STUDIES ON A-AVITAMINOSIS IN CHICKENS

II. Lesions of the Upper Alimentary Tract and Their Relation to Some Infectious Diseases

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Plates 24 and 25

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In the previous paper[1] of this series the lesions in the respiratory tract caused by a lack of vitamin A in the food of chickens was considered, together with a discussion of the clinical findings and their relation to certain infectious diseases. Using the same material and methods, it is the purpose now to deal in the same way with the lesions of the mouth, palate, esophagus, crop, and their associated glands.

Gross Pathology

Because of the close connection between the nasal cavity and the mouth, the seromucous discharge in the former overflows regularly through the cleft palate and produces a clinical and anatomical picture which cannot be differentiated from that found in coryza contagiosa. As secondary invaders appear this exudate becomes transformed into white or slightly yellowish caseous masses which may at times completely plug the cleft palate. In addition, in the region of these nasal plugs and also on the roof of the mouth but seldom in the esophagus, fine thin membranes appear which together with the nasal plug produce a picture which may be easily confused with that of fowl-pox. In A-avitaminosis, however, these membranes may not always be present and when they are they usually are limited to the cleft palate and its adjacent epithelium. They are easily removed leaving no bleeding ulceration, thus differing from fowl-pox.

Early in A-avitaminosis there appear small white or yellowish pustule-like lesions, varying from 0.5 to 2 mm. in diameter, in the region of the excretory ducts of the glands of the mouth (Text-figs. 1 and 2). When discrete they are round, but by confluence they may become longish. They are usually raised above the

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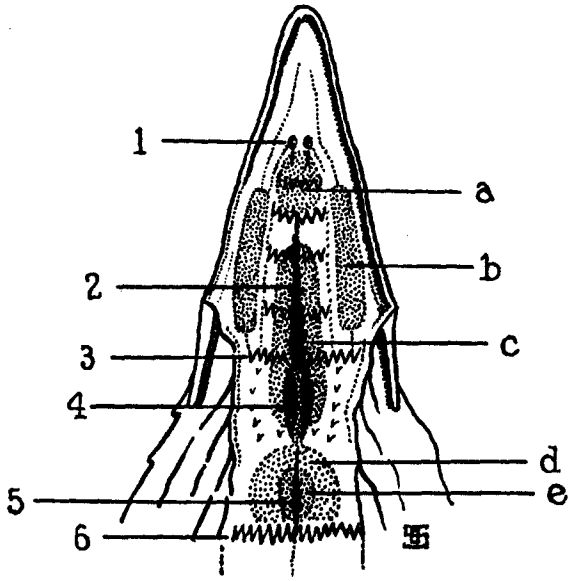
[1] Seifried, O., *J. Exp. Med.*, 1930, **52**, 519.

surface of the mucous membrane and often show a depression in the center (Fig. 1). Later small ulcers may appear at the site of these lesions and these ulcers may be surrounded by inflammatory products. These pustule-like lesions, with or without the inflammatory membranes, very closely resemble lesions of fowl-pox, and it is with great difficulty, if at all, that a differential diagnosis can be made by the macroscopical picture.

The mucosa of the esophagus and crop also shows pustule-like patches which may be even more prominent than those seen in the mouth, probably due to the



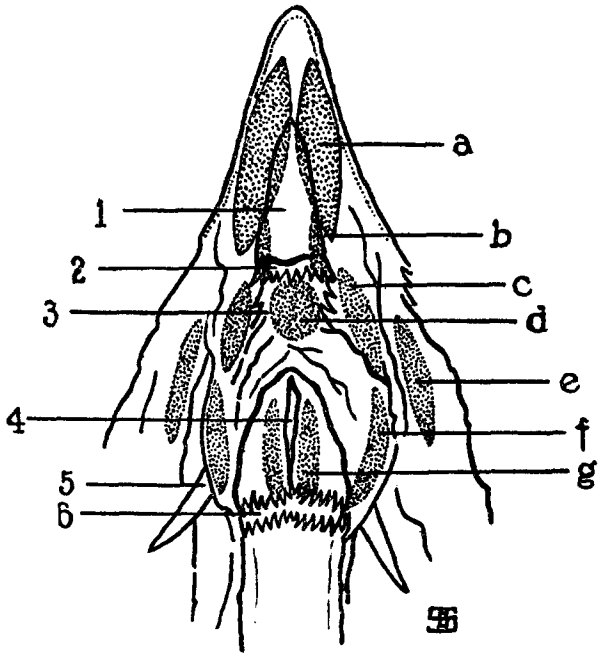
TEXT-FIG. 1. Roof of mouth. 1: excretory ducts of maxillary gland; 2: cleft palate, narrow part; 3: papillae of palate; 4: cleft palate, wide part; 5: infundibulum; 6: papillae of throat. a: glandula maxillaris; b: glandula palatina lateralis; c: glandula palatina medialis; d: glandula spenopterygoida; e: glandula tubaria.

peculiar structure of the glands in these regions (Fig. 1). They may be either localized in the upper part of the esophagus and surrounding the pharynx, or they may cover the mucous membrane of the esophagus throughout its entire length and in cases extend into the posterior part of the crop where the folds of the esophagus are continued. In the main portion of the proventriculus and throughout the intestines no gross lesions have been found. The nodules in the esophagus do not ulcerate as frequently as those in the mouth cavity, but they show a depressed minute opening in the center which corresponds to the excretory duct of the gland. In a few instances they are so numerous in the esophagus that when infected a type of membrane may be formed.

Histology

As noted above the lesions in the alimentary tract occur mainly in the glands. They have been studied in sections made of the roof of the mouth after decalcification.

Glands from the floor of the mouth, and in some instances from the roof, have been dissected out and sectioned in their shortest and longest diameters, while the



TEXT-FIG. 2. Floor of mouth. 1: tongue; 2: papillae of tongue; 3: base of tongue; 4: entrance to larynx; 5: bone of tongue; 6: papillae of larynx. a: glandula submaxillaris anterior; b: glandula lingualis anterior; c: glandula submaxillaris oromedialis; d: glandula lingualis posterior; e: glandula submaxillaris caudolateralis; f: glandula submaxillaris intermedia; g: glandula cricoarytenoidea.

glands of the tongue, pharynx and esophagus have been sectioned *in situ*. The lesions in the glands are essentially the same as those found in the respiratory tract and described in the previous paper. There are, however, some peculiarities which probably depend upon the special structure of the glands. As a rule the early lesions in these glands are not found in the acini but in the collecting spaces and ducts. Lesions may be well marked in the maxillary and submaxillary glands before they appear in the nasal passages, sinuses, trachea or bronchi. Fig. 2 shows

an early lesion of the submaxillary gland in which there is a marked proliferation and degeneration of the original epithelium. At the same time new stratified epithelium appears beneath or in place of the original epithelium. These early lesions may not be very characteristic but as the process is advanced typical pictures occur, as shown in Fig. 3 of the maxillary gland where in the collecting space is found a focus of squamous stratified keratinizing epithelium. In the acini the high mucus-secreting epithelium has begun to degenerate and atrophy and in a few instances there has been a replacement by stratified keratinizing cells. These changes in the acini have never been as pronounced as in the collecting spaces. Fig. 4, a cross section through the glandula lingualis, shows a well marked stage in the process with newly formed stratified epithelium and early keratinization. This newly formed epithelium may be found in isolated islands or in larger foci beneath or in place of the original epithelium lying directly upon the connective tissue. It seems probable that this newly formed tissue is favored in its growth by the abundant supply of capillaries from the surrounding connective tissue. The collecting spaces become filled with degenerated cells, masses of mucus (Fig. 4), and in some cases inflammatory products. This accumulation is undoubtedly in large part due to the lesions in the excretory ducts of the glands, as it is here that the oldest and most pronounced changes have been found. The epithelium of the mucous membrane of the mouth seems to extend into the duct, which is partially filled with the new stratified epithelium, and causes a more or less complete blocking. As a result the glands become dilated and frequently show evidences of bacterial infection, the latter being especially marked in the duct. Since this is an early lesion the gland may continue to secrete mucus for a time and this accumulates in the collecting space. Later desquamated cells from the newly formed stratified epithelium become numerous and the gland, which originally is a sac with invaginations, becomes smoothed out and distended. Finally this distended sac becomes completely filled with stratified keratinized epithelial cells, as shown in Fig. 5 which gives a picture of the final stage of the process. Such a picture is found more frequently in the glands of the tongue, palate and esophagus than in the compound glands of the cavity of the mouth.

The histological picture shows that early infections of the excretory ducts of the glands are relatively common. These infections are manifested by necrosis of the epithelial lining of the duct and of the surrounding mucous membrane, cellular infiltrations, and the presence of numerous bacteria (Figs. 6 and 7). Oftentimes it appears that the lumen of the duct is blocked, thus resulting in an accumulation of inflammatory products in the gland itself. At times the inflammatory process spreads from the mouth of the duct over the surrounding mucous membrane of the mouth (Fig. 8) or tongue, and more rarely over the mucous membrane of the esophagus.

In the submucosa signs of infection such as perivascular and diffuse infiltrations are frequently seen. In some sections through the tongue such infiltrations have been found even in the deeper layers of the muscularis, and in the ventral

portion of the vomer these lesions are particularly pronounced. In the esophagus and crop and sometimes in the proventriculus, the lesions in the glands are the same as those described in the mouth cavity but secondary bacterial infections are not as pronounced.

As pointed out above, the macroscopical lesions in the mouth may closely resemble lesions of fowl-pox. The histological difference between these two is quite striking and there is no difficulty in separating them from sections, but what part the process plays in the development of fowl-pox and other infections by breaking down the resistance of the mucous membranes is a problem that is well worth further consideration.

SUMMARY AND CONCLUSIONS

When fowls are placed on a diet lacking in vitamin A lesions appear in the upper alimentary tract which are confined largely to the mucous glands and their ducts. Histologically it is shown that the original epithelium becomes replaced by a stratified squamous keratinizing epithelium and that secondary infections are relatively common. The ducts of the glands may be blocked leading to distention with secretions and necrotic materials. These lesions macroscopically resemble very closely certain stages of fowl-pox and the two conditions can be separated only by histological examination. It is pointed out that these lesions produced by a lack of vitamin A may enable bacteria and other viruses to enter the body.

EXPLANATION OF PLATES

PLATE 24

FIG. 1. Pustule-like patches at the roof of the mouth, the base of the tongue and the mucous membrane of the esophagus. (Chicken 7, dead after 82 days on experimental diet.)

FIG. 2. Longitudinal section through the glandula submaxillaris of Chicken 11. × 55. Proliferation of the original epithelium lining the collecting space; first stage of process. Hematoxylin-eosin. (Killed after 56 days on experimental diet.)

FIG. 3. Cross section through the maxillary gland of Chicken 1. × 110. Small foci of stratified keratinizing epithelium in the collecting space; degeneration of the epithelium in the acini. Hematoxylin-eosin. (Killed after 30 days on experimental diet.)

FIG. 4. Cross section through the base of the tongue (glandula lingualis) of Chicken 9. × 80. Shows a more advanced stage of the process. The cells, which are to replace the original epithelium, form a continuous vascularized syncytium, lying directly upon the surrounding connective tissue. Beginning keratinization, desquamation and degeneration of the upper layers of the newly formed epithelium. Hematoxylin-eosin. (Dead after 75 days on experimental diet.)

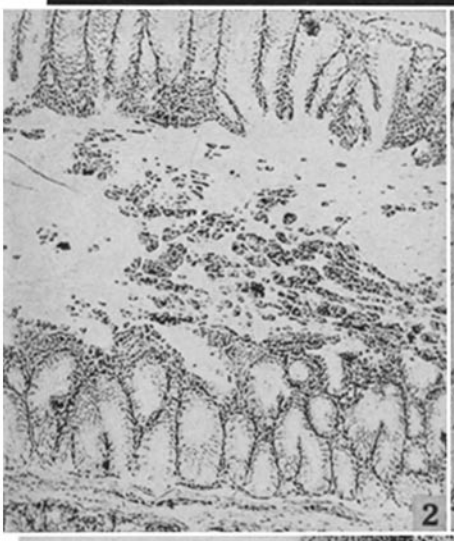
PLATE 25

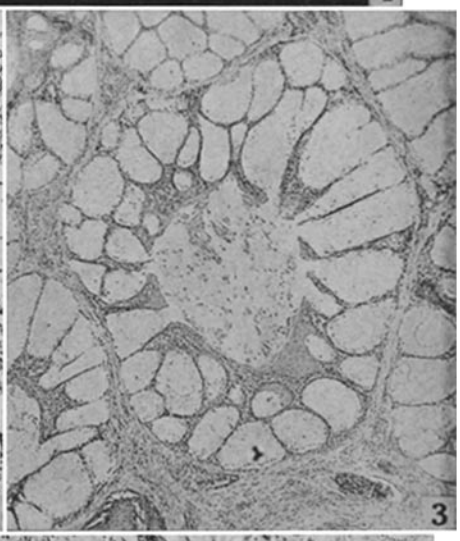
FIG. 5. Cross section through the base of tongue (glandula lingualis) of Chicken 6. × 50. Final stage of the entire process. The glands are completely filled with stratified, more or less keratinized, homogeneous masses. Extreme dilatation of the glands. van Gieson stain. (Dead after 87 days on experimental diet).

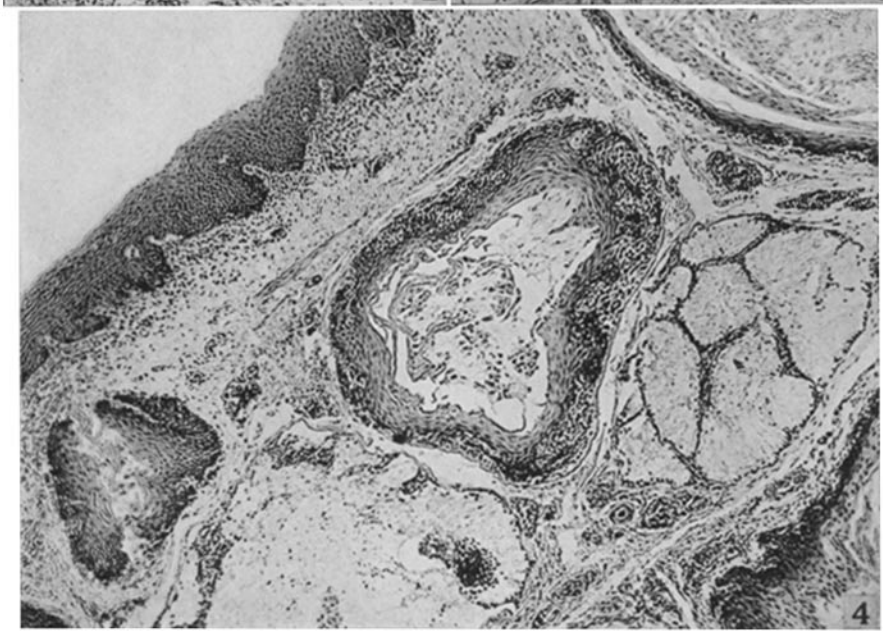
FIG. 6. Excretory duct of the glandula palatina medialis of Chicken 5. × 50. Bacterial infection followed by necrosis of the duct and its surroundings; accumulation of necrotic masses, mucus, inflammatory products and numerous bacteria. Giemsa stain. (Dead after 85 days on experimental diet.)

FIG. 7. Excretory duct of the lateral glandula palatina of Chicken 7. × 875. Invasion with numerous bacteria. Methylene blue stain. (Dead after 82 days on experimental diet.)

FIG. 8. Cross section through roof of mouth (glandula maxillaris) of Chicken 7. × 75. Thick membrane covering the mucous membrane, developing after secondary infection of the excretory ducts. Hematoxylin-eosin. (Dead after 82 days on experimental diet.)







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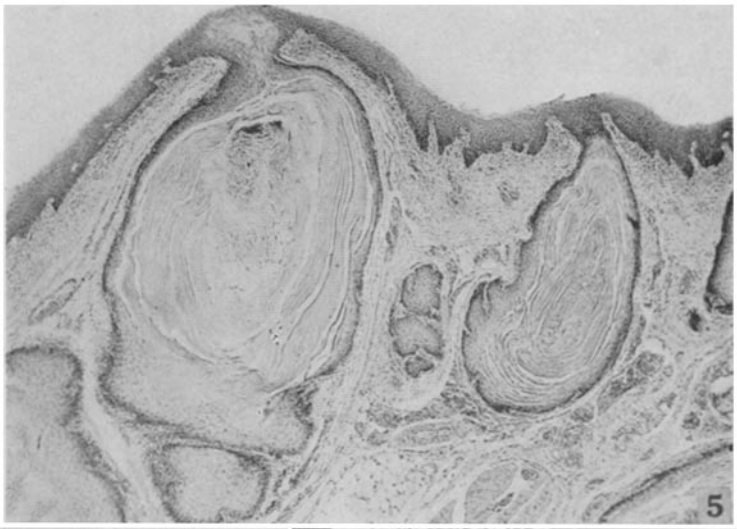

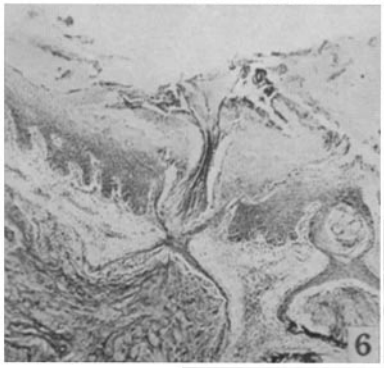

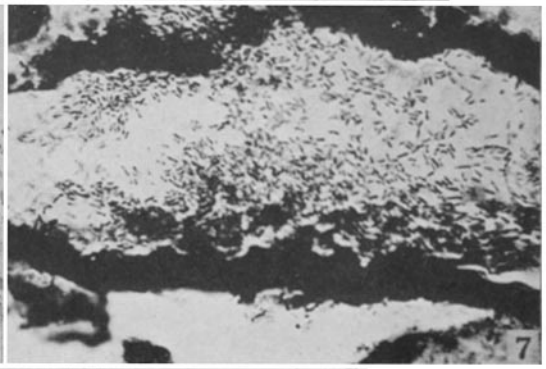

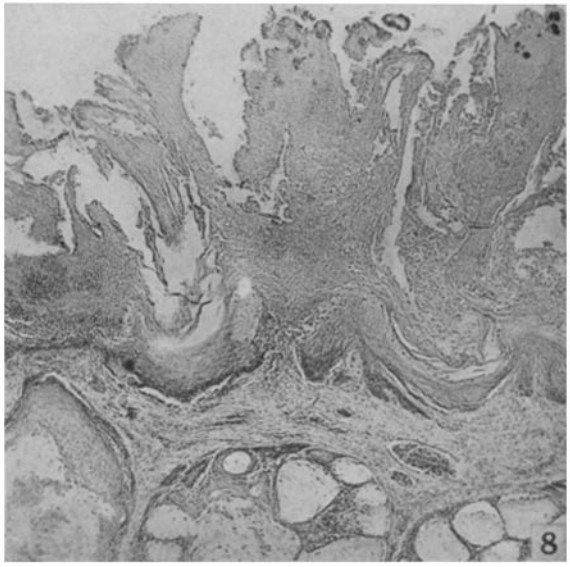

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