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Author(s)	渡邊, 彌三太郎
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七面鳥の黒頭病に就て

農學士 渡邊彌三太郎

ON THE SO-CALLED BLACK-HEAD OF TURKEY.[1)]

By

Y. WATANABÉ.

Agricultural College, Tohoku Imperial University.

Introductory.

Since 1905, the turkeys in our college farm have greatly been destroyed by some unknown disease. When attacked by the disease in question, they fell within a few days. The disease seems to be infectious, and especially perilous. Our farm keeper threw some of the dead animals into weak alcohol, probably weaker than 70%. I have undertaken a work to determine the cause of the mischief and, if possible, to find out the methods in taking care of the suffering individuals. The present paper is embodied of the results so far as obtained in this work.

The material fixed in the above-stated fluid was put in successively stronger alcohols and was imbedded in the ordinary way in paraffin-mixture. It was next divided into sections about 1/100 mm. thick, making use of the Schanze's microtome.

As regards the staining fluids, I employed several kinds, such as Delafield's hæmatoxyline, Hansen's hæmatoxyline, eosin, congo-red, etc., but double staining by Hansen's hæmatoxyline combined with congo-red proved to be the best. For the staining of bacteria, which are found among the cœcal contents, fuchsin, gentiana-violet, methyl-blue and Gram's fluid were preferable.

To my great regret, the present material is very imperfect, indeed, owing doubtless to the imperfect method of fixation. As very well known, tissues thrown

1) This paper was originally presented as a graduating dissertation.
[Trans. Sap. Nat. Hist. Soc., Vol. III. 1910.]

in weak alcohol suffer thorough damages in their microscopic structure: they are quite valueless for histological works. And the present investigation is connected a great deal with the histological studies. In addition to this, I could not obtain a healthy turkey, the viscera of which may, if necessary, be compared with those of the diseased one, in verifying the pathological changes suffered by the animal. In my hope, therefore, to renew my work with the fresh material probably obtainable in the coming summer, I left the present work in an imperfect state.

The disease in question appears in early summer and grows severer towards midsummer when it is most severe. Quite young chicken turkeys often show symptoms of the disease, so that we may conclude that the infection goes on already in the chicken.

The birds attacked by the disease soon become inactive, their plumage is turned into a dirty colour, and sowe feathers become ruffled. At the same time, the belly swells up enormously, and diarrhea soon follows, causing immediate death. There is little room in doubting that the direct mortal cause is diarrhea and decreased absorption of nutriment. In the following lines, I will point out some probable causes of the diarrhea and decreased absorption, together with some other factors standing in connection with these pathological phenomena.

I wish here to express my warmest thanks to Prof. S. HASHIMOTO and to Prof. Dr. S. HATTA and Dr. T. KATO for their courtesy shown by them during the course of my present work.

Part I. **DESCRIPTIVE.**

It should first be mentioned that the cœca as well as the liver undergo, as close examinations of the viscera of the preserved specimens show, manifest pathological alterations. For the sake of convenience, the cœca and liver will be dealt with separately.

A. **The Cœca.**

The cœca affected by the disease in question show a certain thickening, and on the inner surface of their walls are detected curious elevations. The thickening varies in degree according to cases met with: it is, in some cases, confined to the distal or to the proximal part, or to both the parts; in other cases, it forms patches circumscribing the walls; in still other cases, the whole extent of the tube's wall is uniformly swollen up. Wherever the cœcal walls are thickened, the serosa is always

thickened. The tuberculous elevations are, on the other hand, nothiug more than masses of compact but brittle bodies adhering to the mucous membrane of the cœca; they are yellow or yellowish-white in colour and fall off by a slight touch. The elevations occur in more numerous number in the distal part than in the proximal.

I have repeatedly examined these curious bodies detached; neverthless I could not determine, whether they are parasitic in nature or mere coagulated fluid. At any rate, the sections through these bodies are stained intensely by the Hansen's haemaloxyline, but slightly by congo-red. Those points of cœcal walls, on which the bodies are found, become sometimes slightly thickened, owing to the submucosa pathologically thickened; sometimes this is not the case. In either case, the walls are depressed on these points, causing the mucous epithelium to sink down and the tunica propria as well as the submucosa to be strongly pressed, so that there is formed in each point a funnel-shaped depression on the mucous epithelium. I can not tell, what is the cause of these changes of the cœcal walls. It is, however, beyond doubt, that the bodies in question are pathological products, because there was met with, in spite of my efforts, no trace of such strange bodies and such changes of the cœcal wall acompanying them in healthy cœca. I will turn away for a moment from this postulated point, to deal with it in future pages.

Let us now pass to the cause of another kind of thickenings which do not lie under the curious bodies just referred to. On series of sections through the diseased wall of the cœcum, we notice at the first sight a certain abnormity of the mucosa, especially that of the tunica propria. Furthermore such an abnormal feature of tissue is not unfrequently extended into the submucosa: even the mucous epithelium often suffers more or less such changes. A close microscopical examination goes to show that the damages are caused by a parasitic organism.

Numerous ovoid or roundish bodies are suspended in the meshes of the reticulated fibrous elements of the tunica propria. Examined in the preserved specimens, each of them is formed of the compact protoplasmic body enclosing a large distinct nucleus which is highly refractive. The cell body is 1.8-3.0μ. in diameter, and the nucleus 0.8—1.0μ (while the cell of the same kind found in the liver, is 2.5—4.0μ. in diameter, and the nucleus 1.0—1.5μ; *vide infra*). Most frequently the parasites occur in groups consisting of two or four; their solitary occurrence, however, is often met with.

I can tell nothing about their living state and the life-history they pass

through. There are, however, certain facts, by means of which we can infer some of the functions they performed during life. I have not unfrequently observed the solitary individuals possessing obtuse processes on their body surface, which look like pseudopodia suddenly hardened. It is thus highly probable that they creep about during life by means of the pseudopodia which they may produce on the suface of their body. On the other hand, the individuals in groups present no structure comparable with a pseudopodium or other locomotive organs of unicellular organisms. They are, therefore, to be regarded as being in a motionless state; they are, as I believe, in the phase of division or multiplication. This assumption is further supported by the fact that the individuals are united in groups formed of sometimes three, sometimes four and rarely more than six. From these facts the following consideration will prove to be true. The parasites multiply by repeated fissions, until they attain a certain number, perhaps, less than 10. Then they creep out of their cell-nest, sending out and withdrawing their pseudopodia. This may further be the reason why the parasites are rapidly added in number.

If the above considerations are convincing, the parasites in question doubtless represent a species of the group *Amœba*, as will be induced from their life history; but I can not say, however, at present with certainty, whether they are *Amœba meliagridis*, a name which Smith [1] has given to the parasite worked out by him in turkeys.

Concerning the damages which the parasites cause in the structures composing the cœcal walls, the following facts are to be mentioned. In the tunica propria, for instance, there is detected no additional matter which may be looked upon as pathological products. On the contrary, the fibers of this layer become very scanty: consequently the texture is much loosened. The spaces thus brought about are occupied by the scattered solitary parasites or by the cell-nests. This fact makes it intelligible that the fibrous elements of the tunica propria are to agreat extent dissolved: probably the parasites nourish themselves with the dissolved matter of the fibers.

In spite of decrease of the structural elements within, the cœcal wall swells up in its thickness; this is due to nothing else than rapidly progressing increase in number of the parasitic individuals, as shown by the fact that at the maximum of the thickening which I have observed, the interior of the tunica propria is thickly

1) Smith: Infectious Entero-Hepatics in Turkeys: Bulletin of the Bureau of Animal Industry. U. S. A. Dep. of Agr., No. 8. 1895.

loaded with the parasitic cells.

It is noteworthy that during the earlier stages of infection, the parasites are confined to the tunica propria, and that they invade by stages the submucosa and even the blood vessels. Changes caused by the parasites in the submucosa, are, on the other hand, quite different from those suffered by the tunica propria just stated: the structural elements, the fibers, are, in contrast to the case in the tunica propria, enormously added, so that the layer is woven with reticulated fibers of thick meshes. The meshes of the reticulated tissue are filled up with the parasitic cells. It is furthermore not unreasonable that there is, within this layer, produced a certain pressure. This pressure and the added tissue-elements probably take part to a great extent in causing the cœcal walls to be thickened.

Lastly the mucous epithelium is not directly attacked by the parasites, but is broken up in consequence of the enormous bulging of the strata underlying it, i. e. the tunica propria and the submucosa.

The Lieberkühn's crypts, which are formed of mucous epithelium, are also filled up with the parasitic cells; their walls suffer, of course, a damage to a certain extent.

Now we arrive at the question, how the parasites reach the place, the tunica propria, where they are found in earlier stages of the disease. I have no positive evidence in proving the actual invading of the parasites; there are, honever, several negative inferences as to the possibility in regard to their invasion. In the first place, it may be assumed that the parasites arrive at the tunica propria by an indirect way from the blood-vessels which they enter at a far distant place. But this assumption could not have bean proved: so far as my observations extended, nowhere were the parasites found in the blood-vessels at the time when they make their first appearance in the tunica propria.

In the second place, the Lieberkühn's crypts may be taken as the entrance of the parasites; for the tubes are not only in a situation appropiate for the entrance of the parasites, but they are, in fact, very often filled up with the parasitic cells. There is, however, an important factor which should not, at any rate, be overlooked for the elucidation of the problem: the parasitic cells are detected always in the outside as well as in the inside of the tube's wall, i. e. in the tunica propria and in the tube's interior, and they never occur in the latter part alone. It is, therefore, evident that the parasites in the tube's interior push their way from without into the interior, and not in the inversed direction: in other words, they come into the

crypts from the tunica propria, destroying certain points of the crypts walls, which can actually be made out; consequently the present assumption is disproved.

In the third place and lastly, I have mentioned in the foregoing pages that the mucous membrane is excessively depressed at the points where the curious bodies are found. The depression shows for each body the shape of a funnel, so that it encloses the basal half of the body; the underlying tissues, especially the tunica propria and the submucosa, are strongly pressed, so as to be turned into compact layers. When we follow the series of sections through a depression, we see that the mucous epithelium forming the bottom of the depression is, in a certain extent, dissolved. Furthermore, the fibers of the tunica propria adjacent to the depressions are also melted together, and the numerous parasitic cells are found imbedded in this molten mass. This part is the only point which is to be recognised as the entrance of the parasites, as the following considerations will prove.

Suppose that the parasitic cells arrive at the interior lumen of the cœcum, having been carried in by the food ingested; the cells sooner or later fasten themselves on the surface of the mucous membrane of the cœcum. By the influence of their parasitic life on this part, there is produced an abnormal body, as it seems to me, in a similar way as the gall-nut is produced on a plant leaf, to which gall-wasps or gall-mites give their irritating stimuli, in leading their parasitic life on it.

The increasing irritation of the mucous membrane by the parasites causes, as it were, the abnormal body in question to be added in its bulk, so that it presses, at last, strongly upon the cœcal wall, until the wall has been depressed into a funnel-shaped pit, embracing the basal half of the body. If this assumption is correct, the body in question may represent what was spoken of above as the curious body, and it follows that the pit is nothing else than the above-mentioned funnel-shaped depression.

Next, the parasites migrate into the tissues of the cœcal wall, destroying the epithelial lining; in the first step, they come into view in the tunica propria and turn, as above stated, the structure into amorphous masses, in which they are found imbedded. I can not say with certainity, however, how this is brought about; but there is little doubt in assuming that the changes are effected by the parasitism.

The parasites not merely wander about within the tissue of the tunica propria, but force their way, on one hand, into the submucosa, and on the other, into the mucosa layer, under rapid multiplications. In the submucosa they do not give any marked change to the structure at all. On the contrary, the mucous epithelium

suffers injury; this is especially the case in the walls of the Lieberkühn's crypts: the crypt's walls are destroyed at several parts which the parasites attack in multitude to make their way into the interior of the crypt's lumen, as stated in the foregoing lines. I call attention to this point of destruction by the parasites: namely, the parts of the crypt's walls, which are destroyed in consequence of the parasitism, form the only way of escape of the parasites. The animals come to the interior of the cœcum through the crypt's walls broken up and are cast off to the exterior, being intermingled with the cœcal contents, viz. the fæces; in this way they may infect other host individuals.

B. **The Cœcal Contents.**

Having dealt with the diseased features of the cœca, the cœcal contents will briefly be examined, in order to make intelligible the relations of the parasitic organisms which may be found in the contents, to the diseased parts of the cœca.

The contents of the turkey's cœca are hard, owing doubtless to the action of the preserving reagent employed. They are coagulated, though semifluidal in their fresh state, being greenish in colour similar to those taken off from the fowl's cœca. A microscopical examination shows that the cœcal contents consist mainly of undigested parts of their foods, such as the vessels and fibers of plants. Only one species of animal parasite is detected, but numerous in individual number. It is a species of the genus *Trichostomum* of Trichocepalidæ, Nematoda. Bacteria are found in abundance: at least six species are to be distinguished. All these vegetative parasites have, it seems, certain physiological meanings in causing putrefaction to the food undigested. There are found many epithelial cells which show a great similarity with those forming the mucosa layer: they have doubtless fallen off from this layer in consequence of artifact. It is very striking that there are detected only a few individuals of the parasitic protozoön which was recognised, in the foregoing lines, as causing the disease. There is, therefore, little room for doubting that these parasitic cells are on their way escaping from the cœcal walls where they live in colony.

C. **The Liver.**

The liver of a diseased turkey shows resemblance to the fowl's liver in its external configuration. On the surface of the liver, we find numerous coloured spots varying in size. They are in some cases 17 mm in diameter, while in other

cases they are represented by mere points which also vary in size among themselves. These peculiar spots are greater in number on the upper surface than on the under or gastral surface of the liver. In a surface-view they are round or ellipsoidal in outline and flattened or slightly depressed on their surface, and are lemon yellow or light yellow in colour. On this ground colour, are seen dark brown lines of varying forms: in some spots the lines mark a net-work and often show irregular markings; in others they are represented by radial striations sent off from the center of the spot. Besides these sharply defined spots, there is another kind of spots, showing mottled brownish colour and being marked off from the surrounding liver tissue only by their darker colour, while in some other cases, there are found uniformly light yellowish spots, shading away gradually into the surrounding tissue. The spots are often so hardened, that they can easily be taken off, by a slight touch, as plates or scales.

The peculiar occurrences above referred to are due, self-evidently, to the pathological condition. Let us proceed to show, how they are caused.

Observed in sections into which the diseased liver is divided, the liver tissue beneath the above stated spots shows great changes. The hepatic cells are fused together, and the nuclei become bigger, and are feebly stained. In some cases, the liver cells are converted into fibrous net-like structure containing neither nucleus, nor blood capillaries and blood corpuscles, and are still less affected by certain staining reagents such as the Delafield's haematoxyline, etc. The changes of the liver tissue go so further that the whole tissue is transformed into a single homogeneous plate, containing no blood-vessel and no blood corpuscle at all. This plate is to be hardly stained with the above-mentioned haematoxyline or some other staining fluids.

Very curious, however, it is that, in and about the changed tissues of all the kinds above enumerated, I can not make out, in spite of my efforts, anything to be regarded as parasites. On the other hand, amidst the unchanged liver tissue, lying apart from the above-mentioned changed tissues, I made out foreign cells, two or four of which are in groups. Beyond doubt, these cells are parasites; they are, in all their features, quite the same as those pointed out in the cœcal walls, except their bulk slightly greater than the cells of the latter lot, varying from 4.0 to 2.5μ in diameter (see p. 69).

These parasitic cells are detected in the meshes of the hepatic cells as well as in the places which were doubtless occupied formerly by the hepatic cells themselves

and which have now been destroyed. In addition, they occur in bile-ducts and in the interlobular vessels which latter represent probably the branches of the portal veins.

The parasitic cells under consideration are found free in the vessels as well as in the ducts, but those in the hepatic meshes are surrounded by fibrous capsules which are, as it seems, extended by stages. Observations of sections through the liver in several stages of the disease, show that the fibrous corpuscles extend themselves to the peripheral parts of the liver. The fibrous structure in this case can by no means be distinguished from the above-stated scale-like and plate-like structures, containing no parasites. We are justified, therefore, in concluding that the fibrous structure is pathogenous in origin, being caused by the parasites, and that the scale-like and plate-like structures above mentioned represent nothing else than advanced states of the same pathological changes of tissue, although there is detected no trace of parasites. In short, when the disease is advanced, the parasites disappear in the places previously infected, migrating into other parts where the nourishment is not yet exhausted.

I have nothing at present to tell with certainty about the mode, in which this fibrous change of the hepatic tissue is brought about; it is, however, highly probable that this change is due to the abnormal increase of the connective tissue, which forms the support of the hepatic glands, by stimulations of the parasitic life. The host animal dies simply because the liver loses its functional power to a certain degree, when the pathological change in question is extended to a certain extent.

Next we come to explain, how the parasites enter the substance of the liver. I have mentioned in the foregoing lines that the parasites are found in both the bile-ducts and portal branches of vessels. The former constitute, as I believe, their way escaping, while the latter represent their entrance. There is little room in doubting that the parasitic cells do not travel by means of their own activity, but are, to a great extent, transported in a passive way by the medium in which they are found, and this medium is represented by the hepatic juice poured out from, and the portal blood hasting into, the liver. It is, therefore, convincing that the parasites enter the liver through the portal blood, and escape through the bile-ducts.

From the facts above pointed out, it follows that the cœcum is the first to be attacked by the parasites, and then they come, on the way of the portal stream of blood, into the liver. The parasites in the cœcal walls represent, I venture to say,

a generation different from, and foregoing to, the generations to be passed in the liver.

If the considerations above given are valid, the progressing process of the disease may be assumed as follows: in the first stage, the hepatic cells are more or less destroyed; in the second stage, the hepatic tissue is turned into fibrous reticulum, the gland cells being totally absorbed; in the third stage, the reticular structure disappears to a large extent, and the parasitic cells can no longer be detected there at all; while in the fourth stage, the fibers constructing the abnormal tissue is converted into an almost homogenous plate. When the histological changes of the last state advance to a certain extent, then the death of the host animal takes place.

The parasites escaped through the bile-ducts are, it is obvious, cast off, together with the excrement, throngh the vent. They are probably encysted in the exterior to be again taken up, mixed with foods, by other host individuals which will be infected by them.

Part II. HISTORICAL REVIEW AND CONCLUDING REMARKS.

It is rather curious that the striking disease of the turkey above referred to has drawn the attention of comparatively a few scientic observers. So far as I am aware, concerning the disease in question, there are only a few published papers, of which the work by Cushman,[1] that by Smith[2] and that by Moore[3] are very well known. The views advanced by these three authors are in accordance in concluding that the bacteria which are found in the diseased organs of the turkey in large number, can by no means be looked upon as the pathogenetic in the disease under consideration. As mentioned in the foregoing pages, the results of my present work also speak for this view. As to the real cause of the disease, on the other hand, the results arrived at by myself best agree, as seen from the above descriptions, with those by Smith,[4] but I can not, at present, determine with certainty, whether or not the parasite represents the species named by Smith. In spite of

1) Cushman: Nature of Black Head in Turkeys: Reports of Rhode Island Agr. Exp. Station, p. 199, 1894.

2) Smith: Infectious Entero-Hepatics in Turkeys: Bulletin of the Bureau of Animal Industory, U. S. A. Dep. of Agr. No.8. 1895.

3) Moore: The direct Transmission of infectious Entero-Hepatics in Turkeys: Circular No. 5. Bureau of Animal Industory, U. S. A. Dep. of Agr., No.7., 1896.

4) *Loc. cit.*

the morphological harmony of the parasite observed by Smith with that by myself, it differs in size: the parasitic protozoön in my case is smaller, as compared with that in the case of the American observer; this differenc is, however, due, it is probable, to the different reagents of fixation employed in both the cases. A further difference in the results by Smith from mine consists in the frequent occurrence of the exudates on the outer surface of the diseased cœcum, while this is not the case in the specimens observed by myself. I have never met with at all any structure which may answer to the giant cells mentioned by Smith: they are, I think, nothing else than the thickly grouped parasitic cells imbedded in the destroyed tissues.

From the accounts given above, the mortal cause of the host is not difficult to infer. In the case in which the host dies when the cœca alone are attacked, the death is due to diminished absorption of the digested matters; for the cœca of the birds form, as is very well known, a strong organ of absorption. The death in the case of the disease attacking the liver is self-evidently caused by the weakened hepatic function. Very common mortal causes consist in destruction of both the organs.

Among others, Schaudinn worked out *Entamœba hystolyca* occurring in the human body. It is in great resemblance with the parasite of the turkey above mentioned. A comparative study of the parasites in both the hosts will prove of interests.

It is the common opinion in our country, that turkey farming is connected with great difficulties, and we have been informed that the difficulties consist chiefly in protecting the bird from the disease above referred to. To do this, we have only to keep off the chicken from the infected individuals.

Summary.

1. The disease is caused by the parasite called *Amœba* sp., and not by bacteria found in the cœca.

2. The parasite attacks first the cœca, the histological structure of which is destroyed by it.

3. Some of the parasitic organisms escape from the cœca through the Lieberkühn's crypt, into which it comes out, breaking up the crypt's walls.

4. Passing through the portal vein, it comes then to the liver which is converted by it at last into hard plates.

5. The parasite escapes from the liver through the bile-ducts.

6. The parasite from the liver, together with that from the cœca, is cast off

from the enteric canal through the vent, being intermingled with the excrements, to be infected to other individuals.

7. The direct mortal cause is the destruction of the coeca or liver, or of both the organs.

摘　　要

明治三十八年以來、我東北帝國大學農科大學農場に飼養せる七面鳥中、原因不明の疾病に罹りて死するもの多く、殊に、稚雛に其の甚しきを見たり。蓋し、該疾病は傳染性にして且つ猖獗なるが如し。余は其の病源を明かにし、更に進んで之れが豫防並に治療法を確實にせんとして、是れが研究に着手せり。これ此研究の一部なり。

疾病の爲めに侵害されたる部分は盲腸と肝臟となり。此疾病に襲はれたる『盲腸』は、其壁厚くなり且つ異常の隆起を呈す。盲腸壁の此肥厚及び隆起の病的產物たるは、是等が健全なる盲腸に伴はざるを以て明かなり。隆起の下に敷かれたる粘膜は甚しく壓迫せられ表面に漏斗狀の窪處を生ぜり。又隆起の下にあらざる肥厚部を見るに其組織に著しき異狀あり、此異狀の及ぶ區域は、粘膜は素より其下層も多少の害を被れり。此の變化は、實に寄生生物に原因す。

寄生生物として見るべきは粘膜下の網眼中にある、卵形若くは圓形の、無數の細胞なり。此細胞は大にして且つ強く光線を反射する核を含み、直徑1,8乃至3,0μありて、後に述ぶる肝中の寄生細胞より、稍小なり(肝中の寄生細胞は2.5乃至4.0μあり)。寄生細胞は單獨にあることもあれど、多くは二乃至四個づゝ群を爲してあり。此生活狀態は今知るに由なしと雖も、其表面上にある鈍き突起は、虛足が

アルコールの爲めに急速に固定せられたるは疑なし、よりて考ふるに、生活中此虚足によりて這ひ廻りしこと明かなり。また群を爲せるものは、虚足其他原生々物の移動器と見るべきものを有せず、此等は休止の狀態にあり、分裂蕃殖しつゝあるや明かなり。是等の事實を總合して考ふるに、此寄生生物は分裂增殖し、後一つ一つ分離して這ひ出すものなり。然らば則ち此寄生生物はアミーバの一種なること明かなるが、果してスミス氏の七面鳥にて發見せしと同一のアミーバなるや否やは疑問なり。

盲腸壁内の礎膜中には、寄生動物以外には病的生產物として見るべきものなし。之れに反して組織は大にゆるみ、其間隙は彼の寄生細胞と其群体とによりて充たさる。是れ組織成分たる纖維は此寄生動物の爲めに融かされ、其營養物となれるなり。かく成分の減少せるにもかゝはらず壁が肥厚せる所以は、寄生動物が急劇に增加して組織間を埋るによる。疾病初期にありては寄生動物は粘膜下にのみ棲めども、順次其下層に及び粘膜下層並に血管を侵すものなり。此層は礎膜と反對に、組織要素大に增加し、其網眼は寄生細胞を以て充たさる。是れ盲腸壁の厚さを增すに與りて力あること論を俟たず。粘膜表皮は直接寄生動物に侵かされざれども、其下にある礎膜及び粘膜下層の著しく膨大せる結果、破らるゝに至る。リーベルキューン氏腺は又た寄生細胞を以て充たさる。

今また此寄生動物の盲腸壁内に侵入する經路を見んに、

(第一) 血管より來らず、如何となれば疾病の初期に礎膜は血管より先きに侵さるればなり。

(第二) リーベルキューン氏腺内には礎膜より後にて現はる、故に此の腺より侵入せしに非らざること明かなり。

(第三) 余は上に異常なる窪起が附着せる所に於て、粘膜が漏斗狀に凹入することを記せり、此所にては粘膜表皮は殆んど壓し破られ且

つ礎膜の纖維は夥しく融され、其中に多數の寄生細胞を包含す、是れ則ち寄生生物の侵入せし入口と考ふ可き唯一の所なりとす。其の經路を想像するに左の如くならん。

寄生生物は食物と共に胃內に入り、盲腸に達し、フシ蜂或はフシダニが葉上に五倍子を生ずるが如く粘膜に異常物質を生ぜしむ、而して盲腸壁の組織中に表皮を破りて礎膜に出でゝ增殖し、粘膜下層粘膜に移行し、リーベルキューン氏腺を破りて腺內腔に出で、其れより盲腸內容物と共に排泄せらる。

盲腸の內容物は重に食物の不消化部分なり。加之バクテリアの夥多と一種の寄生蟲と、粘膜の脫落せるもの及び先に記せる寄生動物の細胞とあり。寄生生物中バクテリアと一種の圓蟲類は他の健康体にも見出さるゝものなりとす。

此の疾病に罹れる『肝臟』の表面には、大きさを異にせる多數の斑點ありて、大なるは直徑十七粍、小なるは漸く認め得るに過ぎず。其色橙黃色又は淡黃色に、其形楕圓又は圓形なり。斑點の基色中に射出狀、網狀等の紋線あり。此の斑點は肝臟組織の著しく變化せる部分なり。肝臟組織の變化の順次は(1)肝細胞は僅かに破損せられ、核大きくなる。(2)細胞は順次融解し肝の組織は網狀となり核、血管、毛細管を含まず。(3)網狀に變ぜる細胞は次で組織一樣なる板狀となる。寄生動物は直徑2.5乃至4.0μありて單獨に、或は二個乃至四個宛群をなして、肝細胞、膽管並に門脈中に見出さる。即ち寄生物は其運動器を以て自ら遠く移行する能はざる可しと雖も、門脈內の血液に乘じて肝臟に入り、膽管を通じて外界に出で去る。

此疾病に關する研究は僅かにカッシマン、スミス、ムーア氏等のものあるに過ぎず。余の研究の結果はスミス氏のそれと大に似たるものあり、只スミス氏の發見せるものに比し、原生動物は小なりき。是れ或は兩者固定劑の異なれるによるならんか。他方面

に於てはシヤウヂン氏は人に寄生し病原となるエントアミーバ、ヒストリカに就きて極めて有益なる研究をなせり。蓋し此等の疾病及び原生動物の比較研究は彼我利する所大なるを信ず。

以上の説明したることを下に枚擧す。

1. 病原はアミーバの一種に屬する寄生動物の寄生に因る。
2. 病原蟲たるアミーバは最初盲腸を侵して其組織を破壞す。
3. 寄生動物の一部はリーベルキューン氏腺を破りて出で去る。
4. 寄生動物は門脈より肝臟に流入して肝臟に寄生し、其の組織を破壞す。
5. 寄生動物は膽管より肝臟を去る。
6. 肝臟より去れる寄生動物は盲腸の內容物と共に排泄さる。
7. 直接の死因は盲腸或は肝臟の破損若くは兩者の破損にあり。

七面鳥飼養の難事たる原因は實に玆に存す、故に此の疾病の病原たる寄生動物の傳播を防ぐは最も適切なるものなり。