sions, its anatomical course is in the main a progressive one, and the remittent stages often occur under treatment other than that of decapsulation.

Of the above 33 per cent. mortality, 26 per cent. died from chronic interstitial nephritis; and of the remainder, in 25 per cent. death occurred from the so-called chronic parenchymatous nephritis, and in 75 per cent. "diffuse" (?) nephritis was assigned as the cause.

In acute nephritis, anuria, haematuria, puncture and decapsulation of the kidney is said to yield relief and to favourably affect the function of the other kidney. In these cases it is suggested that the improvement is due simply to the relief of tension.

From the standpoint of pathology we may not make an excursion into the field of surgery, and therefore we must not discuss the possibilities of the operation. We feel, however, that the purpose of the paper would not be served unless we mentioned that while many American and some French surgeons regard the procedure as one that has come to stay and as possessing much promise in the treatment of degenerative and interstitial nephritis, such operators as Israel, Nicolayson, and others have already abandoned the method in chronic conditions. It is, of course, a matter of common knowledge that the simpler reni-puncture relieves tension and promotes renal functions, so perhaps the description of the changes observed after decapsulation may give more prominence to the method of simple incision, may emphasize the probability of rapid local reparation when the temporary obstruction is removed, and may suggest the earlier surgical treatment of those post-febrile and acute renal manifestations, which, when unrelieved, often terminate fatally.

Since the completion of our experiments Boncz-Osmolowsky has published a paper on "the changes in the kidney after removal of its capsule." He used rabbits for his experiments, and found that six days after decapsulation the leucocytes and connective tissue were increased between the tubules and that the cells of the superficial tubules showed granular changes in the protoplasm. Twelve days after the operation the superficial tubules and glomeruli were slightly compressed by the connective tissue elements, and there was atrophy of renal cells. He did not observe any new formation of blood vessels in the reformed capsule.

[Some portions of this paper were communicated to the March meeting of the Manchester Medical Society.]

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REPORT LXXXV.

SOME FURTHER OBSERVATIONS ON THE LIFE-HISTORY OF FILARIA IMMITIS, LEIDY.*

By THOS. L. BANCROFT, M.B. EDIN.

I COMMUNICATED to the Royal Society of New South Wales in June, 1901, a paper entitled "Preliminary notes on the intermediary host of *Filaria immitis*, Leidy," since then I have had the opportunity of following up several points in the life-history of this parasite, which remained to be elucidated.

Fig. 1.—Diagram of a "house mosquito" (*Culex fatigans*), Wied, in the act of imbibing blood, drawn from photomicrographs.

It was evident that the young or larval filariae, arrived at maximum development, as far as their life in the mosquito was concerned, passed out, in some way or other, into their final hosts, but the exact manner of exit was a disputed point. I had myself suggested that the worms passed down the proboscis into the wound inflicted by the mosquito; I did not think it possible for such helpless creatures to swim against the blood stream entering the mosquito's stomach by the epipharynx. Afterwards, whilst examining labia of filarated mosquitos, mounted in water and with a cover-glass, it was observed how readily the worms escaped from the apex of the proboscis or labium, and it was suggested that they naturally

Fig. 2.—Diagram of the head of a mosquito imbibing blood, depicting the escape of a young filaria into the skin alongside the stylets. A, thorax; B, head; C, clypeus; D, skin; E, eye; F, filaria; H, labellae; I, antenna; L, labium; N, neck; O, occiput; P, palpi; S, stylets.

thus made their exit. Dr. Geo. C. Low considered it probable that the young filariae passed out of the labium at its base; Grassi, however, could not believe it possible for larval worms, without any special mechanism for boring their way, to pierce the integument of the labium; he had an idea of his own to explain their exit from the labium. As is well known, when a mosquito imbibes blood, the labium or sheath of the stylets is bent upon itself, whilst the stylets are thrust deep into the skin; Grassi considered that when a labium containing filariae was thus buckled up it would be ruptured and the worms extruded. This hypothesis was easily disproved, for upon microscopic examination of the labia of filarated

*The expenses of this investigation have been met in part by a grant of £5 from the British Medical Association, London, July 25th, 1901.

mosquitos, which had bitten and relieved themselves of parasites, one or more openings were seen in the labellae, often very apparent owing to the escape of pigment and granular matter; there were no openings elsewhere.

It remained to be proved by actual experiment, that filarated mosquitos could infect dogs, and it was important to ascertain how long a time would elapse before embryo filariae could be detected in the experimental dog's blood. The time taken from the bite of the mosquito until the embryos can be found in the blood is about nine calendar months; in one instance it was only eight and a-half months. One dog, which had been filarated, was stolen at eight months, up to which time there were no embryos in its blood. To fix the time as accurately as possible, this experiment was made: a puppy, bred from non-infected parents, was kept away from filarated dogs; on December 30th, 1902, it was put into the mosquito house and 183 filarated mosquitos, three weeks old, were liberated; the next morning it was found that 70 mosquitos had bitten; that was the only infection the dog received. After seven months had elapsed its blood was examined weekly, but it was not until October 1st, 1903—that is, nine months—that the embryos were detected; the dog was then killed and the heart and lungs examined. There were 32 fully-developed filariae (16 males and 16 females) in the right ventricle and pulmonary artery.

I was desirous of ascertaining (although the chance of doing so was very slight), where in the dog's body the young filariae lived before betaking themselves to the heart. I had on several occasions, whilst dissecting filarated dogs, found half-grown forms in the lungs. One dog, which had been bitten by 103 filarated mosquitos and in whose body there would be at least fifty worms, was examined three months afterwards; the tissues were sliced with a knife or cut into small pieces with scissors in hope that, were a worm cut, it might move or its body protrude and thus attract the eye; nothing, however, was seen. Mature filariae scarcely move when cut across, and might readily be overlooked, so it can be understood how such delicate bodies as young filariae might easily escape detection. It is probable that the young filariae live in minute arteries in the lung. Another filarated dog was examined seven months after infection, full grown filariae, both males and females—the females 10 in. long, which is the maximum length they attain to—were found in the heart; the uterine tubes were devoid of embryos however. The parasite is at this date probably sexually mature, but a month or two must elapse before embryos are developed.

Two dogs used as a "control" in this investigation, lasting over a period of two years, remain free from filariae.

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DIGESTION AND ABSORPTION OF HAEMOGLOBIN.

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SOME years ago, when Bunge's theory of the part played by inorganic iron preparations in the relief of anaemic conditions was exciting interest, it occurred to me as important to determine whether haemoglobin was capable of absorption. For among organic preparations of iron, haemoglobin is sometimes prescribed for the cure of chlorosis and allied disorders.

Pressure of other work at the time caused me to leave my investigation in an unfinished condition, and I never took it up again, and in the meantime numerous other researchers had taken up the question and published their results.

The main trend of this work has been to show that numerous preparations of iron, both inorganic and organic, including haemoglobin, are really absorbed, though probably in small quantities, and do not, as Bunge taught, merely remain in the alimentary canal, to be the ready prey of bacteria, so protecting the normal haematogen (iron-nucleo-proteid) contained in small quantity in numerous food stuffs.

The evidence adduced by these investigators in favour of the absorption hypothesis may be briefly summarized in the following way:

1. Animals fed on the preparations added to their food, rendered as iron free as possible, increase in weight, and thrive better than the control animals.

2. Their red corpuscles increase in number and in their richness in pigment. This by itself might, however, be equally well explained by Bunge's theory.

3. By macrochemical methods the amount of iron in their spleen and other organs increases more than can be explained by the small amount of iron in the haematogen of their food.

4. By microchemical methods the granules containing iron can be detected in these organs as well as in the intestinal epithelium. Of the microchemical methods, that with ammonium sulphide has been shown to be more trustworthy than that involving the use of potassium ferrocyanide.

5. The duodenum appears to be the situation where most absorption takes place; the spleen the principal situation for storage; the reappearance of the iron reaction in the colon, and to a certain degree in the kidneys, appears to indicate excretion rather than absorption.

I may say briefly that my experiments, as far as they went, supported on all these grounds the hypothesis that haemoglobin given with comparatively iron-free food is really absorbed, and may thus lead to blood formation. Looking through my old notes, I have determined that it is quite worth while to publish the results, not because they now contain anything which is absolutely new, but as confirming the observations of others just alluded to. Moreover, in one or two directions the methods adopted have been somewhat different from those used by others. Particulars, for instance, concerning the action of the digestive fluids on haemoglobin I do not remember as having seen in previous publications.

ACTION OF ARTIFICIAL DIGESTIVE FLUIDS ON HAEMOGLOBIN.

It appeared to me first to be important to determine what changes haemoglobin undergoes when subjected to the action of the various digestive juices. For this purpose I prepared a large quantity of crystalline oxyhaemoglobin from dog's blood and divided it into several portions:

1. On keeping it at body temperature for some days it underwent no change.

2. On adding saliva to it and keeping the mixture at 40° C. there was also no change in twenty-four hours, except that the crystals underwent solution.

3. On adding to a solution of these crystals an equal volume of 0.4 per cent. hydrochloric acid the solution showed almost immediately and in the cold the bands of acid haematin. On keeping this at 40° C. a swollen amorphous precipitate of acid haematin settled out gradually, though twenty-four hours later a good deal still remained in solution. Menzies¹ states that the first effect of dilute acids on oxyhaemoglobin is to form methaemoglobin; this I have confirmed if a few drops only of the 0.4 per cent. solution of hydrochloric acid are added to the solution. But with the amount of acid present in gastric juice this stage, if it occurs, is too transitory to be caught. The spectroscopic bands of acid haematin and of methaemoglobin are very similar, but the two can be readily distinguished by careful measurement, and even more readily by the following test. If a reducing agent like ammonium sulphide is added, the absorption bands of methaemoglobin are replaced by those of oxyhaemoglobin and reduced haemoglobin; whereas the bands of acid haematin are replaced by the perfectly characteristic bands of haemochromogen. I sought for haemin after treatment with hydrochloric acid of this strength, but was never successful in proving it to be present.²

4. In the next experiment pepsin solution was added to the mixture with hydrochloric acid, and the resulting appearances were just the same. On keeping the mixture at 40° C. the acid haematin gradually separated out as a precipitate, the appearance of which closely resembled that of "coffee grounds." In this case the digest within a few hours gave evidence of the presence of albumoses and peptone derived from the hydrolysis of the proteid component of the haemoglobin. These did not appear when the acid alone was employed; the dissolved proteid in that case was simply acid albuminate. So far, we have learnt that the effect of gastric digestion is not only to form hydrolytic products from the proteid constituents of the haemoglobin, but the iron-containing portion is converted into acid haematin, which, as it mainly separates out as a precipitate, is not very likely to be available for purposes of absorption.

5. Sodium carbonate was next added to another portion of the haemoglobin solution until 1 per cent. was present. To half of this a solution of trypsin was added; the result in both cases was the same so far as the iron-containing portion of the pigment was concerned. In the specimen where trypsin was added there was naturally in addition the formation of proteolytic products. No immediate change in the pigment was produced, but on keeping at 40° C. the fluid became reddish-brown and showed the absorption spectrum of alkaline