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MORBIDITY AND MORTALITY WEEKLY REPORT

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Epidemiologic Notes and Reports

Update: Human Immunodeficiency Virus Infections in Health-Care Workers Exposed to Blood of Infected Patients

Six persons who provided health care to patients with human immunodeficiency virus (HIV) infection and who denied other risk factors have previously been reported to have HIV infection. Four of these cases followed needle-stick exposures to blood from patients infected with HIV (1-4). The two additional cases involved persons who provided nursing care to persons with HIV infection. Although neither of these two persons sustained needle-stick injuries, both had extensive contact with blood or body fluids of the infected patient, and neither observed routinely recommended barrier precautions (5,6).

CDC has received reports of HIV infection in three additional health-care workers following non-needle-stick exposures to blood from infected patients. The exposures occurred during 1986 in three different geographic areas. Although these three cases represent rare events, they reemphasize the need for health-care workers to adhere rigorously to existing infection control recommendations for minimizing the risk of exposure to blood and body fluids of all patients (7-9).

Health-Care Worker 1: A female health-care worker assisting with an unsuccessful attempt to insert an arterial catheter in a patient suffering a cardiac arrest in an emergency room applied pressure to the insertion site to stop the bleeding. During the procedure, she may have had a small amount of blood on her index finger for about 20 minutes before washing her hands. Afterwards, she may also have assisted in cleaning the room but did not recall any other exposures to the patient's blood or body fluids. She had no open wounds, but her hands were chapped. Although she often wore gloves when anticipating exposure to blood, she was not wearing gloves during this incident.

The patient with the cardiac arrest died. A postmortem examination identified *Pneumocystis carinii* pneumonia, and a blood sample was positive for HIV antibody by enzyme immunoassay (EIA) and Western blot methods. Twenty days after the incident, the health-care worker became ill with fever, myalgia, extreme fatigue, sore throat, nausea, vomiting, diarrhea, a 14-pound weight loss, and generalized lymphadenopathy which her physician diagnosed as a viral syndrome. That illness lasted 3 weeks. She felt much better 9 weeks after the incident, and, when she was examined 6 months after the incident, all signs and symptoms had resolved. She had donated blood 8 months before the incident and was negative for HIV antibody by EIA. She donated again 16 weeks after the incident and was positive for HIV by EIA and Western blot (bands p24 and gp41). Serum samples obtained 20 and 23 weeks after the incident were also positive for HIV antibody. She stated that for over 8 years her only sexual partner had been her husband, who denied risk factors for HIV and was seronegative for HIV

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antibody. She denied ever receiving a blood transfusion, ever using intravenous drugs, or having any needle sticks or other significant exposures to blood or body fluids in the past 8 years. Her serologic test for syphilis was negative. Fifteen other employees who assisted in the care of the patient were seronegative at least 4 months after the exposure.

Health-Care Worker 2: A female phlebotomist was filling a 10 ml vacuum blood collection tube with blood from an outpatient with a suspected HIV infection when the top of the tube flew off and blood splattered around the room, on her face, and in her mouth. She was wearing gloves to protect her hands and was wearing eyeglasses so she did not think she got any blood in her eyes. She had facial acne but no open wounds. She washed the blood off immediately after the exposure. The outpatient's blood sample was positive for HIV antibody by EIA and Western blot, and a hepatitis B surface antigen test was negative. The phlebotomist's EIA was negative the day after the incident and again 8 weeks later. When she donated blood 9 months after the exposure, she was positive for HIV antibody by EIA and Western blot (bands p24 and gp41). She has had no symptoms. She denied having any sexual contact during the previous 2 years, ever using drugs intravenously, or ever receiving a transfusion. Two months after the incident, she scratched the back of her hand with a needle used to draw blood from an intravenous drug abuser of unknown HIV-antibody status. She did not bleed as a result of the scratch and has not had any needle-stick injuries in over 2 years. Her serologic tests for syphilis and hepatitis B were negative. A coworker who was splattered with blood on the face and in the mouth during the same incident remains seronegative 1 year after the incident.

Health-Care Worker 3: A female medical technologist was manipulating an apheresis machine (a device to separate blood components) to correct a problem that developed during an outpatient procedure when blood spilled, covering most of her hands and forearms. She was not wearing gloves. She does not recall having any open wounds on her hands or any mucousmembrane exposure. However, she had dermatitis on one ear and may have touched it. She washed the blood off herself and the machine several minutes after the spill. The patient undergoing the apheresis had denied risk factors for HIV infection, However, a blood sample from the patient was positive for HIV antibody by EIA and Western blot methods and negative for hepatitis B surface antigen the next day. The technologist's HIV-antibody tests were negative 5 days after the exposure and again 6 weeks later. Eight weeks after the exposure, she had an influenza-like illness with fever, myalgia, diarrhea, hives, and a pruritic red macular rash on her arms and legs. The illness resolved after a few weeks, and her physician thought the illness was probably a viral syndrome. Three months after the incident, she was positive for HIV antibody by EIA and Western blot methods (band p24 alone). Four months after the incident, a Western blot was positive (bands p24 and gp41). She indicated that for more than 8 years her only sexual partner had been her husband, who denied risk factors for HIV infection and was seronegative for HIV antibody. She denied ever receiving a transfusion, ever using intravenous drugs, or having any needle-stick injuries in over 2 years. Her serologic tests for syphilis and hepatitis B were negative. She has an immunologic disorder which had been treated with corticosteroids in the past, but she had not taken any immunosuppressive medication for the past year. A coworker with a similar exposure during the same procedure remains seronegative after 3 months.

Reported by: Hospital Infections Program and AIDS Program, Center for Infectious Diseases, CDC.

Editorial Note: Three instances of health-care workers with HIV infections associated with skin or mucous-membrane exposure to blood from HIV-infected patients are reported above. Careful investigation of these three cases did not identify other risk factors for HIV infection, although unrecognized or forgotten needle-stick exposures to other infected patients cannot be totally excluded. The exact route of transmission in these three cases is not known. Health-

Care Worker 1 had chapped hands, and the duration of contact with the blood of the patient experiencing a cardiac arrest may have been as long as 20 minutes. Health-Care Worker 2 sustained contamination of oral mucous membranes. This individual also had acne but did not recall having open lesions. In addition, she had sustained a scratch from a needle used to draw blood from an intravenous drug abuser of unknown HIV-infection status. Health-Care Worker 3 had a history of dermatitis involving an ear. Health-Care Workers 1 and 3 were not wearing gloves when direct contact with blood occurred. Health-Care Worker 2 was wearing gloves, but blood contaminated her face and mouth.

Three ongoing prospective studies provide data on the magnitude of the risk of HIV infection incurred when health-care workers are exposed to blood of infected patients through needle-stick wounds or contamination of an open wound or mucous membrane. In a CDC cooperative surveillance project (10), a total of 1,097 health-care workers with parenteral or mucous-membrane exposure to the blood of patients with AIDS or other manifestations of HIV infection had been enrolled as of March 31, 1987. Needle-stick injuries and cuts with sharp objects accounted for 969 (89%) of the exposures to blood; 298 of these had paired serum samples tested for HIV antibody. One (0.3%) seroconverted (2), indicating that the risk of transmission during these exposures is very low. In addition, 70 health-care workers had open wounds exposed to blood, and 58 had mucous membrane exposed to blood. Postexposure serum samples from 82 of these 128 workers have been tested for antibody to HIV; none was seropositive.

In a study at the National Institutes of Health (11) through April 30, 1987, none of the 103 workers with percutaneous exposures and none of the 229 workers with mucous-membrane exposures to blood or body fluids of patients with AIDS was seropositive. At the University of California (12), none of 63 workers with open wounds or mucous membranes exposed to blood or body fluids of patients with AIDS was seropositive. Although the precise risk of transmission during exposures of open wounds or mucous membranes to contaminated blood cannot be defined, these studies indicate that it must be very low.

The three cases reported here suggest that exposure of skin or mucous membranes to contaminated blood may rarely result in transmission of HIV. The magnitude of the risk is not known since data on the frequency with which such exposures occur are not available. Skin and mucous-membrane exposures are thought to occur much more commonly than needle sticks, and the risk associated with skin or mucous-membrane exposures is likely to be far lower than that associated with needle-stick injuries. Nonetheless, the increasing prevalence of HIV infection increases the potential for such exposures, especially when routinely recommended precautions are not followed.

It is unlikely that routine serologic testing for HIV infection of all patients admitted to hospitals would have prevented these exposures since two of the three exposures occurred in the outpatient clinic setting, and one occurred during a resuscitation effort in an emergency room shortly after the arrival of the patient. At the time of exposure, Health-Care Worker 2 suspected that the source patient was infected with HIV, but Health-Care Workers 1 and 3 did not. The hospital where Health-Care Worker 3 was exposed has a protocol for apheresis which normally involves HIV-antibody testing of donors, however, such testing was not done in advance of the procedure. Previous CDC recommendations have emphasized the value of HIV serologic testing for patient diagnosis and management and for prevention and control of HIV transmission (13) and have stated that some hospitals in certain geographic areas may deem it appropriate to initiate serologic testing of patients (7). Such testing may also provide an opportunity to reduce the risk of HIV infection to health-care workers, but it has not been established that knowledge of a patient's serologic status increases the compliance of health-care workers with recommended precautions.

These cases emphasize again the need to implement and strictly enforce previously published recommendations for minimizing the risk of exposure to blood and body fluids of all patients in order to prevent transmission of HIV infection in the workplace and during invasive procedures (7-9).

- 1. As previously recommended, routine precautions must be followed when there is a possibility of exposure to blood or other body fluids. The anticipated exposure may require gloves alone (e.g., when placing an intravascular catheter or handling items soiled with blood or equipment contaminated with blood or other body fluids). Procedures involving more extensive contact with blood or potentially infective body fluids (e.g., some dental or endoscopic procedures or postmortem examinations) may require gloves, gowns, masks, and eye-coverings. Hands and other contaminated skin surfaces should be washed thoroughly and immediately if accidentally contaminated with blood (7). These precautions deserve particular emphasis in emergency care settings in which the risk of blood exposure is increased and the infectious status of the patient is usually unknown (14).
- 2. Previous recommendations have emphasized management of parenteral and mucous-membrane exposures of health-care workers*. In addition, health-care workers who are involved in incidents that result in cutaneous exposures involving large amounts of blood or prolonged contact with blood—especially when the exposed skin is chapped, abraded, or afflicted with dermatitis—should follow these same recommendations. Moreover, serologic testing should be available to all health-care workers who are concerned that they may have been infected with HIV.

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^{&#}x27;If a HCW [health-care worker] has a parenteral (e.g., needlestick or cut) or mucous membrane (e.g., splash to the eye or mouth) exposure to blood or other body fluids, the source patient should be assessed clinically and epidemiologically to determine the likelihood of HTLV-III/LAV [sic] infection. If the assessment suggests that infection may exist, the patient should be informed of the incident and requested to consent to serologic testing for evidence of HTLV-III/LAV (sic) infection. If the source patient has AIDS or other evidence of HTLV-III/LAV (sic) infection, declines testing, or has a positive test, the HCW should be evaluated clinically and serologically for evidence of HTLV-III/LAV [sic] infection as soon as possible after the exposure, and, if seronegative, retested after 6 weeks and on a periodic basis thereafter (e.g., 3, 6, and 12 months following exposure) to determine if transmission has occurred. During this follow-up period, especially the first 6-12 weeks, when most infected persons are expected to seroconvert, exposed HCWs should receive counseling about the risk of infection and follow U.S. Public Health Service (PHS) recommendations for preventing transmission of AIDS (15,16). If the source patient is seronegative and has no other evidence of HTLV-III/LAV [sic] infection, no further follow-up of the HCW is necessary. If the source patient cannot be identified, decisions regarding appropriate follow-up should be individualized based on the type of exposure and the likelihood that the source patient was infected (7).

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Epidemiologic Notes and Reports

B-Virus Infection in Humans — Pensacola, Florida

Between March 28 and April 7, 1987, four persons were admitted to hospitals in Pensacola and Gulf Breeze, Florida, with illnesses that were later confirmed to be caused by infection with B-virus (cercopithecid herpesvirus 1, *Herpesvirus simiae* [1]). Three were monkey handlers with the Naval Aerospace Medical Research Laboratory (NAMRL) at the Pensacola Naval Air Station; the fourth was the wife of one of the three handlers.

Patient 1: On about March 4, a 31-year-old male who had been employed as an animal caretaker for 8 years was bitten on the left thumb by a 3-year-old Rhesus monkey that was suffering from severe bilateral conjunctivitis and diarrhea. The employee had occasionally handled smaller monkeys without protective leather gloves, and it is not certain whether he was wearing gloves when he was bitten. Five days later, he developed numbness in his left arm. Eighteen days after being bitten, he developed lethargy, fever, chills, dizziness, and myalgia. At no time did he have skin lesions suggestive of herpesvirus infection. Over the next 4 days, he developed numbness and paresthesia in the left side of his body, diplopia, and leg weakness. On March 28, he was admitted to the hospital. Two days later, he was placed on intravenous acyclovir. Subsequently, 8-virus antibodies were detected in his serum by enzyme immunoassay (titer = 32). Spinal fluid that was collected before the initiation of acyclovir therapy was positive for 8-virus. The patient continued to deteriorate and was put on a respirator. His therapy was changed to 9-(1,3-dihydroxy-2-propoxymethyl)guanine (DHPG) on a compassionate Investigational New Drug protocol granted by the Food and Drug Administration. He is currently semi-comatose.

B-Virus Infection - Continued

Patient 2: On about March 10, a 37-year-old male who had been employed as a biological technician for 13 years suffered a penetrating wound which may have been a monkey bite or scratch on the left forearm. Patient 2 had had frequent contact with the monkey that injured Patient 1, and his wound may have been inflicted by this animal. Patient 2 had also handled smaller animals without leather gloves, but it is uncertain whether he was wearing them at the time he was exposed. Five days after his injury, he developed herpetiform vesicles at the site of the wound. On March 26, after the lesions had become crusted, he was seen by a dermatologist who detected giant cells in scrapings from the lesions (Tzanck preparation) but no distinct viral inclusions. A presumptive diagnosis of herpes zoster versus herpes simplex was made. Topical acyclovir was prescribed, but the patient treated himself only with topical hydrocortisone cream. Over the next several days, he developed numbness in his left arm, chest pain, dyspnea, fever, confusion, lethargy, diplopia, and dysphagia. He made several visits to emergency rooms before being hospitalized on March 28. Later that day, he suffered a respiratory arrest and was placed on mechanical ventilation. A lumbar puncture was consistent with aseptic meningitis. He was placed on intravenous acyclovir, A skin biopsy specimen obtained the day after admission was positive for B-virus. Treatment was subsequently changed to intravenous DHPG. However, the patient's condition deteriorated, and he died on April 28.

(Continued on page 295)

TABLE I. Summary - cases specified notifiable diseases, United States

	1	9th Week End	ing	Cumulative, 19th Week Ending			
Disease	May 16, 1987	May 10, 1986	Median 1982-1986	May 16. 1987	May 10, 1986	Median 1982-1986	
Acquired Immunodeficiency Syndrome (AIDS)	165	250	N	6.717	4,556	N	
Aseptic meningitis	80	90	84	1,627	1,587	1,495	
Encephalitis: Primary (arthropod-borne				•			
& unspec)	14	16	18	288	287	337	
Post-infectious	2	1 :	. 3	23	38	38	
ionorrhea: Civilian	14,549	14,562	16,999	283,204	301,933	301,933	
Military	343	327	526	6.256	5,582	7,738	
fepatitis: Type Á	451	412	412	8.951	8.032	8.032	
Type 8	461	500	500	9.096	9,184	8.979	
Non A, Non B	60	81	N	1,111	1.260	N	
Unspecified	46	107	142	1,185	1.812	1.963	
egionellosis	16	5	N	273	210	N	
eprosy	3	5	4	76	106	101	
Malaria	15	12	12	247	261	261	
Measles: Total*	293	141	64	1.636	2.667	1,118	
Indigenous	262	128	N	1.431	2,554	N	
Imported	.31	13	N	205	109	N	
Meningococcal infections: Total	55	59	61	1.312	1.198	1,284	
Civilian	55	59	60	1,311	1,196	1,273	
Military	l			1,0	2	5	
Mumps	386	117	93	7,181	1.344	1,529	
Pertussis	38	32	31	622	914	654	
Rubella (German measles)	21	10	28	139	187	281	
Syphilis (Primary & Secondary): Civilian	656	573	489	11,912	9.437	10.279	
Military	2	4	6	70	83	127	
Foxic Shock syndrome	6	13	Ň	109	133	N	
luberculosis	359	425	425	7,117	7,215	7.425	
Tularemia	1 4	7-5	4	39	23	38	
Typhoid Fever	11	7	5	105	88	122	
Typhus fever, tick-borne (RMSF)	' ;	30	10	38	50	00	
Rabies, animal	98	132	124	1.800	2.038	2.038	
The state of the s	30	,,,,		,,000	000	4.950	

TABLE II. Notifiable diseases of low frequency, United States

	Cum. 1987		Cum. 1987
Anthrax Botulism: Foodborne Infant (Calif 1) Other Brucellosis (Ark 1) Cholera Congenital rubella syndrome Congenital syphilis, ages < 1 year Diphtheria	3 19 30 3	Leptospirosis Plague Poliomyelitia, Paralytic Poliomyelitia, Paralytic Politacosis (Mo 1, Colo 1, Oreg 1) Rabies, human Totanus Trichinosis Typhus fever, (lea-borne (endemic, murine) (Calif 2)	8 2 29 9 22 10

Five of the 293 reported cases for this week were imported from a foreign country or can be directly traceable to a known internationally imported case within two generations

TABLE III. Cases of specified notifiable diseases, United States, weeks ending May 16, 1987 and May 10, 1986 (19th Week)

1	AIDS	Aseptic Menin-	Encep	halitis	Gono		H	epatitis (V		Legionel	Leprosy	
Reporting Area	Alus	gitis	Primary	Post-in fectious	(Civi	lian)	A	8	NA NB	Unspeci fied	losis	Cepros
	Cum 1987	1987	Cum 1987	Cum 1987	Cum 1987	Cum 1986	1987	1987	1987	1987	1987	Cum 1987
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TABLE III. (Cont'd.) Cases of specified notifiable diseases, United States, weeks ending May 16, 1987 and May 10, 1986 (19th Week)

İ	Malaria			les (flub		r	Menin- gococcal	Mu	mps		Perlussis	.	Rubella		
Reporting Area	Cum	 	Cum	1987	Cum	Total Cum	Infactions	} -	Cum		Cum	Cum	1001	Cum	Cum
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UNITED STATES		262	1,431	31	205	2.667	1,312	386	7,181	38	622	914	21	139	187
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38	, 5	2	20	•	17	. 2	. 61	4	33	2	15	21	•	•	•
N CENTRAL	8	3	125	1	16	515	169	217	4,179	1	76	164	1	19	12
)hio nd	4 2	:	1	:	4		63 20	11	57 590	. !	26 1	63 16			
11 .	1	3	83	ît	12	301	25	58	2,008		5	21	1	18	9
Mich: Nis	1	••	. 23			210	50	38	570	-	24	18	•	1	2
	•		38			210	11	1	956	•	20	46	-		1
VN CENTRAL	7	55	89	6 6 §	12	130 24	63 20	63 49	880 548	-	34 7	43 20	• •	1	7
owa owa	ī					- 1	3	1	233	-	á	6		1	:
Ao ·	2	55	89		. 1	6	17	-	13	• ,	13	4	-		,1
V Dak 5 Dak					:	8	1	13	5 51	:	1 2	2		:	:
łabr		. •	•			_:	2	• .	2	-		1	-		٠.
lans	•	•	•	•	1	91	19	•	28	•	8	7	•	•	. 6
ATLANTIC	45	. •	42		4	363	224	6	124	3	134	358		9	1
Del Md	10	:	:			22	4 20	3	12		2	205 45	:	ž	:
C	6	-	-		. 1		5			5				:	
/a N Va	В	:		:	· :	29 2	37	î	48 18	í	33 27	9		1	
V C	6	-	:		• :	2	29	j	3	ż	55	14	-		
C	3 2	•		•	•	291	21 44		10	•	13	- 6 56	•	í	
ia Ia	. 9	. :	42		3	13	64	i	6 27		4	18		5	i
S CENTRAL	2		2			1	64	68	1,039		7	16		- 2	1
(γ .	-	•	•		-	-	11		202	•	1	1	•	2	1
lenn	1	- :	**		:	1	22 25	66 2	822 15	:	3	10		:	:
Ala Viiss	1	•	2	•	•	•	6	:	,,		2				
NS CENTRAL	14	61	135		1	358	95	14	522	. 1	41	27	1	2	36
Ark	1			•	•	274	10	-	203		2	2	•	1	•
a Okla	3	-	•		i	4	10 15	- 5 N	183 N	i	9 30	4 21	:		
le»	10	61	135			80	60	9	136				1	1	36
MATMUON	9	5	236		12	162	49	5	133	14	58	92	9	15	1
Mont	:	:	42	٠	1	1	4	. :	3	6	1 18	4 26			•
daho Nyo	.1	:				• :	4	1	-		. 2	1	:	i	
Calo	. 1	÷		-	<i>:</i>	.5	15	.:	22	-	17	19		•	
v Mex Arız	5	5	193		9 1	19 137	3 18	N 4	N 101≻	8	- 3 16	23	4	. 4	ī
Jjah .	-		:	-			8		5 2		. 1	10	5	9	:
lev	2		. •	٠	1	•	. 3		. 2	•	-		•		•
ACIFIC	124	6	463	-	44	221	400	. 7	174	15	170	75	9	85	102
Nash Drag	. 4		1	-	77	. 52			29	. 1	25	26 5			2
ald	110	6	460		32 8	147	17 327	. N	131	3	73	41	2	62	99
Maska	,,3	·					4	·	3		2	1			
ławau	•		•	•	4	20	. 2		11	10	56	2	7	22	1
iuam 	:	10	404			3	3 2		4		,;	. :		t t	2
' B	. 1	18	404			8	2	1 2	. 4		11	4		1	58
ac Trust Terr		٠.	-				1	î	4	*	1.			1	
Amer Samoa	-		•						3		-				

^{*}For measles only, imported cases includes both out of-state and international importations

N Not notifiable U Unavailable International 5Qut of state

TABLE III. (Cont'd.) Cases of specified notifiable diseases. United States, weeks ending May 16, 1987 and May 10, 1986 (19th Week)

Reporting Area	Syphilis ((Primary & S	Civilian) Secondary)	Toxic shock Syndrome	Tuber	culosis	Tula remia	Typhoid Fever	Typhus Fever (Tick borne) (RMSF)	Rabies Animal	
	Cum 1987	Cum 1986	1987	Cum 1987	Cum 1986	Cum 1987	Cum 1987	Cum 1987	Cum 1987	
JNITED STATES	11,912	9,437	6	7,117	7,215	39	105	38	1,800	
EW ENGLAND	188	185	3	204	219	1 4	9	1	. 1	
Maine	1	11	• ,	14	19	•	7			
1 H	2	6 6	:	5 4	10 8	•	•	•	•	
Mass	91	89	3	93	107		. 7	1		
1	5	12	•	23	. 14		· .	•		
onn	88	61	•	65	61		, i			
IID ATLANTIC	2,159	1,304	• .	1,299	1,486	*	10		130	
ipstate N Y	78 1,512	64 742	•	200 638	226 714		4	•	. 1	
Y City	239	249		219	277		6	•		
)a 1	330	249		242	269		•	-	12	
N CENTRAL	209	387		861	902	,	17	3	- 50	
Ohio	39	50		175	149	i	6	ž	٠.	
nd	18	43	-	83	106	:	4		•	
1 .	69	217	• .	347	400		. 4		24	
Aich	62	- 56		227	201	100	2			
Vis	21	21	•	29	46	-	ī		18	
VN CENTRAL	55 5	99 17		205 55	202 49	11	7 2	-1	39: 9:	
Ainn Swa	. 9	5	•	10	17	3	2		120	
owa No	25	51	-	103	100	7	3	1	1	
no I Dak	20	2	:	103	4		٠		5	
Dak	5	î		. 9	9		- :		76	
lebr	7	8		11	4			-	12	
ans	Ä	15		16	19	1			24	
ATLANTIC	4,066	2,741	•	1,434	1,386	3	8	- 10	48	
el	36	12		12	18	1				
Λd	222	176	•	127	96		1	2	170	
C	127 93	129 159	•	45 127	51		:	•	. 2	
/a V Va	93 5	8		44	131 47	1	1		14	
i C	229	194		144	. 191	1		ż	2	
c c	270	258		140	159			5	21	
5a	585	513		207	188			•	6	
la	2,499	1,292		588	505		4	1 1	, 3	
S CENTRAL	753	616		597	630	2	1	6 .	160	
4	6 324	26	•	166	159	1		•	7	
enn	192	237 211	-	163	184	•	1	4 ,	-5	
Ala Aiss	231	142		186 82	203 84	, i		2	3	
VS CENTRAL	1,550	1,975	3	800	880	12	6	13	28	
Ark	78	93		86	95	5	1	1	6	
.a	272	315		105	171	1				
Okla	- 61	56	3	81	82	6	2	12	100	
e×	1,139	1,511	•	528	532	. •	. 3	•	18	
MOUNTAIN	271	225 2		175 8	159	7	3	1	14	
daho :	í	í		16	5	i	-		,	
Vyo	22	:					•	-	3	
colo	41	67		-	9	1			, ,	
l Me	21	26		36	34	i .	3			
Ariz	121	94	-	99	76	2	-		3	
llah	. 7	_4		6	13	1		-		
lev	51	31	•	10	. 15					
ACIFIC	2,661	1,905	1	1.542	1.351	3	44	1	16	
Vash	31	49	•	74	75.	1	1	*		
Dreg Cahi	102 2,521	39 1,800	-	1 220	47	2	4.5			
iani Maska	2.521	1,600	•	1,330 22	1,141		41	1	18	
lawan	5	17		73	64		2			
iuam	2	1		4						
	357	301	-	92	91	-		4 - 1 - 1	. 2	
A,										
A. Lac Trust Terr	3 83	115		1 56	1 10	*	9	•		

U Unavailable

TABLE IV. Deaths in 121 U.S. cities." week ending May 16, 1987 (19th Week)

		All Caus	es, By A	gn (Year	e)					All Cause	s, By Aç	a (Years)		0.444
Reporting Area	All Ages	≥65	45-64	25-44	1.24	<1	P&I** Total	Reporting Area	All Ages	≥65	45-64	25-44	1-24	<1	Pal** Total
NEW ENGLAND	676	476	127	38	16	19	63	S ATLANTIC	1,243	764	251	137	45	45	43
Boston, Mass	153	100	34	10	5	4	22	Atlanta, Ga	167	101	32	28	7	.!	. 4
Bridgeport Conn	42	30	6	3	3		3	Baltimore Md	312	205	62	25 9	9	11 6	13
Cambridge, Mass	27	18	8	1			4	Charlotte, N.C.	93	54 45	21 25	23	14	5	1
Fall River, Mass. Hertford, Conn	31 58	26	3 .	2	2		1	Jacksonville Fla Miami Fla	112	59	21	23	'7	٠.	1
Lowell, Mass	34	43 28	6	3	2	3	2	Norfolk Va	56	37	13	ż	i	3	Ś
Lynn, Mass	25	19	ä	i	í	ì	1	Richmond, Va	80	53	12	10	4 :	1	3
New Bedford, Mas		22	4	Å	i	÷		Savannah Ga	29	21	6	2	-	•	2
New Haven, Conn	73	52	13	3	i	4	4	St Petersburg, Fla	75	58	11	3	2	1	5
Providence, RI	51	35	12	1		3	2	Tampa Fla	68	47	12	. 5	2	12	2
Somerville, Mass	.9	9		•	-		1	Washington D.C.	130	8B 16	27 9	18 7	1	15	3
Springfield Mass Waterbury, Conn	45	31	7	4		.3	7	Wilmington, Del	33	10	9	,	'	•	•
Worcester, Mass	34 63	23 40	6 18	3 .	2	i	12	ES CENTRAL	744	504	146	40	22	32	42
***************************************	. 03	-10	10	3	•	•	,,	Birmingham, Ala	97	60	17	10	3	7	1
MID ATLANTIC :	2,891	1.861	614	292	58	66	142	Chattanooga, Tenn		57	10	3	2	4	6
Albany, N Y	39	24	10	1	1	3	2	Knoxville. Tenn	71	57	12	1	1	•	6
Allentown, Pa	15	11	4	2.0	•		-	Louisville Ky	114	82	22	6	3	1	8
Buffalo, N.Y	120	. 82	28	7	2	3	13	Memphis, Tenn	153	106	30	· 7	8	7 6	12
Camden, N.J	48	29 14	9 3	5 .	2	3	-	Mobile Ala	59 41	34 28	15	2	i	1	3
Elizabeth, N.J. Erie, Pa.t.	31	26	4	3	-	í	1	Montgomery, Ala Nashville, Tenn	128	80	31	8	3	6	6
Jersey City, NJ	66	30	17	11	1	ż	,	regarrente, termi	120	- 00	•	_			-
	1,489	944			30	27	67	WS CENTRAL	1,253	770	280	102	51	50	56
Newsik, NJ	85	24	26	29	1	5	3	Austin Tex	50	34	G	5	2	1	6
Paterson, N J	30	17	6	3	1	3	. 3	Baton Rouge La	52	27	19	4	:	2	1
Philadelphia, Pa	493	337	100	38	9	9	26	Corpus Christi Tex	49	30	12	.4	3	.1	4
Pittsburgh, Pa t	100	67	25	7	•	١	2	Dallas, Tex	152	87	37	10 5	- 6 - 4	12	2 8
Reading, Pa	37 96	30 70	4 16	3	4	ī	9	Et Paso, Tex Fort Worth Tex	61 89	38 68	11	. 4	- 4	7 2	5
Rochester, N Y Schenectady, N Y	21	16	4	5	1	1	1	Houston Tex &	308	176	74	34	13	11	7.
Scranton, Pa t	26	17	-6	ź	1	:	i	Little Rock, Ark	71	51	10	5		5	6
Syracuse, NY	108	72	26	5	з	2	3	New Orleans La	108	63	26	7	7	3	
Trenton, N.J.	25	17	3	ē	ĭ	-	2	San Antonio, Tex	168	101	41	12	10	4	10
Utica, NY	23	21	1		1		2	Shreveport La	55	35	. 12	7	1		3
Yonkers, N Y	19	13	, 4	1	•	1	1	Tulsa Okla	92	60	23	. 5	2	. 2	4
	2,318	1,505			61	65	82	MOUNTAIN	692	430	137	61	41	22	26
Akron, Ohia	56	37	13	6	:	•	4	Albuquerque N Me		57	. 12	. 15	17	5	5
Canton, Ohio Chicago, Ill §	35 564	20 362	10 125	4 45	10	22	16	Calo Springs Cula Denver Cala	105	28 61	5 29	3 11	3	2	4
Cincinnati, Ohio	146	97	33	77	7	2	11	Las Vegas. Nev	92	56	20		ź	2	ż
Cleveland, Otho	166	106	35	16	6	3	4	Ouden. 'Jtah	15	12	ã				ī
Criumbus, Ohio	130	79	34	8	ő	3.	4	Phoenix Ariz	145	86	39	. 9	5	6	3
Dayton, Ohio	118	81	22	. 7	3	5		Pueblo Colo	22	17	2	3	-		2
Detroit, Mich	247	140	54	36	11	6	7	Salt Lake City Utah		32	7	8	4	4	•
Evansville, Ind	42	32 44	.7	. 2	5	1	3	Tucson Anz	110	81	20	5	3	. 1	- 8
Fort Wayne, Ind Gary, Ind	67 11	6	13 3	4	1	1	i	PACIFIC	2,080	1,393	385	172	71	53	132
Grand Rapids, Mic		43	12	ź	i	ā	3	Berkeley, Calif	2,080	22	363	1/2	'i		1 1
Indianapolis, Ind	169	101	47	15	3	3	3	Fresno Calif	78	54	17	4	i	2	8
Madison, Wis	35	27	6	ĭ	ĩ	-	2	Glendale, Calif	29	22	5	2			2
Milwaukee, Wis	147	113	23	5		6	4	Honolulu, Hawan	66	43	13	4	2	4	3
Peans. III	45	36	3	3		3	6	Long Beach Calif	119	80	26		. 1	. 6	3
Rockford, III	43	23	12	6	1	1	3	Los Angeles, Calif	582	364	124	65	21	3	27
South Bend, Ind	59	36	17	. 4	1	2	3	Oakland, Calif	. во	49	20		6	3	4
Toledo, Ohio	115	. 79	23	5	4	4	6 1	Pasadena, Calif Portland, Oreg	21 127	16 100	16		3	i	3 9
Youngstown, Ohio	62	43	17	1	. 1	•	'	Sacramento, Calil	143	103	24	6	6	4	15
W N CENTRAL	801	553	168	43	15	22	38	San Diego, Calif	163	103	31		ĕ	6	27
Des Moines, Iowa	61	38	12	5	2	4	2	San Francisco, Cali	1 188	114	29	35	7	3	7
Duluth Minn	32	28	3	1		-		San Jose Calif	180	126	33	- 11	4	6	. 11
Kansas City, Kans	37	24	11	2	•		4	Seattle Wash	166	116	23		6	11	6
Kansas City, Mo	107	69	20	12	1	5	7	Spokane Wash	62	44	. 9		3	2	2
Lincoln, Nabr	35	30	5			4	3	Tacoma, Wash	50	36	10	2	1	1	. 4
Minneapolis, Minn	159	108	31	10	6	. 2	13	TOTAL	12 600	^{††} 8,256	2617	1,063	380	374	624
Omaha, Nebr	141	59 100	21 31	3 .	1	1	1	TOTAL	12,038	0,230	2,017	1,003	300	,3 / 4	024
St Louis, Mo St Paul, Minn	. 75	53	19	1	•	2	i	i .							
Wichita, Kans	68	44	15	à	1	4	3	i .							

^{*} Mortality data in this table are voluntarily reported from 121 cities in the United States, most of which have populations of 100,000 or more. A death is reported by the place of its occurrence and by the week that the death certificate was filed. Fetal deaths are not included. **Pineumonia and influenza.** Because of changes in reporting methods in these 3 Pennsylvania cities, these numbers are partial counts for the current week. Complete counts will be available in 4 to 6 weeks. If 10to a includes unknown ages.

§ Data not available. Figures are estimates based on average of past 4 weeks.

B-Virus — Continued

Patient 3: On March 11, a 53-year-old male laboratory supervisor who had been employed at NAMRL for 12 years handled a clinically healthy monkey. He wore leather gloves to catch the animal but wore only surgical gloves while holding it afterward. He reported no bites, scratches, or contact with monkey body fluids. On March 27, he noted pruritic vesicles on the third finger of his right hand. Three days later the lesions were dry and crusted. A physician at the laboratory referred him to a darmatologist who performed a biopsy and later placed him on oral acyclovir. The tissue obtained during the biopsy was positive for a herpesvirus, and, on April 6, the patient was hospitalized. Intravenous acyclovir was begun on April 10, and the tissue was confirmed positive for B-virus on April 13. The lesions continued to heal, and the disease did not progress further. On April 21, the patient was discharged from the hospital and instructed to continue treatment with oral acyclovir. However, he greatly reduced his dosage a few days later. Routine follow-up cultures of conjunctiva and buccal mucosa obtained on April 28 were positive for B-virus the following day. He was readmitted to the hospital and again placed on intravenous acyclovir. He has remained asymptomatic. All other follow-up cultures except a rectal culture obtained May 8 have been negative.

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Patient 4: The 29-year-old wife of Patient 2 applied hydrocortisone cream to her husband's skin lesions beginning about March 18. During this time, she also applied this cream to an area of contact dermatitis under a ring on her finger. The dermatitis was highly pruritic, and she scratched it to the point of bleeding. On April 1, she was seen by a dermatologist who performed a culture of samples taken from the lesion and prescribed oral acyclovir. On April 7, the culture was reported positive for B-virus, and the patient was hospitalized and placed on intravenous acyclovir. Her dermatitis cleared, and the disease did not progress further. Cultures of oral and conjunctival specimens were performed every 3 to 4 days. The conjunctival cultures became positive for B-virus beginning with the specimen of April 10 and remained positive through April 28. She had no clinical evidence of conjunctivitis, and subsequent cultures have been negative.

Forty-nine persons who had direct (skin-to-skin or body-fluid-to-skin) contact with the patients before diagnosis are under clinical and laboratory surveillance for B-virus infection. No cases of infection or illness suggestive of B-virus have been detected among this group. The ill monkey that bit Patient 1 and that may have bitten Patient 2 and the clinically healthy monkey that was handled by Patient 3 have positive saliva cultures for B-virus.

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Editorial Note: B-virus, a close relative of the herpes simplex viruses of man, is enzootic in macaques and possibly other Old World monkeys. It is most frequently associated with Rhesus monkeys (Macaca mulatta). Like herpes simplex virus infections in man, B-virus infection in monkeys is characterized by intermittent reactivation and shedding, particularly during periods of stress and/or immunosuppression. Fortunately, symptomatic infection in monkey handlers and in persons handling monkey tissue appears to be rare—since the discovery of the virus in the 1930s, only 23 cases of symptomatic human infection have been described in the literature (2). However, the consequences of symptomatic infection are severe—of the 23 patients, 18 have died from encephalitis. The frequency of asymptomatic human infection is unknown.

B-Virus Infection - Continued

In at least one instance, Patient 1 and Patient 2 had handled an ill monkey that had not been anesthetized. It appears that at least one of them had not worn the recommended protective clothing. One was bitten, and the other was either bitten, scratched, or infected through contamination of a preexisting wound. It is, therefore, likely that the use of appropriate protective clothing could have prevented illness in at least one of the men. Patient 3, however, was appropriately protected when he handled the second culture-positive monkey, and he was not aware of any skin contact with the monkey or its body fluids. However, he may have had unrecognized contact with contaminated material.

Patient 4 has the first documented case of human-to-human transmission of B-virus. Infectious fluid from her husband's skin lesions was apparently inoculated directly into macerated skin, similar to the inoculation produced by a monkey bite. Since her infection does not appear to have spread systemically, she may have spread the infection to her eyes when she inserted her contact lenses. Transmission of the virus by less direct contact, such as inoculation of infectious fluid on intact skin or transmission by fomites, although theoretically possible, has not been documented. The lack of detectable infection thus far in persons with such exposures to any of the four patients suggests that transmission from casual contact is unlikely. This information will be important as public health recommendations are developed for releasing Patient 3 and Patient 4 back into the community.

This outbreak serves as a reminder of the inherent risk in working with macaques and possibly with other Old World monkeys. These monkeys should not be used for research purposes unless the handlers can adhere strictly to published guidelines. These guidelines state that persons working with macaques should wear gloves and laboratory coats to avoid bites and scratches (3). To further reduce risk, monkeys, especially large ones, should be anesthetized before handling, when it is feasible, or should be housed in squeeze cages.

The most important control measure is the careful education of animal caretakers and laboratory personnel who handle monkey tissues. The following points should be emphasized:

1) the nature and risk of B-virus infection, 2) the need to rapidly and thoroughly cleanse any penetrating wounds, 3) the need to seek medical attention immediately if suspicious lesions or other symptoms such as intense pruritus or numbness occur, and 4) the need for any physician suspecting B-virus infection to consult public health authorities and to institute appropriate diagnostic and therapeutic measures. So far, acyclovir therapy appears to have prevented the progression of disease in Patient 3 and Patient 4. The apparent responsiveness of these infections to treatment underscores the importance of early recognition and treatment of B-virus infection in symptomatic persons.

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Current Trends

Deaths Among the Homeless — Atlanta, Georgia

Between July 1, 1985, and June 30, 1986, the Office of the Fulton County Medical Examiner in Georgia investigated 40 deaths occurring among the homeless. All of the deaths occurred in Atlanta, 95% of which is located in Fulton County. In 1986, Atlanta had an estimated homeless population of 4,000 to 7,000 (Task Force for the Homeless, unpublished data). Based on these figures, the crude death rate among the homeless for that year was 5.7 to 10,0/1,000.

The medical examiner's (ME's) office identified these deaths by reviewing the 2,380 deaths reported during the 1-year period being studied. A decedent was considered homeless if there was no address available at the time of death or if the available address could not be considered a residence. The city directory was used to verify addresses. The ME's records supplied information on the age, sex, and race of the decedent; the location and date of death; and the results of autopsy (performed in 23 cases) and toxicologic examinations (performed in 35 cases).

Black males accounted for 19 (48%) of the 40 deaths; black females, for three (8%); and white males, for 18 (45%). The age at death was known for 36 of the 40 decedents; the median age for this group was 44 (range = 21-70 years). For black men, the median age at death was 43 (range = 22-56 years), and, for white men, it was 53 (range = 23-70 years). The age at death was known for two of the three women; one was 21, and the other, 63. The address of 11 of the decedents was a shelter.

Twenty-two persons (55%) died or were found dead outdoors; one was in a parked car. Of the 18 persons who died indoors, seven were found in vacant buildings; five, at shelters; three, in houses or apartments; one, in prison; and one, in an alcohol treatment unit; one was killed while in a store during a robbery. Two to five deaths occurred each month except October, when eight persons died—four in a single fire.

Cause of death was determined from the medical history, the scene investigation, circumstances of death, and autopsy and toxicologic studies, when performed (Table 1). The ME categorized the manner of death as either natural (the consequence of a disease or of the aging process), accidental (unintentional), homicidal, or suicidal.

Sixteen deaths (40%) were classified as natural. Six of these were attributed to chronic alcohol (ethanol) abuse. Only one of the six had a measurable level of blood alcohol (198 mg%). The ME determined causes of the other five deaths from the circumstances of death and the medical history. Ten deaths resulted from natural causes other than the direct effect of alcohol. Three of these were from seizures (probably due to alcohol withdrawal); four, from heart disease; and three, from lung disease.

The ME classified 19 deaths (48%) as accidental. Seven of these resulted from acute alcohol toxicity (mean blood alcohol = 498 mg%, range = 296 mg%-610 mg%). Twelve resulted from accidental injuries and included six deaths from fires, two from hypothermia, two from pedestrian-motor vehicle incidents, one from drowning, and one from a fall.

There were four homicides and one suicide. Although blood alcohol was measurable for one of the homicide victims, the ME determined that alcohol was not causally associated with the death.

The ME determined that 28 (70%) of the 40 deaths were alcohol-related. Although blood alcohol was not measured or measurable at the time of death for 11 of these decedents, their deaths were classified as alcohol-related either because they had a history of alcohol abuse

Homeless -- Continued

or because of the circumstances of death. Three of these 11 decedents died from seizures consistent with alcohol withdrawal. One, who died from a fall, had had measurable blood alcohol in a sample taken from a subdural hematoma sustained in the fall. Another, who died 10 days after being struck by an automobile, had had measurable blood alcohol upon admission to the hospital. One, who was hospitalized for burns prior to death, was clinically judged to be intoxicated when admitted to the hospital. The other five died from the effects of chronic alcohol abuse.

No deaths were attributed to drugs other than alcohol. Of 31 decedents screened for barbiturates, benzodiazepines, phenytoin, and other weakly acidic or neutral drugs, three (10%) were positive (one for barbiturates, one for phenytoin, and one for barbiturates and phenytoin). All of the drugs were present at therapeutic or subtherapeutic levels. Thirteen persons were screened for cocaine or cocaine metabolites in their urine, and one was positive. Four (20%) of the 20 screened for cannabinoids were positive.

Reported by: R Hanzlick, MD, Office of the Fulton County Medical Examiner. Surveillance and Programs Br, Div of Environmental Hazards and Health Effects, Center for Environmental Health, CDC.

Editorial Note: Most deaths among the homeless in Atlanta occurred among men <60 years of age and were alcohol-related. The high proportion of alcohol-related deaths reported in this study underscores the potentially serious health consequences of alcohol use or abuse. These consequences include those resulting from the acute intoxicating effects of alcohol, alcohol withdrawal syndrome, and the effects of chronic alcohol abuse.

TABLE 1. Prevalence of alcohol (ethanol) in blood among deceased homeless persons, by cause of death — Atlanta, Georgia, July 1985-June 1986

	Total	Blo	Blood Analysis for Alcohol						
Cause of Death	Deaths	Positive	Negative*	Not Tested	Related †				
Natural									
Chronic Alcohol	6	1	5(1)	_	6				
Heart Disease	4		2(1)	2	_				
Seizures	3	_	3(2)	· —	3				
Lung Disease	3	_	2(1)	1	_				
Accidental									
Acute Alcohol Toxicity	7	7	_		7				
Fire, Burn, Smoke	6	- 5	_	1	6 .				
Hypothermia	2	2	_	_ ,	2				
Pedestrian	2	1	-	1	2				
Drowning	1 .	1	 .		1				
Fall	1	_	1(1)	· ·	19				
Homicide	4	1	3(2)	· -					
Suicide	1 .		1(1)	_					
Total	40	18	17(9)	5	28				

The number in parentheses represents the number of persons who did not have measurable blood alcohol at the time of death but who had received intravenous fluids or lived for more than 2 hours following injury or onset of the condition that directly led to their death (e.g., gunshot, myocardial infarction, seizure). In such a situation, the blood alcohol at the time of death may not reflect the blood alcohol at the time of injury.

[†]Determination that death was related to alcohol was made by the medical examiner on the basis of toxicologic studies, the scene investigation, and circumstances of death. No homicide or suicide deaths were classified as alcohol related because the presence of alcohol in the victim's blood cannot be considered to be causally related.

 $[\]S$ Although the systemic blood was negative for alcohol, blood in a subdural hematoma was positive for alcohol

Homeless -- Continued

Little information is available on the size and composition of the homeless population or the health problems and causes of death among this group. Estimates of the number of homeless persons in the United States vary widely (1,2). However, there is general agreement among health service providers that the number of homeless is increasing and that a growing proportion of these are young and female (3,4).

Homeless persons have been characterized as extremely poor, significantly disabled by mental or physical illness, and socially isolated. Marginal ties to family and others have been identified as a significant contributor to homelessness (5). Forty percent of homeless persons have psychiatric illnesses (6). Physical health problems among the homeless include trauma, respiratory disease, tuberculosis, scabies and pediculosis infestations, peripheral vascular disease, and chronic illnesses, such as diabetes mellitus, that are exacerbated by adverse living conditions and lack of health care (7).

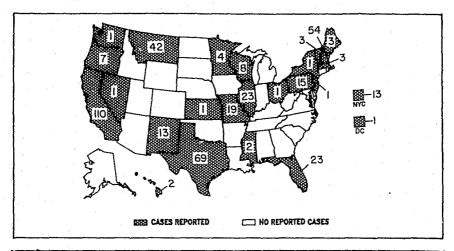
MEs investigate sudden or unexpected deaths, violent deaths, and deaths to persons unattended by a physician. Since the homeless often die suddenly and without a physician, many of these deaths are investigated by an ME. However, an unknown number of homeless persons die while hospitalized, and their deaths are not routinely investigated. Despite this limitation, ME's records are one of the few sources of information available for describing deaths among the homeless.

Additional studies are needed to describe the characteristics of deaths among the homeless more completely. Studies on such deaths in other parts of the country are needed. A better understanding of the causes and circumstances of these deaths would help in developing public health programs to prevent them. For now, this limited study suggests that, although providing shelters might prevent deaths from hypothermia and some fires, this intervention alone will not prevent most deaths among the homeless.

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FIGURE I. Reported measles cases — United States, weeks 15-18, 1987



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The editor welcomes accounts of interesting cases, outbreaks, environmental hazards, or other public health problems of current interest to health officials. Such reports and any other matters pertaining to editorial or other textual considerations should be addressed to: ATTN: Editor, Morbidity and Mortality Weekly Report, Centers for Disease Control, Atlanta, Georgia 30333.

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