pigment cells, even when situated in the capillaries of the liver as the ordinary seats of formation of bile pigment, and pointing away in a corresponding degree from passive destruction of red corpuscles as the source of the hæmoglobin thus transformed. I have now to draw your attention to evidence pointing to the liver cells as the seat of this transformation, and to an active destruction of red corpuscles as a necessary precursor of this process, and that, too, not only in mammals, but also in birds. I have shown you that nothing more clearly indicates such an active destruction of red corpuscles than the presence of minute granules of blood pigment in the liver cells, more especially in those of the portal zone of the hepatic lobule. I have shown you, moreover, that, although not constant, such pigment is very frequently found in the liver cell, more especially in birds; to a much less extent in mammals.

It now remains for me to show, from my experiments, that an increased formation of bile pigments is more frequently associated with an increased deposit of blood pigment within the liver cells than it is with an increase of pigment cells within the capillaries. Further, that such an increased formation is frequently attended solely by an increase of blood pigment in the former situation, not a particle of pigment or a single pigment cell being discoverable within the capillaries. And lastly, that a large increase in the pigment cells within the capillaries can take place without any increased formation of bile pigments (Experiment 89). To illustrate the first two points I now show you a section from Experiments 42 and 43. Two healthy rabbits received at intervals doses of toluyldiamine as follows, the injections being made subcutaneously:

Experiment 42.

Sept. 19th ...	0.18	gramme per kilo.	of weight =	0.5	gramme.
„ 20th ...	0.13	„	„	= 0.35	„
„ 26th ...	0.18	„	„	= 0.5	„
„ 29th ...	0.36	„	„	= 1.0	„

When killed, on Oct. 13th, it had lost 3 oz. in weight.

Experiment 43.

Sept. 15th ...	0.18	gramme per kilo.	of weight =	0.5	gramme.
„ 19th ...	0.15	„	„	= 0.4	„

When killed, on Sept. 20th, it had lost 6 oz. in weight.

In both cases, especially in Experiment 42, you will note the great abundance of fine pigment within the liver cells, all of it giving the reaction of free iron. So small are the individual particles, indeed, that it is only with higher powers (300 or more) that their great number and their precise situation within the liver cells can be properly made out. In both cases, moreover, no less striking than its abundance in the liver cells is the absence of pigment cells from the capillaries. The bearing of these observations will be at once obvious when I state that, as will presently be seen, toluyldiamine causes an increased formation of bile pigments and distinct destruction of blood—viz., an acute destruction of red corpuscles. That the blood pigment within the liver cells owed its origin to the destruction of free hæmoglobin within these cells there can, as we have seen, be no doubt; and the conclusion may, I think, be permitted that it was likewise within the liver cells that the bile pigments so largely excreted were also formed. Evidence of a similar kind pointing to the same deduction was afforded by experiments on pigeons and observations on man. From these observations I infer that it is within the liver cells, and not in the capillaries, that hæmoglobin is transformed into bile pigment. The hæmoglobin thus transformed must have escaped from the red corpuscles before passing into the liver cell. In short, the source of the bile pigment is free hæmoglobin.

It will thus be seen that, although I do not absolutely deny to pigment cells of leucocyte and connective-tissue nature, such as we occasionally find in the capillaries of the liver, the power under certain circumstances of forming pigments similar to the bile pigments from hæmoglobin, I am undoubtedly of opinion, for the reasons above stated, that such a mode of origin of the bile pigments is altogether secondary in importance to their derivation from the liver cells. For not only are such pigment cells frequently entirely absent from the tissues of animals in which an undoubted, it may be even a largely increased, formation of bile pigments is taking place, but also, what is even more significant, a largely increased formation is frequently marked by an increase of the blood pigment in a situation where it can only, in my opinion, be derived from free hæmoglobin—namely, within the liver cell. Such an escape of hæmoglobin

from the red corpuscle into the plasma of the blood being, as we have seen, the chief feature of an *acute* as distinguished from a *passive* destruction of red corpuscles, the answer which my observations supply to the question with which we started is that: (1) the formation of bile pigments by the liver is subserved by an active destruction of red corpuscles and not a passive one; (2) a certain destruction of blood is therefore a phenomenon of daily occurrence.

ABSTRACT OF A Clinical Lecture

ON A

CASE IN WHICH SUCCESSIVE ANEURYSMS WERE TREATED BY LIGATURE OF FOUR LARGE ARTERIES.

By ALEX. O. MACKELLAR, M.Ch. Q.U.I.,
F.R.C.S. Eng. &c.,
SURGEON TO ST. THOMAS'S HOSPITAL.

GENTLEMEN,—The subject of our clinical lecture to-day is a case you are well acquainted with, the patient having been in the hospital three times and having undergone the ligation of no less than four of the main arteries for the treatment of aneurysms. He was first admitted on March 9th, 1888, suffering from a popliteal aneurysm with a large sac, which was rapidly increasing. He was thirty years of age, and by occupation a police constable; previously he had been a soldier in Egypt and also a professional runner, his best time being one hundred yards in ten seconds and a quarter. He had had venereal disease, but there was no certain history of syphilis. The aneurysm was of the right popliteal artery, and was attributed to slipping off the kerb, when he felt “something give way.” This was one month previous to admission. The superficial femoral artery was tied with a silk ligature. All pulsation at once ceased in the aneurysm, and the patient made a satisfactory recovery. He was readmitted a year later, suffering from an aneurysm of the other popliteal artery. There was no history of injury. On this occasion treatment by digital compression was tried for thirty-one hours, but no improvement resulted, and on Aug. 23rd the left superficial femoral was ligatured with silk. Pulsation in the aneurysm at once ceased, and the patient recovered. In February, 1892—that is to say, four years after his first admission—our patient was admitted for the third time; on this occasion he was suffering from a pulsating swelling, clearly an aneurysm, behind and above the manubrium sterni. In the previous November he had fallen from and under his horse. For this aneurysm the right common carotid and the third part of the right subclavian were simultaneously ligatured; this was on Feb. 22nd, 1892. The carotid was tied with silk and the subclavian was tied in two places and divided between. These operations seemed for a time to arrest the progress of the aneurysm, which subsequently gradually increased, and finally ruptured through the skin, the patient dying of hæmorrhage on May 19th, 1892. A minute examination of the ligatured vessels and aneurysms was made by Messrs. Ballance and Edmunds, but instead of reading their report it will, I think, be more instructive for us to go through the case again, discussing the various points that arise in the light of what we now know. The patient, then, was admitted suffering from popliteal aneurysm, which was treated by the operation first practised by John Hunter at St. George's Hospital in December, 1785. Before this, popliteal aneurysms were treated, if treated at all, either by amputation or by laying the aneurysm open, turning out the clot and ligating the two ends of the artery; this latter operation was almost invariably fatal. With a view to reduce this mortality Hunter devised his great operation; as you know, he tied the femoral in Hunter's canal, and this alone sufficed to cure the aneurysm. He had himself lost an apparently favourable case of popliteal aneurysm treated by the old operation from hæmorrhage, and he contrasted this with the freedom from hæmorrhage after amputation. “Why not then,” he asks, “tie the artery where it can be safely tied in amputation and save the limb?” This he did, and the procedure has constituted one of the greatest

advances in surgery that it has ever fallen to the lot of one man to make. Hunter himself saved four out of the five cases on which he operated, and other surgeons were equally successful. French surgeons have claimed that Hunter's operation was merely a revival of that performed by Anel eighty years previously. Anel had been called on to treat a monk suffering from an aneurysm of the brachial artery accidentally caused by venesection; Anel tied the brachial immediately above the aneurysm, which was thus cured.

Now, the differences between Anel's and Hunter's operations are: First, that Anel's was on the arm and Hunter's on the leg; this is a great difference—for instance, the blood-supply of the arm after ligation of its main artery is much better than that of the leg; gangrene is very rare in the arm. A second difference is that Anel tied immediately above the aneurysm, whereas Hunter tied at some distance from it. From a pathological point of view Anel's may be the better procedure, but it is far easier for the surgeon to tie the superficial femoral artery than to seek the popliteal in the ham, in close proximity to a large pulsating tumour, a procedure which Anel, be it understood, neither practised nor recommended. It has sometimes been stated that the cause of the failure of the old operation was that the artery was diseased near the aneurysm. This, however, Hunter himself pointed out was not the case, and you will see the same in the specimens on the table. Another reason for the success of Hunter's operation has been given—namely, that by it a small current of blood is allowed to trickle through the aneurysm after the ligation of the artery, thus causing the deposit of laminated clot. You will remember that in neither of these popliteal aneurysms after the ligation was there the slightest pulsation; this is nearly always the case in popliteal aneurysms. Pulsation in them immediately after ligation is either due to a double superficial femoral, as in Bell's case, or to the artery being imperfectly occluded. Later recurrent pulsation may happen from excess of the collateral circulation, and this may be so extensive as to prevent the cure of the aneurysm. Failure from this cause is happily very rare; the treatment is rest, and if this fails religation nearer the aneurysm.

As to laminated clot, when it is found in a popliteal aneurysm after ligation it has been formed before ligation, and is not due to any trickling of blood through the aneurysm after ligation. On ligation the cavity of the aneurysm and the popliteal artery become filled with clot, which later is converted into fibrous tissue, the whole aneurysm being represented, as you see in the specimens before you, by a shrunken mass of fibrous tissue. The ligation which I used was silk boiled in a perchloride of mercury solution. Innumerable substances have been recommended for this purpose, but silk has this great merit—that it can be readily and certainly made aseptic by boiling, a treatment which catgut and other animal ligatures will not stand. Ever since arteries have been ligated surgeons have had to consider how tightly the ligation should be drawn; whether, in other words, the two inner coats should be ruptured or not. Paré, who reintroduced the ligation, endeavoured not to injure the vessel, and Hunter took the same view; but in the early part of the present century Jones in his classical work on hæmorrhage gave the result of numerous experiments, from which he concluded that the coats should be ruptured. It must be remembered, however, that in these days of antiseptics and absorbable ligatures the problem is widely different from what it was in Jones's time, when the ligation had to work its way through the vessel and be discharged externally. Jones's experiments, too, as he points out, were incomplete. He made no experiments without rupture of the coats; these Scarpa made a few years later, and he arrived at the opposite conclusion. So the controversy has gone on, the latest contribution to it having come from our hospital. For myself, I think it has now been conclusively proved, both by experiments on animals and experience in man, that the coats should *not* be ruptured; in arteries of any size it is unnecessary, in the case of the largest arteries it is to invoke the gravest risk of hæmorrhage. In trying not to rupture the coats one has to be careful not to fail to occlude the vessel. It is clear that with a reef knot the ends must be slackened for an appreciable time in order to make the second hitch, and this gives an opportunity for the pressure of the blood to redistend the loop of the ligation. Tying the second hitch tightly will not tighten the loop. To obviate this the so-called surgeon's knot has been devised and it was this knot that I used for the femoral that was first tied. This knot does not answer this purpose—indeed, some say it is inferior to a reef

knot. Another way of meeting this difficulty has been devised by Messrs. Ballance and Edmunds. The aneurysm needle draws a double ligature under the artery and if the loop is divided two ligatures lie side by side. Both these are to be used. The first hitch of a reef knot is tied on each separately, then the two ends on one side are taken in one hand and the two ends on the other in the other hand, the loops being drawn sufficiently tight to occlude the artery; the knot is then completed as a reef knot by treating the two ends in each hand as single threads. This is called a "stay knot." It has been objected that it is a somewhat bulky knot. I did not find it so with silk; but if you prefer it you can complete each reef knot separately, the principle being that the friction in the two single hitches first made and between the two loops offers so much resistance to distension by the pressure of the blood that ample time is allowed to complete the knot; a further advantage is that with two loops there is a great range between the force which is just sufficient to occlude and that which just stops short of rupturing the coats, thus there is little fear of injuring the vessel. The stay knot, I may say from experience, is not at all complicated—indeed, if the knot is completed as two separate reefs, it is simplicity itself. I used the stay knot for the left femoral and the common carotid—with what result we will now see.

We will commence with the report on the right femoral which had been tied with the surgeon's knot four years before death. For about three-quarters of an inch at the seat of ligation the lumen is occluded, with the exception that a very fine bristle can be made to enter it. This tiny winding channel is crossed by several perforated diaphragms. the coats are uninjured, but apparently were not quite brought into contact by the ligation and the vessel is practically impervious; above and below the seat of ligation the vessel is patent, but is much diminished in size. No trace of the ligation could be seen. Below the vessel was patent down to the aneurysm, where the popliteal artery and aneurysm were together converted into a small mass of connective tissue. The left femoral had been tied with a stay knot three years before. The vessel was blocked at the seat of ligation by fibrous tissue (organised clot) for about half an inch; in this were minute holes, along which a bristle would not travel, owing to the openings not corresponding in what may be called the successive diaphragms. The coats could easily be followed and had not been injured by the ligation, but appear not to have been brought quite into contact, the closure being completed by a clot which subsequently became converted into fibrous tissue. The vessel was patent above and below the seat of ligation; no trace of the ligatures was seen. The condition of the artery below and of the aneurysm was the same as on the right side. The right common carotid: at the seat of ligation for three-quarters of an inch the coats are uninjured, in contact and adherent. The whole length of the vessel above and below this was filled with clot; the ligatures were encapsuled in connective tissue. In all three cases the whole seat of ligation was enveloped in a small fusiform mass of connective tissue. The ligatures on the femorals fully answered their purpose, and that on the common carotid seems to have been ideal.

With respect to the subclavian artery, you will remember that as the aneurysm needle was being passed the vessel gave way, and I had to treat it as a wounded artery—namely, by applying a ligation above and below and dividing the vessel between them; the ends, you will see, are separated by half an inch and lie in a mass of fibrous tissue. On the proximal side there is no clot whatever, although the inner coat has been ruptured and is recurved, and though the nearest branch above is one inch and a quarter distant from the seat of ligation. On the distal side there is a clot extending to the first branch—that is, for half an inch. The silk ligatures have ulcerated through the vessel and lie in minute inflammatory cavities just large enough to hold them at the side of the vessel. On the patient's third and last admission to the hospital there was an obvious aneurysm behind the manubrium sterni. It must not be inferred that an aneurysm in this position is of the innominate artery, for aneurysms frequently spring from the front of the arch and pass up in front of the innominate. This, in fact, was what was eventually found to be the case in this patient. The aneurysm was a sacculated one, communicating by an opening the size of a shilling with the arch of the aorta, just in front of the origin of the innominate, which was perfectly normal. There are several specimens in our museum of these aneurysms; in one, the aneurysm arises, as in our present case, from the

aorta alone; in a second, by an opening common to it and the innominate artery; in a third, the opening of the aneurysm is from the innominate, but only from just within its mouth. It is not possible from the position, then, of the aneurysm to determine whether it is innominate or aortic. With respect to other symptoms, the most important is the comparison of the two radial pulses; if the right is decidedly weaker the innominate is probably involved. The various pressure symptoms may be caused by aneurysm of either artery.

With respect to the treatment by distal ligature, it is to be remarked that if the aneurysm is purely aortic the operation can only offer temporary relief, though several cases are now on record in which much good appears to have been done. In the case of purely innominate aneurysms a cure probably would be obtained if a method could be found of preventing the passage of any blood along the innominate artery; but this is not effected by the ligature of the carotid and the third part of the subclavian. In the present case the innominate and the whole length of the subclavian as far as the seat of ligature were patent, so that a large amount of blood continued to flow through the innominate after the operation. Clearly the ligature of the carotid reduces considerably the amount of blood flowing through the innominate, but the good effect of tying the third part of the subclavian is not so clear, since the collateral circulation for the arm and brain is supplied partly by the branches of the first and second parts of the subclavian. On this account some surgeons have recommended that the vertebral should also be tied, but the operation for these cases, if it were only feasible, would be the ligation of the first part of the subclavian; this operation up to the present time has always been followed by fatal hæmorrhage, but it is encouraging to remember that Smyth of New Orleans has ligatured a larger artery—namely, the innominate—with success; we may therefore be permitted to hope that with the progress of surgery the operation may yet be successful.

squamous fissure often being evident, after stripping off the dura mater, which sends a process into it, and occasionally a fine bristle may be passed through it into the cavity. The roof is generally thin, often cellular under the upper layer of compact bone; to remove it, commencing from behind, is the easiest way of demonstrating the cavity, the lining mucous membrane often remaining intact. The apex abuts on the base of the mastoid process, sometimes pointing straight into the groove for the lateral sinus—the intervening portion of bone being smooth, extremely thin and translucent,—at other times pointing more towards the mastoid mass, being either smooth or cellular and never having any communication with that process. The antero-internal wall formed by the mastoid sulcus is generally reticulated, and is in relation at its upper and posterior aspect with the semicircular canals (the superior on a slightly higher level), being separated by a layer of dense bone which increases in thickness from the front backwards and measuring about one-eighth of an inch behind. In front of the canals and on a lower plane is the facial nerve in its canal, also separated by a layer of dense bone from one-sixteenth to one-eighth of an inch in thickness; the wall soon slopes away from these structures, narrowing to the apex. A gap is left at the upper and anterior angle, forming the opening of the cavity.

Voltolini describes a canal occasionally leading from the fossa subarcuata, or the depression below the superior semicircular canal, into the mastoid cells. I have a specimen in which the canal opens into the antrum on the antero-internal wall. The postero-internal wall forms a portion of the posterior fossa of the skull, corresponding to a roughly triangular surface on the posterior surface of the petrous

THE MASTOID ANTRUM IN CHILDREN.

By ARTHUR H. CHEATLE, F.R.C.S. ENG.,

LATE HOUSE SURGEON AND HOUSE ACCOUCHEUR, KING'S COLLEGE HOSPITAL; AND JUNIOR RESIDENT MEDICAL OFFICER AND REGISTRAR, EVELINA HOSPITAL FOR SICK CHILDREN.

MR. ARBUTHNOT LANE has drawn attention to the great importance of the mastoid antrum in connexion with the functions and diseases of the middle ear. After reading his papers I examined the cavities of a number of children whose ages ranged from one to twelve years, and found that they presented very uniform appearances and relations in each case, and admitted of the following description:—The mastoid antrum is a cone-shaped cavity with rounded angles, situated in the most external portion of the petrous bone, the base being turned towards the middle fossa of the skull, the apex running downwards, backwards and outwards, abutting on the base of the mastoid process, opening at its upper and anterior aspect into the recess immediately above the incus; a line drawn from the centre of the base to the apex, forming about an angle of 45° with the direction of the mastoid process, measuring from a quarter to three-eighths of an inch from base to apex, the same in diameter at the base, gradually narrowing to the apex; lined with mucous membrane continuous with that lining the rest of the tympanic cavity and containing a viscid fluid. As regards its development, it is the cavity enclosed by the junction of the projecting lips of the mastoid sulcus, which join posteriorly with the lamellæ of the horizontal portion of the squamous division, the upper lip with the posterior part of the internal lamella, the lower with the posterior part of the outer, the inner surface of the outer lamella being cellular, where it forms the outer wall of the antrum.

For the purpose of describing its relations to surrounding parts it presents for examination a base or roof, an apex, an opening, and external, antero-internal and postero-internal walls. The roof forms a portion of the floor of the middle fossa of the skull, and corresponds to a surface of bone on the superior surface of the petrous bone from one-fourth to three-eighths of an inch in diameter, slightly external to the eminence formed by the superior semicircular canal, irregularly circular or triangular in outline, and formed by the upper lip of the mastoid sulcus overlapping the internal lamella of the horizontal portion of the squama, the petro-

bone, external to the semicircular canals, the base upwards; it is often smooth, thin and translucent, sometimes cellular towards the cavity. The relation of the groove for the lateral sinus varies considerably, sometimes the groove covering a considerable portion of its outer and lower aspects, at other times only its lower aspect, or not coming into relation with it at all, but skirting external to and below it. Between the roof and the postero-internal wall the superior border of the petrous bone projects into the cranial cavity, along which runs the superior petrosal sinus, enclosed in the tentorium cerebelli. The external wall is roughly triangular in shape and always lined at its superior and anterior aspects with a layer of intercommunicating cells, all of which open eventually into the main cavity; it is formed by the posterior part of the outer lamella of the horizontal portion of the squama, joining with the lower lip of the mastoid sulcus, evidences of the fissura mastoideo-squamosa often being visible. This wall corresponds on the external surface with a depression felt behind the attachment of the auricle at its upper part, above the mastoid process. A small tubercle may often be felt close to the attachment of the auricle, separated from the mastoid process by a slight groove; if a hole be drilled straight in at this tubercle it will open into the antrum, as a rule just above the apex. A rough method of marking out this wall on the exterior is to draw a line along the anterior edge of the mastoid process, parallel to the main direction of that process (see diagram, *a, b*). In this