ON ARTERIAL LESIONS FOUND IN EGYPTIAN MUMMIES (1580 B.C.—525 A.D.)[1]

By MARC ARMAND RUFFER.

(PLATES XLVIII.–L.)

THE mummies examined came from the XVIIIth–XXVIIth Dynasties (1580–527 B.C.), and from the time of the Persian Conquest (500 B.C.). I also dissected a Greek[2] and a Coptic mummy, the latter dating from the fifth or sixth century after Christ.

The investigations therefore range over a period of 2000 years,—namely, from 1580 B.C. to 525 A.D.

I take this opportunity of thanking Sir Gaston Maspero, Professor Flinders Petrie, Professor Elliot Smith, Dr. Derry, and Dr. Keatinge for help. To the last gentleman my thanks are specially due for giving me a number of arms and legs from broken-up mummies of the XXIst Dynasty (1090–945 B.C.). These limbs were of no possible use as museum specimens, and I had no hesitation, therefore, in dissecting them. Most of the preparations came from them.[3]

DIFFICULTIES OF THE INQUIRY.

The chief difficulty consisted in the extensive mutilations made during the process of embalming. Dr. Elliot Smith has shown that at the time of the XXIst Dynasty, the embalmers removed the whole of the viscera, the aorta, and most of the muscles of the body. The body cavity and the holes left in the limbs after removal of the muscles are found filled with mud, sand or rags, or all three.

The sole of the foot is packed with sawdust mixed with some

[1] Received November 26, 1910.

[2] This mummy was given me as coming from the Greek period. Judging from the way it was embalmed, I am of opinion that it really dated from the XXIst and certainly not later than the XXIInd Dynasty.

[3] The only papers I know on the subject are: (*a*) Shattock, "Microscopic Sections of the Aorta of King Menephtheh," *Lancet*, London, January 30, 1909; and (*b*) Armand Ruffer, "Remarks on the Histology and Pathological Anatomy of Egyptian Mummies," *Cairo Scientific Journal*, vol. iv., January 1910. Some of my pathological sections were shown at a meeting of the Cairo Scientific Society in December 1908.

"resinous" material. The muscles and big blood vessels of the neck are also gone, the larynx is either pushed upwards or has been removed, and the neck is filled with mud or rags. The cheeks are filled out with a fatty material mixed with sand and sawdust, and the brain removed. It is only by accident, therefore, that the whole or a portion of the aorta, or one of the large arteries is left behind. Fortunately for our purpose, one artery, namely the posterior peroneal, owing to its deep situation, often escaped the embalmer's knife. In a few cases also, when the embalmers had evidently scamped their work, the arm or leg was untouched.

After the removal of the foreign material, and when the limb is plunged in the softening solution, the walls of the cavity thus left are often found lined with a hard black material, not easily removed by water or a weakly alkaline solution. It must be taken away mechanically, and this often proves a very tedious process. When left standing in the hot and damp summer atmosphere of our laboratory, a brown gummy sticky fluid exudes out of this material. The same gummy substance is also found in bandages kept under similar conditions. I hope shortly to have some chemical evidence as to its nature.

The limbs of such mummies of the XXVIIth Dynasty as I examined were intact, but the body cavity had been almost cleaned out. The thoracic cavity contained an enormous quantity of jet black material showing a glistening surface, which I should certainly never have suspected of containing any tissue. In one case, however, a lump of this substance, which to the naked eye appeared to contain no tissue whatever, was placed in running hot water. The black substance slowly dissolved out, and then a small piece of aorta appeared, which, after long washing, showed exquisite calcareous patches. Mr. Lucas, who has chemically examined some of the material found in mummies, will doubtless, later on, give the result of his researches. Suffice it to say that the black shiny material is not bitumen. Indeed, it is a striking fact that up to the present, I have never found bitumen in any mummy, even in those of the Ptolemaic period.

The Coptic mummy had apparently been simply dried, and there was no evidence of its having been embalmed in any way.

Method of Isolating the Arteries.

The mud, sand, bandages, and gummy material are first picked out with forceps, or slowly scraped away; a most unpleasant task, as the dust floating in thick clouds about the room is most irritating to the lungs. The "packing" does not appear to contain pathogenic microbes, as, in spite of numerous cuts and scratches, no inflammation followed.

The limbs or trunk are thoroughly washed, and deep incisions are made into the skin, wherever necessary. The parts to be examined are then placed in a solution containing carbonate of soda 1 per cent. and formol 0·5 to 1 per cent., and soaked for twenty-four to forty-eight hours, when the skin can be taken off as a rule. After a few days of this treatment, the remaining

muscles, fasciæ, etc., are soft enough to allow the arteries to be dissected out. Unfortunately, the condition of the tissues is very variable, part of one limb, for instance, softening quickly, the remainder more slowly. In some cases, without any apparent reason, the muscles remain as hard as stone.

The arteries, especially the larger ones, such as the aorta, femoral, brachial, etc., are completely flattened out, looking as if they had been well ironed, and are therefore often difficult to find. If they have undergone marked fibroid or calcareous changes, the lumen may be patent and the vessel easily seen.

The arteries are dissected out and placed in a fresh solution of the above fluid for twenty-four hours. All adhering connective tissue is now removed, and the vessels are plunged into glycerine to which a few drops of formol have been added. This solution must be changed two or three times in the course of the next few weeks, as some colouring matter invariably dissolves out.

For microscopic examination small pieces of a calcified artery are placed in alcohol containing nitric acid, or better into Marchi's solution.[1] After twenty-four hours or longer the decalcified piece is washed in water for some hours, hardened, embedded in paraffin, and cut in the usual manner.

Fibrous pieces were hardened in alcohol in the usual way. It is very difficult to know, however, whether a given artery does or does not contain small calcareous patches, so that for practical purposes it is always better to decalcify first. Marchi's solution does no harm, and by adopting this process much time will be saved.

Description of Arteries examined.

1. Aorta (XXIst Dynasty) consists of a piece 4½ inches long, covered almost throughout its whole length by a hard calcareous plate.

2. Aorta (XVIIIth–XXth Dynasties).—The arch had been hacked away by the embalmer, who had also cut right through all the coats just above the bifurcation of the vessel. The thoracic aorta from a point just above the origin of the left subclavian artery and the whole of the abdominal aorta were intact and easily removed. The internal coat is studded with small calcareous patches, and the two largest, each nearly the size of a shilling, are situated just above the bifurcation. The left subclavian artery at a point just above its origin is almost blocked by a raised, ragged, calcareous excrescence, as large as a threepenny-bit (calcified atheromatous ulcer). Small atheromatous patches, not calcified, are scattered through the whole length of the aorta, and these, owing to the dark coloration of the tissues, are more easily felt than seen.

The common carotid arteries show small patches of atheroma, but the most marked changes are found in the pelvic arteries and in those of the lower limbs.

The common iliac arteries are studded with small patches of atheroma and calcareous degeneration. The other arteries of the pelvis are converted by calcification into rigid "bony" tubes, down to their minute ramifications. So stiff and brittle are they that it was impossible to dissect them out entire, and in spite of every possible care they were invariably broken. The minute intramuscular arteries were easily felt on triturating the muscles under the fingers.

Both arms and the legs (about 6 inches below Poupart's ligament) had been lost, but on the right side the common femoral and profunda were dissected out. Both were converted into rigid calcareous tubes.

[1] I have given up alcohol and nitric acid, as Marchi's solution gives much better results.

It is to be noted that, as far as could be made out from the examination of the cartilages of the ribs, the mummy was not that of a very old person.

3. Atheromatous Patches in the Aorta and Brachial Arteries in a Greek Mummy.—From the examination of the cartilages, etc., I concluded at the time that the man was not above 50 years old at the time of death.

4. Piece of Thoracic Aorta (XXVIIth Dynasty), altogether 4½ inches long. It contains seven calcareous patches, two of which are figured in Plate XLVIII. Fig. 3. No other lesion.

5. Aorta from a Coptic Mummy.—Small hard calcareous patches scattered throughout its length. The two largest are just above the bifurcation and are almost the size of a sixpenny-piece.

6. and 7. Pieces of two Aortæ, Thoracic (XXIst Dynasty).—No lesions.

8. Posterior Tibial Artery.—From a woman of the XXIst Dynasty, calcified from end to end.

9. Posterior Peroneal Artery.—A piece about 4 inches long. Artery stiff, lumen patent: evidently calcareous in places. After soaking, artery still very stiff; calcareous patches visible from outside. On opening, internal and middle coats almost completely calcified in places. In other places, vessel studded with minute calcareous nodules projecting into the lumen of the tube, hardly any healthy tissue being left between nodules.

10. Anterior Tibial Artery.—Apparently healthy, though lumen patent. On careful examination with lens, small points about the size of a pin's head, of a darkish brown colour. Microscopically these points were found to be foci of disease. See further on.

11. Posterior Tibial Artery.—One piece about 6 inches long. This is completely calcified, the whole being converted into a rigid calcareous tube.

12. Posterior Peroneal.—Apparently quite normal. The contrast between this smooth, highly flexible artery and the diseased vessels is most striking.

13. Posterior Peroneal Artery.—Quite soft and flexible, but here and there small highly coloured brown patches project into the lumen.

14. A Small Piece of Anterior Tibial Artery and Dorsalis Pedis.—Walls not markedly thickened, but distinctly nodular, with dark brown small nodules projecting into lumen.

15. Femoral, Profunda, and Branches.—Very tortuous and almost completely calcified.

16. Posterior Tibial and Branches.—Almost completely calcified from end to end.

17. A Piece of Artery found mixed with the Packing of the Leg.—Apparently quite normal.

18. Part of Posterior Tibial and Peroneal Artery.—Stiff, but no other changes to the naked eye.

19. Piece of Ulnar Artery, about 3 Inches long.—Lumen patent and artery stiff after soaking and being plunged in glycerine for weeks. It has a curiously mottled, brown and white appearance. On cutting sections fairly extensive calcification was discovered.

20. Several small Pieces of a Brachial Artery.—In glycerine it becomes beautifully transparent, light yellow in colour, but in spite of several weeks' soaking the longitudinal folds do not disappear. From the outside small brownish spots are seen studding it. On opening, these spots are seen to be small nodules projecting into the lumen. Some have a whitish centre with a brown irregular margin.

21. Ulnar Artery.—Apparently quite normal.

22. Part of Palmar Arch, Soft and Flexible.—Small brownish patches in first digital branch.

23. Ulnar Artery.—Apparently normal.

24. Brachial, Ulnar, and Two Inches of Radial Arteries.—Ulnar and radial almost completely ossified. Brachial studded throughout its length with brownish prominent patches projecting into lumen of the tube. These are mostly quite soft, but the centre of some is undoubtedly calcified. The whole artery is markedly thicker than it should be.

N.B.—When not otherwise stated the mummies belonged to the XXIst Dynasty.

The results noted may be summed up as follows:—

1. *Complete or incomplete calcification.*—There is no difficulty in recognising completely or partially calcified arteries. Even before they are placed in the softening solution, or at any rate shortly afterwards, their hard, "osseous" structure is manifest. Arteries, such as are depicted in Plate XLVIII. Fig. 1, are as rigid as calcified arteries of the present day.

When slit up, even with the finest scissors and the greatest care, the calcareous middle and inner coats have a tendency to detach themselves from the adventitia, and to break up into small, brown, roughly rectangular plates (see Plate XLVIII. Fig. 2). In this picture a small artery just branching off shows well-marked calcareous change.

After decalcification in picric acid and staining, microscopical sections of such arteries are most interesting and will be best understood by examining Plate XLIX. Fig. 9. This shows, under a low power, a decalcified posterior peroneal artery, stained by van Gieson's method, from a mummy of the XXIst Dynasty. The section is perhaps not quite satisfactory, in so far that, nearly the whole of the artery being diseased, it is difficult to find points of comparison between healthy and calcareous tissue. Only shreds of endothelium and fenestrated membrane, for instance, are left at *a*, *a*1, and *a*2.

The point of interest is that the muscular coat has been changed almost wholly by calcification, following on degeneration of the muscle fibre, into a magma of no particular structure. The disease clearly did not begin in one spot, but in several foci which coalesced, as at *b*, for example. When the section is stained with hæmatoxylin alone the calcified parts are coloured so black that hardly any structure is recognisable. Plate XLIX. Fig. 10 represents part of a calcified ulnar artery under a high power (same stain as previously). In this section the muscular fibres at *a* have been completely destroyed by calcification, so that no structure is recognisable. At *b*, on the other hand, the annular fibres are still indicated, though somewhat vaguely, whereas at *c* they are plainly visible. To the naked eye this artery appeared to be completely calcified.

Partial calcification was best seen in the aorta, and is well illustrated by Figs. 3 and 4 in Plate XLVIII. Here we see calcareous patches in two aortæ. Fig. 3 represents part of the abdominal aorta of a mummy of the XXVIIth Dynasty, and Fig. 4 a piece of a thoracic aorta dating from the same Dynasty. The flattened vessel did not open out again, in spite of long soaking in glycerine, but remained angular.

The calcareous patches are quite obvious, and it is unnecessary to describe them any further. In the aorta depicted in Plate XLVIII. Fig. 3 they projected to a considerable extent into the lumen of the tube.

Such aortæ are not good objects for microscopical examination, because, however careful the decalcification, the calcified part almost invariably falls off. In the calcified part nothing can be seen except a few shreds of muscular tissue lying between oval or round masses of calcified material staining almost black with hæmatoxylin.

In the coats of the aorta, beneath the wholly calcified parts, one sees almost normal muscular fibres, but here and there are small round darkly-staining masses such as have been already described in the posterior peroneal and radial arteries. These are manifestly patches of incipient calcareous degeneration.

An interesting point is that very often the disease seems to pick (see, for instance, Plate XLVIII. Fig. 4) just the point of origin of the smaller arteries.

On examining carefully the inner lining of such an artery one often sees small brownish nodules. These, however, are much more evident in the smaller arteries and will be described more fully hereafter. Indeed, in the larger vessels they are much more easily felt than seen.[1]

In one subclavian artery of the XVIIIth-XXth Dynasty the lumen of the artery near its origin was almost blocked by a ragged calcareous excrescence, depicted in Plate XLVIII. Fig. 5. There can be no doubt that this person narrowly escaped embolism.

2. *Partial calcification and atheroma.*—When an artery like the femoral or brachial is partly or completely calcified, there can be no difficulty in recognising such a lesion. The case is different, however, when the lesions are slight, as they are completely obscured by the colouring matter and the opacity of the tissue.

Good results can be obtained, however, by soaking pieces in glycerine to which a few drops of formol have been added, when, after a few days, the tissues become transparent. In many cases, even before the artery is opened, one sees through the coat (Plate XLVIII. Fig. 8) small dark brown patches, which are then also felt easily. When the artery is opened these patches are seen to protrude into the lumen (Plate XLVIII. Figs. 6 and 7), and sometimes they have a hard white centre (Plate XLVIII. Fig. 6), which is manifestly calcareous to the touch.

In pieces of such an artery, hardened and stained in the usual way, these patches are found to be just under the fenestrated membrane,

[1] In this connection it must not be forgotten that, for some unexplained reason, air bubbles are often present between the middle and inner coat. These cause the inner coat to bulge outwards, causing an appearance as if the aorta were studded with small atheromatous patches. A little pressure at once causes them to flatten out and disappear.

which is easily recognised at one or both edges of the preparation (Plate L. Figs. 11 *b* and 12). The inner membrane of the artery is often intact; sometimes the lesion has evidently broken through it (Plate L. Fig. 12).

The lesion therefore is in the middle coat of the artery, the muscle fibres of which are transformed into dark, deeply-staining strands, which have evidently undergone some very marked degeneration (Plate L. Fig. 11 *d*, *e*, *f*, and Fig. 12).

Very often nothing more can be seen, and there is no sign of emigration of leucocytes in or around the diseased tissue.

In some arteries, however, I have seen round the degenerated patch small, irregular bodies, which may or may not be leucocytes (Plate L. Fig. 13 *a*).

I do not attach much importance to this absence of leucocytes, as I know from experience that leucocytes are hardly ever found in mummies, even in such tissues and lesions where we know that they must have been present in considerable numbers during life. Why this should be the case need not be discussed here.

I have already drawn attention to the fact that some arteries, although not necessarily showing any sign of calcification or other degeneration, feel like whip-cord and are plainly thickened, though they are not atheromatous. I regret that I cannot show any satisfactory microscopical specimens illustrating this fibroid change. When we remember that the thickness of an artery in microscopical sections of the tissues from fresh bodies depends on many conditions, it will be manifest that in mummified bodies, comparison and inferences are practically impossible. Moreover, at present I cannot always distinguish, with certainty, fibrous from unstriated muscular tissues in mummies. I repeat, however, that to the naked eye and to the touch some arteries are distinctly thickened and fibrous.

Discussion of Results.

Nature of the lesions.—There can be no doubt respecting the calcification of the arteries, and that it is of exactly of the same nature as we see at the present day, namely, calcification following on atheroma.

The small patches seen in the arteries are atheromatous, and though the vessels have without doubt been altered by the three thousand years or so which have elapsed since death, nevertheless the lesions are still recognisable by their position and microscopical structure.

The earliest signs of the disease are always seen in or close below the fenestrated membrane,—that is, just in the position where early lesions are seen at the present time. The disease is characterised by a marked degeneration of the muscular coat and of the endothelium. These diseased patches, discrete at first, fuse together later, and finally

form comparatively large areas of degenerated tissue, which may reach the surface and open out into the lumen of the tube. I need not point out how completely this description agrees with that of the same disease as seen at the present time.

I have already mentioned the absence of leucocytes and cellular infiltration, and need not therefore return to it here.

In my opinion, therefore, the old Egyptians suffered as much as we do now from arterial lesions identical with those found in the present time. Moreover, when we consider that few of the arteries examined were quite healthy, it would appear that such lesions were as frequent three thousand years ago as they are to-day.

Etiology.

The etiology of this disease three thousand years ago is as obscure as it is in modern people. One cause which is supposed to play a part in modern times, namely *tobacco*, can certainly be eliminated, as this drug was not used in ancient Egypt.

Syphilis also can be eliminated with considerable certainty, as no pathological specimens of this disease in ancient Egyptians have as yetbeen discovered.

Alcohol played a part in Egyptian social life, in so far that on festive occasions some of the old Egyptians certainly got drunk, as is shown by pictures found in Egyptian tombs. Beer was a common beverage, and wine was not only made in the country but also imported.

It is clear, however, that the Egyptians as a race are not and never have been habitual drunkards.

If I may be allowed a short digression, I would remark in this connection, that my personal experience has led me to call in question the importance of alcohol as a cause of arterial disease. During the Mussulman pilgrimage, I have made over eight hundred post-mortem examinations of people who had certainly never touched alcohol in their lives, and I have found that disease of the arteries is certainly as common, and occurs as early in total abstainers as in people who take alcohol regularly.

Another favourite cause invoked for the production of arterial disease is the supposed increased wear and tear of modern life. This has always appeared to me an extraordinary theory, considering that people, even as late as the beginning of last century, worked far harder and had much greater difficulty in getting their living than in the present day. In my opinion, the theory that the wear and tear of human life has increased is a myth, the fact being that our life is easier and that we work less than did our ancestors.

There is no evidence that old Egyptians worked hard either mentally or physically. Indeed, the time-tables of workmen which

have been discovered show that the Egyptian navvies of ancient times toiled practically the same hours as the Egyptians do now. They enjoyed a holiday every seven days, as do many nations at the present time.

I do not think we can accuse a very heavy meat diet. Meat is and always has been something of a luxury in Egypt, and although on the tables of offerings of old Egyptians haunches of beef, geese, and ducks are prominent, the vegetable offerings are always present in greater number. The diet then as now was mostly a vegetable one, and often very coarse, as is shown by the worn appearance of the crown of the teeth.

Nevertheless I cannot exclude a high meat diet as a cause with certainty, as the mummies examined were mostly those of priests and priestesses of Deir el-Bahari, who, owing to their high position, undoubtedly lived well. I must add, however, that I have seen advanced arterial disease in young modern Egyptians who ate meat very occasionally. In fact, my experience in Egypt and in the East has not strengthened the theory that meat-eating is a cause of arterial disease.

Finally, strenuous muscular exercise can also be excluded as a cause, as there is no evidence that ancient Egyptians were greatly addicted to athletic sport, although we know that they liked watching professional acrobats and dancers. In the case of the priests of Deir el-Bahari, it is very improbable, indeed, that they were in the habit of doing very hard manual work or of taking much muscular exercise.

I cannot therefore at present give any reason why arterial disease should have been so prevalent in ancient Egypt. I think, however, that it is interesting to find that it was common, and that three thousand years ago it represented the same anatomical characters as it does now.

DESCRIPTION OF PLATES XLVIII.-L.

(For particulars see text.)

PLATE XLVIII.

FIG. 1.—Pelvic and arteries of thigh completely calcified (XVIIIth-XXth Dynasty).

FIG. 2.—Completely calcified profunda artery after soaking in glycerine (XXIst Dynasty).

FIG. 3.—Partly calcified aorta (XXVIIth Dynasty).

FIG. 4.—Calcified patches in aorta (XXVIIth Dynasty).

FIG. 5.—Calcified atheromatous ulcer of subclavian artery (XVIIIth-XXth Dynasty).

FIG. 6.—Patch of atheroma in anterior tibial artery (glycerine). The centre of the patch is calcified (XXIst Dynasty).

FIG. 7.—Atheroma of brachial artery (glycerin) (XXIst Dynasty).

FIG. 8.—Unopened ulnar artery, atheromatous patch shining through (glycerine) (XXIst Dynasty).

PLATE XLIX.

FIG. 9.—Section through almost completely calcified posterior peroneal artery (low power). Van Gieson staining. *a*, *a*1, *a*2, Remnants of endothelium and fenestrated membrane. *b*, Calcified patches. Many more are seen.

FIG. 10.—Section through calcified patch of ulnar artery. Same stain. (Leitz, Oc. 1, $\times \frac{1}{12}$.)

a, *d*, Calcified patches.

b, Partially calcified muscular coat.

c, Annular muscular fibre.

PLATE L.

FIG. 11.—Section through atheromatous patch of anterior tibial artery. Same stain. (Leitz, Oc. 1, $\times \frac{1}{12}$.)

a, Remains of endothelium.

b, Fenestrated membrane.

c, Muscular coat.

d, *f*, Membrane coat undergoing degeneration.

e, Completely degenerated remnants of muscular coat.

FIG. 12.—Section through atheromatous patch of ulnar artery. Same stain. (Leitz, Oc. 1, $\times \frac{1}{12}$.) (Reference letters the same as in Fig. 11.)

FIG. 13.—Section at edge of atheromatous patch. Hæmatoxylin stain (Leitz, Oc. 1, $\times \frac{1}{12}$.)

a, Leucocytes (?). The atheromatous part on the left stains intensely dark with hæmatoxylin.

FIG. 1.

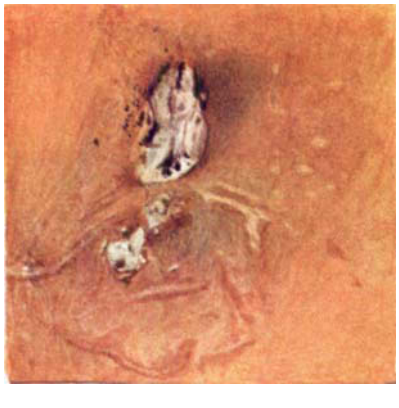


FIG. 3.

FIG. 2.

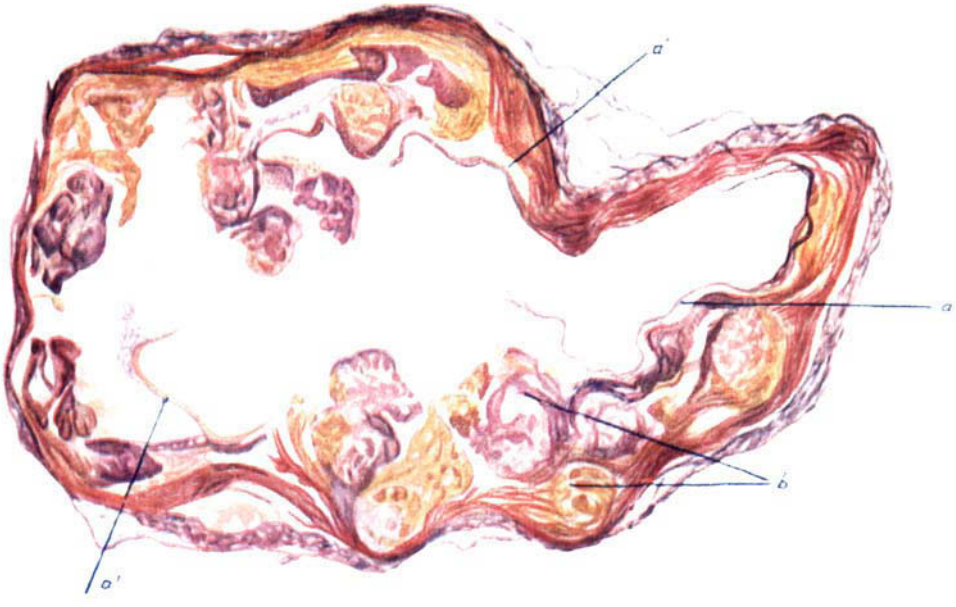
FIG. 4.

FIG. 5.

FIG. 6.

FIG. 7.

FIG. 8.



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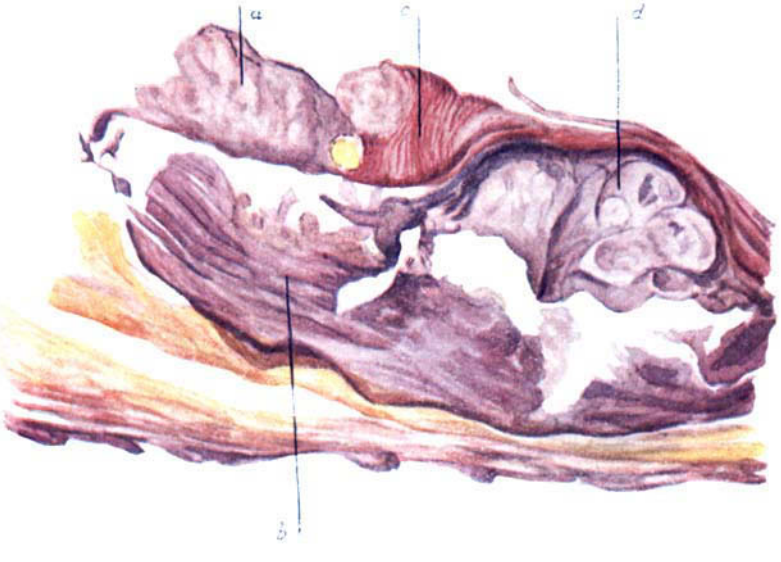

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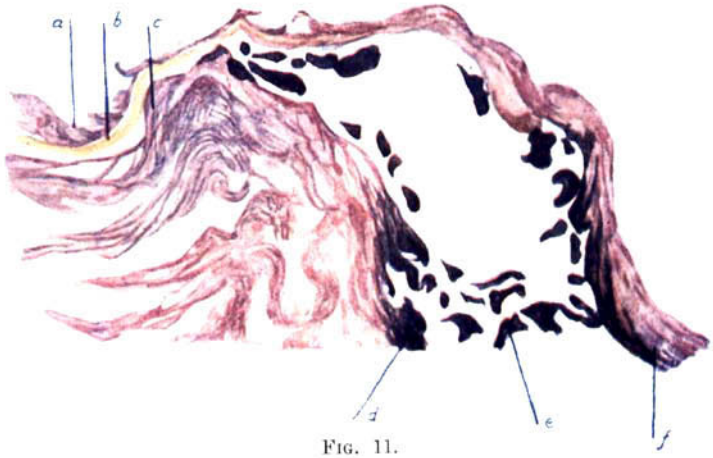

FIG. 11.

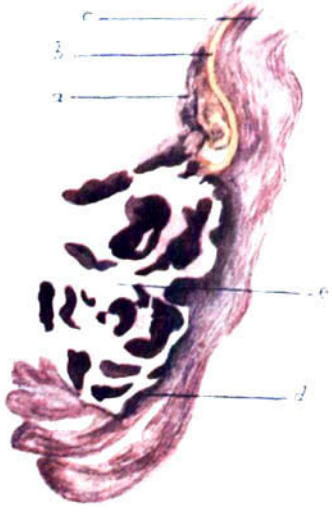

FIG. 12.

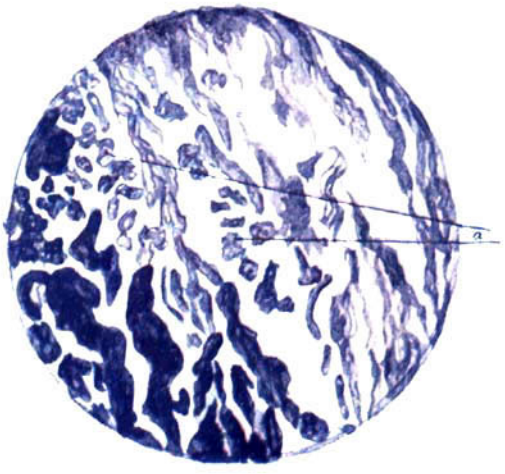

FIG. 13.