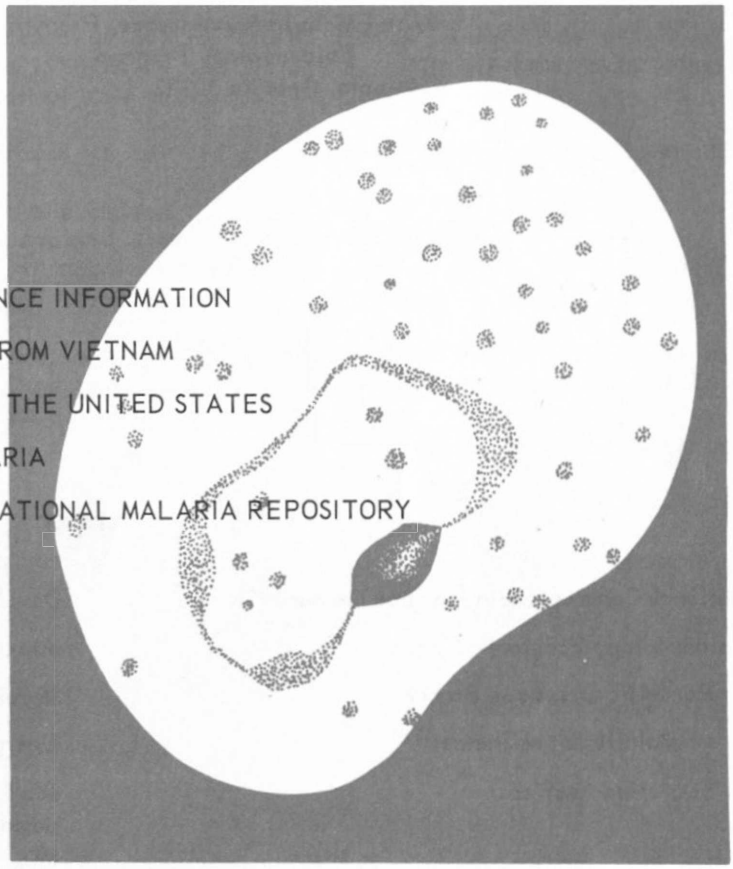
NATIONAL COMMUNICABLE DISEASE CENTER

MALARIA

SURVEILLANCE

TABLE OF CONTENTS



U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
PUBLIC HEALTH SERVICE
HEALTH SERVICES AND MENTAL HEALTH ADMINISTRATION
NATIONAL COMMUNICABLE DISEASE CENTER
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PREFACE

This report summarizes information received from State Health Departments, Medical Departments of the Armed Forces, and other pertinent 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 then to:

National Communicable Disease Center
Attn: Malaria Surveillance, Parasitic Diseases
Epidemiology Program
Atlanta, Georgia 30333

National Communicable Disease Center	David J. Sencer, M.D., Director
Epidemiology Program	Alexander D. Langmuir, M.D., Director
Parasitic Diseases Branch	Myron G. Schultz, D.V.M., M.D., Chief
Malaria Surveillance	George U. Fisher, M.D.
Statistics Section	Ida L. Sherman, Chief James C. Lastinger, M.S. Mrs. Stella S. Sanford

Collaborators

Laboratory Division	
Helminthology and Protozoology Unit	George R. Healy, Ph.D., Chief
National Malaria Repository	Neva N. Gleason, M.S., Supervisor Margaret Welch, M.T. (ASCP)
Fluorescent Antibody Laboratory	Alex J. Sulzer, Ph.D., Chief Marianna Wilson, B.S.
Computer Systems Branch	Charles P. Tyson

MALARIA SURVEILLANCE 1968

I. SUMMARY

The number of malaria cases reported in the United States in 1968 was slightly less than the 1967 total (2,610 in 1968 vs. 2,855 in 1967). Vietnam returnees continued to account for over 90 percent of the cases.

P. vivax accounted for 81 percent of the infections and P. falciparum 13 percent.

Twelve cases were acquired in the United States. Five of these, all due to P. vivax, were introduced cases, and seven, 3 due to P. falciparum, 3 due to P. malariae, and 1 due to P. vivax, were induced cases. Four of the introduced cases occurred in civilians and were epidemiologically related.

There were 6 malaria fatalities, all P. falciparum infections. Two of these cases occurred in seamen, 2 in Vietnam returnees, 1 in an American tourist, and 1 in the recipient of a blood transfusion.

II. TERMINOLOGY

The terminology used in this report is derived from the recommendations of the World Health Organization.[1,2] The definitions of the following terms are included for reference purposes.

1. Autochthonous

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

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

2. Imported

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

3. Induced

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

4. Relapsing

   Renewal of clinical activity occurring after an interval from the primary attack greater than that due merely to periodicity.

III. GENERAL SURVEILLANCE INFORMATION

Between January 1, 1968, and April 1, 1969, 2,610 cases* of malaria with onset of illness in 1968 in the United States and Puerto Rico were reported to the Parasitic Diseases Branch of the National Communicable Disease Center. This compares with 2,855 cases recorded in 1967. Military personnel (including recently discharged veterans) accounted for 2,487 cases and civilians for the remaining 123 (Table I). The number of military cases in 1968 was slightly less than in 1967, but considerably in excess of the annual totals for 1959 through 1966; civilian cases have not increased appreciably in the past 5 years (Figure 1). Only 12 of the 2,610 cases acquired their infection in the United States; five of these were classified as introduced and seven as induced. These cases are described in Section V.

The geographic distribution of the 2,610 cases is shown in Figure 2. The marked concentration of cases in California, Colorado, Georgia, Kentucky, North Carolina and Texas is due to the location of major military centers within these states. The seasonal distribution of cases (Figure 3) shows no distinct pattern and is primarily dependent upon the seasonal variation in the number of troops returning from Vietnam.

* A "case" is defined as an individual's first attack of malaria in the United States, regardless of whether or not he had experienced previous attacks while outside the country. A subsequent attack in the same individual caused by a different Plasmodium species is counted as an additional case, but repeated attacks caused by the same species are counted as relapses, not additional cases. In all instances, the diagnosis must be based on a positive blood smear.

The Plasmodium species was identified in 2,555 of the 2,610 cases (97.9 percent). P. vivax accounted for 81 percent of the infections while P. falciparum was diagnosed in 13 percent; these percentages are identical to those reported in 1967. P. malariae accounted for 32 cases in 1968, while 8 infections were due to P. ovale; this compares with 19 cases of P. malariae and 18 cases of P. ovale in 1967. Mixed infections, generally due to P. vivax and P. falciparum, accounted for 46 cases in 1968 (Table II).

The age and sex distribution of the 123 civilian cases is presented in Table III.

Table I

Military and Civilian Cases of Malaria
United States 1959-1968*

Year	Military	Civilian	Total
1959	12	38	50
1960	21	41	62
1961	45	37	82
1962	75	40	115
1963	58	90	148
1964	52	119	171
1965	51	105	156
1966**	620	144	764
1967**	2698	157	2855
1968	2487	123	2610

*Onset of illness in the United States and Puerto Rico

**The 1966 and 1967 figures have been updated to include cases reported after the publication of previous annual summaries.

Table II

Cases of Malaria by *Plasmodium* Species
United States, 1968

Species	Total	Percent
P. vivax	2125	81.4
P. falciparum	344	13.2
P. malariae	32	1.2
P. ovale	8	0.3
Mixed Infections	46	1.8
Undetermined	55	2.1
Total	2610	100.0

Table III

Age and Sex Distribution of Civilian Malaria Cases
United States, 1968

Age Group	Male	Female	Total	Percent
0-9	4	3	7	5.7
10-19	10	7	17	13.8
20-29	37	18	55	44.7
30-39	16	3	19	15.4
40-49	8	3	11	8.9
50-59	2	2	4	3.3
60-69	3	1	4	3.3
70 +	0	2	2	1.6
Unknown	3	1	4	3.3
Total	83	40	123	100.0

Information regarding the occurrence of previous malaria was available in 2,449 of the 2,610 cases; 921 (37.6 percent) gave a history of malaria while abroad and the remaining 1,528 (62.4 percent) denied previous attacks. These percentages varied significantly depending on the Plasmodium species: Of 2,013 imported vivax cases for which information was available, 794 (39.4 percent) gave a history of a previous malaria attacks, as compared to only 87 (28.2 percent) of 308 falciparum cases.

United States citizens accounted for 81 of the 123 civilian cases, and the remaining 42 cases occurred in foreign visitors. College students or teachers accounted for more cases than any other occupational group (40 cases), followed by merchant seamen (14 cases). Only six cases were reported in Peace Corps Volunteers, as compared to 25 in 1967,* 44 in 1966,* 17 in 1965, and 5 in 1964. (Table IV).

The geographic source of infection for the 2,610 cases is shown in Table V. Most infections (2,444) were acquired in Vietnam. Of the other specified countries, Pakistan and Korea accounted for the second greatest number of cases with 14 each. In 1967, four cases were reported from Korea and 24 from Pakistan.

The onset of illness occurred more than 30 days after arrival in the United States in 74 percent of the 2,368 cases for which both date of onset and date of arrival in this country are known (Table VI). As in previous years, a marked difference was observed between falciparum and vivax malaria: 69.5 percent of the falciparum cases became ill within one month after arrival, as compared with 19.6 percent of the vivax cases. Thirty cases experienced their first symptoms more than one year after return to the United States; 26 of these were due to P. vivax, 2 were caused by P. falciparum, and 2 by P. ovale. The longest interval from return to the United States until onset of illness was 34 months for ovale cases, 32 months for vivax cases, and 21 months for falciparum cases.

Of the 2,610 cases reported in 1968, 74.2 percent were initially treated in a military hospital, 16.7 percent were admitted to Veterans Administration hospitals, and 6.5 percent received care in a civilian hospital (Table VII).

Veterans accounted for 405 cases in 1968, as compared to 291* in 1967 and 47* in 1966. The rising number of malaria cases occurring in veterans and the appreciable number of cases treated in civilian hospitals, coupled with the frequent occurrence of malaria cases during the first 30 days after return to the United States (when many returning servicemen are home on leave) indicates that civilian physicians are particularly likely to encounter cases of malaria.

There were six malaria fatalities in the United States in 1968, all due to P. falciparum, giving an overall malaria case fatality ratio of 0.23 percent and a falciparum case fatality ratio of 1.74 percent. All of the fatal cases are described in detail in Section VI. Information concerning the occurrence of serious clinical complications was available in 234 of the 344 falciparum infections reported in 1968. Serious intravascular hemolysis was reported in 37 cases (15.8 percent). Cerebral involvement occurred in four cases (1.71 percent), renal failure in 3 cases (1.3 percent), and pulmonary edema in four cases (1.71 percent).

During 1968, a total of 247 malaria relapses were reported; 191 relapses were second attacks, 44 were third attacks, 10 were fourth attacks, and 2 were fifth attacks. Of the 191 individuals who experienced their second attack in 1968, 108 also had their first attack in 1968 and the remaining 83 had their first illness in previous years. Thus, a total of 2,857 malaria attacks (2,610 primary attacks plus 247 relapses) was reported in 1968.

*These figures have been updated to include cases reported after publication of previous annual summaries.

Table IV

Occupation and Nationality of Civilian Malaria Cases
United States, 1968

Occupation	U.S. Citizen	Foreign Visitor	Total	Percent
Tourist	11	-	11	(8.9)
Businessman	10	2	12	(9.7)
Government representative	4	2	6	(4.9)
Missionary	8	-	8	(6.5)
Peace Corps	6	-	6	(4.9)
Seaman	5	9	14	(11.4)
College Student or Teacher	22	18	40	(32.5)
Other	6	7	13	(10.6)
Unknown	9	4	13	(10.6)
Total	81	42	123	(100.0)

Table V

Distribution by Plasmodium Species and Area of Acquisition of
Cases of Malaria, United States, 1968*

	vivax	falciparum	malariae	ovale	Mixed	Unknown	Total
AFRICA	16	20	5	8	-	2	51
Africa **	6	3	-	1	-	-	10
West Africa**	2	4	-	1	-	-	7
East Africa**	-	-	2	-	-	-	2
Cameroon	1	1	-	-	-	-	2
Congo	-	1	-	-	-	-	1
Ethiopia	2	-	-	-	-	-	2
Ghana	2	2	-	-	-	1	5
Kenya	-	1	-	-	-	1	2
Liberia	2	6	1	2	-	-	11
Nigeria	-	1	1	2	-	-	4
Rhodesia	-	1	-	-	-	-	1
Rio Munni	1	-	-	-	-	-	1
Sierra Leone	-	-	-	1	-	-	1
Togo	-	-	1	-	-	-	1
Upper Volta	-	-	-	1	-	-	1

Table V continued next page.

Table V (continued)

	vivax	falciparum	malariae	ovale	Mixed	Unknown	Total
ASIA	2074	318	22	-	46	52	2512
Asia**	9	-	-	-	-	-	9
Southeast Asia**	11	4	-	-	-	-	15
Ceylon	1	-	-	-	-	-	1
India	3	-	-	-	1	-	4
Korea	13	-	-	-	-	1	14
New Guinea	1	-	-	-	-	-	1
Pakistan	13	-	1	-	-	-	14
Philippines	3	-	-	-	1	-	4
Thailand	4	2	-	-	-	-	6
Vietnam	2016	312	21	-	44	51	2444
CENTRAL AMERICA AND CARIBBEAN	11	-	-	-	-	-	11
Caribbean**	1	-	-	-	-	-	1
El Salvador	2	-	-	-	-	-	2
Honduras	1	-	-	-	-	-	1
Nicaragua	1	-	-	-	-	-	1
Panama	6	-	-	-	-	-	6
EUROPE	-	-	1	-	-	-	1
Greece	-	-	1	-	-	-	1
NORTH AMERICA	7	3	3	-	-	1	14
Mexico	1	-	-	-	-	1	2
United States	6	3	3	-	-	-	12
PACIFIC**	2	-	-	-	-	-	2
SOUTH AMERICA	7	2	-	-	-	-	9
South America**	2	-	-	-	-	-	2
Brazil	5	1	-	-	-	-	6
Columbia	-	1	-	-	-	-	1
UNKNOWN	8	1	1	-	-	-	10
TOTAL	2125	344	32	8	46	55	2610

*Onset of illness in the United States and Puerto Rico

**Country Unspecified

Table VI

Interval Between Onset of Illness and Date of Entry into the U.S.A.
by *Plasmodium* Species, United States, 1968

Interval (Months)	Plasmodium Species								All Cases	(%)
	Vivax	(%)	falciparum	(%)	malariae	(%)	ovale	(%)		
Less than 1	395	(19.6)	221	(69.5)	7	(24.1)	2	(25.0)	625	(26.4)
1 - 2	842	(41.8)	75	(23.6)	15	(51.7)	3	(37.5)	935	(39.5)
3 - 5	551	(27.4)	16	(5.0)	5	(17.2)	1	(12.5)	573	(24.2)
6 - 11	199	(9.9)	4	(1.3)	2	(6.9)	0	(-)	205	(8.6)
1 year or more	26	(1.3)	2	(0.6)	-	(-)	2	(25.0)	30	(1.3)
All Cases	2013	(100.0)	318	(100.0)	29	(99.9)	8	(100.0)	2368	(100.0)

Table VII

Malaria Cases by Type of Hospital of Initial Admission
United States, 1968

Type of Hospital	Number of Patients	Percent
Military	1938	74.2
Civilian	170	6.5
Public Health Service	25	1.0
Veterans Administration	437	16.7
Not specified	15	0.6
Not hospitalized	25	1.0
Total	2610	100.0

IV. MALARIA IMPORTED FROM VIETNAM

Infections acquired in Vietnam accounted for 2,444 of the 2,598 imported cases (94.1 percent). Only nine of these 2,444 cases were non-military personnel. P. vivax was the etiologic agent in 2,016 of the 2,444 cases (82.5 percent), P. falciparum in 312 cases (12.8 percent), P. malariae in 21 cases (0.8 percent), and mixed Plasmodium species in 44 cases (1.8 percent). No P. ovale cases were reported and in 51 cases (2.1 percent) the Plasmodium species was not identified. Army personnel accounted for 86 percent of the military cases from Vietnam; 10.2 percent were Marines; Navy and Air Force personnel accounted for less than one percent of the cases (Table VIII).

Of the 2,008 military returnees from Vietnam who developed vivax malaria in the United States in 1968, 103 later suffered a vivax relapse, for a relapse rate of 5.1 percent; the corresponding rate for 1967 was 18.4 percent, and for 1966, 29.8 percent. The relapse rate for falciparum infections in military Vietnam returnees in 1968 was 0.96 percent (3 relapses in 312 infections) as compared to 6.5 percent in 1967 and 8.8 percent in 1966. The 1968 relapse rates should be considered preliminary estimates since relapses of 1968 cases will continue to occur in the future. The 1966 and 1967 relapse rates are somewhat higher than those cited in the 1967 Annual Report due to the occurrence of additional relapses.

Table VIII

Military Malaria Cases from Vietnam, by Branch of Service
Unites States, 1968

Branch of Service	Number of Cases	Percent
Army	2094	86.0
Marine Corps	249	10.2
Navy	17	0.7
Air Force	4	0.2
Unknown	71	2.9
Total	2435	100.0

V. MALARIA ACQUIRED IN THE UNITED STATES

Introduced Malaria

Cases 1 through 4

On August 8, 1968, M.W., an 18-year-old male resident of Waverly, Alabama, developed headache and backache, followed two days later by chills and fever. Because of persisting fever, he was hospitalized on August 12; on August 15, P. vivax parasites were identified in his peripheral blood smear. On August 12, 1968, D.D., an 18-year-old female resident of Camp Hill, Alabama, and a close friend of patient M.W., developed headache and fever. She was hospitalized on August 14 and P. vivax parasites were found in her blood smear. On August 12, T.B., a 16-year-old resident of Auburn, Alabama, developed chills and fever which persisted until his hospitalization on August 18. The next day P. vivax parasites were found in his blood smear. L.H., a 15-year-old resident of New York State, had visited relatives in Opelika, Alabama, from July 20 to August 11, 1968. On August 10, she developed chills and fever which recurred daily until her hospitalization in New York on August 16, when P. vivax parasites were found in her peripheral blood smear. Three of the cases were re-exposed to mosquitoes after their onset of symptoms but prior to hospitalization (Figure 4). All four patients responded promptly to appropriate antimalarial therapy. The diagnosis in each case was confirmed by the National Malaria Repository, NCDC.

None of the four patients had ever traveled to malarious areas, received blood transfusions, or used shared syringes. The only activity common to the four cases was attendance at a drive-in movie theater located between Auburn and Opelika, Alabama, during the weekend of July 26 to 28, 1968. The average time from attendance at the drive-in until onset of symptoms was 14 days (Figures 4 and 5).

Within the vicinity of the drive-in theater there were several ponds, two trailer parks accomodating a total of 450 trailers, and a small residential area consisting

of about 30 homes (Figure 6). During the spring and summer of 1968, the population turnover in the trailer courts was rapid since most of the occupants were students at Auburn University. Ft. Benning, Georgia, which receives large numbers of Vietnam returnees lies approximately 30 miles to the southeast of the theater.

Case detection surveys were conducted among occupants of the trailer parks near the theater, among foreign students and Vietnam returnees studying at Auburn University, among patients seen at the Auburn infirmary in July and August, among physicians practicing in the five-county area surrounding the transmission site (Figure 5), and among recently returned Vietnam veterans who had registered at draft boards in the same five counties. In addition, travel histories of vivax cases treated at hospitals in eastern Alabama and western Georgia, including those hospitalized at Ft. Benning, Ft. McClellan, and Ft. Rucker were reviewed. The surveys did not yield the index case or additional introduced cases.

Adult female Anopheles quadrimaculatus mosquitoes were found in the vicinity of the drive-in theater between mid-July and late August 1968. A. quadrimaculatus is an effective malaria vector and presumably was responsible for transmission in this outbreak.

(Reported by J. R. Herring, M.D., Lafayette, Alabama; W. H. Y. Smith, M.D., M.P.H., Alabama State Dept. of Public Health; Julia L. Freitag, M.D., Director, Bureau of Epidemiology, New York State Dept. of Health, and a team of EIS officers).

Case 5

On August 10, 1968, R.P., a 23-year-old Caucasian Army serviceman stationed at Ft. Stewart, Georgia, developed shaking chills and fever. On August 13, he was hospitalized at Ft. Stewart and blood smears were found positive for Plasmodium vivax; the diagnosis was confirmed by the National Malaria Repository, NCDC. He was treated with chloroquine and primaquine and made an uneventful recovery. He had no history of malaria, blood transfusions, use of shared syringes, or travel to malarious areas. A review of his activities during the 8 to 30 day period preceding his onset of symptoms indicated that he had been infected at Ft. Stewart or in the immediate vicinity.

Ft. Stewart is located about 30 miles southwest of Savannah, Georgia, (Figure 7), and serves as one of the Army's flight training centers for pilots of helicopters and small fixed wing planes. The main entrance to Ft. Stewart is located only a few hundred yards north of the city of Hinesville, the seat of Liberty County. During the summer of 1968, there were 4,500 Army troops at Ft. Stewart, many of whom lived in apartments or trailer parks in Hinesville. Many of the 25,000 civilians living in Liberty County were employed at Ft. Stewart.

R.P. lived in an apartment complex in Hinesville and worked at the air traffic control tower at Wright Field, the major airstrip at Ft. Stewart (Figure 8). Many of R.P.'s neighbors were Army personnel, some of whom had served in Vietnam.

The physicians in Liberty County and at Ft. Stewart were interviewed but no additional cases were identified among their patients. Active case detection surveys were conducted at Ft. Stewart and Hinesville (Figure 8); a total of 1,063 individuals were interviewed but no associated introduced cases were detected nor were possible index cases identified. Furthermore, a review of the travel histories of vivax cases treated at Ft. Stewart and other hospitals in southwestern Georgia in the preceding

months did not yield the index case. However, a review of patients with fevers of unknown origin treated at Ft. Stewart's hospital in July yielded one individual who may have been the index case. This man had returned to the United States from Vietnam in late October 1967, and in the subsequent nine months experienced several attacks of chills and fever responsive to self-administered chloroquine. The last of these attacks occurred in mid-July and led to his hospitalization at Ft. Stewart; blood smears obtained at that time were negative for malaria parasites. However, serologic studies performed in September 1968 indicated that this man had indeed experienced a vivax parasitemia some time in the summer of 1968. Furthermore, he frequented the area of the patient's apartment during mid-July, when transmission from the index case to the anopheline vector probably occurred.

Entomologic surveys conducted at Ft. Stewart and in the area of the patient's apartment in Hinesville yielded numerous adult female Anopheles crucians; no A. quadrimaculatus were found. A. crucians is not generally considered an efficient vector, but the species has been found infected in nature and has on occasion been epidemiologically implicated as a vector; in addition, A. crucians has transmitted malaria in the laboratory.

The introduced outbreak in Alabama and the one at Ft. Stewart could not be related.

(Reported by Colonel Harold Chappell, Post Surgeon, Ft. Stewart; Mr. Roger Apel, Sanitarian for Liberty-Bryan counties, Dr. John E. McCroan, Chief Epidemiologist, Georgia State Health Department, and a team of EIS officers).

Induced Malaria

Case 6

On December 4, 1967, a 57-year-old Negro male was admitted to a Washington, D.C., hospital for a laryngectomy because of carcinoma of the larynx. Between December 5 and 19, 1967, the patient received three units of whole blood and one unit of packed cells. His postoperative course was uncomplicated until January 6, 1968, when his temperature spiked to 104° F.; it exceeded 103° F. on each of the next 3 days. On January 9, he became comatose. A routine blood smear obtained on January 11 revealed a 15 percent parasitemia with Plasmodium falciparum. Chloroquine therapy was immediately instituted via naso-gastric tube. The following day, oral pyrimethamine, intravenous quinine, and intramuscular dexamethasone were added to the treatment. On January 12, he was found to have hemoglobinuria and an elevated plasma hemoglobin. His hematocrit dropped from 42 on January 7, to 22 on January 12; the BUN remained normal. Because of the hemolysis, he was treated with mannitol, large amounts of parenteral fluid, and transfusions of packed cells. He died on January 13 without having regained consciousness. Blood films obtained just prior to his death contained only a few P. falciparum parasites. Autopsy findings included pulmonary edema, pigmentation of spleen and liver, and cerebral edema with P. falciparum parasites in the cerebral capillaries.

The patient had given no history of malaria, unexplained fever episodes, or travel outside the Washington, D.C., area. The four persons who donated the blood given to the patient prior to January 6, 1968, were identified and their sera examined. Three were U.S. citizens, none of whom had a history of malaria or unexplained fevers. One had never been overseas; one had been in western Europe in 1945-1946; and one had traveled in Central America in 1962. None of the sera of these donors contained fluorescent antibodies against malaria. The fourth donor was a 29-year-old Negro male student from Nigeria. He donated blood on December 7, 1967, and it was given to the patient on December 19, 1967. The donor had come to the United States

on April 5, 1965, and had not traveled abroad since that time. He denied any history of malaria, unexplained fever episodes, blood transfusions, or use of commonly shared syringes. He had not received antimalarial drugs. His serum contained fluorescent antibodies against malaria, but the Plasmodium species could not be identified. No malaria parasites were found in thick and thin blood smears obtained from this donor in January and March 1968. However, examination of multiple thick and thin blood films, obtained on April 12, 1968, 1 day after he underwent a 500 ml phlebotomy, revealed the presence of very rare P. falciparum trophozoites.

(Reported by John R. Pate, M.D., M.P.H., Chief, Communicable Disease Control, and William E. Long, M.D., Chief, Epidemiological Service Division, District of Columbia Department of Public Health; and an EIS officer.)

Case 7

On October 17, 1967, a 15-year-old girl in Hawaii underwent open heart surgery and received seven units of whole blood. She was hospitalized again in mid-December 1967 for treatment of a gynecologic abnormality and received five more units of whole blood. In January 1968, she began to experience intermittent febrile episodes, which persisted until mid-April 1968, when Plasmodium malariae parasites were detected on a peripheral blood smear. She was treated with chloroquine and made an uneventful recovery. She had never traveled outside the Hawaiian Islands and had never had malaria previously; she denied use of shared syringes.

The 12 blood donors were all contacted and interviewed; none gave a history of malaria attacks and only two had ever traveled to malarious areas. Sera were obtained from these two donors in September 1968 and analyzed for the presence of malaria antibodies by the indirect fluorescent technique. Only one of the two donors had a positive serology; the dilution end points in his serum were 1:4,096 against P. malariae, 1:64 against P. vivax, and 1:16 against P. falciparum. These results indicate that he had a P. malariae infection. This donor was born in the Philippines in 1911 and emigrated to Hawaii in 1930. He remained in Hawaii except for a visit to the Philippines from December 23, 1966, until January 7, 1967. He denied any history of malaria attacks or unexplained febrile episodes. He had donated his blood on October 16, 1967, and it was given to the patient on October 17. Blood smears were not obtained from this donor before he was treated with chloroquine and primaquine in October 1968.

(Reported by Robert Penington, Jr., M.D., Chief, Epidemiology Branch, Hawaii Department of Health.)

Case 8

On January 23, 1968, a 62-year-old woman was admitted to a New York City hospital for insertion of a bypass graft for an occluded left femoral artery. Following surgery, she developed a progressive anemia and, between February 1 and 7, she was given seven units of whole blood. On February 21, her temperature rose to 102° F. Intermittent fever spikes continued every 24-48 hours until March 11, when examination of a routine blood smear revealed Plasmodium vivax parasites. Following treatment with chloroquine, she showed prompt clinical improvement.

The patient was born in Austria and came to the United States many years ago. She had never lived in malarious areas and had no history of unexplained fever or use of commonly shared syringes. Four of the seven donors whose blood she had received

were located. One donor had served in Korea in 1960-1961, and had traveled in Mexico for several weeks in 1965. He did not give a history of malaria and no parasites could be detected in his blood. Two other donors had never visited malarious areas and gave no history suggestive of malaria. The fourth contacted donor was a 22-year-old veteran who had served in Vietnam from July 1966 until July 1967. On September 8, 1967, he developed daily fever spikes and 4 days later was admitted to an Army hospital; vivax malaria was then diagnosed. Over a 3-day period he was treated with a total of 1.5 gm of chloroquine base and was then given eight tablets of chloroquine-primaquine to be taken once a week for eight weeks. On February 1, 1968, he donated the blood which was given to the patient on February 4. The donor had denied both his military duty and his hospitalization for malaria to the blood bank. Examination of blood films taken from this donor in March 1968 revealed the presence of P. vivax parasites.

The Vietnam returnee had also donated blood on December 15, 1967. This blood was given to a patient in New York City on December 28, 1967, together with another unit of blood. This recipient did not experience symptoms compatible with malaria but she did develop hepatitis 6 weeks after hospitalization.

(Reported by Vincent F. Guinee, M.D., Director, Bureau of Preventable Diseases, and Howard B. Shookhoff, M.D., Chief, Tropical Disease Division, New York City Department of Health; Herbert I. Horwitz, M.D., New York City; and Martin Goldfield, Director, Division of Laboratories, and Ronald Altman, M.D., Acting Director, Division of Preventable Diseases, New Jersey State Department of Health.)

Case 9

On April 30, 1968, a 19-year-old white male was admitted to a New York City hospital with a temperature of 102° F. and hepatosplenomegaly. He had experienced four attacks of chills and fever in the preceding 12 days. On May 1, 1968, he had a fever of 104° F. Examination of peripheral blood films then revealed the presence of Plasmodium malariae parasites. Chloroquine therapy was promptly instituted. On May 3 he became afebrile and remained so thereafter.

The patient had no previous history of unexplained febrile episodes or overseas travel, but he was addicted to heroin. Between February 23 and 25, 1968, while in a New York City hospital, he had received three exchange transfusions, consisting of 16 units of whole blood and 26 units of plasma, for treatment of severe hepatitis with pre-coma. By February 29, 1968, he had improved and was discharged from the hospital.

Of the 16 whole-blood donors, only 12 could be contacted; 11 of these had no history of malaria or travel to malarious areas; the twelfth donor had been born and reared in Honduras; attempts to obtain serum from this donor for malaria serology were unsuccessful. Of the 26 plasma donors, 23 were contacted; one had recently traveled in malarious areas but his serum contained no antibodies to malaria (indirect fluorescent technique). Four plasma donors had been born in Puerto Rico and a fifth donor was a native of Greece; all five had resided in their homeland at a time when P. malariae was endemic there; none of the five would consent to serologic screening for malaria.

(Reported by Murray Wittner, M.D., Jacobi Hospital, New York City; Vincent Guinee, M.D., M.P.H., Director of Preventable Diseases, and Howard B. Shookhoff, M.D. Chief, Tropical Disease Division, New York City Health Department).

Case 10

On May 12, 1968, a 54-year-old white American woman entered a hospital in Seattle, Washington, for insertion of an aorto-iliac bypass graft. On May 13, the day of her operation, she received 7 units of whole blood. Her post-operative course was uncomplicated, and she was discharged on May 22. On May 26, she developed low abdominal pain, nausea, vomiting, and fever, and was rehospitalized with a temperature of 104° F. Daily fever spikes continued and on June 4, numerous trophozoites of Plasmodium falciparum were identified on a routine blood smear. She responded promptly to chloroquine therapy. The patient gave no history of malaria, travel in malarious areas, or use of shared syringes.

The donors of the 7 units of blood were contacted, and only one had ever traveled in a malarious area. This man, a 22-year-old American Negro, had served in the U.S. Army in Vietnam from July 29, 1966, until March 31, 1967, when he returned to the United States. While in Vietnam he had taken a combination tablet containing 300 mg chloroquine base and 45 mg primaquine base once weekly in addition to 25 mg diamino-diphenylsulfone (DDS) daily. He continued his chloroquine-primaquine tablets for 6 weeks after returning to the United States. He denied a history of malaria and had experienced no unexplained illnesses except for a self-limited 3-day febrile episode in late May 1967. On May 2, 1968, 13 months after returning from Vietnam, he donated the infected blood. Serum was obtained from this donor on June 14, 1968, and analyzed for malaria antibodies by the indirect fluorescent antibody technique; serum dilution end points were 1:80 against P. falciparum, 1:40 against P. ovale, and 1:20 against both P. vivax and P. malariae. These values are consistent with a recent P. falciparum infection. This donor's hemoglobin electrophoresis was normal, but his red cells were deficient in glucose-6-phosphate dehydrogenase. Blood smears obtained on July 2 were free of malaria parasites; he then underwent a 500 cc phlebotomy and further blood smears were obtained on the next day; again, no malaria parasites were detected.

(Reported by Donald R. Peterson, M.D., M.P.H., Epidemiologist, Seattle-King County Department of Public Health, Seattle, Washington.)

Case 11

In April 1968, a 36-year-old man with chronic renal failure underwent bilateral nephrectomies and incidental splenectomy in preparation for a renal transplant. Post-operatively, he was maintained on biweekly hemodialysis. On July 20, he developed chills and fever, and on July 30, Plasmodium malariae parasites were detected on a routine blood smear. Previous blood smears were then reviewed and parasites were detected as early as July 18. He was treated with chloroquine and primaquine, and he made an uneventful recovery. He had no history of malaria or use of shared syringes and had not traveled outside the United States except for two brief trips across the border from Texas into northern Mexico, 15 years previously. However, in the preoperative treatment of his renal insufficiency and during his postoperative hemodialysis, he had received 56 units of whole blood.

Of the 56 blood donors, 33 were located and interviewed; none gave a history of malaria, but 13 had traveled to malarious areas. Serum was obtained from eight of the 13 and analyzed for malaria antibodies by the indirect fluorescent technique. Only one of the eight, a 21-year-old Nigerian exchange student, had a positive serologic test. The dilution end points in his serum were 1:2,560 against P. malariae, 1:640 against P. falciparum, and 1:160 against P. ovale and P. vivax;

these results indicate a recent P. malariae infection. Blood smears were obtained from this donor on several occasions, but no malaria parasites were detected. On August 23, 1968, 10 ml of his fresh blood were given intravenously to a volunteer recipient, and on September 10, P. malariae parasites were detected in the volunteer's peripheral blood. On repeated questioning, the Nigerian donor denied having had malaria at any time in his life. He had been well since arriving in the United States in June 1966; he had not used antimalarial drugs. The blood which he donated on June 15, 1968, was given to the patient on June 17.

(Reported by James P. Luby, M.D., and Paul M. Southern, Jr., M.D., Department of Internal Medicine, University of Texas Southwestern Medical School at Dallas; Hal J. Dewlett, M.D., M.P.H., Dallas City Health Department; M. S. Dickerson, M.D., M.P.H., Director, Communicable Disease Division, Texas State Department of Health; and Peter G. Contacos, M.D., Head, Section on Primate Malaria, Laboratory of Parasite Chemotherapy, National Institutes of Health, Chamblee, Georgia.)

Case 12

On July 30, 1968, a 25-year-old American woman, the wife of a U.S. Army officer, was admitted to the obstetrical service of the base hospital at Fort Sill, Oklahoma, and delivered a normal child. Because of a postpartum hemorrhage, she received six units of whole blood between July 30 and August 3. She was discharged from the hospital and remained well until August 16, when she developed a persistent severe headache, nausea, and fever. She was hospitalized on August 18 and was treated with antibiotics for suspected endometritis. However, over the next 5 days, she continued to have temperatures to 104° F. On August 22, she became semicomatose. On August 23, trophozoites of Plasmodium falciparum were detected on a routine peripheral blood smear. She was then treated with quinine, pyrimethamine, and diamino-diphenylsulfone (DDS) and made an uneventful recovery.

The patient had no history of malaria or use of shared syringes and had not traveled outside the United States. The six blood donors were all contacted; none had ever experienced malaria attacks, but three had traveled to malarious areas within the past 5 years. Serum was obtained from each donor and analyzed for malaria antibodies by the indirect fluorescent technique. Only one donor had a positive serology; the dilution end points in his serum were 1:256 against P. falciparum and 1:64 against P. vivax and P. brasilianum, thus indicating a recent P. falciparum infection. The positive donor, a 21-year-old white American male, had served with the U.S. Army in Vietnam for 1 year, returning to the United States on January 18, 1968; otherwise, he had never traveled to malarious areas. He had no history of malaria and had not been ill while in Vietnam. In February 1968, he experienced headache and weakness without fever for 4 days; this illness resolved spontaneously. While in Vietnam, he took a combination tablet containing 300 mg chloroquine base and 45 mg primaquine base once weekly in addition to 25 mg of DDS daily. He continued the chloroquine-primaquine for 8 weeks and the DDS for 14-28 days after returning to the United States. He used no antimalarial medication thereafter. He donated his blood on July 17, 1968, and it was given to the patient on July 30. Blood smears obtained from this donor in late August and mid-September 1968 did not contain malaria parasites; after the last blood smears were obtained, he was treated with quinine and pyrimethamine. In March 1969, his hemoglobin electrophoresis, serum protein electrophoresis, and erythrocyte G-6-P-D assay were all normal.

(Reported by James T. Howell, Captain, MC, USA, Chief, and Philip H. Perkins, Captain, MC, USA, Post Sanitarian, Preventive Medicine Division, U.S. Reynolds Army Hospital,

Fort Sill, Oklahoma, R. Leroy Carpenter, M.D., M.P.H., Director, Division of Epidemiology, Oklahoma State Department of Health, and Robert D. Woodson, M.D., University of Washington, Department of Medicine, Seattle, Washington).

VI. DEATHS DUE TO MALARIA IN THE UNITED STATES

Case 1

A fatal case of falciparum malaria is described above under Induced Malaria, Case 6.

Case 2

On March 26, 1968, while on board ship in the mid-Atlantic, a 21-year-old American seaman developed weakness, chills, and fever; his ship had departed from a West African port several days previously. On March 30, he developed headache, severe diarrhea, abdominal cramps, and dyspnea; his temperature was 103.6° F. He was treated with a tetracycline and later penicillin but continued to have daily fevers as high as 106° F; his diarrhea persisted and he became extremely weak. On April 5, his ship arrived in the United States and he was hospitalized. Physical examination revealed an exceedingly thirsty, agitated, dehydrated, hypotensive white male with diarrhea, tachycardia and a temperature of 100° F. The tip of the spleen was palpable and he had marked weakness of the extremities. The hematocrit was 62 percent; no malaria parasites were noted in the initial examination of the admission blood smear. The BUN was 42 mg. percent, sodium 124 meq. per liter, potassium 3.2 meq. per liter, chloride 83 meq. per liter, and CO_2 16 meq. per liter. He was treated with intravenous fluids and belladonna. Twenty-four hours later he had not improved; another blood smear was obtained and numerous P. falciparum parasites were identified. Before chloroquine therapy could be instituted, the patient developed marked hyperthermia (temperature 108° F.), quickly followed by aspiration of vomitus and a respiratory arrest. Attempts at resuscitation were unsuccessful.

Postmortem examination revealed malaria parasites and malaria pigment in red cells and blood vessels throughout the body. The heart showed interstitial edema and hyaline degeneration; there was pulmonary edema; the central nervous system neurons showed anoxic changes. In addition, there was marked congestion of the gastro-intestinal mucosa. A review of the blood smears obtained on the day of the patient's hospitalization revealed P. falciparum parasites.

(Reported by Henry E. Harris, Senior Surgeon, U.S. Public Health Service Hospital, Staten Island, New York, and Dr. Howard B. Shookhoff, Chief, Tropical Disease Division, Bureau of Preventable Disease, New York City Health Department, N.Y.)

Case 3

On May 2, 1968, 12 days after sailing from Liberia for the United States, a 27-year-old West German seaman developed nausea, vomiting, and bilateral costovertebral angle pain. On May 6, 2 days after arrival in Baltimore, he was hospitalized because of persistence of these symptoms and the development of oliguria. Physical examination revealed a healthy appearing man with a temperature of 100° F., bilateral costovertebral angle tenderness, and hepatosplenomegaly. Initial laboratory results included a normal urinalysis and a hematocrit of 48 percent; a peripheral blood smear was obtained but not examined. An X-ray of the abdomen revealed calcific densities in the path of the right ureter, leading to an

initial diagnosis of obstructive uropathy. However, retrograde pyelograms performed several days later showed that the calcific densities were outside the ureter.

On May 7, the BUN was 78 mg percent, and bilirubin was 1.6 mg percent. On May 8 and 10, the patient experienced spiking temperatures to 103° F. By the afternoon of May 10, the hematocrit had fallen to 33 percent, the bilirubin had increased to 20.3 mg percent, the BUN had risen to 180 mg percent, and a urinalysis showed occult blood. Blood smears taken at this time revealed a heavy parasitemia with Plasmodium falciparum; both trophozoites and gametocytes were present. Chloroquine therapy was immediately instituted, but in less than 18 hours, the patient developed pulmonary edema and died.

An autopsy revealed pulmonary congestion and pulmonary edema, calcific deposits in the ileum, and focal tubular necrosis and hemoglobin casts in the kidneys. The heart was normal. P. falciparum parasites were found in the capillaries and small vessels of all organs examined. Postmortem review of the blood smears taken on May 6 revealed the presence of many P. falciparum parasites.

(Reported by J. H. Janney, M.D., M.P.H., Director, Division of Communicable Diseases, Maryland State Department of Health, and E. J. Hinman, M.D., Director, USPHS Hospital, Baltimore, Maryland.)

Case 4

On May 23, 1968, 24 hours after returning from service in Vietnam, a 22-year-old white male American marine, complaining of headache, fatigue, nausea, and vomiting of 5 days duration, presented himself to the emergency room at a military hospital in California. He did not give a history of malaria or other serious illnesses while in Vietnam, and he reported having taken his malaria chemoprophylaxis regularly. On physical examination he appeared moderately ill and had a temperature of 102.4° F. There was slight scleral icterus, and the liver was enlarged and mildly tender; there was no splenomegaly. Initial laboratory studies revealed a hematocrit of 47 percent, a bilirubin of 3.2 mg percent, and SGOT of 262. No malaria parasites were noted at this time on a routine blood smear.

The patient was hospitalized with a diagnosis of infectious hepatitis. He experienced daily fever spikes of 104° F. On the sixth hospital day, he became semicomatose; examination of peripheral blood smears then revealed a 70 percent parasitemia with Plasmodium falciparum. Laboratory values at that time included the following: hematocrit 26 percent, bilirubin 23.6 per mg percent, BUN 120 mg percent, Na 118 meq/L., K 2.0 meq/L., Cl 64 meq/L., and CO_2 8 meq/L. Hemoglobin was detected in the urine. Intravenous antimalaria therapy with quinine and chloroquine was immediately instituted. In addition, dexamethasone, heparin, and transfusions of packed red cells were administered. Because of the electrolyte imbalance and progressive azotemia, peritoneal dialysis was instituted on the seventh hospital day. On the eight day, the patient had once again become lucid, his hematocrit had risen to 37 percent, and his electrolyte abnormalities had been corrected. Examination of blood films at that time revealed only a few malaria parasites. Peritoneal dialysis was discontinued. That evening the patient developed pulmonary edema which responded to treatment with digoxin, morphine, rotating tourniquets, and ethacrynic acid. On the evening of the ninth hospital day, he again developed pulmonary edema which did not respond to treatment, and he expired. No malaria parasites could be found in blood films taken on the day of his death.

Postmortem examination revealed dilatation of both cardiac ventricles, marked pulmonary edema, congestive hepatosplenomegaly, and swollen bile-stained kidneys which showed focal evidence of tubular necrosis. Reexamination of the blood smears obtained on the day of the patient's admission to the hospital revealed the presence of numerous trophozoites of P. falciparum.

(Reported by Philip K. Condit, M.D., M.P.H., Chief, Bureau of Communicable Disease Control, California State Department of Public Health; and George I. Smith, Major, U.S. Air Force Medical Corps.)

Case 5

On June 27, 1968, while on duty in Vietnam, a 41-year-old American serviceman developed chills, fever, blurred vision, and generalized weakness. He returned to his home in Texas on June 28, having received emergency home leave, and on July 1, because of persistent symptoms, he sought medical attention at a military hospital. On physical examination, his temperature was 103° F.; he was drowsy, moderately confused, and appeared somewhat toxic; his liver and spleen were enlarged and tender. Initial laboratory studies showed a hematocrit of 43 percent, a BUN of 32 mg percent, and a bilirubin of 3.8 mg percent. Peripheral blood smears showed that approximately 50 percent of the red cells were parasitized with Plasmodium falciparum.

He was treated with intravenous quinine sulfate, oral chloroquine phosphate, and intramuscular dexamethasone. On July 2 his platelet count was 5,000; the prothrombin time, partial thromboplastin time, and fibrinogen level were normal, and there were no petechiae or signs of blood loss. Intravenous heparin therapy was instituted. Also on July 2, he developed hemoglobinemia and hemoglobinuria, associated with a decline in hematocrit to 30 percent. Despite hydration and treatment with mannitol and diuretics, he became oliguric and azotemic. On July 3 his platelet count was still 5,000 and his hematocrit had decreased to 26 percent; he received four units of packed cells and 12 units of platelets. On the same day he had a grand mal seizure, necessitating treatment with diphenylhydantoin. On July 4 he began to show signs of improvement; his temperature returned to normal, and his mental status improved slightly; his BUN stabilized at 150 mg percent, and his urine output increased to 100-150 cc per hour. Peripheral blood smears at this time showed a 10 to 15 percent parasitemia. On July 6, however, he again became febrile and a chest film showed bilateral pulmonary infiltrates. Blood cultures were drawn, and he was started on cephalosphorin and sodium colistimethate. However, he developed progressive respiratory insufficiency, and despite a tracheostomy, he died on July 7, the seventh hospital day. The blood cultures drawn on July 6 subsequently grew Staphylococcus aureus and Pseudomonas aeruginosa.

On postmortem examination, the lungs showed an acute necrotizing confluent bronchopneumonia with pulmonary edema and congestion. The kidneys showed focal acute inflammation and necrosis with occasional colonies of gram positive cocci; hemoglobin casts were found in the distal tabules. The brain showed multiple small foci of acute inflammation, one of which contained gram positive organisms; in addition, focal perivascular hemorrhage with ischemic necrosis was noted. The heart showed focal acute myocarditis. There was marked hyperplasia of both myeloid and erythroid elements in the bone marrow. P. falciparum parasites were not detected in any tissues, but malaria pigment was found in virtually every organ. Postmortem cultures of the lungs grew S. aureus and Ps. aeruginosa.

(Reported by Charles L. Hedberg, Colonel, MC, Acting Chief, Department of Medicine, Brooke Army Medical Center, Ft. Sam Houston, Texas; and M. S. Dickerson, M.D., Director, Communicable Disease Division, Texas State Department of Health.)

Case 6

On July 13, 1968, following an untreated illness of 8 days duration, a 25-year-old American woman in Minneapolis, Minnesota, died. On July 14, postmortem examination revealed a normally proportioned Caucasian female with slightly icteric skin. The liver and spleen were enlarged and slate grey in color, and the brain appeared slightly congested and edematous. On microscopic examination, the capillaries of all organs were found to contain red cells parasitized with Plasmodium falciparum. Hemoglobin cases were found in the renal tubules, and vacuolar degeneration was noted in the proximal tubular cells. The bone marrow showed marked erythroid hyperplasia. The heart and lungs were normal.

The woman had no history of previous malaria attacks and had not received blood transfusions. She and her husband had lived in Sierra Leone from September 1967 until June 15, 1968, and then had returned to the United States, arriving in San Francisco on July 5. During their return journey they had visited several countries in southeast Asia, including Thailand. While abroad, the woman had not used malaria chemoprophylaxis. On July 5 she complained of dizzy spells and by July 10 appeared febrile and intermittently delirious. Because of her religious beliefs, she had not sought medical attention.

(Reported by Calvin Bandt, M.D., Hennepin County Medical Examiner's Officer, Minneapolis, Minnesota; Fred G. Gunlaugson, M.D., Director, Bureau of Disease Prevention and Control, Minneapolis City Health Department; and D. S. Fleming, M.D., Director, Division of Disease Prevention and Control, Minnesota State Health Department.)

VII. REPORT FROM THE NATIONAL MALARIA REPOSITORY - 1968

The presence of Plasmodium species or agreement that there were no parasites present was confirmed by the National Malaria Repository in blood films from 1,479 of the 1,494 cases (99%) submitted in 1968. Malaria organisms could not be found in blood films from 10 cases (0.65%) submitted as having parasites present and slides from 5 cases (0.35%) were judged to be unsatisfactory for adequate parasitologic diagnosis.

It should be noted that in 77 cases (5.7%) the National Malaria Repository determined that a different species was present than that identified by the laboratory of origin.

Tables illustrating the origin (Table A) and specific diagnosis (Table B) of malaria smears examined by the Repository are shown below. Totals for calendar year 1967 are included for comparison.

Table A

	ORIGIN							
	Army	Navy	VA Hosp.	Air Force	Health Depts. (State, County, City)	PHS Hosp.	Others - Hospitals, Clinics, Physicians, etc.	Cumulative
Cumulative total positive 1968	790**	28	349	31	46	29	71	1344
Cumulative total positive 1967	1585*	24	309	18	57	16	121	2130

Table B

Species	Cumulative Total 1968	Cumulative Total 1967
P. vivax	1135	1836
P. falciparum	154	241
P. malariae	17	8
P. ovale	13	23
Plasmodium sp.	25	22
Negative	145	119
Unsatisfactory	5	3
Total examined	1494	2252
Cumulative positive	1344	2130

*Beginning in October 1967, only 10% of the blood films submitted by 11 large Army Hospitals were examined.

**This includes only 10% of 820 cases submitted from the 11 Army Hospitals.

VIII. ACKNOWLEDGMENT

The Malaria Surveillance Report, prepared annually at the National Communicable Disease Center, is based on information provided on individual case reports. The tremendous support given to the malaria surveillance program 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.

We are particularly grateful to Dr. Hans O. Lobel, the former chief of the malaria surveillance program, and presently assigned to the Malaria Eradication Program, NCDC; the present system of gathering and analyzing malaria surveillance data was conceived and implemented under his direction. We also wish to acknowledge Dr. William E. Collins, Laboratory of Parasite Chemotherapy, NIAID, NIH, for performing some of the fluorescent antibody tests noted in this report.

Thorough and comprehensive evaluation of all cases of malaria reported in the United States constitutes the most effective approach to preventing reestablishment of malaria transmission subsequent to importation.

All cases of malaria, regardless of where they are acquired, should be promptly reported to the appropriate health department. Clinical and epidemiological information on each case should be provided on the Malaria Case Surveillance Report Form PHS 4.80 (NCDC). Extra copies of this form are available on request. Every effort should be made to obtain pretreatment thick and thin blood films for each case. These films may be submitted with the Surveillance Form.

REFERENCES

1. Terminology of malaria and of Malaria Eradication. World Health Organization, 1963, p. 32.

2. WHO Expert Committee on Malaria - Tenth Report. WHO Technical Report Series No. 272, p. 34

Figure 1 MILITARY AND CIVILIAN CASES OF MALARIA, UNITED STATES, 1959-1968

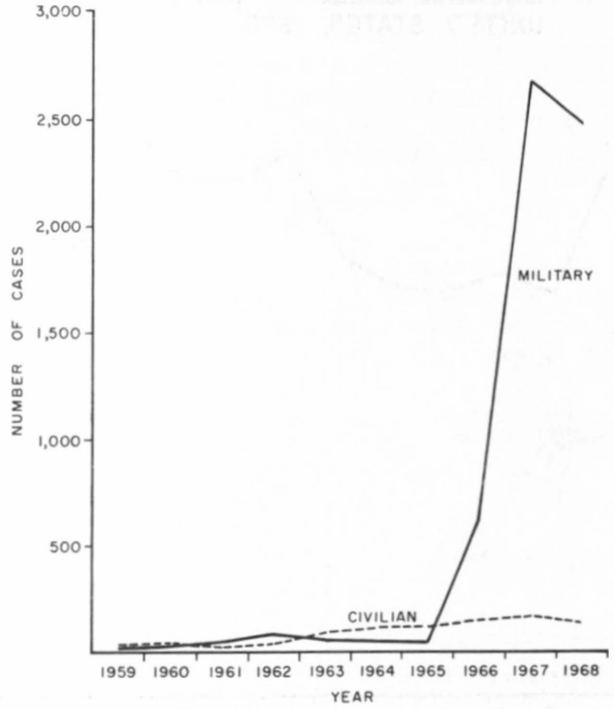

Figure 2 GEOGRAPHIC DISTRIBUTION OF MALARIA CASES WITH ONSET IN UNITED STATES, 1968

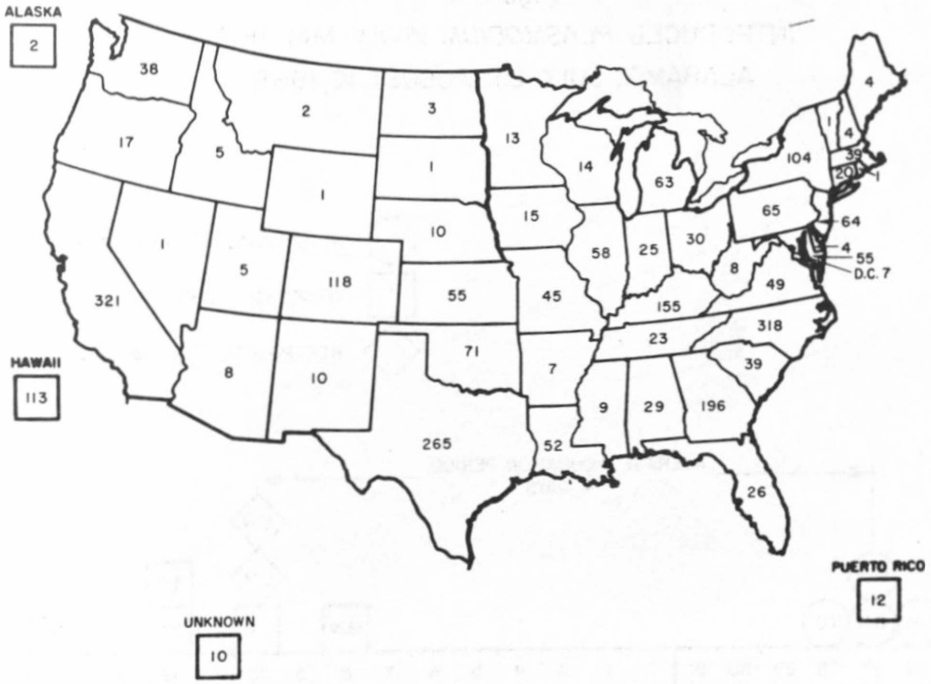

Figure 3 MALARIA CASES BY MONTH OF ONSET, UNITED STATES, 1968

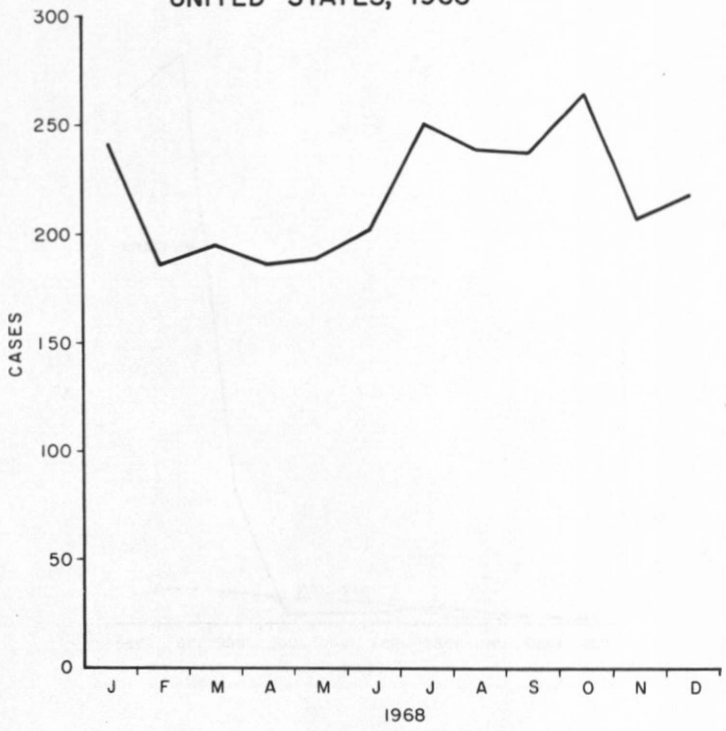

Figure 4
INTRODUCED *PLASMODIUM VIVAX* MALARIA
ALABAMA, JULY 25 - AUGUST 16, 1968

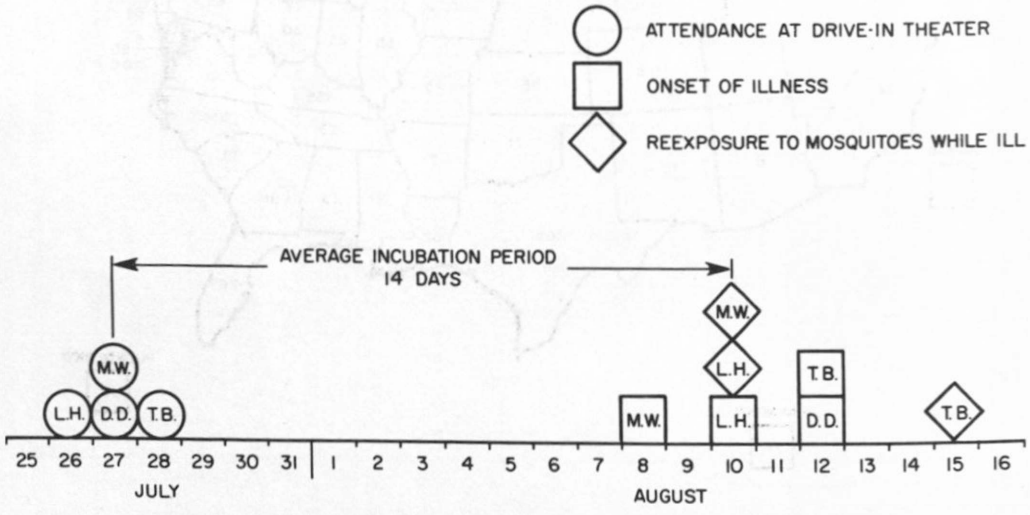

Figure 5 LOCATION OF AREA OF MALARIA TRANSMISSION IN EASTERN ALABAMA, JULY 1968

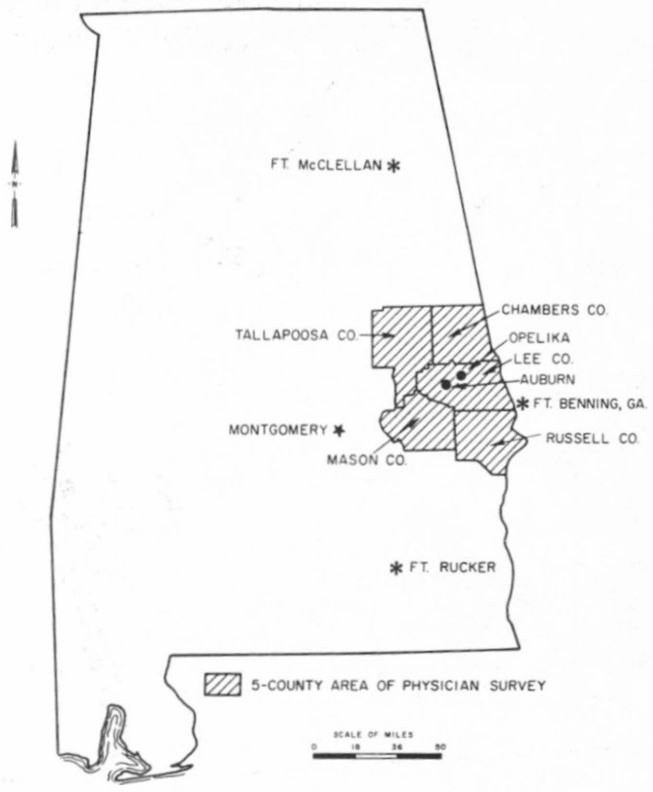

Figure 6 AREA OF MALARIA TRANSMISSION, AUBURN, ALABAMA, JULY 1968

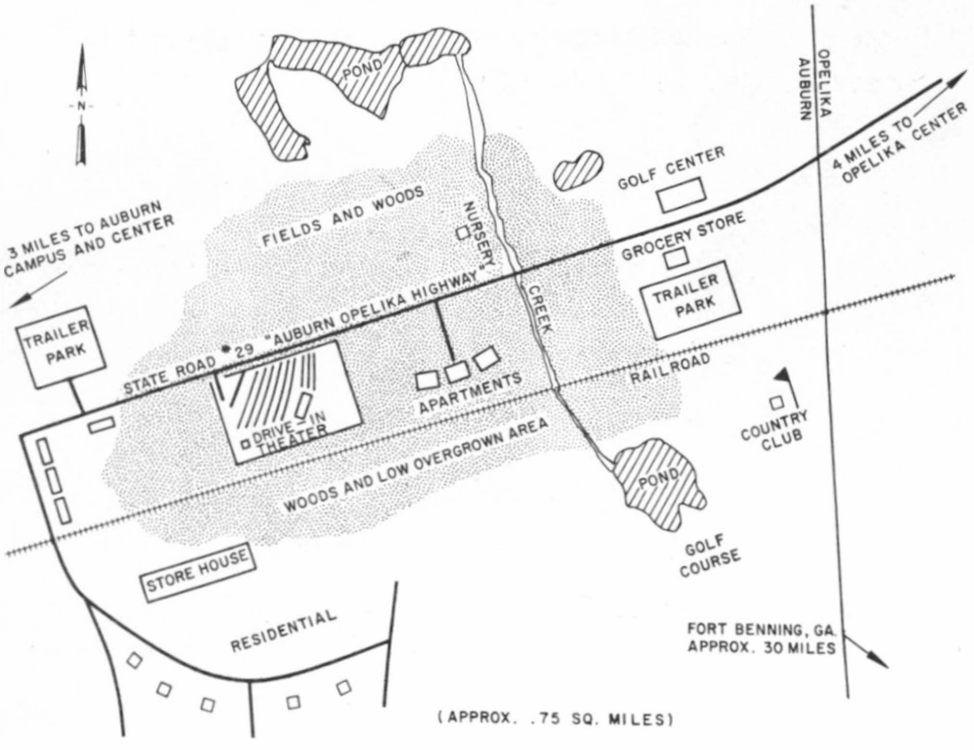

Figure 7

MALARIA PATIENT'S WHEREABOUTS

JULY 11 – AUGUST 2, 1968

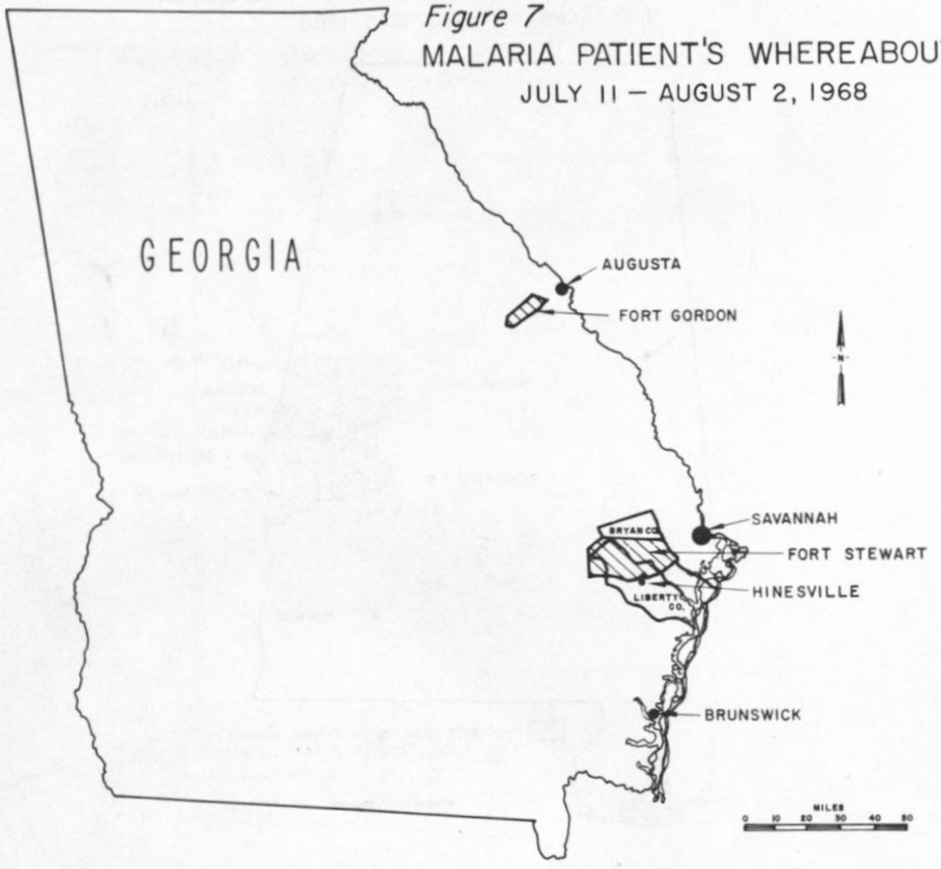

Figure 8

Suspected Area of Malaria Transmission. Fort Stewart–Hinesville, Georgia, July 1968