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Communicable Diseases Intelligence

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Editor Dr I.F. Cook

Contents:

VIRUS REPORTING SCHEME: A total of 1 359 reports were processed for this period.

Herpes simplex virus was isolated from the post mortem specimens of tissues derived from the lungs of a 19 year old female who died from a severe respiratory tract infection following bone marrow transplantation.

Echovirus type 11 was isolated from:-

- The urine of a 5 month old female who presented with bulging fontanelle and enlargement of the head following a one week history of febrile episodes; and
- the nasal aspirate of a 2 month old male infant who was identified as at high risk and subsequently monitored for Sudden Infant Death Syndrome.

In early April, the Commonwealth Serum Laboratories reported an unusual, limited outbreak of 14 cases of influenza type B infection in metropolitan Melbourne. Viral isolates, obtained from throat washings, bore antigenic resemblance to B/VIC/3/85 most closely and appeared to be antigenically distinct from B/USSR/100/83. No further cases have been reported since the outbreak and it is expected that the B/Ann Arbor/1/86-like antigenic component of the current 1987 influenza vaccine remains antigenically relevant. Current FLUVAX vaccine should still provide adequate immune protection against type B viruses despite the observed antigenic drift. Antibody studies and antigenic analysis using ferret antisera are continuing.

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Contributions are solicited, and do not preclude later publication elsewhere.

Figures given may be subject to revision.

An epidemic polyarthritis outbreak has been reported in South East Queensland for the summer months of 1986-87. A comparison of the monthly incidence for the first quarter of this year with that of last year indicates a substantial increase in Ross River virus infections:-

	January	February	March
1985	5	20	25
1986	67	170	138

This limited outbreak is due to a transient mosquito population increase facilitated by the severe floodings in that part of the country during the summer of 1986-87. (Dr Wyatt - personal communication).

CDI Editorial Note:

Epidemic polyarthritis is a notifiable disease in all States and Territories of Australia. Reports should be made in accordance with the procedures established by the States/local health authorities.

DIARRHOEA EPIDEMIC - MALDIVES ISLAND

Intending visitors to the Maldives Islands are advised that, due to recent tidal floodings , a diarrhoeal epidemic has been reported in Male (main town of the Island of Male Atoll).

The epidemic is currently restricted to Male, and no resort islands have been reported to be affected at present.

Local health authorities have identified a few cases of cholera and shigella infections. Health measures and precautions, including mass vaccination of Male residents, are being undertaken to prevent the spread of the epidemic to other islands.

The World Health Organisation is investigating the outbreak and its epidemiology. The CDI is monitoring the situation and will keep readers posted of future developments.

HUMAN RABIES : ex INDIA

(Based on CDR 87/10, 13 March 1987)

In mid-March of this year, an 8 year old male was admitted to a hospital intensive care unit in England with a clinical diagnosis of rabies. The patient had been bitten on the ankle by a dog two months previously while visiting relatives in India. He returned to the UK three weeks following the bite and remained well until four days before the hospital admission. Symptoms developed over the next few days and on the day before hospital admission he attended the casualty department of a local hospital and transferred a few hours later. Symptoms including hydrophobia led to the diagnosis and the patient remained under sedation on a ventilator. Health

care staff treating the patient and other close contacts have been offered vaccination.

This is the nineteenth case of human rabies reported since the disease was eradicated from the domestic dog population of the UK in 1902. Thirteen of these cases were infected in the Indian sub-continent where rabies is common in the semi-domesticated dog population.

CDI Editorial Comment:

Rabies is prevalent in animal reservoirs in Europe, North and South America, Africa and Asia. Human rabies characteristically follows a bite or scratch from a rabid animal, but infection from an aerosol has been reported following exposure in bat caves and in a laboratory; and four cases have resulted from corneal transplants, after the donors died from undiagnosed rabies. Vaccination of humans is limited to those considered to be at special risk, or those who have been exposed to infection.

Rabies Immunising Products

There are two types of immunising products; (i) a vaccine that induces an active immune response, and (ii) immunoglobulins that provide rapid passive immune protection, which persist for a short period of time. both types of product should be used concurrently for rabies post-exposure prophylaxis.

Human diploid cell rabies vaccine (HDCV)(Merieux Inactivated Rabies Vaccine) is an inactivated virus prepared from fixed rabies virus grown in human diploid cell culture. The vaccine is inactivated with B-propiolactone. Vaccine is supplied as 1.0mL single-dose vials of lyophilised vaccine with accompanying diluent.

Rabies immunoglobulin, human (RIG)(Cutter Laboratories' HYPERAB) is antirabies immunoglobulin concentrated by cold ethanol fractionation from plasma of hyperimmunised human donors. Rabies neutralising antibody content is standardised to contain 150 international units (IU) per mL. It is supplied in 10mL (1500 IU) vials.

Merieux Inactivated Rabies Vaccine and Rabies Immune Globulin are available in Australia from the Commonwealth Serum Laboratories (CSL) for post-exposure prophylaxis. They are issued free of charge to patients for post-exposure treatment on an authority from the Commonwealth Department of Health in Canberra, or in the appropriate State capital city, or from the Director of the Commonwealth Serum Laboratories in Melbourne. However, for pre-exposure prophylaxis the costs have to be met by either the individual or his or her employer. Vaccine for pre-exposure prophylaxis may be obtained from May & Baker Australia Pty Ltd.

Recommendations for use

Pre-exposure prophylaxis

Pre-exposure prophylaxis should be considered for veterinarians, agricultural advisers and laboratory workers who

may be exposed to contact with the virus in potentially rabid animals or laboratory specimens. This is particularly relevant to those working in South East Asia where the incidence of rabies is extremely high. It is not recommended for travellers who would have extremely limited chances of such exposure.

Pre-exposure prophylaxis involves three doses of vaccine, the second given seven days after the first and the third 30 days after the first injection. Prolongation of the intervals between doses does not impair the immune response.

Booster doses of vaccine

Persons who work with live rabies virus in research laboratories are at risk of inapparent exposure and should have the rabies antibody titre of their serum determined every six months; booster doses of vaccine should be given, as needed, to maintain an adequate titre. Other laboratory workers, such as those doing rabies diagnostic tests, and those veterinarians, animal control and wildlife officers in areas where animal rabies is epizootic, should have boosters every two years or have their serum tested for rabies antibody every two years and, if the titre is inadequate, have a booster dose. Veterinarians and animal control and wildlife officers, if working in areas of low rabies endemicity, do not require routine booster doses of human diploid cell rabies vaccine after completion of primary pre-exposure immunisation.

Post-exposure prophylaxis

The essential components of rabies post-exposure prophylaxis are local treatment of wounds and immunisation, including administration, in most instances, of both globulin and vaccine.

(i) Local treatment of wounds

Immediate and thorough washing of all bite wounds and scratches with soap and water is perhaps the most effective measure for preventing rabies. In experimental animals, simple local wound cleansing has been shown to reduce markedly the likelihood of rabies.

(ii) Immunisation

Post-exposure antirabies immunisation should always include administration of both antibody (preferably rabies immunoglobulin) and vaccine, with one exception: persons who have been previously immunised with the recommended pre-exposure or post-exposure regimens with human diploid cell rabies vaccine or who have been immunised with other types of vaccines and have a history of documented adequate rabies antibody titre should receive only vaccine. The sooner treatment is begun after exposure, the better. However, there have been instances in which the decision to begin treatment was made as late as six months or longer after the exposure due to delay in recognition that an exposure had occurred.

Post-exposure treatment involves six doses of vaccine on days 0, 3, 7, 14, 30 and 90. Human rabies immunoglobulin should be given once at a dose of 20 IU/kg when the first dose of vaccine is administered.

(iii) Post-exposure therapy of previously immunised persons

Rabies post-exposure treatment in a person who has previously:

. completed the recommended rabies vaccination regimen with human diploid cell rabies vaccine; or
. demonstrated rabies antibody,

consists of two intramuscular doses of human diploid cell rabies vaccine (1.0mL) one given immediately and the second three days later. Rabies immunoglobulin should not be given in these cases.

In cases where the immune status is uncertain because the patient did not receive the recommended human diploid cell rabies vaccine, full primary post-exposure antirabies treatment (rabies immunoglobulin plus six doses of human diploid cell rabies vaccine) should be considered. In these cases, if rabies antibody can be demonstrated in a serum sample collected before administration of the vaccines, two dose treatment as above is appropriate.

Precautions and Contraindications

General precautions

Human diploid cell rabies vaccine should not be administered intravenously or intradermally because the antibody response has been shown to be less predictable after these methods of administration.

Allergies

Persons with histories of hypersensitivity should be given rabies vaccine with caution. When a patient with a history suggesting hypersensitivity to human diploid cell rabies vaccine must be given that vaccine, antihistamines can be given; adrenaline should be readily available to counteract anaphylactic reactions, and the person should be carefully observed.

Use of steroids and immunosuppressive agents

Corticosteroids and immunosuppressive agents can interfere with the development of active immunity and pre-dispose the patient to developing rabies. They should not be administered during post-exposure therapy unless essential for the treatment of other conditions.

Pregnancy

Because of the potential consequences of inadequately treated rabies exposure and limited data that indicate that fetus abnormalities have not been associated with rabies vaccination, pregnancy is not considered a contraindication to post-exposure prophylaxis. If there is substantial risk of exposure to rabies, pre-exposure prophylaxis may also be indicated during pregnancy.

Summary of recommended prophylaxis schedule

	Rabies immunoglobulin (given)	Human diploid cell rabies vaccine (1 mL on days)
Pre-exposure	No	0, 7, 30
Post-exposure	Yes	0, 3, 7, 14, 30, 90

Side effects and adverse reactions

Human diploid cell rabies vaccine (HDCV)

Reactions after vaccination with human diploid cell rabies vaccine are less common than with previously available vaccines. In a study using five doses of human diploid cell rabies vaccine, local reactions, such as pain, erythema, and swelling or itching at the injection site, were reported in about 25 per cent of recipients of human diploid cell rabies vaccine, and mild systemic reactions, such as headache, nausea, abdominal pain, muscle aches, and dizziness, were reported in about 20 per cent of recipients. Two cases of neurological illness resembling Guillain-Barre syndrome that resolved without sequelae in 12 weeks, and a focal subacute central nervous system disorder temporally associated with human diploid cell rabies vaccine, have been reported.

Recently, a significant increase has been noted in 'immune complex-like' reactions in persons receiving booster doses of human diploid cell rabies vaccine. The illness, characterised by onset 2-21 days post-booster, presents with a generalised urticaria and may also include arthralgia, arthritis, angioedema, nausea, vomiting, fever, and malaise. In no cases were the illnesses life-threatening. Preliminary data suggests this 'immune complex-like' illness may occur in up to six percent of persons receiving booster vaccines and much less frequently in persons receiving primary immunisation. Additional experience with this vaccine is needed to define more clearly the risk of these adverse reactions.

Rabies immunoglobulin, human

Local pain and low-grade fever may follow receipt of rabies immunoglobulin. Although not reported specifically for rabies immunoglobulin, angioneurotic oedema, nephrotic syndrome, and anaphylaxis have been reported after injection of immune serum globulin (ISG). These reactions occur so rarely that the causal relationship between ISG and these reactions is not clear.

Management of adverse reactions

Once initiated, rabies prophylaxis should not be interrupted or discontinued because of local or mild systemic adverse reactions to rabies vaccine. Usually such reactions can be successfully managed with anti-pyretic agents such as aspirin.

When a person with a history of hypersensitivity must be given rabies vaccines, antihistamines may be given; adrenaline should be readily available to counteract anaphylactic reactions, and the persons should be carefully observed immediately after immunisation.

Serious systemic anaphylactic or neuroparalytic reactions occurring during the administration of rabies vaccines pose a serious dilemma for the attending physician. A patient's risk of developing rabies must be carefully considered before deciding to discontinue vaccination. Moreover, the use of corticosteroids to treat life-threatening neuroparalytic reactions carries the risk of inhibiting the developing of active immunity to rabies.

Q FEVER: PERSON TO PERSON TRANSMISSION WITHIN A FAMILY

(Based on Thorax 1986; 41: 974-75)

Q fever infection, caused by a rickettsia like organism, Coxiella burnetii accounts for 1% of all hospital admissions with pneumonia in Great Britain[1].

This organism most commonly affects the genital tract of wild and domestic animals, including cows, sheep and goats, but rarely causes disease. Infection in man occurs usually by inhalation of the organism following exposure to domestic animal carriers, by contact with dust or straw, or by drinking unpasteurised milk contaminated with the organism[2]. Infection arising from a contaminated blood transfusion has been reported to occur rarely.[5].

After an incubation period of 14-28 days patients may develop an acute illness with chills and sweating associated with radiological evidence of pulmonary consolidation [2]. Raised complement fixing antibody titres to phase II antigen may provide evidence of acute infection. High titres to phase I antigen are found in chronic infections such as endocarditis.

Person to person transmission of C. burnetii is rare[3,4]. In one case, staff in a hospital were infected by a patient[4] and in the other, pathologists and a mortuary technician were infected during a postmortem examination.

Case report (figure)

. A 79 year old man in Britain (index case - house 2 - case 5) was admitted with a 24 hour history of sharp chest pain, breathlessness, vomiting, and urinary incontinence. Before this illness the patient had been largely confined to his home as a result of Parkinson's disease and a previous stroke. On examination he was febrile and tachypnoeic and a few basal inspiratory crackles were audible on auscultation. The chest radiograph showed no abnormality on admission to hospital but subsequent films showed the development of right upper lobe consolidation. He was treated with erythromycin 500 mg four times daily, after which his condition improved and the chest radiographic appearances returned to normal. A diagnosis of recent Q fever infection was subsequently made

when complement fixation testing indicated C. burnetii antibody titres of less than 16 for phase I and 512 for phase II.

. five weeks before his admission the patient's wife (case 4) had been treated at home (house 2) for pneumonia. Throughout her illness she had noted symptoms of dyspnoea, chest tightness, and confusion. She was subsequently found to have serological evidence of recent Q fever infection, with a phase I titre of 16 and a phase II titre of 512 18 weeks after her illness. Neither of these two patients had had any contact with animals.

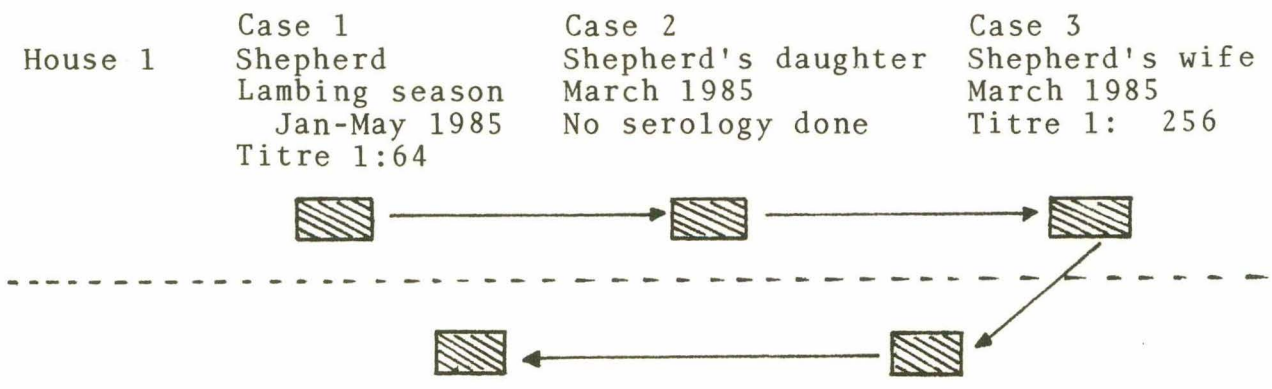
. subsequent inquiry revealed that:

- the patient's married daughter (case 3), who lived six miles away (house 1), had had a similar illness seven weeks before that of her mother; 29 weeks after this illness she had serological evidence of recent C. burnetii infection, with a phase II titre greater than 256.

- the patient's grand daughter (case 2), who lived with her parents, had suffered a similar illness a week before her mother, but no blood specimen could be obtained for serological testing.

- the son in law (case 1), who had remained asymptomatic, had evidence of recent C. burnetii infection with a phase II titre of 64. He was employed as a shepherd at a local veterinary station where lambing was in progress during the period when his wife and daughter had become ill. It is pertinent that he worked in overalls that were always washed at home and that he changed before visiting his wife's parents' house. Sick animals were never brought to the house and none of the family drank unpasteurised milk.

Figure: Proposed disease transmission



DISCUSSION

The son in law (case 1) was probably infected during the lambing season, since the organism may be shed in large numbers

at parturition. Infection has been reported after contact with contaminated clothing[6], and as the shepherd went home in his working clothes, infection of his daughter (case 2) and wife (case 3) may have occurred by this route.

Alternatively, however, the shepherd may have transmitted the organism himself since on the evidence of a raised antibody titre he had an asymptomatic infection with the organism.

Transfer of the organism on clothing could not account for the subsequent infection of the shepherd's relatives who lived several miles away, as he invariably changed from his working clothes before visiting them. In addition, the time course of the illness is suggestive of sequential spread of infection. No other cases of Q fever in the vicinity of cases 4 and 5 have been reported during the time of their illnesses and no sera positive for C. burnetii was received by the regional virus laboratory from the area around the household during the first six months of 1985. The homes of the families were situated in semirural areas not associated with a high density of domestic animals, making infection from another animal source unlikely.

This report appears to provide the first example of person to person transmission of C. burnetii in a family. It also emphasises the need to consider this organism as a cause of pneumonia in what may initially appear to be the unlikeliest of cases.

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2. Infectious diseases, epidemiology, and clinical practice edited by Christie AB (1980): 800-12
3. Lancet (1949) ii: 1028-30
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5. Principles and Practice of Infectious Diseases edited by Mandell GL, Douglas RG, Bennett JE (1979) 1516-20.
6. Bull World Health Org (1955) 13: 781-806.

PENICILLINASE - PRODUCING NEISSERIA GONORRHOEAE - UNITED STATES, 1986

(Based on MMWR Vol. 36 No 8, 6 March 1987)

In 1986, 16 608 cases of infection caused by penicillinase-producing Neisseria gonorrhoeae (PPNG) were reported to Centers for Disease Control (CDC). This accounted for 1.8% of all reported gonorrhoea and represented a 90% increase over the 8 724 cases reported in 1985. PPNG incidence has risen fourfold since 1984. Sixty-four percent of cases in 1986 occurred in the three areas previously identified as hyperendemic: Florida, New York and Los Angeles.

New York City experienced the greatest proportional increase of PPNG incidence despite its policy of treating all patients diagnosed with gonorrhoeae in the public clinics with antimicrobials effective against PPNG. In 1986, 3 986 cases were reported, compared with the 1,567 cases reported in 1985 - a 154% increase. The proportion of total gonorrhoeae attributable to PPNG was 43%. Outbreaks have been identified

in suburban areas of New York City located on Long Island and in New Jersey and Westchester County.

In Florida, 5 629 PPNG cases were reported - 34% of the national total. In Dade County (Miami), Florida, the most severely affected county in the country, reported cases of PPNG increased from 2 455 in 1985 to 2 648 in 1986 - an 8% increase. In 1986, the proportion of gonorrhoea attributable to PPNG in Dade County was 22%. Excluding Dade County, reported cases in Florida increased from 1 710 in 1985 to 2 981 in 1986 - a 74% increase. The number of counties in Flordia reporting hyperendemic PPNG (a proportion of PPNG) (73%) rose from 16 counties in 1985 to 31 counties in 1986. These counties account for 69% of the State's population.

In Los Angeles, the number of cases increased from 488 in 1985 to 942 in 1986 - a 93% increase. Another centre of PPNG activity probably representing secondary spread, has also been identified in suburban Orange County.

Editorial Note:

The incidence of antibiotic-resistant gonorrhoea, and PPNG in particular, continues to increase and is spreading to previously unaffected areas. In earlier PPNG outbreaks, travel to PPNG endemic areas and prostitute contact were cited as risk factors for infection. While these factors may play an important role in the spread of PPNG disease to areas previously free of disease, once PPNG becomes endemic, it has the same epidemiologic characteristics as endemic, antibiotic-sensitive gonorrhoea. PPNG patients have been predominantly inner-city residents, members of ethnic minority groups, and heterosexuals. Although high-risk groups for gonorrhoea have included homosexual men, PPNG outbreaks among homosexual men are rare. The reasons for this are not entirely clear. Recent evidence from a CDC study in Miami has associated PPNG infection with inappropriate use of antibiotics.

Patients with inadequately treated PPNG infection are at high risk for complications. Women are especially at high risk for pelvic inflammatory disease (PID) and subsequent infertility. PPNG is affectively treated with ceftriaxone or spectinomycin, in doses recommended below:

1. Uncomplicated Genital Infections
   . Adults : spectinomycin 2.0g IM as a single injection
   . Children : spectinomycin 40mg/kg IM
2. Anal and pharyngeal infections : ceftriaxone, 350mg
3. Neonatal ocular infections : spectinomycin 40mg/Kg daily for 3 days

   (an ophthalmologist's opinion should also be sought).

Once antibiotic-resistant gonorrhoea becomes endemic, eradication is extremely difficult; it is also expensive. In these areas, all patients with a presumptive diagnosis of gonorrhoea should be treated with ceftriaxone or spectinomycin. Comprehensive recommendations for prevention, surveillance, diagnosis, and control of antibiotic-resistant gonorrhoea have been recently developed by CDC in consultation with an expert advisory panel and are currently being reviewed by state and local health officials.

RAPIDLY PROGRESSIVE DEMENTIA IN A PATIENT WHO RECEIVED A CADAVERIC DURA MATER GRAFT
(Based on MMWR Vol 36/No 4, 6 February 1987)

In mid November 1986, a 28 year old woman developed gait ataxia 19 months after surgical resection of a cholesteatoma (a cyst-like mass with a lining of stratified squamous epithelium, occuring most commonly in the middle ear or mastoid region).

During surgery she received an imported, commercially prepared human dura mater graft (LYODURA, Lot # 2105, processed in 1982 by B. Braun Melsungen AG of the Federal Republic of Germany). By early December, she required assistance with ambulation and had developed dysarthria. Two weeks later she gave inappropriate responses to questions and developed visual hallucinations. By early January 1987, she developed monoclonic jerks and on physical examination was demented. Diagnosis of Creutzfeldt - Jakob disease (CJD) was confirmed by brain biopsy which demonstrated spongiform encephalopathy. She had no family history of degenerative neurologic disease, nor had she received cadaveric, pituitary-derived human growth hormone (hGH). No patient with known CJD had surgery in the same neurological suite in the 3 months prior to this woman's operation.

MMWR Editorial Note:

CJD occurs with a frequency of about 1 in 1,000,000 population per year in the United States and in various populations worldwide. Most cases occur spontaneously in patients over 50 years of age; CJD is rare in persons under 30 years of age.

Iatrogenic transmission of CJD has occurred in one patient by corneal transplant from an infected donor, in two patients who were exposed to intracerebral electrodes after they had been used in a CJD patient, in four patients in neurological suite following procedures on CJD patients and in four recipients of hGH. Onsets of symptoms following direct brain or eye exposure to the CJD agent have ranged from 16 to 28 months; however, patients who received systemic hGH have had onsets of symptoms after 4 to 21 years. No other reports of CJD transmission via dura mater grafts have been identified.

Dura mater harvested from cadavers is used predominantly in neurosurgical procedures, but is also used in orthopaedic otologic, dental, urologic, gynaecologic, and cardiac procedures. Although the number of recipients of LYODURA and other dura mater grafts is not well known, the age of this patient and the 19 month interval between her graft and onset of symptoms of CJD strongly suggest that the dural graft was the vehicle for transmission of the CJD agent. The Food and Drug Administration and the Centers for Disease Control are continuing to investigate the association.

Procedures used to sterilise cadaveric dura such as exposure to ethylene oxide or ionising irradiation are not sufficient to completely inactivate the CJD agent. Until methods to eliminate the CJD agent from dura mater can be better defined, the transmission of this lethal, degenerative neurological disease remains a possibility. Surgeons may wish to consider the alternative use of autologous fascia lata or temporalis

fascia or of synthetic substitutes. Physicians who use cadaveric dura mater should verify that their sources follow stringent donor selection procedures and criteria such as those promulgated by the American Association of Tissue Banks.

The Centers for Disease Control is currently requesting that:-

- previous and current patients who have rapidly progressive dementing illness consistent with CJD and who have received a dural graft during an operative procedure be reported to respective State Health Department; and

- any medical facility finding remaining stock of LYODURA, Lot # 2105, seeks immediate advice regarding its disposition and possible testing.

CDI Editorial Comment:

The safety of natural growth hormone, following the reported deaths from CJD of three US patients who had received NIH-produced product in the 1960s and 1970s, has prompted the regulatory authorities in Australia, Belgium, Finland, Greece, the Netherlands, Sweden, the UK, Canada, West Germany and the USA to suspend the distribution of natural growth hormone (hGH).

Since the suspension of natural hGH in Australia in 1985, a synthetic growth hormone, available as SOMATONORM, has been distributed by Kabi Vitrum via Pharmacia to existing hGH recipients under a clinical trial design protocol, as no general marketing approval has been granted for the product.

Many countries including Australia are carrying out studies to determine whether the product should be allowed back on the market. Completed retrospective epidemiologic studies conducted on 6000 recipients of natural hGH in nine European countries, as well as Japan, Argentina and Israel, had failed to establish a causal link between death from CJD and natural hGH. However, biochemical studies to date have yet to produce conculsive evidence.

MENINGITIS ON AN AIRCRAFT - CANADA

(Based on 'Epi Notes' Vol. 11, No 1, 23 February 1987)

On 1 February 1987, a 21 year old male with meningococcal meningitis, was admitted to the intensive care unit of the Alberta University Hospital, 18 hours after arriving in Edmonton by plane from New Delhi, India (via Frankfurt, Germany).

Following this case detection a follow up plan was initiated by the health authority to identify all persons considered to be possible contacts of the index case. When seating details of the flight were reviewed, the airline identified 35 contacts, 16 of which disembarked in Edmonton while the remainder continued to Vancouver. It was recommended that all 35 contacts be offered a prophylactic course of rifampicin. Other passengers were not considered at significant risk of acquiring the infection, but it was deemed prudent to inform them of the

circumstances and to advise them to seek medical attention should they develop any illness in the week following their arrival in Canada.

Existing air travel registration procedures enabled the airline to only provide 4 telephone contacts for 8 of the listed 16 contacts disembarking at Edmonton. The remaining 8 contacts were traced by media assistance and necessitated 24 interviews to be conducted. Subsequently all 16 contacts were traced and rifampicin prophylaxis given. In addition, two passengers who were not identified by the airline but who were known to have moved during the flight to occupy empty seats located in the area considered to present a high exposure risk, were also contacted and offered prophylaxis.

Rifampicin prophylaxis has been extended to the family of the index case, the family with whom the patient had been travelling and four members of the airline flight crew.

A number of passengers contacted medical staff reporting a relatively high frequency of headache, nausea and fatigue on arrival, however all the reported symptoms were attributed to jet lag. Several other passengers developed suspicious symptoms within 3-4 days of arrival, however subsequent investigations did not implicate meningococcal infection. No secondary cases occurred in Alberta or British Columbia.

CDI Editorial Comment:

In March 1987, CDI 87/5 reported a similar case of meningococcal septicaemia in a 25 year old male returning to Australia from a backpacking holiday in Nepal. The patient had been ill in flight, became comatose soon after hospital admission following airport disembarkation and despite intravenous antibiotic treatment, died a fortnight later without regaining consciousness.

In this instance, contact tracing had not been difficult since the airline carrier was able to:

i) define high risk seating arrangements, and

ii) identify at risk passengers who had been successfully traced through a system of Incoming Passenger Cards which registered contact addresses for all travellers arriving in Australia.

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 20-4-87 to 3-5-87 BULLETIN NUMBER 87/9
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	11		3		2	2	9	2	29
0101 ADENOVIRUS TYPE 1				2					2
0103 ADENOVIRUS TYPE 3	1			2				3	6
0107 ADENOVIRUS TYPE 7	1								1
0110 ADENOVIRUS TYPE 10	1								1
0112 ADENOVIRUS TYPE 12			1						1
0119 ADENOVIRUS TYPE 19								1	1
0137 ADENOVIRUS TYPE 37								2	2
0199 ADENOVIRUS TYPING PENDING			8		2				10
0201 INFLUENZA A VIRUS	1		3						4
0301 PARAINFLUENZA VIRUS TYPE 1					2		4		6
0302 PARAINFLUENZA VIRUS TYPE 2	1		1					3	5
0303 PARAINFLUENZA VIRUS TYPE 3					1	1			2
0399 PARAINFLUENZA VIRUS TYPING PENDING					2	1			3
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)	9	1	1	1	1	5	1	13	32
0500 RHINOVIRUS (ALL TYPES)				4	8	9			21
0600 MYCOPLASMA PNEUMONIAE	8		3		1	3		2	17
0901 COXSACKIEVIRUS B1				1					1
0903 COXSACKIEVIRUS B3						2			2
1000 ECHOVIRUS NOT TYPED						1			1
1005 ECHOVIRUS TYPE 5	3		3	3					9
1009 ECHOVIRUS TYPE 9	1								1
1011 ECHOVIRUS TYPE 11	4	1	1	3					9
1020 ECHOVIRUS TYPE 20						1			1
1100 POLIOVIRUS NOT TYPED			4						4
1101 POLIOVIRUS TYPE 1	3								3
1102 POLIOVIRUS TYPE 2	2			1		1		1	5
1103 POLIOVIRUS TYPE 3	1							1	2
1200 MUMPS VIRUS	1							2	3
1300 HERPES VIRUS GROUP-NOT TYPED	21							5	26
1301 HERPES SIMPLEX VIRUS NOT-TYPED		3					1	1	5
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	4		5		2	2		9	22
1303 VARICELLA-ZOSTER VIRUS	1		2			1		1	5
1306 HERPES SIMPLEX TYPE 1	29		10	33		15	34	5	126
1307 HERPES SIMPLEX TYPE 2	78		27	79		10	82	24	300
1399 HERPES VIRUS TYPING PENDING				2	1			3	6
1401 COXIELLA BURNETI	2					1			3
1502 PICORNA VIRUS-NOT TYPED			27				16	1	44
1521 MEASLES VIRUS						1			1
1522 RUBELLA VIRUS				1			2		3
1532 HEPATITIS B ANTIGEN	66	2	13	14	2	24	13	26	160
1535 HEPATITIS A ANTIBODY	3		5	1		4		2	15
1541 CHLAMYDIA A - C TRACHOMATIS	40		8			55	20	50	173
1555 PAPOVAVIRUS GROUP (PAPILLOMA-HUMAN WART)	2								2
1556 CMV - CYTOMEGALOVIRUS	5		8	43	4	3	15	14	92
1564 ROTAVIRUS	15	5	2		2	16			40
1599 ENTEROVIRUS TYPING PENDING		4	20		4				28
9992 ROSS RIVER VIRUS			2			2	115	4	123
9994 SMALL VIRUS (LIKE) PARTICLE		1							1
Total	314	17	157	190	34	160	312	175	1,359

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 20-4-87 to 3-5-87 BULLETIN NO 87/9

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.....		2				1					
0103 ADENOVIRUS TYPE 3.....	1	3									
0110 ADENOVIRUS TYPE 10.....							1				
0112 ADENOVIRUS TYPE 12.....							1				
0119 ADENOVIRUS TYPE 19.....	1										
0199 ADENOVIRUS TYPING PENDING.....							1				
0201 INFLUENZA A VIRUS.....	1	2									
0301 PARAINFLUENZA VIRUS TYPE 1....		6									
0302 PARAINFLUENZA VIRUS TYPE 2....	1	2									
0303 PARAINFLUENZA VIRUS TYPE 3....		1									
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....	4	27									1
0500 RHINOVIRUS (ALL TYPES).....		19					1				
0600 MYCOPLASMA PNEUMONIAE.....	3	11					1				1
0903 COXSACKIEVIRUS B3.....	1	1									
1005 ECHOVIRUS TYPE 5.....	1	3		5							
1009 ECHOVIRUS TYPE 9.....	1										
1011 ECHOVIRUS TYPE 11.....				3		2	2				
1101 POLIOVIRUS TYPE 1.....	1						2				
1102 POLIOVIRUS TYPE 2.....	2										
1103 POLIOVIRUS TYPE 3.....							1				
1200 MUMPS VIRUS.....			2								
1300 HERPES VIRUS GROUP-NOT TYPED..	2										7
1301 HERPES SIMPLEX VIRUS NOT-TYPED	1	1									2
1302 EPSTEIN-BARR VIRUS (EB VIRUS).	8	1						1			
1303 VARICELLA-ZOSTER VIRUS.....	1					1					2
1306 HERPES SIMPLEX TYPE 1.....	8	5	1							2	53
1307 HERPES SIMPLEX TYPE 2.....	12										80
1401 COXIELLA BURNETI.....	1										
1502 PICORNA VIRUS-NOT TYPED.....							1				
1522 RUBELLA VIRUS.....											1
1532 HEPATITIS B ANTIGEN.....	44						1	99			1
1535 HEPATITIS A ANTIBODY.....	3							9			
1541 CHLAMYDIA A - C.TRACHOMATIS...	16										16
1555 PAPOVAVIRUS GROUP (PAPILLOMA-HUMAN WART).....											2
1556 CMV - CYTOMEGALOVIRUS.....	4	19	1	1		2	2	1		4	1
1564 ROTAVIRUS.....							40				
9992 ROSS RIVER VIRUS.....	29	5					1	1			17
9994 SMALL VIRUS (LIKE) PARTICLE...							1				
Total.....	146	108	4	9		6	56	111		6	184

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 20-4-87 to 3-5-87 BULLETIN NUMBER 87/9
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
0103 ADENOVIRUS TYPE 3.....	2							1	1	
0107 ADENOVIRUS TYPE 7.....	1									
0112 ADENOVIRUS TYPE 12.....				1						
0137 ADENOVIRUS TYPE 37.....	2									
0201 INFLUENZA A VIRUS.....								1		
0302 PARAINFLUENZA VIRUS TYPE 2....	1							2	1	
0303 PARAINFLUENZA VIRUS TYPE 3....									1	
0500 RHINOVIRUS (ALL TYPES).....			1					1	2	
0600 MYCOPLASMA PNEUMONIAE.....			1							
0901 COXSACKIEVIRUS B1.....								1		
1011 ECHOVIRUS TYPE 11.....								2	1	
1020 ECHOVIRUS TYPE 20.....									1	
1102 POLIOVIRUS TYPE 2.....										3
1103 POLIOVIRUS TYPE 3.....										1
1200 MUMPS VIRUS.....			1							
1301 HERPES SIMPLEX VIRUS NOT-TYPED									1	
1302 EPSTEIN-BARR VIRUS (EB VIRUS).			6					3	3	
1303 VARICELLA-ZOSTER VIRUS.....							1			
1306 HERPES SIMPLEX TYPE 1.....	1	53					1	2	3	
1307 HERPES SIMPLEX TYPE 2.....	2	206								
1401 COXIELLA BURNETI.....							2			
1521 MEASLES VIRUS.....										1
1522 RUBELLA VIRUS.....					2	1		1		
1532 HEPATITIS B ANTIGEN.....					1			1	16	
1535 HEPATITIS A ANTIBODY.....									3	
1541 CHLAMYDIA A - C.TRACHOMATIS...	4	137							1	
1556 CMV - CYTOMEGALOVIRUS.....		6			2	8	6	10	34	1
1599 ENTEROVIRUS TYPING PENDING....									1	
9992 ROSS RIVER VIRUS.....					70			34	1	
Total.....	13	402	9	1	75	9	10	59	70	6