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Contributors

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WESTERN INFIRMARY:

NOTES OF CASES IN PROFESSOR BUCHANAN'S WARDS.

By J. CRAWFORD RENTON, M.D.

THE following cases of interest have come under observation and treatment from time to time in Prof. Buchanan's absence, and with his kind permission I record them:—

EXCISION OF HALF THE TONGUE.

A. B., admitted with a well-marked epithelial ulcer on the left lateral aspect of the tongue. Half the tongue removed by Whitehead's method. A silk ligature was passed round the lingual artery before division, and the patient lost very little blood. Whitehead's varnish was applied to the cut surface, and the wound healed well. The patient lived a year, and was killed when at his work by a fly-wheel. He was quite well up to his death.

C. D. was sent to the Infirmary by Dr. Easton, of Stranraer. The left half of his tongue was removed for the same cause as the above case, and he continued well for eighteen months.

In another case the employment of Mr. Scott Riddell's tongue-clamp enabled us to cut away the tongue with very little bleeding, and I can recommend the use of it.

CRANIAL ACCIDENTS.

M. A., æt. 4, was admitted, having fallen one storey, sustaining a compound comminuted fracture of right parietal bone. The child was unconscious, the left eye being drawn inwards. Slight consciousness returned in two hours, but patient remained with paralysis of left arm and leg and left side of face. Ten hours after admission chloroform was given,

and the depressed portions of bone on the right side, after exposure by dissection of a large flap, elevated, the rongeur forceps being used to effect this; the brain was lacerated, and a considerable amount of blood-clot was removed. The portions of bone were placed in position, a catgut drain inserted—the periosteum was so torn that it was not possible to unite it—and the wound was united with catgut sutures.

The child's progress was satisfactory, the power returning to the leg slightly during the night after the operation, and completely so by the fourth day after, as also that of the face; the power returned to the arm more gradually, but two months after the operation the movement was restored in a great measure. The only point noted was that the hand was a little unsteady when used for three minutes, but that is gradually passing off.

G. P., æt. 15, admitted, having fallen from a baker's van an hour previously. Punctured wound was found to exist in the left parietal region above and behind the ear.

Two hours after admission patient was drowsy, dilatation of right pupil and right facial paralysis existing. Tongue deflected to the right when protruded.

Two hours later, operation—a flap of skin turned down, and the skull trephined on site of punctured fracture; no brain pulsation; dura opened, blood escaped freely, and a small collection of blood was evacuated from the brain substance underlying. Catgut drain inserted and the wound closed by catgut stitches.

The day following the operation paralysis was gone, and the tongue could be protruded in the middle line.

The patient progressed satisfactorily, and was dismissed well.

J. B., æt. 15, was admitted, suffering from epileptic attacks affecting the left side of the body. The patient had fallen off a stool 4 feet high, and was confused for a few minutes, but was able to walk home; a fortnight after the injury the fits commenced and she was brought to the Infirmary.

Dr. Walker and the special nurse noted the fits, and found that they always commenced in the left arm, spreading to the face.

Six days after admission trephining was carried out over the arm centre on right side, the opening being enlarged with forceps; the brain pulsation was diminished. On opening the dura and feeling the brain substance it was evident that a

collection of fluid existed, and on penetrating the brain with a narrow knife an eggcupful of blood was evacuated. A catgut drain was inserted, bone partially replaced, and the wound closed.

Convalescence was uninterrupted except for some slight twitchings of the mouth some days after operation, and three months after operation she continued well.

ACUTE INTUSSUSCEPTION.

M. W., æt. 3 months, was sent to the Western Infirmary by Dr. Paterson, of Berkeley Terrace, with symptoms of intussusception which had existed for twenty-four hours.

On examination under chloroform a distinct fulness and some swelling was found along the line of the transverse and descending colon, and *per rectum* a well-marked protrusion was felt. No influence was produced on this swelling by manipulation or injection, so the abdomen was opened, and with some difficulty 18 inches of intussuscepted bowel were drawn out and returned into the abdomen, and the wound closed. The bowels acted six hours after, and recovery was uninterrupted.

REMOVAL OF CÆCUM WITH SUCCESSFUL USE OF MURPHY'S BUTTON.

J. M., æt. 30, had suffered for five years from fistula into the cæcum with subsequent contraction. After various attempts at closure the cæcum was removed and the ileum joined to the colon with Murphy's button. The patient did well, passing the button on the sixteenth day and is now quite well.

I have pleasure in thanking Drs. Beatson, Burnside Buchanan, Walker, and Carstairs for much valuable help in the conduct of these cases.



ENDARTERITIS PROLIFERENS. By W. AINSLIE HOLLIS,
M.D. Cantab., F.R.C.P. Lond., *Senior Physician to the
Sussex County Hospital.*

Pathogenesis.—Endarteritis proliferens vel obliterans is essentially a disease of the smaller vessels; and it so far differs from atheroma, which attacks by preference the aorta and the large central arteries. Besides this difference in the distribution of the two diseases, their incidence locally and their subsequent evolution are dissimilar. These peculiarities have led some pathologists to assume—wrongly, I believe, although perhaps not unnaturally—that their etiology must necessarily be unlike. In a series of papers published elsewhere,¹ I have adduced reasons for the belief that in many instances atheroma is due to the occasional presence of obnoxious foreign particles in the blood stream. Whether these small particles are in every case the true etiological factors of atheroma is likely to remain a debatable question for some time to come. Meanwhile, the provisional acceptance of such an hypothesis appears more reasonable to my mind than to assume a knowledge of its causation by vaguely ascribing atheroma to senility, mental worry, or overwork, as is now too often done.

On the other hand, the etiology of endarteritis proliferens does not present quite the same difficulty that atheroma does, because pathologists are in a general accord when they describe this disease as one “brought about, as a rule, by some general irritant, which affects the arteries, along with the connective tissue of the organ generally.”² Here, however, my interpretation of the word ‘irritant’ may differ considerably from that of the authors quoted. From the examples of this disease which have come under my notice, and to which special reference will be made in this paper, the association of atheroma

¹ *Journ. Path. and Bacteriol.*, London, vol. 3, pp. 1 et seq. *Tr. Royal Med. Chir. Soc.*, vol. lxxvii. p. 69.

² *Ibid.*, vol. iv. p. 83. Paper by Drs Coats and Auld.

with endarteritis proliferens is far from unusual. The two cases from which the photographs were taken to illustrate these remarks were both of them affected with extensive aortic atheroma.

In cases of advanced atheroma, the blood must repeatedly convey, besides its proper corpuscular elements, the granular detritus washed from the surface of a crumbling patch of intima. Whatever may be the ultimate destination of these particles matters not as regards our present inquiry. It is evident that many, if not all, of them will strike against the walls of the vessels through which they pass; and, other things being equal, the smaller the artery, the more frequently will such accidents probably happen. The repeated impact of foreign material against the living walls of a small artery will, I assume, mainly produce two results,—first: an immediate reaction of the endothelium, subjected to the blows. What this reaction consists in remains at present unknown. Possibly it may take the form of active molecular changes in the affected cells, to which the words ‘stimulation’ or ‘irritation’ may be applicable. Subsequently, and as a consequence of this irritation, secondary effects will arise, the chief one of which is probably a spasmodic contraction of the muscular coat. Now, I wish to show in the following pages that the histology of endarteritis proliferens singles out these two structures, the endothelium and the contractile media, as specially affected in this disease. All the evidence, then, at my disposal points to the presence of foreign particles in the blood as an important factor in the causation of endarteritis proliferens, whatever else may occasionally induce it.

Histology.—The sections described in this paper were prepared in a similar manner: they were first hardened in a 10 per cent. solution of formalin; then stained, and finally mounted in canada balsam. Although text-books have repeatedly described and illustrated, oftentimes diagrammatically, the transverse section of healthy artery, perhaps I may be permitted to give a photograph of the intima of a popliteal artery from a 6–7 months foetus which died *in utero*, and was removed at its mother's autopsy. It is important to compare the inner surface of an artery absolutely free from the suspicion of contamination

through the circulation with others which have been admittedly exposed to contamination. I shall not tire the readers of the *Journal* with a description of the figure. It speaks for itself. (See fig. 1.) I shall, however, refer to it hereafter.

The next illustration (fig. 2) represents a transverse section of an upper oesophageal branch of the aorta from a woman aged 69 years, who died of cancer, involving the cardiac end of the stomach. The portion shown in the figure was situated a few millimetres only from a very atheromatous aorta. There is no doubt about the extent or nature of the disease affecting the arterial coats in this figure. We can see the lumen of the

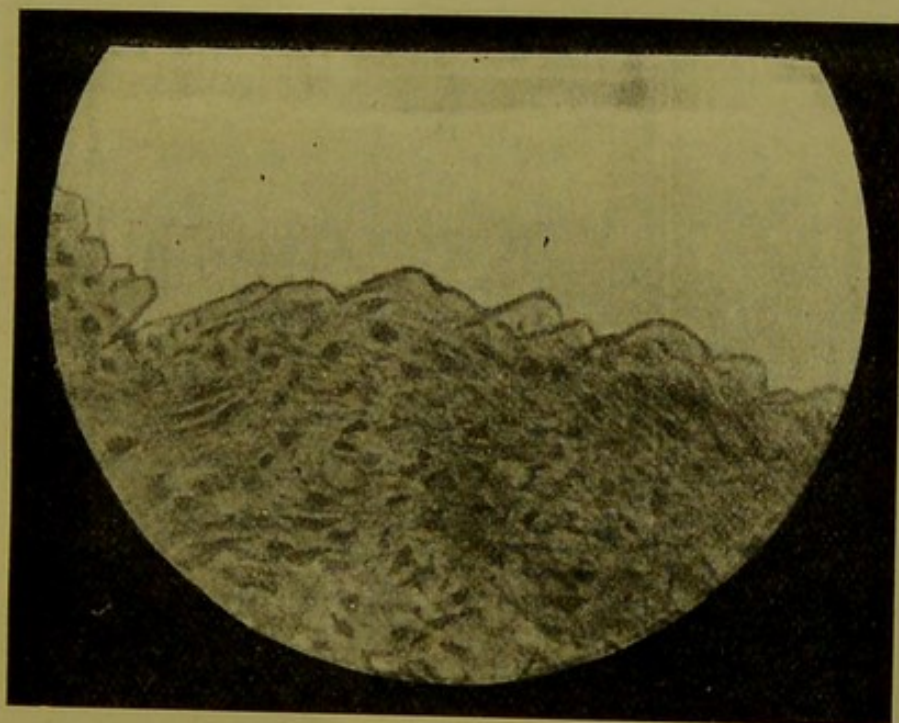


FIG. 1.—Transverse section of popliteal artery, foetus 6 months. The intima only is shown. Zeiss F. $\times 532.9$.

vessel is much diminished by endothelial cell proliferation, occurring mostly in the shape of rounded projections. These are separated from one another by deep indentations, which appear to extend almost to the muscular coat, and probably represent the longitudinal furrows, into which the intima is thrown by the contraction of its muscular ring. As, however, it will be more convenient to consider the histology of this disease with regard especially to the early stages of its growth and development, I pass on to the next illustration (fig. 3). This also depicts a cross-section of an oesophageal branch of the aorta.

The specimen was taken at the autopsy of a man aged 61 years. He suffered from dysphagia for six months before death. At the post-mortem examination a stricture of the œsophagus was found, commencing about an inch above the level of the bifurcation of the trachea, and extending for two inches downwards. The tissues were greatly thickened around the stricture; and opposite the bifurcation there was an opening into the left bronchus, the size of a sixpence, surrounded by fungating, ulcerous walls. The glands adjacent the stricture

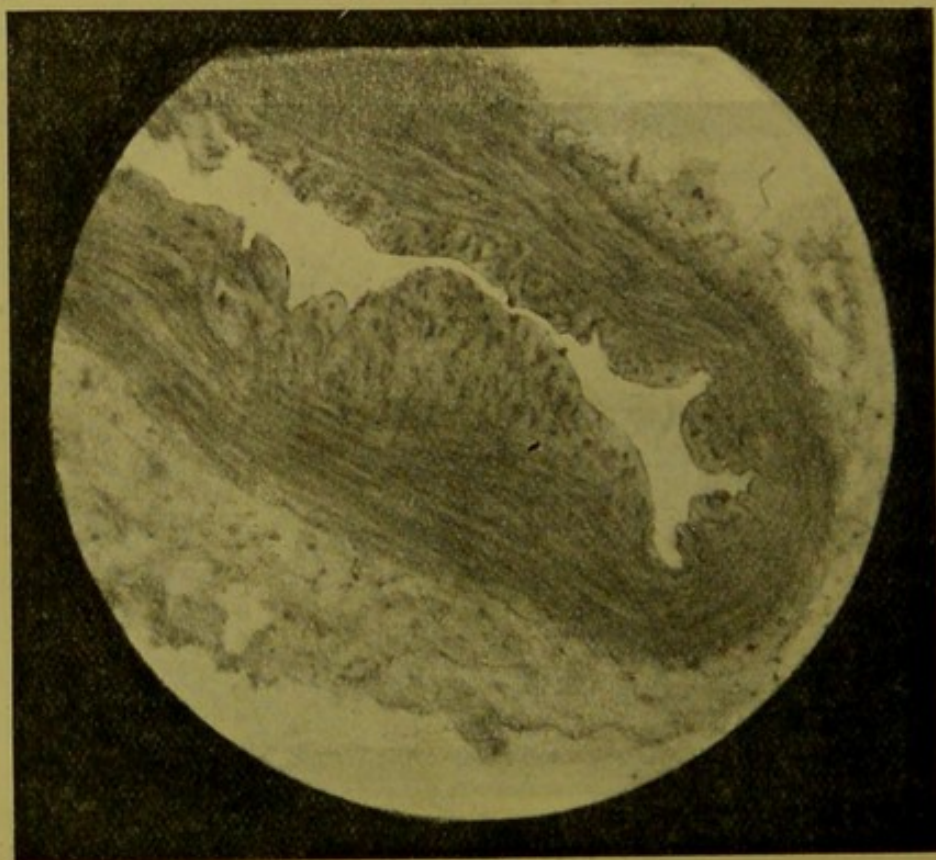


FIG. 2.—Transverse section œsophageal branch of aorta, with advanced endarteritis proliferans, from a case of cancer of stomach cardia. Zeiss DD. $\times 144.4$.

were infiltrated with a soft yellowish-white material. The aorta was very atheromatous.

The endarteritis in this instance has not led to such an excessive proliferation of endothelium as was observable in the previous figure. There is, however, an overgrowth, consisting for the most part of small, ill-developed cells. A sort of leveling up process has then apparently commenced. The longitudinal folds into which the intima of a small artery is naturally

thrown by the contraction of its muscular coat were first filled and subsequently obliterated by the young cell formation. This is well shown in figure 4, where we can trace the original boundary-line of the renal arteriole as it apparently dives into the new tissues. The nuclei were unfortunately not stained in this section. We can, however, observe them in figure 3. Their arrangement is peculiar. Whilst those in the inner lining of the vessel are fortuitously dotted through the new

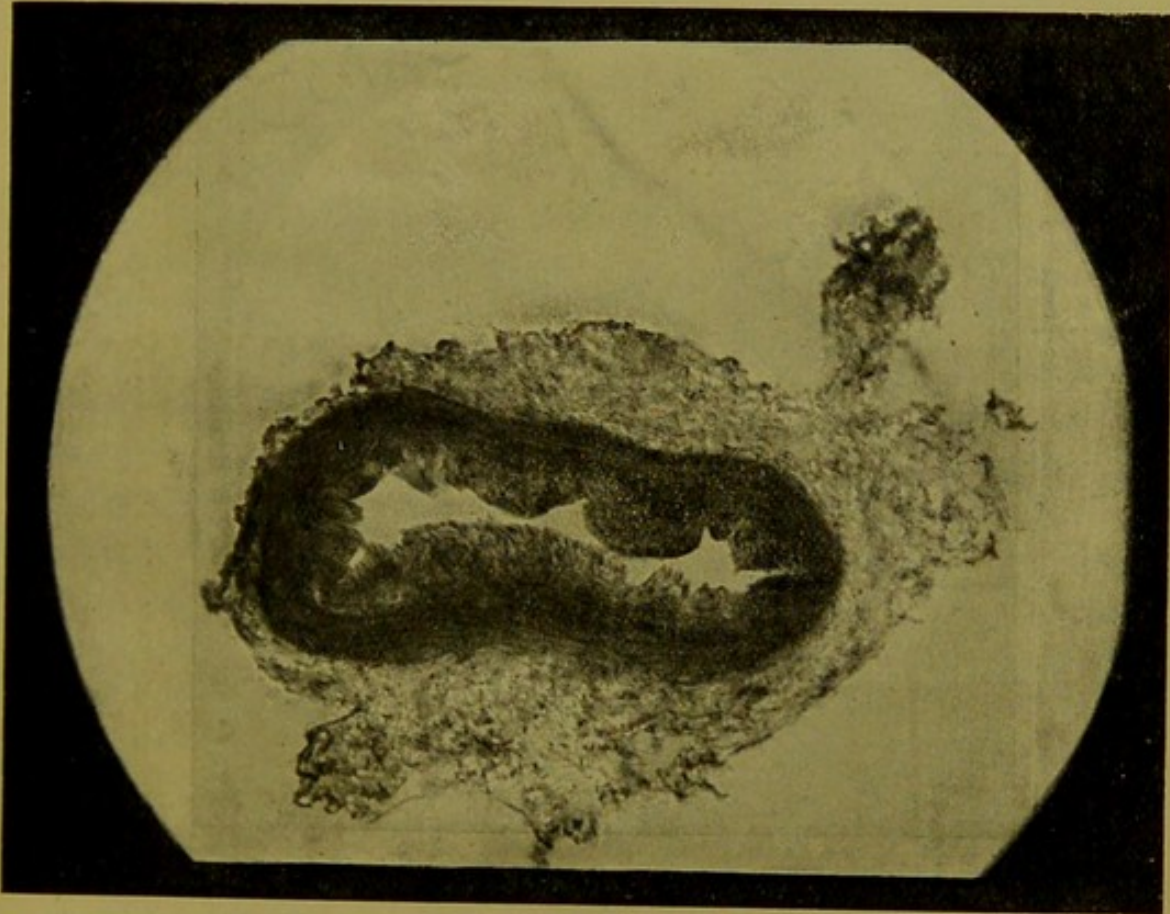


FIG. 2B.—Transverse section of an oesophageal branch of aorta. Case of carcinoma of cardiac end of stomach. Zeiss B.

tissues, a large number of the spindle-shaped nuclei in the outer endothelial layers are arranged with their long axes radially. From one or the other pole of the nuclear body we may, not rarely, see thread-like extensions, which pass oftentimes between two adjacent cells. In the inner and more irregularly shaped cells I have occasionally found two nuclear prolongations from one corpuscle. These highly stained filaments, I presume, are merely pseudopodial extensions of the nuclear plasm. If this

interpretation of their function is a true one, it follows that we have here to deal with little masses of protoplasm, formerly endowed with independent powers of locomotion. To some writers who doubt the presence of vagrant corpuscles among the arterial tissues, except as rare, and hence uninteresting, strangers, this suggestion will not be acceptable; yet the existence of 'free nuclei' in such situations is admitted by many pathologists. If we grant these independent bodies the power of locomotion

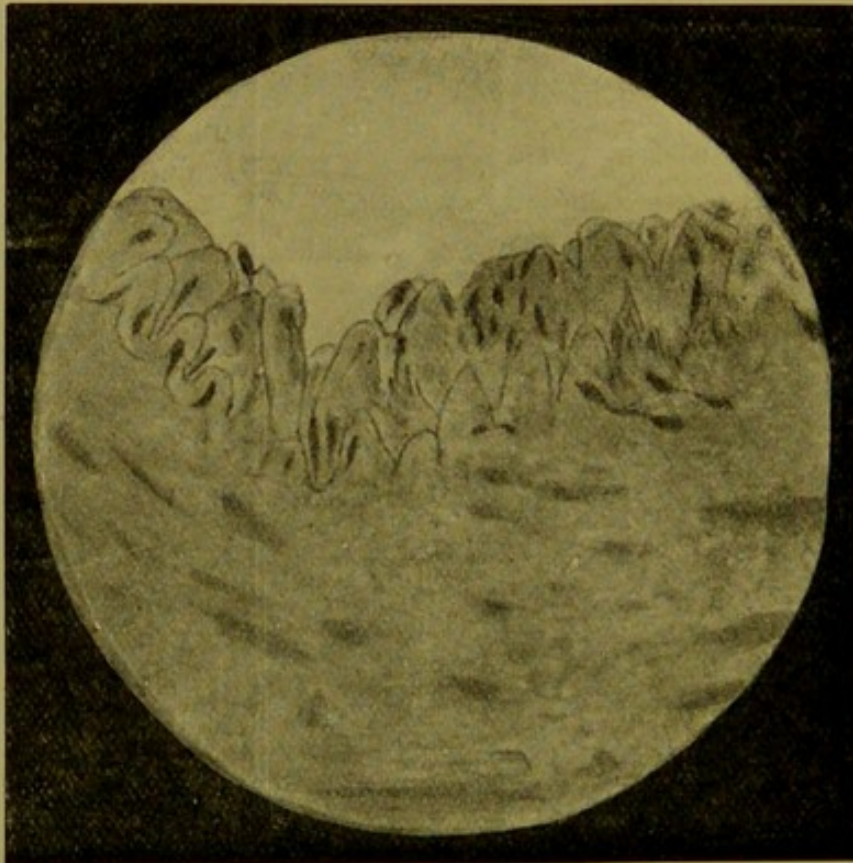


FIG. 3.—Transverse section of an oesophageal branch of an atheromatous aorta. Early stage of endarteritis proliferans. From a photograph. Zeiss F. $\times 490$.

during life, their arrangement in the arterial walls, as above described, gains in significance what it loses in singularity. We can then account for their dissimilarity in the inner and the outer layers of the endothelium. The newly formed cellular lining of the vessel, less cohesive than the older basement cells, permits freer movements among nuclear bodies embedded within it; hence their spheroidal or broadly pyriform shapes when compared with the slender spindle-like corpuscles of the deeper layers, for the latter are laterally compressed in their

passage through the firmer and more resistant tissues of the original arterial wall.

According to my observations, then, it would appear that endarteritis proliferens affects an artery somewhat in the following manner. By the repeated impact of foreign particles against the sides of a small artery a state of 'irritation,' or 'molecular unrest,' as I prefer to call the condition, is established within certain endothelial cells. This unrest manifests itself physiologically by, as it were, rousing the little central mass of nuclear plasm of the cell from its dormant condition to one of active movements. The nuclear disturbance is quickly made known to the vasomotor nerves, and contraction of the artery follows. Now, as we shall see more completely when I come to consider the formation of the tubular clot, this spasmodic contraction of the muscular coat of the vessel is, under such circumstances, about the worst misfortune that could happen. By occurring, as it probably does, during the passage of a cloud of noxious particles through the artery, it enhances the risk that is always present of some being retained within the folds of the intima, to form a still more permanent source of irritation. Hence it happens that the cells at the bottom of a longitudinal fold are more liable to disturbance from this cause than others situated elsewhere. Moreover, from the physical laxness of the tissues in the same situation migrating leucocytes will probably select the furrows as points of departure, a further source of 'molecular unrest' to the neighbouring endothelium.

The existence of free nuclei in the walls of a blood-vessel almost suggests the presence of denucleated cells there. That such cells occur amidst the endothelium of a normal artery I feel confident; the more so, as their presence seems to have been tacitly admitted by some of the leading histologists and pathologists of the present day. I have before me at the time of writing two text-books, selected at random, one upon human histology, the other on practical pathology, each by well-known men. A cross-section of a normal artery forms one of the many beautiful figures with which each work is embellished. The lumen of the vessel, as drawn, is margined in either case by a series of dotted projections, alternating with others which are undotted. The text of each work informs the reader that the

dotted projections represent nucleated endothelium. What the undotted spaces depict is not stated, although in one figure about 50 p.c. of the margin, and in the other fully a fourth, is so drawn. I assume that the artists intended to show by the blank spaces their inability to see any nuclei at those parts. I do not wish to assert that one cell in every four, lining a normal artery, is without a visible nucleus, even in well stained sections, but I maintain that a considerable percentage of endothelial cells are normally without stained nuclei when seen as microscopic preparations (fig. 1). Now, the absence of a visible nucleus may theoretically, I take it, be due to three causes:—(1) It may not take the selected stain. In such cases, however, the outline of the nucleus is always faintly perceptible, if the light is rightly arranged. (2) The cell may never have had a nucleus,—a suggestion of little value. (3) Finally, the nucleus may have escaped from the cell, and consequently the latter was actually denucleated at the time the section was made. The last explanation, in so far as it also seems specially applicable to the new cell formation of endarteritis proliferans, I shall here adopt as the true one.

To resume the progressive history of this disease. When, through over-stimulation, the potential energy of a celled nucleus is converted into active movement, it may lead to the subdivision of the nucleus and the formation of daughter cells on the inner surface of the artery; or a part or the whole of the nucleus may escape through the cell-wall, producing two results, a free nucleus and a denucleated cell. When, by some such process, the natural folds of the intima have been locally obliterated, and the dilation of the artery consequently restricted (figs. 3 and 4), an interesting pathological cycle is apparently induced. As I have elsewhere stated, although we cannot follow the actual movements of a locomotory corpuscle in a stained and mounted section, we may frequently infer the direction in which it was travelling at the time of death from two considerations; namely, that it would probably protrude its pseudopod towards the point it was making; or, secondly, that the vacant burrow it had already made whilst passing through the resistant tissues would be behind it. Viewing their probable movements in this light, I have come to the conclusion, as the

result of many observations, that the little roving protoplasmic masses mainly pass in two directions through the diseased tissues. Whilst a certain number of them apparently force their way outwards between the cells of old endothelium at the bottom of the filled-in furrows, others, starting from the areolar bed, which separates the intima and the media, move inwards, passing, for the most part, through the alternate folds of endothelium dividing the furrows (fig. 3). In cross-sections

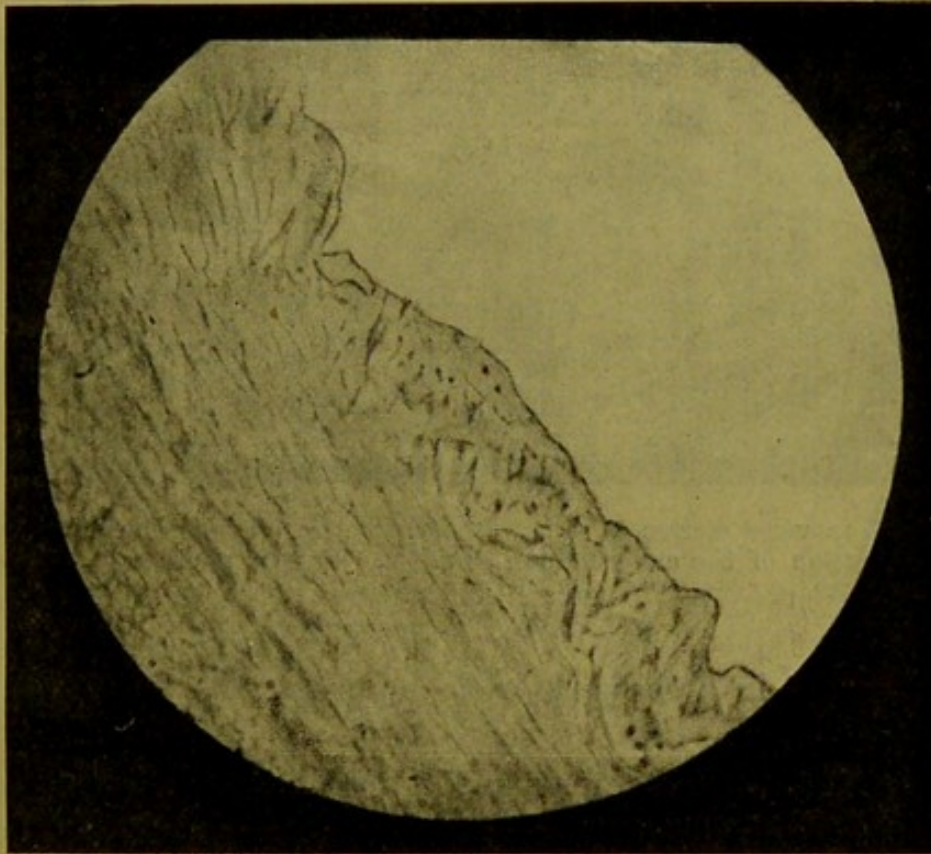


FIG. 4.—Transverse section of a renal arteriole, in a case of kidney infarct, showing endarteritis proliferans in early stage. From a case of cancer of the head of pancreas. A photograph. Zeiss F. $\times 600$.

the apices of these folds are often visibly frayed out, their brush-like tops pointing inwards towards the blood current. This brush-like top is, I consider, due first to the escape of the nucleus from an apical cell into the newly formed tissue beyond; secondly, to the subsequent passage of inward bound motile corpuscles along the same route.¹ Functionally, the frayed

¹ In connection with this subject, compare the observations of M. L. Ranvier on the physiological role of leucocytes in corneal wounds, the formation of 'synaptic' fibres, and the proliferation of the epithelium. (*Sem. Méd.*, March 12, 1897.)

out apex is of importance, as it evidently assists in holding together the feebly cohesive tissues then lining the artery. By such means are the new tissues in endarteritis proliferens welded, as it were, to the old. In the earlier stages of the disease, only here and there can a disrupted fold be seen; when it is advanced, few, if any, of the folds remain intact (fig. 5).



FIG. 5.—Transverse section of an aortic branch showing extensive proliferation of the endothelium. Case 2. From a photograph. Zeiss F. $\times 900$.

This change is in accordance with the progressive nature of the disease.

In offering this interpretation of the chief facts observed by me in the etiology and development of this disease, I do so without prejudice to the many excellent papers already published on this subject. I may perhaps be permitted to state that, in giving again prominence to the acts and wanderings of the locomotory corpuscle, I have in mind the history of a little speck of living protoplasm, endowed with the power of *slowly* forcing a passage through the surrounding tissues, but the direction and the speed of whose movements are mainly deter-

Also a paper by Miss Lily Huie on the changes which take place in the protoplasm and nucleus of the gland cells in the tentacles of the sun-dew, *Drosera rotundifolia*, after the feeding the leaf with pieces of white of egg. The conclusion is drawn, that changes indicative of great activity of the nuclear organs are not exclusively characteristic of cell-division. (*Quart. Jour. Micr. Sc.*, Jan. 1897.)

mined by the physical needs of the living tissues in which it is buried, and of which even itself forms an integral part.

DESCRIPTION OF THE FIGURES, pp. 155-162;

WITH APPROXIMATE MAGNIFICATION.

Fig. 1. Transverse section of the popliteal artery of a 6-7 mos. fœtus. The intima only is shown. From a photograph. $\times 532$.

Fig. 2. Transverse section of an œsophageal branch of an atheromatous aorta, showing advanced endarteritis proliferens. From a photograph. $\times 150$. — Fig. 2B. Another section of the same.

Fig. 3. Transverse section of an œsophageal branch of an atheromatous aorta. Early stage of endarteritis proliferens. From a photograph. $\times 490$.

Fig. 4. Transverse section of a renal arteriole, showing endarteritis proliferens in early stage. From a case of cancer of the head of pancreas. A photograph. $\times 600$.

Fig. 5. Transverse section of an œsophageal aortic branch, showing extensive proliferation of the endothelium. Case 2. From a photograph. $\times 900$.

