A treatise on the ligation of the great arteries in continuity : with observations on the nature, progress and treatment of aneurism / by Charles A. Ballance, and Walter Edmunds.

LIGATION IN CONTINUITY

"But yet be wary in thy studious care."

Henry VI. Part I.

A TREATISE

ON THE

LIGATION OF THE GREAT ARTERIES IN CONTINUITY

WITH OBSERVATIONS ON THE NATURE, PROGRESS AND TREATMENT OF

ANEURISM

BY

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ILLUSTRATED BY 10 PLATES AND 232 FIGURES.

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ANTONIO SCARPA.

PROFESSORE EMERITO, E DIRETTORE DELLA FACOLTA' MEDICA DELLA I. R. UNIVERSITA' DI PAVIA, CAVALIERE DELL' INSIGNE ORDINE AUSTRIACO DI LEOPOLDO, SOCIO DELLA R. ACCADEMIA DELLE SCIENZE DI PARIGI, DI LONDRA, DI BERLINO, DI STOKOLM, ECC., ECC.

TO THE MEMORY OF

ANTONIO SCARPA

THESE PAGES

ARE INSCRIBED.

PREFACE.

THE researches on which the following pages are based were commenced in 1885.

In May of the year following we made a communication to the Royal Medical and Chirurgical Society on *The Ligation of the larger Arteries in continuity.*

Since that time we have continued to work at the subject in its numerous bearings.

The experiments were in part carried out in the pathological laboratory at Leipzig, in part in the laboratory of the Brown Institution, and in part in the physiological laboratory of St Thomas's Hospital.

In 1889 the Erasmus Wilson lectures in Pathology at the Royal College of Surgeons were delivered by one of us on the *Pathology of Hemorrhage after Ligation in Continuity,* but as our investigations in certain directions were still incomplete the lectures were not published. We think however that we are now prepared with our case, though of course there can be no finality in any scientific discussion.

Each chapter we have endeavoured to make to some extent complete in itself, so that it may be read separately: this has entailed the occasional repetition of an argument or a second allusion to a case.

We are indebted to a large number of kind friends for assistance in various ways, and we take this opportunity of tendering them all our sincere thanks.

At Leipzig Professor Birch Hirschfeld gave us every facility in his laboratory, and his assistant, the late Dr Hüber, took much interest in the work. There also we made the acquaintance of Dr William Hunter: he helped us in various ways there and as will be seen we are under obligations to him and his writings.

At the Brown Institution Professor Horsley not only gave us permission to carry on our investigations there but also personally assisted us in them.

In the various questions that arose bearing on physiology we were happy in having the assistance and advice of Professor Sherrington. Chapter VI. indeed is half his work and has been already published as such. We are also indebted to him for the use of his laboratory for making various pressure experiments and for placing his time most freely at our disposal.

The Members of the Staff of St Thomas's Hospital we have to thank for much assistance in various ways, and also Mr Shattock, the lecturer on Surgical Pathology.

To Professor Stewart of the Hunterian Museum and to the Committees and Curators of the Museums of St Bartholomew's, Guy's, and University College Hospitals we are indebted for permission to have specimens drawn.

Sir James Paget was good enough to look out for us notes of cases of ligation which had been under his care at St Bartholomew's.

Professor Roy and Dr Hector Mackenzie aided us by their suggestions and criticisms.

It has been our endeavour in the proper place to acknowledge the drawings and specimens which we received through the courtesy of various friends and professional brethren at home and abroad.

The original drawings for the illustrations have been made by Mr Lapidge, to whose skill and accuracy we are much indebted. We have also to thank him for valuable advice in connection with the illustrations.

The reader will appreciate the care that has been taken by the Typographic Etching Company and the Cambridge University Press in reproducing and printing those figures which are scattered through the text.

The plates have been executed partly by the Cambridge Engraving Company and partly by the Woodbury Photographic Company. They too are faithful representations of Mr Lapidge's drawings.

The portrait of Scarpa is from an engraving in the possession of the Ophthalmological Society.

LONDON,
October, 1891.

CONTENTS.

CHAPTER I.

HEMORRHAGE IN MAN.

CHAPTER II.

VALUE OF EXPERIMENTS.

CHAPTER III.

NATURE OF ARTERIES.

CHAPTER IV.

PHYSIOLOGICAL OCCLUSION.

CHAPTER V.

PATHOLOGICAL OBLITERATION.

CHAPTER VI.

CONDUCT AND FATE OF THE CORPUSCLES.

CHAPTER VII.

CONDUCT AND FATE OF THE CORPUSCLES (CONTINUED).

CHAPTER VIII.

CONDUCT AND FATE OF THE CLOT.

CHAPTER IX.

CONDUCT AND FATE OF THE COATS.

CHAPTER X.

CONDUCT AND FATE OF THE LIGATURE.

CHAPTER XI.

SUPPURATION AFTER LIGATION.

CHAPTER XII.

PATHOLOGY OF HEMORRHAGE.

CHAPTER XIII.

CONDUCT AND FATE OF THE ANEURISM.

CHAPTER XIV.

THE SURGERY OF THE ARTERIES: THE FOUNDATION OF SURGERY.

CHAPTER XV.

CHOICE OF THE OPERATION.

CHAPTER XVI.

CHOICE OF THE LIGATURE.

CHAPTER XVII.

CHOICE OF THE KNOT.

CHAPTER XVIII.

CHOICE OF THE FORCE.

CHAPTER XIX.

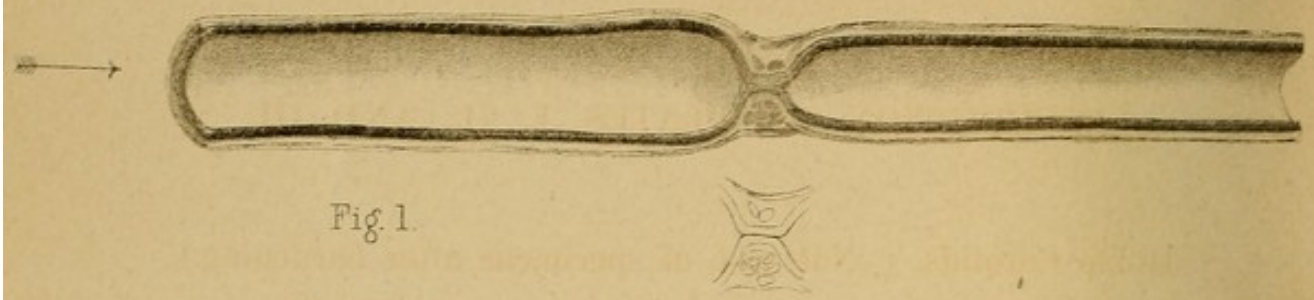
LIGATION EXPERIMENTS.

CHAPTER XX.

CONDUCT OF THE OPERATION AND FATE OF THE PATIENT.

LIST OF PLATES.

LIST OF FIGURES.



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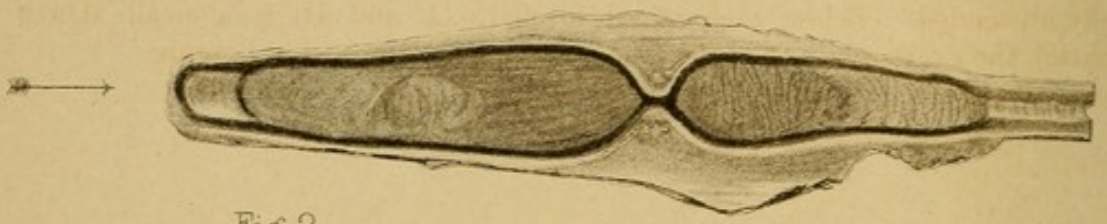

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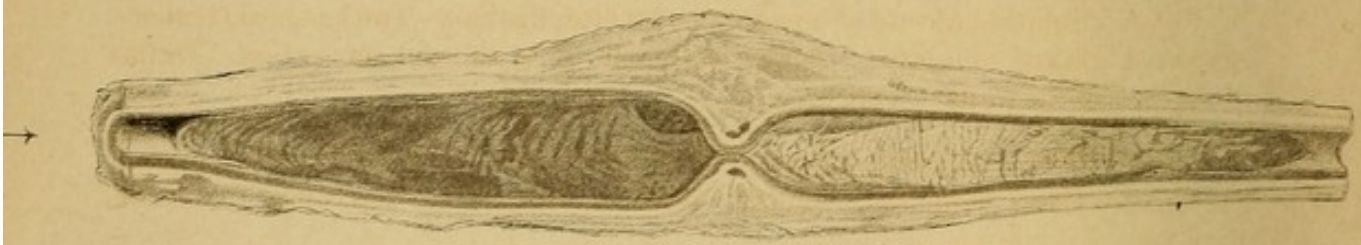

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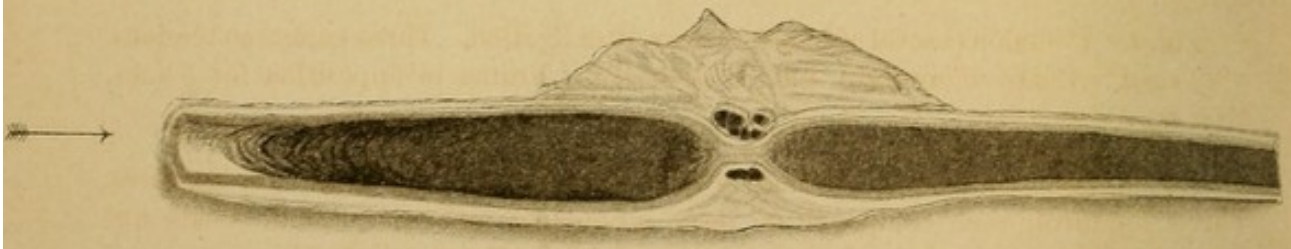

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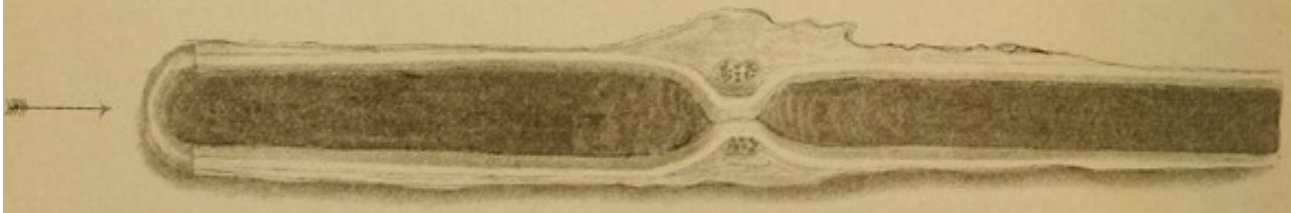

Fig. 5

M. H. Lapidge del. ad nat.

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DESCRIPTION OF PLATES I., II., AND III.

Horse Carotids. (Nat. size of specimens after hardening.)

In all the drawings the cardiac end of the artery is to the left, as shewn by an arrow; it is to be observed that on the cardiac side of the ligature the artery is usually distended. Below each vessel in plates I. and II. is a small sketch indicating the exact relations of the uninjured walls at the seat of ligature.

Plate I.

Fig. 1. Common carotid of horse, 24 hours after ligation. Two kangaroo tendons. Stay-knot. Coats uninjured. Lumen occluded. Clot fell out on section. Intima in apposition for 4 mm. Exp. 64.

Fig. 2. Common carotid of horse, 14 days after ligation. Two floss-silk ligatures. Stay-knot. Coats uninjured. Lumen occluded. Intima in apposition for 3 mm. Exp. 65.

Fig. 3. Common carotid of horse, 21 days after ligation. Two kangaroo tendons. Stay-knot. Coats uninjured. Lumen occluded. Intima in apposition for 2·5 mm. Exp. 63.

Fig. 4. Common carotid of horse, 14 days after ligation. Three kangaroo tendons. Stay-knot. Coats uninjured. Lumen occluded. Intima in apposition for 5 mm. Exp. 62.

Fig. 5. Common carotid of horse, 21 days after ligation. Silkworm-gut ligatures. Three ligatures, each composed of three strands of gut. Stay-knot. Coats uninjured. Lumen occluded. Intima in apposition for 4·5 mm. Exp. 61.

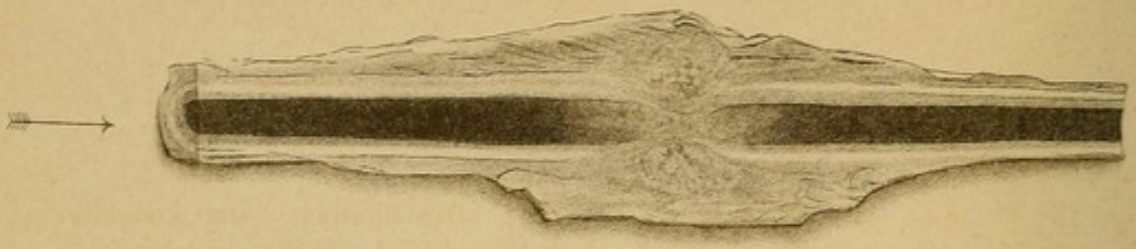

Fig. 1.

Fig. 2.

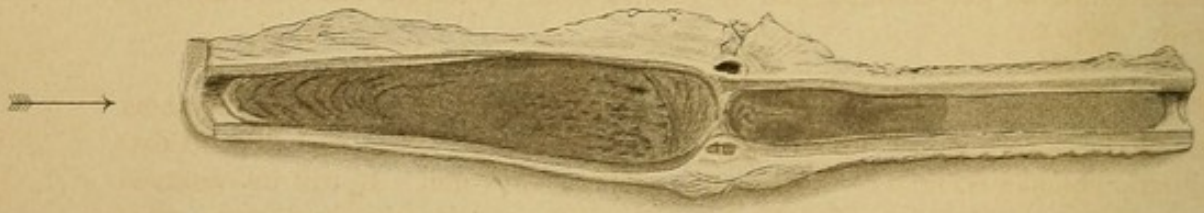

Fig. 3

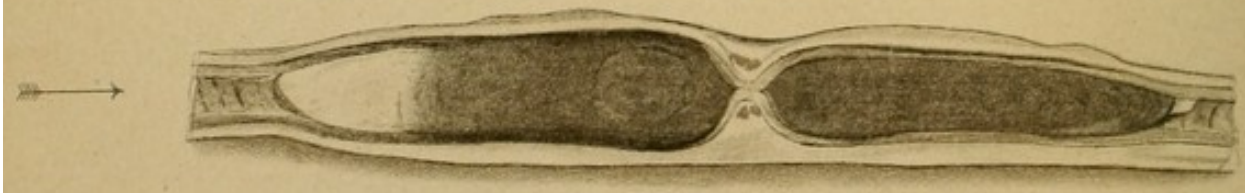

Fig. 4.

M. H. Lapidge del. ad. nat.

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Plate II.

Fig. 1. Common carotid of horse, 131 days after ligation. One kangaroo tendon. Surgical knot. Coats uninjured. Lumen occluded. Intima in apposition for 2 mm. Seat of ligature is now a firm fibrous mass both within and without the artery. The clot, except in the immediate neighbourhood of the ligature, is post mortem. The course of the walls of the vessel, as they pass through and are embedded in the fibrous mass, approaching, touching and receding from each other, can be readily followed in a microscopic preparation with a single lens magnifying 10 diameters. The same can be made out by the naked eye on close inspection. The indications in the drawing of the walls of the artery in the new tissue are more marked than natural. Exp. 53.

Fig. 2. Common carotid of horse, 165 days after ligation. One kangaroo tendon. Reef-knot. Coats uninjured. Intima not in apposition but 1 mm. apart. Lumen occluded by fibrous tissue. No doubt at the operation the first hitch of the reef-knot gave way while the second hitch was being tied: nevertheless the artery was permanently obliterated. It will be noticed that this artery and the preceding (fig. 1) are diminished in size: this is in consequence of their long occlusion. Exp. 54.

Fig. 3. Common carotid of horse, 14 days after ligation. One kangaroo tendon. Clove hitch (the ligature passing twice round). Condition of coats; intima query injured, middle coat intact. Intima not in apposition but 5 mm. apart. Lumen closed by diaphragm. The clove-hitch knot will not run on soft structures: the ends are therefore not jammed, and on tension being taken off, the constriction is relaxed. Exp. 50.

Fig. 4. Common carotid of horse, 14 days after ligation. Two kangaroo tendons. Senn's method: i.e. two reef-knots side by side, the cardiac being tied first. Coats uninjured. Lumen occluded. Intima in apposition for 3 mm. It will be observed that the cut ends of the cardiac ligature are more widely separated than those of the distal ligature. It is likely, from the appearance on close inspection of the artery, that the intima would not have been in apposition if the cardiac ligature alone had been applied. The application of the distal loop was rendered easier by the distending force being held in check by the cardiac ligature. Exp. 51.

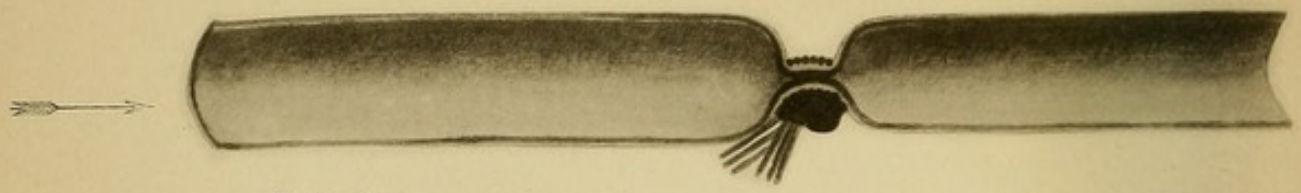


Fig. 1

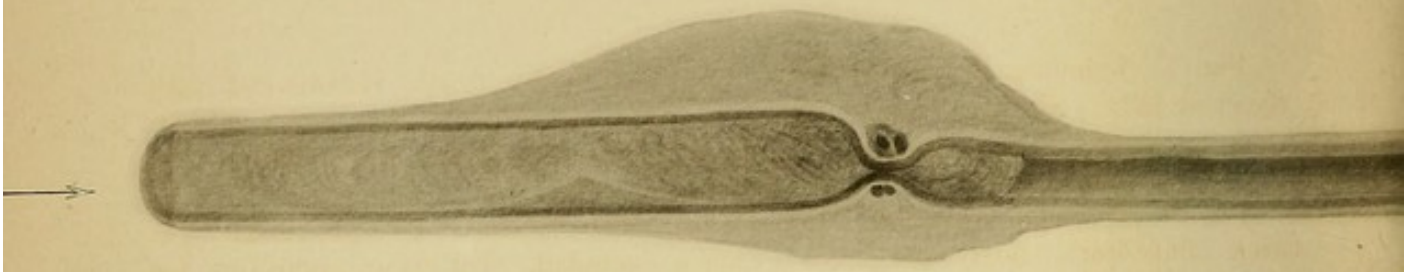


Fig. 2

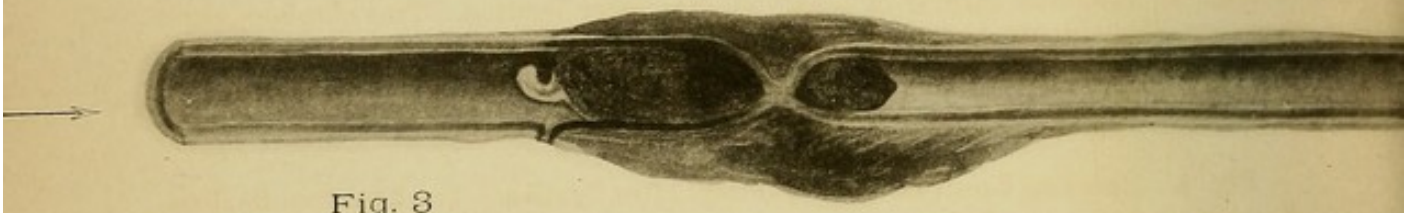


Fig. 3

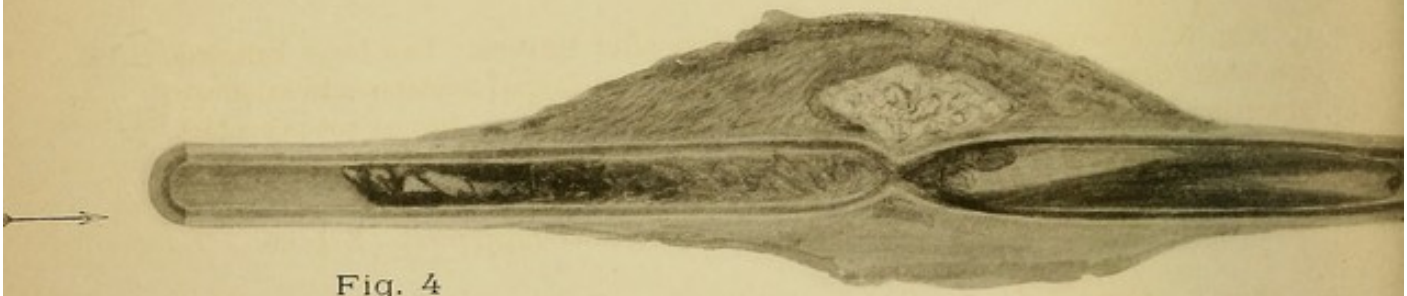


Fig. 4

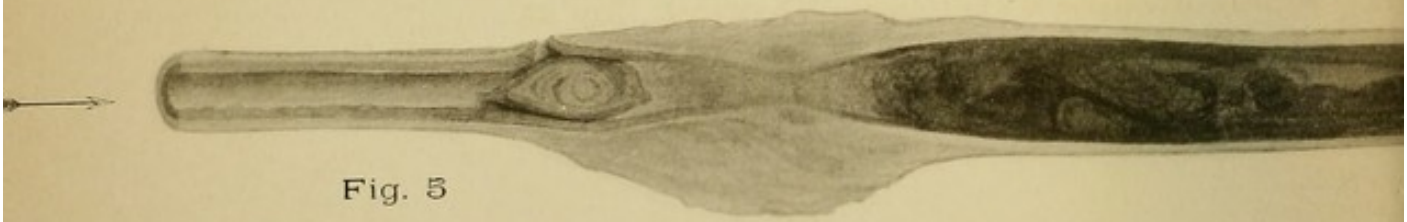


Fig. 5

Fig. 6

Plate III.

Fig. 1. Common carotid of horse, tied just before death, redistended with glycerine jelly. Six silkworm-gut ligatures. Stay-knot, completed as one reef-knot. Coats uninjured. Lumen occluded. Intima in apposition for 3·5 mm. Exp. 71.

Fig. 2. Common carotid of horse, 14 days after ligation. Two peritoneal ligatures. Stay-knot. Coats uninjured. Lumen occluded. Intima in apposition for 2 mm. Exp. 66.

Fig. 3. Common carotid of horse, 36 days after ligation. Two chromic-catgut ligatures. Stay-knot. Coats uninjured. Lumen occluded. Intima in apposition and firmly adherent for 2·5 mm. Clot on proximal side small in consequence of proximity of branch. No trace of ligature to naked eye. Exp. 69.

Fig. 4. Common carotid of horse, 36 days after ligation. Two kangaroo tendons. Stay-knot. Lumen occluded. Coats in apposition and adherent: walls embedded in new tissue inside and outside artery. Exp. 70.

Fig. 5. Common carotid of horse, 58 days after ligation. Two large kangaroo tendons. Stay-knot. Tendons swollen and soft by prolonged immersion in sublimate glycerine, therefore vessel imperfectly occluded at operation. Intima not in contact. Seat of ligature now mass of connective tissue. Uninjured coats dimly seen in it, as also remains of ligature. Branch given off on proximal side. Clot in neighbourhood deposited in concentric manner. Vessel impervious. Exp. 68.

Fig. 6. Carotid of horse, 55 days after ligation. Two peritoneal ligatures. Stay-knot. Ligature not much altered to naked eye. Coats uninjured. Lumen occluded. Intima in contact for 4 mm. About seat of ligature, inside and outside vessel, mass of connective tissue in which coats can be traced. Exp. 67.

CHAPTER I.

HEMORRHAGE IN MAN.

Importance of subject. Hemorrhage in man. Tabular view of statistics. Remarks on the great arteries. Records of London hospitals. Other statistics. Order of investigation.

Although the ligation of arteries has now been practised for upwards of twenty centuries, the best method of performing the operation still remains one of the great questions of surgery: the reason of this is not far to seek; it lies in the frequency in which, in this and every age, hemorrhage has followed the operation.

Much thought and ingenuity have been devoted to ascertaining the best mode of procedure, but no matter what has been the method chosen, and it has varied greatly in different times and in different hands, sooner

or later the dire calamity of hemorrhage has appeared, dashing the hopes of the surgeon and compelling him to consider anew the principles and practice of the operation; but the problem is by no means solved, hemorrhage still occurs with distressing frequency and the question remains as urgent as ever.

How frequent hemorrhage has been in times past we have no means of ascertaining, but for the present century there are statistical records and they tell of a grievous mortality. These statistics, however, present a far too favourable view of the matter, not only on account of the omission of unsuccessful cases, but also because many of the patients in whose cases it is recorded that no hemorrhage occurred, died of some other complication before the most critical period in the repair of the artery had been reached, and there is too much reason to think that had these patients lived longer hemorrhage would in many instances have occurred: however, even as they stand, the figures are very grave.

If to these cases be added those of aneurism which from fear of hemorrhage have been allowed to die without receiving such relief as surgery can offer, it will be seen how important the matter is, and how little apology is to be required of those who venture to approach the question once more.

The introduction of antiseptic surgery has no doubt diminished the danger: but it must not be forgotten that if the arterial wall be injured hemorrhage may occur in a perfectly aseptic wound: and in fact, it is well known that many cases of hemorrhage have occurred since the introduction of Listerian methods twenty years ago.

If asepsis be only maintained, the superficial femoral can probably be ligatured with success by any reasonable method, but the results with the other large arteries are still a matter, to say the least, of uncertainty: indeed the practice of surgeons with respect to the larger arteries, shews a want of confidence in asepsis surely averting hemorrhage.

It is unfortunately true that antiseptics have not always been efficiently used, but that is only a reason for not trusting exclusively to them. Moreover, if the employment of antiseptics is a complete solution of the problem, how comes it that the first part of the subclavian has never yet been tied successfully, and the innominate only once, and that too in the days before Lister? In abdominal surgery the great benefits conferred by asepticism are already fully enjoyed; the mortality from ovariotomy is now almost nil, and to open the abdomen has become one of the less anxious procedures in surgery: in the ligature of the great arteries on the other hand the gain is still mainly in the future, the danger of hemorrhage is still great, and the fact that it is so certainly suggests that there is still something wrong in our practice.

TABLE GIVING THE RESULTS OF THE LIGATURE OF THE MAIN ARTERIES AS SHOWN BY STATISTICS.

Artery	No. of cases	Hemorrhage		Death		Authority
		Total	Per cent.	Total	Per cent.	
Innominate	17	10	59	16	94	Erichsen and Beck, 1888
Common Carotid	789	144	18	323	40	Wyeth, 1888
Right Subclavian (1st part)	14	12	87	14	100	Erichsen and Beck, 1888
Subclavian (3rd part)	251	73	27	134	53	Wyeth, 1888
Abdominal Aorta	7	1	14	7	100	Erichsen and Beck, 1888
Common Iliac	55	?	?	41	75	Kümmel, 1884
Internal Iliac	24	?	?	15	62	Erichsen and Beck, 1888
External Iliac	141	24	17	31	22	Barwell, 1883
Common Femoral	31	18	58	16	51	Barwell, 1883
Superficial Femoral	277	46	16	54	19	Rabe, 1875

Remarks on Individual Arteries.

Innominate:—This artery was first tied by Mott in 1818; since the seventeen cases collected by Erichsen three more have been published, one each by Banks, Durante and Twyman: Banks's case died of hemorrhage from the first part of the subclavian (which was also ligatured), the innominate was found patent after death: Durante's case died of hemorrhage and Twyman's of coma: thus out of twenty cases only one recovered. This was that of A. W. Smyth, house surgeon to the Charity Hospital in New Orleans. It was a case of subclavian aneurism for which Smyth tied the innominate and common carotid simultaneously: severe hemorrhage occurred, for which the vertebral was ligatured: the patient then recovered and remained well for nine years when the aneurism recurred; the internal mammary was now ligatured and later the sac opened but without avail: the patient survived the first operation eleven years: at the autopsy the innominate and subclavian were found reduced to a fibrous cord extending from the point of ligature to the origin of the inferior thyroid.

Many of the cases died directly of hemorrhage, and it is probable that many of the others would have done so too had they not at an early date died of some other complication.

An instructive article by W. G. Spencer, founded upon experiments on monkeys, has recently appeared: he is no doubt right in advising in this operation the median incision and no division of muscles.

Common Carotid:—Wyeth's statistics are from Abernethy's case in 1803 down to 1879: Norris gives 20 hemorrhages in 149 cases: Pilz's statistics coincide: a case of hemorrhage from this artery after ligature in continuity recently came under our observation: the ligature was of silk and the artery gave way under the knot.

The first part of the Right Subclavian has been tied fourteen times, but never with success: of the cases twelve died of hemorrhage, one of pleurisy, and one of pyaemia, the last two on the fourth and fifth days respectively: it seems probable that had they lived longer they too would have had hemorrhage.

Wyeth gives 18 cases, all of which were fatal: besides these a fatal case has recently occurred in London. This artery was first tied by Colles in 1813.

Third part of the Subclavian:—Erichsen and Beck give 48 ligatures of this artery for axillary aneurism with 23 recoveries, and 25 deaths, three of which were caused directly by hemorrhage. Wyeth gives 75 ligatures for axillary aneurism with a mortality of 28 cases or 37 per cent. Barwell gives 90 ligatures for aneurism with 32 deaths—35·5 per cent.: of these deaths ten occurred directly from hemorrhage at the site of ligature.

Abdominal Aorta:—Ten ligatures, all of which terminated fatally (Wyeth): only one died of hemorrhage, that occurred on the tenth day: the others died earlier: the artery was first tied by Astley Cooper in 1817: he had previously ligatured it successfully in a dog, the specimen from which can still be seen in Guy's Museum.

Common Iliac:—Wyeth states that of ligatures for

aneurism 67 per cent. were fatal: Packard and Agnew arrived at a similar conclusion.

Internal Iliac:—This was first ligatured by Stevens of St Croix in 1812: the mortality is about the same as that for the common iliac.

External Iliac:—The first recorded ligature was by Abernethy in 1796: of 141 cases, collected by Barwell, hemorrhage occurred in twenty-four, of which fifteen died.

Norris gives 118 cases of ligature, of these thirty-three died: there was hemorrhage in fourteen, of which seven recovered and seven died: of the 118 cases seventy were for femoral aneurism, of them twenty-two died, four directly from hemorrhage. The mortality with this artery has been much less than that from the common iliac above, or from the common femoral below.

Common Femoral:—The 31 cases in the table were all tied for aneurism: hemorrhage occurred in eighteen, and was fatal in twelve: it has been thought by some that the numerous branches given off about Poupart's ligament constitute a reason for tying the external iliac rather than the common femoral: but if the arterial wall be not injured the nearness of the branches to the seat of ligature is not important: acting on this principle, Pitts has twice ligatured the common femoral with tendon and with success. The operation too has been successfully practised by Porter and Smyley in Ireland, by Laugier in France and Tiffany in America.

Superficial Femoral:—The cases in the table are all Hunterian ligatures for spontaneous aneurism. Suffice it now to add that the low mortality after ligature of the superficial femoral is no evidence of the success of the

operation, because many of the cases of failure recover after amputation.

The preceding statistics do not refer to one country only but are founded on all the cases that are recorded. If they be thought too bulky to grasp and we turn to the Reports of such London Hospitals as publish their results we find that they tell the same tale. By looking through some sixty volumes of annual reports published during the last twenty years, there may be found not less than thirty cases of hemorrhage after ligature in continuity.

Statistics published by individuals also agree: Hutchinson in 1856 collected 45 cases of ligature of large arteries for aneurism, among them hemorrhage occurred five times: twice from the common carotid, once from the subclavian and twice from the femoral. In 1860 he published thirty-two additional cases: amongst these were nine cases of hemorrhage as follow: two from the third part of the subclavian, one from the common iliac, two from the external iliac, one from the common femoral and three from the superficial femoral: thus in a total of 77 cases, hemorrhage occurred in 14.

Now the ligature of the superficial femoral artery is as a rule successful, and from its being the artery which is by far the most frequently ligatured for aneurism, the percentage of hemorrhage is kept down: of the above 77 cases 51 were ligatures of the superficial femoral and in three of these secondary hemorrhage occurred. If the ligatures of the superficial femoral be excluded there remain 26 ligatures with five hemorrhages, or 19 per cent.

of hemorrhages. Further, in these statistics are to be found 23 cases of popliteal aneurism which were cured by compression alone; if instead of this treatment the superficial femoral had been ligatured no doubt a very large proportion of the cases would have done well and the statistics of hemorrhage after ligature would have appeared still better. The prevalent idea that cases of ligature for aneurism do fairly well is entirely due to the combined success and frequency of ligature of the superficial femoral: if this artery be excluded the percentage of hemorrhage is very high.

Haynes, of Philadelphia, collected in 1874 all the recorded cases of ligature of large arteries in continuity with the carbolic catgut which was then in use: they were twenty in number, and in four of them hemorrhage occurred: namely, twice from the subclavian, once from the external iliac and once from the superficial femoral: of this last artery there were seven ligations, so that if these be omitted there remain 13 ligations with three hemorrhages—more than 20 per cent.

Bryant in 1878 recorded 10 ligations by himself of large arteries in continuity with catgut: in two cases hemorrhage occurred: once from the subclavian and once from the external iliac: five of the 10 cases were ligations of the superficial femoral; if these be omitted there remain five ligations with two hemorrhages.

Symonds in 1881 collected from the records of Guy's Hospital 35 cases of ligation of large arteries for aneurism: there were four cases of hemorrhage, once each from the common carotid, subclavian, external iliac, and femoral: 20 were ligations of the superficial femoral:

omitting these there are left 15 ligations with three hemorrhages, or 20 per cent.

Holmes in his lectures at the College of Surgeons in 1874 mentions 77 recent cases of primary ligature of the superficial femoral for aneurism, in only two of which hemorrhage is reported.

Cripps, writing in the same year, collected chiefly from the records of the preceding 30 years no less than 56 cases of hemorrhage from the superficial femoral after ligature in continuity, the operation being performed in nearly every instance for aneurism: he calculates that secondary hemorrhage occurs in 8 per cent. of the cases of ligature of this artery: it may be objected that a large number of the cases occurred before the introduction of antiseptics (in 1870); but Walsham gives four cases of hemorrhage occurring in 54 ligatures which were performed in the years 1870—1885: quite recently Howard Marsh has referred to two cases of secondary hemorrhage since Walsham's paper: and generally speaking, it is well known that cases of secondary hemorrhage still occur from time to time, indeed they may be found recorded in the weekly journals: only the other day another case occurred in London.

The ligature of the vertebral for epilepsy has been practised wholly within the antiseptic era, and yet hemorrhage has occurred once certainly and possibly twice in the course of about 80 ligatures of this small artery.

It will no doubt be said, that all statistics are misleading; but, as Billroth points out, those of hemorrhage understate the case: he reports 23 ligatures in continuity of large arteries in the hospitals of Mannheim and Weissen-

burg during the Franco-German War: three died of hemorrhage, four recovered after hemorrhage, seven recovered without hemorrhage, and nine died of other causes; hemorrhage occurred therefore in 30 per cent. of the cases: but, as Billroth goes on to say, "no one can tell "whether these nine who died of other causes would have "had hemorrhage or not, had they lived, therefore they "should be left out of the calculation:" if this be done, the numbers will stand thus, 14 ligatures, 7 cases of hemorrhage, that is to say, secondary hemorrhage in 50 per cent. of the cases.

To find out and avoid the causes of hemorrhage after ligation in continuity, we have for our guide in the first place the example of Nature, for she, to carry out the necessary changes in the circulation at birth, has herself to close certain vessels: physiological occlusion, therefore, must be our first study. Pathological occlusion (as it occurs apart from surgical interference) will come next. Then the various changes, great and small, which in the artery, clot and ligature, follow ligation, must be investigated by experiments on the lower animals. Finally, from the knowledge thus obtained, and in the light of the experience of those who have gone before, a choice must be made from among the various methods of operating of that which seems least likely to be followed by the sad sequel of hemorrhage.

CHAPTER II.

VALUE OF EXPERIMENTS.

Objections stated. Processes identical in man and brutes. Hemorrhage in brutes. Remarks of Scarpa and Jones. Anaesthetics. Conclusion.

As the conclusions that are arrived at in the following pages depend largely on the results of experiments on the lower animals, it will be well, at the outset, to meet the objection that the tissues and constitutions of animals are so different from those of men that these experiments are of little value.

It is hardly to students of surgery, to whom this volume is addressed, that we need demonstrate the value of experiments upon the lower animals in order to elucidate problems in pathology or in surgery. It will be sufficient

for the present to assert that the processes of healing and inflammation are for all practical purposes identical in the higher animals which we have employed, with that which is observed following upon the wounds of men. So far as we know there is no foundation for a contrary opinion. We do not stay to prove that secondary hemorrhage is as frequent in the lower animals as in man; or that all pathological changes are to the last detail identical in man and brutes; but we do maintain that the broad characteristics of the constructive process and the elemental actions proceeding in a ligatured artery are the same, be the vessel in a horse, an ass, a sheep, or a man. It may also be mentioned that diseases of arteries common in man, such as atheroma, are not rarely met with in old horses, their aortæ for example becoming greatly thickened, dilated and

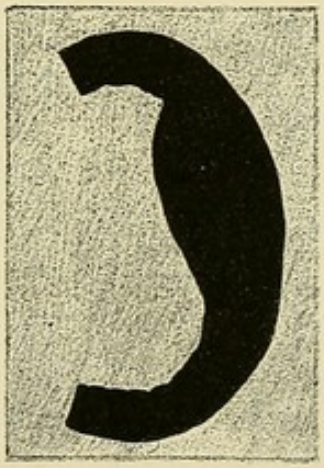


FIG. 1. Aorta of horse. (Size of specimen.)

Portion of a transverse section of the thoracic aorta of an old horse. The diseased state of the vessel is evident from the variations in the thickness of its wall.

irregular (Fig. 1), and in small rodents, disease of the arteries has been produced experimentally, for Israel, to

give one example, studied endarteritis in rabbits by causing contraction of one kidney (Fig. 2).

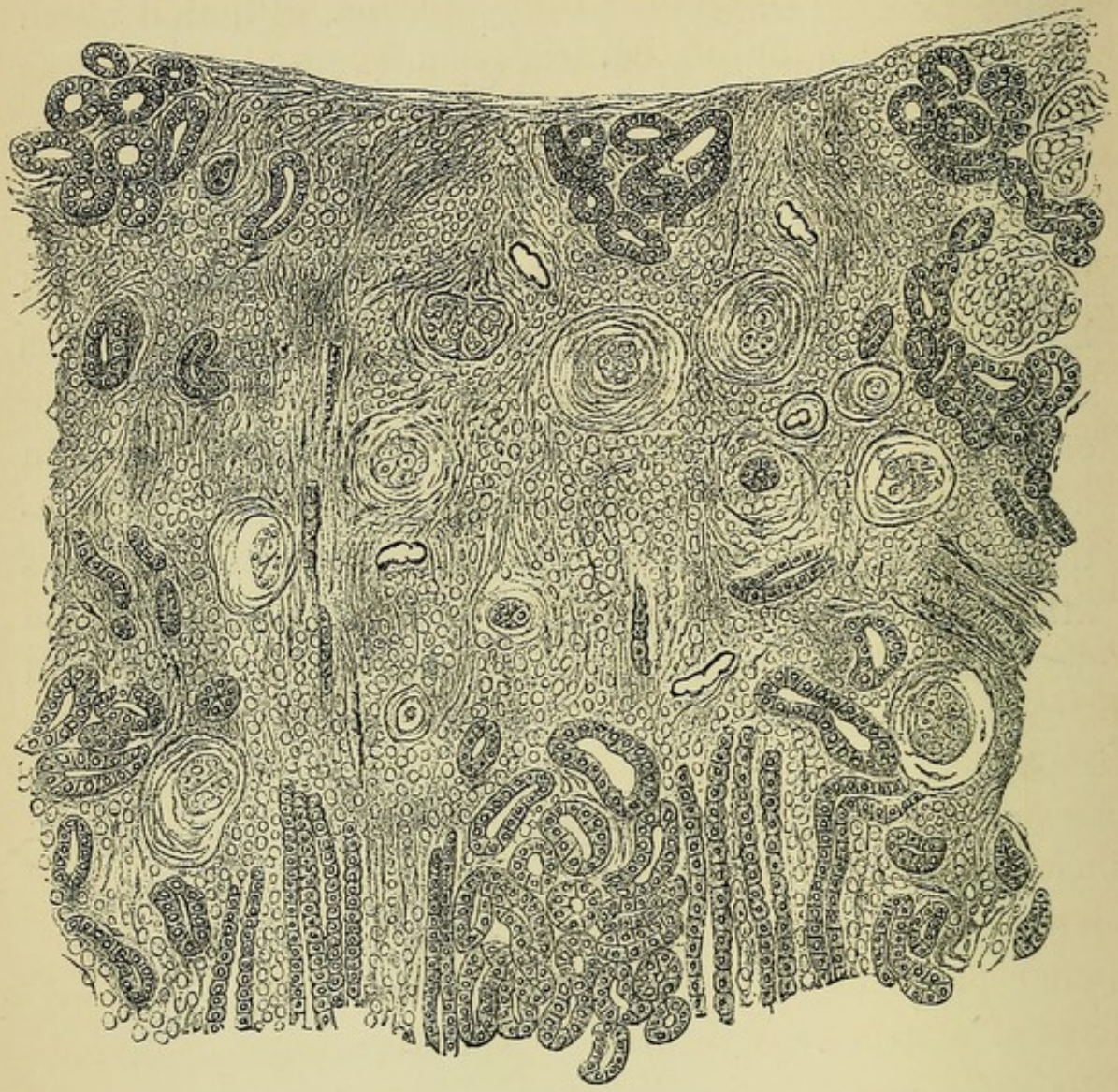


FIG. 2. Chronic Nephritis in the rabbit.

Reproduced by kind permission of Dr Oscar Israel.

The vessels are becoming blocked by growth of endothelium and clotting: precisely the same appearance is seen in human arteries in figures 46 and 47. Israel also figures endocarditis and endoaortitis in the rabbit.

From Virchow's *Archiv für path. Anat. und Phys.* Band 86 (1881).

With respect to the formation of aneurism in the arteries of brutes there is to be seen in the Hunterian

Museum an aneurism of the aorta of a turtle (No. 3150) and also an aneurism of the abdominal aorta of a jaguar (No. 3212). Mr Harrison Cripps informs us that he has known a hound die of thoracic aneurism, and Mr Jones, Veterinary Surgeon of Leicester, says that he has seen an aneurism in the thigh of a horse as large as a child's head. William Williams in his work on Veterinary Surgery states that many aneurisms in horses are recorded and that Bruckmüller of Vienna found verminous aneurism of the mesenteric artery in 91 per cent. of the horses on which he made a post mortem examination. Williams too found an aneurism of the mesenteric or cæliac arteries in every ass which he examined: the vessels were thickened, calcareous and aneurismal: the disease being due to a parasite called Strongylus Armatus. Cobbold agrees with this. Williams also has seen many cases of aneurism of the aorta in dogs. "Staggers," too, in horses is stated to be due to degenerative changes in the cerebral arteries. The accompanying illustration (Fig. 3) shews the iliac and femoral arteries of a horse diseased, thrombosed and aneurismal: a somewhat similar preparation shewing thrombosis of both common iliacs of a deer may be seen in the Hunterian Museum (No. 3267).

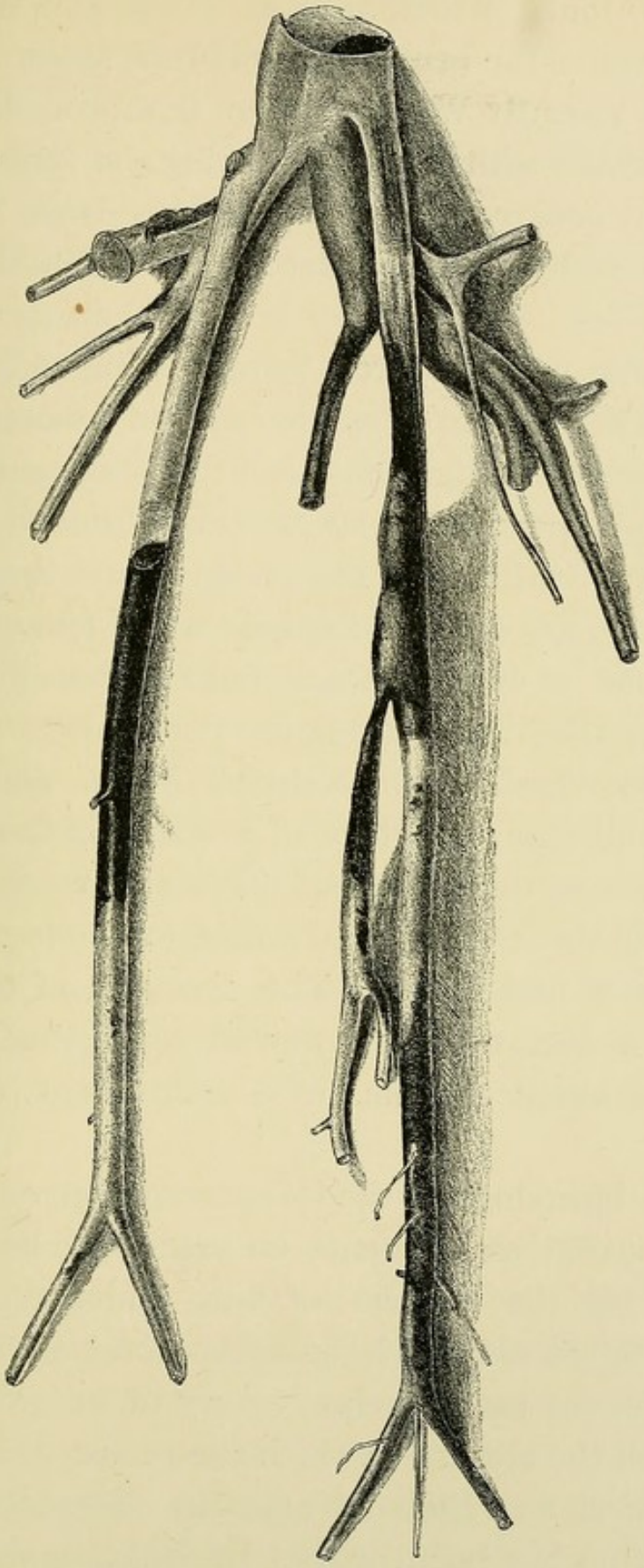
The picture which we shall attempt to draw of the processes which happen in, say, a ligatured horse carotid are identical with those which the microscope reveals to us when we have the opportunity of examining human arteries under like conditions.

It may further be remarked that not only has hemorrhage dogged the footsteps of the surgeon in operations upon man, but the same pathological event has followed the ligation of the arteries of brutes.

FIG. 3. The iliac and femoral arteries of a horse.
($\frac{1}{3}$ nat. size.)

The vessels are diseased, thrombosed and aneurismal. The horse was 4 years old and the arteritis followed and was possibly due to an attack of "strangles."

Drawing reproduced from a water-colour sketch by Clark Stanton now in the possession of the Brown Institution: see also *Edinburgh Veterinary Review*, 1858, page 147.



Pouteau, Jones, Mislei, Travers, Porta and Warren record such events, the brutes being horses, asses, goats, and dogs. Only recently W. G. Spencer ligatured the innominate of a monkey with catgut, rupturing the tunics; a little suppuration occurred, and seven days later secondary hemorrhage suddenly took place from which the animal died.

Jameson, Howard, Barwell, Senn, Stirling and Dent, and ourselves, it is true, have had no cases of hemorrhage following experimental ligations; but then Jameson, Howard, Barwell, Senn and ourselves were careful not to injure the arterial wall. Stirling and Dent did rupture the coats, but their experiments were conducted on the femoral arteries of sheep and it by no means follows that if they had operated in that way on horses that they would have escaped the disasters which befell Mislei and Travers. Moreover the femoral artery of a sheep is too small for the purposes of the argument. For this reason we gave up the carotids of sheep and made our experiments on those of asses and horses. The diameter of the carotid artery of a sheep when removed from the body and collapsed is about 5·5 mm., of a man 7 mm., of a horse 12 mm.

Scarpa, speaking of the Hunterian operation, says: "After repeated experiments on animals I had no hesi-"tation as to the success of this undertaking in the "human subject also. The investigation was directed to "the ligature of the principal artery of an extremity, to "accomplish the closure of which the powers of life in man "are not inferior to those of animals. The correctness of "my induction has been proved by facts, as shall appear

"from the histories of the three operations performed by "Paletta at Milan, followed by a fourth operation at "Pavia."

These experiments may be also objected to on moral grounds: on this subject we cannot do better than quote the words used by F. J. D. Jones in his classical work.

"The author has," he writes in his preface, "only a "few more words to say addressed to men out of the pale "of his profession into whose hands this little book may "fall whose opinions he esteems and whose sentiments he "honours. He regrets the necessity of obtaining even this "important knowledge by the sacrifice of brutes. But "when we remember the incessant scourge of war which "has followed man through all the ages of his history, not "to mention the consequences of accident and disease, it "is not too much to assert that thousands might have been "and may still be saved by a perfect knowledge of these "subjects; which can only be directly obtained by experi-"ments on brutes; indirectly, and very slowly, by observa-"tions on the injured arteries of man; and even these "cannot be made, until he has fallen a sacrifice to the "want of assistance or to the imperfect knowledge of the "surgeon."

Since Jones's time anaesthetics have been discovered, and they were of course used in our experiments.

Sometimes we employed chloroform and sometimes ether, and there is no difficulty in administering these to animals. Chloroform is, however, much more dangerous, for under its influence we have lost horses, asses and sheep, whilst with ether we have only lost one sheep. Latterly we have only used ether and have had no fatalities. From

the time the animals recovered from the anaesthetic they took their food well and did not appear to be inconvenienced.

The moral question involved is not, however, whether experiments on animals are justifiable, but whether it is right to perform operations on man without being first satisfied of their feasibility, and endeavouring to perfect them and save life by performing experiments on animals.

Every advance in surgery must be at first of the nature of experiment, and the only question is whether the experiment shall be made on man or brutes.

CHAPTER III.

NATURE OF ARTERIES.

Minute anatomy. Differences due to age. Outer coat. Middle coat. Inner coat. Vasa vasorum. Elasticity. Drawings of walls of human arteries. Measurements of the collapsed coats. Experiment of Roy. Tenuity of distended walls. Measurements and scale drawings. Effect of different pressures. Nature of change in wall. Longitudinal tension.

It is desirable, before entering on our subject, to make one or two remarks on the minute anatomy of the vessels. The structure of the larger arteries, with which alone we are now concerned, differs somewhat from that of the smaller vessels. Thus, if the common carotid be compared with the radial, it will be seen that the wall of the larger vessel is proportionately thinner, and that while in the radial the demarcation of the inner coat from the middle is clearly marked by the fenestrated membrane of

Henle, in the carotid the distinction is not so clear on account of elastic membranes being present in great numbers in the middle coat, and extending into the sub-endothelial layer of the inner coat.

In the measurements of the thickness of the arterial coats, we have always taken the innermost continuous elastic film as the boundary between the inner and middle coats: the thickness of the arterial wall is due mainly to the middle coat: it is said that in addition to certain sexual and individual differences, the calibre of the large arteries, and the thickness of their walls, increases gradually with advancing years: the following table gives the thickness of the three coats of certain arteries when collapsed, from two men, one aged 25, the other 45 years. The numbers refer to hundredths of a millimetre, and were estimated from microscopic sections by means of the camera lucida.

Artery	Man aged 25				Man aged 45			
	Outer coat	Middle coat	Inner coat	Total	Outer coat	Middle coat	Inner coat	Total
Innominate	5	73	20	98	10	109	9	128
Common Carotid	3	60	15	78	4	79	18	101
1st part Subclavian	5	50	18	73	5	49	15	69
3rd part Subclavian	7	51	5	63	9	74	8	91
Superficial Femoral	20	45	7	72	35	74	18	127

It will be noticed that the first part of the subclavian appears to be an exception.

The outer coat of the larger arteries is composed of a closely-woven network of connective tissue, in which numerous elastic fibres can be seen, which have connections with the elastic laminae of the middle coat. This coat in the large arteries is proportionately thinner than in the smaller vessels, and containing many more elastic fibres is more brittle.

The middle coat of the larger arteries, such as the common carotid, consists of elastic films, twenty or more in number, quite similar to the fenestrated membrane of Henle; the actual number of these films has been given as follows: aorta 65: innominate 20: common carotid 23: subclavian 15: axillary 13, and common iliac 13: these are disposed at equal distances, and united with each other by separate elastic fibrils passing obliquely between the muscular bundles: these elastic membranes alternate with thin muscular layers: bundles of white connective tissue occur also in the middle coat, the proportion increasing with the size of the artery: with these fibres are associated a corresponding number of connective tissue corpuscles: these, when stimulated in any way, will proliferate and form connective tissue. Warren, in his work on the Healing of Arteries, endeavours to shew that the most important factor in cicatrization is the multiplication of the involuntary muscular cells: but with this portion of his conclusions we are unable to agree: the muscle cell can be distinguished from a connective tissue corpuscle, by its distinct rod-shaped nucleus, and the presence in its protoplasm of a linear series of granules at the extremity of the rod, on the other hand the nucleus of the connective

tissue corpuscle when fixed by osmic acid is always large, oval, and vesicular, as will be fully described in the chapter on the Conduct and Fate of the Corpuscles.

The inner surface of the inner coat is composed of a continuous plate of connective tissue cells, beneath which is a delicate fibrillated layer, in whose numerous spaces lie branched cells of a like character: so that the inner coat, as far as its power of growth is concerned, may be thought of as made up of layer upon layer of connective tissue cells: the elastic element predominates in the framework which supports these cells: and according to Schäfer, the chief substance of the intima in the larger arteries is formed of elastic tissue: on section the fenestrated membrane of Henle is seen as a wavy line, as it forms the outermost portion of the inner coat: this wavy line is not present during life, for then the pressure of the blood stretches out the membrane.

This may be seen by injecting a vessel with wax under the pressure of the blood: sections will shew that all the wavy membranes are now straight: in the same way it can be shewn that the crinkled contour of the endothelial cells of the pleura on the apex of the lung is a post-mortem appearance, for if the lung be distended their outer border is found even.

The vasa vasorum ramify through the outer coat: observers differ as to whether or not they extend also into the middle coat: we hold that they do: it has even been stated that capillaries may be seen in the intima. Senn says that, "whatever may be the exact truth, it is evident "that the intima, though devoid of blood-vessels, yet "receives sufficient nutritive supply to render it capable of

"regenerating its own elements in case of loss, and to "assume inflammatory changes like other similar meso- "blastic structures:" these changes will of course be accompanied by the growth of new capillaries into the inner coat.

The following illustrations shew the construction of the principal arteries of man: the sheath of each vessel having been removed as it would be in the ligature of an artery. They are transverse sections and the magnification is one hundred times, except where otherwise stated. In the drawings the outer coat is placed uppermost, and the brackets indicate the limits of the three coats.

List of Arteries drawn.

Aorta near Valves.
Innominate.
Common Carotid.
Facial.
Middle Meningeal outside Skull.
Middle Meningeal in Groove of Sphenoid.
Contents of Parietal Groove.
Middle Meningeal in Groove of Parietal.
Basilar.
Arteria Centralis Retinæ external to nerve.
Arteria Centralis Retinæ in nerve.
Subclavian first part.
Subclavian third part.
Axillary second part.
Brachial.
Ulnar.
Radial.
Abdominal Aorta near bifurcation.
Superior Mesenteric.
Common Iliac.
Umbilical at birth.
Uterine of virgin.
Uterine at term.
Dorsalis Penis.
External Iliac.
Common Femoral.
Superficial Femoral.
Popliteal.
Posterior Tibial.
Nutrient of Tibia external to bone.
Contents of Nutrient Foramen of Tibia.
Nutrient of Tibia in Foramen.
Anterior Tibial.

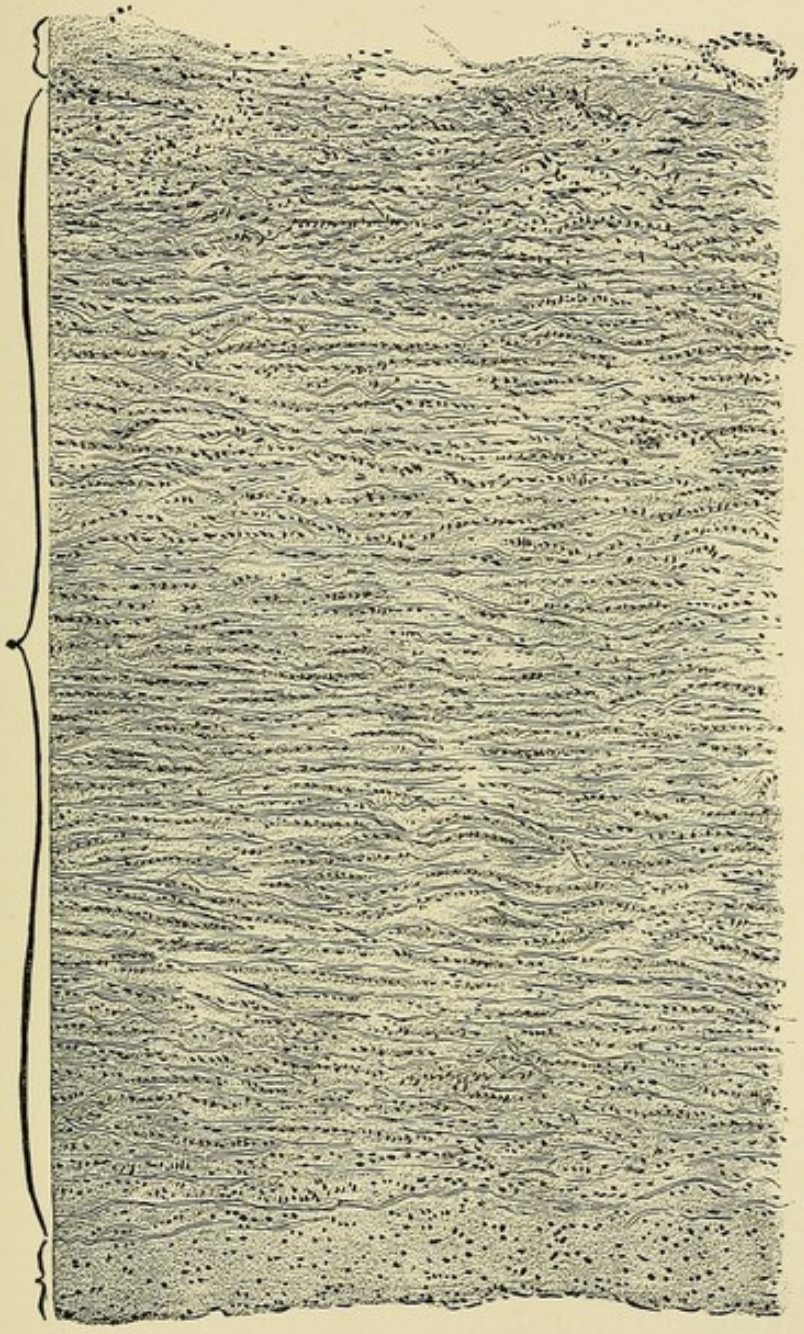


FIG. 4. Aorta near Valves.

Outer coat very thin.
Middle coat very stout.

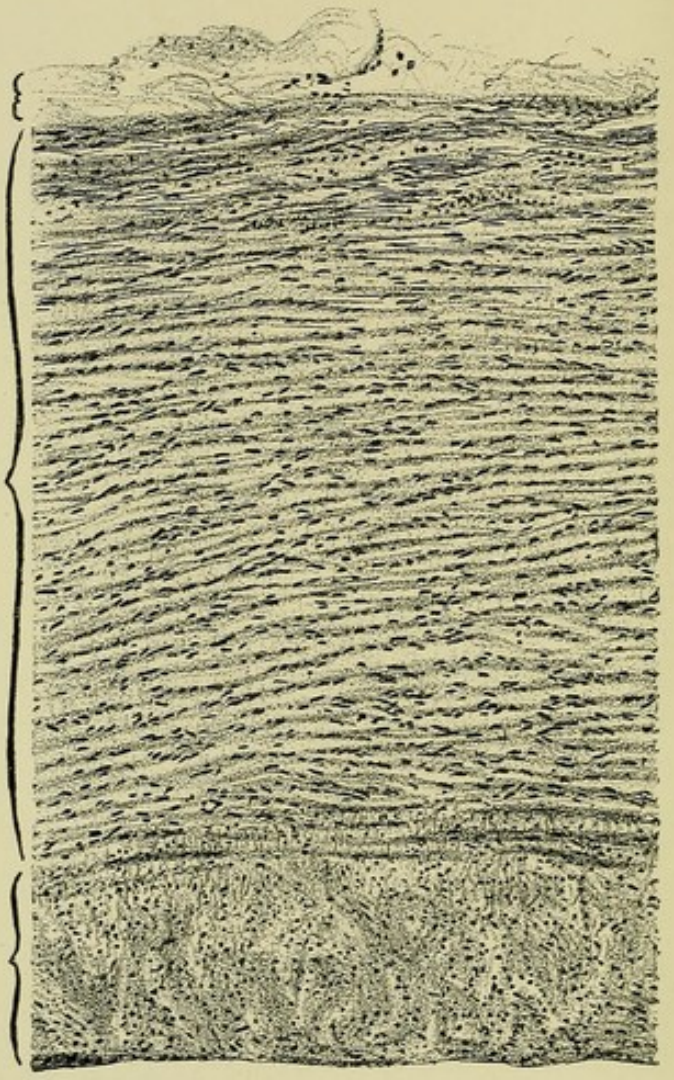


FIG. 5. Innominate.

Outer coat very thin.
Middle coat stout.
Inner coat varies much in thickness.
Drawing shews a thick part near bifurcation: vessel was apparently normal.

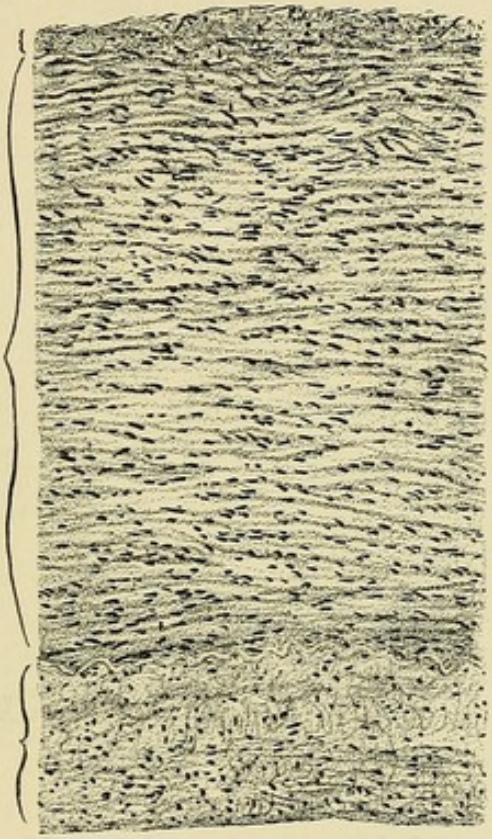


FIG. 6. Common Carotid.

Outer coat thin.
Middle coat thick.
Inner coat varies greatly: sometimes, as represented, very thick, sometimes very thin.

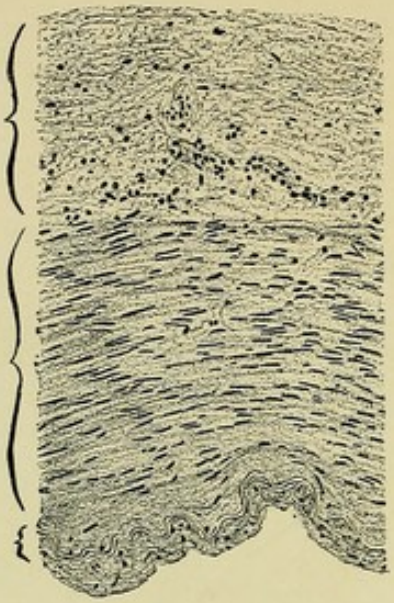


FIG. 7. Facial.

Outer coat very thick.

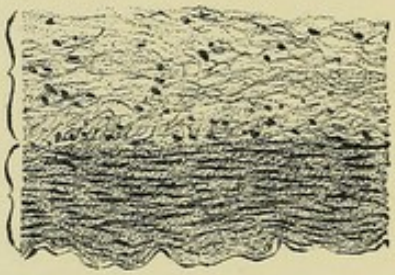


FIG. 8. Middle Meningeal outside Skull.

Outer coat very thick.

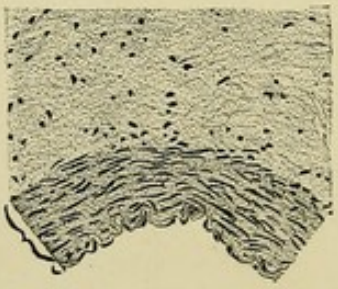


FIG. 9. Middle Meningeal in Groove of Sphenoid.

Outer coat continuous with dura mater.

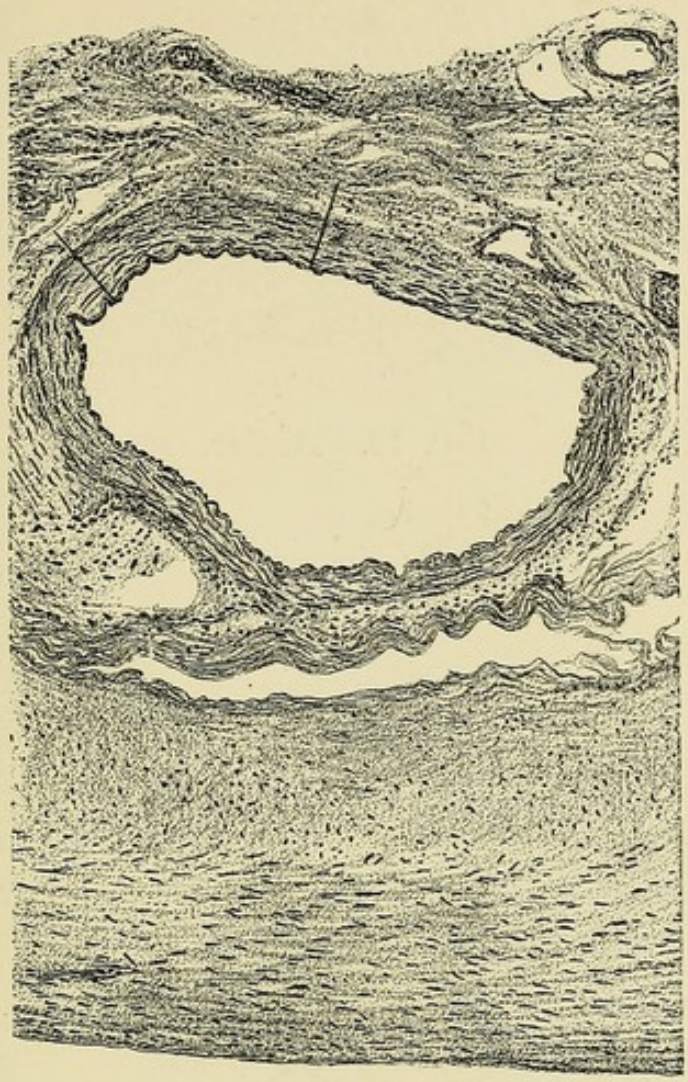


FIG. 10. Contents of Parietal Groove (× 75).

Dura mater below: groove above.

The portion marked off in black lines is seen × 100 in next figure.

FIG. 11. Middle Meningeal in Parietal Groove.

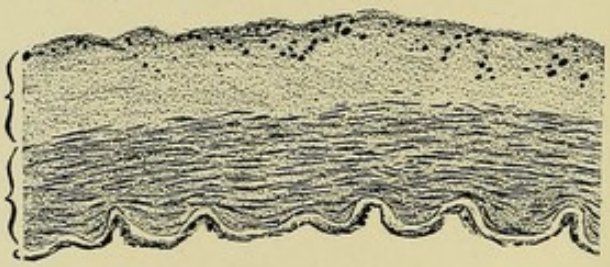


FIG. 12. Basilar.

Outer coat thick.
Inner coat thin: a single layer of cells.

FIG. 13. Arteria Centralis Retinæ outside nerve.

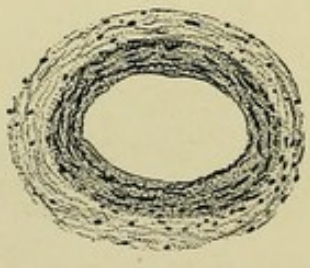


FIG. 14. Arteria Centralis Retinæ within nerve.

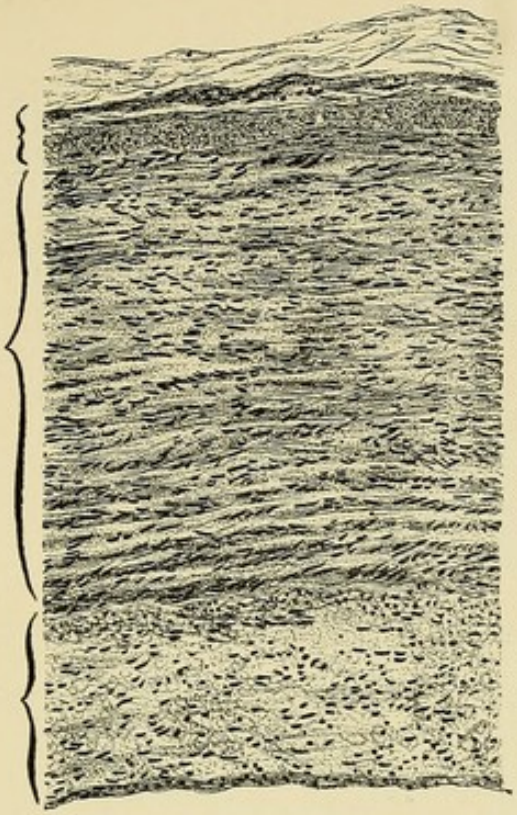


FIG. 15. First part of Subclavian.

Outer coat very thin.
Inner coat varies: thick (as shewn in figure) near origin of branches.

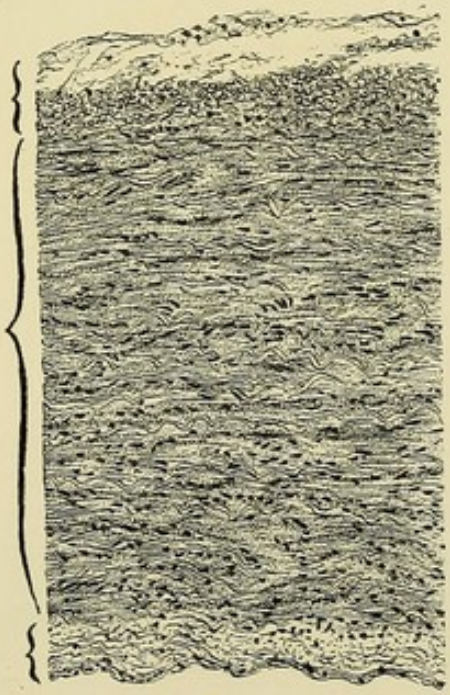


FIG. 16. Third part of Subclavian.

Outer coat thin but thicker than in first part of artery.

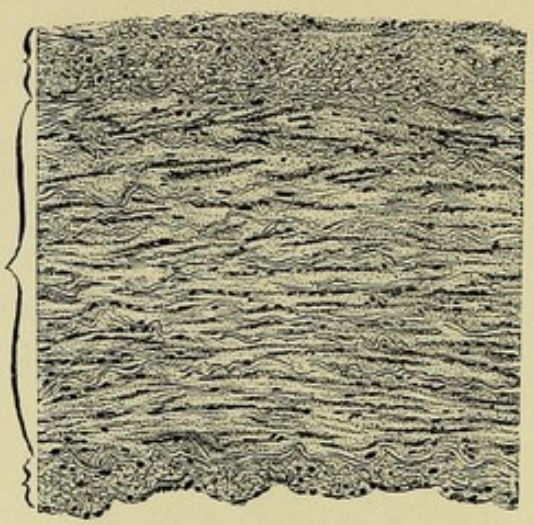


FIG. 17. Second part of Axillary.

Outer coat fairly thick: at one part of all transverse sections thinner: query opposite humerus?

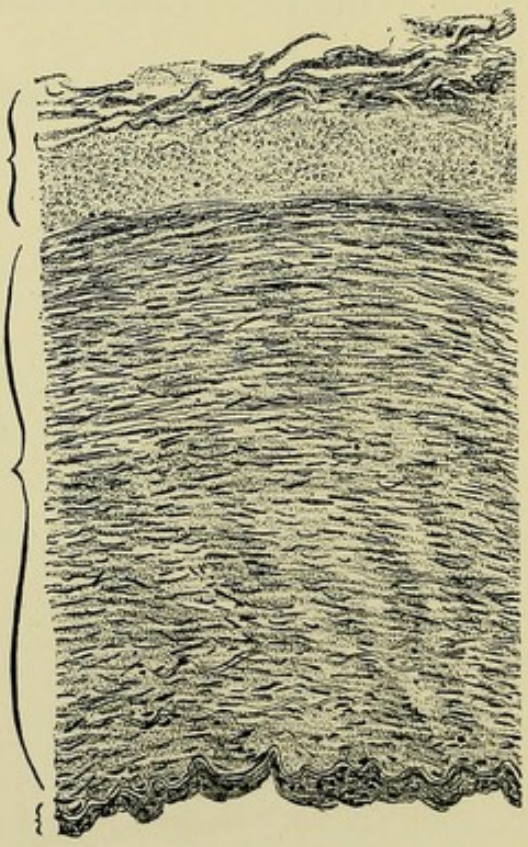


FIG. 18. Brachial.

Outer coat thick.

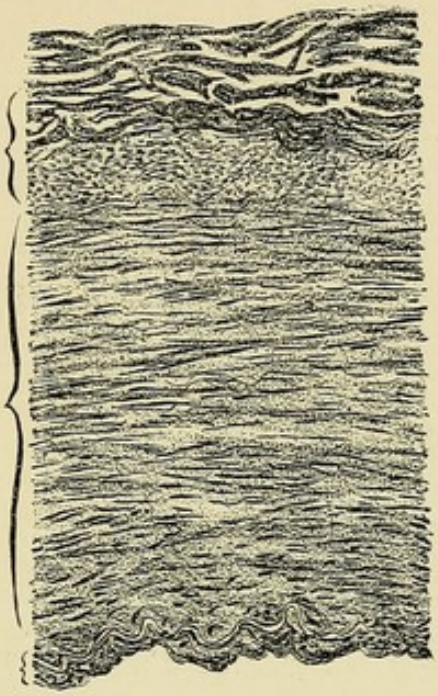


FIG. 19. Ulnar.

Outer coat thick.

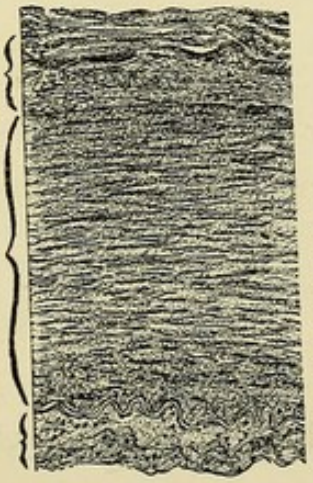


FIG. 20. Radial.

Outer coat thick.

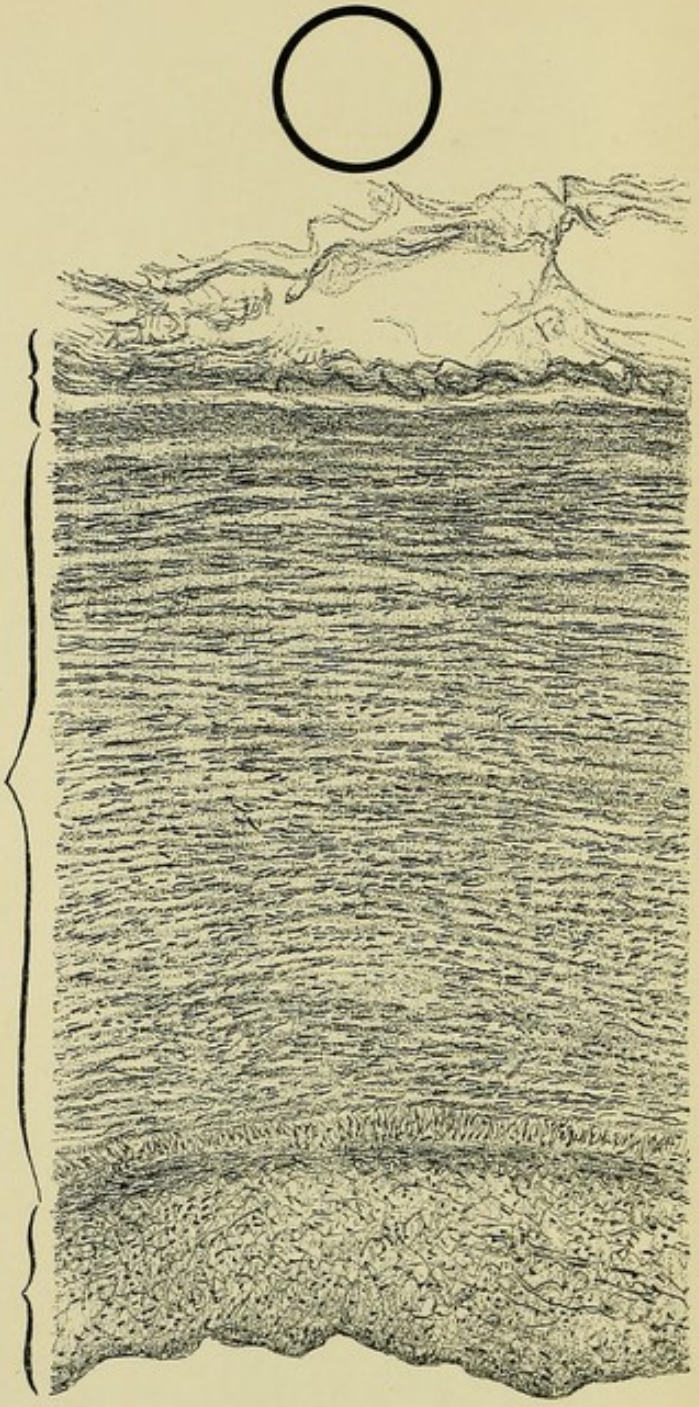


FIG. 21. Abdominal Aorta near termination.

Outer coat very thin.
Inner coat varies greatly: thick (as represented) at bifurcation.
Circle above represents post-mortem thickness: compare with Common Femoral.

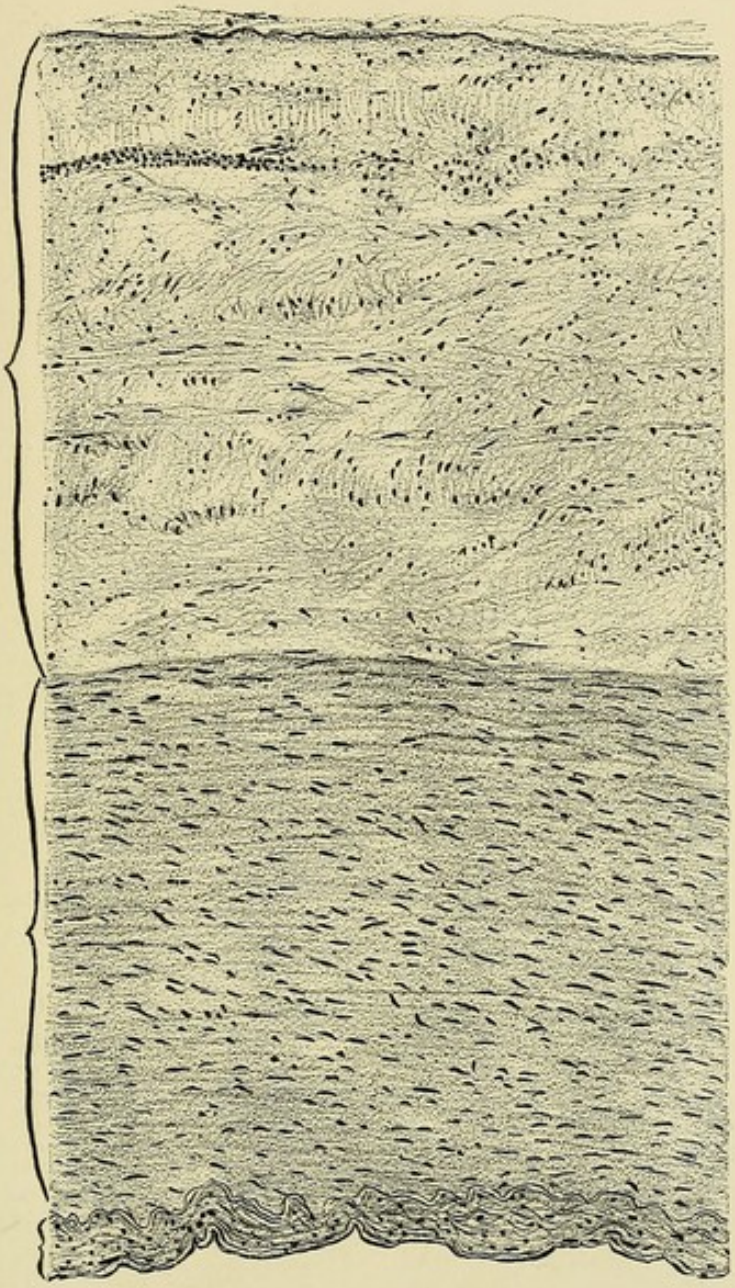


FIG. 22. Superior Mesenteric.

Outer coat very thick: the thickest in the body: half the thickness of the artery.

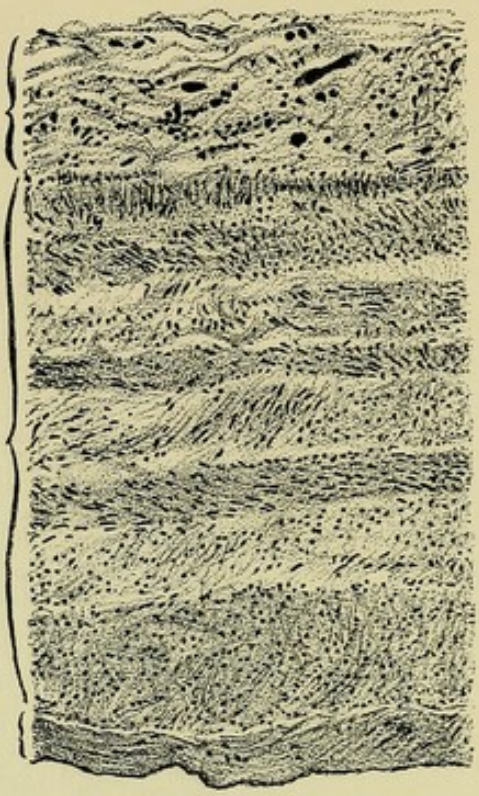


FIG. 23. Common Iliac.

Outer coat well marked.

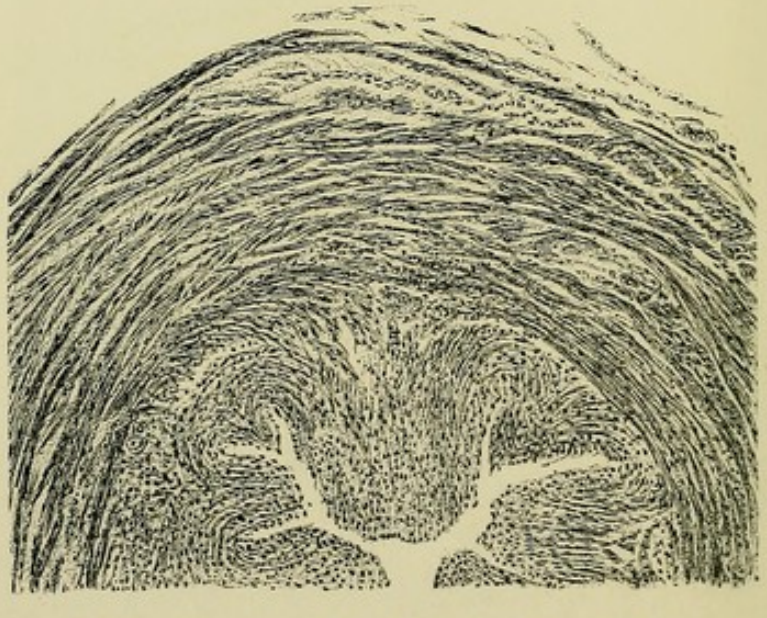


FIG. 24. Umbilical at birth (× 65).

Vessel collapsed.
Inner coat very thick and folded.

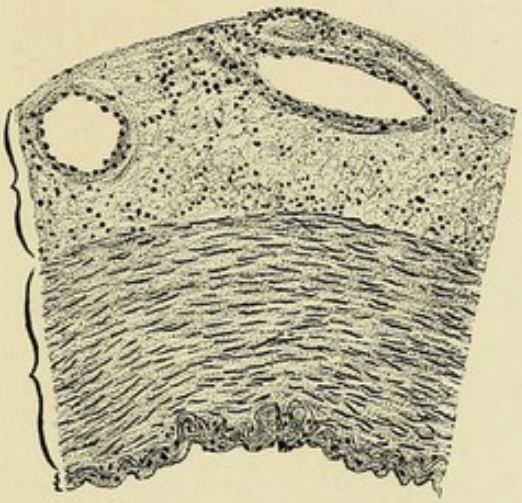


FIG. 25. Uterine of Virgin (ætatis 18).

Outer coat thick: contains large vessels.

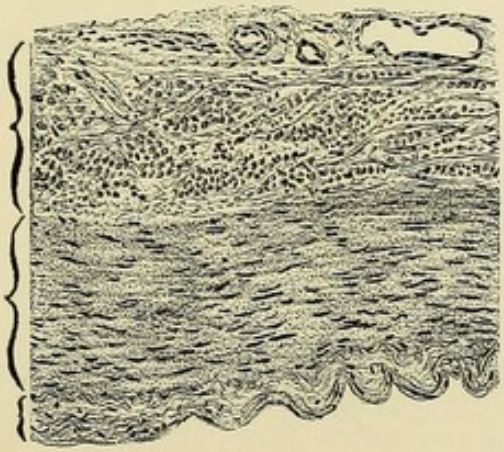


FIG. 26. Uterine at term.

Outer coat thicker and denser than in virgin.

FIG. 27. Dorsalis Penis.

Outer coat very thick.

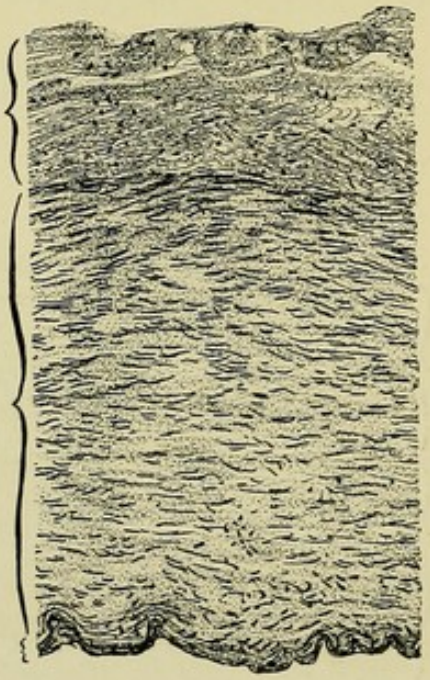


FIG. 28. External Iliac.

Outer coat moderately thick.

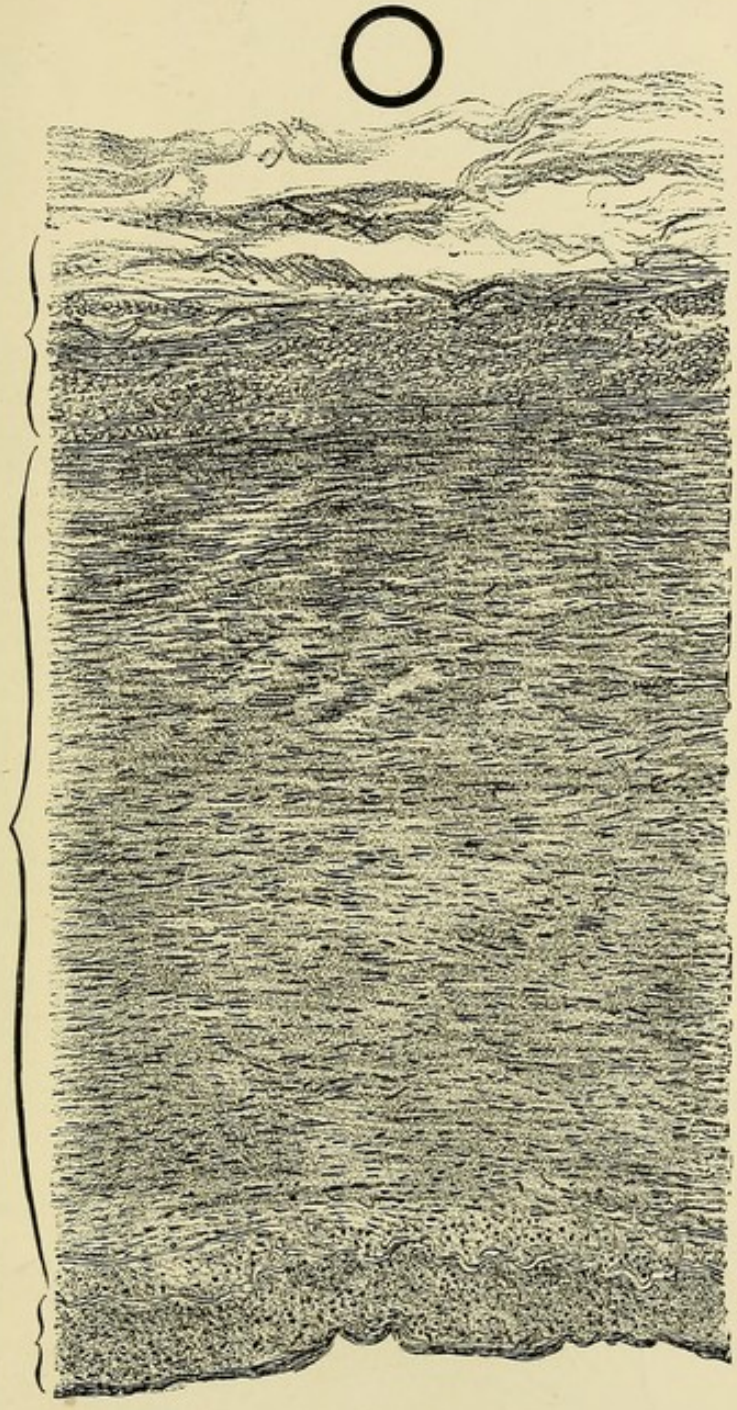


FIG. 29. Common Femoral.

Outer coat very thick: this artery is one of the stoutest in the body.

The circle above gives the actual thickness of the vessel when collapsed, it is thicker than the Abdominal Aorta.

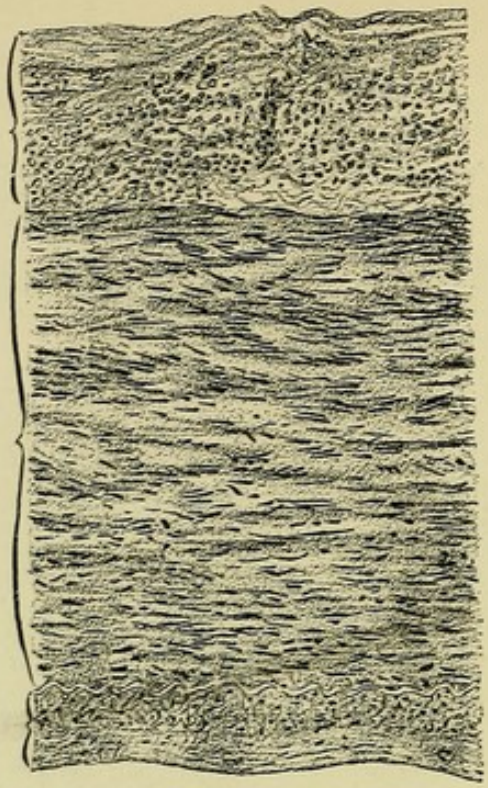


FIG. 30. Superficial Femoral.
Outer coat very thick.

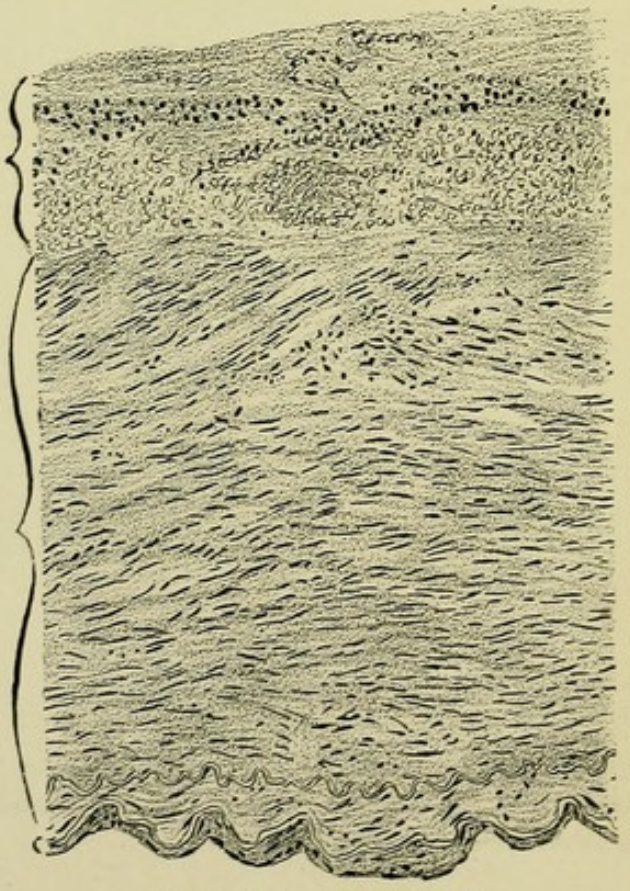


FIG. 31. Popliteal.
Outer coat thick.

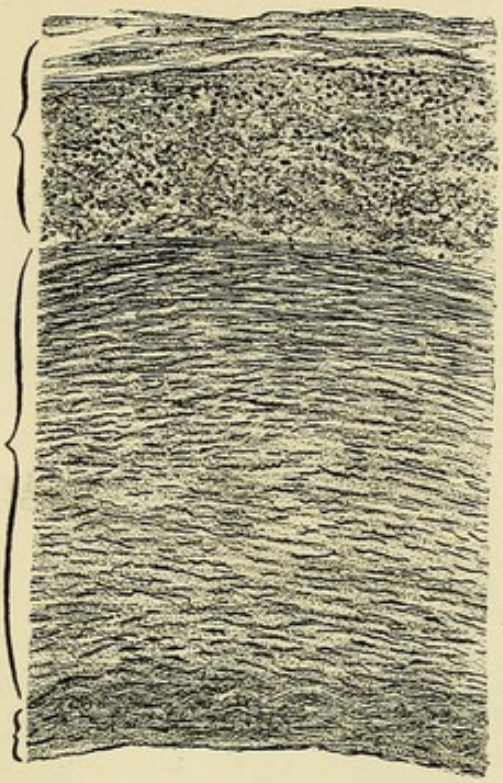


FIG. 32. Posterior Tibial.

Outer coat very thick.

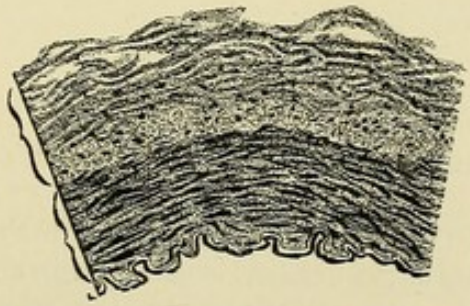


FIG. 33. Nutrient of Tibia external to bone.

Outer coat very thick.

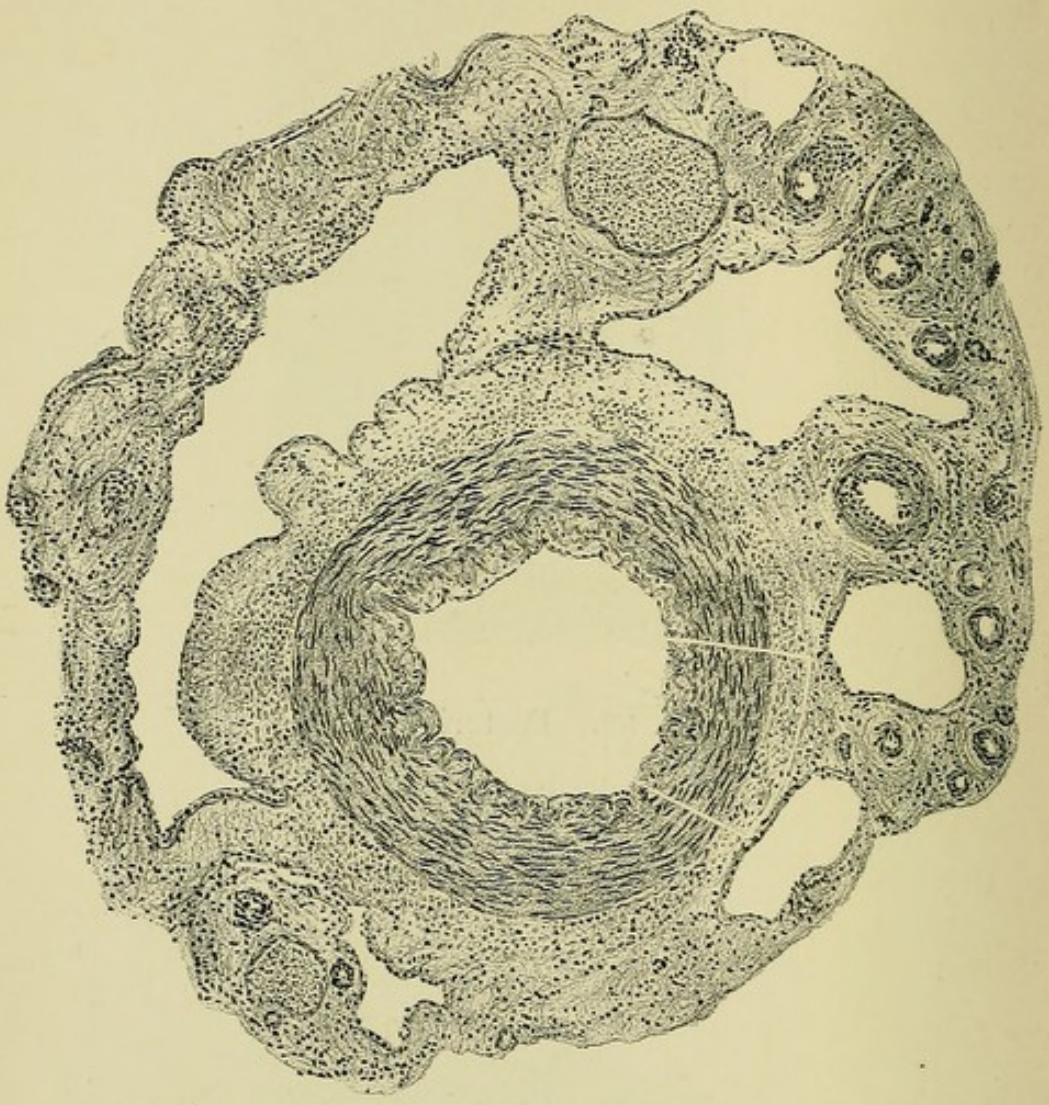


FIG. 34. Contents of Nutrient Canal of Tibia (×75).

The outer coat of the artery is continuous with the periosteum: the section shews the whole contents of the nutrient canal in transverse section: there are to be seen the artery with its venæ comites, also nerves and a few small vessels in the periosteum: the part marked off by white lines is shewn in the next figure.

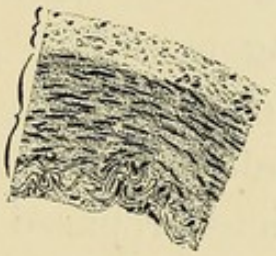


FIG. 35. Nutrient of Tibia in bony canal (× 100).

Portion marked off by white lines in preceding figure.

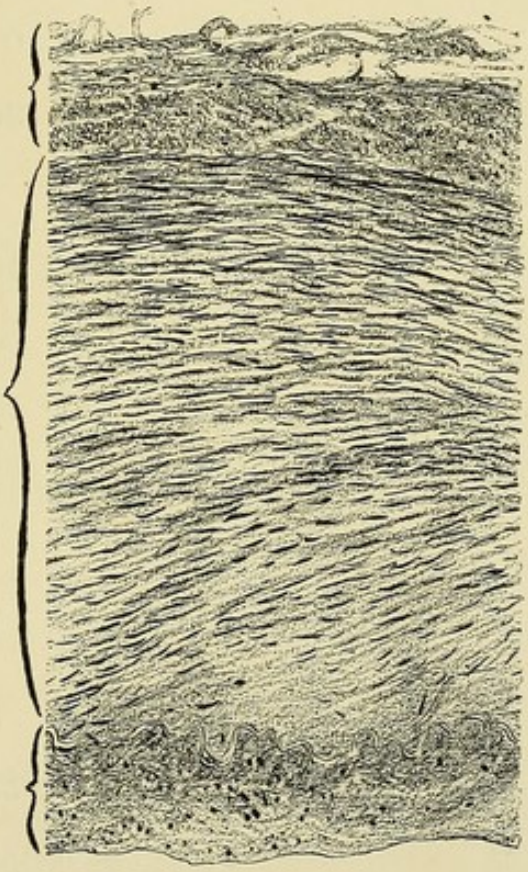


FIG. 36. Anterior Tibial.

Outer coat not so thick as in the posterior tibial.

The following table gives in hundredths of a millimetre the thickness of the coats, and the total thickness of the wall of the principal arteries when in a collapsed condition, the sheath being removed. Every endeavour was made to obtain the healthy arteries of adult men. The measurements were made, with the aid of a camera lucida, from microscopic sections prepared in the usual way.

	Outer coat	Middle coat	Inner coat	Total of wall
Aorta near valves	5	110	8	123
Innominate	5	73	20	98
Common Carotid	3	60	15	78
Facial	20	30	3	53
Middle Meningeal				
outside skull	12	10	0·5	22·5
in groove of sphenoid	continuous with dura	8	0·5	+8·5
in groove of parietal	3	7	0·5	10·5
Basilar	9	10	1	20
Arteria Centralis Retinæ				
external to nerve	7	6	0·5	13·5
within nerve	3	3	1	7
First part Subclavian	5	50	18	73
Third part Subclavian	7	51	5	63
Second part Axillary	7	36	4	47
Brachial	15	55	3	73

	Outer coat	Middle coat	Inner coat	Total of wall
Ulnar	12	40	3	55
Radial	6	30	5	41
Aorta near bifurcation	10	75	20	105
Splenic	8	46	5	59
Superior Mesenteric	60	53	5	118
Renal	10	22	1	33
Common Iliac	15	52	5	72
Umbilical at birth	5	35	15	55
Uterine of virgin	18	20	2	40
Uterine at term	17	17	3	37
Uterine of multipara	17	17	± 8	42
Dorsalis Penis	4	4	1	9
External Iliac	14	45	2	61
Common Femoral	20	80	10	110
Superficial Femoral	20	45	7	72
Popliteal	17	57	2	76
Posterior Tibial	20	42	5	67
Nutrient of Tibia				
external to bone	11	10	0·5	21·5
in canal	?4	12	2	18
Anterior Tibial	14	53	10	77

It will be seen from the foregoing drawings and table that the thickest artery in the body is, as might be expected, the arch of the aorta: the next in thickness is (at least in such arteries as we have measured) the superior mesenteric: next the common femoral: these are both thicker than the lower end of the abdominal aorta or the innominate.

The actual thickness of the outer coat varies greatly in different vessels: it is thickest in the superior mesenteric, next in the common femoral, superficial femoral, and posterior tibial, in all of which it is about equal: it is markedly thin in the aorta, innominate, common carotid and subclavian.

In the smaller arteries the relative thickness of the outer coat to the total thickness of the wall is much greater than in the larger vessels: it is at its greatest in the facial, middle meningeal before the foramen spinosum, basilar, uterine, dorsalis penis, nutrient of tibia external to bone, and arteria centralis retinæ external to nerve.

The probable explanation of these variations is that the thickness of the outer coat is adapted to resist the pressure of joint, muscular and visceral movement; this is well seen in the common femoral, facial and superior mesenteric respectively: the thickness of the outer coat of the basilar is probably related to the pulsations of the brain and of the vessels in the orbit to the movement of the eyeball.

This view is confirmed by the fact that the outer coat of the nutrient artery of the tibia is much thicker where the artery is external to the bone than where it is within its canal: the same applies to the arteria centralis retinæ

without and within the optic nerve. The outer coat of the posterior tibial which lies amongst muscles is much thicker than that of the anterior tibial where it lies on the interosseous membrane: the outer coat of the ulnar which lies amongst muscles is much thicker than that of the radial where it lies between tendons.

Donders and Jansen, Kölliker, Gimbert and Henle have made measurements of the coats of various arteries.

In making these measurements there is a difficulty in deciding first where the sheath ends and the outer coat begins, and secondly where the middle coat ends and the inner begins: Henle, indeed, says that the outer coat cannot be measured with precision. We found however that, with the larger arteries, it is possible to be fairly accurate.

It is interesting to tabulate the different observations (see next page).

It will be noticed in the table overleaf that with respect to the outer coat the observations of Gimbert and ourselves closely correspond, while Donders and Jansen find it thicker: this must be due to their including in outer coat what Gimbert and we considered sheath: Henle and Kölliker agree with Donders: the question as it bears on surgery is where an aneurism needle would pass, and that is quite internal to the sheath if it be properly opened: this we bore in mind in obtaining our arteries for section, with the larger vessels actually using an aneurism needle. Gimbert points out how very thin is the outer coat of the aorta, innominate, subclavian and common carotid: in the case of the aorta it is thinner on the arch than elsewhere:

(MEASUREMENTS IN HUNDREDTHS OF A MILLIMETRE.)

Artery	Outer coat	Middle coat	Inner coat	Total	Observer
Aorta near valves	15	54	50	119	Donders & J.
	very thin	78	10	—	Gimbert
	5	110	8	123	B. and E.
Innominate	—	60	—	100	Donders & J.
	very thin	61	thin : variable	—	Gimbert
	5	73	20	98	B. and E.
Common Carotid	25	45	23	93	Donders & J.
	very thin	44	thin	—	Gimbert
	3	60	15	78	B. and E.
Subclavian	20	38	15	73	Donders & J.
	very thin	33	thin	—	Gimbert
	7	51	5	63	B. and E.
Common Iliac	50	55	5	110	Donders & J.
	32	40	13	85	Gimbert
	15	52	5	72	B. and E.

on the other hand arteries which are much disturbed have a very thick outer coat: that of the external carotid being (he says) 0·45 mm. and that of the internal maxillary being 0·32 mm., in one case 9 times and in the other 6 times thicker than the outer coat of the innominate, and in both cases double the thickness of their own middle coats: the actual measurements he gives are, for the external carotid, outer coat 0·45 mm., middle coat 0·22 mm., and for the internal maxillary, outer coat 0·32 mm., middle coat 0·16 mm.

The outer coat of an artery probably limits its size under pressure. Roy found that when the pressure within the carotid of a rabbit is increased from 10 mm. to 170 mm. Hg. that "the increase in volume with each "successive increment of pressure becomes greater till "about 60 mm. is reached, that the maximum increase is "between 60 and 80 mm., and that with higher pressures "the increments in cubic capacity become less and less: "so much so, that raising the intravascular pressure from "110 to 170 mm. produces only about an equal increase "in contents to that produced by raising the pressure "from 60 to 70 mm. Now 70 or 80 mm. of mercury is "about the normal mean blood-pressure of the rabbit, "and this experiment shews that above this the arteries "become more and more rigid-walled."

With respect to the elasticity of arteries Roy also says that, "considered as tubes it is found that the arteries are "most elastic and most distensible with internal pressures, "such as during the life of the animal have existed in their "interior: the maximum increase in capacity with unit "increase of internal pressure, is reached in the case of

"the large arteries with a pressure at or near the medium "blood-pressure of the animal."

The reason that an artery does not dilate proportionally with pressures above normal, is that the gradually increasing inextensibility of the outer coat prevents the further distension of the vessel and to some extent determines its size.

It is necessary to clear our minds of the appearance of arteries as seen in the post-mortem room and to think of them as they exist during life, subject to the pressure of the blood: for few recognise the extreme tenuity of the arterial wall under natural circumstances.

After death, when the arteries are empty and the pressure of the blood is withdrawn, they collapse; also, the middle coat, being no longer pressed against the outer coat by the blood, becomes more bulky and succulent; the elastic laminae too, being no longer on the stretch, wrinkle up and are seen in sections as wavy lines: the artery after death thus appears smaller than during life and its wall much thicker.

In order to determine the thickness of the arterial wall during life, we distended human arteries, some with melted glycerine jelly, some with wax made thin with oil, some with liquid cocoa-butter, and some with plaster of Paris, and allowed them to set; this distension was done with a manometer attached, at a pressure varying from 150—240 mm. of mercury and at a temperature about that of the blood: the arteries were then hardened in Müller's fluid and dilute spirit, and microscopic sections made in the ordinary way: in other cases the thickness of the walls was measured with a camera lucida, as soon as the in-

jection was set: some of the results are shewn in the annexed table. Before the measurements were made, care

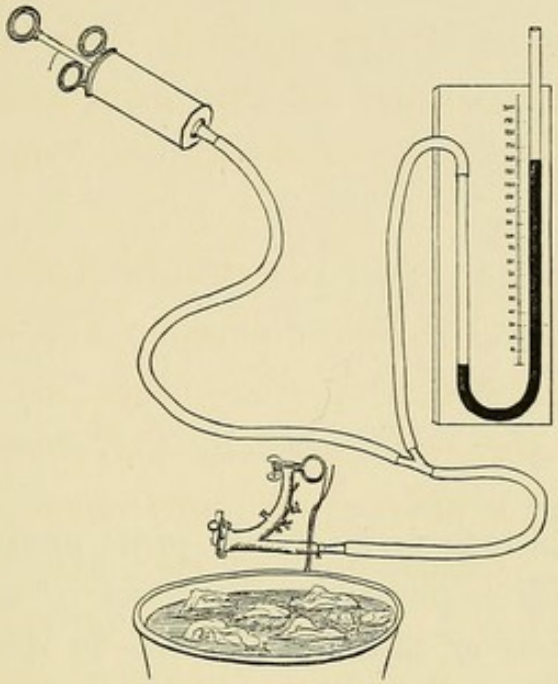


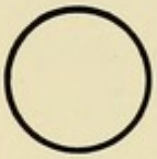
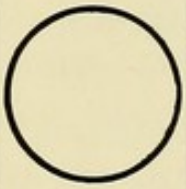
FIG. 37. Method of distending Arteries under normal pressure. (Scale $\frac{1}{8}$.)

Below is seen a bowl of ice: above it is the innominate, subclavian and common carotid arteries: the last is attached by tubes to a syringe for injecting the distending fluid and a manometer to shew the pressure: the free ends of the innominate and subclavian are closed by clips, and the branches of the subclavian are ligatured by silk: in using the apparatus the clips are opened and some fluid injected by the syringe and allowed to flow through till the system is free from air: they are then closed and the injection continued till the required pressure as shewn by the manometer is reached (in the figure this is 160 mm. Hg.): the ligature round the carotid is now tightened and tied, and the distended vessels plunged into the ice below in order to set the distending fluid.

was taken to remove the sheath as it would be previous to the application of a ligature.

The question of what is the pressure of the blood in man, will be found alluded to in the chapter on the Choice of the Force; suffice it to say here that it is, as far as is known, in the great arteries about 200 mm. Hg. The pressure does not vary much in the main arterial system,

Table shewing the actual size of the great arteries of man when distended with a pressure of 240 *mm. of mercury: also the actual thickness of their walls when so distended, and also when not distended, as found in the post-mortem room: the circles representing the arteries were drawn with a camera lucida: the undistended arteries are represented the same size as the distended, to facilitate the comparison between the thickness of the wall of the living and the dead artery. The measurements are in millimetres.*

Name of Artery	Diameter under distension	Thickness of wall under distension	Thickness of wall of collapsed vessels	Scale drawing actual size of vessels during life, and actual thickness of their walls	Actual thickness of coat of collapsed vessel
Innominate	14	0·51	0·98		
Common Carotid	8·5	0·29	0·78		
1st part Subclavian	11	0·31	0·73		
3rd part Subclavian	9	0·35	0·63		
Abdominal Aorta (near division)	17	0·5	1·05		
Common Iliac	12	0·5	0·72		
External Iliac	11	0·43	0·61		
Common Femoral	10	0·52	1·1		
Superficial Femoral	8	0·51	0·72		

it is probably only 20 or 30 mm. less in the radial than in the aorta, and must therefore remain fairly constant in the length of each artery.

Artery	Healthy male adult. 240 mm. Hg.	Male æt. 32. Tubercular peritonitis. 175 mm. Hg.	Male æt. 60. Renal and cardiac disease. 160 mm. Hg.
Innominate	51	57	—
Common Carotid	29	36	43
Subclavian, 1st part	31	32	82
Subclavian, 3rd part	35	20	37
Abdominal Aorta	50	53	73
Common Iliac	50	30	56
External Iliac	43	29	—
Common Femoral	52	30	82
Superficial Femoral	51	37	59

In disease the pressure may greatly increase. In cardiac hypertrophy Roy says it may rise as high as 350 mm. Hg.: it is associated with a compensatory hypertrophy of the arterial wall.

As it does not appear settled what the normal mean blood-pressure in man is, we repeated the observations at various pressures. Some of the results are embodied in the preceding table: the figures give the total thickness of the walls of the distended arteries in hundredths of a millimetre.

It will be noticed that the change of pressure does not make any material difference in the thickness of the distended wall, and also that in chronic renal disease the arteries are exceptionally thick: these arteries before distension appeared more than proportionately stouter, so that the lesser pressure had comparatively more effect upon them than the greater pressure upon the normal arteries.

Vierordt in his *Daten und Tabellen* quotes from Bollinger (1886) the results of the measurement of certain human arteries under distension with a salt solution, the pressure being about equal to 120 mm. Hg. He found the thickness of the common carotid to be 0·29 mm., which is exactly what we made it, the common iliac and the common femoral he gives as 0·32 mm. as against our 0·50 and 0·52 respectively. Thus, with a less pressure, he makes these arteries only slightly thicker than the common carotid: our results are more in accord with the relative thickness of the undistended vessels.

It has next to be considered which coat of the artery undergoes most alteration by the collapse of the vessel at death. The following table gives the thickness of each coat of some of the main arteries under normal pressure and as they are found in the post-mortem room (unit of measurement 0·01 mm.).

Artery		Outer coat	Middle coat	Inner coat	Total
Innominate	Distended	3	47	1	51
	Collapsed	5	73	20	98
Common Carotid	Distended	2	26	1	29
	Collapsed	3	60	15	78
Subclavian, 1st part	Distended	2·5	28	0·5	31
	Collapsed	5	50	18	73
Subclavian, 3rd part	Distended	5	29	1	35
	Collapsed	7	51	5	63
External Iliac	Distended	12·5	30	0·5	43
	Collapsed	14	45	2	61
Common Femoral	Distended	14	37	1	52
	Collapsed	20	80	10	110
Superficial Femoral	Distended	14	36	1	51
	Collapsed	20	45	7	72

It will be seen that the increase in thickness of the wall after death occurs in the greatest degree in the middle coat. This is due partly to internal folding depending upon the reduction in the size of the artery, as evidenced by the wavy contour of the elastic membranes, and partly also to the imbibition of fluid which becomes possible when the pressure of the blood ceases, and which was displaced in the experiments by the measured pressure: in like manner the elastic membranes become straightened out.

In the same way as the collapsed artery removed from the body is increased in diameter by a fluid pressure equal to that of the blood, it is also increased in length. By means of the pressure of a column of water 9 feet in height, the following table was drawn up: marks were

Artery.	
Innominate	17 per cent.
Common Carotid (right) .	20 „ „
Common Carotid (left) . .	25 „ „
1st part Subclavian (right) .	25 „ „
1st part Subclavian (left) .	25 „ „
3rd part Subclavian (right) .	12 „ „
Brachial	20 „ „
Abdominal Aorta . . .	5 „ „
Common Iliac . . .	17 „ „
External Iliac . . .	17 „ „
Common Femoral . . .	25 „ „
Superficial Femoral . .	17 „ „

made on the artery while collapsed and the distance between them determined: pressure was then applied and the separation of the marks again measured: in this way the percentage increase in length of each artery was

calculated: it will be seen that the increase is about 20 per cent. The table is mainly compiled from experiments on the arteries of a healthy man between 40 and 50 years of age.

The variations are presumably mainly dependent on the amount of elastic tissue in the different arteries.

The above table may be taken to represent an average case with healthy vessels: in some arteries at earlier periods of life the longitudinal elasticity of the dead artery is greater than represented: on the other hand, old age and disease have the opposite effect.

Table shewing the increase in length produced by distension of the arteries of a man aged 34 *years who had died of tubercular peritonitis after a short illness: the vessels were healthy: a pressure of* 170 *mm. Hg. was applied with melted cocoa-butter at a temperature of* 100° *F.* (38° *C.*).

Arch of Aorta (transverse portion)	
anterior surface . . .	41 per cent.
posterior surface . . .	17 „ „
Innominate	30 „ „
Common Carotid (right) . . .	32 „ „
1st part Subclavian	20 „ „
3rd part Subclavian	11 „ „
Abdominal Aorta (near bifurcation)	
anterior surface . . .	32 „ „
posterior surface . . .	11 „ „
Common Iliac	30 „ „
External Iliac	25 „ „
Common Femoral	35 „ „
Superficial Femoral	15 „ „

Table shewing the increase in length produced by distension of the arteries of a man aged 59 *who had died from renal disease: the heart was greatly hypertrophied, and the arteries thickened and dilated: distension with cocoa-butter at* 100° *F.* (38° *C.*) *to a pressure of* 160 *mm. Hg.*

Common Carotid	14 per cent.
1st part Subclavian . . .	5·5 „ „
3rd part Subclavian . . .	none
Abdominal Aorta (near bifurcation) anterior surface . . .	12·5 „ „
Common Iliac	20 „ „
External Iliac	20 „ „
Common Femoral	34 „ „
Superficial Femoral	1 „ „

It will be noticed how greatly disease of the arterial wall reduces its elasticity.

It does not follow from what has just been said that the arteries during life put much tension on their attachments. After death the arteries cannot contract in their length owing to their anatomical attachments, but if these be divided they will at once shorten, shewing that there is considerable external tension, but this is only after death, when distension by the pressure of the blood is withdrawn, and it does not follow that there is much external tension during life when the pressure of the blood is distending the arteries.

To test this point we made the following experiment. The carotid arteries of a healthy young adult who had died of acute peritonitis were fully exposed *in situ* before the thoracic viscera were interfered with, and on

each of the arteries two marks were made; on the right carotid it was found that these marks were 20·5 mm. apart, and on the left 34 mm. These measurements were made with the head in the normal anatomical position; it was found that even extreme flexion of the head made no difference; extreme extension of the head, however, increased the distance between the marks on the right carotid by 1·5 mm., on the left by 0·5 mm. The arteries were now divided near their extremities (far beyond the marks) and removed from the body: they contracted so that the marks on the right carotid were now 13 mm. apart, and on the left 24·5 mm. Next, the arteries were attached in turn to a manometer, and distended with water to a pressure of 150 mm. Hg. They lengthened out so that the marks became on the right carotid 20·5 mm. apart, and on the left 34 mm., that is to say, exactly what they were at first before being divided; in other words, whilst when collapsed the arteries exert much external tension, yet when distended by a pressure about equal to that of the blood they are of such a length that they occupy the whole extent of their anatomical position in the living body: there is therefore, as far as this experiment goes, little or no strain on their external attachments.

The following experiment is also of interest. If in the artery while distended with water a longitudinal incision be made, the water will spurt out, and it will be seen that the aperture is very wide, nearly circular in fact. On the other hand, if a transverse incision is made, it will be seen that the water escapes in a flat sheet, and that the aperture is linear. The explanation of the difference

seems to be that the transverse contractility which exists in a distended artery widens out the longitudinal slit but there is little or no longitudinal contraction to do the same for the transverse slit.

In order to keep up the pressure of water in this experiment a syringe, as represented in fig. 37, p. 53, is not sufficient; it is necessary to have the manometer attached to a column of water coming from a reservoir at the requisite height so as to have a continuous and ample stream.

Another experiment: the common carotid of a large animal under ether was exposed in two places, near its bifurcation and at the root of the neck: these wounds were about 4 inches (10 cm.) apart: on the distal portion exposed two ligatures were placed; on the proximal portion exposed two marks were made, these on measurement with callipers were found to be 13·5 mm. apart; the artery was then divided between the two ligatures, thereupon at the proximal opening the marks were observed to recede towards the chest for about 4 mm.: on measuring it was found that the marks were now 13 mm. apart, that is to say, they had approximated by 0·5 mm. or about 4 per cent. of the distance between them. The cardiac ligature also receded 4 or 5 mm. Thus the shortening of the vessel, the distension being maintained, is only about 4 per cent. of its length, while the shortening of vessels when the distending force is withdrawn is, as we have seen, about 25 per cent. The small degree of shortening in the experiment is not to be explained by the vessel being held by branches, for there were none; neither is it to be explained by the vessel being held by

attachments to the sheath, for the least movement, such as produced by wiping gently with a sponge, of the part exposed in the proximal opening was readily communicated to the distal part, as seen by the movement of the ligatures before the vessel was divided.

On this occasion it was also found that the blood spurted out from a transverse and a longitudinal incision in an artery in the manner above described as occurring during distension with water.

Arteries when distended outside the body never appear quite straight: and when divided within the body it is possible that some curving within the sheath may occur which would account for a certain amount of apparent shortening.

It seems clear then that the blood pressure almost neutralizes the external tension that there would be if the artery were collapsed from the withdrawal of this pressure: a small amount of tension exists, but not more than is possessed by other tissues; even the spinal cord on division retracts.

Indeed it is not to be expected that there should be any considerable external tension of an artery during life, because if there were it could fulfil no useful function and would therefore soon be lost from disuse. The alteration produced in the length of an artery by the bending of a joint is very slight, and not more than could be met by the tension under which all living tissue exists, and which is supposed to be necessary for nutrition.

The points which in this chapter bear most on the practice of surgery are, firstly, the extreme tenuity of the

arterial wall in its natural state; secondly, the smallness of the external tension of an artery when distended with the pressure of the blood; thirdly, the greatness of that tension when the artery is not so distended; fourthly, the variation in the amount of the longitudinal contraction of the different arteries when collapsed (that of the third part of the subclavian being less than half that of the common carotid); and lastly, the reduction or even abolition by disease of the elasticity of the collapsed artery.

The tension produced in the artery itself by the pressure of the blood within will be considered in the chapter on the Choice of the Force.

CHAPTER IV.

PHYSIOLOGICAL OCCLUSION.

Ductus Arteriosus. Hypogastric Artery. Umbilical Vein. Remarks on Nature's method.

It is a matter of common knowledge that at birth certain changes occur in the vascular mechanism in consequence of the expansion of the lungs, and cutting off of the placental circulation. The ductus arteriosus, the umbilical arteries and other vessels become physiologically obliterated as they are of no more service to the economy. The changes which take place in these vessels have been studied by Thoma, Langer, Warren and many others.

Ductus Arteriosus. All observers seem to agree that the obstruction of the ductus is caused first by a mechanical process, secondly by histological changes. Chevers thought

it was pressed upon by the recurrent laryngeal nerve, which curves round the fifth left fœtal arterial arch, the outer and back part of which forms the ductus arteriosus. King thought that pressure was exercised upon it by the left bronchus. Virchow says definite obstruction is caused at the first inspiration. F. Schanz, in a recent and interesting paper in Pflüger's *Archiv*, suggests that the sudden blocking of the ductus is shewn by, and is the explanation of, the enormous rise in the pulse during the first 15 or 20 minutes of life, the subsequent fall corresponding with complete obstruction. Kewisch thought the obstruction was due to active muscular contraction under nervous influence, but this is impossible, as the muscular band in the ductus is very thin, less strong indeed than that in the femoral artery. The presence of muscle cells is denied by Walkhoff and Schultze, who regard the diminution of the blood-stream, the stagnation of blood during the short period of equal pressure in the pulmonary artery and aorta, and the general rearrangement of the thoracic contents at birth, as the most important factors in obliteration. In microscopical preparations of the ductus arteriosus a thin muscular layer can always be discerned. (Fig. 41.) F. Schanz has shewn by numerous experiments that the ductus is greatly stretched by the first inspiration. His plan of experiment consists not only in observing the condition of the ductus in the bodies of children who had died before and after lung respiration had been set up, but also in cutting holes in the chest-walls of still-born children so as to be able to directly observe the change in the ductus when the lungs were artificially inflated from the trachea.

The aortic end of the ductus is fixed by the attachment of the termination of the aortic arch to the spine by areolar tissue. The pulmonary end of the ductus on the other hand is moveable, and at the first inspiration is carried forwards, firstly, by the forward movement of the roots of the lungs which advance 1 to 1½ fingers' breadths, secondly, by the forward movement of the sternum, the

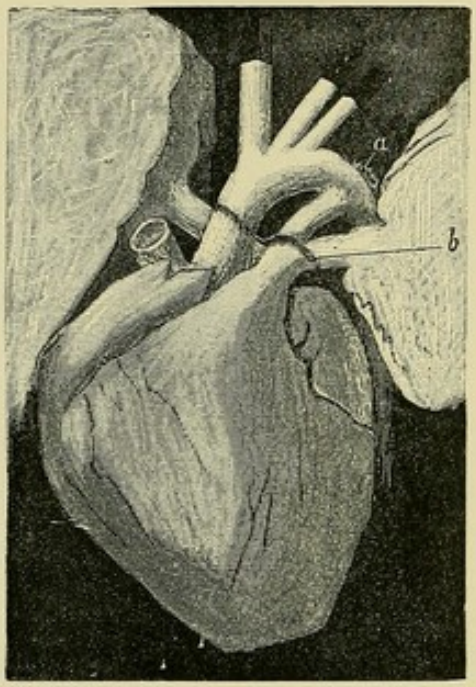


FIG. 38.

Reproduced by kind permission of Prof. Schanz from the Archiv f. Physiol. v. Dr Pflüger, 1888. (*Slightly modified by request.*)

Heart and great arteries of a new-born child before inspiration. *a* shews the firm attachment of the aorta posteriorly to the spine: and *b* the reflexion of the pericardium. The drawing also shews how the right and left pulmonary arteries are given off from the back of the main trunk. *a* being fixed, the effect of the first inspiration is to draw *b* forwards and by putting the ductus on the stretch to occlude it.

pericardium being attached to the sternum and pulmonary artery, and thirdly, by the downward movement of the diaphragm, the pericardium being attached below to the

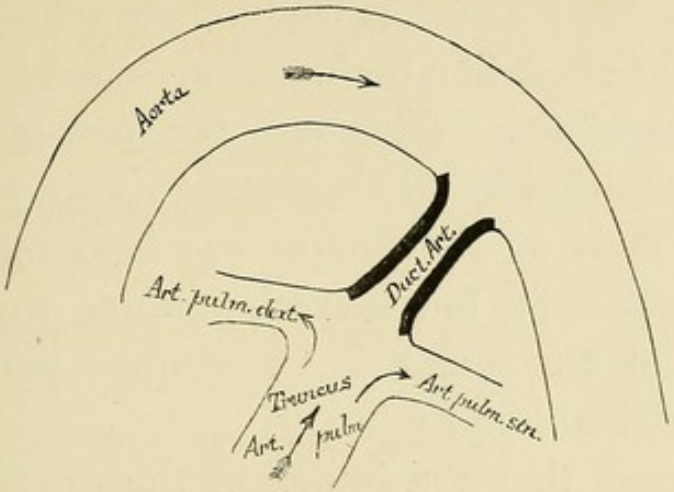


FIG. 39.

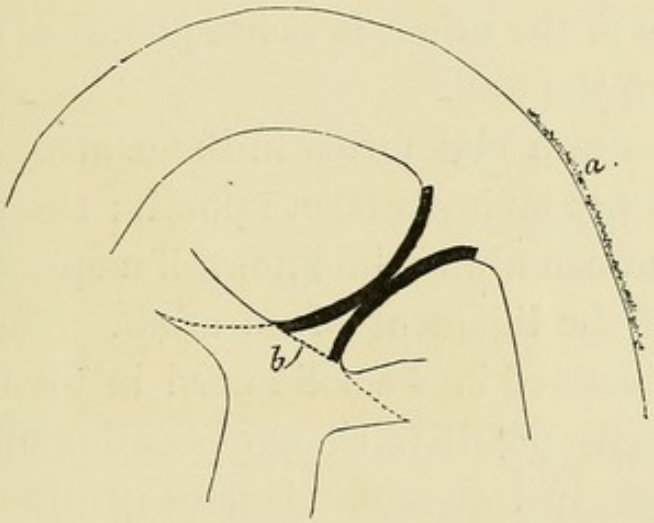


FIG. 40.

Reproduced by kind permission of Prof. Schanz from the Archiv f. Physiol. c. Dr Pflüger, 1888. (*Slightly modified.*)

Fig. 39 shews the aorta, the pulmonary artery (dividing into right and left pulmonary arteries) and the ductus arteriosus as immediately before the first inspiration.

Fig. 40 shews the same vessels immediately after the first inspiration: *a* point of attachment of aorta to spine by connective tissue. The dotted line *b* is the line of attachment of the pericardium: *a* being fixed posteriorly and *b* moving forwards the ductus is stretched and its walls brought into contact midway between the aorta and the pulmonary artery.

diaphragm and at the other extremity to the pulmonary artery. (Fig. 38.) This longitudinal stretching, combined with the slight alteration in direction, causes a constriction in the centre of the ductus, and the hour-glass shape of the lumen admitted by all observers. (Figs. 39 and 40.)

The two ends of the ductus are held open by their anatomical relations, but there is nothing to prevent the obliteration of the lumen in the central part when the vessel is stretched longitudinally.

Warren says that the lumen of the ductus, being no longer kept open by the large stream of blood, is blocked midway between the pulmonary and aortic attachments by the approximation of the tunica intima of one side to the tunica intima of the other, in consequence of a twisting or convolution of the wall.

In rare cases a clot forms in the centre of the ductus and then the two ends contract upon it; the pre-existence of a coagulum can always be known if a space be found in the centre of the ligament of an adult. The ductus appears to be changed into a solid cord in about six weeks, but retrogressive nutritive changes continue for months or even for years, just as such changes continue for a long time in and about a ligatured artery, in order that a slow perfecting of the newly-formed scar tissue may take place.

The wall of the ductus at birth is in a less firm and perfect state than, for example, are the walls of an artery of an adult limb. Its texture is much looser; but it is quite easy to make out the three coats, the intima especially being very thick and distinct. (Fig. 41.) There is a good deal of elastic tissue, but the muscular band, as has already been pointed out, is narrow, and generally speaking the

middle coat is not so clearly differentiated as that of vessels in later life. The reduced lumen of the ductus after the first inspiration causes the coats, especially the tunica intima, to be thrown into pleats or folds, much like those that are seen after the application of a ligature in

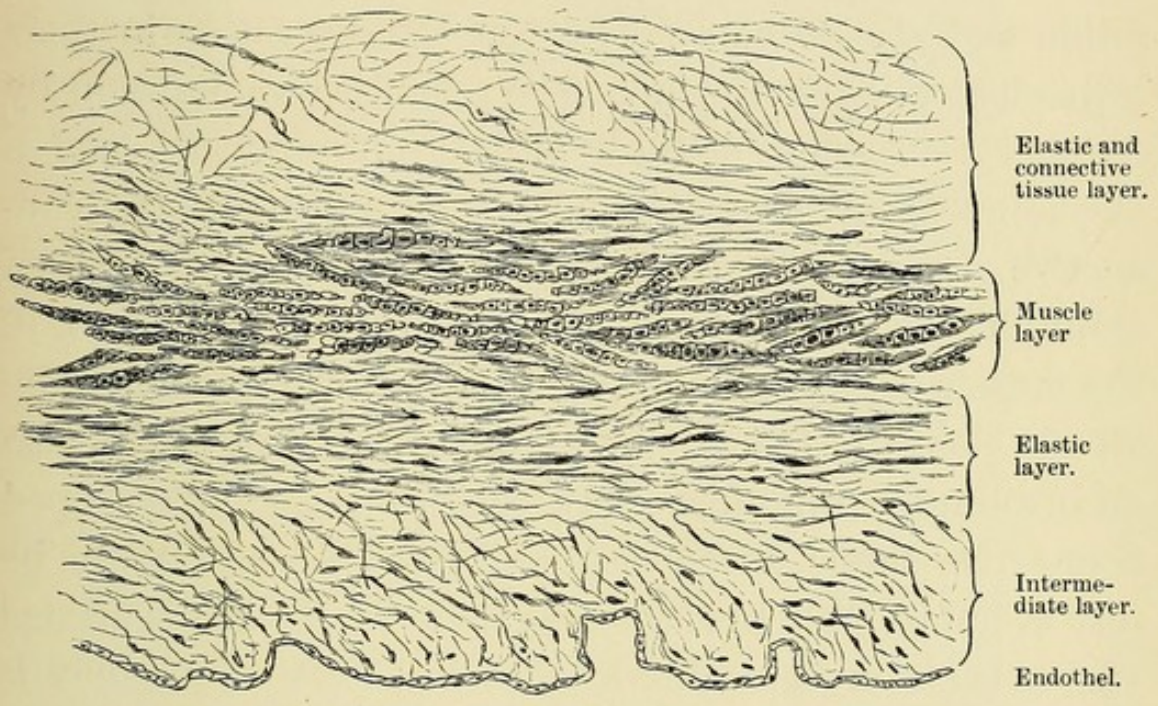


FIG. 41. Section of the Ductus Arteriosus from a still-born infant.

Reproduced by kind permission of Prof. Fritz Schanz from the Archiv für Physiologie von Dr Pflüger, 1888. (*Slightly modified.*)

When respiration has occurred some separation or rupture is visible between the inner elastic coat and the middle coat or muscular layer in consequence of the stretching to which the ductus is subjected at the first inspiration. It is specially to be noted that the intima is very thick.

continuity. Immediately after birth the intima proliferates, so that very soon the walls of the ductus are in contact in their whole length. If clot be formed it is absorbed and replaced in a manner similar to that which obtains in ligated arteries. Not unfrequently after the ductus has been transformed into a ligament, a small central arteriole

remains which is distributed to the fibrous tissue. Histologically the cicatrising and obliterating process is seen on section to be due to the great growth and vascularisation of the intima. (Fig. 42.)

The outer and middle coats proliferate as well as the inner. This is owing to a change in their nutritive condition and also in consequence of the alteration in their physiological environment. The result is a diminution by degeneration from non-use of the muscle cells, and a development of fibrous tissue by the activity of the connective tissue cells of the whole arterial wall. Warren maintains that the process of obliteration is the work of the muscle cells of the middle coat, but in this opinion he stands alone: it is often by no means easy to distinguish an involuntary muscular fibre cell from a spindle-shaped connective tissue corpuscle: and it would appear to be just as improbable that the scar tissue of arteries is formed of a mass of muscle cells, as that an intestinal cicatrix is muscular and not fibrous. It is, however, possible that some muscle cells may be found sparsely just as elastic fibres are sometimes seen in old scar tissue, but they could take no active part in the early and important stages of the process of occlusion of the ductus.

The proliferation of the intima is one of the earliest and most marked characters which are to be noted in the ductus a few days after birth, and later it is by means of this proliferation that the final obliteration of the vessel occurs. The necessary vascularisation to enable the new tissue to maintain its vitality is supplied to it from the vessels of the outer coat. When after a lapse of some months the ductus is changed into a simple fibrous cord,

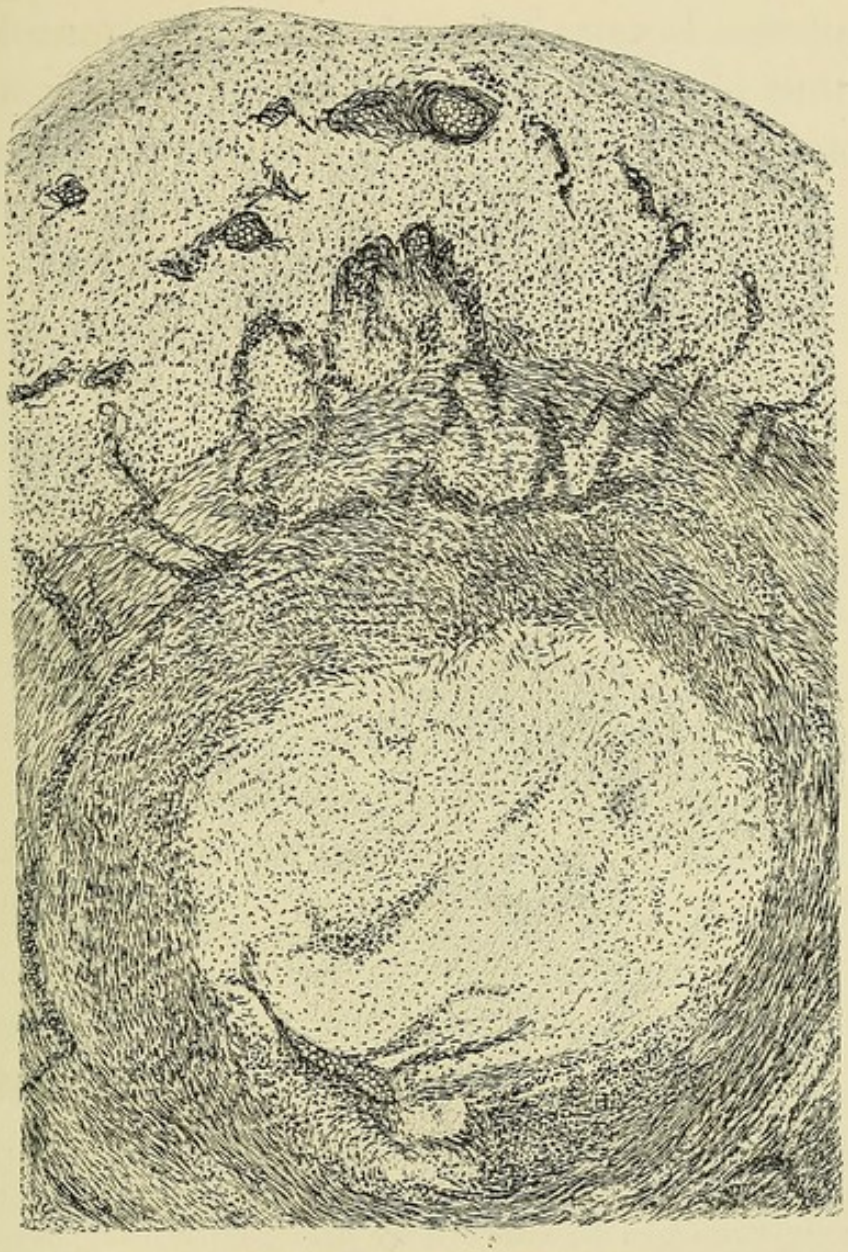


FIG. 42. Ductus arteriosus 5 months after birth (× 50).

In the upper part of the figure is seen the outer coat with numerous capillaries: internal to this is the middle coat: through its centre runs circularly a capillary: the central light-coloured area is formed by the hypertrophied intima: to one side of it (below and to the left) is a small vessel containing post-mortem clot, this is all that remains of the original lumen of the vessel.

the capillary network diminishes and is maintained at that standard which is sufficient to keep up the life of the part, and this is very different to the abundant capillary supply that is required in effecting the earlier stages of occlusion.

FIG. 43. Hypogastric artery 3 weeks after birth (× 50).

The limits of the three coats are readily recognised: the intima is thickened and its cells are invading the central clot: the clot is attached only at certain points to the intima: capillaries from the outer coat are passing through the media to supply nourishment to the growing intima.

Hypogastric Artery. The next vessel to which reference must be made is the hypogastric; the obliterating process in it has been described as comparable to that which ensues in a large artery after amputation of a limb, as the vessel is, so to speak, ligatured at the umbilicus. (Fig. 43.) The artery becomes filled with clot; great thick-

ening of the intima takes place; columnar masses of endothelial cells arise from the intima and permeate the clot. Fibrillation of this tissue gradually occurs and in the end a fibrous band alone remains, through the centre of which a small vessel can be followed as far as the umbilicus. At first the process of obliteration depends upon the activity of the inner coat and the presence of clot, but soon some proliferation of the outer coat and of the connective tissue part of the middle coat occurs, which has for its object the supply of new capillaries for the new tissue, which by substitution is taking the place of the coagulum. (Fig. 44.) It has been said that the clot in the hypogastric artery is at first of traumatic origin, and it would be interesting in view of the theories of the connection between sepsis and clot formation lately advanced, to know if clot forms under complete antiseptic conditions of the umbilicus. It is pointed out, however, elsewhere that the stagnation in the current and the growth of the intima is amply sufficient to produce a coagulum.

Umbilical Vein. The process of obliteration in arteries and veins is the same. The microscopical changes are absolutely identical, though the naked eye appearances are sometimes apparently different, in consequence of the disturbance produced in an artery by the breaking of the blood-waves upon the clot and the fury and rapidity of the blood-stream. (Fig. 45.)

To return to the ductus, it has been said that one cannot argue from its occlusion to that of a ligatured artery because the force of the stream of blood has been diverted from the ductus, which is not the case in the recently-ligatured artery, but it must be remembered that

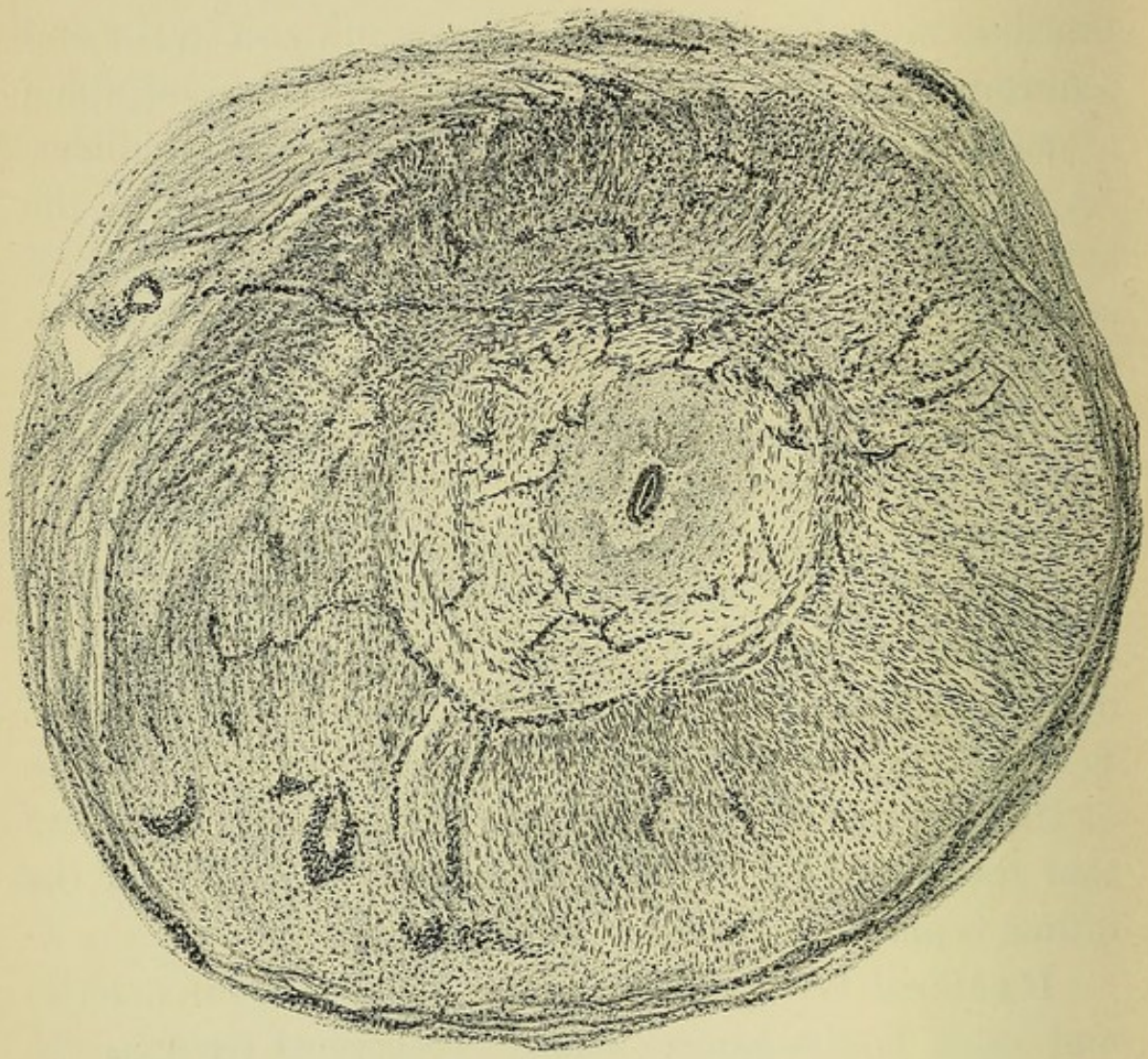


FIG. 44. Hypogastric artery 4 years after birth (× 50).

Specimen was taken 1 inch (24 mm.) from the umbilicus. At the centre of the figure a small vessel is seen in which there is a post-mortem clot: the wall of this newly-formed vessel is dark in colour and oval in section: external to this is a light-coloured area which is nourished by the blood circulating in the central capillary: external to this again is a darker area composed of thickened intima which has undergone hyaline degeneration. External again is a light area composed of hypertrophied intima which has not degenerated because it is well supplied with blood by numerous capillaries continuous with those of the outer and middle coat; some of these are arranged radially, others circularly: the outer margin of this light area corresponds to the original inner surface of the intima.

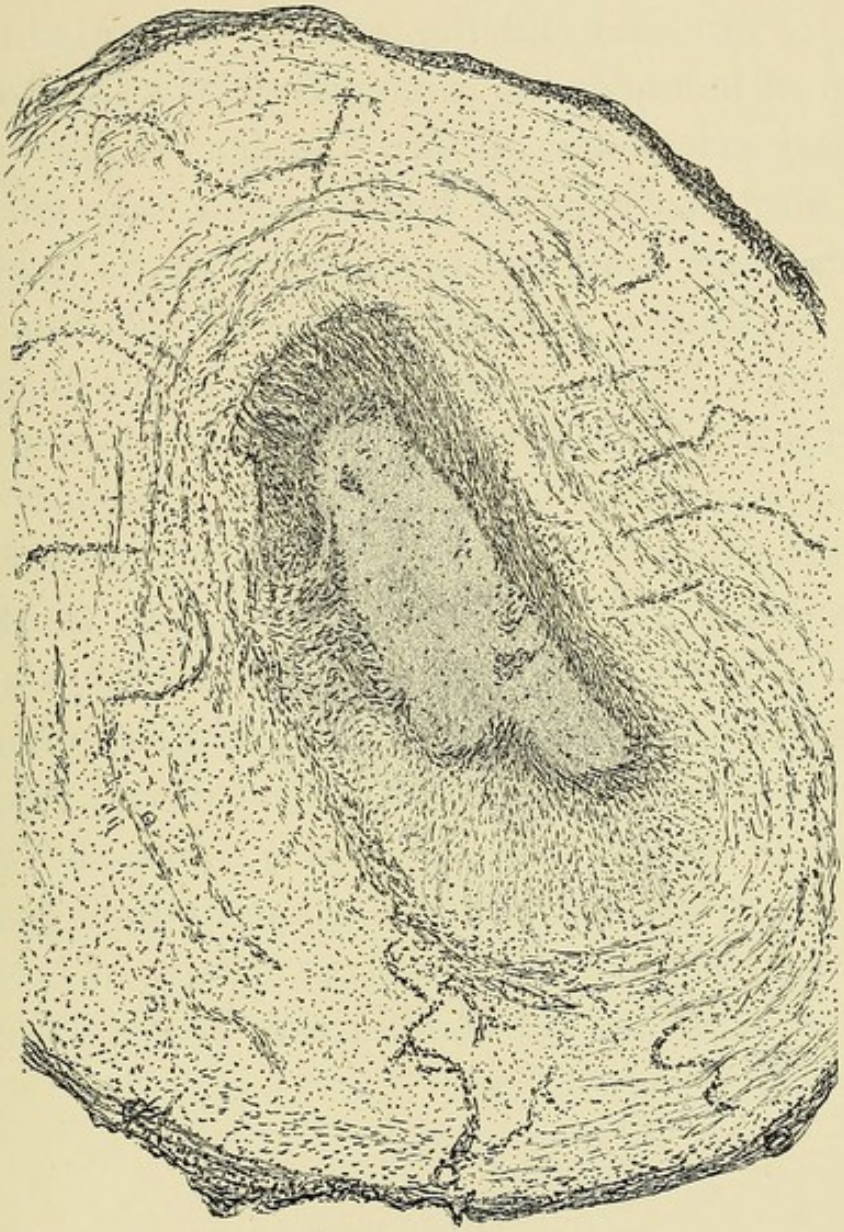


FIG. 45. Umbilical vein 3 weeks after birth (× 50).

The lumen of the vessel is occupied by clot, but its original size has been greatly encroached on by the proliferating and thickened intima: groups of plasma cells derived from the endothelium are seen in various parts of the clot: the outer limit of the intima is bounded by circularly arranged capillaries: the middle coat is highly vascular, new capillaries being formed to supply blood for the rapid growth proceeding in the intima.

for many days after birth the ductus is found pervious to a stream of water, and during life though closed in the centre, yet at either end the walls are separated by blood which must be under the same pressure as that within the pulmonary artery and aorta. The central portion of the ductus may therefore be looked upon very fairly as in much the same condition as a large artery is in after ligation in continuity without rupture of the tunics. The parallel may be continued further, for the longitudinal stretching must interfere with the vasa vasorum, in much the same manner as a ligature which obstructs them as well as the vessel in the outer coat of which they ramify.

In conclusion, it may be pointed out to the followers of J. F. D. Jones, that Nature does not think it necessary when occluding the ductus to rupture the two inner coats in order to produce sufficient intimal growth; and to the followers of Celsus and Abernethy, that she does not divide the artery to reduce the longitudinal tension, on the contrary, she does not hesitate greatly to increase it; nevertheless, in her hands, failure to occlude very rarely occurs, hemorrhage never.

CHAPTER V.

PATHOLOGICAL OBLITERATION.

Renal Endarteritis. Syphilitic Arteritis. Tubercular Arteritis. Chronic Arteritis. Arteritis Obliterans. Periarteritis. Obliteration by pressure. Remarks on Nature's method.

Pathology offers many instances of the extraordinary power of proliferation possessed by the tissues of the arterial wall, when excited by other stimuli than the application of a ligature.

In renal disease small vessels in various parts of the body are often found obliterated: the obstruction is partly produced by the growth of the intima, and partly in the later stages by the formation of clot. (Figs. 46 and 47.)

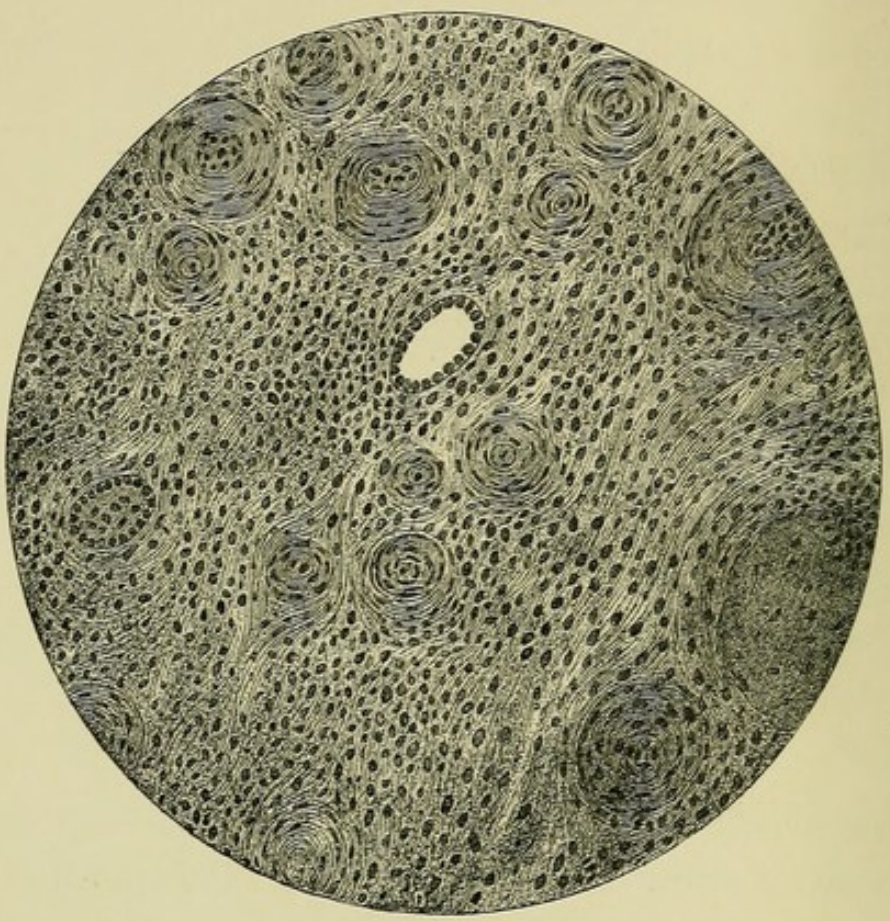


FIG. 46. Section near hilum of a kidney affected with Bright's disease.

Drawing reproduced from paper on retinal changes in Bright's disease by Brailey and Edmunds. Ophthalmo. Soc. Trans. Vol. I. (1881).

Numerous arteries are seen in transverse section: their outside diameters are enlarged; all their coats are hypertrophied, while their lumina are diminished or entirely obliterated by growth of the intima: in the latter case the obliteration is probably assisted finally by the formation of a central clot in which connective tissue developes.

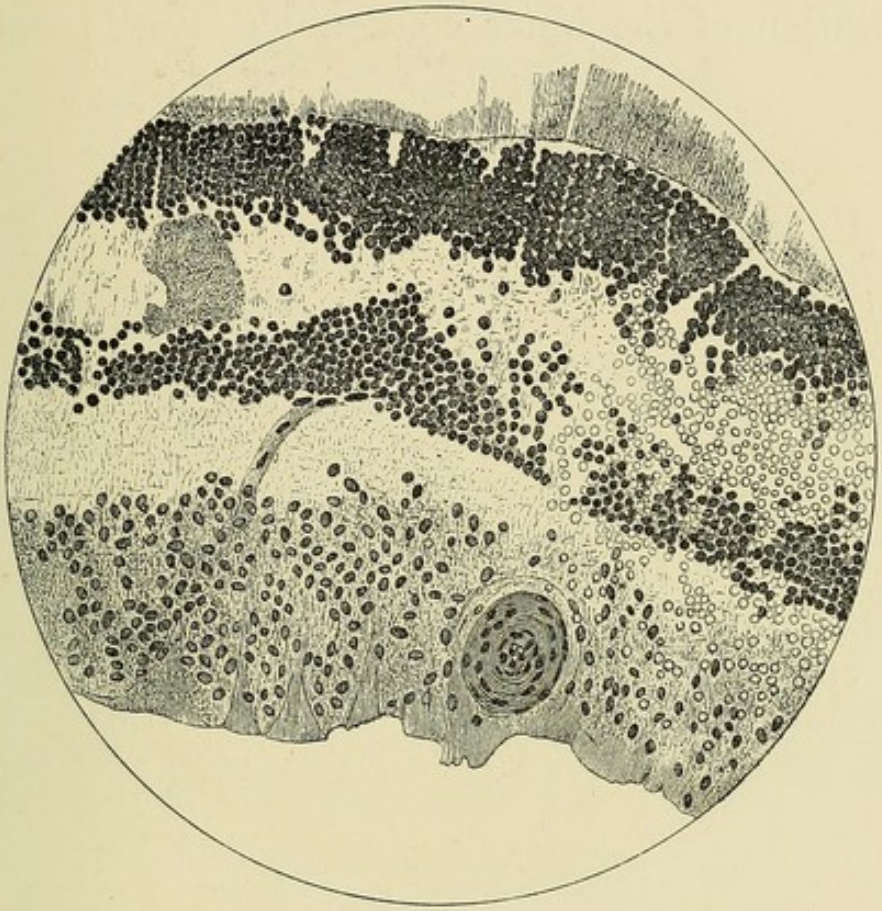


FIG. 47. Section of retina from a case of Retinitis Albuminurica.

Drawing reproduced from paper on retinal changes in Bright's disease by Brailey and Edmunds. Ophthalmo. Soc. Trans. Vol. I. (1881).

A retinal artery is seen much altered: its outside diameter is increased, while its lumen is completely obliterated by growth of the subendothelial layer of the intima, possibly assisted by a small central clot which became the scene of connective tissue formation.

In syphilitic arteritis the disease seems chiefly to affect the inner coat, though the outer is sometimes also thickened: in the artery from which the drawings were made it is seen that the lumen was nearly obliterated by the proliferation of the inner coat, aided very possibly (in

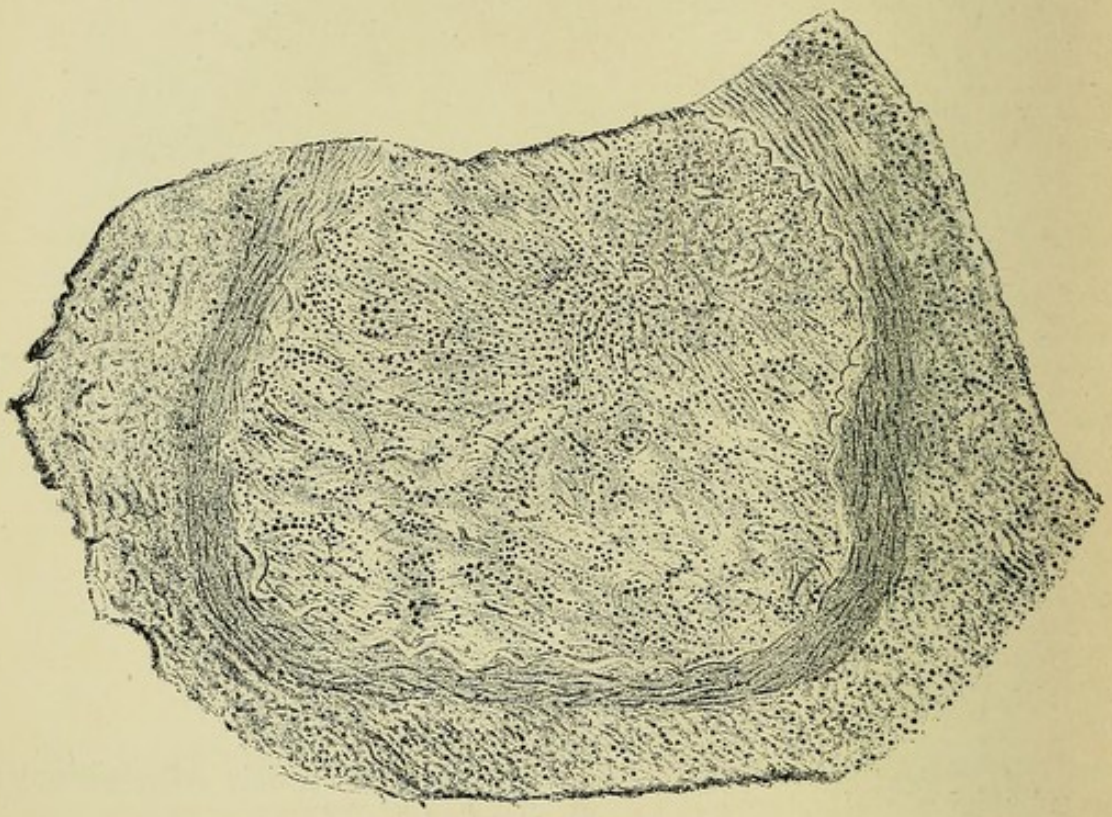


FIG. 48.

(For description see opposite page.)

figure 48) by the replacement of clot, which may have formed as the disease advanced in the vessel wall. A vessel thus diseased may ultimately become transformed into a fibrous cord. (Figs. 48 and 49.)

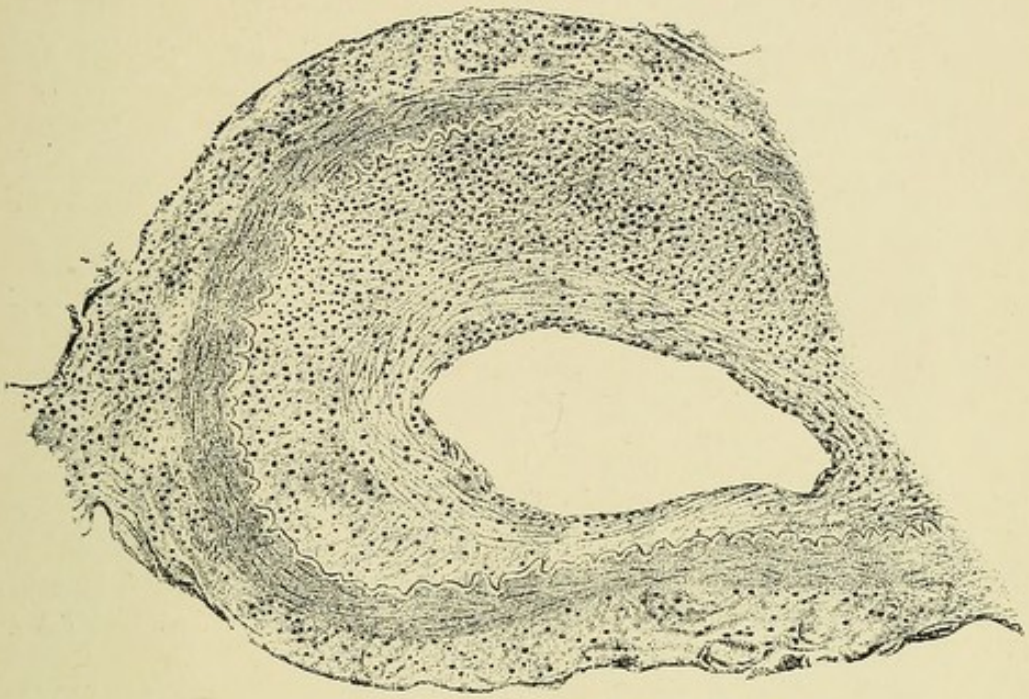


FIG. 49.

FIGS. 48 and 49. Syphilitic Arteritis.

Reproduced by kind permission of Dr Sharkey from Path. Soc. Trans. Vol. XXXVIII. (1887).

Transverse sections of left Sylvian artery from man aged 25 who had contracted syphilis four years previously. The external coat is hypertrophied. The middle coat is unaltered. The intima is greatly thickened. The two small blood-canals in Figure 48 indicate that a clot has formed and become organized. If the lumen had been occluded simply by a growth of the intima only one opening would have existed, as is the case in Figure 49.

In tuberculosis a similar proliferation, chiefly of the inner coat, is seen. (Figs. 50, 51 and 52.)

Figure 50 shews a small artery from a tubercular lung: the intima is greatly thickened, and in it are seen numerous tubercle bacilli.

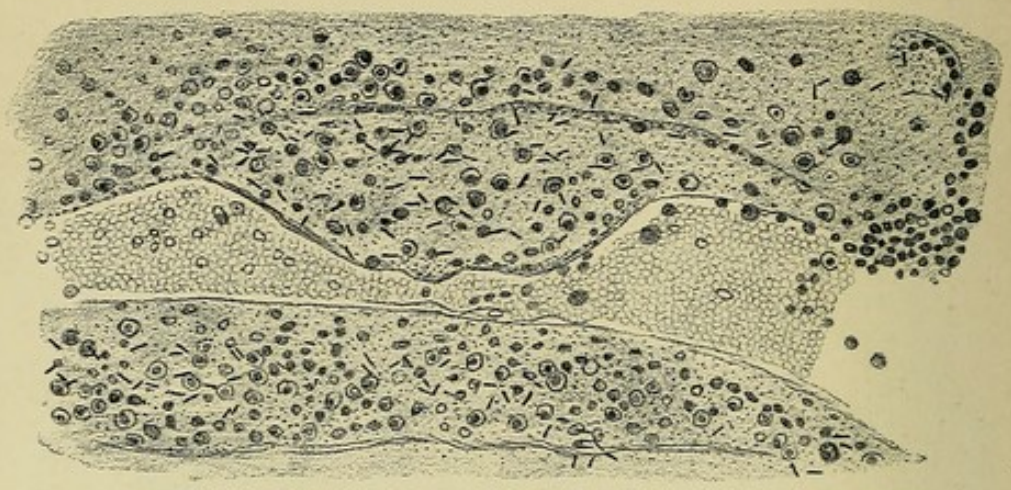


FIG. 50. Arteriole from the lung (× 200).

The original drawing was kindly lent to us by Dr Percy Kidd, by whom it has been already reproduced in the Trans. Roy. Med. Chir. Soc. Vol. LXVIII. (1885).

From a case of tuberculosis: the bacilli are seen in the intima, in consequence of which there is an active growth and thickening of this coat.

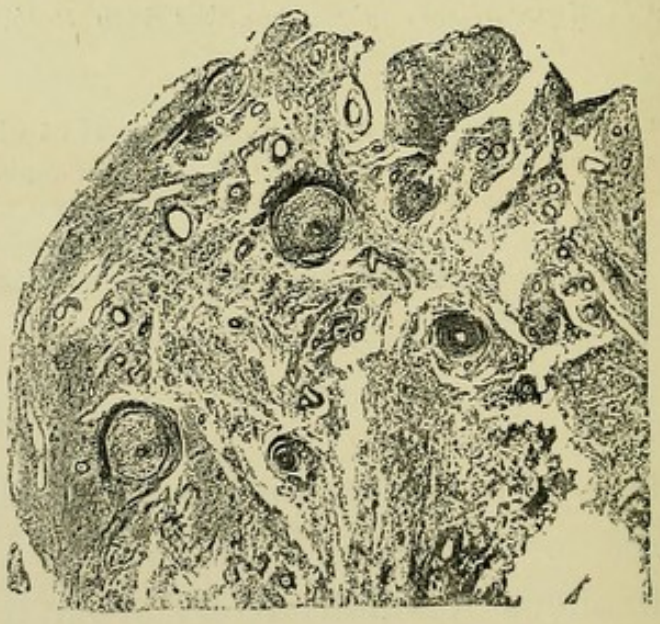


FIG. 51.

(For description see opposite page.)

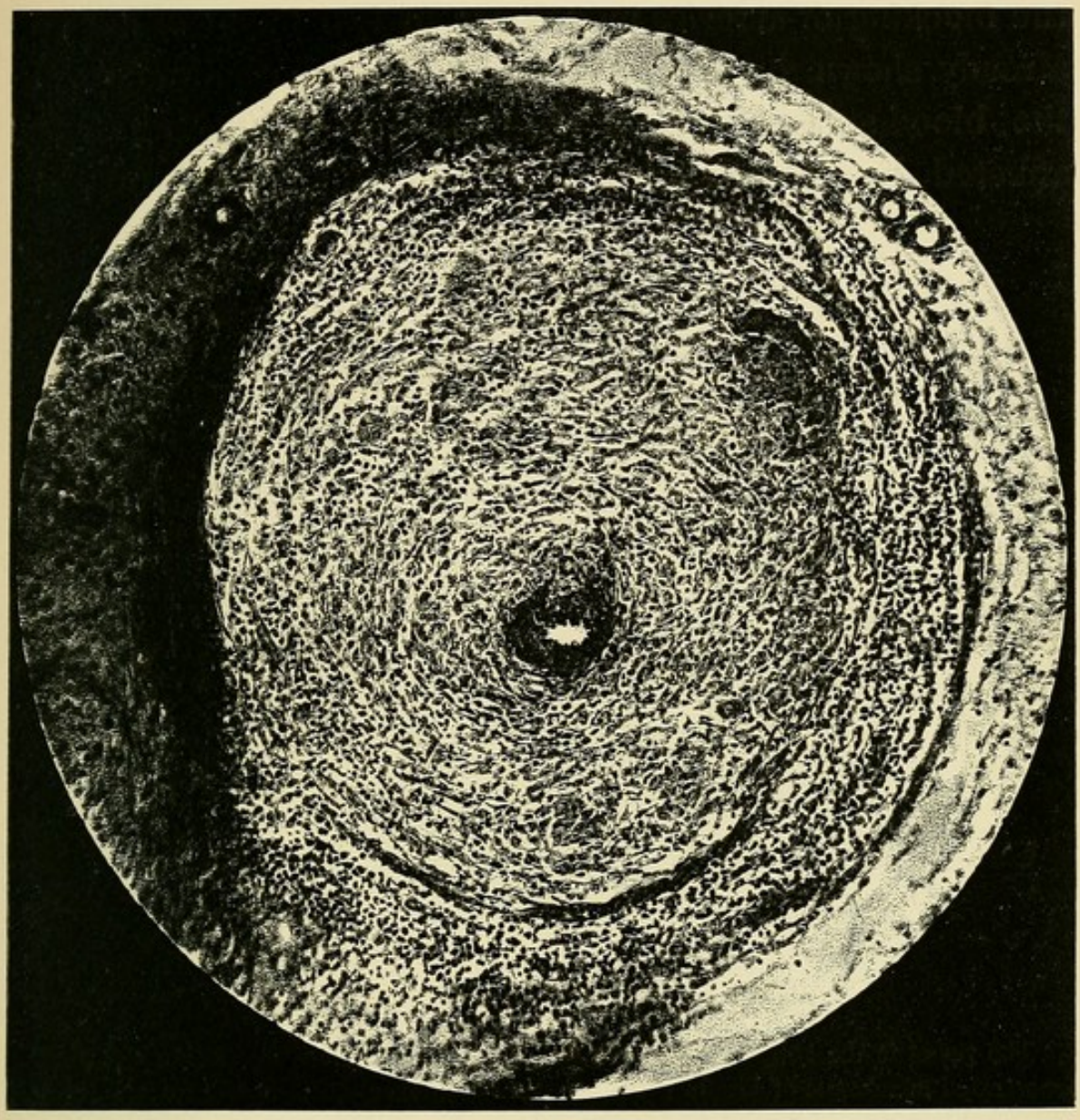


FIG. 52.

FIGS. 51 and 52. Tubercular Arteritis.

The figures were reproduced from microphotographs kindly lent to us by Mr Watson Cheyne.

Tuberculosis of a joint was produced experimentally by inoculation. Figure 51 shews a portion of synovial membrane the arteries of which are seen blocked by growth of their intima.

Figure 52 represents one of these vessels more highly magnified. The great thickening of the intima is seen; only a small central aperture remaining. The structure of the rest of the vessel is obscured by inflammatory changes.

Both in syphilis and tuberculosis it is probable that the blocking of arteries may occur in two ways: either as already shewn, by the infection of the endothelium from the blood-stream; or by the vasa vasorum being first affected by the involvement of the outer coat of the

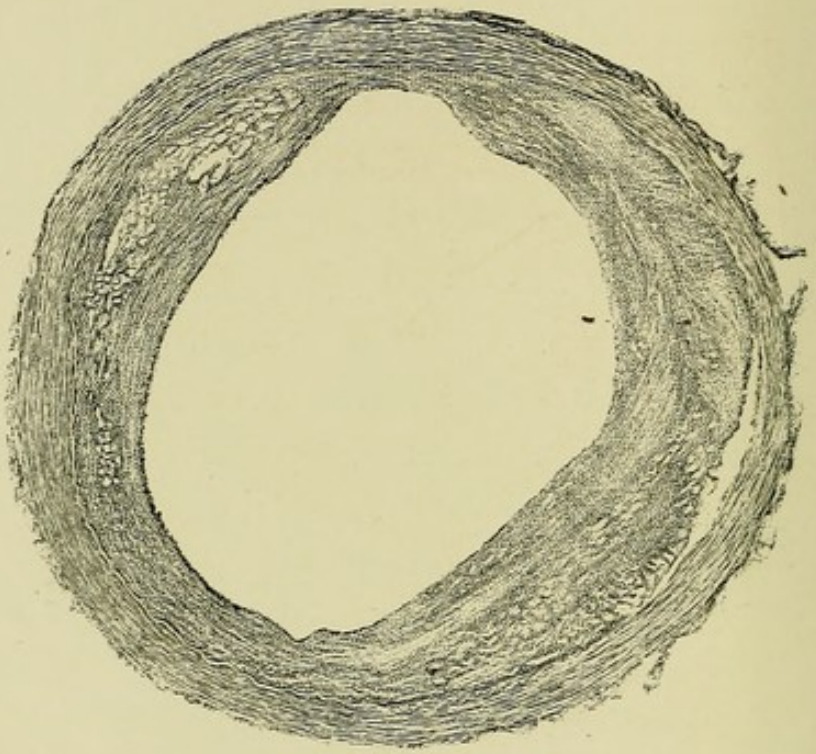


FIG. 53. Basilar Artery (× 20).

The vessel is the subject of chronic endarteritis. On close inspection the wavy fenestrated membrane of Henle can be seen, and internal to it is the intima enormously thickened. At some places the outer part of the inner coat is undergoing degeneration. We are indebted to Dr Sharkey for this specimen.

artery in neighbouring disease, but even in this case the blockage appears mainly due to the secondary hypertrophy of the intima, which also is what occurs when a ligature is applied so as to occlude without rupture. (Figs. 90, 94.)

In atheroma or chronic arteritis the thickening is at first confined to the deeper layers of the intima as shewn in figure 53: its origin is generally ascribed to increased

arterial pressure: in conditions of diminished arterial pressure, on the other hand, as in the obliteration of the ductus arteriosus and the hypogastric arteries, and after ligature with the formation of clot, the increase of the intima is chiefly in its innermost layers.

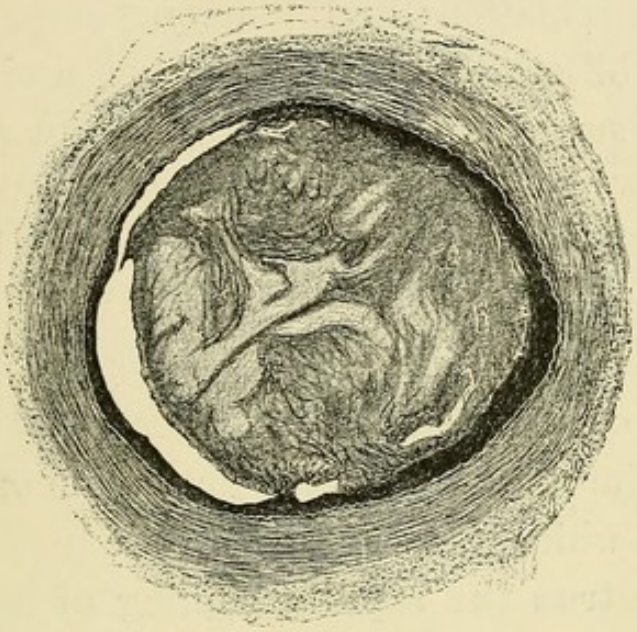


FIG. 54. Transverse section of Popliteal Artery (× 8).

From a man aged 35 who had gangrene of the right foot. There was no visceral disease and the case was apparently an example of the "Endarteritis Proliferans" of Friedländer. In the section the wavy fenestrated membrane of Henle can be seen on close inspection. Internal to it the thickened intima is represented black. A clot nearly fills the vessel. The darker area of the clot is permeated by cells derived from the endothelium which have partly transformed it into connective tissue. The narrow white spaces are all that is left of the lumen of the vessel: here alone the circulation continued.

In the endarteritis proliferans or arteritis obliterans of Friedländer and von Winiwater, which the latter had an opportunity of examining microscopically, it is stated that the obliteration is the result of a great development of the endothelium which narrows the lumen of the vessel until the final obstruction is brought about by thrombosis.

New vessels were found by v. Winiwater penetrating from the outer coat into the proliferating endothelium; which, with the thrombus, was seen in the older parts to be undergoing changes similar to those occurring in closure of an artery after ligature. (Fig. 54.)

Billroth states that the disease is preceded by feebleness of the circulation; and Warren compares it to the process of obliteration of the ductus and hypogastric arteries, and suggests that in both cases a similar cause may explain the phenomena observed; namely, that a diminished blood supply is accompanied by a partial cessation of the function of the arterial walls, and that the formative process is simply a physiological participation of the coats of the vessels, made necessary by the diminished vascular activity or requirements of the tissues or organs to which they are distributed.

If this be true the rapid shrinking of a great artery after amputation and the growth of the intima within it, is easily explained by the abolition of its main functions.

Ziegler says that obliterative endarteritis "may take "place not only in the smaller arteries, but even in the "largest trunks, especially at points where branches are "given off; thus, for example, the subclavians and carotids "affected with endarteritis obliterans may be occluded by "a thickening of the intima at their points of origin from "the aorta."

This disease, though it may occur in old people, cannot properly be termed malum senile arteriarum, for it also occurs in young adults, as is shewn by Pearce Gould's case at the age of 19 years, and Hadden's case at the age of 35 years. Langhans and Köster assert that a certain amount

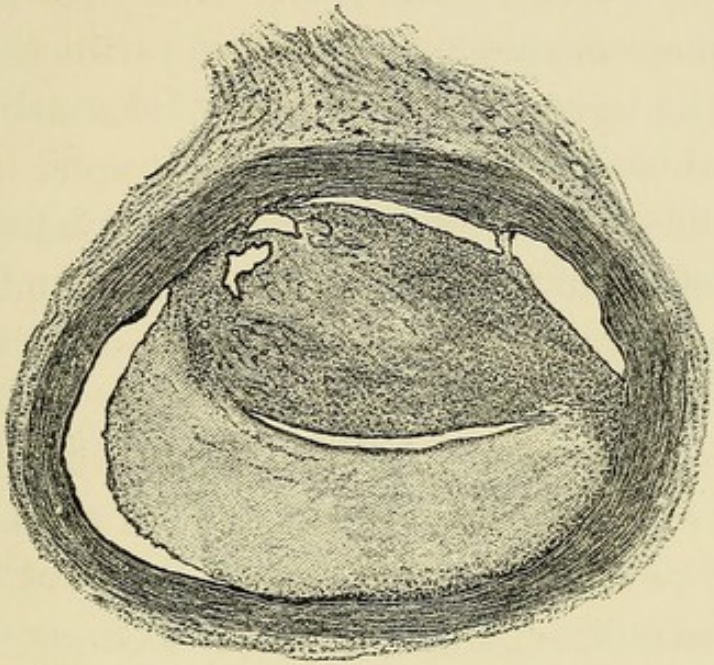


FIG. 55. Obliterative Arteritis of Femoral Artery (× 10).

A man aged 60 first suffered from severe pain in the left leg and foot: two months later the foot became very cold, and no pulsation could be detected in the tibials at the ankle. One month later dry gangrene appeared in the great toe and on the dorsum of the foot: one month later foot became quite warm, and gangrenous patches became moist: pain continued in limb, femoral pulse good: two months later amputation at lower third of thigh: femoral artery here quite blocked: ligated with stay-knot of floss silk: stump healed by first intention.

On examination of the limb the femoral, popliteal, and the greater part of both tibials were blocked: at the ankle both tibials were patent for a short distance: the popliteal artery was considerably calcified.

The drawing shews a transverse section of the femoral artery immediately below where divided: the outer and middle coat are easily recognised: the thickened intima is represented by a black line: it will be seen that there are two masses of clot within the vessel, one dark, the other light: they are both undergoing absorption and replacement by connective tissue, and are of different ages: a week previous to amputation pulsation was felt in this vessel; it is probable that at that time the younger of the two clots was not then deposited: the white spaces are blood channels lined by endothelial cells.

We are indebted for this specimen to Mr Pitts, under whose care the patient was.

of thickening of the intima is common in old age, and may be regarded as physiological; and as changes in the vessel wall from disease affect the nutrition of the parts which they supply, so conversely, a diminution in the nutritive activity of the tissues produces a physiological compensatory textural change in the wall of the vessel. (Fig. 55.)

These illustrations of pathological conditions amply shew the power which the inner coat possesses of proliferation independent of traumatic lesions such as rupture. The only other disease which need be mentioned is periarteritis. All writers, however, admit the power of the cells of the adventitia to develop and form scar-tissue, so that there is no need to dwell upon the affections which implicate the outer coat, and cause thickening of it.

Periarteritis acts in the same way as a non-cutting ligature—causing proliferation of the intima and thus producing obstruction.

Wyeth writes, "if argument be needed to strengthen "this position" [that there is no necessity to rupture the coats to obtain obliteration], "how unanswerable is this "that this process of proliferation of the intima into granu-"lation projections is so extensive in some cases of periar-"teritis that occlusion by thrombosis occurs. Here surely "there is no division of the coat, not even contact of "surfaces, but an inflammation of the intima resulting "from pre-existing periarteritis."

Finally, there are certain cases recorded in which large arteries have been occluded by the pressure of tumours. In this way the tunica intima of one side is brought into relation with the tunica intima of the opposite side, the result being that if the sides are kept in apposition for a

sufficient length of time, the lumen becomes permanently occluded, and the artery at the point of pressure converted into a solid fibrous cord.

There are several specimens illustrating this in the various museums of London; for instance:—

1. Guy's Museum, 1486[80]. An aneurism of the aorta has in this way quite blocked the left carotid.

2. St Bartholomew's Museum, 1498. Shews obliteration of the subclavian and vertebral by simple outside pressure by an aortic aneurism.

3. Guy's Museum, 1519[55]. Shews obstruction of femoral artery from pressure of a psoas abscess. The pressure only lasted a short time and now partial expansion of the vessel and commencing canalisation of the clot are seen. If the pressure had continued for a longer period, or had been permanent, no doubt the occlusion would have been permanent also.

4. There are many instances of the natural blocking of arteries involved in aneurismal tumours in the London Museums, especially those affecting the great arteries at the root of the neck; and there is no more remarkable specimen in existence, in illustration of this point, than the one numbered 1501[10] in Guy's Museum, which shews a large thoracic aneurism which had completely obliterated the orifices of the innominate and left carotid arteries: the patient lived one year in fair health without a carotid or right radial pulse.

This specimen is encouraging for it seems to shew that if only the coats are not ruptured, the great vessels at the root of the neck may be successfully ligated.

Such, then, is the method by which Nature obliterates vessels in disease, and here, as in the case of physiological occlusion, two things are to be noted; firstly, that the rupture of the coats is by no means necessary to occlusion, and secondly, that the coats not being ruptured hemorrhage does not occur, and that too, although grave pathological changes are in active progress. Ruptured aneurisms, great and small, no doubt occur, but when once Nature has succeeded in obliterating the vessel, the danger from hemorrhage is at an end: not so in surgery, where hemorrhage too often occurs not only from the aneurism but also from the ligated vessel above: if surgeons would only follow Nature's method they might attain a like success.

CHAPTER VI.

CONDUCT AND FATE OF THE CORPUSCLES.

Question stated. Methods of Investigation. Ziegler's Chamber. Contents of Chambers at different dates. Further changes in Sealed Chambers. Conclusions as to conduct and fate of Leucocytes and Plasma Cells.

As the ligation of an artery results in the formation of connective tissue, it is desirable at the outset to ascertain as far as may be the nature of this process: especially is it advisable to determine if possible the parts played respectively by the connective tissue corpuscles of the arterial wall and by the migrated leucocytes of the blood. The formation of scar-tissue being the same in all regions of the body, it is convenient to study the process, firstly, by the ingenious method devised by Ziegler, and secondly,

by the examination of such small arteries as lend themselves readily to the most exact methods of histology.

In these investigations we were happy in obtaining the advice and aid of our friend Dr Sherrington: a full account of the results obtained is given in a paper by him and one of us on the "Formation of Scar-tissue" in the *Journal of Physiology* for 1889.

The main question at issue is whether the new tissue is formed from migrated leucocytes or from connective tissue corpuscles, or from both. The view that the leucocytes are the primary source of the new tissue has been maintained by Cohnheim, Senftleben, Heidenhain, Schede, Tillmans, Ziegler, and others: the opposite view by Baumgarten, Böttcher, Thiersch, Riedel, Hamilton, Heuking and Thoma, and others. It is worthy of remark that on this latter side are to be found nearly all those who have studied the process as it occurs in the occlusion of blood-vessels.

Methods employed.

Two circular cover-glasses, each $\frac{5}{8}$ of an inch (15 mm.) in diameter and 0·006 of an inch (0·15 mm.) in thickness, were fastened together so as to form a little flat glass chamber, in the manner employed by Ziegler. A strip of tin-foil placed between them at their edge along $\frac{11}{12}$ of their circumference was cemented by shellac on each face to the corresponding surface of the cover-glass. The tiny chamber thus formed had therefore between the two ends of the strip of tin-foil an opening into the interior. Tin-foil $\frac{1}{20}$ mm. in thickness was employed. With this thickness membranes were obtained between the cover-glasses

that made satisfactory microscopical specimens. Plate VI., Figs. 3 and 5.

These chambers for eight-and-forty hours before use were emptied of air and filled with distilled water previously sterilized, or with nutrient broth containing peptone according to the recipe of Koch. Both the chamber and fluid in which it was kept were again sterilized by heat an hour or so before being used for experiment. In a few instances the air was not entirely expelled.

The animals employed have been in all cases rabbits or guinea-pigs. During every experiment the animal has been deeply under the influence of an anaesthetic. Antiseptic precautions were vigorously maintained throughout all the operations. No suppuration ever occurred. Had it done so in any experiment we should have excluded the results of that experiment. In the earlier experiments the chambers were placed in the peritoneal cavity; in the later into the subcutaneous connective tissue of the flank.

The chambers were allowed to remain within the animals for various periods, from four hours at shortest to 18 days at longest. When the chamber was removed its contents were examined either fresh upon a warm stage under the microscope, or after appropriate treatment with hardening and staining reagents. The outsides of the chambers were often covered with thin films of young fibrous tissue—these films were examined by the same methods as were the contents. The reagent chiefly employed was osmic acid, either in freshly made 0·5% watery solution, or in vapour from a 1% solution. In the former case the chamber taken warm from the body was at once

plunged into the osmic acid solution or was rapidly split open, and with the contents so exposed, placed in osmic acid. In the solution of osmic acid they remained for an hour in the dark. Where osmium vapours were used the same plan was adopted, except that the chamber was always opened unless bubbles of air happened to have got ingress previously, as sometimes happened. Exposure to the vapour was ensured by placing the specimen between two watch-glasses, at the bottom of which was osmic acid solution, or by suspending from the cork of a bottle containing the solution. The action of the osmic acid was allowed about two hours' play, always in the dark. The preparations with osmic acid were, after washing in water, mounted either in Farrant's gum solution, or after dehydration, in xylol-balsam. They were in some instances after-stained, with picrocarmine, with fuchsin, methylene blue, eosin, or most frequently with hæmatoxylin (Ehrlich's solution). Other preparations were made without the use of osmic acid. In these after hardening in chromic acid, or in alcohol, or Flemming's solution, picrocarmine, methylene blue, eosin, fuchsin, hæmatoxylin, etc. were used.

For the observations on living specimens a warm stage of Stricker's pattern was adopted.

Contents of the Chambers.

One of the first steps which we took was to examine the serous moisture of the abdominal cavity of the rabbit and guinea-pig, and of subcutaneous wounds and the blood, in order to ascertain the characters of the cellular elements

contained therein. The examination was conducted by the cover-glass method first recommended by Koch and Löffler. Eosin, fuchsin, saffranin and methylene blue were used as stains. Some preparations were made by exposing the moist film on the cover-glass to vapours of osmic acid solution.

The examination revealed the presence in the serous moisture and in the tissue-plasma of at least two kinds of cells. The one kind resembled in all respects the leucocyte, the colorless cell of the blood. This kind was in fact indistinguishable from the leucocyte.

A second kind of cell was also present and sufficiently numerous. This cell, which is much larger in size than is the former kind, when fixed by osmic acid vapour, often presents a discoid figure, some 30μ to 40μ across; not infrequently however, and more frequently than not when fixed by plunging into osmic acid solution, or by drying, as in the Koch-Löffler method, the cell outline is irregular, often angular, with especial prominence of one angle or of two; in the last case the cell might be described as fusiform. Whatever the shape of the body of the cell may be, there always lies within it, generally towards the centre, a large oval vesicular nucleus, itself somewhat larger than the red corpuscle of the blood. It does not stain so darkly as does the nucleus of a leucocyte. The substance of the flattened plate-like cell-body is markedly granular, particularly so when prepared by the rapidly drying method. The protoplasm reduced to a flattened flake as it generally is, may be of such tenuity near the margin that in the fresh condition and in many osmic acid preparations the outline of the cell is somewhat difficult to distinguish. The

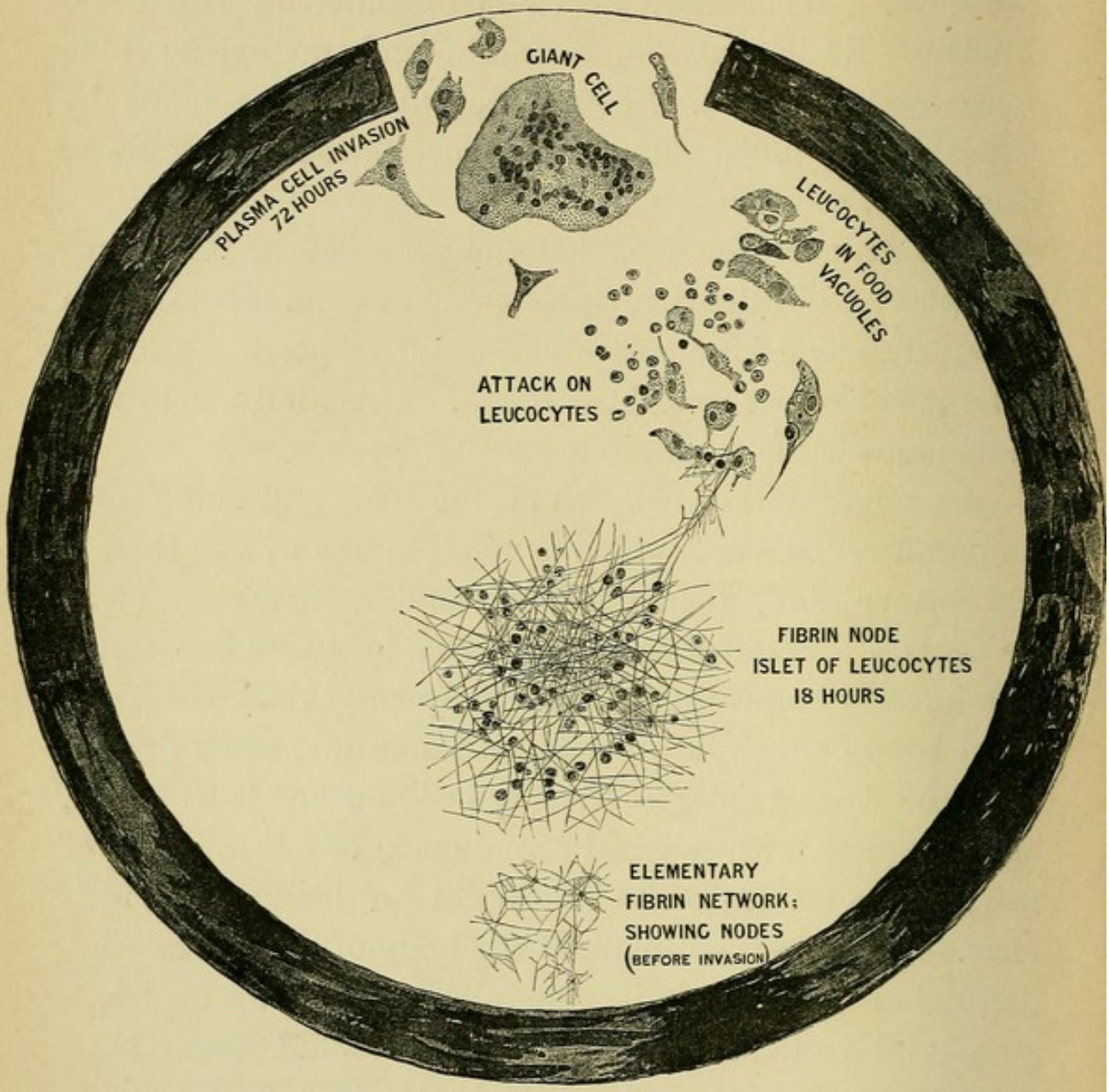


FIG. 56. Ziegler chamber as used in the experiments.

The opening is seen at the upper part: the contents of the chamber are drawn to the same scale (× 150), with the exception of the "elementary fibrin network," but the chamber itself is drawn to a much smaller scale.

The appearances represented have been taken from different chambers of various dates, the times being shewn in the figure.

It will be noticed that in the "islet of leucocytes" the nuclei of many of the corpuscles are triboled or crescentic, a condition indicative of loss of vitality: thus they fall a ready prey to the more virile plasma-corpuscles which follow and devour them.

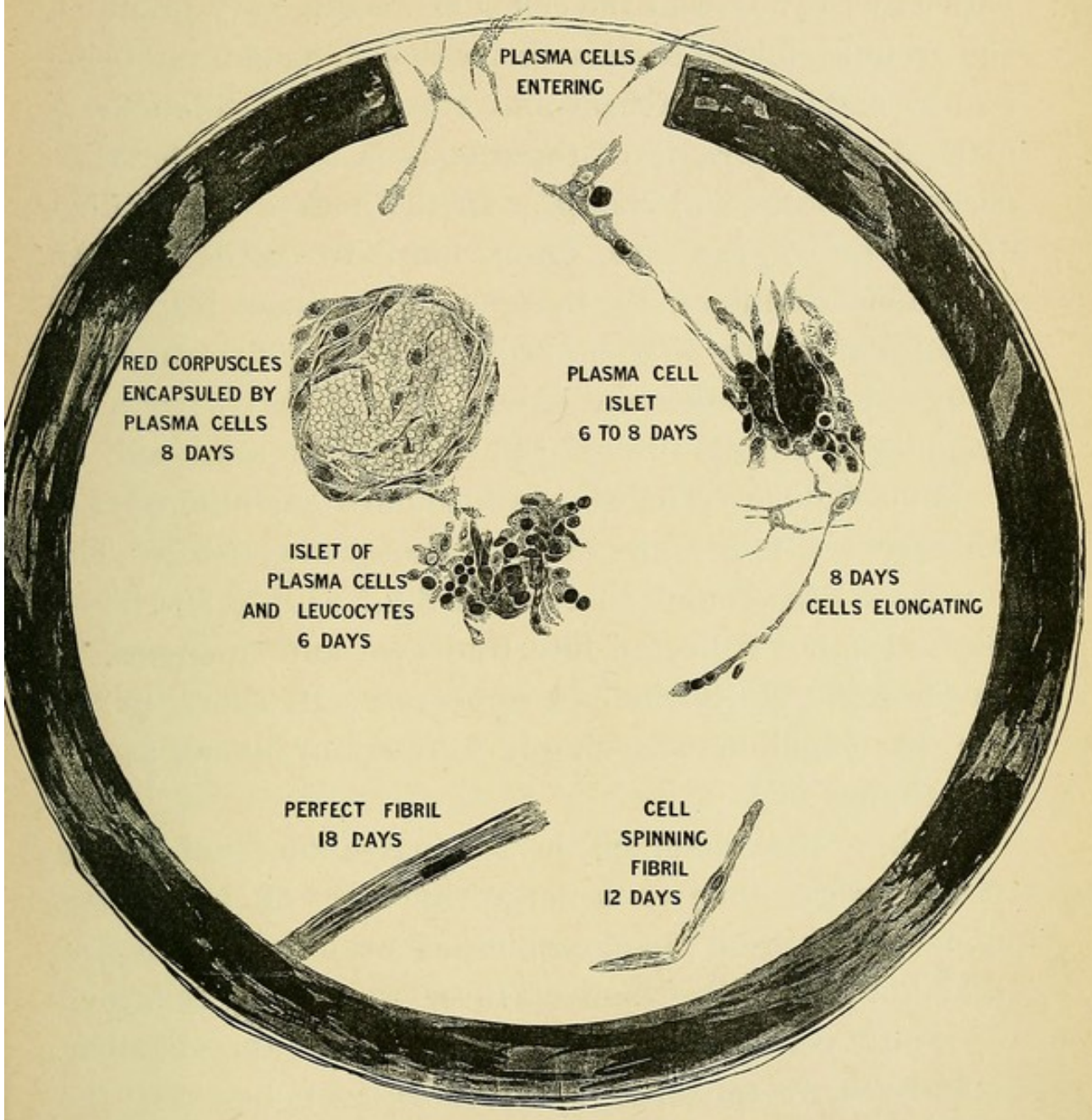


FIG. 57. Ziegler chamber as used in the experiments.

The opening is seen at the upper part: the contents of the chamber are drawn to the same scale (× 150), but the chamber itself is drawn to a very much smaller scale. The appearances represented have been taken from different chambers of various dates, the times being shewn in the figure: it must however be borne in mind that the length of sojourn of the chamber in the tissues is no guide to the date of entry of a plasma-corpuscle or to the age of the corpuscle if born in the chamber.

determination of the limits of the cell is however rendered easier by the fact that the cell substance is very granular, the granules taking a sepia tint with osmic acid, and being readily stained by fuchsin and other aniline colours. A marked characteristic of these cells is their tendency to occur in masses and clumps. In the masses the outlines of the individuals are often hard to recognise. In specimens examined fresh upon the warm stage the granules are many of them brilliant and highly refracting, though not to such an extent as are fatty particles. Granules of various size exist in one and the same cell.

Four Hours.—The shortest sojourn we allowed the chambers was four hours in the subcutaneous tissue. The chambers were found to contain fluid with a few blood corpuscles, not collected into rouleaux, and unaltered in appearance. No fibrin had appeared. In short, mixed with the bouillon was simply a trace of blood, as yet unclotted.

Within the chamber in the neighbourhood of its opening were however a large number of leucocytes, unmixed with red blood-corpuscles or indeed with any other kind of cell. None of these wandering cells had apparently penetrated far into the recess of the chamber, because there appeared an obvious gap between the position of their pioneers and the diluted blood elements occupying the chamber elsewhere.

Nine and a Half Hours.—Nine and a half hours after insertion into the abdominal cavity, the diluted blood was also found unclotted—although in this instance the chamber had been filled not with bouillon but with water sterilized. The trace of blood which almost unavoidably

found its way into the chambers at the time of their insertion had remained unclotted in all specimens examined earlier than fifteen hours. The bouillon containing peptone, seemed, as indeed one might have expected, to retard the clotting of the blood that entered the chamber. In chambers filled with nutrient peptone-bouillon no clot was ever found earlier than twenty hours after the original implantation. On the other hand we found a very considerable formation of fibrin in a chamber that had been filled with sterilized water and then allowed a sojourn of eighteen hours in the subcutaneous tissue. The nutrient bouillon contained commercial peptones, and we think the peptones may not so rapidly diffuse but that this retardation of the clotting may be explained by their presence.

In the early specimens of fibrin production in the tissue-plasma which filled the chambers we had abundant examples of the formation of fibrin filaments as fine straight or very slightly curved lines, irregularly radiating from granular nodal masses. The nodal granular *débris*, under various reagents, appeared to consist, as the generally accepted view affirms, of altered blood-platelets, and leucocytes undergoing alteration.

About the nodal points of the network of fibrin the leucocytes were grouped. Both on the warm stage and after fixation by osmic acid these leucocytes displayed only rarely any deviation from the spheroid form. There were indeed some instances in which they presented a fusiform outline, or possessed a tiny process jutting from the cell-body. Such examples were extremely sparse, and occurred only in the neighbourhood of the opening of the

chamber, and in certain situations to be specified immediately. For the most part the cells seemed in an inert condition, as far as one can judge of their activity from their form. In the short time between extraction of the chamber from the body and the fixation in osmic acid or the observation of the cell upon a warm stage under the microscope, their vitality had suffered sufficiently for the cell to have assumed its zero of shape, the subspheroid figure. Or for some reason existence with the Ziegler's chamber was not conducive to activity of the protoplasm, yet from what we shall relate there is no good reason for thinking that the leucocytes within the chamber are in a very different state from those invading the inflamed tissue without. The latter supposition is favoured by the fact that the larger cells, plasma-cells as we shall term them, also found in the chambers under similar circumstances, although subjected to the same *technique* of preparation as these leucocytes, shewed well-marked amœboid movements.

Indeed if the latter of the two suppositions just suggested be not accepted, it becomes necessary to assume that of these two kinds of cell the leucocyte is much the more perishable and delicate, and was practically annihilated by a simple procedure that did not appear to interfere with the vitality of its co-occupant the plasma-cell.

Here must be mentioned another sign of degeneration in the leucocytes examined in these chambers. Many of them shewed the triple and multiple nuclear bodies that are universally regarded as evidence of the lethal disintegration of the nucleus, or, as Flemming names it, the

"fragmentation" of the nucleus. On the other hand the cell-body of the leucocyte was not granular or fatty, but fairly evenly though deeply tinted by the osmium. These points are seen in Plate IV., Fig. 1.

Eighteen Hours.—In chambers removed after the appearance of fibrin within them, but before the stay within the body had exceeded eight-and-forty hours, it was usual to find a number of areas in which leucocytes were present in much greater numbers than elsewhere. Plate IV., Fig. 5.

The tendency to collect to certain points which the leucocytes evinced in even very early specimens was more marked in these later preparations. About the nodal points of the fibrinous network crowds of them were present. The outlying individuals were frequently arranged in lines along the converging filaments of fibrin. The older within certain limits these films of coagulum the more obvious the aggregation of the leucocytes into certain groups. For convenience on account of their prominence and apparent importance in subsequent stages we have been accustomed to refer to these groups shortly as the cell-islets. Cf. Plate IV., Fig. 3. They are little collections of cells, occurring constantly, scattered about in the thin cellular membranes which grow over and within the glass chambers. Some are obvious to the naked eye, especially when the film has been treated with carmine or with hæmatoxylin, which shew them as deeply colored points. They vary in size from a small pin's head downward. The larger islets are often compounded of smaller ones. The smallest display best what we believe to be the structure originally characteristic of all:—a centre of

amorphous albuminous *débris* surrounded by leucocytes; less frequently one or two altered red blood corpuscles form the centre.

It was in specimens of this date that the first evidence of the presence of another cellular element than the leucocytes and red cells of the blood was found. Cells similar to the large flattened plate-like forms of the peritoneal moisture, already adverted to, began to be found in the chamber. The time of their advent varied within narrow limits when the chamber rested in the subcutaneous tissue; when in the peritoneal cavity there was much greater variation in respect to time. This we believe was due to the chamber not coming to rest in one particular spot for some time after introduction into the abdomen. The movements of the viscera seemed able to shift it and prevent its forming adhesions. When put into the abdomen we always placed it about an inch to right or left of the little wound in the linea alba through which it was inserted. But we never found it anywhere near that situation after a sojourn of more than a few hours. In the course of four-and-twenty hours the chamber had nearly always passed toward or actually into the position which it almost constantly came permanently to take, that is, a little distance from the median line in front of the psoas muscle at the very root of the mesentery. Once there it appeared in a few hours to contract adhesions, and become fixed in a permanent fashion. Until bound down by adhesions, the full complement of cells did not reach the interior of the chamber.

In one instance, in a chamber exposed for eighteen hours in the subcutaneous tissue, plasma-cells were

found in considerable numbers near the opening. They were indistinguishable in appearance from the plasma-cells of the normal subcutaneous tissue, except that a greater variety of individual form was to be seen in them. In later specimens the plasma-cells were found scattered throughout the whole chamber, although most numerous near the opening.

Seventy-two Hours.—In a preparation from a chamber which had been seventy-two hours in the subcutaneous tissue, plasma-cells entered into the formation of the islets even in the portions furthest removed from the opening. At the opening, however, no other kind of cell was mixed with them, which was not the case elsewhere. No forms intermediate between the leucocyte and the plasma-cell were to be found; they were repeatedly expected and repeatedly looked for, but the search was unsuccessful.

The preparations gave an almost bewildering number of examples of the infinite variation in shape of the large amœboid plasma-cells, which also varied very considerably in size, and as to granules. The body of the cell was for the most part plate-like, being in many instances extended into so thin a film that its exact limit was hard to determine, especially when, as occasionally happened, the granules of the cell-body were less pronounced towards the periphery. Some idea of the wide diversity of outline exhibited by individual cells may be gathered from the figures.

It must not be thought, however, that in any of its forms the plasma-cell could not be distinguished with certainty from the leucocyte. In the same way as in the peritoneal moisture and in the plasma of subcutaneous

tissue, so here the former both on the warm stage and in osmic preparations is characterised by larger size, coarser granules, the constant presence of a single clear nucleus of oval figure, and by the differences in staining qualities and mobility already referred to. We may here add that these differential characters may be obscured by faulty methods of examination.

In the specimens obtained from chambers that had rested for seventy-two hours in the subcutaneous tissue of the guinea-pig, we found individuals among the plasma-cells, which shewed well-marked vacuolation. Plate IV., Figs. 1, 2, 4. For the most part the matter within the vacuole was a granular *débris* that furnished no sufficient clue as to its nature. But in a few it was indisputable that the vacuole contained, more or less altered but still perfectly easily recognisable, a leucocyte or red blood corpuscle. In Fig. 2 is shewn the appearance presented by one of these cells. A large vacuole contains a somewhat faintly-stained body, which is finely granular and indistinctly nucleated. It is a little smaller than is the nucleus of the plasma-cell itself. Fine threads seemed to pass from the sides of the vacuole across the cavity to the substance of the included leucocyte. Taken with the context afforded by examination of other cells in the neighbourhood we believe that this and other similar instances were examples of leucocytes lying in vacuoles in the plasma-cells. Many stages of ingestion could be found. Simple approximation, the hollowing out of a little bay in the side of the plasma-cell into which the leucocyte was as it were drawn, partial inclusion, total inclusion—all these were exemplified. And further there

were many vacuoles in which mere granular *débris* lay. This *débris* was, we think, probably the still undigested remnant of the ingested leucocyte or red blood cell. We doubt whether without very special apparatus the cells of the tissues of mammalia can be kept in sufficiently normal condition for sufficient length of time to compass observations on ingestion by living cells; we were however much assisted in the interpretation of the appearances of the osmic fixed preparations by the processes described by Miss M. Greenwood for the Rhizopoda. Her observations were conducted on living specimens of Amœba proteus and Actinosphærium, and she was able to follow in these animals under the microscope all the visible phenomena accompanying the ingestion of prey. In our preparations we had as it were a number of amœbæ, many of which had been actively engaged in ingesting living prey, immediately before the reagent had been used that killed them so rapidly as to allow no time for any great departure from their previous aspect.

Nor were leucocytes the only bodies to be found within the substance of the plasma-cell. Red corpuscles of blood were recognisable in them. Very frequently along the border of the space in the chamber, the plasma-cells lying in great numbers near the cement (shellac glue) which fixed the strip of tin-foil to the glass, were filled with tiny droplets of oil that became deep black under the treatment with osmic acid. Sometimes the entire cell was dotted, except just round the nucleus, with fine fatty particles of a fairly equal size: sometimes the oil was collected into a few much larger globules. The cement itself turned deep black under osmic acid treatment.

There was little room for doubt that the black particles in the plasma-cells were derived from the cement near the cells; whether the cells took up the particles without altering them, or whether the particles were in any degree a food for the cells are points we can give no answer to.

Contiguous plasma-cells or even those a little distance apart were often connected together by their processes. The bands of connection might be short thick arms or long gossamer threads of protoplasm. By similar arms and threads the cells seemed to adhere to the most diverse objects in their surrounding. The surface of the cover-glass, a filament of fibrin, a hair, a fibre of cotton, a lump of the cement fastening the sides of the chamber together, all afforded points to which the processes from the plasma-cells would cling. Plate VI., Figs. 1 and 2.

There were present also in chambers of eighteen hours', twenty-two hours', twenty-six hours', forty-eight hours', and seventy-two hours' standing, as also in others of older date containing well-formed granulation tissue, many giant cells (Plate V., Fig. 1)—huge multi-nucleate cells, that obviously in many instances were cell-fusions. Congregations of large plasma-cells as before mentioned were frequently met with. They adhered one to another in groups. And here many collections of them existed intermediate in character between those groups in which the individual cells were agminated but easily distinguishable from one another, and giant cell masses in which the nuclei were the only guides to the individual position of the coherent members. Some appeared to be cell-fusions; many did not. In these latter the nuclei were gathered together into an irregular heap. The ring-like arrange-

ment of the nuclei frequently found in the giant cells of tubercle was never observed in these membranes.

Of nuclei in these giant cells there existed apparently two kinds. One was large, clear, and oval, having all the characters of the nucleus of the separate plasma-cell; it was invariably present in all the giant cells. The other sort was smaller, round, more darkly tinted by osmium treatment, and was not invariably present, that is, did not exist in every giant cell, but was in some cells even more numerous than the larger oval variety. We doubt very much the accuracy of describing the latter smaller bodies as true nuclei. We incline to believe, from their great similarity to some of the leucocytes observed in the plasma-cells, that they are nothing but leucocytes surrounded by the substance of the giant cell and somewhat altered in appearance. Against this supposition is the fact that there was often no indication of a vacuole-space around the ingested cell, but in support of it the substance of the plasma-cell was seen sometimes very closely applied to the ingested leucocyte in instances in which there was very little doubt as to the nature of the included body. In osmic preparations there is generally a light space free from granules immediately around the oval nucleus of the plasma-cell that simulates somewhat closely the appearance of a vacuole about the nucleus itself.

Miss Greenwood, in a critical and suggestive letter written after perusal of the paper in the *Journal of Physiology*, says, "Apropos of vacuoles, you mention that in some "giant cells there are bodies which but for the absence of "surrounding fluid might be leucocytes undergoing diges- "tive change. Now I used to notice that there was some-

"times loss of vacuole in Amœba while digestive change "was still incomplete, and this loss of course brought the "substance of the prey into direct contact with the endo- "derm: in fact I thought the digestive activity was "obscurely phasic. I suppose the same disappearance of "fluid might occur in plasma-cells for unknown reasons; "and since your osmic preparations perpetuated one "moment in the life of your specimens, there might have "been a vacuole earlier, or it might be destined to appear "later. You see I am trying to get in a vacuole somehow, "believing that a fluid medium is essential for the mani- "festation of primitive digestive activity."

Miss Greenwood also suggests that the absence of vacuole around ingested material may indicate that it is innutritious, and that thus nutritive material may be distinguished from innutritive.

Advancing further into the chamber, in the specimens of more than forty-eight hours' duration, the plasma-cells begin to apply themselves to the islet-groups of leucocytes. They surround the leucocytes. The islets come to consist of a central portion made up of leucocytes and an outer zone of large and granular plasma-cells. In this way the islets seem to increase rapidly in size. Neighbouring islets appear to become merged together. Giant cells are frequent in them, especially, it would appear, near, although not actually at, the centre. Most of the growth that went on in the membrane appeared to consist in enlargement of individual islets, and the fusion of neighbouring islets. The islets appeared to be the chief growing points of the tissue. But it is true that gradually a more or less continuous sheet of plasma-cells is formed over the intervening

space between the islets. When very thin the inflammatory membrane consisted of a layer of scattered cells lying separated by considerable but fairly regular distances one from another. Each individual cell was of a discoid or fusiform figure, and granular, with a large clear nucleus. The edge of the disc was thin and often deeply scalloped; it merged, under all methods of staining used by us, at certain points quite imperceptibly, in a tenuous film which composed the bulk of the membrane proper. When fixed with osmic acid and after-stained with hæmatoxylin (Ehrlich's), this membrane is shewn to contain, if not to be entirely made up of, a feltwork of filaments, like filaments of fibrin. These cross in every direction in the plane of the membrane, without prominent arrangement in any one particular sense. The individual filaments vary a good deal in size.

It was among the plasma-cells of the fringe of the islets that we noticed the earliest regularly fusiform cells, the immediate precursors of fibrous elements in the new tissue. It is true that plasma-cells of an irregular spindle-shape were observable not rarely among even the earliest of the plasma-cell swarm entering the chamber. But in those instances the outline was probably but one of many which the amœboid cell successively assumed, and generally it was not of the same character as the regularly fusiform type prevailing among these plasma-cells in the outskirts of an islet. In that latter the majority of the cells lay in lines concentrically set about a core of ill-stained, broken-down matter that composed the centre of the mass. Plate V., Fig. 4. The fusiform fibroblasts began in fact the encapsulation of the *débris* of the breaking-down blood-

cells, &c. The lengthening out and assuming of a regular spindle form took place also very early in those cells that had become attached to hairs and cotton fibres, and lumps of the shellac glue. They were soon found adhering there in rows of regular disposition, the rows consisting entirely of typical young fusiform fibroblasts.

Later than seventy-two hours.

Older specimens revealed further progress in the formation of a fibrous-tissue membrane. After a stay of eight days, or ten days, or fourteen days in the subcutaneous tissue in many instances the islets consisted of plasma-cells alone. The leucocytes had disappeared. The pigmented remnants of the red blood corpuscles were much longer traceable. In many places along certain lines the spindle-shaped cells had become attenuated, and formed distinct bands and often long and delicate cords. In many places in the tenth day specimens, and in some of the eighth day ones an inter-cellular substance shewing fibrillation exists. This extra-cellular matter is well seen where, as occasionally happens, a single chain of fusiform fibroblasts, set in end-wise series, has produced a thread-like tiny cord. Each fibroblast appears to lie in a sheath of fibrillated matter. The delicate lines marking the fibrillæ run parallel to the contour of the cell. The fibrillated matter was not tinted by osmic acid or by any of the stains employed by us to the same depth as the granular substance of the cell itself. The granules of the cell-body, the clear oval nucleus, were still marked characters of the plasma-cell, although it might be considered at

this stage to have become a fixed corpuscle of connective tissue.

We were unable to satisfy ourselves on the question as to whether the fibrillated extra-cellular matter had been formed by direct transformation from the surface portion of the cell-body, or whether it had arisen as a secretion from the protoplasm of the cell. But the latter view appears to us the most probable, if only for the reason that the fibroblast cell and its new capsule of fibrillated matter are when taken together much larger than, so far as we have observed, the individual naked fibroblast ever is.

From the islets the bands of spindle cells spread away in various directions. The determination of the direction of the earliest-formed chains of spindle cells seemed to us greatly due to the lines taken by the filaments of the original fibrin-network; the radiation from the same nodal points, the interlacing not always at acute angles but frequently in rectangular fashion.

In membranes of ten, fourteen, and even eighteen days' growth, not all the cells nor even the majority were spindle-shaped. A vast number were triradiate, and multiradiate; some had but one process; very few were rounded. Many recalled to mind the branched fixed corpuscles of the cornea. Long tapering branches united cell to cell, not only the cells of one plane with another, but the cells of different planes also. A meshwork of infinite variety and complexity was thus established. But in all these examples of plasma-cells in the stable as well as in the previously described labile forms, the granular nature of the cell substance and the clear oval nucleus were characters never lost.

In the same manner as did the more delicate strands of fibrous tissue, larger, broader sheets and beams arose. In all the spindle cells side to side as well as end to end are separated by intervening matter fibrillated in a direction parallel to the longer axes of the cells.

It may have been noticed that no mention has been made of any developing blood-vessels in the membranes examined. It is a striking fact that in none of the preparations, not even in the preparations of eighteen days' growth, taken from the peritoneal cavity, did we find in any instance any trace of a formation of blood-vessels. Nowhere were capillaries to be found; although the chambers were bound by adhesions and in the later specimens encapsuled in cicatricial tissue. This observation seems to furnish a negative to the view advanced by Creighton that the giant cells of granulation-tissue are exclusively vaso-factive. Here we had giant cells in abundance, but never any capillary formation. Perhaps the film of tissue in the chamber was thin enough to allow sufficient nutriment to reach the cells by fluid soakage only.

Abstract of some of the Notes of the Experiments

The number of hours mentioned corresponds to the time during which the chambers rested in the bodies of the animals.

1. 4 *hours.* Subcutaneous. No fibrin. No rouleaux of red cells. A large number of leucocytes in the neighbourhood of the mouth of the chamber.

2. 18 *hours.* Subcutaneous.
   High power. Fibrin network very extensive. Crowds of leucocytes at the nodal points of fibrin. Cells circular in

outline, nuclei crescentic or trilobed. At the periphery of the islet a few leucocytes still in an active state and of irregular form. At the mouth of the chamber are a few large plasma and giant cells.

Low power. The islets of cells in the fibrin film are well seen. Indeed they are visible to the naked eye, about the size of pins' heads.

3. 72 *hours.* Subcutaneous.

Islets more marked. Fibrin network very extensive. Numerous large plasma-cells encircling the islets of leucocytes at the nodal points. In the neighbourhood of the mouth of the chamber the plasma corpuscles are more numerous, and the islets are partly made up of these cells. Moreover, numerous red and white blood-cells are visible in the vacuoles of giant cells and in those of separate plasma-cells.

72 *hours.* Peritoneal cavity.

The chamber was quite *free.* No trace of an adhesion.

The chamber seems to have escaped the leucocytic immigration. The islets are formed almost entirely of plasma-cells.

4. 8 *days.* Peritoneal cavity. Fixed by an adhesion; ? how long fixed.

The islets are formed of plasma-cells alone. The leucocytes have all disappeared, a few only are visible in the vacuoles of the larger cells. The cells at the circumference of the islets are lengthened out along the lines of fibrin, and joined with others free of the islets to form a giant cell field, or plasmodium. Every cell is connected by long processes with others, so that looked at in one way the whole field is one giant cell. The islets consist often of one giant cell together with numerous plasma corpuscles. It is only at the periphery of the islets that the contour of the individual plasma-cells can be made out. Foreign bodies such as blood-clot, shellac, etc., are surrounded by a capsule of spindle cells.

5. *Several chambers.* 6 to 18 days in peritoneal cavity. Some fixed, and some *not* fixed by adhesions.

Fibrillation advanced. Every stage can be observed, from the simple fusiform elongation of the plasma-cell to the development of a perfect fibril.

The above experiments seemed to point to a certain definite period at which the migration of leucocytes and connective tissue corpuscles occurred.

In order to examine somewhat further the behaviour of the leucocytes and of the plasma-cells respectively toward the chamber, a slight modification of the mode of experiment was used on two occasions. Two chambers (or four) were placed side by side in the subcutaneous tissue. At the end of twenty-two hours they were taken out, one was dropped into osmic acid, and the other was sealed with warm paraffin. The sealing was done by dipping the mouth of the chamber into a soft paraffin melting at 107° Fahr. (42° C.). Only the part of the chamber immediately next the opening was touched by the paraffin, which was just above the temperature of solidification. After being sealed the chamber was placed in the abdominal cavity of a second guinea-pig, there to remain for incubation.

In this way it was possible to compare the contents of two chambers which had been placed side by side in the subcutaneous tissue, and whose contents were presumably the same at the time of withdrawal. One was then fixed for histological examination. The other incubated for a longer period, no new cells being allowed to enter during this second incubation.

The imperviousness of the chambers after sealing was tested in the following two ways:

(*α*) A little 1% hydrochloric acid was introduced in the chamber, the outer edge of the opening carefully dried, and then the paraffin applied by dipping the mouth just as in the experiment above. The chamber was then placed in blue litmus. No change took place in the litmus, although the chamber remained a week in the solution.

(*β*) A little of an active culture of Spirillum Finkleri was introduced into the chamber, the edge of the opening cleaned, and then sealing performed as before. The chamber was then placed in nutrient broth (in another case in nutrient gelatine) for a week, at a temperature of 35° C. No growth appeared in the broth (or gelatine). In a control tube the broth was turbid in two days.

In the experiments performed in this way the appearances observed in the chambers were alike. The chambers were withdrawn at the end of twenty-two hours, one was then incubated further for another forty-four hours.

The contents at the end of twenty-two hours were as follows, viz.:—

A large number of leucocytes and several patches of red corpuscles. Plasma-cells are also present but very sparsely; they are most numerous at the mouth of the chamber. They are scattered at long intervals. In one place a few plasma-cells are collected around some red cells, and a fibre of wool. Fibrin filaments are present in the chambers taken from the rabbit, but none in those from the guinea-pig.

Contents of sealed chambers after forty-four hours' further incubation:

Fibrin network extensive. The leucocytes lie around the red cell masses. The leucocytes possess for the most

Description of Plate IV.

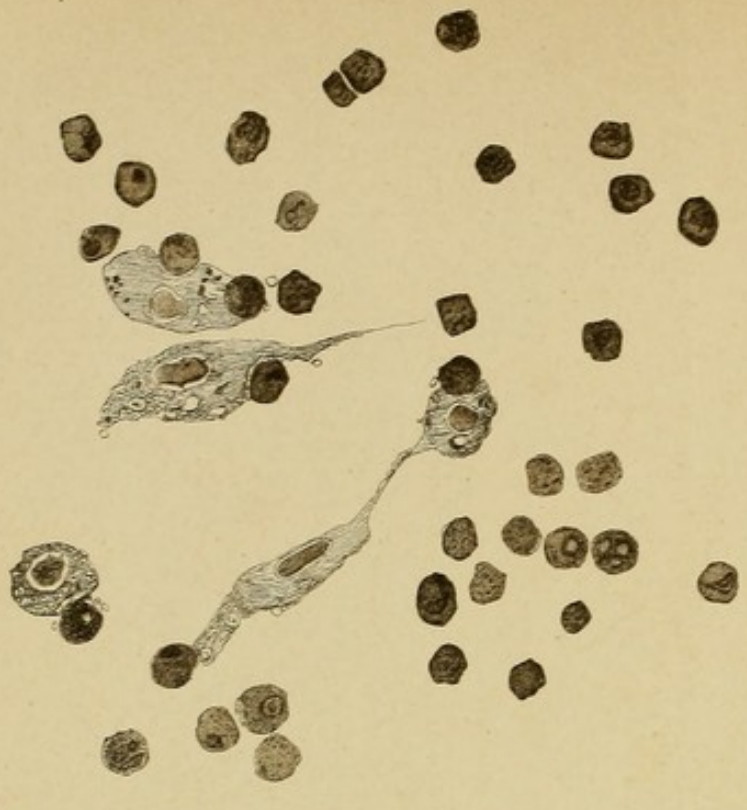
Fig. 1. Contents of experimental chamber that had remained 72 hours in the peritoneal cavity of the rabbit. Five large amoeboid plasma-cells, with altered red corpuscles and apparently dead leucocytes. Outlined with camera lucida. Apochromatic oil immersion and ocular No. 4. Zeiss. Prepared over osmic vapour.

Fig. 2. Contents of same chamber as in Fig. 1. Close to the opening of the chamber. Five plasma-cells, one of them containing a leucocyte within a large vacuole. Magnification and method of preparation as in Fig. 1.

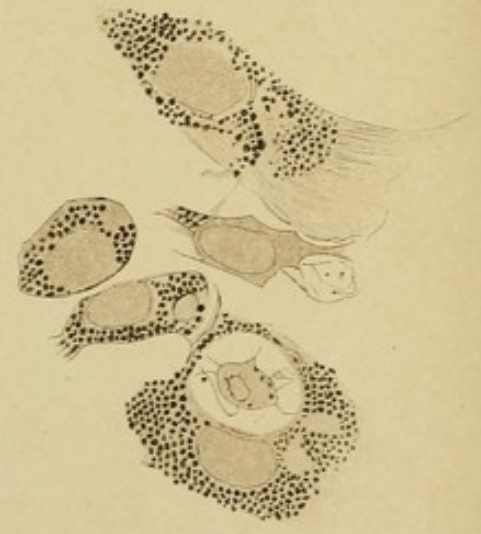
Fig. 3. Fragment of inflammatory membrane formed within a chamber placed for three days in the subcutaneous tissue (guinea-pig). Islets and groups of islets scattered through the membrane. Zeiss, Obj. A, Oc. 2. Osmic acid solution, and Ehrlich's logwood.

Fig. 4. Contents of same chamber. Two plasma-cells and two red corpuscles; the plasma-cells are indistinguishably united with fine filaments of fibrin in their surrounding, some of which are given in the figure. Osmic acid vapour. Zeiss apochr. system, oc. No. 2.

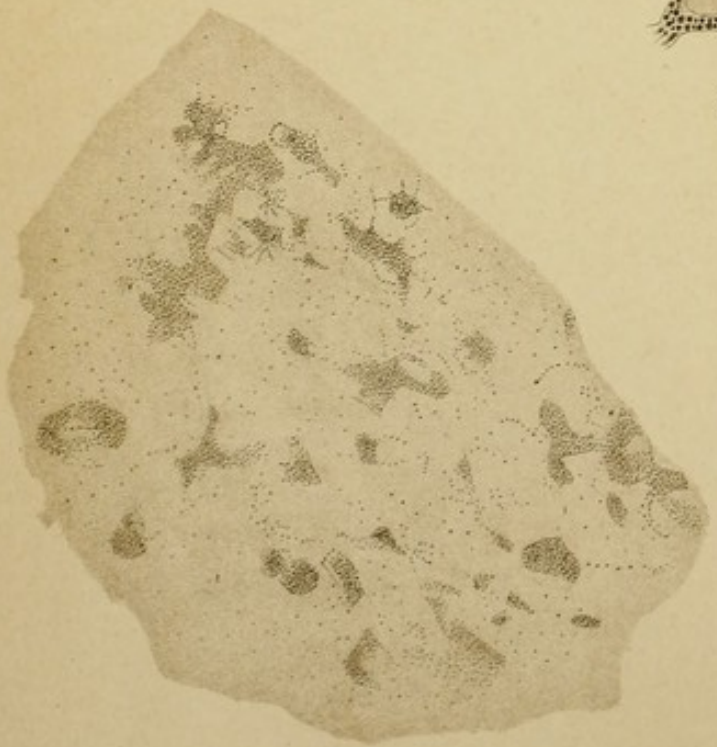
Fig. 5. Contents of a chamber for 18 hours in the peritoneal cavity (rabbit); near the centre of the chamber. Fibrin filaments, leucocytes, red corpuscles, and an ill-defined granular mass forming a nodal point in the fibrinous network—the beginning of a "cell-islet." Outlined under camera. Similar method of preparation, and similar magnification to Fig. 1.



1_(72 hours)



2_(72hours.)



3_(72hours.)

4_(72 hours.

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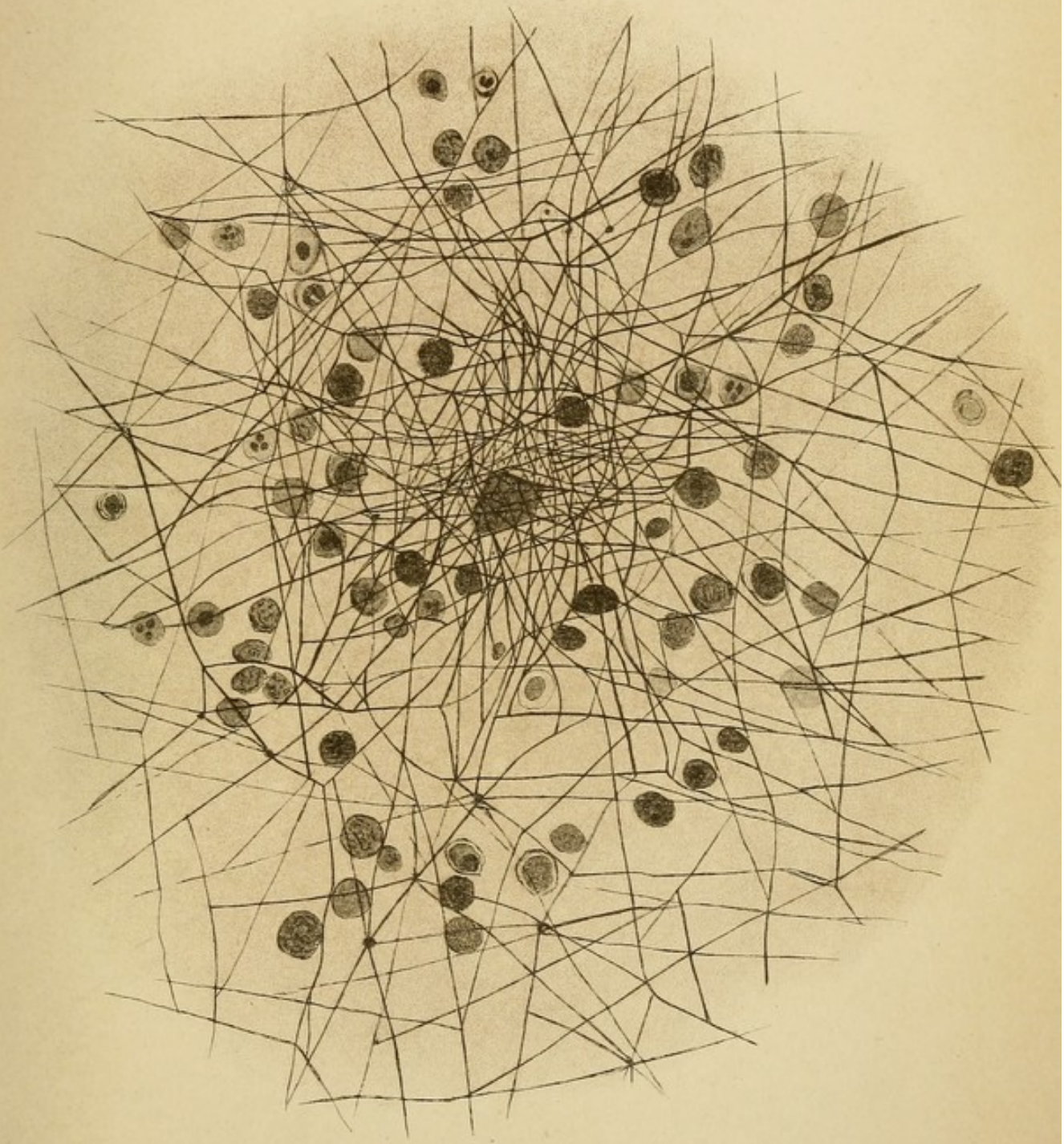

5_(18 hours.)

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part "fragmented" nuclei. The plasma-cells are far more numerous. They exist in patches and groups quite apart in many cases from the clots or the leucocytes. Many plasma-cells lie mingled with the leucocytes around the little blood-clots.

Remarks.

We have pointed out that there appears to be a definite sequence of events in the processes induced within the tissue by implantation there of the experimental chamber—processes which must according to ordinary terminology be designated as inflammatory in nature. At a definite time and in a definite order occur the immigrations respectively of leucocytes and of the daughter cells of the tissue corpuscles. The former had commenced at the end of four hours. In our experiments the fibrin within the chamber was crowded with leucocytes within eighteen hours of the time of insertion in the subcutaneous tissue of rabbit or guinea-pig. But in those eighteen hours scarcely a plasma-cell could be found to have penetrated into the chamber. On the other hand, after the lapse of seventy-two hours the nodal points, which had been previously the centres of aggregation of leucocytes, had been transformed into islets consisting chiefly of plasma-cells. This primary leucocytic invasion and the subsequent appearance of "larger cells with clear vesicular nuclei" has also been noted by Ziegler and by others. W. Hunter passed by transfusion all the blood of one rabbit into the peritoneal cavity of a second. At the end of a few hours he was able to find scarcely a white

Description of Plate V.

Fig. 1. Giant cell from chamber 72 hours in the peritoneal cavity (rabbit). Zeiss apochr. system, ocul. 5. Osmic acid vapour.

Fig. 2. Plasma-cells from same preparation which furnished Fig. 1.

Fig. 3. "Cell-islet" from inflammatory membrane obtained from chamber six days in the peritoneal cavity of the rabbit. Osmic acid solution. Zeiss oil imm. and oc. 2.

Fig. 4. Mass of blood-cells (? clot) surrounded by fibroblastic cells, and invaded by them at four places. Inflammatory membrane from chamber eight days in subcutaneous tissue. Magnification as in preceding, and prepared in similar manner.

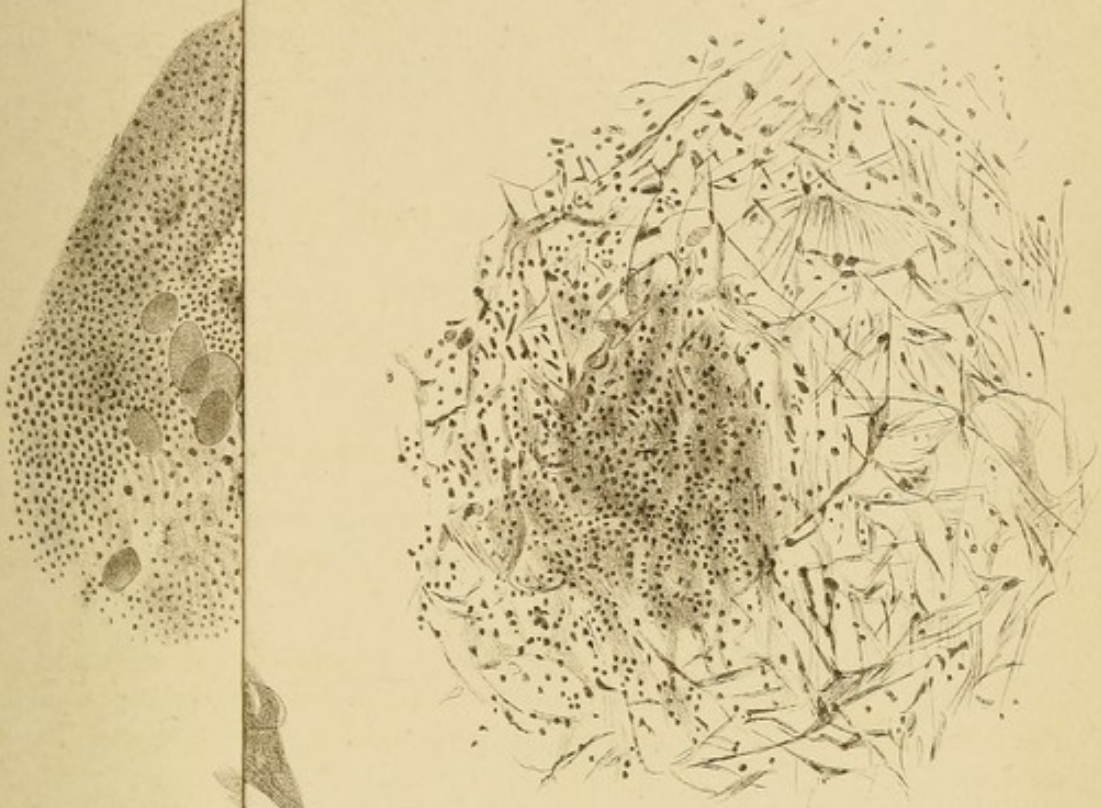
Fig. 5. "Cell-islet" from inflammatory film obtained in a chamber left eight days in the subcutaneous tissue of the guinea-pig. At the margin it is united to outlying plasma-cells. Zeiss oil, oc. 4. Osmic acid vapour.

Fig. 6. Young cicatricial tissue of anastomosing branched cells, some of which are represented under the higher magnification in Plate VI., Fig. 6. From a thrombosed artery (syphilis) near the centre of the thrombus. Zeiss A, oc. 3. Logwood. Preparation kindly lent us by Dr Sharkey.

Fig. 7. Fusiform plasma-cell (fibroblast) surrounded by a fibrillated material which forms a thread-like band of connective tissue. Zeiss oil and oc. 4. Osmic vapour. From chamber 12 days in subcutaneous tissue.

Fig. 8. Similar but larger and thicker fibrous band from 18 days' specimen. Similar preparation and magnification.

PLATE V.



6

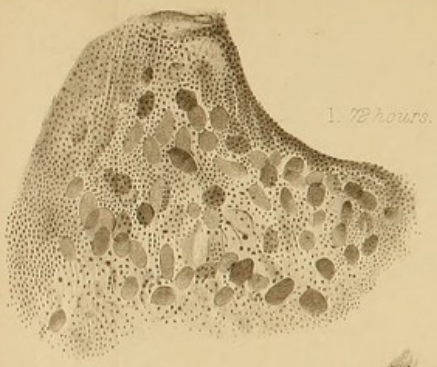
2. 72 h

7. 12 days.

3. 6 days.

LIGATION IN CONTINUITY.

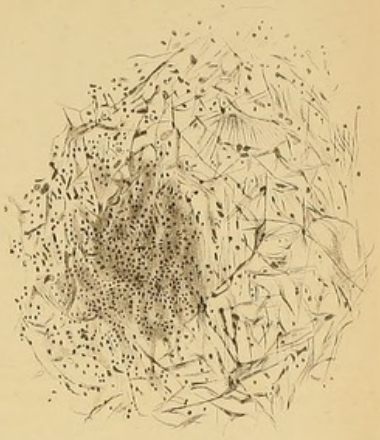
PLATE V.



1. 72 hours.

2. 72 hours.

5. 8 days.



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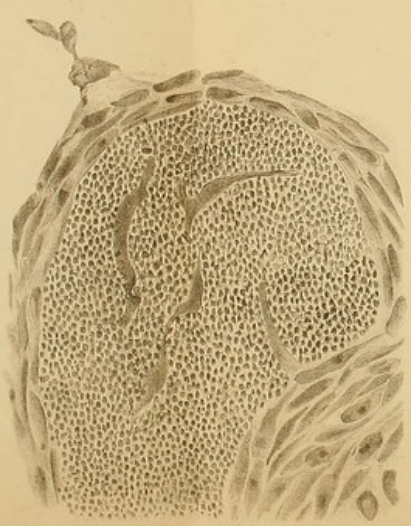

3. 6 days.

7. 12 days.

8. 18 days.

M. H. Lapidge del. ad. nat.

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corpuscle in the circulating blood; the amœboid cells had migrated into the peritoneal cavity where the foreign body—the fluid blood or the coagulum—was resting.

This observed order in the occurrence of events serves to explain "the periods of repose" that are known to the surgeon. The fluid which oozes from the surface of a wound is at first blood-tinged, but soon becomes pale, until at the end of a few hours the surface is covered with a whitish film. This film is a fibrinous network, containing within its meshes leucocytes in enormous numbers, and ever increasing as the first few hours pass by subsequent to the development of the film. "Such a "calm," says Paget, "continues from one day to eight, ten, "or more, according to the nature and extent of the "wounded part, and the general condition of the body. "The calm may be the brooding time for either good or "evil; whilst it lasts the mode of union of the wound will "in many cases be determined. Moreover in open "wounds the time at which on each tissue granulations "are produced is determined by this calm; for they begin "to be distinctly formed at its end." The share which we think the white corpuscles have in the constructive process of repair will be evident from what we mention elsewhere in the paper. "Apparently they do not hinder it." And previous to the advent of aseptic surgery it was believed by many that to leave the cut-surfaces of a wound exposed until they bore a whitish, glassy film, and not to put them into contact until then, was to give a condition favorable to union by primary adhesion.

Indeed, whatever view be adopted regarding the fibroblastic value of the leucocyte, certain other purposes

Description of Plate VI.

Fig. 1. From chamber five days in subcutaneous tissue. Plasma-cells adhering to a cotton fibre. Osmic vapour and carmine. Zeiss apochr. oil and oc. 4.

Fig. 2. From chamber eight days in subcutaneous tissue. Plasma-cells adhering to a hair, which had accidentally been allowed to get into the wound. Zeiss obj. D, oc. 2. Osmic acid solution and haematoxylin.

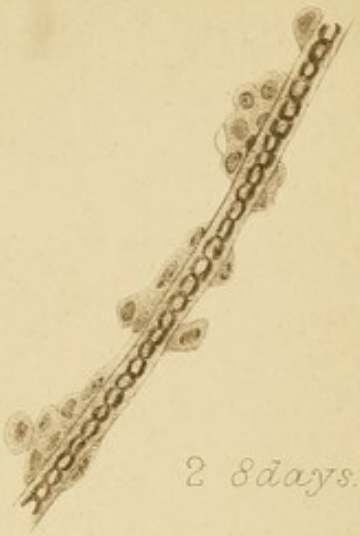
Fig. 3. The modified Ziegler chamber; the sketch shews the actual size employed.

Fig. 4. Inflammatory membrane from chamber eight days in the abdominal cavity; taken from a tenuous portion of the membrane. Four fibroblasts, in a film which is composed of an extremely irregularly arranged network of filaments resembling fine fibrin threads. The processes from the cell-body are continuous apparently with the fibrils of the matrix. Osmic acid vapour and haematoxylin. Zeiss. apochr. oil imm. and oc. 4. Outlined with camera lucida.

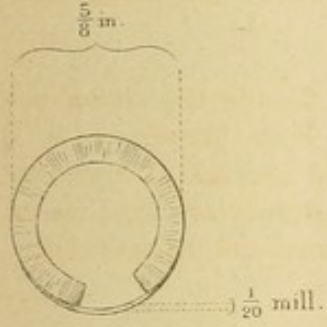
Fig. 5. Portion of the chamber seen edgewise, shewing the opening between the cover-glasses. Enlarged 12 times.

Fig. 6. Stellate fibroblasts and two leucocytes from same preparation as in Plate V., Fig. 6, more highly magnified. Zeiss apochr. oil and oc. 4. Outlined with camera lucida.

1. 5 days



2. 8 days.



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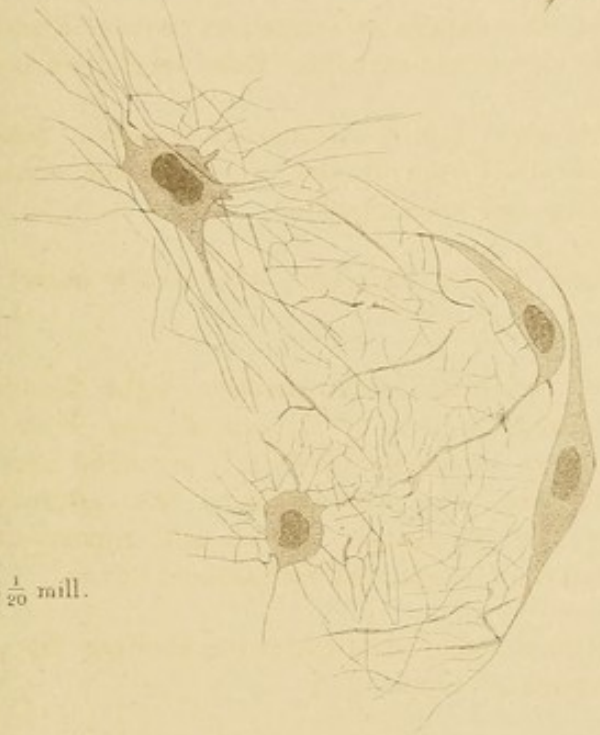


4. 8 days.

5

6

which it may subserve in the process of repair were in the experiments extremely obvious. It was the pioneer of all the wandering swarm of cells that visited the intruding occupant of the tissue. Whatever causes, intrinsic or extraneous, guided its early voyaging, the route it traversed and the position it assumed seemed to determine almost absolutely the course of aftercoming plasma-cells that appeared in great measure to be simply followers along the track thus broken for them. Where the intruding body was of penetrable nature, as in the case of blood-coagulum, these leucocytes entered it in the van of a destroying army, that in turn attacked it from channels that leucocytes had prepared. By leucocytes the mass to be absorbed was in part previously divided up and made to offer a greater surface for absorption by plasma-cells. Where larger masses of clot are concerned cracks and fissures occur from chemical causes, which in the same way allow of the entrance of the plasma-cells among whose functions in the clot mass are absorption and substitution. The filaments of fibrin when they were present appeared to direct to a certain extent the path travelled by the cells. Certainly to group themselves about the granular nodal points of the fibrinous network was quite characteristic of the distribution of the leucocytic swarm, and this directly influenced the formation of islets in the cellular membrane, the islanding being the direct outcome of the original grouping.

By plasma-cells, as before stated, is meant all kinds of connective tissue corpuscles and their progeny: they are included by Metschnikoff in his group of phagocytes, which he divides into two classes, the makrophagen and the

mikrophagen. The latter he affirms are for the most part leucocytes. Wyssokowitsch came to the conclusion that bacteria were attacked most vigorously by the tissue corpuscles, and Carl Hess has figured in *Virchow's Archiv* gland cells destroying bacilli—the leucocytes having been simply carriers to the glands. In the chambers when bacteria happened to be present they were taken up by the plasma-corpuscles.

The leucocytes served also as a pabulum for the active plasma-cells. Just as, in the extremely interesting observations given by Miss Greenwood; little monads, Euglenæ and Algæ coexisting in the same water with Amœba proteus were by it ingested, so leucocytes become the prey of the plasma-cell, and are by it included and ingested. And if the growth and proliferation of the plasma-cells be of importance in the process of repair, what circumstance more propitious than the presence in abundance of nutriment so delicately adapted and so highly organized as the substance of the leucocytic cell? Of Amœba and Actinosphærium it was remarked that the food most suitable to these forms is unshielded non-coagulated proteid matter. A low degree of vitality, a diminished activity of its protoplasm, renders an organism easier prey, more readily captured and more readily absorbed. The plasma-cell may in some respects be taken as a hothouse variety of amœba; it finds its unshielded non-coagulated proteid in the dead or dying leucocyte. It will be remembered that within the chambers the leucocytes revealed striking signs of lowered vitality.

Not that the number of instances in which we could detect an actually included or a partially ingested leu-

cocyte would, we think, account for the large disappearance of them that does actually occur. Is it not probable that the plasma-cell can exert digestive action upon material which it does not incept? Suppose a proteolytic ferment secreted by the plasma-cell, and leucocytes that are dead or dying as in the above experiments; a gradual solution of their substance in the tissue plasma will occur, yielding to it an abundance of rich food for other cells that are in a thriving condition.

Passing in review the chief points observed in regard to plasma-cells, it became clear enough that in the study of their origin and development lies the best key to the problems of the formation of tissue of repair. We found them traceable up from forms of an amoeboid kind, different in many ways from the amoeboid cell-forms of blood and lymph, through individual types of almost endless diversity of figure with the utmost variety of combination and interdependence, onward finally to the fixed corpuscle of fusiform or of stellate shape imbedded in fibrillated material.

As to giant cells, often it was obvious that the large cell had resulted from a fusion more or less complete of the bodies of several smaller cells, the nuclei of which remained distributed regularly through the substance of the aggregate. In other instances a massing of the nuclei of the giant cell about one point appeared to denote a mode of origin from a single cell that had grown and undergone nuclear multiplication without actual separance of the daughter cells from the parent as they had been produced.

Again, by the union of cell with cell, by means of long

pseudopodium-like processes, it was sometimes found that a whole field under the lens was occupied by the net-like ramifications of one huge multi-nucleated cell—better described perhaps as an unbroken sheet of anastomosing cells. The characters of the giant cells in the implanted Ziegler-chambers resembled in this particular those of such giant cells as occur in marrow, growing bone, the splenic pulp, myeloid sarcoma, and in granulation tissue. In no cases did the arrangement of the nuclei in them bear resemblance to the ring-like or other regular disposition often seen in the giant cells of tubercle.

Upon the position of the giant cells depends partially the arrangement of the fibrillated tissue which is ultimately produced. The run of the bundles of fibrillæ is often from and between giant cells. The cells range themselves previous to fibrillation in lines spreading for some distance from the giant cells; in fact in many ways the resemblance of giant cells to cell-islets is a close one. Just as in some cases, if not in all, the so-called giant cell is really but a congeries of smaller coherent cells, attracted to one and the same spot for the purpose of participation in a common prey, so is it with the cell-islets also. The groups of leucocytes from which the cell-islets arise appear to be originally formed under the common attraction which is offered to these cells by the albuminous *débris* present at the central nodes of the fibrinous network. Later, the leucocytes themselves becoming from some cause or another effete and of low vitality, exert a similar attraction upon the wandering plasma-cells, and afford to them a rich and easy quarry. By this arrival of fresh cells the islet is increased in bulk. The more centrally

situated individuals feed upon the leucocytes they have surrounded, and the latter rapidly merge to an amorphous kernel for the entire mass.

The outlying cells become disposed along definite lines, and as it were sketch in in its main outlines the general plan which the adult arrangement of the new fibrous tissue will display.

The cell-islets are the centres of most active growth and proliferation in the young cellular tissue. They contain the stores of nutriment that are gradually dissolved and digested. They may contain also innutrient matters, and matters such as are not only innutrient but incapable of solution. At first the shape of those cells which are immediately next to the kernel of nutritious matter in the islet is irregular, and suggests amœboid properties in the cell; later the cell becomes almost regularly fusiform, and is applied by its side to the material which gradually disappears. The material comes to be encircled by chains of fusiform cells set concentrically around it. It becomes encapsuled in the same way as is the ligature placed around an artery by the surgeon, or as is any foreign body placed within a wound that heals around it. The fusiform fibroblasts slowly exert the same solvent action upon the imprisoned material as did their amoeboid ancestors. No doubt the more easily affected portions of the material are the first to go into solution and disappear, leaving a constantly less amenable residue and a less nutrient one; and perhaps it is in accordance with the decreasing supply of food from this source that the cells in contact with it undergo gradual change and lose their pristine elasticity of form. They

assume the spindle-shape, and a fibrillated intercellular cement substance comes into existence between them. We have already seen reason to think that this "matrix" is a secretion from the cell. Prominent among the conditions under which the young fibroblasts begin to form it is, it would seem, a diminution in the amount of pabulum at hand to support growth. Much as amœba under adverse conditions assumes an encysted form, so where food is scanty do the inherited tendencies of the fibroblast lead it into states of quietude and encystment. The less nutritious, the more inert the foreign body which the plasma-cells surround, the sooner do they become fixed cells, the earlier do they elongate, and make around themselves the bed of fibrillated matter, which commits them to immutability of form. In the same specimen in which plasma-cells preying upon remnants of blood-clot were still actively amœboid, it often happened that around innutritious matter as hairs, and cotton fibres, the cells were already perfectly developed into young fibrous tissue.

When embedded in the fibrillated secreted substance all digestive and absorptive activities within the cell do not cease. Encapsulation does not arrest absorption. This question is further discussed in the chapter on the Conduct and Fate of the Ligature.

Unfortunately it was only when the present experiments had been concluded, and the present work very nearly so, that we were able to obtain copies of Professor Ziegler's monographs from the Würzburg Institute. We had been obliged to satisfy ourselves with the results of his work in abstracts of the original papers. As a matter of fact our work has not been a repetition of his quite to the extent

we had imagined. A great part of the observations deal with periods which his do not touch or only slightly so. His first communication is based upon observations on chambers implanted in sixteen dogs at thirty-six different intervals. But of these only five, upon four individual animals, refer to the first two weeks after implantation, and he records no observations prior to the seventh day. Of our observations with rabbits the major part refer to the first two weeks after implantation, and our earliest observations were made only four hours after implantation. We imagine too, judging from the beautiful illustrations to the original papers, that the cell-masses that we have so frequently referred to as cell-islets are included by him among the giant cells. It must be remembered also, that his experiments date prior to the acceptance of antiseptic surgery, and eleven times he records pus, either in the implanted chambers or in the wound. In no case, as we have already said, did we ever find the slightest trace of pus.

It will have been seen that in most points our observations entirely confirm the original observations made by Ziegler. One particular there is however, and that one of fundamental importance, in which we are in disaccord with the descriptions furnished in his paper. As far as we observed, there are in the tissue plasma of a part subjected to irritation such as that described in the experiments two kinds of cell. On the one hand there are present leucocytes indistinguishable from and probably identical with the colorless corpuscles of the blood; on the other hand are plasma corpuscles, cell-elements proper to the connective tissue of the part offended. The

cell that plays, as we incline to believe, the only active part in the construction of new tissue that goes on, is the plasma-cell, a corpuscle absolutely distinct from the colorless corpuscle of the blood. The cover-glass preparations lead us to believe that these cells in small number exist free in the tissue plasma even under normal circumstances. Where the connective tissue corpuscles are proliferating, as for instance within an inflamed area, there these free tissue-cells are enormously more numerous. In the experiments, out of them arose the permanent membranes, to be designated inflammatory if the ordinary unsatisfactory use of the term be sufficient, which spread themselves over and inside the Ziegler's chambers when lying in a subcutaneous space or in the peritoneal sac—membranes composed at first of cells entirely similar to the corpuscles of the normal tissue plasma. Colorless blood-cells doubtless wandered in the surrounding of the chamber, and doubtless entered in plenty the space within it. But these leucocytes had no permanence of possession. The fibroblast of the new tissue was not of leucocytic origin. Our observations yield no support to Cohnheim's view of the genesis of cicatricial tissue from leucocytes.

This indeed is only what is to be expected when we remember that it is probable that the whole life of a leucocyte is at most three weeks, while a plasma corpuscle may live for an indefinite period: and it is not to be supposed that a cell having such an ephemeral existence as a leucocyte could effect much in the slow work of construction.

Another fact in favour of the view that leucocytes have no power of themselves to build new tissue is that in

the clot in aneurism, where they exist in abundance and have, as Professor Hamilton forcibly points out, every opportunity for carrying out constructive changes, nothing of the kind occurs: no new tissue being formed even after the lapse of years, unless the clot becomes adherent to the wall of the aneurism and thus allows the entrance of connective tissue cells.

The analogy with the healing process in the vegetable kingdom points in the same direction: here are no migratory cells or any representatives of the leucocytes; all repair therefore is carried out by the fixed cells and their descendants, as has been clearly shewn by Shattock in communications to the Linnæan Society.

When pus is formed in an inflamed focus many of the migrated colorless corpuscles of the blood become, as is well known, pus-corpuscles. It does not appear strange that where pus is not produced those of the swarm of leucocytes which do not drain off by lymphatic channels from the tissue they have temporarily invaded but remain behind should not thrive within it. Many circumstances might, we conceive, render their sojourn perilous. The high carbonic acid tension, the comparatively stagnant character of the fluid, the presence of, to them, unwonted chemical bodies, and of others in unwonted percentages —these are instances of conditions which might, we conceive, constitute an environment of disadvantage. And that the migrated leucocytes should rapidly be not merely acclimatized in the new locality, but should actually become fixed elements of the part, and generate the cells of a fibrous tissue, is to our minds improbable. The cells of fibrous tissue and the colorless corpuscles of the blood are

both of mesoblastic origin, but we have no evidence that they are more nearly related one to another than are the fibres of a striated muscle to the endothelial cells of an artery. No one advances the view that of these latter one can by any means be made to reproduce the other. Even in tumours with their apparent departure from the normal type of growth the principle of heredity is in reality religiously observed. "The secondary growths in car-"cinoma are identical with the primary, for it is the "epithelial element which is infected, and it is this element "which determines in the normal process of development "the general anatomy of the parts around, be they gland-"ular or otherwise. So a columnar-celled carcinoma of "the rectum produces in its metastatic growths intestinal "crypts in the liver; a thyroid cancer produces in its "secondary tumours thyroid tissue in the bones; an "osteoid sarcoma shows in its secondary manifestations "osteoid tissue; and it might even be conjectured that if "the epithelium over the papilla of a hair received the "carcinomatous infection hair-like structures would be "found in the primary and secondary tumours." In the production of scar-tissue it seems to us of transcendent significance that such tissue is characterised by the possession of cells of which each tends to secrete a collaginous capsule for itself, so that around the cells a more or less solid and fibrillated intercellular matrix comes to be characteristic. Cells with a similar tendency characterise broadly the connective tissues wherever found. It is in accordance to laws of natural descent for the cells of connective tissue, when thrown into renewed and extraordinary genetic activity in what is termed plastic

inflammation, to produce a progeny of cells possessed of the same tendencies as themselves. And among all these tendencies, which one is more unfailingly repeated in them than to spin a semi-solid fibrillated collaginous capsule, in short, to build up fibrous tissue? But the cells of the blood nowhere show signs of any such propensity. The colorless corpuscle of the blood is conspicuously endowed with a character apparently opposed to, even incompatible with, the formation of a semi-solid circumcellular test—the whole story of its normal life, so far as we know that, is associated with a ceaseless change of form.

The invasion of leucocytes and plasma corpuscles occurs in the healing of all wounds and has therefore much interest for the surgeon. Of whatever other use the extravasated white blood-cells may be, the number of them invading the tissues in the healing of subcutaneous wounds, and in those that unite by the first intention, seems to some extent adapted to the nutritive requirements of the plasma-corpuscles whose food they constitute, and which alone spin the connective tissue that repairs the injury.

The conclusion then is that the scar-tissue which occludes the artery is formed, not from the leucocytes of the blood, but from the plasma-cells of the arterial wall. Later it will be shown that these cells will do their work perfectly if the vessel be simply occluded, and that it is by no means necessary that any injury be inflicted on the arterial wall.

Note 1. The preparations and original drawings referred to in this Chapter were exhibited at the Royal College of Surgeons in

March, 1889, in illustration of part of the Erasmus Wilson Lectures on "Ligation in Continuity."

Note 2. Since the above was written the view that the work of construction is effected by the migrated leucocytes has been generally abandoned. At the International Medical Congress held in Berlin in August, 1890, it was stated by Ziegler that the connective tissue corpuscles are the sole active agents in the formation of the scar-tissue, and this view was unanimously accepted by the distinguished pathologists present.

CHAPTER VII.

CONDUCT AND FATE OF THE CORPUSCLES (CONTINUED).

Object of further Experiments. Ligatured Rabbits' Carotids. Histological Methods. Observations and Conclusions.

Having studied the parts played by the plasma-corpuscles and leucocytes in embryo scar-films of various ages and of great tenuity, and endeavoured to obtain a clear idea of the conduct and fate of these cells in the formation of scar-tissue, the next step is to observe their conduct and fate in the wall and coagulum of a ligated artery.

For this purpose the carotids of rabbits were selected, partly because the arteries being so small all the cells of the wall and clot could be readily brought in a living state under the influence of osmic acid, so that the individual corpuscles in the specimens could be compared and identified with those previously observed in the Ziegler

Description of Plates VII. and VIII.

All the drawings are made from transverse sections of the common carotid arteries of the rabbit.

Fig. 1 is a normal artery; the others have been ligatured, and the date of ligation is marked against each artery.

The drawings were made with an apochromatic 1-12th oil immersion lens by Ziess, with oculars 4 and 8, giving a multiplication of about 600 diameters.

Description of Plate VII.

Fig. 1. Part of a Transverse Section of a Normal Common Carotid of Rabbit.

The innermost of the elastic lamina is well seen, and serves as a landmark in comparing the different sections: internal to it (that is, in the drawing below) is seen the single layer of flattened cells which constitute the whole of the active intima in this artery.

Fig. 2. Carotid 24 hours after ligation, section close to ligature.

Internal to membrane of Henle is seen a multiplication of the endothelial cells, so that this layer is now two or three cells deep: internal to this are seen a few of the red corpuscles of the coagulum: one of the endothelial cells has entered the coagulum.

Fig. 3. Carotid 5 days after ligation.

The innermost elastic lamina is well marked, and internal to it is seen the endothelial layer, now several cells deep. In this layer and in the middle coat and also in the clot are large plasma-cells; these are daughter cells, some from the endothelium and some from the connective tissue corpuscles of the middle and outer coats. The nuclei of these cells are large, oval, vesicular, faintly staining and granular. The amount of protoplasm round the nucleus varies in amount. It will be seen in this and also in the next drawing (Fig. 4) that the outlines of the red corpuscles in the clot are well marked in its more central portion, but that at its surface they are less distinct, and instead of being round have become oval. The want of definition is probably due to the action of some ferment, and the change of shape possibly to pressure.

Fig. 4. Carotid 6 days after ligation.

The upper drawing is at the surface of the clot and includes the membrane of Henle, and the lower drawing is from nearer the centre of the clot.

In the upper drawing the coagulum has lost all trace of outline of the individual red corpuscles. Internal to the elastic lamina are seen the endothelial cells, some of which are projecting into the clot, while others have entered deeply into it in a long string two or three cells thick.

The lower drawing is from a more central portion of the clot. Here the outlines of the red corpuscles are well seen. In the centre is an islet of cells. The large cells in the centre are plasma-cells: they are known from leucocytes by their larger size, by their oval nuclei, by the vesicular appearance of their nuclei, and by the faint staining of both their protoplasm and nuclei. The two cells to the left are leucocytes; they are smaller, round, and deeply stained.

Figs. 5 and 6 described overleaf.

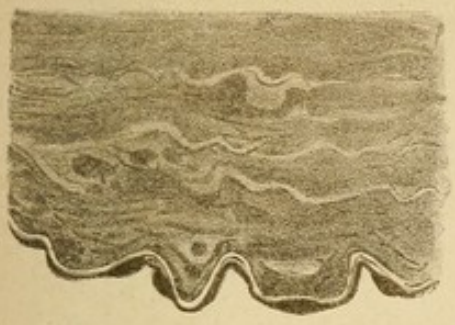


Fig. 1 *Normal.*

Fig. 2. *1 day.*

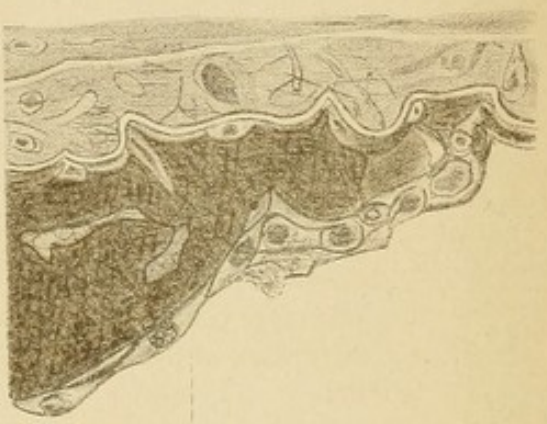


Fig. 4. *6 days.*

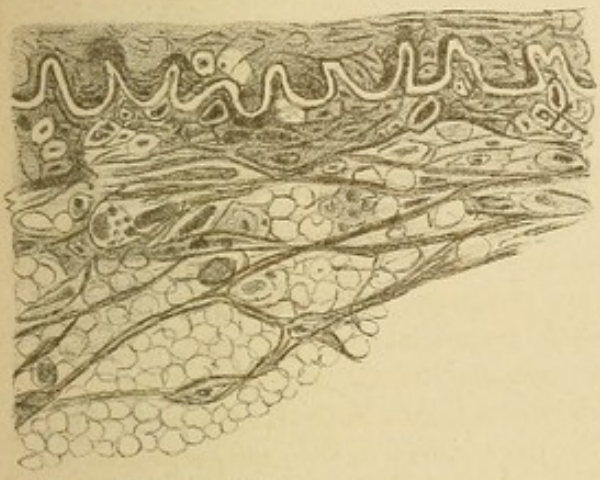


Fig. 5. *7 days.*

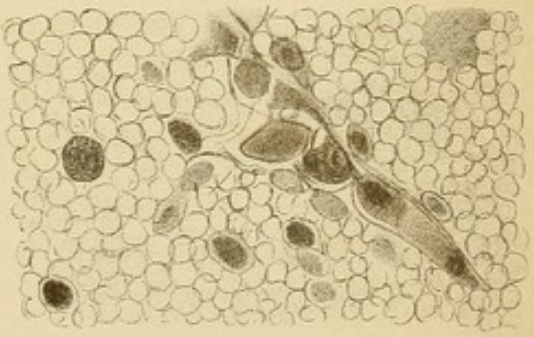


Fig. 3. *5 days.*

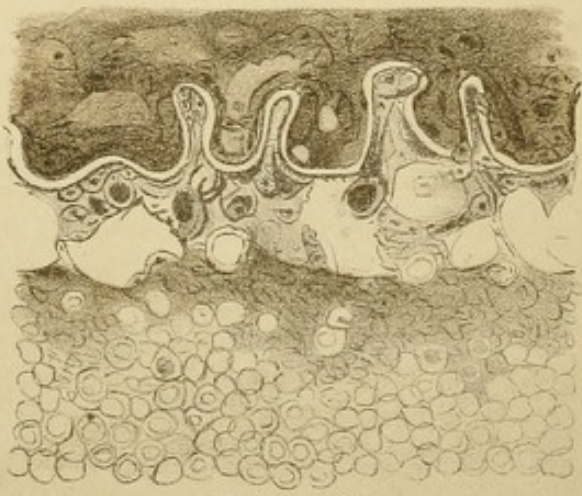


Fig. 6 *14 days.*

chamber films; partly because complete transverse sections can readily be made of these small vessels; and partly because in them the intima consists, as Baumgarten pointed out, of a single layer of endothelial cells resting directly on the elastic lamina, and the process can therefore be viewed in its simplest form.

The common carotids of several rabbits were ligatured with fine silk or thread, with antiseptic precautions, and the specimens secured at dates varying from one to twenty-nine days. Before removal the vessels were secured at some distance above and below the seat of ligature. In order to obtain the cells in as natural a condition as possible the specimens were placed at once in Flemming's solution. While in this, they were kept, as is usual, in the dark: after twenty-four or forty-eight hours they were transferred to dilute alcohol, and finally, to complete the dehydration, to absolute alcohol. In order that the arteries should remain straight, and not curl up during hardening, they were suspended in test-tubes with a small weight attached to their lower end.

Finally, the arteries were transferred to turpentine, embedded in paraffin, and cut with a Cambridge rocking microtome, which delivers the sections adherent to one another in ribbons. In this manner specimens were obtained from rabbits' carotids at nine different dates, namely, twenty-four hours, and three, five, six, seven, fourteen, fifteen, twenty-two, and twenty-nine days after ligation.

The result of the examination of the sections is seen in the accompanying drawings, which shew the process better than any verbal description.

Fig. 5. Carotid 7 days after ligation.

Internal to the innermost elastic lamina the endothelium is several layers deep. The young plasma-cells which have advanced furthest into the clot have thrown out processes which connect them one with another and divide up the clot into spindle-shaped areas. In those of these districts which are nearest to the artery the process of absorption of the clot is nearly completed, the spaces being occupied by plasma-cells.

Fig. 6. Carotid 14 days after ligation.

The appearances are an advanced stage of those seen in Fig. 5. In the membrane of Henle are openings through which daughter cells of the connective tissue corpuscles of the middle coat appear to pass. Internal to the elastic lamina the endothelial cells are multiplied, and the spaces referred to in the text are seen. At its outer part the clot is nearly devoid of all structure, but more deeply the outlines of the red corpuscles are well seen.

Description of Plate VIII.

Fig. 1. Carotid 15 days after ligation.

Internal to the elastic lamina the endothelial cells are seen proliferating. Towards the centre of the vessel the appearances are the same as those seen in Plate VII. Fig. 5 (7 days), with the exception that the areas of the clot are less defined in consequence of the process being more advanced; between this and the proliferated endothelium immediately in contact with the membrane of Henle the plasma-corpuscles have extended themselves into long narrow spindle-shaped cells. This is a stage preliminary to the formation of perfect fibrils.

In the text, further remarks are made about this specimen, with regard to the reopening of the lumen of the artery and the attachment of the clot to only one part of the wall of the vessel.

Fig. 2. Carotid 22 days after ligation.

The artery wall is to the right, the clot to the left. The fenestra in the membrane of Henle are well seen, and cells can be observed passing through them. The multiplying endothelium forms a thick layer with some cells projecting into the clot. As in Plate VII. Figures 3 and 6, clear spaces are seen in the periphery of the clot; and here too is a general blurring of the structure of the coagulum. It must be borne in mind that although the ligation had taken place 22 days previously it does not follow that this particular portion of clot is of that date.

Fig. 3. Carotid 29 days after ligation.

In this drawing the wall of the artery is at the lower part, the dark wavy line being the membrane of Henle. Internal to (that is, in the drawing above) this is a mass of young connective tissue formed of fusiform cells and fibres lying in various directions. The peripheral portion of this young connective tissue is formed from the thickened intima which has become blended with the new tissue which has taken the place of the clot, so that it is now no longer possible to distinguish the line of union between them. In the new tissue are seen capillaries, one in the centre of the figure having fairly well-developed walls.

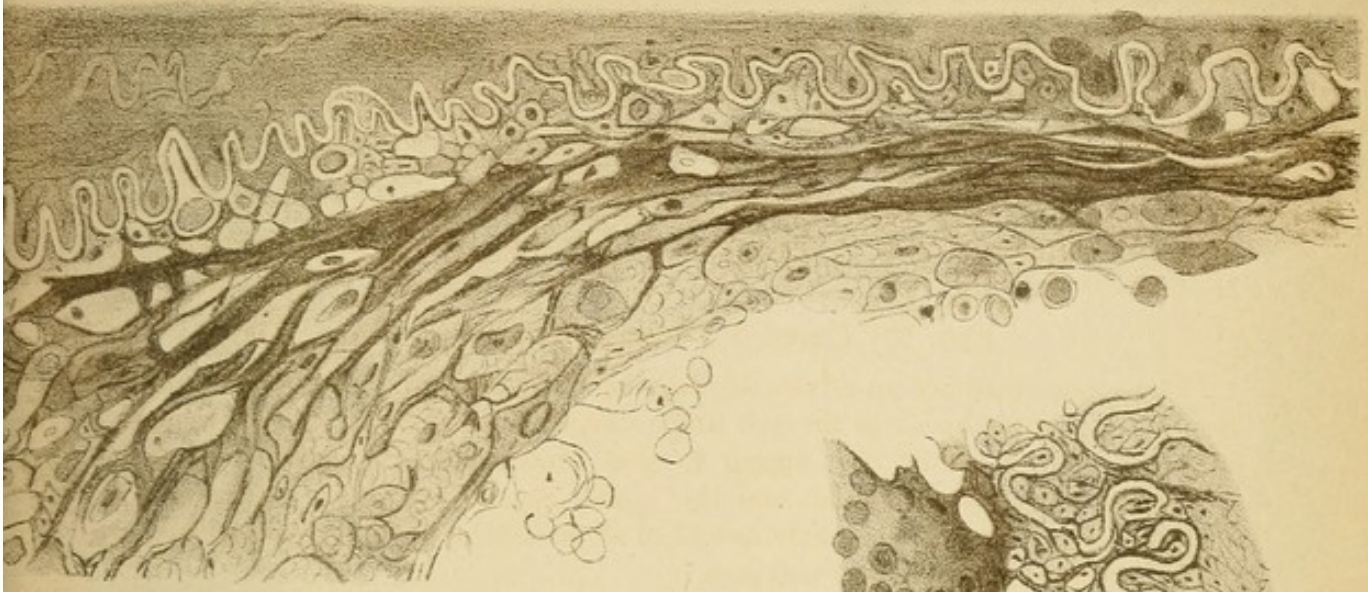


Fig. 1. *15 days.*

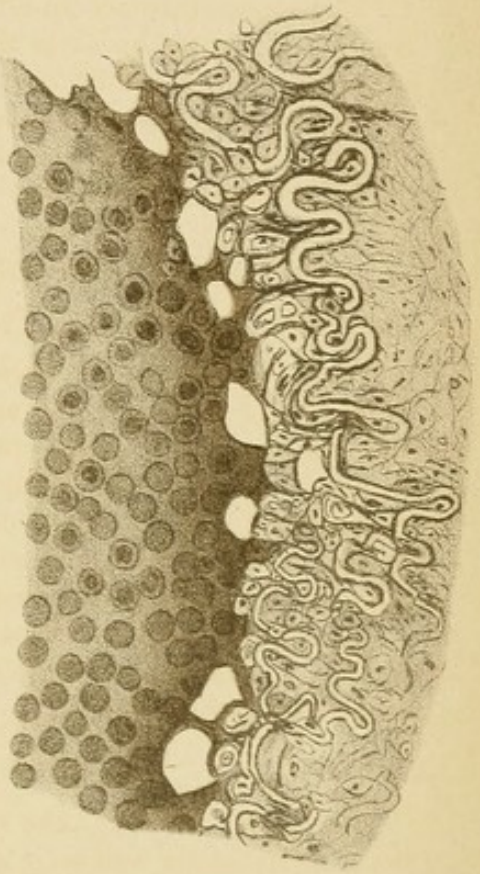


Fig. 2. *22 days.*

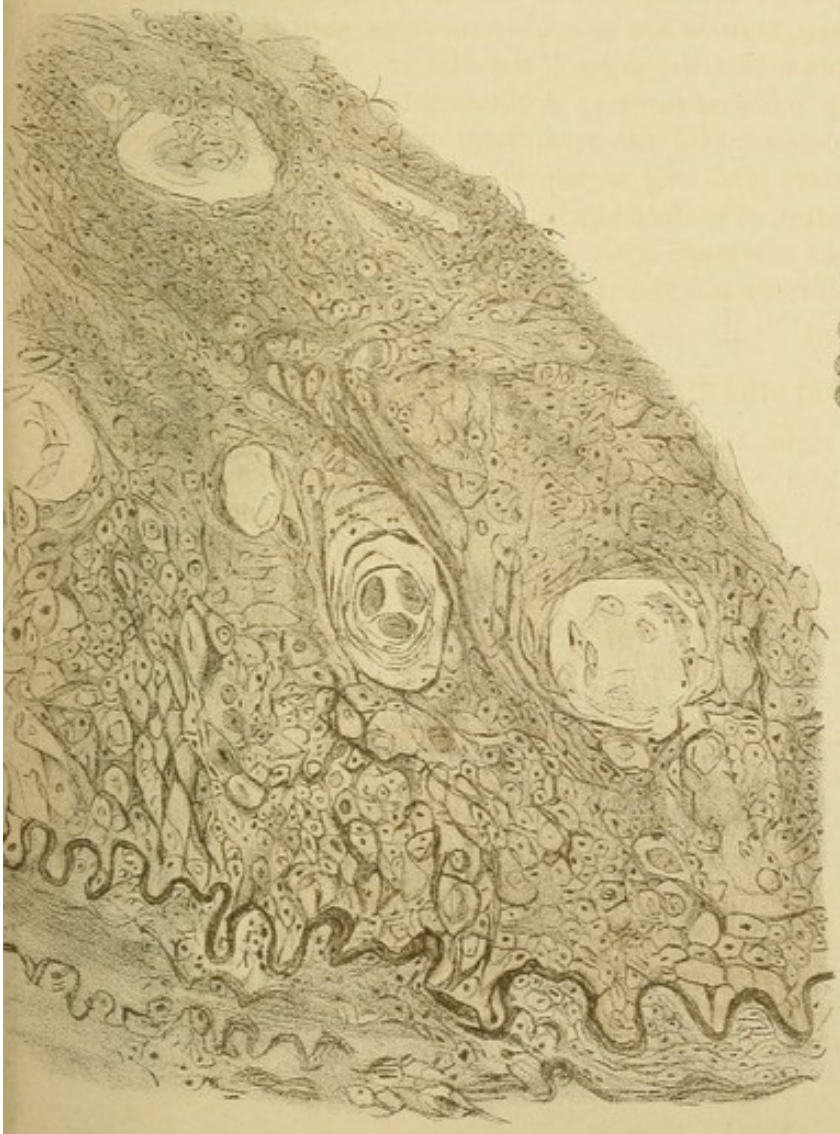


Fig. 3. *29 days.*

The carotid of a rabbit consists of an outer coat, a middle coat containing several elastic laminæ, and an inner coat. The thickest and most continuous of the elastic films in the wall of the rabbit carotid is the membrane of Henle: it is very conspicuous in all the sections, and forms a valuable landmark, being, as it is, the outer layer of the inner coat: internal to it is a single layer of flattened cells with large oval nuclei. (Plate VII. Fig. 1.)

One of the first results of ligation is to cause a multiplication of the endothelial cells, so that even in twenty-four hours the layer becomes two or three cells deep. (Plate VII. Fig. 2.)

Other changes soon occur; the connective tissue corpuscles in the middle coat become active and subdivide, and the daughter cells of these corpuscles pass through the openings in the membrane of Henle and enter the clot; leucocytes also take the same course, but they do not seem to be numerous, which for our purpose is an advantage, since the plasma-corpuscles are thus not obscured, and can be seen with great distinctness.

Besides undergoing this invasion, other changes occur in the clot: at its centre the red corpuscles retain, for a considerable time, their appearance and shape, but at the periphery near the arterial wall they undergo two changes; firstly, they lose their sharpness of outline, and break down into a granular mass: this is probably due to a peptonising fluid, excreted by the neighbouring plasma-cells; secondly, they, instead of remaining round, become oval, or fusiform, and possibly swollen. As the long axis of the oval is parallel to the arterial wall, it is possible that the change of shape is due to pressure from the rapid multiplication

of the endothelium. This pressure could hardly be effective unless the endothelial development is extremely rapid, and, as it were, overtakes the natural contraction and shrinkage of the coagulum. The pressure effects can easily be understood if a firm adhesion takes place and a small portion of clot ("clot district") is cut off from the rest of the coagulum by a circlet of plasma-corpuscles; or, if the clot does not contract, as in the experiment of Lister, in which he showed that horses' blood, drawn under perfectly sterile conditions in glass vessels, clots, but does not contract—a phenomenon which, at present, is utterly inexplicable. The grounds for the statement that a proteolytic ferment is excreted from the surface of the invading cells will be given in the chapter on the Conduct and Fate of the Clot.

The difference between the central and peripheral clot is seen in Plate VII. Fig. 4, and the oval shape of the corpuscles is shewn in the left-hand part of Plate VII. Fig. 3.

Further, there are to be seen in the periphery of the clot small oval spaces, best seen in Plate VII. Figs. 3 and 6, and Plate VIII. Fig. 2. The possibility of these being caused by shrinkage from the action of the reagents must be borne in mind, but although some of them may be due to this, we are inclined to think that the majority of these spaces are not to be so explained, partly because of their regularity in position and shape, partly because they often correspond in extent to the border of one or more plasma-corpuscles, indicating possibly their origin from the action of material produced by these cells, and partly because on carefully focussing the remains of a cell, possibly its most

indigestible portion, can be made out in many of the spaces, which, in fact, seem not to be empty.

In these changes, just described, may possibly be found one of the reasons why the clot so readily becomes detached from the vessel-wall.

All these changes are established by the fifth or sixth day: but the plasma-cells continue to invade the clot, often collecting into groups in it, forming islets at the nodes of fibrin, as in the Ziegler chambers. From the islets, processes of elongated cells are thrown out, which, uniting with similar processes from other islets, divide the clot into small, regular areæ, or "clot districts." The subdivision of the foreign body is to aid in its absorption and substitution.

In Plate VIII. Fig. 1, the fibrous tissue formation of the thickened intima and clot is permanent only on one side of the vessel, the remainder of the lumen having been opened again to the blood-stream. The section was cut some distance from the ligature. The original clot did not here form a permanent adhesion to the entire circumference of the artery wall, and the blood flowed again through the diminished artery at the site of the section. It will be observed that the cell-covering of the fibrous patch which encroaches on the calibre of the artery is gradually approaching the smooth character of an endothelial surface.

Lastly, about the fourth week capillaries appear in the clot, and ultimately the vessel becomes converted into a mere cord of fibrous tissue. (Plate VIII. Fig. 3.)

These observations confirm the conclusions derived

from the Ziegler chamber experiments. It is clear that, although two kinds of cells invade the tissues about the seat of ligature, the whole of the work of construction is carried out by the tissue corpuscles and their descendants.

CHAPTER VIII.

CONDUCT AND FATE OF THE CLOT.

Methods of inquiry. Formation of Clot. Conduct of Blood arrested between Two Ligatures. Living Test-tube. Asepticity and Intravascular Coagulation after Ligature. Extent and Date of the Clot. Ampulla of Bryant. Functions of the Clot. Collateral Branches. Strata and Fissures of the Clot. Absorption and Substitution of Clot. Vascularization of Clot. Extent of Artery obliterated. Re-establishment of Circulation. Diaphragms.

Methods of inquiry.

The knowledge of the structure and fate of blood coagulated either within its own proper channels or external to them is of comparatively recent date. Petit, writing in 1735, states that clot is essential for the arrest of hemorrhage and the permanent occlusion of vessels.

The latter statement has been controverted by many writers since his day. Petit made the first research into the intimate nature of clot; but the method he adopted for preparing the clot for examination was not likely to lead to satisfactory results. It consisted in placing the clot in water for two months, the water being changed two or three times a day: at the end of the two months brandy was substituted for water; and what remained of the clot after its treatment by water was left undisturbed in the brandy for three years. The plan of Petit is interesting historically, and contrasts with the modern methods of histology at our disposal. For the study of the conduct and fate of blood shed within the living body, we have:—

(1) The warm stage and osmic acid preparations of the Ziegler chamber films in which small blood-clots were often present.

(2) The osmic acid preparations of ligatured rabbit carotids.

(3) The clot in aneurisms, and in other situations outside the vessels, both in museum specimens and microscopic sections variously prepared.

(4) Each great artery, experimentally ligated in sheep, asses or horses (see Abstract of Experimental Ligations) was after removal from the body of the animal hardened in Müller's fluid and spirit. It was then divided longitudinally. One-half was immersed in a saturated solution of chloral hydrate to remove all trace of the discolouration by Müller's fluid and then mounted in a glass dish filled with glycerine jelly for macroscopic observation. The other half was used for the purpose of making microscopic sections, longitudinal and transverse, both through

the ligatured point and at various distances on either side of it. In this way a large number of sections of clot at different periods after ligation were obtained in which the conduct and fate of clotted blood in arteries could be studied. The use of Müller's fluid as a hardening medium is very advantageous, as it preserves the outline of the red corpuscles in a remarkable way, and thus the stages of their absorption and disintegration can be followed without difficulty: the only disadvantage being that, unlike osmic acid, it to a large extent prevents the observation of the karyo-kinetic changes in the invading and multiplying plasma-cells which by the processes of absorption and substitution are destined to replace the clot.

Formation of Clot.

At the outset of the inquiry into the conduct and fate of clot in ligated arteries it seems desirable to briefly refer to the most recent views with regard to the intimate causes which determine the coagulation of the blood. Many observers have been of opinion that clotting did not necessarily take place when no injury is inflicted upon the arterial wall. In all our ligation experiments, in the great majority of which no such injury was inflicted, a proximal and distal clot were always found.

Physiologists are not yet agreed as to the processes which cause coagulation, but the most generally accepted view is that of Hammersten, which has recently been confirmed and defended by Halliburton in the *Journal of Physiology* in reply to our friend the late lamented Wooldridge. According to this, coagulation is due to the action on the fibrinogen of the blood-plasma of a ferment

derived from the lethal disintegration of blood-platelets, a special variety of white blood-corpuscles of small size.

It is probable that various circumstances may cause the death of the blood-platelet, and therefore if this be the true explanation of the process, many different events may determine the coagulation of the blood.

When the coats of an artery are ruptured, fibrin is first deposited on the damaged recurved tunics: these then will initiate the disintegration of the cell containing the fibrin ferment.

When the coats are uninjured the problem of clottage presents greater difficulties. Riedel states that he has made the opposite sides of an artery cohere by multiplication of the opposed endothelial cells without the formation of any clot. Dent and Delépine, too, as the result of the study of ligatured sheep's femorals, sent to them by Stirling from Australia, state that "coagulation is not a "necessary phenomenon of ligation, that it may be entirely "absent if a collateral branch sufficiently large is im- "mediately above the ligature, and that it only occasion- "ally occurs beyond the ligature." Paul Bruns states that if the coats of an artery be not ruptured by the ligature clotting does not take place. In our experiments, as has been already stated, clot was always formed, whether the coats were ruptured or not. Frequently too we found collateral branches of not inconsiderable size in the immediate neighbourhood of the seat of ligature (e.g. arteries to the thyroid gland) filled with clot continuous with that in the main carotid vessel. For exceptions to this condition see Plate III. Figs. 3 and 5. To test the statement that if a ligature be applied immediately

below a large branch no clot is formed, we applied a ligature on several occasions as close below a large branch as we could (e.g. to the common carotid, as close as possible

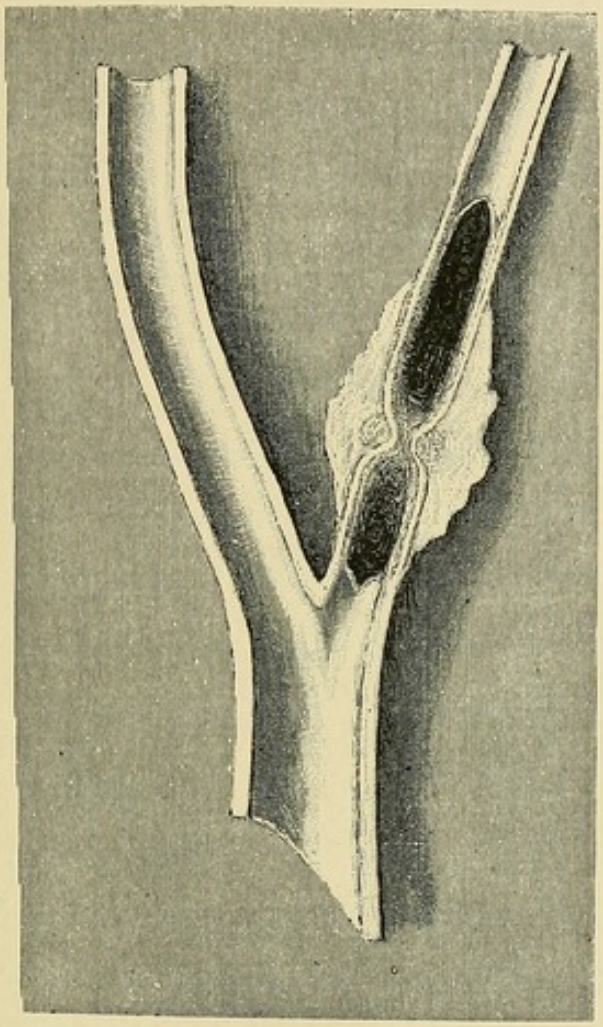


FIG. 58. Anterior Aorta and two Common Carotids of an Ass 42 days after ligature. (Nat. size.)

The ligature used was a clove hitch of kangaroo tendon. It was applied $\frac{5}{8}$th of an inch (15 mm.) from the bifurcation. The arterial wall is uninjured and the intimæ of opposite sides are in apposition; firm occlusion has occurred; some suppuration took place, which accounts for the amount of new tissue round the seat of ligature. The difference in size of the two vessels will be noticed. Owing to the suppuration much of the ligature had disappeared. Its track was partly occupied by a minute quantity of pus, from which a small sinus passed through the external sheath of plastic material.

The operation was performed through a central incision. The anterior aorta lies deeply in the chest behind the sternum. The object of the operation was to test the result of ligation near a large collateral branch, the coats being unruptured.

to the bifurcation of the anterior aorta [see Fig. 58]), and in every instance a clot was deposited; indeed, it seems to us impossible to apply a ligature so close to a branch that there will not be recesses between the pleats into which the vessel is thrown where clot will form.

When a ligature is applied so that the tunics are undamaged and yet the lumen of the artery is occluded, a profound alteration in nutrition is at once established at the ligatured point. The vasa vasorum are blocked and the plastic effusion which ensues is often sufficient in thirty hours to bury the loop of the ligature, which as a foreign body is itself of course a stimulus to active plastic effusion. Simultaneously the opposed endothelial surfaces proliferate and commence to cohere. Thiersch has observed proliferation of the intima a few hours after ligation. Physiologists agree that any alteration in the endothelial lining of a vessel is enough to account for the formation of clot, in other words, for that fatality amongst the blood-platelets which precedes the production of fibrin. Michael Foster says, "that any treatment of a "blood-vessel tending to alter its normal condition causes "local coagulation, and that between the nearest collateral "branch and the ligature there is stasis: though this may "not be complete. The consequence is that the walls of "the artery suffer from want of renewal of the blood, which "favours coagulation." Lister has shown that horses' blood drawn under conditions of absolute asepticity into sterile glass vessels will always clot, but will not contract even after many weeks: a phenomenon which we think we have observed in some of our completely aseptic ligature operations.

To test this point we drew the arterial blood of a horse into several sterile test-tubes; in every case the blood soon clotted, but in other respects it behaved differently. In one tube (Fig. 59) the red corpuscles remained uni-

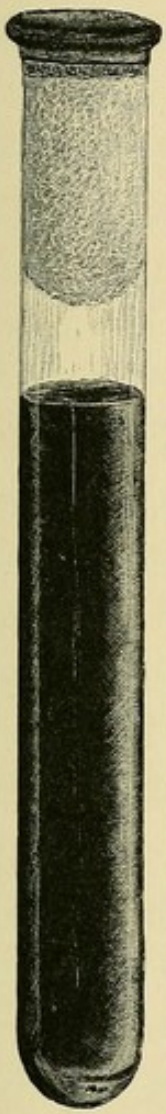


FIG. 59.

FIG. 60.

FIG. 61.

These test-tubes were rendered sterile and into them under aseptic precautions blood was drawn from a thyroid artery of a horse. The drawings were made some weeks later. All the tubes remained sterile and in all the blood clotted. In tube (fig. 59) the red corpuscles did not sink, no buffy coat formed, no contraction occurred and no serum was expressed: in tube (fig. 60) the red corpuscles sank, a buffy coat formed, no contraction occurred and no serum was expressed: in tube (fig. 61) the red corpuscles sank, a buffy coat formed and contracted, expressing serum. The cause of these variations is not known.

formly distributed throughout the clot, which did not contract: in another tube (Fig. 60) the red corpuscles fell to the lower part of the clot, the upper part in consequence becoming of a buffy color, but neither part of the clot contracted: in a third tube (Fig. 61) a buffy coat formed in the same way, but it contracted, expelling a considerable quantity of clear serum. Whatever may be the cause of these varieties it is certainly not connected with septic changes, for the tubes remained sterile and unaltered for many weeks. The blood of a guinea-pig and of a snake drawn in the same manner was found to clot, and the clot to contract, but no buffy coat was formed.

It has been said that a vital equilibrium exists between the blood and the vessels which contain it, and that coagulation will not occur while this equilibrium is maintained. Brücke's idea that the fluidity of the blood was due to some mysterious action of the intact living wall of the vessel again emphasizes the importance of an unaltered and natural state of the wall of the vessel, for otherwise coagulation will sooner or later take place. The nutritive changes at the point of ligature even when the tunics are undamaged are, we think, an ample explanation of the occurrence of the proximal and distal coagulation that was found in all our experiments.

Zahn has pointed out that the application of a crystal of common salt to the exterior of a frog's artery will cause the colourless blood-corpuscles within the artery to collect at the point which corresponds to the crystal, and the number continuously increases until a thrombus plugs the vessel; and Lister has shown that if a vein be ligatured in two places and a caustic be applied at a point between

the ligatures, coagulation will first take place at that spot in the interior of the vein which is nearest to the place where the caustic has been applied. If such slight stimuli effect coagulation it is difficult to conceive how a ligature, even when so applied as merely to make the artery impervious to blood and yet not to interfere with the integrity of its wall, could be employed without sooner or later causing the formation within the artery of a proximal and distal thrombus.

Conduct of Blood between Two Ligatures: living test-tube.

Whether blood arrested between two ligatures in a living vessel within the body will remain fluid or coagulate has long been discussed. Hewson tried the experiment in the last century and found the blood clotted: Gulliver found the same, in fact the experiment has often been tried in this country and always with the same result: recently, however, Baumgarten and Böttcher in Germany have come to the opposite conclusion. It might be thought that the fluid hæmatomata in man, which occasionally persist for months after injury, decide the question, as far at least as blood remaining aseptic in the living tissues is concerned; but, as Morrant Baker points out, there is a fallacy in this: in the first place clot does form, for whenever these blood tumours have been opened, clot has always been found, and when the tumour has not been opened, but merely aspirated, the sac is found to contain, in addition to the coagulated blood, a fluid derived partly from disintegrated clot, partly from effused serum, and partly from recently effused blood; for it is probable that in some, at least, of these cases, there is recurrent hemor-

rhage, the fluid drawn off containing not old and shrivelled red corpuscles, but others young and well formed.

In 1877 Baumgarten stated that blood confined in a vessel between two ligatures remained fluid, and was gradually absorbed in the fluid state by the advance of the new tissue.

Böttcher in 1887 repeated some of Baumgarten's experiments, and found that, without exception, the blood was fluid between the two ligatures. These experiments were made on the carotid arteries and veins of rabbits. The arteries were cut out 7, 14, 24 or 28 days after ligation, and sections were placed at once in Flemming's mixture or in chromic acid, or the liquid blood was squeezed on to cover-glasses, and then fixed by osmic acid or by the flame. In one specimen, when the animal was killed on the tenth day, he says that the coagulation observed was caused by the fixation fluid and that the red discs where the endothelium was proliferating to the greatest extent were indistinct in outline. This alteration in the appearance of the red corpuscles seems to point to the usual ante-mortem change of the red cells in the presence of plasma-corpuscles, if not to ante-mortem coagulation.

The most interesting part of Böttcher's research, however, is his endeavour to discover the blood-platelets in the still liquid blood. He adopted Schimmelbusch's method of fixation and methyl violet staining, and was able to discover them in large numbers up to the seventh day, but not afterwards. He sums up his work as follows:—"That intravascular blood arrested between ligatures "remains liquid and becomes of a dark venous colour. The

"leucocytes in it undergo fatty degeneration, but the "red discs may appear normal, even at the end of four "weeks. Large endothelial cells can be seen traversing "the liquid blood, or they may be seen in colonies and "not singly. The blood-platelets could always be de- "monstrated up to the seventh day, but never after the "fourteenth."

Baumgarten has also observed the invasion of fluid blood by plasma-cells, and says that the cells are cast off and become free of the intima by means of the act of cell division. He observed the process especially during the investigations on which his work on the *Histogenesis of the Tubercular Process* is founded.

The fluid blood in a vessel between two ligatures described by Böttcher and Baumgarten as invaded by plasma-cells is not, we submit, blood, but a mixture of expressed and effused serum and any remnant of clot not yet disintegrated: in the tiny vessels used by them the clot would soon disappear.

The experiments we have performed on this point are not sufficiently numerous to justify any positive conclusion. Carotids and veins of rabbits and carotids of monkeys were tied, the ligatures being fixed about half-an-inch (12 mm.) apart. In some experiments the coats were ruptured, in others not: the animals were killed in from one to several weeks: suppuration never occurred. The piece of vessel between the ligatures was generally found converted into a fibrous cord (as was the case in one of Böttcher's experiments at the end of 24 days). In no instance was the blood discovered fluid, but in some a clot, partly replaced by young connective tissue, was visible, but

this had contracted away from one side of the vessel, the remaining space containing fluid.

One view is that coagulation does take place, but only after a shorter or longer period of delay. This conclusion, however, appears to be opposed to Böttcher's experiments. If it be confirmed that the blood-platelets disappear after the seventh day, and that their presence is essential to the production of clot, then coagulation, if it occurs, must be completed by the seventh day and cannot happen afterwards. There may be factors, however, at work, of which we are at present ignorant, and possibly in all cases there may not be a complete or an apparent uniformity in the phenomena observed. We have so often found coagulation to follow proliferation of the endothelium, that it is difficult to think that coagulation does not happen in blood confined in an artery or vein between two ligatures, seeing that all observers agree that under these circumstances there is a marked multiplication of the endothelium. Indeed, we believe that the blood does clot, but even if occasionally it seemingly remains fluid, as in hæmatomata, its digestion and replacement by the plasma-cells are likely to be at least as easily accomplished as when the blood has become solid by clotting.

The old experiment of the "living test-tube" bears on the question: for if coagulation be delayed in a vein external to the body it might be expected to be also delayed in blood confined within a living vessel between two ligatures. The living test-tube experiment was repeated by us in the following manner. The large jugular vein of a horse when full of blood was ligatured in two places about 8 inches (20 cm.) apart. This section of

vein was then removed from the body of the animal and suspended in a glass vessel in which arrangements were made for keeping the air moist. The blood never clotted firmly, but in less than a week became of a thick treacle-like consistence. In this experiment it must be remembered that no multiplication of the endothelium occurs.

Asepticity and Intravascular Coagulation after Ligature.

It has been more than once suggested that primary union of a wound in certain cases, such as occurs under a Listerian dressing, is in some respects disadvantageous. A most striking instance of an attempt to uphold this view is to be met with in an account of a case of ligature. The paper is entitled, "A case illustrating the condition of large arteries after ligation under antiseptic and non-antiseptic measures," and its present interest lies in the suggestion by its author that "the failure to effect "permanent occlusion of a great artery was consequent on "the primary union of the wound, and, in fact, that "surgically the healing of the wound was too perfect; "whereas if suppuration had occurred [as it did after the "ligature of the subclavian in the same case] the intra-"vascular clot would have been much larger and the "chances of the re-establishment of the circulation would "have been correspondingly diminished." The common carotid had been tied with catgut, and on the death of the patient, 108 days later, the vessel was found to be patent. Its lumen was contracted by the development of a diaphragm with a small central aperture. In the section dealing with diaphragms in arteries we have shewn

that the production of a complete or partial partition across the lumen of an artery depends upon causes other than that of suppuration. If the wound, by which the common carotid had been tied, had suppurated, it is quite possible that a diaphragm might not have formed, and the vessel have been obliterated; but it is also possible that the pus would have destroyed the outer tunic, and the other two having been cut across, the complete solution of the arterial wall thus effected in an artery only partially obstructed, might not improbably have been followed by fatal hemorrhage. (Cf. fig. 85.) Safety therefore is to be sought not in suppuration and complete division of the arterial wall, but in asepticity and maintaining intact the coats of the vessel.

We have already mentioned our own experiments which show that the blood of different animals invariably clots if drawn into glass tubes or vessels under sterile conditions, and that after ligature the blood in veins and arteries always coagulates even when no injury has been done to the coats by the use of the ligature, and when the wounds have healed by first intention. Lister, moreover, showed long ago that sterility or asepticity has nothing to do with the process of coagulation of the blood. Some observers have, however, thought differently.

Baumgarten, from numerous experiments, comes to the conclusion that no thrombus is formed if healing takes place without suppuration. Böttcher agrees with him.

Suppuration is now allowed on all hands to be associated with the presence of bacteria, whether these reach the wound directly or through the medium of the circulating blood; so that, if it be true that a clot in an artery has

an essential connection with the presence of pus outside it, we should be almost driven to conclude that clot formation was associated in some way or other with bacteric conditions. Such a conclusion is, however, with our present knowledge impossible to accept, for it would mean that the germs of bacteria were latent in every tissue of the body and were capable of producing coagulation in healthy bodies in which healing by primary adhesion occurred without formation of pus.

Moreover, in numerous experiments we have ligatured arteries through small wounds with the minimum amount of disturbance of the parts and with antiseptic precautions: in a large number of these experiments no damage was done to the arterial wall and no suppuration took place, and yet a proximal and distal coagulum formed above and below the seat of ligature. The same result was obtained in many instances, in which the tunics were invariably ruptured, by Stirling, in Australia, who ligatured many sheep carotids for Dent, who has kindly allowed us to see them. If, therefore, coagulation in the living body depends on suppuration or septicity, which is scarcely conceivable, the essential bacterium of clot must be at all times latent in the fluid blood. There is no time for it to gain entrance into the blood-stream from the respiratory or intestinal tracts in consequence, for example, of the onset of fever, for the coagulation process occurs at once on the recurved edges of the tunics if the coats be ruptured and very shortly also if they be not injured. We have ourselves observed a fragment of clot already formed in one hour on the proximal side of the ligature, the coats being uninjured; and this is easily understood, since distinct en-

dothelial proliferation at the seat of ligation has been observed within the same short time.

It seems probable then that the presence of bacteria has no relation to the coagulation of the blood, that primary union of a wound made in order to ligate a great artery is a safeguard against hemorrhage, and that the healing of a wound, and still more of a wound leading to a ligatured artery, cannot be too perfect.

Extent and Date of the Clot. Ampulla of Bryant.

When the coats are uninjured the coagulum does not form as rapidly as when they are ruptured by the ligature. It is probable that in the former case fibrin is not deposited till the blocking of the vasa vasorum and the consequent exudation of fluid and diapedesis of leucocytes has induced a sufficient alteration in the arterial wall at the site of the ligature. No doubt also plastic changes occur in consequence of the presence of the ligature as a foreign body. In one of the Ziegler chamber experiments the leucocytes had entered and were clustering round the mouth of the chamber in large numbers at the end of only four hours. Again, W. Hunter states that six hours after intraperitoneal injection of blood scarcely a white corpuscle was to be found in a field of several hundred squares of the circulating blood; they had migrated into the abdominal cavity. These observations appear to indicate the period beyond which it would not be likely for clotting to be delayed within a vessel ligated without rupture of the tunics; and our experiments accord with this. For example, fine fragments of clot were found on each side of the point of constriction in the carotid of a

horse one hour after the artery had been ligatured, and a considerable proximal and distal clot had formed in a sheep's carotid 9½ hours after ligation.

The extent to which coagulation takes place varies. In small arteries the old view that it occurs as far as the nearest collateral branch is no doubt usually correct, but in large vessels it is not so. In the common carotid arteries of horses and asses we observed, as a rule, a clot for four or five inches (10—13 cm.) on the proximal, and for two or three inches (5—8 cm.) on the distal, side of the ligature; the thyroid branches being also blocked. In some cases, especially those examined within a short time (say one to three weeks) of ligation, the proximal clot occupied the artery for many inches and even extended in it nearly to, or as far as, the anterior aorta. The fact that these long proximal clots were not found at later periods after ligation seems to show, in some instances at least, that their tapering ends were broken up and washed away by the blood-stream. The distal clot is almost invariably smaller, shorter and narrower than the proximal, and it does not generally distend the artery in the same way. An exception to this statement occurs in Experiment No. 68 (Plate III. Fig. 5). Here a distal clot was found distending the artery for many inches, whilst the proximal was only one inch (24 mm.) in length, reaching only as far as the origin of an unusually large thyroid branch. The proximal coagulum distends the artery immediately above the ligature in a remarkable way, forming the ampullary enlargement described by Bryant. From the ampulla the clot becomes gradually smaller until it ends usually in a free extremity. We have in-

vestigated the ampulla-like dilatation and we agree with Warren that in reality there is no dilatation at all, but the appearance is produced by the contraction of the vessel above the ampulla, where it is not completely filled with clot. We find that the extreme diameter of the ampulla is not greater than that of the artery before the ligature is applied; the pressure of the clot in the ampulla cannot be greater than that of the blood, and moreover it seems probable that the extreme diameter of a great artery is maintained by the pressure of the circulating blood and is not likely to be suddenly increased against the inelastic resistance of the outer coat.

The artery beyond the distal and above the proximal thrombus is always contracted and considerably diminished in size. The obstruction being permanent, the amount of permanent diminution of calibre of the artery is very noticeable: though it must be borne in mind that our experimental ligations were all done on the common carotid arteries, which are of great length and have comparatively small branches.

In one experiment in which the left common carotid artery of a horse had been ligatured 55 days before the death of the animal, its outside diameter at the post-mortem examination was 9 mm., and when distended with liquid cocoa-butter under a pressure of 300 mm. of mercury the outside diameter was 13 mm. The unobstructed right common carotid artery of the same horse measured under similar circumstances 14 mm. and 18 mm. respectively. (See Exp. 67.)

When the coagulum had in part disappeared, its original extent, as far as it was in contact with the

inner wall, we thought, could be accurately gauged by the limit of the thickened endothelium: at the same time it must not be forgotten that some observers have stated that, with a diminished blood-pressure, proliferation and consequent thickening of the intima occurs as a kind of physiological adaptation to the changed functions of the vessel.

The clot, when to the naked eye it seemed to fill the artery, is often found to be attached to the intima only in one, two, or a few places: indeed, it is often possible to make a transverse section in which the lumen of the artery is still unobstructed in its entire periphery to the extent of a slit-like space intervening between the clot and the inner aspect of the tunica intima. This interval may extend up to the point of ligature, and as it contains fluid blood has an important bearing on the occurrence of hemorrhage.

Function of the Clot: Collateral Branches.

The clot which forms above and below the seat of ligature takes no active part in the constructive process by which an artery is obliterated: this is now generally admitted; Ziegler, Cornil and Ranvier and others, all agree in this. Böttcher even states that not only does the thrombus play a passive part, but it may actually be a hindrance to cicatrisation. Travers experimentally proved its non-essential nature, though many before his day had combated the doctrine of Petit. The coagulum within a deligated vessel is as much a foreign and dead substance as the clot on the flap of an amputation stump. It, like the aseptic animal ligature encircling the artery, is gra-

dually absorbed. The clotting of the blood is an indication of the death of that tissue, and has been described as the rigor mortis of the blood. The use of a coagulum in a deligated artery appears to be threefold: firstly, to act as a barrier or buffer between the impulse of the blood-stream and the seat of ligature, where important plastic actions are in progress which might otherwise be disturbed or interrupted; secondly, to afford some support, ladder-like, to the plasma cell invasion as the latter climbs across the cavity of the vessel; and thirdly, to serve as food for the invading and actively proliferating army of plasma-cells, since, as has already been pointed out, lack of nutriment arrests the activity of the plasma-corpuscle and causes it to assume a condition of quietude and encystment. Though the clot is dead and takes no part in those living processes by which the final obliteration of a vessel is secured, a deficiency in its amount when the arterial wall is damaged may be a predisposing factor of an attack of hemorrhage, and especially is this said to occur when a large collateral branch is close to the deligated point.

As, however, the formation and extent of the coagulum have really nothing to do with the adhesive changes by means of which permanent occlusion of an artery is obtained, the presence of a large branch near the seat of ligation, if the coats be not weakened by rupture, is of trifling importance, or more truly perhaps of no importance at all. The living and uninjured wall is the only true safeguard against hemorrhage.

The plastic actions which proceed at the place of ligation, are practically the same whether the tunics be ruptured or not. In the former case, however, any retar-

dation of the constructive process on account of the general condition of the patient or from septicity of the wound may, especially if the rupture be in the vicinity of a large branch, be attended with grave risk to life—a risk which can by no means be made light of even when the course of events in the wound is apparently favorable. On the other hand, when the tunics are undamaged, the nearness of a collateral branch is immaterial, and even suppuration in the wound is much less serious. The presence of pus is always associated with an increase in the size of the fusiform swelling around the artery, and this constitutes, when the arterial wall is intact, an additional protection against hemorrhage.

Strata and Fissures of the Clot.

The clot is deposited gradually and is always stratified, the oldest parts being placed centrally and nearest to the ligature: on transverse section a number of concentric lamellæ are seen, the outermost being the most recently deposited. (Fig. 62.) The rush of blood between the proximal clot and the wall of the artery tends to prevent the formation of adhesions and is the cause of the constant addition of layers of fresh clot to the exterior of the thrombus. The chief characteristic of clot outside the body is its tendency to contract; contraction goes on until putridity sets in. This tendency apparently existed in most of the intravascular coagula which have been studied by us, unless indeed the seeming centripetal movement of the layers of clot was due, not to contraction, but to the force exercised on them by the peripheral blood-stream. Lister showed that horses' blood drawn

under aseptic conditions will clot, but does not contract. If this be true, and our own experiments in part confirm it,

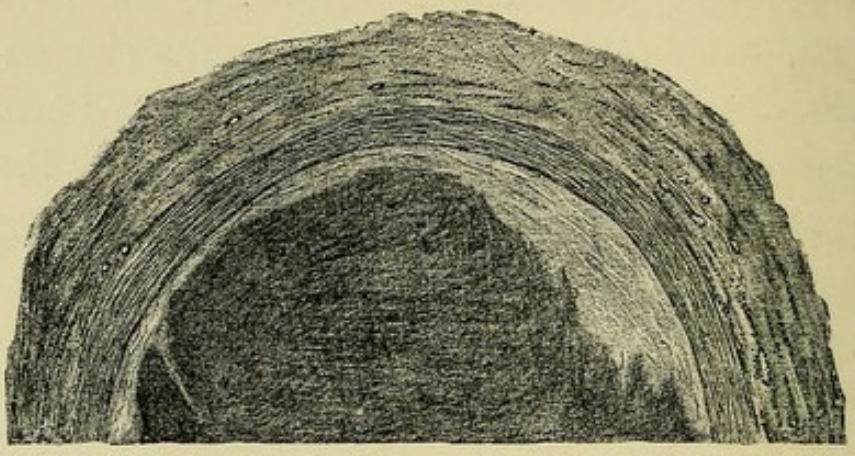


FIG. 62. Carotid Artery of a Horse 14 days after ligature (× 6).

It is a transverse section: the wall of the vessel is well defined: the dark part in the centre is the earliest clot, which is now free from the artery wall in its whole circumference: between it and the intima are several layers of fresh coagulum concentrically deposited, the outermost being the most recent: the separation between these layers is marked by fine white lines.

surely after perfectly aseptic operations the intravascular coagula in our experiments ought not to have exhibited any tendency to contraction. We think that possibly the specimens resulting from a few of our experiments lend some support to Lister's remarkable statement, namely, those in which not far from the ligature the coagulum entirely fills the vessel and is attached by strong intimal prolongations to the wall of the artery in its whole circumference. Just as in the test-tube experiments some clots contracted while others did not (Figs. 59, 60 and 61), so it is possible that intravascular coagula may or may not contract. To tell whether or not a small clot contracts (especially when surrounded by moving blood under pressure) is a very difficult problem. Confirmation of the truth of Lister's experiment is offered by the stability

of clots of blood in aseptic wounds, which do not appear to contract.

Between the strata endothelial cells can sometimes be seen which have been perhaps carried off by the "contracting" clot, or, what is more probable, a few cells are carried off when the surface of the clot leaves the vicinity of the intima and the remainder are due to the active proliferation of these. (Fig. 63.)

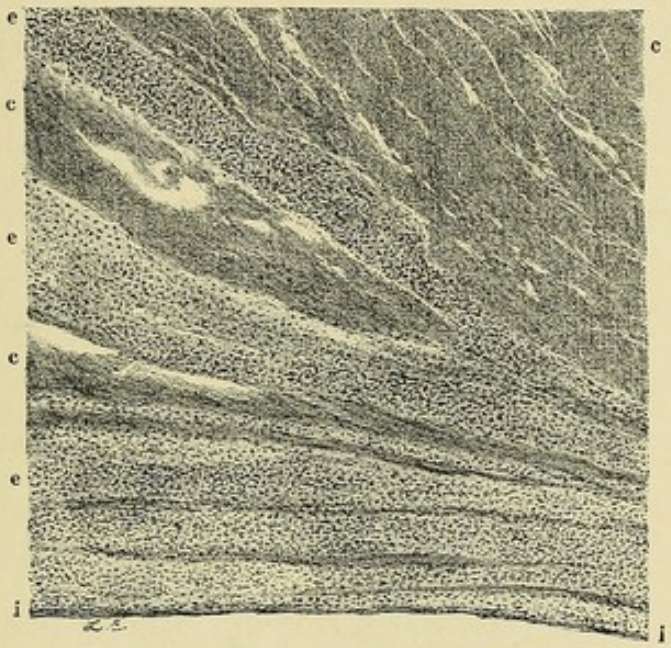


FIG. 63. Proximal Coagulum from Carotid of Horse 14 days after ligature (× 40).

At the lower part of drawing *ii* represents the innermost layer of the thickened intima: the ligature was some distance to the right and the clot extended a little further to the left, the portion selected for representation being near its conical end.

The coagulum is represented in different layers by *c*, *c*, *c*, and between these are layers of plasma-corpuscles (*e*, *e*, *e*), forming young connective tissue: the process by which this arrangement has arisen is as follows: the coagulum (*c*) which is uppermost in the drawing was originally in contact with the arterial wall; later, by a process described in the text, it became detached, carrying with it endothelial cells: on its detachment the second coagulum (*c*) formed between it and the arterial wall, and it in its turn became detached and its place taken by the third coagulum: in this way numerous layers are formed and the endothelial cells in the different layers continuing to grow and to absorb the clot, the whole becomes eventually converted into connective tissue.

The phenomena observed in the intravascular coagula in arteries have been described by Heuking and Thoma as occurring in venous thrombi in a most interesting paper on "The substitution of the thrombus in veins by connective tissue." They state that venous thrombi are constantly permeated by a system of minute cracks and fissures, and that the central layers of the coagulum are often quite granular whilst the outermost are composed of clot in which the outline of every cell can be easily distinguished.

The cracks and fissures are found in all parts of the clot and exhibit a distinct regularity in arrangement; they divide the clot up into little portions of fairly equal size. (Fig. 64.)

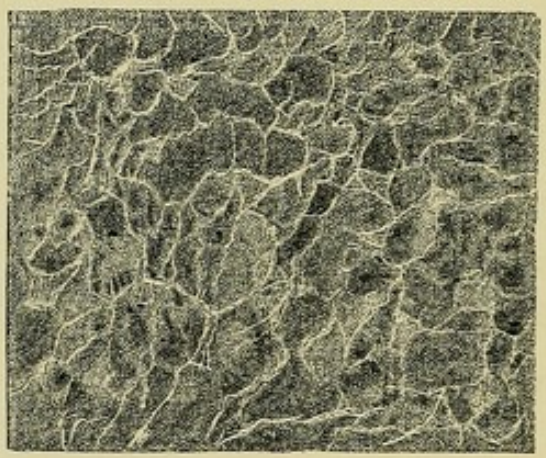


FIG. 64. Cracks and Fissures of the Clot (× 90).

The drawing was made from a portion of clot in the carotid artery of a horse 14 days after ligation. It was taken 4 inches from the seat of ligature, where it was not adherent to the artery wall and was as yet uninvaded by leucocytes or plasma-corpuscles.

Although the ligation had taken place 14 days previously, this portion of clot was probably of much more recent date. This is the stage spoken of by Dent and Delépine as mechanical vacuolation.

There are also cracks between the layers of clot or spaces of various sizes and irregular in shape or outline.

If they be near a point where the thrombus is adherent to the intima they soon become lined or filled by the invading plasma-cells.

The fissures, we think, do not occur till some days after the formation of the clot, but the exact date of their appearance is uncertain. Sometimes fresh blood-corpuscles can be seen in them, indicating their connection, at some point in the periphery of the clot, with the lumen of the artery.

When the clot is loosened from its attachment to the vessel wall in any place, a partial peripheral re-opening of the lumen of the vessel for the current of blood takes place. The consequent inrush of blood allows either of further clot-strata being deposited, or else of a layer of fluid blood establishing itself between the arterial wall and the clot, it may be, as far as the deligated point.

Doubtless there are mechanical and chemical causes, not yet properly understood, which determine the breaking up of clot into little masses, or, as we term them, "clot districts." The more irregular spaces between layers of clot may be the result of shrinkage or of displacement of portions of the coagulum, combined with the unequal attachment of the clot to the arterial wall and of the numerous strata with each other. The importance of the cracks and fissures of the clot in the absorption and substitution process is paramount. The plasma-cells travel without difficulty through the network of lanes thus laid down for them (Fig. 65), and their onward progress is possibly assisted by feeble currents of fluid which pass along these channels of the clot.

We have thought that the primary cellular invasion of

the clot previous to the occurrence of "mechanical vacuolation" is along the fibrin trabeculæ, and W. Hunter has

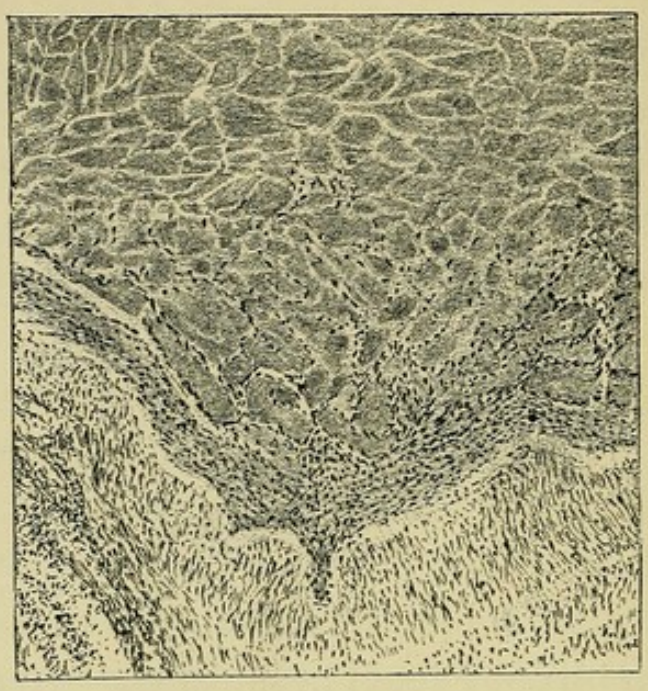


FIG. 65. From the Carotid of a Sheep 21 days after ligature (× 95).

In the lower part of figure is seen the middle coat: above that the white line shews the membrane of Henle; above that, again, a darker band is the thickened intima; the remainder of the figure is coagulum: this is a recent clot which has been deposited between the original clot and the vessel wall: the coagulum is fissured by a mechanical or chemical process, and near the endothelium corpuscles may be seen invading it along the cracks and fissures.

observed the same phenomenon in small adherent clots examined 48 hours after the injection of blood into the peritoneal cavity (Fig. 67, page 172). We think it highly probable that the later cleavage of the clot occurs also along the lines of the fibrin trabeculæ from node to node, for in whatever way the plasma-corpuscles enter a section of a clot, whether by making a passage for themselves along the fibrin trabeculæ, or by passing freely along cracks and channels already formed, the little masses of clot or "clot districts" into which the whole clot is divided

up by the advancing cells invariably appear alike in the matter of size and shape. (Figs. 64, 65 and 94.)

It will thus be seen that while the cracks and fissures depend for their production for the most part upon obscure mechanical and chemical conditions, yet at an early stage in the history of the clot some channels may be formed by a kind of vital process, viz. the tunnelling of the clot by the invading army of cells.

It is worthy of remark that the attack of the living cells upon the clot must be greatly facilitated by the splitting up of the whole foreign body into small portions, which can be attacked separately and, as it were, absorbed in detail.

Absorption and Substitution.

In the Ziegler chamber experiments the formation of fibrin previous to the cellular invasions was observed. At certain points, presumably the site of the disintegration of a blood-platelet, a fibrin node showed itself, and spreading from it rosette-like in all directions the fibrin trabeculæ appeared. The trabeculæ, spreading radially from a node, met at two, three or more places, the fibrin filaments advancing from neighbouring nodes. As a consequence, the bands of fibrin running between contiguous nodes were much more numerous and more thickly grouped together than the filaments of fibrin which were distributed in other and less definite directions. The origin of the fibrin nodes from groups of dying blood-platelets has been described by Schimmelbusch and confirmed by Böttcher, as has also the arrangement and development of the fibrin network which we also have observed. Subsequently the

leucocytic and plasma-cell invasion occurs, both kinds of cells travelling to the position occupied by the fibrin node, being in part perhaps guided by the lines of fibrin and in part attracted by the abundance of suitable food provided, in the one case by the fibrin node and in the other by the leucocytic islet.

It is not surprising that the arrangement of the fibrin network, which in the Ziegler chamber films played such an important part in the distribution of leucocytes and plasma-corpuscles, should also exert an influence on the manner the red corpuscles group themselves when blood clots. This question is the more difficult of examination since the red cells are so numerous that no fibrin can be seen in ordinary sections of clot.

Fig. 66 shows the arrangement of the red cells of coagulated frog's blood, the way in which the red corpuscles form "rosettes" around the nodes of fibrin, and the bands of cells (rayons) which are gathered, as we believe, along the numerous trabeculæ of fibrin which pass from one node to another. Ranvier says that the same appearance can be demonstrated in human blood-clot, but adds that with it the preparation of such a specimen is far more difficult.

It may be observed that as in the Ziegler film the lines of fibrin between contiguous nodes walled in, as it were, little spaces and gave to the whole membrane the appearance of an open network, the little open spaces being of fairly equal size, visible everywhere and invariably present, occupied by comparatively few fibrin filaments and bounded on all sides by dense lines of fibrin which stretched from node to node, so we have reason to think

that in all blood-clot a similar laying down of the fibrin network must occur, which has the effect of breaking up the mass of clot into little portions or "districts" bounded on all sides by the internodal fibrin bands. In the chamber

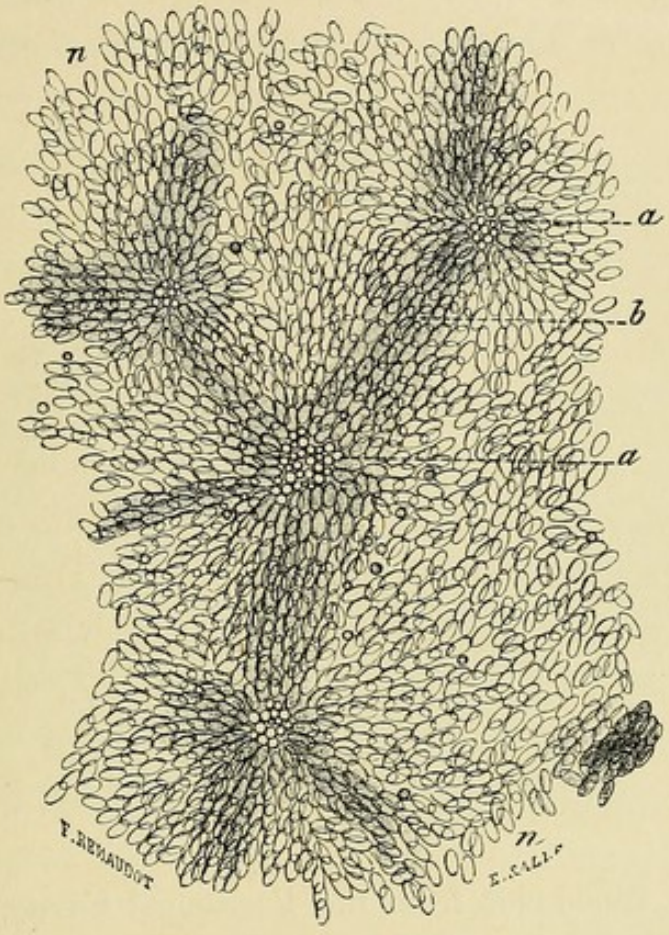


FIG. 66. Blood from Frog's Heart examined 24 hours after being taken (× 100).

Copied from Ranvier's Traité technique d'Histologie (by permission of M. Savy, Paris).

(*a a*) "Rosaces centrales:" arrangement of red blood-corpuscles in rosettes around nodal points of fibrin.

(*b*) "Rayon:" red blood-corpuscles arranged along rays of fibrin.

(*n*) Isolated red blood-corpuscles.

experiments the invading cells were observed travelling towards the position occupied, or once occupied, by the fibrin node, and we believe that the invading cells, passing

into intravascular coagula, travel along the lines of the fibrin trabeculæ: the peptonised trabeculæ forming an excellent pabulum for them in their journey onward to the node. It is probable, moreover, that the voyage of the invading cell in this direction would encounter little resistance; no transverse obstructing filaments of fibrin

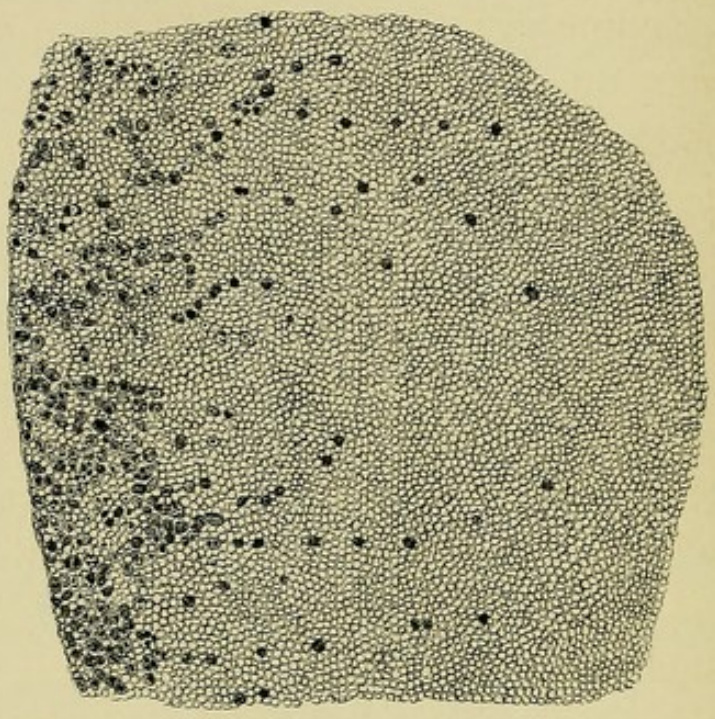


FIG. 67. Blood-clot from the Peritoneal Cavity (× 200).

(*From a specimen kindly lent by Dr Wm. Hunter.*)

The specimen was obtained from the abdominal cavity of an animal into which a large quantity of blood had been injected 48 hours previously. The above was taken from a small adherent clot: the adherent border is to the left. The corpuscular invasion is commencing: it appears to have a tendency to occur in lines.

On looking closely it is possible to recognise many of these corpuscles as leucocytes and others as plasma-cells. The exact date at which this portion of clot formed cannot be known: it is probably much less than 48 hours old.

would bar the way, and forward the cell would worm itself in the tiny interval left void of solid matter by the gradual solution of the fibrin trabecula to which it is applied.

Fig. 67 shows a section through the attached edge of a clot at the end of 48 hours. The primary cellular invasion is seen. The advancing lines of cells appear to occupy a definite position and each line seems to be separated from others by intervals of fairly equal size. The position taken by these lines of cells is occupied in turn by the ever increasing army of invading corpuscles until in place of single lines solid cylinders of cells appear; these divide the clot up into "districts," and indicate also the points at which the primitive capillaries of clot may first be seen.

Fig. 68 shows these parallel lines of cell-columns or embryo capillaries entering a clot on the tenth day.

What we venture to suggest is that the primary cellular invasion, the solid cylinders of cells and, later, the capillaries, all occupy the place once held by the bands of fibrin which stretch from node to node. The invading cells, leucocytes or plasma-corpuscles, or both, collect in little groups through the substance of the clot, and from these groups bands of cells pass off to other groups, so that it is almost impossible not to fancy that the groups of cells indicate the position once occupied by the fibrin nodes, and that the bands of cells stretching between the cell-islets have replaced the trabeculæ of fibrin that formerly connected together the nodes of fibrin. The knowledge gained by the study of the invasion of the fibrin network of the Ziegler chambers by leucocytes and plasma-corpuscles is the key to the explanation of the paths taken by the same cells when invading a clot which is but fibrin network and red corpuscles. In each instance the voyaging of the invading cells is excited by the same stimuli, con-

tinued under like influences and controlled by the same laws.

The plasma-cells mostly come from the proliferating endothelium of the vessel, and the previous invasion of

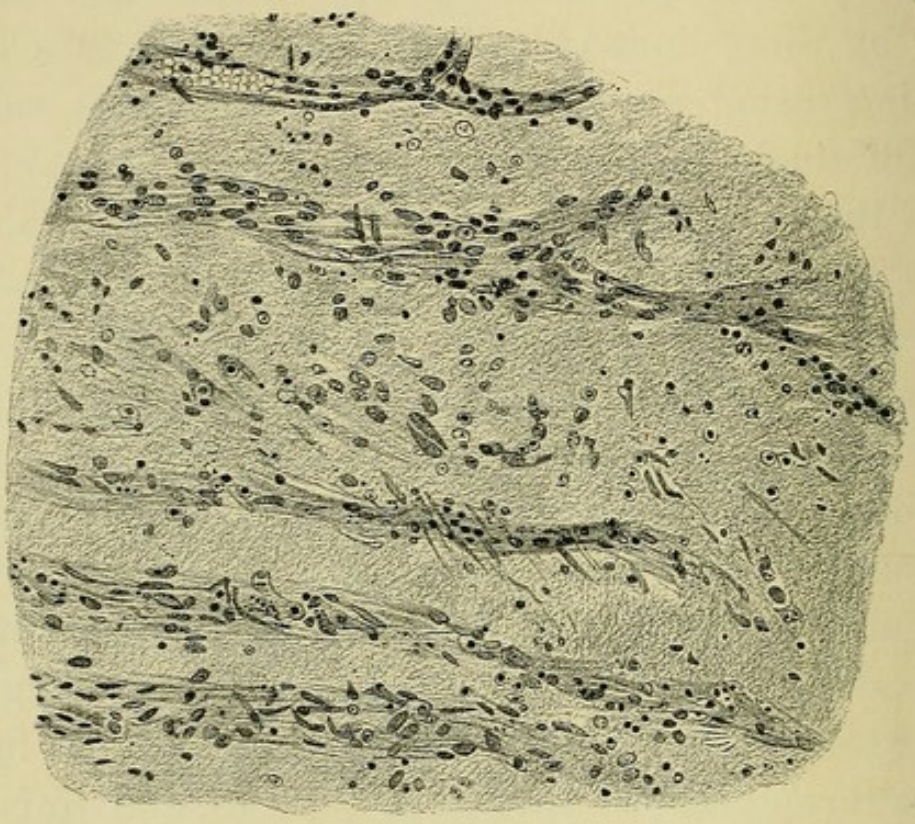


FIG. 68. Blood-clot from Peritoneal Cavity (× 200).

(*From a specimen kindly lent by Dr Wm. Hunter.*)

The specimen was obtained from the abdominal cavity of an animal into which a large quantity of blood had been injected 10 days previously: the drawing was made from a small clot: its adherent border is to the left. The outline of the red corpuscles can be no longer seen: plasma-cells are scattered throughout, but are specially arranged in linear masses which are about to form capillaries, indeed, in the uppermost, circulation has already commenced, as shown by the blood within it.

white blood-corpuscles may in some instances have opened a way for the larger after-coming corpuscles. Subsequently the fissuring of the thrombus facilitates the permeation of the whole mass by the plasma-cells.

The points of contact of the coagulum and vessel wall are of infinite importance in the absorption and substitution process, as it is at these places that the plasma-cells are able to enter into and attack the foreign body.

To the naked eye the first indication of a cellular invasion of the clot is a change in colour from the original dark claret colour to chocolate. Sometimes chocolate striæ can be seen showing the parallel lines of the cellular invasion. Gradually, as the changes become more marked, the chocolate colour fades until the grey tint of connective tissue takes its place.

Cohn was the first to assert that the obliteration of a vessel after ligation was due to the proliferation of the endothelial lining of the intima. Lancereux, Förster Thiersch, Riedel, Auerbach, Raab, Pick, Waldeyer, Baumgarten, Cornil and Ranvier, and others, adopted these views. More lately Dent and Delépine, and W. Hunter, Arris and Gale Lecturer at the Royal College of Surgeons, have denied that the leucocyte possesses any fibroblastic power, and with these opinions, as has already been seen, our own observations agree.

We have followed the fibroblastic plasma-corpuscles as they throng down the channels of the clot towards the nodal points of the fibrin to form there cell-islets or giant cells, and have watched the manner in which they spread through all the substance of the foreign body, dividing it by the inter-islet columns into little portions or "clot districts." Every portion of the clot, leucocytes, red corpuscles, &c., seems to be looked upon ultimately by the plasma-corpuscles as alien material and treated accordingly. We think also that, as in the Ziegler chambers, the

invading leucocytes become the food of the more virile plasma-cells; and, too, we have often observed that while some of the original leucocytes of the clot gradually become less and less distinct in the red cell mass, others make their way (as in the chambers) to the surface of the coagulum, exhibit there signs of feeble vitality and soon fall a prey to the neighbouring plasma-corpuscles.

The process of mapping out the coagulum into "clot districts" is most advanced where the columns of cells, or where at a later stage the capillaries, pass from the wall into the clot. Near the ligature the process is almost invariably more advanced than away from it. Delépine, in opposition to Cornil and Ranvier, states that the most active changes in the clot occur in the region furthest removed from the ligature. This however is only true when the termination of the coagulum is adherent to the vessel wall.

The clot is always encapsuled very rapidly by cells from the intima, just as any other foreign body would be. The cells are spindle-shaped, but the fine adjustment shows that they are flat with one surface lying on the thrombus. If a proximal clot in a large artery extends for some distance from the point of ligature without any attachment to the intima, the cells covering the free end may be only one layer thick or there may be no covering of plasma-cells, the fury of the blood current being apparently sufficient to bar their advance over the free and mobile end of a long tape-like clot, rest being apparently essential for the action of plasma-cells on clot. (See chapter on Aneurism). Thus the clot furthest from the ligature may show no signs of absorption, while in that nearest to it

absorptive and constructive changes may be far advanced. The fact being that cell activity in and on the coagulum depends not on the nearness or distance from the seat of ligation, but upon the points of contact and vital attachment between the clot and the intima.

The cells surrounding the little "clot districts" multiply so that the area of each district is gradually encroached upon. Some of the plasma-cells invade the "districts," as was seen in the Ziegler chambers. The first change in the red cells is that their outline becomes obscure and very soon is quite lost, nothing remaining but an amorphous, yellow, coarsely or faintly granular, badly staining mass. The red corpuscles appear to undergo this change simply from proximity to or contact with the plasma-cells, as if a ferment were secreted by the latter which acts on the corpuscles, as does the neutral ferment in the food vacuole of Miss Greenwood's Amœba upon unprotected proteid.

That the plasma-cells do secrete a ferment which is the cause of the disintegration by contact of the red corpuscles is highly probable, and the following considerations appear to us to afford evidence that it is so.

(1) The clot, often recent, which is breaking down owing to an invasion of plasma-corpuscles, has a totally different appearance to the formless *débris* of cells which is seen in sections of uninvaded clot under a high power, say from a large and old aneurism.

(2) The red cells at the periphery of a "clot district" lose their outline before those at the centre—in other words, those nearest to the plasma-cells succumb to the ferment first. (Plate VII., Fig. 4.)

(3) It is hardly possible to conceive how absorption of

foreign bodies takes place unless it be by the action of some substance secreted by the cells. Take an encapsuled piece of tendon, for instance: it is surrounded by spindle-shaped cells, the surface of each spindle being applied to the foreign body which is being slowly dissolved.

The powers of cells while the healthy life of the organism is unimpaired may be said to be co-extensive with the duties they have to perform; and certainly a duty of plasma-corpuscles is under all circumstances to dissolve, if possible, a foreign body.

(4) All the known ferment-manufacturing cells within the body pour their secretions into the world outside themselves; and why should not plasma-cells do the same when imperatively called upon to cause solution of a foreign body, just as the gastric cells do when required to change an insoluble proteid into soluble peptone?

(5) Moreover, though we have been able to watch the digestion of blood-corpuscles in the food vacuoles of plasma-corpuscles, the enormous number of red cells in a clot make it almost impossible that they should all be taken into the food vacuoles of the invading cells. Such a process would be slow and tedious, and it is manifest from even a superficial examination of sections of invaded clot that a much more rapid process is going on: the red cell masses vanish and are replaced by an amorphous, granular yellow *débris*, and the conclusion is forced upon the observer that this effect is produced by the agency of the neighbouring plasma-cells.

(6) This change is not one due to the age of the clot, because, as has been already pointed out, it is quite different to that seen in very old uninvaded coagula; and

because we have frequently observed normal red cells in the central layers of an intravascular coagulum into which no plasma-cells had as yet found their way. These, though the oldest in the clot, were quite perfect in outline and general appearance, whilst the peripheral stratum or youngest portion of the clot had suffered an extensive invasion, was partly mapped out into districts, and the outline of the red cells had entirely disappeared with the advent of the phenomena of disintegration by contact or proximity.

(7) There are many analogies supporting this view of the action of plasma-corpuscles:—

The osteoclasts (plasma-cells) of Kölliker cause absorption of the walls of the primary areolæ, in the typical process of ossification, by means of a secretion from their surface (? a peptonising ferment), and in this way also the myeloplaxes of the marrow cause absorption of bone. The same thing also occurs in rarifying osteitis. In the Ziegler chamber experiments we observed that the proximity of a plasma-cell to a leucocyte distinctly diminished the activity of the leucocyte, and if continued the leucocyte became quiescent and showed evidence of segmentation of the nucleus. "Diminished vitality is "favourable for ingestion, sluggishness induced by dying "allows enclosure," and proximity to a plasma-cell alone had a peculiar disabling and devitalising effect upon the leucocyte. Miss Greenwood relates the escape of a flagellate Infusorian after contact with an Actinosphærium for one hour, and adds that its movements were less rapid than usual. She also says that quiescence on the part of a motile prey may occur previous to determinate ingestion, both facts which would apparently indicate an influence of

the animal cell on the world outside itself. Carl Hess has figured pathogenic bacteria quiescent or dead in contact with (not within) the tissue-cells of liver and spleen, and if we pass from the protozoa to the protophyta the great group of pathogenic bacteria all owe the effects they produce not to their own dynamic activities but to the specific chemical poisons and ferments which they elaborate from the surrounding lymph when they gain access to the body. The two following phenomena, often witnessed in the laboratory, exhibit the peculiar influence exerted by the lowest forms of vegetable life on the world around them; and is it not likely that if such an influence is possessed by the unicellular forms of plant life it should be present also in the lowest forms of animal life? (A) Organisms grown in colonies on coloured gelatine plates decolourise the jelly further than either growth or liquefaction extends. (B) The colonies in plate cultivations are always larger if they are well separated, as they appear to produce something which is inimical to the growth of neighbouring colonies. Watson Cheyne, in his recent work on *Suppuration and Septic Diseases* (1889), supports this view. He says, "It may be that the micro-organisms are killed by "chemical substances given off by the leucocytes or the "tissues and not by inclusion and ingestion in the cells."

(8) The recent experiments of Buchner, proving that serum possesses the power of destroying micro-organisms, seem to point in the same direction, for the active constituent of the serum is no doubt derived from tissue-cells. Other observers also agree. A summary of the work that has recently been done on this subject has been published by Kanthack.

Such then are briefly the reasons which have induced us to attribute the disintegration of each "clot district" to the permeation through it of a ferment thrown off by the surrounding plasma-corpuscles. The fluid products of this disintegration stream off into the lymphatics, while much of the solid doubtless is ingested by the plasma-corpuscles or further acted upon by the fluids which induced the initial change in the clot. The blood pigment is taken up by the cells and much of it is carried to the nearest lymphatic glands. (Fig. 69.)

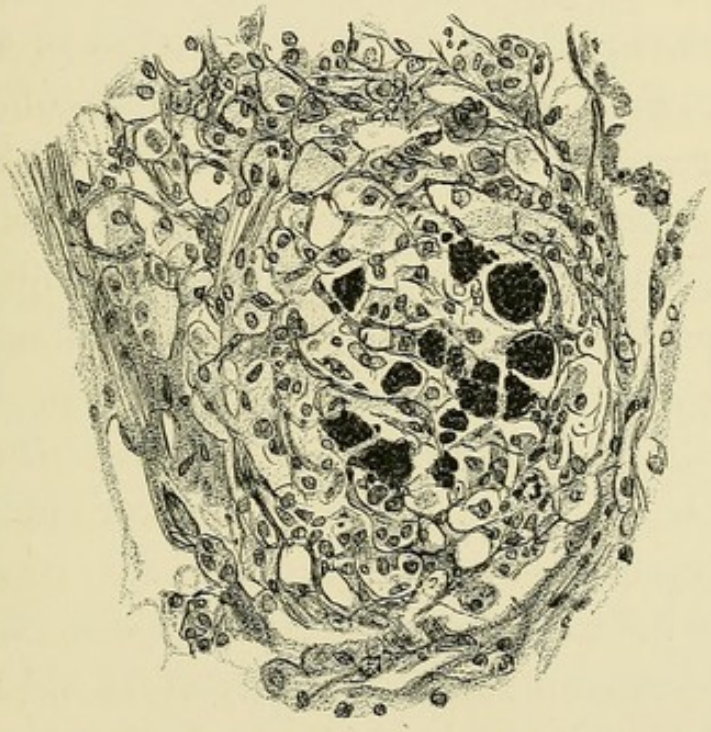


FIG. 69. Lymphatic Gland from the neighbourhood of a cured Popliteal Aneurism (× 300).

The gland on section was of a rusty colour; under the microscope patches of dark amorphous blood pigment were seen: some of this pigment was contained in the cells, while some was free in the loculi.

The case was under the care of Mr Wm. Anderson, and we are indebted for the specimen to Mr Shattock.

The connective tissue corpuscles round a "clot district" remain active as long as food is abundant, that is to say,

as long as there is yet any part of the little clot mass still unabsorbed. When this period is passed, the cells elongate and there commences that process of spinning fibres which ultimately transforms the "district" into connective tissue.

Vascularization of the Clot.

As soon as the plasma-cells have ramified through the clot, mapping it out into well-defined and equal areas, the need for capillary development becomes urgent. Heuking and Thoma state "that all capillary formation in the "thrombus is by vacuolation and germination as in the "embryo; so that the pathological process of vascularization of clot finds a parallel in the domain of physiology, "and the results of physiological and pathological examinations are confirmatory one of the other." The process originally described by Thiersch and mentioned by Paget and Billroth is that the primitive blood channels of the clot are formed by the lining of the cracks and fissures of the clot by plasma-corpuscles. (Fig. 70.) It appears to be doubtful, however, whether this method of capillary development ever occurs. All recent writers describe the hollowing out of the connective tissue-cells to form blood-vessels as in the embryo. If this be always true, a cell blocking one of the paths in the clot would divide into two parts, each of which would limit opposite sides of the primitive capillary. At the point of meeting of several capillaries (? nodal points) more or less irregular spaces are formed which communicate with these capillaries. It does not seem to us, from the views expressed with regard to the determinate hereditary tendency of cells *a priori* difficult to imagine the daughter cells of the endo-

thelial elements lining the cavity of a ligated artery, adjusting themselves against the boundary of a crack in the clot, thus forming a capillary without vacuolation which results in a part of each cell occupying opposite sides of the primitive capillary.

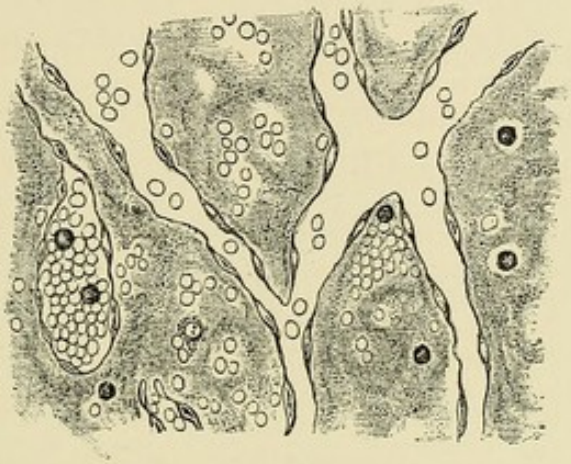


FIG. 70. The newly-formed Capillaries in a Thrombus (× 310).

Reproduced, by kind permission of Prof. Thoma, from Virchow's Archiv, Bd. CIX. (1887).

Spindle-shaped plasma-cells encapsule each district of the clot, and at the same time form a plate of cells which bound the channels through which the circulation of the blood is first established. The red corpuscles are no longer recognisable in the districts of the clot, their place being occupied by a granular material.

The solid cylinder of cells between the districts of the clot begin to vacuolate and form channels which communicate with each other, and with the capillary loops which have been developed by germination and which can be observed passing down through the middle and inner tunics to the points at which the clot and intima are in permanent connection. On the other hand, columns of cells reach the cracks and fissures at points of the clot not in contact with the intima. These also show after a time the same hollowing out; and circulation through the plugs

of young connective tissue becomes possible. In this way the capillaries of the clot communicate with the lumen of the obstructed artery. (Fig. 71.) The current of blood, as

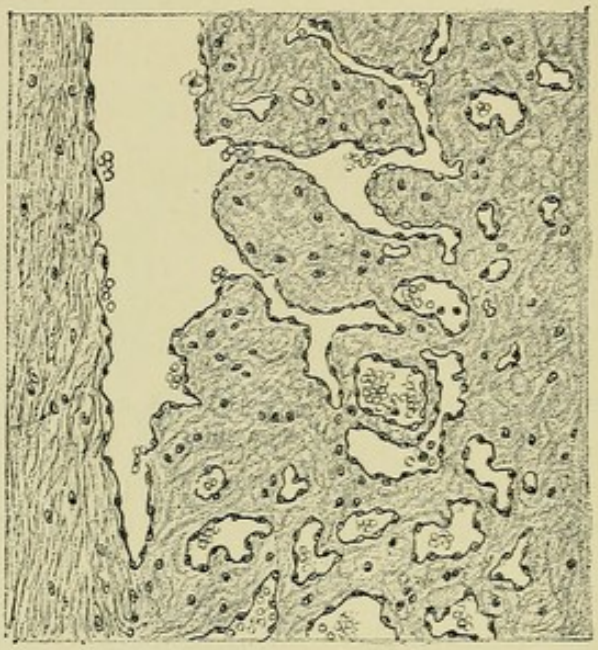


FIG. 71. Thrombosed external Jugular Vein of Man (× 140).

Reproduced, by kind permission of Prof. Thoma, from Virchow's Archiv, Bd. CIX. (1887). (*Slightly modified.*)

To the left is seen the wall of the vessel with its intima much thickened. To the right a coagulum is shown in which the absorption and substitution process is going on. Several capillaries and spaces in the clot lined by plasma-cells derived from the endothelium are seen. These all communicate with each other (see text), with the lumen of the vessel, and with the newly-formed capillaries entering the clot from the wall of the vessel.

soon as the network of channels is complete, will be from the proximal lumen of the artery and out through the venules which pass from the coagulum into the vessel wall. There may also be a current of blood passing into the clot from the arterial capillaries developed from the vasa vasorum; and the venules corresponding to these arterial capillaries may be either separate or identical with those which carry away the blood which enters the coagulum

from the lumen of the vessel. Heuking and Thoma, O. Weber, and Kocher, hold that these two systems of capillaries only inosculate with each other and are otherwise independent. In the fusiform swelling external to the ligature numerous vessels are developed, and these are in connection with capillaries which traverse the wall and clot on the proximal and distal sides of the ligature.

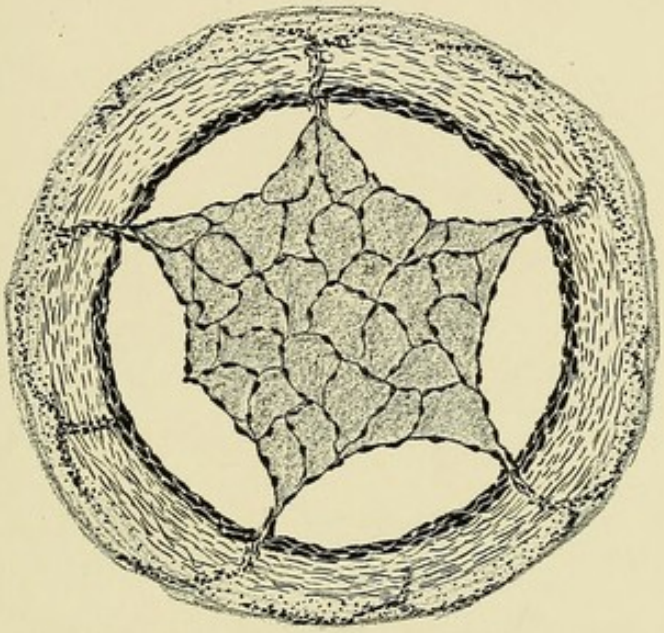


FIG. 72.

Diagram to show the attachment of the central clot to the wall of the vessel, and the peripheral reopening of the lumen of the vessel by the blood current: the clot is star-shaped and vessels are entering it at its points of attachment; one of these has been recently broken by the stream of blood: the clot is represented mapped out into districts and its surface covered with plasma-cells: the clear space between the wall and the clot is where the fluid blood penetrates.

The direction of the blood current in the newly-formed capillaries which open into the lumen of the artery will be reversed in the case of a thrombus in a vein; but otherwise the phenomena of vascularization of clot in arteries and in veins are identical.

The capillary development increases, is stationary, or withers in accord with the physiological needs, first of clot absorption and secondly of the mature state of cicatrisation.

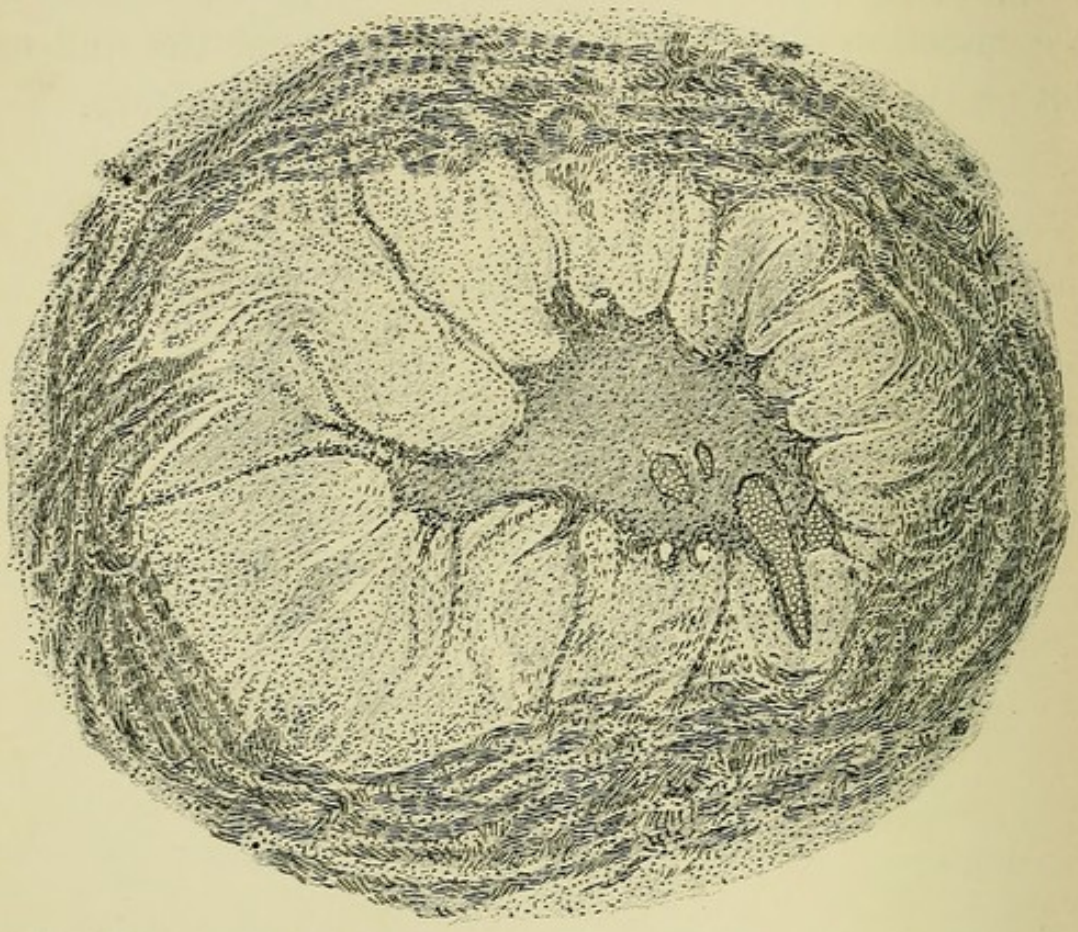


FIG. 73. Artery from Amputation Stump (× 50).

From a specimen kindly lent by Dr Sharkey.

Five months after operation. The first clot is seen of a darker colour and irregular shape in the centre of the vessel: it is attached to the vessel wall by a few capillaries which indicate the original points of attachment of the clot to the intima: external to the original clot is more recent clot, of varying age and varying stages of substitution: three spaces occupied during life by circulating blood are to be seen: coats of vessel indefinable and changing into connective tissue.

Three stages of vascularization of clot have been described: (1) mechanical vacuolation, which corresponds to the cracks and fissures already mentioned; (2) potential

vascularization, which is the stage in which solid columns of plasma-cells fill the cracks and fissures and surround the clot districts; and (3) true vascularization, which follows the vacuolation process by which true blood channels are developed.

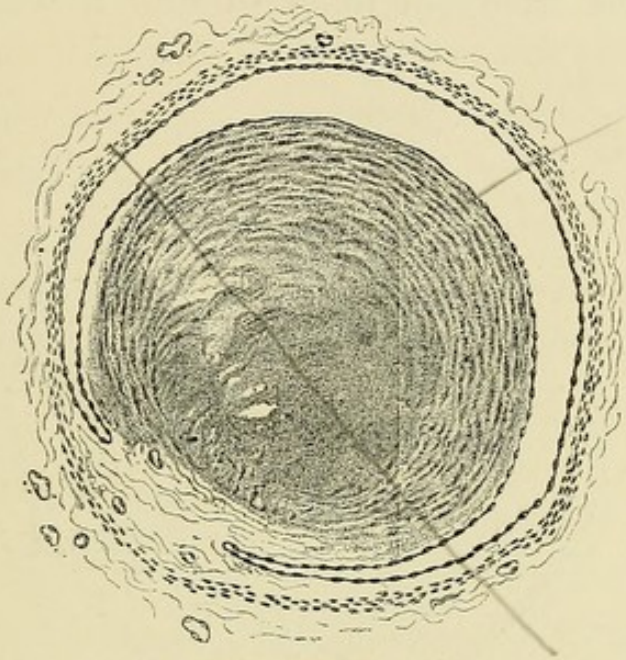


FIG. 74. Thrombosed Femoral Vein of Man (× 9).

Reproduced, by kind permission of Prof. Thoma, from Virchow's Archiv, Bd. CIX. (1887).

The thrombus is attached at one point only to the vein. It is surrounded by cells derived from and continuous with the endothelium. By the shrinking of the coagulum the vessel is patent. The lumen is crescent-shaped and the fluid blood is at all points limited by endothelial cells. The concentric deposition of the clot is illustrated.

True circulation through the clot does not apparently take place before the 20th day. The end of the third week and the commencement of the fourth week being the time, according to our observations, at which blood appears in newly-formed capillaries in large intravascular clots. Ziegler, in his original papers, did not find that vacuolation occurred before the 18th day, but W. Hunter observed

it in small clots on the 10th day. The time at which true blood circulation occurs through a clot or foreign body, which is a dead thing and has to be replaced bit by bit by living tissue-cells, is quite different to the time at which capillaries appear in actively living structures, as for instance in the fusiform mass of new material which so

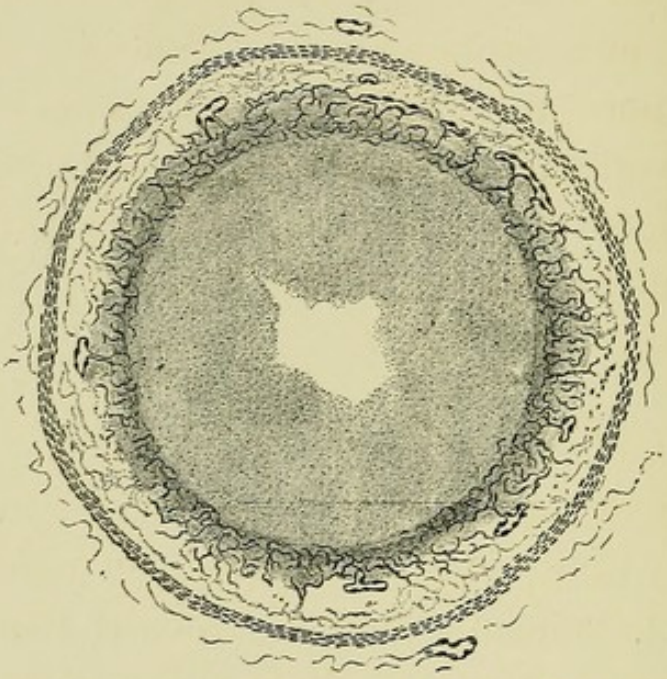


FIG. 75. Thrombosed Femoral Vein of Man (× 11).

Reproduced, by kind permission of Prof. Thoma, from Virchow's Archiv, Bd. CIX. (1887).

The central portion of the thrombus has dropped out. The peripheral portion is attached by its whole circumference to the intima, which is highly vascular. The absorption and substitution of the clot by connective tissue-cells is progressing circumferentially and not centrally, which is the usual course of events when the coagulum is only attached at a few places to the vessel wall. Compare fig. 73.

rapidly collects external to and around the deligated point of an artery. In this living mass of cells capillaries commence to form after the third day and may be injected as an extensive network on the fifth or sixth day.

If the clot be only in contact at one or a few points of the intimal wall the capillaries will pass into the clot at

these places, and join by oft-repeated anastomoses in the central portion of the coagulum. (Figs. 72, 73, 74.) If however the clot is adherent by its whole circumference (which rarely happens) the intima becomes full of capillaries, which pass down from the media and streaming through the intima enter the clot at short intervals and in almost parallel lines. (Fig. 75.) In this case the circumference of the clot becomes sooner absorbed and vascularized than the central portion. This is an exceptional event in veins, and it is a question whether it ever happens in arteries except close to a deligated point, as the pressure of the blood-stream in arteries is always tending to lift the clot away from the vessel wall.

Extent of Artery Obliterated.

The extent of artery which becomes temporarily or permanently obliterated varies with the force of the blood current, the arrangement of branches and the freedom of the collateral circulation. It is much more extensive in veins than in arteries, as in veins the chances of disturbance of the plastic processes are reduced to a minimum. It is well known that considerable lengths of venous trunks become permanently blocked. Similarly, long portions of small arteries may be occluded, but the large trunks with which we are now concerned are each differently circumstanced, and the amount of obliteration varies accordingly. The distal section of an artery conforms to the same laws as the proximal, and does not appear more prone to be transformed into fibrous tissue than the proximal.

Of all large arteries, the external iliac seems to be subject to obliteration to a greater and more uniform

extent than others. Clot fills the artery at first as high as the bifurcation of the common iliac, and as low as the giving off of the deep external circumflex and deep epigastric arteries, or even beyond this point. (See two figs. in Chapter XVIII.) Permanent obliteration of the whole length of the artery often follows. (See Hunterian Museum, No. 3082.) In this specimen the external iliac is seen to be changed into a thick fibrous cord. When the artery is divided between two ligatures, the ultimate condition seems to be, in cases of survival, that the two ends are united by a narrow, fibrous thread (Fig. 76). If the subclavian artery be examined after ligature, it will seldom be found to be obliterated for more than half-an-inch or one inch: (12—24 mm.). (See Barwell's two cases in the Hunterian Museum, Nos. 3168 and 3097, one of which is figured on page 248.) In Heath's case of simultaneous ligature of the right subclavian and right common carotid arteries (see Hunterian Museum, No. 3097), in which the patient survived four years, the subclavian artery is obliterated for a distance of one inch (24 mm.). The short length of the subclavian artery, which is permanently blocked after ligature, doubtless depends upon the number and importance of the branches which the vessel gives off.

The common carotid artery is seldom occluded for a longer distance than the subclavian. On the proximal side, the great expansile force of the blood current must be taken into account; and the distal extremity of the vessel is exposed to a moving stream of blood dependent upon the free anastomosis between the branches of the internal and external carotid arteries of opposite sides. However, while obliteration to a considerable length does

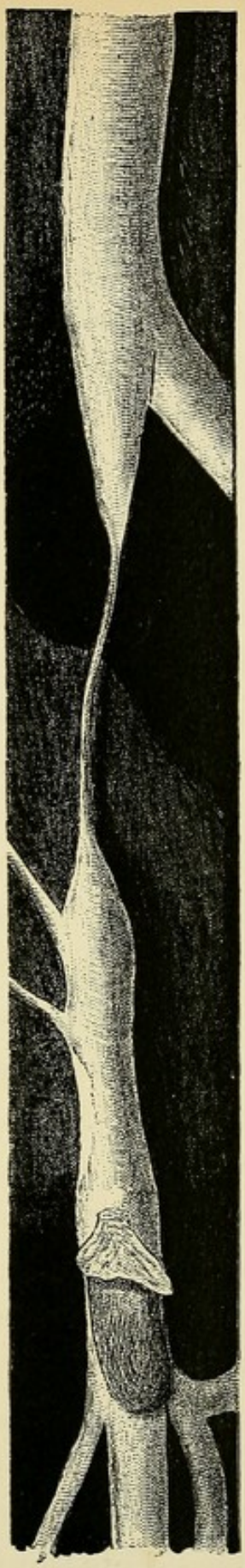


FIG. 76. Ligature of the External Iliac Artery by Sir Astley Cooper.

The artery was tied in two places and divided between: a fine cord connects the upper and lower portions of the artery. Patient survived operation 18½ years. For further details see description of figure (156) in Chapter XIII.

not commonly occur, permanent diminution of calibre below and above the deligated point may take place. Moreover, it is important to remember, as Astley Cooper says, that the obliteration of the trunk of a great artery above and beyond a ligature, is a very gradual process. Sometimes clotting may take place on the cardiac side of the ligature for considerable distances, but the violence of the current sweeps away all its connections with the arterial wall, except where it is in juxtaposition with the plastic processes at the seat of ligation. Such a specimen can be seen in the Hunterian Museum, No. 3167 (Fig. 77). A tape-like clot extends in the left carotid from the point of ligature to the aorta, and is adherent at no place, except at the point of ligation. These long, tape-like tongues of clot are not at all uncommon in large arteries after amputation, often extending with no peripheral attachment for considerable distances. Such a clot can be seen in Guy's Museum, No. 1506[65], which shows a femoral artery from a stump nine days after amputation.

The amount of proximal clotting which happens in the common carotid after ligation is of much importance, as Holmes has endeavoured to explain, the local improvement which undoubtedly has taken place in some cases of aneurism after distal ligature, by the extension of the coagulation process backwards in the artery until the tumour is reached, when the blood in it also clots.

Savory, in a communication to the Royal Medical and Chirurgical Society, has pointed out that after ligature of the superficial femoral for popliteal aneurism, the artery intervening between the ligature and the aneurism is in

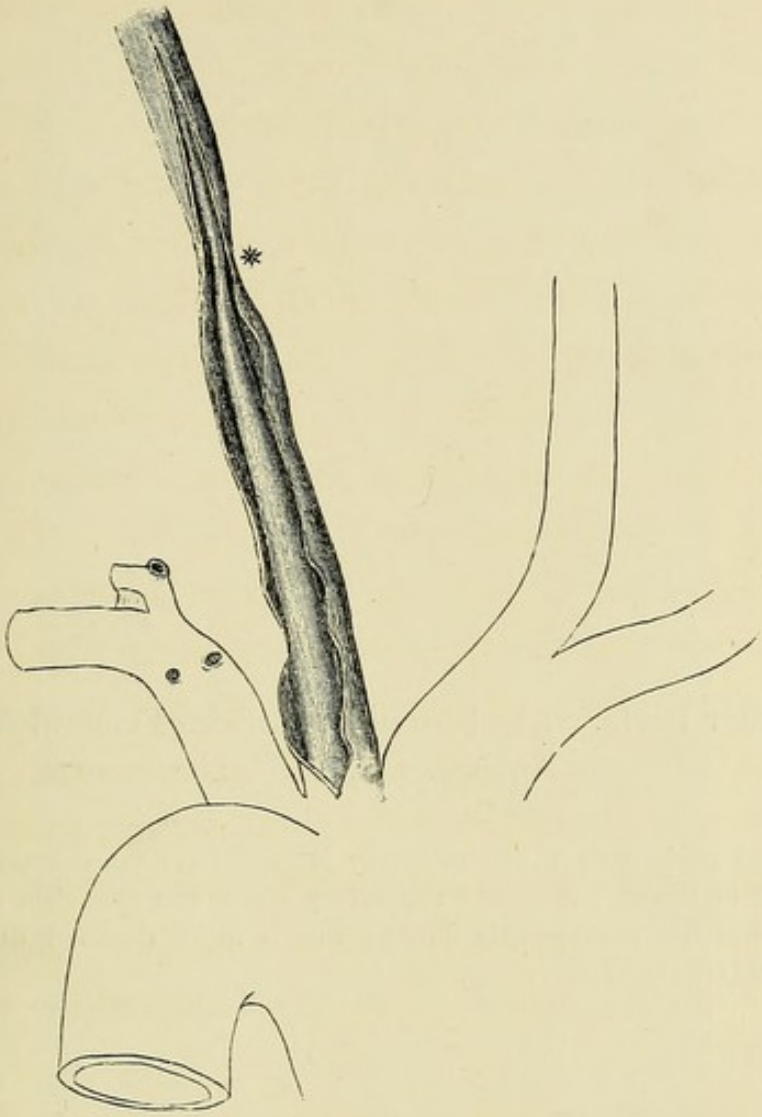


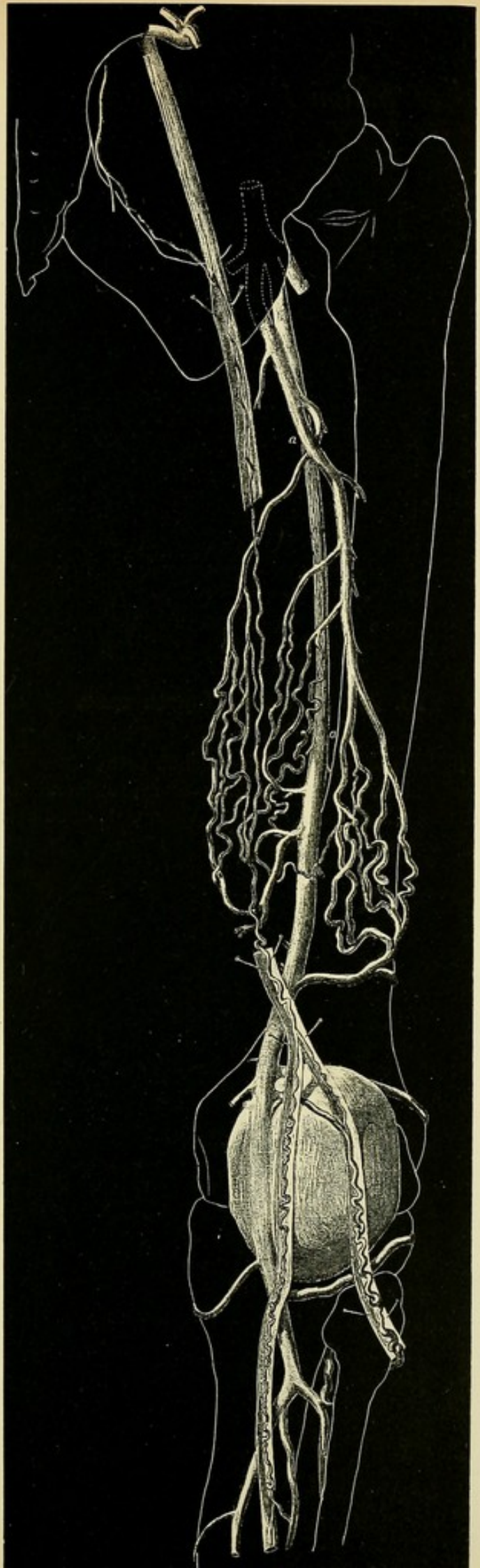
FIG. 77. Ligatured Left Common Carotid Artery.
($\frac{2}{3}$ size of specimen.)

The vessels are viewed from behind: the innominate to the right, and the descending part of the arch of the aorta to the left. The left common carotid artery was ligatured with carbolic catgut four years before death by Mr C. Heath: a clot extends from the point of ligature, indicated by a star, to the aorta: it is only adherent at the seat of ligature: the coats of the vessel appear to be uninjured, and its lumen was not completely occluded at the operation, nevertheless the circulation was permanently arrested: this was probably an example of the giving way of the first hitch of the reef-knot while the second half was being tied. All trace of the ligature has disappeared.

The specimen is in the Hunterian Museum, No. 3167. See also *Trans. Clin Soc.* Vol. v. (1872) and Vol. x. (1877).

FIG. 78. After Porta. Ligature of Superficial Femoral for Popliteal Aneurism by Miloni.

Only a short portion (*a a*) of the artery is occluded: the collateral circulation round the seat of ligature re-enters the artery above the aneurism so freely that the aneurism was not permanently cured. For a more detailed description see the same figure (159) in Chapter XIII.



13—2

FIG. 79. Hunter's fourth case.

Patient survived ligature of superficial femoral 50 years. The whole length (*a a*) of the superficial femoral and popliteal arteries are obliterated. The popliteal aneurism is reduced to a small fibrous mass. A more detailed description will be found under same figure (160) in Chapter XIII.

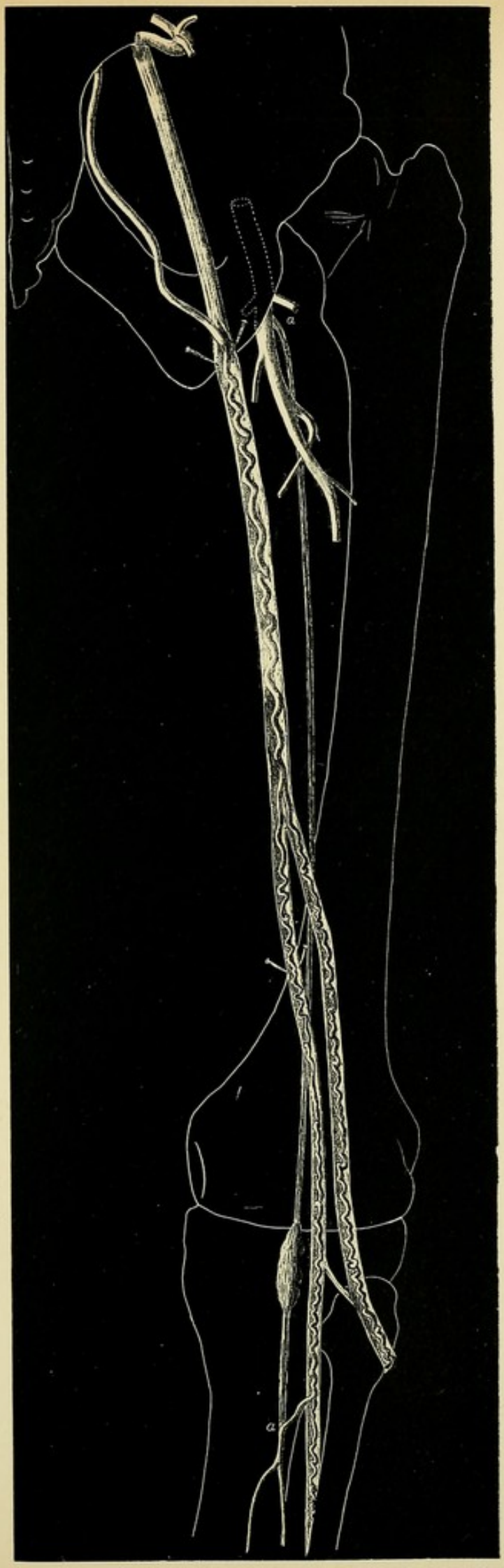
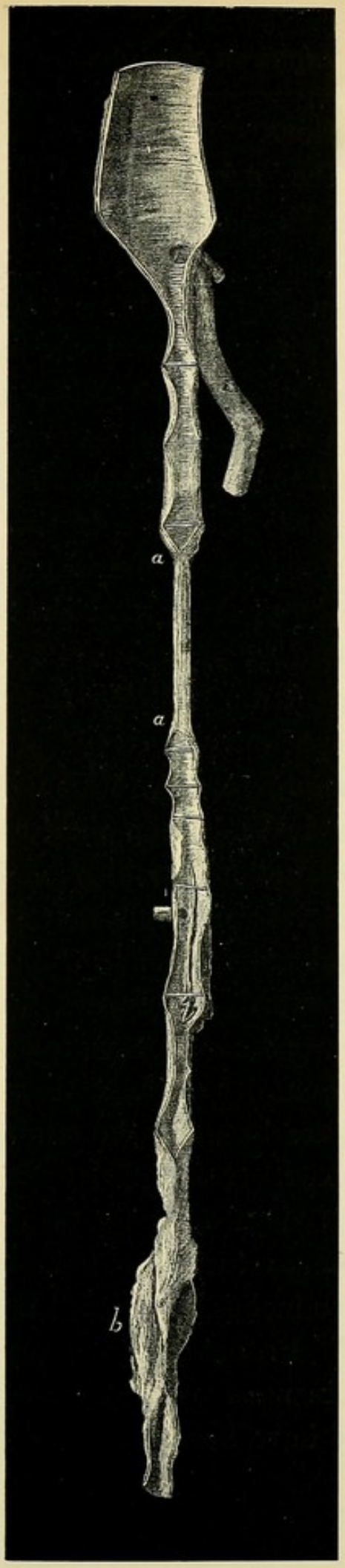


FIG. 80. Ligature of Femoral.

Stanley's case of ligature of superficial femoral twenty years before death: (*a a*) length of artery obliterated, (*b*) remains of aneurism. For further description see the same figure (161) in Chapter XIII.


a
a
b

most cases pervious. The vessel was found, in 15 cases in which the aneurism was cured, pervious to some extent at this part in all, and completely so in eleven.

In Porta's case (Fig. 78) the common amount of permanent closure of the superficial femoral after ligation is seen. Obstruction of the artery does not occur upwards beyond the origin of the deep femoral. Hunter's case is exceptional; for the main artery of the limb is obliterated from the origin of the profunda to the bifurcation of the popliteal. (Fig. 79.) In Stanley's patient (Fig. 80), the superficial femoral was changed at the site of ligation into a thick fibrous band for $1\frac{3}{4}$ inches (4·5 cm.), but the artery is patent for some distance below the profunda. On the other hand, in specimen No. 1407 in St Bartholomew's Museum, the femoral artery, which had been ligatured 18 months before death, is seen obstructed for four inches (10 cm.), the obstruction reaching as high as the origin of the profunda. It is probable that after ligature of the superficial femoral artery, much of the distal clot which forms at first, afterwards disappears, the free anastomosis, as shewn in the drawing of Porta's case, allowing the circulation of a large quantity of blood through that section of the artery which lies between the site of ligation and the aneurism. Specimen 1519[32] in Guy's Museum shews a femoral artery 11 days after ligature, and in it a distal clot, extending many inches, can be seen, to within—in fact—two or three inches (5—8 cm.) of the aneurism and then it tails off. Probably, if the patient had survived, this would have in most part ultimately disappeared and the artery would have become, in accordance with what

usually happens, more or less patent again between the site of ligature and the aneurism.

As far as the cure of aneurism is concerned, the important point is the permanent occlusion of the artery opposite the aneurism: if this be occluded the extent of artery obliterated above or below is immaterial. This question and its practical bearings on the ligature of the femoral and other arteries will be found discussed in the chapter on Aneurism.

Re-establishment of the Circulation.

If the ligature does not occlude the artery at the time of the operation, or if the knot of the ligature gives way shortly after ligation, or if the material of the ligature be such that it is rapidly softened and absorbed by the living tissue around, the circulation through the artery may be re-established. This applies to all cases in which the tunics are not ruptured, and also to some in which the damage done implicates the whole thickness of the middle coat. Both the internal coats were ruptured by James Adams, in a case of ligature of the common carotid: the artery became again patent and a diaphragm with a central opening was found when the patient died, 108 days later. (Fig. 86.) This result however does not necessarily follow: in some of our ligation experiments obliteration occurred permanently, though the intimæ of opposite sides were not quite in juxtaposition.

In the chapter on the Choice of the Knot, it is pointed out how difficult it is to occlude a great artery by means of the ordinary ligatures and knots when the coats are not

ruptured; and also the way in which the difficulty may be overcome.

In our first experiments we found that the greater size of and the increased blood-pressure in the common carotid, when the opposite one had been tied some time previously, added to the uncertainty which attended the attempt to completely close the lumen of the artery: but here again the employment of the "stay-knot" met the difficulty and obviated all uncertainty as to the complete closure of the artery by the non-rupturing ligature.

Too much stress can hardly be laid upon this point, which is one of great practical importance. We have witnessed the deligation of two superficial femoral arteries for aneurism, in both of which this event happened. In one we distinctly saw the first half of the reef give way before the second half was tied. In both instances silk was the material of ligature: in both, on the morning following the operation, pulsation was noticed in the aneurism, and the blood could be heard whizzing through the constricted part of the vessel.

Some of the older surgeons held that a ligature loosely applied to an artery produced inflammation of the vessel wall and clotting in its interior; but this could only happen in septic wounds in which a septic ligature had to make its way out through the arterial wall.

When the ligature and wound is aseptic and the lumen of the vessel is only slightly narrowed, no coagulum forms, as in the cases above related. In such instances, one can hardly speak of the re-establishment of the circulation, for it was only momentarily interfered with and continued subsequently with a diminished stream.

Re-establishment of the circulation may take place, too, after a clot has formed, but the process by which this occurs is by no means simple.

On making a transverse section of an artery in which a clot is being replaced by living tissue, it will be seen that the central portion is irregular in outline, star-shaped in fact, in consequence of its attachment at more or less regular intervals by capillaries to the intima (Fig. 73, page 187), but if the clot be free, its transverse section is circular in form. The central mass is mapped out into "clot districts" by the cellular invasion. Between the points of attachment are spaces, bounded internally by clot covered with endothelial cells, and externally by the intima. These are true blood channels, lined by endothelial cells. They may be blocked at a later date by proliferation of the surrounding plasma-cells, or further clotting may ensue; or, if the force of the blood-current suffices, they may be all thrown into one by the breaking of the young and slender fibrous connections between the wall of the vessel and the central clot. In this manner, a peripheral re-opening of the lumen of the artery (not a canalisation of the clot) may occur as far as the place of ligation. If these bands give way, a long tapering clot is left free in the blood-current, sometimes for a considerable distance, being attached only at the point of ligation. Such a tape-like coagulum is shewn in Figure 77, page 193, and is often to be seen in the large arteries of amputation stumps.

The vessel may become pervious in another way,—by an opening forming down the centre of the clot. The central clot above described is mapped out into "clot districts," and if development of connective tissue does not

proceed in an effective manner, the cells, or granular material of which the central part of each district is made up, may be exposed to the full force of the blood-current and washed away. The "districts" of the clot may be thus violently transformed into lacunæ filled with moving blood. This phenomenon may happen in one of three ways: before the endothelium has plugged or encapsuled every part of a long and unattached coagulum; or by rupture of a capillary leading from the lumen of the vessel into the clot; or by breaking off of the end of the clot, or other small damage to its surface. Thus, all the districts of the coagulum may be gradually changed into blood-lacunæ, and the circulation takes place through a kind of cribriform or sieve-like membrane, which, looked at in longitudinal section, has often the appearance of ropes of fibrous tissue crossing the lumen of the blood-vessel and being free in the blood-stream. (Fig. 81.)

In the near neighbourhood of the ligature, in consequence of the great adhesion here of the clot to the intima, the connective tissue development is always more advanced at the periphery of the clot than at the centre, so that the central districts of the coagulum are the first to give way and allow the blood-current to circulate in them. In this way a true central "canalisation of the clot" occurs, for, at the site of ligature, the peripheral portion of the coagulum is always the last to be swept away. The entrance of the blood-current into the clot districts is speedily followed by rupture of the delicate fibrous network which surrounds them—partly owing to the violence of the current, partly in consequence of degeneration due to nutrition failure, and in part to the

FIG. 81. Longitudinal section of the Carotid Artery of an Ass 69 days after ligature with a large kangaroo tendon tied in a reef knot (× 26).

The coats were unruptured and not in apposition, but the vessel is completely occluded for some distance by fibrous tissue. The drawing is made from a part some distance from the ligature: the intima is much thickened, and extending from its inner surface are seen bands of connective tissue; these are in reality sections of diaphragms, some complete, some perforate, which stretch across the lumen of the artery.

The specimen is an example of the partial giving way of the first half of the reef-knot from the pressure of the blood before the second half of the knot was finished: nevertheless in this case the vessel was occluded.

fact that blood can again dilate the artery in the portion filled with clot—and thus place an excessive strain upon the delicate fibrous trabeculæ. When the canalisation has reached the ligatured part of the artery, if the lumen of the artery be not entirely obstructed, one of two things may happen: either a diaphragm of fibrous tissue which has already formed across the lumen successfully resists the blood impulse, or the canalisation process continues and again opens the cavity of the vessel at the point of ligation. (Fig. 82.)

The time at which canalisation occurred and was completed, in our experiments, appeared to be from the 50th to the 70th day, though no doubt this period would vary with the vessel ligatured, the amount of constriction of the wall, and the extent of the original coagulum.

True canalisation seems to occur always after the lapse of some weeks, and supposing that the operation was performed for the cure of aneurism, the re-establishment of the circulation would happily be too late to interfere with the cure of the disease as far, that is, as that is dependent upon the stoppage of the blood-current through the vessel tied.

In some cases when circulation recurs a diaphragm is developed, and the volume of blood passing along the vessel never attains to its former dimensions. In other cases, the calibre of the vessel is completely re-established, as in specimen No. 3168 in the Hunterian Museum, which shows a common carotid, patent and undiminished in size, after ligature with ox aorta. (Fig. 104.) This is probably to be explained, in part, by the giving way of the first half of the reef, and in part by the rapid softening and

absorption of ox aorta. Again, in Guy's Museum there is a specimen in which the great artery of the lower limb is seen obstructed by the pressure of a psoas abscess. The specimen shows beautifully the phenomenon of the central canalisation of clot. (Fig. 82.)

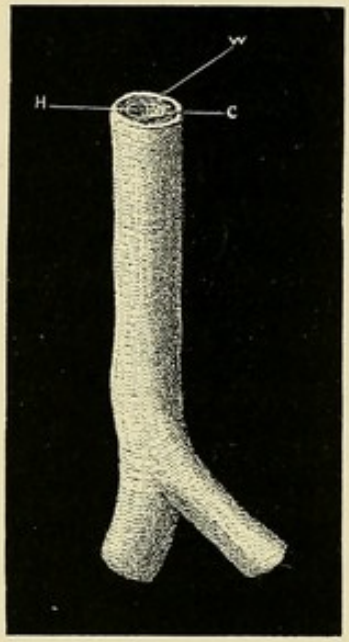


FIG. 82. Popliteal Artery, with Canalised Clot. (Nat. size.)

From male aged 23. The thrombosis was attributed to the pressure of an abscess, higher up.

(*W*) wall of vessel: (*C*) peripheral portion of clot in which organization is taking place: (*H*) central canal: the organization in the clot being in this instance more advanced in the periphery, the central part more readily yielded to the blood, and thus canalization took place.

(Guy's Hospital Museum, 1519[55].)

Many cases of re-establishment of the circulation after ligature are on record, among them may be mentioned Barwell's ligature of the common carotid with ox aorta, already alluded to, and Porcher's and James Adams's cases relating to the same artery. In Porcher's case, the ligature catgut was loose on the 7th day, on which day the patient died. In Adams's case, the artery was probably

obstructed for some weeks. Heath and Thomas Smith have related cases of catgut ligature of the femoral artery, which have been followed by a re-establishment of the circulation. Rivington had a similar experience, one month after ligature of the external iliac, so had Elliott with the same artery, and Thomas Smith found that the first half of a tendon ligature used by him around the common iliac gave way and allowed of renewed pulsation in the aneurism: we have already referred to two cases in which the femoral artery was ligatured but not occluded at the time of operation. Some of the above cases come probably under the heading of re-opening of the artery after some weeks by canalisation of the clot, in consequence of some defect, either in the material of the ligature, or in its mode of application, whereby the lumen of the artery was not completely closed at the time of the operation. In a few instances, doubtless, no clot formed at all, the ligature when the knot was completed insufficiently constricting the artery, though as long as the first half of the reef was being actually pulled upon the artery might have been occluded and even the coats ruptured. When carbolic catgut was in use there may have been another category of cases, namely, those in which the ligature was broken down, loosened, or absorbed in from two to three days. This is decidedly too short a time for the tunics to be held in contact, whether they be lacerated or not. Scarpa long ago showed that when the coats are not ruptured they must be supported by the ligature, if that be broad, for four days, if it be narrow this period must be increased; Paul Bruns, on the other hand, with his temporary ligature and with rupture of the coats, maintains that

70 hours is sufficient. Much, however, must depend upon the size of the artery to which a ligature is to be applied and something on its proximity to the heart. An aseptic ligature, on the other hand, applied by means of the stay knot, which holds the undamaged coats in contact and lasting as does kangaroo tendon for some weeks, makes both hemorrhage and the re-opening of the artery to the current of blood impossible.

Diaphragms.

In some ligatured arteries the calibre is seen after death to be constricted or occluded at the seat of ligature by a circular ridge of tissue constituting a diaphragm: it is probable that in these cases the walls of the artery were not held in apposition by the ligature at the time of operation, but the same result would possibly be produced by a catgut ligature (which had or had not ruptured the coats) becoming too rapidly absorbed. These diaphragms were found in several of the carotids of sheep and asses which we ligatured, and in these we think, for reasons given elsewhere, that the ligature was not tied tight enough to occlude the lumen of the vessel.

The following table shews the cases: in all we think it probable that a clot formed for a longer or shorter period. Every stage of diaphragm development can be followed in the specimens, from a slight fibrous thickening of the intima at the point of ligation up to a complete membrane bridging across the lumen of the artery.

1.	Carotid of Sheep	44 days after ligation			(exp. 16)	Slight fibrous thickening of intima.
2.	„	„	58	„	„	(exp. 5)	
3.	„	„	73	„	„	(exp. 6)	Thickening of intima more evident and commencing formation of a ridge or diaphragm.
4.	Carotid of Ass		77	„	„	(exp. 36)	Diaphragm with central opening. (See Fig. 88.)
5.	„	„	76	„	„	(exp. 35)	
6.	„	„	85	„	„	(exp. 41)	Imperforate membrane across lumen of vessel.
7.	„	„	79	„	„	(exp. 39)	
8.	„	„	64 days after ligation			(exp. 27)	Distinct diaphragms which are imperforate; and one ridge (or diaphragm with large opening) at some distance from ligature.
9.	„	„	42	„	„	(exp. 24)	Succession of thin membranes across lumen, some complete (?) and some cribriform.
10.	„	„	42	„	„	(exp. 25)	

In all of these we believe the coats of the artery were uninjured, but in some of the cases in which diaphragms occur the coats are ruptured by the ligature and then allowed to separate again by the first half of the knot slipping. These latter cases would be very similar to those in which a ligature is applied so as to rupture the coats and then at once removed: experiments of this nature were made by J. F. D. Jones, and in one a dia-

phragm with a central perforation was found (Fig. 83): in another, in which four ligatures were applied, the vessel was occluded. (Fig. 84.)

It is difficult to offer anatomical proof that when a diaphragm forms there has not been the slightest injury to the intima. If for many weeks a vessel be constricted though not occluded, by a ligature, a proliferation of the underlying intima will surely take place, and if the irritation be continued for some time it might well be that in the end a permanent ridge of connective tissue or a diaphragm would be formed. In Adams's case the artery is very weak at the point of ligature and allows of considerable angular bending. This is owing to the almost complete division of the artery by the ligature. If, however, a diaphragm be developed when the coats are unruptured, or when the inner tunic is only superficially injured, no bending at the seat of ligature is allowed: the strength and integrity of the arterial wall is not impaired. As is shewn elsewhere, the injury to the arterial wall varies greatly from a simple dislocation of the endothelium or a rupture of the elastic lamina of Henle to complete division of the two inner tunics, or of the whole arterial wall. We believe in our experiments the intima was undamaged, and if it were so, a diaphragm can be produced quite independently of gross injury to the coats of the vessel. Generally speaking, when a diaphragm forms the outer contour of the dead artery is unaltered or only slightly so; though in our specimens we think a constriction might well have been visible if they had been examined distended to their natural living size.

The discovery of diaphragms and of pervious arteries

after ligature of several ass carotids, when we knew that at the time of the operations the vessels had all been occluded by the first hitches of the reef or surgical knots

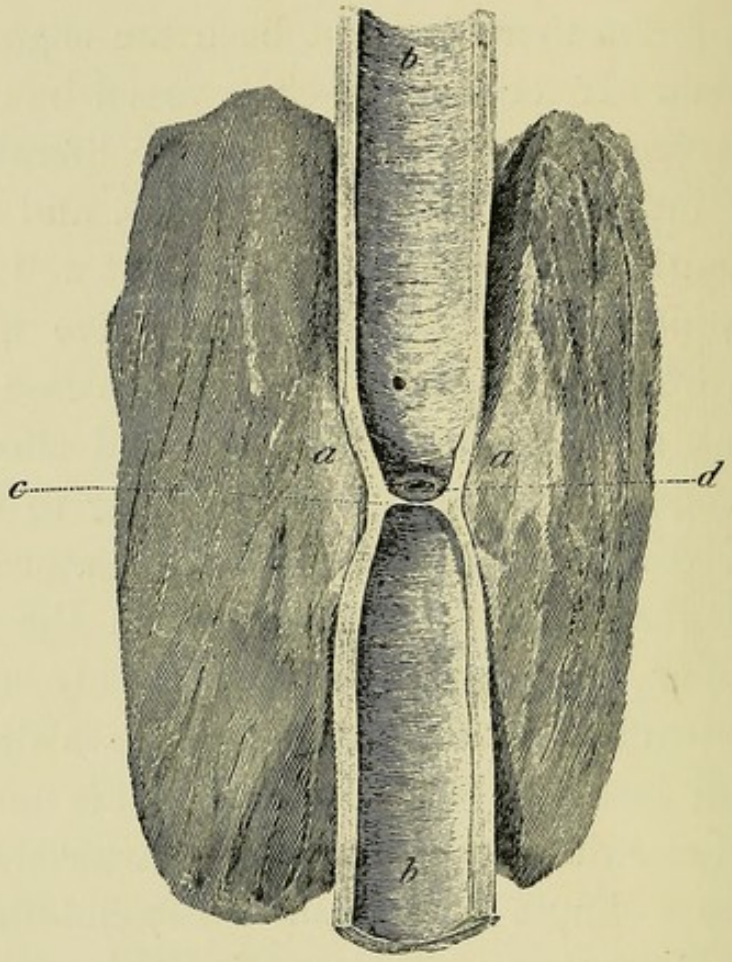


FIG. 83. Carotid of a Horse after one momentary ligature.

Jones tied the carotid of a horse with a single ligature so as to rupture the coats and immediately removed it: the circulation became re-established at once. The specimen was obtained on the third day; there is a very small clot (*d*); across the vessel at seat of ligature is a diaphragm (*c*), through the centre of which is a small perforation—smaller than represented in drawing. (*a*, *a*) plastic effusion.

From J. F. D. Jones *On Hemorrhage* (1805). Plate XII. reproduced with description.

employed, led to the investigation described in the chapter on the Knot, the result of which was the adoption of a method of applying the ligature that is free from the

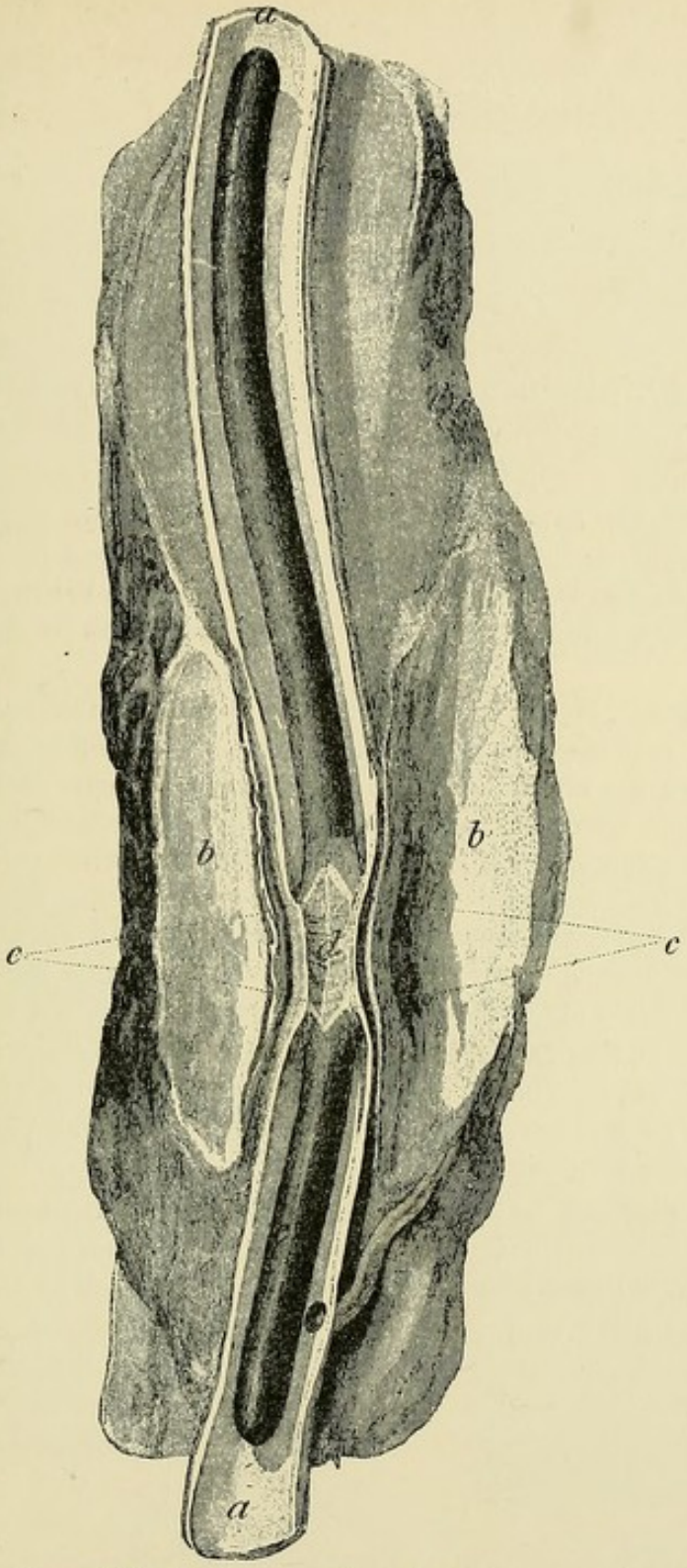


FIG. 84. Carotid of Horse after four momentary ligatures.

Jones tied with four ligatures, side by side, the carotid of a horse so tight as to rupture the inner and middle coats; the ligatures were immediately removed and the circulation became again established. Three days later the specimen was obtained: at the seat of ligature (*d*) the coagulum was adherent, (*c*) for ½ inch (12 mm.): the clot extended a long distance above and below this but it was not adherent. (*b*, *b*) plastic effusion.

From J. F. D. Jones *On Hemorrhage* (1805). Plate XI. reproduced with description.

Figs. 85, 86, 87. Human Carotid Artery 108 days after ligature with stout catgut.

The left-hand lower figure (Fig. 86) shows the naked eye appearances. The artery is patent, but its lumen is contracted by a diaphragm leaving an opening 1½ lines (3 mm.) in diameter: the outer contour of the vessel shews a depression at the seat of ligature, and the free border of the diaphragm is depressed in the centre.

The upper figure (Fig. 85) is a longitudinal section of the wall of the vessel (× 20): the outer coat is above, the intima below. It is obvious that the ligature completely divided the middle coat: no doubt the inner coat also was ruptured, for the middle coat never ruptures alone: the injured intima has reunited and become thickened, forming a diaphragm. In the connective tissue between the ends of the middle coat were found remains of the catgut ligature: these are represented more highly magnified (× 100) in the right-hand lower figure (Fig. 87). At the seat of ligature the artery was very weak and bent like a hinge: connective tissue has formed round and about the ligature, constituting a new outer coat.

In tightening the first half of the reef-knot the coats were ruptured; but before the second half of the knot of the *stout* catgut ligature was completed it is probable that the first half gave to the pressure of the blood, which in this case was increased by the occlusion of the opposite carotid.

The carotid was tied by James Adams for innominate aneurism, the subclavian being subsequently ligatured by Treves. The specimen is in the Hunterian Museum (3084 A). Professor Stewart kindly gave us a portion for the microscope.

The case is recorded by Treves in the *R. Med. and Chi. Soc. Trans.* vol. LXIV. 1881.

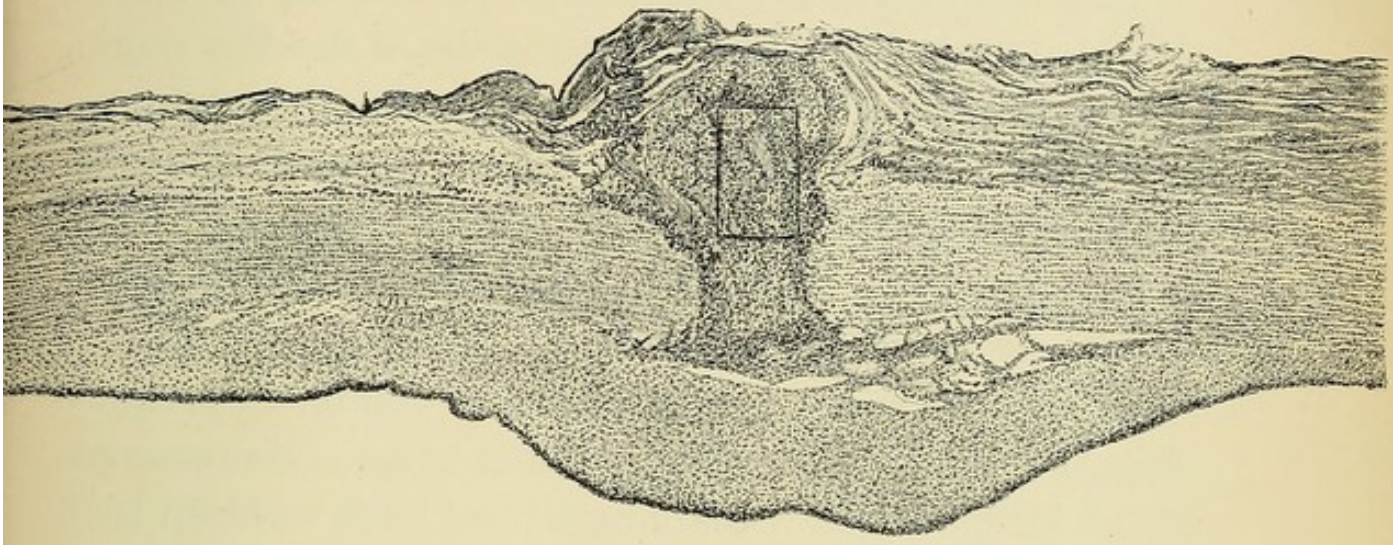


FIG. 85.

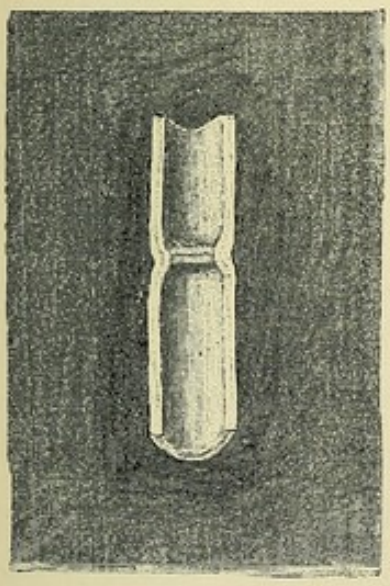


FIG. 86.

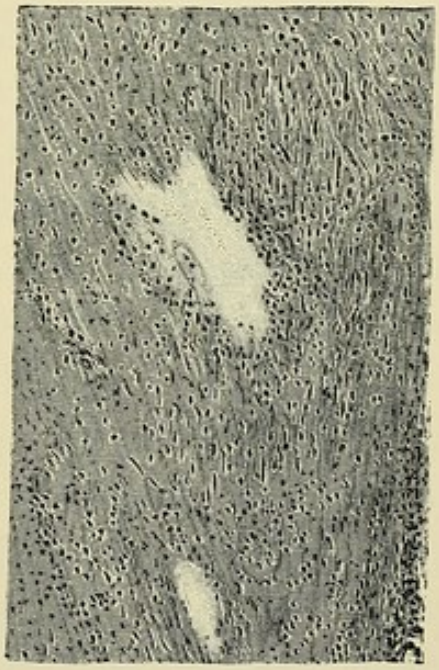


FIG. 87.

imperfections attending the use of the reef and surgical knots.

In some of the arteries the thickening of the intima extended for long distances from the site of the diaphragm. In others, in which a longer interval had elapsed between ligation and death, this intimal growth had in part disappeared. This primary intimal development may depend not only on the irritation of the clot, but also on diminished arterial pressure; and its subsequent disappearance may be consequent on the return of the normal blood pressure when the coagulum has been washed away. The changes of the intima where a diaphragm has been built up have reached the fibrous stage, and so no alterations of pressure could be thought of as likely or potent enough to cause resolution of such tissue.

Treves, at the Royal Medical and Chirurgical Society on February 8th, 1881, reported the case in which James Adams ligatured the common carotid for innominate aneurism. Catgut ligature of large size was used, and it was tied in a "surgical" knot. The wound healed without suppuration. The death of the patient took place 108 days later. At the post-mortem examination it was found that the ligature had completely disappeared, and that externally the contour of the artery was uninterrupted and apparently normal at the seat of ligation. On examining the interior of the vessel it was seen to be patent, but narrowed at the seat of ligature by a kind of diaphragm having a central aperture 1½ lines (3 mm.) in diameter (Figs. 85, 86, 87). The diaphragm was produced by the incurvings of the ruptured ends of the middle coat.

Through the kindness of the Curator of the Hunterian

Museum we have had the opportunity of examining this specimen microscopically. It is quite clear that both the inner and middle tunics were divided by the ligature. (Fig. 85). Between the ruptured ends of the middle coat traces of the catgut ligature are distinctly visible, still unabsorbed. (Fig. 87.) The intima for a considerable distance on each side of the point of ligation is greatly thickened, and opposite the position of the ligature is raised into a distinct eminence The tissue forming the base of this ridge of intima is continuous with the young connective tissue surrounding the remains of the ligature between the ruptured and separated extremities of the tunica media.

This diaphragm formed in a human carotid is exactly comparable to those which we have had the opportunity of studying in the carotids of asses, except that in the ass carotids the middle coat was in all cases uninjured. The groove on the summit or inner border of the diaphragm in the human carotid is due without doubt to the separation of the ruptured ends of the inner coats on either side of the ligature. (Fig. 88).

A diaphragm in the common carotid of a horse which had been tied with a clove hitch of tendon is shown in Plate II. Fig. 3.

Similar conditions in man have been described by MacCarthy, Lane and others, but perhaps the most interesting case is that of Holmes, which is given and figured in his *Principles and Practice of Surgery:* it was a simultaneous ligature of the carotid and subclavian arteries for intrathoracic aneurism; carbolized catgut was used; the patient survived 56 days; it was found

at the autopsy that the subclavian artery was perfectly occluded by a diaphragm of opaque white tissue; within the carotid was also a diaphragm, but in this artery the

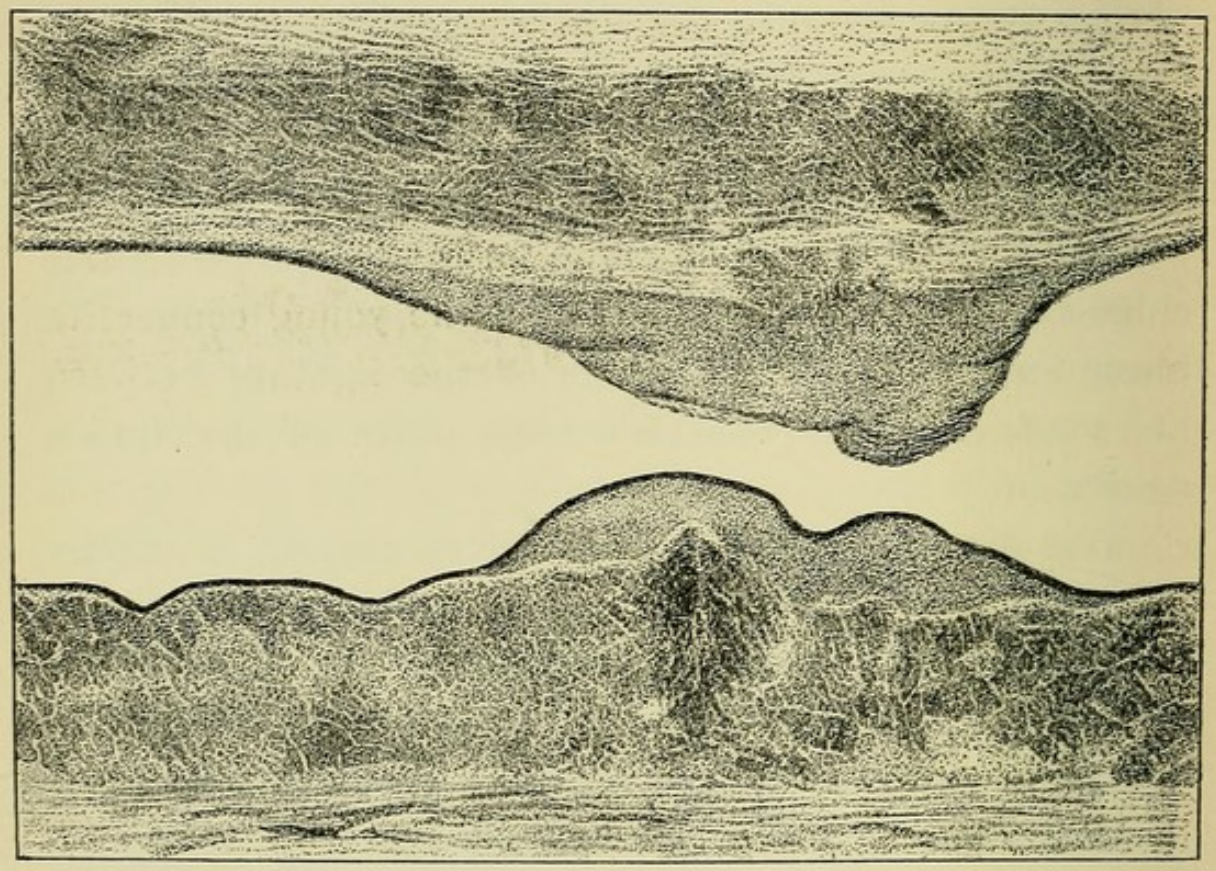


FIG. 88. Longitudinal section of the Carotid Artery of an Ass 77 days after ligature with a small kangaroo tendon tied in a reef-knot (× 26).

The vessel was patent, though greatly diminished in calibre, the lumen at the seat of ligature being still further reduced by a diaphragm which is formed by the hypertrophied inner coat. The groove on the free border of the diaphragm suggests that the intima was ruptured in some degree at the operation.

The specimen is an illustration of the partial giving way of the first half of the reef from the pressure of the blood before the second half of the knot can be tied tight.

lumen was only narrowed: there were also found passing through the wall of the carotid at the seat of ligature two small openings which led to a minute cavity external

to the vessel containing blood clot. "This is, if I mistake not," says Holmes, "the first definite anatomical proof "that arteries can be obliterated at the seat of ligature "without being divided. If this result could be attained "in every case secondary hemorrhage would of course be "unknown."

The conclusion of the matter is then that the formation of a diaphragm is due to the failure of the ligature to completely close the artery. If the artery be occluded of course there is no space for a diaphragm to form. Diaphragm production is not therefore related to the question of ruptured or intact arterial walls, but must depend on some fault in the way the ligature is applied, or on its too rapid absorption.

CHAPTER IX.

CONDUCT AND FATE OF THE COATS.

Wound septic: ligature silk or hemp. Wound septic: ligature tendon or catgut. Wound aseptic: coats ruptured: silk or catgut. Wound aseptic: coats ruptured: catgut ligature. Temporary ligature. Loose ligature. Wound aseptic: arterial wall intact. Plastic effusion; coats ruptured: coats not ruptured. Endothelial proliferation. Plasma-cell proliferation in middle and outer coats. Vascular changes in wall. Fibrillation and final state of the parts. Aneurism and arterio-venous aneurism at seat of ligature. Arterio-venous aneurism at seat of compression. Aneurism caused by new growth.

The conduct of the coats will depend partly on whether suppuration occurs, partly on whether they are ruptured, and partly on the absorbability of the ligature.

If suppuration occurs a silk or hempen ligature will work its way across the vessel and be discharged, the artery becoming completely divided and the two ends

retracted from each other; this will certainly occur if the coats are ruptured and may do so if they are not, the difference being that in the latter case the process will take longer. In no instance, however, have we known a ligature, whether septic or aseptic, which did not lacerate the coats, cause death of the section of the arterial wall included in the loop of the ligature and the consequent division of the vessel. If, however, an absorbable ligature such as chromic catgut or tendon be used and the coats be uninjured the continuity of the wall of the vessel will be maintained and it will not be divided. If the ligature be of tendon the artery will be permanently closed (Fig. 58), but if it be of a very absorbable nature, such as carbolic catgut, it may disappear so rapidly that the artery will become patent and the circulation through it be re-established.

When suppuration does not occur the fate of the vessel will depend on whether the coats are ruptured or not. If the coats are ruptured the adventitia in the loop of the ligature may be absorbed, the vessel being thus completely divided and its two ends retracted. That this occurs was pointed out long ago by Bryant in an article in the *Transactions of the Clinical Society* on the Catgut Ligature: and Senn states that a silk ligature always works its way across a vessel and becomes encapsuled to one side of it: but this we opine only occurs when the coats are ruptured.

The fate of the adventitia in the loop of the ligature depends upon the rate of absorption of the latter. The longer the time required for the completion of the absorption processes, the longer will inflammatory changes

FIGS. 89, 90. Carotid of Sheep 10 days after ligature with chromic catgut (× 26).

The coats are uninjured and the lumen is all but obliterated. A clot has formed on both sides.

The lower figure (Fig. 90) represents more highly magnified (× 300) the angle of approximation of the coats, and the great proliferation of the endothelium uniting the intima of opposite sides: (*f*) the penetrated membrane of Henle.

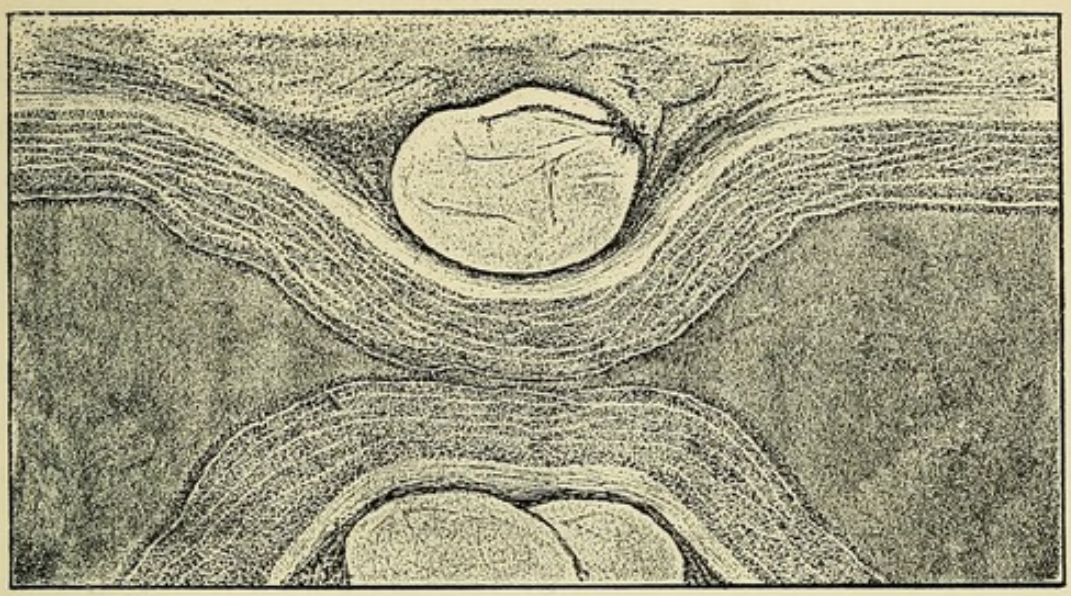


FIG. 89.

FIG. 90.

be active in the neighbourhood of the portion of adventitia in question, and the greater the likelihood of the occurrence of the solution of this tissue. Both Lister and Holmes hold that the catgut ligature, when the coats are ruptured and the wound heals by first intention, does not kill the included adventitia. Lister wrote, in 1869, "that by ap-"plying a ligature of animal tissue antiseptically upon an "artery, whether tightly or gently, we virtually surround it "with a ring of living tissue and strengthen the vessel "where we obstruct it." And Holmes (1888) claims to have "offered the "first definite anatomical proof that arteries "can be obliterated at the site of a catgut ligature without "being divided." His case and others are referred to on page 217.

If a temporary ligature be used, that is to say, if the ligature be removed at the end of 70 hours, the coats being ruptured, the adventitia will live and the vessel will not be divided. This has been shown by Bruns and Walter, who assert that not only does the adventitia not die, but that it is actually strengthened by the deposit of new connective tissue outside it. The continuity of the vessel, in fact, depends in the case of rupture on the life or death of the adventitia in the loop of the ligature; and this in its turn will depend on the nature of the ligature used, on the thickness of the adventitia of the particular artery tied, and the tightness of the constriction it is subjected to. There is a specimen in the Museum of the College of Surgeons, to which reference is made elsewhere, in which the coats were ruptured by a catgut ligature, but in which not only is the continuity of the vessel preserved, but the circulation is re-established through it. (Fig. 86, page 215.)

The "loose" ligature, which the younger Cline and South tried, has no effect whatever on the circulation unless suppuration ensues, in which case the ligature may

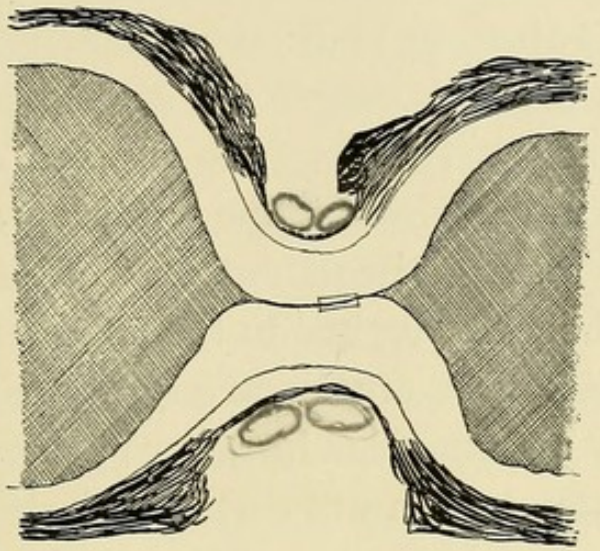


FIG. 91.

FIG. 92.

FIGS. 91, 92. Carotid of Horse two weeks after ligature with two kangaroo tendons tied with the stay-knot.

The upper figure (Fig. 91) shews the coats not injured, and in close apposition for some distance. A clot has formed above and below.

The lower figure (Fig. 92) shews highly magnified (×200) the part marked off by lines in the upper figure. The multiplication and cohesion of the endothelia of opposite sides is seen.

work through and divide the vessel, as no doubt occurred in some experiments. South writes (1847): "A thread "applied around the carotid artery of a dog so loose as not "to interfere with the passage of the blood, is sufficient to

"cause inflammation, which will block it up completely, as "was proved by an experiment made by my able master, "the younger Cline, and which I myself repeated with the "like result." A case recorded by Porcher shows the effect of the loose ligature in man: the common carotid artery was tied with carbolic catgut for carotid aneurism: the patient died on the 7th day from rupture of the sac: at the post-mortem examination the ligature was found to be loose and the vessel to be patent, no clot being present.

The rule then with regard to the fate of the continuity of the ruptured wall of the artery appears to be that asepticity may maintain the integrity of the adventitia, but even a little suppuration will certainly destroy it.

If suppuration does not occur and the coats are not ruptured the continuity of the vessel is maintained: not only is the vessel not injured in any way by the ligature, but it is strengthened by the new tissue which is thrown out around it. The arterial wall is strongest at the point where it is strengthened by a scaffolding of ligature and young connective tissue (see *Transactions of the Royal Medical and Chirurgical Society*, 1886), and Holmes states that "the lymph thrown out around the vessel is a great "support to it, and when the coats are injured is the chief "protection against secondary hæmorrhage."

To maintain intact the continuity of the vessel, then, two things are clearly indicated: that the coats be not ruptured and that the wound be aseptic; if these conditions be complied with there is no danger of hemorrhage, for the vessel is whole and the new tissue formed round it only strengthens it. It has been said that more plastic inflammation occurs if the coats be ruptured, and this is

employed as an argument in favour of rupture. Indeed, some statements in recent works are still more dogmatic: for example, it is said, "that plastic lymph is effused as a "consequence of the injury done to the coats, and upon "the amount and vitality of the effusion depends the safe "closure of the vessel:" "that the injury done to the "intima is of cardinal importance for the formation of "thrombus and the development of adhesive inflamma- "tion:" and again, "if these coats be not lacerated it is "probable that no lymph will unite their opposed sur- "faces;" but the fact is, the amount of lymph effusion is of no importance if without rupture the vessel can be satisfactorily occluded, and if the operation be properly performed this can be done.

Whether the coats are injured or not the fusiform swelling around the artery and ligature is found apparently of equal size in all cases. The lymph may completely obscure the ligature in 30 hours. In the interior of the vessel, if the wall be extensively damaged, no doubt more lymph is thrown out than if it remains intact. The question is, however, as above stated, not whether more or less lymph be effused, but whether, when the coats are not ruptured, the quantity is equal for the end in view, namely, the obliteration of the vessel: and our experiments distinctly answer this question in the affirmative.

It has also been stated that "when two endothelial sur- "faces are brought into contact they unite with difficulty, "and that therefore it is necessary to interrupt the con- "tinuity of the tunics;" and that "it is an advantage to "bring by means of the cutting ligature the adventitia of "one side into close relation with that of the opposite side,

"because union between areolar structures is rapidly "effected." The results of our experiments are quite opposed to these theoretical considerations. With regard to the latter it may be noted that in some arteries there is scarcely any outer coat to deal with (see p. 46); and in respect to the former, that most endothelial surfaces are known to adhere with very little provocation, indeed Ziegler likens the interior of a blood-vessel to that of a serous cavity, and the intimal growth after ligature to the plastic inflammation of a serous membrane.

One of the most important changes which results from the application of a ligature is the multiplication of the cells of the inner coat. This occurs to such an extent that the intima is seen thickened to the naked eye, appearing as a thin pinkish membrane. This thickening of the intima extends as far along the vessel as the clot does, and the thickening may be as great some inches from the ligature as near it. The intimal thickening is always most marked where the clot is adherent, so that if the extremity of the clot is adherent to one side of the arterial wall, the intima may be as thick far away from the ligatured point as it is near.

Dent and Delépine say that the internal coat is always altered whether there is thrombosis or not. We think that the intimal thickening may shew the extent of the original thrombosis, but at the same time it may be that Warren's explanation of the thickening of the intima in arteritis obliterans holds also in this instance, that is to say, that it is due to the diminution of blood-pressure and is a natural or physiological adaptation of the artery to its diminished functional activity.

The amount of the endothelial multiplication is remarkable. Soon the cells become six or more layers deep; and the whole clot surface, and every crack and fissure of the clot, may be covered by plasma-corpuscles. The rapidity of cell development is well known in the lower forms of vegetable life, such as the Fungi; and it has been estimated recently that one bacillus subtilis will increase under favourable conditions to 800,000,000,000,000 of organisms in 24 hours. Flemming states that animal cells can multiply by the indirect or karyokinetic division of their nuclei with a rapidity equal to that of the vegetable cell, so that the rapidity of the endothelial proliferation is explained. The thickening of the intima is mainly caused by the endothelial cells becoming many layers in thickness. The same thing occurs to the cells covering the outer surface or lining the cracks of the coagulum. The first layer of cells, forming the primitive capsule of the clot, and which are for the most part the daughter cells of the proliferating connective tissue corpuscles of the intima, becomes thicker by the addition of cells arising either from the intima or from the multiplication of those cells which have already reached the clot. The thickening of the intima or of the cells on the surface of the clot is caused by new cells being laid on immediately beneath the surface layer; exactly as is described in arteriosclerosis by Thoma; he says, also, that he has seen the same thickening of the intima of arteries in amputation stumps. The daughter cells of the endothelial corpuscles, having large oval vesicular nuclei and little surrounding protoplasm, have been seen by Heuking and Thoma immediately beneath the endothelial plates, and it is these

FIGS. 93, 94. The Carotid Artery of a Sheep 21 days after ligature with kangaroo tendon.

The upper figure (×26) shews that the coats of the vessel are uninjured, the intimæ of opposite sides being in apposition. The white lines shew the position of the fenestrated coat of Henle. The part inclosed by black lines is represented more highly magnified in the lower figure.

In this figure (×160) the intima is greatly thickened, solid columns of cells have passed from the endothelium of one side to that of the opposite side through the substance of the coagulum, which is mapped out into districts. Vacuolation and capillary formation has not yet occurred. The stage is that of "potential vascularization." The outlines of the red corpuscles are no longer visible: all that can be seen is a yellow granular material.

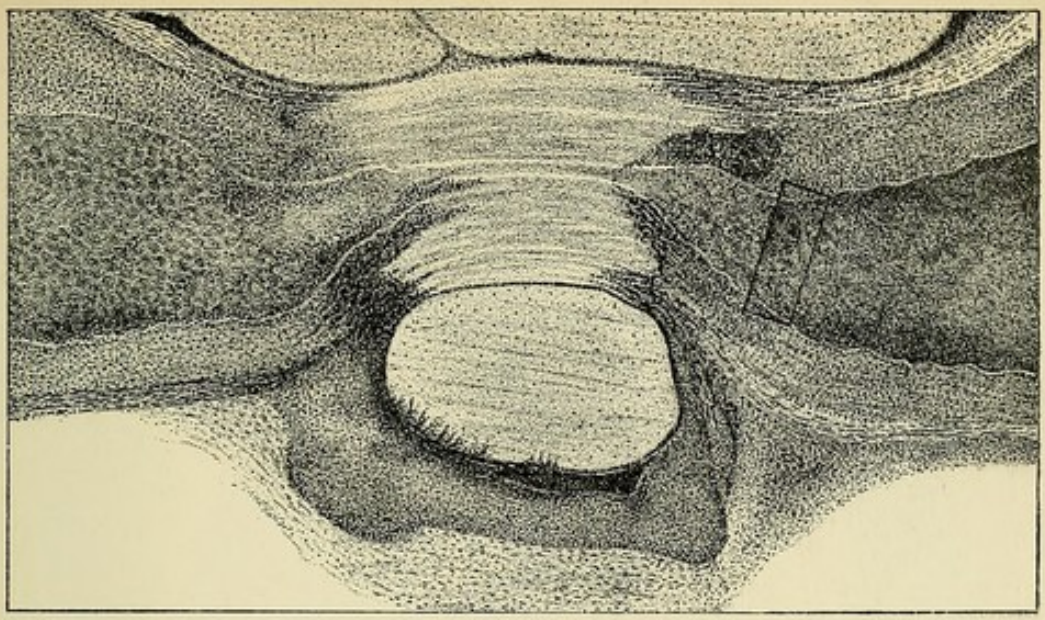


FIG. 93.

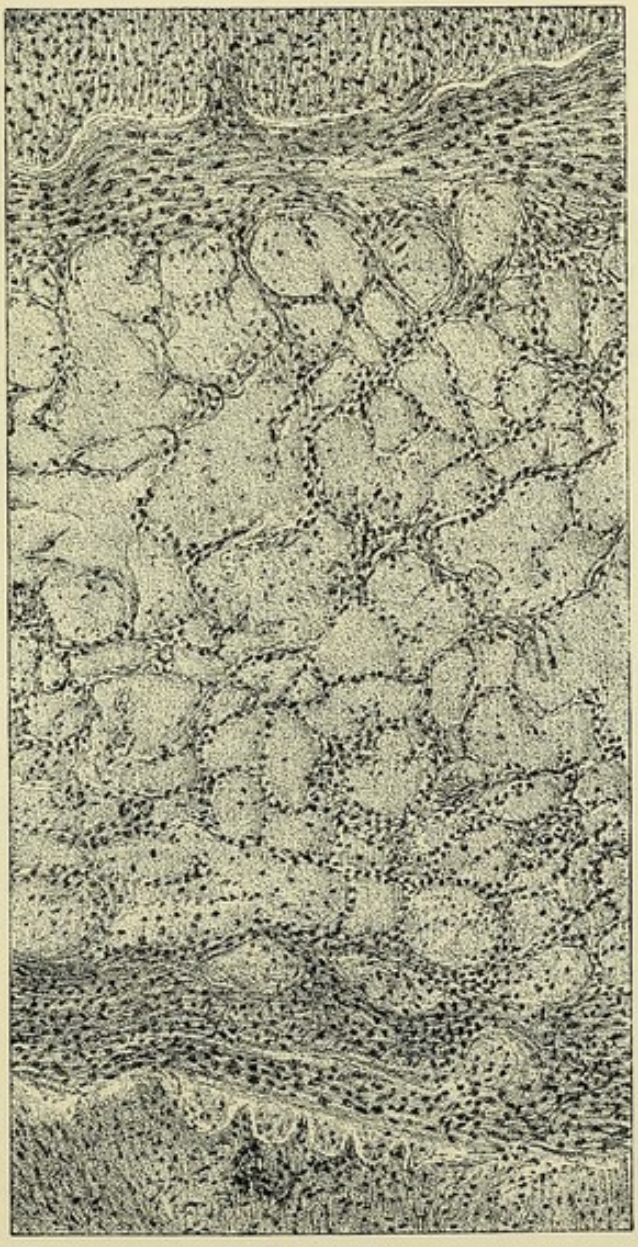


FIG. 94.

FIGS. 95, 96. Longitudinal section of a Carotid of a Horse (× 12) 51 days after ligature with chromic catgut.

The coats of the vessel are uninjured, and the lumen of the vessel was, as far as can be made out, not quite obliterated by the ligature. No trace of the catgut can be discovered even with the microscope. The place of the clot is taken by connective tissue material, which is completely fused with the intimæ of the opposite sides. * Probable site of ligature. The lines indicate the part which is represented more highly magnified (× 60) in figure 96.

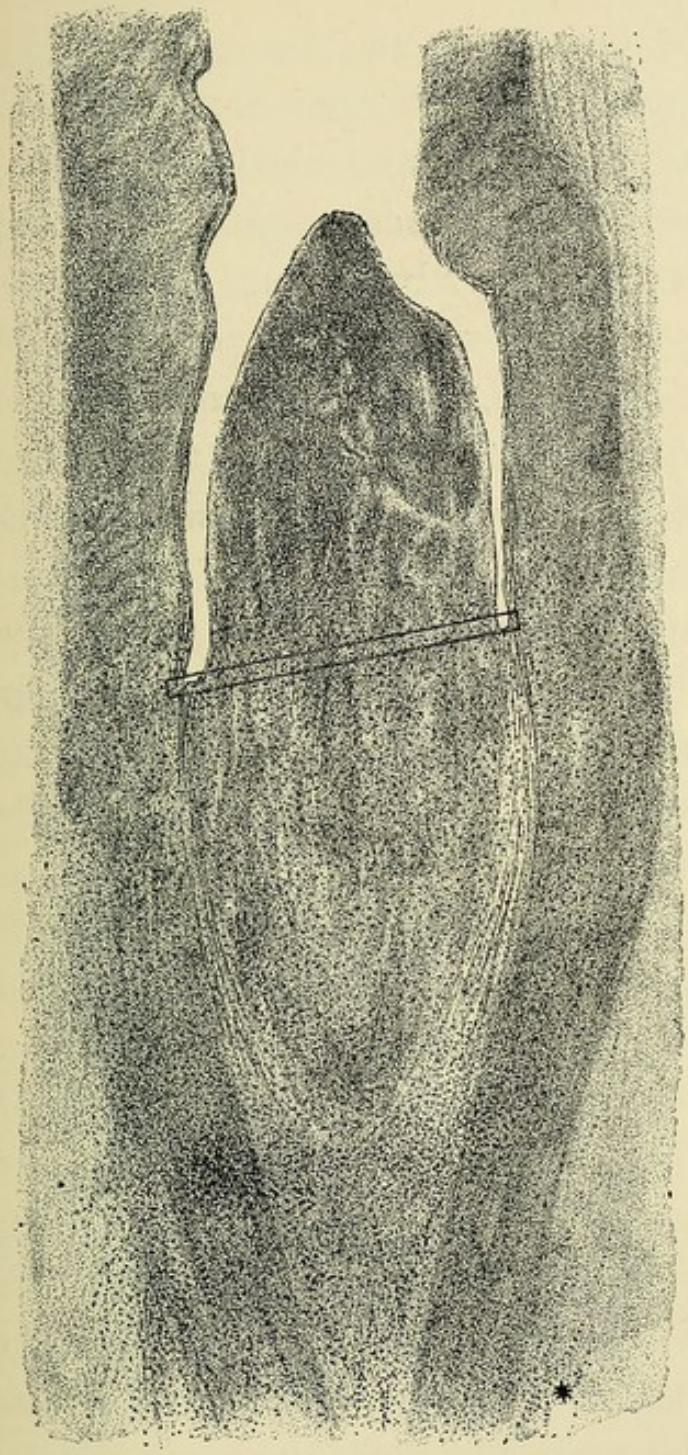


FIG. 95.

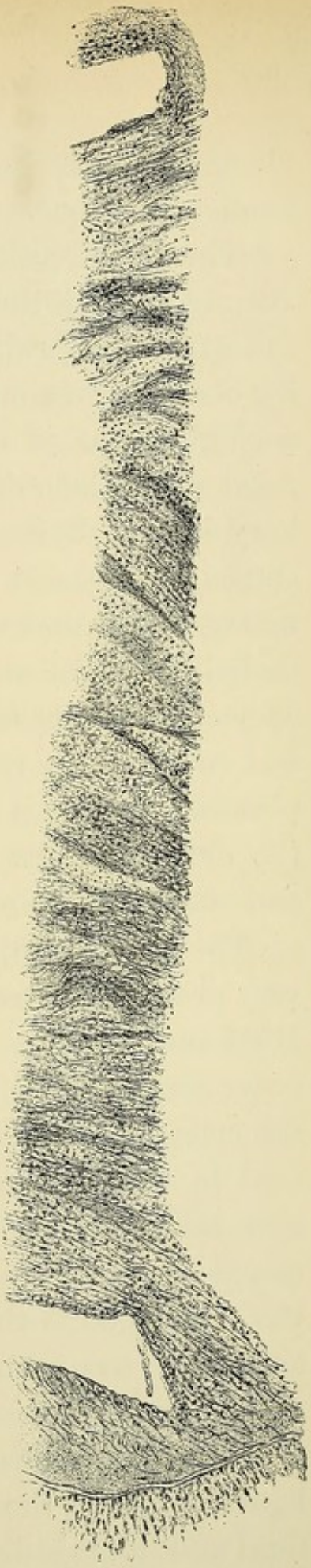


FIG. 96.

elements which reach the clot and ultimately replace it by connective tissue.

The proliferation of the intima has been studied by Baumgarten, Böttcher and ourselves (see Chapter VII.) in the carotids of rabbits, which are particularly favourable for observation since the intima in these vessels consists of a single layer of cells. Böttcher, by means of sections fixed with Flemming's mixture, was able to observe the karyokinetic division of the cells, the stellate and rosette stages being most frequently seen. He says that there was no doubt that what he saw was the division of endothelial cells and not leucocytic proliferation; for the leucocytes were very few in number in the wall of the artery and could always readily be distinguished from the larger plasma-cells. It is by the act of cell division, he says, that the daughter cells of the plasma-corpuscles become free and competent to migrate into the clot or elsewhere.

The effect of injury to the vessels of the adventitia is very prettily illustrated by an unpublished experiment of Mott and Horsley. They applied some nitric acid to the outer coat of the carotid of a horse: great proliferation of the cells of the underlying intima followed and the inner coat in this region became permanently very thick, the new tissue undergoing fibrillation. The action of the caustic is probably due in part at least to the arrest of the circulation in the vasa vasorum; a ligature, even if not so tight as to rupture the coats, produces the same effect.

The proliferation of the endothelium already described is not the only change produced. The application of a ligature blocks the vasa vasorum, causing exudation of fluid and diapedesis of leucocytes. The result is that the

whole thickness of the wall of the vessel is saturated by the fluid and cellular invasion; and thus a proliferation of the connective tissue-cells of the deeper layers of the intima and of the middle and outer coats is excited.

This process has been carefully studied by both Baumgarten and Böttcher. The former describes first the growth of the intima, and secondly the inflammatory proliferation of the plasma-cells through the whole thickness of the arterial wall: and the latter has observed the karyokinetic division of the connective tissue-cells through all the thickness of the artery wall. The large vesicular nucleus of the daughter cells of the plasma-corpuscles and their other characters, clearly differentiating them from the white blood-cells.

Some or many of the muscle-cells of the middle coat may disappear, and in a short time the new connective tissue, covering the ligature and replacing the wall of the artery and the clot, is a continuous fibrous mass. When the ligature has not injured the arterial wall it is easy to follow with the aid of a lens the course of the middle or chief arterial tunic through the connective tissue mass, and this may be done for many days after ligature. (Plate II., Fig. 1, and compare with Fig. 85, p. 215.)

During the stage of active connective tissue development important alterations occur in the vascular supply of the arterial wall, and it may be pointed out that normally capillaries from the adventitia dip down into the middle coat, and that the endothelial cells nearest to the capillary loops will therefore obviously be in a position of vantage with regard to the vascularisation process in the clot.

In sections of the arterial wall made from 12 hours

and onwards after ligation streams of cells are seen passing down on either side of the ligature through the inner half of the media and the whole of the intima. These streams probably pass through openings in the fenestrated membrane of Henle, and are made up at first probably of leucocytes escaped from the blocked vasa vasorum and looped capillaries, and ultimately by plasma-cells which come from the proliferating connective tissue corpuscles of the whole arterial wall. The position occupied by these solid columns of cells is subsequently taken by the capillaries which pass from the adventitia through the wall of the vessel into the clot. In this manner the firm attachment of the coagulum to certain places of the arterial wall, and the facility with which the current of blood is able to separate the clot from the intima in other situations is capable of explanation.

After a time, the fibrillation of the new tissue within and without the artery tends to make it more compact and less vascular. The parts assume the character of ordinary scar-tissue and are not, *tace* Warren, made up of a mass of involuntary muscular fibres. The scar-tissue soon becomes more elastic, looser, and adhesions are freed. The tissue, in fact, becomes more like natural connective tissue. The artery itself cannot indeed be reconstituted, but the branches which connect the patent upper and lower ends exhibit the effort of Nature to return to the state which obtained prior to the application of the ligature: so that gradually, as Paget says, a "nearer "conformity of the tissue fabric with the specific character "of the parts it would replace, is established till repair "becomes almost reproduction."

In certain rare cases an aneurism forms at the seat of ligature. Fig. 97 is taken from Porta's work and shews an aneurism on a dog's femoral artery which he had ligatured a year previously. Thomas Smith has recorded a case in which after ligature with catgut of the superficial femoral for popliteal aneurism a secondary aneurism formed at the seat of ligature. A case in which a secondary aneurism formed on the subclavian artery after the application of a single silk ligature is recorded by Cowell. A case in which an arterio-venous aneurism formed on the superficial femoral at the seat of ligature is related by Oliver Pemberton: "The femoral artery had formed an "aneurism at the seat of operation as large as a hen's "egg, and the femoral vein communicated with the artery "by a large opening." (Fig. 98.) Akin to this is another case recorded by Pemberton, in which an aneurismal varix formed at the seat of digital compression. (Figs. 99, 100.) This formation of aneurism as the result of inflammation is analogous to its formation from the invasion of the arterial wall by new growth. Fig. 101 shews this somewhat rare condition. In some of these cases an error of diagnosis would seem to be inevitable.

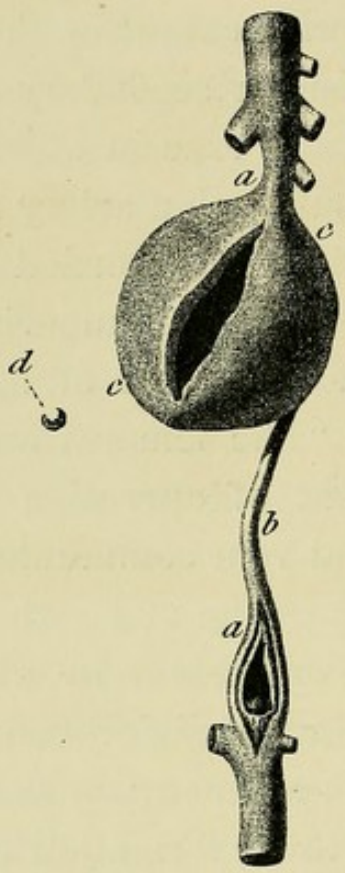


FIG. 97. Aneurism at seat of ligature.

(Reproduced from Porta's Delle Alterazioni Patologiche delle Arterie per la Legatura e la Torsione. Plate I., Fig. 4. Milano, 1845.)

Secondary aneurism at seat of ligature. The femoral artery of a dog ligatured one year before death.

The cyst (*cc*) contained "thick lymph," and also a remaining shred (*d*) of the silk ligature. It communicated freely with the upper end of the artery. Beyond the ligature the artery was reduced to an impervious cord (*b*).

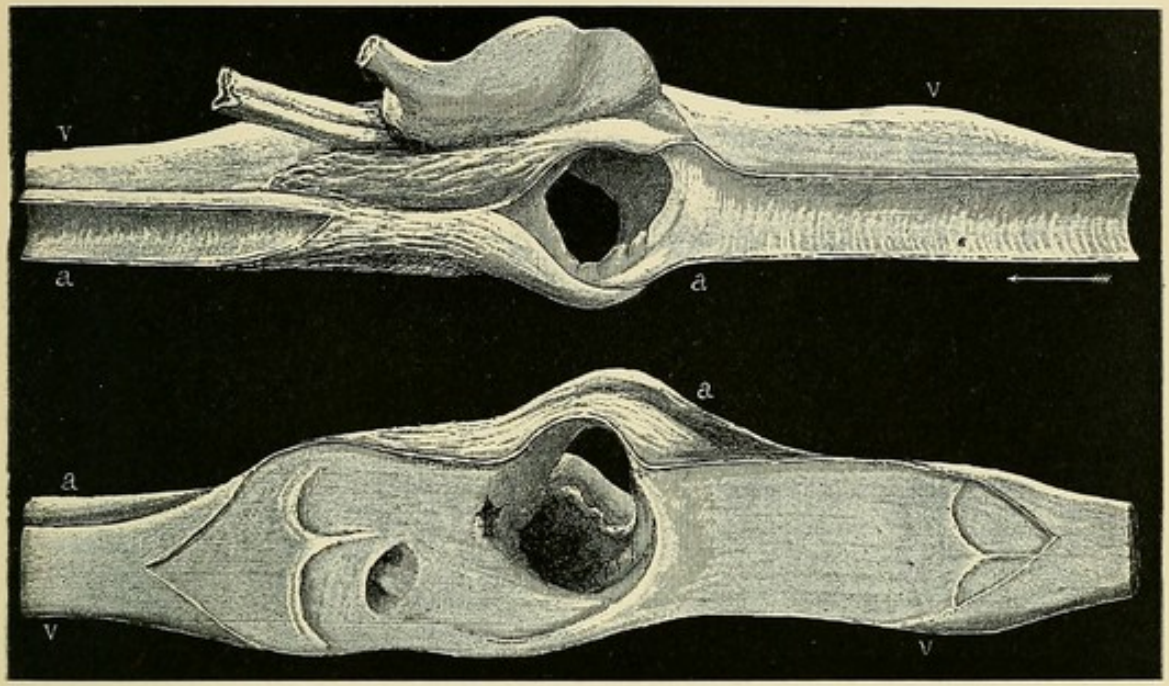


FIG. 98. Arterio-venous aneurism of superficial femoral vessels at seat of ligature. ($\frac{3}{4}$ size of specimen.)

Aneurism of the superficial femoral artery at entrance of Hunter's canal in a male patient, ætat. 41. Ligature of superficial femoral artery by Mr Amphlett in 1844: ligature separated 19th day: arterial hemorrhage from seat of ligature on the 29th, 39th and 53rd days: patient recovered. Three years later arterio-venous aneurism at seat of ligature: two years later died of pleurisy: specimen removed by Mr Pemberton. The superficial femoral artery is completely obliterated for one inch (24 mm.) at presumably the seat of ligature: immediately above this the artery is somewhat dilated and communicates with the accompanying vein by a large opening; the vein is also dilated, but the opening between the artery and vein is so free that there is only one aneurismal sac: below its obliteration the artery is quite patent into the aneurismal sac, from which the clot has now been removed; below the sac the artery is greatly contracted but allows of the passage of a probe: the vein is dilated below the aneurism. The drawings represent the specimen from two different views: (*a*) artery, (*v*) vein. See Oliver Pemberton, *Address in Surgery to British Medical Association*, 1872. The specimen was kindly lent to us by Mr Pemberton.

Figs. 99, 100. Aneurismal Varix of Common Femoral vessels resulting from compression.

Male aged 50 was admitted with aneurism of the upper part of posterior tibial artery. This was treated with instrumental compression of the common femoral artery below Poupart's ligament, lasting more or less continuously for nine months, when aneurism was cured. Ten months later well-marked symptoms of arterio-venous communication at seat of former pressure. In 20 more months patient died. Autopsy showed that the original aneurism was cured; but that there was an aneurismal varix on the common femoral vessels. In the figures, *A* is the femoral artery, *B* the femoral vein, *C* the saphena vein, *D* the profunda and external circumflex arteries arising by a common trunk. As seen in the right-hand figure, there is a large varix on the vein, communicating with the artery by a large opening. There is also some dilatation of the artery.

See Oliver Pemberton, "On a Case of Aneurismal Varix" in the *Transactions of the Royal Medical and Chirurgical Society*, Vol. XLIV. (1861), from which the figures have been reproduced by kind permission.

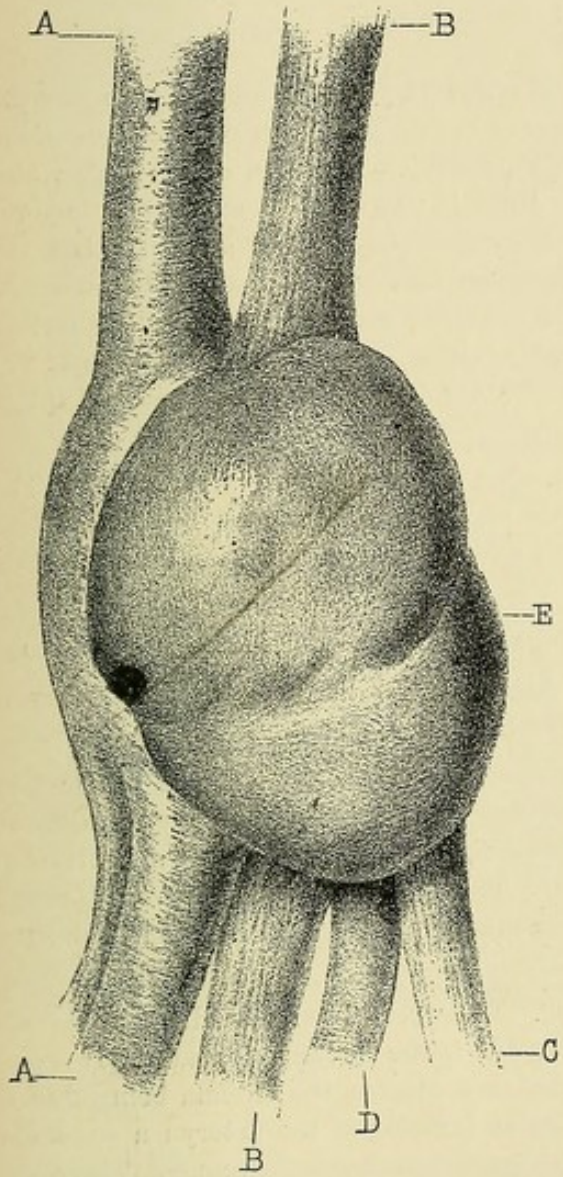


FIG. 99.

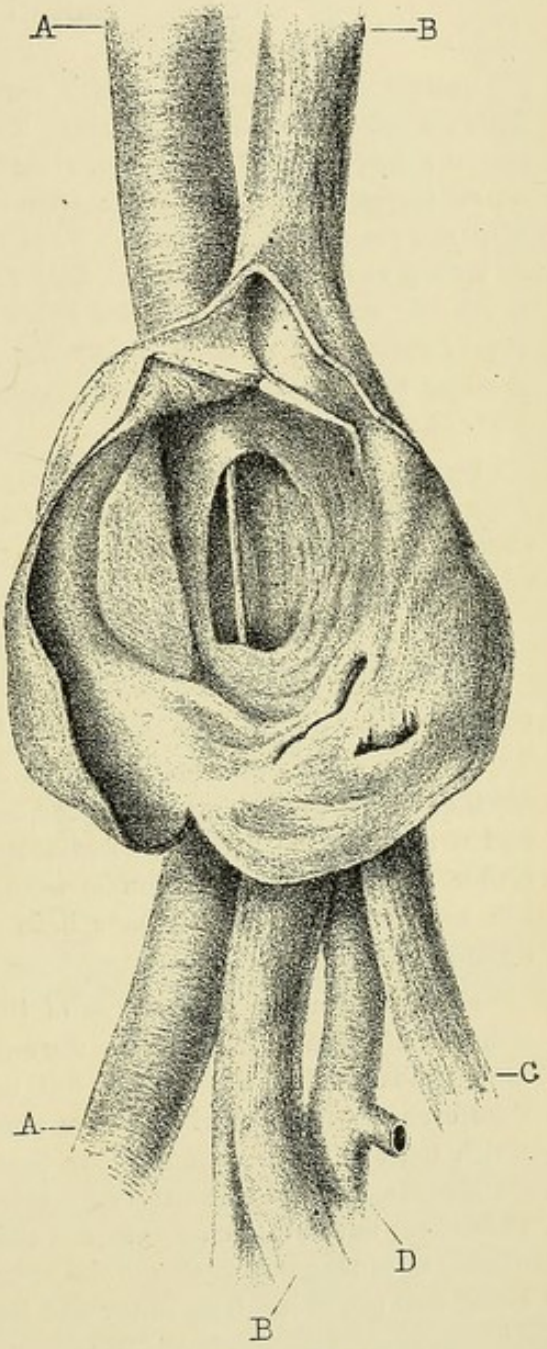


FIG. 100.

Figs. 101, 102. "Popliteal Aneurism." Ligation: recurrent pulsation: sarcoma.

Patient was under Mr Croft's care. He was 71 years of age. Three months before admission œdema of left leg. Two weeks before admission poplite alaneurism, size of a marble. On admission, June 4, 1888, aneurism now size of small egg; also mitral incompetence and aortic stenosis. June 16: ligation of superficial femoral with silk; coats not ruptured. Next day recurrent pulsation in aneurism, but not so strong as before operation. July 7: aneurism harder and smaller but pulsation continues; religation above and below first ligature; coats ruptured; all pulsation ceased and never returned. July 27: some swelling in popliteal space. Sept. 27: swelling has increased; pain of gnawing character. Oct. 15: amputation of thigh. Nov. 14: well; discharged.

Figure 101 shows section of limb ($\frac{1}{2}$ nat. size). The aneurism is seen at *C*. The popliteal artery passes first through a mass of sarcoma (*SS*), then by a circuitous channel through the clot, as shown by the arrow, and finally into the lower portion of the popliteal artery.

The vein (*V*) is seen plugged with sarcoma both above and below the aneurism.

Figure 102 shews the part (*m*) more highly magnified ($\times 100$). The artery is seen with the clot within and the sarcoma without (to the left in drawing).

The recurrent pulsation after the first ligation must have been due to the first hitch of the knot slipping before the second was completed, for there was pulsation and a thrill in the artery immediately below the ligature indicating a contracted orifice. Although no pulsation followed the second ligation, blood still passed through the aneurism; there must have been an excessive collateral circulation round the ligatures.

The first sympton was œdema of the leg. Ten weeks later a small aneurism was noticed; after another ten weeks the swelling due to the sarcoma appeared. It seems most probable that the tumour was the primary disease, the œdema being due to interference with the vein and the aneurism to invasion of the artery: a condition which must be more common than that of an aneurism causing sarcoma. There was cardiac disease, and this suggests a third explanation—that the aneurism was due to embolism; but in that case it is probable that the aneurism would have formed immediately above the bifurcation of the artery, whereas that portion is healthy, the disease being well above it. The femur and its periosteum are healthy.

Specimen in St Thomas's Hospital Museum. See *Trans. Pathological Society*, 1890.

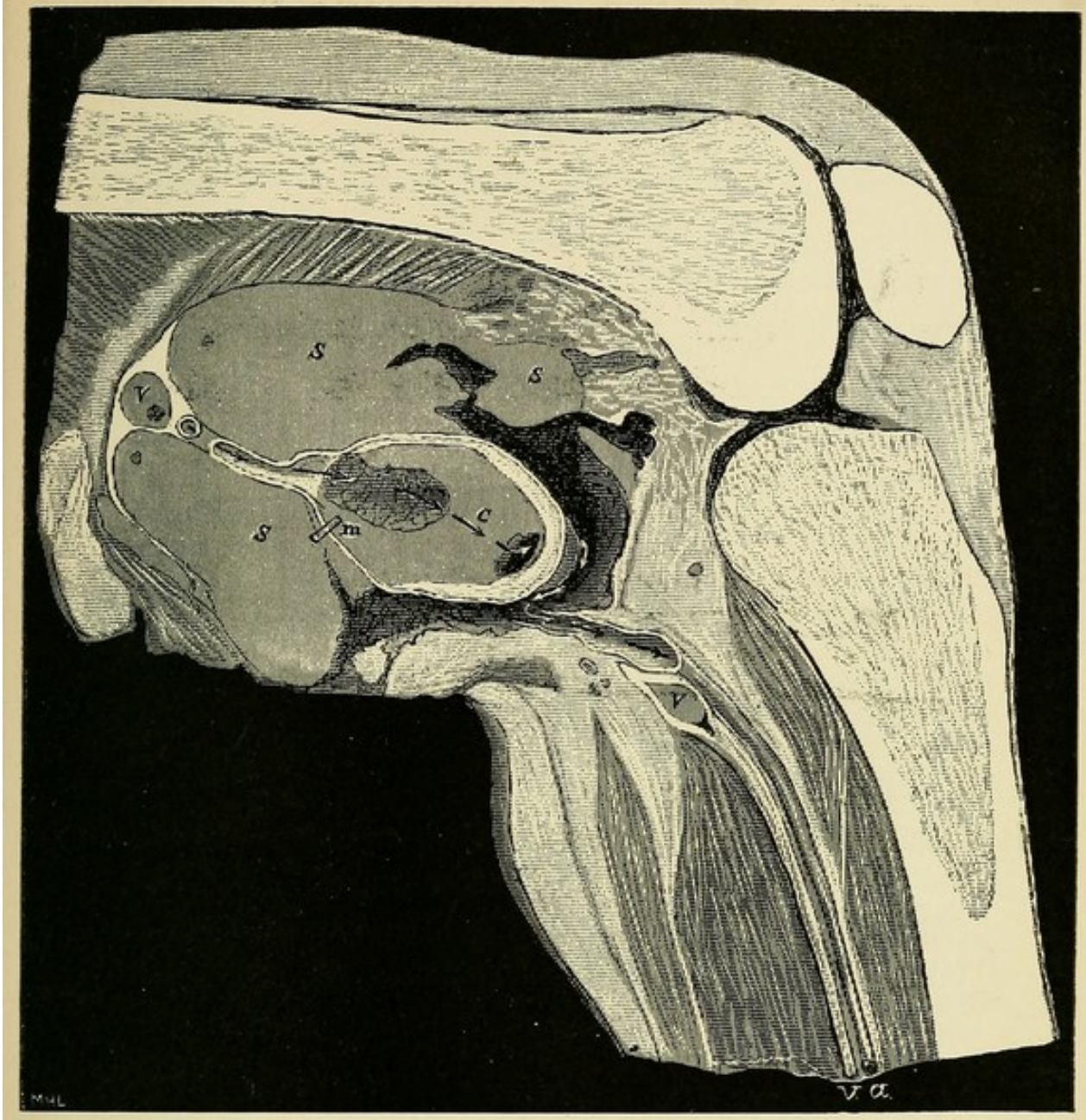


FIG. 101.

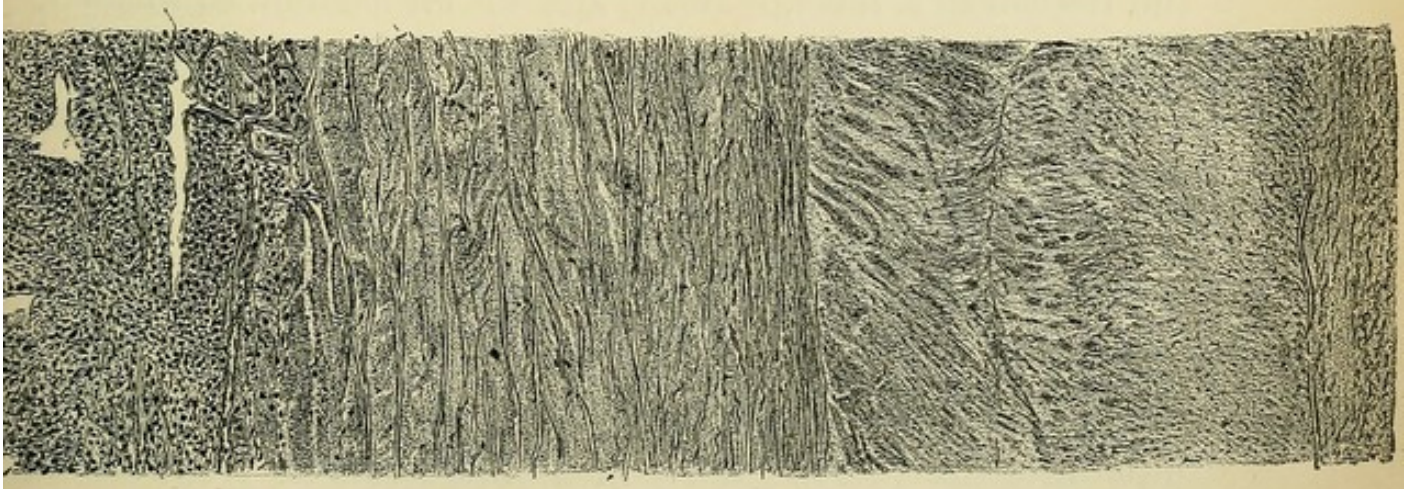


FIG. 102.

CHAPTER X.

CONDUCT AND FATE OF THE LIGATURE.

Most ligatures absorbable. Exceptions. Absorption and Substitution. Catgut. Kangaroo tendon. Reindeer tendon. Silk. Floss silk. Silkworm gut. Ox aorta. Peritoneal ligature. Rate of absorption. Observations of Porta and others.

A ligature applied round a blood vessel is treated by the tissues as a foreign body, and an endeavour to absorb it is made at once by an attack of cells. The success of this attack will depend on the nature and structure of the ligature. In the case of gold wire it is wholly unsuccessful, and the ligature will remain permanently encapsuled: the same is probably true of platinum, for R. H. Clarke has found that tracheotomy tubes plated with platinum, used for the treatment of roaring in horses, will last for an indefinite time, keep their polish, and cause no irritation;

whereas silver tubes become after a time thin where they pass through the wound and consequently untrustworthy.

Wires of silver, lead, iron and probably other metals become sooner or later completely absorbed: we have found a silver wire in an aseptic case used for the suture of the olecranon distinctly pitted from absorption at the end of four months: compare figure 103. Haynes ligatured the carotid of a large dog with lead wire: no trace of it could be seen at the end of 42 days. All animal and vegetable ligatures also disappear in time: and ivory pegs were found by Glück deeply eroded in 14 weeks.

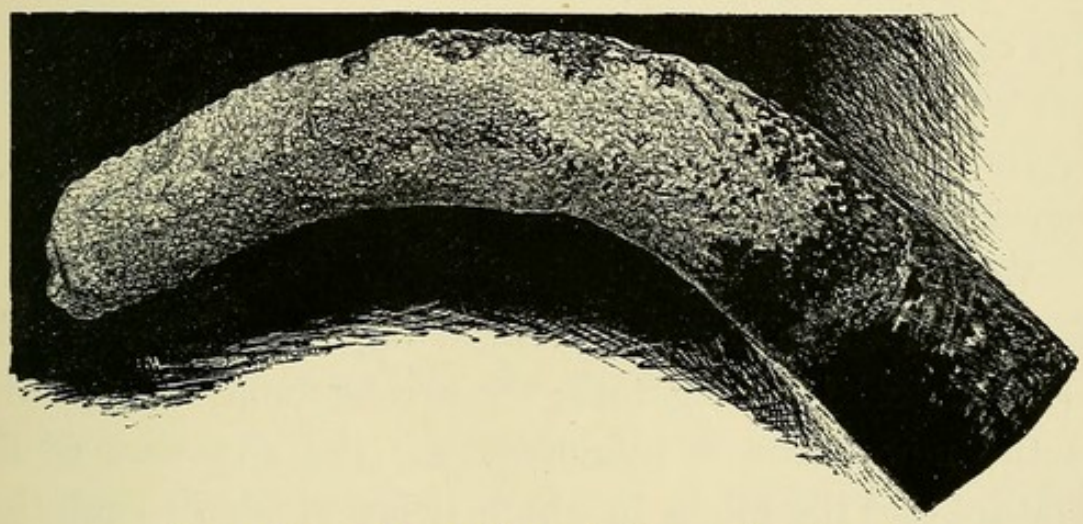


FIG. 103. Absorption of silver wire (× 45).

From specimen kindly presented by Mr R. H. Clarke.

A silver wire was placed in the larynx of a horse and allowed to remain 3½ months. The wire is thinned, brittle, corroded and pitted on surface; the free end (to the left) was bathed in pus.

Our experiments have been chiefly with kangaroo tendon and chromic catgut, but we have also used ordinary silk (Chinese twist), floss silk, reindeer tendon, silkworm gut, sulphurous catgut, ox aorta and a ligature made from the peritoneum of the ox.

If no septic organisms have been introduced at the operation the wound will unite by first intention, and a mass of plasma-cells will rapidly encapsule the ligature. This collection is much less on the vessel side of the ligature in consequence of the pressure exerted by it. The plasma-cells will cause absorption of the surface of the ligature with which they are in contact, and if the structure of the ligature permit of it, they will also penetrate into its interior, and by thus attacking a larger surface cause its more rapid absorption.

If from its nature and bulk the ligature be not readily absorbed, the delay in the process will lead to the formation of connective tissue, that is to say, to encapsulation; but absorption is not on this account arrested: on the contrary, with the exceptions mentioned, it is always carried to a successful issue.

Where the ligature is absorbed it is replaced by new tissue and there is no interval at any stage between the living material and ligature: the arrangement of the fibres in the new tissue is influenced by the structure of the ligature and the mode in which it has yielded to solution. Although it is well known that skin and bone grafts may live for an hour or so detached from the body, yet it is not possible, as some have supposed, for a dead animal ligature to come to life again; but, as Lister explains, "the new tissue takes as a model the old and forms "at its expense, the old tissue is absorbed by the new, "and as the old is absorbed new is put down in its "place."

Figures 113 p. 256 and 115 p. 257 shew tendon ligatures entirely replaced by young connective tissue.

This is also well seen in figure 104: the common carotid and subclavian were tied by Barwell with ox aorta, and what is now visible in the specimen round the arteries is probably partly the ox aorta itself and partly young connective tissue which has taken its place (see also figures 127 and 128).

The accompanying illustrations shew sections of various ligatures at different dates and in various stages of absorption. When catgut was re-introduced by Lister it was prepared by soaking in carbolic acid, but was found to be too quickly absorbed: it is now prepared with chromic acid as well, which renders it much more resistant. The results obtained with carbolic catgut may be seen in Haynes' table (1874): in 20 ligations of large arteries he gives four cases of hemorrhage, two cases in which the artery was patent and not constricted, one case in which the artery was patent and constricted, two cases in which the artery was not constricted but was occluded by clot, and one case in which the artery was constricted and occluded by a clot. Callender (1878) also brought to the notice of the profession the doubt existing as to the trust to be placed in carbolic catgut ligatures when used for tying an artery in its continuity. Arnaud's experiments pointed (1880) in the same direction.

Figure 105 is a chromic catgut ligature which had been applied for three days round the carotid artery of a sheep: it shews very well the villi of the sheep's intestine from which the catgut was made: this mucous tissue does not add to the strength of the ligature and makes it more difficult to asepticise: the fissures, too, are objectionable, as they allow the cells to penetrate into the ligature and

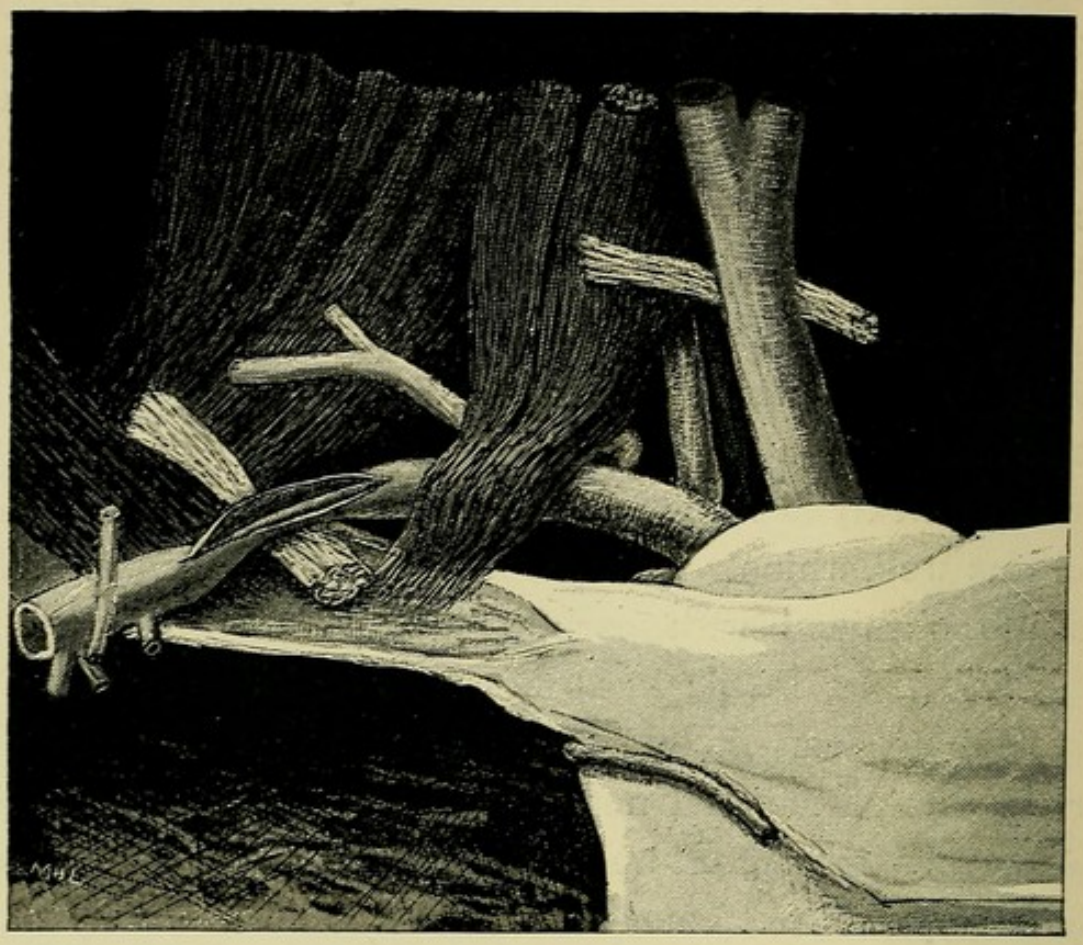


FIG. 104. Ligation of Common Carotid and third part of Subclavian. (Natural size.)

The patient survived the operation 15 months. The ligature used was ox aorta. The common carotid is quite pervious and unconstricted in any way. The third part of the subclavian is occluded; its coats were uninjured, and the lumen was not completely obliterated at the operation, but it is obstructed by new tissue substituted for the original clot. Two bands of white tissue which before dissection encircled the arteries at the seats of ligature are seen opened out: these are not wholly the ox aorta ligatures themselves, but partly living connective tissue which has been substituted for the dead ox aorta which has been absorbed. See figure 128.

This case is probably an example of the giving way of the first hitch of the reef-knot while the second hitch was being tied.

The case was under the care of Mr Barwell. Specimen in the Hunterian Museum, 3168. See also *Trans. Med. Chi. Soc.* Vol. LXII., 1879, and Barwell "On Aneurism," p. 393.

attack it from within. A dense mass of corpuscles has formed on the outer side of the ligature.

In figure 106 (7 days) the villi are fainter, but the mass of corpuscles (leucocytes and plasma-cells) are denser and beginning to find their way into the interior of the catgut: some absorption has already taken place on the outer surface.

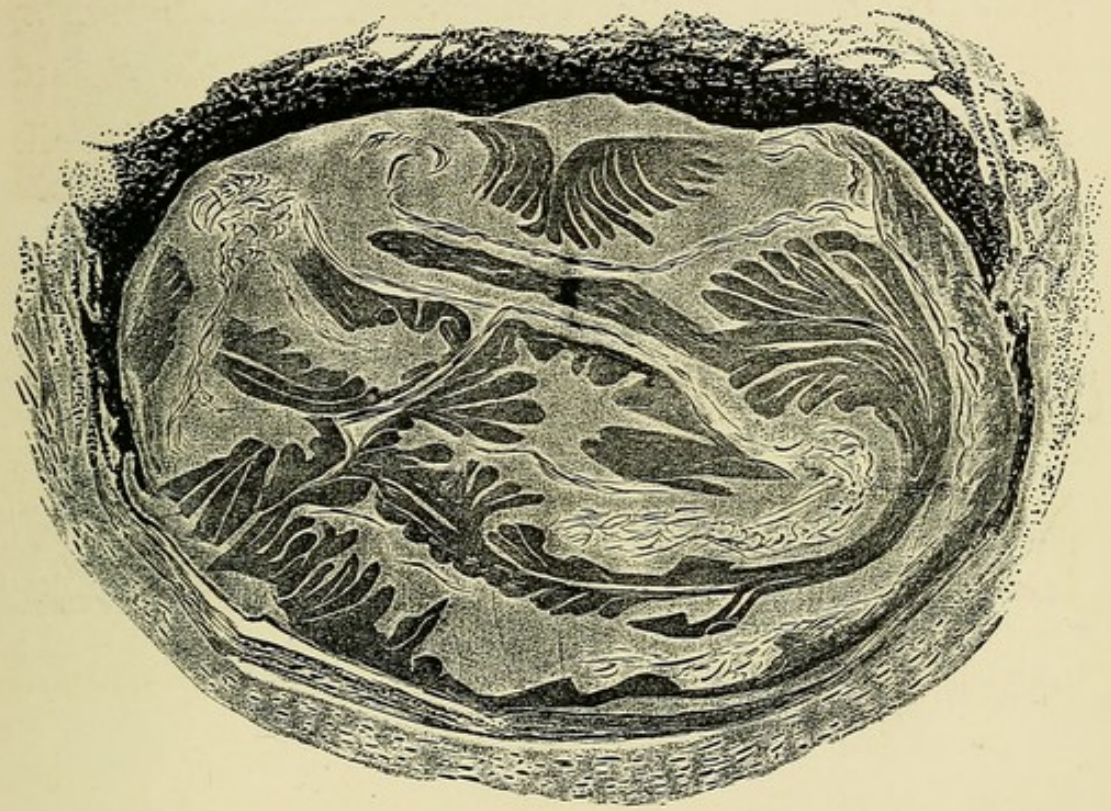


FIG. 105. Chromic catgut ligature which had been applied for 3 days round the Carotid of a Sheep (× 90).

The villi of the mucous coat of the sheep's intestine, from which the ligature was made, are clearly seen ; as are also the fissures formed by the twisting of the gut. A dense mass of cells has collected on the side of the ligature away from the artery.

Figure 107 is after ten days: the villi have disappeared, but large fissures are seen in the catgut, and into these the invading cells will soon find their way, indeed they have already done so in some parts.

Figure 108 is after 14 days: absorption has gone on very rapidly, at least three-quarters of the ligature having disappeared: the reason of this is that suppuration had occurred, which causes the rapid solution of catgut: contrast with this figure 109, from an aseptic case 44 days

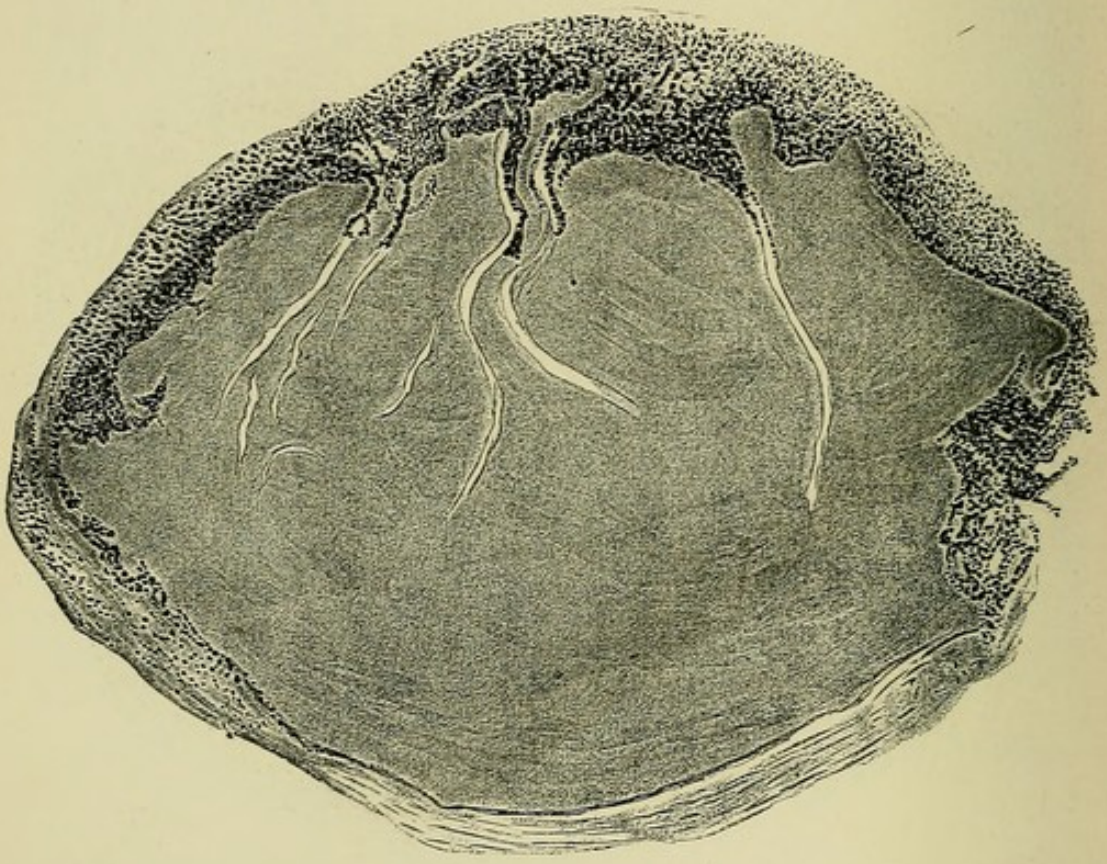


FIG. 106. Chromic catgut ligature which had been applied for 7 days round the Carotid of a Sheep (× 90).

The position of the mucous coat can just be discerned, but the villi themselves are indistinguishable. Corpuscles are invading the substance of the ligature on the side away from the artery, and are penetrating deeply into it along the interstices formed by the twisting of the catgut.

after ligation; absorption is proceeding very slowly; this must have been an exceptionally resistant piece of catgut.

Figure 87 page 215, shews a catgut ligature which had been applied round a human carotid; the wound healed

by first intention and the patient survived 108 days; it is seen that a small portion of the ligature is still unabsorbed.

On the other hand, in figure 91, page 225, the carotid of a horse 51 days after ligation, the large chromic catgut

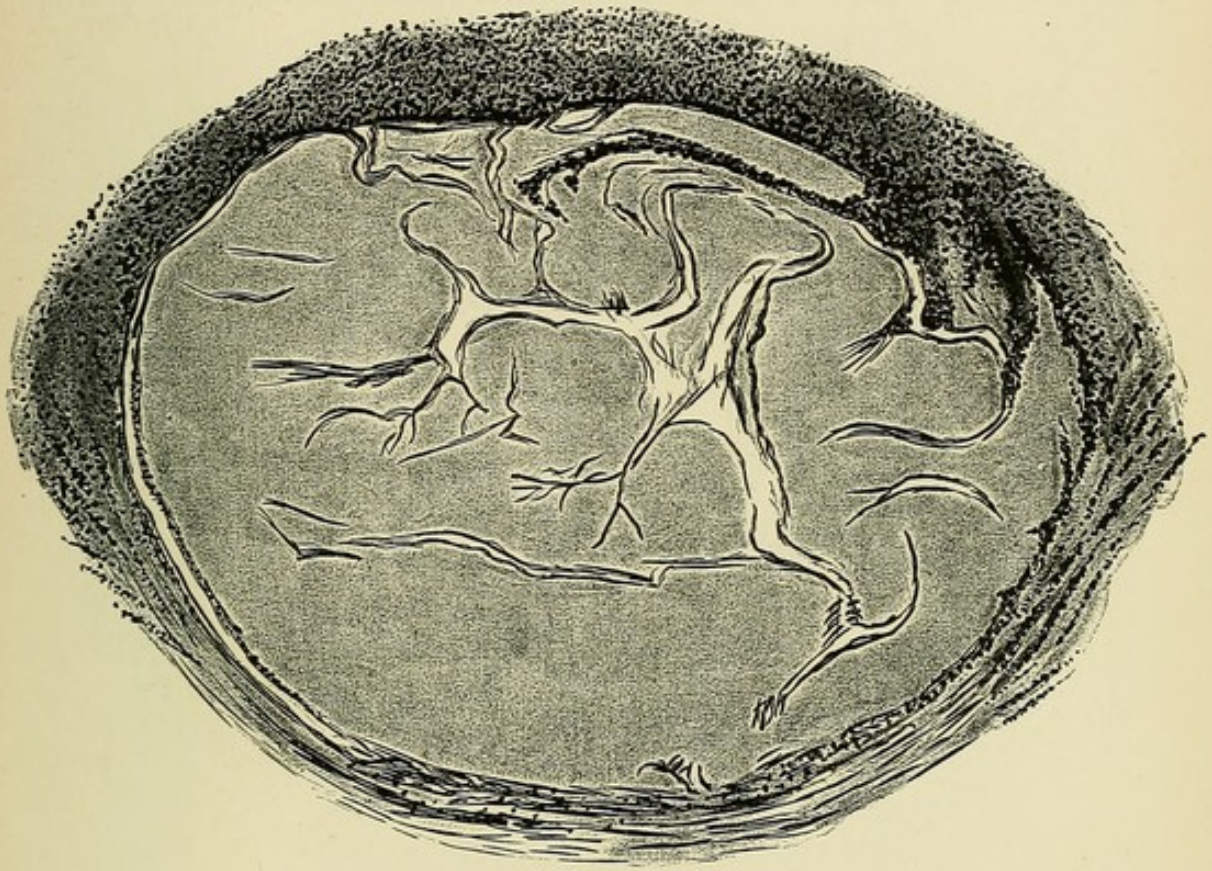


FIG. 107. Chromic catgut ligature which had been applied for 10 days round the Carotid of a Sheep (× 90).

Corpuscles are seen, as usual, in greatest number on the side away from the artery: they are penetrating into the substance of the ligature along its fissures. All indication of the mucous coat of the intestine, from which the catgut was made, has now disappeared.

which has been used has entirely disappeared; in this instance slight suppuration occurred.

It may be taken, then, that good chromic catgut of such a size as would be generally employed for a large artery, say nos. 4 to 6, will not be completely absorbed, if

the wound heal by first intention, till at least two months have elapsed.

The next ligature we tried was kangaroo tendon: it was originally recommended by Girdlestone and has many advantages: it is very strong, is homogeneous, that is to say, has no fissures into which the corpuscles can pene-

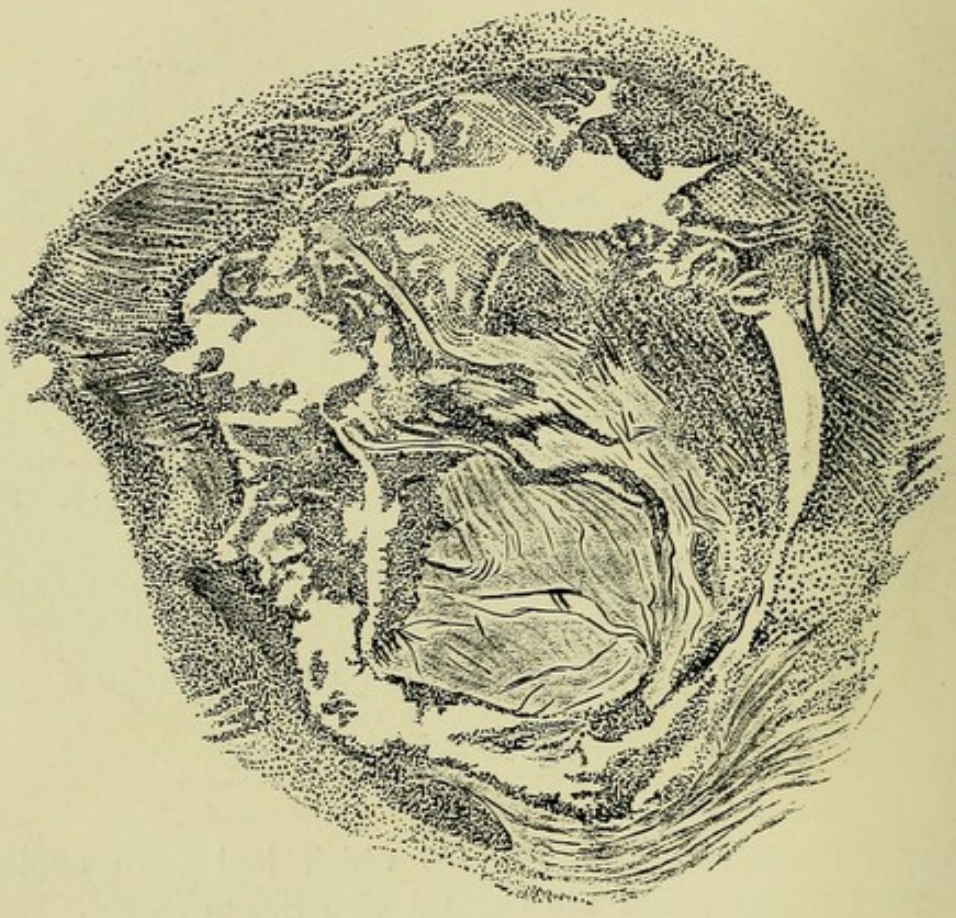


FIG. 108. Chromic catgut ligature which had been applied for 14 days round the Carotid of a Horse (× 45).

Suppuration occurred, and the catgut is being rapidly broken down.

trate: it can easily be made aseptic, and undergoes absorption from the surface only, except at the cut end.

Figure 110 shews this ligature 14 days after application, but as the drawing is made from the cut end it must not be taken as a guide to the rate of absorption of the loop.

Figure 111 shews a portion of the loop of kangaroo tendon which had been applied for 21 days: it is to be noticed that there are no fissures in it along which the

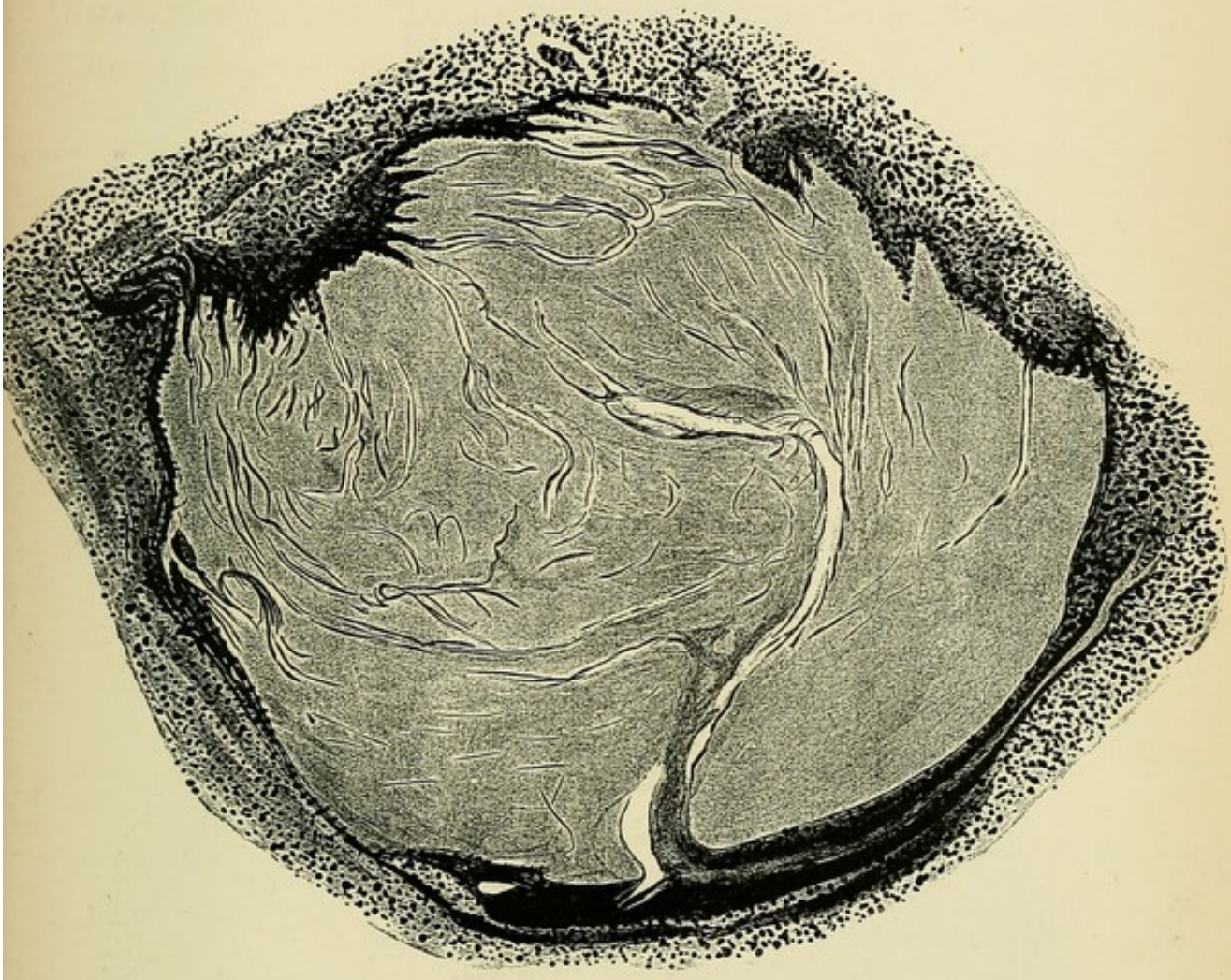


FIG. 109. Chromic catgut ligature which had been applied for 44 days round the Carotid of a Sheep (× 90).

Absorption by corpuscles is progressing all round the ligature, and at one point the cells have penetrated into the interior of the ligature. This is an exceptionally resistant specimen of catgut, otherwise it would have been broken up by this date.

corpuscles can find their way, absorption therefore only occurs at the surface: this should be contrasted with figure 107, shewing catgut after only ten days.

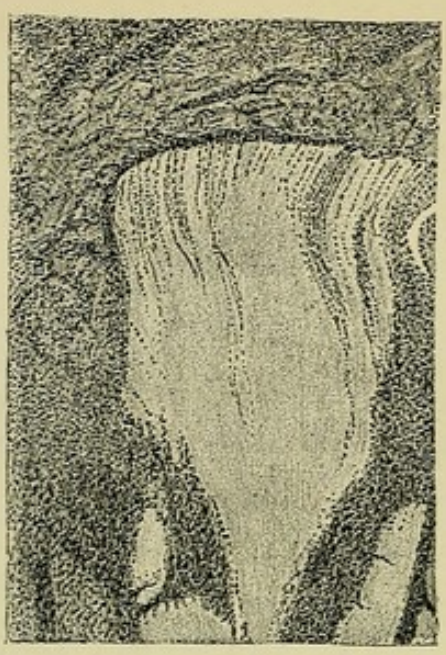


FIG. 110. Kangaroo tendon—14 days (× 90).

The portion of ligature represented is the cut end, the section being longitudinal: it shews the invasion from the free end.

The tendon was used to ligature the carotid of a sheep, and was allowed to remain 14 days.

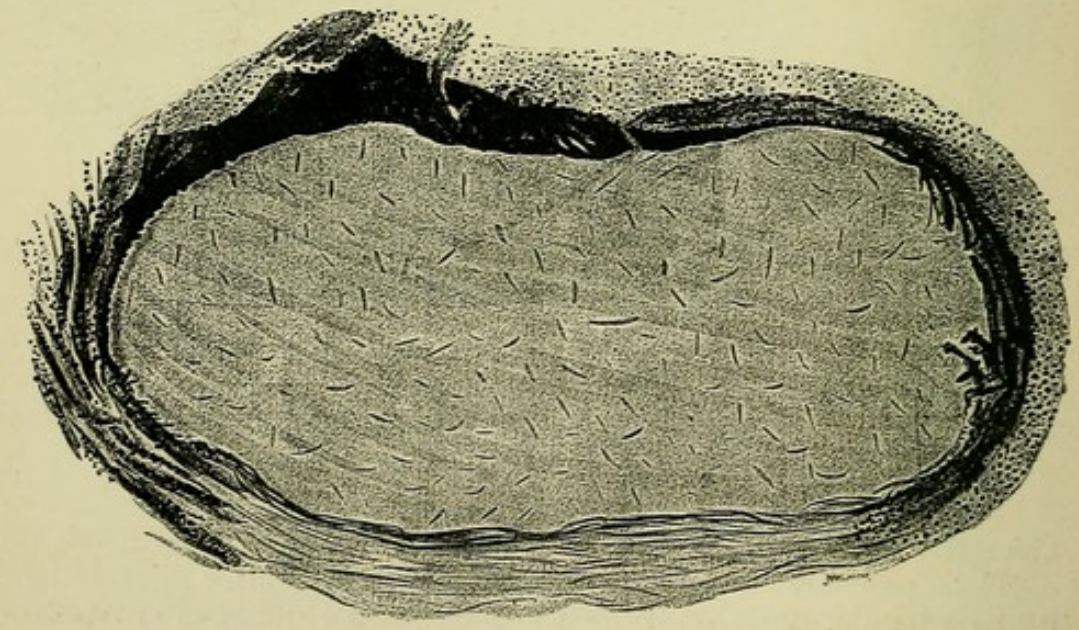


FIG. 111. Kangaroo tendon ligature which had been applied for 21 days round the Carotid of a Sheep (× 45).

A mass of corpuscles is seen covering the ligature on the side away from the artery. The superficial part of the ligature is being invaded, especially to the right, where absorption has commenced, it is to be noted, at the surface only.

Figure 112 shews kangaroo tendon after 73 days: there is still a considerable portion of the ligature left: it is seen how the absorption has occurred only on the surface, and the ligature is not split up.

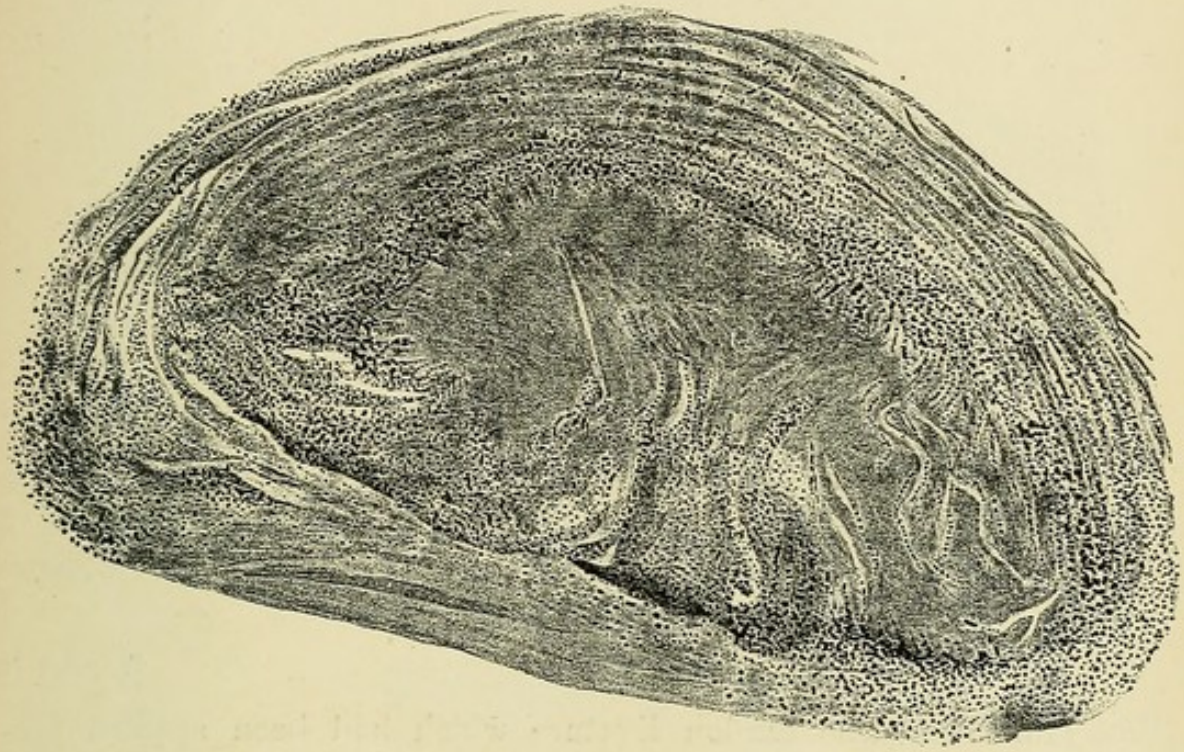


FIG. 112. Kangaroo tendon ligature which had been applied for 73 days round the Carotid of a Sheep (× 45).

The tendon has undergone considerable absorption at its surface. Its original size and shape can be discerned. The cells are only now beginning to penetrate deeply into the ligature.

Figure 113 shews the site of a kangaroo tendon 77 days after application: the ligature has completely disappeared and its place is taken by a mass of cells.

Figure 114 is the site of a kangaroo tendon 79 days after application: a few scattered fibrils are all that remains of the ligature.

Figure 115 illustrates the manner in which new connective tissue is laid down in the place previously occupied by the ligature.

Figure 116 shews what remained of a kangaroo tendon 131 days after application: there is a small portion in the

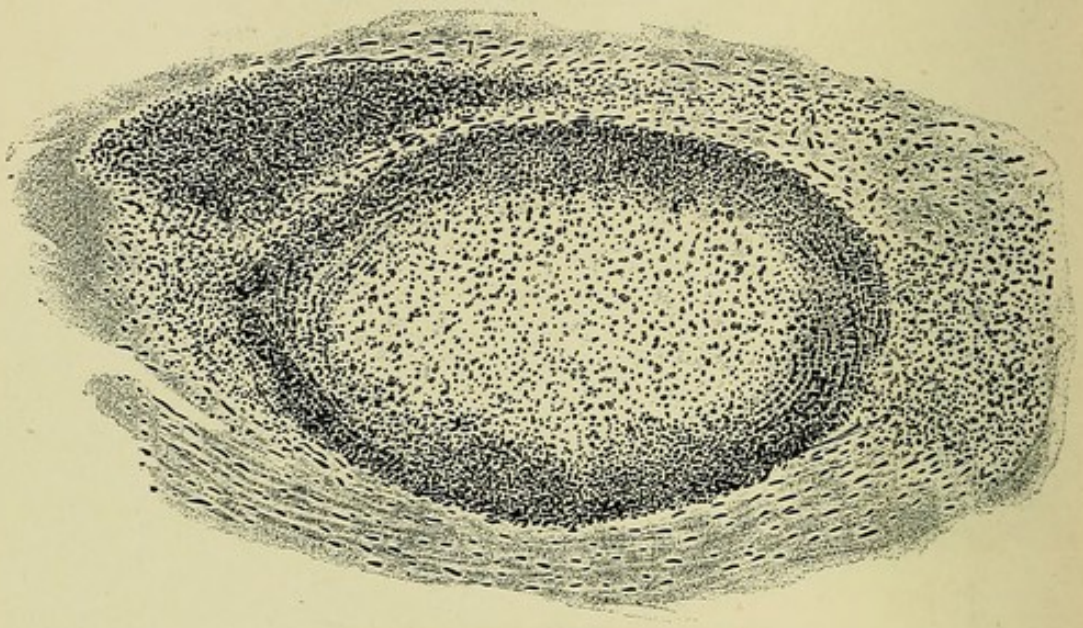


FIG. 113. Kangaroo tendon ligature which had been applied for 77 days round the Carotid of an Ass (× 90).

The tendon has entirely disappeared, but its site is clearly marked by the presence of a crowd of corpuscles.

The same appearance is shewn in other specimens, notably in a case in which a large kangaroo tendon had been applied round the carotid of a horse 165 days before death.

centre which has not yet been absorbed, and is as yet unaffected: absorption must have gone on from the surface only, or the ligature would have been more quickly absorbed.

Pitts ligatured the common femoral of a man with kangaroo tendon and 13 months later, when the specimen could be examined, no trace of the ligature could be found

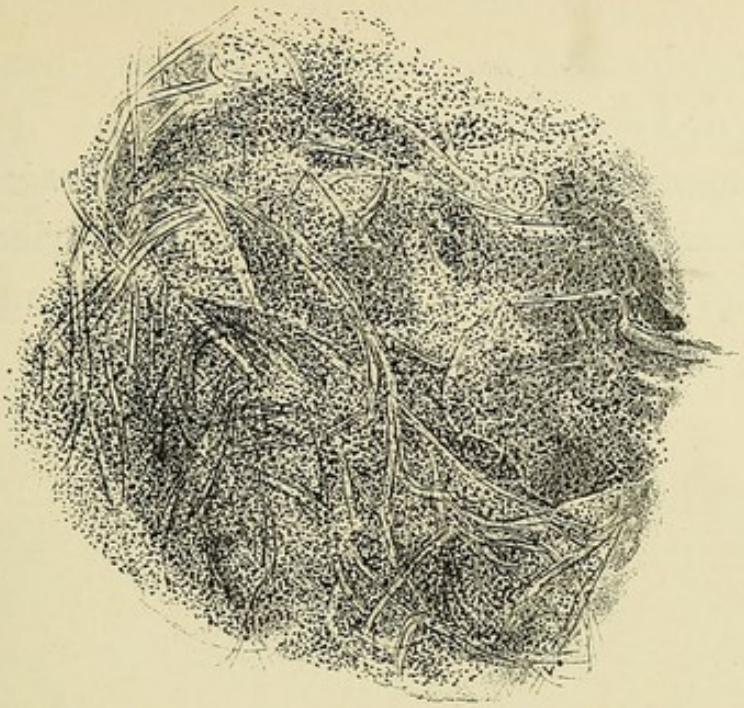


FIG. 114. Section of Kangaroo tendon which had been applied for 79 days round the Carotid Artery of an Ass (× 90).

The fibres are lying in all directions, and the site of the ligature is occupied by a mass of corpuscles.

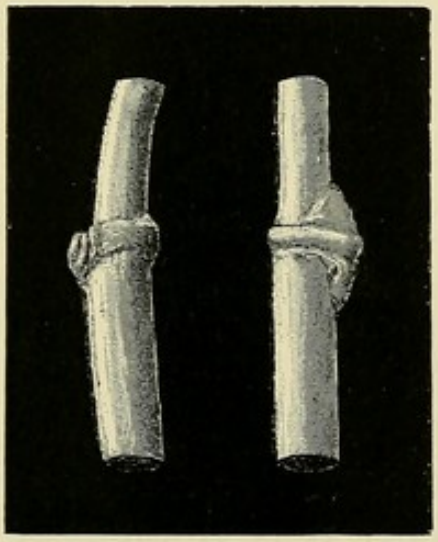


FIG. 115. Two Carotids of the Ass: ligatured with Kangaroo tendon.

The left-hand artery is 77 days after operation, and is from experiment No. 37. The right-hand artery is 67 days after operation, and is from experiment No. 29.

On transverse section of the loops of the ligatures they are seen to be entirely replaced by young connective tissue, the tendon band being replaced by one of fibrous tissue.

even with the microscope. We may infer then that kangaroo tendon will not have totally disappeared till at least three or four months have elapsed.

A specimen of reindeer tendon was kindly sent to us by Dr Kriedener of Archangel, in Russia: we tried it on the carotid artery of a horse, and figure 117 shews a section of it after 14 days: it is becoming absorbed somewhat

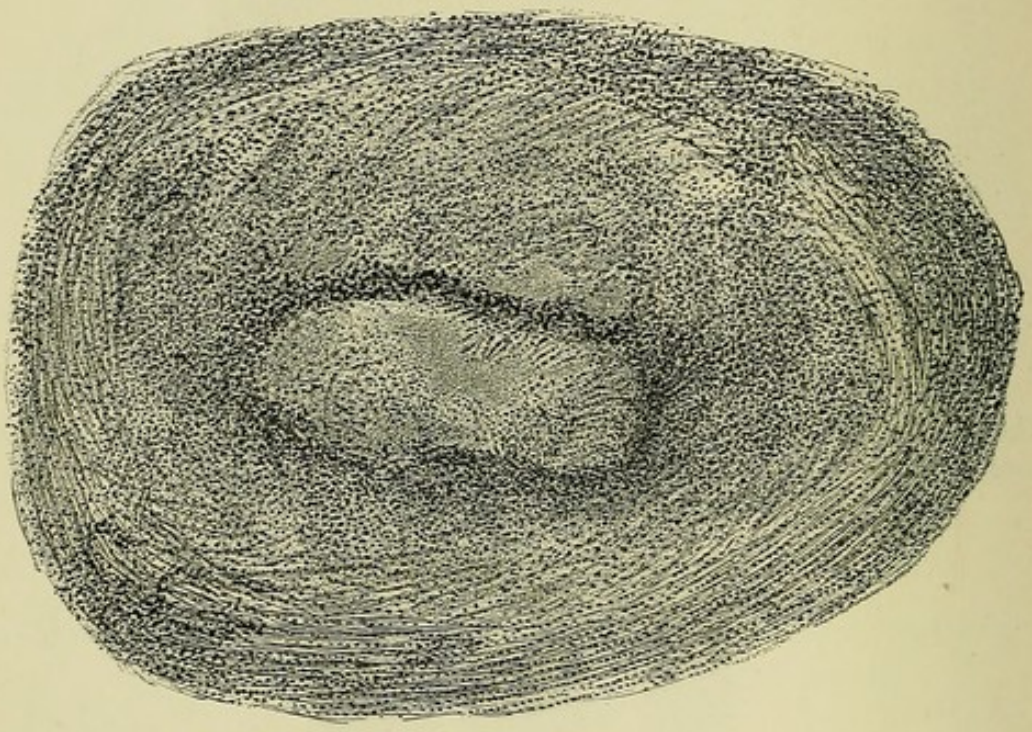


FIG. 116. Kangaroo tendon—131 days (× 90).

The tendon was applied round the carotid artery of a horse and allowed to remain 131 days. Portions of the tendon are still seen to be unabsorbed; it originally occupied nearly the whole of the part illustrated. The lower side was that next the artery.

rapidly: the structure of this tendon is seen in the lower part of the drawing: it is subdivided by a network of connective tissue: into these fissures the corpuscles find their way, and for this reason reindeer tendon is inferior to kangaroo. Contrast figure 111, a kangaroo tendon after 21 days.

The next ligature is silk: figure 118 shews an ordinary silk ligature (Chinese twist) which had been applied round the carotid of a sheep for 42 days: the corpuscles are beginning to find their way into its interstices. Floss silk has been used by Langton on account of its softness: it

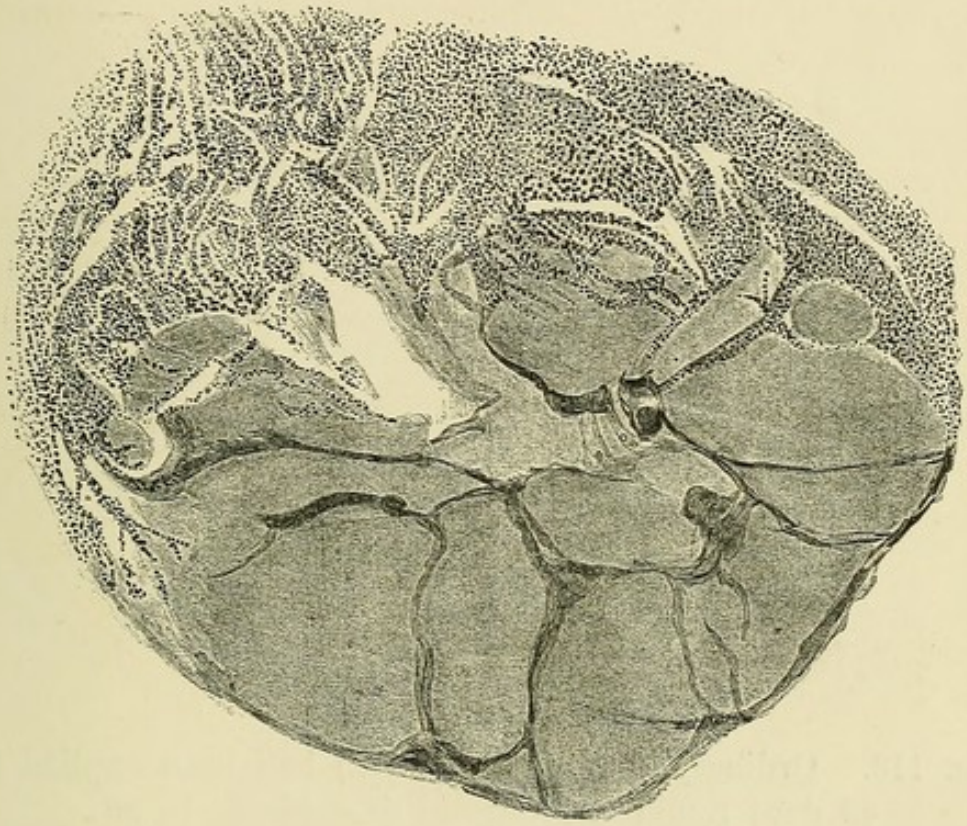


FIG. 117. Reindeer tendon ligature which had been applied for 14 days round the Carotid of a Horse (× 90).

The tendon is not homogeneous like that of the kangaroo, but is divided into separate fasciculi, which are united by connective tissue. The ligature is undergoing rapid absorption by corpuscles, which have collected in large numbers on the side away from the artery, their attack being facilitated by their advancing along the areolar planes between the fasciculi.

makes a very good ligature: figures 119, 120, 121 shew portions of such a ligature 14 days after application: owing to its loose structure the corpuscles quickly get into its interior.

Silkworm gut is shewn in figures 122, 123, 124, 125; one specimen was examined 21 days after application: absorption has not yet commenced. We have no experiments shewing whether this ligature becomes absorbed or not, but no doubt it does after a long time, perhaps a year or two (see figures 123, 124, 125).

FIG. 118. Ordinary silk ligature which had been applied for 42 days round the Carotid of a Sheep (× 90).

Leucocytes have collected in considerable numbers on the side away from the vessel and are commencing to work their way into the substance of the ligature. The drawing shews the structure of a silk ligature and, on the left, the commencing penetration of the corpuscles between the cords composing it.

Mr R. H. Clarke informs us that plaited horsehair makes an excellent ligature, and after immersion in oil for some time becomes beautifully supple. It undergoes slow absorption: compare figure 126.

Ox aorta ligature is shewn in figure 127: it had been placed in the connective tissue of a rabbit for 24 days: it is undergoing rapid absorption and splitting up readily.

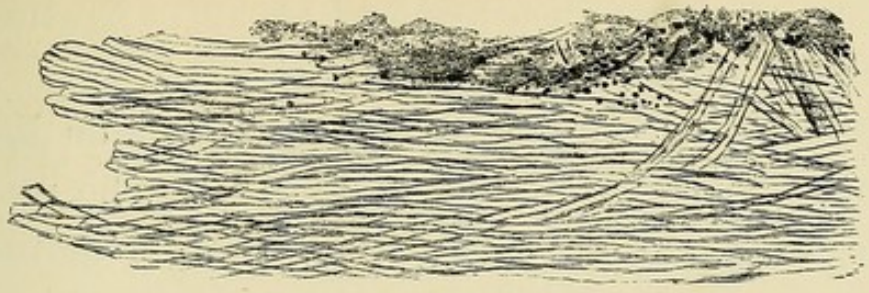


FIG. 119.

FIG. 120.

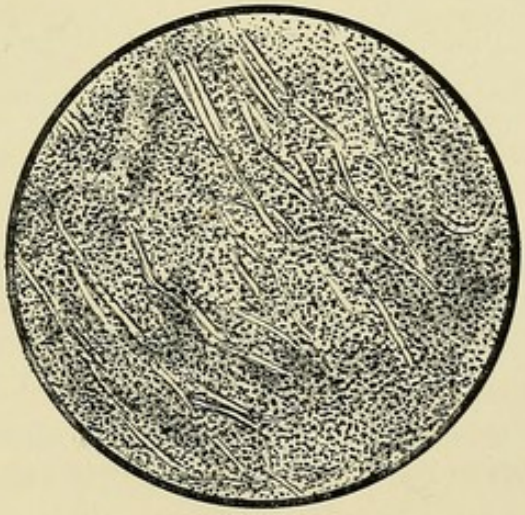


FIG. 121.

FIGS. 119, 120, and 121. Floss silk ligatures after use (× 95).

The ligature had been applied round the carotid of a horse for 14 days.

Fig. 119 shews the floss silk fibrils almost uninvaded by cells. It was taken from the interior of the encircling loop.

Fig. 120 shews the silk fibrils moderately invaded. It is from the surface of the encircling loop.

Fig. 121 shews the fibrils lying in a mass of plastic material. It is from the free (cut) ends of the ligature.

Figure 128 shews absorption of ox aorta ligature after 15 months' sojourn in the human tissues.

When shewing sections of kangaroo tendon ligatures undergoing absorption to Sir Joseph Lister, he remarked that the best material for an absorbable ligature was white fibrous tissue, of which the outer coat of an artery

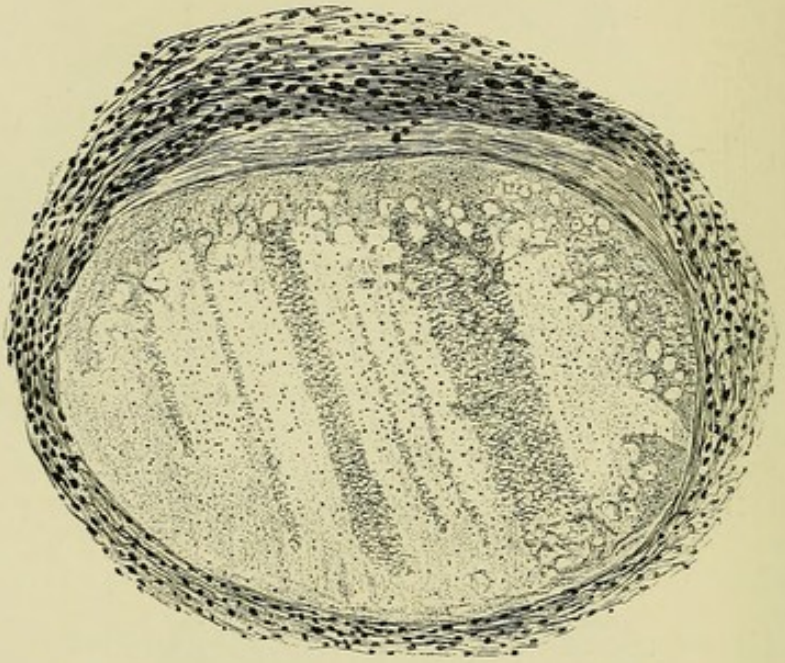


FIG. 122. Silkworm gut ligature which had been applied with eight others for 21 days round the Carotid of a Horse (× 200).

Corpuscles have collected, especially on the outer side, but no absorption whatever has occurred. The oblique markings were probably produced by the razor; the other appearances are due to the structure of the silkworm gut.

consists, and that the purest white fibrous tissue in the body was the peritoneum; he thought therefore that the best material for a ligature would be peritoneum: indeed he had himself tried it and with success on an artery of a calf.

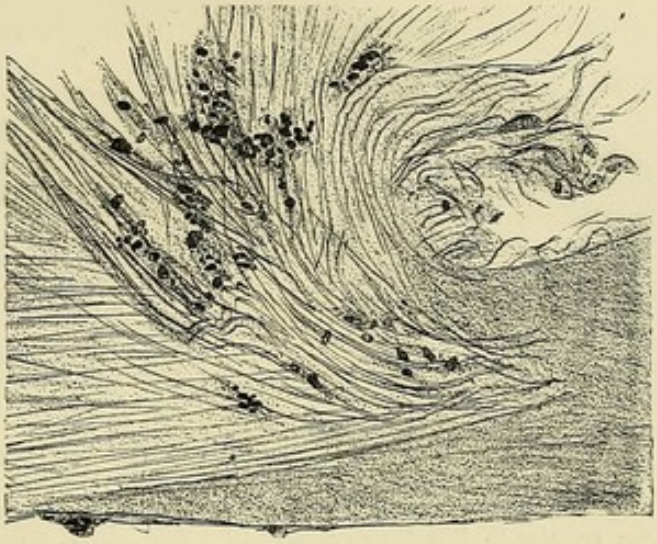


FIG. 123.

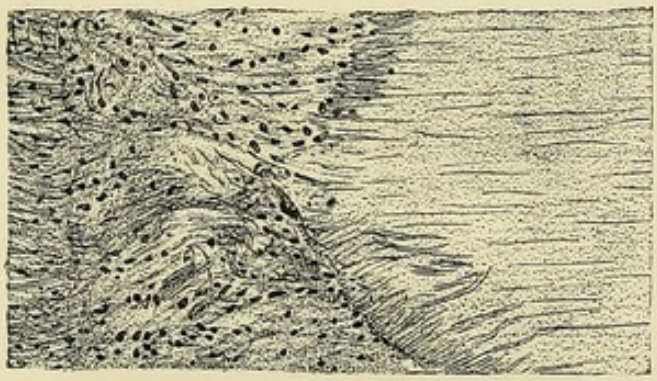


FIG. 124.

FIGS. 123, 124. Absorption of silkworm gut (× 250).

Drawings shew portions of a silkworm-gut ligature which had been embedded for 15 months in the tissues of a man. There was very little change, but at the ends and a few other places the capsule was firmly adherent and absorption was in progress.

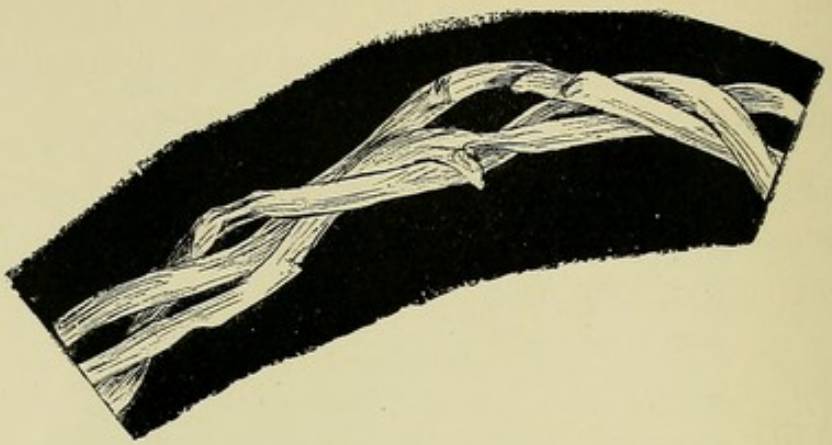


FIG. 125. Absorption of silkworm gut (× 9).

From specimen kindly presented by Mr R. H. Clarke.

A ligature composed of three strands of silkworm gut twisted together was introduced into the larynx of a horse (for the cure of roaring) and allowed to remain 16 months.

The gut is thinned, roughened and brittle.

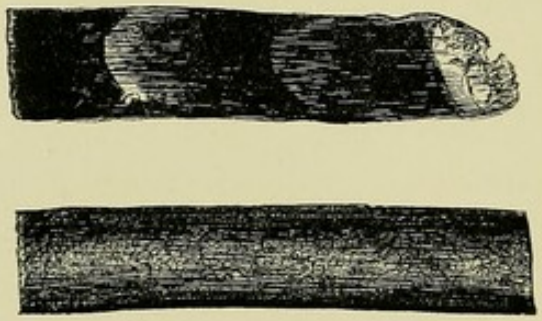


FIG. 126. Absorption of horsehair ligature (× 45).

From specimen kindly presented by Mr R. H. Clarke.

A plaited horsehair ligature was introduced into the larynx of a horse and allowed to remain five months. Only portions of the ligature could be found; they were greatly thinned, but the surface is even.

Clarke has found a horsehair ligature in the larynx of a horse more than a year after introduction.

Accordingly we obtained from the butcher a large piece of the parietal peritoneum of an ox: it was stretched out, dried, cut into strips and twisted into ligatures, which were prepared in chromic and carbolic acids. Messrs M^{c}Farlan of Edinburgh also kindly prepared some for us: the ligatures were made of various sizes and from 18—20 inches (45 to 50 cm.) long.

FIG. 127. Ox Aorta ligature, shewing absorption after 24 days (× 90).

The ligature was kindly presented by Mr Barwell.

The ligature is splitting up rapidly and the corpuscles are penetrating deeply into its substance.

Figure 129 is a peritoneal ligature which has been applied for 14 days: it is surrounded with plasma-cells and their progeny, but very little absorption (if any) has yet taken place.

Figure 130 shews the same ligature after 55 days: absorption is going on, but only at the periphery; the ligature is still holding well: this should be compared with

FIG. 128. Ox Aorta ligature 15 months after application (× 90).

The ligature was obtained from the subclavian in Barwell's case, the specimen from which is now in the Hunterian Museum (3168). *See figure* 104.

It is evident that at least to a great extent the ox aorta has been replaced by new tissue, in which there are many vessels. There is also to be seen a large amount of yellow elastic tissue passing in different directions. It is not certain whether this is the remains of the ligature or a part of the new tissue replacing it.

Paget says that elastic tissue is not commonly formed in the first construction of a scar, but appears in it as late as twelve months after its first formation.

figure 112 page 255; (kangaroo tendon after 73 days); the peritoneal ligature seems to resist absorption longest.

With respect to the time taken in the absorption of the various ligatures it would seem that chromic catgut will resist absorption sufficiently to continue to hold the artery for a month or more; remains of it will be found after 100 days: kangaroo tendon will remain efficient for two months, and silk for longer still; reindeer tendon is about equal to chromic catgut and peritoneal ligature to kangaroo tendon; silkworm gut lasts longest of all, but it is not very easy to use.

The rate of absorption of ligatures has received a good deal of attention from time to time. Porta, as the result of 400 experimental ligatures, found that in from one to two years after operation 70 per cent. of catgut ligatures had become absorbed, 36 per cent. of silk ligatures, 66 per cent. of hemp and flax ligatures, and 20 per cent. of horsehair; the order of rapidity of absorption then was catgut, hemp, silk and horsehair.

Senn experimented on sheep with catgut, silk, silkworm gut and horsehair. "Catgut," he says, "appeared to "constitute itself a part and parcel of the vessel-tissues "until it was replaced by substitution by a ring of or-"ganized tissue, which served as a material support to the "vessel until cicatrization was completed, thus preserving "the continuity of the vessel. All the remaining kinds of "ligatures appeared to act as foreign bodies, as far as the "vessel tunics were concerned, and invariably produced a "solution of continuity after a sufficient length of time had "elapsed. They were usually found encysted in the mass "of connective tissue between, and some distance from, the

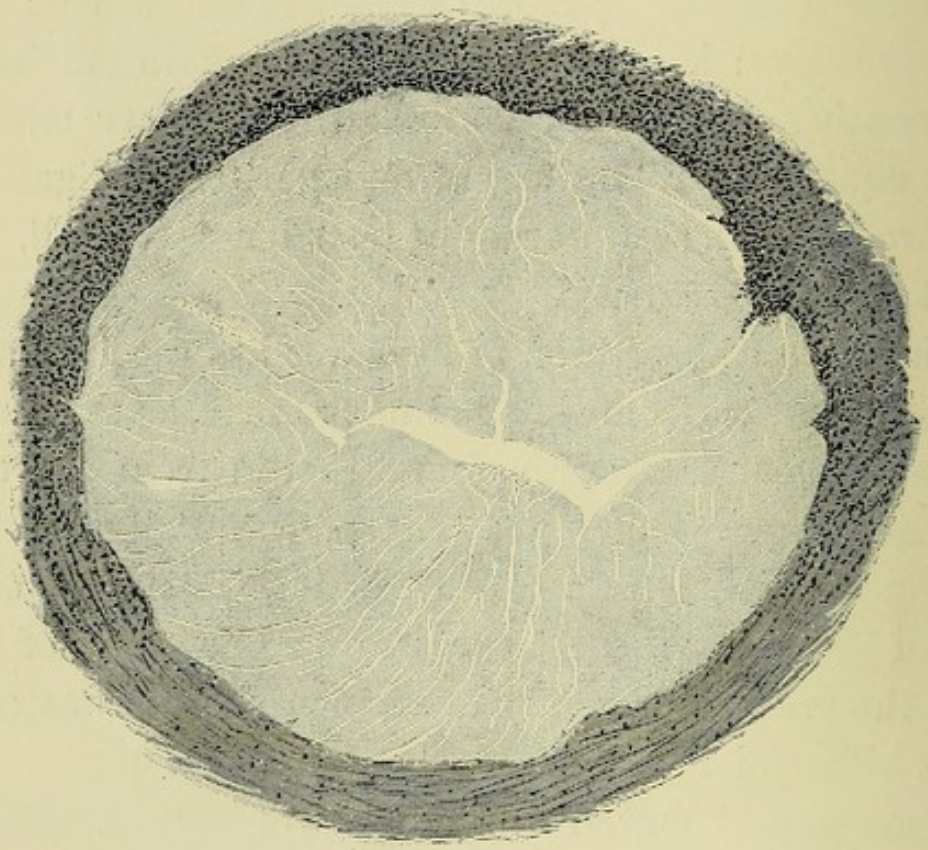


FIG. 129. Peritoneal ligature after 14 days (× 50).

The ligature had been applied for 14 days round the carotid of a horse. The folds formed in the manufacture of the ligature by twisting the membrane are seen separated by (white) spaces. The side from the artery is uppermost. Absorption has scarcely begun.

FIG. 130. Peritoneal ligature after 55 days (× 50).

The ligature had been applied for 55 days round the carotid of a horse. The spaces between the folds into which the membrane was thrown in manufacture are seen as white streaks: and it is along them that on the side away from the artery that invasion is occurring. It is only on this side that absorption is taking place.

"ends of the vessel." Senn therefore decides in favour of catgut: he found the earliest time in which the loop became absorbed was 21 days, the knot alone remaining; after 52 days no trace of the ligature could be found.

A case of innominate aneurism, treated by Pollock by distal ligature of the carotid and third part of the subclavian, is related by Dent: kangaroo tendons were used for the ligatures, and they were examined microscopically after the death of the patient, which occurred on the tenth day.

Dent found the tendon "infiltrated with small round "granulation cells or leucocytes. The cells, which are "readily stained by logwood, have penetrated into the "interfascicular spaces, and tend to split up the tendon "into longitudinal bands." The paper is illustrated by two good plates. "The operation was not performed "under the antiseptic system," but the tendon seems to have been acted upon more rapidly than in our experiments, for even in those cases which did not heal by first intention, the tendon by the tenth day was scarcely altered except at its cut end.

Alban Doran has described the process by which ligatures applied round the peduncle in ovariotomy become absorbed: the portion of the peduncle beyond the ligature does not die but becomes adherent to neighbouring structures and to the portion of peduncle proximal to the ligature, which thus becomes bridged over and finally absorbed. There is a specimen in St Thomas's Hospital Museum (FF 48[1]) shewing this process in an ovarian peduncle which had been tied nine months previously with silk, which is now partly absorbed.

In two post-mortem examinations on cases in which ovariotomy had been performed some considerable time previously, we had the opportunity of looking for the silk ligatures which had been used for the peduncles: one case was 18 months after operation, and here only the knot of the ligature could be found; the other was three years after operation, and in this case no trace of the silk ligature was discoverable.

With respect to hempen ligatures, Doran found in one case in which whip-cord had been used, that after a year only the knot remained; in another after seven months not a trace of the ligature could be found. (See Hunterian Museum Specimen, No. 4560.)

Recently Thomson of Dorpat has made experiments with different ligatures to ascertain which were best for suturing the uterus after Caesarean section: he found that with carbolised catgut the loop was absorbed in ten days, and the knot seven days later; chromic catgut was unabsorbed at the end of 64 days, and during the same time silkworm gut remained quite unaltered: silk was partly absorbed in fifty days and wholly absorbed in sixty-four days: he concluded that silk was best at least for his purpose, as it became absorbed in a suitable time and could be easily rendered sterile: chromic catgut and silkworm gut he rejected as unabsorbable, while carbolic catgut was too quickly absorbed to be trustworthy.

It seems then that many ligatures will remain unabsorbed for a sufficient time to do their work when applied to a great artery; which is best will be considered when we come to the question of the choice of the ligature.

CHAPTER XI.

SUPPURATION AFTER LIGATION.

Inflammation. Bacteria and Suppuration. Suppuration and Ligatures. Sepsis as a cause of hemorrhage. Ulceration of Arterial Wall from without: Pyæmic Pus, Bubo, etc. Sepsis as a cause of aneurism. Condition of wall in acute Arteritis. Ulceration of the Arterial Wall from within: Ulcerative Endocarditis. Sepsis as a cause of gangrene. Conclusions.

Whenever a damage is done to any tissue of the body inflammation ensues. The virulence of the process varies greatly: the injured part may heal without the formation of pus and without constitutional disturbance; or suppuration may take place of varying intensity from a few drops of so-called laudable pus to a profuse ichorous discharge of an infective and pyæmic character.

John Hunter divided wounds into two sorts, "those "which do and those which do not communicate ex-

"ternally," and he says, "those which do not communicate "externally seldom inflame, while those that do commonly "inflame and suppurate." In these sentences are embodied not only the principles of subcutaneous surgery, but also of those improvements in the treatment of wounds for which we are indebted to Sir Joseph Lister. Perhaps it would be more convenient if the term inflammation did not include the process of healing without suppuration, if we had, in fact, two words to express the two states of wounds which appear clinically so different. The minute processes, however, which occur in an injured part are so alike, be the healing by immediate union or by suppuration, and the term is so universally recognised, that any change is impracticable.

Suppuration is allowed on all hands to be associated in some way with the agency of bacteria. These form the injurious substances which, when present in an active state, delay healing and produce suppuration. They may reach the wounded surfaces in various ways from outside, or, if the organism be not healthy, they may be brought to the wounded spot by the blood. For practical purposes suppuration in surgical wounds may be looked upon as due to the entrance into them of germs from without.

The object of all antiseptic measures may be said to be to limit the inflammatory process—the leucocytic extravasation, etc.—and thus to obviate all interference with the plastic proliferation of the plasma-cells, upon which the cicatrisation process is dependent; if these measures are successful, the healing of wounds will approximate to what Senftleben described, namely, the healing of minute corneal wounds in rabbits by multi-

plication of the corneal corpuscles without the intervention of leucocytes.

In the old days one end of a ligature was brought out through the wound, and a process of ulceration was desired in order that the ligature might be discharged; with it came often a segment of the artery. This happened usually in from 5 to 20 days. It was always the period of greatest peril, because when the ligature became free it meant that complete severance of the artery had been effected. (Fig. 131.) Paul Bruns, in an elaborate monograph on the use of the temporary ligature, which his father and he practised at Tübingen, well says that the danger of the ligature is to be found, firstly, in suppuration, and secondly, in the complete solution of the arterial wall. Bruns maintains that by his method the adventitia is never cut through, and that therefore the continuity of the outer coat of the artery being maintained hemorrhage cannot happen. Holmes, Senn, and others have advocated the catgut ligature with the same idea.

When the coats are ruptured and suppuration happens, the thin adventitia in the loop of the ligature, whose ends have been cut short, becomes infiltrated and softened. Thus a complete solution of the vessel-wall is effected and the two sections of the artery are able to retract from each other. When the coats are not ruptured this does not happen unless the pus is of a very irritating character and the ligature is of silk or some material which resists absorption for a long time. In some of our experiments on horses and asses suppuration occurred: the tendon ligature became infected, a ring of pus formed around the

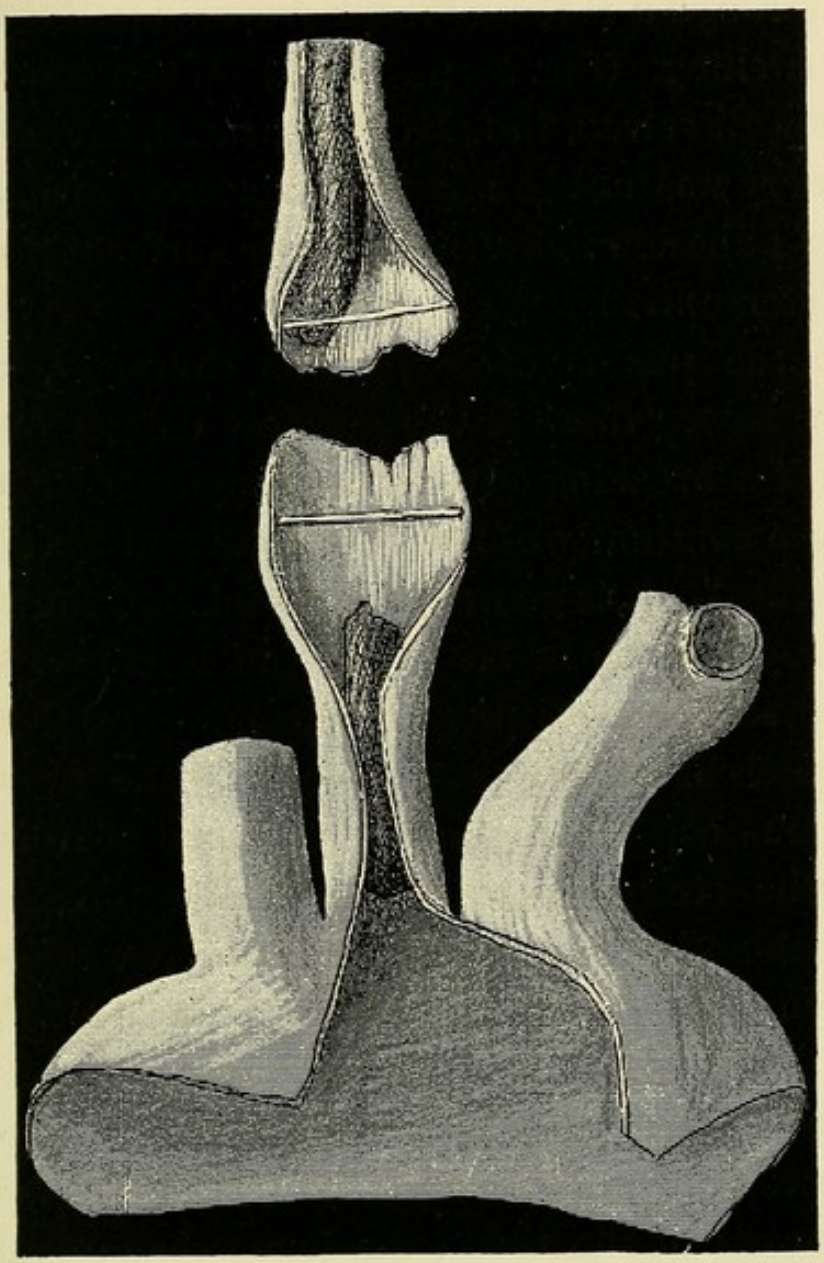


FIG. 131. Arch of Aorta with great vessels seen from behind. (Nat. size.)

From S. Bartholomew's Hosp. Museum, Spec. 1393.

The left carotid was ligatured for epilepsy in an old man. No hemorrhage occurred, but patient died 6 weeks later from mediastinal abscess. The vessel was found completely divided and the ends separated, open and softened. There were no adhesive changes, and hemorrhage was only prevented by non-adherent clots.

artery corresponding to the loop of the ligature and escaped through a fistulous track traversing the external fusiform swelling. (Fig. 58, page 147.) When, however, the tendon was dissolved, the pus disappeared, the continuity of the artery was not in any way interfered with, and the process of adhesion between the inner surfaces proceeded without interruption.

When the pus is of a pyæmic character, then, as Stromeyer says, "there is the greatest danger of hemor-"rhage by the breaking down of the coagulum, the destruc-"tion of the continuity of the vessel-wall and the softening "of the tunics." It is generally believed, and the view was endorsed by Cheyne in his Hunterian Lectures, that the infective and ichorous pus of pyæmia owes the special effects which it produces not only to the presence of ordinary pus cocci, but also the presence of other cocci, such as the streptococcus of spreading inflammations, whose combined actions on the organism cause the condition known as pyæmia. There have been at St Thomas's Hospital within the last three years two cases of pyæmic ulceration of arteries: in both the pus made its way from without through the vessel-wall. The first case was one of acute necrosis of the tibia; the hemorrhage came from the posterior tibial artery, the pus around the vessel having caused solution of its wall. The second case was one of hemorrhage from a pyæmic abscess of the neck due to ear disease. (Fig. 132.) The common carotid was ligatured, but the wound made to tie it soon suppurated. The drawing shews the acute arteritis in this case. It is noteworthy that the suppurating process commenced outside the vessel and not in the clot, which might be

supposed to offer a very nutritious soil for the growth of pyæmic cocci.

Symonds, in the *Pathological Society's Transactions* for 1884, describes a case in which sloughing of the tissues

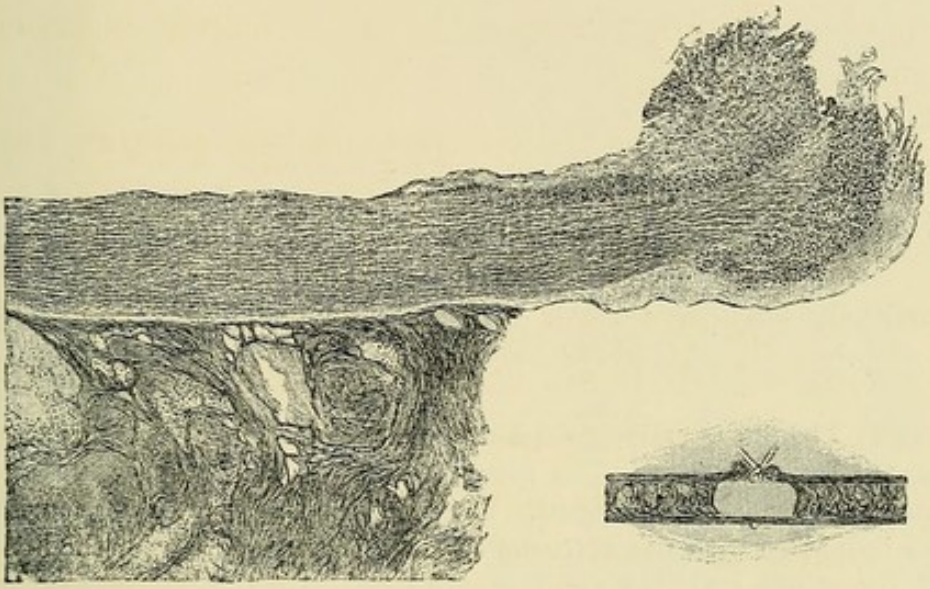


FIG. 132. Acute Arteritis of Human Carotid (× 16).

The common carotid of a boy was ligatured for hemorrhage: he was suffering from pyæmia, secondary to ear disease, from which he died 13 days after ligation. The skin incision was healed, but the artery at the seat of ligature was surrounded by pus. The small diagram to the right represents the condition of the artery. The wall had given way under the knot and the pus had entered the interior of the artery there, but hemorrhage had not occurred as the carotid was blocked on each side by clot. The inflamed and infiltrated condition of the frayed edge of the vessel-wall is seen in the drawing. The pyæmia had infected the seat of ligation through the blood. The giving way of the vessel appears to have been from without inwards, for columns of cells are seen penetrating the middle coat and between the middle and inner coat, the intima itself being only thickened.

From a patient under the care of Mr Clutton.

and hemorrhage followed a severe lacerated wound and compound fracture of the forearm. After amputation there were discovered two aneurismal dilatations of the radial artery and one on the interosseous artery. The hemorrhage had come from one of the dilatations of the

Figs. 133, 134, 135, 136. Effects of Septic Inflammation on Arteries.

Reproduced from Trans. Path. Soc. Vol. xxxv. (1884) *by kind permission of Mr Charters Symonds.*

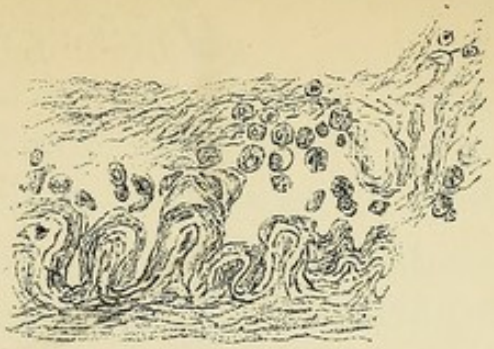
Fig. 133. The radial artery from a man aged 40 who had sloughing of soft parts of forearm following injury. The artery is laid open shewing two aneurismal dilatations, each containing coagula. The outer surfaces of the aneurisms were black and connected with the surrounding slough: from the larger the hemorrhage occurred, for at one point the wall was perforated. On the interosseous trunk was a similar small sac, a transverse section of this is given in Fig. 134, where (*a*) is the clot, (*c*) middle coat, (*d*) inflammatory cells due to suppuration in outer part of middle coat, (*e*) outer coat.

Fig. 135 is an enlargement of (*b*) in Fig. 134; it shews the wavy membrane of Henle and internal to it the proliferation of the endothelium.

Fig. 136 is from the brachial artery of another patient—a man (æt. 52) with suppuration in the stump of an amputation through arm: the wavy fenestrated membrane of Henle is seen, and external to it suppuration in the inner part of the middle coat of the artery: in the interior of the artery purulent clot was found; which alone prevented hemorrhage. (*a*) internal coat, (*b*) middle coat, (*c*) outline of external coat.

Fig. 133.

Fig. 135.



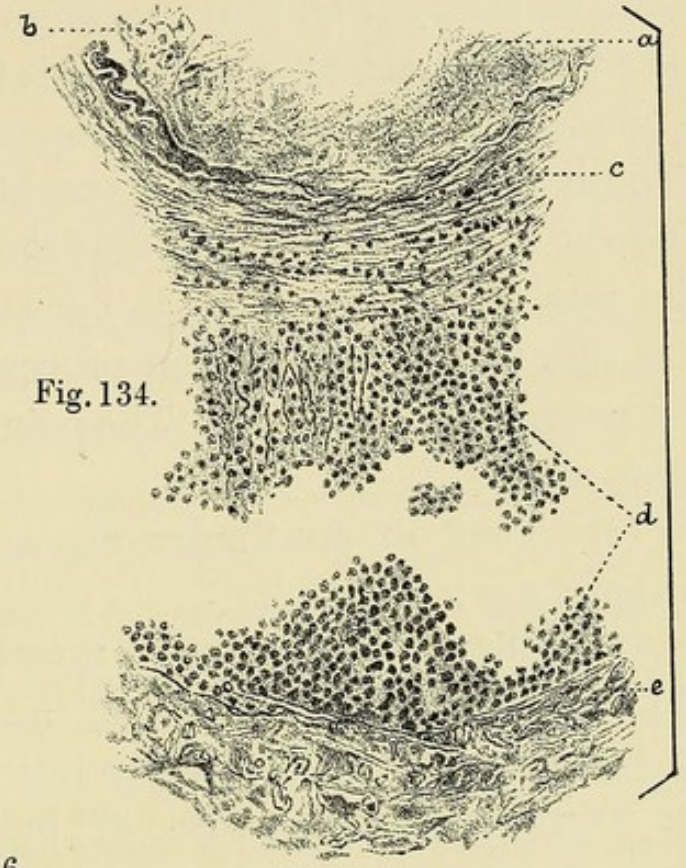
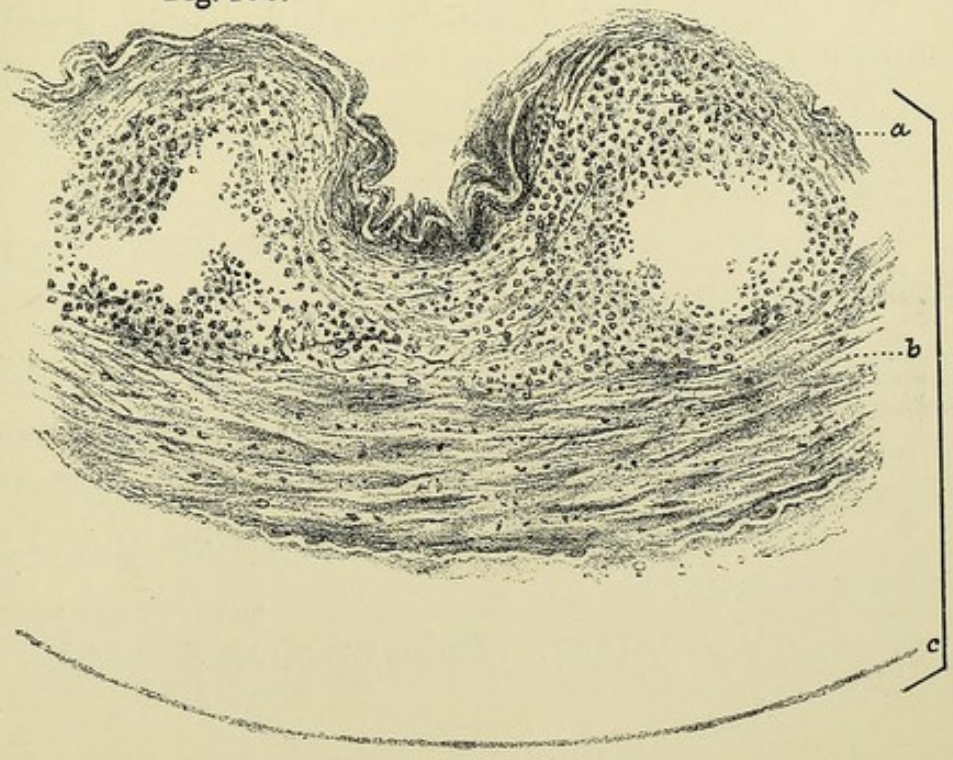


Fig. 134.

Fig. 136.



radial. (Fig. 133.) The aneurismal swellings were accompanied by separation of the middle and outer tunics by pus. Intimal proliferation and clotting had occurred in the aneurisms. In another case acute arteritis of the brachial artery (Fig. 136) was observed in an amputation stump. The artery was not constricted and a thin coagulum of blood had alone prevented hemorrhage. The deposit of pus was between the inner and middle coats, and the suggestion is made that the different situation of the pus in this instance as compared with the former was due to the suppuration process being able to attack the cut end of the artery. The points illustrated by these cases are:—

(1) That in continuity an aneurism forms before hemorrhage;

(2) That the suppurative arteritis and not the failure of clot formation is the cause of the hemorrhage.

Spreading ulcerative suppurations of other kinds, as for example Sheild's case of bubo opening into the femoral artery and vein, and the other similar cases mentioned by him, are instances of ulceration of arterial walls from without. In the same category may be cited the following Museum specimens:

1. St Bart.'s, No. 1388. Perforation of aorta by a mediastinal abscess.

2. St Bart.'s, No. 1439. Perforation of aorta by a psoas abscess.

3. St Bart.'s, No. 1540. Bubo opening femoral.

4. „ „ No. 1440. Suppurating gland of neck opening carotid.

5. Guy's, No. 1507. Bubo opening femoral.

6. Guy's, No. 1504[55]. Aneurism of femoral due to tubercular ulceration, in a case of hip-joint disease.

Further, the hemorrhage that commonly occurs in phthisis is due not solely to a tubercular process, but often, if not always, to a suppurative arteritis affecting the arterial wall where it is exposed in a vomica, producing first an aneurism and lastly rupture. It is necessary, however, to have a specific irritating pus before the wall of a large artery which is intact will be softened or injured by suppuration. We have known the popliteal artery to be bathed in pus for some weeks, and the only change which was noticeable (after amputation) was a thickening of the outer coat. If, however, suppuration (of a so-called healthy character) ensue when the coats are ruptured the adventitia soon gives way and the continuity of the vessel, which is the great safeguard against hemorrhage, is abolished.

A weakening of the vessel-wall, from septic processes occurring within it, is best seen in the embolism of ulcerative endocarditis. The septic embolus is followed at its point of lodgment by an aneurism. Several such cases can be seen in the Guy's, St Bartholomew's and St George's Museums. An interesting account of such a case is given by Langton and Bowlby, in the *Royal Medical and Chirurgical Transactions* for 1887; dilatations of the vessels first occurred and these then ruptured to form large, soft, thin-walled, false aneurisms. (See St Bart.'s Hosp. Museum, No. 1460 b.)

The Figures 137 and 138 shew very well an early and late stage of the process.

Spencer and Horsley, in an interesting paper on the control of hemorrhage from the middle cerebral artery

FIG. 137. Femoral Artery obstructed by Embolus. (Nat. size.)

From a man aged 50, admitted with gangrene of the left foot and also left hemiplegia. Post mortem: large vegetations of mitral valve, left femoral artery blocked by a clot, at one point the vessel is dilated. This was probably the site of a septic embolus, the greater part of the clot being due to a secondary thrombosis.

(Guy's Hospital Museum, 1506[31].)

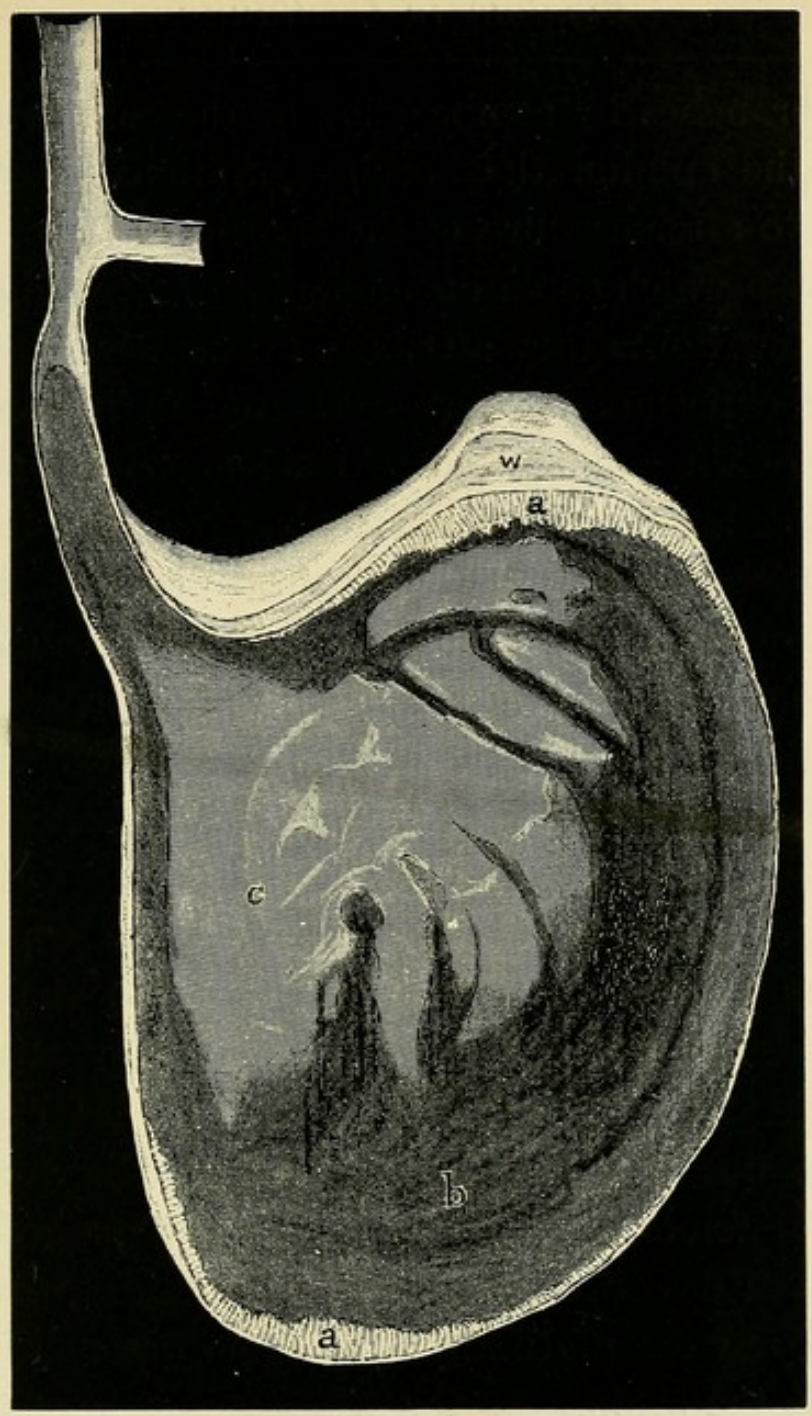


FIG. 138. Embolic Gluteal Aneurism. (Nat. size.)

From a woman aged 25. The aneurism was treated by ice and other means, but was not cured; the patient was too ill medically for further treatment of the aneurism, and five months after it was first noticed she died of heart disease, with embolism of the spleen and kidneys. The clot in the aneurism is comparatively recent; at both the upper and lower part there has grown into it for a considerable space a narrow fringe of connective tissue (*aa*), which has sprung from the wall (*w*). This is well seen, for at the time the drawing was made the aneurism had not been long in spirit and the recent clot (*b*) still retained its red colour, which contrasted with the white of the young connective tissue: the lighter part marked (*c*) contained blood.

The patient was under the care of Sir W. Savory. The specimen is in St Bartholomew's Museum (No. 1460 E) and was exhibited by Mr Edgar Willet at the Pathological Society, Feb. 1890.

and its branches by compression of the common carotid, express the opinion that brain symptoms coming on a day or two after ligature of the carotid are due to sepsis, while those which come on immediately after ligature or within a few hours they attribute to anæmia of the brain. In their experiments they shew that in monkeys compression of the common carotid artery arrests the pulsation of the brain in that part only, which is supplied by the branches of the middle cerebral artery of the same side; in other words, that the communication through the circle of Willis is not sufficient to compensate for the compression of the common carotid as far as the middle cerebral artery is concerned, the contrary being the case with the anterior and posterior cerebrals.

This explains, they say, the cases in which brain symptoms come on early after ligature of the common carotid. The cases which occur later they attribute to sepsis; but it would seem that some of the later cases also might be due to anæmia. In a case of ligature of the superficial femoral which we have seen gangrene came on the 18th day after ligature; the wound having healed aseptically by the ninth day: the same presumably might happen in the brain.

In such cases as are septic the sepsis would find a weak spot in the anæmic portion of the brain. In the great majority of the cases no doubt the hemiplegia is due to sepsis and anæmia combined. Any cause which increases the anæmia would increase the danger of paralysis: thus the hemorrhage for which the ligation may have been undertaken, or a recurrence of such hemorrhage after ligation, or a hemorrhage from the seat of

ligature, may predispose to the arrest of the already enfeebled circulation in the branches of the middle cerebral, and thus death of the portion of brain affected might occur, even without sepsis. Statistics seem to confirm this view.

Wyeth in his tables gives 772 cases of ligature of the common carotid, 231 for wounds, 264 for tumours, 237 for aneurisms, and 40 for nervous disorders. We have counted up the cases in which hemiplegia is said to have come on, and find that it occurred 23 times after ligature for hemorrhage, 17 times after ligature for tumours, 16 times after ligature for aneurism, and once only after ligature for nervous disease. The classification is not very satisfactory for our purpose, for it is probable that hemorrhage had occurred before ligature in some of the tumour and aneurism cases, whilst in others hemorrhage occurred after ligation from the seat of ligature; still it seems that hemiplegia is most common in the hemorrhage cases occurring in 10 per cent. of them, and rarest in the nervous cases occurring in 2·5 per cent. of them.

If we assume that the percentage of septic cases was about the same in all four classes, the infrequency of paralysis in the nervous class may be explained by the absence of hemorrhage.

Further, it is obvious that sepsis is by no means necessary for the occurrence of gangrene: it occurs, for instance, without any breach of surface in some cases of aneurism which have not been treated; in some cases which have been treated by digital compression, as in a case of a man aged 38, with a large popliteal aneurism, for which digital compression was tried for nine hours;

gangrene followed and necessitated amputation. It occurs also in some cases after treatment by Esmarch's bandage, as in a case related by Pemberton to the International Congress in London. A man aged 45 years had a popliteal aneurism: Esmarch's bandage was applied and kept on for one hour. Digital compression was used subsequently: gangrene rapidly supervened and necessitated amputation.

It is clear then (as is to be expected) that gangrene may occur in the limb from local anæmia only, and presumably the same might happen in the brain. Spencer and Horsley shew conclusively that the anastomoses at the base of the brain are insufficient to compensate for the ligature of a carotid as far as the portion of brain supplied by the middle cerebral of that side is concerned.

Whenever the question of the ligation of the common carotid arises, from whatever cause, the practical lesson of Spencer and Horsley's work is that care should be taken, firstly, to keep (or make) the wound aseptic; and secondly, never to ligate the common carotid when the ligation of the external carotid or one of its branches will suffice. It might in cases of ligature for external hemorrhage be advisable to put a ligature temporarily on the common carotid while the bleeding-point is being searched for, as has been successfully practised by Rivington and Treves.

If the temporary ligation of the common carotid is continued much over an hour, there is a danger of a clot forming on the proximal side and causing embolism of the brain on the relaxation of the loop. The loop after loosening may be left *in situ* for a few days if thought desirable.

The suggestion of Spencer and Horsley of compression or ligature of the common carotid for cases of ingravescent apoplexy is certainly worthy of being borne in mind.

The danger of suppuration in the neighbourhood of a great artery, especially when the coats are not intact, must now be evident; it may occasion hemorrhage, aneurism, or gangrene. Though it be possible that with Listerian precautions any vessel up to the size of the superficial femoral may be ligated by almost any method with success, yet this cannot be employed as an argument in favour of rupturing the coats, nor can any security which antiseptics may afford justify the practice.

CHAPTER XII.

PATHOLOGY OF HEMORRHAGE.

Causes of hemorrhage. Use of the coagulum. Collateral branches. The time at which bleeding occurs. The fall of the ligature. Comparative immunity of certain vessels. Conclusion:—no injury to the coats and asepsis.

The causes of hemorrhage after ligation in continuity are two, laceration of the coats and suppuration: it might be thought that to these should be added disease of the arterial coats; we, however, know of no case of hemorrhage following ligation in which there has not been either laceration of the coats or suppuration.

With regard to suppuration, the acute arteritis which it causes may so weaken the wall as to lead to the formation on either the proximal or distal side of the ligature of a minute aneurism, the bursting of which is the immediate cause of hemorrhage. (Fig. 139.)

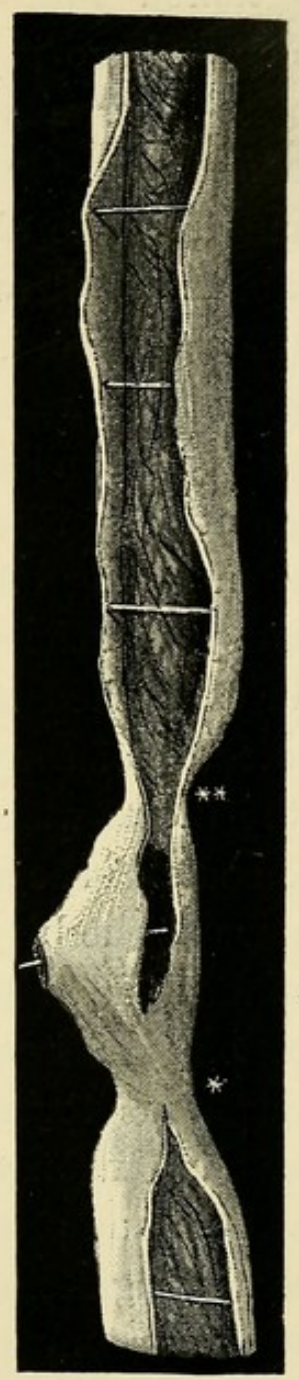


FIG. 139. Femoral Artery from a stump. (Nat. size.)

From S. Bartholomew's Museum, spec. 1413.

The artery was ligatured in the first instance at point marked *. Secondary hemorrhage occurred and the vessel was re-ligatured at point marked **. Here is now firm connective tissue and above this clot. Between the two ligatures is a small aneurism from which the hemorrhage probably occurred. The coats of the vessel were diseased.

It cannot be questioned that when the coats are ruptured secondary hemorrhage is promoted by suppuration, and the complete severance of the continuity of the artery thus effected.

It has been thought that the absence or scanty formation of clot was a cause of hemorrhage, but this by itself would not be a sufficient cause. A deficiency in the amount of clot formed when the vessel is ligated near to a collateral branch has always been supposed to be an adequate explanation of an attack of hemorrhage. The clot may, for a time, assist in taking off the pressure of the blood from the weakest spot of the arterial wall, but it so soon contracts away or is pushed away from the intima that its value as a protector of the ruptured coats must be small indeed: for the blood which penetrates between the vessel wall and the coats to the seat of ligature would be under the full pressure of the blood-stream.

The diversity of opinion as to the formation of clot after experimental ligations has been referred to before. Porta says that no thrombus formed in 35 out of 250 operations, and Walter that in only one instance out of 28 did he fail to find a distal and proximal thrombus.

In these experimental ligations the percentage of hemorrhage was a little over one per cent. In all our experimental operations thrombi formed, though in most the tunics were uninjured: and the non-occurrence of hemorrhage in any, whether suppuration did or did not occur, may fairly be said to be in part due to the integrity of the arterial wall.

The time at which hemorrhage takes place varies greatly, but it is not likely that the period of its onset

differs much now from what it was years ago, when it was customary to recommend a ligature which by suppuration should cut its way through the adventitia and thus be loosened and cast off in the discharges.

Porta, while Professor of Clinical Surgery in Pavia (1845), collected 70 cases of hemorrhage in man after ligation in continuity. Of these 43 happened between the 5th and 20th day, or the time at which the ligature was most likely to come away, 7 from the 1st to the 5th day, and 20 from the 21st to the 155th day.

The time which the ligature takes to come away varies, says Bruns, with the calibre of the artery, that is to say, with the amount of tissue which it has to cut through. This is not strictly true, however, because with a wound healing by first intention it may be most difficult to get the ligature, one end of which is hanging out of the wound, away at all. So that really the time of the fall of the ligature in former and even recent times depends for the most part upon the intensity of the septic process represented by the presence of pus. The actual cutting through of the vessel allows of retraction of the severed ends of the artery, and any sudden movement or exertion of the patient at this time has often been followed by hemorrhage.

The greatest injury to the vessel is always just under the knot. It is here that are found the rents from which blood has issued; and it is in this situation that the thin film of tissue representing the original, firm, thick wall gives way by reason of its thinness or from its being sodden with pus.

The blood frequently comes from the distal side of the

ligature, especially when the collateral circulation is free: the smallness of the distal clot, too, adds to the danger.

Cripps made an interesting experiment bearing on this question. He says: "Hemorrhage occurs most frequently "from the distal end of the tied femoral. All fluids have "a tendency to move in the direction of least resistance. "It is obvious that if there be a patent orifice in the "femoral the blood would have a greater tendency to flow "in this, the direction of less resistance, than towards the "capillaries, in which the resistance is considerable. The "following experiment illustrates this tendency to move "through the anastomoses of the arterial branches rather "than towards the capillaries, in which the resistance is "considerable. If water be injected into the common iliac "of a rabbit, it will circulate through the capillaries, re- "turning by the vein. If the external iliac and the super- "ficial femoral be now tied, the water will still return by "the vein, only much more slowly. If the femoral artery "be now divided below the ligature, and water still injected "by the common iliac, it will be found that a greater quan- "tity of the water will return through the cut (lower) end "of the femoral than by the iliac vein. Less resistance "is therefore offered by the anastomoses of the arteries "than is afforded by the capillaries."

The importance of the collateral arterial circulation is also insisted on by Pitts. Having seen a case of hemorrhage from the internal carotid, in which the common carotid was tied, but without preventing a fatal return of the hemorrhage, he with Reid made experiments on the dead subject. They injected a coloured fluid into one common carotid, and found that it came more readily

through the opposite external than through the opposite internal carotid.

It would then appear that if in such a case it is thought right to ligature the common carotid, the external should also be tied; but, as Pitts points out, hemorrhage from the neighbourhood of the tonsil, even when severe, is not necessarily from the internal carotid, and, if it is not, ligature of the external vessel will control it.

This quite agrees with the views of Henry Morris as to the free communication between the external carotid arteries. He says that in aneurism of the carotid near its bifurcation it is not sufficient to tie the common trunk, but it is advisable to ligate also such branches of the external carotid as are easily accessible.

Erichsen says that in 14 cases of ligature of the first part of the subclavian, death took place in all: in 12 from hemorrhage, which in every case came from the artery on the further side of the knot. It is impossible to give a statistical contrast of the frequency of proximal and distal hemorrhage, but it is probable that, except in those cases in which a large branch is given off just above the ligature, the hemorrhage more frequently occurs from the distal than from the proximal side.

In seven out of Porta's 70 cases, fatal hemorrhage was noted as occurring from soon after the operation to the 5th day.

In the cases of bleeding from the first part of the subclavian, the shortest time after ligature, according to Erichsen's table, was 24 hours, and the longest 36 days.

In the ligation of a great artery in continuity the blood has been known to spurt out as soon as the ligature was

tied, and that too, although the artery did not appear to be diseased to the naked eye; moreover, it will be shewn that the force necessary to rupture does not vary materially from the normal if atheroma (not calcification) be present. In former times the most anxious period was when the ligature separated, but hemorrhage may occur after the patient has left the hospital and returned to his vocation.

Such a case will be found in the London Hospital Reports; the external iliac had been ligatured for a femoral aneurism: the patient returned home, and some two months after operation fatal hemorrhage occurred.

Sir James Paget has shewn us notes of a case of popliteal aneurism, for which the superficial femoral was tied: all went well till after the patient had left the hospital, when (six weeks after operation) a small hemorrhage occurred for which the patient was re-admitted, but no further hemorrhage occurring, he again left; nine weeks after operation a severe and fatal hemorrhage occurred through a small opening in the scar.

The danger of hemorrhage does not depend solely upon the size of the artery, indeed certain great vessels appear to enjoy a comparative immunity from hemorrhage: for example, it is much less frequent from the external iliac than from the first or even the third part of the subclavian. The reason for this appears to be as follows: the diameter of the external iliac when distended is given as 10 mm., that of the first part of the subclavian as 11 mm., and that of the third part as 9 mm.: the total thickness of the wall of the external iliac when collapsed is 0·61 mm., of the first part of the subclavian 0·73 mm., and

of the third part 0·63 mm.; but by far the most marked difference is found in the thickness of the outer coat; that of the external iliac is 0·14 mm., of the first part of the subclavian 0·05 mm., and of the third part 0·07 mm.: thus the outer coat of the external iliac is double as thick as that of the third part of the subclavian: besides this, the branches of the subclavian are much larger and more numerous than those of the external iliac; in the latter, also, there is a long stretch of artery without any branches of any size; also, it must be remembered that the subclavian being nearer to the heart, the blood in it is at a somewhat higher pressure.

With antiseptic wounds we think the smaller arteries could be tied with or without rupture of the tunics and without fear of hemorrhage: but this is no argument for the infliction of useless damage on their walls. It is indeed probable that few recognise the extreme tenuity of the wall of the larger arteries, not as they appear in the dissecting-room, but as they actually exist during life, distended by a pressure of about 5 lbs. to the square inch. When one considers how thin the walls of the large arteries are, the wonder is not that hemorrhage does occur when the coats are ruptured, but that it does not occur more often.

Early hemorrhage from an artery, say on the first or second day, when sepsis has not had time to cause solution of the adventitia, must depend upon the rupture of the remaining fragment of the arterial wall.

The experiments in Chapter XVIII. shew how the wall of a large artery may be readily and completely ruptured under the knot, without the use of extraordinary force;

and a slightly less severe injury than this to the wall of the artery would result without question in hemorrhage some short period after the operation was completed.

To conclude,—when the coats are ruptured hemorrhage will happen most often with those arteries in which the outer coat is thinnest, the collateral branches most numerous, and the minimum of clot is deposited; that in these cases the full force of the blood-current breaks upon and rends the outer tunic, where it alone confines the blood within its natural channel; further, by antiseptics alone the great arteries of the body cannot be ligated as far as hemorrhage is concerned with absolute safety, but this result may be expected when with asepsis is combined the employment of a suitable ligature, so applied as to occlude the artery without damaging its wall.

In Chapter XVII. it will be considered how this can be surely done.

CHAPTER XIII.

CONDUCT AND FATE OF THE ANEURISM.

Quotation from Scarpa. Mode of formation of aneurism. Structure of wall. Structure of clot. "White clot." Blood external to clot. Apparent exceptions to Scarpa's rule. Cases of spontaneous cure. Principle of Hunter's operation: his cases. Causes of failure. Treatment by other methods. Conclusion.

"It is a certain and incontrovertible fact in practical "surgery that a complete and *radical* cure of aneurism "cannot be obtained in whatever part of the body this "tumour is situated unless the ulcerated, lacerated, or "wounded artery from which the aneurism is derived is "by the assistance of nature, or of nature combined with "art, obliterated and converted into a perfectly solid liga- "mentous substance, for a certain space above and below "the place of the ulceration, laceration, or wound."

So wrote Scarpa, and, although nearly a century has passed away since this bold statement was made, its truth

has never been disproved: for our own part we have failed to find the record of a single case in which cure has taken place in any other way; there does not exist in any museum which we have visited a single specimen shewing any other mode of cure, and it may be added that any exception to the rule is not to be expected from what is known of the processes which occur in the disease.

The first step in the formation of an aneurism is the occurrence of some injury to or disease in the arterial wall, by which it becomes weakened and yields to the pressure of the blood within: a bulging occurs, round which, as round a foreign body, a capsule forms which constitutes the wall of the aneurism, but this in its turn is not strong enough to resist the pressure of the blood, and the disease continues to advance. As it does so the shape of the aneurism alters, and there arise in it bays and back-waters in which the blood circulates but feebly and finally deposits a clot. As the aneurism continues to enlarge, another layer of clot is deposited internal to the first, and so on, the aneurism gradually increasing, and successive layers of clot being deposited in its quieter parts: in this way the sac may become wholly filled with laminated clot: even so the aneurism is by no means cured: there is a constant danger of the blood finding its way between the wall and the clot. The only thing that can obviate this danger is for the wall and the clot to become adherent, and the only way this can occur is by extension of connective tissue growth from the wall into the clot: this does actually occur at certain parts of the aneurism, but only in those places where there is perfect stillness, for the slightest movement of the clot on the wall will prevent

the invasion of plasma cells, and therefore also the necessary adhesion of the clot to the wall. Experience shews that there is always too much pulsation in an aneurism for this adhesive process to be carried out effectively unless the artery on which it is situated is occluded. The reason why the clot in an artery can become adherent is because it entirely fills the artery; all circulation and pulsation is arrested, and stillness is obtained: when the same condition is produced in an aneurism by the occlusion of the artery, adhesion occurs between the wall and the clot. It must be remembered too that the intima of an artery is a more active tissue than the inner layers of the sac of an aneurism: since in the latter the greater number of plasma-cells have passed into a stage of quietude in fibrous tissue. In fact, in an artery, just as in an aneurism, it is necessary, if adhesion of the clot is to be established, that the vessel be occluded.

If, however, the coagulation extend from the aneurism into the artery, and this also become filled with clot, then all pulsation in the aneurism ceases, invasion of plasma-cells from the wall occurs over the whole extent of the clot, the formation of connective tissue and absorption of coagulum proceed concurrently throughout the whole thickness of the clot, which, together with the wall, becomes partly absorbed and partly converted into a mass of connective tissue: this gradually shrinks, so that after the lapse of years all that remains of the aneurism is a small fusiform nodule of connective tissue. This last stage is well shewn in Figure 160, page 334, in which is seen the remains of a popliteal aneurism fifty years after ligation.

FIGS. 140, 141, 142 and 143. Diagrams to shew the process of growth of an aneurism and the mode of deposition of clot within it.

The diagrams are sketches of the aneurisms represented in Figures 153, 150, 148, 149.

Figure 140 shews the first stage of aneurism: it is a simple sac (*w*) filled with laminated clot (*a*). The space (*x*) between the wall and the clot contains fluid blood, and is in communication with the main vessel.

Figures 141 and 142 shew the second stage. The portion of the clot first deposited is marked *a*, the later *b*; as the aneurism extends by the pressure of the blood, the clot (*a*) is pushed further away from the mouth of the aneurism and more recent clot is deposited beneath it, as seen in Figure 142: or the clot (*a*) may become situated on one side in consequence of the unequal extension of the sac: this is seen in Figure 141.

Figure 143 shews a yet further stage: *a*, *b* and *w* as before: but the space (*x x x*) has become much enlarged from the pressure of the fluid blood within it, which, as in the other drawings, is in communication with the blood in the artery. The fluid pressure is in all directions as shewn by the arrows, but the direction of the growth is modified by the resistance of surrounding structures. By this process the clot (*a*) has been left in this case in the centre of the aneurism: and a new layer of clot (*c*) has been deposited on the inner side of the wall (*w*); a further stage still is seen in the ruptured cœliac aneurism shewn in Figure 147.

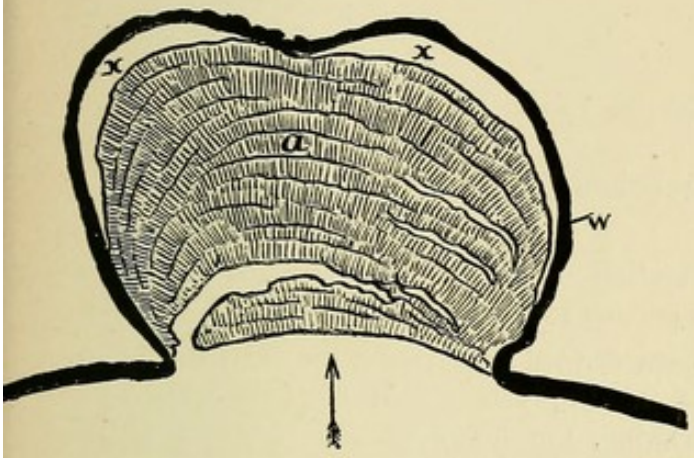


FIG. 140.

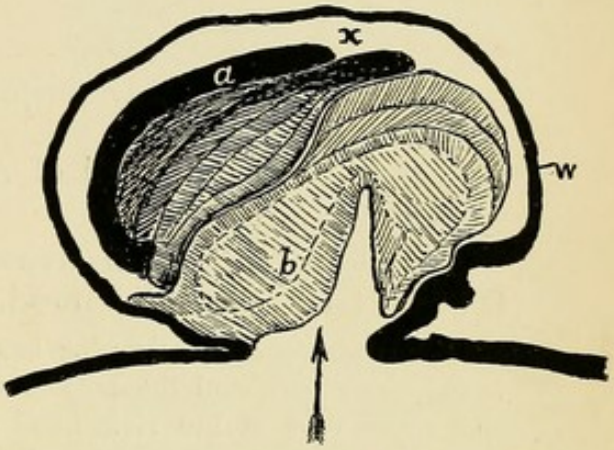


FIG. 141.

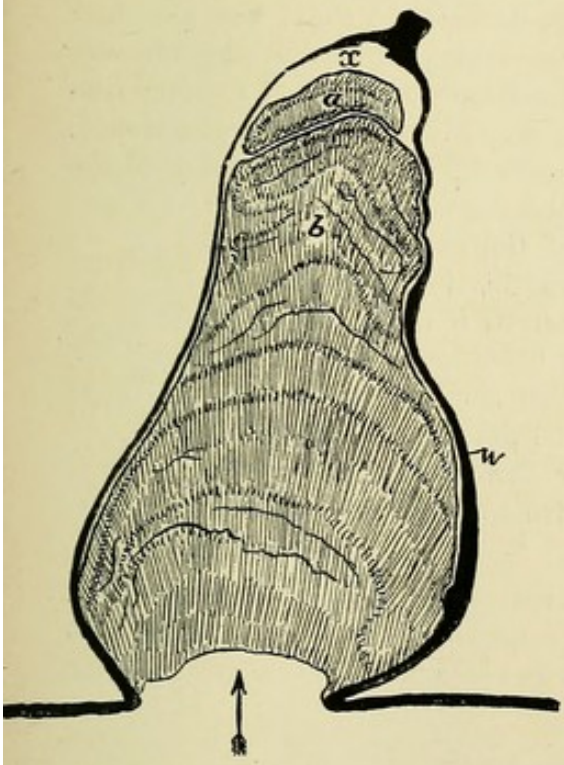


FIG. 142.

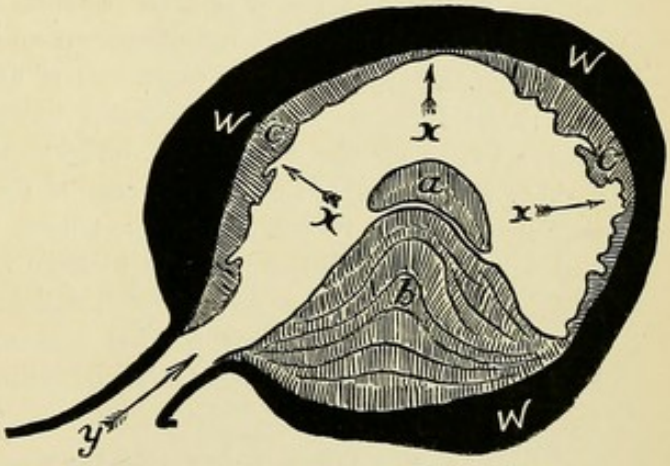


FIG. 143.

Description of Plate IX.

Wall of Aneurism (× 150).

The drawing is from a transverse section of the wall of the aneurism shewn in Figure 164, page 345: it is from the portion enclosing the space *b*.

For convenience the drawing has been divided: the top of the left-hand figure is the outer part, and the bottom the middle; this should be continuous with the upper part of the right-hand drawing, the lower portion of which is the inside of the wall with some clot adhering.

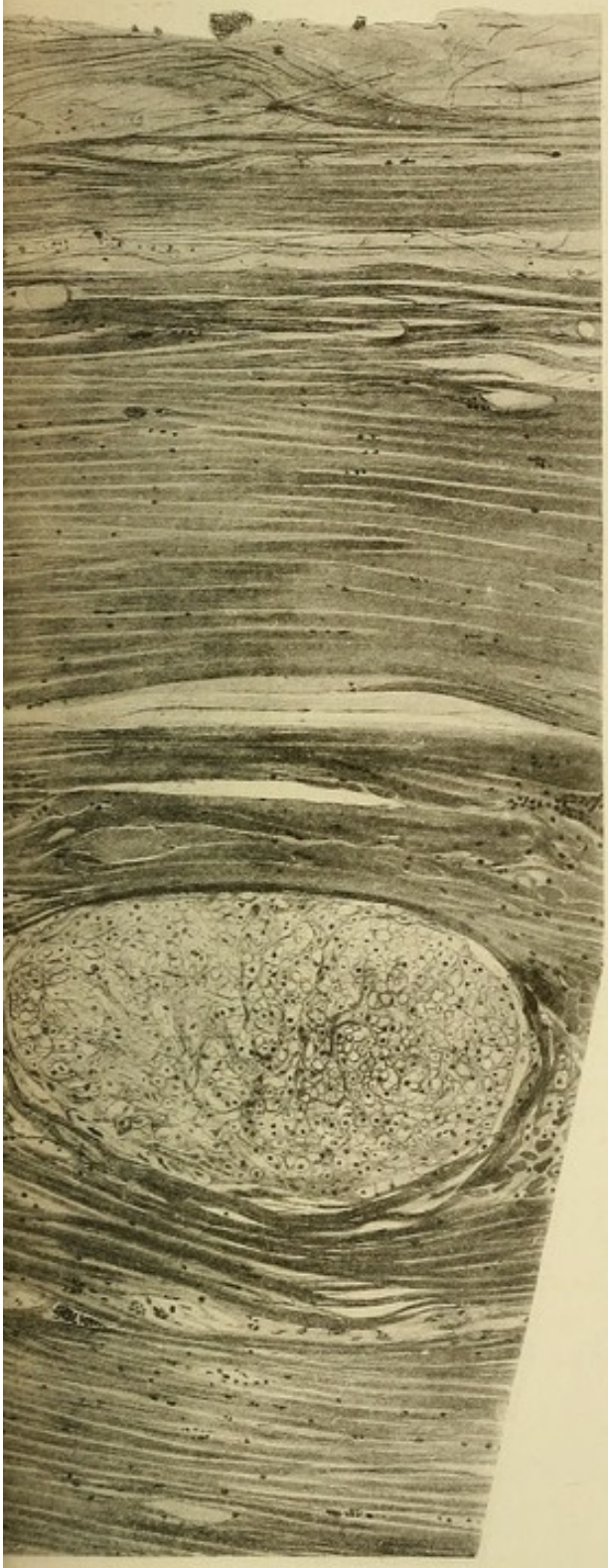
The wall consists mainly of parallel fibres; these are seen under a high power to be composed of smaller fibres.

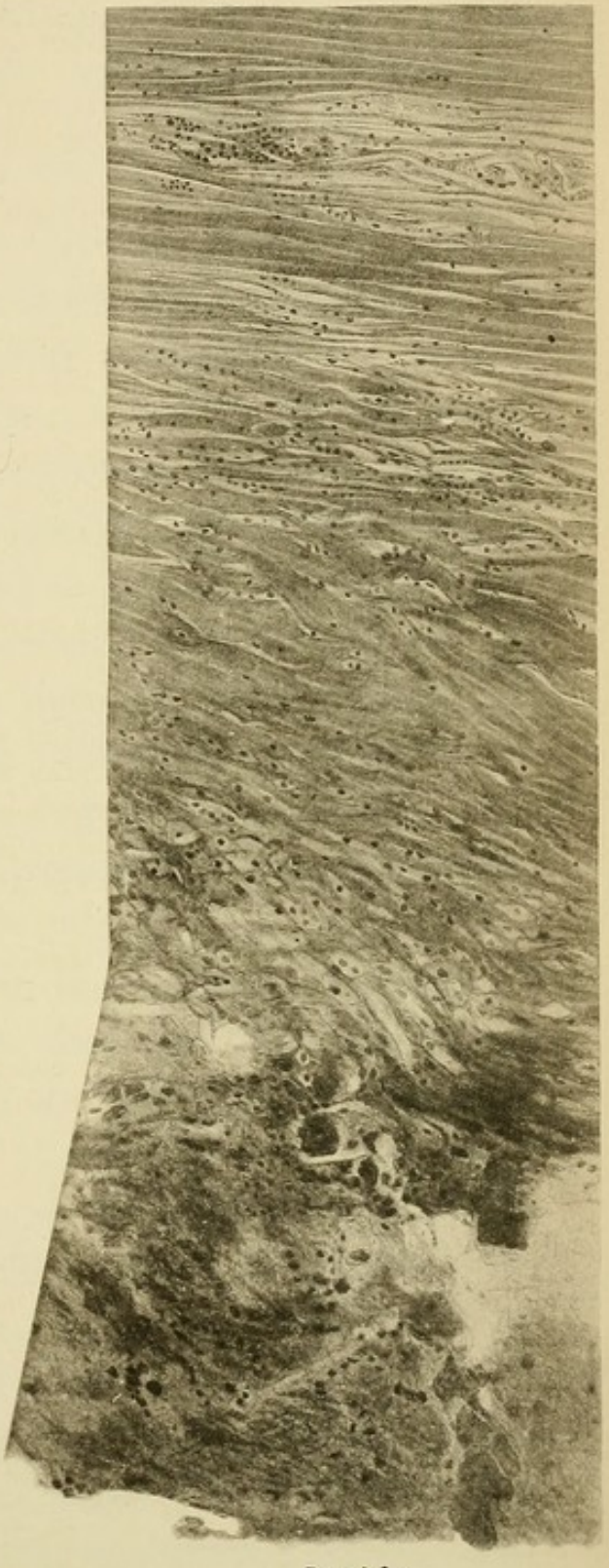
The large body in the left-hand drawing is an old blood channel in which a clot has formed and become absorbed, being now replaced by young connective tissue. It will be noticed that the amount of loose tissue between the fibres gradually increases from without inwards: and that the outer part of the adherent clot has become invaded and is undergoing absorption, being replaced by young connective tissue: this tissue in time becomes fibrous and constitutes part of the wall: there is thus no sharp line of demarcation between the wall and the clot (compare Plate VIII. Fig. 3, page 138). The wall of an aneurism is formed partly from within and partly from without, the relative extent of the two processes depending on the strength of the current of blood: where this is strong there will be no clot and the wall will distend, and if the formation of fibres externally is not sufficiently rapid it will rupture: where the blood current is slow or absent, clot will be deposited, which will become invaded from the wall and be absorbed and replaced by fibrous tissue which will become part of the wall. Where circumstances are such that this process can occur throughout the aneurism, it will be in time absorbed, a little mass of connective tissue being all that is left (see Figure 160).

Compare Fig. 116, page 258, shewing the absorption of a kangaroo tendon ligature. The capsule is situated not external to the original site of the ligature but in the place of the absorbed portion, there being no space between it and the portion now undergoing absorption.

Outside.

Middle.





Middle.

Inside.

WALL OF ANEURISM.

In transverse sections of old and large aneurisms the clot is seen to be arranged in a very complicated manner. It is difficult sometimes to decipher its history, but the general principle of its growth will be seen from the accompanying diagrams (page 301), which have been drawn from actual specimens.

That the foregoing is the process which occurs in aneurisms will be seen too from the accompanying drawings: Plate IX. shews the wall of an aneurism: it is seen to consist of coarse, parallel fibres: under a higher power each of these is found to be composed of smaller fibres. On the inner side of the wall a portion of clot is adherent; this on its outer side is undergoing invasion from the wall, and new connective tissue is forming in it, so that no line of demarcation can be found between the wall and the clot: this part of the drawing should be compared with Plate VII. Fig. 5, and Plate VIII. Fig. 1, pages 136 and 138, where the same process is occurring in a clot in an artery.

Wherever clot is adherent to the wall, connective tissue growth extends into the clot, and the new fibres thus formed become in time part of the wall: the same process occurs when a ligature is absorbed, the new tissue forms in and takes the place of the portion of ligature absorbed, and thus the capsule always closely surrounds the remaining portion of the ligature. (See Fig. 112, page 255.)

The successive fibrous laminæ forming the wall of an aneurism are separated by considerable intervals, in which all that is seen is a little loose connective tissue. The existence of these wide spaces is due to the pulsation

FIGS. 144, 145 and 146. Laminated clot from Aneurism (× 200).

The clot was removed from an aortic aneurism of considerable size and age; it was not adherent to the wall, on which it formed a layer 9 mm. thick.

Fig. 144 represents the outer portion of the outer layer, that portion which was in contact with the aneurism wall. In this case this was the portion of clot first deposited. No invasion of plasma-cells or any indication of the formation of connective tissue is to be seen, even in this, the outermost layer.

Fig. 145 is taken from one of the middle layers of the clot.

Fig. 146 is from the innermost and most recent layer of clot. In this are seen numerous red corpuscles whose outlines are still distinct. This portion of clot was to the naked eye red, while the other layers were of a light brown colour.

In the other two Figures the red corpuscles cannot be recognised except at the lower part of Figure 144, where a rent in the clot has allowed fluid blood to penetrate.

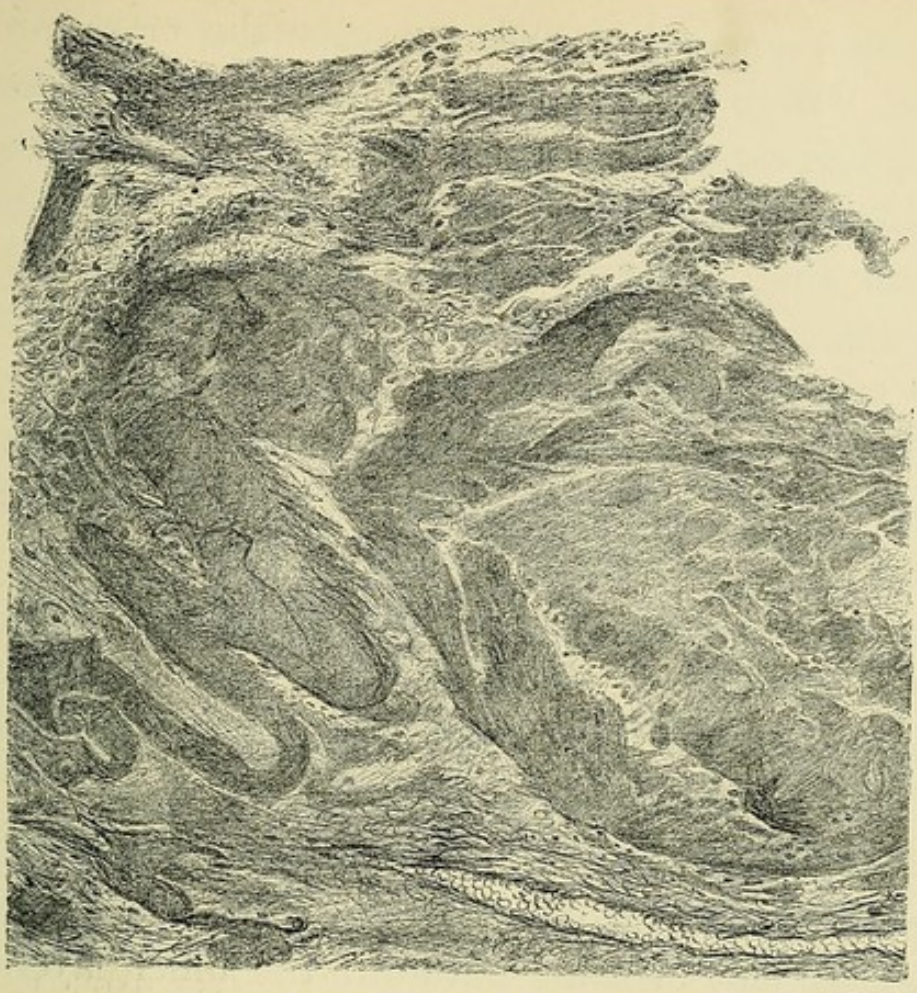


Fig. 144.

Fig. 145.

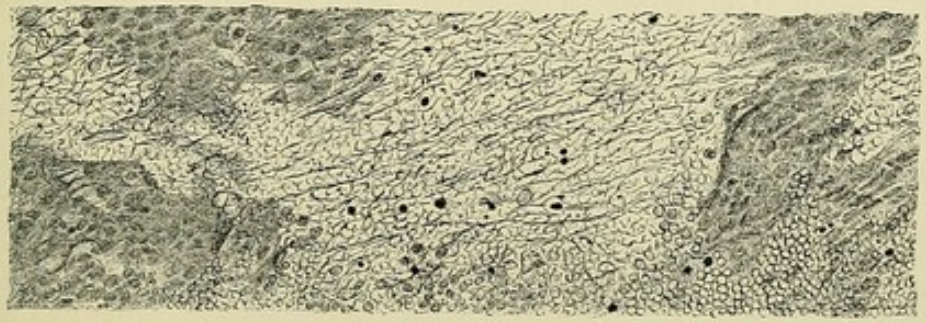


Fig. 146.

of the aneurism, and in their presence the capsule of an aneurism differs from that of all non-pulsating foreign bodies. The capsule is formed under constant movement, and is adapted to it; with each pulsation the fluid in the spaces in the wall is no doubt displaced, the fibrous lamellæ being pressed together.

The structure of laminated clot is shewn in Figures 144, 145, 146. In Figure 146, which is from the innermost and most recent lamina, the red corpuscles can still be recognised; in the others they cannot, except in a rent in the clot in Figure 144: the clot has not a homogeneous appearance, but that is not due to organization taking place within it, for there is no sign of anything of the kind.

The outer layers of old clot become hardened and discoloured and constitute the "active" clot of Broca and the "white" clot of some authors: the inner layers and the central mass of clot are red and constitute the "passive" clot of Broca and the "red" clot of others. This latter clot is both red and passive; but the "white" outer layers of clot are also passive, and the only part they can play in the cure of an aneurism is the passive one of being absorbed and replaced by connective tissue cells from the aneurismal wall, when by chance this "white" clot becomes adherent to the wall of the aneurism.

These outer layers of clot are only "white" because they have become decolourized; when they were first deposited they were red like the inner layers, indeed they then were the inner layers. Elsewhere we have shewn how the colour leaves the clot, the pigment being found in

neighbouring lymphatic glands: this process occurs independently of the cellular invasion of the clot.

There is another way in which the appearance of "white clot" is produced. The outermost layers of clot in an aneurism may become invaded by plasma cells, and thus be replaced by young connective tissue. This young tissue is bright white on section; but it is not "white "clot," for it is not clot at all. (Fig. 138, aa, page 283.)

It must not be assumed that because a clot is white when seen, that it was so when first deposited: unless the date at which the clot was formed is known for certain no inference can be drawn. It must not be thought, too, that because a clot appears white that it is or was mainly composed of white corpuscles. The experiments of Eberth and Schimmelbusch shew how in capillaries small clots, composed mainly of white cells and blood-platelets, may form; but these collections are merely microscopic and probably no larger than the groups which form the starting points of fibrin when ordinary clots form. The collection of leucocytes at the periphery of the blood-stream, and their becoming attached to the aneurismal wall, is an essential preliminary to the formation of red clot. This red clot is homogeneous and constitutes a new layer. Later leucocytes become adherent to its surface, and then another layer of red clot is deposited. In our experiments we have never seen any clot that we knew must have been recent to be anything but red: wherever we found clot white it had had ample time to become decolourized. In fact, as far as we know, no clot of any size anywhere, when deposited, contains anything but its due proportion of red corpuscles; indeed, there is only one kind of clot.

The nearest approach to white clot is that which forms in leucocythemia, but this clot does not, as far as we know, exhibit greater activity than ordinary clot (Fig. 146[A]).

Lastly, it may be urged that from the conduct of the white corpuscles which exist in red clot there is no reason to suppose that a clot composed exclusively of

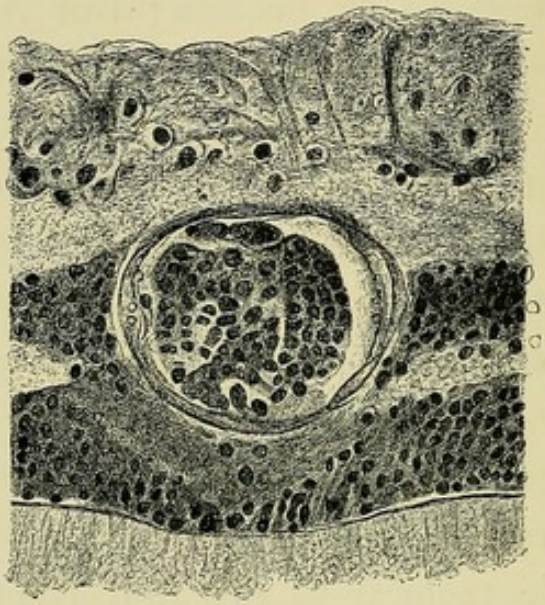


FIG. 146[A]. Retina in Leucocythæmia (× 300).

The drawing is reproduced from the Transactions of the Ophthalmological Society, Vol. X., where the case is recorded.

The section passes through a retinal vein in which is seen an uninvaded clot containing a large excess of white corpuscles.

The patient was a married woman, aged 22 years: she suffered from leucocythæmia with enlargement of liver, spleen and lymphatic glands: there were numerous retinal hemorrhages: infarcts were found in the spleen, and petechial hemorrhages in the pia mater and other parts. Shortly before death the white corpuscles equalled the red in number.

white corpuscles would behave otherwise than a red clot, or render more assistance in the cure of an aneurism.

Attention has recently been directed to the subject of white clot by an important surgical paper by Macewen. He attributes the cure of aneurism to the formation of white clot, and in certain cases to produce this he has

introduced into aneurisms fine steel needles. He allows them to slightly scratch the inner surface of the sac or lining clot. Macewen gives several interesting cases which have been thus treated. He had, however, no sure means of knowing either that the "white" clots he found after death in those cases which were subsequently examined were not present before his operation, or that if they resulted from it they were not red at first and became subsequently decolourized.

If the irritation produced by the needle causes adhesion between the aneurismal wall and the clot and the more certain and rapid invasion by plasma cells, the treatment is admirable; at least the wall of the aneurism would be strengthened at that part by new connective tissue; but further experience of this treatment is required before a definite opinion of its value can be arrived at.

Figure 147 is from an aneurism of the cœliac axis: the current of blood came out from the aorta in a forward and slightly downward direction: it impinged on the opposite side of the aneurism, and it was there that the aneurism eventually ruptured: in the quieter parts above and below the direct stream clot has formed, and here and there has become adherent to the wall.

Figure 148 is an aneurism arising from the aorta before the giving off of the innominate: it is entirely filled with clot, but is not cured: the clot is adherent to the true left-hand side, but elsewhere it was not, and blood could get between the clot and the wall: the varying thickness of the wall will be noticed, on the true right it is as thin as paper.

Figure 149 is an aneurism of the internal iliac artery:

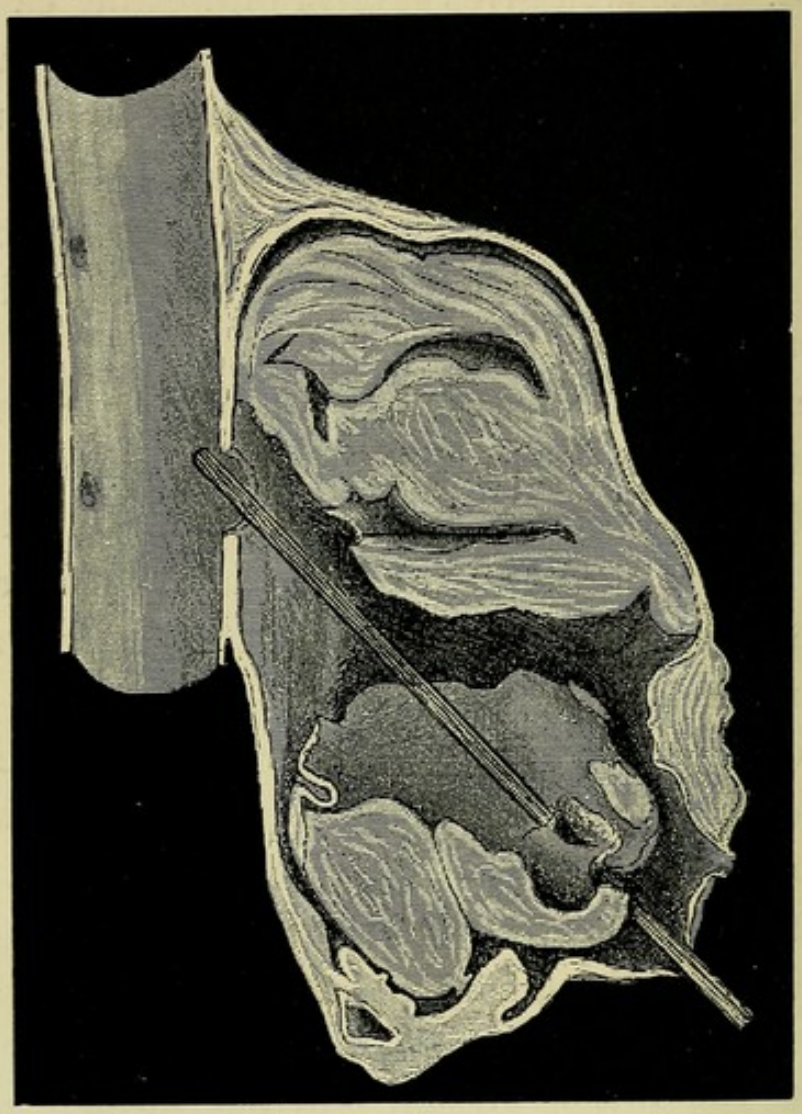


FIG. 147. Aneurism of Cœliac Axis. (Nat. size.)

Specimen in S. Thomas's Hospital Museum, Y 84.

It shews that the direct impact of the blood fell on that portion of aneurismal wall which was in front of and below the opening into the aorta; it was here that the aneurism ruptured: there is abundant clot on either side out of the main stream. The glass rod passes from the aorta through the opening in the wall of the aneurism. Death was caused by rupture into the abdominal cavity.

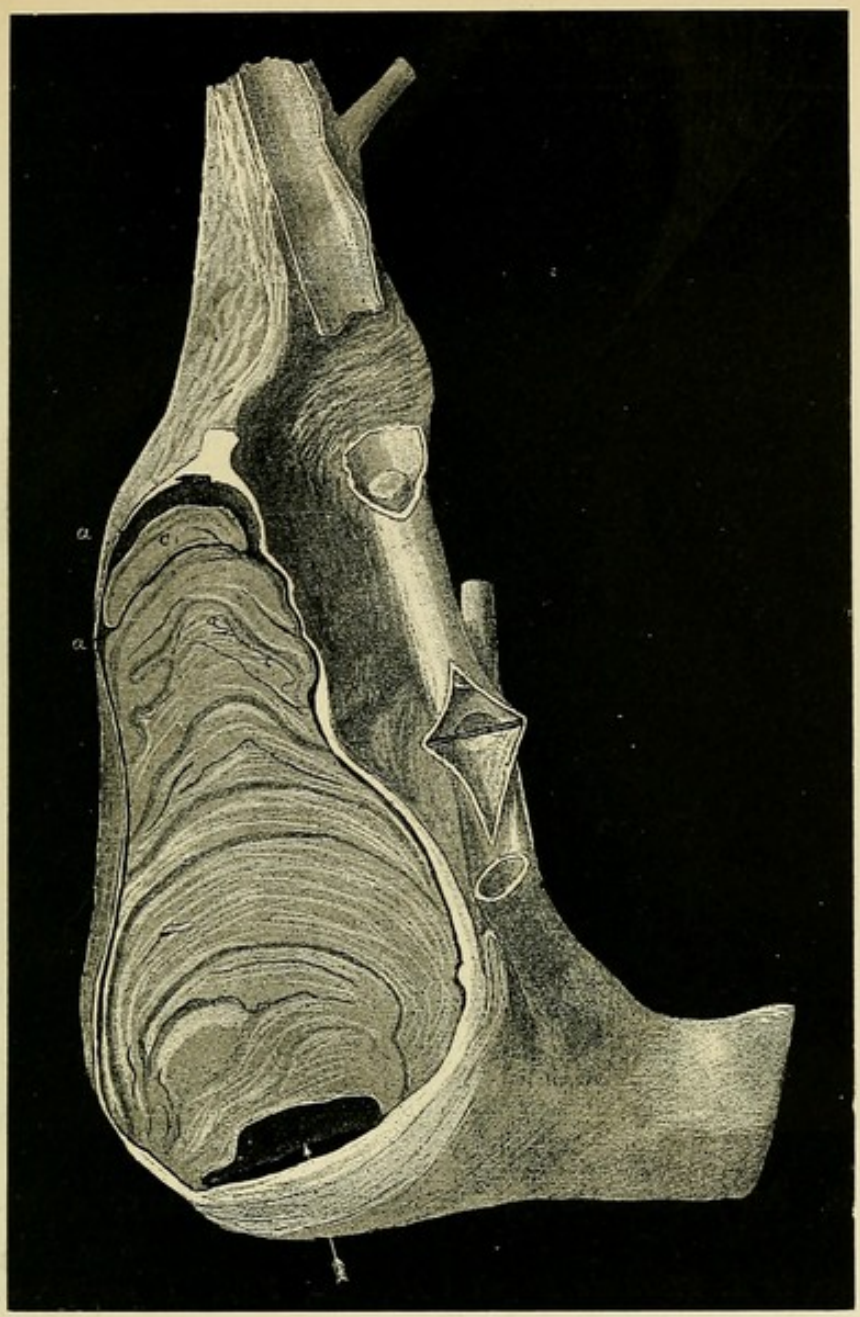


FIG. 148. Aortic Aneurism (× $\frac{2}{3}$).

The aneurism was thought during life to be of the innominate artery, and the right common carotid was ligatured; thirty days later patient died with cerebral symptoms. The parts are seen from behind.

The common carotid was tied in two places and divided between. In the drawing the two ends are seen widely separated; the lower end of this artery has been opened to shew the extent of the proximal clot, and the origin of the right subclavian: there is no distal clot. In front of the innominate is the left common carotid. The aneurism arose from the aorta in front and to the right of the origins of the great vessels. The layers of the clot in the aneurism are well seen: they are crescentic and their shape is due to the pressure of the blood, as indicated by the arrow. The uppermost part of the clot marked *c* was probably that first formed, and may have filled the whole aneurism where this was quite small. The aneurism yielding in an upward direction, this first formed clot was forced upwards and new layers were deposited below it. It will be noticed that an interval exists between the clot and the aneurismal wall: here fluid blood found its way and caused the disease to progress. It will be seen also that the wall varies greatly in thickness: at *a a* it is extremely thin and on the verge of rupture. See also fig. 142, page 301.

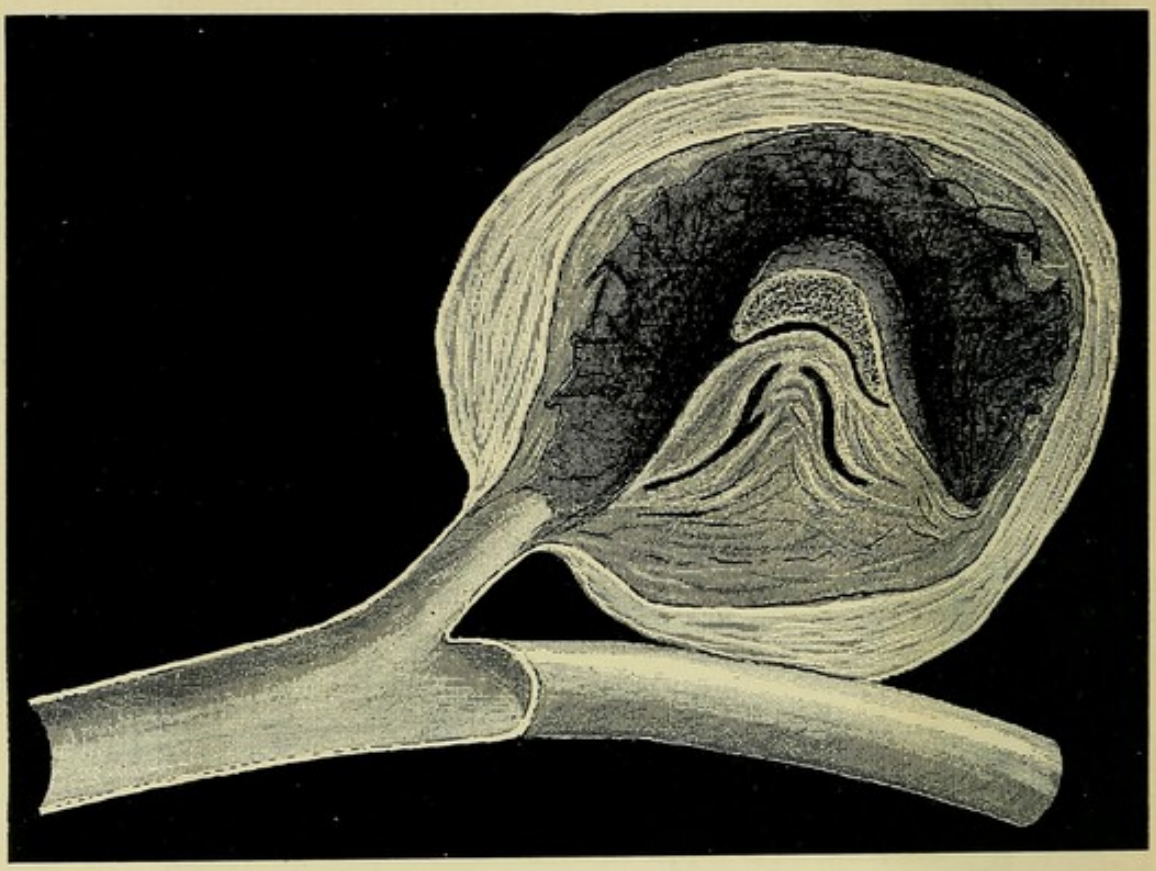


FIG. 149. Aneurism of Right Internal Iliac Artery. (Nat. size.)

Specimen in S. Thomas's Hospital Museum, Y 95. It was presented by Travers.

The probable explanation of the specimen is that the cap-shaped portion of clot now at the centre of the aneurism was formed first and when the aneurism was only of its size; the laminated clot below this was formed later as the aneurism increased, the first-formed clot being undisturbed, and as it were lying in a pouch of the aneurism; subsequently the blood forced its way external to the clot, and caused an expansion of the tumour and a separation of the wall from the clot. A new layer of clot is forming on the inner surface of the distended wall; this is represented light: the darker part of the drawing is where the blood was circulating. The distal part of the internal iliac is not in the portion of the aneurism preserved. See also fig. 143, page 301.

when the aneurism was smaller it was probably nearly filled with clot, but the blood got external to the clot, and caused the aneurism to distend, leaving the clot in

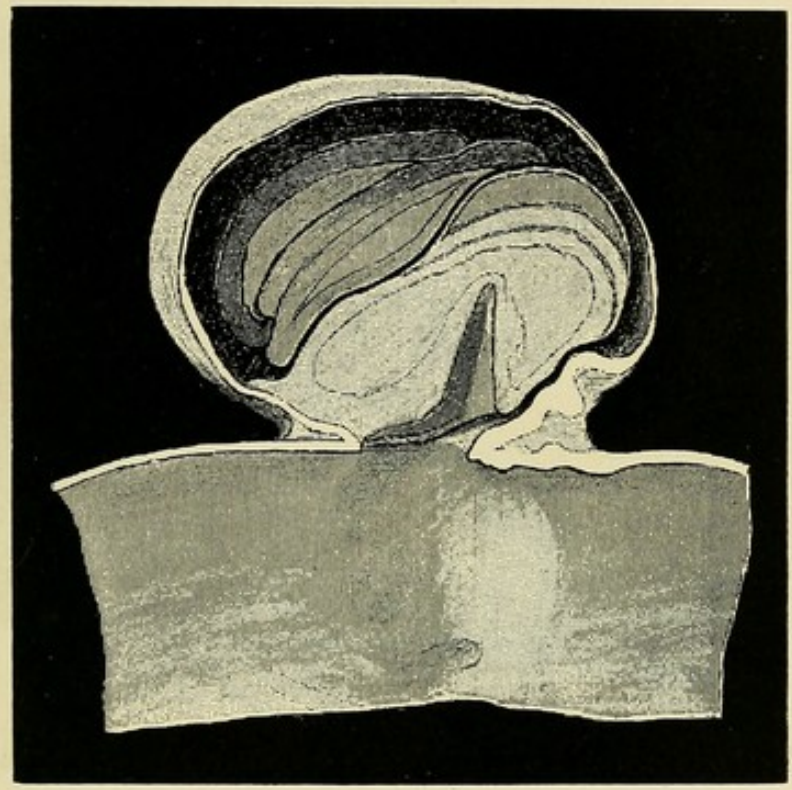


FIG. 150. "Cured" Aortic Aneurism. (Nat. size.)

The Specimen is in S. Thomas's Hospital Museum, Y 36[1].

The ascending portion of the aorta is dilated and ulcerated. There is a globular aneurism, size of a Tangerine orange, projecting from the right side of the dilatation; it is filled with laminated clot. There is a narrow space between the aneurism wall and the clot where fluid blood penetrated. For further description see diagram, Figure 141, page 301.

the centre; a more recent clot is seen forming on the now distended wall of the aneurism.

Many apparent exceptions may be cited to the proposition that if the artery remains pervious the aneurism cannot be cured. In the Museum of Guy's Hospital may be seen an aneurism of the heart (Fig. 152), which is described as cured, but it is very doubtful if it was, for a free

communication existed with the ventricle. There are also to be found in most museums specimens of small aortic aneurisms which are full of clot, and described as cured. Figures 150 and 151 represent such specimens in St Thomas's Museum, but it will be seen from Figure 151,

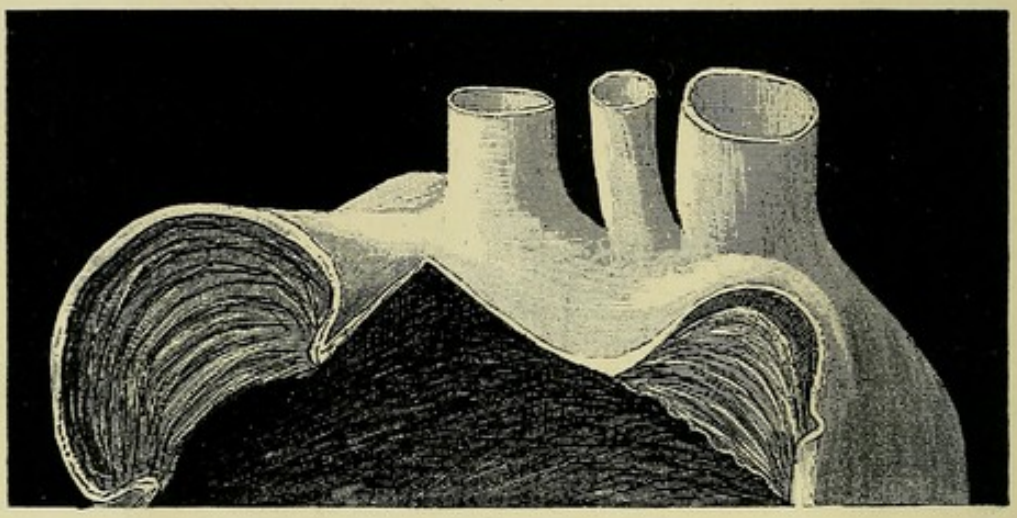


FIG. 151. "Cured" Aneurisms of Aorta.

The Specimen is in S. Thomas's Hospital Museum, Y 41.

The whole of the upper portion of the aorta is dilated and on it are seen two small aneurisms which are filled with laminated clot.

These aneurisms are not really cured; that to the right is seen to have a dark crescentic lamina between the aneurismal wall and the lighter coloured clot: this is recent clot.

In the aneurism to the left a similarly situated dark lamina is a space into which fluid blood could penetrate; the drawing does not shew this well.

that in one of the two small aneurisms represented, a space exists external to the clot where blood penetrated. In the other aneurism the same space had existed, but is now filled with recent clot; in Figure 153 also a considerable space exists external to the clot.

Any doubt that the blood really does find its way between the clot and the wall will be removed by examining specimen No. 3097 in the College of Surgeons

FIG. 152. "Cured" Aneurism of Heart (× $\frac{2}{3}$).

Reproduced from Trans. Path. Soc., Vol. VIII. (1857), *by kind permission of Dr Wilks. Specimen in Guy's Hospital Museum,* 1395[30].

The aneurism is at the apex of the heart: a probe is passed through the small communication between it and the left ventricle; patient died (ætat. 52) of tuberculosis.

Although the aneurism is described as cured and its contents are partly calcified, yet from there being a free communication with the ventricle, and from the fact that the contained clot is not everywhere adherent to the wall, it is not certain that the disease is cured.

Museum: it is an aortic aneurism, in which ligation of the large vessels had been practised by Barwell; the specimen is injected, and the injection can be seen to have passed

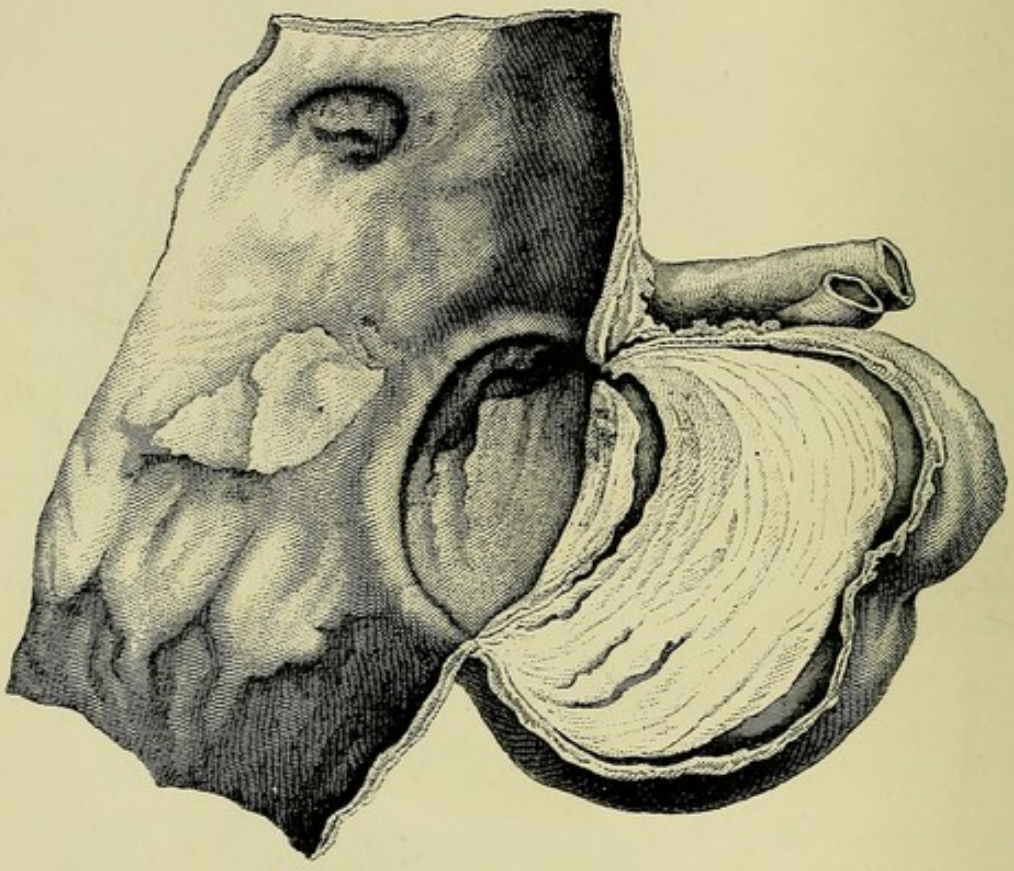


FIG. 153. "Spontaneous cure" of Aortic Aneurism.

The figure and description is from "Engravings illustrating Diseases of Arteries by Joseph Hodgson" (1815).

"Section of an aorta in which an aneurism was situated at the origin of the "cœliac artery. This aneurism was filled with firm lamellated coagulum which "entirely closed the communication between the sac and the artery. The coagulum "terminated at the part where the coats of the artery had given way in a smooth "surface, which had a membranous appearance."

It will be noticed that there is an interval between the clot and the wall into which there can be no doubt that fluid blood found its way. Compare figure 140, page 301.

between the sac and the clot. The same thing must occur when rupture takes place in aneurisms which are nearly filled with old standing clot.

A "spontaneous cure" of an aneurism of the abdominal aorta is seen in Figure 153.

A case of "spontaneous cure" of a femoral aneurism, in which the artery remained pervious and continued to

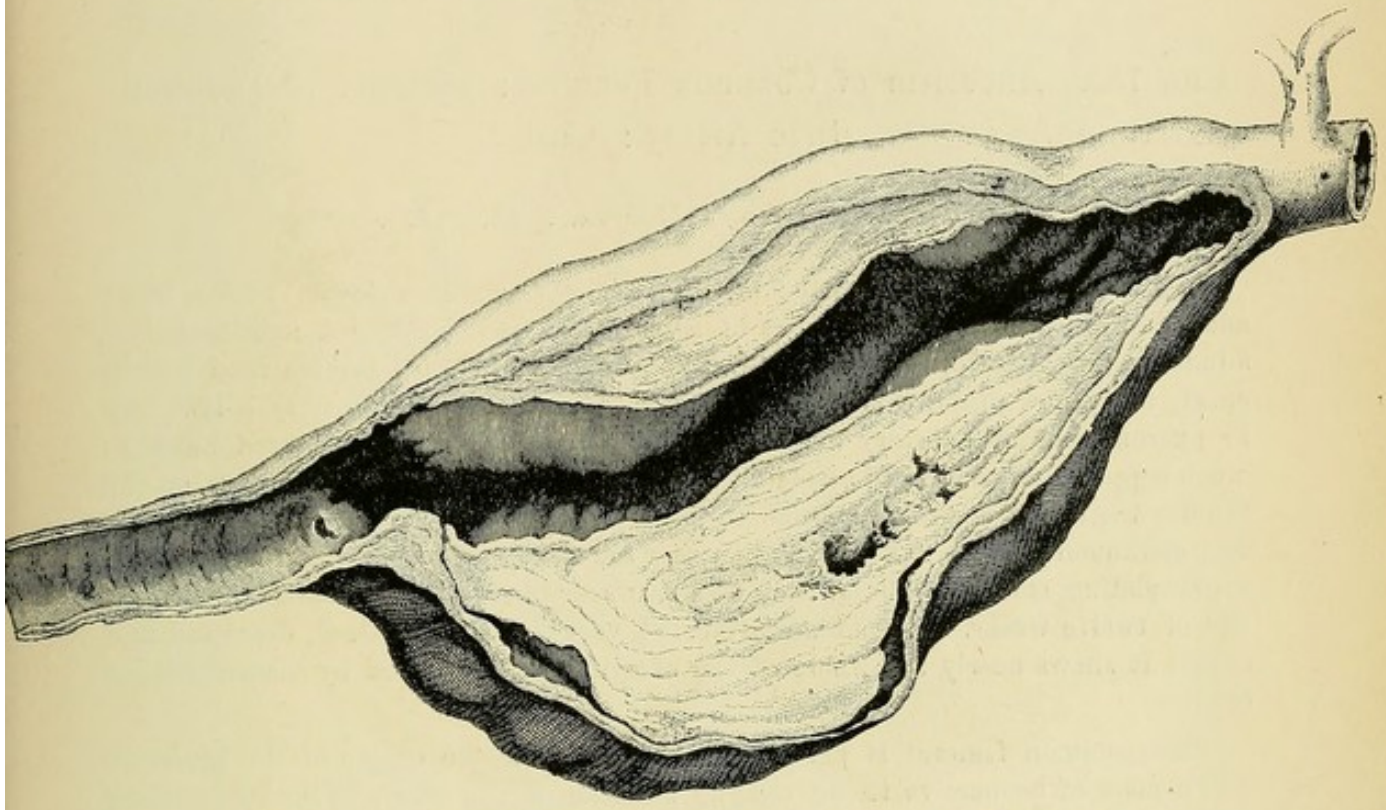


FIG. 154. "Spontaneous cure" of Femoral Aneurism.

From Hodgson's Plates (1815), *Plate VII. Figure* 4. *For an account of the case "see Hodgson on Aneurisms"* (1815), *page* 134.

The aneurism had been noticed 6 or 7 years previously and soon grew to the size at which it remained, till patient's death from axillary aneurism: it pulsated strongly but caused no inconvenience to the patient. It extended from the origin of the epigastric to that of the profunda. The sac is lined with laminated clot, through the centre of which a canal for the blood remains.

It will be seen that there is a distinct interval between the clot and the aneurism wall at the lower part of the drawing. Under these circumstances the aneurism cannot be regarded as cured.

pulsate, is described by Hodgson. He obtained the specimen and gives a drawing of it, which is reproduced in Figure 154: it will be seen that a considerable space exists in one part between the clot and the wall, into this space

Fig. 155. Aneurism of Common Femoral: Ligature of External Iliac Artery: Cure.

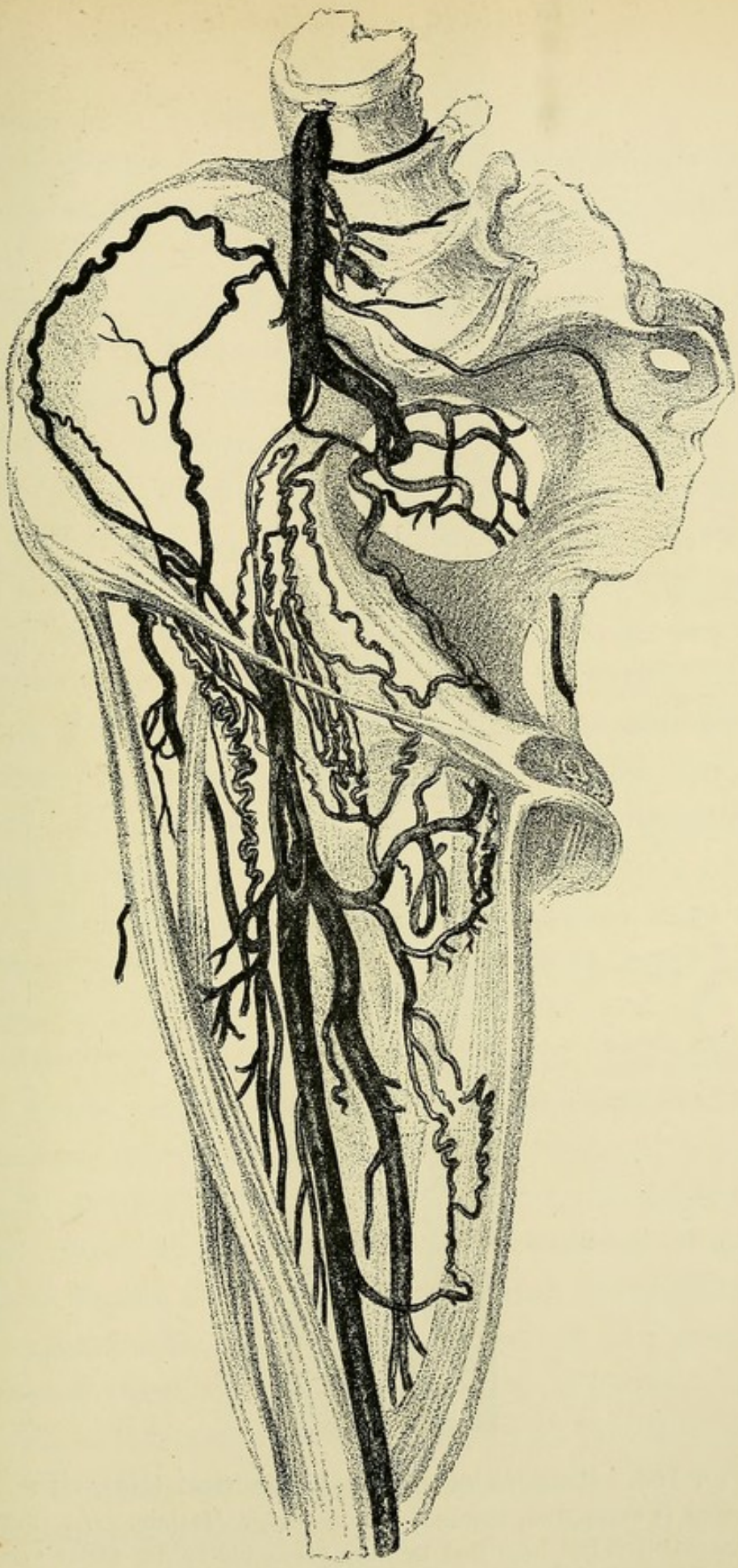
Specimen in Guy's Museum. No. 1519[12].

A gardener, aged 39, admitted to Guy's under Sir Astley Cooper with a large aneurism of common femoral size of a cocoa-nut, first noticed 6 months before admission; ligature of external iliac: June 22, 1808. Two ligatures used $\frac{3}{4}$ inch apart, the upper ligature after being tied was passed through artery below loop to prevent slipping: artery divided between ligatures: vessel appeared healthy: much suppuration occurred in wound: ligatures discharged on the 17th day: on the 8th day the sac of aneurism opened through the skin and discharged dark blood: this continued till the 16th day, when the sac sloughed and came away, leaving a granulating surface: suppuration was profuse: finally both wounds healed at end of twelve weeks. Patient survived $18\frac{1}{2}$ years. Limb injected, dissected and dried; it shews nearly the whole of the external iliac replaced by a small fibrous cord.

The common femoral is pervious, and on it near the origin of the profunda was a mass of connective tissue, causing adhesion of the artery to the surrounding parts: "there can be little doubt that the original opening of communication "between the sac and the femoral trunk had existed at this spot, but it would "seem equally apparent that as the aneurismal tumour became obliterated in the "progress of the cure after the operation, the opening into the vessel also became "closed, while the integrity of the arterial trunk above and below the sac was "maintained continuous and entire." See also figure 156, page 320.

The specimen shews clearly that the artery is pervious and of normal calibre opposite the site of the aneurism.

See also *Med. Chi. Trans.*, Vol. iv. (1813), and *Guy's Hospital Reports*, Vol. i. (1836), from which this drawing is reproduced.



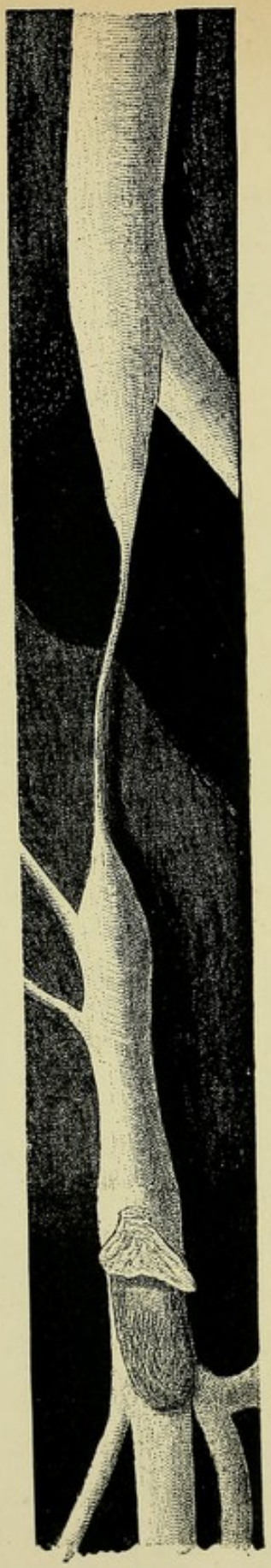


FIG. 156. Remains of sloughed Aneurism (Nat. size).

Drawing is made from specimen now in Guy's Museum. No. 1519[12].

The external iliac had been tied 18 years previously by Sir Astley Cooper for aneurism of the common femoral: (see also figure 155 which is from the same case). At the lower part of the artery is seen, attached to its wall, some loose connective tissue which is the remains of the aneurism which had burst and discharged externally.

no doubt blood found its way during life; the aneurism, therefore, cannot be regarded as cured although it had remained stationary, or nearly so, for a considerable time.

There is in Guy's Museum a very interesting specimen from a case of femoral aneurism, for which Sir Astley Cooper ligatured the external iliac; the sac subsequently ruptured and discharged its contents, the patient recovering. After death from another cause the femoral artery was found to be pervious; here, then, it may be said, is a case in which the artery was patent and yet the aneurism was cured, but it was not cured by becoming filled with clot in which organization occurred; strictly speaking, it was removed, the wound in the artery healing. The artery must have been blocked at the time this occurred, or there would have been fatal hemorrhage. (Figures 155 and 156.)

A somewhat similar case of spontaneous cure of an aneurism by sloughing has recently been recorded by Oliver—

A man aged 52 had an aneurism of the right subclavian artery: eight months after it was first noticed it was treated by electrolysis; pulsation ceased, but soon recurred: nine months later the tumour extended considerably both above and below the clavicle; it suppurated and finally a huge slough weighing more than one pound separated: the mass consisted of the aneurism, the middle third of the clavicle, and a portion of the subclavian artery and brachial plexus: in the deep wound that was left the two ends of the subclavian artery could be seen plugged with clot.

Figure 157 is from a case in which a subclavian aneurism, involving the second and third parts of the artery, underwent spontaneous cure. Here it is seen that the artery at each extremity of the aneurism is closed with connective tissue.

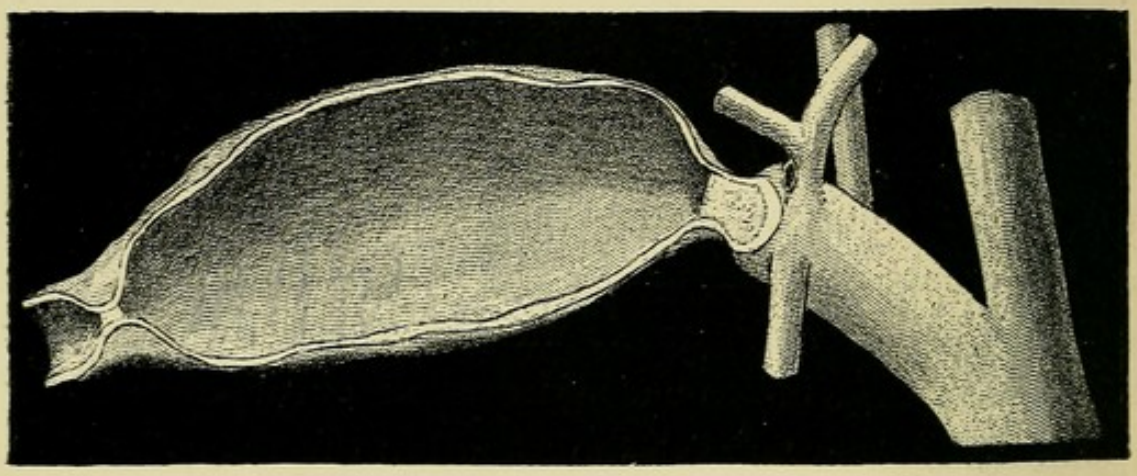


FIG. 157. Spontaneous cure of Subclavian Aneurism.

From S. Bartholomew's Hospital Museum. No. 1549.

From a woman, ætat. 39. The first part of the artery is healthy. The aneurism contained laminated fibrin. At each extremity the aneurism is occluded by a plug of fibrous tissue adherent to the wall of the artery. The specimen hangs vertically in the bottle.

It has been thought by some that the success of the Hunterian operation for popliteal aneurism is due to its not causing a complete arrest of the circulation in the popliteal artery but allowing a small stream of blood to flow through it, and thus causing the aneurism to become slowly filled with laminated clot; this if it occurs will constitute a fatal exception to Scarpa's law; it is therefore worth while to consider the question at some little length.

By far the most common of the external aneurisms is that of the popliteal artery; and it was these that Hunter

had chiefly in his mind when he devised his famous operation. At that time the only methods of treatment were amputation, and what is now known as the old operation, or that of Antyllus. It consists in incising the aneurism, turning out the clot and securing both ends of the artery; as far as popliteal aneurism was concerned it was almost invariably fatal. Hunter himself performed it several times, but never with success; the estimate that Percivall Pott, at that time the leading surgeon in London, expressed of the operation was no doubt strictly just. In one of his lectures he asks how femoral and popliteal aneurisms are to be cured, and replies:

"If a man was to answer from theory he would say "that the skin is to be divided, the extravasated blood "is to be cleared away, and the artery to be tied above "and below the dilatation; in short, what is called the "operation for the aneurism is to be performed.

"Sorry I am to find myself obliged to say that as far "as my observation and experience go, such operation, "however judiciously performed, will not be successful, "that is, will not save the patient's life.

"In both these aneurisms, the femoral and the popli- "tean, it most frequently happens that the artery is not "only dilated and burst, but it is also distempered some "way above the dilatation, particularly the poplitean. "This may very probably be one reason why the ligature "is in general so unsuccessful.

"The want of collateral branches of sufficient size to "carry on the circulation is another very powerful im- "pediment. Whether these may be allowed sufficient to "prostrate the attempt by the operation, I will not take

"upon me to say, but certain I am that it does not "succeed. I have tried it myself more than once or twice. "I have seen it tried by others; but the event has always "been fatal. Excessive pain, a high degree of sympto- "matic fever, great tension of the whole limb rapidly "tending to gangrene, and ending in mortification both "upwards and downwards have destroyed all those, whom "I have seen, on whom the operation of tying the artery "has been practised."

Pott advised amputation and Bromfield, one of Hunter's colleagues at St George's, went further and regarded all interference as inadvisable.

What Hunter himself thought may be seen from one of his lectures.

"When the aneurism," he says, "has arrived at the "stage Mr Pott describes, perhaps the only thing is to "amputate above the dilated part of the artery; but "Mr Pott should have considered that before these "threatening symptoms there is a stage when all the "surrounding parts are sound. * * * The earlier, "therefore, the operation for the aneurism is performed "the better, not waiting with the expectation that the "increased size of the aneurism will produce an increased "size of the collateral branches. That the popliteal "artery, according to Mr Pott, is oftener diseased above "the aneurism than other arteries I cannot well determine, "but can see no reason why it should be so. If the artery "cannot be tied above the aneurism in the operation "where can it be tied if the limb be amputated? *Why "not tie it up higher in the sound parts where it is tied "in amputation and preserve the limb?* * * * *

"Mr Bromfield objects to every operation, either ampu-
"tation or for the aneurism; this would be just, if what
"he asserts was true, viz. that the whole of the arterial
"system is in general diseased, which, however, is certainly
"not the case. He says, too, that 'the injecting of parts
"'in dead bodies having shewn that in particular subjects
"'the branches sent off have now and then formed anas-
"'tomoses with other branches given off lower down, has
"'led to very extravagant notions of the smaller branches
"'being always able to carry on the circulation; and an
"'extravagant proposition has been suggested by some
"'people to tie up the principal trunk of an artery in the
"'extremities. I once saw an attempt of this kind in a
"'true aneurism of the ham, in which I shall only remark
"'that the patient died, and I do believe that the embar-
"'rassments which occurred as well as the events of the
"'operation, will deter the gentleman (meaning me) who
"'performed it from making a second attempt in a similar
"'case?' Now unfortunately, either for Mr Bromfield or
"myself, this is the very case from which I have formed
"favourable ideas of the success of future operations of a
"similar nature. A young man had for two years a pain
"in the calf of his leg similar to cramp; at length he
"received a blow in the ham, and a swelling with pulsation
"appeared soon after. I gave it as my opinion that the
"old operation should be performed, and if it did not
"succeed then to amputate. The operation was per-
"formed and the patient apparently was going on well till
"the fifth day, when, the ligature giving way, the artery
"burst above, either from its being tied too tight or too
"loose. I was now obliged to dilate the wound still

"higher, and take up the artery higher up; but, appa-
"rently from the loss of blood before the tourniquet could
"be applied, the patient died a day or two after. The
"leg and thigh were both found to be putrid and emphy-
"sematous, and that was the case even above the aneurism.
"I do not know exactly how to account for the failure of
"this operation, whether from the unsoundness of the
"vessel, or the tightness or the smallness of the ligature;
"but from the appearance of the limb before the bursting
"of the artery and the injection of the vessels after death,
"all seemed favourable for the operation. The artery
"also was sound above the part where the first ligature
"was applied. From these considerations I should cer-
"tainly be encouraged to perform the operation again,
"though this was the case which led Mr Bromfield to
"condemn the operation." Hunter goes on to mention a case of femoral aneurism, which was cured by the old operation by Mr Bromfield, jun., and a case of popliteal aneurism cured by the same operation by Mr Martin, "the patient soon got well and was able to dance." He concludes the lecture by stating that "In December, 1785,
"I performed the operation at St George's Hospital in a
"case of popliteal aneurism in a manner different from
"that ordinarily practised and with success. The particu-
"lars of this case are given in the *London Medical Journal.*
"I would only observe that in future I would advise only
"tying the artery in one part, and not to endeavour to
"unite the wound by the first intention. In that case
"four ligatures were applied to the artery."

It will be noticed that although this lecture was delivered more than a year after his historic operation,

Hunter does not yet recommend his method but modestly alludes to it as "a manner of performing the operation "different from that ordinarily practised."

The account referred to in the *London Medical Journal* is not by Hunter himself but by Sir Everard Home who was at that time his assistant. Home relates how the aneurism itself was not touched, how the femoral artery was exposed from the front of the thigh and how it was tied with four ligatures which were drawn "so slightly "as only to compress the sides of the vessel together;" the reason for using four ligatures being "to compress "such a length of artery as might make up for the want of "tightness, as Mr Hunter chose to avoid great pressure on "the vessel at any one point." Home, however, says nothing about Hunter's doubts as to the popliteal artery being really diseased, nor his surmise that the hemorrhage which followed the old operation might be due instead to tying too tightly, but he boldly asserts that the popliteal artery is always diseased and thus misses the point of one of the great advances Hunter made.

It is clear then why the artery was taken up in the thigh and why its coats were not injured; it remains to be seen why it was thought unnecessary to interfere with the sac.

As early as 1710 Anel had cured a traumatic aneurism at the bend of the elbow by ligaturing the brachial artery above it, the sac being left untouched; 60 years later Sabatier cured a femoral aneurism by instrumental compression: in the autumn of 1785 Desault treated a popliteal aneurism by merely ligaturing the artery immediately above the sac. Desault failed in his object for

the sac ruptured. Sabatier's case was not recorded: Anel's was duly published and it is one of which our Gallic brethren are justly proud. Hunter however knew nothing of it, he was too busy reading in the book of Nature.

It appears that about this time Ford, surgeon to the Westminster Dispensary, had under his care a patient with a femoral aneurism on the right side and a small popliteal aneurism on the left; the femoral aneurism gradually got worse and eventually caused the death of the patient. Meanwhile the popliteal aneurism had disappeared but at the post-mortem examination it was found on cutting down to the popliteal artery that it was enlarged to the size of a hazel nut; "on opening the artery "both above and below this tumour and endeavouring to "pass a director and a probe, it was found to be quite "impervious to the instruments although some force was "used; and upon further examination it was found "plugged up by a substance of a firm and hard con-"sistence." Ford kept the specimen and shewed it to Hunter, and a drawing of it was made which was reproduced in the plates to Hunter's works (Fig. 158).

Somewhat later Ford had another case of femoral aneurism which Hunter saw in consultation with him; on this occasion Ford no doubt told Hunter of the spontaneous cure of the popliteal aneurism: in this second case they agreed to try instrumental compression on the artery above the aneurism, thereby shewing that Hunter thought it possible that an aneurism might be cured without being opened: the pressure could not be borne and the aneurism was treated simply by rest in bed,

FIG. 158. Spontaneous cure of Popliteal Aneurism.

Reproduced from Plate XXIV. figure 2, of Hunter's works edited by Palmer (1835).

Hunter's notes say "This was shewn to me by Mr Ford. It is the aneurism "which got well, but by what means was not known; or whether it had really "been an aneurism or only a contraction of the artery as I once saw in a young man, "a patient in St George's Hospital, and the small dilatation of the artery took place "after the obliteration of the artery, or whether the aneurismal part had contracted "to this size, is not now easily ascertained."

That it really was an aneurism, seems to be established by Ford's account of the case in the *London Medical Journal*, Vol. IX., and by its resemblance to a specimen now in the Hunterian Museum (No. 3265), the description of which is: "Parts of a femoral and popliteal artery and vein. The lower part of the artery "is completely closed and in nearly two inches of its length is contracted into a solid "cord. Above this for two inches the canal is full of firm dark coagulum: still "higher up the artery is pervious, but its inner coat is thickened and corrugated "and has fatty deposits beneath its surface. It was believed that there was an "aneurism of the popliteal artery eleven years before death, and that it had under-"gone a spontaneous cure by the sac becoming full of coagulum. From the museum "of John Howship."

Ford had also under his care another case of popliteal aneurism, which underwent spontaneous cure. Desault also described a similar case.

under which treatment it underwent "spontaneous" cure, but this result could not have influenced Hunter in planning his operation, for it was not attained in time to have done so.

Hunter performed his operation five times in all; in every case for popliteal aneurism; in the first, that of a coachman aged 45, he used four ligatures, they were tied only sufficiently tight to bring the sides of the artery in contact; the vein was included in the ligatures; the case did well, but portions of the ligatures continued to discharge for six months; the patient survived the operation 15 months, dying of a remittent fever; the specimen is in the Hunterian Museum (no. 3258); the popliteal artery is impervious where it enters the aneurismal sac.

The second patient was a trooper, 40 years of age; one strong ligature was used so as not to injure the coats of the vessel, the vein was included in the ligature; the wound was plugged from the bottom as it was thought that the slow convalescence of the first case was due to the shutting in of the ligatures by the premature healing of the wound. On the 14th day the ligature came away, on the 19th day hemorrhage occurred and again on the 20th, when the artery was religatured in the wound; but on the 26th day another and fatal hemorrhage occurred.

The third patient was a postillion aged 35, a single ligature was used and the wound brought together, the fatal result in the second case being attributed to the plugging of the wound; the ligature separated on the 14th day and the patient made a rapid recovery.

The fourth patient was a coachman aged 36 years; a single ligature was used and for the first time the vein

was not included; the ligature separated on the 29th day, but some irritation lasted about the wound so that the patient did not leave the hospital till the 14th week: he survived the operation 50 years; the limb is now in the Hunterian Museum (no. 3259: see also fig. 160, p. 334).

The fifth and last patient on whom Hunter performed his operation was a man aged 42 years; a single ligature was used, and the vein was not included; the ligature separated the eleventh day, the wound healed by first intention, the local irritation was very slight, and the patient recovered without any complication.

Thus Hunter cured four out of the five patients on whom he operated, and his last case ran an almost ideal course.

Equally brilliant results were obtained by other surgeons and the operation soon became established throughout Europe, in fact it constituted as Erichsen says, "one "of the greatest and most direct advances in surgery that "has ever been made by the single act of one man."

Now it has been thought that the success of Hunter's operation depends in part at least on a small current of blood continuing to flow through the popliteal artery after the ligation of the femoral; it being supposed that the result of this would be to cause the clot to be deposited slowly and to be firm and laminated, and thus less likely to be washed away than uniform clot: whatever truth there may be in this it is certain that Hunter had no such idea; the expression used by Home "taking off the force of the circulation" is seen from the context to mean, taking off the whole force and not merely reducing it. (See Note p. 347.) Moreover there is no truth in the above

view, for the formation of "white" laminated clot in an aneurism is a matter of days and weeks if not of months and years, and the result of the Hunterian operation is nearly always to immediately occlude the vessel; sometimes some slight pulsation can be felt after the operation but this completely ceases in a day or two, and long before such clot could be deposited. In all the specimens in museums of popliteal aneurisms cured by ligature of the femoral the popliteal artery is seen to be impervious; it is so in Hunter's first and fourth cases in the Royal College of Surgeons' Museum; it is so in four specimens in St Bartholomew's Museum, 18 months, 6 years, 6½ years, and 20 years after operation. No doubt it does occasionally happen that the collateral circulation round the seat of ligature enters the femoral and popliteal arteries above the aneurism to such an extent that the popliteal artery remains pervious and the aneurism continues to pulsate; but then these cases are not cured; this in fact is the one weak point of Hunter's operation; these failures are happily rare but a considerable number are now on record. Soon after the introduction of the operation Chopart had a case in which the aneurism continued to pulsate and was not cured. Sir Astley Cooper had a case in which secondary hemorrhage necessitated amputation; it was found on examination of the limb that the popliteal artery was pervious down to and into the sac of the aneurism. Gunning had a case in which the aneurism ceased to pulsate for four years and then recommenced as strongly as ever; at the wish of the patient the limb was amputated; the artery was found to open into the aneurism; the specimen was placed in St George's Hospital

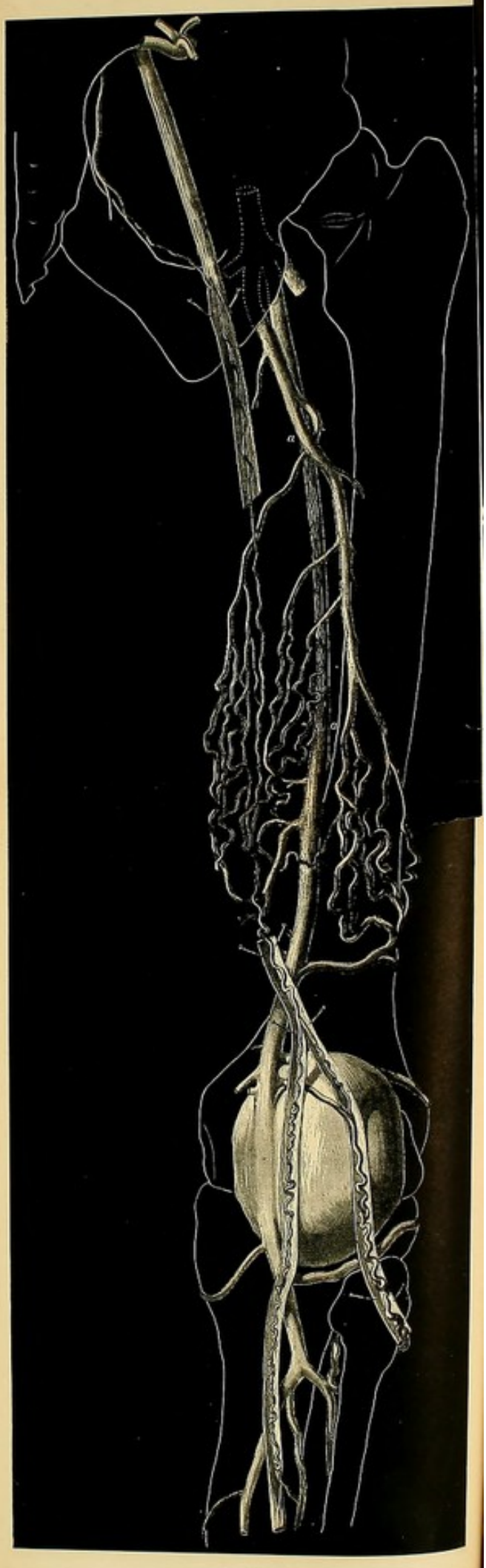


Fig. 159.

FIG. 159. Failure of Hunterian method. (After Porta.)

From Porta's Delle Alterazioni patologiche delle arterie per la legatura e la torsione, plate XIII.

From a patient in Milan Hospital. The superficial femoral artery was ligatured for a popliteal aneurism: pulsation disappeared for four years, but then recurred. Seven years after the first operation, the external iliac artery was ligatured. Patient only survived operation 78 hours. The parts are seen from behind.

It shews a large aneurism in the popliteal space. The superficial femoral artery is impervious from (*a*) to (*a*); there is a large collateral circulation round the seat of ligature, but the blood conveyed by it re-enters the femoral and popliteal arteries above the aneurism. The result was that a large quantity of blood passed through the aneurism, the popliteal artery remained pervious, and a cure did not take place.

FIG. 160. Success of Hunterian method. (Hunter's fourth case.)

Specimen in Hunterian Museum. No. 3259.

Male, ætat. 36 years. The popliteal aneurism was somewhat lower than usual: leg swollen, veins turgid: femoral artery ligatured: vein not included in ligature as it had been in the three previous cases: ligature separated 29th day: left the hospital at end of 14th week.

Patient survived operation 50 years. Specimen was presented to the Museum of R. Coll. Surgeons, by T. Wormald, Esq. The drawing, which is a back view, shews that the aneurism is shrunk to a small fusiform mass of connective tissue at the back of the tibia; also that the whole length of the superficial femoral and popliteal arteries from the origin of the profunda femoris (*a*), to the division into the tibials (*a*), was quite impervious and had dwindled into a fine fibrous cord. The comes nervi ischiatici and the branches from the deep femoral, were the chief vessels which carried on the collateral circulation. There is a small branch from the comes artery to the lower end of the popliteal just above its bifurcation; another branch of the comes runs to the front, round the neck of the fibula: vide *Trans. Soc. Improvement Med. and Chi. Knowledge*, Vol. I., 1793.

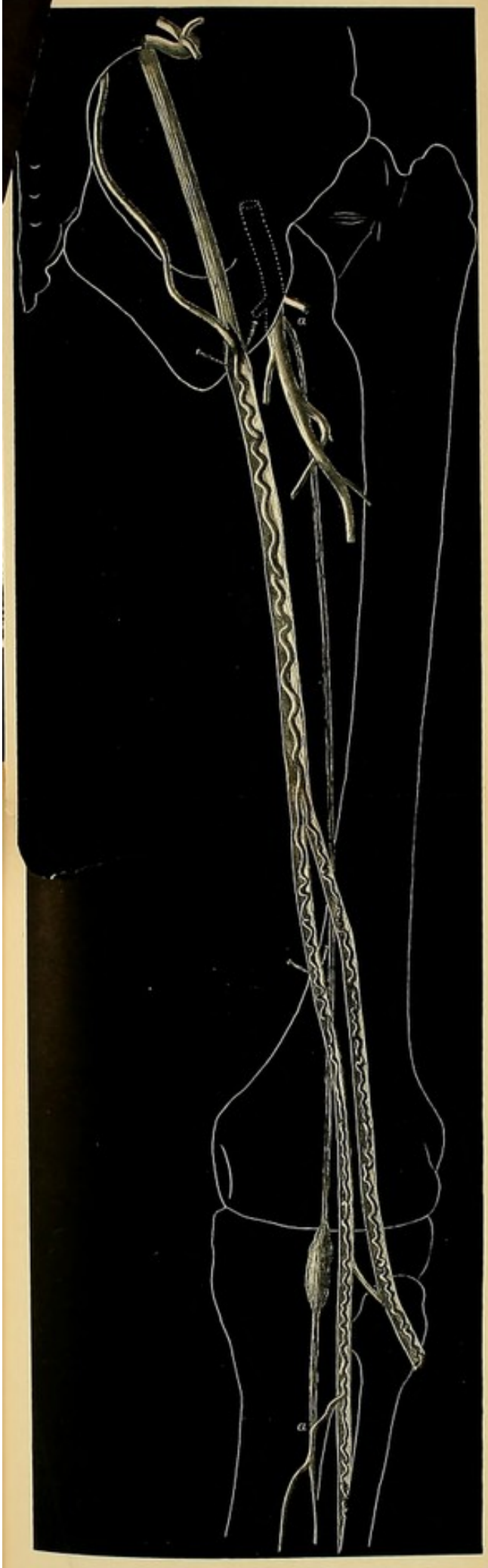


Fig. 160.

Museum where it may still be seen. Another such case is described by Porta who shews the collateral circulation in a beautiful plate from which figure 159 has been drawn; an almost identical specimen may be seen in St Mary's Hospital Museum; failures also happened to Roux and Spence.

Although in successful cases the femoral artery is obliterated at the seat of ligature and the popliteal at the seat of aneurism, yet there is generally a certain extent of the femoral and popliteal arteries pervious between the two occluded parts, as seen in Stanley's case (Figure 161). It is seldom that the whole length of the arteries from the origin of the profunda to the bifurcation of the popliteal is obstructed; this however does sometimes occur, as in Hunter's fourth case, where the whole length of the arteries is reduced to a small fibrous cord (see Figure 160): the opposite extreme is seen in Miloni's case, in which the operation failed (Fig. 159). To what extent the arteries will become obliterated cannot be told before operation, and indeed the circumstances which determine it are not very clear, for in Sir Charles Bell's case, in which there were two superficial femorals, only one of which was ligatured—a case in which of all others it would be expected that the operation would fail—although immediately after ligature the aneurism pulsated as strongly as before, yet it completely ceased on the third day; after death (from septicæmia) on the sixth day the aneurism was found completely filled with clot. (Fig. 162.)

The occasional failure in this way of Hunter's operation has recently attracted the attention of Sir William Savory; he points out how seldom it is that the popliteal artery is

FIG. 161. Femoral and Popliteal Arteries from a case in which the Superficial Femoral was ligatured.

From S. Bartholomew's Hospital Museum. Specimen 1551 c.

Patient was under the care of Stanley, who ligatured the femoral at the apex of Scarpa's triangle. Patient survived the operation 20 years, dying of rupture of aortic aneurism.

The aneurism (*b*) is reduced to a small fusiform mass of connective tissue, about the size of half a walnut. The femoral artery is obliterated for $1\frac{3}{4}$ in. (4 cm), from *a* to *a*, forming a fibrous cord. Small portions of altered clot are seen adherent to vessel wall in the patent portion of the vessel between the seat of ligature and of aneurism. The popliteal artery is also obliterated opposite the remains of the aneurism and for a short distance above and below.

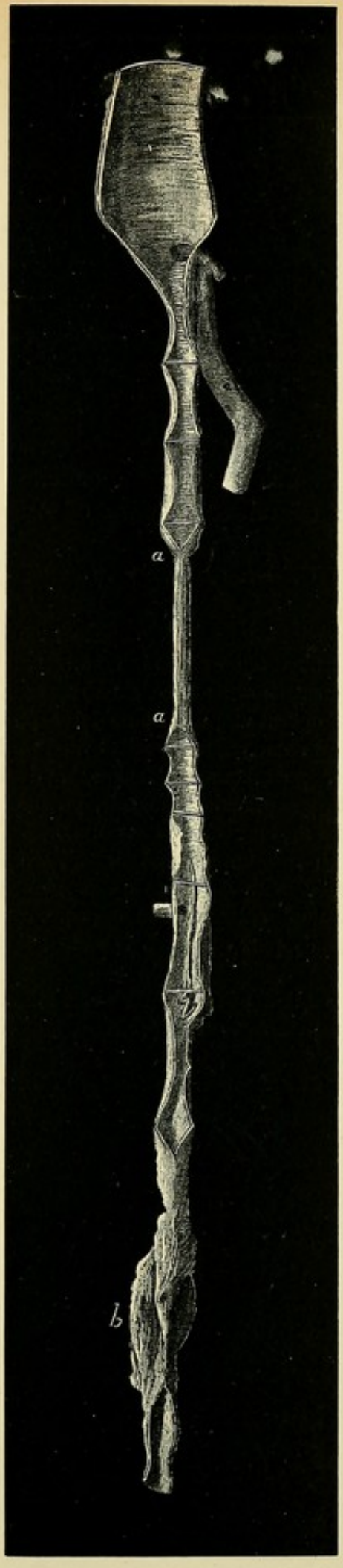

a
a
b

FIG. 162. Popliteal Aneurism: two Superficial Femorals. Ligature of one: commencing cure of Aneurism.

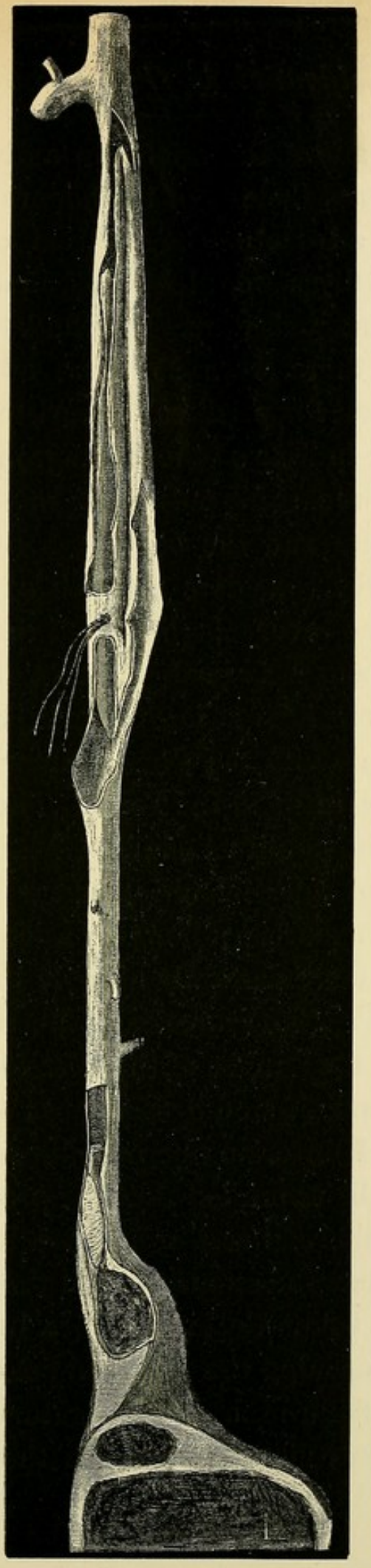
Specimen in University College Museum. No. 1289.

Patient was a negro ætat. 42. Left popliteal aneurism, noticed 4 years: Hunterian operation: superficial femoral easily found: pulsation in aneurism ceased for a few seconds after application of ligature, then recurred and in half an hour was nearly as strong as before operation; 66 hours later pulsation in tumour ceased: symptoms of septicæmia now came on, and patient died on the 6th day.

Figure shews at upper part common femoral and origin of profunda, which is cut short; there are two superficial femorals, on one of which a ligature has been applied: this contains a long clot in its upper part, and a lower clot extending to its reunion with the unligatured artery, which is quite patent. The aneurism is completely filled with clot: its upper part and smaller portion was in the usual position for popliteal aneurism opposite the bend of the knee, the lower and larger portion, only part of which is shewn in the drawing, is unusually low.

The popliteal artery is filled with clot for some distance above the aneurism, from this the femoral is patent to the point at which the superficial femorals unite.

See also Charles Bell, *The London Medical and Physical Journal*, Vol. LVI. (1826).



found diseased above the aneurism and suggests that it should be ligatured instead of the femoral.

Bowlby too advocates the ligation of the popliteal: he argues that there would be less chance of gangrene and of recurrent pulsation. He finds that during the last ten years there were 23 cases of ligature of the superficial femoral for popliteal aneurism at St Bartholomew's Hospital: in four of these gangrene ensued and in three there was recurrent pulsation: one of these last was cured by pressure and the other two by ligation of the popliteal. Bowlby considers the risk of gangrene would be less when the artery is ligatured below the anastomatica magna: but it is probable that the proximal clot would in some instances at least extend into this vessel as we have seen it do into the thyroid arteries after ligation of the common carotid in the horse. The ease with which the ligation of the popliteal can be effected in the cadaver is no measure of the difficulties of the operation in the presence of an aneurism: however as the ligature of the popliteal would more certainly occlude that artery it must be conceded that it is theoretically the better procedure: but considering the danger of the operation in the vicinity of a large tumour, it would seem that the practical advantages of the Hunterian operation must prevail.

It may here be remarked that the proper treatment of these cases of failure, if rest in bed does not succeed, is to religature between the aneurism and the seat of ligature, and preferably below the anastomica magna: this does not necessitate opening the popliteal space.

In cases of failure too of the ligature with aneurisms of other arteries, religature between the seat of previous

ligation and the aneurism is no doubt the first operation to be considered: if this second ligature arrests all pulsation in the sac there is no need to do more; excision would offer no advantage, for if the supplying artery can be safely tied after excision it can also be so tied in continuity without excision: if it is found that the ligature does not arrest all pulsation, excision can be proceeded with at once. An interesting case in which a femoral aneurism was excised is recorded by Rose: for an aneurism of the superficial femoral the external iliac was tied with catgut: a slight fluttering pulsation was noticed the next day; it continued and increased so that in two years' time the aneurism was pulsating as freely as at first: the aneurism was now excised; the femoral vein was found adherent to the sac and had to be removed with it: no œdema or gangrene resulted and the patient made a rapid recovery.

An aneurism which continues to pulsate after the ligature of its artery above it, and in such a position that no large branch is given off between the ligature and sac, must be regarded as of cirsoid character. If operative treatment is considered advisable the best course, if practicable, is to excise the aneurism: a case in which this was done by one of us for a cirsoid aneurism of the dorsalis pedis artery will be found in the *Transactions of the Royal Medical and Chirurgical Society* for 1885.

In some cases although pulsation is arrested the tumour remains as a large fluctuating swelling, which cripples the patient; the treatment for this condition is to incise the aneurism aseptically and turn out the contents, allowing the sac to granulate from the bottom. A case of

this description was recently in St Thomas's Hospital, and will be found reported in the *Lancet* of April 19th, 1890.

We may conclude then that, as in all cases of popliteal aneurism which are cured by ligature of the femoral the popliteal artery is obliterated, and as in all cases which

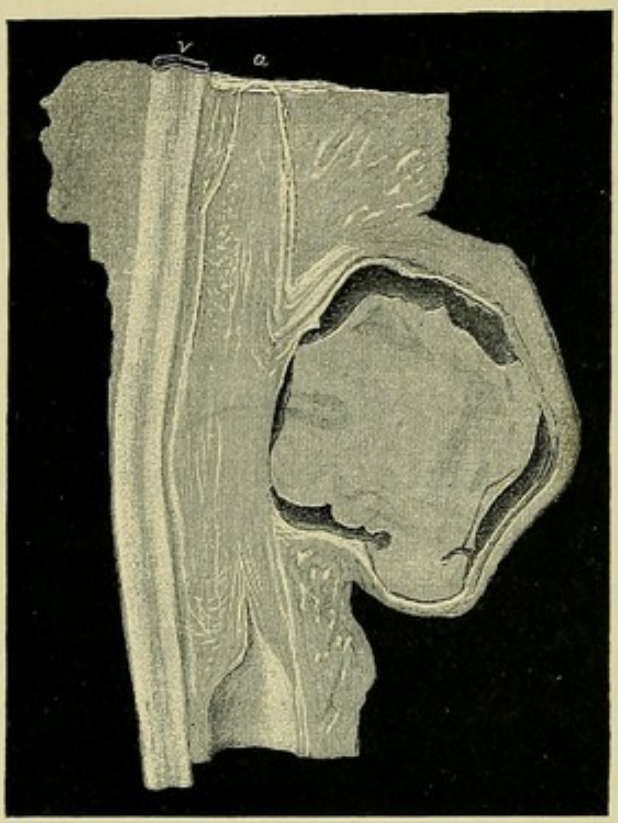


FIG. 163. Popliteal Aneurism cured by digital compression (× $\frac{4}{3}$).

From Guy's Hospital Museum, 1519[36].

A man ætat. 32 was admitted into Guy's Hospital with left popliteal aneurism under Mr Bryant. It was cured by digital compression by students. Subsequently he had a right popliteal aneurism, which patient himself cured at home by digital compression. He died 2 years later. Cause of death unknown. Specimens were sent to Guy's.

The drawing shews the cured popliteal aneurism on the left side. The popliteal artery (*a*) is completely obliterated, and the vein (*v*) is normal.

are not cured it remains pervious, the results of the Hunterian operation offer no exception to Scarpa's law, but on the contrary strongly confirm it.

The same is true of the treatment by compression.

There are in Guy's Museum two specimens of popliteal aneurisms cured by digital compression, and in both of them the popliteal artery is occluded; one of these is represented in Figure 163.

There is also in St Thomas's Museum a specimen (Y 104^3) from a case of popliteal aneurism treated by digital compression; so extensive was the obstruction to circulation that gangrene ensued and the leg had to be amputated; the aneurism and artery are both filled with clot.

On the other hand there exist in St George's Hospital Museum two specimens of popliteal aneurism "cured" by compression in which the popliteal artery remained pervious; they are figured in Holmes's *System of Surgery* (Vol. III. p. 68). "This method of cure," Holmes remarks, "is however not to be desired, since the clot may yield "again to the force of the circulation and thus the disease "may recur." These cases then cannot be regarded as radically cured and constitute therefore no exception to Scarpa's law.

With respect to the method of flexion, we have failed to find a specimen of an aneurism which has been cured in this manner, but in St Bartholomew's Museum (no. 1552) there is a popliteal aneurism for which this treatment was adopted: pulsation was completely arrested, but symptoms "thought to be those of gangrene" came on and the limb was amputated; the specimen shews that the sac and the artery are both filled with clot, as was to be expected from the history of the case.

So also for the treatment by manipulation: this was introduced by Fergusson; the principle of the operation

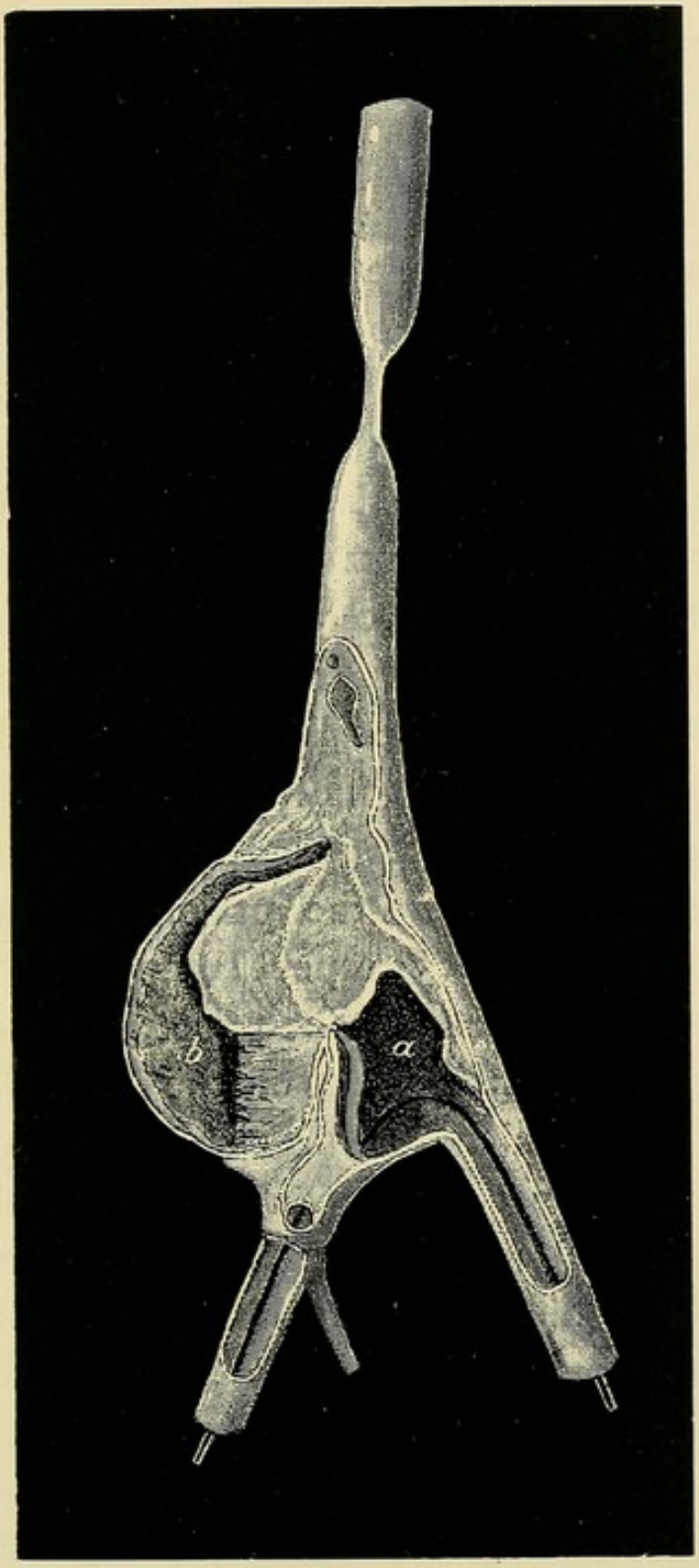
FIG. 164. Inguinal Aneurism not cured by Ligature. (Nat. size.)

Specimen in St Thomas's Hospital Museum, Y 97[1]*: see also Lancet, August,* 1887.

Man aged 42 was admitted to St Thomas's Hospital, under the care of Mr Anderson, with a large inguinal aneurism on the right side, which had been noticed 8 weeks, and a popliteal aneurism on the left side. The right external iliac was ligatured with stout chromic catgut. Pulsation ceased. One month later the popliteal aneurism was treated and cured by digital compression combined with electrolysis.

Two months after ligation of external iliac patient was allowed to get up; then pulsation in inguinal aneurism reappeared and continued till death of patient, 15 months later, of aortic aneurism.

Drawing shews artery above aneurism occluded: below there was free communication through the aneurism between the superficial and deep femorals, as shewn by a black bristle: in the half of the specimen preserved no obvious communication can be traced between the cavities in the aneurism marked *a* and *b*, the latter of which was in front: it is however highly probable that the regurgitant stream filled both cavities.


b
a

is to displace a portion of the laminated clot in the sac of the aneurism in the hope of thus causing the obstruction of the vessel; we know of no specimen of an aneurism cured in this manner, but the following account of one of the most successful cases points to the circulation through the artery being completely stopped.

An albino aged 53 came under the care of Mr Little of Donegal with a large aneurism of the right subclavian artery; gentle but steady pressure was made with the thumbs alternately over the sac. For two days after this treatment no change of any kind was perceptible; on the third the radial pulse was weaker and the arm colder; these symptoms increased so that on the tenth day there was no pulse in the radial, brachial or axillary arteries. The aneurism became more solid and finally all pulsation ceased, and the tumour dwindled to the size of a small walnut. The arm, which was partially paralysed and very cold, recovered, and when the patient was seen a year later the cure was in every respect complete.

In successful cases of galvano-puncture also the artery seems to be obliterated; one of the most satisfactory cases was that in which Abeille cured by this method a subclavian aneurism the size of a hen's egg; the tumour became quite solid, all pulsation ceased, the arm became cold and the radial pulse could not be felt for three days; the collateral circulation was enlarged; the brachial pulse remained permanently smaller than in the other arm; the aneurism too shrank and finally disappeared; there was no return of the disease, at least for the two years the patient remained under observation; it is true it was thought three months after the operation that on

deep pressure a return of pulsation could be felt in the affected artery, but it is clear from the symptoms that it was occluded at the time of the operation and in all probability it remained so.

It is not even sufficient for the artery to be closed from the seat of ligature right down to the mouth of the aneurism, but the artery beyond the aneurism must be closed also. Figure 164 shews an inguinal aneurism for which the external iliac artery had been ligatured: the aneurism however was not cured, for the blood re-entered it from below through some of the lower branches which carried the collateral circulation.

It seems then that Scarpa is right and that whatever the method of treatment adopted, be it rest, ligature, compression, galvano-puncture, manipulation or any other plan, it will only be permanently successful if the artery at the seat of aneurism is obliterated, and conversely it may be said that any method of treatment which effects this will be successful, that method being best which most surely attains this end with least danger and suffering to the patient.

NOTE (see page 331).

That Sir Everard Home means by "taking off the force of the circulation" taking off the whole force and not merely reducing the current will be clearly seen from the following extract from *A supplement to the Account of Mr Hunter's Method of performing the Operation for the Popliteal Aneurism inserted in the Seventh Volume of this Work. Communicated in a second Letter to Dr Simmonds by Mr Everard Home, Surgeon, F.R.S.*, in the *London Medical Journal*, Vol. VIII. p. 130 et seq. (1787).

"The conclusion to be drawn from the above account appears to "me to be a very important one, viz. that the simple taking off the

"force of the circulation from the aneurismal artery is sufficient to "effect a cure of the disease or at least to put a stop to its progress "and leave the parts in a state from which the actions of the animal "economy are capable of restoring them to a natural one.

"In confirmation of this account, that the cure of an aneurism "depends on taking off the force of the circulation, I shall mention "a case that recovered without any assistance from art and which "I consider to have got well upon the same principle. This case "was more particularly under the care of Mr Ford, Surgeon, in "Golden Square, who will, I hope, lay a particular account of it before "the public: I mean to notice it no farther than by endeavouring "to account for the recovery, which may be explained by Mr Hunter's "observations on mortification.

"The aneurism was in the femoral artery and the swelling "appeared upon the anterior part of the thigh a little above the "middle, extending upwards as it increased in size nearly to the "brim of the pelvis. Every attempt towards a permanent com- "pression of the artery above the tumour just as it passes over "the brim of the pelvis proved ineffectual: the tumour enlarged "to a very considerable size; a great degree of inflammation and "swelling took place in the sac and common integuments; and "mortification appeared to be taking place in the skin which lay "over it: while in this state the pulsation, before very evident in "every part of the tumour, was no longer to be felt, nor even "in the artery immediately above it, so that the steps preceding "mortification had certainly taken place, the blood in the artery "above having coagulated[1]; and this circumstance was sufficient to "prevent the absolute mortification coming on, for the artery above "becoming impervious, put a stop to the dilatation of the sac and all "its consequences.

"From the time the pulsation stopped, the swelling and inflam- "mation subsided, although exceedingly slowly, and the tumour "diminished, becoming more firm and solid, and at the time of "writing this paper is very much reduced in size, and to the feel "resembles that found in the ham of the patient who is the subject "of this paper."

[1] "In those patients who die in consequence of mortification of any part of their "body the artery leading to that part is found always completely stopped up for "several inches in length by a firm coagulum: this must take place prior to the "mortification and seems intended for the wisest purposes to prevent hemorrhage.

"*Taken from Mr Hunter's Lectures.*"

CHAPTER XIV.

THE SURGERY OF THE ARTERIES: THE FOUNDATION OF SURGERY.

Views and practice of the earlier and later surgeons: Erasistratus: Herophilus: Celsus: Galen: Paulus Egineta: Harvey: Paré: Monro: Heister: Platner: Callisen: Benjamin Bell: Hunter: Deschamps: Desault: Abernethy: Jones: Scarpa: Charles Bell: Assilini: Crampton: Formy: Guattani: Simpson.

The surgery of the arteries, and indeed the science of surgery itself, dates from the foundation of the schools of Alexandria, by Ptolemy Soter, king of Egypt (B.C. 285), for it was then that, by Erasistratus and Herophilus, human anatomy was first systematically studied and taught. Previous to this, but little was known of the anatomy of the soft parts, but Hippocrates (B.C. 460) had worked at the skeleton, and Aristotle (B.C. 334) had an extensive

acquaintance with the vascular system from the dissection of the lower animals.

The operation of the ligature of arteries was the natural outcome of the Alexandrian studies, but it is not known by whom it was first practised. It is described by Celsus, writing B.C. 50, and by Galen, A.D. 130.

Celsus says, "But if pressure and astringents are in-"effectual to restrain the hemorrhage, the bleeding vessel "is to be taken up, and a ligature applied on each side "of the wound in it: the vessel is then to be divided, the "two parts of the vessel will become united by anasto-"mosing branches and the orifices will become oblite-"rated."

Galen writes, "If the artery be large, and if it be "cicatrized beyond the aneurism, the whole of it should "be cut through, and oftentimes that very practice "prevents the danger from hemorrhage, for it appears "plainly that when a complete transverse division is made, "both portions of the artery retract on either side, the "one above the part, the other below."

Paulus Egineta (A.D. 600) also practised the operation: "The artery having been cleared of the surrounding parts "is to be exposed with the same scalpels with which the "membranes have been divided; a needle being then "passed under it, the artery is to be tied with a double "ligature having previously been punctured in the middle: "suppuration must then be promoted till the ligatures "fall out."

The mode of operating recommended by these early surgeons is then to tie the artery in two places and to divide it between the ligatures.

It is strange that the ligature of arteries both for wounds and aneurisms should have been practised centuries before the discovery of the circulation of the blood: Harvey's work, "*De motu cordis et sanguinis,*" was published in 1618, but it does not appear to have had any immediate effect upon surgery.

A great diversity of opinion has always existed as to the best method of performing the operation: the practice of surgeons from early times to the present day seems to have been based on one or other of two great opposing principles: either of tying with considerable force in the belief that damage to the arterial wall was essential to obliteration, or a safe-guard against hemorrhage by tying the blood in: or of treating the artery with gentleness in the endeavour to cause its obliteration without inflicting any injury upon it.

In the middle ages the operation seems to have fallen into disuse, but it was revived by Paré about 1570. Referring to amputations he says, "The ends of the "vessels lying hid in the flesh must be taken hold of, "and drawn with this instrument [forceps] forth of the "muscles whereinto they presently, after the amputation, "withdrew themselves. In performance of this work you "need take no great care if you, together with the vessels, "comprehend some portion of the neighbouring parts, "as of the flesh, for hereof will ensue no harm, but the "vessels will so be consolidated with more ease than if "they being bloodless, the parts should grow together by "themselves," and with reference to aneurisms he says, "Divide the skin above the aneurism, and separating the "artery, pass a seton needle armed with a strong thread

"under it and allow the ligature to fall of itself. Na-
"ture will then generate flesh which will block up the
"artery."

Elsewhere he says, "Wherefore I must earnestly entreat
"all Chirurgeons, that leaving this old and too cruel way
"of healing [actual cautery], they would embrace this new
"which I think was taught me by the special favour of
"the Sacred Deity: for I learnt it not of my masters, nor
"of any other, neither have I at any time found it used by
"any: only I have read it in Galen that there was no
"speedier remedy for stanching of blood than to bind
"the vessels (through which it flowed) towards their roots,
"to wit the liver and the heart. This precept of Galen,
"of binding and sewing the veins and arteries in the new
"wounds, when as I thought it might be applied to
"those which are made by the amputation of members,
"I attempted it in many."

Paré carried out the details in a different way to that of the earlier surgeons, for he used a bundle of fine ligatures and included with the artery some of the surrounding structures, his object being to close the vessel without injuring it: and for the same purpose he used a small compress between the artery and the knot: "Then
"you shall tie your ligature sufficiently tight over a small
"compress of lint two or three folds thick and of the
"size of a finger. This will prevent the knot cutting
"into the flesh and at the same time certainly occlude
"the vessel: I can assure you that never after this
"operation have I seen a single drop of blood from
"vessels thus tied."

To Paré then belongs the credit, not only of re-

discovering the operation, but of being the first to insist on the advantage of avoiding injury to the arterial wall.

The same gentle treatment was recommended by Alexander Monro in 1725: he advised to avoid injury to the vessel a wide ligature not drawn too tightly.

Heister also (1718) was so impressed with the importance of this point that he recommends the use of a small pad between the artery and the knot.

Platner (1758) also advises the use of small pledgets of lint next the artery for the purpose of protection.

Callisen (1780) appears to have been on the same side, for he declared that the Celsian operation was both injurious and unnecessary: "Arteriam inter vincula mediam "discindere ob extremorum retractionem nocuum et super- "fluum merito habitur."

Benjamin Bell writes, in his *System of Surgery*, in 1787, "There is no occasion whatever for making the ligature "so tight on arteries as to run any risk of dividing them; "a much less degree of pressure than is commonly applied "or could have any influence in hurting them, being fully "sufficient for compressing them in the most effectual "manner."

John Hunter, too, was exceedingly careful only to bring the sides of the artery in apposition and with this object he, in his first case (1785) used a series of ligatures.

To Deschamps (1793) is due the principle of complete isolation of the vessel, and Desault (1798) was the first to state and demonstrate that the ligature might divide the two inner tunics of an artery.

At the commencement of this century, however, the operation of Celsus was again recommended by Abernethy

of St Bartholomew's Hospital, where this method still retains, we believe, its popularity, and has recently been advocated by Walsham and Howard Marsh.

Contemporary with Abernethy was Jones (1805). He carried out a large number of experiments on the arteries of lower animals: the conclusion at which he arrived was, that a single small round ligature should be used, and that it should be drawn sufficiently tight to rupture the coats. He writes, "if the two inner coats be not ruptured, "hemorrhage will occur": but this opinion was only a matter of inference: he made no experiments in which the coats were not divided: and he adds, "it cannot be "expected that I should illustrate these opinions by cases "or experiments; nor would it be easy to do so in the "case of dogs for whom Nature does so much." In fact, Jones practically admits that as far as experiments would go, the rupture of the coats is unnecessary.

In Italy, Scarpa (1817), Emeritus Professor in the Imperial and Royal University of Pavia, warmly advocated the gentler methods. "I do not hesitate to declare from "my own experience, as well as the experience of others, "that for obtaining the quick and permanent closure "of the principal arteries of the extremities, the choice "falls upon the single ligature with the interposition of "the small cylinder of linen spread with ointment between "the tape and the artery. This mode of ligature already "recommended by the greatest masters of the art of "surgery, Paré, Heister, and Platner, preserves all the "coats of the artery entire, it is easily and quickly applied, "and it is sufficient of itself alone, or by means of simple "pressure, to produce quickly in the artery the proper

"degree of *adhesive* inflammation which is required for "the speedy union and complete closure of the arterial "tube. And, considering the great number of cases of "this kind which have terminated favourably, compared "with others, in which a totally different mode of ligature "had been employed, I do not hesitate again to assert "that the good effects of this practice will always corre- "spond with our expectations."

Scarpa was the first to demonstrate that the rupture of the coats of an artery is not necessary in order to surely occlude it in continuity. His tape ligature (so called) was composed of from 4 to 6 waxed threads according to the size of the artery to be ligatured: his method of operating was as follows: (1) the artery was isolated and detached only sufficiently to allow the tape to pass round it: (2) the tape was tied over a cylinder of linen, as broad as the artery, and slightly longer than the tape was wide: the linen cylinder was smeared with ointment: (3) the constriction of the ligature brought the two opposite internal healthy parietes of the artery into actual contact: (4) the ligature was removed on the fourth day, "except in weak and extenuated patients" when it was not removed till the fifth or sixth.

It may be mentioned, that Sir Charles Bell, in his *Great Operations of Surgery* (1821), advises that "the "loop and knot of the ligature be sunk into the coats, "sufficiently to prevent the pulsation of the vessel, shifting "the ligature, but not drawn so tight as to cut the inner "coats of the artery."

The same idea of not injuring the artery underlay the use of metallic instruments for holding the opposite

sides of the vessel in firm contact; compressors for effecting this were devised and used by Deschamps (1793), Desault (1798), Assilini (1812), Crampton (1819), and others.

The coats too are not ruptured by the use of graduated compresses of linen or touchwood (Buzani, 1770), applied to the denuded artery: this method has been in past times successfully practised by Formy (1652), Guattani (1772), and others.

Acupressure of arteries depends also on the same principle. It was originally introduced by Simpson. His *Essay on Acupressure* was published in 1860: Pirrie and Keith supported him with a work published in 1867: by this method numerous successful results were obtained: but there is a great objection to the operation, as was pointed out by R. Lawson Tait (1865). "It happened," he says, "in one of my earliest experiments that I had "pushed a needle, as I thought, under both artery and "vein of a dog's leg. There it remained for eighteen "hours, and then was removed, as I intended allowing "the dog to live for some time after. But to my astonish-"ment, the dog fell dead in about a minute, and when "I examined the body, I found that the needle had "included scarcely half the vein, that it had pierced it, "that a clot had formed about the needle which had been "set loose on its removal and got impacted in the pul-"monary circulation. The same thing happened in another "experiment, where the needle had been in nearly thirty-"six hours."

As the result of his experiments, Tait concluded that the coats should on no account be ruptured, and as the

best method for effecting occlusion he recommended a form of arterial compressor.

Lastly, it may be added, that a like process occurs in Nature, when she effects the occlusion of great vessels by the pressure on them of tumours, such as an aneurismal sac or an abscess: examples of this mode of obliteration are to be found in the museums.

CHAPTER XV.

CHOICE OF THE OPERATION.

Three methods to select from. Celsian operation. Comments of Jones, Scarpa, and Bell. Accidents following this operation. Jones's operation. His views and argument. Conditions of Occlusion. Experiments of Howard. Effects of suppuration. Conclusion. Opinion of Erichsen.

There are three methods of ligating an artery in continuity, namely:—

1. Tying in two places and dividing between.
2. Tying in one place with rupture of the coats, and
3. Tying in one place without rupture.

Which is best?

With respect to the earliest operation, that of double ligature with division, it is to be observed in the first place that it is by far the most severe procedure, so much

so, indeed, that before it can be entertained it is necessary to shew that the milder operations are more dangerous: in other words, the whole burden of proof must rest with those who advocate this method.

Further, it is to be noted, as Scarpa and others long ago pointed out, that there are many arteries in the body on which this operation cannot well be performed. This is the case with the lower third of the common carotid, the first and third parts of the subclavian, the innominate, and in cases of inguinal aneurism, the iliacs. Indeed if this is to be regarded as the only safe method, all hope of bringing the ligature of the subclavian and other great arteries within the domain of uniformly successful surgery must be abandoned.

In sufficiently exposing, for example, the third part of the subclavian or the external iliac for the operation of double ligature and division, much more disturbance of the parts must ensue, and far more risk must be run of injury in the one case to the pleura and the sac of an axillary aneurism, and in the other to the peritoneum and the sac of an inguinal aneurism, than if the sheath of the artery were only exposed enough to allow an opening to be made in it just sufficiently large to admit the aneurism needle; for to this amount alone should the sheath be stripped. Indeed, the argument that the Celsian method "diminishes the risk of hemorrhage by "insuring that no part of the artery above the upper and "below the lower ligature is deprived of the nourishment "it receives from its sheath," ignores the proper way of ligating an artery in continuity. Even if it were proved (which it is not) that the separation of the sheath from

the artery increases the danger it would shew nothing, for the sheath should not be separated. Further, we have on many occasions removed the sheath for several inches from the carotids of horses before ligating, and have subsequently been unable to observe any difference, either by the naked eye or under the microscope, from those vessels in which the sheath has been left undisturbed. Senn's experience is the same.

It has often been pointed out that a disadvantage in the Celsian operation is, that one at least of the two ligatures must be nearer a collateral branch, than would be a single ligature if placed on the vessel at the site of the proposed section: the danger of ligation when the coats are ruptured near a branch is well recognised.

It has been maintained that the chance of hemorrhage is lessened by division because the longitudinal tension of the artery is removed: but this is only a matter of inference, and is not supported by either experiments or statistics: the statement too requires important qualification; we will however reserve this question for the chapter on the Choice of the Force.

The ends of the subclavian artery after the Celsian operation do not retract to anything like the same extent as do the ends of the smaller arteries such as the brachial and superficial femoral. These latter have long and straight courses, and the separation of their ends after division is considerable, but with them the method of ligature, if only asepsis be maintained, is not so important.

It is sometimes stated that by this method of operating the artery is placed in the same condition as after amputation, and that therefore the risk of hemorrhage

will be reduced to that in amputations, but the fallacy of this reasoning was long ago pointed out by Sir Charles Bell. "Has the profession," he asks, "still to learn the "difference of the condition of an artery where the limb "is cut off, and with it is removed the stimulus to the "activity of the vessels, and of an artery tied in the centre "of a limb, where the member still influences the con- "dition and activity of the trunk and its branches?"

Scarpa says, "The comparison which some authors "draw between the effects of division of a large artery "after amputation of a limb, and the shortening of the "same artery after the division between two ligatures for "the cure of aneurism is not accurate. For after the "amputation of a limb the divided artery retires in con- "sequence of its elasticity and tonic power, and at the "same time by the simultaneous shortening of the divided "muscles, as well as of the cellular substance to which "the artery is conjoined. On the contrary, in the opera- "tion for the cure of aneurism the divided artery retires "only in consequence of its own elastic and tonic power: "the muscles and interposed cellular substance remaining "in their natural position," and again, "the advantage "which some surgeons expect from the retraction of the "divided artery between the two ligatures will be equally "obtained from placing the operated limb merely in a "proper relaxed posture. Heister, Callisen and Richter "were well acquainted with this fact, and therefore did not "hesitate to assert *that the division of the artery between "the two ligatures is at the most an indifferent operation.*"

Another advantage claimed for the Celsian operation is that the circulation cannot be re-established along the

vessel. This is no doubt true, but it is a grave disadvantage, for if the blood passes the seat of ligature it must flow, not within the vessel, but into the cellular tissue, and externally—hemorrhage, in fact: and this unfortunately is what has often followed this operation.

Even in the case of the superficial femoral, which is exceptionally well suited for this method, or indeed, as we have seen done, for excision of a length of the artery, it has been found in practice by no means free from risk. Abernethy himself lost a patient on the third day from hemorrhage after performing this operation on this artery. Astley Cooper, too, relates how he ligated the superficial femoral in this manner, "tying with such a degree "of force as could be used without the risk of cutting the "coats." While the dressings were being put on both ligatures slipped off, Cooper reapplied them, and the case did well. The same accident happened to Cline; he ligated the superficial femoral in this way, "using "extraordinary pains in securing the ligatures;" three hours after the operation severe hemorrhage occurred, a dresser compressed the artery while Cline was sent for: when he came he found that the upper ligature had slipped off: the artery was re-ligatured and the patient recovered. Similarly Monteggia, Brenja and Morigi all had hemorrhage after ligature of the superficial femoral in this manner.

The same with other arteries: Monteggia had hemorrhage from the brachial; and two cases of fatal hemorrhage from the common carotid and one from the third part of the subclavian have occurred in recent years at a London hospital.

Several surgeons in the early part of the present century say they knew of many cases in which hemorrhage has followed this operation. Jones writes: "There is yet "another important consideration which it is necessary to "notice here. I allude to the slipping off of a ligature. "It is well known that this accident has often happened; "and it may be adduced in confirmation of the observa- "tions which I have offered, on the improper and careless "application of the ligature; for however surgeons may "endeavour to excuse themselves, by referring it to the "violent impulse of the blood, I believe that a candid "inquirer into the cause of it, will find a much more "rational and satisfactory explanation, either from the "clumsiness of the ligature which prevented its lying com- "pactly and securely round the artery, or from its not "having been applied tight enough, lest it should cut "through the coats of the artery too soon; or, finally, from "its having that very insecure hold of the artery, which "the deviation from the circular application above "alluded to, must necessarily occasion. It is obvious that "these causes may be variously combined in the same case, "and if one be adequate to occasion the slipping off of the "ligature, how much more likely is that event to happen "when they are so combined." And Scarpa says, "I could "enumerate many instances of the unfortunate result of "the Celsian operation in the hands of the most celebrated "surgeons in which there was neither a diseased state of "the artery or a sickly constitution of the patient."

If too, as some have asserted, hemorrhage never or very rarely happened in consequence of this mode of ligature, is it probable that the younger Cline would have

suggested—as had Richter long before—a third ligature, made by passing one of the ends of the thread through the end of the divided artery, thus making a knot which would prevent the slipping off of the ligature; or that the "ligature of reserve" would have been devised?

It may be said that the cause of the ligature slipping in some of these cases was that it was not drawn tight enough to rupture the coats: it is certainly necessary in order to be secure against the upper ligature slipping either to pass the ligature beyond a small collateral branch or to rupture the coats: but even rupture may not be effectual with smooth tendon ligatures, which indeed are unsuitable for use in ligating a terminal portion of any tissue, such as the omentum for instance.

"There is no motive," writes Holmes, "for this return "to an ancient practice: the cause of hemorrhage is not "the tension of the artery but ulceration about the liga- "ture; the greater exposure of the vessel increases the "chance of injury to the vein and so predisposes to "phlebitis and gangrene."

For these reasons then the operation of division must be rejected, and the vessel ligated in continuity: it only remains now to consider whether the coats should be ruptured or not.

Jones, whose brilliant work has had a great influence on English surgery, as the outcome of his experiments advises that the coats be ruptured, the object being to cause plastic exudation which should securely close the vessel by the time the ligature separates. "I think we "may fairly conclude," he says, "that if the ligature does "not completely cut through the internal and middle coats

"all round the artery adhesion cannot take place between "its internal surfaces, and therefore secondary hemorrhage "will take place as soon as the ligature has ulcerated "through any part of the parietes of the artery, and that "it will of course become more frequent and copious as "the process of ulceration advances." Jones we think erred in two ways, firstly by not keeping his animals long enough after operation, and secondly by not making experiments without rupture of the coats, but possibly he would have done this had his life been spared. Indeed he says, "the experiments on the several subjects require to "be extended to ascertain certain events connected with "them which are not yet fully explained. This it was the "author's intention to have done, but before he could ac- "complish it he was called away to the West Indies...He "particularly regrets that he was obliged to hurry over "that part of his work which demanded the most laborious "investigation and to limit it merely to the abuse of the "ligature." The title of his work is "A treatise on the "process employed by Nature in suppressing the hemor- "rhage from divided and punctured arteries," but as far as ligation in continuity is concerned, the question is what is the process employed by Nature in arresting the current of blood in an uninjured artery.

The conclusions drawn by Jones do not coincide with those which some of his contemporaries equally experienced in the surgery of the arteries arrived at. Dalrymple of Norwich repeated one of Jones's experiments—that of rupturing the coats with a ligature and immediately removing it: he tried it ten times, seven times on horses, and three times on sheep: no clot formed and the artery

remained pervious—"but in every instance its calibre was "contracted; still however it remained pervious in some "degree and capable of transmitting a lessened column "of blood."

In pre-antiseptic times when one end of the ligature was left hanging out of the wound and it necessarily worked its way by suppuration through the artery wall, the question of rupture of the tunics occupied a very different position to what it does now. It can hardly be reasonably disputed that the temporary ligature of Scarpa was incomparably safer than the ulcerative arteritis which followed the application of the round cutting ligature of Jones. There is no case on record in which hemorrhage followed the occlusion of a great artery by Scarpa's method, when it was strictly carried out.

Whether or not it is advisable to rupture the coats, it is certain that it is by no means necessary for the occlusion of the vessel. Since Jones's time a very large number of experiments have been made by various observers and the following facts now seem clearly established with respect at least to arteries of moderate size:—

1. That if a ligature be applied to a vessel and then immediately removed, the vessel will not as a rule be occluded, even if the coats are ruptured.

2. That if a ligature, such as silk, be applied to an artery so as to rupture the coats and left on the vessel for 70 hours or more and then removed, the vessel will be occluded. (Bruns.)

3. That if a broad ligature be applied to a vessel without rupturing the coats, and left in situ for four days and then removed, the vessel will be occluded. (Scarpa.)

4. That if a vessel be occluded without rupture by a ligature which is not removed and is not quickly absorbed the vessel will be permanently obliterated. Our experiments clearly demonstrate this.

5. That if a ligature be applied to an artery so that the lumen is greatly reduced but not absolutely closed, the vessel will nearly always be occluded: many of our own experiments shew this; the same observation had been previously made by Howard of New York; he even goes so far as to recommend that in ligating an artery in man the lumen should not be completely closed.

Howard made a considerable number of experiments on sheep with silver wire ligatures. (See Figures 165, 166, 167, 168.) These were applied so as not to completely close the vessel: in every instance the artery became impervious: but it must be remembered that the carotids of sheep are comparatively small and one cannot argue directly from them to larger vessels with higher blood pressures. It is possible, even when the coats are ruptured, if a too absorbable ligature is used, that the vessel may ultimately become again patent at the seat of ligature. The chances in favour of re-establishment of the circulation through the ligatured point have been thought to be increased by not rupturing the coats: but this will not occur with a proper ligature, properly applied: from some defect it may occur, but then probably not till ample time has been allowed for an aneurism to become consolidated: in other words the cure of the disease will have been effected as far as it depends on the passage of blood through the artery tied. Permanent obliteration of an artery at the seat of ligature is not necessary for the cure of aneurism, as is shewn by

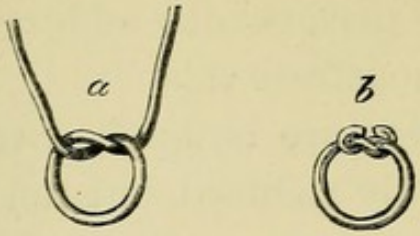


FIG. 165. Constricting silver wire Ligature.

(*a*) the ligature before the ends are cut off.

(*b*) the ligature after the ends have been cut off, and remaining parts turned down to remain permanently.

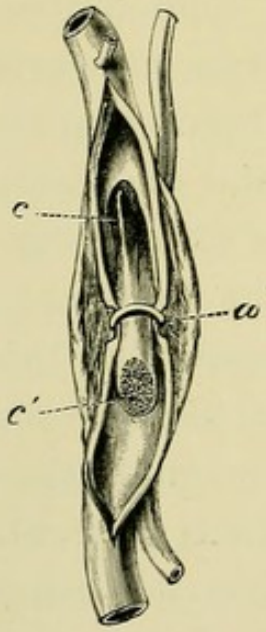


FIG. 166. Carotid of Sheep constricted with silver wire 10 days previously.

The wire is seen at *a*. The compression of the vessel wall by the ligature has been purposely exaggerated in the figure: the artery was constricted by about two-thirds of its lumen. *cc.* represents the clot.

FIG. 167. Carotid of Sheep constricted by silver wire $10\frac{1}{2}$ months previously.

Round the carotid artery of a sheep, a silver wire ligature was applied so as to diminish the calibre by two-thirds. The specimen was obtained $10\frac{1}{2}$ months later; it shews the artery reduced to a mere cord, and fibrous tissue (*c*, *c*) obstructing the lumen for a considerable distance both above and below the ligature (*a*).

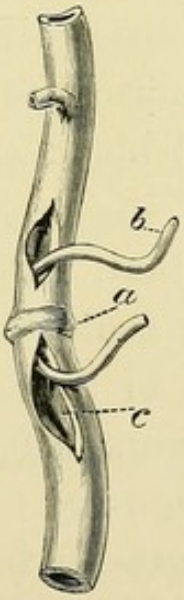


FIG. 168. Carotid of Sheep encircled with wire $13\frac{1}{2}$ months previously.

Round a carotid artery of a sheep, a silver wire (*a*) was placed without avoidable constriction of the lumen of the vessel. The artery was found pervious as shewn by the passage of the stout wire (*b*). (*c*) points to interior of vessel.

Figs. 165 *to* 168 *are reproduced from blocks kindly lent by Dr Howard of New York; see also his work "On the Treatment of Aneurism"* (1870).

the success of digital compression and other methods of treatment, which do not produce occlusion of the artery except at the seat of aneurism? Moreover, it must be remembered that in the Hunterian operation the vessel between the aneurism and the ligature generally becomes more or less patent; for example, Savory found with aneurisms of the popliteal artery that out of the seventeen specimens he examined, the artery was pervious in this situation in fourteen.

Clearly then the rupture of the coats is not necessary for the occlusion of the vessel, though it may be thought that it makes occlusion doubly sure. It, however, is not advisable, for it greatly weakens the vessel, occasioning serious danger from hemorrhage.

When a vessel is tied the blood pressure on the cardiac side rises to that of the artery of which the vessel is a branch: if the coats are ruptured, the outer coat alone has to withstand this abnormal force: even after a length of clot has formed fluid blood still finds its way to the ligated point.

It has been argued that, although there may be no necessity to rupture the coats when the wound heals by first intention, yet that in those cases in which suppuration unfortunately occurs the danger of hemorrhage will be greater if the coats are not ruptured. The effect of not rupturing the coats is to leave a larger amount of tissue for the ligature to work through, thus at least postponing the division of the vessel and allowing more time for consolidation to take place, and is thus a gain and not a loss. Moreover it is not certain that a silk ligature would ulcerate through the vessel: its loop, like

that of tendon, might be dissolved in situ: in the case of a ligature becoming rapidly dissolved the vessel may become patent and the operation fail, but this with modern ligatures is not probable, and even if it occurred would be a much less serious disaster than hemorrhage, which cannot happen so long as the vessel remains whole.

But the argument in favor of rupturing the coats because suppuration may occur, rests on no firm basis, for we have several times in our experiments observed suppuration to occur for a few days and occasionally profusely and for a long time, and yet the tendon ligature has held the coats in contact, for a time sufficient before its destruction by the pus to cause permanent occlusion of the artery, without any solution of continuity of its wall. Moreover, we have had the opportunity of watching a similar process in man: a tendon was put round the superficial femoral artery without injuring its tunics; the skin and fascia of Scarpa's triangle sloughed in consequence, probably, of the previous attempt to cure the aneurism by digital compression, and the ligatured artery was exposed in the bottom of a septic wound: nevertheless the tendon held, the artery was occluded and the aneurism cured.

Lastly, it must be repeated that it is hardly possible for any operation to yield worse results than the rupturing method has given in the case of the larger arteries.

The coats then should not be ruptured, and in support of this conclusion may be cited the authority of Erichsen. Having related in his work on the Science and Art of Surgery, how out of 14 cases of ligature of the first part of

the subclavian, 12 died of hemorrhage, he expresses the opinion that "this operation should be abolished from "Surgery, unless further experience shews that absorbable "ligatures can be applied in such a way as to occlude "the artery without risk of ulceration or division of its "coats," and in the last edition of this work it is added that "it may reasonably be hoped that the average of "success [in ligating the third part of the subclavian] "will be materially increased by the use of ligatures which "do not divide the coats."

CHAPTER XVI.

CHOICE OF THE LIGATURE.

Views of Galen. Catgut. Wires. Hemp. Silk. Hair. Skin. Tendons. Nerves. Arteries. Peritoneum. Requirements of a perfect Ligature. These tests applied to the foregoing. Decision.

The choice of the material used for the ligature has always received, as indeed it deserves, the careful attention of surgeons. Galen, speaking about hemorrhage, says, "But if on laying bare the vessel it should appear "to you large, and to pulsate strongly, it is safer for "the operator to put a double loop round it and to divide "between, and let these ligatures be of a material which "will not readily decompose. Such a material in Rome "can be got from the Gaietans, who bring it from the "country of the Kelts and sell it in the Via Sacra, which "leads from the Temple of Roma to the markets. This is "the easiest thing to get in Rome, and it is sold very "cheaply there; but if you are practising your art in "another city prepare for yourself some of the threads

"known as silk: rich women have these in many parts "of the Roman Empire and especially in large cities. "If you cannot get this, choose the material least liable "to decompose from among those that you can get where "you are, such as fine catgut; for materials which easily "decompose fall quickly out of the vessels, but we wish "the knot only to fall out when the vessels have been well "covered round with flesh, for the flesh, which grows up in "the parts of the vessels which have been cut off, acts as a "covering and stops their mouths, and when this has "happened is the time for the ligatures to separate "without danger."

Thus, although in the absence of hemp and silk Galen used catgut (graciliū chordarū), yet it was not on account of its absorbability; indeed, that is the very quality he did not want.

A very large number of substances have been used at various times. The first to recommend absorbable ligatures, as such, was Physick who in 1814 used untanned buckskin: he cut the ends short, but in this he had been anticipated by two surgeons of the Royal Navy, the practice having originated with Lancelot Haire and a colleague at Haslar in 1780. Haire writes, "An intimate "friend of mine, a surgeon of great abilities, proposed "to cut the ends of the ligatures close, and thus leave "them to themselves. By following this plan we have "seen stumps healed in the course of 10 days. The "short ligature thus left in, commonly made its way out "by a small opening, in a short time, without any trouble, "or the patient being sensible of pain."

In 1817 Astley Cooper used catgut for the same

purpose. His first case was a perfect success. The patient was a man aged 80 years with popliteal aneurism. The femoral artery was ligatured: the ends of the catgut ligature were cut short: and the man was up and about in three weeks. Astley Cooper remarks, "I confess that this "case gave me great pleasure; the great age of the "patient, the simplicity of the operation, the absence of "constitutional irritation and consequently of danger, and "his rapid recovery, lead me to hope that the operation "for aneurism may become, at some future period, in-"finitely more simple than it has been rendered to the "present moment." In his second case, however, hemorrhage occurred and he gave up the use of catgut.

In 1818 MacSweeny recommended silkworm gut: besides this, wires of gold, platinum, silver, iron, and lead, have been used, also hemp and silk, and hair, the untanned skin of various animals, tendons of the horse, whale, reindeer and kangaroo; nerves and strips of arteries.

To decide between these it is necessary to consider the requirements of a perfect ligature: it must be sufficiently strong not to break: inelastic: round: smooth: pliable and easily tied into a knot: not too bulky: absorbable and yet not too readily so; and lastly capable of being rendered certainly aseptic.

With respect to strength most ligatures are satisfactory; but catgut occasionally breaks in the surgeon's hands. In the case of ox aorta, too, this accident happened to Mr Bennett May, in ligaturing the innominate artery. Although no great strength in the ligature is necessary to occlude the vessel, it is desirable to pull

pretty tight in finishing the knot, doing which, it must be remembered, does not tighten, at least in the case of a reef knot the loop in which the artery is held. The ligature breaking off short at the knot is a most serious accident, for it makes it possible for the knot to come undone, and therefore necessitates the application of another ligature.

No. 2 silk (which is the finest any one would think of using for a large artery) breaks at a strain of 11 lbs. (5 kilogrammes), while No. 4 breaks at 25 lbs. (12 kilos.), No. 4 catgut (which is pretty stout) breaks with 12 lbs. (5·5 kilos.). Kangaroo tendon (1/20th of an inch or 1·2 mm. in diameter) at 18 lbs. (8 kilos.): peritoneal ligature of about the same size at 17 lbs. (7·7 kilos.).

Hemp and silkworm gut are also strong; but the same cannot be said of ox aorta ligature, for a specimen, which Mr Barwell kindly supplied, ruptured at 5 lbs. strain (2·3 kilos.).

The next requirement is inelasticity; if the ligature stretches, the surgeon cannot gauge the force he is applying to the artery. Ox aorta fails in this respect, for a piece which we tried stretched to nearly double its length with a pull of only two pounds (1 kilog.); a force which is not sufficient to occlude a large artery. This ligature has the apparent advantage of being flat, but at the knot it does not lie flat but gathers up into a lump. The same objection applies to all flat ligatures, and to oval ones also, as we found with some large oval catgut kindly prepared for us by Messrs Macfarlan of Edinburgh: the loop is well enough, but at the knot, if soft, they tie up into a lump, or if hard, turn on edge, cut into the artery wall, and are

then more dangerous to the integrity of the coats than a round ligature. The ligature, therefore, must be round or nearly so: it should also be smooth, for the greater the friction at the points of contact of the strands in the first hitch of the reef knot, the less can the surgeon judge of the effect which tension on the ends of the ligature is producing on the artery, and the less likely is it that his fingers will convey to him, by the resistance offered by the meeting of the coats, the exact moment at which the artery becomes occluded.

The next quality is pliability. All kinds of wire are to be condemned on this score; silkworm gut also is not easy to tie a knot with; but by soakage in a watery medium (carbolic lotion) for some weeks it becomes much less rigid: more pliant and supple. We endeavoured to obtain some modification in the process of manufacture, so that it might be made of larger size, more pliant, and generally more easily manipulated as a ligature, but we failed. The largest salmon gut is nothing like the size of the kangaroo tendon (1/20th to 1/16th of an inch, 1·2 to 1·5 mm. in diameter) recommended for use.

Further, the ligature should be absorbable, and yet not too rapidly so; the exact time the process takes is not very material so long as the ligature remains a sufficient time to do its work. Chromic catgut lasts long enough if no suppuration occurs, but if it does, owing to the cells working into the interstices of the catgut, it becomes rapidly absorbed and is not to be relied on: this objection does not apply to kangaroo tendon which is absorbed only from the surface.

It was at one time thought that all animal substances

in aseptic wounds, with the single exception of silk, were absorbable, and remained quiescent till they disappeared, and that silk and vegetable substances were not absorbed, and in consequence, worked their way out.

It is now known however, that all ligatures, except gold and platinum wire, are absorbed, and if sterile lie quiet and do not cause suppuration.

One of the reasons why silk and hemp work out more often than tendon ligatures, is that from their structure they require more care to render them aseptic. They will, however, stand boiling in perchloride of mercury solution, in which particular they are superior to catgut and tendon, and can thus be rendered aseptic, and then will not excite suppuration, becoming at first encapsuled and finally absorbed, for encapsulation is only a stage previous to absorption. Kölliker of Leipzig, in 80 amputations with antiseptic precautions, found that in 40 cases the silk ligature came away. Both Lister and Clutton report in cases of ligature of the external iliac with silk, abscess, and discharge of the ligature after many weeks, and after the primary wound had healed without suppuration. The suppuration which occasionally occurs in the holes made for silk sutures is well known, and is in striking contrast to the behaviour, under the same circumstances, of horsehair or silver wire.

That the ligature may remain quiescent and not cause suppuration, it is necessary that it be aseptic: the best method of preserving tendon ligatures and catgut is in glycerine containing 0·1 per cent. of perchloride of mercury: this is a far stronger antiseptic than carbolic oil, which was shewn years ago by Koch to have very

feeble antiseptic powers, and moreover the ligatures come out of the glycerine pliable and perfectly ready for use, whereas if they be kept in oil they are stiff and unmanageable and require to be soaked for some little time in a watery medium before they become sufficiently soft.

Catgut can be kept for any length of time in glycerine perchloride, but if kangaroo tendon be allowed to remain in it for many months it swells up and becomes almost too soft, hence the tendon should not be kept in the glycerine for more than two or three months.

It is necessary to be very careful in the matter of asepsis, for Lister long ago demonstrated bacteria in catgut, and Kocker is so convinced that catgut infected his wounds that he has given it up entirely: Volckmann had a patient inoculated with anthrax from the catgut used.

In both silk and catgut there are fissures and cracks in which bacteria can lodge. Silk can be boiled but catgut is destroyed by that treatment: Larochette recommends the sterilization of catgut by dry heat: his plan is as follows, the temperature is gradually raised to 284° F. (140° C.): plenty of space must be left for the evaporation of the water contained in the gut. When the temperature has been maintained at this height for two hours the sterilization is complete: the catgut should then be placed by flamed forceps in a 10 per cent. solution of carbolic oil.

In recent years sulpho-chromic (green) catgut has been extensively used: it is kept dry and soaked in carbolic at the time of operation: but even with this precaution it is not satisfactory, for in several cases which have come under our observation in which it has been

used for the ligature of veins, it has been discharged by suppuration: in one case the knots were all discharged, the last coming away a year after the operation. Fine Chinese silk boiled in perchloride of mercury has given, on the other hand, very satisfactory results: green catgut, however, preserved in glycerine perchloride (1 in 1000) is perfectly pliant and no doubt also aseptic.

The method of preparation originally recommended by Lister was to soak the catgut in an emulsion of carbolic acid in olive oil: the catgut had to remain in this for at least two months before use. It was thought that catgut thus prepared was too quickly absorbed in the tissues and the method which Lister now advises is to make a solution of 1 part of chromic acid, 200 parts of absolute phenol and 4000 parts of water: the catgut added is to equal in weight the phenol: it remains in this for 48 hours, and is then kept in carbolic oil.

In Germany catgut is prepared by immersion for from 6 to 12 hours in a 1 per cent. solution of corrosive sublimate: it is then preserved in absolute alcohol.

Without disparaging the use of catgut, it must be pointed out that much care must be given to its preparation. The surgeon cannot control its manufacture, but he is responsible for its asepticity: for our own part, we prefer for daily use fine silk which has been boiled in mercuric perchloride solution (1 in 500).

The reason why wire ligatures so seldom cause suppuration, is that their hard smooth surface can so readily be rendered aseptic. Some experiments by Knapp of New York in this connection are very interesting. He purposely made septic a cataract knife, and found on using

it on the eye of one of the lower animals, that, as was to be expected, it caused suppuration and opacity of the cornea: he then tried placing the septic blade in antiseptic solutions and it still caused suppuration, finally he tried wiping the knife firmly with dry cotton wool but not applying any antiseptic, no evil then followed the use of the knife.

To conclude: as the outer coat and sheath of an artery consist chiefly of white connective tissue, the most appropriate of the foregoing ligatures will be of this material, that is to say, will be either tendon or peritoneal ligature: and these satisfy all our tests: they are strong, inelastic, round, smooth, pliable, the proper size, slowly absorbed and easily rendered aseptic: of the two, peritoneum is slightly the better as it lasts longer: floss silk is a smooth and trustworthy ligature: failing these, ordinary silk and chromic catgut are good, but their rough surfaces and the presence of mucous and muscular tissue in the latter are defects; much care too is needed to render them aseptic: wire must be rejected, as being too rigid and as difficult to handle: with respect to silkworm gut, it is not easy to manipulate round an artery unless it has been soaked for a long time in water, and moreover, several strands must be employed to make a ligature which will not cut the coats, and this adds to the difficulty. When, however, these obstacles are overcome, it makes a very beautiful ligature. (See Plate I. fig. 5, and Plate III. fig. 1.) Ox aorta too must be rejected, on account of its weakness, bulk, and elasticity.

The choice then must fall on ox peritoneum, kangaroo tendon or boiled floss silk; and failing these on boiled Chinese twist, chromic catgut, or silkworm gut.

CHAPTER XVII.

CHOICE OF THE KNOT.

"Granny." Reef knot. Non-occlusion of vessel. Surgical knot. Objection to it. Slip knot. Carrick bend. Clove hitch. Senn's method. Authors' knot: its action: why trustworthy. Ligation of peduncles. Staffordshire knot.

We have seen how recurrence of pulsation after ligation may be due to the collateral circulation re-entering the original channels above the aneurism instead of below: it may also possibly be due to the too rapid absorption of an animal ligature; a third cause suggests itself, namely, that by some defect in the method of tying the ligature may fail to close the lumen of the vessel.

The most natural knot to tie and that which was probably used by the earlier surgeons is a "granny"; this is not a perfect knot and may yield, but there is no evidence that it has ever done so when used in the

ligation of an artery; it has, we believe, never been found either untied or even loose: still it is inferior to a reef knot and must therefore be rejected.

A reef knot once completed cannot yield: to test the security of knots we tied stout india-rubber tubing with catgut, tendon and silk, using various knots, and then placed the tubes in putrid and purulent fluids, keeping them in an incubator at 100° F. (38° C.) for some weeks: in no case did the knot become loosened or undone: it is clear then that a knot once completed does not slip in the living body, even in the most unfavourable conditions: similar experiments were made by Callender and with the same result.

The difference between a granny and a reef knot is seen in Figs. 169 and 171; it will be noticed that the loop in the reef knot goes round and outside the two parts, whereas in the granny it passes between them and thus holds them less securely: a reef knot is always preferred to a granny by sailors for another reason—it never jams: when it has to be untied it can be loosened by a jerk and can then be readily undone: this is no advantage whatever for a surgeon but, on the contrary, a source of danger: it will be seen from fig. 169 how a reef knot can, by pulling on one free end, the loop being held firm, be converted into a slip knot which can come undone. Some time ago in a case of strangulated hernia in which a portion of omentum had been ligatured and cut off, a fatal hemorrhage from the stump occurred which was supposed to have been due to the knot of one of the ligatures having been deranged by the surgeon pulling on one of the ends, which had not yet been cut off.

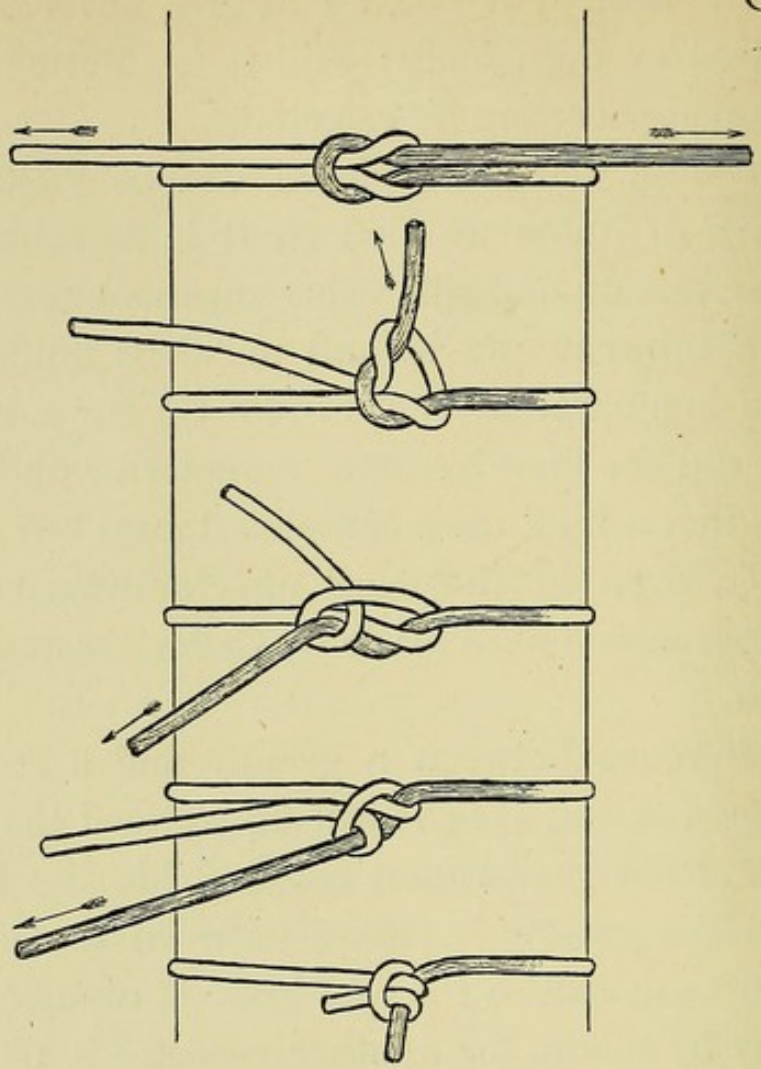


FIG. 169. Conversion of reef into slip knot.

Fig. 169 shews the various stages of the conversion of a reef knot into a slip knot by pulling or jerking on the dark end as indicated by the arrow: the lowest drawing shews how unsafe the knot then becomes when the ends are cut close.

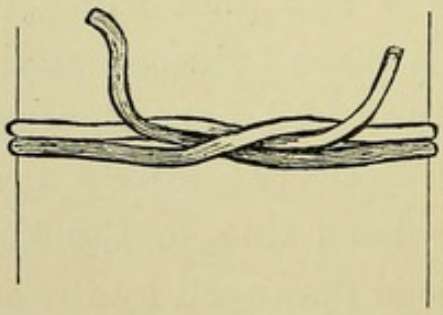


FIG. 170. Clove hitch.

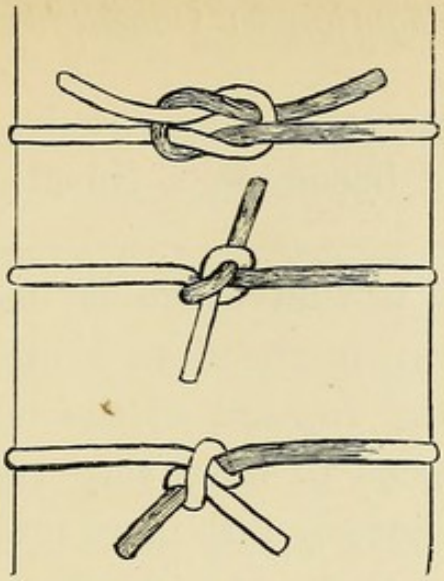


FIG. 171. Conversion of "granny" into slip knot.

Fig. 171 shews the stages of conversion of an ordinary "granny" knot into a slip knot, one end being a clove hitch on the other: this is effected in the same manner as with a reef knot.

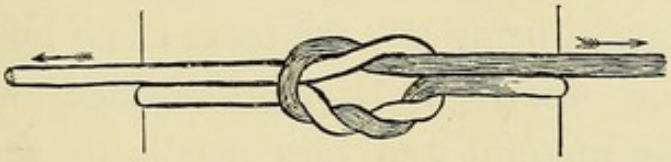


FIG. 172. Surgical knot.

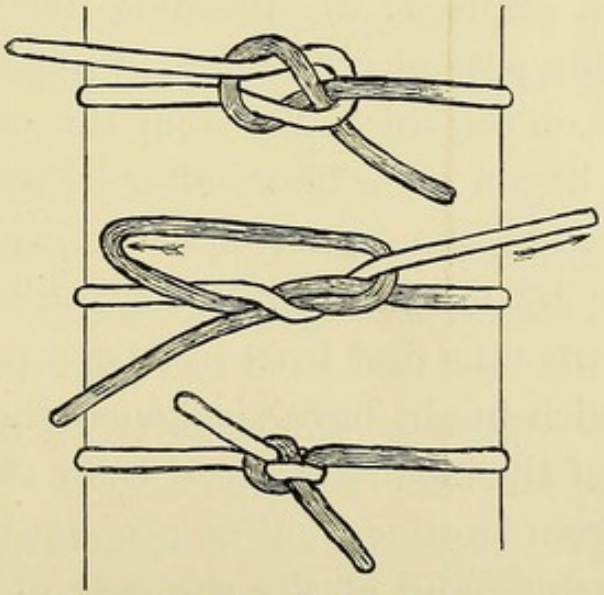


FIG. 173. Carrick bend.

The upper drawing shews the construction of the knot: contrast it with the reef and "granny": to apply the knot to an artery it is necessary to arrange the ligature as shewn in the middle drawing and to constrict the vessel by pulling in the direction indicated by the arrows: the dark loop is then let go, the finger placed on the knot, and the knot drawn tight by pulling on the two ends: the lowest figure shews the knot tightened.

In the same way a granny can be converted into a slip knot: one end becoming a clove hitch on the other (see Fig. 171).

The reef knot is that which is usually used in the ligature of arteries: in the days before absorbable ligatures and when the rupture of the coats was generally practised there seems to have very rarely been trouble with the knot, but latterly with more bulky ligatures which are less pliant, smoother and more slippery, cases in which the vessel has not been occluded have from time to time occurred.

Brenja in 1825 ligated a superficial femoral by the Celsian operation, using silk ligatures: two hours after the operation the dressings became stained with arterial blood but the amount was very small: on the third day alarming hemorrhage occurred: the artery was compressed and again secured: the bleeding had been from the upper end: the patient recovered. Brenja remarks "The "hemorrhage on the third day from the operation (which "indeed had begun a few hours after it) was produced by "the looseness of the upper ligature, which I can only "account for by my having slightly pulled up the two "ends of it after the first twist had been tied to make the "second,—which might have slackened the first. In fact "the oozing of the blood appeared soon after the patient "recovered from fainting; but on account of the tightness "of the dressings, and of the slowness of the oozing the "blood coagulated and opposed a temporary obstacle to "further hemorrhage. When the dressings and consequently the pressure were removed the ligature was cast "off altogether and hemorrhage ensued."

Thomas Smith, in a case of inguinal aneurism, ligatured the common iliac artery with kangaroo tendon; the next day pulsation had recurred: the artery was again exposed and the ligature found somewhat loose; the vessel was again tied with the result of stopping all pulsation.

There is a case recorded in St Thomas's Hospital Reports in which the superficial femoral was ligatured with silk it being intended not to rupture the coats: the next day pulsation was found in the aneurism and a thrill could be felt and heard at the seat of ligature; on the 18th day the artery was again exposed and tied above and below the original ligature, the intervening portion of vessel being excised. On examination of this it was found that though constricted the vessel was pervious and that the coats had not been ruptured.

In the case illustrated in Fig. 101, page 243, the same accident occurred; the superficial femoral artery was found to be pervious on the day after the operation and the vessel had to be religatured.

In the case too from which Fig. 86, p. 215, is taken the carotid artery was found to be pervious, and yet (as seen in the drawing) the coats had been ruptured at the operation.

Thomas Smith attributed the looseness of the ligature in his case to the first part of the reef knot slipping before the second part was completed. Thus although the knot was secure the loop was loose: this too was no doubt what occurred in the other cases. Even if the ligature while loose be placed obliquely round a vessel it will assume a transverse position during tightening; this then cannot be the cause of failure.

In the earlier of our own experiments the same thing happened from time to time. In the course of our work we ligatured several carotids of sheep and asses and were surprised subsequently to find that in many the walls were not in apposition. Some were occluded at the point of ligature by a diaphragm of connective tissue, but four were at the time of examination pervious, though constricted. For some time we were in doubt as to the cause of this, which might have been the slipping of the knot, the too rapid absorption of the ligature, or not rupturing the coats. One day it happened that a horse had to be killed at the Brown Institution and the following experiment was carried out which made the matter clear, and explains many of the cases of recurrence of circulation through the artery at the seat of ligature. The horse being chloroformed, Mr Horsley was good enough to dissect out about one foot of the common carotid; as the vessel lay upon his hand the question of obstruction without rupture was tested by various ligatures with different knots. It was found that it was almost impossible to tie in the living subject the second half of a reef or surgical knot quickly enough to prevent the partial giving way of the first hitch before the second was tight. The materials employed were tendon, catgut and silk: gentle tension on the first half of the knot for forty seconds or longer made a difference, for then with the utmost rapidity the second half of the reef or surgical knot could occasionally be satisfactorily applied. Not only is this true when the coats are undamaged but with a large ligature, say number 6 catgut (Macfarlan), the same is true when the coats are ruptured. (Compare figure 174 with fig. 86, page 215.)

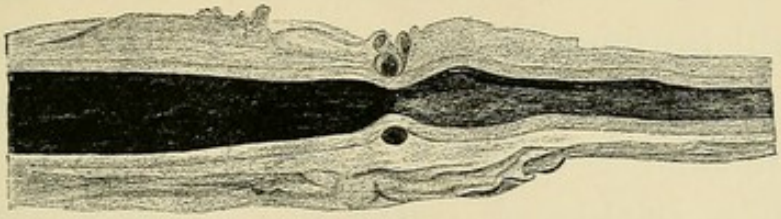


FIG. 174. 'Giving way of first hitch.' Carotid artery of horse 10 days after ligature with kangaroo tendon (nat. size).

The coats are not in contact but the artery is filled with clot. The microscope shews cells from the endothelium at the seat of ligature invading the coagulum: a reef knot was used and the open condition of the vessel is due not to the ligature having been drawn insufficiently tight but to the first half of the reef knot having given while the knot was being completed: the figure shews the extent (2mm.) to which this has occurred.

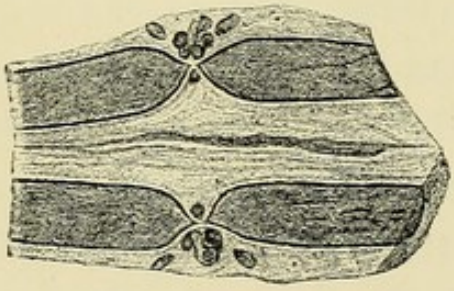


FIG. 175. 'Rare instance of success of first hitch.' The carotid artery of a sheep 13 days after ligature with chromic catgut tied with a surgical knot (nat. size).

(*St Thomas's Hospital Museum, No.* 414).

There is approximation of the coats without injury and with complete occlusion of the vessel. Clot has formed for a considerable distance on each side of the constriction. The ligature is embedded in young connective tissue.

Let any large artery be obtained and distended with a pressure of ten feet of water and the first half of a reef be tied it will be found that so long as the tension on the ends is maintained no water will escape, but as soon as this tension is relaxed, as it must be to complete the knot, a trickle of water will occur which will not be stopped by the completion of the knot. The knot itself will be secure but the loop will be somewhat loose: how loose it will be will vary with the nature and size of the ligature and the rapidity with which the second hitch is put on, but the difficulties are clearly much greater in the living subject than in an experiment like this.

This then is the usual cause of return of pulsation after ligation, and although it does occur as we have seen with silk, and also when the coats are ruptured, it is much more likely to happen when a catgut or tendon ligature is used, and when it is intended not to rupture the coats of the artery.

This danger has long been known and what is called the surgical knot has been devised to obviate it (fig. 172): it is not, however, effectual as experiments with water pressure show,—the two turns slip in the same way as one does: putting the finger on the first half of the knot while the second is being tied is of no use in the living subject because there is no firm structure against which to compress the artery, also the ligature is sunk in so deep a groove that the finger does not reach it. Clamping the first hitch with a pair of artery forceps is also a clumsy and ineffectual procedure. Another method which suggests itself is to pass a fine silk ligature under the first hitch before tightening and to tie it over the

main ligature while tension is being maintained, and before the second hitch is tied: this we found to be successful in the case of tendon; the great objection to it is that it makes the surgeon dependent on his assistant.

Lidell writes "The surgeon's knot must not be "employed in tying arteries because it may fail to close "the lumen of the vessel. Boyer remarks that the "surgeon's knot has the inconvenience of being tightened "with difficulty while at the same time it fails to entirely "close the artery even though great force be employed in "tightening it; and that its disadvantage is proved by the "following case: Chopart operated on a gardener for "popliteal aneurism in presence of the most distinguished "professors of the Ancient School of Surgery, by the old "method. The ligatures being placed and the first being "tightened by the surgeon's knot, the compression was "withdrawn but notwithstanding the blood escaped abun- "dantly. A second and a third ligature were placed and "tightened in the same manner and with as small success. "The failure after a moment's deliberation was attributed "to the ossification of the artery or to some unknown "cause and it was decided to amputate. On examining "the limb however the artery was found in a natural "state; it was embraced by the three ligatures; but "although these ligatures had been tightened in the most "forcible manner neither of them had perfectly effaced "the lumen of the vessel, but so incompletely that a large "stylet could easily penetrate it. This fact is sufficient to "proscribe the surgeon's knot for ever in operations for "hemorrhage as well as in those for aneurism. I have my-

"self often observed that ligatures tied with the surgeon's "knot do not completely close arteries in the cadaver."

In Adams's case (p. 215) a surgeon's knot was used and the vessel was not occluded, although the coats were ruptured.

We tried a slip knot in one instance (Exp. 68), but it is not to be recommended partly because it gives in the same way as the first hitch of a reef knot, partly because it is not possible with it to judge of the force being used, and partly because the loop saws the vessel as it is being tightened.

Though sailors always use the reef knot for the purpose its name indicates and for many others, they do not use it for the very important duty of fastening two hawsers together in towing: for this a Carrick bend is recommended; it is shewn in fig. 173 and only differs in one detail from a granny; the merit of the knot appears to consist in the free end being held firm between the two long portions. To use this knot on an artery it is necessary to pass the end into its position before tightening the ligature, by pulling on the end and the loop in opposite directions: the ligature being drawn sufficiently tight the loop is let go and the end pulled instead and the knot thus completed. We tied an artery with this knot and the result was quite satisfactory (see Exp. No. 52), the knot is perfectly secure but cannot be recommended: partly because it is complicated, partly because it requires the pressure of the finger, and partly because its final tightening leads to a further tightening of the loop round the artery which might produce a rupture of the coats, which is the very thing to be avoided.

It may be remarked that the difficulty might be overcome in a quite simple way, namely, by compressing the artery above the point at which the ligature is being applied. As a matter of fact this could only be done in the case of the superficial femoral, and even then the natural resilience of the artery would remain to endanger the occlusion of the vessel.

It seems then that there is no satisfactory way of tying a ligature which goes only once round the vessel, and it is necessary to use either, as Thomas Smith recommends, a clove hitch or two or more separate ligatures.

We tried the clove hitch on animals: the artery was always obliterated, but the coats were not always in apposition: (see Plate II, fig. 3); the knot cannot be recommended because it will not run properly on a soft structure; as the ends are pulled on, instead of the ligature tightening, the artery flattens up, and when the ends are finally relaxed the artery opens out again to a certain extent and becomes patent. It is not possible moreover to judge of the force being applied to the artery and the coats may be unintentionally ruptured.

In the ordinary method of tying an artery the unthreaded needle is passed, threaded and drawn back; the loop being divided there remain two ligatures side by side: the use of two ligatures therefore entails no additional disturbance of parts. Hunter, as we have seen, (page 327) used four ligatures passed in this way, and recently Senn has recommended that two should be used, the proximal being tied first: we tried this method with a satisfactory result (see Plate II, fig. 4), but it will be

observed that the distal ligature has more closely constricted the vessel than the proximal, which in all probability did not quite occlude the lumen.

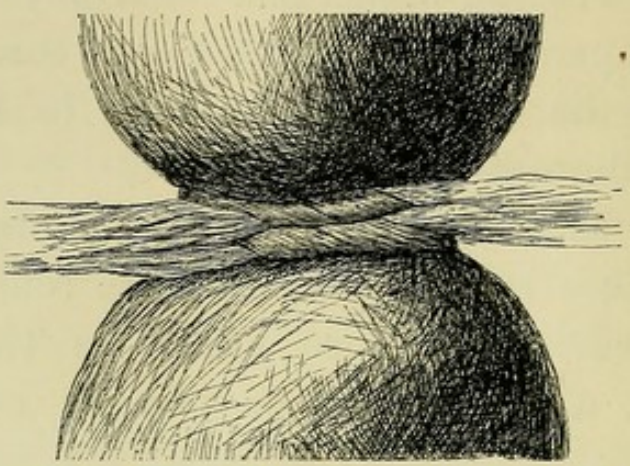


FIG. 176. Floss silk: Stay knot (first stage).

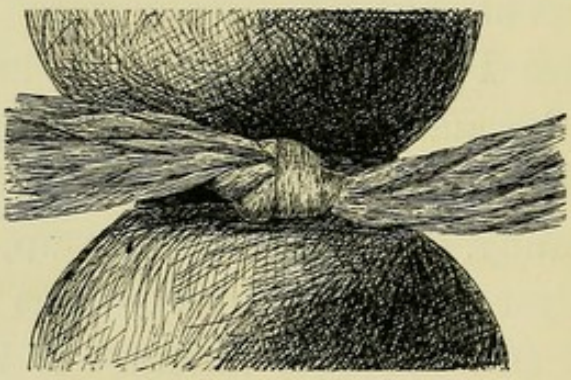


FIG. 177. Floss silk: Stay knot (completed).

Fig. 176 represents two floss silk ligatures side by side (×5). The first half of a reef is tied on each in the same way. The two ends on either side being treated as one, are drawn upon to occlude the vessel. The hitches lie at the bottom of a deep groove and are seen to fit into one another.

Fig. 177 shews the knot completed by using the two ends on each side as a single cord and by tying the second hitch as if completing an ordinary reef knot.

There is another advantage in using two ligatures—a greater length of the intima of opposite sides is brought into contact.

The best way of tying two ligatures is to make on each separately, and in the same way, the first hitch of a

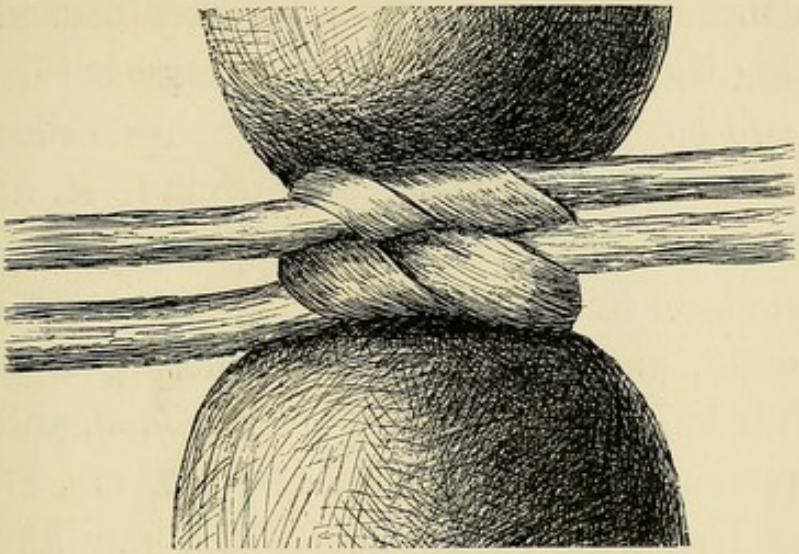


FIG. 178.

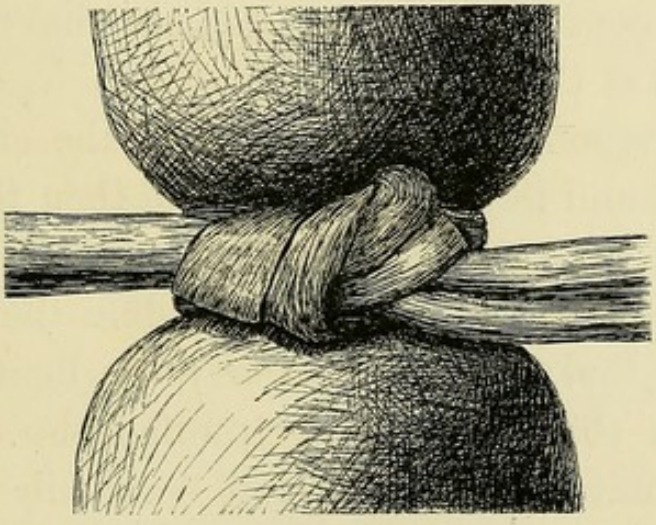


FIG. 179.

FIGS. 178, 179. Kangaroo tendon: Stay knot.

Fig. 178 represents two kangaroo tendon ligatures each $\frac{1}{20}$th inch (1·2 mm.) wide (×5). The first half of a reef is tied on each in the same way. The two ends on either side being treated as one are drawn upon to occlude the vessel. The hitches lie at the bottom of a deep groove and fit into one another.

Fig. 179 shews the knot completed by using the two ends on each side as a single cord and tying the second hitch as if completing an ordinary reef knot.

reef knot, and to tighten each separately so that the loop lies in contact with the vessel without constricting it; then, taking the two ends on one side together in one hand and the two ends on the other side in the other hand, to constrict the vessel sufficiently to occlude it, and finally to complete the reef knot. The simplest method of completing the knot is to treat the two ends in each hand as a single thread, and to tie as if completing a single reef knot. This knot we have called a *stay knot*, and it is this that we recommend. (See Figs. 176, 177, 178, 179.)

To accelerate the completion of the stay knot and to prevent the mistake of tying a 'granny' instead of a reef knot, it is well, immediately after passing the first hitches and before constricting the vessel, to make loosely the second hitch of the knot. (See Exp. 70.)

If the surgeon prefer, he can let go the ends of one of the ligatures and tie first one reef and then the other, but he must be careful to make no mistake about the ends, and it would be much better to complete the knot on the cardiac side first. Three, four or more ligatures can be employed in this manner; by pulling the ends of the ligatures simultaneously they will lie evenly side by side and constrict a greater length of the vessel than if they were tied separately and were lying as they then would, one on the top of the other in a deep groove in the artery.

It is necessary in using stay knots not to make any mistake as to which side the ends belong to. The best method of avoiding this is by clamping together all the ends on each side with a pair of forceps. (Exp. 62.)

With ordinary ligatures three hitches can be completed as one knot, four should as a rule be treated as

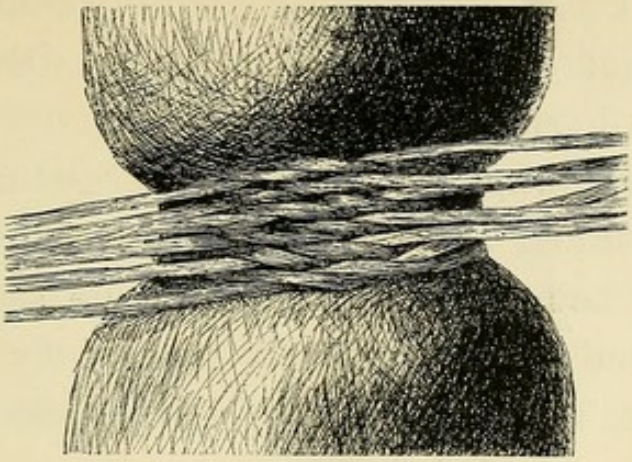


FIG. 180. Stay knot.

The figure represents six silkworm-gut ligatures (×5). The first half of a reef is tied on each in the same way. The six ends on either side being treated as one are drawn upon to occlude the vessel. The knot is completed by using the six ends on either side as a single cord and tying the second hitch as if completing an ordinary reef knot. We used this knot for ligating a horse's carotid and it answered well, but it is not recommended since the tying and arranging of the six half hitches is tedious: the six strands lie close together at the bottom of the groove and appear as a single stout round ligature. Exp. 71, see Plate III, fig. 1.

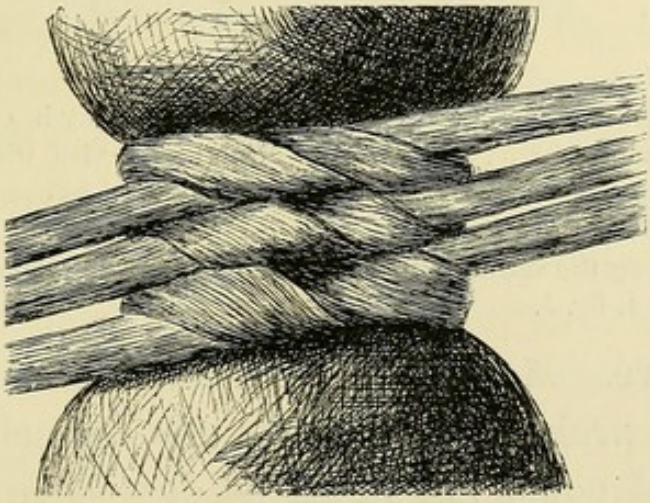


FIG. 181. Stay knot.

The figure represents three kangaroo tendon ligatures each $\frac{1}{20}$th inch (1·2 mm) wide (×5). The first half of a reef is tied on each in the same way: the three ends on either side being treated as one are drawn upon to occlude the vessel: the hitches lie at the bottom of a groove and fit into one another: the knot is completed by using the three ends on each side as a single cord and tying the second hitch as if completing an ordinary reef knot.

The above is recommended for very large vessels, such as the Innominate.

two pairs, but with fine ligatures, such as silkworm-gut, six can be included in the single final hitch of the knot.

(Compare Figures 180, 181 and 182.)

The reasons why treating the two ligatures simply as one will not meet the difficulty of the expansion of the first hitch is because they would not lie side by side on the artery, but would be likely to entirely overlap, and thus not more intima would be in apposition than with a

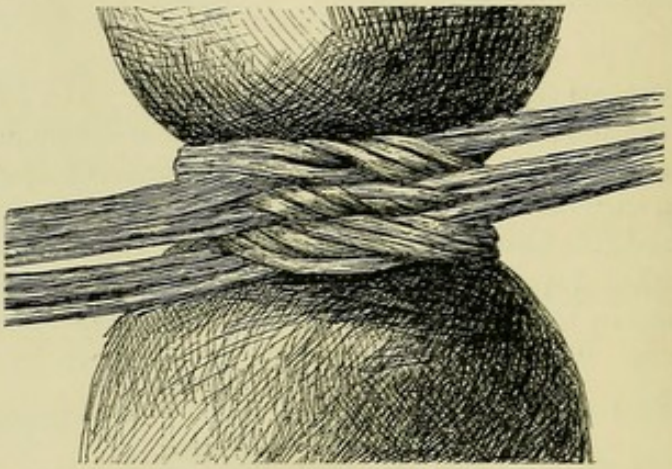


FIG. 182. Stay knot.

The figure represents a stay-knot of three ligatures each composed of three strands of silkworm-gut (× 5). The first half of a reef is tied with each ligature in the same way: the nine ends on either side being treated as one are drawn upon to occlude the vessel: the knot is completed by using the nine ends on either side as a single cord and tying the second hitch as if completing an ordinary reef knot. See Exp. 61 and Plate I, fig. 5.

single ligature. Moreover, the knot would be clumsy, the loops would probably not be equally constricted and the force applied could not be so accurately judged as with the stay knot.

The principle upon which the stay knot depends is that the mutual support, which the ligatures afford one another by friction and interlocking, prevents the first hitches of the knot slipping when the ends are relaxed, as they must be to complete the knot. The loops do not expand

in the least, and abundant time is allowed to tie the second hitch without risk of the artery being left imperfectly constricted.

Recently "the uncertainty of occlusion" of the vessel when it is intended not to rupture the coats has been put forward as an argument in favour of rupture, but if the stay knot be used with a suitable ligature there need be no such uncertainty.

It is not only in ligation in continuity that the reef and surgical knots fail the surgeon. In the ligature of ovarian and other peduncles we have seen the first hitch give before the knot was completed; and it is well known that fatal hemorrhage has from time to time occurred in these cases. A mass of tissue like an ovarian peduncle is highly elastic and acts similarly to an artery under blood pressure. A common method is to transfix the pedicle and tie the two halves separately, finally tying the ends of one knot round the whole. But the difficulty with the first hitches remains. To prevent the two halves tearing apart in the tying, and opening a vessel in the pedicle above the ligatures, it is recommended to interlock the two loops. There is no objection to this if only two loops are used, but if the pedicle is so large that it is thought well to use three a new danger arises.

In a very instructive case the pedicle of an enlarged spleen was treated in this manner; first one and then the other of the two lateral ligatures was tied and then the middle. No hemorrhage occurred when the spleen was removed, but while the wound was being closed the

whole omentum and mesentery rapidly filled with blood. The patient died the same evening. At the post-mortem

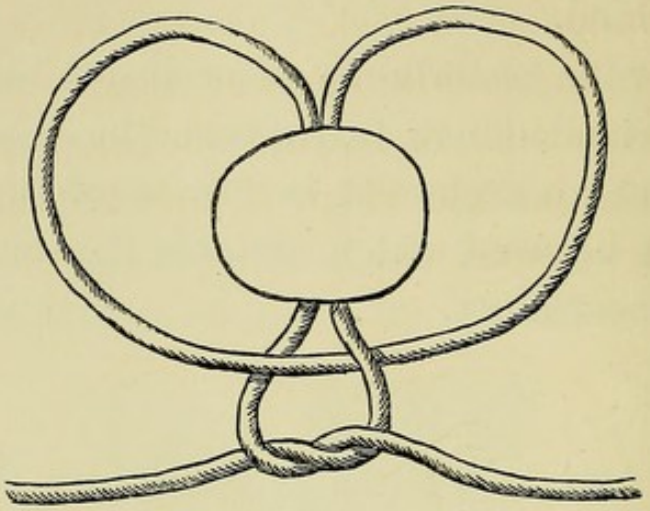


FIG. 183. Staffordshire knot for pedicle (first method).

An armed aneurism needle is passed through the pedicle, the loop is held and the needle withdrawn: a large loop is then pulled through and passed over the tumour: one of the ends is passed through the loop: the whole is pulled tight and a reef knot tied external to the loop.

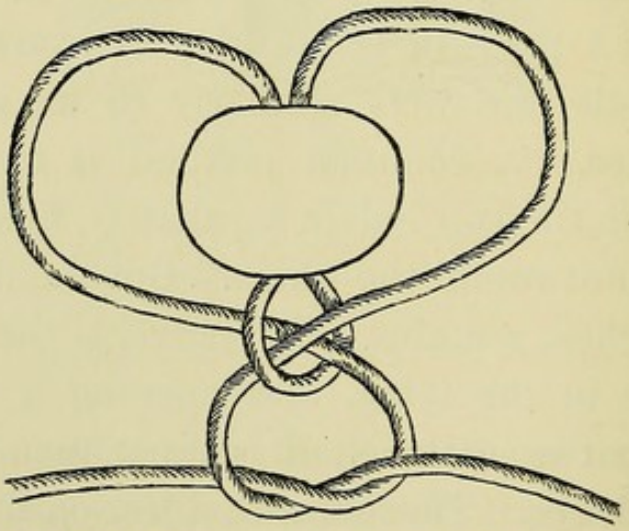


FIG. 184. Staffordshire knot for pedicle (second method).

An armed aneurism needle is passed through the pedicle: the loop is caught and the needle withdrawn: the ends are brought round the pedicle, one on each side, and passed through the loop: all is pulled tight and finally a reef knot is tied external to the loop.

It will be noticed that in the first method the operator must pass the armed needle from him; in the second towards him.

examination it was found that a very small artery had retracted from the middle loop of the ligature, and great

hemorrhage had taken place between the layers of the omentum. "The mistake that was made was in tying the "two outer loops of a locked chain before the middle "one, as when the middle was tied there were two fixed "points on each side of it, and the small membranous "portion of the pedicle which it enclosed was not suffi- "ciently tightly constricted."

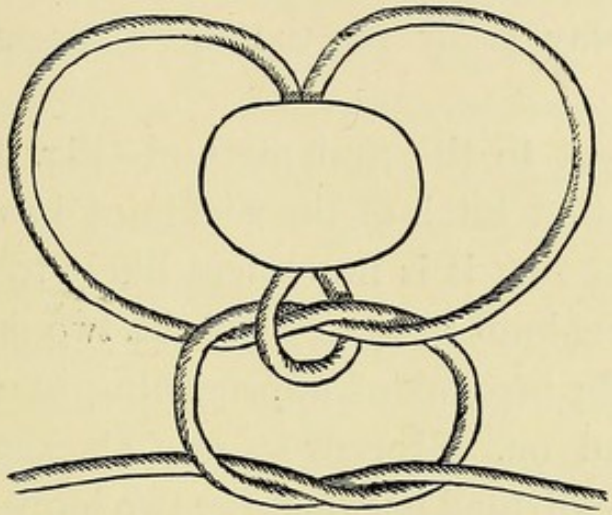


FIG. 185. Staffordshire knot for pedicle (third method).

This method only differs from that shewn in Figure 184 by the ends being hitched as they pass under the loop: the advantage of this is that when all is pulled tight the loop holds the hitch by pressing on it while the first half of the final reef-knot is being tied: it is safer to tie a complete reef-knot external to the loop.

Lawson Tait uses the Staffordshire knot. "An ordi- "nary handled needle armed with a long piece of the silk "required is passed through the pedicle and then with- "drawn so as to leave a loop on the distal side. This "loop is then drawn over the ovary or tumour, and one of "the free ends drawn through it so that one end is above "while the other is under the retracted loop. Both ends "being seized in the hand, they are drawn through the "pedicle, against which the thumb and forefinger of the

"left hand are pressed as a fulcrum, a simple hitch is then "made as in the drawing (Fig. 183) and tightened, and "that is followed by another as in ordinary ligature tying. "There is another and more complicated way of making "the knot, by passing each end of the thread round the "corresponding half of the pedicle and crossing them "within the loop in front, which is equally effective, and "which may be used in cases of large solid tumours; but "the former way is by far the more elegant and rapid "method."

With respect to the first method (Fig. 183) it will be seen that the first hitch of the reef-knot has some chance of slipping, but that it is much less likely to do so than in an ordinary reef-knot because here the two ends to be tied lie side by side instead of approaching each other from a distance end on. Moreover, any slackening that did occur would be divided between the two loops. Figures 184 and 185 are intended to represent the two ways in which the second method might be applied, but there is a vital difference between them, the mere passing of the ends under the loop in Figure 184 being much inferior to hitching them, as shewn in Figure 185, for the loop holds the hitch firm while the reef-knot is being tied. It is best to tie a complete reef-knot external to the loop, that is to say, not to count the hitch under the loop as part of the reef-knot.

This, therefore, is the best method of using the Staffordshire knot, but a stay-knot is equally efficient. Another method is to pass two ligatures side by side, to make one hitch on each and to let an assistant constrict with one while the other is being completed.

Sinclair of Manchester has tried the Staffordshire knot and has watched it and seen it slip before his eyes.

William Duncan also thinks it unsafe. He relates a case of ovariotomy in which the pedicle had been tied with the Staffordshire knot. As he was about to close the wound he noticed a little blood, and on separating the edges he found that the abdominal cavity was full of blood: the ligature had slipped. The pedicle was religatured in another way, and the case did well.

Tait, too, says: "As to the Staffordshire knot, like other "knots, it could not be tied with negligence. Once or "twice I have failed with it through not attending to my "own directions for its use."

An advantage the usual method of tying the peduncle in two halves has over the Staffordshire knot is that by it each half is constricted by a ligature, both ends of which can be and are pulled on; with the Staffordshire knot the central part is fixed, and only the outside portion of the loop is pulled on and constricts.

Finally it must be remembered that however well a ligature may be applied, if the pedicle is cut off too close the loop will slip over the cut end: this, indeed, has happened.

CHAPTER XVIII.

CHOICE OF THE FORCE.

Formation of Pleats. Elasticity of Arteries persists after death. Degrees of rupture. Arterial blood pressure. Excursion of the Ligature. Blood pressure beyond Ligature. Tension in the walls of Arteries and Aneurisms. Force sufficient to occlude the Artery. Force necessary to rupture. Collapsed Arteries no guide. Efficiency of the Stay-knot and its safety with respect to rupture.

Having decided on the ligature and the knot, it has next to be considered with what force the ligature shall be applied, so as to completely occlude the vessel and yet not inflict any injury upon it.

When a ligature round an artery, distended with blood or with water at the natural pressure, commences to constrict the vessel, the first thing that happens is for the wall to be thrown into longitudinal pleats, half a dozen or more in number (Figs. 186 and 187): the surface

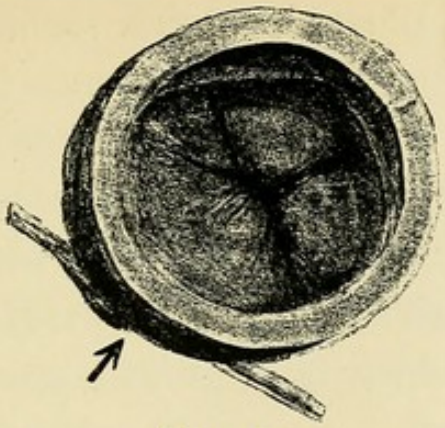


FIG. 186.

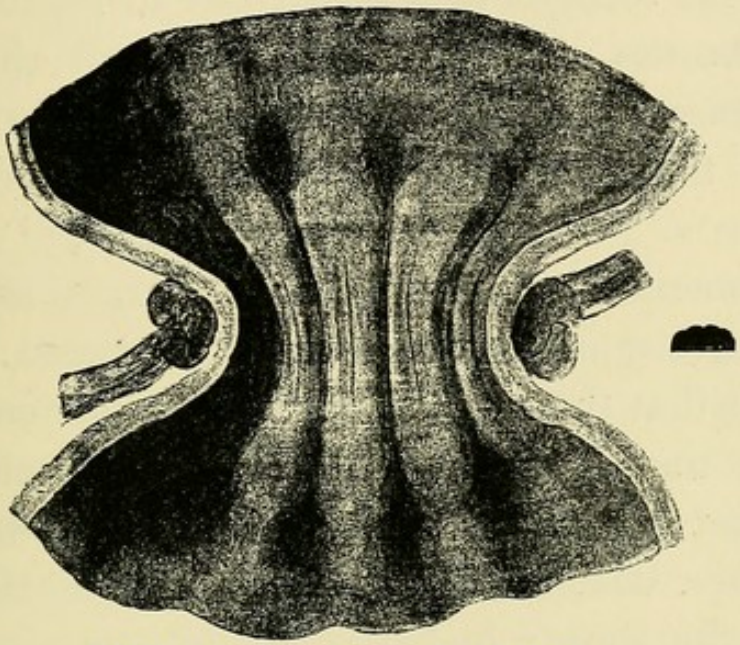


FIG. 187.

FIGS. 186, 187. Artery ligatured with kangaroo tendon (× 4).

The drawings are made to shew the folds into which the wall of the artery is thrown by a ligature of kangaroo tendon when the coats are uninjured.

The folds vary somewhat with the size of the vessel.

The artery was distended with liquid gelatine under pressure, ligatured, and placed in alcohol for a few days.

Fig. 186 is a transverse section made immediately above the ligature: there are three main folds, the middle of the largest of which is under the knot.

Fig. 187 shews the same artery opened out after a longitudinal incision passing through the knot and the artery wall beneath it: the arrow in the upper figure indicates the line through which this incision was made.

The knot is seen cut in two and the folds of the artery wall are exposed, the largest being divided and the halves turned aside: each fold has secondary folds, as indicated by the longitudinal lines on their surface and by the small diagram to the right, which shews a transverse section of a single fold.

of each of these pleats presents secondary foldings: it results from this that when the lumen is just occluded without any injury to the wall the diameter of the constricted part of the vessel is from 3 to $4\frac{1}{2}$ times the thickness of the artery wall. (See Plate X.) The pleats are all of the same size, with the single exception of that immediately under the knot, which is larger, its size depending upon the size of the knot.

When the force is increased so as to rupture the two inner coats, the pleats are destroyed and the external coat assumes a more or less crescentic form around the knot, the concavity being immediately under the knot.

Experiments on human arteries shew that the most various degrees of rupture can be produced: it might be thought that these experiments are fallacious, but Roy has shewn that it is not so. From his observations he states that "from the time of death to that when putre-"faction has commenced the elasticity of the arteries "remains the same: in experimenting on the elasticity "of human arteries, therefore, no error is introduced, "owing to the interval which has necessarily elapsed "between death and the experiment." We may take it then that for our present purpose a human artery after death is the same as a living vessel.

Arteries were tied with various ligatures and with different degrees of force: microscopic sections were made, and the following injuries were found:—

1. Superficial abrasion of intima.

2. Rupture of Henle's membrane, as described by Warren.

3. Short ruptures of intima, sometimes extending into media.

4. Continuous rupture of intima.

5. Continuous rupture of intima and inner half of media.

6. Continuous rupture of both intima and media.

7. Complete rupture of all three coats, that is to say, complete division of vessel.

Some of the injuries inflicted on human arteries under pressure by the ligature are shewn in the accompanying drawings, which are strictly to scale. (Figs. 188—194.)

In rare instances instead of any rupture a deep indentation on the inner surface of the artery is seen: in discontinuous rupture the injuries occur at the apices of the folds, and appear first and are greatest under the knot.

Another apparent result of applying a ligature to an artery is to reduce the thickness of the wall in the immediate vicinity of the rupture: and this is not recovered from when the force is taken off. The following measurements are taken from the same carotid artery, portions

Unit used is 0·01 *millimetre. Measurements made with camera lucida.*

	Outer coat	Middle coat	Inner coat	
Common carotid tied with silk :				
1st degree of force (slight)	3	74	5	82
2nd „ (moderate)	4	70	7	81
3rd „ (complete rupture)	4	53	8	65

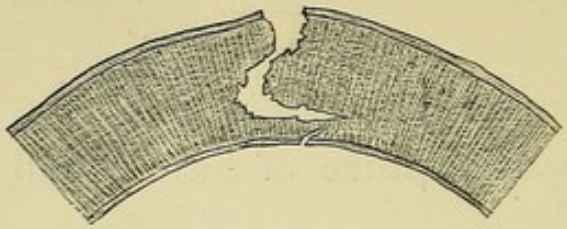


FIG. 188. Innominate.

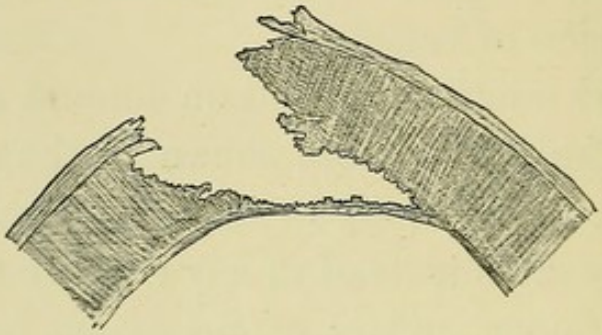


FIG. 189. Innominate.

FIG. 190. Common Carotid, slight force.

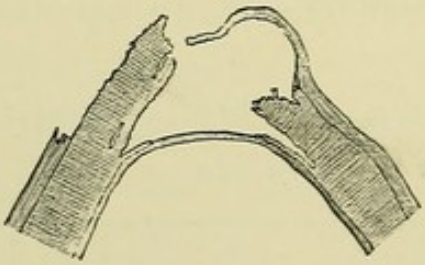


FIG. 191. Subclavian, 1st part.

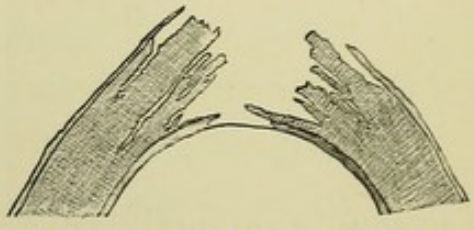


FIG. 192. Subclavian, 3rd part.

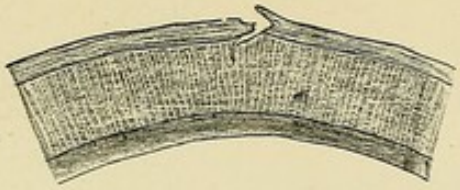


FIG. 193. Superficial Femoral.

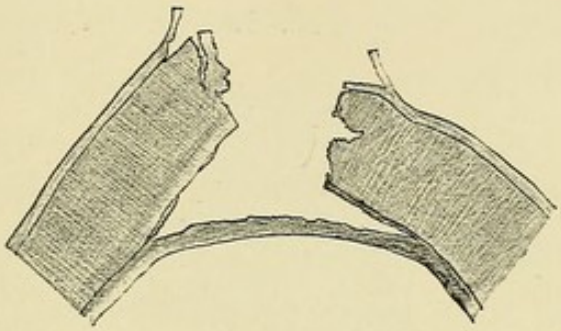


FIG. 194. Superficial Femoral.

FIGS. 188, 189, 190, 191, 192, 193, 194. Drawings of longitudinal sections of various human arteries after ligature with kangaroo tendon (× 10).

The arteries were distended with the pressure of 10 feet of water (220 mm. Hg.). They were ligatured with a kangaroo tendon $\frac{1}{20}$ inch (1·2 mm.) in width; the force used was never more than sufficient to give the sensation of rupture.

The sections were made, stained, mounted, and examined under the microscope, in the usual way.

Figs. 188, 189 shew the ruptures in two innominate arteries: the ligature was applied in both cases external to the thin sheath which this vessel has. In 188, in addition to the usual rupture of inner and middle coats, there is a separate rupture through the sheath and external coat which nearly completes the division of the vessel at this point.

In Fig. 190 (the common carotid) is seen a broad shallow depression in the inner coat: the extreme thinness of the outer coat will be observed.

In Figs. 191, 192 (subclavian) the ligatures were applied external to the sheath. In 192 the sheath alone remains to maintain the continuity of the vessel.

In Figs. 193, 194 (superficial femoral) different degrees of rupture are shewn: note the thickness of the outer coat.

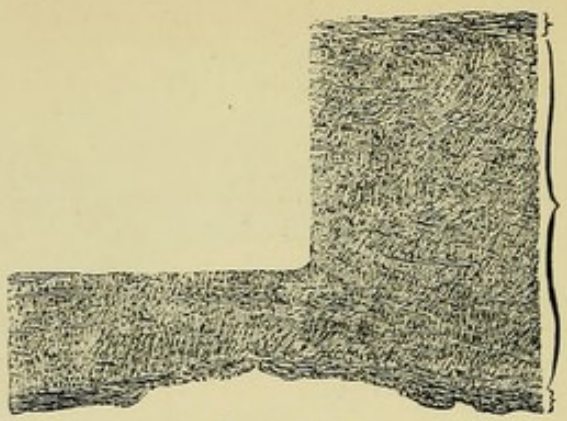


FIG. 195.

FIG. 196.

FIG. 197.

FIGS. 195, 196, 197. Human Carotid ligatured with silk (× 50).

The vessel was distended with a pressure of 10 feet of water (220 mm. Hg.): it was then ligatured with silk of medium size (No. 4) at three different points: no excessive force being used. The drawings illustrate different degrees of rupture.

The sections were stained and mounted in the usual way.

Fig. 195 bruising of intima.

Fig. 196 rupture of inner and half middle coat.

Fig. 197 complete rupture of both middle and inner coats. The comparative thinness of the whole wall in 197 is probably due to the compression exercised by the ligature.

of which are represented in figures 195, 196 and 197. With the greater force the total thickness of the artery is reduced from 0·82 to 0·65 mm.; the compression occurs wholly in the middle coat which is reduced from 0·74 to 0·53 mm.

In distending some of the great arteries and intentionally rupturing their coats during ligation, we were surprised to find that even when wide ligatures were used, with only reasonable force, the whole thickness of the vessel sometimes became divided under the knot; this occurred with the innominate, the first and third parts of the subclavian, the carotid, the abdominal aorta, and the common iliac; the vessels employed were perfectly healthy and from young adults (see Figure 198): so much was this the case that to occlude the end of the artery and collateral branches, before distending it for the experiment, we found that silk so often cut through the vessel and caused it to leak under pressure, that its use had to be partly given up and flat metal clamps used instead.

John Lidell, late Surgeon to Bellevue Hospital and Inspector of the medical department of the army of the Potomac, in giving directions for the deligation of an artery in continuity, writes (1883): "One assistant takes "a position where he can compress the artery on the "proximal side if by any accident it should be wounded or "if the ligature should cut through its tunic." We have heard, too, of a case in London in which the ligature so completely cut the artery that the blood spurted out there and then.

When the two coats of a dead artery are ruptured with a ligature its strength is destroyed, it bends sharply

at the site of injury instead of merely curving, and in fact is like a broken stick.

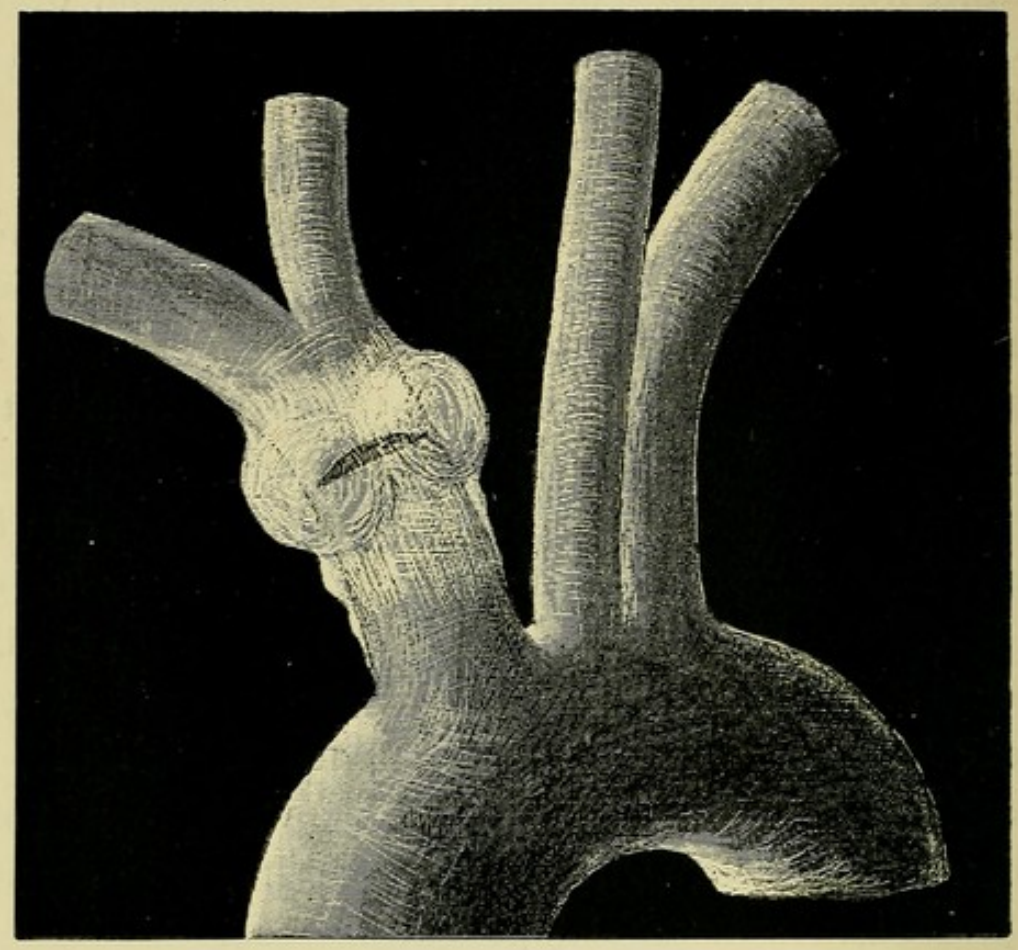


FIG. 198. Arch of Aorta with great vessels. (Nat. size.)

(*Specimen in Museum of St Thomas's Hospital.*)

The parts were removed from the body of a healthy young male adult: the vessels were distended with a fluid of same specific gravity as the blood by a pressure of 5 lbs. to the sq. inch (240 mm. Hg.): round the innominate external to its sheath were placed three kangaroo tendons, each $\frac{1}{20}$th inch (1·2 mm.) wide; the first half of a reef-knot was tied on each, the three ends on each side were pulled as one, the force (which was measured) being gradually increased up to 10 lbs. (4·5 kilos), when the vessel suddenly gave way, and the sheath became distended with the fluid as represented in the figure: on removing the ligatures there was seen immediately under the knot a transverse rupture of the artery passing completely through all three coats; the inflated portion seen in the drawing is the sheath of the vessel and not the outer coat (which was ruptured). This is not an isolated experiment (see text, p. 411).

When, however, such an artery is distended with water, at the pressure of the blood there is no bulging or

irregularity of contour, but a white line surrounding the vessel indicates the place where the adventitia alone prevents the escape of fluid. (Fig. 199.)

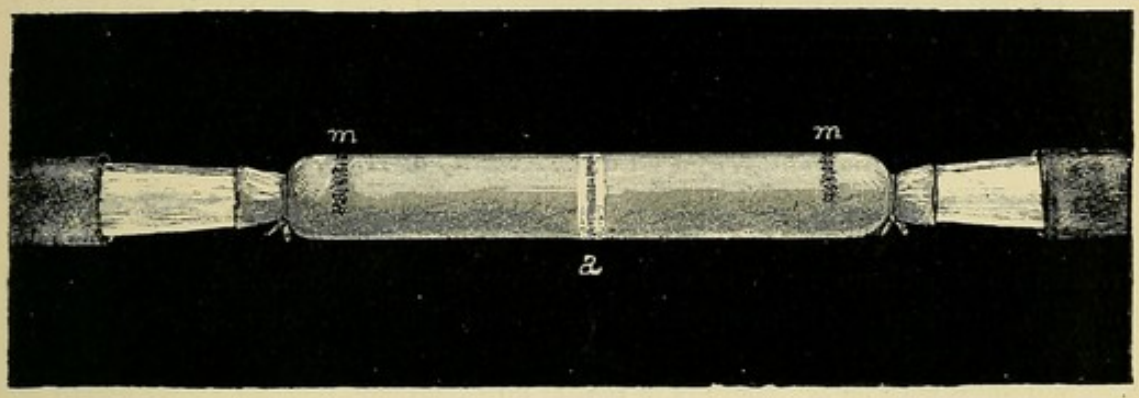


FIG. 199. Distended and ruptured Human Carotid.

The figure shews a carotid artery of man distended with water from both ends, as shewn in fig. 206, p. 424: the marks *m m* have resumed their original distance apart at a pressure of 170 mm. Hg.: a rise to 400 mm. Hg. made the marks only very little more apart: at *a* the two inner coats had been ruptured by a small round silk ligature: even with a pressure of 400 mm. Hg. the vessel does not bulge in the least, but the outer coat is so thin as to be translucent at the part shaded dark: the light part on each side is opposite the recurved coats.

Figure 201 is from Warren's monograph. A carotid artery of a horse had been ligatured and the coats purposely ruptured: it appears as if broken in two. Figures 200, 201, 202 should be contrasted with those in Plates I., II., and III.

Besides the local effect, the application of the ligature has an influence on the blood pressure within the vessel. If, for instance, a ligature be applied to an artery which gives off no branches, it results that the artery becomes a cul-de-sac, and the blood pressure in it thus becomes that of the artery from which it arises: for instance, ligating the left common carotid converts it into a cul-de-sac from the aorta, and raises the blood pressure in it to that

FIGS. 200, 201, 202. The Common Carotid Arteries of Horses at various periods after ligation: the silk ligature having in each case ruptured the coats.

(*Reproduced by kind permission of Dr Collins Warren of Harvard University, from his monograph on the "Healing of Arteries,"* 1886.)

Fig. 200. 14 *days after ligature.* "The proximal thrombus is white, for what "reason does not appear. The walls of the vessel are continuous around the base "of each thrombus and they appear to have united by first intention, which is not "the case. On either side of the ligature the ends of the vessel are distended "ampulla-like with clot. There is in reality no dilatation, but the vessel above and "below the thrombi has greatly contracted. The adventitia may be traced under "the microscope down the sides of the vessel into the callus, but apparently it has "been absorbed at the point of ligature."

The silk can be seen. The wall of the artery is completely divided.

Fig. 201. "60 *days after ligature*—shewing the external and internal callus "and the open ends of the vessel.

"One month after the ligature was applied the wound reopened, and a small "sinus continued to discharge until the animal was destroyed. The sinus was found "to communicate with the ligature, traces of which could still be seen. The condi- "tions are favourable for a breaking down of the material which plugs the interior of "the vessel, but this tendency has been overcome by the excessive callus formation. "The balance between the suppurative and reparative process is thus maintained to "an extent sufficient to prevent secondary hemorrhage."

The drawing shews the sinus leading to the remains of the ligature between the ruptured ends of the artery. It is also a typical example of the appearance presented by a great artery which has been subjected to the cutting ligature: broken and crippled, it can bend like a hinge to an acute angle when taken hold of on one side of the oval mass of new tissue. The danger of such a sinus as is here described when the artery wall is not intact must be evident.

Fig. 202. "120 *days after ligature.* The specimen shews absorption of the "external callus." Warren's 3rd stage of repair.

"A differentiation of the cell elements of the internal callus is however already "taking place, and cells, closely resembling muscular cells, can be seen in large "numbers."

Thus in time Warren's "muscular" cicatrix is formed.

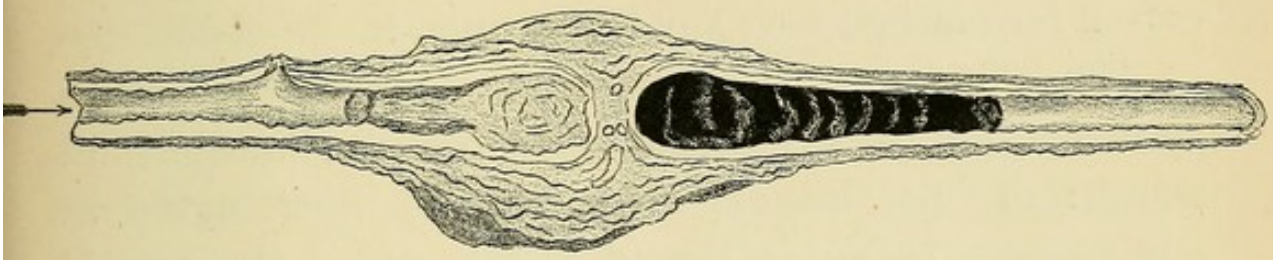


FIG. 200. 14 days.

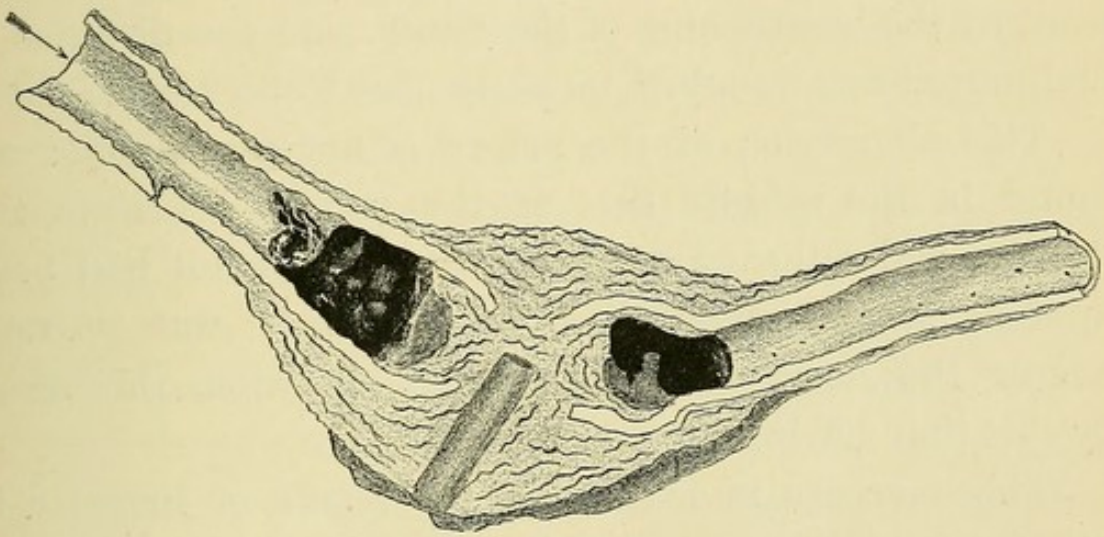


FIG. 201. 60 days.

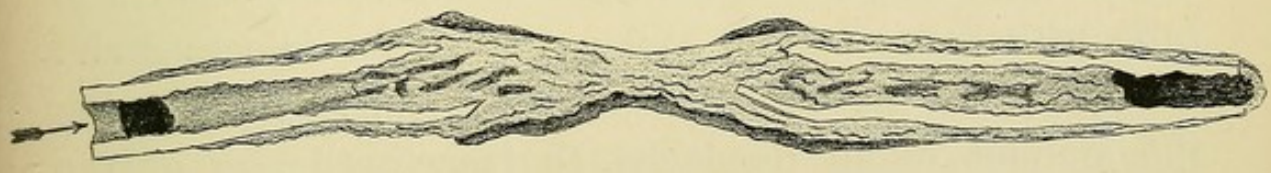


FIG. 202. 120 days.

of the aorta, that is to say, to a pressure to which it has never been subjected before, and which it is not adapted to withstand, and this in itself constitutes a reason for not inflicting any injury upon it.

The effect of this increase of pressure is to cause an increase in the size of the vessel, both in diameter and length: to test this experimentally Dr Sherrington at our request, passed a thread loosely round the carotid artery of a rabbit and accurately noted its position: on tightening the loop so as to occlude the vessel the ligature was seen obviously to be carried towards the head by the lengthening of the vessel, and the distance it had moved was found on measurement to be 2 mm.

This elongation of the artery is not permanent: we found in the rabbits that at the end of six days the ligature had returned to the position in which it had been applied, and at the end of two months it was 3·5 mm. nearer the heart than its original position, the vessel by this time having shrunk.

This increase in length, as the result of increase in pressure, we have frequently observed in the human artery when experimenting with water pressure.

Roy and Adami have found the same: they removed the carotid of a rabbit and connected it by means of a T tube with a manometer and a syringe for injecting air: the artery was placed in a vessel full of oil: by measuring the amount of oil displaced the increased size of the artery at different pressures could be determined: it was found that the vessel was most elastic at about its normal blood pressure, and that at higher pressures it became more rigid: thus the change in size from a pressure of 60 mm.

to 70 mm. of mercury, or from 70 mm. to 80 mm., was equal to the whole increase from 110 to 170 mm.

On the carotid of a dog, too, we have seen the same movement of the single ligature away from the heart: no doubt it always occurs to a certain extent.

With respect to the effect of the ligature on the pressure of the blood, Dr Sherrington made the following experiment for us: a dog was placed under an anæsthetic and the abdominal aorta, the two common iliacs and the large middle sacral artery were exposed: a cannula was inserted into the left common iliac and connected with a manometer and a recording drum: the blood pressure was now seen to be about 85 mm. Hg.: on occluding the aorta above, the pressure fell to about 18 mm. Hg.; the time taken in this fall was about two minutes: on releasing the aorta the pressure returned to 85 mm. Hg., and on occluding the middle sacral and right common iliac the pressure rose 10 mm., that is to say, to 95 mm. Hg.

The above shews that the effect of the application of a ligature to a large artery is to greatly reduce the blood pressure on the distal side, and that this reduced pressure continues at least as long as would be taken in the completion of the knot. Owing to the clotting of the blood, it would not be possible to determine by this method whether, and if so, when and to what extent, the pressure would return.

It appears, too, that the ligature of the common iliac and also of the middle sacral only increases by very little the pressure in the opposite common iliac; presumably a like small rise would occur in one carotid of man when the other is occluded.

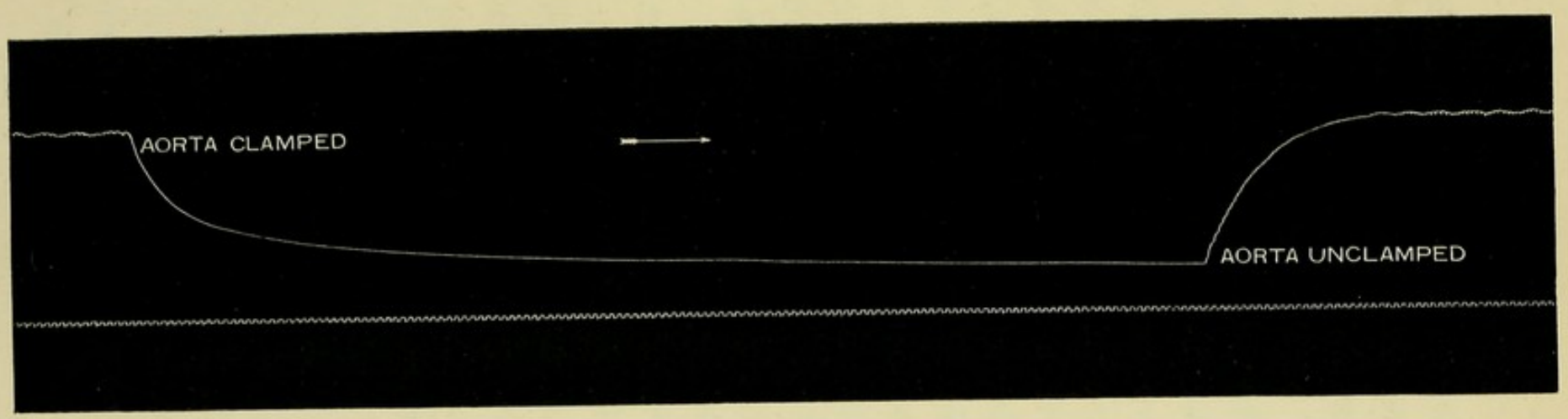


FIG. 203.

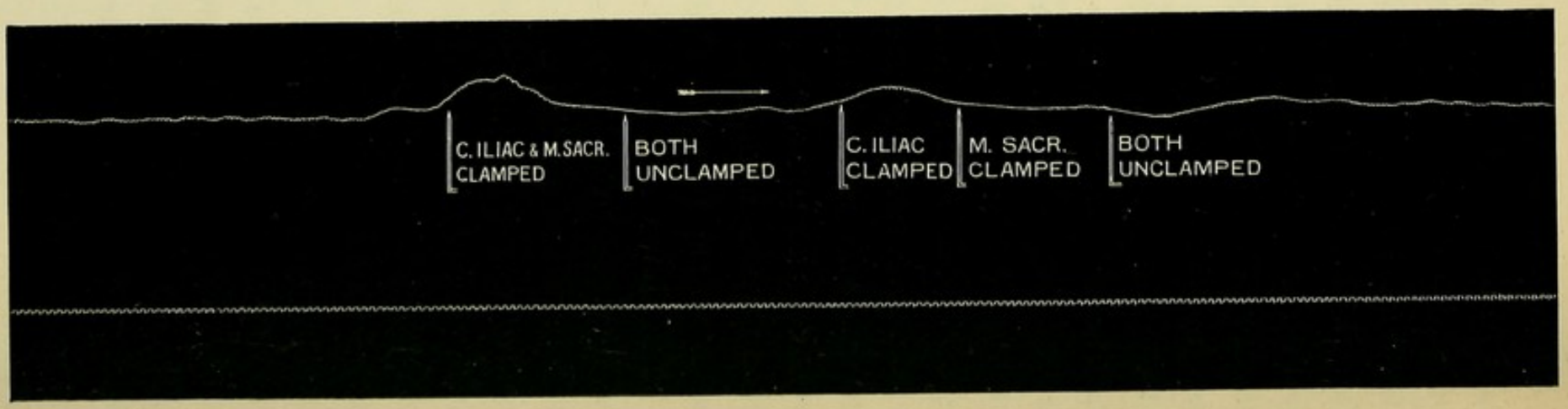


FIG. 204.

Descriptions of Figures 203 and 204.

Figures 203 and 204 shew blood-pressure curves from a dog. A cannula was inserted into the left common iliac artery of a small dog, and the blood pressure was recorded in the usual way by a mercurial manometer with a float and a pen attached writing on a revolving drum.

The tracings have been reduced one-half: the seconds' line is also the base line, that is to say, the level at which the needle would mark if there was no pressure: at the commencement of the experiment the blood in the common iliac artery was at about 90 mm. Hg. pressure: the aorta was then clamped, a great fall of pressure ensued and continued till the aorta was unclamped, when it immediately rose: the pressure fell to about 20 mm. Hg., that is, it fell 70 mm. Hg.

The aorta being unclamped, the effect of clamping the right common iliac and the middle sacral artery was tried: this latter vessel is of large size in the dog: when both these arteries were clamped at the same time the blood pressure rose about 10 mm. Hg., when either was clamped separately the pressure rose 6 mm. Hg.: by looking closely at the tracing in Figure 204 it will be seen that the effect of unclamping the vessels is to take away all pulsation for a time from the artery.

With respect to the Celsian operation: the carotid artery of a dog was exposed, two ligatures were placed on it 8 mm. apart, and the artery divided between them: it was found that the proximal ligature had moved towards the heart 3 mm. and the distal in the opposite direction 4 mm., so that the two ligatures were now separated 15 mm.

In another experiment (on the common femoral), in which the ligatures were 14 mm. apart, the upper ligature moved 4 mm. and the lower 5 mm., making the total separation of the ligatures 23 mm.

Before discussing the force that should be used in ligating, it will be well to get a fair idea of the force which has to be overcome, of the force, namely, that is distending the vessel: also, as the longitudinal tension in the artery is the ground for recommending the Celsian operation, it is desirable to be clear about it. In Chapter III. it was shewn how, when an artery is distended during life with the blood at its usual high pressure, it occupies the whole or nearly the whole length of its bed with little or no assistance from its terminal attachments. What assistance (if any) there is, we spoke of as external tension. What is true of one artery is true also of the whole arterial system. Suppose, for example, all the arteries of a limb freed during life from all surrounding structures, and closed at their ends; the normal blood pressure being maintained the alteration (if any) from their normal anatomical lengths would depend upon the amount (if any) of the external or anatomical tension. Under normal circumstances the obstruction to the circulation by the

friction in the arterioles and capillaries is the main cause of the blood pressure in the arteries.

Apart from this anatomical tension, the pressure of the blood produces, as we shall see, considerable tension in the wall itself,—this may be regarded as the internal or blood tension.

The blood tension is not in addition to the anatomical tension but partly or wholly relieves it.

With regard to the nature of the anatomical attachments, it is probable that the branches of the arteries are the main, if not the only means, by which they are fixed. The coats of each artery support, we believe, alone the lateral pressure of the blood without outside help. The large arteries are separated from their sheaths by a distinct lymph space, and the delicate fibres which cross this are not strong enough to withstand any tension.

If the carotid artery be exposed after death, and two marks made on it, it will be found, when the artery is divided at both ends well beyond the marks, that they have approximated. In one of our experiments the marks were 28 mm. apart, and after the removal of the artery were only 21 mm. apart: if now this artery be attached to a tube leading from a reservoir of water 10 feet in height and the end of the artery be left open, the water will flow out, and the artery will neither dilate nor lengthen.

If a clamp be applied on the free end of the artery, it will be seen to both lengthen and dilate considerably.

If now, in order to more accurately measure the pressure, the artery be attached to a syringe and manometer (as shewn in figure 37, page 53) and the end of the

artery again clamped, it will be seen, as the pressure is gradually raised, that the artery enlarges both in diameter and length; in the experiment of which we are speaking it was found that the original space of 28 mm. between the marks was regained, when the pressure was raised to 170 mm. Hg.: a further rise to 200 mm. Hg. made no appreciable further separation, and a rise to 400 mm. Hg. only made the marks 2 mm. more apart.

The tension on the walls of any given artery for any given blood pressure can be calculated.

The diameter of the common carotid is about 8 mm. Let us consider in the first place a length of the artery 1 mm. long (see figure 205).

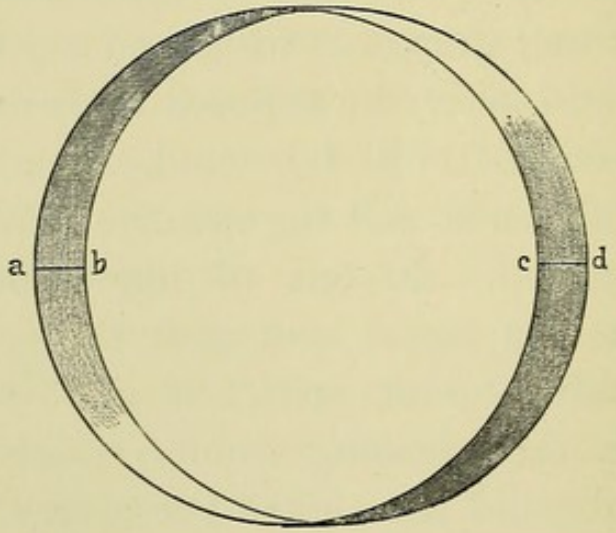


FIG. 205.

(*ab, cd, to represent* 1 *mm. each.*)

Now the force derived from the pressure of the blood, tending to lift off the upper half of the circle of artery shewn, will be the force, tension or strain at *ab* and *cd*. The pressure of the blood will act vertically to the inner surface of the tube and its vertical component will be

equal to the same force applied to the parallelogram *abcd*, the area of which is 8 sq. mm. If we take the pressure of the blood to be 200 mm. Hg. (which is 2·7 grammes to the square millimetre) the lifting force will be $8 \times 2{\cdot}7 = 21{\cdot}6$ grammes; this force will be divided equally between *ab* and *cd*; at each the tension is therefore 10·8 grammes: and this is the transverse tension on any one millimetre in the length of our artery.

Again, if we imagine a cork fastened in the end of the artery, the diameter of the base of the cork will be 8 mm. and the area of the base will be $8^2 \times 0{\cdot}7854 = 50{\cdot}2656$ square millimetres: the pressure of the blood being 2·7 grammes per sq. mm., the total pressure on the cork or longitudinal strain on the artery will be 135·7171 grammes (= 5 oz.), the circumference of the base of the artery will be $8 \times 3{\cdot}1416 = 25{\cdot}1328$ mm., and the longitudinal strain on each millimetre in the circumference of the artery will be 5·4 grammes. This is just half the transverse tension, which was 10·8 grammes.

Thus we see that the tension or strain on any artery is dependent not only on the blood pressure but also on the diameter of the artery; in fact, it is directly proportional to both: the transverse tension, too, is seen to be double the longitudinal: it is a well-known law for cylindrical vessels that the tension is proportional both to the fluid pressure and the diameter, and that the transverse tension is double the longitudinal.

In the living body the friction of the blood in passing through the arterioles and capillaries plays the part of the cork, which we have supposed fixed in the artery; if the blood could pass freely along the capillaries, it would

do so and there would be no blood pressure in the arteries. Contraction of the arterioles raises the blood pressure, dilatation diminishes it. We found that injecting the fluid at both ends of the artery (as shewn in figure 206) produced the same effect as closing one end

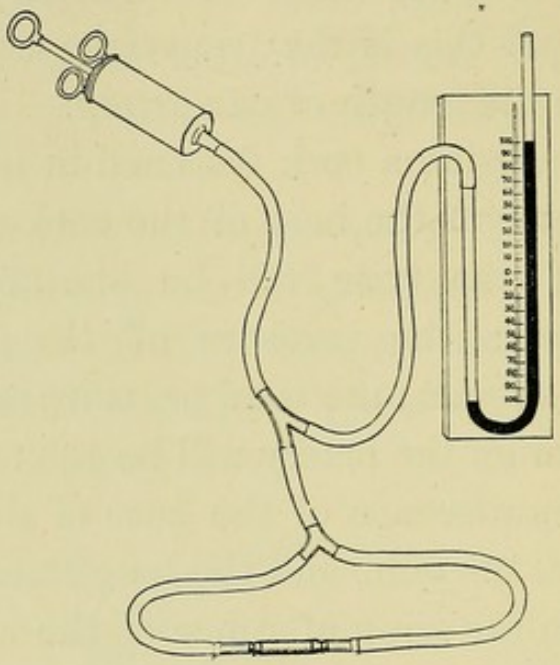


FIG. 206. (See fig. 199.)

and injecting at the other, and indeed it was only to be supposed that it would.

It might be objected that the escape of the blood through the capillaries is not completely blocked, but what has been said applies only to the actual amount of blood pressure, which is caused by the obstruction that does exist. If the arterioles and capillaries were completely blocked, the blood pressure, and consequently the tension, would be higher.

We found in the experiment to which we have been referring that the carotid regained its normal length at a pressure of 170 mm. Hg., that is to say, it requires a total

longitudinal tension or strain of 115 grammes (4 oz.) to maintain it at its natural length; and as it is maintained at that length both during life and after death, it follows that it has always upon it at least that strain, which must be derived either from the blood pressure within or from the strain on its attachments or partly from one and partly from the other. When (if ever) during life the blood pressure in this artery was 170 mm. Hg. or higher, there was no tension on the attachments of the artery: when it fell below this, there must have been some such tension, and finally, after death, when the blood pressure ceased, the whole tension of 115 grammes must have fallen on the anatomical attachments.

The blood pressure varies in different individuals, and at different times, and in different positions in the same individual.

And now for the bearing of all this on surgery.

The first effect of ligating a large artery is immediately to arrest the current of blood so that the ligature acts like a cork, and the blood pressure rises to that in the vessel of which the artery is itself a branch. This tends to cause a slight lengthening and helps to account for that movement of the ligature away from the heart, which has been already mentioned. See page 416.

On the distal side the blood pressure, as we have seen on page 417, is at first greatly reduced, and this puts a strain on the attachments of the collapsed portion of the artery. "Anatomical" tension to a great extent replaces the "blood" tension. Now one of the attachments of this collapsed portion of the artery is the distal extremity

of the proximal part, and this too may have something to do with the movement of the ligature.

If the force required to hold out the distal portion of the artery at its proper length is the same when it is collapsed as when it is distended, the collapse would have no effect on the position of the ligature or the tension of the wall: if otherwise it would.

The application of a second ligature beyond the first produces little or no further alteration in the tension.

The division of the artery between the two ligatures will produce various alterations.

On the proximal side, that part of the tension of the wall, which is due to the pressure of the blood within, will remain unaltered, but any anatomical tension of the distal part on the proximal part of the artery will cease, and the artery will in consequence contract. That there is such tension, and the effect of relieving it, has been seen in the experiment related on page 63.

On the distal side the blood pressure being less, the "anatomical" tension will be more and it will be relieved by the division.

The followers of Celsus are therefore entitled to claim that the division of the artery does relieve tension, but it must be remembered that the tension due to the pressure of the blood on the proximal and distal sides will remain: the former is somewhat more than natural, and the latter may be considerable after the establishment of the collateral circulation.

But as has been said before, the Celsian operation is only practicable in certain arteries, and in them is unnecessary.

Ligation with a single ligature rupturing the two inner coats, leaves the tension to act on the sole remaining outer coat. There is, however, no need to relieve tension if the coats are not ruptured, for they then are not weakened and are well able to resist the tension, which they are adapted and constructed to bear.

The diameter of a tube having such a great influence on the tension of its walls, explains why aneurisms are so much more common in the aorta and other large vessels than elsewhere; the difference is far more than the mere difference in blood pressure, which does not vary much in the principal arteries.

The diameters of the aorta, innominate and carotid arteries are about 28, 14 and 8 mm. respectively, and the strength of their walls should also be in this proportion, and so perhaps it may be, but the thickness of the walls is 1·23, 0·98 and 0·78 mm. respectively.

It may here be remarked that the tension on the walls of the large arteries affords yet another argument against rupturing their coats in ligating.

With respect to aneurisms, it will be seen what a strain must fall on the walls of those that are fusiform (such as is shewn in figure 154 page 317): if the artery be dilated for a certain length to three times its diameter the strain on the wall will be three times as great.

In a sphere containing fluid, the tension of the wall is proportional to the diameter of the sphere and to the fluid pressure, but the tension is only half the transverse tension in a cylinder of the same diameter.

If an artery of say 10 mm. diameter has on it a spherical aneurism of 40 mm. diameter, and if there is

free communication between the two and no clot in either, the tension in the wall of the aneurism will be double that in the artery: this explains in part the growth of aneurisms; the reason why large aneurisms do not grow more rapidly than they do is because they are in great part filled with clot.

In many cases of aneurism, as the result of the disease of which the aneurism itself is only a part, there is a considerable rise in the blood pressure, and this would induce a further rise of tension in the wall of the aneurism; thus there is no difficulty in understanding the rapid rate at which some aneurisms grow and the rupture of others.

It seems strange that when a vessel is ligated and the whole pressure of the blood is thrown upon the small collateral vessels, that they do not rupture: this they appear never to do: the explanation seems to be that the tension in their walls, being proportional to their diameter, is too small to do harm.

In the early days of steam-power the makers of the boilers did not design them proportionately strong for their diameters—they not unnaturally only took into consideration the steam pressure with which they were intended to be used—the larger boilers therefore were prone to burst.

As has been said before (see page 64) a longitudinal incision in a distended artery causes a wide opening, while a transverse incision causes a very narrow opening: the explanation seems to lie in the fact that the transverse tension is double the longitudinal: the vessel remaining distended, the transverse tension is constantly tending to separate the edges of a longitudinal incision and to

pull together the edges of a transverse incision, and the longitudinal tension is tending to do exactly the reverse: the transverse tension being double the longitudinal, the longitudinal incision is widened out and the transverse held with its edges nearly touching.

The elasticity of arteries has been made the subject of experiments by Roy: he cut strips of arteries 1 cm. wide both in the longitudinal and transverse directions, and by attaching weights to them measured the elongation by a recording apparatus: he found that "in all of them it can "be seen that the longitudinal strip expands with light "weights more readily than the transverse strip, but that "with heavier weights this relation is reversed; for example, "with weights below 90 grammes the longitudinal strip "was longer than the transverse, while with weights above "90 grammes the reverse was the case."

Our own observations have been confined to small weights: as far as they go they agree with Roy's. The method we adopted was merely to stretch separate squares of artery wall, first in a longitudinal and then in a transverse direction, using a spring balance to shew the force used: a pull of 1 oz. (30 grammes) increased 6·5 mm. of a human common carotid to 10·5 mm. in the transverse, and to 15 mm. in the longitudinal direction.

It has been already seen (page 423) that the tension of a human common carotid is approximately 10 grammes per mm. (i.e. 100 grammes per centimetre) in the transverse direction and 5 grammes in the longitudinal: therefore, at the tension at which arteries are during life, they are somewhat more extensible in the longitudinal than the transverse direction.

It will be remarked, however, that the wall of the artery is not in the same condition in the above experiments as during life, for then the transverse and longitudinal tension are acting at the same time.

There seems to be little doubt that when secondary hemorrhage occurs after the use of a single ligature the blood generally comes from the distal side.

The cause of this is not clear, but it is probably in part due to there being a smaller clot on that side: it may be remarked that the secondary movement of the ligature towards the heart (page 416) would seem to imply, and indeed be due to, a shortening of the proximal part; this would necessitate a lengthening of the distal section, and this would be associated with an abnormal amount of longitudinal tension in it: this in its turn may have to do with the occurrence of hemorrhage from the distal side.

To determine the force which in the human subject is necessary to occlude, and yet not so much as to inflict injury on the vessel, it is not sufficient to experiment with collapsed arteries after death, for the pressure of the blood makes a great difference. Thus, if a collapsed human carotid be taken and the first hitch of a reef-knot be tied on it with silk, and drawn and held with a spring balance, it will be found on trying to inject water through it with a syringe, that when the force being used is less than 4 oz. (114 grms.), the water passes through; when the force is 4 oz. or more the vessel is occluded; and further, that when the force is $2\frac{1}{4}$ lbs. (1 kilog.), the coats are ruptured. (See page 435.) Hence it is seen that the elastic resilience of the artery itself (apart from the

blood pressure) which opposes occlusion amounts to 4 oz., while the natural strength of the wall yields to a force of 2 lbs. (880 grms.) more.

If, however, the same artery be attached to a column of water, 10 feet high (220 mm. Hg.), it will be found that a force of 4 oz. is now quite insufficient to occlude, and that to effect this a force of $1\frac{1}{4}$ lbs. (570 grms.) is necessary, and also that $2\frac{1}{4}$ lbs. (1 kilo.) will not rupture the coats, but that to do so a force of 3 lbs. (1365 grms.) is requisite.

It is necessary, therefore, to make our experiments under the pressure of the blood. This in man cannot often be determined directly: in animals the first observations were made by the Rev. Stephen Hales, Rector of Farringdon (1730). He connected a glass tube with the crural artery of a horse and found the blood to rise in it to a vertical height of more than 8 feet: this would give a pressure of about 200 mm. Hg. A large number of experiments have been made since, and as a result of them, Mr Langley, of Cambridge, informs us that the blood pressure in the common carotid of large animals is approximately as follows:—

Horse	160—220 mm.	of mercury.
Sheep	155—210 mm.	„
Man	150—200 mm.	„
Large dog	140—180 mm.	„

The height of the mercurial barometer is 760 mm.; thus the average pressure in the carotid of a man may, according to Langley, be taken at about a quarter of an atmosphere, or 190 mm. Hg.: this would be about 8 ft. 6 inches of water.

There are some reasons to think that these estimates are rather low, and we have used in the following experiments the pressure of a column of water 9 feet high, or 200 mm. Hg., or about 4 lbs. on the square inch.

These pressures are those on the sides of the artery: the pressure onwards is probably the same as the lateral pressure in the artery from which the artery in question is given off: when the artery is occluded by a ligature, the lateral pressure rises till it becomes that in the artery above: when clot forms and other changes occur the pressure diminishes and may ultimately cease. In connection with this subject a paper by Cybulski may be read with interest.

Vierordt, in his *Daten und Tabellen*, estimates the pressure in the great arteries near the heart as about 200 mm. Hg.: by direct experiment during an amputation Faivre found the pressure in the femoral 120 mm. Hg.; in the brachial on one occasion 120, and on another 100 mm. Hg. Albert found the pressure in the anterior tibial 100—160 mm. Hg.: Esmarch's bandage applied to the other leg raised the pressure 15 mm. Hg., which agrees with the ligature experiment mentioned on page 417. With Basch's sphygmomanometer Eckert found the blood pressure in the superficial temporal artery to be 174 mm. Hg.: in the radial Basch calculated it at 145—180 mm. Hg.

For determining the blood pressure in men Roy has devised an instrument which he calls the sphygmometer: a description of it will be found in a paper in the *Practitioner* (1890) by Roy and Adami, entitled *Heart Beat and Pulse Wave.*

Professor Roy writes to us "that the blood pressure "varies much in the same individual with the degree "of muscular activity, being for instance higher when "standing than when sitting, and higher when sitting than "when lying down: several other influences physiological "and pathological affect it: a fair average in the radial is a "minimum of 100 mm. Hg. with a maximum of 140 mm. Hg. "for a healthy man sitting down: in a case of double "aortic disease I found a minimum of 50 mm. Hg. and a "maximum of 200 mm. Hg. It has been clearly proved "that the blood pressure as measured by the mercury "manometer falls only very little between the root of the "aorta and the large arteries, shewing that the loss of "energy by friction in them is relatively very small."

It must be remembered that these pressures are taken from the radial; some allowance also must be made for the resistance of the skin.

It would appear to us that the most satisfactory measurements are those taken directly from arteries divided in amputations, but these too would be below the normal in consequence of the patient being anæsthetised. It seems, therefore, that the pressures used for distending vessels in the experiments related in the chapter on the Nature of Arteries are sufficiently exact for the purpose in view.

While the ligature is being tightened the elasticity of the artery at first assists and later resists, also the blood pressure is gradually rising, and it is not possible (we gather), the diameter of the ligature being given, to calculate the force necessary to occlude the vessel: it must therefore be determined by experiment.

For this purpose a method was adopted, by means of which it was possible for a vessel to be distended with water to about its normal pressure. (See figure 207.) A strong saline solution, to which glycerine was added,

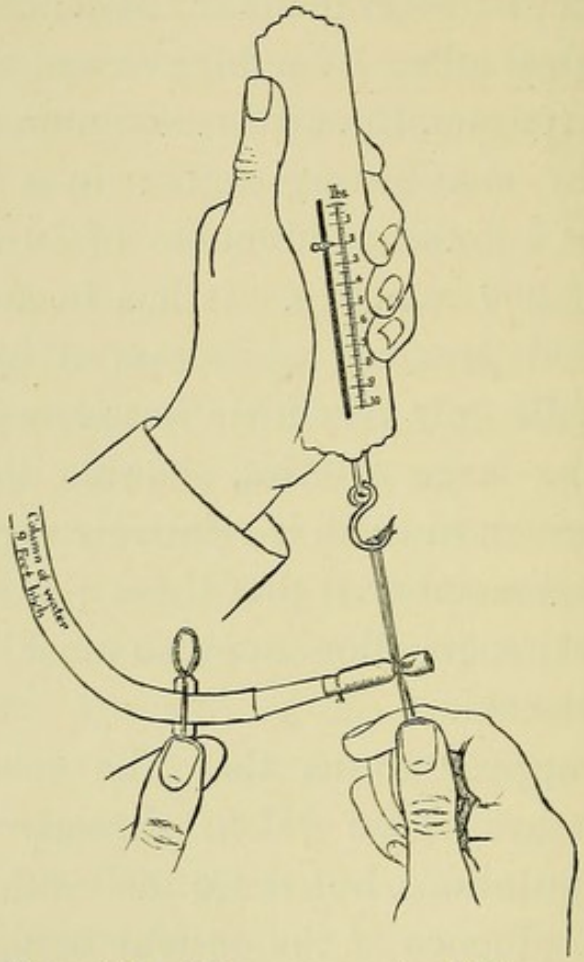


FIG. 207. Measurement of Force.

Fig. 207 shews roughly the method adopted to determine the force necessary to occlude an artery under pressure and the relative value of the various knots (scale $\frac{1}{4}$ nature): the portion of artery is tied on to a glass tube in connexion with a column of water 9 feet high (200 mm. Hg.); the first hitches of a stay-knot have been tied, and the artery is being constricted by a force of 2 lbs. (910 grms.), as indicated on an accurate spring balance. No water escapes from the artery while this force is exerted and no injury is inflicted on the vessel.

was made to the specific gravity of the blood, but the results were the same as with plain water.

Experiments with this shewed that the size and material of the ligature make a difference in the force required:

the results are given in the following tables, where they are contrasted with experiments on undistended arteries.

EXPERIMENTS ON THE COMMON CAROTID OF ADULT MAN.

Artery not distended.

Material of Ligature	Force which occludes	Force which ruptures both coats
Silk, No. 6 (large)	4 oz. (114 *grms.*)	2¼ lbs. (1021 *grms.*)
Kangaroo tendon $\frac{1}{10}$ inch (2·5 mm.)	4 oz. (114 *grms.*)	2¼ lbs. (1021 *grms.*)

Artery distended with a force equal to that of a column of water 9 *feet in height.* (200 mm. Hg.)

Material of Ligature	Force which occludes	Force which completely ruptures both coats
1 Kangaroo tendon $\frac{1}{20}$ inch wide (1·3 mm.)	¾ lb. (340 *grms.*)	3 lbs. (1361 *grms.*)
2 ditto (stay knot)	1 lb. (454 *grms.*)	5 lbs. (2268 *grms.*)
3 ditto (stay knot)	1½ lbs. (681 *grms.*)	Query. No rupture with 9 lbs. (4082 *grms.*)
1 Chromic catgut, No. 3 (fine)	¾ lb. (340 *grms.*)	1½ lbs. (681 *grms.*) (aetatis 11 yrs.)
1 Silk, No. 2 (fine)	¾ lb. (340 *grms.*)	2 lbs. (907 *grms.*)
1 Silk, No. 6 (large)	1¼ lbs. (567 *grms.*)	3 lbs. (1361 *grms.*)
1 Silkworm-gut (ordinary size)	¾ lb. (340 *grms.*)	1½ lbs. (681 *grms.*)
Ditto, 6 strands (stay-knot)	3 lbs. (1361 *grms.*)	7 lbs. (3175 *grms.*)

The presence of atheroma in the vessel does not seem to make much difference. If, however, an atheromatous patch is included in the circle of ligature, the rupture appears first in it: the condition of calcification of arteries seems to be comparatively rare, we endeavoured to obtain a specimen of it for a long time without success.

To occlude, then, a human carotid with a ligature, and at the same time not to rupture the coats, it is necessary to apply a force intermediate between those given in the table, and as there is a fairly wide range, there should be no great difficulty in doing this. Of course it is not suggested that the operator should make use of a spring balance, but only that he should previously inform himself, through his muscular sense, of about the amount of force requisite: moreover, the surgeon has to assist him, the cessation of the pulsation in the aneurism and in the artery below the seat of the ligature, and the obstruction felt when the coats come into contact.

But here another and a serious difficulty presents itself; having with the first half of the reef knot occluded and yet not ruptured the vessel, to complete the knot it is necessary to relax the two ends of the ligature, and with them the tension on the knot, whereupon the blood pressure distends the loop, and when the knot is completed the ligature is found either not to occlude the vessel at all, or only partially to do so. Hitching the ends twice round, making what is called a surgeon's knot, does not meet the difficulty: the surgeon's knot holds no better than the reef knot. Putting a finger on the knot while it is being tied is also of no use: the knot is so sunk down

in the vessel that the finger cannot reach it; the vessel, too, is moveable and slips away. (See page 386 et seq.)

With a fine silk ligature tied so tight as to rupture the coats, the same difficulty does not arise, for the loop of the ligature lies down between the recurved intima and media, and the pressure of the blood does not distend it: the same is not true for stout silk, stout catgut, or kangaroo tendon, which will re-distend even if the coats are ruptured.

This probably explains what has happened in some cases of ligature the specimens from which are now in museums: whether the coats were ruptured or not, it would appear that the first hitch of the knot 'gave' before the second was 'home.' (See figures 208 and 209 and fig. 77, page 193.) We have frequently seen this happen when experimenting with large ligatures on dead arteries under pressure, even when the coats were ruptured.

The introduction, then, of animal ligatures has brought with it a new difficulty, and it is undoubtedly the case, as Savory has repeatedly pointed out, that the re-establishment of the circulation through the ligatured vessel is much more common nowadays than in the time of silk and rupture. Of the reality of this danger anyone can satisfy himself by practising on human arteries with water pressure, or on the arteries of the lower animals. Two instances have come under our own observation, in which from this cause an artery has not been occluded, and in which a second operation has been necessary: and it is probably the cause of the recurrence of circulation at the seat of ligature when it occurs, a condition which must not be confounded with a return of pulsation in the

FIG. 208. Ligature of External Iliac Artery. (Nat. size.)

From St Bart's. Hosp. Museum, spec. 1397. *See also Lawrence, Med. Times and Gaz. July* 2, 1853.

The ligature was applied for hemorrhage after a punctured wound in a man aged 55 years.

Source of hemorrhage not found; main trunk ligatured immediately above epigastric. Clot extends the whole length of external iliac and 1 inch below ligature. At the seat of ligature the coats are not injured and not quite in contact: probably the first hitch of the reef knot gave before the second hitch was completed. Patient died on the 5th day.

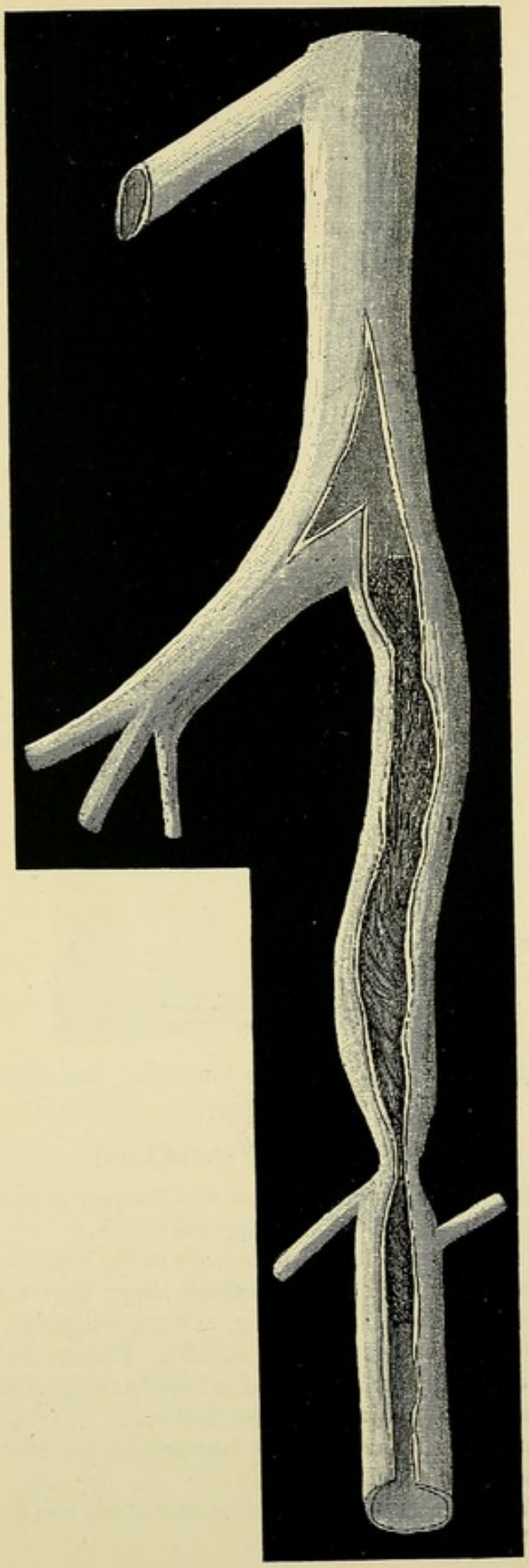


FIG. 209. Ligature of External Iliac for Inguinal Aneurism. (Nat. size.)

Guy's Hospital Museum 1519[1].

The drawing shews the common, external and internal iliac arteries, and below part of an aneurism involving the common femoral artery.

The patient was a man aged 39, under the care of Mr Bryant.

The external iliac was tied a short distance above the aneurism with a silk ligature. Slight pulsation in sac continuing, a second ligature was applied. All pulsation then ceased. Wound suppurated slightly. Patient died on 8th day.

The position which the ligatures occupied is readily recognized.

The wall of the artery is uninjured by the ligatures. The lumen of the vessel is not completely occluded: but the artery is impervious, the whole of the external iliac and the aneurism being filled with clot.

It seems probable that in each case the first half of the reef knot gave before the second hitch was pulled tight.

aneurismal sac from collateral circulation. The diagnosis can be made by pulsation being felt in the vessel at and below the site of ligature, and if there is constriction, by a thrill being felt there, and also by a loud murmur being audible with the stethoscope.

As stated elsewhere, the result of experimenting with various knots is to decide in favour of the stay knot. In the following table is given the force sufficient to occlude and that requisite to rupture the various arteries of man when tied with different ligatures with this knot.

The table is compiled from the results of numerous experiments.

Experiments on some of the arteries of man distended under normal pressure and tied with stay knots of two or three strands of kangaroo tendon, each about $\frac{1}{20}$th inch (1·3 mm.) wide.

Two kangaroo tendons.

Artery	Force which occludes		Force which ruptures	
	Momentarily	For 3 minutes	Superficial rupture	Through both tunics
Innominate	$2\frac{1}{2}$ lbs. (1135 *grms.*)	$3\frac{1}{2}$ lbs. (1588 *grms.*)	8 lbs. (3629 *grms.*)	10 lbs. (4536 *grms.*)
Common Carotid	1 to $1\frac{1}{4}$ lbs. (454—567 *grms.*)	$2\frac{1}{2}$ lbs. (1135 *grms.*)	4 lbs. (1814 *grms.*)	5 lbs. (2268 *grms.*)
Subclavian, 1st part	2 lbs. (907 *grms.*)	$2\frac{1}{2}$ lbs. (1135 *grms.*)	7 lbs. (3175 *grms.*)	Query. No further rupture with 10 lbs.* (4536 *grms.*)
Subclavian, 3rd part	$1\frac{1}{4}$ lbs. (567 *grms.*)	$1\frac{1}{2}$ lbs. (681 *grms.*)	5 lbs. (2268 *grms.*)	$5\frac{1}{2}$ lbs. (2495 *grms.*)
Common Iliac	$2\frac{1}{4}$ lbs. (1021 *grms.*)	3 lbs. (1361 *grms.*)	$6\frac{1}{2}$ lbs. (2949 *grms.*)	8 lbs. (3628 *grms.*)
External Iliac	$1\frac{1}{2}$ lbs. (681 *grms.*)	2 lbs. (907 *grms.*)	6 lbs. (2722 *grms.*)	8 lbs. (3628 *grms.*)
Superficial Femoral	$1\frac{3}{4}$ lbs. (794 *grms.*)	2 lbs. (907 *grms.*)	7 to 9 lbs. (3175—4082 *grms.*)	Query. No further rupture with 10 lbs.* (4536 *grms.*)

Three kangaroo tendons.

Innominate	Not occluded with less than 3 lbs. (1361 *grms.*)	3 lbs. (1361 *grms.*)	9 lbs. (4082 *grms.*)	10 lbs. (4536 *grms.*)
Common Iliac	Ditto	3 lbs. (1361 *grms.*)	$6\frac{1}{2}$ lbs. (2949 *grms.*)	8 lbs. (3628 *grms.*)

* In some experiments no rupture whatever.

It will be noticed what a wide range exists between the smallest force which is sufficient to occlude and the smallest force which produces rupture of the coats. It is clear that when the stay knot is employed all the surgeon has to do is to use a force between these two extremes; with a little practice on distended vessels he will have no difficulty in educating his muscular sense to this.

There is another advantage in using two ligatures, namely, that there is less danger of rupturing the coats. Thus, with a stay knot on a human carotid (with 9 feet of water pressure or 200 mm. Hg.) with one kangaroo tendon 3 lbs. (1361 grms.) ruptures, with two 5 lbs. (2268 grms.), and with three tendons, as much as 9 lbs. (4082 grms.) is necessary: the force necessary to occlude is also somewhat increased, as seen from the following table.

Ligature	To occlude	To rupture
One kangaroo tendon	$\frac{3}{4}$ lb.	3 lbs.
Two kangaroo tendons (stay knot)	1 lb.	5 lbs.
Three kangaroo tendons (stay knot)	$1\frac{1}{2}$ lbs.	9 lbs.

The force necessary to occlude the various arteries of the body, with different ligatures, will be seen in the tables. When the pressure is diminished the force necessary to occlude is diminished; thus while with 9 feet of water a force of $\frac{3}{4}$ lb. is necessary to occlude with one tendon, if the pressure is reduced to 3 feet a force of $\frac{1}{2}$ lb. is sufficient.

The force then which should be used is any that is intermediate between that which is sufficient to occlude and that which ruptures. The surgeon should know approximately what force he is using. With a stay knot—that is to say, with two ligatures pulled on at once—the force necessary to merely occlude puts a considerable strain on the index-fingers, and the force required to rupture is so much as in some cases to exceed 10 lbs. (4·5 kilos), which Lister says is the maximum force which anyone can apply under the conditions of ligation in continuity: the operator, therefore, with this knot, is not likely to err on the side of excess.

The inference to be drawn from the experiments related in this chapter appears to be that there should be no difficulty in occluding a great artery without injuring its coats and avoiding the risks both of hemorrhage and repatency.

Note to page 430. Lister has recently pointed out that the cause of the frequency of hemorrhage from the distal side of the ligature is the smallness of the clot there: this in its turn is due to the fact that it is undisturbed: he finds as the result of experiment that undisturbed aseptic clot does not propagate itself whereas disturbed clot (as that on the cardiac side is) causes further coagulation.

CHAPTER XIX.

LIGATION EXPERIMENTS.

Experiments on the Common Carotids of Sheep. Experiments on the Common Carotids of Asses. Experiments on the Common Carotids of Horses.

The following experiments were made to ascertain the changes produced in an artery by its ligation without rupture of its two inner coats.

In only two experiments (nos. 19 and 20) were the coats intended to be, or subsequently found, ruptured.

In the great majority of cases the vessels were found on examination to be impervious: but to this there were six exceptions.

In experiments 55 and 58 a minute opening allowed the blood to pass: in these two cases the animals were destroyed immediately after the operation, but it is almost

certain that if they had been preserved the arteries would have become temporarily, if not permanently, occluded.

In one of these cases a surgical, and in the other a slip knot, had been tried.

In experiments 5, 6, 16, and 35, the arteries were found when obtained several weeks after the operation, in some degree patent. In every case the intima was thickened for some distance on either side of the ligature, shewing that the artery had been closed with clot, and again become pervious: the knot used was in three instances a reef, and in one a surgical knot.

In all the other cases the arteries were occluded.

With the knots ordinarily used in surgery we were surprised to find that although the artery was almost always impervious, yet in nearly half the experiments the coats were not in apposition at the point of ligature, the occlusion being completed by connective tissue. This was the case in nine of the experiments on sheep; in five a reef knot had been used, and in four a surgical knot. It happened also thirteen times in the experiments on asses: in nine of these a reef knot, and in three a clove hitch had been used, and in one Senn's method had been followed except that the knots were tied in the reverse order.

In eleven of the experiments on horses the same thing was found: in five a reef knot, in three a clove hitch, in one a surgical, in one a slip knot and in one a stay knot had been used. In the case of the stay knot (Exp. 68) it was found at the operation that the ligatures were unsuitable and that the artery could not be occluded with them on account of their bulk, softness and slipperiness: when

the knot was completed a thrill could be felt in the vessel within the loop: notwithstanding this we left it to see what would happen: the stay knot did not yield, and the vessel eventually was found closed by connective tissue.

In the earlier experiments sheep were used; but as it did not seem safe to argue directly from the common carotids of sheep to the greater arteries of man, asses and horses were substituted in the later experiments.

The vessel tied was always the common carotid: the animals were anæsthetised with chloroform or ether.

On page 144 will be found the method by which the arteries were prepared for examination.

A short abstract of each experiment will now follow.

A. *Experiments on Sheep* (20 in number).

Kangaroo tendon: reef knot, six experiments. 7 to 73 days.
Catgut: surgical knot, ten experiments. 9½ hours to 44 days.
Silk: reef knot, four experiments. 21 to 42 days.

Exp. 1. Kangaroo-tendon ligature. Reef knot. Specimen obtained at the end of seven days. It was found that the coats were uninjured, the intima of opposite sides nearly, but not quite, in contact, the vessel being closed with clot which extended for some distance above and below ligature. Marked cellular invasion of clot, and proliferation of endothelium in neighbourhood of ligature. The kangaroo-tendon ligature is surrounded by cells, especially on its outer side: the absorption of the ligature has commenced on its surface, but only there, and there only to a very small extent. Surrounding the seat

of ligature is an oval mass of young connective tissue completely hiding the ligature.

Exp. 2. Kangaroo tendon. Reef knot. 10 days. Coats not ruptured. Lumen not quite obliterated but vessel closed by clot. Connective tissue forming in clot. Absorption of ligature has commenced at surface and there only: interior of ligature not invaded.

Exp. 3. Kangaroo tendon. Reef knot. 14 days. Coats not ruptured and opposite sides in contact. Connective tissue forming in clot. Intima proliferating. Ligature being absorbed at surface only except at one point, which is probably one of the free ends of the ligature; here the invasion stretches for a considerable distance in parallel lines in the length of the ligature. (Fig. 110, page 254.)

Exp. 4. Kangaroo tendon. Reef knot. 21 days. Coats not ruptured: opposite sides all but in contact. Lumen occluded. Clot is composed of strata of varying dates. Near ligature, and at points of attachment, connective tissue fibrillæ extend completely across the vessel. Intima greatly thickened: the ligature attacked only on surface; middle and inner coats also contain numerous plasma cells. The tissue surrounding ligature is fibrillated, in fact fibrous tissue with numerous capillaries. (See figs. 93, 94 and 111, pages 231 and 254).

Exp. 5. Kangaroo tendon. Reef knot. 58 days. Coats not ruptured. Lumen not occluded. Vessel now patent. For some distance on either side of ligature intima much thickened, indicating former presence of clot. The calibre of vessel greatly reduced throughout, but especially opposite the ligature where intima is thickest, and forms a diaphragm with a large opening. Absorption of ligature

has proceeded so far that about half has disappeared. The absorption has taken place, however, only from the surface which is frayed.

Exp. 6. Kangaroo tendon. Reef knot. 73 days. Coats not ruptured. Lumen not occluded. Vessel now pervious. Intima greatly thickened for some distance on either side of ligature, indicating former presence of clot. Calibre of vessel greatly reduced throughout; opposite ligature a distinct diaphragm is seen formed by hypertrophied intima. About three-quarters of the ligature absorbed, the remainder encapsuled: the absorption has taken place only from the surface. (Fig. 112, page 255.)

Exp. 7. Chromic catgut. Surgical knot. 9½ hours. Coats not ruptured: opposite sides in contact. Clot in artery on both sides of ligature. Catgut not yet invaded, but on its outer side is a great collection of cells, which must be leucocytes, since 9½ hours is too early for invasion by plasma cells. Permeation of inner coat and adjacent clot with invading leucocytes.

Exp. 8. Sulpho-chromic catgut (green). Surgical knot. 24 hours. Coats not ruptured: opposite sides in contact. Clot above and below ligature. Near the ligature well stained invading cells are seen in both the inner coats and the adjacent clot: those in the clot are not its white corpuscles, for these can be distinguished by their fainter staining and uniform distribution. Ligature unaltered; corpuscles in large numbers collected on its outer side.

Exp. 9. Chromic catgut. Surgical knot. 3 days. Coats not ruptured: opposite sides in contact. Proliferation of intima: cells from it invading clot and near ligature completely traversing clot; these are darkly stained.

Leucocytes of clot now indistinguishable. Spaces between clot and intima described in text seen, and the primary clot is at places separated from the artery wall by younger clot. Ligature shews villi of sheep's intestine. Mass of cells on outer side. (Fig. 105, page 249.)

Exp. 10. Chromic catgut. Surgical knot. 7 days. Coats not ruptured: opposite sides not quite in contact, but lumen obliterated by clot which extends above and below ligature. Ligature becoming invaded by cells which are proceeding along its fissures: in it the villi of the sheep's intestine are faintly seen. (Fig. 106, page 250.)

Exp. 11. Chromic catgut. Surgical knot. 9 days. Coats not ruptured: opposite sides not quite in contact. Lumen, clot and ligature as in preceding experiment. Intima much thickened.

Exp. 12. Chromic catgut. Surgical knot. 10 days. Walls not ruptured: opposite sides not quite in contact but lumen closed by clot. Clot in strata of different dates. Ligature—commencing invasion at surface and along fissures. Intima thickened close to and on each side of ligature. (See Figs. 89, 90 and 107, pages 223 and 251.)

Exp. 13. Chromic catgut. Surgical knot. 13 days. Coats not ruptured: opposite sides in contact. Lumen obliterated. Clot above and below. (Fig. 175, page 389.) Specimen in St Thomas's Hospital Museum, No. 414.

Exp. 14. Chromic catgut. Surgical knot. 14 days. Coats not ruptured: opposite sides in contact: lumen obliterated. Connective tissue formation further advanced in clot. Ligature splitting up.

Exp. 15. Chromic catgut. Surgical knot. 21 days.

Coats not ruptured: opposite sides in contact. Extensive organization of plastic material in clot near the seat of ligation. Ligature undergoing absorption.

Exp. 16. Chromic catgut. Surgical knot. 44 days. Coats not ruptured. Vessel now patent. Intima much thickened, indicating previous presence of clot: this thickening is greatest opposite the ligature, forming a diaphragm with a large opening. Ligature encapsuled and undergoing slow absorption: probably this was an exceptionally good piece of catgut. (Fig. 109, page 253.)

Exp. 17. Silk ligature. Reef knot. 21 days. Coats not ruptured: opposite sides not quite in contact: lumen closed by clot which extends above and below ligature. Silk becoming encapsuled: numerous cells on its outer side. Corpuscles have invaded only outer surface of silk.

Exp. 18. Silk ligature. Reef knot. 42 days. Coats not ruptured: opposite sides in contact. Lumen obliterated. Clot above and below ligature. Corpuscles invading surface of silk, especially at one spot. (Fig. 118, page 260.)

Exp. 19. Silk ligature. Reef knot. 21 days. Two inner coats purposely ruptured. Vessel completely divided: ends of artery retracted, ligature in space between. Connective tissue formation in clot, wound healed by first intention.

Exp. 20. Silk ligature. Reef knot. 42 days. Two inner coats purposely ruptured. Condition of artery similar to that in experiment 19, but changes more advanced.

B. *Experiments on Asses* (21 in number).

Catgut: clove hitch, one experiment. 42 days.

Kangaroo tendon: clove hitch, four experiments; reef knot, fourteen experiments; Senn's method, one experiment; ditto modified, one experiment. 21 to 85 days.

Exp. 21. Chromic catgut. Clove hitch. 42 days. Coats not ruptured: opposite sides not quite in contact: but lumen closed by clot. Organisation proceeding in clot in transverse layers extending across from side to side, forming a succession of complete diaphragms. Ligature completely absorbed, but its site can be recognized from a mass of corpuscles which have replaced it.

Exp. 22. Kangaroo tendon. Clove hitch. 21 days. Ligature was applied to common carotid $\frac{5}{16}$ths of an inch (7·5 mm.) from the bifurcation of anterior aorta, which is deeply placed in the mediastinum: on removal round seat of ligature was found a mass of young connective tissue through which a small sinus led down to the ligature, which lay in a tunnel of pus, which thus surrounded the artery: the ligature could not be examined, as what remained of it fell out on section. Coats not ruptured: opposite sides in contact. The vessel was completely occluded. Clot above and below ligature.

Exp. 23. Kangaroo tendon. Clove hitch. 42 days. Ligature was applied to common carotid $\frac{5}{8}$ths of an inch (15 mm.) from bifurcation of anterior aorta which lies deeply in the thorax. Coats uninjured. Walls in apposition. Clot above and below: firm occlusion: much external callus: some suppuration. Ligature partly ab-

sorbed: where absorbed, it is replaced by a mass of cells. (See fig. 58, page 147.)

Exp. 24. Kangaroo tendon. Clove hitch. 42 days. Coats not ruptured. Walls not in apposition. Extending across interior of vessel are diaphragms of connective tissue, some complete, some cribriform: in the latter some of the spaces are filled with recent clot. Ligature completely replaced by plasma cells; at least this was so in portions examined microscopically. Artery impervious.

Exp. 25. Kangaroo tendon. Clove hitch. 42 days. Result same as in preceding experiment.

Exp. 26. Kangaroo tendons. Senn's method: that is, two ligatures side by side each tied with a reef knot; the proximal being tied first. 42 days. Coats uninjured. Walls in apposition. Vessel completely occluded. Great length of clot. Ligature completely replaced by young connective tissue.

Exp. 27. Kangaroo tendons. Two ligatures side by side, but the distal ligature tied first (differing in this respect from Senn's method). Reef knot. 64 days. Coats uninjured. Walls not quite in apposition. Vessel occluded. Complete diaphragms stretch across interior of vessel and are continuous with intima. Some portions of the ligatures still remain unabsorbed. The clot is of various dates.

Exp. 28. Kangaroo tendon. Reef knot. 64 days. Coats uninjured. Opposite sides in contact. Ligature replaced by young connective tissue, except a shred or two. It is easy to recognize site of ligature. Considerable length of vessel occluded.

Exp. 29. Kangaroo tendon. Reef knot. 67 days. Coats uninjured. Opposite sides in contact. Ligature

completely absorbed, but its former site is readily recognized by a mass of cells which have replaced it. Vessel firmly occluded. (Fig. 115, page 257.)

Exp. 30. Kangaroo tendon. Reef knot. 67 days. Coats uninjured. Opposite sides not in contact. Loop of ligature completely absorbed: part of knot still remains. Vessel impervious.

Exp. 31. Kangaroo tendon. Reef knot. 67 days. Coats uninjured. Opposite sides not in contact. Ligature all but absorbed: site clearly recognizable. Vessel closed.

Exp. 32. Kangaroo tendon. Reef knot. 67 days. Coats uninjured. Opposite sides in contact. Clot extensively replaced by connective tissue continuous with, and in fact, intima. Ligature completely absorbed, and replaced by fusiform plasma cells. Vessel closed.

Exp. 33. Kangaroo tendon. Reef knot. 69 days. Coats uninjured. Opposite sides not quite in contact. Clot traversed in all directions at and on both sides of, and for some distance from, the ligature by a network of connective tissue, which springs from the intima on all sides. Remains of ligature distinguishable. Vessel closed.

Exp. 34. Kangaroo tendon. Reef knot. 75 days. Coats uninjured. Opposite sides not quite in contact. Extensive clot above and below ligature. Ligature almost completely absorbed, but shreds still visible. The artery is encircled by a band of fibrous tissue which has replaced the band of tendon. Vessel closed.

Exp. 35. Kangaroo tendon. Reef knot. 76 days. Coats uninjured. Opposite sides not quite in contact. The knot of the ligature is still visible. Vessel almost

completely occluded by diaphragm of thickened intima, in the centre of which is a small opening.

Exp. 36. Kangaroo tendon. Reef knot. 77 days. Coats uninjured. Opposite sides not quite in contact. Remains of ligature just recognizable. Lumen almost completely closed by diaphragm formed from the thickened intima at the level of the ligature: in this diaphragm is a small central opening. (See fig. 88, page 218.)

Exp. 37. Kangaroo tendon. Reef knot. 77 days. Coats uninjured. Opposite sides of vessel in contact. Clot for considerable distance above and below ligature. Nothing can be seen of the ligature, which is replaced in the sections examined by a crowd of cells. Vessel closed. (Figs. 113 and 115, pages 256 and 257.)

Exp. 38. Kangaroo tendon. Reef knot. 77 days. Coats uninjured. Opposite sides in contact. Ligature almost completely absorbed, but its site is marked by a mass of cells. Vessel closed.

Exp. 39. Kangaroo tendon. Reef knot. 79 days. Coats uninjured. Opposite sides not quite in apposition. For $\frac{3}{4}$ inch (18 mm.) at seat of ligature interior of artery filled by firm connective tissue which has replaced clot, and is continuous with the intima on all sides: above and below this the vessel is patent. Site of ligature occupied by cells, among which lie loose interlacing single filaments of the tendon. Vessel closed. (Fig. 114, page 257.)

Exp. 40. Kangaroo tendon. Reef knot. 81 days. Coats uninjured. Opposite sides not quite in contact. Remains of ligature still visible, but absorption is far advanced. Fibrous capsule round remnants of ligature. Vessel closed by connective tissue.

Exp. 41. Kangaroo tendon. Reef knot. 85 days. Coats uninjured. Opposite sides not quite in contact. At level of ligature a fibrous diaphragm, continuous with the inner coat, completely occludes the artery. Some remnants of ligature visible. Much external callus. Vessel closed.

C. *Experiments on Horses* (30 in number).

Mixed ligatures: reef knots, one experiment. 30 minutes.

Catgut: reef knot, two experiments; clove hitch, one experiment. 14 to 51 days.

Kangaroo tendon: reef knot, four experiments; clove hitch, two experiments; Senn's method, one experiment; two Carrick bends, one experiment; surgical knot, one experiment. 30 minutes to 165 days.

Reindeer tendon: surgical knot, one experiment; reef knot, one experiment. 15 minutes and 14 days.

Kangaroo tendon + two silkworm-guts: modified Senn, one experiment. 15 days.

Silk: slip knot, one experiment. 15 minutes.

Silk + catgut: clove hitch + reef knot, one experiment. 15 days.

STAY KNOTS (twelve experiments).

Six silkworm-guts. 30 minutes.

Nine silkworm-guts, applied in sets of three. 21 days.

Three kangaroo tendons. 14 days.

Two kangaroo tendons. 21 days.

Two kangaroo tendons. 24 hours.

Two floss silk ligatures. 14 days.

Two peritoneal ligatures. 14 days.

Two peritoneal ligatures. 55 days.

Two kangaroo tendons. 58 days.
Six silkworm-guts (size for salmon). 30 minutes.
Two chromic catgut. 36 days.
Two kangaroo tendons. 36 days.

Exp. 42. The carotid artery of a horse was exposed in its whole extent immediately before death. Commencing distally ligatures of silk, catgut and kangaroo tendon (two of each) were applied at intervals, and so as to rupture the coats; reef knots; the force used varied, and different degrees of rupture were thus obtained. Microscopic sections were prepared in the usual manner, and the exact extent of the injuries shewn. The degrees varied from a superficial abrasion of the intima to a complete rupture of the two inner coats. In one case a deep dent seems to have taken the place of the rupture.

Exp. 43. Catgut. Reef knot. 14 days. Coats uninjured. Opposite sides not in contact. Much suppuration. Large fusiform swelling around ligature: ligature being rapidly absorbed. Lumen closed by clot. Vessel impervious. (Fig. 108, page 252.)

Exp. 44. Catgut. Clove hitch. 28 days. Coats uninjured. Opposite sides not quite in contact. The ligature was placed just below a collateral branch. There is considerable clot in the carotid both above and below the ligature and also in the collateral branch; on the cardiac side the vessel is distended, on the distal side somewhat collapsed: in tying, the ligature did not run properly. Thrombus opposite ligature replaced by connective tissue. Ligature not yet absorbed. Artery impervious.

Exp. 45. Catgut. Reef knot. 51 days. Coats uninjured. Opposite sides not quite in contact. Fibrous

tissue (replacing clot) completely occludes the vessel. Ligature completely absorbed, not a trace of it left. Artery impervious. (See figs. 95 and 96, page 231.)

Exp. 46. Kangaroo tendon. Reef knot. ½ hour. Coats uninjured. Opposite sides in contact. Traces of clot between the folds into which the vessel is thrown by the ligature. Vessel impervious.

Exp. 47. Kangaroo tendon. Reef knot. 1 hour. Coats uninjured. Opposite sides in contact. Vessel impervious.

Exp. 48. Kangaroo tendon. Clove hitch. 9 days. Coats uninjured. Opposite sides not quite in contact. Ligature apparently unaltered to the naked eye. Extensive clot on both sides of ligature. Vessel impervious.

Exp. 49. Kangaroo tendon. Reef knot. 10½ days. Coats uninjured. Opposite sides not quite in contact. Extensive invasion of clot by plasma cells at seat of ligation. Ligature unaltered. Vessel impervious. (Fig. 174, page 389.)

Exp. 50. Kangaroo tendon. Clove hitch. 14 days. It is probable that the inner coat was ruptured. Opposite sides not in contact: the clove hitch would not run well on the soft wall of the artery, which gave before it. It could not be judged at the time whether the coats were ruptured or not. Considerable clot above and below. A diaphragm has formed across artery opposite ligature, composed partly of inner coat, partly of new formed tissue. Ligature unchanged. Vessel impervious. (See Plate II., fig. 3.)

Exp. 51. Two kangaroo tendons. Senn's knot: i.e. a reef knot on each ligature separately, the proximal

being tied first. 14 days. Coats uninjured. Opposite sides in contact. On careful examination it appears that the proximal ligature did not hold the coats in contact, but that the distal did, it having less pressure to contend with. Abundant clot above and below. Ligatures unaltered to naked eye Vessel impervious. (See Plate II., fig. 4.) Specimen in St Thomas's Hospital Museum.

Exp. 52. Two kangaroo tendons, each tied with a Carrick bend. 15 days. Proximal tied first. Coats uninjured. Opposite sides in contact. Ligatures apparently unaltered. Vessel impervious.

Exp. 53. Kangaroo tendon. Surgical knot. 131 days. Coats uninjured. Opposite sides in contact for 2 mm., as seen with a single lens. Wall of vessel in neighbourhood of ligature embedded in a fibrous mass which completely obstructs the artery. There is a small portion of the ligature still unabsorbed. (See fig. 116, page 258.) Vessel impervious. (See Plate II., fig. 1.)

Exp. 54. Kangaroo tendon. Reef knot. 165 days. Coats uninjured. Opposite sides not quite in contact (as seen with a single lens). Wall of vessel in neighbourhood of ligature embedded in a fibrous mass which completely obstructs the vessel. Beyond this fibrous mass is a more recent clot. Ligature absorbed, but site marked by collection of cells. Vessel impervious. (See Plate II., fig. 2.)

Exp. 55. Reindeer tendon. Surgical knot. 15 minutes. Coats uninjured. Opposite sides not quite in contact.

Exp. 56. Reindeer tendon. Reef knot. 14 days. Coats uninjured. Opposite sides in contact. At first it was attempted to tie the tendon with a clove hitch, but as this

would not run, a reef knot was used instead. Clot above and below: it is being replaced by young connective tissue. Ligature being absorbed somewhat rapidly: it is not homogeneous, like kangaroo tendon. Vessel impervious. (Fig. 117, page 259.)

Exp. 57. One kangaroo tendon and two silkworm-gut ligatures. Reef knots. 15 days. These were applied, first the kangaroo tendon, and secondly a gut on each side, the idea being to diminish the steepness of the groove in which the tendon lay: in this the method failed as the gut ligatures slipt down into contact with the tendon. Coats unruptured. Opposite sides not in contact. Abundant clot, especially on cardiac side. A white band of young connective tissue may be seen with the naked eye stretching across clot from side to side at seat of ligation. Ligatures unaltered. Vessel impervious.

Exp. 58. Silk ligature. Slip knot. 15 minutes. A slip knot was tied on the vessel and a hitch put on above the knot. Coats uninjured. Opposite sides not quite in contact.

Exp. 59. A silk ligature was applied with a clove hitch: it did not run properly on the soft wall of the artery: a catgut ligature was therefore applied distally with a reef knot. 15 days. Coats uninjured. Vessel found closed by the catgut ligature.

The coats were in apposition within the catgut ligature but not within the silk.

Exp. 60. Six silkworm-gut ligatures. 30 minutes. The ligatures were tied by the authors' stay knot, that is to say, each ligature was passed round the vessel and

the first half of a reef knot tied on it; it was then drawn tight enough to be in contact with the vessel wall all round and yet not to compress it in the least: the ligatures having been thus arranged side by side, all the ends on one side were taken together as one end and the same on the other side; the two ends thus formed were drawn upon so as to simply occlude the vessel; the ends were then let go to see if the hitches would give to the pressure of the blood and allow it to flow again along the vessel; this they did not do although allowed several minutes: the knot was then finished by treating all the ends on each side as a single end and completing the reef knot, making in fact a second half of the knot common to all the six ligatures: they did not lie side by side, but came together at the bottom of the groove in the artery, making a beautiful round ligature of the size of No. 6 catgut. On examining the vessel it was found that the coats were uninjured and the opposite sides in contact. The only objection to this method is that it takes a long time to arrange the first halves of the reef knots side by side, the silkworm gut being stiff and troublesome to handle: in all other respects the knot is perfect. Extensive clot above and below. Artery impervious.

Exp. 61. Three ligatures, each composed of three strands of silkworm gut. The first half of a reef was tied on each ligature in the same way: the nine ends on either side were then treated as one, and drawn upon to occlude the vessel: the knot was completed as described under figure 182, page 398. As in Exp. 60, the ligatures ran together in the groove to form a circular ligature. 21 days. Coats uninjured. Opposite sides in contact.

Extensive clot above and below. Artery impervious. (See Plate I., fig. 5, and fig. 122, page 262.)

Exp. 62. Three kangaroo-tendon ligatures. Stay knot as described in text: in tying this knot one of the ends got crossed over and was pulled in the wrong direction; this was discovered and corrected: to avoid such a mistake it would be well to clamp together the ends on each side with forceps. 14 days. Coats uninjured. Opposite sides in contact. Much clot above and below. Artery impervious. (See Plate I., fig. 4.)

Exp. 63. Two kangaroo tendons. Stay knot. 21 days. Much suppuration. Coats uninjured. Opposite sides in contact. Much clot above and below. Artery impervious. (See Plate I., fig. 3.)

Exp. 64. Two kangaroo tendons. Stay knot. 24 hours. Coats uninjured. Opposite sides in contact. Clot above and below. Artery impervious. (See Plate I., fig. 1.)

Exp. 65. Two floss-silk ligatures. Stay knot. 14 days. Coats uninjured. Opposite sides in contact. Clot above and below. Artery impervious. (See Plate I., fig. 2, and figs. 119, 120 and 121, page 261.)

Exp. 66. Two peritoneal ligatures as described in text. Stay knot. 14 days. Coats uninjured. Opposite sides in contact. Clot above and below, but chiefly above. Some suppuration. To naked eye ligatures unaltered. Artery impervious. (See Plate III., fig. 2, and fig. 129, page 268.)

Exp. 67. Two peritoneal ligatures as described in text. Stay knot. 55 days. Coats uninjured. Opposite sides in contact. A little altered clot above and below ligature.

Coats in neighbourhood of ligature embedded in a fibrous mass. Wound healed by first intention. Ligatures not much altered to naked eye. Artery impervious. Artery cardiac side (collapsed after death) outside diameter 9 mm.; opposite carotid (collapsed) outside diameter 14 mm. (See Plate III., fig. 6, and fig. 130, page 269.)

Exp. 68. Two large soft kangaroo tendons. Stay knot. Condition of ligatures owing to soaking in glycerine for too long a time prevented proper manipulation, and when knot was completed a thrill beyond could be felt, the vessel not being completely occluded. 58 days. Healed by first intention. Coats uninjured. Clot above and below: short on proximal side. Artery impervious, being blocked by connective tissue. (See Plate III., fig. 5, and page 446.)

Exp. 69. Two chromic catgut ligatures (No. 8 Mac Farlan). Stay knot. 36 days. Healed by first intention. Coats uninjured. Opposite sides in contact, and adherent. Clot above and below. Artery impervious. (See Plate III., fig. 3.)

Exp. 70. Two kangaroo tendons, each $\frac{1}{20}$th inch (1·3 mm.) in diameter. Stay knot. 36 days. Healed by first intention. Coats uninjured. Opposite sides in contact. Clot above and below. Artery impervious. Second part of knot was tied loosely before first half was drawn tight: in this way the stay knot can be completed rapidly. (See Plate III., fig. 4.)

Exp. 71. Six silkworm-gut ligatures. The largest size, called salmon gut. Stay knot. Completed as one reef knot. Horse destroyed at once. Artery removed and distended with glycerine jelly. Coats uninjured. Opposite

sides in contact. Artery impervious. (See Plate III., fig. 1.)

In none of these experiments did secondary hemorrhage occur: all the wounds in the sheep healed without suppuration: many of those in asses did so also: in some asses however the superficial parts suppurated, and in two experiments (nos. 22 and 23) a sinus leading down to the artery persisted. In the case of the horses, in nearly every wound a certain amount of suppuration occurred: it is very difficult to asepticize the skin of a horse in consequence of the size and depth of the hair follicles: no doubt, too, animals differ as to their liability to suppuration.

In all cases the steam spray was used; the skin of the animal was shaved and cleansed with soap and ether: the antiseptics used were carbolic acid (1—20) and mercuric perchloride (1—1000): the wounds were irrigated; and in some instances dusted with boracic acid powder; finally they were stitched up with stout catgut, no drainage tube being inserted. In all the sheep and in some of the other animals a dressing of gauze saturated with collodion was applied: the horses seemed to do best without any dressing, the wound being merely washed with carbolic twice a day.

With respect to the force used in tying the ligatures, the first hitches of the reef and surgical knots were pulled on until an obstruction was felt by the coats coming into contact; at the same time the pulsation in the facial ceased; the knot was then completed: a few minutes later, on examining the facial or the vessel beyond the

ligature a feeble but distinct pulsation could sometimes be felt: at the time we attributed the return of pulsation in the facial to the collateral circulation.

Although we have no doubt that in every case the vessel was occluded by the tightening of the first hitches (for we took great pains to make sure of this), yet in some cases we subsequently found that the coats were not in apposition: the explanation of this is given on page 388: the experiment mentioned on that page is not included amongst those abstracted in the present chapter.

From these experiments it is clear that arteries can be permanently occluded without the rupture of any of their coats: to effect this with certainty it is essential to employ a suitable ligature, a suitable knot tied with appropriate force, and to preserve the strictest asepticity.

CHAPTER XX.

CONDUCT OF THE OPERATION AND FATE OF THE PATIENT.

Results in man of the rupturing and non-rupturing operations: experience of Hunter, Scarpa, Jameson, Barwell and Wyeth: recent cases: opinions of Senn and Erichsen: repatency: cases of Travers and Roberts: gangrene: aneurism at seat of ligature: case of Warner.

Ligation of the greater arteries: first part of subclavian: Sédillot's incision: details of the operation for the first part of the right and left subclavians, the innominate, the abdominal aorta, and the iliacs.

Plate shewing immediate effect on arteries of non-rupturing ligation.

Digital compression: temporary ligation: ligatural compression: Esmarch's bandage: galvano-puncture: Wooldridge's discovery.

Essentials of the operation: times of discharge of rupturing and non-rupturing septic ligatures: cases of Lynn, Butcher, Rivington, Paget, Holden, and Spence: complete severance of artery by ligature: case of Bryant: transfusion and infusion.

We have now seen that to occlude an artery in continuity with a ligature it is not necessary to rupture the coats, and further that there is grave danger in so doing.

We have also noted the sources of danger and failure, and from these have endeavoured to infer the best method of operating: it only remains to enquire what results have attended these methods in such cases as they have already been applied in, and to consider what further progress may reasonably be hoped for in the future.

Our own experiments with rupture have been very few, but numerous experiments in this direction have been made at various times throughout this century, and the effects of this procedure in animals have been thoroughly worked out by many observers. The rupture of the coats, too, being the usual practice in surgery, operations which have been performed on man may be regarded as experiments with rupture. The result of these operations on the larger arteries has, as we have seen in Chapter I., been most unsatisfactory. But such experiments are not necessary to our argument, for if it be shewn that there is no advantage in rupturing the coats, the cardinal principle of inflicting the least amount of injury which suffices to cure the disease will apply and decide the question.

The method of operating without rupture is also by no means new, and the results already obtained in animals and man have been very good.

Hunter's cases have been already given. He appears always to have intended not to injure the artery. Silk ligatures were used and the ends brought out of the wound. Four out of the five cases recovered. The unsuccessful case was one in which he plugged the wound from the bottom and to this he attributed, and probably rightly, the fatal hemorrhage.

The results of operations by Scarpa's method have been referred to on page 355.

Professor Jameson of Baltimore "more than fifty years "ago shewed that the buckskin ligatures which he em- "ployed obliterated the arteries without cutting their "inner and middle coats, and without destroying their "continuity. Hence he opposed all indissoluble ligatures "of whatever material; he declared it to be not only "unnecessary but highly hazardous to cut the inner and "middle coats of arteries." For a "series of years he "employed the animal ligature in an extensive surgical "practice: he tied the carotid, the iliac and the femoral "and other arteries with buckskin, and in no instance had "secondary hemorrhage. He states that he has never "seen anything of his ligatures, and his wounds generally "healed by first intention. His observations and ex- "periments on sheep, dogs, and other animals shewed "that the ligature would be dissolved."

Barwell, with his ox aorta ligature, has been, as is well known, very successful. In his article on Aneurism in Ashhurst's *Encyclopædia of Surgery* (1883), he gives a list of nineteen ligations of great arteries with this ligature. In none of these did hemorrhage occur, but in one the vessel was subsequently found patent. (See Figure 104, page 248.)

Wyeth says, "the tension of the ligature to such a "degree as to divide the inner or middle coat, or both, is "unnecessary. I have tied arteries (carotid and subcla- "vian) in human beings, and in horses and dogs, and have "specimens which demonstrate successful occlusion of the "vessel without division of either of the 3 tunics. Scarpa

"advanced this idea years ago, but surgeons generally "have decried it. None the less is it true, and I am "fully convinced by experience that it is safer than the "division of one or two coats of a vessel by tightly "drawing a narrow cutting ligature around an artery."

The cases of ligation in continuity in man which have come under our own observation, in which it has been the intention of the surgeon not to rupture the coats, are thirteen in number. Two were of the common femoral and the remaining eleven of the superficial femoral. In no case did hemorrhage occur, but in two of the superficial femoral ligations (cases which have been already referred to on page 387) there was return of pulsation due to patency of the artery at the seat of ligation, necessitating the religature of the artery.

The operations were by Clutton, Croft, Horsley, Keetley, Lunn, MacCormac, Mackellar, Pitts and one of us.

The ligatures employed were silk, catgut, or kangaroo tendon. The knots used were the reef-knot, the surgical-knot, the clove-hitch, and in two instances, Senn's method with catgut.

As far as we know there is no case on record in which a surgeon has ligatured with an absorbable ligature one of the arteries usually tied and with the intention of not rupturing the coats in which hemorrhage has occurred.

In no case in man has a stay-knot been employed for ligation in continuity, but in one case of amputation it has been used for the femoral, which was much diseased, and all went well (Fig. 55, page 89). We have frequently tried the knot on the large common carotid of the horse and it

has behaved perfectly: no rupture of coats—no hemorrhage—no repatency. (See Chapter XIX.)

Senn in his "Cicatrisation in blood-vessels after ligature," says, "I am firmly convinced that in many of my "experiments [on sheep] the internal tunics of the arteries "remained intact after ligation, and yet cicatrisation pro- "gressed in a satisfactory manner. Hence it is no longer "necessary to tie the ligature so firmly as to crush the "tunics of the vessel. All that is necessary is to tie with "sufficient force to approximate the inner surfaces of the "intima."

The opinion expressed in the latest (1888) edition of Erichsen's Surgery (revised by Beck) is in favour of not rupturing. "The probability is that in all arteries of the "size of the superficial femoral and below it, it matters "little, as far as safety is concerned, whether the coats are "divided or not, provided the wound follows an aseptic "course and unites by the first intention.....In larger "arteries, however, secondary hemorrhage has occurred "with great frequency after ligature with division of the "coats, and in some, such as the first part of the sub- "clavian, the innominate, and the aorta, with such con- "stancy that the only hope of success seems to be in not "injuring the arterial wall."

The arteries now usually tied are the common carotid, the third part of the subclavian, the external iliac, the common and superficial femorals; as far as they are concerned the danger of hemorrhage would, with asepsis and non-rupture, seem to be a thing of the past. There remain the dangers of repatency and of gangrene.

As to repatency the cases already quoted shew that it is not confined to those in which the coats have not been ruptured (pages 207 and 216).

In the cases in which repatency has followed the use of carbolic catgut, its cause might have lain either in the ligature or the knot. Travers had repatency and return of pulsation in a popliteal aneurism in a case in which he ruptured the coats with a silk ligature which he removed at the end of twenty-seven hours: but Roberts of Carnarvon three years later reported a case in which a popliteal aneurism was cured by a temporary ligature which was left on the superficial femoral for only twenty-four hours. But it would seem that with a suitable ligature and a stay-knot there need be no fear of repatency occurring.

In gangrene there are generally two factors at work, anæmia and sepsis; anæmia is, in some degree a necessary consequence of treatment by ligature, and of itself is able to cause gangrene; but the risk to the distant anæmic parts is greatly increased by sepsis in the wound; happily, however, this can be avoided. It has often been stated that a wound of the main vein necessitating its ligature during the operation greatly increases the danger of gangrene, but the evidence for this is not very strong. In a case under the care of one of us it was necessary to divide both the common femoral artery and vein in order to remove a sarcoma from behind them: no evil consequences followed. A case in which an aneurism of the superficial femoral and a length of the corresponding vein were excised without evil consequences has been referred to on page 341.

Septic phlebitis would, no doubt, increase the danger of

gangrene, and indeed, to life, but on account of the sepsis rather than of the thrombosis.

The rare accident of secondary aneurism at the seat of ligature has been already referred to (page 237).

It is fair to infer that this sequela would be more likely to occur in those cases in which the coats have been ruptured than in those in which the ligature has been applied so as to act as a support to the arterial wall.

In the following case a secondary aneurism occurred no less than three times after the ligation of the brachial artery: no doubt a silk ligature was used and the coats ruptured. It is from *Cases in Surgery* by Joseph Warner, Surgeon to Guy's Hospital (1754).

"In a few days after the operation [amputation of the "arm] the humeral artery became so dilated above the "ligature as to endanger its bursting. Upon this account "it was judged necessary to perform the operation for "aneurism, which was done and the vessel was secured by "ligature above the upper extremity of its distended "coats. After this operation everything went on seem-"ingly well for some time when suddenly the artery "appeared again dilated and was in danger of bursting "above the second ligature. These circumstances made "it necessary to repeat the operation for aneurism, and "from this time everything went on successfully till the "stump was on the point of healing, when quite unex-"pectedly the artery appeared a third time diseased in "the same manner as before for which reason a third "operation was performed. The last operation was near "the axilla. The patient continued well from this time "without any relapse."

Besides the arteries now usually tied, it is very desirable that surgeons should be in a position to undertake with fair prospect of success the ligation of the greater arteries.

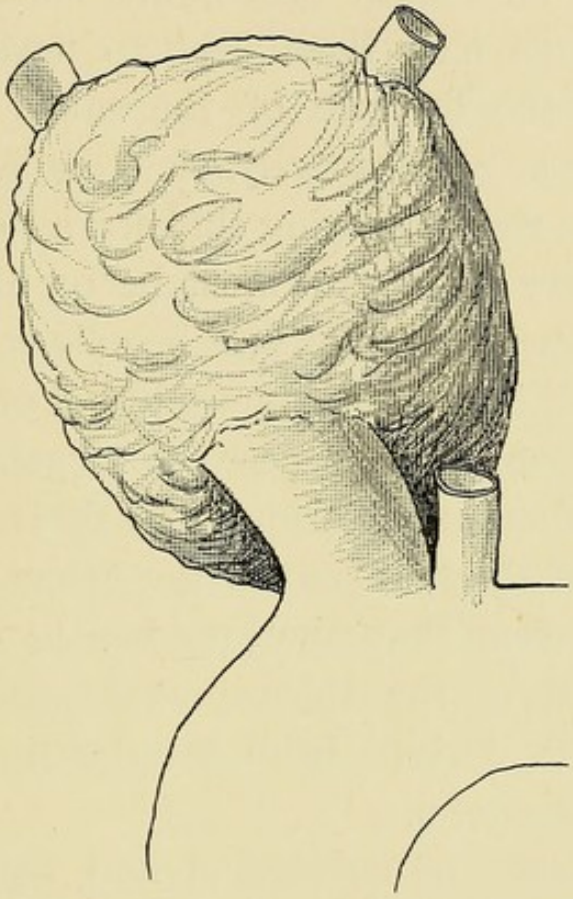


FIG. 210. Aneurism of the Innominate Artery. (Nat. size.)

From St Bartholomew's Museum, Spec. 1507. *See also Lawrence, Med. Chi. Trans. Vol. VI. p.* 227 (1815).

From a girl aged 20. The disease involved the whole circumference of the vessel. The first three-quarters of an inch of the artery was free from disease. Patient died of dyspnœa from pressure on the trachea.

Specimen No. 1506 in the same Museum shews an innominate aneurism the size of an orange, from a man aged 39 years.

Cases have been recorded by Burney, Wharton and Ashhurst in which aneurisms supposed to have been confined to the innominate have been apparently cured by

the distal ligature of the carotid and third part of the subclavian arteries.

The innominate aneurism shewn in Figure 210 could possibly have been cured by distal ligature.

There are on record also numerous cases in which aortic aneurisms have been said to have been greatly benefitted by the ligature of the same arteries either simultaneously or consecutively, or of one carotid alone.

If Scarpa is right in saying that for an aneurism to be cured the artery at its site must be occluded, it follows that aortic aneurisms can at best be only relieved.

It is difficult to see how the occlusion of the third part of the subclavian in these cases does good, for the collateral circulation for the arm will be derived mainly from the branches of the first part of the artery; be that as it may, it seems clear that whatever benefit may be gained by the ligation of the third part of the vessel, much more would be derived from the ligation of the first part, if only successful.

In many cases of subclavian and axillary aneurism (Fig. 157, page 322), the best operation by ligation (if successful) would be to tie the first part of the subclavian. In St Bartholomew's Hospital Museum are two other specimens, one of a subclavio-axillary aneurism on the right side (No. 1513), and the other of a subclavian aneurism on the left side (No. 1514): in each of these the first part of the subclavian appears to be quite healthy. Ligation of the first part of the right subclavian would be preferable to that of the innominate, for the cerebral circulation would not be materially interfered with; it would also be far better than the treatment suggested by Fergusson for

desperate cases—amputation of the arm and distal ligature. As far as the circulation in the arm is concerned, it would seem that it would be as good, after the ligation of the first part of the subclavian, as after that of the third part.

It must be admitted that the operation is difficult, but it was not on this account that Erichsen was led to the conclusion that it should be banished from surgery: indeed, surgeons have never given up an operation merely because it was difficult.

Mitchell Banks has performed the operation and gives a by no means encouraging account of it.

A man aged 50 had an aneurism of the third part of the right subclavian: the innominate and common carotid were ligatured simultaneously: pulsation returned, and on the 67th day after the ligature of the innominate, Banks tied the first part of the subclavian; "as to go down "through the old cicatrix in search of the innominate, "with a view of tying it a second time, seemed very "impracticable.....very slowly and after an infinity of "anxious picking and teasing, the artery was exposed "about half an inch from the aneurism," and secured by two catgut ligatures. "The knots were drawn very "gently with the intention of merely closing the artery "and not of injuring any of its coats. The aneurism at "once became still. A very few lines have sufficed to "describe this operation, but it took more than an hour to "perform, while the difficulty, danger and anxiety that "attended it are almost impossible to describe: owing to "the fact that the parts had been already interfered with, "there was a great deal of thickened and cicatricial tissue

"present.....Compared with this performance, tying the "innominate was a mere surgical amusement." Hemorrhage occurred from a sinus on the 31st day, and after frequent recurrence proved fatal on the 37th day.

The difficulties in this case must have been greatly increased by the previous operation, but however great they were, Banks overcame them and the patient survived 37 days, when he died of hemorrhage. If, therefore, the danger of hemorrhage can be surmounted, the operation may be considered practicable.

It would appear to us that the best mode of reaching the artery is between the sternal and clavicular attachments of the sterno-mastoid muscle. This method has been recommended by Sédillot. It is shewn in Figures 214 and 215.

Sédillot says that the operation affords easy access not only to the first part of the subclavian and its branches, such as the inferior thyroid and vertebral, but also to the primitive carotid and innominate. For the two latter vessels, however, the best operation is from the median line.

But if it be desired to ligate simultaneously the lower part of the right common carotid and the first part of the right subclavian it is easy to reach them both through a single incision by passing between the two origins of the sterno-mastoid, as may be seen on the cadaver.

When this method is used for tying both vessels, it is advisable first to arrange the ligatures with their stay-knots loosely around both vessels before constricting either, and to tie the subclavian first. If the carotid be tied first, the pressure in the subclavian would

be very high and the force required greater: the same no doubt would happen with the carotid, but then it is a smaller vessel and therefore easier to occlude.

We will now consider the steps for the ligation of the first part of the right subclavian, of the innominate and of the first part of the left subclavian.

Procedure for the ligation of the first part of the right subclavian.

A firm pillow should be placed beneath the shoulders: the head allowed to drop backwards, the face turned to the opposite side and the right arm pulled down so as to lower the clavicle. The operator should stand on the patient's right. An oblique incision four inches (10 cm.) long, is made parallel to the outer border of the tendon of the sterno-mastoid, and terminating below at the inner end of the clavicle. The incision is carried through the subcutaneous tissue and the platysma. The fascia covering the sterno-mastoid is divided and the two parts of the muscle separated by the ivory handle of a scalpel for the whole extent of the wound. Beneath the sterno-mastoid the anterior jugular vein may be met with. The sterno-hyoid is then exposed, and along its outer border is seen through the fascia the internal jugular vein (Fig. 214). To avoid the vein the deep fascia is divided over the sterno-hyoid: the muscle is then reflected inwards and held under the inner retractor (Fig. 215). On pressing the outer border of the sterno-hyoid muscle inwards the vagus nerve is dimly seen along the inner border of the internal jugular vein, which on this side of the neck and at this level is some distance

external to the common carotid artery. Coming from beneath the sterno-hyoid is often seen a moderate-sized vein (the inferior thyroid) passing obliquely downwards and outwards to join the right innominate vein. The right innominate vein is behind the clavicle and is not seen. The thyroid vein is tied in two places and divided between. Then a little connective tissue is picked through with two pairs of forceps, or better divided with the knife, immediately over the artery, keeping rather to its inner side to avoid the pleura: the first part of the subclavian is thus reached. No branches need be seen nor any nerve nor any vein except those mentioned. In our operations on the cadaver the vertebral vein was never seen. There is a full inch (25 mm.) of artery without branches, and round the mid-point of this portion the ligature should be placed. The mid-point of the portion of artery exposed is one inch (25 mm.) from the surface and three-quarters of an inch (18 mm.) above the upper border of the clavicle. With the patient in the position described the artery is therefore well above the clavicle and easy of access. The artery should be fully exposed and the point of the aneurism needle should in passing be kept close to it, the needle being passed from below upwards. No attempt must be made to pass the aneurism needle until the sheath has been cut completely through on the front of the artery, so that the point of the needle may pass close to the vessel where it can travel readily.

Anderson and Makins have pointed out that a distinct lymph space separates the inner surface of the sheath from the outer surface of the tunica adventitia: it is this

interval that must be opened so that the aneurism needle may freely travel round the artery. The space has the function of allowing the movement of the vessel with the minimum of friction, it is analogous indeed to a synovial sac or to the pleura.

It is clear from this that there should be no difficulty in distinguishing where the outer coat ends and the sheath begins. This is seen in some of the figures of arteries in Chapter III.

By this way of passing the aneurism needle the danger of injuring the pleura, of including the recurrent laryngeal nerve, or one of the filaments of the sympathetic, is avoided. The needle must be passed from below and without, to avoid the pleura and large veins. In the living subject the distension of the veins would, to some extent, increase the difficulty of the operation, but the pulsation of the artery would materially aid the surgeon in exposing it. The wound recommended is small in extent, is not complicated by the division of any muscular fibres, with proper precautions it would not require the use of a drainage tube and should certainly heal by first intention.

Procedure for the ligation of the innominate artery.

(See Figures 216 and 217.)

The shoulders should be raised on a pillow and the head thrown back. The operator should stand on the patient's left; but some will prefer to commence on the patient's right. A median incision is made from the cricoid cartilage to a point half an inch (12 mm.) below the upper border of the manubrium. The two layers of

the deep cervical fascia are then divided, and in doing this the transverse branch between the two anterior jugular veins may or may not be seen. When the second layer of fascia has been divided the adjoining margins of the sterno-hyoids above and of the sterno-thyroids below are seen and separated (Fig. 211). A large inferior thyroid vein comes into view embedded in the connective tissue in front of the trachea. This vein may extend from the thyroid isthmus across the artery and open into the right innominate vein. It must be divided between two ligatures. By pulling with the retractor on the right side of the wound, the right common carotid artery can be dimly seen or felt through its fascia, and on following this down the commencement of the right subclavian artery can also be distinguished. There is no need to open the sheath of the carotid. The bifurcation of the innominate in this position of the head is quite three-quarters of an inch (18 mm.) from the middle of the episternal notch and quite half an inch (12 mm.) vertically above this.

The innominate lies higher and more obliquely than seems generally to be thought. Where it emerges from behind the sternum its left border is exactly behind the mid-point of the interclavicular notch in the upper border of the sternum. The bifurcation is directly behind the superior angle of the inner end of the clavicle. (See Fig. 211.) The sheath is carefully opened on the anterior surface about half an inch (12 mm.) from the bifurcation, and the aneurism needle is passed from right to left to avoid wounding the pleura and large veins. In this way, unless the patient is fat and the neck short, the innominate can be tied without difficulty in the neck. The

operation does not involve the division of any muscular fibres, the retracted muscles after the ligation would fall again into their place, and the wound should heal by first intention; in many cases probably a drainage tube could be dispensed with.

Procedure for the ligation of the first part of the left subclavian artery.

This part of the artery can be reached in three ways:

(1) from the middle line:

(2) between the two origins of the sterno-mastoid by passing between the adjoining margins of the internal jugular vein and sterno-hyoid muscle, the former being retracted outwards and the latter inwards as already described in the procedure for exposing the first part of the right subclavian artery,

(3) between the origins of the sterno-mastoid by passing external to the jugular vein, between it and the inner border of the scalenus anticus.

By the two first methods the artery will be reached internal to its branches, by the third external to them and just as it passes underneath the scalenus. By the second method the artery is sometimes exposed at the origin of some of the branches, as these commonly come off earlier on the left than on the right side.

First method. (Fig. 218.) With the shoulders raised and the head thrown back there is quite an inch (25 mm.) of the artery situated in the neck. The operator should stand on the patient's right. A median incision as described for the ligation of the innominate is made, and after the cervical fascia has been divided the left border

of the wound, with the left carotid (which can be felt), is retracted outwards. The finger must then feel for the subclavian: the connective tissue between the carotid and trachea is next divided. The exact position of the artery will be found to be behind, and slightly external to the common carotid. (See Figs. 212 and 213.) The artery lies deep and the chief danger would be of injury to the pleura.

This operation is very grave, but might justifiably be attempted under certain conditions.

Second method. (Fig. 219.) The shoulders should be raised, the head thrown back and the face turned towards the opposite side. The artery is reached between the two origins of the sterno-mastoid. When they are retracted the sterno-hyoid muscle and the jugular vein are seen beneath the fascia. This is divided over the sterno-hyoid and the muscle retracted inwards and the vein outwards. Under the inner retractor too is taken the carotid artery in its sheath. The first part of the subclavian is here about 1·2 inch (30 mm.) deep. The steps of the operation are the same as on the right side of the neck, the main difference between the two sides being that on the left the jugular vein is nearer the median line and partly in front of the carotid artery. (Fig. 212, p. 489.)

Third method. (Fig. 220.) The difference between this and the second method is that in this the jugular vein is retracted inwards, and the artery exposed external to it. The part of the artery thus tied will be at the inner border of the scalenus anticus and external to the branches. The second part of the artery too can be reached by this incision.

Care must be taken to avoid the phrenic nerve, which on this side passes in front of the first part of the artery. The thoracic duct too issues from behind the carotid and arches outwards above the subclavian and in front of the scalenus anticus muscle and phrenic nerve to open into the subclavian vein just external to its union with the internal jugular. It must, of course, be avoided.

It would appear that the effect of the ligature of the subclavian here would not differ much from that of the ligature of the third part of the artery—a far easier operation. In the case, however, of an axillary aneurism so high as to prevent the ligature of the third part of the subclavian, this method of ligating the first or second part of the artery would not present any insurmountable difficulty.

It will be seen from the drawings that the seat of these operations is entirely in the neck, there is no necessity for the surgeon to enter the mediastinum: the large veins must be carefully avoided; the main danger is wounding the pleura, and this can be obviated by the surgeon going straight down to the artery and performing no manipulation outside the region of the artery.

There is no excessive danger in a deep dissection at the root of the neck: operations for the removal of diseased glands and enlargements of the thyroid body do as a rule very well: the disturbance of parts in these operations is much more extensive than in the ligation of a large artery: the only cause that deters the surgeon from undertaking the latter is the fear of secondary hemorrhage.

In performing the operation it is advisable to bring the operating table close to a window, so that the light falls on to the patient's face and into the wound. The surgeon should stand on the right side of the patient (except perhaps in the case of the innominate and the second and third methods for the left subclavian), and he should be provided with a small electric lamp, which can be worn on the forehead, so as to light up the deeper parts of the wound. A pillow should be placed under the shoulders and the patient's head thrown back, that is to say, if this throws no undue strain on the aneurism and does not interfere with respiration.

Figures 211 to 220.

The drawings are intended to shew the relative positions and the steps for the ligation of the three great arteries at the root of the neck, namely the innominate and the first part of the right and left subclavians. Figure 211 is in outline and gives the position of the structures as seen from before. Figures 212 and 213 are transverse sections.

The other figures shew the dissections to expose the arteries. The drawings were made from numerous operations on the cadaver (male adults) and the study of ordinary dissections and transverse sections: the variations found were comparatively slight, and the figures practically correspond though taken from different subjects. In every case the shoulders are supposed to be raised on a firm pillow and the head to be thrown back. The figures are life-size and to scale, and the arteries are represented the size they would be when distended with blood during life.

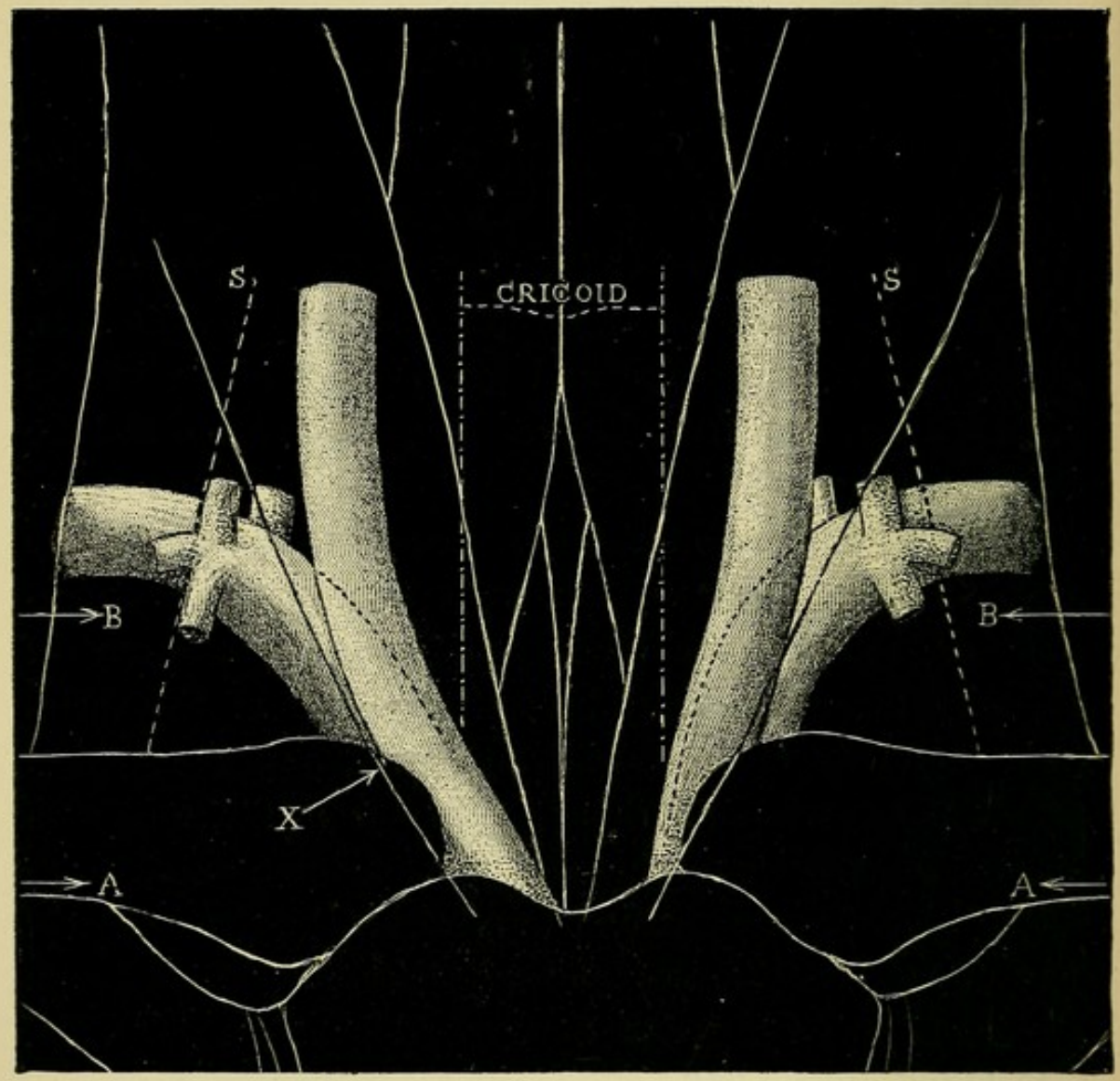


FIG. 211.

FIG. 211. Front view of the great arteries at the root of the neck, with the bones and muscles shewn in outline. (Nat. size.)

Outline drawing shewing the position of the great arteries at the root of the neck: a block being placed under the shoulders, the head thrown back and the outer ends of the clavicles depressed.

The manubrium sterni, the clavicles and the first ribs are seen: the sternal origin of each sterno-mastoid is shewn and also the outer edge of its clavicular origin, but the inner edge of this attachment is not marked. The inner border of each scalenus anticus is indicated by the dotted line S.

In the median line above are the adjoining margins of the sterno-hyoid muscles, lower they separate and expose the inner edges of the sterno-thyroids which meet and sometimes even overlap in the middle line at the upper border of the sternum. The lower border of the cricoid and the sides of the trachea are indicated by broken lines.

The cricoid in this position of the neck is nearly two and a half inches (6 cm.) from the notch of the sternum.

The left border of the innominate artery first appears in the neck immediately behind the middle of the episternal notch, that is to say, exactly in the middle line: the bifurcation of the innominate is indicated by the arrow X: it is directly behind what Ward names the superior angle of the inner extremity of the clavicle: the subclavian arises from the back of the innominate and passes upwards and outwards, and also backwards so that in the drawing it is foreshortened: there is a full inch (25 mm.) of the first part of the subclavian without branches between the bifurcation of the innominate and the origin of the vertebral artery.

It will be seen that the lower margin of the second part of the subclavian is 0·7 in. (18 mm.) above the upper border of the clavicle.

At the episternal notch the innominate is 1·25 in. (32 mm.) from the surface.

The first part of the right subclavian before the branches are given off is 1·1 in. (28 mm.) from the surface.

The left subclavian at the level of the notch is 1·6 in. (40 mm.) from the surface vertically, but 1·8 in. (45 mm.) from the surface at the middle line, that is to say, from where an incision would be made to tie it at or about this level.

The depth from the surface of the left subclavian just before the branches are given off is 1·2 in. (31 mm.), and at the inner border of the scalenus 1·1 in. (28 mm.) which latter is the same as on the right side.

The left common carotid at the level of the notch is 1·3 in. (33 mm.) from the surface vertically and 1·4 in. (35 mm.) from the surface at the middle line.

The two arrows AA. represent the level at which the transverse section shewn in Fig. 213 is taken.

The arrows BB. represent the level of the section shewn in Fig. 212.

Fig. 212. Transverse section of neck. (Nat. size.)

Transverse section seen from above at the level of the arrows BB. in Fig. 211: this line is half an inch (13 mm.) above the clavicle, and 1·2 in. (31 mm.) above the episternal notch.

The section passes below the branches of the first parts of the subclavian arteries.

On either side in front is seen the cross section of the sterno-mastoid muscles: a white line separates the sternal from the clavicular origins: behind these are the sterno-hyoid muscles; they do not meet in the middle line at this level: behind these again are the sterno-thyroids: at the outer border of each sterno-hyoid is seen the collapsed jugular vein.

In the median line are the trachea, œsophagus and the vertebral column: on either side of the vertebra the longus colli muscle is seen in cross section: external to this the grey shaded area represents the section of the apex of the lung and pleura: this is seen as far outwards as the scalenus anticus, which is oval in section.

The arteries are represented at their natural (distended) size and the coats with the thickness they have when under the blood pressure during life: the arteries on the right side, it will be noticed, are a little anterior to those on the left: the subclavians are cut obliquely and the shading shews the direction they are about to take behind the scaleni muscles and over the dome of the pleura, which they in this part groove.

All other structures omitted for the sake of clearness.

Fig. 213. Transverse Section of neck. (Nat. size.)

Transverse section seen from above at the level of the arrows AA. in Fig. 211.

In front on either side of the median line the two white ovals are transverse sections of the tendinous sternal origins of the sterno-mastoids. External to these are in section the two sterno-clavicular joints with their inter-articular cartilages; at the back of the drawing the vertebral column is seen.

Behind the bones in front are found in the middle line the two sterno-thyroids and external to them the sterno-hyoids: behind the sterno-hyoids are the collapsed jugular veins, the left being nearer the median line than the right and overlapping the left carotid artery.

To shew clearly the position of the main arteries all other structures besides those mentioned have been omitted.

The innominate is seen cut obliquely, the shading indicates its direction: the left carotid and left subclavian are divided transversely: the drawing shews that if the artery be approached directly from a median incision there would be no risk of injury to the pleura: the depths of the arteries from the surface is given in the description of Fig. 211.

The small artery near the jugular vein on each side is the internal mammary.

The anterior border of the trachea is 1·4 in. (35 mm.) from the surface: the shading shews how the trachea is directed somewhat backwards as well as downwards. Behind and to the left is the œsophagus. On either side (shaded grey) is the lung and pleura.

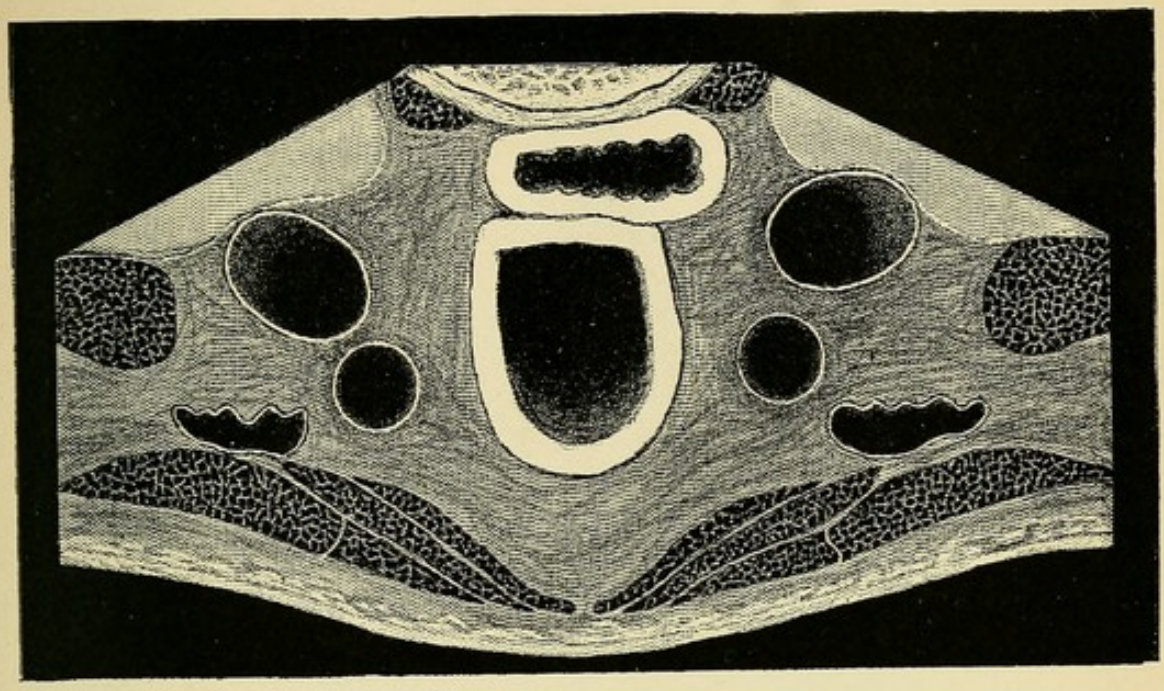


FIG. 212.

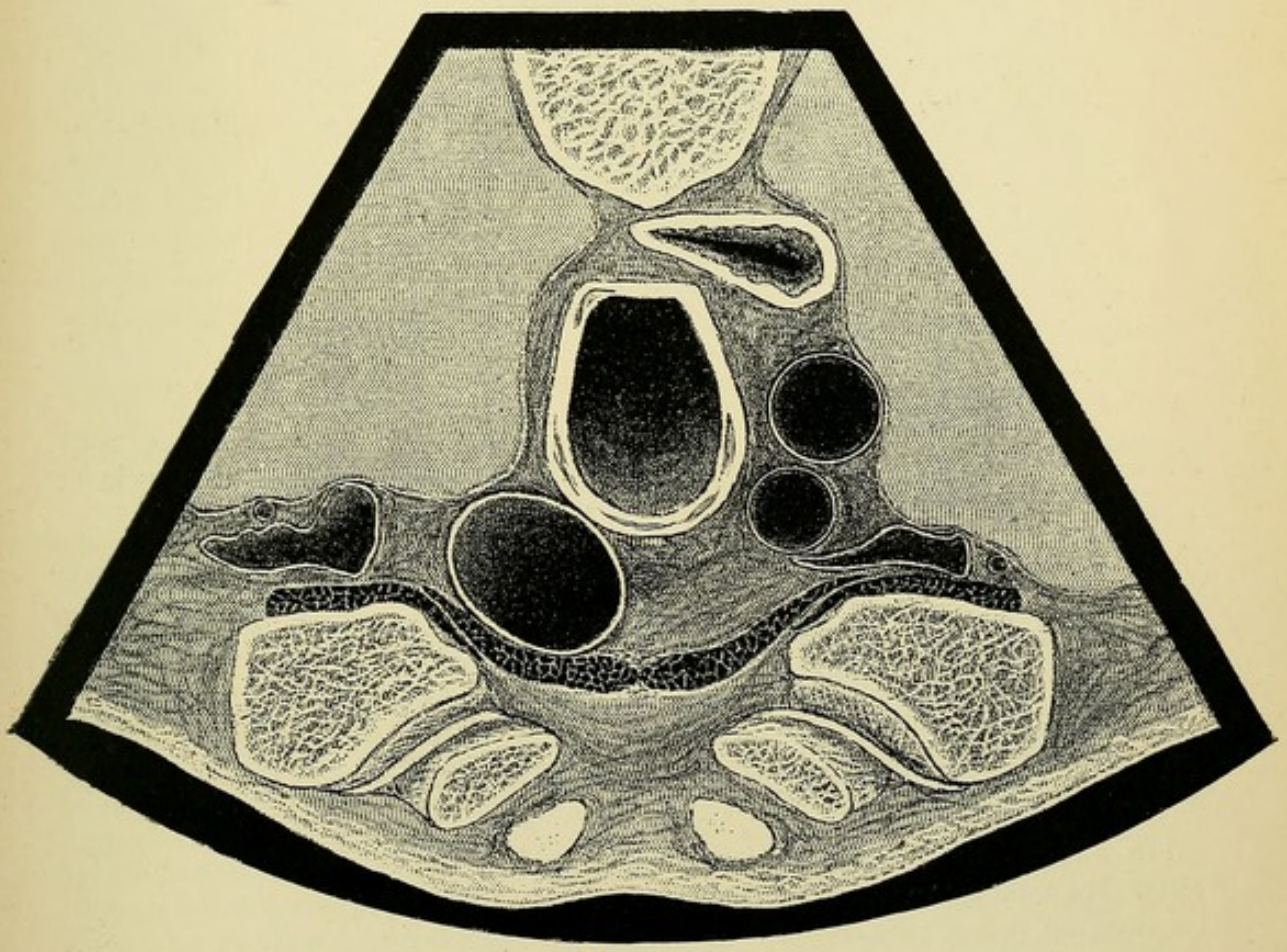


FIG. 213.

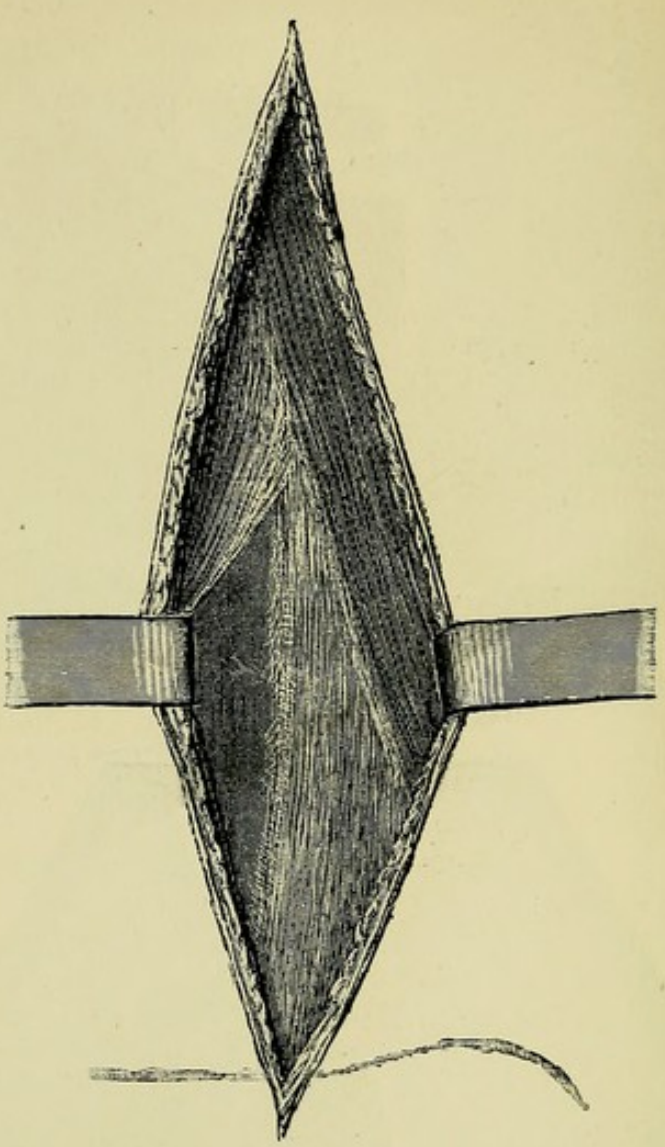


FIG. 214. First stage of exposure of first part of right subclavian: front view. (Nat. size.)

Figs. 214 and 215. To shew Sédillot's operation for the exposure of the first part of the right subclavian artery. Below is seen the outline of the upper border of the clavicle: it will perhaps be noticed that this does not correspond exactly with the outline in Fig. 211; this is merely because the drawings were made from different subjects: in this case the shoulders were more than usually broad, and the incision is therefore farther from the inner end of the clavicle.

In Fig. 214 an incision has been made dividing the superficial structures for $4\frac{1}{2}$ in. (114 mm.): it extends a little below the clavicle and passes over the interval between the two origins of the sterno-mastoid: these are held aside by retractors. There is thus exposed a layer of the cervical fascia and externally is seen, appearing

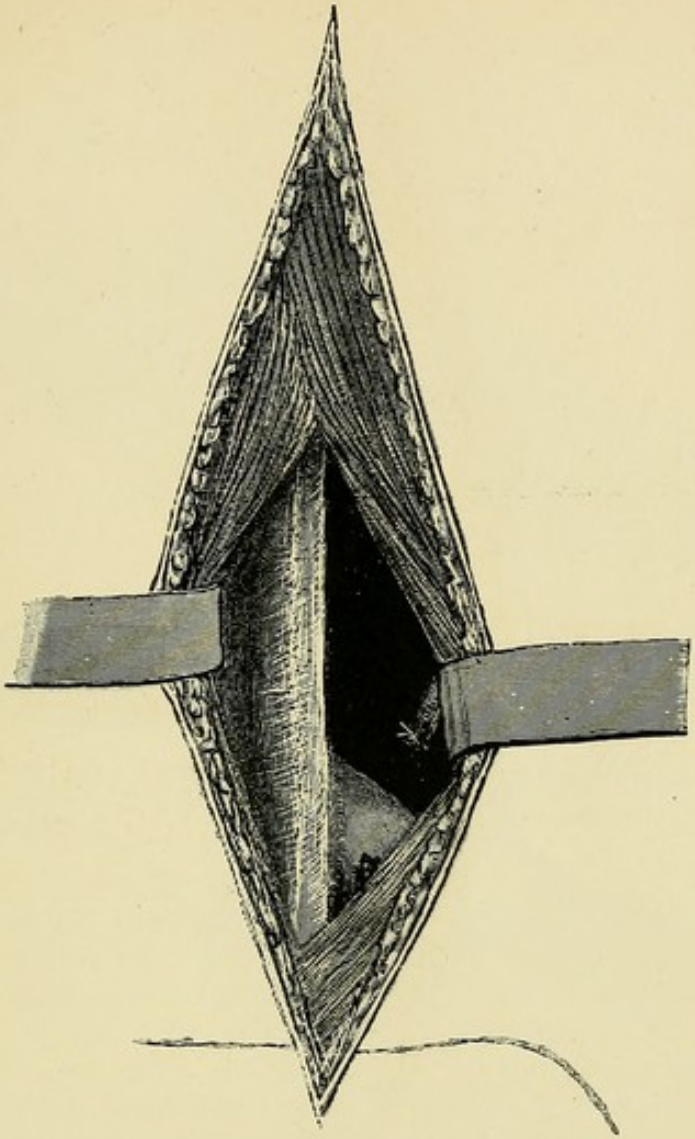


FIG. 215. Second stage of exposure of first part of right subclavian: front view. (Nat. size.)

dark through it, the internal jugular vein; internal to this are the fibres of the sterno-hyoid muscle.

In Fig. 215 the fascia over the sterno-hyoid has been divided vertically and in this way it is easy to pass between that muscle and the vein: the vein occupies the same position as in Fig. 214, and the vagus nerve is seen along its inner border: internally the sterno-hyoid is retracted: beneath it the inferior thyroid vein has been ligatured in two places and divided between: by cutting or tearing through some loose connective tissue, the first part of the subclavian artery has been exposed at the level where it is seen in the transverse section in Fig. 212. The mid point of the portion of artery exposed is one inch (25 mm.) from the surface and the lower border of the artery at this point is half an inch (12 mm.) above the upper border of the clavicle.

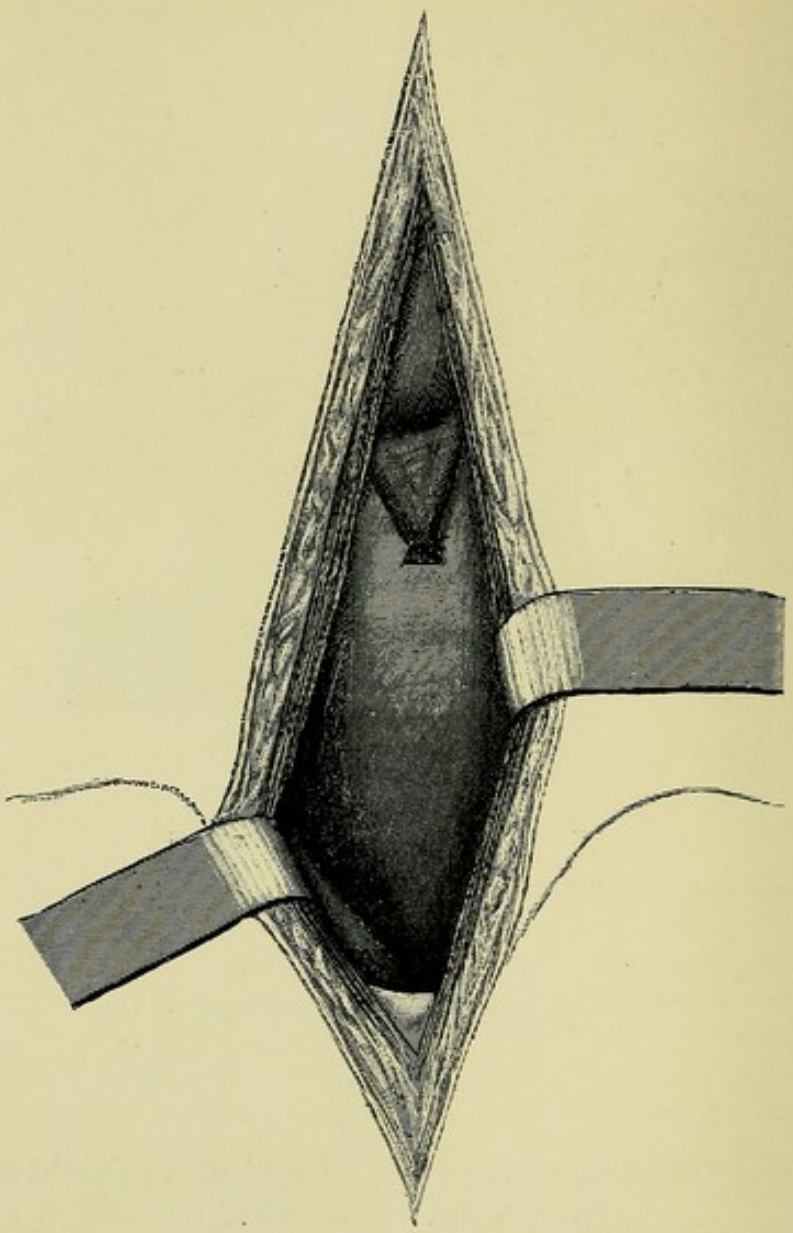


FIG. 216. Exposure of Innominate by median incision. Front view (Nat. size.)

Figs. 216 and 217. To shew the method of exposing the innominate by a median incision.

In Fig. 216 the parts are seen directly from the front.

In Fig. 217 they are seen as they would appear to the surgeon standing on the left side of the patient's neck and looking into the wound, not vertically downwards, but at an angle of 35^0 from the vertical, and not at right angles to the median incision at its mid point, but at an angle 15^0 nearer the head.

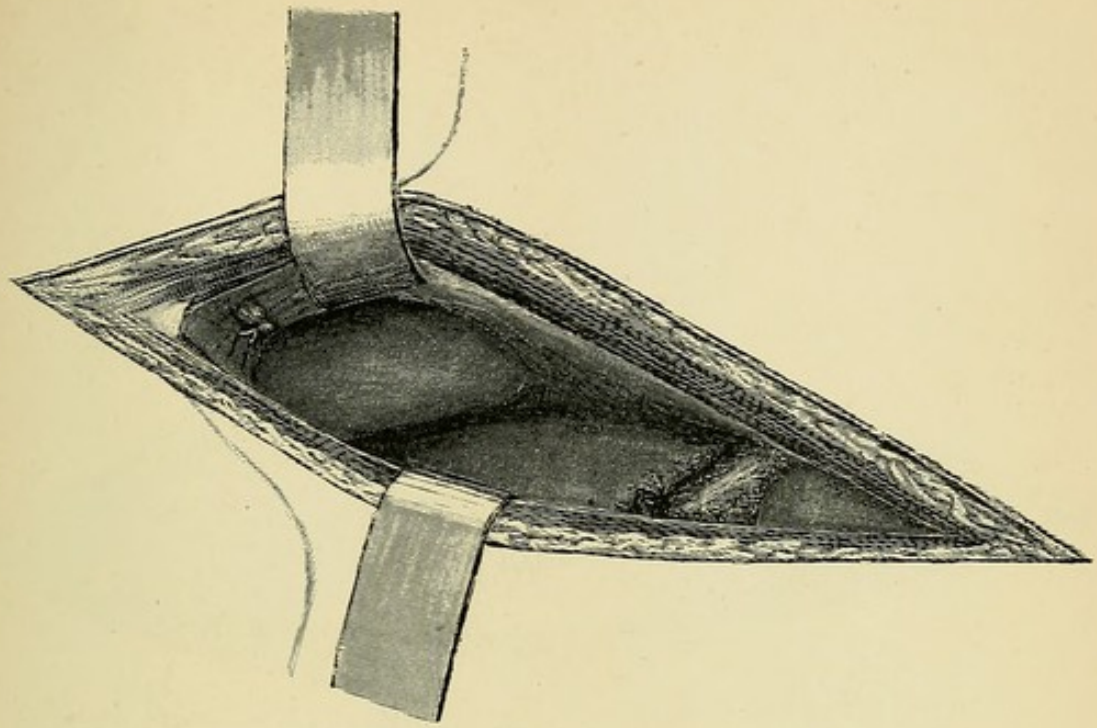


FIG. 217. Exposure of Innominate by median incision. Oblique view. (Nat. size.)

The figure is placed on the page so that the depths of the wound appear to the reader as they would to the surgeon during an operation.

A vertical median incision about 4 in. (100 mm.) long is made from the cricoid to half an inch (12 mm.) below the upper border of the manubrium.

The superficial structures and two layers of the deep cervical fascia are divided, the adjoining margins of the sterno-hyoids above and the sterno-thyroids below are separated and held apart by retractors. Some areolar tissue in front of the trachea is seen and divided. This is the stage shewn in Fig. 216: the thyroid with the divided inferior thyroid vein is above, and below this is the trachea in its sheath. By retracting the right side of the wound the position of the right carotid can be readily found, and by following it downwards the position of the innominate. The artery is a full inch (25 mm.) from the surface. The horizontal plane in which it bifurcates is quite half an inch (13 mm.) above the notch.

Fig. 216 shews how little of the artery can be seen by looking vertically into the wound. If this figure be compared with Fig. 211 it will be seen that in the subject from which that drawing was made another eighth of an inch of the artery would have been visible towards the bifurcation.

Fig. 217 shews how by looking into the wound obliquely much more of the artery is seen: quite three-quarters of an inch (19 mm.) of the left border is thus exposed in the neck where it is easily accessible to the surgeon.

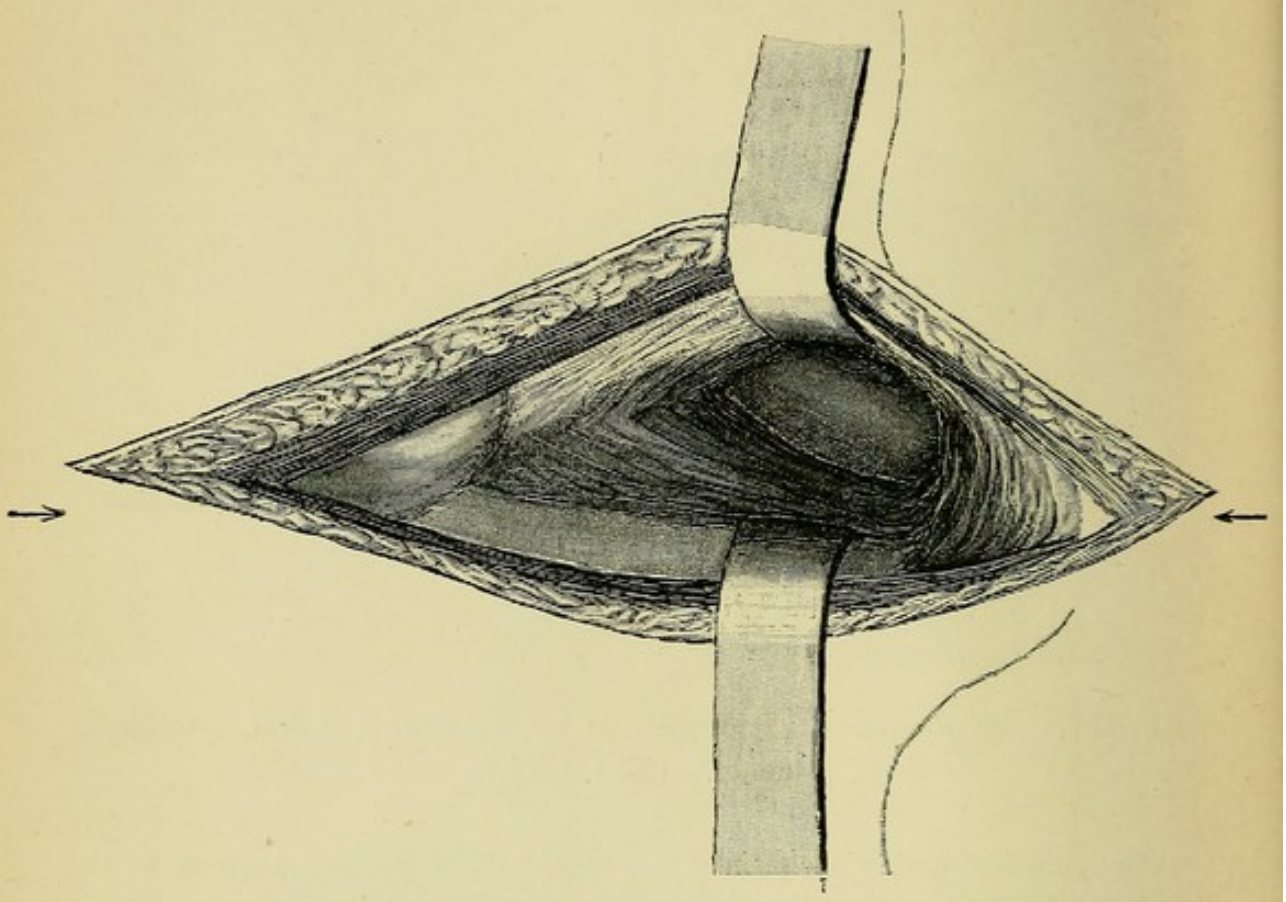


FIG. 218. Exposure of first part of left subclavian by median incision: oblique view. (Nat. size.)

The wound is drawn as it would appear to the surgeon standing on the patient's right, and looking into it, not vertically, but at an angle of 25° from the vertical and at right angles to the middle of the incision. The figure is placed on the page so that the artery appears to the reader as it would to the surgeon when operating.

The incision is made as for the innominate artery (Fig. 216): the retractor on the patient's right is holding back the right sterno-hyoid and sterno-thyroid muscles and the trachea. The left retractor is holding back more strongly the corresponding muscles on the left, and the left common carotid artery within its sheath.

On working through the areolar tissue over the artery the vessel is exposed at the level shewn in Fig. 212. It is here at a depth of 1·6 in. (40 mm.) from the middle line, where the incision has been made. It will be seen that the portion of artery exposed is at a considerable height above the manubrium. The arrows indicate the median line.

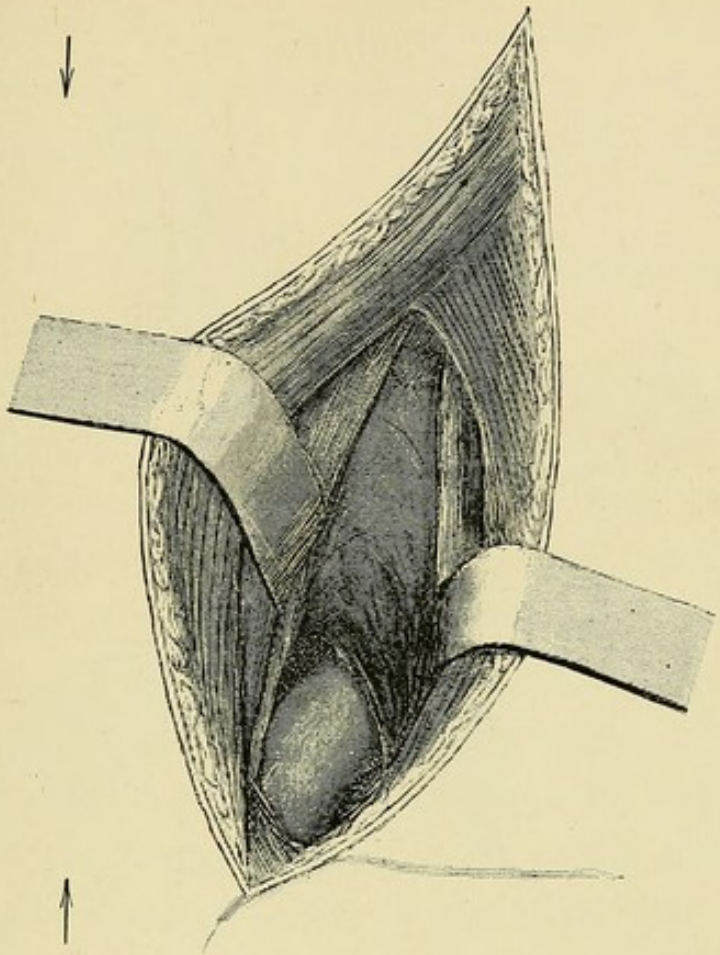


FIG. 219. Exposure of the first part of the left subclavian between the heads of the sterno-mastoid: front view. (Nat. size.)

To shew Sédillot's method for the exposure of the first part of the left subclavian artery.

The same procedure is adopted as on the right side (see Figs. 214 and 215). The view is directly from the front. The arrows indicate the median line. The outline of the clavicle is seen below.

The retractor on the patient's left pulls outwards the internal jugular vein with the vagus nerve (shewn white) on its inner border. The right-hand retractor holds back the sterno-hyoid muscle and also the left common carotid artery (its edge indicated light). At the bottom of the wound is areolar tissue some of which has been removed to expose the vessel, which here lies at a depth of 1·2 in. (30 mm.) from the surface and well above the clavicle. The artery will generally be reached here as by the median incision before its branches are given off.

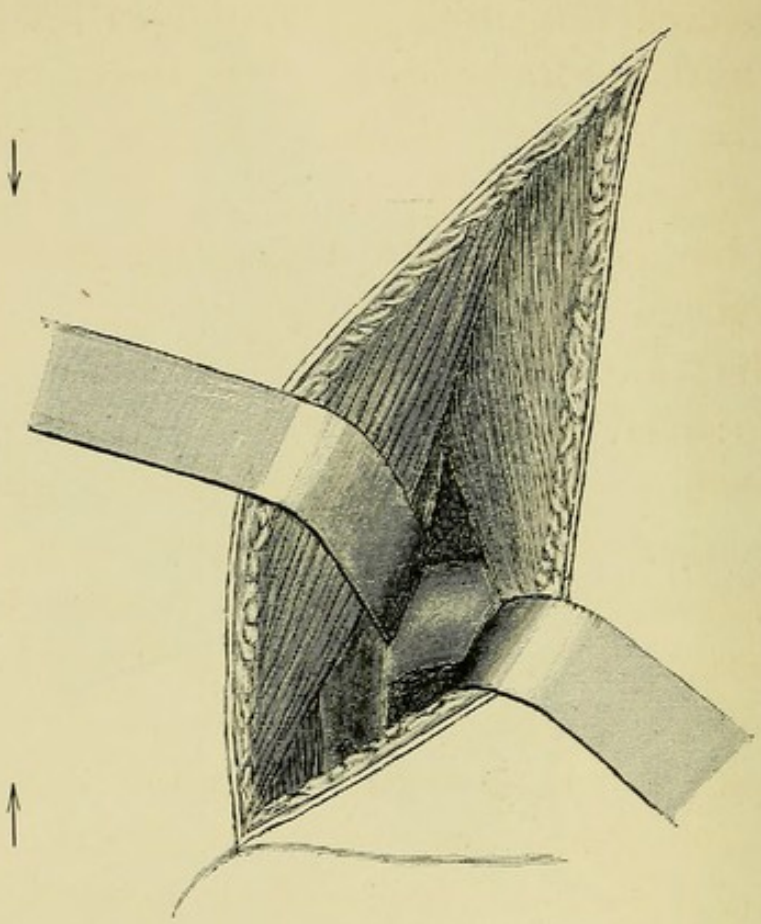


FIG. 220. Exposure of first part of left subclavian artery at inner border of scalenus: front view. (Nat. size.)

Ligation of first part of left subclavian external to the branches and external to the jugular vein, immediately before passing behind the scalenus anticus.

The outline of the clavicle is below: the arrows indicate the median line. The same incision as in Fig. 219 is made. The two heads of the sterno-mastoid are seen separated: the inner border of the scalenus anticus has been made out and the fascia over it divided, in this way it is possible to pass between the scalenus and the jugular vein. The artery then comes into view as seen in the drawing: it is at a depth of 1·1 in. (28 mm.) from the surface and its lower border is $\frac{3}{4}$ in. (19 mm.) above the clavicle. The retractor on the patient's right is holding back the jugular vein, the scalenus muscle is not in view. It will be noticed that the artery is reduced in size owing to the branches having already been given off. Compare Figs. 218 and 219.

By this method the second part too of the subclavian can be reached.

As in the root of the neck so in the lower part of the trunk the ligation of the main arteries has been attended with a considerable mortality. The abdominal aorta has never been tied with success. The mortality with the common iliac has been 75, and with the external iliac 22 per cent. (See page 4.)

The causes of death have been peritonitis, gangrene and hemorrhage.

In order to avoid the risk of peritonitis which was involved in opening the peritoneal cavity, the extra-peritoneal operation was devised. For femoral aneurisms, in which it suffices to ligate the external iliac just above Poupart's ligament, this method can be followed, but in inguinal aneurisms it involves the necessity of operating in the immediate vicinity of the tumour.

Another way has now been found of avoiding peritonitis, and in these days of antiseptics the danger of opening the peritoneal cavity ought to be nil.

Gangrene is in some cases a sequela of ligation which the surgeon can neither foresee nor avert. It is not however, a necessary consequence even of ligation of the abdominal aorta.

This was shewn, as far at least as the lower animals are concerned, by the experiments of Sir Astley Cooper already mentioned. In man the same thing is proved by the case of Monteiro of Rio de Janeiro. He ligated the abdominal aorta, and during the ten days the patient survived there was no sign of gangrene. The cause of death was hemorrhage.

The third and chief remaining cause of death is hemorrhage; if therefore some way of obviating this can be

devised, the patient will have a fair chance of recovery.

The surest method of treating most aneurisms is by the ligation of the artery above: but for a certain number of cases of iliac aneurism it is the abdominal aorta that would have to be tied.

The operation does not appear to be as formidable as it sounds. In persons of ordinary stoutness the abdominal aorta is from two to three inches from the surface. The management of the intestines would offer no great difficulty.

The common iliac, too, can be most satisfactorily reached through the abdomen, the upper part of the artery from the linea alba, the lower part from the linea semilunaris.

The details of these procedures are appended. The operations for the abdominal aorta and common iliac are, on the cadaver at least, easier than the extraperitoneal—one comes down directly on the artery.

The incision in the linea semilunaris gives easy access also to the first part of the external iliac artery. This operation may be useful in some cases, we have therefore described and figured it.

Procedure for the ligation of the abdominal aorta.

(Figure 223.)

The aorta as a rule bifurcates on the left side of the fourth lumbar vertebra, and about three-quarters of an inch (19 mm.) below the level of the umbilicus.

The operator should stand on the patient's right.

A median incision is made four inches (10 cm.) long, extending an inch and a-half (4 cm.) above, and two and a-half (6 cm.) inches below, the umbilicus.

The incision is carried through the linea alba and parietal peritoneum in the usual way; the abdominal cavity being opened, broad retractors are inserted on the right and left; the transverse colon is pushed upwards and kept there by means of a sponge. The small intestine with its mesentery is then all drawn over to the left and held back by the left-hand retractor and by a sponge at the lower part of the wound. The right-hand retractor holds back the cæcum. This plan of dealing with the intestine is illustrated by the transverse section shewn in Figure 221, page 502.

The mesentery being attached obliquely to the front of the spine from the left side of the second lumbar vertebra to the right sacro-iliac synchondrosis the small intestine is easily displaced to the left; the root of the mesentery being at this level well to the left, the parietal peritoneum over the aorta becomes exposed. In those cases in which the attachment of the mesentery crosses the median line high up it is as easy to expose the aorta by turning the mesentery over to the right as to the left.

The distance of the aorta from the surface of the abdomen is surprisingly little, especially in a thin patient. In one subject we found it an inch and a-half (4 cm.), in another two and a-half inches (6 cm.), that is, measuring from the skin to the front of the distended aorta.

The position of the aorta being recognized, a vertical incision an inch and a half (4 cm.) long is made through the peritoneum covering it. The sheath must be now

opened in the usual way, and the aneurism needle passed. When the stay-knot has been completed, and the parts sponged and dried, the sheath may be closed by two fine silk stitches and three or four Lembert's stitches should be applied to adjust the peritoneal incision. The abdominal cavity is thus closed behind. The opening in the abdominal wall in front is brought together in the usual way.

Procedure for the ligation of the Common Iliac.

The common iliac can be exposed by an incision either in the middle line or in the linea semilunaris. To reach the upper part of the artery the median incision is better, for the lower part the lateral. (See Figures 225 and 226.)

Median incision. An incision four inches (10 cm.) long is made from the umbilicus downwards. After the abdominal cavity has been opened and the position of the artery defined the intestines are retracted in the same manner as already described in the case of the abdominal aorta; in fact all the steps of the operation are similar.

Lateral incision. An incision four inches (10 cm.) long is made in the linea semilunaris starting from the level of the umbilicus.

In this case it will be advisable to hold the retractors somewhat obliquely to the body so as to expose the artery in its length. (See Figure 226.)

The cæcum is held back by the right-hand retractor and the small intestines by the left. The lower sponge presses down the end of the ileum, where it passes to the right to join the cæcum.

The ureter passing downwards almost vertically crosses the artery at its bifurcation. It is adherent to the peritoneum, and its position may be sometimes defined.

The details of the operation are the same as for the abdominal aorta.

On the left-hand side the upper part of the common iliac can be tied as described above, but for the lower part, and in some cases for the whole of the artery, a different management of the intestines might be advisable. When the attachment of the mesentery crosses the middle line at or above the bifurcation of the abdominal aorta it would be best to reach the left common iliac by turning the mesentery over to the right.

Procedure for the ligation of the upper part of the external iliac.

An incision (see Figure 228) four inches (10 cm.) long is made in the linea semilunaris, commencing one inch (2·5 cm.) below the level of the umbilicus. The cæcum with the end of the ileum is pushed upwards by the upper sponge, the small intestines held back by the left retractor. The position of the vessel is defined and a ligature placed upon it in the manner described for the abdominal aorta.

The ureter and genito-crural nerve must be avoided.

On the left-hand side the vessel will be sought, either below or above the meso-sigmoidea, according as the attachment of this is at, or below, the bifurcation of the common iliac artery, in either case the small intestines must be retracted to the right.

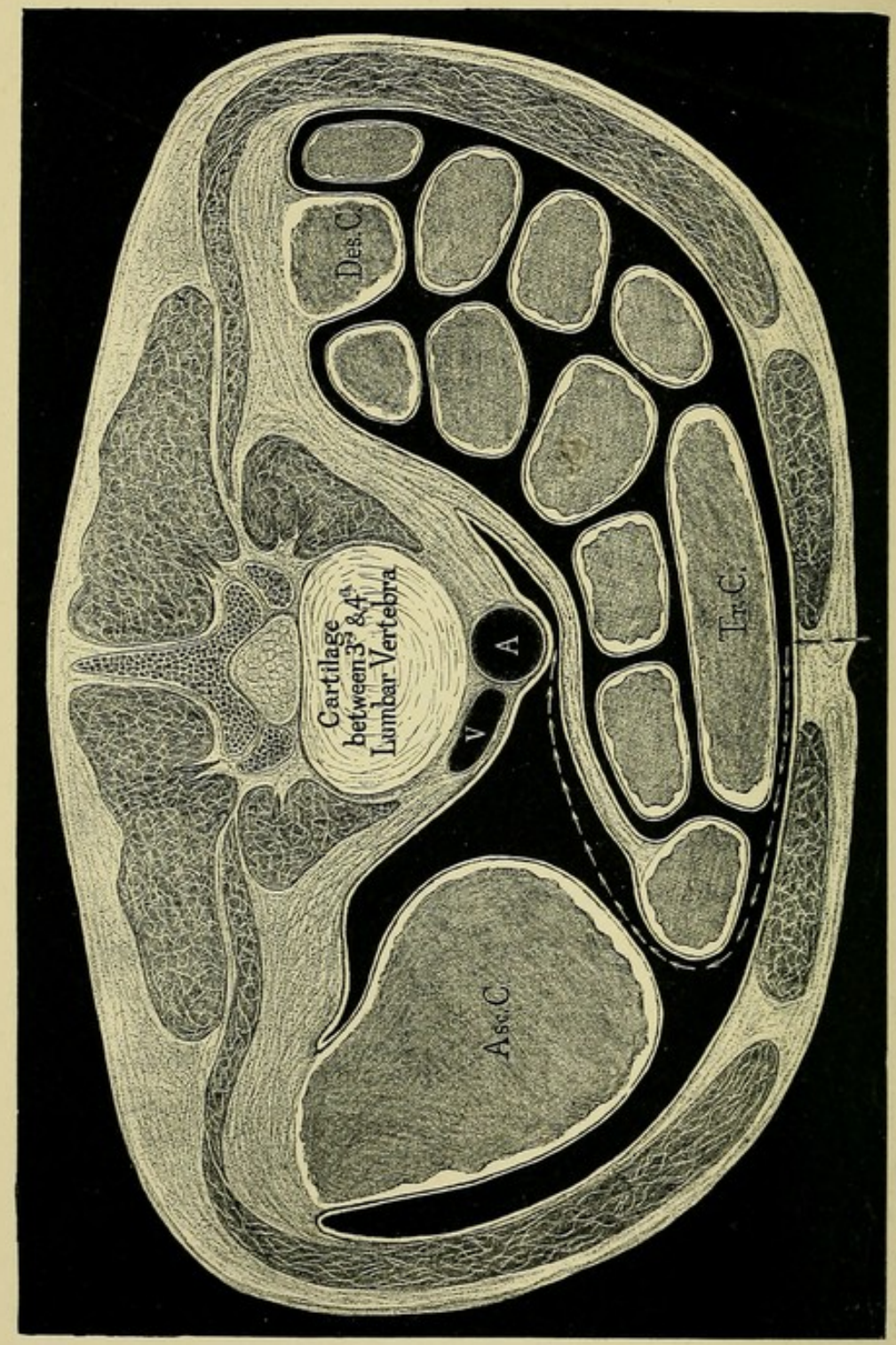


FIG. 221.

FIG. 221. Transverse section of trunk through umbilicus.
(Half nat. size.)

The section passes through the cartilage between the third and fourth lumbar vertebræ: the positions of the ascending, transverse and descending colon are indicated. A and V mark the aorta and vena cava. The aorta is drawn to scale: the parietal peritoneum can be followed, also the attachment of the mesentery to the posterior boundary of the abdomen behind and to a loop of the small intestine in front. The other unmarked sections of bowel are small intestine. In order to reach the aorta the transverse colon is pushed upwards and the small intestine and mesentery are retracted to the left: this is shewn by the arrows. In this way the aorta is exposed with a single layer of parietal peritoneum in front of it. The drawing is taken from a specimen at the Royal College of Surgeons: the attachment of the mesentery is to scale, but the sections of small intestine are somewhat diagrammatic.

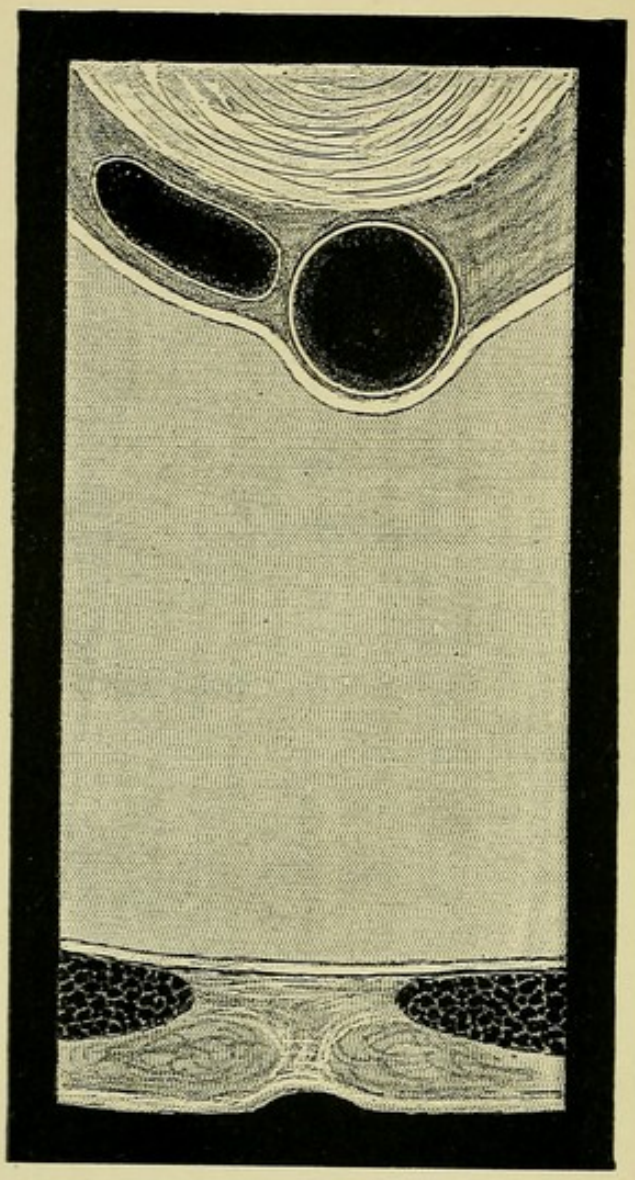


FIG. 222. Part of transverse section shewn in figure 221. (Nat. size.)

The intestines are omitted, they being supposed to be retracted. The diameter and thickness of the wall of the aorta are drawn to scale: its section is represented circular, but in some distended specimens we found it one millimetre more in the transverse than in the antero-posterior diameter. The distance of the front of the aorta from the skin varies much, in this specimen it is $2\frac{3}{4}$ inches (7 cm.).

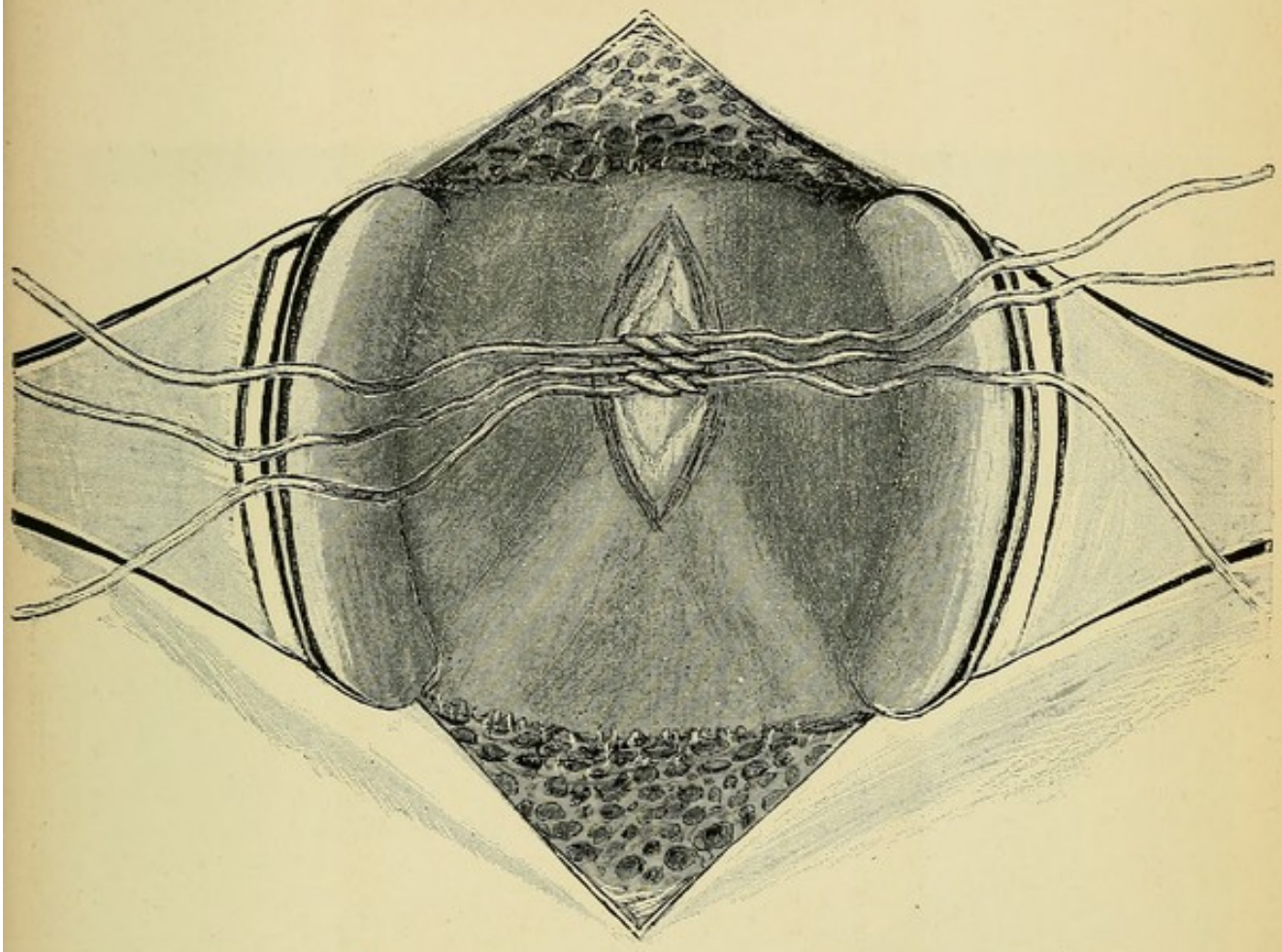


FIG. 223. Ligation of abdominal aorta by median incision. (Nat. size.)

The vessel has been exposed by the method described in the text (page 498). The posterior layer of the parietal peritoneum has been incised, the sheath of the artery opened and three kangaroo tendon ligatures ($\frac{1}{20}$th inch—1·3 mm. wide) have been passed round the artery. The first hitches of the stay-knot have been adjusted. In this case the ligatures were placed immediately behind the umbilicus, the lowest being half an inch (12 mm.) from the bifurcation. The common iliac arteries raise the peritoneum over them into a low ridge: the drawing was made from a thin subject and the front of the distended aorta was only $1\frac{1}{2}$ inch (37 mm.) from the surface of the abdomen.

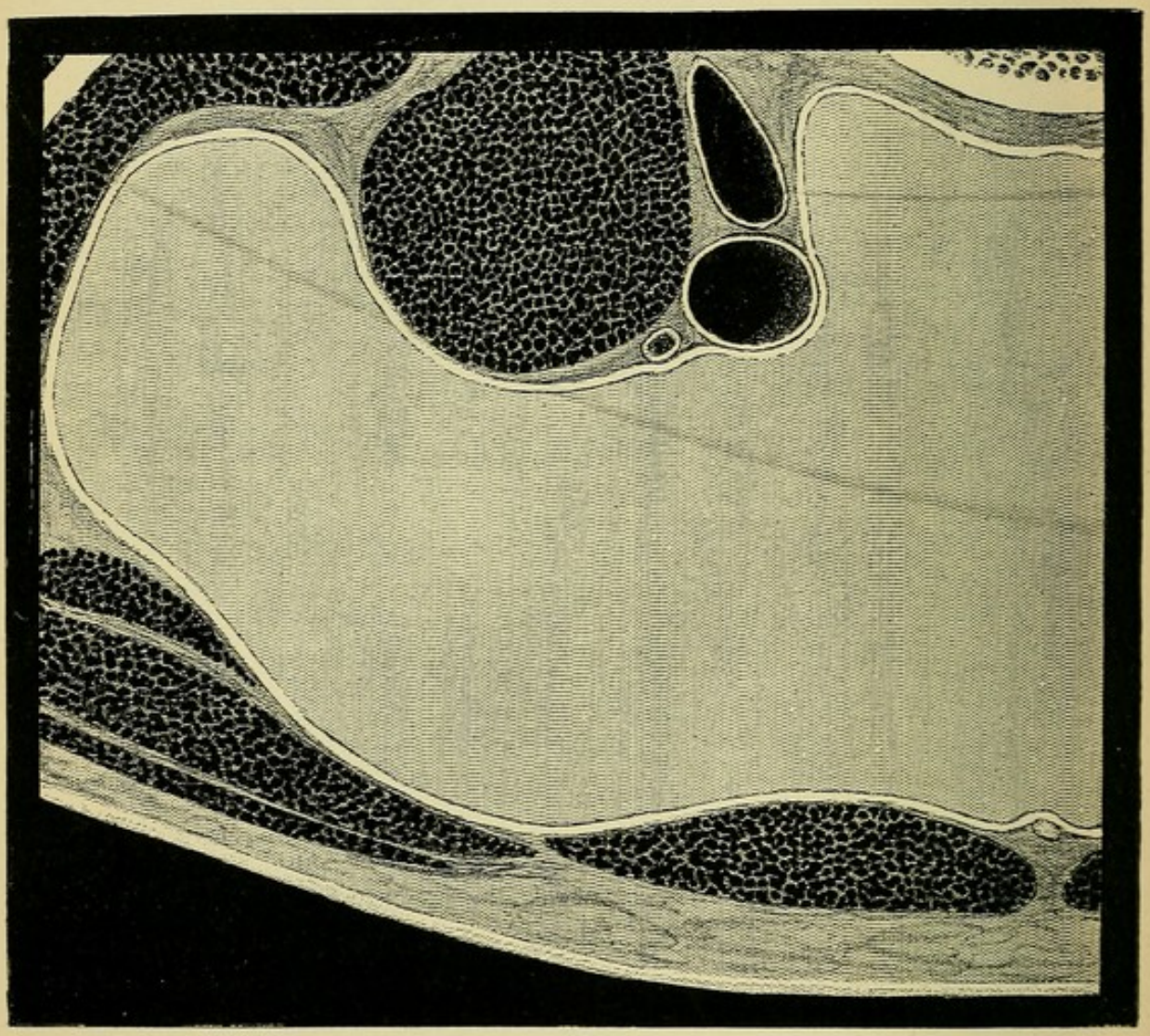


FIG. 224.

FIG. 224. Transverse section of trunk about $1\frac{3}{4}$ inch (4·5 cm.) below Umbilicus. (Nat. size.)

The drawing is made to shew the relation of the common iliac artery to the linea alba and the linea semilunaris. The section extends a little beyond the middle line in one direction and just far enough in the other to include the limits of the abdominal cavity.

In front is seen a little of the left and the whole of the right rectus muscle: external to the right rectus are seen the sections of the external and internal oblique and the transversalis muscles.

In the extreme upper and left-hand corner is a portion of the os innominatum: internal to that is the iliacus and internal to this is the round projecting mass of the psoas: in the middle line behind is the upper part of the sacrum: the parietal peritoneum is indicated by the thick white line bordering the grey shaded space of the abdominal cavity which would contain externally the cæcum and internally the small intestine.

At the inner border of the psoas is seen the common iliac artery: behind it is the corresponding vein and external to it is the ureter: the size and thickness of the artery are to scale: the vessel is cut obliquely as is shewn by the shading: it runs downwards and outwards at an angle of 35° with the vertical. The section is made about the mid-point of the common iliac where it is behind the middle of the rectus: above this level it would be easier to reach it from the middle line, below from the linea semilunaris.

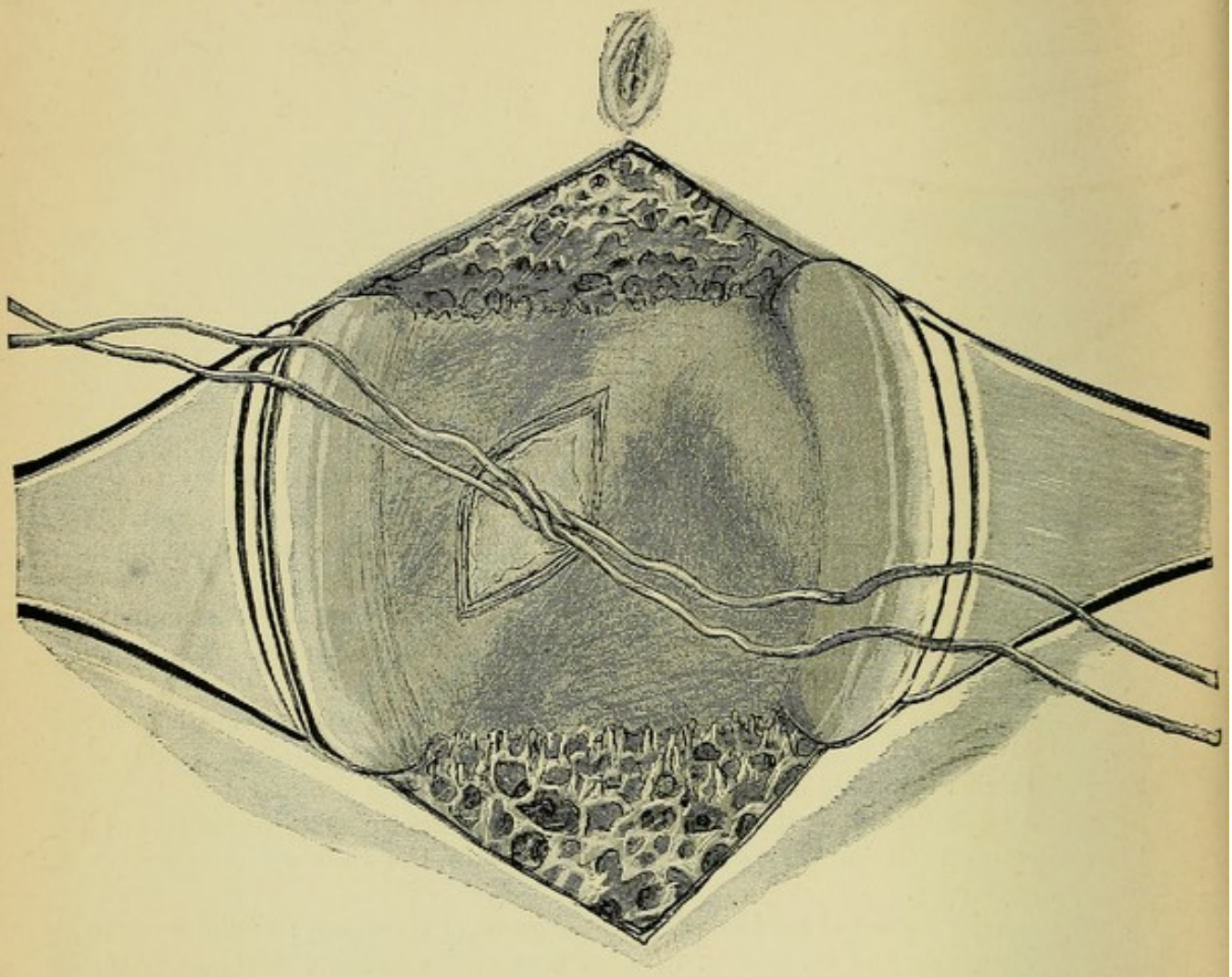


FIG. 225. Ligation of common iliac through a median incision. (Nat. size.)

A median incision is made commencing at the umbilicus and extending 4 inches (10 cm.) downwards.

The management of the intestine is the same as in the ligation of the abdominal aorta as described under figure 221.

In the figure the right artery is being ligated and accordingly the right retractor is more pulled on than the left. In this case the aorta bifurcates somewhat lower than usual.

The peritoneum and the sheath of the vessel have been incised so as to expose the artery, and the first hitches of a stay knot of kangaroo tendon adjusted.

The operation can be completed as described on page 500.

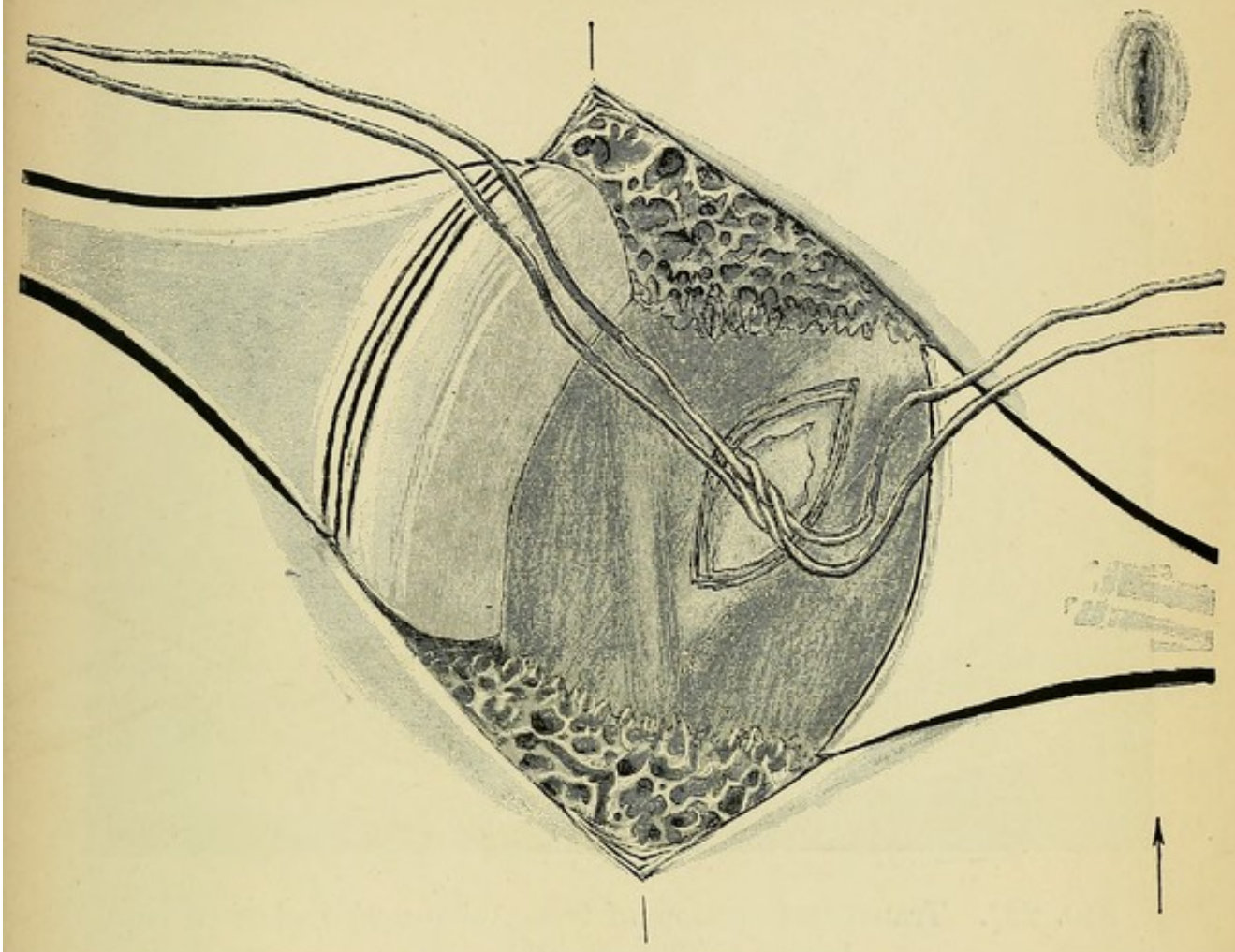


FIG. 226. Ligation of common iliac through a lateral incision. (Nat. size.)

An incision is made in the linea semilunaris, commencing at the level of the umbilicus and extending 4 inches (10 cm.) downwards and slightly inwards.

The cæcum is retracted to the right, the small intestines to the left; the lower sponge keeps down the end of the ileum just before it joins the cæcum : the upper sponge too holds back intestine. The right retractor is held obliquely so as to expose the artery in its length. The course of the ureter is faintly indicated : it crosses the artery just where it bifurcates. The stay knot has been applied three quarters of an inch (19 mm.) above the bifurcation. The median line is indicated by the position of the umbilicus and the arrow below.

The exposure of the artery, the application of the ligature and the completion of the operation as before described.

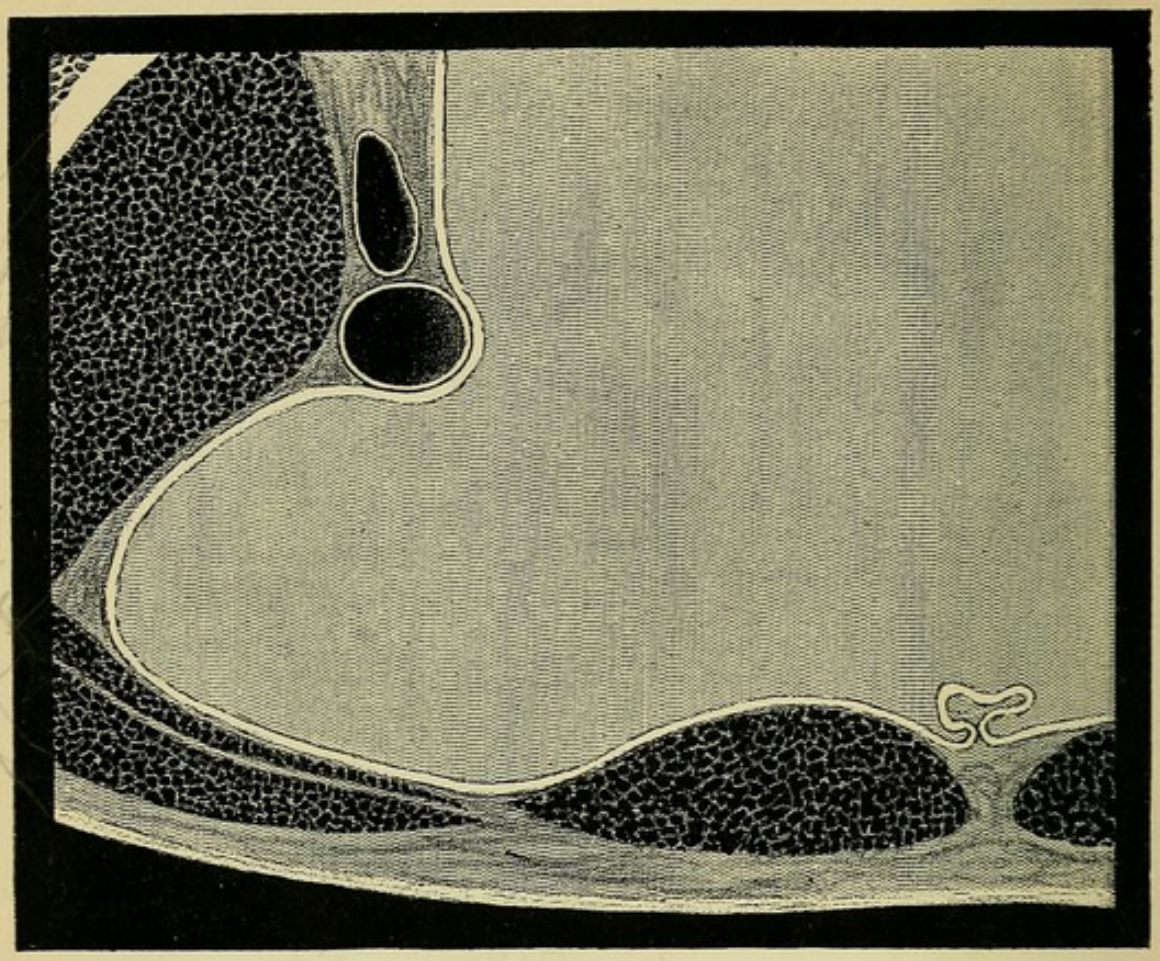


FIG. 227. Transverse section of trunk about 3½ inches (9 cm.) below Umbilicus. (Nat. size.)

The drawing is made to shew the relation of the first part of the external iliac artery to the linea semilunaris. The section extends beyond the median line in one direction and just far enough in the other to include the lateral limit of the abdominal cavity.

In front is seen a little of the left and the whole of the right rectus muscle: external to the right rectus are the internal oblique and transversalis muscles, the section being below the muscular fibres of the external oblique.

In the extreme upper and left-hand corner is a portion of the os innominatum: internal to that is the fleshy mass of the ilio-psoas muscle: the parietal peritoneum is indicated by the thick white line bordering the grey shaded space of the abdominal cavity.

On the inner border of the psoas is seen the transverse section of the external iliac artery, behind it is its companion vein: the size and thickness of the arterial wall are to scale. The vessel is cut obliquely as shewn by the shading; it here runs downwards and outwards at an angle of 32° with the vertical. It will be noticed that the artery is immediately behind the linea semilunaris.

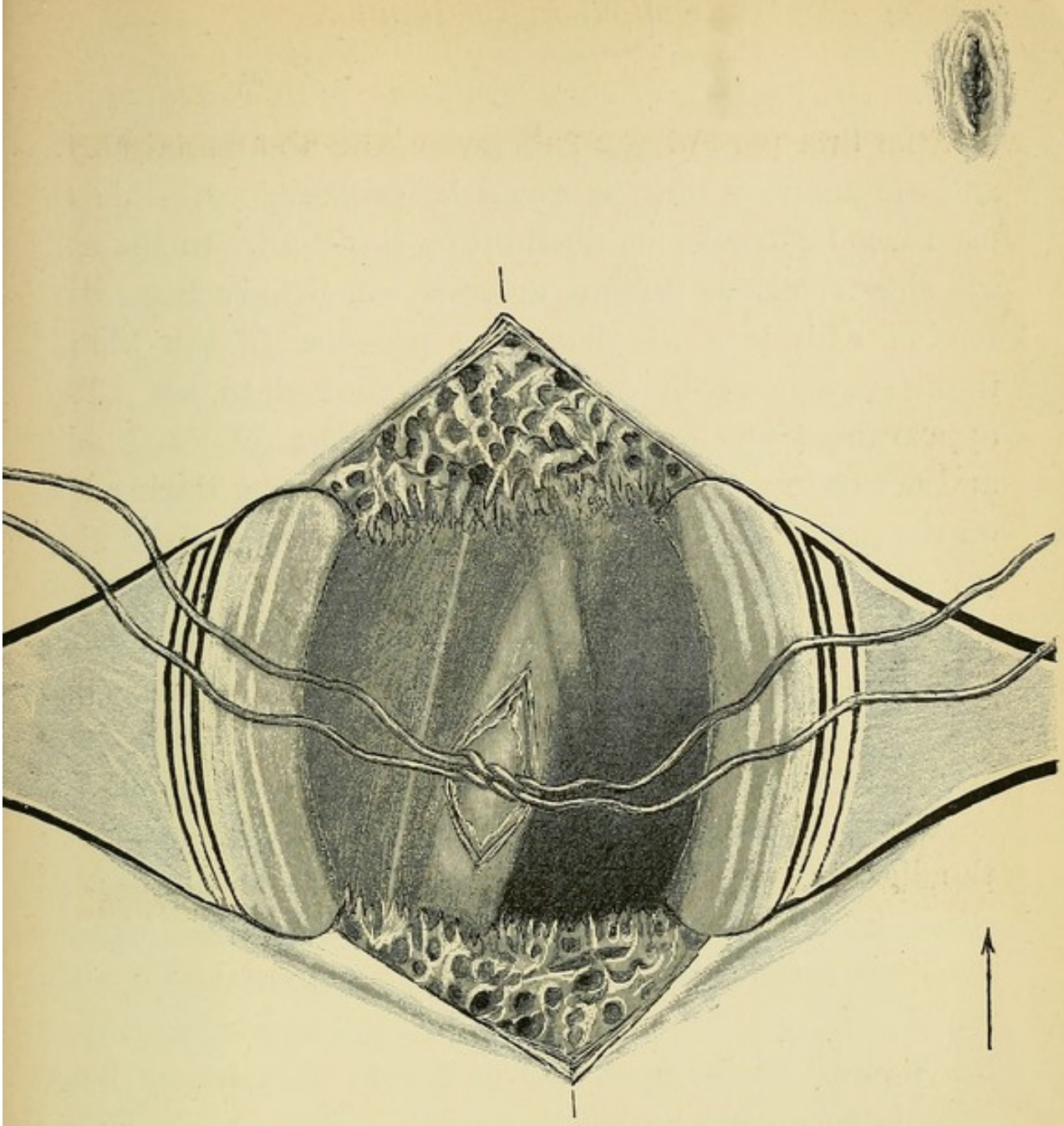


FIG. 228. Ligation of external Iliac.

Ligation of the upper part of the external iliac through the linea semilunaris. An incision four inches (10 cm.) long is made commencing one inch (25 mm.) below the level of the umbilicus. The median line is indicated by the position of the umbilicus and the arrow below. The sides of the wound are held back by retractors, the left holds back the small intestines, and the upper sponge the cæcum and termination of the ileum: the artery makes a distinct elevation of the peritoneum over it: external to this is seen through the peritoneum the psoas with the genito-crural nerve lying on it. The peritoneum over the artery has been incised and the sheath of the vessel opened: the first hitches of a single stay knot have been adjusted. About an inch (25 mm.) above the ligatures a nearly vertical ridge of peritoneum indicates the position of the ureter where it crosses the bifurcation of the common iliac: the ligatures are therefore applied about one inch (25 mm.) from the commencement of the external iliac. The internal iliac comes off from the back of the common iliac, and therefore does not interfere with the direction of the main trunk.

The depth of the retractors shewn in this figure and in figures 223, 225 and 226 is $2\frac{1}{2}$ inches (6·5 cm.).

For the ligation of such large vessels as the innominate, the first part of the subclavian, the abdominal aorta and the common iliac, it would be probably advisable to use three ligatures combined into a stay-knot. In Plate X are shewn various human arteries which have been distended with wax to about the pressure of the blood, ligated with stay-knots and then allowed to set. The appearances are the same on both sides of the knots, and accurately represent what would be seen during life on the proximal side of the ligatures. On the distal side (as we have seen on page 417), immediately after ligation the blood-pressure falls and the artery, to a great extent, collapses. Later, the pressure is partly re-established, but the degree to which this occurs will vary in different cases according to the freedom of the collateral circulation.

There are other methods of curing aneurisms than by the ligation of the main artery above. The simplest of these is digital compression. It has the advantage of avoiding an operation, and is very appropriate for such cases as offer a fair prospect of being thus cured within a few hours. These cases will as a rule be those of large sacculated aneurisms which already contain a good deal of clot: and which at first sight seem worst. Digital compression has also another advantage; by it, when successful, only one obstruction is raised to the circulation, namely, at the seat of aneurism. Whereas, after the Hunterian operation there are two, one at the aneurism, and another at the seat of ligation. The immediate danger of gangrene therefore is greater in the latter case.

Temporary ligation of the artery for (say) one or two hours would possess the same advantage with respect to

a single seat ot occlusion as digital compression, but a clot might form above the ligature and on its removal be carried along the artery. If the artery were blocked at the aneurism, the embolus would do no harm, but if the artery were patent, the embolus would pass on and might cause gangrene.

Next in the order of advancing severity comes ligation without rupture. Like digital compression it causes no weakening of the arterial wall, and might, therefore, be called ligatural compression. With respect to other methods, it would seem that Esmarch's bandage, by completely arresting the circulation throughout the limb for the time that it is applied, entails too great a risk of gangrene. Of galvano-puncture it must be said, that although this apparently simple method has been before the profession for upwards of half a century, it has failed to secure confidence—it is too uncertain.

The methods of Fergusson—digital manipulation of the aneurism in order to displace the coagulum and thus block the vessel—and of Macewen—scraping the interior of the sac by the points of fine steel pins in order to stimulate the growth of the wall and the formation of clot—have been already referred to. They may be suitable for certain cases.

Possibly in the future, Wooldridge's discovery of a substance which will instantly produce coagulation may become of service, but at present there is no means known, of restricting the clotting to the blood in the aneurism.

We are left, then, in the great majority of cases to the use of the ligature, either proximal or distal. The latter acts by means of the proximal clot extending into the sac,

Description of Plate X.

Human Arteries tied with stay-knots.

The arteries drawn are healthy arteries of men; they were distended to the normal pressure of the blood with wax, cocoa butter or glycerine jelly: they were then ligatured with either kangaroo tendon or stout catgut, the ligatures being tied with stay-knots. After the distending material had set the external appearance was drawn; longitudinal sections were then cut, and the internal appearance drawn. The sections shew the actual size of the vessels and the actual thickness of their walls under distension.

It will be seen that the arteries represented in Figures 1 to 14, and 27 and 28 have been tied with two ligatures. Those represented in Figures 19 to 26 were tied with three, those in Figures 15 and 16 with four, and those in 17 and 18 with six ligatures.

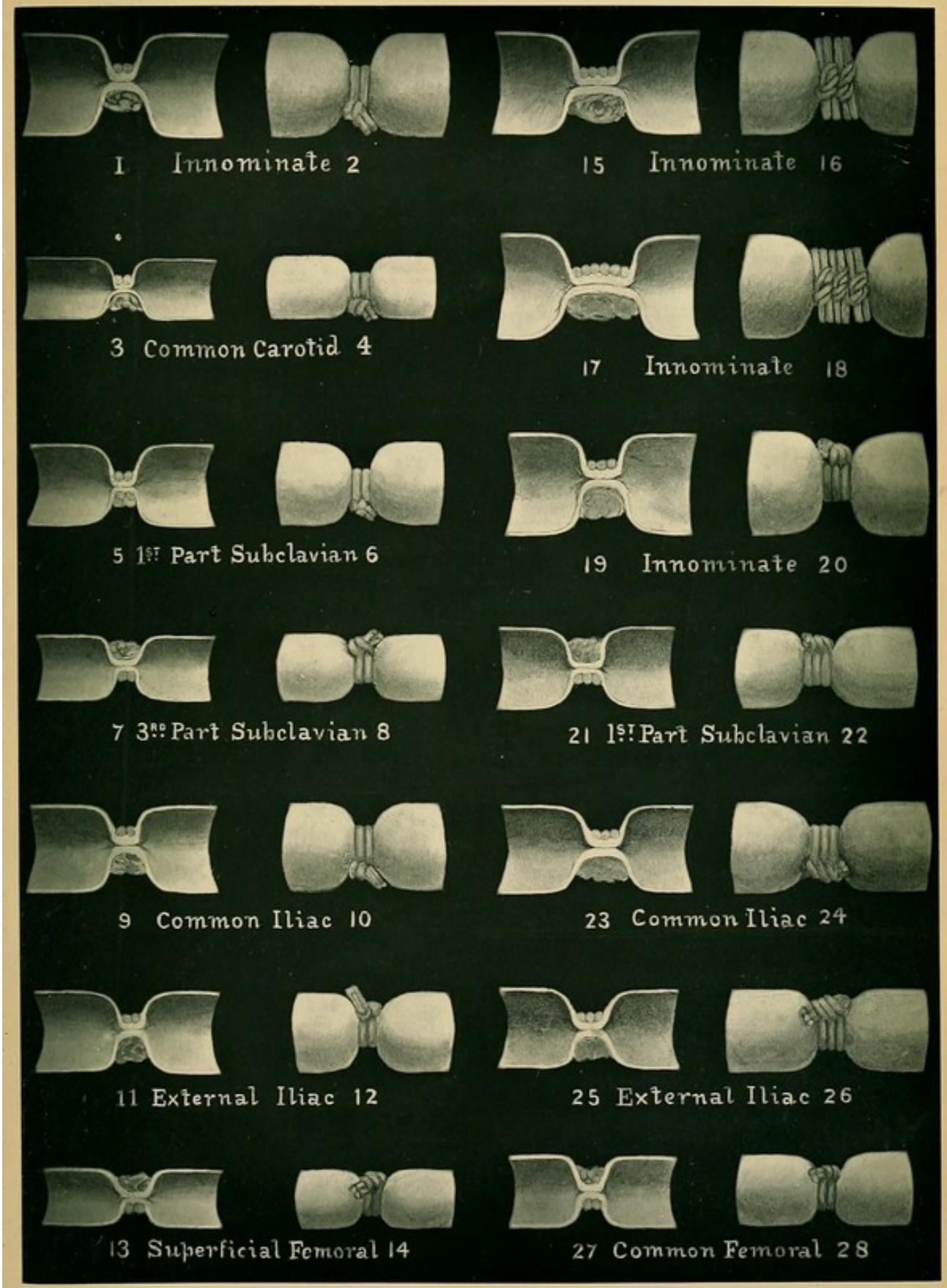
In all the cases in which either two or three ligatures were used, the ends after the tightening of the first half of the stay-knot were combined into a single final hitch. In Figures 15 and 16 the four ends on each side were combined in pairs to form two distinct final hitches. In Figures 17 and 18 the six ends on each side were treated in pairs, so that three distinct final hitches were formed.

It will be noticed that the sections of the loops of the ligatures on opposite sides of the vessel are separated by a distance considerably more than twice the thickness of the wall of the vessel. The exact extent of the separation is given in the following table:

Innominate	4·5	times thickness of wall.
Common Carotid	3·1	„ „ „ „
First part of Subclavian	4	„ „ „ „
Third „ „ „	3·5	„ „ „ „
Common Iliac	4·5	„ „ „ „
External Iliac	4	„ „ „ „
Common Femoral	3·75	„ „ „ „
Superficial Femoral	3	„ „ „ „

LIGATION IN CONTINUITY.

PLATE X.



and starting fresh coagulation in it. It is less effectual and has the disadvantage of increasing, temporarily at least, the blood-pressure in the aneurism, whereas proximal ligation immediately greatly reduces it.

In proximal ligation, therefore, lies our best hope of curing most aneurisms. It only remains to consider how best to secure, in the case of the greatest arteries, such as the innominate, the full measure of success. The secret lies in a painstaking attention to every detail.

In the first place, the surgeon must have a clear picture in his mind of the structures among which he has to find his way to the artery. "I always determine in "my own mind," said the sculptor Chantrey, "the ex- "pression to be given, and unless I can see the face "distinctly and with that expression when I close my "eyes, I can do nothing."

Secondly, the surgeon must have a fair idea of the amount of force that will be required to effect his object of occluding the vessel without rupturing its coats. He should experiment with human arteries distended with water to the pressure of the blood.

Thirdly, he should hold himself responsible for the asepticity of the operation. The skin, the instruments, the "sponges," and the ligatures must all be rendered aseptic. If the handle of a scalpel is used for the separation of muscles it should not be the scalpel in the surgeon's hand, but another fresh from the carbolic solution.

Fourthly, he must make sure that the ligature is sufficiently strong for the purpose: the breaking of the ligature is a trying accident.

Fifthly, he must provide himself with an assistant with whom he is accustomed to work. The main duty of the assistant will be to hold the wound gently open with retractors. Every step of the operation can be and should be performed by the surgeon himself, sponging, clamping small vessels and tying them; indeed, none but the operator's fingers should touch the wounded surfaces.

Sixthly, as it is often impracticable to render aseptic the skin over the aneurism, or undesirable to attempt to do so, it is well to bespeak the presence of some trustworthy friend, who shall place his hand over the aneurism just before the ligatures are tightened and inform the operator when all pulsation has ceased.

Lastly, for sponging we ourselves prefer cotton wool pledgets which have been previously soaked for some hours in mercuric perchloride solution (1 in 1000). The wound should be irrigated with perchloride, which causes less serous oozing than carbolic solution. It is of the first importance, if possible, not to divide muscular fibre or use a drainage tube. In this way primary union may be confidently hoped for.

With respect to the actual performance of the operation, the only subject that calls for remark is the passing of the ligature. If silk or catgut is used, the surgeon will pass the needle empty and thread it with a piece of silk long enough to make two ligatures when the loop is divided to free the needle. He should watch which way the ligature passes, so that there may be no question as to which end belongs to which, or whether the ligatures are crossed behind the artery.

If kangaroo tendon or peritoneal ligature is employed,

the surgeon will probably not have a sufficient length to divide into two ligatures. After passing the first, it will be quite easy, by lifting the artery a little with it, to pass the unarmed needle a second and, if need be, a third time.

When the operation is thus conducted, our belief is that hemorrhage will not occur. Even if the surgeon should unfortunately have failed to keep the wound aseptic, there is far less danger of hemorrhage than if the coats had been ruptured.

If a kangaroo tendon or peritoneal ligature is used, the result of sepsis will be, not the division of the vessel, but the more rapid absorption of the ligature, causing a danger, not of hemorrhage, but of repatency. If silk be employed, it may be discharged thus involving the division of the vessel but if this happens it will be later than if the coats had been ruptured, and therefore when there is more chance of the vessel being closed.

Sepsis and the discharge of a silk ligature was the recognized course of a case in the old days.

In the four cases of his operation in which Hunter used a single ligature, and in all of which he did not intend to injure the vessel, the ligatures separated in one on the eleventh, in two on the fourteenth, and in one on the twenty-ninth day (page 330).

Lynn, a pupil of Hunter's and surgeon to the Westminster Hospital, ligatured a superficial femoral for popliteal aneurism: he used a broad ligature, doubtless with the object of not rupturing the coats: the ligature separated on the thirteenth day and the patient did well.

Earle, of St Bartholomew's Hospital, had, in 1792, a

case of rapidly increasing aneurism in the lower part of the popliteal space. "Having noticed with much satisfac-"tion the success which attended Mr Hunter's method in "a similar case, I decided in favour of that operation, but "as in the present instance the artery appeared to be in "its natural and perfect state in the ham, till it reached "the dilatation below the knee, I determined to take it up "in that part rather than to tie it in the middle of the "thigh. * * * Having discovered it I passed a liga-"ture round it, about two inches above the tumour; I "now again examined, and being convinced that the artery "was included alone in the ligature, I gradually made it "tight till I felt a pulsation above it and none below, "when I desisted, concluding that any pressure beyond "this degree would be useless and dangerous." No further pulsation occurred: the ligature separated the fifteenth day, and at the end of six weeks a small fluctuating swelling alone remained to mark the site of the aneurism.

Scarpa in his earlier days did not remove his ligature, but allowed it to discharge itself. He says: "Experience "has demonstrated to me that the ligature applied in the "first way [without rupture and with a pad under the "knot] is not detached from the femoral artery till the "eighteenth, twentieth, twenty-first day, very rarely or "never before the fourteenth day after the operation; "while it is well known that the ligature applied upon "the same artery in the ordinary way quits its hold most "frequently the seventh or tenth day; a period at which "surgeons are usually on their guard from fear of a "secondary hemorrhage."

If the coats are ruptured and the wound becomes

septic there is grave risk. On the same shelf in one of our London museums are two specimens of popliteal aneurism, obtained within recent years from cases in which the superficial femoral had been ligated, and in which the patient had died of hemorrhage.

The following cases illustrate some of these risks; they are hemorrhage, secondary aneurism and repatency.

There is a case, related by Holden, of ligature of the superficial femoral with carbolic catgut; on the sixth day a discharge of thin greenish pus from the wound was noticed, and on the eighth day a sudden and fatal hemorrhage occurred. It was found that the ligature had divided the two inner coats, and that at the point of ligature there was a small perforation with jagged edges in the wall of the artery. The ligature had completely disappeared. The opening in the artery was probably where the knot had been.

Sir James Paget has shewn us notes of a case of axillary aneurism: the third part of the subclavian was ligatured with silk: the wound suppurated. After seven attacks of secondary hemorrhage the patient died on the sixty-fifth day. At the autopsy it was found that the hemorrhage had occurred from the distal side, and that a small secondary aneurism had formed there.

An interesting case of popliteal aneurism is related by Butcher of Birkenhead. The patient was admitted in 1885 and was treated by pressure with weights: the aneurism was improved but not cured. He returned a year later and the artery was tied with a silk ligature, the coats being ruptured. In thirty hours a recurrence of pulsation was noticed, and the artery was on the second day

religatured one inch (25 mm.) higher up: all pulsation now ceased, but six days later secondary hemorrhage occurred; the artery was again ligated, but the patient died. Butcher attributes the return of pulsation after the first ligation to the freedom of the collateral circulation caused by the previous treatment by pressure. But if this had been the cause, it is not clear why religature one inch higher up (and "below the profunda") should at once have stopped all pulsation in the aneurism. It seems possible that the first hitch of the silk ligature slipped, as in Brenja's case (p. 386), and this although the coats were ruptured, and that the artery thus became again patent at the seat of ligature.

Rivington records a case of repatency. He ligatured the external iliac artery with stout catgut for a femoral aneurism: suppuration occurred in the wound, and the patient died five months later of hemorrhage from the aneurism. It was found that the canal of the external iliac was completely restored, there was no trace of the ligature, and its former site could only be recognized by a line across the artery. On holding the artery up to the light, it was seen that it was thin where the ligature had been applied. (Compare figure 199, page 413.)

Staff-Surgeon Elliott, R. N., relates the case of a marine, aged 32, suffering from a femoral aneurism. The external iliac was ligated with the largest sized carbolic catgut ligature: on the fifth day faint pulsation was detected in the sac, which increased considerably during the next few days. Three months later the artery was religatured with a hempen ligature, one end of which was brought out of the wound; slight pulsation was felt in the

sac on the second day but it passed off: the ligature separated on the thirty-ninth day. Faint pulsation recurred on the eighty-seventh day, but again disappeared in a few days, the aneurism being cured.

In this case it appears that the sole cause of trouble was the too rapid absorption of the carbolic catgut ligature, which seems to have given way by the fifth day: it was therefore a case of temporary ligation, in which the ligature was removed on the fourth day. If it had been a sacculated aneurism it would probably have been cured, as were Scarpa's cases, but being a fusiform aneurism, the ligature held for too short a time.

There is a case of ligation of the common carotid artery with carbolic catgut recorded by Spence: he took great care to prevent the first half of the knot slipping. The patient did well for twenty-four hours, but then suddenly became comatose and died the next day but one. At the autopsy it was found that the ligature was pulpy, and that there was no constriction of the artery; there was an embolic clot in the middle cerebral artery, which Spence thinks must have come from the proximal side of the ligature, it being allowed to pass in consequence of the softening of the ligature. This case illustrates the danger of temporary ligation. (See page 286.)

Further, it must be remembered that the rupture at the time of ligation may not be confined to the two inner coats, but that the outer coat too may be ruptured, and the whole wall therefore cut through. (See Fig. 198, page 412.)

In the St George's Hospital Reports will be found a

case of popliteal aneurism, in which the superficial femoral was ligated with silver wire. Secondary hemorrhage occurred on the tenth day and proved fatal.

The artery was found "cut through almost in its entire "calibre."

A similar effect of the ligature is related by Bryant—

"The patient, 29 years of age, was admitted into Guy's "Hospital with ulcerative endocarditis and an aneurysm "of the right common femoral artery. The aneurysm "burst, and as the man's life was threatened from con- "cealed hæmorrhage, I was induced to apply a ligature to "his external iliac artery. The operation, however, failed "to save life, and the man died from heart-affection four- "teen hours subsequently. In the operation I employed "a medium-sized carbolized catgut ligature, and did not "employ more force than I thought sufficient to occlude "the vessel: and what do we find? That the inner and "middle coats of the artery are completely divided and "the external coat partially so; that is, it is completely "divided in parts. A fair amount of clot exists on both "sides of the ligature, and the ligature itself is intact."

Finally, putting aside all experimental work and relying only on the records of human surgery, it cannot be gainsaid that the method of rupture leads, with certain arteries, almost inevitably to the dread sequel of hemorrhage and death; and further, that the rise of Listerian surgery has not abolished the danger. The choice is therefore before us of abandoning further progress in the surgery of the arteries or of adopting that way of ligation which leaves intact the arterial wall. Con-

sidering the extreme thinness of the wall during life this method seems reasonable: and Nature, too, in the occlusions she effects at birth and in disease, appears to point in the same direction. Moreover subsidiary questions, such as the amount of clot formed, the presence of atheroma, and the nearness of the ligature to a collateral branch, about which so much discussion has arisen among the advocates of the cutting ligature, sink into insignificance when the wall is left whole.

The first indication of hemorrhage is, in many cases, a slight stain on the dressing or discolouration of the discharge. The temptation is to attribute this to bruising of the granulations, or the giving way of some minute vessel. If it increases and is to prove the precursor of hemorrhage, it is clear that now is the time to operate. Otherwise the next thing will be a severe hemorrhage; after the patient has sufficiently recovered from the collapse, the wound must be reopened, and the vessel religated above and below.

For the collapse caused by the loss of blood, the best treatment, as William Hunter has shewn in his recent lectures on transfusion, is the injection of a saline solution.

This is far better than the transfusion of blood, a procedure which is not without considerable danger and difficulty.

The best solution to use for injection is a 0·75 per cent. solution of chloride of sodium. This is about one drachm to a pint, and as distilled water is not necessary the solution can be made in any house. The simpler the apparatus to be used for injection the better; a glass

cannula to be introduced into one of the veins at the bend of the elbow, a short piece of india-rubber tubing and a syringe are all that is necessary. Care must be taken not to inject any air.

The fluid should be at the temperature of the blood, but it had much better be three or four degrees below than the least over that temperature.

The amount of fluid injected should be from one to three pints (600—1800 cc.), or even more, continuing till a good effect is produced. Its main use is to replace the bulk of the blood lost, for the danger from hemorrhage seems to arise from interference in some way with the mechanism of the circulation; there are said always to be plenty of corpuscles left to carry on the respiratory functions of the blood.

The records of the ligation of great arteries contain not a few cases of recovery after hemorrhage, bearing brave witness to the untiring care of many an unnamed nurse, to the ready resource of many a forgotten dresser; but in the majority of cases of hemorrhage, care and skill are unavailing, and the surgeon arrives on the scene only to find that he is too late, too late for all but remorse.

Note 1 (page 516). Messrs Macfarlan have recently made peritoneal ligatures for us from gold-beater's skin. They are very smooth, strong, 30 inches in length, and appear admirable. We have however not yet tested them by experiment.

Note 2 (page 524). Successful cases of the intravenous injection of saline solutions for the collapse caused by hemorrhage have been recorded during the course of the last few years by Jennings[1], Coates[2], Szuman[3], Brown[4] and Lane[5]. In one of Coates's two cases only water was injected. Lane's case was for hemorrhage after a cleft palate operation in a girl of thirteen. Three and a half pints of a saline solution were injected and the patient who was pulseless and apparently moribund improved "in a marvellous manner." The first pint injected produced no obvious effect.

In 1872 Wagstaffe[6], for the collapse due to the hemorrhage arising from severe injuries, injected a pint and a half of water to which a little milk had been previously added: the patient temporarily improved but later died of collapse.

[1] *Lancet*, Sept. 1882.
[2] *Ibid.* Dec. 1882.
[3] *Ibid.* June, 1883.
[4] *Lancet*, March, 1889.
[5] *Ibid.* Sept. 1891.
[6] *Obstetrical Journal*, 1874.

APPENDIX A.

BLOOD PRESSURE IN ANEURISMS.

On page 427 the tension in the walls of arteries and aneurisms is discussed: it was assumed that the blood-pressure in an aneurism is the same as that in the artery above; this would be true if the blood was stationary but it being in motion alters the problem materially.

Robinson in his *Hydraulic Power* (1887) in speaking of the flow of water under pressure says: "When the pipe ceases to have a uniform "internal diameter, important fluctuations of velocity and pressure "arise, which are too frequently forgotten. A sudden enlargement or "reduction in a pipe produces eddies in the current, which result in a "corresponding diminution or increase in the velocity, and therefore "in the pressure. A vein of water flowing at a uniform velocity is "influenced throughout its mass by a uniform pressure acting upon it "from behind. If this vein suddenly reaches an enlargement in the "pipe, the velocity will be diminished, as the velocity in a pipe varies "inversely with the area. The reduced velocity through the enlarged "portion of the pipe implies that a force is opposing the forward "movement of the water; or in other words, that the force from "behind (which produced the forward movement) meeting a force in "front which arrests or diminishes it, implies that a pressure must be

"produced in front of the fluid increased beyond that which existed "at the time it was flowing in the uniform portion of the pipe. "Similarly, when a vein of water meets a contraction in a pipe, the "diminished sectional area of the pipe necessitates an accelerated "velocity proportional to the reduced area. This shews that the "pressure behind the fluid is greater than that in front, and con- "sequently the pressure throughout the length of pipe which has the "reduced area is less than it was before, in proportion to the extent "of the reduction.

"It follows, therefore, that in a system of pipes of varying areas, "in which a fluid is circulating, the pressure that is exerted by that "fluid will vary at any point in inverse proportion to the velocity "at that point, or in other words, to the sectional area at that point. "In the lengths of pipes where the sectional areas are the same, there "will be found the same pressures (friction not being considered). "Where the areas are greatest, there will be the greatest pressures, "and where the areas are least, there will be the least pressures. "Experiments by Mr Froude prove this. He inserted a series of "vertical glass tubes in a horizontal length of pipes which had "enlargements and contractions in them. It was seen that the heights "to which the fluid rose when it was flowing through the pipes varied "(with a uniform head) as the area of the pipe varied. The tube "which was placed over an enlargement in the pipe had a higher "column standing in it than was the case with the tube which was "placed over a contracted length * * * In other words, a pipe "filled with water and subjected to a head, has a greater pressure in "it when the water is at rest than when the water is in motion; "again, it has a greater pressure when the velocity is checked by an "enlargement in the pipe, and has a less pressure when the velocity "is accelerated by a reduction in the pipe."

The paper by Froude referred to was his Inaugural Address to the section of Mechanical Science of the British Association at its meeting at Bristol in 1875. It will well repay perusal as will also Robinson's chapter "On the Flow of Water under Pressure" from which we have just quoted.

FIG. 229 ($\frac{1}{8}$th scale).

The glass sphere at the lower part of the drawing with the tubes on each side of it is intended to represent an aneurism with the artery above and below it: a current is maintained by water flowing from the reservoir on the left through the sphere and up through the tube into the tank on the right: it is clear that the level to which the water rises in the tubes Nos. 1, 2 and 3, will give the pressure of water in the tube before the sphere, in the sphere, and in the tube beyond the sphere respectively: the tubes at the base were of about the same internal diameter as the human common carotid, and the level at which the water is drawn in the tubes is what it stood at when the outflow at *B* was equal to the flow of blood through the carotid during life.

The dotted line, crossing the manometers, is horizontal: below it the continuous oblique line shews the height at which the water in the middle tube would have stood at if there had been a tube instead of a sphere at its base. (See page 530.)

The effect of closing the clamp *A* was to cause a fall in all three tubes, but it occurred most rapidly in that connected with the sphere, similarly opening *A* and closing clamp *B* caused a rise in all the tubes, but it was most rapid in No. 2 tube.

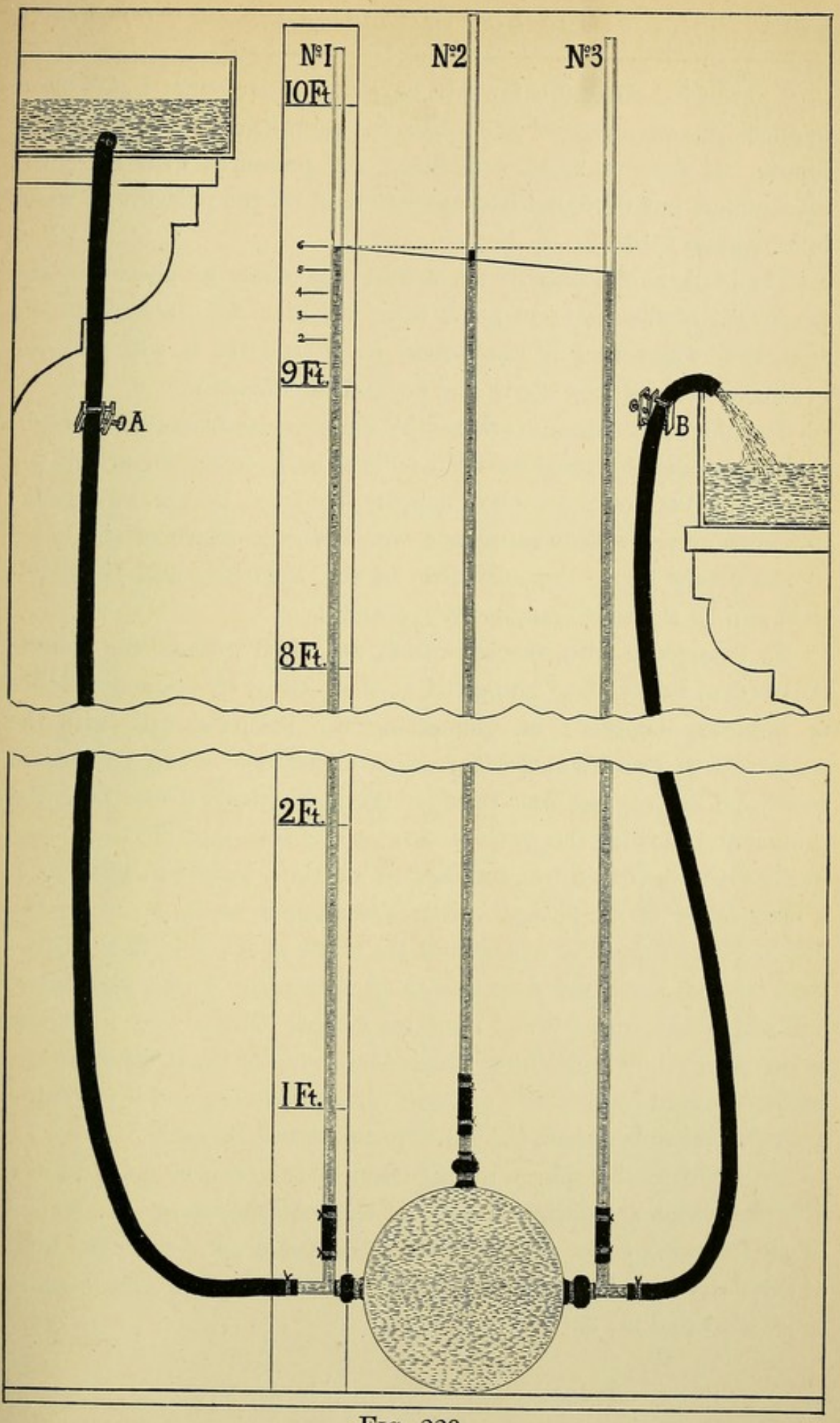


FIG. 229.

To see what effect this law will have at the pressures which occur in aneurisms we arranged the apparatus shewn in the accompanying figure. At first we tried estimating the pressures with mercurial manometers but we found it simpler to read off the pressure of water in long glass tubes.

The globe at the base of no. 2 tube represents an aneurism, and the height of the water in no. 2 tube will give the pressure in the aneurism, while that in the tubes, no. 1 and no. 3, will give the pressures in the artery above and beyond the aneurism.

The flow of the water is effected by the pressure from the reservoir, this represents the pressure of the blood as it enters the artery, the circulation beyond the artery being represented by the exit tube; but what we are mainly concerned with is the pressure of the blood in the artery above the aneurism, in the aneurism, and beyond it as shewn by the tubes numbered 1, 2 and 3.

To commence with, the apparatus was tried without the sphere, the vertical tubes being connected at their bases by horizontal tubes of uniform diameter: on connecting the left-hand tube with the water tap of the laboratory, giving a pressure of about 25 feet of water and holding the exit tube on the right about 8 feet from the ground and allowing the water to flow, it was seen that the water rose in the three vertical tubes, standing at a certain height in tube no. 1, a little lower in no. 2, and a little lower again in no. 3: where the height of the exit tube was altered the levels of the fluid in the tubes were altered, but it was seen that in no. 2 tube the height was always half way between that in no. 1 and no. 3 tube. This is only what was to be expected, because the gradual diminution of the pressure in the tubes is caused by the 'loss of head' due to friction and if the tubes are equal distances apart the fall of pressure will be equal.

On inserting the sphere or aneurism at the base of no. 2 tube a different result was obtained, some of the experiments are embodied in the following table. It will be noticed that in all of them the level of the water in tube no. 2 was higher than half way between those in tubes no. 1 and no. 3.

Table shewing result of experiments on pressure in aneurisms.

Pressure of water.	Height of water in tube				Outflow during 1 minute
	No. 1	No. 2	No. 3	Exit tube	
About 25 feet (tap)	10 ft. 1 in.	10 ft. 5 in.	9 ft. 6 in.	7 ft.	—
„ „ „	4 ft. 8 in.	5 ft. $2\frac{1}{2}$ in.	3 ft. 7 in.	not raised	—
„ „ „	5 ft. 3 in.	5 ft. $3\frac{1}{8}$ in.	4 ft. $8\frac{1}{2}$ in.	not raised	4700 c.c.
16 feet (reservoir)	9 ft. $2\frac{3}{4}$ in.	9 ft. $2\frac{3}{4}$ in.	9 ft. $1\frac{3}{4}$ in.	7 ft. 2 in.	1900 c.c.
„ „	7 ft. $7\frac{7}{8}$ in.	7 ft. $7\frac{7}{8}$ in.	7 ft. 6 in.	5 ft. 7 in.	2500 c.c.
„ „	3 ft. $0\frac{1}{8}$ in.	3 ft. $0\frac{1}{8}$ in.	2 ft. $9\frac{1}{2}$ in.	$10\frac{1}{2}$ in.	3940 c.c.
11 feet „	10 ft.	10 ft.	9 ft. 10 in.	9 ft. 6 in.	1550 c.c.
10 feet „	9 ft. 6 in.	9 ft. $5\frac{7}{8}$ in.	9 ft. 5 in.	9 feet	750 c.c.
9 ft. 7 in. „	8 ft. 7 in.	8 ft. 7 in.	8 ft. $5\frac{1}{4}$ in.	6 ft. 8 in.	1400 c.c.

Michael Foster in his *Physiology* (Part 1, page 220) says: "In a "large artery of a large animal such as the carotid of a dog or horse "and probably in the carotid of a man the blood flows at the rate of "300 or 500 mm. a second." The diameter of a human carotid is about 8·5 mm., the area of its transverse section would therefore be $3{\cdot}14 \times 4{\cdot}25^2$, in one second there would pass through this artery at the rate of 400 mm. a second 22608 cubic mm. or 1356·5 cc. per minute.

In the last experiment in the table the exit tube was adjusted to give about this rate of flow: and as the tubes were of the above internal diameter (8·5 mm.) the difference in the pressures in the tubes 1, 2 and 3 would roughly correspond to the difference in the pressures in a carotid with a large aneurism: now the difference between 1 and 3 was $1\frac{3}{4}$ inches, therefore no. 2 would have stood had there been no aneurism at its base $\frac{7}{8}$ inch below no. 1, but it stands at the same level as no. 1, therefore the presence of the aneurism causes an increased pressure of about 1 inch of water or less than 2 mm. Hg, a pressure which may be disregarded.

But the pressure in a human artery is not constant, it varies in a large artery about 30 mm. Hg. during a cardiac cycle. The effect of a change in the pressure in our apparatus is very interesting: a sudden reduction of pressure caused by closing the clamp A caused of course the fluid to fall in the manometers, but the fluid in the tube over the glass sphere fell much the most rapidly: on unclamping the inlet tube the fluid rose in tube no. 2 the most rapidly; thus it would seem that any variation in the pressure of the blood in the carotid would produce more effect, and be more severely felt in the aneurism than in the artery above or beyond.

The eccentric pulsation that is so well known as a symptom of aneurism and the friction produced by the eddies and obstruction by clot in the aneurism causes a marked reduction in the arterial pressure beyond, which is often easily recognized at the bedside, constituting an important diagnostic symptom.

APPENDIX B.

THE BLOOD CURRENTS IN ANEURISMS.

When water falls over a weir into an expanded portion of a river it rapidly travels down the centre of the stream, but on each side there are eddies and back-currents which may be seen to carry floating objects upwards towards the weir again: here and there too are little bays along the banks in which the water is motionless or nearly so.

It is easy to see that the blood in aneurisms behaves in a similar way. If a glass sphere with two openings in it on opposite sides be attached by tubing to a water tap and solid particles of the same specific gravity as the water be placed in the sphere, when the water is allowed to flow the movements of the particles will shew the various currents in our aneurism. (See figure overleaf.)

Some of these currents are represented in the accompanying diagram. For this experiment rape seed will do, but what seems best is small balls of cork so covered with modelling wax that they neither sink nor float.

The currents may also be well shewn on the screen, when carbon particles are suspended in the water by throwing a strong light through the sphere.

In figure 147, page 310, it is clear that the aneurism has ruptured at that point in its wall which is opposite the opening in the aorta and which had to bear the main stress of the stream of blood. It will be observed too that it is in the back-currents that clot has formed.

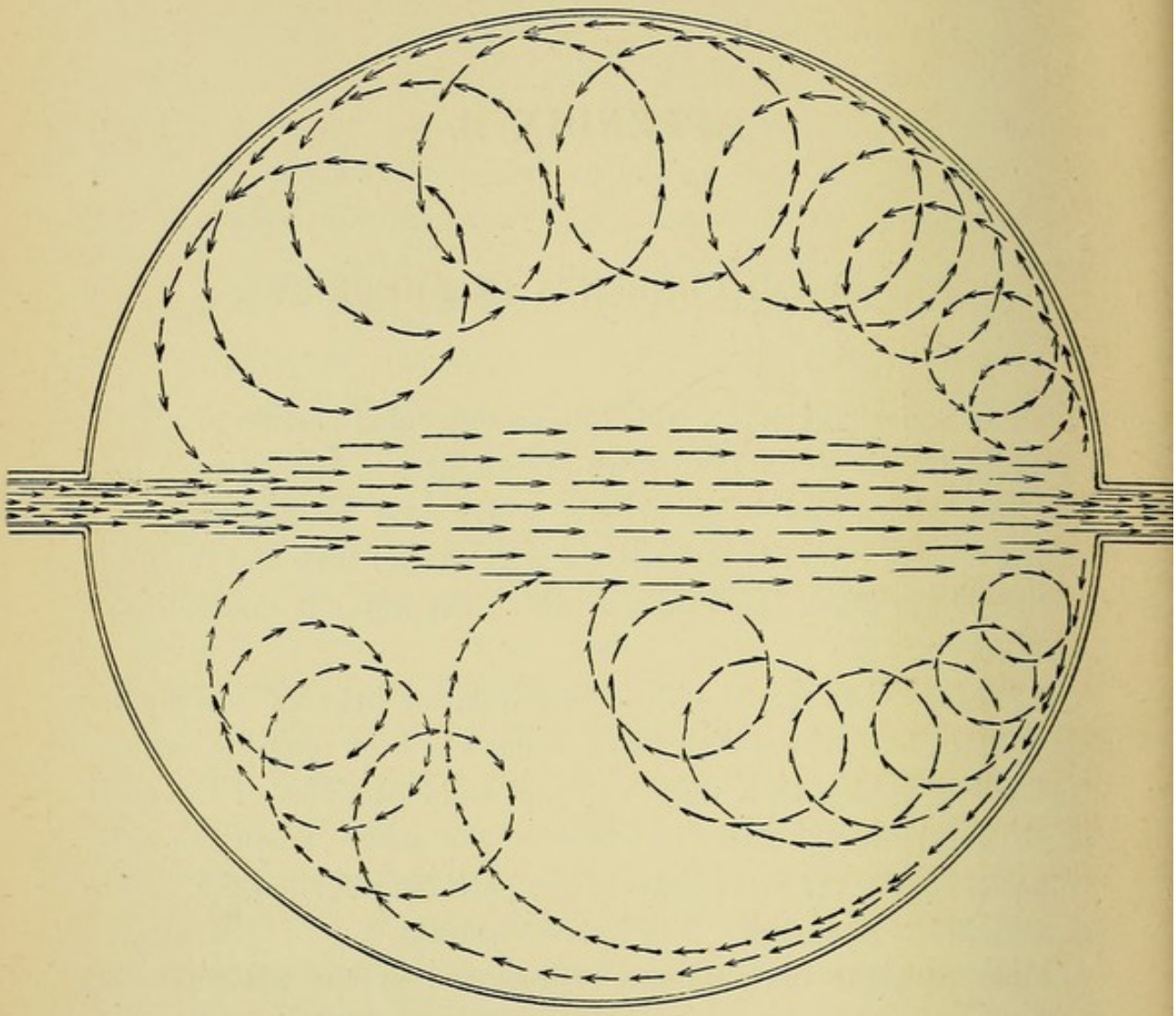


FIG. 230. Diagram to shew the course of blood in aneurism.

The blood is supposed to enter on the left: it then passes straight across the aneurism and some passes out through the opposite opening: the rest returns in eddies round the sides: the arrows and circles represent the varying courses which particles suspended in water can be seen to take: later they re-enter the central stream. Close to the surface of the aneurism were very small eddies. The only effect of making the inlet tube extend into the centre of the aneurism was to make the fluid behind the entrance more stagnant.

Besides the main eddies shewn in the diagram there are others which keep in contact with the inner surface of the glass.

APPENDIX C.

CHOICE OF THE ANTISEPTIC.

In the early days of antiseptics carbolic acid was mainly used, but later Koch conducted an elaborate series of experiments which seemed to shew that perchloride of mercury was a more powerful disinfectant. (*Mittheilungen aus dem Kaiserlichen Gesundheitsamte*, 1881.)

Koch's method was as follows: he infected silk ligatures with sporulating anthrax cultures, and placed the various agents to be tested in separate test-tubes, he then dropped pieces of his silk into the test-tubes, and left them there for a certain time. The silk was then removed and placed on a culture medium.

The results of the experiments with carbolic acid were as follow:

1 per cent. watery solution, no action after 15 days' exposure.
2 „ „ „ „ slight retardation of growth.
3 „ „ „ „ killed spores in 7 days.
4 „ „ „ „ „ „ „ 3 „
5 „ „ „ „ „ „ „ 1 „

This slight action of carbolic acid on spores is quite different, however, to its action on anthrax bacilli devoid of spores.

The fresh blood of an animal dead of anthrax contains bacilli but no spores, if it is mixed with an equal bulk of 1 per cent. carbolic acid, it will not give anthrax when injected, if with 0·5 per cent. solution of carbolic acid it will: therefore the limit lies between 0·5 and 0·25 per cent. for the mixture.

Koch found that 1 per cent. aqueous solution of corrosive sublimate killed anthrax spores in 1 or 2 days, therefore as far as anthrax spores are concerned corrosive sublimate is the better.

In the experiments on *hindering* growth various disinfectants were added to blood serum and the anthrax silk was then put into the mixture.

With carbolic acid	1 in 1250	hindered growth of anthrax bacilli.
„ „ „	1 in 850	prevented growth of anthrax bacilli.
„ corrosive sublimate	1 in 1,000,000	hindered growth of anthrax bacilli.
„ „ „	1 in 300,000	prevented growth of anthrax bacilli.

Crookshank disapproves of the method of using silk threads infected with anthrax because he thinks the antiseptic might be acting (the silk being wet) for some time after the silk was transferred to the culture medium. He made cultures in broth, and when the culture had settled at the bottom of the test-tube the supernatant liquid was carefully poured off. The antiseptic solution was then poured in. After the solution had acted for the stated time a fluid was added which would at once stop the action of the antiseptic—ammonium sulphide, as suggested by Geppert (*Berliner Klin. Woch.* 1889) being used in the case of corrosive sublimate. The results were not at all in favour of corrosive sublimate. Crookshank concludes by strongly recommending a solution of carbolic acid (1 in 50) for use in antiseptic surgery in preference to any solution of corrosive sublimate.

Max Gruber also objects to the silk thread method, and especially to the plan of testing such threads by inoculating them into animals. This latter plan, however, appears from the surgical point of view to be of quite peculiar fitness. In Max Gruber's experience carbolic acid, lysol and kresole were the best antiseptic agents.

Armand Ruffer relates experiments which he had conducted with

a view of determining the effect which various antiseptic agents have of favouring or hindering the cellular invasion in areas of aseptic inflammation. For this purpose he introduced subcutaneously small pieces of sterile sponge saturated with the antiseptic to be tested. "Generally speaking, the chemical substances which combine readily "with the salts and albuminoid bodies of the blood, e.g. carbolic acid "and sublimate, do not repel the cells so energetically or for so long "as those which do not readily combine, e.g. aniline, benzol, etc."

Behring, in his remarkable paper on "disinfection in the living body," points out the various ways in which disinfection may be effected. He tells how the fatal result of anthrax infection in mice may be postponed or prevented by the *local* injection of a mixture of corrosive sublimate and chloroborate of soda; and how with neither solution alone are the same results obtainable. The *local* injection of a solution of trichloride of iodine has been shewn to be similarly efficacious in tetanus and diphtheria. The blood of animals rendered immune from these diseases was still more effective [containing the defensive proteids of Hankin], and it was not necessary in this case to inject near the seat of inoculation.

These results seem to be of great importance, for antiseptics no doubt act not only by killing the micro-organisms, but also by rendering the wound for a time immune.

The experiments of Crookshank, Max Gruber, Ruffer, and Behring, were communicated to the International Congress of Hygiene at its session in London in 1891, and will be found in its *Transactions*.

Abbott in the *Bulletin* of the Johns Hopkins Hospital of Baltimore (April, 1891) contributes a paper on corrosive sublimate as a disinfectant against the staphylococcus pyogenes aureus. After referring to the work of others, he narrates his own experiments, which seem to have been carried out with the greatest accuracy and care. He concludes that the disinfectant process is a chemical one, taking place between the protoplasm of the individual bacteria and the sublimate in the solution, that the organisms which survive exposure to the sublimate experience a temporary attenuation, that although few substances possess higher disinfectant properties than sublimate

solution, yet against staphylococcus pyogenes aureus its disinfectant property is not so great as was supposed, and that in surgical practice sublimate solutions do not possess all of the advantages hitherto attributed to them.

In the year 1887 when perchloride of mercury was coming into general use, one of us carried out with Dr Sherrington a series of experiments with the view of determining the result of adding $HgCl_2$ in aqueous solutions of various strengths to fresh warm blood-serum: one idea being of finding a solution of mercury which might be injected subcutaneously in the treatment of syphilis without producing local induration, the *antiseptic filtrates* mentioned below would probably answer this purpose. It was found

1. That the addition of a small quantity of $HgCl_2$ to blood-serum produces a fine white precipitate, which did not as a rule appear to redissolve. The filtrate is clear, it contains albumen and is antiseptic.

2. The previous addition of a trace of HCl to the $HgCl_2$ solution either prevents precipitation, or, if it occurs, the precipitate is coarse and flocculent, and is easily dissolved by agitation.

3. In all the experiments the precipitates, the filtrates and the clear solutions remained unchanged and free from growth, though exposed to the air for weeks.

As an example the following experiments may be mentioned:—

A. 5 c.c. blood-serum + 5 c.c. (5 c.c. $HgCl_2$ 1 per cent. + 5 c.c. HCl 12 per cent.) slight precipitate dissolved in excess.

Do. + 5 c.c. more, no permanent precipitate, the solution perfectly clear.

Do. + 5 c.c. 1 per cent. $HgCl_2$—no permanent precipitate.

B. 5 c.c. blood-serum + 5 c.c. 0·5 per cent. $HgCl_2$. Slight precipitate dissolved in excess.

Do. + 5 c.c. more. Slight precipitate dissolved in excess.

Do. + 5 c.c. 1 per cent. $HgCl_2$. Slight precipitate apparently permanent.

To test the antiseptic power of the filtrates, $HgCl_2$ solution was added to serum to an amount just short of producing the maximum precipitate. The precipitate was then filtered off, leaving a clear solution. As examples of the experiments the following may be cited—Four tubes of nutrient gelatine were then liquefied by being placed in the incubator at 37°·5 C., and then each was inoculated by a platinum needle from a culture of the bacillus of green pus. To three of the tubes some of the above filtrate was added, with the following result:

Tube	Amount of filtrate added	Condition after 24 hours at 37°·5 C.
A	None (control)	green
B	one drop	green
C	two drops	green
D	five drops	unaltered

Five drops of the filtrate, therefore, were sufficient to prevent the growth of grün Eiter in a tube of nutrient jelly.

In another experiment blood serum was used instead of nutrient gelatine—the tubes being infected as before.

Tube	Contents of tube	Result at end of 48 hours at 37°·5 C.
A	Blood Serum + Nil (control)	green
B	4 c.c. blood serum + 3 c.c. "filtrate" as before	unaltered
C	5 c.c. blood serum + (2·5 c.c. of 0·75 per cent. $HgCl_2$ + 2·5 c.c. of 33 per cent. HCl) (clear solution)	unaltered

Owing to the importance of the subject we asked Mr White the pharmaceutist to St Thomas's Hospital to reinvestigate the subject. This he did and has embodied his results in the following table:

Table prepared from Mr White's Experiments:

Number of Experiment	Proportions in which blood serum (ox or sheep) and sublimate solution were mixed		Amount of perchloride in filtrate[1]	More serum gradually added to filtrate	More perchloride added to filtrate
	corrosive sublimate solution (1 in 1000)	Serum			
1.	100	1	1 in 1250	copious precipitate	no precipitate
2.	100	2·5	1 in 1330	precipitate less than Exp. 1	„ „
3.	100	5	1 in 2000	„ „ „ Exp. 2	„ „
4.	100	10	1 in 4000	„ „ „ Exp. 3	„ „
5.	100	15	1 in 6250	precipitate very slight	„ „
6.	100	20	1 in 5880	no precipitate	„ „
7.	100	30	1 in 2850	„ „	„ „
8.	100	40	1 in 2220	„ „	„ „
9.	100	50	1 in 2000	„ „	„ „
10.	100	100	mixture clear	—	—

[1] Estimated by comparison with the colouration produced by ammonium sulphide in solutions of known strength.

The filtrate (No. 5 expt.) containing in solution 1 in 6250 $HgCl_2$ was dialysed. The dialysate shewed no traces of $HgCl_2$ after 3 days.

The solution and precipitates exposed to the air for days and weeks did not decompose at all but remained unaltered except for evaporation.

It is clear from these experiments that the precipitate (Albumen + $HgCl_2$) formed when solution of $HgCl_2$ is added to serum is always in part at least soluble in the serum, and that thus even when there is a large excess of serum, $HgCl_2$ is found in the filtrate. That this mercury is in combination with certain constituents of the serum is shewn by the dialysis experiment: it is held by the colloid and so cannot pass through the membrane. In this state the mercury as has been shewn still acts as an antiseptic.

It is interesting to compare with these laboratory experiments the result of careful clinical observation. The records of the General Lying In Hospital during the seven years 1882 to 1889 have been analysed and tabulated by Boxall and communicated to the Obstetrical Society. During the whole of the seven years antiseptic precautions of one kind or another were systematically carried out at the hospital: in 1882, 1883 and part of 1884 carbolic acid and Condy's fluid were used: in 1884 corrosive sublimate was substituted for these and the change was accompanied by a marked decline in the frequency of cases of pelvic inflammation and of cases of febrility.

In 1888 owing to some symptoms in one or two cases of mercurial poisoning Salufer was used instead of perchloride: (salufer is a silico-fluoride of soda which has been highly spoken of as a disinfectant). The death and febrility rate immediately rose, and after eight weeks salufer was given up and the sublimate again used, then the health of the hospital at once improved. Although these experiences are not conclusive as against carbolic yet they shew the great value of perchloride as an antiseptic.

For our own part, as has been already said, we think the best practice is to use perchloride for irrigation: the application of a strong carbolic solution causes a whitening of the surface of the wound due to its caustic action: this in itself must be injurious and

accounts for the large amount of serous oozing which occurs in wounds thus treated: this oozing is very objectionable, for it necessitates in many cases the use of a drainage tube: the best dressing for wounds seems to us to be Lister's cyanide of zinc and mercury gauze wrung out in carbolic acid solution (1—40), the use of perchloride solution for this purpose is apt to cause irritation of the skin.

Supposing a wound made for, say an amputation to be irrigated with the perchloride solution, the perchloride will be in excess during the operation: when the flaps are brought together the serum will soon be in excess and there will be between the raw surfaces an antiseptic fluid,—serum containing perchloride in solution: and even if it were proved that the disinfectant action of this fluid was only sufficient to *hinder* the growth of organisms which may have gained a footing in the wound during the operation, yet it might still be the best disinfectant for use in surgery, for the invading cells are well known to be able to destroy bacteria in an inactive or quiescent state.

After all we cannot be guided in the use of disinfectants simply by the laboratory test as to which substance destroys anthrax bacilli or the streptococcus pyogenes most rapidly, but given the laboratory test that the substances in question are really good disinfectants, the clinical experience of the wards as to which of them is the best must be for surgeons the crucial test: the clinical experience of both of us is in favour of perchloride of mercury.

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INDEX.

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