Formation of blood pigment need not, in my opinion, necessarily attend this destruction of hæmoglobin within the liver cells. I have time after time satisfied myself that a rapid formation of bile pigments may be going on without any trace of blood pigment being present in the liver cells.

The peculiar feature of this process, then, as distinguished from the one just described, is that formation of blood pigments need not necessarily attend the destruction of the hæmoglobin, and that if this does occur, as we have seen from the presence of pigment within the liver cells it undoubtedly does, only a part of the hæmoglobin may be thus transformed, the remainder being broken up in other ways. It is the pigment so formed from free hæmoglobin that is distinguished by the smaller and more uniform size of its granules.

A change in the red corpuscles, attended by a liberation of its hæmoglobin, is altogether more rapidly brought about than a slow gradual decay. Moreover, it implies some activity on the part of plasma or leucocytes. Hence, to this process the terms "acute" or "active" destruction of red corpuscles may be applied, according as we view the change in relation to its rapidity or to the rôle of the blood as a whole in bringing it about.

It is clear then that blood pigment must have a very varying significance as an indication of hæmolytic change. (By hæmolysis we naturally mean those changes by which the blood, as a whole, is affected injuriously, and not one particular element of it).

For the process that leads to the formation of most blood pigment—namely, slow death of the red corpuscle—affects only the red corpuscle, and implies, indeed, an absence of change in the plasma. Such a process cannot be termed "hæmolysis." The other process is the only one, therefore, which we can rightly term "hæmolytic," in so far as it is marked by changes, not in the red corpuscles only, but, as we shall afterwards see, also in plasma and leucocytes. And yet it is this process which we have seen favours the disposal of hæmoglobin in ways which may leave no trace of blood pigment behind. The conclusion then is: hæmolysis is a process which may be unattended by the formation of any blood pigment; and hence it follows that the absence of such pigment in health, as is the case in young mammals, may be and, as I will now proceed to show, is quite compatible with the occurrence of a very considerable daily hæmolysis.

BILE PIGMENT AS AN INDICATION OF HÆMOLYSIS.

Having arrived at the conclusion that, probably in health, and certainly in disease, a destruction of blood takes place, the question naturally arises, What is the extent of this destruction in health? We have seen how close is the relation between hæmoglobin and bile pigment; and this may be regarded as affording at least presumptive evidence in favour of a certain daily destruction of hæmoglobin. Such evidence is, however, no more than presumptive, for we have seen that in the slow death of red corpuscles a certain amount of hæmoglobin becomes effete; and yet this slow death is by no means an evidence of hæmolysis. If bile pigment can be formed from such effete hæmoglobin, it is clear, then, that bile pigment as an evidence of hæmolysis loses much of, if not all, its value.

As I have shown, a large body of evidence points to the liver as the seat of conversion of hæmoglobin into bile pigments. The matter at issue thus resolves itself into a consideration of the relative importance of pigment cells within the capillaries of the liver and the liver cells themselves as seats of formation of bile pigments. For it is the capillaries that are, according to my observations, exclusively the seat of blood pigment formed in the above way—namely, from a slow destruction of red corpuscles.

It may appear at first sight a matter of little moment where within the liver this change takes place, whether through the agency of leucocytes in the capillaries or of the liver cells themselves. I cannot so regard it. On the contrary, nothing has impressed me so much in the course of these studies of the liver as the necessity for distinguishing sharply between leucocytes and liver cells—between processes carried on in the capillaries exclusively by leucocytes and endothelial cells, and processes carried on within the liver cell itself. Carmine or ultramarine blue injected into the circula-

tion is arrested to a very large extent by the liver, but only by its cells of leucocyte and endothelial nature. The liver cells take no part whatever in this process. Such a function of the liver, valuable as it is, cannot be put on a level with one carried out by the liver cells—for example, the glycogenic.

If, then, the formation of bile pigment can be effected equally well by leucocyte or liver cell, and probably chiefly by the former, as Minkowski and Naunyn maintain, the process would cease, in my opinion, to be a truly hepatic one, and no forcible reason could be adduced why it should not take place in other organs equally rich in leucocytes and endothelial cells, in the spleen, for example, where, however, these two observers admit that no evidence of such a formation can be found.

When we pass from these general considerations to the facts, we find they are as follows. It cannot, in my opinion, be doubted that a certain power is possessed by cells of leucocyte and connective tissue nature of forming pigments apparently identical with bile pigment from effete hæmoglobin (Langhans, Cordua, Quincke). Moreover, a pigment, apparently biliverdin, has been found (Minkowski and Naunyn) in the large pigment cells of the liver of the goose—an observation from which its authors conclude, apparently justifiably, that a formation of bile pigments goes on within these cells.

While admitting the accuracy of both these facts, I nevertheless cannot admit that such a method of bile-pigment formation is even a common—still less the usual—one, and for three reasons. First, that in a large number of animals I have failed to find any pigment cells in the capillaries of the liver. Their presence, especially in young mammals, is, as I have said, the exception. Secondly, the experiments I now quote show that a very large formation of bile pigments may be induced in mammals without the appearance of a single pigment cell in the capillaries, but with, on the contrary, the appearance of blood pigment within the liver cells—a situation in which, as I have shown, it can only be derived from free hæmoglobin. Lastly, as the experiment I now quote shows, a very large increase in the pigment cells may be brought about unattended by any increased formation of bile pigments.

It is therefore within the liver cells themselves, and not in the capillaries that, according to my observations, hæmoglobin is transformed into bile pigments. And this conclusion carries with it the further one, that whatever hæmoglobin is daily broken up to form the bile pigments must have escaped from the red corpuscle into the plasma, and thence passed into the liver cell. In other words, some degree of active hæmolysis is an event of every-day occurrence.

LIGATURE OF THE ILIAC ARTERIES THROUGH THE PERITONEUM.

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In the *BRITISH MEDICAL JOURNAL* of October 29th, Mr. Marmaduke Sheild started a very interesting question as to the propriety of ligaturing the iliac arteries through the peritoneum in view of the great safety with which laparotomy can now be performed. It very commonly happens that, unknown to each other, the same idea occurs to several people, who either partially or completely carry it into practice; but naturally it is not until the details of several cases are collected and collated that a definite conclusion can be arrived at. Mr. Sheild's letter is obviously eliciting the necessary experience, as evidenced by Mr. Clement Lucas's communication, in which he reminds us of his successful case of ligature of the common iliac through the peritoneum for aneurysm of the external iliac artery done three years and a half ago. Again, Messrs. Ballance and Edmunds have brought to our recollection three cases of ligature of the internal iliac artery performed by Dennis, of New York, so far back as 1886, and described by Jacobson in his *Operative Surgery*. I am glad to be able to contribute a case to the general tale of experience.

My patient was a farm labourer, aged 62, sent to me in Sep-

tember of this year by Dr. Laing, of Burscough. He was a fairly healthy-looking man of spare frame, who had never had an illness in his life, and whose family history presented nothing remarkable. Some weeks before his admission, while engaged in ditching, he felt pain and a feeling of beating in the right groin. This occurred after midday, but he continued to work for the remainder of the day, getting relief from the pain and beating by occasionally resting. On reaching his home at night he examined his right groin, and found a lump there about the size of a small nut; he also noticed that the lump throbbed. He struggled to keep at his work for about a fortnight, the pain meantime becoming much worse, and the swelling daily increasing in size. At the end of that time he was compelled to give up work, and remained at home resting. Finally, he consulted Dr. Laing, who sent him into the Liverpool Royal Infirmary. A large pulsating tumour as big as the fist occupied the upper part of the right triangle of Scarpa, pushing its way upwards beneath Poupart's ligament. The pulsation was expansile, and there was a distinct *bruit*. The general signs left no doubt that the tumour was aneurysmal.

On September 20th the abdominal cavity was opened by an incision about 3 inches in length along the right linea semilunaris. The cæcum and small intestines came into view, and were held apart by the hands of an assistant. My finger immediately detected the position of the external iliac artery. The peritoneum over it was raised and slit up for about $\frac{3}{4}$ of an inch, and the artery came clearly into view. It was exposed high up in its course, and seemed perfectly healthy. It was tied with catgut, which had been soaked in carbolic oil, with just the exact amount of pressure necessary to stop all pulsation in the aneurysm, and no more. No attempt at tight ligaturing with the object of dividing the internal coat was made. The peritoneal incision was stitched up with a fine bit of catgut, so as to make the artery and ligature once more extraperitoneal. The abdominal wound was closed with fishing gut and dressed with cyanide gauze. The patient's recovery was uninterruptedly successful except for one incident. On the eleventh day after the operation my house-surgeon was hastily summoned to him at 8 A.M., and found that he had suddenly experienced acute pain of a stabbing nature in the precordial region, while his breathing had become painful and difficult. His face was deeply cyanosed, and he was in a state of great collapse. Ether and brandy were administered, and after about twenty minutes he rallied. I imagine that a piece of clot had become detached from the neighbourhood of the ligature, and by its lodgment in the circulatory round had caused the sudden and alarming symptoms mentioned. No investigation of the chest was made for the simple reason that, while it might satisfy curiosity, it could do no real good to the patient, whose best chance of recovery consisted in absolute repose. This was the only drawback. The limb, which had been wrapped up in cotton wadding, remained warm all the time. The temperature never rose a single degree. The pain disappeared and the appetite returned. For some time the aneurysm contained fluid at one part, evidently blood serum, but this became absorbed and only a hard swelling remained, steadily contracting in size. He left the infirmary on November 1st, 1892, the forty-second day after the operation, in good general health, and asserting that he could walk as well as ever.

About this aneurysm and its cure there is nothing of any special interest, because ligature of the external iliac is always a very successful operation. But the idea of tying an iliac artery through the peritoneum had many times occurred to me, and I thought this a fair and proper case to try it in, for the reason that the aneurysm pushed well up beneath Poupart's ligament, and I could not make out with any certainty whether the vessel in its immediate vicinity was healthy or whether it was thinned and dilated. It was obvious that the artery being once exposed through the peritoneum, I could select any point of it for ligature, going as high up as I chose till I came on sound vessel. As it happened, I struck the artery high up, and found it quite healthy. Nothing could have been simpler than the operation; nothing more satisfactory than the cure.

I have tied the external iliac artery twice previously by the ordinary method; once for the cure of aneurysm, and once in the hope of retarding the growth of a pulsating tumour of the

iliac bone. It is true that it is a sufficiently easy operation, but I think the operation through the peritoneum easier still. As for ligature of the common and internal iliac arteries, in future these will always be tied through the peritoneum; and the next editions of works on operative surgery will be able to dispense with operose descriptions of the old, dangerous, and difficult methods of reaching them. As Mr. Keetley most justly observes, it is Listerism that has enabled us to reach this pitch. For ligature of the external iliac artery the ordinary operation is so safe and simple, that it will probably be retained in all cases where the aneurysm is low down, and where there is a good probability of sound vessel being found immediately above Poupart's ligament. This is reasonable, because a patient may quite well recover from the ordinary operation, even when it has been clumsily done, or when the wound has become septic; but if by evil chance an abdominal wound becomes septic it is quite a different matter. Because laparotomy has succeeded *à merveille*, that is no reason why we should do it recklessly. But where the aneurysm is high, and there is a doubt as to the soundness of the artery near it, as in my case, then I think it will be the most judicious thing to ligature the vessel through the peritoneum.

Mr. Footner's case of iliac aneurysm, which burst just as he was going to tie the common iliac through the peritoneum, is most interesting. I may be prejudiced, but since catgut ligatures and surgical cleanliness became understood, I have never seen compression of arteries for the cure of aneurysm: do anything which well-conducted ligature could not do more easily, more quickly, and more safely. Moreover, it has seemed to me that where compression has been tried and failed, subsequent ligature has never had the same fair play as if it had been done at first. I have never yet seen anything but absolute and satisfactory cure after tying the femoral for popliteal aneurysm, for instance, until I have come to regard it as one of the safest and easiest operations in all surgery. What better is desired? The only cases where I have seen trouble has been where flexion has been tried and failed; so that I am sure Mr. Footner will agree with me that if he chances on another iliac aneurysm he will promptly tie the common iliac through the peritoneum, and not waste any time over aortic compression.

Some fifteen years ago a man came into the infirmary under my care in a dreadful state of pain and distress generally. He had been out of work, and half starved. He had noticed a swelling in the left side of his abdomen for some time. In urgent search of employment he had performed a very long journey on foot, and reached Liverpool just in time to crawl exhausted into hospital. The swelling had increased rapidly, and was daily growing bigger. It was made out to be an aneurysm occupying the left iliac region, reaching to the middle line in front and to the umbilicus above. It was impossible to say where it sprang from, but as the man evidently had only a very short time to live it was necessary to act promptly. I opened the abdomen in the middle line, which was thought rather an adventurous proceeding in those days, with the intention of tying the common iliac or the aorta itself if I got a chance. But it was found impossible to do anything. The aneurysm overlapped the left common iliac and the lower portion of the aorta, so that neither of them could be reached. It was a gigantic thing, and had been leaking for some time at the back, tearing up the tissues behind the peritoneum in all directions. I shut the wound up, when I found the case hopeless, and the patient lived for a few days, which was all that we expected him to do under any circumstances. So that what was done did no real harm, and showed us that we had omitted no reasonable chance of cure.

CONGENITAL SYPHILIS AS A CAUSE OF NERVOUS DISEASES IN CHILDREN.¹

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SCANTY as are the records of morbid anatomy, there is, nevertheless, reason to believe that "nearly every variety of nervous affection of acquired syphilis has its parallel among con-

¹ Read before the Medical Society of London, November 21st, 1892.