Occurrence of Leprosy in U.S. Veterans After Service in Endemic Areas Abroad

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MORE U.S. CITIZENS than ever before are living in or traveling to virtually every country of the world—many are members of the Armed Forces who are stationed in areas where leprosy is endemic. Thus, it is important to know the incidence of leprosy among U.S. veterans who have served abroad in endemic areas.

This report is concerned with cases of leprosy known to have occurred in U.S. veterans, probably as a result of exposure to the disease while in service. The data were obtained from the records of the Public Health Service Hospital at Carville, La., Public Health Service outpatient clinics, and State departments of health. No information is available on the number of unreported cases which might exist. It is also probable that among veterans, as among nonveterans even in endemic areas, there are undiagnosed cases of leprosy.

Although leprosy was introduced into the Americas from the Old World by explorers, settlers, and African slaves, no case was reported in U.S. veterans of any war until the Spanish-American War in 1898, despite the fighting in the War of 1812 in New Orleans where leprosy had been present for at least 70 years (1).

A 1940 report by Hasseltine (2) dealt with leprosy in U.S. veterans. His study, based on records of those who had been admitted to the hospital at Carville, included 32 veterans of the Spanish-American War and 51 of World War I.

One of the Spanish-American War veterans did not serve outside the United States, one had no record of such service, and the remaining 30 had served in leprosy endemic areas outside the United States. Five of these veterans were born outside the United States, eight came from southern States, including Georgia, and 19 came from northern States.

Of the 51 World War I veterans, 33 had no military service outside the United States. None were born in northern States—18 were from other countries and 33 were born in southern States.

In 1944 Faget (3) reported 14 additional U.S. veterans with leprosy who were admitted to Carville. However, he did not attribute their disease to foreign service.

In 1965 the Veterans' Administration published a report of a study of 90 cases of leprosy

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in U.S. veterans with military service in 1940 or later (4). (The study, conducted by members of the Leonard Wood Memorial staff, was started by Dr. J. A. Doull and continued after his death by Dr. R. Guinto and Dr. C. H. Binford.) Before their service, 12 of the 90 veterans had signs of leprosy and 15 had a history of exposure to the disease. Twenty-eight had lived in endemic areas before 1940, and 35 probably had leprosy as a result of exposure while serving in endemic areas.

Since the 1965 report (4), 26 additional U.S. veterans with leprosy were admitted to the Carville hospital. Also, 124 veterans with leprosy, not admitted to Carville, were reported to State health departments as follows: California, 27; Hawaii, 71; Texas, 14; and other States, 12. Thus 150 additional cases of leprosy in veterans have come to our attention. Including the 90 cases reported in 1965, a total of 240 cases in veterans with military service from 1940 through December 31, 1968, have been reported.

In 46 of the 240 veterans the infection probably occurred as a result of exposure to leprosy during military service outside the United States. Of the 46 whose cases were considered to be service connected, 35 were reported in 1965, seven were admitted to Carville from June 1, 1964 through December 31, 1968, and during this period four were reported but not admitted to Carville.

The veterans with leprosy who were born or had lived where leprosy was endemic were not included in the number believed to have been service connected. The U.S. endemic areas generally are considered to be southeast Texas, southern and southwest Louisiana, southern Florida, and Hawaii. Other veterans excluded were those born in or who had lived in Mexico, Puerto Rico, Guam, Samoa, and the Philippines.

In summary, from 1940 to 1968 of a total of 240 cases of leprosy in U.S. veterans, 194 were not attributed to exposure while in service and 46 were considered to be service connected.

Cases Considered Service Connected

The following patients with leprosy regarded to be service connected were admitted to the Public Health Service Hospital at Carville.

Case 1: White male, born 1904, Ohio. Moved

when he enlisted in U.S. Army. During his 21 years of service he was stationed in Panama for 3 years; discharged 1945. The first sign of anesthesia in skin on right elbow was noticed in 1936. In 1939, diagnosis of syringomelia was made; at the same time he noted partial nasal obstruction. Diagnosis of leprosy was made in 1942. Admitted August 18, 1946.

Case 2: White male, born 1919, Montana. No other residence prior to enlistment in U.S. Marine Corps in 1939. Served in American Samoa September 1, 1942 to December 7, 1943; discharged 1945. Onset probably in 1945 when loss of sensation of pain was noticed in right arm. Admitted February 27, 1949. Diagnosis was tuberculoid leprosy.

Case 3: White male, born 1894, Missouri. No known foreign residence prior to enlistment in U.S. Army in 1917. Stationed in Philippines, 1934–39; Hawaii, 1939–43; discharged 1946 (retired). Onset probably in 1948 when he noticed numbness of right foot and toes. Diagnosis of leprosy was made in June 1950 when skin lesions appeared. Admitted August 25, 1950. Diagnosis was lepromatous leprosy.

Case 4: White male, born 1895, Indiana. Lived there until 1916 when he enlisted in U.S. Army. From 1916 to 1942 he was stationed in San Antonio and Brownsville, Tex. Onset probably in 1947 when numbness of arms and legs was first noticed. Admitted July 19, 1948. Diagnosis was tuberculoid leprosy.

Case 5: White male, born 1911, Arkansas. Lived in nonendemic area in northern Texas from 1915 to 1940. No record of other residence or of travel prior to enlistment in U.S. Army in 1938. Stationed in Philippines where he was a prisoner of war in Bilibid and Cavanaguan, November 1941 to March 1945 (in contact with leprosy cases); discharged 1946. Onset probably in 1950 when he noticed red spot on left foot. Admitted January 19, 1951. Diagnosis was tuberculoid leprosy.

Case 6: White male, born 1923, Texas. Lived there until he joined U.S. Army in 1944. Served in Honolulu, Saipan, Okinawa, and Japan; discharged 1946. Onset probably in 1947 when areas of numbness were noticed on left leg. Admitted August 3, 1951, and discharged in 1952. Diagnosis was tuberculoid leprosy.

Case 7: White male, born 1913, Alabama. Professional soldier for 14 years, 1935-45 and 1949-53. During this period he spent 2 years in Philippines, 2 months in Africa, and 2 years in Japan and Korea; discharged 1953. Onset probably in 1951 while in Korea when he noticed nonpruritic red spots on trunk and extremities. Admitted February 12, 1953. Diagnosis was lepromatous leprosy.

Case 8: Negro male, born 1920, Georgia. Lived there until 1943 when he entered U.S. Army. During service he was stationed in New Guinea, Luzon, and Japan; discharged 1946. Onset probably in early 1953 when small light-colored areas appeared on right shoulder and abdomen. Diagnosis of leprosy made in July 1953. Admitted August 5, 1953. Diagnosis was lepromatous leprosy.

Case 9: Negro male, born 1917, Pennsylvania. Lived there until 1943 when he joined U.S. Navy. Served in New Guinea and Philippines (1 year); discharged 1946 and returned to Pennsylvania. First sign in 1952 when an erythematous macule appeared on right forearm. Diagnosis of leprosy made in 1953. Admitted October 28, 1953. Diagnosis was lepromatous leprosy.

Case 10: White male, born 1895, Kentucky. Lived in Kentucky and Tennessee until he joined U.S. Army in 1942. Served in European theater during 1944 and 1945 and in Philippines 1946-47; discharged 1947 and returned to Kentucky. Onset probably in 1951 when an erythematous macule appeared on left ankle. Admitted May 3, 1954. Diagnosis was lepromatous leprosy (5).

Case 11: White male, born 1922, North Carolina. Entered U.S. Army in 1942. Served in New Caledonia, Fiji, Guadalcanal, Luzon (1 year to 18 months), Japan; discharged 1945. Onset probably in 1948 when an injury to right knee caused no pain. In 1951 hypopigmented areas appeared on abdomen and lower extremities. Diagnosis of leprosy made by private physician in 1952. Admitted May 31, 1955. Diagnosis was lepromatous leprosy.

Case 12: White male, born 1918, Georgia. Lived in Georgia until he joined U.S. Army in 1942. Served in New Caledonia from January 1943 to May 1945, Philippines (5 months), and Japan (1 month); discharged 1945 and returned

to Georgia. Onset probably in 1948 when he noticed anesthesia of right elbow. Admitted January 18, 1956. Diagnosis was lepromatous leprosy.

Case 13: White male, born 1912, Virginia. Entered U.S. Army in 1945. During service spent 1 year in Philippines, otherwise service was in the United States; discharged 1946. Onset probably in 1950 when he noticed a flat red spot on right hip. Diagnosis of leprosy was made in 1956 by skin specialist. Admitted December 14, 1956. Diagnosis was lepromatous leprosy.

Case 14: White male, born 1927, Kentucky. Lived in Michigan from 1932 to 1955 except for about 18 months when he served in the U.S. Army during 1946-47. Stationed on Luzon, Philippines, for about 12 months. Onset probably in 1948 when anesthesia of the left ankle was first noted. Diagnosis of leprosy was made in 1954 when an outpatient at Carville hospital. Admitted October 23, 1958. Diagnosis was tuberculoid leprosy.

Case 15: Negro male, born 1924, North Carolina. Lived in North Carolina until he entered U.S. Navy in 1942. Served 2 years in North Carolina, 1½ years in Hawaii; discharged 1945 and returned to North Carolina. Onset probably in 1954 or 1955 when a small numb spot on back of right leg was observed. Diagnosed as leprosy in 1956. Admitted March 27, 1957. Diagnosis was lepromatous leprosy.

Case 16: White male, born 1915, Indiana. Lived in Indiana until he entered U.S. Army in 1941. Stationed in United States except for 1 year (1944) on Guam; discharged 1946 and returned to Indiana. Onset probably in 1.57 when numbness of foot was noticed. Admitted August 1, 1960. Diagnosis was lepromatous leprosy.

Case 17: White male, born 1924, Pennsylvania. Entered U.S. Army in 1942. Between 1943 and 1945 served in New Guinea, Dutch East Indies, Philippines, and Australia. Deserted in 1945 and remained in the Far East (Philippines and Japan) for 2 years, then returned to Pennsylvania. Treated for "ringworm" in 1944 in the Philippines. First definite sign of leprosy was anesthesia of the forearm in 1957. Admitted August 3, 1960. Diagnosis was lepromatous leprosy.

Case 18: White male, born 1923, South Car-

olina. Lived there until he entered U.S. Army in 1942. During service spent 15 months in South Pacific islands; discharged 1944 and returned to South Carolina. Onset probably in 1959 with hypesthesia of the left knee. Admitted September 28, 1960. Diagnosis was lepromatous leprosy.

Case 19: White male, born 1924, Maryland. Moved to Georgia in 1939. Entered U.S. Army in 1945. Stationed in Philippines for 14 months; discharged 1946 and then lived in Miami, Fla. Onset probably in 1959 when he noticed "numbness" of arm. Admitted October 10, 1960. Diagnosis was borderline (dimorphous) leprosy.

Case 20: Negro male, born 1916, Alabama. Lived there until he entered U.S. Army in 1942. During service he was stationed in California, 1942-43 and India, 1944-45; discharged 1946 and returned to Alabama. Onset probably in 1958 when he noticed nodular lesions on his legs. Admitted May 25, 1961. Diagnosis was lepromatous leprosy.

Case 21: White male, born 1931, Alabama. Lived there until he joined U.S. Army in 1948. Served in South Carolina, 1948-49; Japan, 1949-50; Korea, 1950-51; and Georgia, 1952; discharged 1952 and returned to Alabama. Onset probably in 1960 when he noticed an erythematous plaque on leg. Admitted January 16, 1962, Diagnosis was lepromatous leprosy.

Case 22: White male, born 1921, Missouri. Lived there until he joined U.S. Air Force in 1942. During service he was stationed in the United States and in 1944-45 was on Espíritu Santo Island (2 months), other New Hebrides islands (2 months), Okinawa (3 months), and Philippines (5 months); discharged 1946 and then lived in Oregon. Onset probably in 1955 when a hyperpigmented macule on forehead was noted. Admitted February 22, 1962. Diagnosis was lepromatous leprosy.

Case 23: White male, born 1925, Florida. Lived there until 1943 when he entered U.S. Army. Served in India during 1944 and 1945; discharged 1946 and returned to Florida. Onset probably in 1952 when anesthesia of the arm was noted. Admitted October 22, 1962. Diagnosis was tuberculoid leprosy.

Case 24: White male, born 1925, Ohio. Lived there until he entered U.S. Marine Corps in 1942. Served for 2 years (1943-45) in South Pa-

cific; discharged 1945 and then lived in Florida. Onset probably in 1955 when numbress of arm was noted. Admitted December 10, 1963. Diagnosis was lepromatous leprosy.

Case 25: White male, born 1910, North Carolina. Lived there until he entered U.S. Army in 1942. Served in Australia (6 months), New Guinea (7 months in 1944), and the Philippines (10 months 1944-45); discharged 1945. Onset probably in 1950 when nodular lesions of the skin were noticed. Admitted April 30, 1964. Diagnosis was lepromatous leprosy.

Case 26: White male, born 1929, Missouri. Lived there until he entered U.S. Army in 1945. Served in the Philippines (1946-48); discharged 1948. Lived in Arizona 1948-56 and in California 1956-64. Onset probably in 1959 when a nodule on the right shoulder was noticed. Admitted August 10, 1964. Diagnosis was lepromatous leprosy.

Case 27: Negro male, born 1921, Maryland. Served in U.S. Army 1942-46 in New Guinea (14 months), Australia (8 months), Philippines (12 months), and Japan (6 months). Onset in 1955 with infection of right great toe. Admitted March 2, 1965. Diagnosis was dimorphous tuberculoid leprosy.

Case 28: White male, born 1917, Arkansas. Served in U.S. Army April 1945 to July 1946 in Texas (17 weeks) and in Hawaii and New Caledonia. Lived in Civilian Conservation Corps camp in California 1942–45. Onset in 1963 with numbness of right hand; later both feet were numb. Admitted April 12, 1965. Diagnosis was dimorphous leprosy.

Case 29: White male, born 1923, New York City. Served in U.S. Air Force March 1940 to 1965 in Louisiana, Texas, California, Guam, Philippines, China, Japan, Okinawa, Hawaii, and numerous European countries. First noted numbness of his toes in 1956. In 1963 he noted small skin lesions on all extremities, chest, and back. Admitted November 11, 1965. Diagnosis was dimorphous lepromatous leprosy.

Case 30: White male, born 1921, New Jersey. Served in U.S. Army 1942–45, 1946–52, 1961–66, and in U.S. Marine Corps 1952–55, in Mississippi, Panama, Texas, Philippines, Japan, Korea, Indonesia, California, Washington, Kentucky, and Georgia. First noted anesthesia in left thigh in June 1964. In August had skin

changes in both legs and thighs. In October 1965 noted brownish, swollen area below both eyes. Admitted July 12, 1966. Diagnosis was dimorphous leprosy.

Case 31: White male, born 1936, Pennsylvania. Served in U.S. Army January 1955 to January 1958 in New Jersey, Kansas, and Georgia. Served in U.S. Air Force November 1961 to December 1966 in Okinawa (30 months) and in Vietnam (12 months). Onset in April 1965 as paresthesia of the right cheek, but diagnosed as leprosy by biospy March 1967. Admitted April 10, 1967. Diagnosis was indeterminate leprosy.

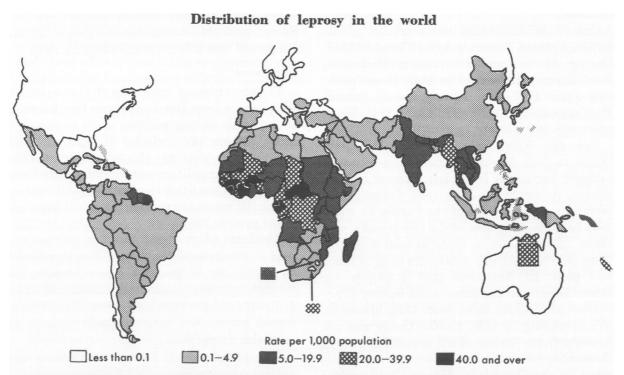
Case 32: White male, born 1924, Iowa. Lived there until he entered U.S. Air Force in 1943. From 1943 to 1963 served in Okinawa, Philippines, Guam, Cuba, Guatemala, Mexico, Texas, Florida, and Puerto Rico. Onset probably in 1964 when he became aware of a skin rash and sensory loss involving both lower extremities. Admitted February 27, 1968. Diagnosis was lepromatous leprosy.

The following patients were not admitted to the Public Health Service Hospital at Carville. Case 33: White male, born 1929, Louisiana. Lived there until he joined U.S. Navy in 1948. No record of foreign travel or other residence prior to service. During service spent 16 months in Pacific theater, with short periods in the Philippines, Hawaii, Okinawa, and Japan; discharged 1952. Onset probably in 1953 when an anesthetic red spot on right forearm was noted. Was examined as outpatient at Carville hospital, December 1953. Diagnosis was tuberculoid leprosy.

Case 34: White male, born 1914, Kansas. Lived in Kansas and Missouri until he joined U.S. Army in 1942. During service spent about 2 years in South Pacific theater; discharged 1946. Date of onset unknown but in 1945 was treated for "ringworm." By 1948 this lesion was about the size of a quarter and oval in shape. Diagnosis was tuberculoid leprosy.

Case 35: White male, born 1924, western New York State. Lived there until he joined U.S. Army in 1942. Served in Pacific theater—Philippines, 1943—45; discharged 1945. Onset probably in 1958 when sensory loss of both hands occurred, also thickening of facial skin. Diagnosis was lepromatous leprosy in 1959.

Case 36: White male, born 1933, Virginia.



Source: Adapted from Bechelli, L. M., and Martínez Domínguez, V.: The leprosy problem in the world. Bull WHO 34: 811-826 (1966).

Lived there until he joined U.S. Army in 1954. Spent 15 months in Korea; discharged 1955. Onset probably about 1958 when numbress of left little finger was noticed; later finger became contracted. Diagnosis was tuberculoid leprosy.

Case 37: White male, born 1907, Kansas. Lived there until 1940, then in Salt Lake City, Utah (1940-43). Joined U.S. Army in 1943. Was stationed in California, New Guinea, Leyte, and Luzon, Philippines. While on Luzon was quartered on the second floor of a native house. Discharged 1945. Onset probably in 1945 while in the Philippines, when an ulcer appeared on left calf. Lesion healed spontaneously leaving a hypopigmented, anesthetic, atrophic scar. During next 7 years the area of anesthesia enlarged slowly to involve most of the left calf. Diagnosis was indeterminate leprosy in 1953 (6).

Case 38: White male, born 1913, Tennessee. Lived there until he joined U.S. Army in 1940. During service spent 1943-45 in South Pacific theater. Discharged 1945 in Brisbane, Queensland, Australia. Onset probably in 1955 with the appearance of pigmented macules and papules on face and ears. Diagnosis was lepromatous leprosy. Died in 1960 of an acute heart condition.

Case 39: White female, born about 1911, California. Entered Women's Army Corps in 1942. During service spent 8 months in Hollandia, New Guinea; discharged in 1945. Onset probably about 1947 when leprosy was diagnosed. May have started in New Guinea in 1945. Diagnosis was lepromatous leprosy.

Case 40: White male, born 1922, Michigan. While in U.S. Marine Corps was tattooed on extensor surface of lower left forearm in Melbourne, Australia, in June 1943. Subsequently served in endemic areas of the Pacific. In April 1946 he noticed the area of the tattoo and a zone about 1.5 centimeter in width around it had become pale red and was insensitive to light touch and pain. In November 1946 a biopsy was taken. Diagnosis was tuberculoid leprosy (7).

Case 41: White male, born 1922, Michigan. While serving in U.S. Marine Corps was tattooed on flexor surface of left arm in Melbourne, Australia, in June 1943. Served in endemic areas of the Pacific. In January 1946 noticed area of tattoo and a zone about 1.5 centimeter

in width was becoming dusky red and numb. In November 1946 a biopsy was taken. Diagnosis was cutaneous tuberculoid leprosy (7).

Case 42: Male, race unknown, born 1930, Pennsylvania. Served in U.S. Air Force 1948-52 and U.S. Army 1952-68, in California, Mississippi, Japan (12 months), and Korea (24 months). Served 12 months in Vietnam and then lived in Georgia, Texas, Virginia, and California. Onset late 1967. Diagnosis was tuberculoid leprosy.

Case 43: White male, born 1944, California. Served in U.S. Air Force February 1964 to November 1965. Leprosy believed to have been contracted while serving in the Pacific. Diagnosis was lepromatous leprosy.

Case 44: White male, born 1907, California. Served in U.S. Army 1942-45 in Hawaii, Makin, and Saipan. Red spot on right deltoid region was diagnosed as leprosy, July 16, 1965. Type not reported.

Case 45: Negro male, born 1931, Tennessee. Military service in Korea 1951-52. Burned left forearm without experiencing pain in 1955. Diagnosis of tuberculoid leprosy (clinical and nerve biopsy) was made in Cleveland in 1960.

Case 46: White male, born 1924, Connecticut. Lived there until he entered military service. Served from 1943 to 1945. Spent 1 year in Guam. Diagnosis was tuberculoid leprosy in 1963.

Discussion

The occurrence of leprosy in 46 U.S. veterans with service from 1940 to 1968 can be attributed to exposure during military duty in endemic areas. Others not included because of their geographic origins may also have contracted the disease during military service rather than their place of residence before enlistment. All but one of the 46 veterans reported here had been exposed prior to 1960.

The total effect of the Vietnam war on leprosy in members of the Armed Forces will not be known for 10 years or longer because the incubation period is generally considered to be 3-5 years and the time lag between the first observed lesions and diagnosis will perhaps be several more years.

The prevalence of leprosy in Vietnam and the neighboring countries of Southeast Asia is estimated at 5 per 1,000 or higher; therefore, from the experience of World War II and the Korean war, during the next decade some new cases can be expected among U.S. veterans. Also in this period, a few cases will probably occur in former members of the Peace Corps.

No study has been reported of contacts of veterans whose disease was probably the result of exposure while in military service. One as yet unreported family study has come to our attention (personal communication from Dr. P. Fasal, Public Health Service Hospital, San Francisco, December 13, 1968). A U.S. veteran (case 22) apparently contracted the disease while serving 1 year in the Philippines, Okinawa, and New Hebrides with the U.S. Air Force. In 1962 he was admitted to Carville, 7 years after the onset of his disease. His son, born in 1952, was admitted at the same time with a diagnosis of tuberculoid leprosy of recent origin.

Examination of the other members of the veteran's family revealed that his wife had a reddish-brown and slightly infiltrated macule on the right side of her face; it had been present for several months. The histopathological diagnosis was tuberculoid leprosy. Lesions in a daughter, age 9, and a son, age 7, were diagnosed as indeterminate leprosy with histopathological confirmation. A fourth child, age 14, had no clinical evidence of the disease. The mother and three of the four children apparently became infected from contact with the father who contracted leprosy while serving in the U.S. Armed Forces overseas. The family lived in a nonendemic area in the United States.

The foregoing family study illustrates how leprosy can be transmitted to susceptible individuals. It also illustrates the importance of early diagnosis. Fasal, in his communication mentioned earlier, stated that the father was under medical care for 4 years before the diagnosis of leprosy was made. The 4-year delay in diagnosis prevented the father from receiving effective treatment while the disease was in the early stage; had he been treated at this time, the other members of his family probably would have been spared.

The long delay in diagnosis cited in the foregoing case is not unique. A study of admissions to Carville revealed that in many cases patients had been under medical observation for several

years before leprosy was considered by physicians (8). A similar delay has been reported in outpatients by Fasal (9).

Pathologists likewise may fail to consider leprosy among the differential diagnoses of skin lesions. One of us (C.H.B.) reported six cases of leprosy (10) in which pathologists on initial or subsequent biopsy examinations did not consider leprosy.

If leprosy can be overlooked by both the clinician and the pathologist, especially in its earlier stages, then delays on the part of the patient can be also expected. Even if the patient suspects leprosy, he may choose to hide the disease, hoping it will go away. A study of patients admitted to the hospital at Carville from 1955 to 1965 showed an average delay of 14.5 months after the onset of signs or symptoms before a physician was seen (8). Marshall (11) and Brubaker (12) and their associates reported factual knowledge of widespread leprosy in all age groups among Ryukyu islanders. Despite this knowledge, however, leprosy continues to be a crippling and deforming disease in the Ryukyu Islands—probably because of late diagnosis and late treatment (12, 13). Too often such is the case in the United States as elsewhere in the world.

As Marshall's study (11) pointed out, and what is now almost universally recognized, one reason for delay in seeking medical aid when the disease is suspected is because of the fear of social ostracism or of being sent away when leprosy is discovered. The high proportion of patients coming to light with deformity indicates delayed diagnosis and treatment. Failure of physicians to promptly suspect or recognize leprosy is to a great extent the result of the lack of emphasis on leprosy in medical schools. The reluctance of a person who suspects leprosy in himself or a member of his family to seek medical assistance has been fostered by archaic laws and public health regulations dealing with the person who has the disease. Changes in these legal restrictions are taking place in many States and foreign countries concerned with the problem, yet much remains to be done to match public health and humane concern with the present knowledge of the disease.

Irrespective of the absence of clinical activity,

a history of leprosy excludes a citizen from being able to enlist or to be commissioned in any branch of the U.S. Armed Forces. With rare exceptions, when leprosy is diagnosed in a person on active duty in a military service, regardless of how mild or amenable to treatment the disease may be, he is given a medical discharge.

Of interest is a young man who had been treated at the hospital at Carville and discharged because his disease was arrested. He applied for duty in one of the services and was accepted. He stated in his medical history that he had had Hansen's disease. After a few months of duty, the discovery was made that Hansen's disease was leprosy. Based on his history of leprosy, despite the lack of signs of active disease or deformity, he was discharged immediately. This and other examples, in and out of the service, indicate needed change in attitude as well as regulations if leprosy is to be placed in the mainstream of medicine and acceptable public health practice. Only with these essential changes will the medical profession gain the confidence of patients and potential patients that will encourage them to seek medical care at the earliest possible moment. When leprosy is diagnosed in its early stages and treatment begun promptly, public health and other physicians can give patients assurance that the disease can be arrested, that deformities can be prevented, and that they can live normally.

Summary

Before 1940, 83 cases of leprosy were reported in U.S. veterans. Thirty of these cases were considered to be the result of exposure to the disease outside the continental United States during the Spanish-American War.

From 1940 through 1968, 240 cases of leprosy were reported in U.S. veterans. As indicated in a résumé of their cases, 46 veterans were considered to have service-connected leprosy as a result of their exposure outside the United States.

No study has been reported of contacts of veterans with leprosy. However, one situation was brought to our attention in which leprosy was diagnosed in the wife and three children of an infected veteran. The family lived in a nonendemic area.

Delay in the early diagnosis of leprosy is caused by the failure of both patients and physicians to suspect the disease. Early diagnosis and treatment assure the best possible opportunity for arresting the disease and preventing disability and further spread by reduction of the infectious reservoir.

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Tearsheet Requests

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