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By D'ARCY POWER, M.B. (Oxon.), F.R.C.S., F.S.A.

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SOME POINTS IN THE MINUTE ANATOMY OF INTUSSUSCEPTION.[1]

By D'Arcy Power, M.B. (Oxon.), F.R.C.S., F.S.A., *Hunterian Professor of Pathology and Surgery at the Royal College of Surgeons of England.*

From the Conjoint Laboratories of the Royal Colleges of Physicians (Lond.) and Surgeons (Eng.).

(Plates XXXVII. to XL.)

I cannot but feel that I am courting criticism by attempting to reconsider so well-worn a subject as is that of intussusception. It is an affection which has been known and treated ever since medicine has had a literature, for Hippocrates himself recommended enemata for its relief, and Paraxagoras of Cos is said to have been bold enough to open the belly for its cure. I have before me, however, the noble example of our great master of pathology and surgery, after whom the chair I now occupy is so worthily named. John Hunter did not scorn to employ his time and his great talents upon such subjects as the natural history of the teeth, the inflammation of the internal coats of veins, the treatment of abscesses, and the cure of popliteal aneurism,—subjects which his contemporaries were accustomed to think of as threadbare, but from which his genius and his patience enabled him to evolve far-reaching conclusions which have withstood the test of time, bringing with it methods he could not even imagine. I cannot pretend to imitate him in any single point. It is only possible for me to follow humbly in his footsteps and to bring forward such facts as I have obtained by clinical observation and from pathological research in a subject to which he himself devoted both time and thought.

My clinical observations have been made at the Victoria Hospital for Children, and I am greatly indebted to my colleagues there for the readiness with which they have allowed me to use their cases. The pathological facts have been gleaned from an examination of the specimens preserved in the museums attached to the various medical

[1] The first of a course of lectures on the "Pathology and Surgery of Intussusception," delivered in February 1897 at the Royal College of Surgeons of England.

schools in London and the provinces, whilst my task has been greatly simplified by the liberality with which the Royal College of Surgeons of England and its sister College of Physicians have allowed me to work in the laboratories they maintain with such noble munificence.

Very little[1] is known of the minute anatomical changes in the intestine which take place in strangulated hernia, still less is known about those which occur in the rarer condition of intussusception, yet the Hunterian Museum and the museums attached to the medical school of every large hospital contain plenty of material for examination. The kindness of the conservator of the Hunterian Museum, Professor Stewart, and of the curators of the various museums have afforded me the opportunity to examine such specimens as seemed to be in a good state of preservation. I propose to describe the results I have obtained with such conclusions as it seems lawful to draw from them. Verbal descriptions of unknown objects are wearisome, and convey but little knowledge when it is impossible to study the preparations at leisure. I have therefore obtained photo-micrographs of many of the sections, to render my remarks somewhat more interesting. I may say at the outset that an obvious fallacy attends these observations. The work is new, and it has been carried out by a single observer upon a comparatively small number of specimens, 31 in all. These specimens have not always been preserved with the minute attention to details of a hardening process which marks the histology of our own time. Pathological histology is of such recent growth that the older surgeons were rarely anything more than morbid anatomists. Spirit, however, was cheap and strong at a time when its retailers offered their customers to be "drunk for a penny, dead drunk for twopence, and straw for nothing" upon which to sleep off the inexpensive debauch. Some of the specimens preserved in these long by-gone days are therefore better fitted for microscopical examination than are those obtained more recently. I cannot hope to have made more than a beginning, and I do not doubt that I have often fallen into grievous error in my interpretations, but others with better material will soon correct my mistakes, and will advance more quickly along the paths I indicate.

In Plate XXXVII. Fig. 1 are seen the earliest changes in an intussuscepted intestine which I have been able to obtain. The sections were taken from the intestine of a child 8 months old[2] who had suffered for 13½ hours from the symptoms of an intussusception. I opened the abdomen and reduced the invaginated gut with such success that the baby digested his food and passed a perfectly natural motion. Symptoms of fresh obstruction appeared 45 hours after the operation, and the child died 6 days after the abdominal section, and 7 days after the onset of the illness. The post-mortem examination showed

[1] Nicaise, "Des lesions de l'intestin dans les hernies," Paris, 1866.

[2] *Brit. Med. Journ.*, London, 1895, vol. ii. p. 1356.

that there was neither peritonitis nor intussusception. The small intestines were distended with flatus, but the distension ceased suddenly, and without any visible cause, 12 in. from the ileo-cæcal valve. The colon, like the lower part of the ileum, was collapsed.

The collapsed intestine was at once hardened in Foà's solution (Müller's fluid saturated with corrosive sublimate), the process being completed by alcohol. Sections taken through the colon three-quarters of an inch from the ileo-cæcal valve show (Fig. 1) that the stress of the disease has fallen upon the submucous coat. In the mucous coat the crypts of Lieberkühn are either single or double. Each is lined with a layer of columnar epithelium which in some parts is normal, but in others contains an increased number of goblet cells, showing that the glands have been subjected to irritation. The increase in the number of goblet cells accounts for the excessive secretion of mucus, which is often so marked a symptom of intussusception. Each crypt of Lieberkühn, as is usual in children, has its own sheath of adenoid tissue separating it from its neighbour upon either side. This tissue is a little swollen, for it is rather more distinct than it should be in a healthy intestine, whilst the masses of lymphadenoid tissue forming the solitary follicles are greatly increased in size (Plate XXXVII. Fig. 2).

The muscularis mucosæ forms a thick, continuous, and well-defined layer, which in the hardened section is depressed at the solitary glands. Its cells are unaltered. The submucous tissue is abundant in quantity, and the presence of an increased number of small round cells shows that it is slightly inflamed. It contains numerous large and rather thick-walled arteries, but its veins are neither large nor conspicuous by their congestion. The meshes of its connective tissue are widely open and are filled with an extravasation of blood, which, though considerable in quantity, has not yet exerted any great pressure upon the walls of the vessels, for even the capillaries are patent and contain blood corpuscles. The extravasation of blood is not uniform, and the mucous membrane with its submucous tissue is thrown into ridges which run transversely to the axis of the intestine, the ridges being due to the thickening caused by the extravasation. The effusion is most abundant just above the layer of circular muscle, and it fades away (Plate XXXVII. Fig. 3) towards the muscularis mucosæ, though it is easy to see how it might have tracked through this layer and along the inflamed and softened adenoid tissue lying between the crypts of Lieberkühn, until it had made its escape into the lumen of the intestine.

The circular layer of muscle has not undergone any visible change. It is separated from the longitudinal band of fibres by a large lymphatic vessel. This vessel and the plexus myentericus are normal histologically. The longitudinal muscle is very œdematous, for the fibres are separated one from another by stellate spaces, but

the individual fibres have undergone but little if any change. The serous coat is separated from the muscular layer by many large venous channels, but for the most part it is healthy, and it only contains patches of extravasated blood at long intervals.

The ileo-cæcal valve has undergone changes in every respect similar to those described as occurring in the colon. The general structure is thickened and an extravasation of blood has taken place into its centre, whilst its lymphatics are blocked with leucocytes, showing how easily an inflammation might begin in the valve. The blood vessels, however, are not enlarged, and the epithelium lining its crypts of Lieberkühn do not contain an undue proportion of goblet cells.

The changes in the ileum are similar to those which have taken place in the large intestine. The villi, however, are reduced to mere tags of connective tissue. The submucous layer is thickened by an extensive extravasation of blood, which has not yet undergone any change. The circular layer of muscle is comparatively healthy, though blood has been effused into its substance, but the longitudinal layer of muscle is completely disorganised. Its fibres are widely separated from each other by the effused corpuscles, and the protoplasm of the muscle cells is becoming degenerate. The serous coat is also greatly thickened by the effused blood.

The next section (Plate XXXVII. Fig. 4) is taken from an intussusception, which was made experimentally in a cat by drawing the colon over the cæcum and the lower part of the ileum, and securing it with a few point sutures at the neck of the invagination. The specimen was obtained by resecting the invaginated intestine a week afterwards. The animal made an excellent recovery and is still alive.

The minute anatomical changes are again more marked in the mucous and submucous layers than in the muscular and serous coats, but this specimen differs from the previous one in the fact that the mucous membrane has suffered more severely than the submucous tissue.

The villi covering the mucous membrane are much congested (Plate XXXVII. Fig. 5), and the blood corpuscles are escaping from them in many parts into the lumen of the intestine. The diapedesis of white corpuscles is only just beginning, and as there has not been any great multiplication of cells, it may be assumed that the inflammatory process has hardly advanced beyond the stage of congestion and stasis. The apices of many of the villi are often curiously invaginated, a change which is due, in all probability, to unequal shrinkage during the hardening process, though I do not remember to have seen it in any of the numerous sections I have examined when teaching classes of normal histology. The columnar epithelium of the villi is converted almost wholly into goblet cells. The crypts of Lieberkühn are quite healthy, their epithelium is normal, and they are not

surrounded by any inflammatory zone of small round cells. The muscularis mucosæ is unaltered, and both its layers of muscular fibres are clearly seen. The submucous coat is swollen; its blood vessels—large and small—are filled with normal blood corpuscles, and its lymphatic channels are enormous. The swelling of the submucous coat is chiefly due to œdema, for it is not associated with any great infiltration of small round cells, nor has there been much extravasation of blood into the meshes of its connective tissue. The circular layer of muscle is greatly congested, and its individual fibres are separated one from another. Blood has been extravasated into it to a slight extent, but the œdema is much less marked than it was in the previous case. The longitudinal layer of muscle is scarcely altered, and the serous coat is continuous with the slightly congested mesentery.

The section from the experimental invagination in a cat may be compared very profitably with one[1] (Plate XXVIII. Fig. 6) taken from the ileum of a dog killed by an intussusception which occurred spontaneously. An extensive extravasation of blood has done serious injury to the mucous membrane by surrounding and compressing the glands and the villi. It has separated the fibres of the muscularis mucosæ one from another, and it has caused great thickening of the submucous tissue. The circular layer of muscle is profoundly affected (Plate XXXVIII. Fig. 7). Its blood supply is greatly increased, and its lymphatics are so much enlarged that the muscle has become œdematous. The œdema, in turn, has reacted upon the muscle fibres, so that hardly one in ten is healthy (Plate XXXVIII. Fig. 8), the remainder having undergone an inflammatory change which has led to a loss of all structural details in the transverse section of its fibres. The longitudinal layer of muscle (Plate XXXVIII. Fig. 7) is hardly altered. The structure and outlines of the individual fibres are distinct, but there has been a slight extravasation of blood continuous with an extravasation which has occurred between the longitudinal layer of muscle and the serous coat of the intestine.

The changes shown by these specimens form an interesting series, and are typical of many others which I have examined. They show that the mucous membrane, the submucous tissue, and the circular layer of muscle suffer more severely in the early stages of an intussusception than the longitudinal layer of the muscle or the serous coat.

The changes in the submucous coat are the most extensive, but those occurring in the circular layer of muscle are the most important, for they militate against the recovery of the patient even after the intussusception has been reduced.

The series of changes taking place in a case of simple intestinal invagination appears to be as follows:—The vessels in the mucous

[1] St. Barth. Hosp. Museum, No. 2192.

and submucous layers become congested in consequence of the obstruction to the free circulation of blood which is caused by the bending of the intestine upon itself at the apex, and again at the neck of the intussusception. The congestion affects the veins and the capillaries more than the arteries, and after a varying length of time it is followed by an escape of blood from the congested vessels. This extravasation of blood is due to two causes, each acting at a different period in the course of the affection. The first extravasation is due to simple over-distension of the capillaries, resulting from the venous congestion. This over-distension makes them yield, for their endothelial walls are only surrounded by a delicate layer of connective tissue which affords but a minimum of support even in health, whilst in cases of intussusception it is so softened by œdema as to be still less capable of preventing or counteracting any undue dilatation of the vessels. In the most acute cases, however, where the pressure at the neck of the intussusception has been so severe as to cut off the blood supply, the capillaries may be empty. No bleeding occurs in these cases, and the surgeon is sometimes misled as to the nature of the affection he is called upon to treat. If he recognises it, however, opens the abdomen at once and reduces the intussusception, bleeding may afterwards occur. The capillaries, in these cases, having been empty for some time have lost what, for want of a better term, must be called their "vitality," and when the blood passes through them again it either escapes, or so much plasma exudes as to cause a fresh œdema, which may be sufficient to paralyse the bowel and kill the patient. Paralysis of the bowel, from hæmorrhage, is rare, and it is more often due to œdema of the circular muscle, for small extravasations of blood may be absorbed in simple cases, leaving the wall of the intestine thickened, but still capable of doing its duty.

Some of the later stages in an intussusception are seen in a section (Plate XXXVIII. Fig. 9) taken from[1] the cæcum of a child, æt. 9 months, who died 60 hours after the first symptoms of intestinal obstruction had been recognised. The specimen is of great interest. It was shown by John Hunter on the 18th August 1789 at the Society for the Improvement of Medical and Chirurgical Knowledge, one of the most select medical societies which has existed, for it consisted of twelve members who met once a month at Slaughter's Coffee-House in St. Martin's Lane. Persons to be eligible for election were required to attend regularly, and to be physicians or surgeons of five years' standing on the staff of St. Bartholomew's, St. George's, Guy's, or St. Thomas's Hospitals. The society existed from 1783 to 1818, and during this time it admitted only eighteen members. John Hunter read some of his best papers at its meetings, and it is a proof of the importance he attached to this specimen, that he brought it forward at this society rather than at any of the others to which he belonged.

[1] Hunterian Museum, No. 2703.

The specimen formed the text upon which he wrote his celebrated paper, "On Introsusception," and thanks to the interest which it excited in the mind of our master as well as in that of his unworthy assistant (Sir) Everard Home, it has been so well preserved from the time it was removed from the child's body that I have no difficulty in describing its histological characters, though it is now 108 years since the patient died.

The mucous membrane is very slightly if at all thickened, but it is covered in parts with a layer of coagulated blood. The muscularis mucosæ is normal, and beneath it is a considerable quantity of adenoid tissue. The whole of the submucous layer is thickened, partly by œdema, partly by the extravasation of blood, and partly by the infiltration of inflammatory products. The veins in the submucous layer are congested, and there are many huge capillaries from which a considerable diapedesis of white corpuscles has taken place. The circular and longitudinal layers of muscle are œdematous, but the circular layer is the more affected, and the meso-cæcum is thickened by threads of fibrin. It contains a lymphatic gland surrounded by healthy fat. The gland, seen in section, is itself inflamed, for its vessels are congested.

The later stages of intussusception are also seen in a section [1] (Plate XXXVIII. Fig. 10) taken through the ileum of a child, æt. 5 months, who had suffered for three weeks from intestinal disturbance, which had culminated in symptoms of acute intussusception six days before her death. The case is carefully recorded by Dr. Howship,[2] who says that gentle aperients were repeatedly given, and that there was great frequency in the passing of blood. The mucous membrane is congested, and much blood has been poured out into its substance. The submucous tissue is greatly thickened and intensely congested. The thickening is due to an extravasation of blood, and to an infiltration of inflammatory cells, for the submucous tissue is in a state of diffuse suppuration. The circular and longitudinal layers of muscle are congested and œdematous, whilst many of their fibres are degenerating. The serous layer is thickened, congested, and suppurating.

This section (Plate XXXIX. Fig. 11) shows a still later stage than the one just described. It was taken [3] from a child, æt. 8 months, who had been ill for 10 days, during the last three of which it suffered from the symptoms of acute intussusception. Every part of the invaginated intestine is seriously injured, though the mucous membrane has undergone somewhat less change than the other tissues. The crypts of Lieberkühn with their epithelial cells are quite distinct, and, with the exception of some conversion into goblet cells, their lining

[1] Mus. Roy. Coll. Surg. Eng., No. 2711.
[2] *Edin. Med. Journ.*, 1812, vol. viii. p. 129.
[3] Mus. Roy. Coll. Surg. Eng., No. 2710.

epithelium has undergone but little change. The connective tissue between the crypts is intensely congested, and in some parts the walls of the vessels have given way, so that blood has been extravasated into the lumen of the intestine. The muscularis mucosæ is so infiltrated with inflammatory products, that in many places it is no longer present as a uniform layer. The submucous and muscular coats are enormously congested, and are in a state of diffuse suppuration, for they are greatly thickened by a large number of small round cells mixed with extravasated red blood corpuscles. At the part where the section is made, the serous coat of the intussusception has formed dense adhesions to the serous coat of the middle layer of the invaginated intestine, whose submucous coat is itself thickened by an extravasation of blood.

The changes which take place in the invaginated intestine of adults are in some points identical with those which occur in the more delicate tissues of children, but in others they are widely different. These sections (Plate XXXIX. Fig. 12) were taken from the ileum of a married woman, æt. 30,[1] who was seized with a sudden abdominal pain on 21st November. Her abdomen was opened on 25th November, and the small intestine was found to be invaginated and irreducible for 5 in. at a point situated 2 ft. above the ileo-cæcal valve. The intestine was cut away, and with it a small fibrous polypus undergoing calcareous degeneration, which was attached to the mucous and muscular coats of the ileum just above the beginning of the intussusception. The patient died of peritonitis on the day after the operation. The histological changes in the intussuscepted intestine[2] are very great, and must have been in progress for some time, though the actual duration of the symptoms was short. The villi (Plate XXXIX. Fig. 13) are mere shrivelled tags of connective tissue, from which the columnar epithelium has disappeared, whilst (Fig. 12) the adenoid tissue, the involuntary muscle, and the central lymphatic have been replaced by small round cells, the products of the chronic inflammatory change which has affected the walls of the intestine. Congested vessels are visible here and there, and in many places blood is extravasated into the connective tissue. The submucous layer, too, is full of extravasated blood, which has pushed aside all the other structures except the greatly dilated veins filled with blood corpuscles which still remain conspicuous objects in the section. The pigmented granules surrounding the margin of each blood vessel, show the chronic nature of the irritation, for the granules consist of altered blood pigment derived from the disintegration of red blood corpuscles. The extravasation of blood has involved the muscular coat of the intestine, so that the individual fibres of the circular and longitudinal layer of muscle are

[1] *Trans. Path. Soc. London*, 1892, vol. xliii. p. 74.
[2] St. Barth. Hosp. Mus., No. 2191 (*a*).

separated from each other by the red blood corpuscles which are lying free between them. The serous coat of the intestine also contains a few blood corpuscles in the meshes of its connective tissue.

The Museum of the Royal College of Surgeons of England contains (No. 2704) an interesting preparation presented by Sir Stephen Hammick. It was taken from the body of a sailor, æt. 27, who had suffered from symptoms of chronic intestinal obstruction for six months before his death. A tumour was felt to the right of his navel for two months before he died of emaciation. The post-mortem examination of the body proved that this tumour was an intussusception of the ileum, cæcum, and colon, into the lower part of the large intestine. Microscopic examination of the ileum from this specimen shows that the submucous tissue beneath the muscularis mucosæ is congested, but that the chief change is a conversion of the submucous and muscular coats of the intestine into dense fibroid tissue, containing little, if any, cellular infiltration. The villi upon the mucous membrane are greatly congested, but they do not seem to have undergone the mummifying changes seen in Plate XXXIX. Fig. 13.

These two cases are examples of chronic intussusception, which end either in the death of the patient (as in the last case from exhaustion), or in exfoliation of the invaginated blood. Recovery sometimes occurs after exfoliation, but in about 40 per cent. of these cases death results from the secondary accidents which attend the process of repair when the separated gut has been discharged. Such a separation of the bowel is more usual in adults than in children, but the Museum of the Middlesex Hospital contains (No. 1537) 5 in. of ileum cast off from the intestine of a child, æt. 7 months. The gut is turned inside out.

Another specimen of exfoliated bowel was presented to the Museum of the Royal College of Surgeons (No. 2715) by Mr. Hacon of Hackney, who has very courteously given me details additional to those which he furnished to Dr. Peacock when he showed the preparation at the Pathological Society of London in 1864.[1] It consists of a piece of bowel measuring 40 in. in length, with a polypus attached to its upper end. The piece of bowel was passed by a lady, æt. 32, who was suddenly attacked with vomiting and abdominal pain on 28th December 1863. The symptoms recurred at intervals for two or three weeks, and at the end of this time she had complete intestinal obstruction, lasting for two or three days. The bowels then acted regularly, and without the passage of blood. The piece of intestine was passed by the anus, enveloped in fæcal matter and without any blood, upon the eighteenth day after the supervention of the severe obstruction, and 15 days after the resumption by the bowel of its normal habtt. The patient died unmarried, 30th July 1889, from

[1] *Trans. Path. Soc. London*, vol. xv. p. 113.

some lung trouble. She became very thin before her death, and at times was subject to constipation, but she never again suffered from obstruction of the bowels. No post-mortem examination of the body was made. The specimen consists of the whole thickness of the intestinal wall, for portions of the mesentery are still attached to it, and the mucous membrane is seen covering its inner side. The microscopic sections (Plate XXXIX. Fig. 14) show that all the coats of the intestine have undergone sclerosis. The line of the mucous membrane is distinct, though none of the details of its structure can be seen, but the submucous coat is indistinguishable from the muscular, and the muscular layers from each other, or from the serous coat. The whole thickness of the section consists of dense fibroid tissue, like that found in an old scar. This tissue is a little more cellular, and rather more vascular just beneath the mucous membrane, and along its outer border; but otherwise it is a dense hyperplasia of the connective tissue, containing a few large vessels which appear black in the section, for they are filled with stained celloidin.

The Museum of Guy's Hospital[1] contains a specimen of equal interest to that just described. It consists of a piece of intestine exfoliated from a child, æt. 1 year, who recovered from an intussusception. The piece of intestine measures 12 in. in length, of which 1 in. is small intestine, the remainder being cæcum and ascending colon. It forms a single tube, which is turned inside out in such a manner that the vermiform appendix lying within its lumen opens directly upon its outer or mucous surface. The microscopical sections which I have examined show that the walls consist of fibrous tissue.

It is not necessary, however, that the intestine should undergo a process of sclerosis before it can be cast off with safety to the patient. The very next specimen at Guy's Hospital[2] consists of 18 in. of small intestine, passed by a woman, æt. 33, 12 days after she had been seized with symptoms of intussusception. The patient afterwards recovered. The bowel has not been turned inside out, for the mesentery is still visible upon its outer side. Microscopic sections show that the exfoliated piece of bowel contains each layer of a normal intestine, but merely as a skeleton of digested fibrous tissue or reticulin, for everything has disappeared, except the connective tissue, some nuclei, and the débris of a few cells. All the coats of the bowel have been intensely congested; but the muscles, the blood vessels, and the cells have been completely removed.

A similar specimen is in the Cambridge Museum, and Dr. Joseph Griffiths has kindly allowed me to take a section from it. It is a portion of the whole circumference of the bowel—measuring 26 in. in length—which was passed by a governess between 20 and 30

[1] Guy's Hosp. Mus., No. 1105.
[2] *Ibid.*, No. 1106.

years of age, who is still alive. She had suffered for some time from an obscure intestinal affection. The bowel, as in the preceding case, was passed without being turned inside out, for the mesentery is seen on the outside of the specimen. The sections show that every layer of the intestine has been intensely congested, but that only its connective tissue skeleton of reticulin remains.

Almost identical appearances are seen in a section (Plate XL. Fig. 15) taken from a piece of intestine [1] passed by a woman, æt. 48, who had been seized with signs of internal strangulation 17 days previously. The symptoms of strangulation continued for 6 days and then abated, but the piece of intestine was not passed until 11 days after the improvement began to show itself. There were fæcal evacuations after the intestine had been passed, but the patient died exhausted 10 days later. A section of the intestine examined under the microscope shows that the villi are mere ghosts of their former selves, that they have been intensely congested, and that their epithelial covering has completely disappeared. All the crypts of Lieberkühn have vanished, but there are still traces of solitary follicles, though their former position is very often only indicated by empty spaces. The submucous tissue is moderately thickened, but its cellular elements have disappeared so completely that it also is reduced to a skeleton of reticulin riddled with holes, marking the position of the congested blood vessels. The muscular fibres have entirely disappeared, and the outer margin of the intestine is represented by an irregular border of connective tissue.

The changes which have taken place in these three specimens are identical. All the elements of the intestinal wall have disappeared, with the exception of the connective tissue. The connective tissue has itself become converted into reticulin, a substance which can be obtained artificially by digesting it with trypsin—the pancreatic proteolytic ferment—or with papain. I have, therefore, no hesitation in saying that these pieces of intestine have undergone digestion within the body before they were eliminated, and that the changes which they show are not due to any process of maceration.

The ileum and a portion of the cæcum have also been preserved [2] from the last case, so that it is possible to examine sections taken through the scar left after the separation of the intestine. The sections show that the mucous membrane is greatly congested, and is undergoing a series of changes similar to those seen in Plate XL. Fig. 15, whilst the submucous layer is infiltrated with small round cells. The muscular layers are in a state of advanced degeneration, but the adhesion has taken place by the serous surfaces, which have become converted into layers of such well-formed connective tissue that it is no longer possible to say where they blend.

[1] St. Barth. Hosp. Mus., No. 2189.
[2] St. Barth. Hosp. Mus., No. 2190.

Similar changes have taken place in another piece of intestine[1] obtained from the body of a man who had voided a part of his bowel many years before his death. The canal of the colon at the point where the section was made only measured half an inch in diameter. The mucous membrane is continuous and is everywhere provided with crypts of Lieberkühn, and healthy solitary glands are present. The muscularis mucosæ has disappeared, and the submucous layer is greatly thickened. It consists of dense scar tissue, free from cells and not very vascular at the part nearest the mucous membrane; more vascular, and containing many inflammatory cells in the part lying just above the circular muscle. The circular muscle itself is still inflamed, for it is invaded by small round cells. It is greatly thinned in parts, and long bundles of connective tissue run through it to join the submucous with the serous coat. The longitudinal layer of muscle, like the circular coat, is interrupted by strands of fibrous tissue, whilst the serous coat is unduly vascular, though it is not greatly thickened.

These specimens show that the intestine may undergo at least two kinds of pathological change before it is passed from the bowel. The one a process of sclerosis of its connective tissue, which is probably associated with the long-continued irritation of such a growth as a polypus; the other, a process of tryptic digestion, which leads to the disappearance of everything but the connective tissue framework of the bowel. This is probably associated with the more acute forms of inflammation. The actual process of repair after a piece of bowel has been cast off stands in need of further elucidation, for I have only examined two specimens, and neither of them was in a sufficiently good state of preservation to give a series of satisfactory sections. It is evident, however, that the submucous tissue takes a large share in the reparative process, and the inflamed condition of the connective tissue, even after a long period of convalescence, gives clear evidence that a small amount of additional irritation would be sufficient to light up a fresh and acute inflammation of the bowel. We are, therefore, warned of the prolonged care which is needed to bring these cases to a successful issue.

All the sections which have been described so far have been taken from cases of intussusception, running the ordinary course. I propose now to deal with some of the less common forms.

The specimen from which the following sections are taken was from a case of double intussusception, which was shown some years ago at a meeting of the Pathological Society of London.[2] It was sent to me by Dr. Emmerson of Biggleswade, and it shows an intussusception of the ordinary ileo-colic type, with a second intussusception situated in the colon 3 in. nearer to the anus than the first one.[3] The second

[1] Mus. Roy. Coll. Surg. Eng., No. 2717.
[2] *Trans. Path. Soc. London*, 1886, vol xxxvii. p. 240.
[3] St. Barth. Hosp. Mus., No. 2181, *a*.

intussusception is retrograde; that is to say, it is an invagination of the more distal into the proximal portion of the colon. Both intussusceptions have a layer of lymph over the invaginated portions of the intestine. Plate XL. Fig. 16 is from a section of the first or ileo-colic invagination. It presents all the characters of an ordinary acute intussusception. Blood has been extravasated into the connective tissue between the crypts of Lieberkühn, which have thus been more widely separated from each other than usual. The submucous coat is also greatly thickened by congestion and by the extravasation of blood into its connective tissue, but the muscular coats are not greatly affected, nor is the mesentery inflamed. The greatest effusion of blood has taken place near the muscularis mucosæ, for the deeper portions of the submucous tissue and the muscular coats are merely congested and œdematous. The longitudinal layer of muscle is much less affected than the circular layer, as is usual in intussusceptions.

Plate XL. Fig. 17 represents a section taken from the second or retrograde intussusception. The relative thickness of the mucous, submucous, and muscular layers is unaltered, but the serous coat is congested and the mesentery is inflamed. It is, therefore, fair to assume that the retrograde invagination was formed later than the ileo-colic intussusception. It seems to have been produced when the child was so exhausted that the wall of its alimentary canal hardly responded to the effects of pressure, yet the patient was not absolutely moribund, for there is a slight congestion of the blood vessels in the submucous tissue, and the vessels in the serous coat and in the mesentery are greatly enlarged.

The patient was a boy, æt. 5 months, who was so suddenly attacked with illness five days before his death, that he was thought to have had a "fit." He suffered from the usual symptoms of intussusception, and on the day following the attack his motions consisted of "blood and slime." No abdominal tumour was felt until the third day of his illness. Injections and inflation failed to relieve him; but his parents refused to allow an operation for his cure.

Intussusception in children is nearly always spontaneous, and post-mortem examination rarely reveals any definite cause for its occurrence. A specimen[1] in the Museum of St. George's Hospital, however, is an exception to the rule. It is an ileo-cæcal invagination, which caused the death of a child, æt. 5 months. The cæcum contains three growths, each about the size of a split almond. Microscopical examination of one of the growths shows that it is a small, round-celled sarcoma, growing from the submucous tissue immediately beneath the muscularis mucosæ. The stress of the invagination has fallen upon the serous coat and the mesentery, which are greatly thickened by a plastic exudation, whilst the muscular and submucous layers are healthy. When an intussusception occurs in an adult, on the other hand, a

[1] St. George's Hosp. Mus., Ser. 9, No. 80, *i*.

determining cause can nearly always be found. It may be physiological, for the invagination may follow upon a direct injury to the abdomen, and in some cases it is as truly spontaneous as it is in child life, but far more frequently the cause is some form of new growth, either innocent, like a polypus or a papilloma, or malignant, like a cancer, less often a sarcoma.[1] Vierhoff,[2] and more recently, Dr. Sutherland, have seen intussusceptions begin during an attack of purpura; and in such a disease the mechanism of its production is easy to understand in the light of the histological details which I have just demonstrated. Polypi and carcinomata are the usual growths associated with intussusceptions in adults; polypi when the intussusception occurs in the small intestine, carcinoma when it is found in the large intestine, though Mr. Bryant has lately called attention[3] to papillomata causing intussusception of the large intestine in old people.

Many of the spontaneous intussusceptions occurring in adults are found to be in the small intestine, at any rate as the specimens are seen in the various museums; but I cannot help thinking that this is because they are preserved on account of their rarity, until, when they are all seen together, they form such a goodly array as to give a false idea of their relative frequency, for in reality they are very rare. Polypi are a much more frequent cause of adult intussusceptions, and of these I have found two unusual cases, the one rare in virtue of its position, the other on account of its course.

The first is a specimen preserved in the Museum of Guy's Hospital (No. 1091). It is an intussusception measuring 18 in. in length, and situated at the top of the jejunum. It contains a polypoid growth at the apex of the intussusception, and other polypi are present in the duodenum. Dr. Shaw, the curator of the museum, has kindly allowed me to make a microscopic examination of one of the polypi, and I find that it is of a glandular nature. The specimen was obtained from the body of a girl, æt. 19, who had suffered for a year and a half before her death from periodical attacks of vomiting and abdominal pain, associated with progressive wasting. She was decidedly benefited by enemata of sulphate of magnesia, as obstinate constipation was a prominent symptom throughout her illness. Dr. Goodhart,[4] who examined her shortly before she died, found that she had a dilated stomach, with a tumour in the left flank, extending obliquely towards the pubes. This tumour underwent slow rhythmic alterations, being alternately hard and well defined, and soft and ill defined. At the autopsy the coats of the intussuscepted intestine were found to be thickened but not obviously inflamed. There were two other invaginations in the small intestine, but both were apparently of quite recent origin.

[1] The Middlesex Hosp. Museum, No. 1534, *Trans. Path. Soc. London*, 1867, vol. xviii. p. 114.

[2] *St. Petersb. med. Wchnschr.*, 1893, s. 320.

[3] *Med.-Chir. Trans.*, London, 1894, vol. lxxvii. p. 169.

[4] *Trans. Clin. Soc. London*, 1886, vol. xix. p. 146.

The second specimen appears to be most rare. It was presented to the Royal College of Surgeons of England by John Taunton, the founder of the City of London Truss Society, a most enthusiastic morbid anatomist, and the last of the great dispensary doctors in London. It is described in the catalogue of the museum (No. 2719) as a "part of the ileum, in which is an intussusception about 6 in. in length. At the lower end of the intussuscepted intestine there is a firm tumour, of oval form, 2½ in. in length, and 1 in. in diameter, attached to the mucous membrane, and projecting into the canal of the ensheathing part of the intestine. . . . The coats of all parts of the intestine are thickened." The patient, æt. 45, had been ill for 3 months before her death, complaining of extreme pain in the left side and back, as if something within the abdomen were pressing the ribs and spine outwards. She had also occasional nausea and vomiting, with constipation and a sensation of heat in her abdomen. She was variously treated, but without advantage, and gradually sank; vomiting of a fæcal character supervened shortly before her death. All the organs, with the exception of the part preserved, were found to be healthy. Microscopical examination of the tumour shows that in spite of its age—John Taunton died 5th March 1821—it is in an excellent state of preservation. The thickening of the coats of the bowel is due to a new growth—apparently a sarcoma—which has begun to grow in the serous coat, and has extended into the longitudinal and circular layers of muscle, completely disorganising them. The submucous tissue is greatly thickened, the result of a chronic inflammation, but it is not invaded by the new growth. The muscularis mucosæ forms a continuous layer, except at the points where it is penetrated by large blood vessels passing into the mucous membrane. These vessels, as well as those in the submucous tissue, contain blood corpuscles, aggregated by post-mortem clotting. In the mucous membrane the crypts of Lieberkühn are healthy, and present no unusual appearances. The villi are large and well defined. They have lost their coats of columnar epithelium, but they contain (Plate XL. Fig. 18) numerous oval and circular bodies, of whose appearance I am unable to give quite a satisfactory explanation. I thought at first that they were the results of parasitic infection, and were either psorosperms or amœbæ, but more careful examination leads me to believe that they are thrombosed vessels cut in various directions.

Intussusception of the colon, associated with cancer of the large intestine, runs a very chronic course, and may be present without giving rise to any symptoms except those which may fairly be attributed to the cancerous infiltration. In a specimen,[1] from a case where such a condition was not in the least suspected during life, and the cause of the invagination remained obscure until a section was made for the purposes of these lectures, the preparation consists of parts of the

[1] St. Barth. Hosp. Mus., No. 2188, *b*.

descending colon and sigmoid flexure, which have been invaginated into the rectum. It was taken from a woman, æt. 40, who had suffered for the last two years of her life from attacks of obstruction of the bowels, sometimes accompanied by fæcal vomiting. She ultimately died of gangrene of the foot and leg. A microscopical examination showed that the intussusception was associated with an adenoid cancer of the rectum. The mechanism of such an invagination appears easy to explain. It seems to be of the paralytic variety, for the infiltrated portion of the intestine would be less capable of peristaltic contraction than the healthy gut. It would therefore be more liable to be engulfed by the distal part of the alimentary canal, a liability which would be increased by the constant forcing movements of the proximal part of the intestine which attend so chronic a form of obstruction, while the distal end of the bowel became invaginated in a similar manner, forming a retrograde intussusception. The apex of the invagination in each of these cases protruded through the anal. omit

Such, then, are some of the minute anatomical changes which occur in the intestine as a result of intussusception. It is clear that any part of the intestinal wall may be affected. One portion usually suffers more than the others, and the stress of the affection falls most often upon the submucous tissue and upon the circular layer of muscle. The mucous membrane, too, may be seriously injured, but the longitudinal layer of muscle and the serous coat are the least often affected. The earliest histological changes are correlated with an effusion of blood, but the amount of the extravasation varies greatly, at one time so slight as hardly to displace the tissues, at another so considerable as utterly to destroy them. The seat of the extravasation, too, varies. It may be in the mucous membrane, and it seems to me that this occurs in the most acute cases; it is usually in the submucous coat, though it may be in the muscular layers or in the serous coat. The extravasation is followed by inflammatory changes, in which the submucous tissue and the circular layer of muscle are chiefly involved. These changes terminate in hyperplasia of the connective tissue, leading to sclerosis; in a tryptic digestion, leading to the disappearance of every element in the wall of the bowel, and the conversion of its connective tissue into reticulin; in diffuse suppuration or in sloughing of the inflamed bowel, which is then separated and cast off by the ordinary process of ulceration.

DESCRIPTION OF PLATES XXXVII. TO XL.

PLATE XXXVII.

FIG. 1.—Section through a piece of colon which had been invaginated in a child. The submucous tissue is thickened by an extravasation of blood, whilst the blood vessels and lymphatics supplying the muscle are enlarged. Leitz, Oc. 1, Obj. 4.

Fig. 2.—Section through a piece of colon which had been invaginated in a child, showing the thickened submucous tissue, the disarrangement of the muscularis mucosæ, and the enlargement of a solitary follicle.

Fig. 3.—Section through a piece of colon which has been invaginated. The extravasated blood corpuscles are seen to lie in rows in the deeper parts of the submucous tissue.

Fig. 4.—A portion of the ileum from a case of experimental invagination, produced in a cat 7 days previously. The villi are greatly congested. The submucous coat is congested and thickened, and its lymphatic vessels and veins are enlarged.

Fig. 5.—Two of the villi from the previous section, more highly magnified, to show the congestion of their blood vessels and the invagination of their apices. Most of the columnar epithelium has been converted into goblet cells.

Plate XXXVIII.

Fig. 6.—Section through the ileum of a dog which died of intussusception. The details of the structure of the mucous membrane are destroyed by an extensive extravasation of blood. The submucous tissue is greatly thickened by effused blood, and its blood vessels are engorged. The circular layer of muscle is very œdematous and congested, but the longitudinal layer is unaffected.

Fig. 7.—Section through the muscular layer of the intestine, from the same case as the preceding specimen. The circular muscle is œdematous and degenerate, whilst the longitudinal coat is healthy.

Fig. 8.—Section through the circular muscle from the same specimen, more highly magnified.

Fig. 9.—Section through the cæcum of a child, æt. 9 months, who died 60 hours after the onset of symptoms (John Hunter's specimen). The mucous membrane is almost unaffected, but it is covered with clotted blood. The muscularis mucosæ is normal. The submucous layer is thickened. The circular layer of muscle is more œdematous than the longitudinal layer. The mesocæcum is thickened by threads of fibrin, and it contains a congested lymphatic gland.

Fig. 10.—Section through the ileum of a child, æt. 5 months, to show the later changes occurring in intussusception. The mucous membrane is congested, and blood has been poured out into its substance, whilst the submucous tissue is beginning to suppurate. The muscular layers are œdematous and the serous coat is thickened and suppurating.

Plate XXXIX.

Fig. 11.—Section through the cæcum of a child, æt. 8 months, who died of intussusception. The mucous membrane is intensely congested, the submucous and serous muscular layers are suppurating. The serous coat is adherent to the serous layer of the returning layer of the intussusception. The submucous tissue of the returning layer is thickened by an extravasation of blood.

Fig. 12.—Section from the ileum of a woman who had an enteric intussusception associated with a polypus. The villi have become mere processes of connective tissue. The submucous layer is full of extravasated blood, but the mucous and muscular coats maintain their normal thickness.

Fig. 13.—Some of the villi from the same specimen as the preceding, to show the changes which have taken place in them.

FIG. 14.—A section from a part of the small intestine passed by a lady who survived for many years. The whole thickness of the section consists of dense fibroid tissue, except along the upper border where the outline of the mucous membrane is still visible, though none of its details can be made out.

PLATE XL.

FIG. 15.—A section through a piece of bowel which was passed by a woman, æt. 48. She died 10 days later. The bowel has been reduced to its connective tissue elements which have become converted into reticulin by a process of tryptic digestion.

FIG. 16.—Section through the large intestine of a child who died with a double intussusception. The mucous membrane is not much affected, but the submucous tissue is greatly thickened. The chief extravasation of blood has taken place near the muscularis mucosæ, where the section appears darker in the drawing.

FIG. 17.—Section through the second or retrograde intussusception, from the same case as the preceding. The relative thickness of the mucous, submucous, and muscular layers is unaffected, but the serous coat is thickened and its vessels are congested.

FIG. 18.—Section from the ileum of a woman who died of an intussusception associated with a polypoid growth. The villi contain numerous oval bodies, which appear to be thrombosed vessels.

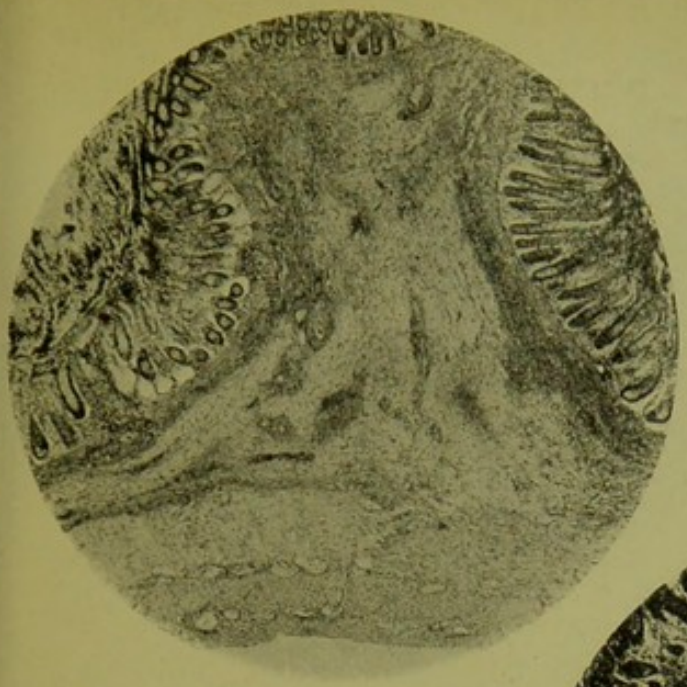
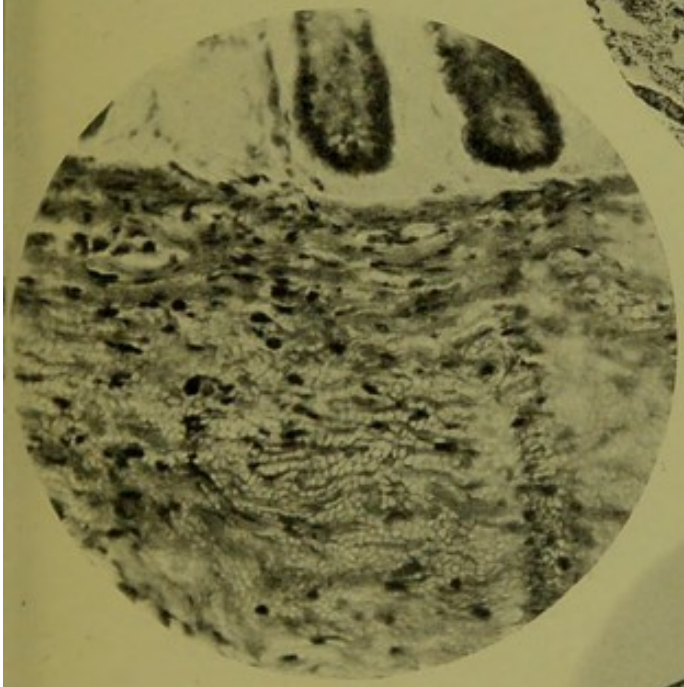


FIG. 1.

FIG. 2.



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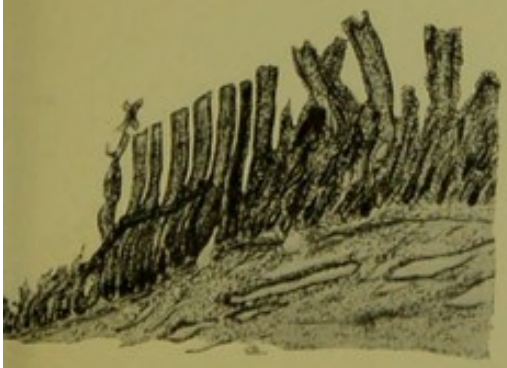

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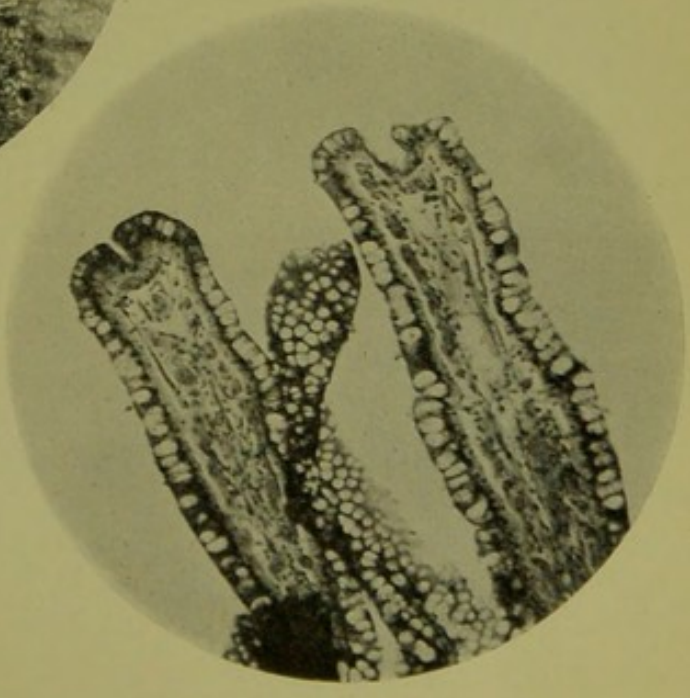

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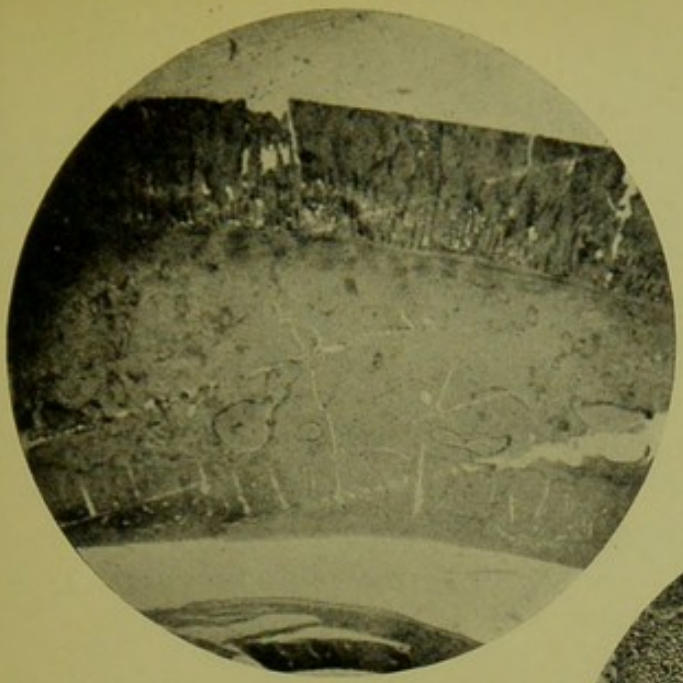

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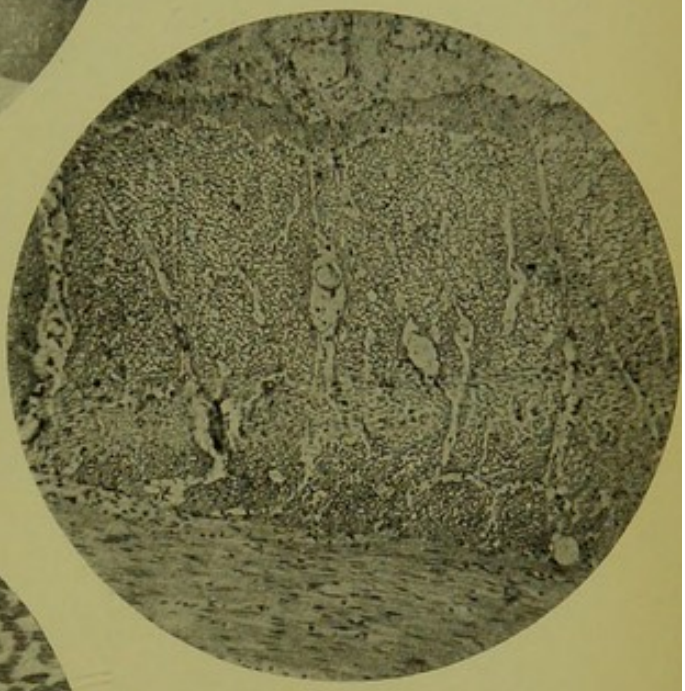

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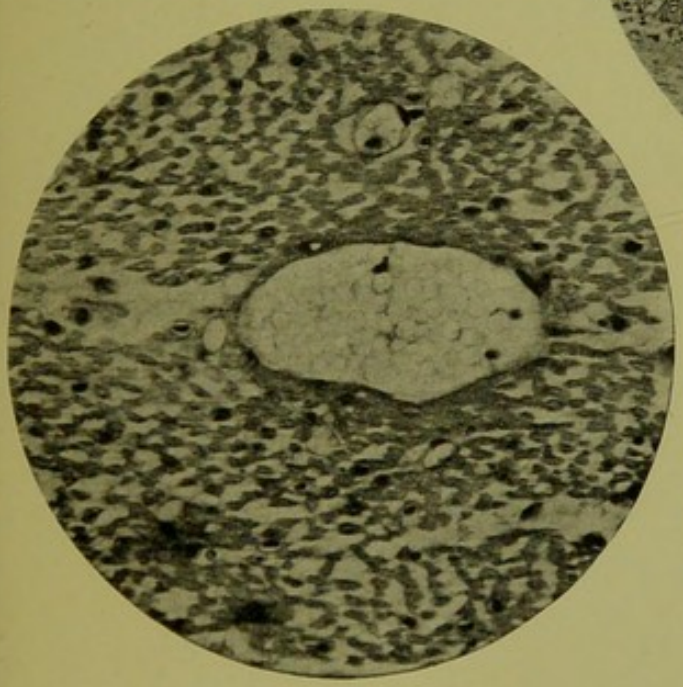

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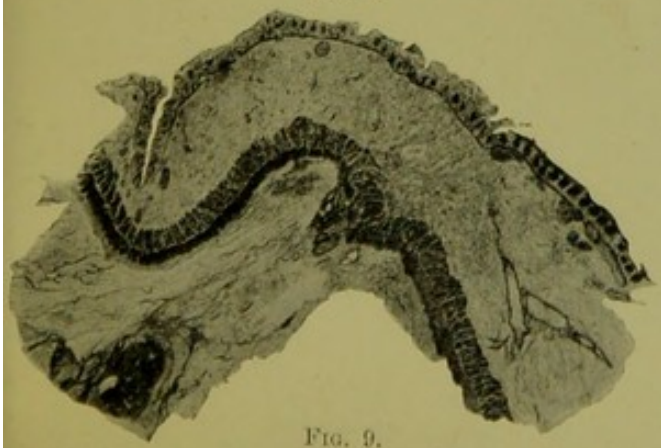
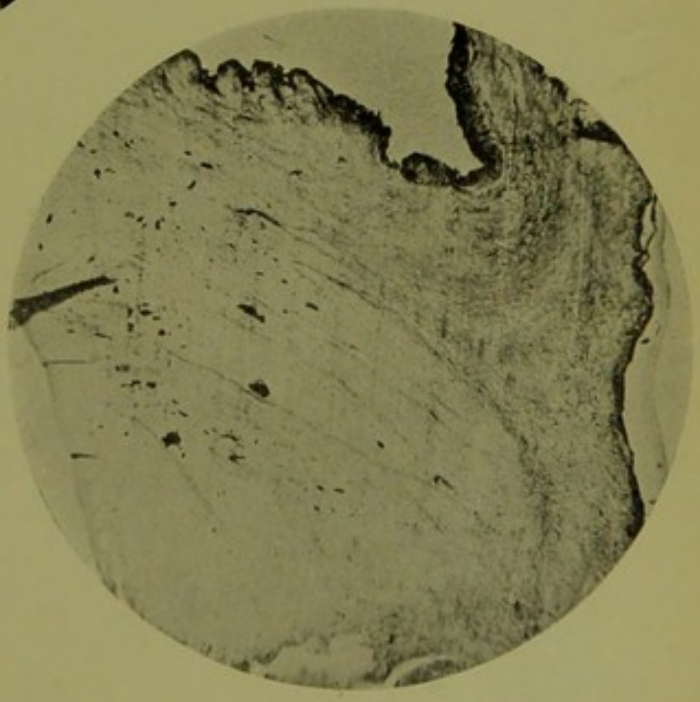


FIG. 9.

FIG. 11.

FIG. 12.

FIG. 13.



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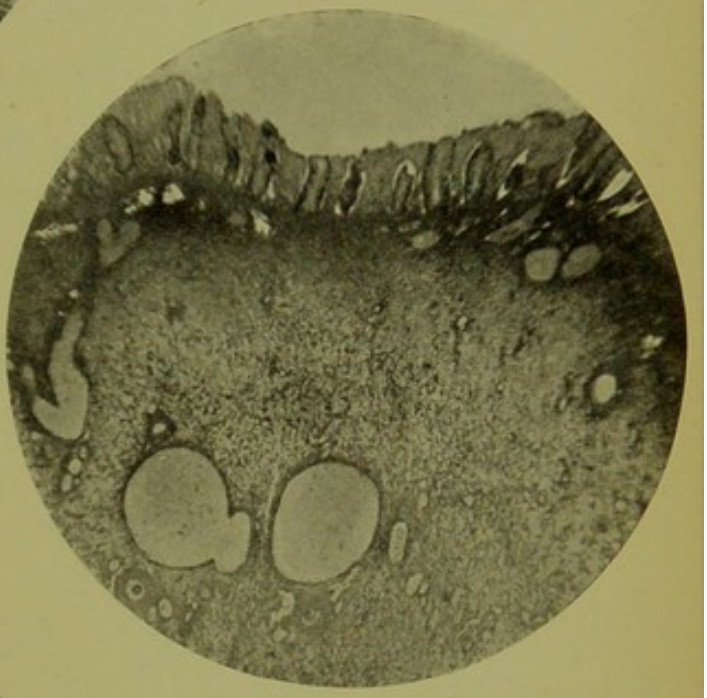

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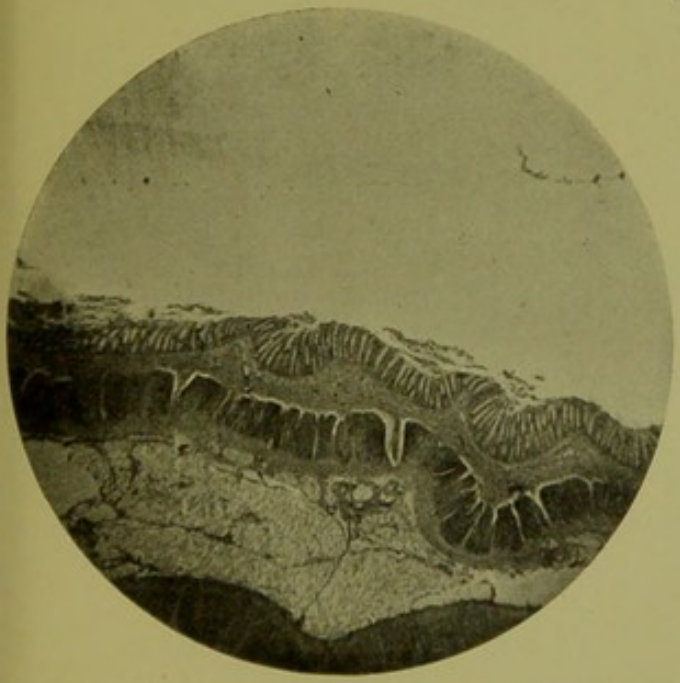

FIG. 17.

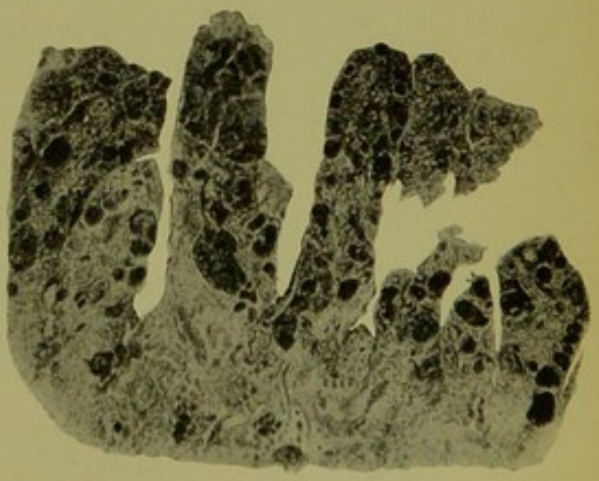

FIG. 18.