Infectious Rhinitis in Swine (Bull Nose)

By C. E. Phillips *

In RECENT years, from Western Canada and areas of the United States, a form of rhinitis has been reported which must be differentiated from the ordinary type of Bull Nose caused by *Actinomyces necrophorus.* Doyle et al in the September 1944 issue of the *A.V.M.A. Journal* reported a dystrophic rhinitis in five herds of swine in Indiana—cause unknown. Connell, in the August 1945 issue of this magazine, refers to the incidence and increasing importance of a similar disease in Western Canada. Jones in Alberta, and Kernkamp in Minnesota, have mentioned, by personal communication, the incidence of a transmissible rhinitis in their respective districts.

All communications and literature would indicate that little is known about the etiology and spread of this infection. Within the past few months our field observations, post-mortem clinic, and correspondence indicate the rapid spread of a similar disease in Ontario. We have no definite evidence as to when this disease started in Ontario, but breeders insist that it is a comparatively new condition of the past three years, and that it can be traced to the introduction of breeding stock.

While we may not be able to trace its introduction, we certainly can trace its spread and the story is always the same. I bought a sow last fall, or I bought a litter of pigs and within six months (or a year, depending upon the number of pigs farrowed) all my pigs had it. This disease has spread so rapidly that in some districts it is the exception to find a clean herd, and it continues to take an increasing toll of profits in all types of piggeries, including those of our foremost Yorkshire breeders.

Spread of Infection

A survey of at least fifty premises on which this disease is enzootic would indicate that the disease is spread through the introduction of infected animals. These carriers include any animal raised in an infected piggery, whether or not they show visible signs of disease. The introduction of a brood sow, boar or shoat will spread the infection. Whether an adult contact animal will serve as a spreader has not yet been determined, but the contagious nature of this disease would indicate that it is quite possible.

The course of the disease, upon the introduction of an infected animal, varies with several variable factors. It usually takes from six months to one year before all suckling pigs show visible indication of infection. Factors such as the number of litters farrowed, type of housing and contact, intimacy of the breeding stock, and infection in the sow are all related to the percentage of infected animals.

It would appear that the infection becomes established shortly after

* Ontario Veterinary College.

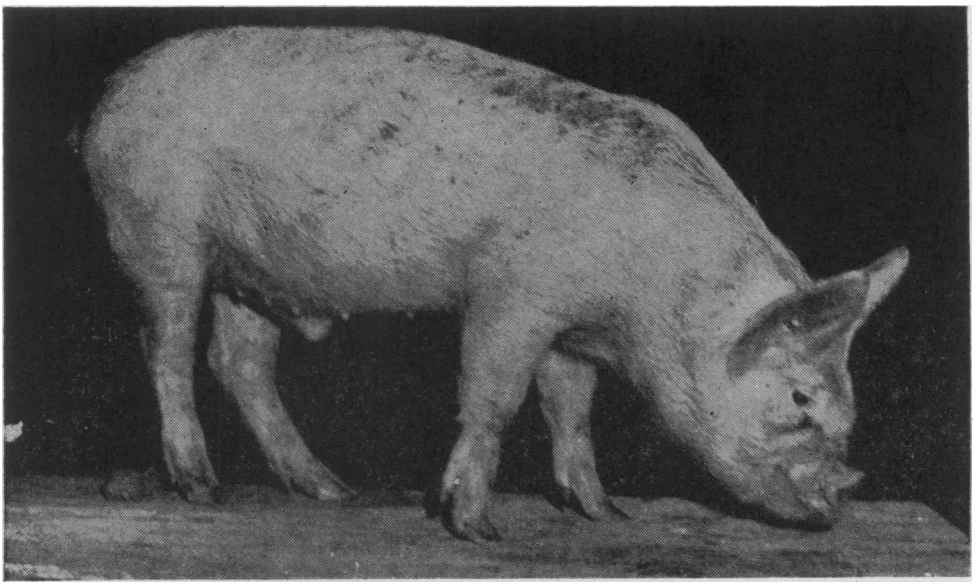

Figure 1.—Rhinitis pig, age 3½ months. Note wrinkled snout and dark area below the eye.

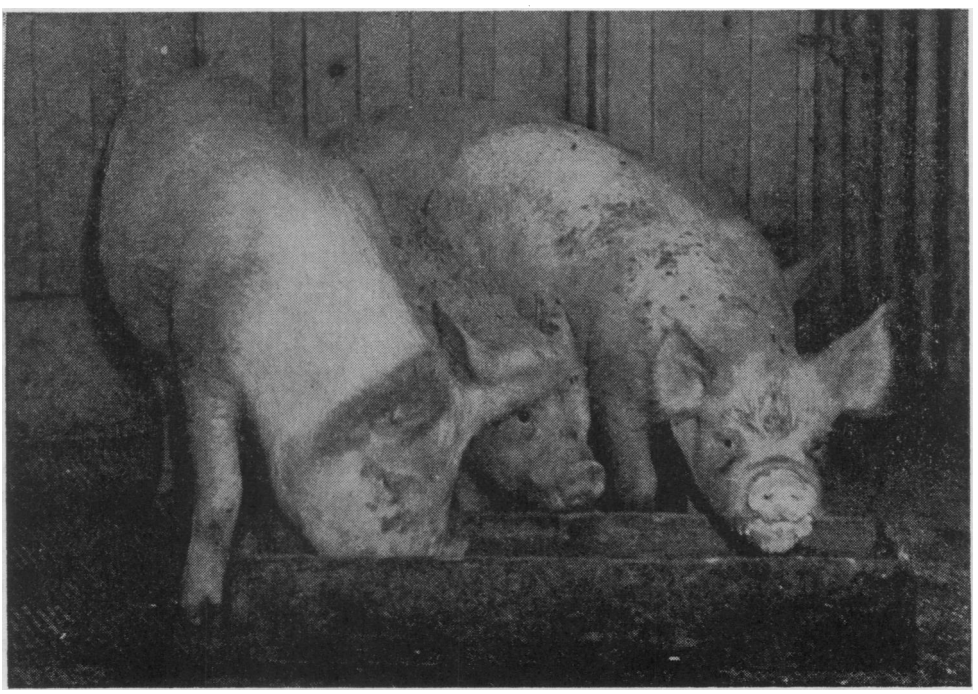

Figure 1A.—Group of infectious rhinitis pigs.

Course of the Disease

birth, although visible signs are not observed until the animal is from 2-4 weeks old.

The first indication is that of sneezing, a sneeze more persistent than that required to dislodge dust; and this endeavour to dislodge foreign material from the nostril becomes more exaggerated as the pig grows older. At from three to eight weeks of age the skin on the snout begins to wrinkle, a symptom best observed in short and medium nosed pigs. The snout usually appears to bulge or thicken—best observed in long nosed pigs. The dis-

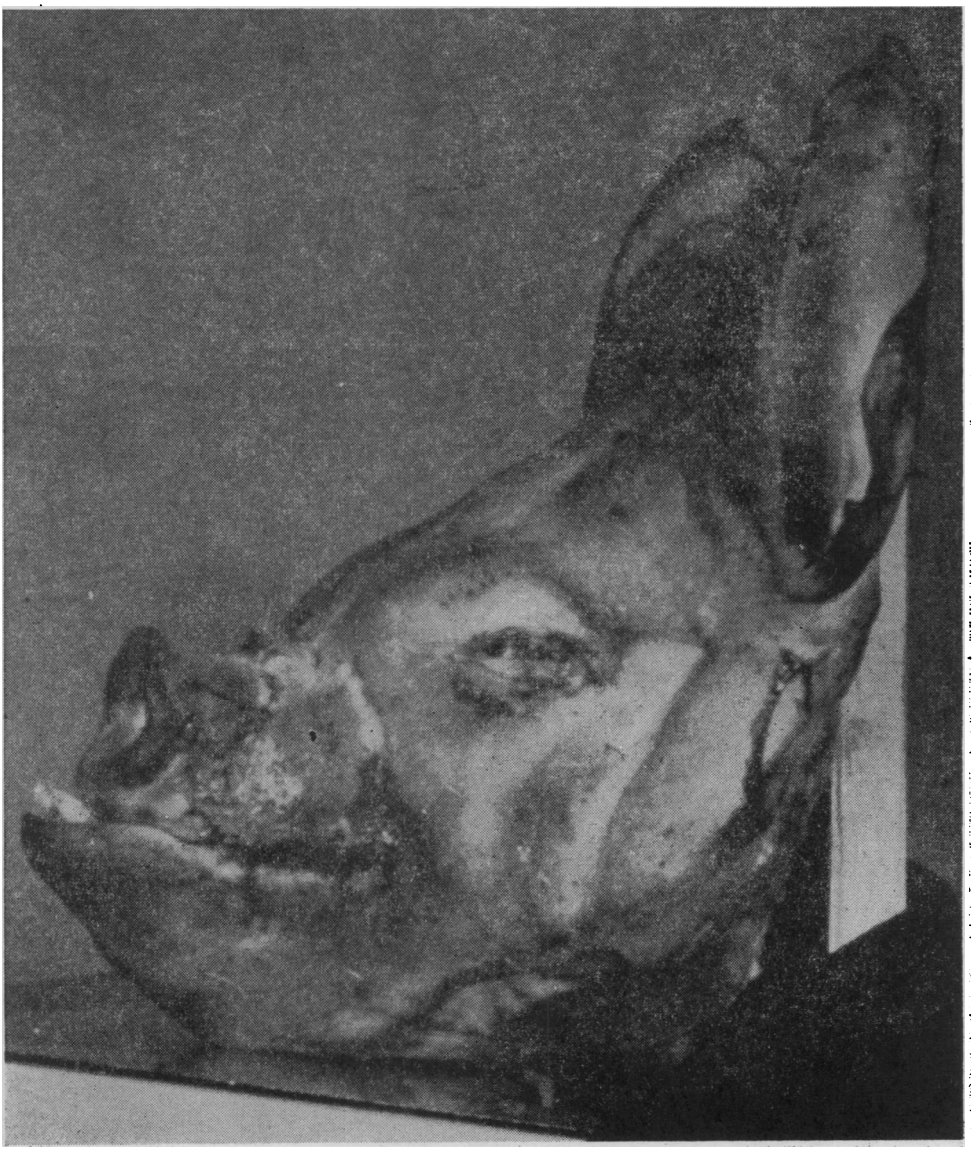

Figure II—Head of medium short nosed type showing damage.

tortion of the snout, when present, will be observed anywhere from six weeks to four months, depending upon the type of head and, to some extent, upon the husbandry (Figures I and IA). The same statement can be made about the general appearance of the pig; however, the majority become rough prior to weaning, and are progressively poor doers until marketed. The larger number will market at from 7 to 9 months of age; however, an inventory in a large piggery will reveal many boarders 12 months of age, and still not ready to go.

Figure III—Nose showing little distortion.

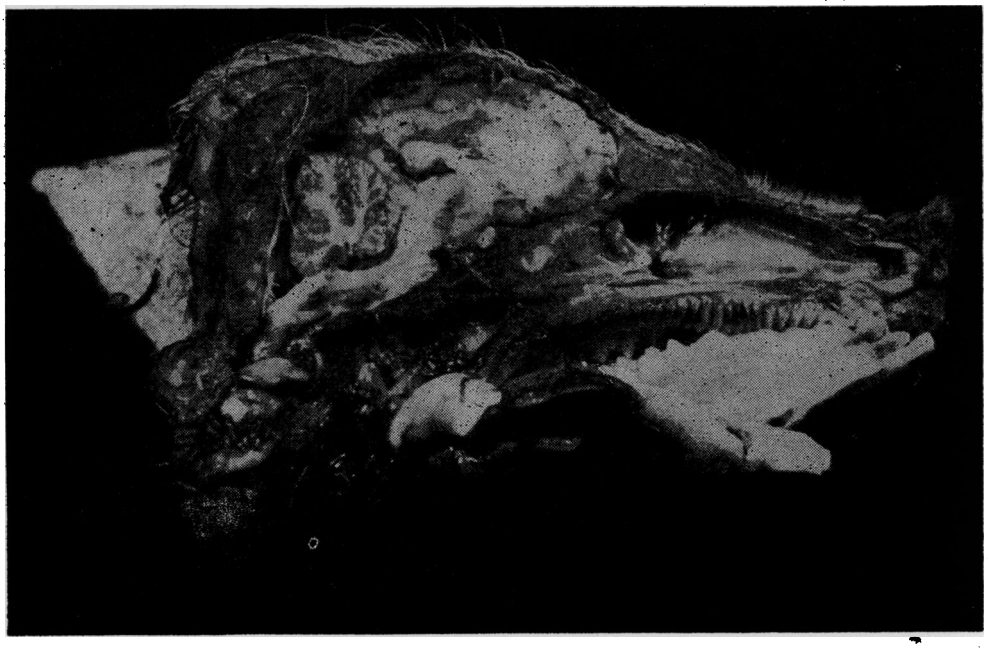

Figure IIIa—Cross section of Figure III showing destruction of turbinates and ethmoid bone

This disease favours long-nosed hogs because of head conformation, but they are just as susceptible to the incidence of infection as are the short-nosed type.

The observable snout distortion will vary according to the head type. The short and medium nosed types present a dished appearance, with the snout receding up to an inch in older animals (Figure II). The snout in the medium to long nosed types may turn to either side, slightly or acutely.

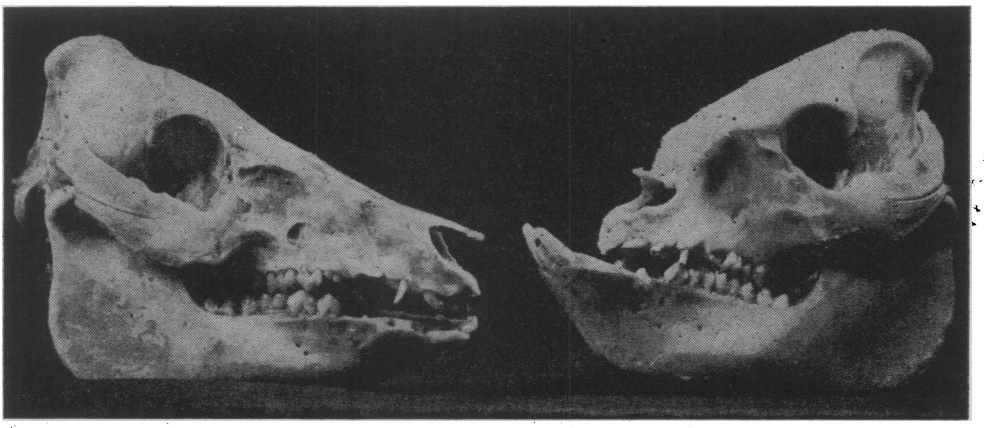

Figure IV—Skull of normal and medium short nosed pig showing distortion of nasal bones

It must be kept in mind that not all cases show observable distortion of the snouts and such animals quite readily pass as suitable breeding stock (Figures III, IIIA). Most animals show a black area below the eyes, caused by occlusion of the tear duct and collection of dust. While this is not confined to rhinitis pigs, it is quite characteristic when observed in a large group of pigs.

Gross Pathology

Skull.—Examination of the skull in the majority of pigs shows a marked distortion of the nasal bones (Figure IV).

Cross section of head.—The macroscopic changes which take place in the nasal cavity are characteristic and present a constant progressive dissolution of the softer bony structures of the nose. An early chronic inflammation of the nasal mucosa is followed by decalcification of the turbinates and ethmoid bones. This is apparent from both macroscopic and microscopic studies. In many older cases, remnants of the turbinate and ethmoid mucosa are observed, completely devoid of bony structure (Figures V, VI). This decalcification and disintegration of the turbinates and ethmoid structures is a gradual process, the turbinates disappearing at from 2 to 4 months of age, while the disintegration of the ethmoid is more variable,—all showing some damage and many completely disappearing before marketable weight is reached. The infection may favour one nostril; this more severe unilateral damage being responsible for some of the characteristic distortions of the snout.

It is logical to presume that while this observable decalcification and

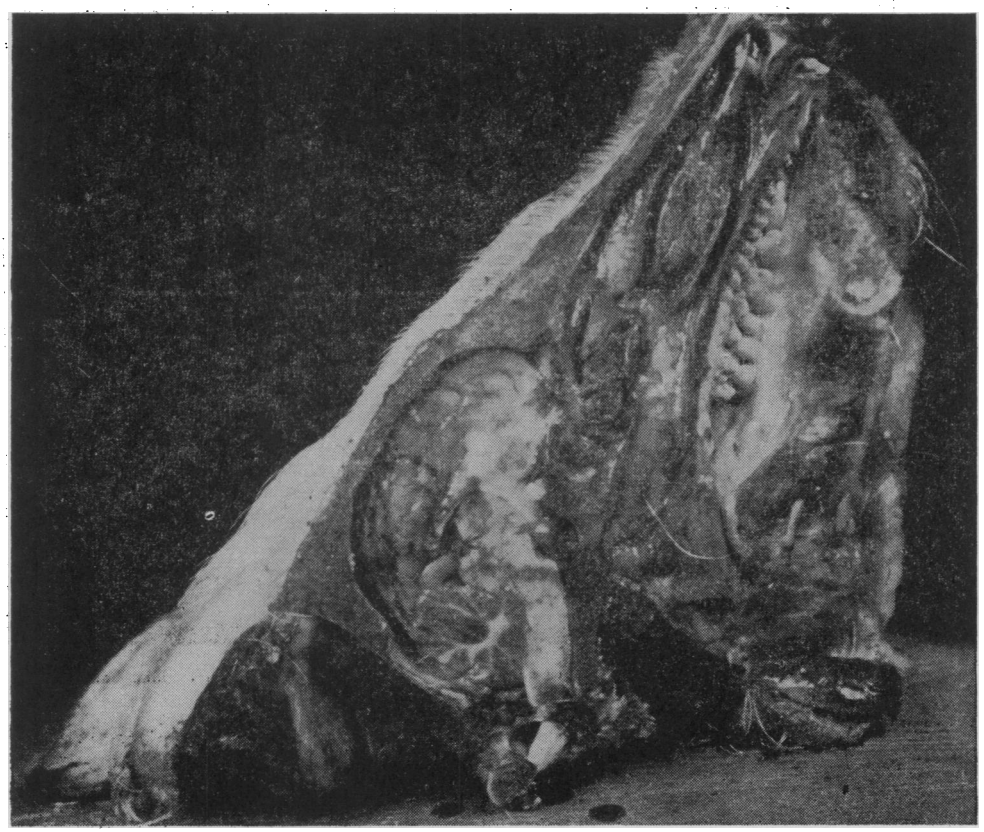

Figure V—Normal cross section with turbinates and etmoid intact.

absorption of the softer bony structures of the nose is taking place, a similar modified change is taking place in the harder facial bones which would account for the distortion of this structure. Whether this is a process of rarefication, or interference with the proper function of the periosteum and osteoblasts in the laying down of calcium, is a point requiring further study.

Exudate.—The type and consistency of the exudate varies from a yellowish, muco-purulent discharge present in a comparatively small amount to a yellow inspissated pus largely filling the posterior part of the nasal cavity, and embedded in the damaged structure of the ethmoid. This may be blood tinged. The former is the more common exudate observed, the latter being mainly present in older chronic cases. It should be kept in mind that we do not consider this disease phenomenon that of necrosis.

Encephal tis.—The tissue damage, especially that of the ethmoid, and the proximity of the cribriform plate with its nerve channels to the brain present a convenient channel for bacterial invasion with resultant encephalitis. This encephalitis takes a minor toll of rhinitis pigs in the early stages and it is rather remarkable that this percentage is not higher. I have isolated pure and mixed cultures of pathogenic bacteria from the brain

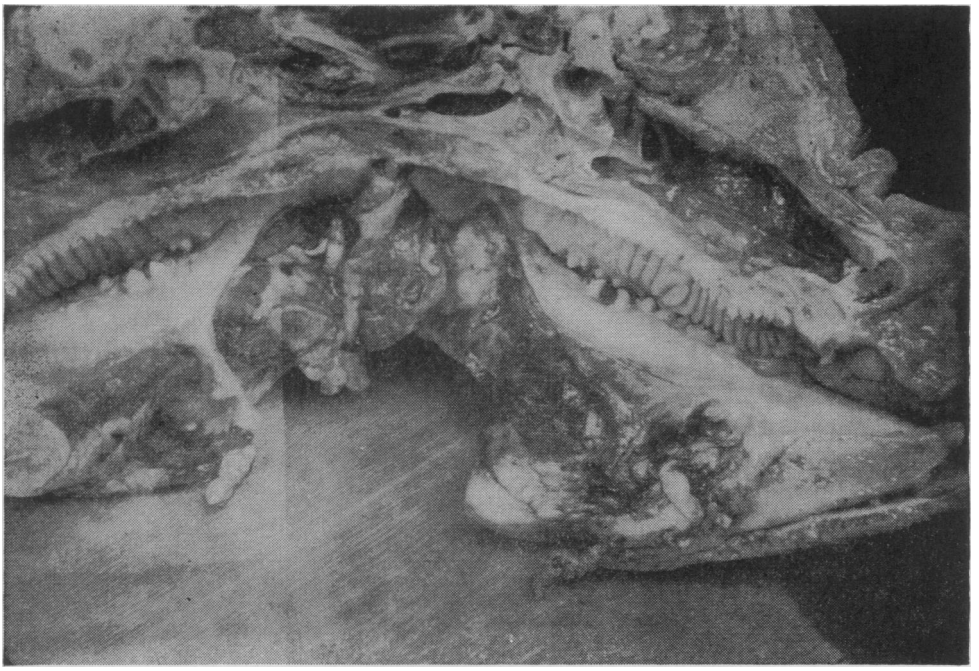

Figure VI—Cross section of medium short nosed head (FigureII) showing distortion. Turbinates have completely disappeared and only remnants of the ethmoid remain.

tissue of recently dead and killed prostrate rhinitis pigs. This lesion should always be kept in mind when pigs die suddenly in an infected herd without apparent cause.

As previously stated the majority of rhinitis pigs are rough and chronically poor doers, requiring up to 12 months to market. This fact makes the raising of pigs economically unsound, and is serving to force a large number of our hog raisers out of business, and others to consider seriously disposing of their herds. From field observations and a large number of post mortems, it is my opinion that secondary bacterial invaders are the main cause of stunting and poor growth. It has been my experience that secondary pneumonias and in long-standing cases arthritis and abscess formation with their bacterial toxic products are responsible. Also, the damaged nasal mucosa presents a fertile field for bacterial growth and toxic absorption.

Bacteriological Cultures

The most consistent organism isolated from the nose and secondary lesions is *Corynebacterium pyogenes.* I have isolated this organism from all herds under observation and experiment, and from 90% of rhinitis pigs submitted to the post mortem clinic, from various sections of the province. Unless masked by *Proteus* or *Pseudomonas aeruginosa,* it can usually be isolated from the nostril; it may be found with other organisms in a purulent pneumonia or abscessed lung; it may be present in caseated lymph nodes or body abscesses in chronic cases. This organism has been isolated

from pigs on at least forty rhinitis infected premises in the last six months. It does not appear to be a factor in the usual porcine pneumonia or arthritis as found on non-infected premises. It would appear that this organism is the most important secondary factor, although we must include *Pasteurella suis, Alcaligenes bronchisepticus* and *Bacillus pyogenes* when considering factors responsible for curtailed growths and secondary lesions, especially pneumonia.

Experiments

Observations on the contagious nature and spread of this disease and initial experiments would indicate that a filtrable factor is the primary etiological agent. It is my opinion that secondary organisms play a large part in the destructive nature of the disease as they do in other virus diseases, but that a primary factor; i.e., a filtrable factor is necessary. I have succeeded in reproducing atypical cases by injecting a bacteria-free filtrate into the nasal cavity of 5-day old pigs. When post mortemed 30 days later, macroscopic and microscopic examinations showed lesions comparable with field cases of that age. The remainder of two litters did not progress beyond this stage. Pigs in a similar experiment with *Corynebacterium pyogenes* remained normal except for a secondary abscess in one pig.

Control

1. If possible, dispose of the herd, and re-establish with clean breeding stock.

2. Advise your clients not to buy breeding stock from an infected herd.

3. Avoid breeding infected sows, as a matter of fact, badly infected sows and boars often fail to breed.

4. Have the sows farrow on clean premises away from the infected piggery, and with as little contact as possible. Do not move these pigs into the piggery until they are at least 2 months old. Individual colony houses, when practical, are the best shelter for this purpose. Pigs at weaning age, and older, are much less susceptible to the observable nasal damage, although they are susceptible to the secondary invaders. When older, pigs or breeding stock are introduced into an infected herd, it is not uncommon to observe a transient arthritis; and/or other lesions produced by the secondary invaders of infectious rhinitis.

Summary

A preliminary report on an extremely infectious atrophic rhinitis in pigs which is spreading at an alarming rate in Ontario. This disease is carried by all pigs raised in an infected piggery and the incidence of infection is unusually high. It is hoped that this article will acquaint veterinarians in rhinitis free areas with the disease in an endeavour to prevent its spread.

Acknowledgements

Thanks are expressed to Dr. A. L. MacNabb for his cooperation in sup-

plying experimental animals and to Dr. F. W. Schofield, department of pathology, for his cooperation in preparing pathological slides and microscopical interpretations.

References

L. P. DOYLE ET AL, Journal A.V.M.A. Sept. 1944.
R. CONNELL, Canadian Journal of Comparative Medicine, August 1945.

Résumé

L'AUTEUR signale l'apparition en Ontario d'une affection du porc rapportée jusqu'ici comme n'existant que dans l'ouest du Canada et dans certaines régions des Etats-Unis. La maladie se manifeste par de la rhinite et, sous cette forme, est à différencier de la rhinite à *Actinomyces necrophorus*. La maladie est fortement contagieuse. Les élevages se contaminent par l'introduction de sujets infectés mais ne présentant pas toujours des symptômes apparents. La rapidité de la propagation de la maladie semble dépendre de plusieurs facteurs: le nombre de portées par année, le genre d'habitation des animaux, la promiscuité dans laquelle vit le stock d'élevage, infection chez la truie portière.

Dans les élevages infectés, les premiers symptômes apparaissent chez les porcelets de 2 à 4 semaines après la naissance: éternuement dont la persistance et l'intensité augmentent avec l'âge. Vers l'âge de 3 à 8 semaines, la peau du groin devient ridée (ce fait est mieux observé chez les porcs à groin court ou moyen); le groin paraît épaissi. Ce dernier fait est plus fréquemment observé chez les porcs à groin long. Dans certains cas, on remarque aussi une déformation du groin. Cette déformation, varie avec la conformation de la tête de l'animal. La croissance est ralentie et du point de vue économique l'élevage s'avère une affaire non-payante. A l'autopsie, l'étude macroscopique et microscopique révèle une décalcification progressive et un ramollissement des os du nez. Les lésions peuvent être unilatérales ou bilatérales et, souvent, le processus pathologique s'étend aux os de la face. On peut aussi observer un exsudat de coloration, de densité et de quantité variables.

Les dommages causés aux os du groin et de la face ouvrent la voie aux infections secondaires; ainsi, chez un ctrtain nombre de porcs infectés, on peut observer de l'encéphalite. Ces infections secondaires ralentissent la croissance, expliquent l'apparence rabougrie des animaux infectés, les troubles de l'appareil respiratoire et des articulations. L'agent microbien le plus souvent isolé est *Corynebacterium pyogenes* (90% des cas); viennent ensuite *Alcaligenes bronchisepticus* et *Bacillus pyogenes*. D'après l'auteur, la cause première de l'infection serait un virus filtrable, dont les germes d'association seraient les microbes mentionnés plus haut.

En milieu infecté, le contrôle consiste à se débarrasser de tous les sujets de l'élevage. En milieu sain, à ne pas introduire dans les troupeaux des animaux provenant d'un milieu infecté et à ne pas faire d'élevage avec des reproducteurs infectés ou même exposés à l'infection. La mise bas devrait se faire dans des locaux isolés et éloignés des porcheries infectées et l'éleveur devrait s'efforcer de réduire au minimum les contacts entre les deux endroits. Les porcelets ne seront transportés à la pocherie où il y a déjà eu des animaux infectés qu'après l'âge de deux mois. Après cet age, en effet, les porcs sont beaucoup moins susceptibles et, s'ils s'infectent, les dommages sont moins considérables.