Human Psittacosis in Milwaukee County Associated With Parakeets and Pigeons

FRANK F. PIRAINO, Ph.D., H. WISNIEWSKI, Ph.D., C. ABEL, B.S., and A. HAITA, B.S.

EIGHTEEN of the 27 cases of human psittacosis reported in 1961 in the East-North Central area of the United States (Michigan, Illinois, Ohio, Indiana, and Wisconsin) occurred in Wisconsin. The 18 cases were 17.6 percent of the 102 cases of human psittacosis that occurred in the United States that year. In 1962 Wisconsin reported 20 of the 29 human psittacosis cases that occurred in the East-North Central area, or 25 percent of the 79 human cases reported in 1962 in the United States (*1*).

Most of the Wisconsin cases occurred in the Milwaukee County area, with a population of 1,073,610. Thus it would appear that Milwaukee County either is an endemic area for human psittacosis or is more efficient in detecting this disease.

Our clinical, laboratory, and epidemiologic data were obtained from eight cases of human psittacosis, four in 1962 and four in 1963, all in Milwaukee County. Four occurred after exposure to parakeets and four after exposure to pigeons. All the patients required hospitalization at some period during their illness. Serologic evidence of psittacosis infection also was found among caged and wild pigeons in Milwaukee County.

Serum specimens were obtained from 40 caged pigeons associated directly or indirectly with the psittacosis cases and from 95 wild pigeons collected in various parts of the city of Milwaukee and adjacent areas in Milwaukee County. All were tested for psittacosis complement fixing antibody.

Dr. Piraino is chief virologist, Dr. Wisniewski is the director, and Mr. Abel and Mr. Haita are virologists, bureau of laboratories, City of Milwaukee Health Department.

Materials and Methods

Serologic evidence of psittacosis infection in the eight patients was first obtained by viral diagnostic tests of paired serums in the City of Milwaukee laboratory. Serums generally are mailed to the laboratory, where those of respiratory disease patients are tested routinely for complement fixing antibody to a battery of respiratory virus antigens, including the psittacosis Lederle antigen, which is prepared from allantoic fluids of infected chick embryos. For all cases of confirmed viral infections, a medical history questionnaire is obtained from the patient's physician. Additional information is obtained from the patient by personal visit to his home or by telephone.

The procedures used for detecting psittacosis complement fixing (CF) antibody in human and avian serums and neutralizing antibody in avian serums have been described in detail elsewhere (*2*, *3*). With rare exceptions, the indirect complement fixation test was used in testing avian serums and the direct complement fixation test was used for human serums. A Lederle laboratory antigen preparation of the 6 BC parakeet strain of psittacosis virus was used for detecting CF antibody. Tests for psittacosis virus neutralizing antibody in avian and human serums were based on the ability of a 1 : 10 dilution of serum to prevent plaques by 6 BC virus

in chick embryo cell monolayers. A positive serum reduced the number of plaques by 80 percent or more.

Results

Clinical and Serologic Findings

Tables 1 and 2 give the clinical and serologic findings associated with the eight patients with psittacosis. The symptoms in these patients were generally similar (table 1). The onset of illness was rather sudden, with chills, sweating, and fever which persisted from 1 to 2 weeks. X-ray examinations of the chest generally showed density in either the right or lower lung, most often in the left lower lung region, and often in the absence of auscultatory signs. All the patients eventually recovered completely after receiving tetracycline or chloromycetin therapy over a period of 4 to 14 days.

Tests for influenza were included in table 2 because Milwaukee had a rather severe episode of influenza during the winter of 1962–63. Results of our serologic tests for influenza infection and cold hemagglutinins were not diagnostically significant (table 2) although the case 1 patient had a concurrent high titer of cold agglutinins. Serum dilutions of 1:32 through 1:128 were used for influenza tests because only rarely have we found influenza associated with a CF titer of less than 1:32 dilution.

Patients 1, 3, 4, 5, and 7 had significant psittacosis reactions (fourfold or greater antibody titer rises between paired serums) in the absence of significant reactions to cold agglutinins and influenza. Patients 2, 6, and 8 had suggestive

Table 1. Clinical data on eight human psittacosis cases in Milwaukee County, Wis.

Case No.	Bird contact	Onset date of symptoms	Clinical diagnosis	Age	Sex	Maximum fever, °F. (days with fever)	Chills, sweating, headache, malaise
1	Wild parakeet	Dec. 23, 1961	Pneumonitis	56	Female	102	+
2	Caged parakeet	Oct. 1, 1962	Psittacosis	37	do	103 (5)	+
3	Wild and caged pigeons.	Sept. 24, 1962	Viral pneumonitis	48	Male	105 (14)	+
4	Caged parakeet	Dec. 6, 1962	Pneumonitis	46	do	104 (6)	+
5	Caged pigeons	Apr. 19, 1963	Virus pneumonia	45	do	103 (5)	+
6	Caged parakeet	Dec. 29, 1963	Pneumonitis	72	Female	104 (5)	+
7	Caged pigeons	Oct. 17, 1963	Psittacosis	49	Male	105 (5)	+
8	Caged pigeons	Nov. 22, 1963	Pneumonitis	25	do	102 (7)	+

	Anorexia	Sore throat	Cough	Sputum	X-ray findings	Therapy
1	+	−	+	Mucoid	Density in base of left lung [1](14); no change day 18.	Achromycin, 250 mg. q.i.d., 7–10 days.
2	+	−	+	Scant	Patchy pneumonia	Tetracyclines.
3	−	−	+	----------	Extensive pneumonia in left lower lung region [1](8); cleared by day 11.	Chloromycetin [1](8), 1 capsule q.i.d., 14 days.
4	+	−	[2]	Scant	Patchy bronchopneumonia-like consolidation in right lower lung [1](20).	Tetracycline, ½ gram q.i.d., 2 days; 250 mg. q.i.d., 12 days.
5	+	+	+	----------	Consolidation in upper right lobe.	Declomycin, 150–250 mg., with dramatic improvement in 24 hours, 10 days.
6	+	−	+	Scant	Hazy density in lower ⅔ of left lung [1](5).	Tetracycline [1](5), 250 mg. q.i.d., 4 days.
7	+	−	+	Scant	Density in lower left lobe of lung.	Penicillin, 1.2 million units per day, 10 days; chloromycetin, 500–1,000 mg. per day, 4 days.
8	+	+	[2]	Unknown	Minimal increase in density in right upper lung region [1](6).	Chloromycetin, 14 days.

[1] Day of illness.
[2] Unknown.

psittacosis reactions in the absence of significant influenza or cold agglutinin reactions. Patient 2, with a severe pneumonia, had a twofold increase in psittacosis antibody titer with no other significant serologic reaction. Patient 6 had a twofold rise in psittacosis antibody titer with severe pneumonitis, and the first blood specimen tested on the eighth day of illness already had a titer of 1:32. Patient 8 had a severe pneumonia with a twofold rise in psittacosis antibody titer.

Psittacosis reactions in the blood specimens of patients 2, 6, and 8 revealed suggestive evidence of psittacosis infection. This was supported by the absence of blood reactions to influenza antigens and cold agglutinins, by the clinical history of the patient, and by evidence of exposure to a bird shortly before illness. Histories of all eight cases are discussed in the following section.

Source of Infection

Case 1. Approximately 1 month before the onset of symptoms (Dec. 23, 1961) the patient found a parakeet outdoors and took it home as a pet. At the time the bird appeared healthy. About the time the patient became ill, her husband developed an upper respiratory infection, but his infection cleared in a few days. The bird was killed before it could be examined in the laboratory for elementary bodies.

Case 2. On and off for 2 years, until the time of her illness, the patient helped clean parakeet cages for a friend who raised them commercially. No bird was kept in her home.

Case 3. No animals other than dogs were kept in the home of the patient. However, he used trapped wild pigeons and culls from various sources in the city to train his dogs at a local sportmen's club. The patient's last exposure to pigeons occurred within 3 weeks before his illness.

Case 4. On November 21, 1962, the patient purchased a parakeet from a pet shop in Milwaukee. Fifteen days later the patient developed typical symptoms of psittacosis. The parakeet was killed for examination, and typical psittacosis elementary bodies were found in impression smears of the spleen. No attempt was made to isolate the virus. The case 4 patient

Table 2. Results of serologic tests of eight human psittacosis cases in Milwaukee County, Wis.

Case No.	Influenza A CF [1,2]	Influenza B CF [1,2]	Cold hemagglutinins [2]	Psittacosis CF [1,2]
1	<1:32 (14) <1:32 (20)	<1:32 (14) <1:32 (20)	1:2,048 (14)	1:8 (14) 1:128 (20)
2	<1:32 (4) <1:32 (9)	<1:32 (4) <1:32 (9)	<1:10 (4) <1:10 (9)	1:8 (4) 1:16 (9)
3	<1:32 (9) <1:32 (22) <1:32 (33)	<1:32 (9) <1:32 (22) <1:32 (33)	[3] [3] [3]	1:8 (9) 1:16 (22) 1:64 (33)
4	<1:32 (9) <1:32 (18)	<1:32 (9) <1:32 (18)	<1:10 (9) <1:10 (18)	1:64 (9) 1:256 (18)
5	<1:32 (5) <1:32 (13) <1:32 (54)	<1:32 (5) <1:32 (13) <1:32 (54)	<1:10 (5) <1:10 (13) <1:10 (54)	1:4 (5) 1:32 (13) 1:16 (54)
6	<1:32 (8) <1:32 (27)	<1:32 (8) <1:32 (27)	<1:10 (8) <1:10 (27)	1:32 (8) 1:64 (27)
7	<1:32 (7) <1:32 (28)	<1:32 (7) <1:32 (28)	<1:10 (7) <1:10 (28)	<1:4 (7) >1:32 (28)
8	[3] [3]	[3] [3]	<1:10 (7) <1:10 (14)	1:16 (7) 1:32 (14)

[1] The CF results are expressed as the highest serum dilution fixing guinea pig complement.
[2] Day after onset of illness when blood sample was taken is in parentheses.
[3] Not done.

donated a pint of blood, which we subsequently used as a source of psittacosis indicator serum for pigeons in studies discussed later in this paper.

Case 5. The patient lived in a rural area near Milwaukee. He kept a few ducks, about a dozen chickens, two dogs, and four pigeons. The pigeons had been purchased from dealer X about 3 weeks before his illness. The ducks and chickens had been on the premises about 6 to 8 months.

On June 7, 1963, 49 days after the onset of his illness, we visited the patient and obtained the third set of blood samples from him, and blood samples from his wife, his two children, and the four pigeons. The human serums were tested for psittacosis reactions by the direct CF method. The pigeons were tested by the indirect complement fixation procedure. Positive human serum from case 4 patient was used as the indicator serum. The pigeon serums and the patient's serum also were tested for neutralizing antibody to the 6 BC parakeet strain, as described earlier in materials and methods (table 3).

The patient developed an eightfold rise in CF titer for psittacosis, which can be interpreted as evidence of an active infection. Serums of the other family members had negative reactions. Complement fixing antibody in high titer and neutralizing antibody to psittacosis virus was present in all pigeon serums tested. The presence of the four ornithosis reactor pigeons on the premises of the patient shortly before the onset of symptoms is strong evidence that the pigeons were the source of the infection. The pigeons had been handled exclusively by the patient and his wife.

The pigeons were kept for observation in the laboratory an additional 4 months. One emaciated bird died suddenly after 1 month. The remaining pigeons were killed, and groups of white mice were inoculated intraperitoneally with liver and spleen emulsions of each bird. Tissue emulsions of livers and spleens of inoculated mice were used for two additional blind mouse passages, but no virus was recovered. On the second mouse passage, one pigeon inoculum induced an enlarged necrotic spleen. However, examination for elementary bodies was negative.

Case 6. The patient kept a parakeet in her home for 2 years, until the time of her illness. The parakeet was killed after psittacosis was suspected but was not available for examination.

Case 7. On October 9, 1963, 8 days before the onset of symptoms, the patient killed and dressed about a dozen caged pigeons obtained as a gift from a friend. No birds were kept in the home as pets. The patient's wife and son, who helped dress the birds, did not become ill.

Case 8. In July 1963, approximately 4 months before the onset of his illness, the patient purchased nine pigeons for breeding purposes and put them in a vacant garage. Two birds were from breeder A and seven birds from

Table 3. Tests for complement fixing and neutralizing antibody to the 6 BC parakeet strain of psittacosis virus, case 5, Milwaukee County, Wis.

Serum	Day after onset of patient's illness	CF titer	Percent survival 6 BC virus	Neutralizing antibody
Patient:				
First specimen	5	1:4	100	[1]
Second specimen	13	1:32	[1]	[1]
Third specimen	49	1:16	100	
Wife	49	<1:8	[1]	[1]
Child 1	49	<1:8	[1]	[1]
Child 2	49	<1:8	[1]	[1]
Pigeons:				
Female tumbler	49	1:32	12	+
Male tumbler	49	1:64	10	+
Female homer	49	[1]	5	+
Male homer	49	1:128	14	+

[1] Not done.

Table 4. Psittacosis CF reactions of wild pigeons from various locations in Milwaukee, Wis.

CF titer [1]	Number of reactive serums				
	Mitchell and 43d Sts.	Canal and 8th–20th Sts.	Forest Home Ave. and 35th St.	Brewery	Railroad repair shops
<1:8	11	10	12	21	35
1:8	0	0	0	1	0
1:16	0	0	0	0	2
1:32	0	0	0	0	3
Total	11	10	12	22	40
Percent positive	0	0	0	4. 5	12. 5

[1] No CF titers greater than 1:32.

breeder B in Milwaukee County. He kept no other birds. About 3 weeks before his illness, the patient transferred the birds from the garage to a loft, where he subsequently experienced heavy exposure to the pigeons by handling and feeding them.

The wife of the case 8 patient operates a pet shop where parakeets are sold. Although she claimed that her husband had little or no close contact with any of the parakeets, this source of infection could not be ruled out.

After the onset of his illness on November 22, 1963, the birds were returned to the original owners. The two pigeons of breeder A were killed, and no examination was possible. However, these birds had been in continuous contact with other birds in his loft that were available for serologic tests. The seven birds of breeder B were still alive and available for study. Blood samples from the seven birds as well as the other birds from breeder A were tested for psittacosis CF antibody. The CF titer and number of reactive serums from these birds are tabulated below:

CF titer	*Number of reactive serums*	
	Breeder A	*Breeder B*
<1:8	3	4
1:8	2	0
1:16	1	2
1:32	0	1
1:64	2	0
Total	8	7
Number positive	5	3
Percent positive	63	43

While the number of birds sampled was rather small, both groups had a significant fraction of psittacosis reactors. These data are consistent with our interpretation that the pigeons were the probable source of infection.

Table 5. Psittacosis complement fixing and neutralizing antibody (NA) found in blood specimens of caged pigeons in Milwaukee County, Wis.

CF titer	21 serum specimens from dealer X loft		25 serum specimens from town V loft	
	CF	Positive NA	CF	Positive NA
<1:8	6	0	21	0
1:8	3	0	1	1
1:16	2	0	0	0
1:32	2	2	1	0
1:64	3	2	1	0
1:128	2	0	0	0
1:256	1	0	0	0
>1:256	2	2	1	1
Percent positive	71. 5	28. 6	16	8

Serologic Reactors Among Wild and Caged Pigeons

Sufficient evidence was established that caged pigeons, kept for breeding and racing purposes, were associated with human psittacosis infections in patients 3, 5, 7, and 8. Therefore, it was of some interest to determine the relative incidence of psittacosis reactors among the caged pigeons associated with the human cases and among wild pigeons. This was done to gain some information on the potiential hazard of exposure to the two classes of birds.

Case 5 provided the opportunity for such a study. We visited the loft of dealer X, who had sold the four infected birds to the patient. None of the four pigeons were progeny of birds from his loft although they had been kept in close contact with other birds in the loft for about 6 weeks before they were sold to the patient. Dealer X had obtained the four birds from two other dealers. One had a pigeon loft in town V and the other had a loft in town W, both in Wisconsin. Serum specimens were collected from 21 of approximately 60 birds in dealer X's loft and from 25 of approximately 50 birds in the town V loft.

Wild pigeons were trapped in five areas in the city of Milwaukee: Two were areas where pigeon populations appeared to be rather heavy, near a railroad repair shop and around a brewery; three were areas on the south side of the city.

All blood specimens from both caged and wild birds were obtained by heart puncture and tested for psittacosis complement fixing antibody. Positive human serum from the case 4 patient was used as the indicator serum. Serums from caged birds were also tested for neutralizing antibody (NA). The results of these tests are given in tables 4 and 5.

Pigeons in the loft of dealer X, where the infected pigeons sold to the case 5 patient had been kept, had a CF reactor rate of 71.5 percent and NA reactor rate of 28.6 percent; pigeons in the town V loft had a CF rate of 16 percent and NA rate of 8 percent. It appears likely that the pigeons originally from the lofts in town V, and probably from town W, became infected while being held for 6 weeks in dealer X's loft, where his pigeons had experienced heavy infection by ornithosis virus.

The wild pigeons (table 4) were infected much less frequently than the caged pigeons associated with human cases and tested in this study (case 8 and table 5). Wild-pigeon CF reactor rates were 0, 0, 0, 4.5, and 12.5 percent compared with caged-pigeon CF reactor rates of 16, 43, 63, and 71 percent.

It is interesting that the level of infection (12.5 percent) for the wild pigeons tested at the railroad repair shops was near that of caged pigeons in the town V loft (16.0 percent, table 5). Nevertheless, the results of our study suggest that caged pigeons kept for breeding and racing purposes are potentially more hazardous sources of psittacosis infection than the wild pigeons trapped in Milwaukee County.

The presence of neutralizing psittacosis antibody in serums of pigeons positive for complement fixing antibody (table 5) is additional evidence of the relative rates of infection in these birds. Neutralizing antibody was not usually present in the blood of pigeons with CF titers of less than 1:32 and was not uniformly associated with CF antibody levels of 1:32 or greater.

Discussion

The eight patients with psittacosis have in common a clinical history of severe respiratory disease compatible with psittacosis infection, significant serologic reactions to psittacosis virus CF antigens in the absence of significant influenza and cold hemagglutinin reactions, and a history of exposure to either pigeons or parakeets, usually 1 to 3 weeks before the onset of typical symptoms of psittacosis infection. If the suspect bird or birds were available for laboratory examination, it was possible to show evidence of ornithosis virus infection based on the presence of psittacosis complement fixing or neutralizing antibody.

The initial symptoms in humans generally resembled those of influenza, with sudden high fever, which persisted from 5 to 14 days, chills, sweating, and headache. The convalescent period was characterized by a long, slow recovery, with extreme tiredness and general weakness. None of the patients died. Response to antibiotic treatment varied from a gradual but marked improvement to a dramatic change in the clinical condition almost within a day. The declomycin given to the case 5 patient had a dramatic effect; overnight his fever dropped from 105° F. to normal.

Of particular interest in this study were the cases associated with pigeon exposures. Even though Meyer (*4*, *5*) showed many years ago that pigeons carry a Bedsonia agent associated with human disease, this source of infection is not generally regarded as a serious public health problem. However, the more recent report of Japanese workers (*6*), and our studies reported

here, indicate that in certain areas where there is little control over large populations of caged and wild pigeons a significant public health problem may exist.

Our studies show that in the Milwaukee County area psittacosis infections have been associated with caged pigeons kept by hobbyists for racing and breeding. Meyer (*4*) found that 60 percent of caged pigeons have CF antibodies to psittacosis virus. Our own data show that 70 to 100 percent of caged pigeons associated with human psittacosis cases have CF antibodies, and 30 percent of these have neutralizing antibody. However, we also suspect wild pigeons because of evidence of human clinical episodes, similar to psittacosis, associated with exposures to wild pigeons. Such cases were not included in our data because we lacked conclusive serologic evidence of active infection, and it was impossible to examine the birds suspected. Case 3 involved contact with both wild and caged pigeons, but we could not establish the most probable source of infection. We have no definitive data to establish the role of wild pigeons in psittacosis disease in Milwaukee County.

The frequency of psittacosis reactor pigeons must depend to a large extent on the population density, flock susceptibility, and transmissibility of the virus. In particular, population density would explain in part the generally high incidence of CF reactors among caged pigeons compared with wild pigeons. Crowding could obviously facilitate bird-to-bird spread of the virus.

In cases 5 and 8, the frequency of reactors among caged pigeons directly in contact with the patients varied from 43 to 100 percent compared with wild pigeon reactor rates of 0 to 12 percent. At this time it seems prudent to consider caged pigeons with reactor rates of 50 percent or greater as potentially harmful sources of psittacosis infection, and probably some effort should be made to control the disease in these flocks.

What seems unusual is the relatively few cases of psittacosis observed when one considers the high incidence of reactor birds among certain caged pigeons. In our study none of the four cases occurred in persons who raise or sell pigeons commercially. All were in people who apparently had their first continuous exposure to infected birds. Studying the complexity of the infection process will certainly prove to be worthwhile.

The methods used for detecting human psittacosis are important from an epidemiologic point of view. Investigators in Japan first discovered that pigeons were infected with Bedsonia agents, and they then began looking for human cases (*6*). The psittacosis cases we have reported were first detected serologically by virus screening tests in which we routinely employed a battery of virus CF antigens in all cases of human respiratory disease. Followup studies later showed exposures to infected birds. Possibly this same approach, if used by other laboratories, would result in detecting more cases of psittacosis.

Summary

Eight cases of psittacosis in human beings occurred in Milwaukee County in 1962 and 1963, four attributed to exposures to pet parakeets and four to infected caged pigeons. Six of the eight cases were confirmed serologically; of the six, three were associated with pigeons and three with parakeets.

Serum specimens were obtained from 40 caged pigeons associated directly or indirectly with the psittacosis cases and from 95 wild pigeons collected in various parts of the city of Milwaukee and adjacent areas in Milwaukee County. All were tested for psittacosis complement fixing antibody. A group of caged pigeons also were tested for neutralizing antibody. Complement fixation reactor rates associated with the caged pigeons ranged from 16 to 71 percent compared with 0 to 12 percent for wild pigeons, from which we conclude that caged pigeons are potentially more hazardous sources of human infections than wild pigeons.

REFERENCES

(*1*) U.S. Public Health Service: Morbidity and mortality weekly reports. Vol. 10, 1961, pp. 1–28; vol. 11, 1962, pp. 1–28. Communicable Disease Center, Atlanta, Ga.

(*2*) Meyer, K. F., and Eddie, B.: Psittacosis. *In* Diagnostic procedures for virus and rickettsial diseases. American Public Health Association, New York, 1956, pp. 399–403.

(3) Piraino, F., and Abel, C.: Plaque assay for psittacosis virus in monolayers of chick embryo fibroblasts. J Bact 87: 1503–1511 (1964).
(4) Meyer, K. F., Eddie, B., and Yanamura, H. Y.: Ornithosis (psittacosis) in pigeons and its relation to human pneumonitis. Proc Soc Exp Biol Med 49: 609–615 (1942).
(5) Meyer, K. F.: Some general remarks and new observations on psittacosis and ornithosis. Bull WHO 20: 101–119 (1959).
(6) Matumoto, M., et al.: Miyagawanella: Psittacosis-lymphogranuloma group of viruses. IV. Serologically diagnosed human cases of psittacosis in Japan. Japan J Exp Med 28: 41–49 (1958).

Conference Calendar

May 2–7, 1965: International Academy of Pathology—American Association of Pathologists and Bacteriologists, Washington, D.C.

May 3–7, 1965: American Psychiatric Association, New York, N.Y.

May 3–7, 1965: National League for Nursing, San Francisco. Information: E. F. MacDonald Co., NLN Post-Convention Tours, 36 South Wabash Avenue, Suite 1301, Chicago, Ill., 60603.

May 4, 1965: World Health Assembly, Geneva.

May 4–7, 1965: U.S. Public Health Service Clinical Society (annual meeting), Staten Island.

May 10–13, 1965: National Conference on Interstate Milk Shipments, Louisville, Ky. Information: Shelby Johnson, Kentucky State Health Department, Frankfort, Ky.

May 21–23, 1965: International Congress of Exfoliative Cytology, Paris, France.

May 23–27, 1965: International Federation for Information Processing, New York, N.Y.

May 23–28, 1965: National Conference on Social Welfare (annual forum), Atlantic City, N.J.

May 25–29, 1965: American Association on Mental Deficiency, Miami, Fla.

May 30–June 2, 1965: National Tuberculosis Association—American Thoracic Society—National Conference of Tuberculosis Workers, Chicago.

May 31–June 3, 1965: Canadian Public Health Association, Edmonton, Canada.

June 3–4, 1965: Sanitary and Water Resources Engineering Conference (1-day concurrent session on Atmospheric Pollution and Control), Vanderbilt University. Information: Dr. Peter Krenkel, Associate Professor and Director of Sanitary and Water Resources Engineering, School of Engineering, Vanderbilt University, Nashville, Tenn.

June 7–10, 1965: U.S.-Mexico Border Public Health Association, Los Angeles.

June 14–18, 1965: Conference of State Sanitary Engineers, Washington, D.C.

June 20–24, 1965: American Medical Association (annual meeting), New York, N.Y.

June 20–24, 1965: National Association of Sanitarians (annual educational conference), Miami Beach, Fla.

June 27–29, 1965: International Congress on Smoking and Health, New York, N.Y.

June 29–July 2, 1965: International Data Processing Conference and Exposition, New York, N.Y.

July 10–17, 1965: International Conference on Health and Health Education, Madrid. Information: ANCHEP, 800 Second Avenue, New York, N.Y., 10017.

July 11–15, 1965: American Veterinary Medical Association, Portland, Oreg.

Announcements for publication should be forwarded to Public Health Reports 6 months in advance of meeting.