swallowing by the animal of considerable amounts of virus and hence juxtaposition of virus also with the pharyngeal and gastrointestinal mucosa) is unsuccessful if the connections of the olfactory nerves with the central nervous system are severed, thus completing the demonstration of the olfactory nerves as the essential and probably exclusive pathway of invasion from the body surfaces when such surfaces are not mechanically injured or broken; that is, when conditions of infection are comparable with those occurring in man.

The theory of gastrointestinal invasion has few remaining advocates since the very convincing study of Clark, Roberts and Preston.[5] However, Dr. J. A. Toomey[6] has recently reaffirmed it in somewhat modified form. As an argument against my own views he states: "In the majority of human beings and monkeys, paralysis first develops in the muscles that receive their nerve supply from the lumbar enlargement and only secondarily in those whose nerve supply comes from the cervical area. This fact forms the basis for a fundamental objection to Faber's theory of virus spread."

Through the kindness of Dr. E. W. Schultz and L. P. Gebhardt, of the Department of Bacteriology at Stanford University, I am able to present data on the region of initial paralysis in 57 monkeys, selected at random, all inoculated intranasally by the method they have devised.[7] In these, the infection beyond any reasonable doubt entered through the olfactory nerves and passed through the brain-stem and spinal cord *from above downwards.*

Arms first paralyzed	25, or 43.9	per cent.
Legs first paralyzed	27, or 47.3	per cent.
Arms and legs paralyzed at about the same time	5, or 8.8	per cent.
Total	57	

These experiments in which entrance of infection from the gastrointestinal tract can be ruled out with some certainty and in which, nevertheless, the legs were more often first involved than the arms demonstrate clearly that initial involvement of the lumbar cord can not properly be used as evidence for the theory of the gastrointestinal portal of entry. On the contrary, they prove that the virus in descending through the cord can, and does in more than half the cases, produce its first manifestations in the lower segments. Knowing from previous experiments that the virus is actually present in all levels of the cord,[8] including the cervical, just before as well as when paralysis appears, I see no other possible explanation of the usually earlier and greater lumbar area paralysis than that offered by Fairbrother and Hurst:[9] the anterior horn cells in that area are somewhat more susceptible than others to attack by the virus of poliomyelitis.

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[5] P. F. Clark, D. J. Roberts and W. S. Preston, Jr., *Jour. Prev. Med.*, 6: 47, 1932.
[6] J. A. Toomey, *Ann. Int. Med.*, 8: 854, 1935.
[7] E. W. Schultz and L. P. Gebhardt, *Proc. Soc. Exp. Biol. and Med.*, 30: 1010, 1933.
[8] Faber and Gebhardt, *loc. cit.*

COCCO-BACILLIFORM BODIES ASSOCIATED WITH AN INFECTIOUS FOWL CORYZA

A STUDY of fowl coryza, based on material secured from infected flocks in the vicinity of Princeton, N. J., has shown two distinct clinical types of the disease. The interval elapsing between the injection of exudate in susceptible fowl is short in one case, 1 to 3 days, and prolonged in the other, 1 to 4 weeks. In both types the nasal discharge generally persists for 2 months and often for a longer period.

The fowl coryza bacillus, *Hemophilus gallinarum,* originally described by De Blieck,[1] is constantly associated with the coryza of rapid onset. Intranasal injection of this organism in normal birds is followed by a coryza which, unlike that produced by exudate, is generally of short duration. Moreover, while recovered birds may be resistant to reinfection with the bacillus they are not protected against a subsequent injection of exudate. These two facts have militated against the acceptance of *Hemophilus gallinarum* as the sole cause of the coryza of rapid onset and long duration.

Hemophilus gallinarum is not associated with the coryza of slow onset; attempts to isolate it from the nasal passages of infected birds have repeatedly failed. Generally the exudate does contain other bacteria, most of which grow freely in cultures. It can be said with certainty that all these bacteria are secondary invaders and of no direct etiological significance. It can also be stated that the responsible infective agent is unable to pass through Berkefeld V candles of average permeability.

It was found, however, that sterile filtrates from a V candle of abnormal permeability contained the infective agent and produced a coryza of slow onset in normal fowl. Bacteria free exudate, which is sometimes present in the orbital sinuses of infected birds, also proved to be infective.

Exudate from two birds originally infected with filtrate has been carried on in series by passage from infected to susceptible birds. Films made directly from this exudate, which regularly contains few bac-

[9] R. W. Fairbrother and E. W. Hurst, *Jour. Path. and Bact.*, 33: 17, 1930.
[1] L. De Blieck, *Vet. Jour.*, 88: 9, 1932.

teria, have constantly shown minute Gram negative cocco-bacilliform bodies. They are commonly extracellular; occurring singly, in pairs, or in loosely formed aggregates, but may also be found intracellularly within both phagocytic and epithelial cells. They vary somewhat in size, with an estimated range of 0.1 to 0.5 μ, many of them approaching the limit of visibility with the ordinary microscope. Early in the disease the bodies are generally numerous, but in some cases their detection requires a prolonged search. The same bodies are also present in films made from the nasal passages of birds injected with unfiltered exudate. Their detection is often more difficult in this case, due to the presence of numerous bacteria.

All attempts to cultivate these bodies, either aerobically or anaerobically, in artificial media have met with failure. In several instances, however, there has been isolated a minute Gram-bacillus which tends to form compact clumps in fluid cultures. This organism, which is not pathogenic, bears a superficial morphological resemblance to the cocco-bacilliform bodies, and like them is capable of passing through certain Berkefeld V candles. Whether or not it is in any way related to these bodies can not be stated at present.

The injection of exudate from birds infected with the coryza of rapid onset into birds which have recovered from the coryza produced by injection of *Hemophilus gallinarum* is often followed by a coryza of slow onset which may be reproduced in series. *Hemophilus gallinarum* is not present in the exudate in these cases. Stained films, however, show cocco-bacilliform bodies indistinguishable from those which characterize the more naturally produced disease. These findings suggest that the coryza of rapid onset and long duration is in reality a mixed infection in which both *Hemophilus gallinarum* and the present virus, of undetermined nature, are operating. Such a relationship would offer an adequate explanation for the previously noted discrepancies in the coryzas produced by exudate and culture, respectively.

Gibbs[2] has recently stated that the causative agent of a fowl coryza with which *Hemophilus gallinarum* was not associated was capable of passing through certain celloidin membranes and estimated that the diameter of the agent was between 80–120 mμ. Filterability is not, however, a trustworthy characteristic for the classification of an infective agent. The present bodies may also be considered as filterable. The smaller forms certainly do not exceed 100 mμ in diameter. They are morphologically similar to the bodies which characterize fowl pox, vaccinia and psittacosis, but also resemble the Rickettsiae of typhus and allied diseases. The present knowledge of the coryza bodies is, however, too meager to warrant a statement as to their nature. It is realized that the implied relationship of them to the etiology of coryza rests on purely circumstantial evidence and that a final appraisal must await their recovery or cultivation in a pure state.

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THE ROLE OF LIPOIDS IN THE X-RAY DIFFRACTION PATTERNS OF NERVE

IN order to obtain greater detail with respect to the 40–45 Å equatorial spacings previously reported for medullated nerve,[1] photographs were made with very small pinholes (0.2 mm), with well-centered small beads and with distances of 10–20 cm from specimen to plate (copper $K\alpha$ radiation). These photographs revealed large well-oriented spacings previously unsuspected for animal tissues. Because of the light which this work throws on the interpretation of nerve diffraction patterns and because of the biological significance of the lipoids, to which these large spacings apparently are due, we wish to describe briefly these results.

The rôles of the myelin and the axis cylinder in the diffraction patterns of nerve were investigated by studying photographs obtained from the lipoid and protein constituents of nerve separately. When rubbed up with water the solids obtained by evaporation of a benzene-alcohol extract of dried cow spinal cord gave patterns similar to those of fresh medullated nerve as exemplified by corpus callosum or motor roots. The dried lipoid patterns are similar to those of dried medullated nerve, with the exception of the equatorial points at 11.5 Å observed in the latter. This spacing was duplicated by fibers spun from nucleoprotein extracts of cow spinal cord and lobster claw nerves and presumably corresponds to the side-chain distance of these protein macromolecules.[2]

The lipoid constituents of the myelin sheath of fresh medullated nerve appear to be organized in a manner such as to form a single type of fluid crystal which gives a spacing in its long direction of approximately 171 Å. This is strongly indicated by the fact that the spacings observed are 85.5, 56.8, 42.7 and 34.2 Å with the even orders strongest and the odd orders weakest; each of the rings has strong equatorial accentuation. Upon drying nerve yields principally

[1] F. O. Schmitt, G. L. Clark and J. N. Mrgudich, SCIENCE, 80: 567, 1934.

[2] The protein extraction and fiber spinning was performed as described by F. O. Schmitt and R. S. Bear, *Proc. Soc. Exper. Biol. and Med.*, 32: 943, 1935.

[2] C. S. Gibbs, SCIENCE, 81: 345, 1935.