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Protection Against Viral Hepatitis (Part I)*

Recommendations of the Immunization Practices Advisory Committee (ACIP) of the U.S. Public Health Service

The following statement updates all previous recommendations on protection against viral hepatitis, including use of hepatitis B vaccine and hepatitis B immune globulin for prophylaxis of hepatitis B, universal screening of pregnant women to prevent perinatal hepatitis B transmission, and use of immune globulin to prevent other types of viral hepatitis. Part I covers hepatitis A and non-A, non-B hepatitis. Part II will appear in a future issue and will cover hepatitis B.

INTRODUCTION

The term "viral hepatitis" is commonly used for several clinically similar diseases that are etiologically and epidemiologically distinct (*1*). Two of these, hepatitis A (formerly called infectious hepatitis) and hepatitis B (formerly called serum hepatitis), have been recognized as separate entities since the early 1940s and can be diagnosed with specific seriologic tests. A third category, currently known as non-A, non-B hepatitis, includes two epidemiologically distinct types of hepatitis: parenterally transmitted and enterically transmitted non-A, non-B hepatitis. Parenterally transmitted non-A, non-B hepatitis is associated with both post-transfusion and sporadic cases of acute hepatitis and may be caused by at least two different agents. Part of the genome for one of these agents has recently been cloned, and a candidate serologic assay for antibody to this virus (proposed as hepatitis C virus) has been developed (*2,3*). Enterically transmitted non-A, non-B hepatitis, which is spread by the fecal-oral route and is different from the types seen in the United States, has been reported in parts of Asia, Africa, and Mexico (*4*). Another distinct type of hepatitis, delta hepatitis, is an infec-

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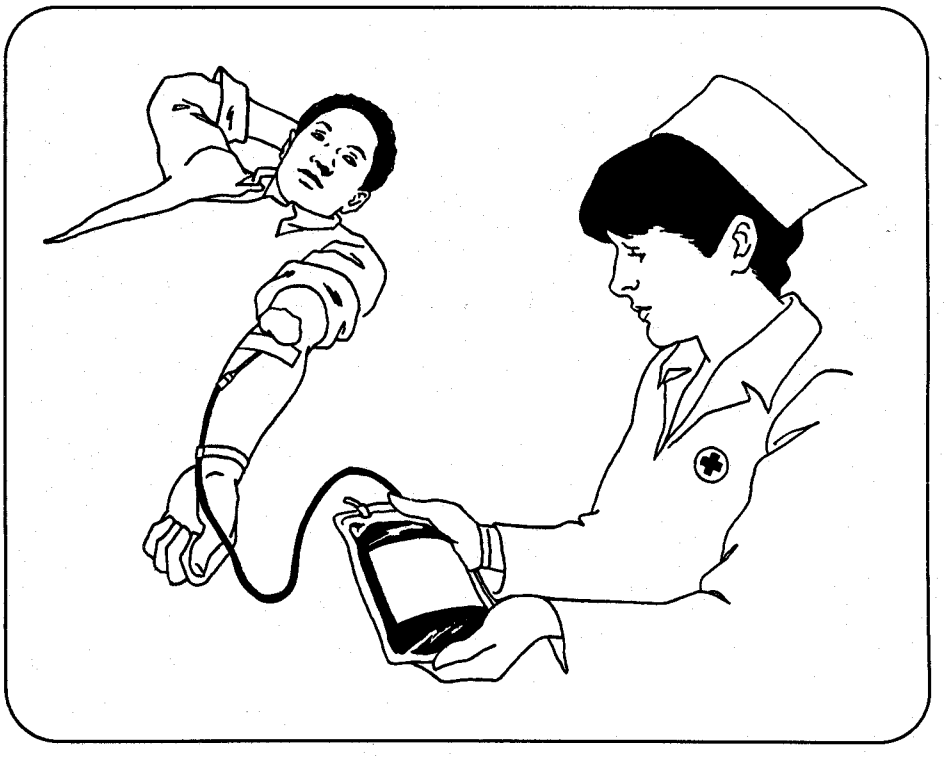

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tion dependent on the hepatitis B virus. It may occur as a coinfection with acute hepatitis B infection or as superinfection of a hepatitis B carrier (5).

Hepatitis Surveillance

Approximately 28,500 cases of hepatitis A, 23,200 cases of hepatitis B, 2,620 cases of non-A, non-B hepatitis, and 2,470 cases of hepatitis type unspecified were reported in 1988 in the United States. Most cases of each type occur among young adults. Since reporting from many localities is incomplete, the actual number of hapatitis cases occurring annually is thought to be several times the reported number.

Immune Globulins

Immune globulins are important tools for preventing infection and disease before or after exposure to hepatitis viruses. Immune globulins used in medical practice are sterile solutions of antibodies (immunoglobulins) from human plasma. They are prepared by cold ethanol fractionation of large plasma pools and contain 10%-18% protein. In the United States, plasma is primarily obtained from paid donors. Only plasma shown to be free of hepatitis B surface antigen (HBsAg) and antibody to human immunodeficiency virus (HIV) is used to prepare immune globulins.

Immune globulin (IG) (formerly called immune serum globulin, ISG, or gamma globulin) produced in the United States contains antibodies against the hepatitis A virus (anti-HAV) and the HBsAg (anti-HBs). Hepatitis B immune globulin (HBIG) is an IG prepared from plasma containing high titers of anti-HBs.

There is no evidence that hepatitis B virus (HBV), HIV (the causative agent of acquired immunodeficiency syndrome [AIDS]), or other viruses have ever been transmitted by IG or HBIG commercially available in the United States (6). Since late April 1985, all plasma units for preparation of IGs have been screened for antibody to HIV, and reactive units are discarded. No instances of HIV infection or clinical illness have occurred that can be attributed to receiving IG or HBIG, including lots prepared before April 1985. Laboratory studies have shown that the margin of safety based on the removal of HIV infectivity by the fractionation process is extremely high (7). Some HBIG lots prepared before April 1985 have detectable HIV antibody. Shortly after being given HBIG, recipients have occasionally been noted to have low levels of passively acquired HIV antibody, but this reactivity does not persist (8).

Serious adverse effects from IGs administered as recommended have been rare. IGs prepared for intramuscular administration should be used for hepatitis prophylaxis. IGs prepared for intravenous administration to immunodeficient and other selected patients are not intended for hepatitis prophylaxis. IG and HBIG are not contraindicated for pregnant or lactating women.

Hepatitis A

Hepatitis A is caused by the hepatitis A virus (HAV), a 27-nm ribonucleic acid (RNA) agent that is classified as a picornavirus. Patients with illness caused by HAV characteristically have abrupt onsets of symptoms including fever, malaise, anorexia, nausea, abdominal discomfort, dark urine, and jaundice. Severity is related to age. Among children, most infections are asymptomatic, and illness is usually not accompanied by jaundice. Most infected adults become symptomatically ill with jaundice. The case-fatality rate among reported cases is about 0.6%.

Hepatitis A is primarily transmitted by person-to-person contact, generally through fecal contamination and oral ingestion. Transmission is facilitated by poor personal hygiene, poor sanitation, and intimate (intrahousehold or sexual) contact. In recent years, cases of hepatitis A among intravenous drug users, most likely due to person-to-person contact, have been reported with increasing frequency (9). Common-source epidemics from contaminated food and water also occur. Sharing utensils or cigarettes or kissing is not believed to transmit the hepatitis A virus.

The incubation period of hepatitis A is 15-50 days (average 28). High concentrations of HAV (10^8 particles/g) are found in stool specimens from infected persons. Virus in the feces reaches its highest concentration late in the incubation period and early in the prodromal phase of illness, and it diminishes rapidly once jaundice appears. Greatest infectivity is during the 2-week period immediately before the onset of jaundice. Viremia probably occurs during the period that the virus is shed in feces. Virus has not been found in urine. A chronic carrier state with HAV in blood or feces has not been demonstrated. Transmission of HAV by blood transfusion has been reported but is uncommon (10).

The diagnosis of acute hepatitis A is confirmed by finding IgM anti-HAV in serum collected during the acute or early convalescent phase of the disease. IgG anti-HAV, which appears in the convalescent phase of the disease and remains detectable in serum thereafter, confers enduring protection against the disease. Commercial tests are available to detect IgM anti-HAV and total anti-HAV in serum.

Although the incidence of hepatitis A in the United States in the 1980s was lower than that in the 1970s, a 26% increase in incidence was observed between 1983 and 1988. It is still a common infection among older children and young adults. In 1988, 50% of reported cases of hepatitis in this country were attributable to

hepatitis A.

Recommendations for IG Prophylaxis for Hepatitis A

Numerous field studies conducted in the past 4 decades confirm that IG given before exposure or during the incubation period of hepatitis A is protective against clinical illness (*11-13*). Its prophylactic value is greatest (80%-90%) when given early in the incubation period and declines thereafter (*13*). Recent tests have shown slightly decreased titers of anti-HAV in current IG lots compared with lots tested 8 years previously; however, no differences in IG efficacy have been noted.

Preexposure Prophylaxis

The major group for whom preexposure prophylaxis is recommended is international travelers. The risk of hepatitis A for U.S. citizens traveling abroad varies with living conditions, length of stay, and the incidence of hepatitis A infection in areas visited (*14-16*). In general, travelers to developed areas of North America, western Europe, Japan, Australia, and New Zealand are at no greater risk of infection than they would be in the United States. For travelers to developing countries, risk of infection increases with duration of travel and is highest for those who live in or visit rural areas, trek in back country, or frequently eat or drink in settings of poor sanitation. Nevertheless, recent studies have shown that many cases of travel-related hepatitis A occur in travelers with "standard" tourist itineraries, accommodations, and food and beverage consumption behaviors (*16* and CDC unpublished data). In developing countries, travelers should minimize their exposure to hepatitis A and other enteric diseases by avoiding potentially contaminated water or food. Travelers should avoid drinking water (or beverages with ice) of unknown purity and eating uncooked shellfish or uncooked fruits or vegetables that they did not prepare.

IG is recommended for all susceptible travelers to developing countries (*17*). IG is especially important for persons who will be living in or visiting rural areas, eating or drinking in settings of poor or uncertain sanitation, or who will have close contact with local persons (especially young children) in settings with poor sanitary conditions. Persons who plan to reside in developing areas for long periods should receive IG regularly.

For travelers, a single dose of IG of 0.02 ml/kg of body weight is recommended if travel is for <3 months. For prolonged travel or residence in developing countries, 0.06 ml/kg should be given every 5 months. For persons who require repeated IG prophylaxis, screening for total anti-HAV before travel is useful to define susceptibility and eliminate unnecessary doses of IG for those who are immune. IG produced in developing countries may not meet the standards for purity required in most developed countries. Persons needing repeat doses overseas should use products that meet U.S. license requirements.

Postexposure Prophylaxis

Hepatitis A cannot be reliably diagnosed on clinical presentation alone, and serologic confirmation of index patients is recommended before contacts are treated. Serologic screening of contacts for anti-HAV before they are given IG is not recommended because screening is more costly than IG and would delay its administration.

For postexposure IG prophylaxis, a single intramuscular dose of 0.02 ml/kg is recommended. IG should be given as soon as possible after last exposure; giving IG more than 2 weeks after exposure is not indicated.

Specific recommendations for IG prophylaxis for hepatitis A depend on the nature of the HAV exposure.

1. Close personal contact. IG is recommended for all household and sexual contacts of persons with hepatitis A.
2. Day-care centers. Day-care facilities attended by children in diapers can be important settings for HAV transmission (*18-20*). IG should be administered to all staff

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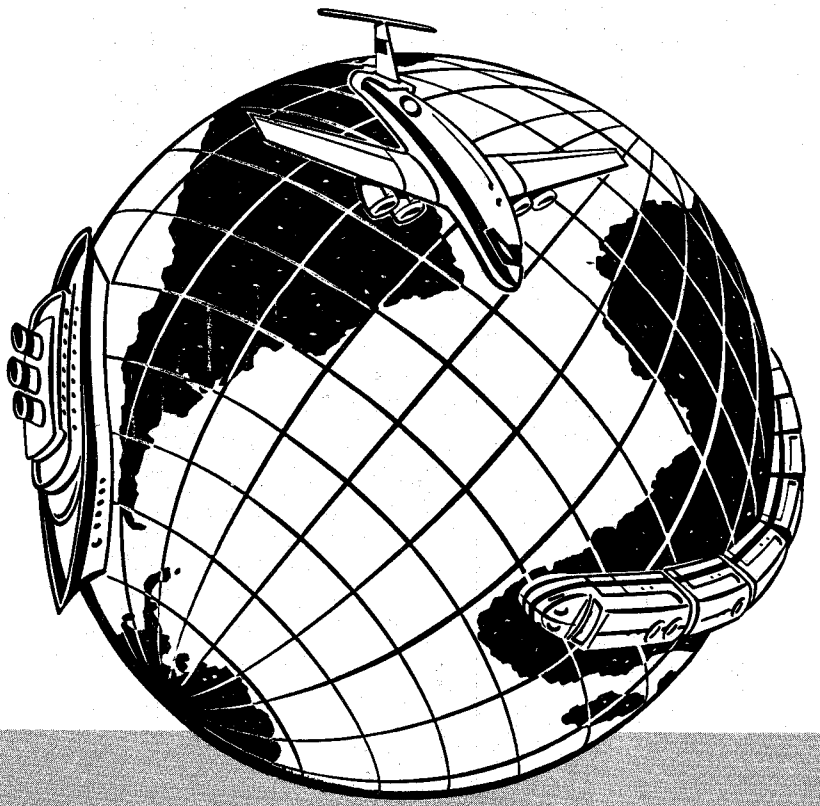

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and attendees of day-care centers or homes if a) one or more children or employees are diagnosed as having hepatitis A, or b) cases are recognized in two or more households of center attendees. When an outbreak (hepatitis cases in three or more families) occurs, IG should also be considered for members of households that have children (center attendees) in diapers. In centers not enrolling children in diapers, IG need only be given to classroom contacts of an index patient.

3. Schools. Contact at elementary and secondary schools is usually not an important means of transmitting hepatitis A. Routine administration of IG is not indicated for pupils and teachers in contact with a patient. However, when an epidemiologic investigation clearly shows the existence of a school- or classroom-centered outbreak, IG may be given to persons who have close contact with patients.
4. Institutions for custodial care. Living conditions in some institutions, such as prisions and facilities for the developmentally disabled, favor transmission of hepatitis A. When outbreaks occur, giving IG to residents and staff who have close contact with patients with hepatitis A may reduce the spread of disease. Depending on the epidemiologic circumstances, prophylaxis can be limited or can involve the entire institution.
5. Hospitals. Routine IG prophylaxis for hospital personnel is not indicated. Rather, sound hygienic practices should be emphasized. Staff education should point out the risk of exposure to hepatitis A and should emphasize precautions regarding direct contact with potentially infective materials (*21*).

   Outbreaks of hepatitis A occur occasionally among hospital staff, usually in association with an unsuspected index patient who is fecally incontinent. Large outbreaks have occurred from contact with infected infants in neonatal intensive care units (*10*). In outbreaks, prophylaxis of persons exposed to feces of infected patients may be indicated.
6. Offices and factories. Routine IG administration is not indicated under the usual office or factory conditions for persons exposed to a fellow worker with hepatitis A. Experience shows that casual contact in the work setting does not result in virus transmission.
7. Common-source exposure. IG use might be effective in preventing foodborne or waterborne hepatitis A if exposure is recognized in time. However, IG is not recommended for persons exposed to a common source of hepatitis infection after cases have begun to occur, since the 2-week period during which IG is effective will have been exceeded.

   If a food handler is diagnosed as having hepatitis A, common-source transmission is possible but uncommon. IG should be administered to other food handlers but is usually not recommended for patrons (*22*). However, IG administration to patrons may be considered if all of the following conditions exist: a) the infected person is directly involved in handling, without gloves, foods that will not be cooked before they are eaten, and b) the hygienic practices of the food handler are deficient or the food handler has had diarrhea, and c) patrons can be identified and treated within 2 weeks of exposure. Situations in which repeated exposures may have occurred, such as in institutional cafeterias, may warrant stronger consideration of IG use.

NON-A, NON-B HEPATITIS

Parenterally Transmitted (PT) Non-A, Non-B Hepatitis

Parenterally transmitted non-A, non-B hepatitis accounts for 20%-40%

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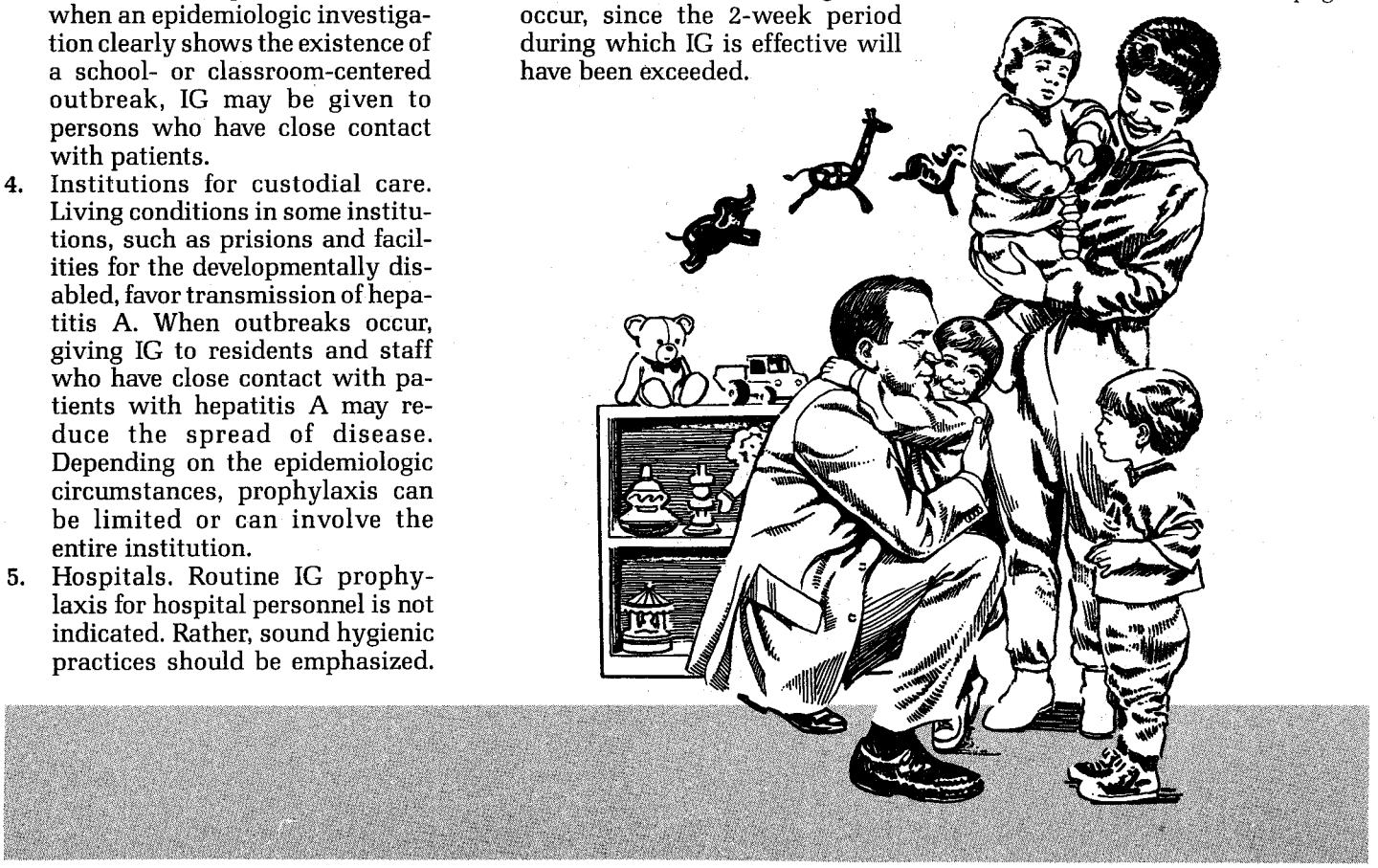

TABLE 1. Hepatitis nomenclature

	Abbreviation	Term	Definition/Comments
A. Hepatitis A	HAV	Hepatitis A virus	Etiologic agent of "infectious" hepatitis; a picornavirus; single serotype.
	Anti-HAV	Antibody to HAV	Detectable at onset of symptoms; lifetime persistence.
	IgM anti-HAV	IgM class antibody to HAV	Indicates recent infection with hepatitis A; detectable for 4–6 months after infection.
B. Hepatitis B	HBV	Hepatitis B virus	Etiologic agent of "serum" hepatitis; also known as Dane particle.
	HBsAg	Hepatitis B surface antigen	Surface antigen(s) of HBV detectable in large quantity in serum; several subtypes identified.
	HBeAg	Hepatitis B e antigen	Soluble antigen; correlates with HBV replication, high titer HBV in serum, and infectivity of serum.
	HBcAg	Hepatitis B core antigen	No commercial test available.
	Anti-HBs	Antibody to HBsAg	Indicates past infection with and immunity to HBV, passive antibody from HBIG, or immune response from HB vaccine.
	Anti-HBe	Antibody to HBeAg	Presence in serum of HBsAg carrier indicates lower titer of HBV.
	Anti-HBc	Antibody to HBcAg	Indicates prior infection with HBV at some undefined time.
	IgM anti-HBc	IgM class antibody to HBcAg	Indicates recent infection with HBV; detectable for 4–6 months after infection.
C. Delta hepatitis	HDV	Hepatitis D virus	Etiologic agent of delta hepatitis; can cause infection only in presence of HBV.
	HDAg	Delta antigen	Detectable in early acute delta infection.
	Anti-HDV	Antibody to delta antigen	Indicates present or past infection with delta virus.
D. Non-A, non-B hepatitis	PT-NANB	Parenterally transmitted	Diagnosis by exclusion. At least two candidate viruses, one of which has been proposed as hepatitis C virus; shares epidemiologic features with hepatitis B.
	ET-NANB	Enterically transmitted	Diagnosis by exclusion. Causes large epidemics in Asia, Africa, and Mexico; fecal-oral or waterborne.
E. Immune globulins	IG	Immune globulin (previously ISG, immune serum globulin, or gamma globulin)	Contains antibodies to HAV, low-titer antibodies to HBV.
	HBIG	Hepatitis B immune globulin	Contains high-titer antibodies to HBV.

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of acute viral hepatitis in the United States and has epidemiologic characteristics similar to those of hepatitis B (*60*). Recently, a portion of the genome of a virus thought to be responsible for PT non-A, non-B hepatitis was cloned (*2*). A candidate seriologic assay for antibody to this virus (proposed as hepatitis C virus) has been developed. This assay appears to detect a substantial number of persons with chronic infection and is being evaluated for screening potential blood donors (*3*). Although PT non-A, non-B hepatitis has traditionally been considered a transfusion-associated disease, most reported cases have not been associated with blood transfusion (*61-64*). Groups at high risk of acquiring this disease include transfusion recipients, parenteral drug users, and dialysis patients (*62-63*). Health-care work that entails frequent contact with blood, personal contact with others who have had hepatitis in the past, and contact with infected persons within households have also been documented in some studies as risk factors for acquiring PT non-A, non-B hepatitis (*63-65*). However, the role of person-to-person contact in disease transmission has not been well defined, and the importance of sexual activity in the transmission of this type of hepatitis is unclear.

Multiple episodes of non-A, non-B hepatitis have been observed among the same individuals and may be due to different bloodborne agents. An average of 50% of patients who have acute PT non-A, non-B hepatitis infection later develop chronic hepatitis (*66*). Experimental studies of chimpanzees have confirmed the existence of a carrier state, which may be present in 1%-3% of the population (*67-68*).

The risk and consequences of perinatal transmission of PT non-A, non-B hepatitis are not well defined. Only one small study has been published in which infants born of 12 women who had acute PT non-A, non-B hepatitis during pregnancy were followed. Six infants developed transient alanine aminotransferase (ALT) elevations at 4-8 weeks of age (*69*).

The results have been equivocal in several studies attempting to assess the value of prophylaxis with IGs against PT non-A, non-B hepatitis (*70-72*). For persons with percutaneous exposure to blood from a patient with PT non-A, non-B hepatitis, it may be reasonable to administer IG (0.06 ml/kg) as soon as possible after exposure. In other circumstances, no specific recommendations can be made.

Enterically Transmitted (ET) Non-A, Non-B Hepatitis

A distinct type of non-A, non-B hepatitis acquired by the fecal-oral route was first identified through investigations of large waterborne epidemics in developing countries. This ET non-A, non-B hepatitis, which has occurred in epidemics or sporadically in parts of Asia, North and West Africa, and Mexico, is serologically distinct from other known hepatitis viruses (*4-73*). Young to middle-aged adults are most often affected, with an unusually high mortality among pregnant women. The disease has been transmitted to experimental animals, and candidate viruses have been identified; however, no seriologic tests have yet been developed (*74*).

ET non-A, non-B hepatitis has not been recognized as an endemic disease in the United States or Western Europe, and it is unknown whether the causative agent is present in these areas. Cases have been documented, however, among persons returning from travel to countries in which this disease occurs (*75*).

Travelers to areas having ET non-A, non-B hepatitis may be at some risk of acquiring this disease by close contact with infected persons or by consuming contaminated food or water. There is no evidence that U.S.-manufactured IG will prevent this infection. As with hepatitis A and other enteric infections, the best means of preventing ET non-A, non-B hepatitis is avoiding potentially contaminated food or water.

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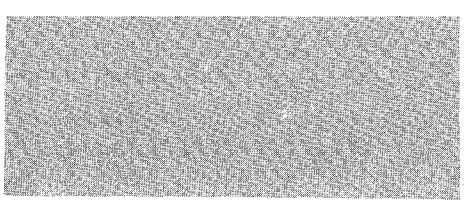
References 70 through 75 may be obtained by writing to the Hepatitis Branch, Division of Viral and Rickettsial Diseases, Center for Infectious Diseases, Mailstop A33, Centers for Disease Control, Atlanta, Ga. 30333.



Cases of selected notifiable diseases, Virginia, for the period March 1 through March 31, 1990.

DISEASE	TOTAL CASES REPORTED THIS MONTH						TOTAL CASES REPORTED TO DATE		
	STATE	REGIONS					THIS YEAR	LAST YEAR (STATE TOTALS)	5 YEAR AVERAGE (STATE TOTALS)
		N.W.	N.	S.W.	C.	E.			
Acquired Immunodeficiency Syndrome	48	3	14	1	23	7	155	105	65
Campylobacter Infections	44	14	12	6	8	4	112	97	89
Gonorrhea	1962	—	—	—	—	—	4687	3974	4018
Hepatitis A	35	0	5	2	12	16	52	46	61
B	27	2	6	3	6	10	60	73	97
Non A-Non B	4	0	2	1	0	1	9	12	17
Influenza	51	8	0	7	19	17	688	1656	1917
Kawasaki Syndrome	3	0	0	0	0	3	5	4	7
Legionellosis	3	1	1	0	0	1	5	1	3
Lyme Disease	4	0	0	0	1	3	7	1	1
Measles	14	1	13	0	0	0	19	0	10
Meningitis — Aseptic	16	3	6	2	1	4	50	47	39
Bacterial*	25	5	2	7	3	8	43	59	61
Meningococcal Infections	3	1	2	0	0	0	16	17	24
Mumps	10	2	4	0	1	3	19	31	12
Pertussis	3	0	0	3	0	0	7	3	10
Rabies in Animals	24	8	1	1	10	4	48	71	68
Reye Syndrome	0	0	0	0	0	0	0	1	<1
Rocky Mountain Spotted Fever	0	0	0	0	0	0	0	0	<1
Rubella	0	0	0	0	0	0	0	0	0
Salmonellosis	60	11	16	7	13	13	225	222	238
Shigellosis	10	1	3	1	2	3	36	152	62
Syphilis (Primary & Secondary)	84	3	14	17	37	13	196	144	104
Tuberculosis	58	1	13	7	11	26	82	77	83

Localities Reporting Animal Rabies: Augusta 2 skunks; Chesterfield 2 raccoons; Clarke 1 skunk; Cumberland 2 raccoons; Dinwiddie 1 raccoon; Fairfax 1 raccoon; Frederick 1 skunk; Hopewell 1 raccoon; Lunenburg 2 skunks; Nelson 1 raccoon; Newport News 2 raccoons; Nottoway 1 raccoon; Orange 1 skunk; Prince George 1 raccoon; Rockingham 1 horse; Shenandoah 1 raccoon, 1 skunk; Warren 1 fox; Washington 1 skunk.

Occupational Illnesses: Asbestosis 15; Carpal Tunnel Syndrome 46; Coal Workers' Pneumoconiosis 32; De Quervain's Disease 1; Dermatitis 1; Hepatitis B 1; Loss of Hearing 6; Repetitive Trauma Disorder 3; Thoracic Outlet Syndrome 1.

*Other than meningococcal

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