Recent Studies on Psittacosis*

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PSITTACOSIS as a problem attracted the attention of the public health authorities during the pandemic of 1929–1930.[1] Single or group infections followed the exposure to parrots. At first, it appeared that the shipments of these birds from Argentina, South and Central America, with their ensuing distribution in pet shops and the contact infections in these places, would explain the pandemic. It is now evident that South America was not the only source of infection. Although the influence of the Argentine epidemic, with its subsequent rapid disposal of infected birds from the stores in South America, on the total number of human cases is great, it is quite obvious that other unknown factors must have operated, and other primary foci must have existed.

Under the influence of the universal and effective publicity which was given in the lay and scientific press, undoubtedly sporadic cases, which would ordinarily have escaped recognition, were brought to light. The data of the past 2 years amply support this statement. Following a case of psittacosis in a prominent person, attention is called to this supposedly rare disease. Several cases and even epidemics have thus, in rapid succession, been discovered by health departments.

Even under California conditions where the interest has been kept alive among physicians and health officers, the report of one case is usually followed by the recognition of others. It is, therefore, the belief of those who have dealt with this malady that, during inter-epidemic periods, many cases have escaped detection and notification.

That birds other than South American parrots were involved in the epidemic of 1929–1930 in the United States is now fully realized. In December, 1929, Dr. Dannenbaum diagnosed a case of psittacosis (clinical and X-ray findings typical) in a patient who had received several shell parrakeets from a San Francisco pet shop. In January, 1930, Sandoer and Coburis [2] saw a mild and a severe psittacosis infection in a mother and daughter who had handled " love birds " (shell parrakeets) originally shipped from Los Angeles. Dr. Chisholm at Vancouver and Dr. Ridewood at Victoria, B. C., made similar observations in March, 1930 (McIntosh [3]).

Late in December, 1931, the Division of Epidemiology of the California State Department of Public Health was notified of the death of 3 people and the illness of a fourth, all of whom had been together shortly before their illness at a home in Grass Valley. The clinical

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history of a typhoid-pneumonia-like disease, in form of a house epidemic, suggested the diagnosis of parrot fever. Inquiries established the fact that a healthy shell parrakeet was in the home but that the mate had died on or about December 1, and that the husband, who was seriously ill, had buried it. A clinical examination of the patient in the light of these facts left no doubt that the symptoms were those of severe psittacosis. Subsequently, the presence of the virus in the sputum, and at autopsy in the lung, liver, and spleen conclusively proved the correctness of the diagnosis. Furthermore, the parrakeet, .which had been found in the house and held under observation for 43 days after the death of its mate, revealed on autopsy an enlarged spleen from which the psittacosis virus was readily obtained by mouse passage.

Incident to the publicity which was given to the dramatic deaths through contact with shell parrakeets, reports of clinically typical cases of parrot fever, observed by physicians during the past few months, gradually filtered in from the southern part of the state. The victims had been exposed to parrakeets of their own breeding or to birds which had been raised in the aviaries of Southern California.

Investigations conducted by the health authorities during the month of January, 1932, disclosed a vast bird breeding industry in parrakeets and other birds, concerning which very little had been known. Laboratory examinations of sample birds obtained from some of these aviaries showed that a fairly large percentage of the shell parrakeets carried the psittacosis virus in their spleens and livers. There was no doubt that psittacosis, as an infectious disease of the shell parrakeets, existed in the California aviaries. Drastic measures of control could not be instituted since thousands of people, directly or indirectly, derived their living from the breeding and raising of these birds. Aside from the public health aspect, the control of psittacosis assumed the importance of an industrial problem. It is this phase of the subject which had to be considered continuously in the investigations which were undertaken.

These data, together with previous and subsequent reports, rather forcibly call attention to the importance of the shell parrakeet as a source and spreader of the psittacosis virus. One recalls the epidemic observed by Friedrich (1892) at Dühringsdorf near Landsberg, the house epidemic at Krefeld (1899),[4] and at Zülpich (1909),[5] the peculiar malady on the campus of the Veterinary College at the University of Zürich in January, 1930,[6] and the isolated cases at Geneva (1929),[7] Graz (1929),[8] at Vancouver (1930),[3] in England (1930),[9] and in New York (parrakeets of Cuban,[10] Japanese and Dutch origin). More recently Fortner and Pfaffenberg[11] discussed their investigations undertaken in connection with 29 psittacosis family epidemics of 62 cases (54 in Berlin) and a mortality of 12 or 19.5 per cent. Equally significant are the reports from Saxony from 1931 to 1934.[11] Sixty-six cases of psittacosis with 15, or 22.7 per cent, deaths were caused by locally bred and raised parrakeets.

It is apparent that the breeding establishments of many countries, dealing principally with the much desired shell parrakeets (*Melopsittacus undulatus*), are infected with psittacosis and are a definite menace to the owners, the dealers of pets, and the patrons of these distributing agencies. Until it was recognized that domestic birds carry psittacosis the control of the disease, by preventing the traffic in tropical birds, appeared relatively simple. In view of the newer knowledge, the procedures have to be changed and the evil must be attacked at its root. The California studies furnish data which may serve as

guides in the formulation of adequate protective measures.

THE CAUSE OF PSITTACOSIS

The investigations conducted during the pandemic of 1929–1930 established as the etiologic agent of psittacosis a corpuscular virus, in part, filterable. In particular, the discovery of the minute, Gram-negative ovoid or spherical bodies in virulent human and bird tissues by Levinthal, Cole, and Lillie,[12] and the recognition of the white mouse as a very susceptible test animal for the virus by Krumwiede, McGrath, and Oldenbusch[13] have greatly facilitated the practical diagnosis of psittacosis. The intraperitoneal injection of mice with the suspected material constitutes the best and safest procedure to demonstrate the presence of the virus.

No examination can be considered complete without a careful search for the elementary bodies described first by Levinthal, which possess in the diagnosis of psittacosis the same value as the Negri bodies in rabies. The particle size of the elementary bodies and their intracellular location (Bedson[12] and personal observations) render filtration of the virulent specimens an irregular procedure. Consequently, infectious sputa or other secretions or excreta cannot be safely freed from their bacterial contaminations by passage through infusorial or asbestos filters without the risk of losing the virus in part or in toto. Fractional centrifugalization of broth suspensions and repeated subcutaneous injections must be employed in the search for the psittacosis virus in contaminated specimens. The virus resists the action of glycerine (pH 7.4) for at least 66 days when held at 5° C. Desiccated bird spleens remain virulent at 5° C. for 277 days. Virulent contents of the cloaca rapidly lose their infectiousness on desiccation at room temperature.

The Salmonella psittacosis has been isolated in one instance only in the parrakeets of one local breeding establishment. In a shipment of 132 South American paroquets and parrotlets, 12 per cent of the birds harbored *S. psittacosis,* an organism closely related to "*S. aertrycke*" in the intestines, liver and spleen.[14] Certain strains of the psittacosis virus increase in virulence for mice by passage and may infect in a dilution of 10^{-12}. Sputum strains and those from "epidemic" birds are more rapidly "passed" than the Australian parrakeet and the South American carrier strains.

SUSCEPTIBILITY OF BIRDS AND MAMMALS

A phylogenetic study of the natural hosts of psittacosis is still in progress. From a practical standpoint it is important to recognize that a great many species of birds, various rodents, monkeys, etc., may be infected by injection or by feeding. By exposure to infected parrakeets, the susceptibility of the fowl (*Gallus gallus* belonging to the order *Galliformes*), parrotlet, conure and cockateel (order *Psittaciformes*), Java sparrow (order *Passeriformes,* family *Ploceidae*), canary, nonpareil, Bengalese (family *Fringillidae*), and Pekin robin (family *Timaliidae*) has been definitely established by placing them in the breeding pens of the budgerigars. Particularly susceptible are the Java sparrows. These birds may serve as excellent sentinels for the detection of the presence and the dissemination of the virus in a room or pen in which parrakeets with latent psittacosis are held. Young chickens intimately exposed may succumb to psittacosis. The virus grows freely in the chick embryo when placed on the chorion-allantoic membrane of the developing egg.

EPIDEMIOLOGY OF HUMAN PSITTACOSIS

Since December, 1929, 189 cases of psittacosis and 14 suspects with 40

deaths, or 21.28 per cent mortality, have been reported in the United States and Canada. One hundred and seventy cases (14 suspects) had direct or indirect contact with diseased shell parrakeets. In California, 70 cases resulted from exposure to locally raised birds, while in other parts of the Union or in Canada shipments of the California birds were responsible for 98 additional cases. During the same period 17 infections due to contact with parrakeets from Cuba, " the Orient," Yokohama, and Holland have been recorded. The origin of the parrakeets in 2 cases (Wisconsin and Minnesota) remained undetermined. Two patients contracted their infections in California through exposure to canaries.[15] The virus was demonstrated in the spleen of 3 of 12 canaries. In the California series, at least 3 and in all probability 6 cases were human to human transmissions.

In the Pittsburgh outbreak, a non-fatal case transmitted a fatal infection to the nursing sister. For the first time, the transmission chain was conclusively proved in 2 instances. The psittacosis virus was demonstrated in the sputum of the patient and in the blood and sputa, respectively, of the 2 nurses who became infected. In another instance, the lung of the fatal case was virulent and the sputum of the nurse contained the same infective agent. The clinical manifestations repeated themselves with their usual variability in intensity; in this respect they differed in no way from the classical descriptions available.

The incubation time, although in many instances difficult to establish, is definitely known in 15 cases and varied from 7 to 14 days after initial contact. In 3 cases a single exposure occurred and the incubation time was 7, 8, and 9 days, respectively. In human to human transmission the interval between the onset of the illness and the discharge of the nurse from the case was 8 and 13 days. Important is the observation that 30 days elapsed between the death of a woman and the first symptoms of psittacosis in her nurse. This must be considered an exceptionally long incubation period since it is reasonable to suspect that the transfer of the virus occurred a few days before the death.

No explanation can be offered for the noteworthy fact that the same degree of contact and the same virus may induce diseases of varying degrees of severity. It is not unlikely that the susceptibility of man is quite variable and that a fair percentage may pass readily through the disease which remains in a latent or sub-clinical stage. In isolated cases, exposure to sick or dead shell parrakeets may induce clinical manifestations which, in any one patient or in any stage of the malady, are insufficiently characteristic to enable the physician to make a diagnosis. The recommendation, therefore, may be safely made that, until the sale of non-infected birds can be guaranteed, it is well to be biased and to suspect psittacosis whenever a patient has recently brought birds liable to this disease into his or her household and suffers from severe influenza, complicated by a " migrating " pneumonia. However, it is imperative that this suspicion or a clinical diagnosis be confirmed even though present laboratory methods frequently decide merely in retrospect. A definite laboratory diagnosis is desirable for epidemiologic reasons.

An examination of the blood for virus has little diagnostic value. In 28 clinical cases examined, the citrated blood of 3 patients infected mice when the blood was collected on the 1st, 2nd, and 4th day, but not on the 9th, 16th, or 17th day of the illness. On the other hand, the examination of sputa, first introduced by Rivers and Berry,[16] has proved quite valuable. In a series of 60 sputa, the virus was conclusively demonstrated in 14 patients whose expectorations were collected on

the 5th, 6th, 7th, 9th, 10th, 12th, 14th, 16th, 23rd, and 37th day, respectively, after the onset. One patient furnished a positive sputum on the 5th and on the 10th day but a negative one on the 16th day, while in a fatal case, the secretion was infectious on the 14th, 19th, and 23rd day of the disease. The elimination of the virus is irregular, and repeated examinations of 24 hour specimens should be made. The autopsy material from 6 human cases was studied. The lungs invariably contained the infective agent in such concentrations that the mice succumbed in from 6 to 8 days with typical lesions. The pathologic and histologic lesions correspond to those described by Lillie.[17]

Further experiments and observations are needed before a definite opinion can be expressed concerning the pathways of transmission of the virus from birds to man. The apparent high infectivity of the dispersed psittacosis virus is shown in the histories in which very short exposures occurred in pet shops, households, and baggage cars where diseased birds were kept. There is considerable evidence that *aerial convection* plays a very important rôle. Monkeys are non-susceptible to subcutaneous administrations of virus while intratracheal injections may induce a disease resembling human psittacosis. The ordinary means of contagion cannot be excluded since the handling of dead birds, feathers, excreta, etc., is frequently reported.

An interesting characteristic of psittacosis outbreaks is the occurrence of multiple cases in the same household. The great epidemics of the past occurred during the winter months. Epidemiologists, therefore, have expressed the opinion that the prevailing disposition to respiratory infections during the colder months of the year favored the spread of psittacosis. Recent observations in California and the United States at large have shown that the seasonal fluctuations of the disease are influenced by the prevalence of infected birds. Many cases have been seen in September and October, and severe psittacosis is not uncommon in midsummer. Usually, in the fall and early winter, immature carriers, sick birds and their mates, which cannot resist the rigors of transportation, reach their destination. The climatic factors are of significance only in so far as they affect the resistance of the birds and the frequency with which human beings may be brought in contact with them through prolonged exposure in the closed rooms of a winter household.

The majority of psittacosis infections have occurred in people of middle age. The lower susceptibility of children is well known. Intimate exposure of children to the same parrakeets which infected parents or older relatives indirectly, has repeatedly been noted. Careful investigations indicate that these contacts failed to produce disease in children. The greater frequency of psittacosis in women (California 48 ♀ : 22 ♂) is, in part, due to the fact that they are either engaged in the breeding of parrakeets for their livelihood, or as lovers of pets they come more closely in contact with birds. That persons engaged in the breeding, raising, transportation and sale of birds are particularly liable to psittacosis is fully recognized. Of the 70 cases of psittacosis infection reported in California, 27 or 38.5 per cent were in owners of large or small parrakeet aviaries or in members of their families. Three other groups—nurses, physicians, and laboratory workers—are equally exposed to the risk of occupational psittacosis.

The case mortality rate in the 187 cases was 40, or 21.28 per cent, or for the California cases of the period 1929 to 1934, 70 with 14, or 20 per cent, deaths. No fatality occurred below age 38. Very much higher rates have been

reported in the past. In one house epidemic in California, the rate was 100 per cent. It appears reasonable that the systematic inquiries which followed the discovery of 1 fatal case usually revealed 1 or 2 others but milder cases, and thus helped to ameliorate the staggering mortality.

THE EPIDEMIOLOGY OF AVIAN PSITTACOSIS

Although the histories of the California single or group infections of psittacosis mention exposure to sick or dead shell parrakeets, it has unfortunately been possible in but 4 instances to secure the virus for examination from these sources. At the time the cases had been clinically recognized, the carcasses had been buried or destroyed and only the mates of the pairs of budgerigars, which brought the disease into the household, or parrakeets from the same breeding establishment which furnished the epidemic birds, were available for examinations. Thus in 3 instances, apparently healthy shell parrakeets were submitted for study. According to the epidemiologic inquiries, 40 or at least two-thirds of the owners of birds had been exposed to sick or dead parrakeets, while 19 had merely had contact with "healthy" birds. Previous observations left no doubt that apparently healthy parrakeets may transmit a disease to man, but since the fatalities among parrakeets in aviaries are by no means uncommon despite emphatic denials by the bird raisers, it cannot be denied that probably a far larger number of human psittacosis cases resulted from exposure to sick and dead birds. However, it must be kept in mind that Fortner found 30 of the 35 parrakeets involved in the German outbreaks to be clinically normal.

It has been demonstrated in a large series of tests that the acutely ill parrakeets or those suffering from relapses are more liberal disseminators of the virus than those with latent infections, but clinically normal. Unfiltered suspensions of the spleen, liver, contents of cloaca and nasal mucosa, removed at autopsy with separate instruments, have been tested on white mice with the following results:

1. Epidemic birds: spleen and liver contain virus 4; cloaca content infectious 4; nose infectious 3.
2. *Acute* fatal infections not connected with outbreaks: spleen and liver contain virus 36; cloaca content infectious 25; nose infectious 26.
3. *Latent infections:* (64) spleen and liver contain virus 59; cloaca content infectious 21 (2 weak); nose infectious 30.

In the acute stage, both the nasal and fecal elimination of the virus may be demonstrated to occur simultaneously, although birds may be encountered in which either no virus discharge may be demonstrated or it may be found only in the nasal mucus or the cloacal contents. The latter are very rich in virus when the birds exhibit signs of diarrhea or polyuria. Since the kidneys are as a rule highly infectious, while the small intestines and colon are not, it is reasonable to assume that, in some of the birds at least, the urine may be the vehicle for the virus. Parrakeets with splenic or hepatic localization eliminate the psittacosis virus slightly more frequently by the nasal than by the anal route. In a small percentage of birds, the viscera were free from virus while the nasal mucosa was infectious; several presented anatomically the residuals of past infections. Breeding hens may carry the virus in the ovaries or in the yolk of the eggs found in the oviduct; congenital transmission of the psittacosis virus is thus suspected. As a whole, the studies lend support to the epidemiologic observations that a visibly diseased bird is more dangerous than carriers on account of the abundant contamination of the environment with highly infectious fecal material.

The epidemic birds or their mates submitted for examination were found to be immature, less than 6 months old. Experimental infections and numerous surveys have shown that the immature and sexually undeveloped parrakeets succumb more readily to psittacosis than the mature breeding birds. For example, the mortality in a flock of 33 *immature* parrakeets from infected and non-infected aviaries, injected with a passage of virus active in a dilution of 10^{-8}, was 10, or 33 per cent, while by comparison under identical experimental conditions, 72 *mature* birds from the same breeding establishments showed a mortality of 3, or 4.1 per cent. Equally corroborative are the data collected from the numerous surveys of aviaries. A sample of 100 birds from one aviary consisted of mature and immature parrakeets. Anatomically and by mouse tests, 8 per cent of the mature and 48 per cent of the immature birds were found to harbor the virus of psittacosis. In 163 aviaries, the 699 parrakeets anatomically suspected of psittacosis were classed according to age as follows: mature: 378 of 3,814, or 9.9 per cent; immature: 321 of 1,850, or 17.3 per cent.

Relatively little is as yet known concerning the flock infection and flock immunity. Mature and immature shell parrakeets selected from pens in which latent psittacosis has been demonstrated may be experimentally infected. By comparison with birds from non-infected aviaries, the mortality is lower (infected 17.3 per cent; non-infected 35 per cent). Aside from the lower mortality, the rate of recovery is slightly greater. Within 28 to 36 days, 33 per cent, and within 135 days, 77 per cent of the parrakeets from infected aviaries free their organs of the virus. Only 39 per cent of the birds from non-infected premises recover within 51 to 144 days. In flocks of old birds, one may find parrakeets which dispose of the virus inoculated within 4 to 5 days. Our present knowledge is entirely inadequate to explain this resistance; the preliminary studies merely indicate that it is not associated with neutralizing antibodies. Ample data are available to support the general observation that the latent infections in a flock of parrakeets may, within 6 to 8 months, be reduced to 1 or 2 per cent. As a rule, a very weak virus may be demonstrated in the spleen and liver. The cloacal content and nasal mucus is, as a rule, free from virus. These carriers are non-infectious; ricebirds exposed in the same cage remain well. However, psittacosis is not eliminated from the aviary.

With the resumption of breeding operations after an elapse of from 6 to 8 months, psittacosis will flare up again and acute parrot fever in young and even old birds will be noted. A variable percentage up to 50 per cent of the 2 to 4 months old parrakeets show at autopsy acute or chronic lesions of the disease. The virus is present in the spleen, liver, cloaca, and nose. When mingled with susceptible birds, they readily disseminate the infective agent. Any aviary once infected remains so, despite excellent sanitary management and systematic removal of sick birds. Complete isolation for 1 year may reduce the number of carriers as judged by the anatomical lesions and the demonstration of the virus by mouse tests, but it never eradicates the disease.

That isolation and quarantine, particularly the customary 2 or 4 weeks, offer little or no protection has been amply attested by the experiences of recent years. It will suffice to submit some observations to illustrate the prolonged intrinsic infectiousness of the avian psittacosis carriers and the relatively long incubation time.[18]

1. A pair of apparently healthy "parakeets," though suffering from latent psittacosis, infected ricebirds ex-

posed in the same cage on the 1st, 21st, and 40th, but not on the 87th day. The incubation time in the ricebirds was at first 8 to 10 days with death on the 14th and 15th day; later it was 40 days with death on the 48th day.

Of 2 ricebirds exposed to 2 shell parrakeets with latent psittacosis, 1 contracted the disease on the 50th day and was sacrificed in a moribund stage on the 55th day of exposure.

2. The longest time intervals between injection and death observed by Meyer and Eddie were 41, 61, and 98 days following the intramuscular application of the virus. In exposure tests, sickness observed on the 95th day was followed by death on the 106th day.

In order to detect acute avian psittacosis in a shipment of birds, it is obviously necessary to extend the quarantine from 2 to 3 weeks, preferably 5 months. Of course, it must be realized that even this precaution gives no assurance that latent psittacosis is nonexistent in a foreign shipment, as observations on Australian [19] and South American psittacine birds [14] have clearly demonstrated. Finally, it is of some interest to know how psittacosis may have invaded the California aviaries. The original stock came from Japan and various other sources, even from Australia. It is now proved that the native Australian budgerigar is spontaneously infected, and thus it is reasonable to assume that avian psittacosis was introduced into the breeding establishments, and since it was not recognized, it spread, through no fault of anyone, to a large percentage of the aviaries. As long as only carefully selected mature birds were distributed, the closed latent infections presented no menace to man. Mass breeding by the inexperienced, distribution and mixing of immature birds from various aviaries, perhaps cyclical factors in the infectiousness of the virus, finally lead to the situation with which the public health authorities and the industry are now confronted.

PROTECTIVE MEASURES

Theoretically, psittacosis is a disease which could be easily controlled, provided the public would appreciate the possible danger inherent in contact with birds, particularly those of unknown origin. This is obviously impossible when a group of people earn their living by raising parrakeets. Earnest attempts were, therefore, made to eliminate from the traffic those birds which are particularly dangerous spreaders of the disease. After 2 years of struggle to accomplish some degree of protection by means of restrictive measures, such as isolation for 30 days, it became apparent that without an extensive force of inspectors and an expensive system of continuous control the diseased birds cannot be eliminated from the usual local and national traffic.

As already emphasized, in California, psittacosis is not only a public health but also an industrial problem. Both interests are best served by the creation of an aviculture free from psittacosis. The speed with which this program may be executed depends entirely on the fund which may be available to make surveys and to eliminate the aviaries which harbor infected birds.

A recent survey of 164 aviaries by the Department of Public Health has shown that approximately 55 per cent of the establishments yielded by repeated examinations parrakeets which were anatomically, and by mouse tests, free from latent psittacosis. Furthermore, several aviaries, held under observation for nearly 2 years, have furnished non-infected immature birds every time they have been sampled. It is, therefore, reasonable to hope that in due time a nucleus of aviaries free from disease may be created. Plans are in progress to establish a number of so-called " rehabilitation aviaries " which

will furnish the breeding stock for the building up of the industry. These aviaries, managed and maintained by a group of bird breeders, remain under the supervision of the Department of Public Health laboratory in order to insure the permanent elimination of psittacosis in the breeding pens. Until the desired goal—no psittacosis in the American bird industry—has been reached, California will restrict the sale and distribution to lots of parrakeets which have been sampled and tested in the laboratory. At the same time, every effort will be made to alleviate the hardship and suffering imposed upon the industry by the restrictive measures which of necessity had to be adopted in order to protect the public health.

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