THE SIGNIFICANCE OF A PLEUROPNEUMONIA-LIKE ORGANISM IN KENNEL COUGH[2]

By A. S. GREIG[1]

Kennel cough refers to an incompletely defined respiratory condition of dogs recognized by many small animal practitioners in Canada, the United States and England. Reference to kennel cough in literature is scanty, but there are a few clinical reports of respiratory conditions in dogs described under several different names. All of the descriptions contain elements of similarity which suggest that the same disease is being considered in each case.

Whitney (1) in 1943 was the first to differentiate the disease on a clinical basis as "House dog" disease or pharyngo-laryngotracheitis. The early symptoms of infection were a type of retching cough and mild diarrhoea accompanied by only a slight elevation in body temperature. These symptoms usually disappeared in a few days, but occasionally persisted for several weeks, and included tonsillitis and swollen parotid glands. In some of the 309 cases which formed the basis of his paper, pneumonia developed around the seventh day after the start of symptoms. In a large percentage of his cases apparent recovery of the respiratory condition was followed in a month or so by acute signs of nervous disorder. Twitching of groups of muscles, vertigo, lethargy or convulsions usually led to death of the animal in a few days. Throughout the course of the disease the appetite remained good.

Microscopic examination of brain sections of those showing nervous signs revealed "non-suppurative encephalomyelitis with considerable demyelinization".

Whitney differentiated this condition from distemper on clinical grounds, and on the lack of distemper inclusion bodies in histological sections. He felt that the causative agent was probably viral in nature because of the obvious infectiousness of the disease coupled with inconclusive bacteriological findings.

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2. Presented before the Meeting of the Ontario Veterinary Association, Jan. 15, 1954.

CORRECTION

The caption under figure II in the article "Swine Rhinitis Studies" by Dr. G. R. Carter in the July issue on page 249 should have read as shown here.

"Pig 719A killed six days after inoculation with the PPLO. Note adherence of the lung to the thoracic wall and the presence of fibrinous bands along the borders of the spleen and liver."

In 1947 Gustafson (2) working in the Yale University School of Medicine was able to transfer the disease through eleven serial passages in dogs. He obtained his original material from nasopharyngeal washings of an infected dog, and inoculated his experimental animals by the intranasal route. The first two nasal irrigations were made with sterile broth, but later irrigations were made with broth containing penicillin and streptomycin in amounts up to 2500 units of each per ml. The incubation period before first noticeable symptoms varied from 5 to 19 days, with the majority occurring between 8 and 11 days. The duration of disease was from 3 to 28 days, and many of the inoculated dogs died. Guinea pigs and mice inoculated with nasal washings from infected dogs remained normal. Bacterial free chorioallantoic fluids from one group of eggs inoculated with nasopharyngeal washings of the 9th dog passage when given intranasally to a dog produced respiratory infection after an incubation period of 14 days.

An organism of the Shigella species isolated from feces of some of the dogs did not appear to have any significance in regards the respiratory symptoms of the inoculated dogs or the diarrhoea which frequently accompanied the disease. Several of the inoculated dogs developed bluish corneal opacities which in the light of more recent knowledge of dog disease is suggestive of canine infectious hepatitis. A few of his dogs developed nervous symptoms, but the etiological agent could not be recovered from brain material.

In England at the same time, 1947, Townson (3) published in the Veterinary Record a paper on a condition he termed "Infectious Catarrhal Fever of Dogs". He mentioned that the condition was known in England also as "X-disease", German distemper and American distemper. The symptoms and course of infectious catarrhal fever were similar to those of kennel cough. There was a slight cough, diarrhoea, and in this instance some inappetence. There was acute tonsillitis and pharyngitis, enlargement of submaxillary, inferior and superior cervical glands, rhinitis and conjunctivitis. Body temperature varied from 103°F. to 105°F.

Townson differentiated the condition from distemper on the grounds that many of the cases had been immunized against distemper by the Field Method or had previous histories of distemper infection. Also, inclusion bodies of distemper were not found by histological examination. *Brucella bronchiseptica* organisms were not recovered, but beta hemolytic streptococci were frequently found. However, penicillin was ineffective in treating the disease.

Fourteen to 28 days after apparent recovery many cases would develop nervous symptoms consisting of twitching of the lips, salivation and epileptiform fits which at first would last about 2 minutes. The fits increased in severity and duration with time and usually eventually terminated in death of the dog. Townson held the opinion that although streptococci were involved in the initial stages of the disease, some other agent, probably a virus, was the cause.

In 1948, Leask (4) in England, published on a highly contagious disease of dogs which came to his experience during three years of private practice. The disease resembled distemper, but had a tendency to recur. In fact, the recurrent feature of the disease, and the fact it occurred in dogs of all ages and in dogs previously immunized against distemper led him to believe he was dealing with a condition distinct from distemper. He described two forms of the disease, the acute form exhibiting intense upper respiratory symptoms and elevated temperature, and a chronic form characterized by a slight cough but little elevation in temperature. In only a few instances did nervous symptoms develop.

Ray (5) in 1948, considered that kennel cough was due to *Brucella bronchiseptica*, and the earlier work of Torrey and Rahe (6) indicates that this organism unassociated with distemper can indeed cause respiratory distress in dogs.

In 1951, at Cornell University, during the study of respiratory complications of the dog both in association with distemper and in the absence of distemper, an agent was isolated in the lungs of mice which subsequently proved to be a member of the pleuropneumonia-group of organisms (7). Although only one other strain of this organism was found capable of growing in mouse lungs, several strains were isolated directly from dogs in media especially designed for the propagation of pleuropneumonia-like organisms. (P.P.L.O.)

P.P.L. organisms, although by no means new in the field of bacteriology, have received prominence lately by their association with chronic respiratory disease of chickens and sinusitis of turkeys (8), and by their presence in the turbinates of pigs affected with atrophic rhinitis. (9). In previous years, members of the group have been isolated from the respiratory passages of mice, rats and dogs (10). In regards the dog isolations, Shoetensack (11) in 1934 obtained two serologically distinct strains and was convinced that one of these was the virus of distemper. At that time there was no clear differentiation between the viruses and PPLO, the latter because of their filterability being considered viruses capable of cultivation on non-living medium. The interesting feature of his work was the degree of pathogenicity exhibited by his organisms. Inoculation of cultures by the intranasal route resulted in acute respiratory distress which frequently terminated in death. The course of the disease produced was indistinguishable to him from distemper.

In 1951 Edward and Fitzgerald (12) in England reported the isolation of 3 serologically distinct types of PPLO from the throats and vaginas of dogs. Although Edward did not report any studies on pathogenicity, he was able to isolate the organism from apparently normal animals.

The strains of PPLO isolated at Cornell University were used in several pathogenicity trials. Dogs were inoculated intranasally, subcutaneously and intravenously with infected mouse lung suspensions and cultures of the organisms. At most only transient bouts of sneezing and occasional coughing de-

veloped, and in general the results did not indicate that the organisms possessed any great degree of infectivity for dogs. Freshly isolated cultures of *Brucella bronchiseptica* were instilled intranasally alone and combined with PPLO material, but none of the dogs developed respiratory symptoms. Indeed, the significance of PPL organisms in the dog is not at all clear at present.

It must be mentioned that many clinicians do not credit kennel cough as an entity distinct from distemper. The overlapping symptomatology of the two conditions gives justification to this feeling, especially since all the ramifications of distemper have not as yet been clearly defined. It is likewise true that before kennel cough should be accepted as a distinct disease, more than clinical evidence must be developed. Of course, the isolation and characterization of an agent of proven etiological significance must be the eventual result of research on the disease. Strong evidence, however, could be gathered from study along several lines of research to help clarify the true nature of kennel cough cases as they are recognized by the practitioner, even without the isolation of a specific organism. Transmission trials, for example, either by contact exposure or inoculation of infective material should be undertaken in dogs of known susceptibility and immunity to distemper and other canine infectious diseases. Serological trials in clinical or experimentally infected cases should be studied, specifically in relation to distemper virus neutralizing antibodies.

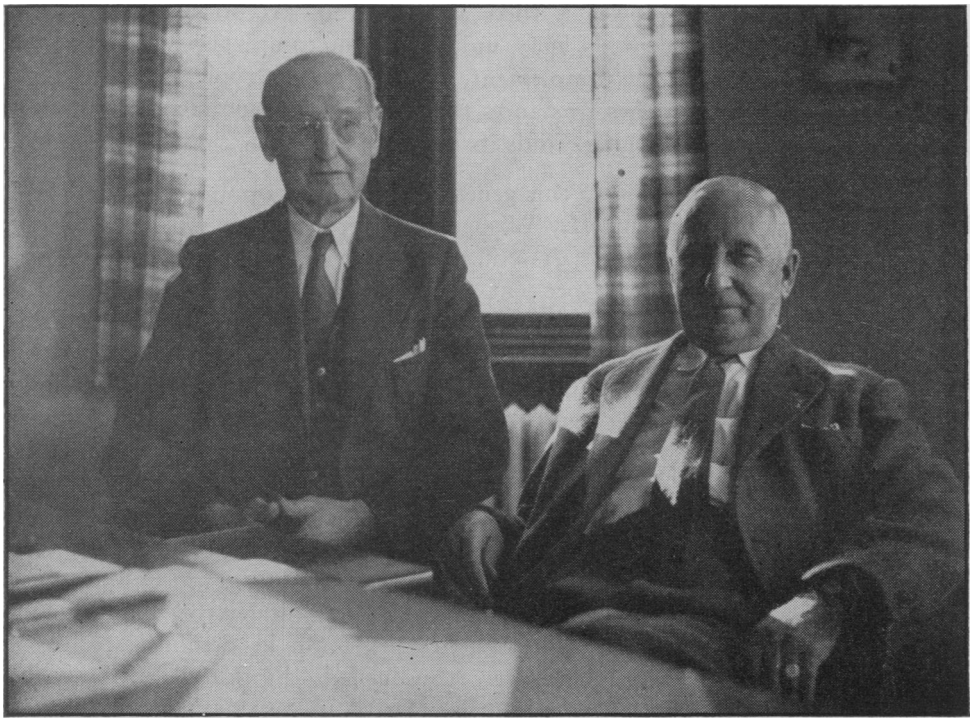
To those clinicians who have encountered and recognize kennel cough, catarrhal fever, virus tracheitis or whatever it may be called, there remains a problem of considerable economic importance. The role of PPL organisms in canine disease is obscure, but similarly the role of these organisms in other hosts in which they have been encountered, including man, is still not clear. Perhaps as knowledge of PPLO is increased and new methods of approach and technique are developed, these organisms will find their true place in the processes of disease.

REFERENCES

1. WHITNEY, L. F. "Housedog" disease. Vet. Med. 38: 419, 1943.
2. GUSTAFSON, W. B. Preliminary studies on the agent of canine pharyngo-laryngo-tracheitis. Yale J. Biol. Med. 20: 12, 1947.
3. TOWNSON, R. S. Infectious catarrhal fever of dogs. Vet. Record. 59: 203, 1947.
4. LEASK, D. E. A specific, highly contagious disease in dogs. Vet. Record. 60: 16, 1948.
5. RAY, J. D. Discussion. Proc. Kansas Vet. Med. Assn. 192. 1948-1949.
6. TORREY, J. C. and RAHE, A. H. Studies in canine distemper. Jour. Med. Res. 27: 291, 1913
7. GREIG, A. S. Unpublished data.
8. MARKHAM, F. S. and WONG, S. C. Pleuropneumonia-like organisms in the etiology of turkey sinusitis and chronic respiratory disease of chickens. Poultry Sci. 31: 902, 1952.
9. SWITZER, W. P. Studies on infectious atrophic rhinitis of swine. I. Isolation of a filterable agent from the nasal cavity of swine with infectious atrophic rhinitis. Jour. Am. Vet. Med. Ass. 123: 45, 1953.
10. SABIN, A. B. The filterable microorganisms of the pleuropneumonia group. Bact. Rev 5: 1, 1941.

11. SHOETENSACK, M. Pure cultivation of the filterable virus isolated from canine distemper. Kitasato Arch. Exp. Med. 11: 277, 1934.
12. EDWARD, D. G. and FITZGERALD, W. A. The isolation of organisms of the pleuropneumonia group from dogs. J. Gen. Microbiol. 5: 566, 1951.

GUEST AT C.C.V.A. MEETING



Left to Right: Dr. C. H. Higgins and Dr. Chas. A. Mitchell, the present Chief of the Division.

Dr. C. H. Higgins of Pearl River, N.Y. was a guest at a recent meeting of the Central Canada Veterinary Association. For many years he served in Canada, being appointed in 1899 as Assistant Pathologist to the Department of Agriculture and served under Dr. George Adami of McGill University until transferred to Ottawa in 1902. In this year he became the first Chief of the newly formed Pathological Division and continued in this capacity until 1917.