ANNUAL SUMMARY 1982 Issued June 1984



CENTERS FOR DISEASE CONTROL MALARRA SURVEILLANCE



U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES • Public Health Service

PREFACE

This report summarizes information received from state health departments, medical departments of the Armed Forces, and other sources. It is intended primarily for the use of those with responsibility for disease control activities. Anyone desiring to quote this report should contact the original investigator for confirmation and interpretation.

Contributions to the Surveillance Report are most welcome. Please address them to:

Centers for Disease Control Attn: Malaria Branch Division of Parasitic Diseases Center for Infectious Diseases Atlanta, Georgia 30333 Telephone: (404) 452-4046 (FTS) 236-4046

SUGGGESTED CITATION

Centers for Disease Control: Malaria Surveillance Annual Summary 1982 Issued June 1984

Publications & Graphics Frances H. Porcher, M.A., Chief Lynn W. Herring, Writer-Editor

I. SUMMARY

During 1982, 930 cases of malaria diagnosed in the United States were reported to the Centers for Disease Control. This compares with 1,103 cases reported in 1981, a decline of 15.7%, primarily due to fewer imported cases of malaria in foreigners. A total of 574 cases with onset in 1982 were reported in foreigners compared with 809 in 1981, a 29.0% decrease.

<u>Plasmodium vivax</u> was the parasite identified in 66% of the 930 cases, and <u>P. falciparum</u> was identified in 20% of the cases. <u>P. malariae</u> and <u>P. ovale</u> were reported in 5% and 3% of cases, respectively. The species was not determined in the remaining cases.

Only 17 of the 930 cases acquired their infection in the United States. Congenital transmission occurred in 7 cases, and infection through blood transfusion occurred in 9 cases. In one isolated case in Washington, DC, the infection was accidentally acquired in a laboratory from infected anopheline mosquitos.

Two deaths attributed to malaria were reported for 1982, compared with 7 fatal cases in 1981. One death occurred in a civilian who had been infected with <u>P. falciparum</u> in Nigeria, and one occurred in a woman who had lived in Iran and traveled to the United States via Pakistan and Spain.

II. TERMINOLOGY

The terminology used in this report is derived from the recommendations of the World Health Organization.¹ The definitions of the following terms are included for reference purposes.

A. Autochthonous

1. Indigenous--malaria acquired by mosquito transmission in an area where malaria is a regular occurrence.

2. Introduced--malaria acquired by mosquito transmission from an imported case in an area where malaria is not a regular occurrence.

B. Imported

Malaria acquired outside a specific area (the United States, Puerto Rico, and Guam in this report).

C. Induced

Malaria acquired through artificial means, i.e., blood transfusion, common syringes, or malariotherapy.

D. Relapsing

Renewed manifestation (of clinical symptoms and/or parasitemia) of malarial infection, separated from previous manifestations of the same infection by an interval greater than any interval due to the normal periodicity of the paroxysms.

E. Cryptic

An isolated case of malaria not associated with secondary cases as determined by appropriate epidemiologic investigation.

¹World Health Organization. Terminology of malaria eradication, 1963. World Health Organization, Geneva, p. 32.

III. GENERAL SURVEILLANCE

A total of 930 cases* with onset of illness in 1982 in the United States were reported to the Division of Parasitic Diseases, Center for Infectious Diseases, Centers for Disease Control; this represents a 15.7% decline over the 1,103 cases reported for 1981. Only 8 of the cases occurred in U.S. military personnel. Civilian cases have accounted for the majority of cases each year since 1973 (Table 1).

Malaria in foreign civilians accounted for 574 (61.7%) of all reported cases (Table 1). There was a decline of 29% in the number of cases among foreign civilians compared with 1981. In contrast, the number of malaria cases in U.S. civilians increased by 27% from 1981 and was larger than in any year since 1966 (Figure 1). CASES OF MALARIA IN U.S. CIVILIANS AND FOREIGNERS,



Table 1 All Primary Malaria Cases in Civilians and Military Personnel with Onset of Illness in the United States, 1966-1982*

Year	Military	U.S. Civilians	Foreign Civilians	Unknown	Total
1966	621	89	32	22	764
1967	2,699	92	51	15	2,857
1968	2,567	82	49	0	2,698
1969	3,914	90	47	11	4,062
1970	4,096	90	44	17	4,247
1971	2,975	79	69	57	3,180
1972	454	106	54	0	614
1973	41	103	78	0	222
1974	21	158	144	0	323
1975	17	199	232	0	448
1976	5	178	227	5	415
1977	11	233	237	0	481
1978	31	270	315	0	616
1979	11	229	634	3	877
1980	26	303	1,534	1	1,864
1981	21	273	809	0	1,103
1982	8	348**	574	0	930

*includes Puerto Rico, the Virgin Islands, and Guam.
**includes 17 cases acquired in the United States.

*A "case" is defined as: 1) an individual's first attack of malaria in the United States, regardless of whether or not he/she had experienced previous attacks of malaria while outside the country, and 2) the presence of a positive peripheral blood smear examined in the local or state health department laboratory. Blood smears from doubtful cases were referred to the National Malaria Repository, CDC, for confirmation of the diagnosis. A subsequent attack in the same person caused by a different <u>Plasmodium</u> species is counted as an additional case. A repeated attack in the same person in this country caused by the same species is not considered as an additional case. The number of cases of malaria in U.S. civilians increased from 273 in 1981 to 348 in 1982, a 27.5% increase. In contrast, malaria in foreign civilians declined from 809 reported cases in 1981 to 574 cases in 1982, a 29.0% decrease.

Only 17 of the 930 cases acquired their infection in the United States. Congenital transmission occurred in 7 cases, and infection through blood transfusion occurred in 9 cases. In one instance, a laboratory worker acquired an infection with <u>P. falciparum</u> from infected mosquitoes at an insectary.

Two deaths attributable to malaria were reported for 1982 compared with 7 fatal cases in 1981.

The <u>Plasmodium</u> species could be determined in 875 of the 930 cases (94.1%). There were no significant changes in the ratios of cases caused by any <u>Plasmodium</u> species from 1981 to 1982. <u>P. vivax</u> was identified in 66% and <u>P. falciparum</u> in 20% of the infected individuals (Table 2).

Table 2 All Malaria Cases by Plasmodium Species, United States, 1981-1982

	198	31	198	32
Species	Total	Percent	Total	Percent
P. vivax	819	74.3	613	65.9
P. falciparum	164	14.9	189	20.3
P. malariae	34	3.1	43	4.6
P. ovale	10	0.9	25	2.7
Mixed	1	0.1	5	0.5
Undetermined	75	6.7	55	5.9
TOTAL	1,103	100.0	930	100.0

The countries of origin of the 930 cases are listed in Table 3. The geographic distribution of the malaria cases within the United States is shown in Figure 2 by the State in which the patient first developed clinical symptoms of malaria.

Table 3 Malaria Cases by Distribution of Plasmodium Species and Area of Acquisition, United States, 1982*

*Includes Puerto Rico, Virgin Islands and Guam

**Country Unspecified

The exact interval between the date of arrival in the United States and the date of onset of illness was known for 756 of the imported cases for which the infecting <u>Plasmodium</u> species was identified also. Clinical malaria developed within 1 month after arrival in 83.4% of the patients with <u>P</u>. <u>falciparum</u> malaria and in 30.2% of the patients with <u>P</u>. <u>vivax</u> infections (Table 4). Only 5.9% of the 756 patients became ill 1 year or more after their arrival in the United States.

Table 4	Imported Malaria	Cases by Interval	between Date of	Entry and Onset
	of Illness and b	y Plasmodium Specie	es, United States	, 1982

PLASMODIUM SPECIES								
Inter	val							
(in mor	iths)	vivax (%)	falciparum(%)	malariae(%)	ovale(%)	Total(%)		
< 1	163	(30.2)	136 (83.4)	9 (26.5)	3 (15.0)	311 (41.4)		
1-2	114	(21.2)	16 (9.8)	8 (23.5)	3 (15.0)	141 (18.7)		
3-5	87	(16.1)	7 (4.3)	5 (14.7)	8 (40.0)	107 (14.2)		
6-11	138	(25.6)	3 (1.8)	7 (20.6)	4 (20.0)	152 (20.1)		
<u>></u> 12	37	(6.9)	1 (0.6)	5 (14.7)	2 (10.0)	45 (5.9)		
TOTAL	5 39	(100.0)	163 (100.0)	34 (100.0)	20 (100.0)	756 (100.0)		

The 2 deaths due to malaria that were reported in the 930 cases in 1982 are discussed in Section VII.

IV. MILITARY MALARIA

Eight cases of malaria were reported in U.S. military personnel in 1982. The Army accounted for 2 cases, the Navy for 3, the Air Force for 1 and the Marine Corps for 2 cases.

V. IMPORTED MALARIA IN CIVILIANS

Malaria in U.S. citizens accounted for 331 of the 905 imported cases in civilians (36.6%), whereas 574 of the cases occurred in citizens of other countries (Table 5). A total of 131 of the 331 imported cases in U.S. civilians were acquired in Africa (39.6%), and 26.9% of the 331 cases were acquired in Asia.

Asia was the area of acquisition of infection in 46.7% of the 574 cases in foreign civilians. Infections acquired in India accounted for 172 of the malaria infections in foreign civilians as compared with 236 cases in 1981.

	United States		Foreigners		Total	
Area of Acquisition	Cases	Percent	Cases	Percent	Cases	Percent
Africa	131	39.6	84	14.6	215	23.8
Asia	89	26.9	334	58.2	423	46.7
Central America	43	13.0	115	20.0	158	17.5
Caribbean	19	5.7	4	0.7	23	2.5
Mexico	21	6.3	30	0.5	51	5.6
South America	9	2.7	4	0.7	13	1.4
Oceania	18	5.4	1	0.1	19	2.1
Unknown	1	0.3	2	0.3	3	0.3
TOTAL	331	100.0	574	100.0	905	100.0

Table 5 Imported Malaria Cases in Civilians, by Area of Infection, United States, 1982

The major changes in several categories of imported civilian cases from the corresponding 1981 data are shown in Table 6. Among imported U.S. civilian cases the major increases were observed among tourists (up 216%) and missionaries (up 100%). Malaria cases in the categories of U.S. business representatives and teachers/students declined by 28 and 35%, respectively.

The 29% decline from 1981 of imported malaria cases in foreign civilians is due to decline of malaria cases among refugees, from 350 cases in 1981 to 136 cases in 1982 (62%).

		U.S. Cit	izens	zens		Foreign Civ	ilians	lians	
	1981			1982		1981		1982	
Category	Cases	(Percent)	Cases	(Percent)	Cases	(Percent)	Cases	(Percent)	
Tourist	25	(9.6)	79	(23.9)	7	(0.9)	6	(1.0)	
Business Representative	32	(12.3)	23	(6.9)	13	(1.6)	11	(1.9)	
Government Employee	9	(3.5)	9	(2.7)	2	(0.2)	4	(0.7)	
Missionary	16	(6.2)	32	(9.7)	1	(0.1)	2	(0.3)	
Peace Corps	10	(3.8)	12	(3.6)	_	-	-	-	
Seamen	-	-	4	(1.2)	8	(1.0)	5	(0, 9)	
Teacher/Student	49	(18.8)	32	(9.7)	57	(7.1)	38	(6.6)	
Refugee	-	-	-	-	350	(43.4)	136	(23.7)	
Other	77	(29.6)	67	(20.2)	162	(20.0)	119	(20.7)	
Unknown	42	(16.2)	73	(22.0)	209	(25.7)	253	(44.1)	
TOTAL	260	(100.0)	331	(100.0)	809	(100.0)	574	(100.0)	

Table 6 Imported Malaria Cases by Category, United States, 1981-1982

VI. MALARIA ACQUIRED IN THE UNITED STATES

A. Congenital malaria

Seven cases of congenital malaria with onset of illness in 1982 were reported in the United States. There were 10 cases of congenital malaria with onset of illness in 1981. All cases in 1982 were caused by <u>P. vivax</u>. All of the mothers had come to the United States from malarious countries (i.e., Mexico, El Salvador, Laos, and India). The onset of illness varied from 20 days to 2 months after birth. All patients were treated with chloroquine.

<u>Case 1</u>--On January 14, 1982, a 20-day-old boy was admitted to a Connecticut hospital with fever, hepatosplenomegaly and jaundice. Peripheral blood smears contained <u>P. vivax</u> parasites. The infant was treated with chloroquine and had an uneventful recovery. During the second and third trimester of her pregnancy, the mother, who had arrived from India in October 1980, had a history of six episodes of chills and fever for which no treatment was sought.

(Reported by M. Masiello, M.D., Bridgeport, Connecticut, and the Connecticut Department of Health.)

<u>Case 2</u>--A 25-day-old boy was admitted to a California hospital on June 16, 1982, with a 4-day history of fever. <u>P. vivax</u> parasites were found in a peripheral blood smear. The infant was treated with chloroquine. The mother, a 22-year-old woman from El Salvador, had had vivax malaria diagnosed in California at 6 months of pregnancy; she had been treated with chloroquine initially, and then given chloroquine tablets to take once weekly until delivery.

(Reported by Harbor General Hospital, Torrance, California, K. Mackey, M.P.H., Long Beach City Health Department, and R.A. Murray, Dr.P.H., California Department of Health Services.)

Case 3--A 45-day-old boy was admitted to a New York City hospital on March 24, 1982, with a 3-day history of fever. The baby had splenomegaly, and <u>P. vivax</u> parasites were found in a blood smear. The patient was treated with chloroquine. The mother, a 32-year-old Mexican woman, had come to the United States in October 1981. She had a history of fever and chills at 8 months gestation. No parasites were found in the peripheral blood of the mother, but she was treated with chloroquine and primaquine.

(Reported by N. Amaran, M.D., St. Lukes-Roosevelt Hospital, New York City, and the New York City Department of Health.)

<u>Case 4--A 2-month-old boy with a history of fever and anemia was</u> admitted on March 20, 1982, to an Illinois hospital. <u>P. vivax</u> parasites were found in a peripheral blood smear. The mother was a Laotian refugee with a history of fever and chills intermittently during the pregnancy for which no treatment was sought. No parasites were found in the peripheral blood, but she was treated with chloroquine and primaquine.

(Reported by C. Balbarin, M.D., Joliet, Illinois, and the Illinois Department of Health.)

<u>Case 5--On February 17, 1982, an infant boy was admitted to a</u> Massachusetts hospital with fever and anemia. <u>P. vivax</u> parasites were found in a peripheral blood film. The patient was treated with chloroquine. The mother had come to the United States from India and had a history of malaria about 1 year prior to the infant's birth.

(Reported by Lawrence C. Kaplan, M.D., Boston, Massachusetts, and the Massachusetts Health Department.)

Case 6--A 40-day-old girl was admitted to a California hospital in December 1982, with fever and hemolysis. <u>P. vivax</u> parasites were identified on a peripheral blood smear. The patient was treated with chloroquine.

The mother had emigrated to the United States from Guatemala and had a history of fever 1 month prior to the delivery. When tested in January 1983, her blood smears were negative, but serologic testing showed an IIF titer of 1:256 for <u>P. vivax</u> and was negative for other <u>Plasmodium</u> species. She moved to Mexico and was lost to followup.

(Reported by A. Tyndell, M.D., Torrance, California, N. Tormey, M.P.H., Los Angeles County Health Department, and R.A. Murray, Dr.P.H., California Department of Health Services.)

<u>Case 7--A 19-day-old boy was admitted on November 14, 1982, to a</u> California hospital because of fever and increasing drowsiness. Peripheral blood smears contained <u>P. vivax</u> parasites. The infant was treated with chloroquine and primaquine. The mother had arrived in the United States in March 1982 from El Salvador and had experienced a parasitologically confirmed episode of vivax malaria in August 1982. She was treated with chloroquine.

(Reported by the Los Angeles County/USC Medical Center, Los Angeles, California, M. Tormey, M.P.H., Los Angeles County Health Department and R.A. Murray, Dr.P.H., California Department of Health Services.)

B. ACCIDENTAL LABORATORY INFECTION

On June 30, 1982, a 52-year-old man, who worked as a laboratory technician in a research insectary complex in Washington, D.C., was admitted to a hospital with a 9-day history of fatigue, headache, and periodic episodes of fever. P. falciparum parasites were found on a peripheral blood smear. The patient was treated with the antimalarial, mefloquine, because it was considered likely that the patient had acquired the infection from <u>Anopheles</u> <u>freeborni</u> mosquitoes that had been infected in the insectary with a strain of multi-drug-resistant P. falciparum malaria. His recovery was uneventful.

The patient denied ever having had malaria, leaving the United States during the preceding 4 years, ever receiving a blood transfusion, or using intravenous drugs.

(Reported by Capt. J.L. Williams, MSC, Department of Immunology, Walter Reed Army Institute of Research (WRAIR); Major Bruce T. Dennis, M.D., M.CC., Division of Infectious Diseases, Department of Medicine, Walter Reed Army Medical Center; Thomas R. Burkot, Ph.D., David E. Hayes and Imogene Schneider, Ph.D., Department of Entomology, WRAIR; and the District of Columbia Health Department.)

C. TRANSFUSION MALARIA

A total of 9 cases of malaria following administration of blood and/or blood products with onset in 1982 in the United States were reported.

<u>Case 1</u>--On May 7, 1982, a 29-year-old woman developed chills and fever. <u>P. malariae</u> parasites were identified on a peripheral blood smear. She was successfully treated with chloroquine. The patient denied ever traveling outside the United States. Following an auto accident on March 16, she received 18 units of red blood cells and 10 units of platelets.

Records of the 28 donors whose blood or platelets had been administered indicated that none had given a history of malaria, malaria therapy, or prophylaxis, nor had any been outside the United States for the preceding 3 years. Serum specimens and blood smears were obtained from 26 of the 28 donors. Serum specimens from these donors were negative when tested for the presence of antibodies to malaria with the indirect immunofluorescence (IIF) test, except for serum from one donor of a unit of packed red blood cells; this serum had a titer to <u>P. malariae</u> of 1:1024. The serologically implicated donor was a student from Liberia who had been in the United States since 1978. Although no parasites could be found in peripheral blood films, he was treated with chloroquine.

(Reported by J. Santoro, M.D., Bala Cynwyd, Pennsylvania; M. Dahlke, M.D., American Red Cross Blood Services, Philadelphia; and the Pennsylvania Department of Public Health.)

<u>Case 2</u>--On May 29, 1982, a 74-year-old man in a hospital in Pennsylvania began to experience fever, chills, shaking, and anorexia. Peripheral blood films showed the presence of <u>P</u>. <u>malariae</u> parasites. The patient had never had malaria and had not been out of the United States during the preceding 4 years. He was treated with chloroquine and recovered from his malaria infection.

The patient had been hospitalized from March 14 to 24, 1982, for repair of a ruptured abdominal aneurysm, during which time he received 10 units of platelets. He was readmitted on March 27, 1982, with phlebitis. While in the hospital, he experienced upper gastrointestinal bleeding. During this period, he received a total of 23 units of packed red cells.

Examination of the information available on the donors showed that a unit of platelets had been obtained from the same donor implicated in Transfusion Case 1. It was assumed that this unit of platelets was responsible for this infection.

(Reported by W. Murphy, D.O., Oxford, Pennsylvania, M. Dahlke, M.D., American Red Cross Blood Services, Philadelphia, and the Pennsylvania Department of Health.)

<u>Case 3</u>--On August 15, 1982, a 63-year-old man received 26 units of blood and 15 units of platelets during and shortly after repair of a ruptured abdominal aortic aneurysm. He developed episodes of fever and nausea 19 days later, and <u>P. ovale</u> parasites were identified in a peripheral blood smear. The patient was treated with chloroquine and had an uneventful recovery from his malaria infection. The patient had served with the U.S. Army in Korea in 1950, 1953, and 1955. During that time, he took malaria chemoprophylaxis intermittently but he denied having had clinical episodes of malaria before.

Only one blood donor had given a history of travel to malarious countries. This donor served with the Peace Corps in Sierra Leone from 1977 to 1979, during which time he took chloroquine as chemoprophylaxis. He had not taken primaquine to prevent relapses but denied having experienced febrile illnesses compatible with malaria since leaving Africa. Of the 39 serum samples that were available from identified donors, only the specimen from this individual was positive when tested with the IIF test for the presence of antibodies to malaria. The IIF titer against <u>P</u>. ovale antigen was 1:256. Repeated blood smears from this donor were negative, but he was treated presumptively with chloroquine and primaquine.

(Reported by Lt. Col. B. Johnson, Major J.H. Brown and Major R. Yoedino, Martin Army Hospital, Ft. Benning, Georgia, and R.K. Sikes, D.V.M., State Epidemiologist, State Department of Health, Georgia.)

<u>Case 4--On</u> June 11, 1982, a 73-year-old woman developed chills and fever. Examination of the peripheral blood showed <u>P. falciparum</u> parasites. The patient was successfully treated with chloroquine, quinine, and sulfadoxine-pyrimethamine.

The patient denied recent travel. However, between January 29 and June 3, she had received 11 units of packed red blood cells as therapy for chronic lymphocytic leukemia and gastrointestinal bleeding. A review of blood donors identified a man who had returned from a visit to Ghana 2 weeks prior to his blood donation. Although his peripheral blood smear was negative for parasites, his serum had an IIF titer of 1:16,384 when tested for malaria antibodies.

(Reported by M. Tapper, M.D., G. Bahr, M.D., Lenox Hill Hospital, New York City, and the New York City Department of Health, New York, New York.)

<u>Case 5--On April 20, 1982, a 46-year-old woman was admitted to an</u> Illinois hospital with a 1-week history of fever and chills. A peripheral blood smear was positive for <u>P. malariae</u>. She was successfully treated with chloroquine.

The patient denied traveling outside the continental United States, although she was born in Puerto Rico. She was being treated for chronic renal failure and had been maintained on hemodialyis for 2 years. She had received over 20 units of blood during 1981, following multiple hospitalizations for kidney transplantation, graft failure, and postoperative complications. Because of the length of time beween the transfusions and onset of acute malaria, all blood donors were not found, and the supposed responsible donor was not identified.

(Reported by J. Semel, M.D., St. Joseph Hospital, and U. Diekamp, M.D., University of Illinois Med Center, Chicago, IL, and the Illinois Department of Public Health, Springfield, Illinois.) <u>Case 6</u>--On May 31, 1982, a 3-day-old infant boy born in Los Angeles, California, received whole blood during an exchange transfusion for the treatment of hyperbilirubinemia. Thirty-four days later the child was noted to be febrile, anemic, and thrombocytopenic. A blood film demonstrated trophozoites of <u>Plasmodium vivax</u>. After treatment with chloroquine, he made a complete recovery.

The patient's mother had never been out of the United States. The blood donor was a 22-year-old man from Guatemala who had come to the United States in September 1981. He reported having episodes of shaking chills, nausea, and vomiting between May 1981 and April 1982 for which he had received chloramphenicol. Pyrimethamine was subsequently given when symptoms recurred. In May 1982 he donated blood, at which time he was asymptomatic. He did not give an accurate history during the screening for blood donation. A blood smear subsequently taken from the donor on July 13 showed <u>P. vivax</u>, and he was treated with chloroquine and primaquine.

(Reported by I.A. Schulman, M.D., USC-LA County Medical Center, Los Angeles, U. Ben-Zeev, M.D., Encino, California, I.A. Whitney, R.N., M. Anglim, PHR, M. Tormey, E. Soprapena, R.N., N. Sochi, M.D., L. Gong, M.D., Los Angeles County Health Department, R.A. Murray, Dr.P.H., California Department of Health Services, Berkeley, California.)

<u>Case 7</u>--On August 3, 1982, a 2-day-old infant boy born in Los Angeles, California, received a whole blood exchange transfusion for hemolytic disease of the newborn and resultant hyperbilirubinemia. A temperature of 103° F developed 14 days later. A blood film showed parasites of <u>P. vivax</u>, and the patient was treated with chloroquine.

The donor was a 27-year-old man from Central America who had been in the merchant marines and was in El Salvador, Brazil, and Mexico from 1978 to May 1981. He had a history of "flu" in 1980 for which he was treated with an unknown drug. He had donated blood at the end of July 1982. Follow-up blood smears were positive for malaria parasites, and he was treated with chloroquine and primaquine. The mother had no antibodies to malaria.

(Reported by I.A. Schulman, M.D., R. Maris, USC-LA County Medical Center, Los Angeles, N. Tormey M.P.H., Los Angeles County Health Department, and R.A. Murray, Dr.P.H., California Department of Health Services, Berkeley, California.)

<u>Case 8--On</u> February 2, 1982, a 10-week-old infant was diagnosed with <u>P</u>. <u>malariae</u> after a febrile illness. The baby had been born in Massachusetts, and had been hospitalized for a month after birth for prematurity and respiratory distress. The child had received multiple blood transfusions during that time.

Serologic testing of blood from 11 identified blood donors demonstrated a positive IFA antibody titer from only 1 donor; this donor was a student who had come to the United States from Uganda 8 years prior to blood donation. Examination of the donor's blood showed <u>P. malariae</u> parasites. Both donor and recipient were successfully treated with chloroquine.

(Reported by L. Wolfe, M.D., Boston Children's Hospital, Boston, Massachusetts; and the Massachusetts State Department of Health.)

<u>Case 9--Screening</u> of recipients who had received blood from the donor implicated in Case 8 revealed a second premature infant who had received a volume of blood from that donor. <u>P. malariae</u> parasites were found in the peripheral circulation, and the infant was successfully treated with chloroquine.

(Reported by L. Wolfe, M.D., Boston Children's Hospital, Boston, Massachusetts; and the Massachusetts State Department of Health.)

VII. MALARIA DEATHS IN THE UNITED STATES

Two deaths in 1982 due to malaria were reported in the United States; 7 fatal cases were reported in 1981.

<u>Case 1--A 42-year-old man who was a missionary was admitted to a Nigerian</u> hospital on July 26, 1982, with a history of nausea and vomiting of 2 days duration and oliguria of 1 week duration. He did not respond to conservative treatment and was evacuated to a New York hospital on July 31 in a comatose state and in renal failure. He was diagnosed as having meningitis, hepatosplenomegaly, renal failure, and disseminated intravascular coagulation. Blood smears on July 31 and August 1 reportedly did not show malaria parasites. The patient died on August 2. Microscopic examination of spleen-smears revealed much pigment and objects that appeared to be aberrant malaria trophozoites as well as a P. falciparum gametocyte.

(Reported by Joseph J. Guarnieri, M.D., and P. Della-Latta, M.D., Queens Hospital Center, Jamaica, New York.)

<u>Case 2</u>--On August 18, 1982, a 29-year-old woman who was a resident of California developed weakness, chills, and fever. The patient was not hospitalized and died on August 31, 1982. Autopsy was performed by the Los Angeles Department of Chief Medical Examiner. In the lungs, heart, adrenals, cerebral cortex, bone marrow, and kidney, large numbers of red cells contained a single and sometimes 2 or 3 irregular ovoid basophilic bodies, usually less than 1/10 of the diameter of the red cell. These bodies were consistent with the trophozoite stage of <u>Plasmodium</u>. Schizont forms were rare. Large amounts of brownish-yellow granular pigment were noted in the vessels, heart adrenals, pancreas, cerebral cortex, bone marrow, kidney, and liver. The IIF test on postmortem blood gave a titer of \geq 1:4096 to <u>P. falciparum</u>, 1:256 to <u>P.</u> <u>malariae</u>, 1:64 to <u>P. ovale</u> and \leq 1:64 to <u>P. vivax</u>. The cause of death, as determined by the Medical Examiner's Office, was acute hemolytic anemia due to anemia, consistent with <u>P. falciparum</u> malaria. The patient had lived in Iran from 1979 until August 3, 1982, and arrived on August 13 in the United States via Pakistan and Spain.

(Reported by Ira A. Shulman, M.D., Los Angeles County/University of Southern California Medical Center; M.P. Tormey, M.P.H., B. Agee, M.D., Los Angeles County Department of Health Services; R. Murray, Dr.P.H., California Department of Health Services.)

VIII. PREVENTION OF MALARIA

Guidelines for the "Prevention of Malaria in Travelers" have been published in a Supplement to the <u>Morbidity and Mortality Weekly Report</u> (MMWR), dated April 16, 1982, Vol. 3, No. S, p. 24S. This Supplement also provides information about countries and, where applicable, areas within each country, where malaria risk exists. In addition, areas in the world where chloroquineresistant strains of <u>P. falciparum</u> are known to exist are listed. "Revised Recommendations for Malaria Chemoprophylaxis for Travelers to East Africa" have been published in the MMWR issue of June 25, 1982, Vol. 31, No. 24. Copies of these issues of the MMWR may be obtained upon request from the Malaria Branch, Division of Parasitic Diseases, Center for Infectious Diseases, Centers for Disease Control, Atlanta, GA 30333.

IX. MICROSCOPIC DIAGNOSIS OF MALARIA

Early diagnosis of malaria requires a high level of clinical suspicion and, in particular, a comprehensive travel history taken from every patient with a fever of unknown origin. Once malaria is suspected, a Giemsa-stained smear of peripheral blood should be examined for the presence of parasites. Since the accuracy of diagnosis is dependent on the quality of the blood film, the following guide is offered for the proper preparation of thick and thin blood smears.

1. Manufacturers' "pre-cleaned" slides are not considered clean enough for use in malaria diagnosis. Before use, wash these slides in mild detergent, rinse them thoroughly in warm running water, then in distilled water, and dip them in ethyl alcohol (90% to 95%). Then, wipe slides dry with a lintless cloth or tissue for immediate use or store them in 95% alcohol until needed.

 Clean the patient's finger with alcohol and wipe the finger dry with a clean cloth or gauze.

3. After puncturing the finger with the blood lancet, allow a large globule of blood to form.

4. Place the cleaned surface of the slide against the drop of blood and with a quick circular motion, make a film the size of a dime in the middle third of 1 end of the slide. Ordinary newsprint should be barely legible through such a wet drop (Fig. 3). (Excessive mixing or stirring with a second slide leads to distortion of blood cells and parasites.)

5. Wipe the finger dry and gently squeeze a <u>small</u> drop of blood from the puncture, placing it at the edge of the middle third of the same slide (Fig. 4).

6. Apply a clean "spreader" slide to the edge of the <u>small</u> drop at a 45° angle and allow the blood to extend about two-thirds of the slide width; then, keeping even contact, push the spreader forward along the slide. This will produce an even layer of red blood cells with a "feathering" at the lower edge (Fig. 5).

 While the thick blood film dries (minimum of 6 hours at room temperature)*, keep the film horizontal and protected from dust and insects.

8. Label the slide in the upper part of the thin film with the date and the name or initials of the patient as illustrated (Fig. 5).

*If a rapid diagnosis is desired, make the thick and thin films on separate slides. The thin film can be air dried, fixed with methyl alcohol and stained immediately. If no parasites are found on the thin film, examine the thick film for organisms not detected on the thin preparation.

Fig. 3



Fig. 5



ACKNOWLEDGMENT

The Malaria Surveillance Report, prepared annually at the Centers for Disease Control, is based on information provided in individual case reports. T excellent support given to malaria surveillance by state and local health departments and personnel of the preventive medicine services of the U.S. Army, Navy, and Air Force is greatly appreciated.

STATE AND TERRITORIAL EPIDEMIOLOGISTS

Key to all disease surveillance activities are the State and Territorial Epidemiologists. Their contributions to this report are gratefully acknowledged.

Alabama Alaska Arizona Arkansas California Colorado Connecticut Delaware District of Columbia Florida Georgia Guam Hawaii Idaho Illinois Indiana Iowa Kansas Kentucky Louisiana Maine Maryland Massachusetts Michigan Micronesia* Minnesota Mississippi Missouri Montana Nebraska Nevada New Hampshire New Jersey New Mexico New York State New York City North Carolina North Dakota Northern Mariana Islands* Ohio Oklahoma Oregon Palau* Pennsylvania Puerto Rico Rhode Island South Carolina South Dakota Tennessee Texas Utah Vermont Virginia Virgin Islands Washington West Virginia Wisconsin Wyoming

Wallace E. Birch, DVM John P. Middaugh, MD Philip M. Hotchkiss, DVM, Acting John Paul Lofgren, MD James Chin, MD Richard S. Hopkins, MD Vernon D. Loverde, MD Paul R. Silverman, DrPH Martin E. Levy, MD Jeffrey J. Sacks, MD, Acting R. Keith Sikes, DVM Robert L. Haddock, DVM Mona Bomgaars, MD, Acting Charles D. Brokopp, DrPH Byron J. Francis, MD Charles L. Barrett, MD Laverne A. Wintermeyer, MD Donald E. Wilcox, MD M. Ward Hinds, MD Charles T. Caraway, DVM Kathleen F. Gensheimer, MD, Acting Ebenezer Israel, MD Nicholas J. Fiumara, MD Kenneth R. Wilcox, Jr., MD Eliuel K. Pretrick, MO, MPH Andrew G. Dean, MD William E. Reicken, Jr., MD H. Denny Donnell, Jr., MD John S. Anderson, MD, Acting Paul A. Stoesz, MD John H. Carr, MD, Acting John M. Horan, MD William E. Parkin, DVM Jonathan M. Mann, MD Richard Rothenberg, MD Stephen M. Friedman, MD Martin P. Hines, DVM Kenneth Mosser Jose T. Villagomez, MO Thomas J. Halpin, MD Gregory R. Istre, MD John A. Googins, MD Anthony H. Polloi, MO, Acting Ernest J. Witte, VMD Antonio Hernandez, MD Jason Weisfeld, MD, Acting Richard L. Parker, DVM Kenneth A. Senger Robert H. Hutcheson, Jr., MD Charles E. Alexander, MD, Acting Richard E. Johns, Jr., MD Richard L. Vogt, MD Grayson B. Miller, Jr., MD John N. Lewis, MD John M. Kobayashi, MD Loretta E. Haddy, MS Jeffrey P. Davis, MD Harry C Crawford, MD