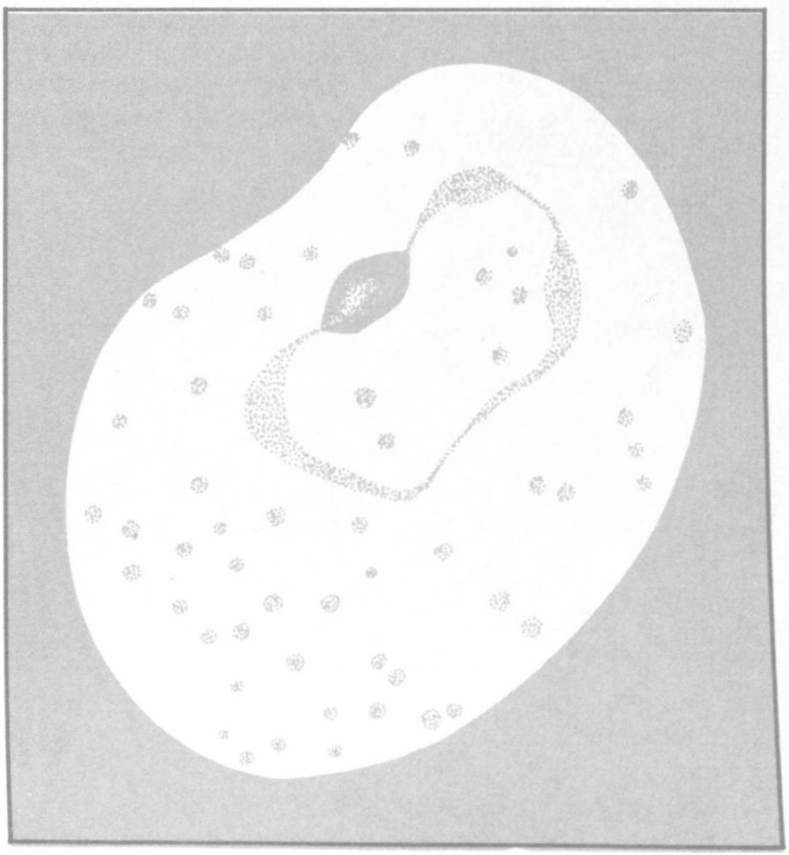
ANNUAL SUMMARY 1981

Issued November 1982

CENTERS FOR DISEASE CONTROL

MALARIA

SURVEILLANCE



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

P R E F A C E

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:

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SUGGGESTED CITATION

Centers for Disease Control: Malaria Surveillance Annual Summary 1981
Issued November 1982

I. SUMMARY

During 1981, 1,103 persons are known to have become ill with malaria in the United States. This compares with 1,864 cases reported in 1980, a decline of 40.8%, primarily due to fewer imported cases of malaria in foreigners. A total of 809 cases with onset in 1981 were reported in foreigners compared with 1,534 in 1980, a 47.3% decrease.

Plasmodium vivax was the parasite identified in 74% of the 1,103 cases, and P. falciparum was identified in 15% of the cases. P. malariae and P. ovale were reported in 3% and 1% of cases, respectively, the remaining cases being undetermined as to species involved.

Only 13 of the 1,103 cases acquired their infection in the United States. Congenital transmission occurred in 10 cases, and infection through blood transfusion occurred in 2 cases. In one isolated case in California, the source of infection could not be determined.

Seven deaths attributed to malaria were reported for 1981 compared with 1 fatal case in 1980. All deaths occurred in civilians; 5 of the patients had been infected with P. falciparum, and in 2 persons the Plasmodium species was reported to be P. vivax. The case-fatality ratio for P. falciparum malaria was 3%.

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.

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

III. GENERAL SURVEILLANCE

A total of 1,103 cases* with onset of illness in 1981 in the United States were reported to the Division of Parasitic Diseases, Center for Infectious Diseases, Centers for Disease Control; this represents a 40.8% decline over the 1864 cases reported for 1980. Only 21 of the cases (1.9%) occurred in U.S. military personnel. Civilian cases accounted for the majority of cases in each year since 1973 (Table 1).

Malaria in foreign civilians accounted for 809 (73.3%) of all reported cases (Table 1). There was a decline of 47.3% in the number of cases among foreign civilians compared with 1980. In contrast, the decline of malaria in U.S. civilians was only 9.9% (Figure 1).

Fig. 1 CASES OF MALARIA IN U.S. CIVILIANS AND FOREIGNERS, UNITED STATES, 1970–1981

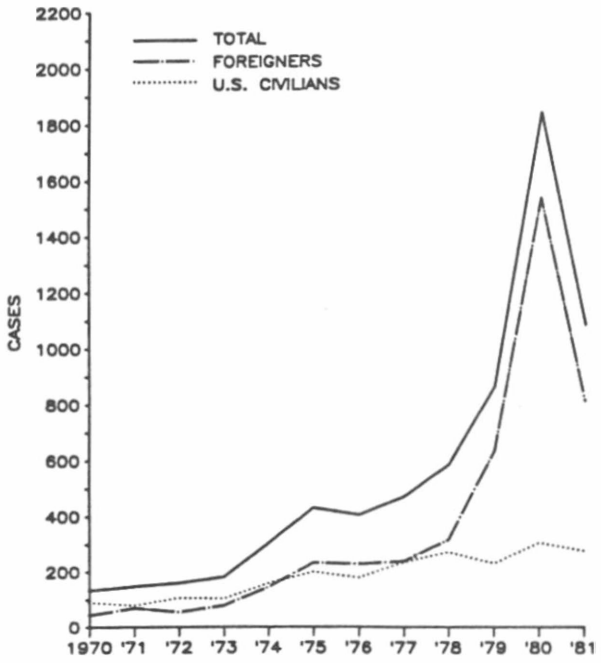


Table 1 All Primary Malaria Cases in Civilians and Military Personnel with Onset of Illness in the United States, 1966-1981*

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

*includes Puerto Rico, the Virgin Islands, and Guam.
**includes 3 cases acquired in the United States.
*** includes 10 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 Plasmodium 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.

Ten congenital cases of malaria were reported for 1981, which is more than have been recorded in any year since 1957 when the Communicable Disease Center (now the Centers for Disease Control) initiated publication of annual malaria surveillance reports. All congenital cases had P. vivax malaria; 8 were born to Indochinese refugee mothers, and 2 were born to mothers from Central and South America. Two cases of transfusion malaria were reported, one was due to P. falciparum and the other to P. vivax. One cryptic case of P. vivax malaria was reported from California.

The Plasmodium species could be determined in 1,028 of the 1,103 cases (93.2%). There were no significant changes in the ratios of cases caused by any Plasmodium species between 1981 and 1980. P. vivax was identified in 74% and P. falciparum in 15% of the infected individuals (Table 2).

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

Species	Total	Percent
P. vivax	819	74.3
P. falciparum	164	14.9
P. malariae	34	3.1
P. ovale	10	0.9
Mixed	1	0.1
Undetermined	75	6.7
TOTAL	1,103	100.0

The countries of origin of the 1,103 cases are listed in Table 3. The majority of cases were acquired in Asia (61.6%), followed by infections acquired in Africa (16.4%) and Central America (15.4%).

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. The large proportion of cases which occurred in California (42.1%) was due to the large influx there of Indochinese refugees and persons from India.

Fig.2 GEOGRAPHIC DISTRIBUTION OF MALARIA CASES WITH ONSET IN UNITED STATES, 1981

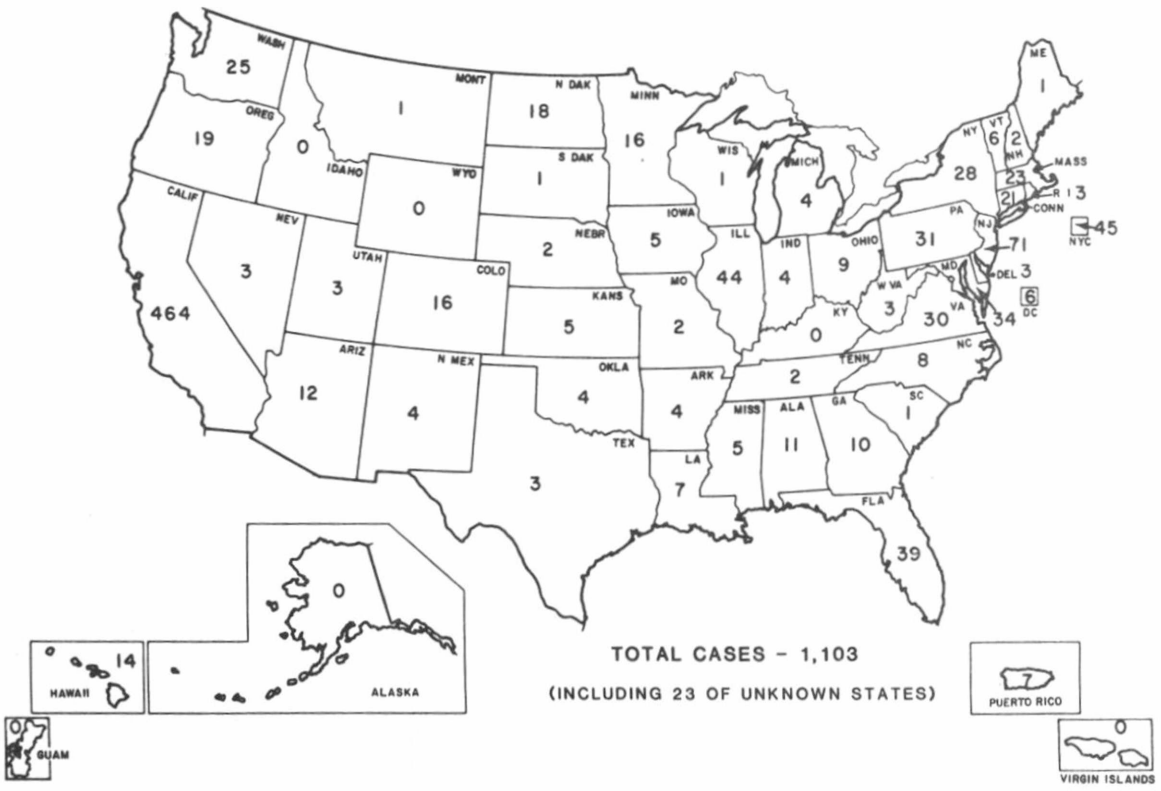


Table 3 Malaria Cases by Distribution of *Plasmodium* Species and Area of Acquisition, United States, 1981*

Area of Acquisition	*vivax*	*falciparum*	*malariae*	*ovale*	Mixed	Unknown	Total
AFRICA	**48**	**103**	**8**	**7**	**1**	**14**	**181**
Africa, East**	1	1	1	0	0	1	4
Africa, West & Central**	5	9	2	0	0	1	17
Africa, Unspecified**	5	9	0	2	0	2	18
Cameroon	1	1	0	0	0	0	2
Central African Republic	1	0	0	0	0	0	1
Congo	0	1	0	0	0	0	1
Egypt	1	0	0	0	0	0	1
Ethiopia	6	0	0	0	0	0	6
Ghana	6	10	2	0	0	0	18
Ivory Coast	1	0	0	0	0	0	1
Kenya	1	19	0	1	0	1	22
Liberia	7	5	0	1	1	1	15
Libya	1	0	0	0	0	0	1
Niger	0	1	0	0	0	0	1
Nigeria	5	30	2	3	0	7	47
Senegal	0	2	0	0	0	0	2
Sierra Leone	2	1	0	0	0	1	4
Sudan	3	2	0	0	0	0	5
Tanzania	0	9	0	0	0	0	9
Uganda	1	2	1	0	0	0	4
Upper Volta	1	0	0	0	0	0	1
Zaire	0	1	0	0	0	0	1
ASIA	**580**	**27**	**21**	**3**	**0**	**48**	**679**
Asia, Southeast**	252	18	8	3	0	21	302
Middle East**	0	0	1	0	0	0	1
Afghanistan	2	0	0	0	0	1	3
Bangladesh	10	0	0	0	0	0	10
Burma	2	0	0	0	0	0	2
China	0	0	0	0	0	1	1
India	259	8	10	0	0	18	295
Indonesia	7	0	0	0	0	1	8
Israel	0	0	0	0	0	1	1
Kampuchea	6	1	0	0	0	0	7
Lebanon	1	0	0	0	0	0	1
Malaysia	1	0	0	0	0	0	1
Pakistan	11	0	0	0	0	1	12
Philippines	9	0	0	0	0	2	11
Sri Lanka	3	0	0	0	0	0	3
Thailand	1	0	0	0	0	1	2
Vietnam	16	0	2	0	0	1	19
CENTRAL AMERICA AND CARIBBEAN	**134**	**25**	**4**	**0**	**0**	**7**	**170**
Belize	1	0	0	0	0	0	1
Carribbean, Unspec.**	0	0	1	0	0	0	1
Central Amer. Unspec.**	23	2	1	0	0	1	27
Dominican Republic	0	0	1	0	0	0	1
El Salvador	68	2	1	0	0	0	71
Guatemala	15	2	0	0	0	1	18
Haiti	3	15	0	0	0	4	22
Honduras	14	3	0	0	0	1	18
Nicaragua	9	1	0	0	0	0	10
Panama	1	0	0	0	0	0	1
NORTH AMERICA	**33**	**1**	**0**	**0**	**0**	**1**	**35**
Mexico	21	0	0	0	0	1	22
United States	12	1	0	0	0	0	13
SOUTH AMERICA	**5**	**1**	**0**	**0**	**0**	**2**	**8**
South America, Unsp.**	1	0	0	0	0	0	1
Brazil	0	0	0	0	0	1	1
Chile	0	1	0	0	0	0	1
Colombia	3	0	0	0	0	0	3
Surinam	1	0	0	0	0	1	2
OCEANIA	**6**	**1**	**0**	**0**	**0**	**0**	**7**
New Guinea	5	1	0	0	0	0	6
Pacific**	1	0	0	0	0	0	1
UNKNOWN	**13**	**6**	**1**	**0**	**0**	**3**	**23**
TOTAL	**819**	**164**	**34**	**10**	**10**	**75**	**1,103**

*includes Puerto Rico, Virgin Islands and Guam
**Country Unspecified

A history of previous illness was obtained in 483 of the 1,090 imported cases (44.3%). Patients with P. vivax malaria were more likely to give a history of previous malaria than those with P. falciparum malaria (45.9% vs 34.1%).

The exact interval between the date of arrival in the United States and the date of onset of illness was known for 798 of the imported cases for which the infecting Plasmodium species was identified also. Clinical malaria developed within 1 month after arrival in 75.3% of the patients with P. falciparum malaria and in 24.7% of the patients with P. vivax infections (Table 4). Only 4.4% of the 798 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 by Plasmodium Species, United States, 1981

Interval (in months)	PLASMODIUM SPECIES									
	vivax	(%)	falciparum	(%)	malariae	(%)	ovale	(%)	Total	(%)
< 1	158	(24.7)	95	(75.3)	8	(34.8)	3	(42.9)	264	(33.1)
1-2	127	(19.8)	18	(14.3)	8	(34.8)	4	(57.1)	157	(19.7)
3-5	141	(21.9)	5	(4.0)	5	(21.7)	0		151	(18.9)
6-11	184	(28.7)	5	(4.0)	2	(8.7)	0		191	(23.9)
≥12	32	(4.9)	3	(2.4)	0	)	0		35	(4.4)
TOTAL	642	(100.0)	126	(100.0)	23	(100.0)	7	(100.0)	798	(100.0)

Seven deaths due to malaria were reported in the 1,103 cases in 1981. These fatal cases are discussed in Section VII.

IV. MILITARY MALARIA

Only 21 cases of malaria were reported in U.S. military personnel in 1981 (Table 5). Since 1974, the number of cases in military personnel has ranged between 5 and 26 per year, and this category accounted for only 1.9% of all cases in 1981.

Table 5 Malaria Cases in Military Personnel, by Branch of Service, United States, 1981

Branch of Service	Cases	(%)
Air Force	0	-
Army	4	19.0
Navy	6	28.6
Marine	9	42.9
Unknown	2	9.5
TOTAL	21	100.0

V. IMPORTED MALARIA IN CIVILIANS

Malaria in U.S. citizens accounted for 260 of the 1,069 imported cases in civilians (24.3%), whereas 809 of the cases occurred in citizens of other countries (Table 6). A total of 103 of the 260 imported cases in U.S. civilians were acquired in Africa (39.6%), and 32.3% of the 260 cases were caused by P. falciparum. As in 1981, U.S. civilians acquired their malaria infection most frequently in Africa during the preceding decade.

Asia was the area of acquisition of infection in 71.5% of the 809 cases in foreign civilians. Infections acquired in India accounted for 236 (51.5%) of the 458 cases which occurred among non-refugee foreigners, and 88.6% of these 236 infections were caused by P. vivax.

Table 6. Imported Malaria Cases in Civilians, by Area of Infection, United States, 1981

Area of Acquisition	United States		Foreigners		Total	
	Cases	Percent	Cases	Percent	Cases	Percent
Africa	103	39.6	76	9.4	179	16.8
Asia	83	31.9	579	71.6	662	61.9
Central America	32	12.3	113	13.9	145	13.7
Caribbean	16	6.2	8	1.0	24	2.2
Mexico	7	2.7	14	1.7	21	1.9
South America	5	1.9	3	0.4	8	0.7
Oceania	7	2.7	-	-	7	0.6
Unknown	7	2.7	16	2.0	23	2.2
TOTAL	260	100.0	809	100.0	1,069	100.0

The number of malaria cases in refugees declined to 350 in 1981 from 1,034 in 1980. Of the 350 imported malaria cases among refugees, 284 (81.1%) were in refugees from Southeast Asia.

Table 7. Imported Malaria Cases in Civilians, by Category, United States, 1981

Category	U.S. Citizens		Foreigners	
	Cases	(percent)	Cases	(percent)
Tourist	25	(9.6)	7	(0.9)
Business Representative	32	(12.3)	13	(1.6)
Government Employee	9	(3.5)	2	(0.2)
Missionary	16	(6.2)	1	(0.1)
Peace Corps	10	(3.8)	-	
Seamen	-		8	(1.0)
Teacher/Student	49	(18.8)	57	(7.1)
Refugee	-		350	(43.4)
Other	77	(29.6)	162	(20.0)
Unknown	42	(16.2)	209	(25.7)
TOTAL	260	(100.0)	809	(100.0)

VI. MALARIA ACQUIRED IN THE UNITED STATES

One cryptic case of malaria, 2 transfusion (induced) malaria cases, and 10 cases of congenital malaria with onset of illness in 1981 were reported in the United States.

A. Cryptic Malaria

On September 5, 1981, a 46-year-old, long-term resident of Yuba County, California, became ill with fever, chills, headache, sore throat, nausea, and abdominal pain. Pharyngitis was diagnosed, and he was treated with penicillin. The symptoms persisted, and on September 17 he still complained of nausea, fever, and shaking chills that recurred daily at about 3:00 p.m. and lasted 30 to 45 minutes.

On examination, he was found to be jaundiced with a tender liver and abdomen. Malaria was suspected, and P. vivax parasites were found on peripheral blood smears. Treatment with chloroquine and primaquine resulted in uneventful recovery.

The patient had no history of blood transfusion, drug abuse, or travel outside the United States. He lived in a semi-rural setting within 1/4 mile of rice fields and orchards. In the spring and summer of 1981 he fished and camped extensively throughout Sutter and Yuba counties and often received mosquito bites.

Examination of peripheral blood smears and serum specimens from the patient's wife, 2 children, and 3 members of an adjacent household did not indicate past or present infection with malaria. Intensive surveillance in the Sutter-Yuba area failed to reveal any other cases of malaria that could have been acquired locally through anopheline mosquitoes. The Sutter-Yuba Mosquito Abatement District reported that A. freeborni (an efficient P. vivax vector) was abundant throughout the summer in Sutter and Yuba counties, including the patient's neighborhood. Mosquito-control efforts in the area included insecticide spraying around the patient's house and insecticide cold-fogging on 3 occasions for a 1/2-mile radius from the house.

Many cases of P. vivax malaria have been detected in Yuba County in recent years in immigrants from India. It is possible that infections in Indian immigrants resulted in P. vivax infection of A. freeborni, resulting in transmission of malaria to this patient.

(Reported by A.F. Taylor, S. Gaspers, L.E. Mahoney, M.D., San Bernardino County Health Deptartment; T. Roswell, M.D., Loma Linda University Medical Center; L.E. Pine, D. Dragoni, M.D., Glenn County Health Department; K. Whitesell, Glenn County Mosquito Abatement District (MAD); J. Buckingham, Diablo Valley MAD; A. Hibbard, M.D., Yuba General Clinic, Marysville; L. Eberhardt, J. Hornstein, MPH., P. Stotler, R.N., M. Cusick, M.D., Sutter-Yuba County Health Department; E. Kauffmann, MPH, Sutter-Yuba MAD; R.R. Roberto, M.D., D. Womeldorf, M.D., California Department of Health Services; J. Chin, M.D., State Epidemiologist, California Department of Health Services; Malaria Branch, Parasitic Diseases Division, Center for Infectious Diseases; Quarantine Division, Center for Preventive Services; and the Field Services Division, Epidemiology Program Office, CDC.)

B. Induced Malaria

The standards for donor selection used by the American Association of Blood Banks and the American Red Cross appear to be effective in limiting the incidence of transfusion malaria. During the period 1971-1980, only 32 cases of transfusion malaria were reported (3.2 cases per year), and in 1981 only 2 such cases were reported.

Case 1--On March 31, 1981, a 53-year-old man was admitted to a Louisiana hospital with a 4-day history of intermittent spiking fevers, shaking chills, sweats, and malaise. P. falciparum parasites were identified on a peripheral smear. He was successfully treated with chloroquine.

The patient had never been outside the United States but had a history of 2 coronary bypass operations, in 1975 and in March 1981. During the last admission, he had received 8 units of whole blood, 5 units of packed red blood cells, 3 units of fresh frozen plasma, and 10 units of banked platelets.

Records on the 26 donors whose blood or blood products had been given to the patient revealed no unusual travel or history of malaria or antimalarial therapy during the past 3 years. Questionnaires mailed to the donors revealed that 2 had been to Europe, 3 to Canada, and 5 to Mexico within the last 5 years,

but none indicated travel in a malarious area. Thick and thin blood smears from the 25 donors contacted were negative for malaria parasites. Serum specimens from these donors were negative when tested by the indirect immunoflorescence (IIF) test, except one which had an antibody titer to P. falciparum of ≥ 1:4096.

The serologically implicated donor was a 26-year-old African man from Upper Volta who had moved to France in 1974. Although he had no documented history of malaria, he recalled having had many courses of quinine and chloroquine during his youth. The donor had indicated on his questionnaire that France was the only country outside the United States that he had been in during the last 3 years. However, during an interview he stated that while living in France, he had returned to Upper Volta each summer until entering the United States in September 1979. The donor's blood, his first and only such donation, had been obtained on March 13, 1981, and was administered as whole blood to the patient on March 18, 1981. Repeat thick and thin smear examinations of this donor were negative for plasmodia. He was nevertheless treated with chloroquine and primaquine for presumed asymptomatic malaria.

(Reported by Dean L. Winslow, M.D., Assistant Staff Physician, and T.W. Gay, M.D., Clinical Fellow, Section of Infectious Diseases, Department of Medicine, Ochsner Foundation Hospital; E. Shannon Cooper, M.D., Medical Director, Blood Bank, Ochsner Foundation Hospital; Ana Carrera, M.D., Director, Hematology Laboratory, Ochsner Foundation Hospital; and Field Services Division, Epidemiology Program Office, CDC.)

Case 2--In June 1981, a 68-year-old woman in Pennsylvania underwent open heart surgery. Chills and spiking fevers developed 19 days after the operation, and P. vivax parasites were identified on peripheral blood films. The patient was treated with chloroquine and primaquine and had an uneventful recovery. The patient had not been abroad but had received 7 units of packed red cells, 4 units of fresh frozen plasma, and 4 units of platelets for the operation. The 15 donors were contacted, and their sera were screened for the presence of antibodies to malaria. Only one serum, of a 40-year-old Indian immigrant, was positive with an IIF titer against P. vivax of 1:256. The patient had received a unit of packed red cells from this donor 11 days before the onset of the symptoms. The donor had a history of malaria in 1976 in India for which he had been treated, and he had been asymptomatic since that time.

(Reported by R. Tronzo, M.D., Central Blood Bank of Pittsburgh; M. Slifkin, Ph.D., D. Horne, M.D., and E. Rotheram, M.D., Allegheny General Hospital, Pittsburgh, PA; T. Angle and M. Ritter, M.D., Johnstown Regional Red Cross Blood Center, Johnstown, PA; J. Sarandria and E. Streiff, Allegheny County Health Department, Pittsburgh, PA; and Field Services Division, Epidemiology Program Office, CDC.)

C. Congenital malaria

Ten cases of congenital malaria with onset in the United States during 1981 were reported; all were caused by P. vivax. The interval between birth and the onset of symptoms in the infants ranged from 15 to 30 days. The presenting signs and symptoms included fever (in 9 cases), cough (1), lethargy (1), anemia (4), jaundice (1), hepatomegaly (3), and splenomegaly (1).

All mothers had immigrated to the United States from malarious countries in Southeast Asia (8), Central America (1), or South America (1). The interval between entry into the United States and the delivery ranged from 4 months to 1 year. In 4 cases, the mothers were found to have P. vivax parasitemia at the time of delivery.

The occurrence of congenital malaria has increased considerably since 1979. During the entire 14-year period from 1966 to 1979 only 14 cases of congenital malaria were reported in the United States compared with 7 cases in 1980 and 10 cases in 1981. This increase reflects the increasing number of refugees admitted into the United States; 15 of the 17 cases of congenital malaria in 1980 and 1981 were in infants born in the United States to refugees.

Case 1--On August 6, 1981, a 25-day-old girl with a 4-day history of fever was admitted to a Delaware hospital. The mother, a refugee from Kampuchea, had lived there until 1979 and then stayed in Thailand for 1 year before emigrating to the United States in October 1980. During pregnancy she experienced episodes of fever and chills for which no treatment was sought.

Blood smears from the infant showed P. vivax parasites. The infant was treated with chloroquine, and recovery was uneventful. Blood films from the mother were negative.

(Reported by Lawrence A. Virgilio, M.D. and Maureen Edge, Beebe Hospital; Santosh Reddy, M.D., Pediatrician, Lewis, Delaware; and the Delaware Department of Health & Social Services.)

Case 2--On February 21, 1981, a baby girl, born on February 2, 1981, was admitted with fever to a hospital in California. Blood smears revealed the presence of P. vivax parasites, and the infant was treated with chloroquine. The mother arrived in the United States in July 1980 from a refugee camp in the Philippines which she had entered about February 1980 after leaving Kampuchea or Thailand. She had malaria symptoms on January 26, 1981, for which no medical attention was sought. After the delivery, the diagnosis of P. vivax in the mother was confirmed by blood-smear examination. She was treated with chloroquine and primaquine. At the time of birth, cord blood was negative for malarial parasites. The child was followed with repeated blood smears which remained negative until February 21.

(Reported by S.O. Smelsey, M.D., French Camp, California, and the California Department of Health Services.)

Case 3--A 1-month-old girl was admitted to an Oregon hospital; she had a history of increased irritability during the week prior to admission. Symptoms included a temperature of 103.2 F, jaundice, and hepatosplenomegaly. Peripheral blood smears contained P. vivax parasites. The infant was treated with chloroquine and had an uneventful recovery. The mother, who arrived from Laos in March 1981, had a history of self-treatment for malaria about 2 years before.

(Reported by J. DiLiberti, M.D., Al Roller, M.D., Emanuel Hospital, Portland, Oregon, and the Oregon Department of Human Resources.)

Case 4--A 29-day-old boy was admitted to a Texas hospital on January 7, 1981, with a 1-week history of jaundice and fever. The infant was anemic, and P. vivax parasites were identified in a blood smear. Treatment consisted of chloroquine, and the patient recovered satisfactorily. The patient was born on December 20, 1980, to a Vietnamese mother who had a spiking temperature at the time of the delivery. P. vivax parasites were found in a blood smear of the mother. She was treated with chloroquine and primaquine after the delivery.

(Reported by K.Q. Nguyen, M.D. and E.B. Heyer, M.D., Houston, Texas, and the Texas State Health Department.)

Case 5--A 3-week-old boy with a 5-day history of periodic fevers was admitted to a Florida hospital. The infant had a temperature of 100.2 F, hepatosplenomegaly, and anemia. P. vivax parasites were found in a peripheral blood smear, and chloroquine treatment was given. The mother had a history of malaria in Surinam. She had chills and rigors one day after delivery, and P. vivax parasites were identified in her blood smear. Repeated blood slides were made of the infant, but they remained negative until the infants' admission to the hospital.

(Reported by R. Villadiego, M.D., Orlando, Florida, and the Florida State Department of Health.)

Case 6--A 37-day-old boy was admitted to a California Hospital on February 26, 1981, because of a cough. The child's mother was a 18-year-old Laotian woman, who had immigrated into the United States in May 1980, via refugee camps in Thailand and Malaysia. She recalled having had febrile episodes in Laos but not since her arrival in the United States. On admission, the infant was febrile, and he had hepatosplenomegaly and anemia. P. vivax parasites were identified in a smear of the peripheral blood. The infant was treated with chloroquine and made a satisfactory recovery.

Blood smears from the mother made at the time of delivery and at admission of the infant, did not contain malaria parasites. The mother was treated presumptively with chloroquine and primaquine.

(Reported by John Nackley, M.D., Charles J. Berletti, M.D., Contra Costa Health Services, both in Martinez, California, and the California Department of Health Services.)

Case 7--On October 7, 1981, a 2-week-old boy developed fever. P. vivax parasites were identified on a blood smear 1 week later. The patient had an uneventful recovery following treatment with chloroquine. The mother was a native of Guatemala who came to the United States in 1980. She had a history of previous malaria in Guatemala and had an attack of P. vivax malaria 2 days after the boy was born. She was treated successfully with chloroquine and primaquine.

(Reported by E. Martin, M.D., Torrance, California, and the California State Department of Public Health.)

Case 8--A 2-week-old girl in California developed fever on July 24, 1981. P. vivax parasites were found in a blood smear. The mother had arrived in the United States several weeks before the delivery as a refugee from Southeast Asia. The infant was treated with chloroquine. The mother did not have symptoms, but P. vivax parasites were found in her blood.

(Reported by P. Lu, M.D., Monterey Park, California, and the California State Department of Health Services.)

Case 9--On June 6, 1981, a 3-week-old boy in Washington State was hospitalized because of fever and hemolysis. P. vivax parasites were identified on a peripheral blood smear. The patient was treated with chloroquine. The mother had immigrated to the United States from Southeast Asia.

(Reported by M.S. Sigley, M.D., Seattle, Washington, and the Washington State Department of Social & Health Services.)

Case 10--A 6-week-old girl in Maryland developed fever and a light hemolysis. Blood-smear examination revealed the presence of P. vivax parasites. The patient was successfully treated with chloroquine. The mother came from Southeast Asia.

(Reported by M. Turner, M.D., Columbia, MD, and the Maryland State Department of Health.)

VII. MALARIA DEATHS IN THE UNITED STATES

Seven deaths in 1981 due to malaria were reported in the United States, more than in any year since 1971. Four of these deaths occurred in U.S. residents who acquired malaria abroad. None had taken appropriate malaria chemoprophylaxis although information for preventing malaria in travelers is readily available (Section VIII, page 14). Two deaths occurred in visiting foreigners, and 1 in a recently arrived Southeast Asian immigrant.

Case 1--A 46-year-old businessman from Michigan visited Nigeria for 1 week. He did not take malaria chemoprophylaxis. On January 30, 1981, one day after departure from Nigeria he became ill with nausea, vomiting, fever and chills. Two days later he consulted a physician because of continued malaise, nausea, fevers, and chills; malaria was not diagnosed at that time. Four days later the patient became comatose and was admitted to a hospital in Michigan. A 10% to 15% P. falciparum parasitemia was found. He was treated intramuscularly with chloroquine, and intravenous dexamethasone, and required ventilatory support. There were signs of disseminated intravascular coagulation, hemolysis, and thrombocytopenia, and the patient developed acute renal failure. On February 8, 1981, the patient had an episode of ventricular tachycardia and died.

(Reported by H. Gunner Deery II, M.D., John Sheagren, M.D., University Hospital, Ann Arbor, MI; Eugene Willoughby, M.D., Jackson, MI; and the Michigan State Department of Health.)

Case 2--On May 19, 1981, a 4-year-old boy from Nigeria became ill in New York City. He had arrived in the United States in December 1980. He had a history of malaria in Nigeria. The illness was reportedly due to P. vivax and was treated with chloroquine. The patient died on May 21, 1981. The cause of death was attributed to the malaria infection.

(Reported by C. Chakrabarti, M.D., and the New York City Department of Health.)

Case 3--A 61-year-old woman arrived on June 4, 1981, in Puerto Rico from Haiti for treatment of P. falciparum malaria. The patient was admitted with signs of cerebral malaria and renal failure. Despite treatment with chloroquine, the patient died on June 21, 1981.

(Reported by B. Betancourt, M.D., Santurce, P.R., and the Health Department of the Commonwealth of Puerto Rico.)

Case 4--A 60-year-old man, a resident of Indiana, visited Kenya and Tanzania on a safari in the summer of 1981. He reportedly had taken pyrimethamine as malaria chemoprophylaxis. Twelve days after his return, he became ill and presented with hemolysis, cerebral involvement, renal failure, and splenomegaly.

The diagnosis of P. falciparum infection was made. He was treated with chloroquine and intravenous quinine, resulting in parasite clearance, but he died 3 days after admission.

(Reported by Thomas G. Slama, M.D., Indianapolis, IN, and the Indiana State Board of Health.)

Case 5--A 44-year-old woman who was a resident of New York City, born in India, visited India from August 1 to 20, 1981. She did not take malaria chemoprophylaxis. On September 4, 1981, the patient developed general malaise, jaundice, and fever. The patient was admitted to a hospital on September 5. After the diagnosis of P. vivax malaria was made, she was treated with 2 grams of chloroquine but died on September 13, possibly due to the malaria infection.

(Reported by M. Patel, M.D., and the New York City Department of Health.)

Case 6--A 25-year-old Vietnamese woman arrived in the United States 1 week prior to the onset of her illness. On September 21, 1981, she was brought to a hospital comatose and in shock. Her blood smear had a 10% to 15% parasitemia of P. falciparum. She was treated intramuscularly with quinine, and with trimethoprim/sulfamethoxazole. The peripheral parasitemia cleared, but the patient died on September 26, 1981, due to ventricular fibrillation, renal failure, and cerebral edema.

(Reported by Dr. R. Yaeger, M.A. Richter, M.D, Tulane Medical School, New Orleans, LA, and the Louisiana State Department of Health.)

Case 7--A 60-year-old man from Minneapolis was admitted to the hospital on December 2, 1981, because of fever, confusion, weakness, and dyspnea. He had returned on November 28, 1981, from a 6-month stay in Liberia. While there, he used chloroquine prophylaxis irregularly. En route home, he developed fever and malaise. He was seen at a clinic on November 30, 1981, because of nausea, vomiting, and diarrhea. A blood film was then negative for malaria parasites and he received antibiotics. On the evening of December 1, he developed fever and shaking chills. The following day, he was admitted to a hospital in shock, confused, and in respiratory distress. A blood slide was positive for P. falciparum. He was treated for shock and acute cerebral malaria with intravenous fluids, corticosteroids, and chloroquine. The patient died 4 hours after admission. An autopsy demonstrated morbid changes in multiple organ systems consistent with severe acute malaria.

(Reported by F. Wong, M.D., and T. Semba, M.D., Minneapolis, MN, and the Minnesota State Department of Health.)

VIII. PREVENTION OF MALARIA

Guidelines for the "Prevention of Malaria in Travelers" have been published in a Supplement to the Morbidity and Mortality Weekly Report (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 chloroquine-resistant strains of P. falciparum 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.
2. 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 one 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 small 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 small 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).
7. 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

in all their phases. The importance of the examination of blood films for the presence of malaria parasites will be fully understood

Fig. 4

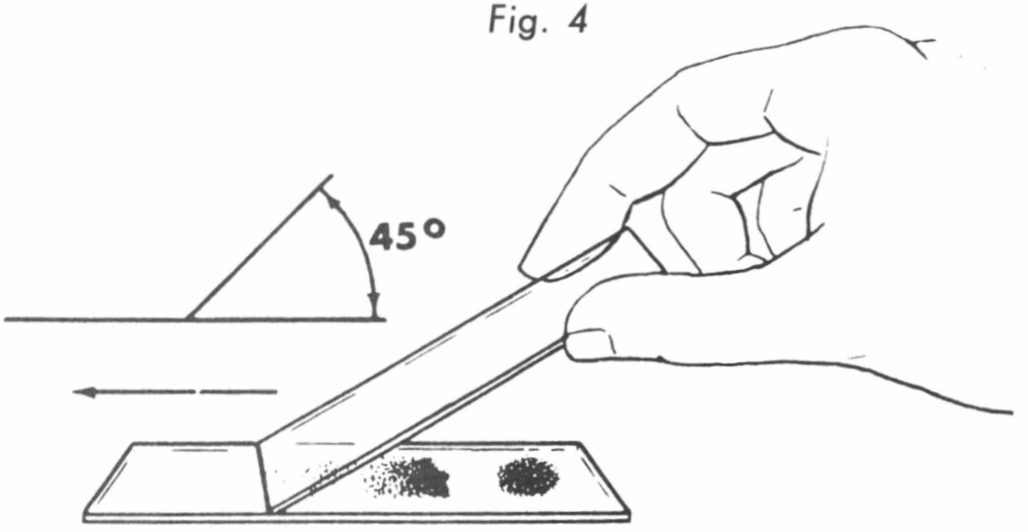
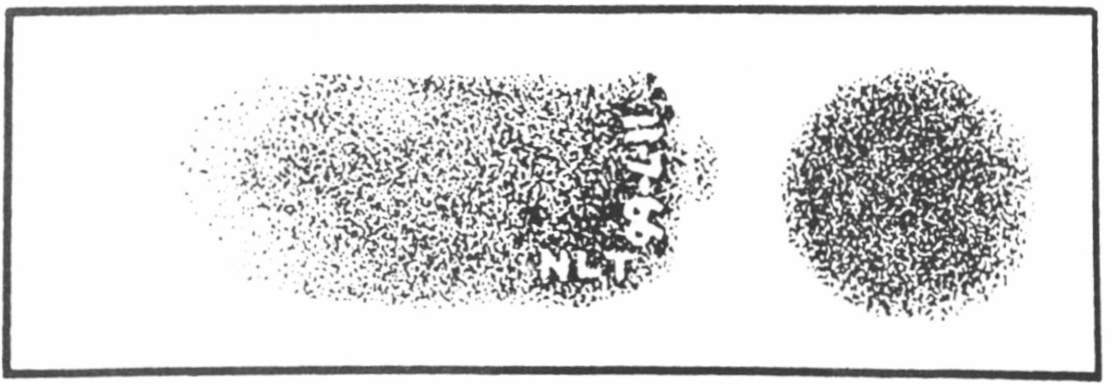


Fig. 5



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