doubtless thus occasioned in this particular case, although we had no opportunity of verifying the diagnosis. This patient is fortunately still alive, and somewhat better. You will thus see, from what I have already said, that attention to the particular combinations and degrees of paralysis of cranial nerves, both sensory and motor, in association with hemiplegic forms of paralysis, is of the highest importance, in enabling or assisting us to arrive at a regional diagnosis in cases of brain disease.

I will now call your attention, gentlemen, to the various forms of *rigidity* which may be met with in the paralysed limbs. This condition is seen not only more frequently but also to a more marked extent in the arm than in the leg. It was a sign much dwelt upon by the late Dr. Todd, and he very properly distinguished between two varieties of it whose pathology is altogether different. To the one variety he gave the name "early rigidity," and to the other variety that of "late rigidity." In some cases hemiplegia comes and goes without the appearance of either form of rigidity; the muscles continue flaccid, whilst power is gradually regained. In other instances, where complete recovery does not occur, there may have been no early rigidity, although a condition of this kind (late rigidity) is apt to manifest itself after the lapse of many months.

"Early rigidity" may be limited to one or two fingers, or it may be so slight as only to show itself when we proceed to move the limb. When the arm is bent, for instance, if we attempt to straighten it there is resistance on the part of the biceps. On other occasions if we try to flex the arm, the triceps responds, and partially resists our effort by some amount of contraction of its fibres. In other cases the rigidity may be very marked, the arm is drawn to the side of the chest, the elbow and wrist are firmly bent, and the fingers are flexed upon the palm. In the most marked cases any attempts to straighten the limb only seem to increase the spasm, and may cause pain as well as set up some amount of tremor or slight clonic spasms in the limbs of one or both sides; though at other times, as you may have seen in the case of an aphasic man lately in our wards, the spasm yields considerably under steady pressure, and this, moreover, produces no pain. Now, this kind of spasm may persist for weeks or months, with only slight variations from day to day. The muscles do not show any signs of wasting for a long time, and they respond to faradisation generally rather more readily than those of the healthy side. For one or two hours also after the extensors have been well faradised, the spasm may continue notably lessened. And we occasionally find with this form of rigidity that the spasm relaxes altogether during sleep, the muscles resuming their rigid condition during the act of awaking. When such a form of rigidity exists, moreover, the reflex excitability of the limbs, but more especially of the leg, is often decidedly increased. Tickling of the sole of the foot or contact with a hot spoon will then cause decided reflex movements. This early rigidity may show itself after some days, or even immediately after the commencement of the attack, and whilst the patient is still in a state of apoplectic or epileptiform stupor. When occurring thus early, the arm may be flexed and rigid either persistently or at times; or the rigid condition may alternate with one of clonic spasm. Sometimes, again, the arm may be rigid whilst the leg is affected with clonic spasms, and *vice versa*. Early rigidity may affect the leg at the same time as the arm, and when it occurs we find the thigh drawn up and the knee firmly flexed so as to bring the heel close against the buttock. The arm, however, is much more frequently affected than the leg. As to the precise meaning of this "early rigidity" we know very little. It is said to be indicative of an "irritative" influence of the brain lesion, although it may occur with lesions of varying nature. It is doubtless in the majority of cases a grave symptom, and its appearance diminishes the chance of the patient's complete recovery. After a time, if the spasm does not relax, changes begin to take place in the muscles, tendons, and joints of the limb, which may ultimately leave it permanently contracted and useless. The lesion of the brain in these cases is often a large one, seriously implicating some extent of the cortical grey matter, and destroying a considerable area of subjacent white substance. Lesions in the optic thalamus also not unfrequently produce this kind of rigidity.

"Late rigidity" supervenes in some cases after many weeks or months, although previously all the muscles may

have been in a flaccid condition. This form of rigidity is also, like the other, generally more marked in the arm than in the leg. It commences after the muscles have undergone some wasting. It may be limited to the flexors of the fingers, or it may extend to other muscles, so that in the most extreme cases the arm becomes immovably bent at the elbow, the wrist is firmly flexed, and the fingers are pressed against the palm of the hand—so forcibly, it may be, as to cause some amount of ulceration in this situation. Attempts to straighten the contracted parts produce much pain, and are usually ineffectual owing to the shortening which has taken place in the muscles and tendons. In the leg, a similar contraction of the hamstring muscles may exist with rigid tendons, and at the same time there may be more or less contraction of muscles about the ankle, and perhaps also some rigidity of these about the hip-joint. Dr. Todd thought that these signs, characteristic of "late rigidity," were brought about under the influence of the process of cicatrisation taking place at the seat of brain lesion. The contracting and indurating changes by which such a process of cicatrisation is distinguished were supposed to occasion an "irritation" of adjacent nerve-tissue, which thence became communicated to the paralysed muscles. As to the mode of conduction of this influence, or indeed as to the precise degree of causal connexion between the process of cicatrisation in and about a brain lesion, and the phenomena of late rigidity, there is much room for doubt. It is unquestionably true that where a limb remains permanently paralysed, certain nutritive changes are apt to be set up after a time in its nerves, its muscles, and its joints, though it is not always easy for us to decide whether these changes are directly occasioned by the brain lesion, or under the influence of those "secondary" changes in the spinal cord which are so often immediately occasioned by the brain lesion. The latter view, and strong evidence in favour of it, have been forthcoming during the last few years.

Clinical Lecture

ON

AMYLOID AND FATTY LIVER IN RELATION TO OPERATIONS.

By RICHARD BARWELL, F.R.C.S.,

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GENTLEMEN,—I have on several occasions pointed out to you certain cases of prolonged and severe suppuration produced by caries and necrosis or by disease of joints, and have shown to you that in several of these the liver has been plainly and palpably enlarged, sometimes to a very great extent. I will now give you shortly the details of two such instances, as a groundwork to the study of the forms of malady which produce this enlargement.

CASE 1.—Caroline L—, aged four years and a half, was admitted into Charing-cross Hospital on July 5th, 1870, under the care of Mr. Hancock, with hip disease. The case was severe, and, in spite of treatment, became worse; eight months after admission the child was greatly reduced by excessive discharge from large abscesses extending down the thigh. Mr. Hancock proceeded to excise the head of the femur, but during the operation that bone broke in two places; it was, in fact, a mere shell. The limb was placed on an interrupted splint; the bone reunited; the patient gained in flesh and strength, and left the hospital on June 4th, 1872, with still an open and discharging sinus on the outer side of the thigh, and another at the operation wound.

She was readmitted into the hospital on Sept. 4th, 1872, under my care. The left leg was very much wasted and shortened. Six open sinuses communicated, three with diseased bone at the upper part of the femur and acetabulum, and three lower in the thigh at the places of fracture. She was much wasted and anæmic; the skin of an opaque

yellowish hue; the pulse, very small, varied at different parts of the day and night from 98 to 130; appetite bad and capricious. The liver was much enlarged, extending through a considerable part of the right lumbar and a great portion of the umbilical region; it was smooth and hard. Urine healthy. No perceptible enlargement of the spleen. There was no evidence of tubercle in the lung.

Having watched this child with great care, I concluded that, in spite of the unfavourable condition, it was my duty to propose removal of the limb at the hip. Some time elapsed before consent could be obtained, but late in October it was given.

On Nov. 1st I examined her carefully. She was emaciated to a mere bag of bones, and was evidently weaker. The liver extended to a finger's breadth of the crest of the ilium, hence crossed hardly half an inch above the umbilicus, and disappeared under the left hypochondrium; it was hard, but smooth; its shape apparently not changed. No evidence of affection in any other organ.

On the 2nd November I amputated at the hip, and, owing to care in my arrangements and the skill of my assistants, scarcely an ounce of blood was lost. The child rallied rapidly; a few drawbacks occurred in the first fortnight, but after that her progress was uninterrupted and rapid. She left the hospital on Feb. 6th, 1873.

In April, 1874, I examined this patient, having seen her occasionally in the interval. She was, save for the loss of the left limb, a very healthy, strong-looking girl. The liver was examined both by myself and Dr. Pollock, and was found to be perfectly normal in size, and its functions duly performed.

Caroline T—, aged fourteen, was admitted on the 14th of April, 1873, into Charing-cross Hospital, under my care, suffering from a recurrence of inflammation after old hip disease. The child was greatly emaciated and æmic; the left hip was much swollen, and around the joint was deeply-seated matter; also the inguinal region was swollen and fluctuated, showing the presence of pus within the pelvis. Several sinus mouths were situated around the trochanter and on the outer side of the thigh. She was treated for six weeks by rest and tonics, in the hope that such improved condition might benefit, but at the end of that time she was found to be weaker and thinner, while the abscesses, especially that within the pelvis, had become larger. She was then more fully examined. The probe was passed into three or four of the sinuses, and two of these were found to enter a cloaca and so pass to a necrosis situated deep in the head and neck of the femur. It was evident that unless this could be removed the child would die of exhaustion. At this time the liver was greatly enlarged; it extended downward and through two-thirds of the right lumbar region; in the middle line its edge was two fingers' breadth above the umbilicus; it was smooth and considerably indurated. The urine was nearly neutral, and contained phosphates, but neither albumen, sugar, nor any casts of the tubuli; there was no diarrhoea; no enlargement of the spleen could be detected.

July 21st.—She was placed on the operating table, and, chloroform being administered, it became evident that the hip was ankylosed (true ankylosis); but the central parts of the head of the bone being necrosed, and there being a large collection of matter within the pelvis, I nevertheless made a semilunar incision behind the trochanter, as for resection of the hip, sawed through the neck of the bone as close as possible to the pelvis, applied a trephine to the truncated head, and with it cut out the necrosis, perforated the acetabulum, and let out of the pelvis a large quantity of dark-coloured pus.

The child rallied from the operation with difficulty, but after the first few days went on very well, regaining appetite, and beginning to put on flesh, till two months after the operation, when a patient having undergone the operation for ruptured perineum was placed in the same ward. The considerable fetor arising from this case affected her markedly; the discharge, hitherto diminishing, increased; her appetite failed, and she began to lose flesh. Removal to another ward changed all these conditions, and she went on uninterruptedly well until the 6th of March, 1874, the state of her discharge. At this time the child was comparatively rosy and stout; the liver had very nearly gained its normal size, and its functions were duly performed.

Both these cases are operations somewhat remarkable,

especially the latter (for I am not aware that any such has been performed or described); yet it is not to that part of the subject to which I would now direct your attention, but rather to the condition of the liver and other abdominal organs.

A patient who has suffered long from free suppuration, whatever be its origin, falls into a state of considerable cachexia: the body loses much of its fat; the skin becomes pale, of an opaque hue, often also dry, rough, and inelastic. In spite of the general emaciation, the abdomen, more especially of children, is generally tumid; percussion returns over the greater part of its surface a loud, hollow note. On passing, however, to the right side, this loud note will be dulled over a certain distance below the hypochondrium according to the extent of enlargement; occasionally, as was the case in the girl C. L—, this dullness, marking the extent of the liver, will reach as low as the crista ili, or close to it. By using the mode of manual examination called "palpation" you will find the edge of the viscus, and feel its surface more or less distinctly according to the thickness of the abdominal walls. Observe that the surface of the gland is quite smooth, and the organ not altered in shape, save that its lower edge may appear rather rounder and thicker than usual; while again, in some cases, the organ seems firmer and harder, in others softer, than normal.

There is, then, in these cases a uniform increase in size, which does not affect by pressure or obstruction the portal circulation, since it is not accompanied by ascites; and though it may diminish, it does not annihilate the secreting function of the gland, since the fæces are coloured with bile, and since there is no jaundice.

This enlargement is due to the deposition in the constituents of the organ either of oil-globules (the fatty liver), or of a material called amyloceous (the amyloid or lardaceous liver). It is not my intention now to detain you with a minute description of the pathological anatomy of either of these states. I need only say that the latter consists in the gradual infiltration of the structure with a material which appears to consist of homogeneous molecules—apparently a low form of protein, which hardens, thickens, and whitens the organ, and in some cases no doubt destroys its function. The tissue thus affected is changed from its normal stage of high organisation into the lowest type of organic matter, which hardly possesses life or function, since it can scarcely undergo change—with difficulty even that last change of dead organism, putrefaction. Therefore, as you will see at once, change from this state back to healthy condition would seem barely possible, and indeed most writers on this subject consider the malady irrecoverable. On the other hand, fat, in the shape of oil-globules, is frequently present in the liver as a normal and temporary sequela of absorption from the intestine of oily ingesta. The accumulation of such substance in the cells of the gland to a morbid extent may be due to a variety of causes: it is common in phthisis and in heart disease, probably as a result of interference with the changes which the blood should undergo in the lung; it is also not unusual in wasting suppurative disease as of bones or joints. The tendency thereto is, I believe, increased, especially in children, by confinement to bed, combined with the high feeding which is usually employed in such cases. Since, then, in healthy life, fat is frequently present in, and as often removed from, the liver, it is plain that recovery from its morbid accumulation in that organ is a much more easy process, a far more likely event, than restoration to the norm of a liver infiltrated with so immobile a material as that which constitutes the amyloid deposit. Therefore we are often presented with certain problems which are at once of high importance and of extreme difficulty. The first concerns our power, or want of power, to discriminate during life between these two forms of liver enlargement in the cases we are considering; the second, our faith, or want of faith, in the power of nature to cure the secondary malady if it be amyloceous, when its exciting cause, the source of suppuration, has been removed.

Concerning the former problem, I can only say that our means of differential diagnosis are very imperfect, the greater hardness or softness of the liver being our chief guide; and I need not tell you that such a sensation conveyed through abdominal walls may be deceptive, especially if those walls be thick. But fatty liver does not reach the

size to which an amyloid organ may attain, as exemplified in the cases above given.

About the second problem much might be said if space allowed me to quote and to remark upon the views of many authorities. The whole balance of opinion is that amyloid disease, once commenced, is of necessity a progressive and fatal malady. With this conclusion, as far as the liver is concerned, I cannot agree; but it is impossible to prove absolutely such position, because if a person having an amyloid liver recover there occurs no opportunity of showing by chemical or microscopic examination the condition of the organ; and this is the position to which I am forced in regard to the above cases. The remarkable hardness and size of the organ, together with the condition of the patient's skin, &c., convinced me that the enlargement of the liver was in neither case due to fatty change; but I cannot prove it.

Although, then, it may in some cases be impossible to diagnose with certainty during life between these two forms of enlarged liver, we have, nevertheless, a certain and sure guide to practice. Since prolonged suppuration causes such enlargement, the liver is in every case to be carefully examined, as are also other organs, for the liver itself is not often attacked alone by the amyloid change. This organ may, as to size, be easily interrogated. Any considerable enlargement of the spleen may be detected without difficulty in young persons. Albumen in the urine is an early symptom of the amyloid kidney, and not unfrequently the peculiarly altered epithelium and sometimes casts may be found sufficiently characteristic to give the key to the form of Bright's disease whence the albuminuria arises. Amyloid intestines are in their earlier phases with difficulty made out. Obstinate, even recurrent, diarrhoea is a suspicious sign, and careful observation of the excreta may disclose cast off villi, even clumps of villi, with the characteristic hyaline appearance, and this even without any pus or sign of ulceration. If, after careful investigation on these points, the absence of any sign of amyloid disease save the enlargement of the liver be determined, any operation for the removal of suppuration is not only justifiable, but our imperative duty. Without operation the patient must die; after operation, however hard, large, and changed be the liver, there is a fair, indeed a considerable, chance of recovery, as the above cases sufficiently indicate.

SUDDEN DEATH FROM EMBOLISM OF THE PULMONARY ARTERIES:

PHLEBITIS OF THE LEG FOLLOWING A BLOW.

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MR. A—, aged forty-four, had never suffered from serious illness, except a severe attack of syphilis twenty years ago; but he had long been in the habit of drinking freely. On October 24th, 1873, he struck his left knee against the leg of a dining table, which caused him some pain. He went to business as usual until the 26th, when, the limb having become more painful, he sent for Mr. J. R. Lynch, of Notting-hill, his usual medical attendant. Mr. Lynch found no bruising of the limb, but a little swelling about the ankle. Movement, or pressure over the popliteal space, caused pain at the back of the knee. Rest and an evaporating lotion were recommended, but these directions were not carried out. Although lame, Mr. A— persisted in going to the City daily, and the limb became more swollen. On the 2nd of November a slight attack of jaundice confined him to the house, but this passed away in about a week. He had no pain except in walking, and could bend his knee to a slight extent without discomfort. The leg, however, continued swollen; and at one time the calf measured nearly an inch more than its fellow. There was, however, no pitting nor pain on pressure. He walked down stairs daily, took his food as usual, and made no complaint whatever, except of hindrance from business. Becoming intolerant of the confinement, on Nov. 19th he went into the City, where he walked about, and called on

several persons. On his return in the evening, whilst ascending the steps at the railway station, he suddenly became faint and unable to proceed. He was placed upon a chair, and was observed to gasp and struggle for breath, striking his chest frequently as if to remove some obstruction within. When he had somewhat recovered, a friend, who happened to pass, put him into a cab, and accompanied him home. He walked into his house with assistance, and, having taken some brandy, was helped to the watercloset and then upstairs to his bedroom, where he lay down upon the bed, and almost immediately expired. Those who were about him stated that he was perfectly conscious throughout, and did not complain of pain; but his face was very livid, and the difficulty of breathing extreme, as though there were a direct impediment to the entrance of air into the chest.

An autopsy was made on the 21st, about forty-eight hours after death, several medical men being present. The body was that of a remarkably fine, well-developed man. There was great hypostatic congestion of the dependent parts of the trunk and limbs. Much discoloration marked the course of the superficial veins, and decomposition was rapidly advancing. The left leg was swollen as high as the knee, but it did not pit under the finger. There was no bruising about the seat of injury. Much fat was deposited throughout the body. The blood was perfectly fluid, so that a large escape took place from the internal mammary veins when the sternum was removed. The heart was small and flaccid; it contained fluid blood, but no clot; the valves were normal. Both the lungs were congested, though crepitant throughout; the trunk and main divisions of the pulmonary artery were empty, but the branches which penetrate the lungs and their subdivisions, as far as they were traced, were completely plugged with dark black clot, which was dry and friable. The liver was large and congested, and its sharp anterior margin rounded and thick; on section it appeared natural. The kidneys were also much congested, with some little deposit in their cortical structure. The inferior vena cava and the iliac veins were largely distended with fluid blood. On tracing the left femoral vein, it was found to be filled with a firm dry clot from the profunda downwards. This clot extended throughout the popliteal and tibial veins, even into their small branches, completely filling them and adhering to their inner coat. The sheath of the femoral vessels was thickened and adherent to the vein, which felt like a hard cord to the finger. The arteries were patent. The internal saphena vein was free, and the external nearly so; the clots found in some of its branches did not completely block them. The fascia over the popliteal space was thickened and brawny from recent inflammation. There was slight extravasation of blood into the fat and areolar tissue around the peroneal nerve and biceps tendon. No blood escaped from the limb during examination, thus presenting a marked contrast with the other parts of the body, from which fluid blood dropped copiously. The head and brain were examined, but nothing abnormal was detected.

Remarks.—Apart from the interest which must always attach to cases of sudden death from embolism, this appears to present certain points which are worthy of consideration. The trifling injury which led to death; the absence of symptoms which could excite suspicion as to the very grave condition of the limb, as shown by dissection; the large extent to which the deep veins were involved without local discomfort or serious interference with health; the complete transference of the circulation from the deep to the superficial system of veins, and the capacity of the latter to carry on the entire venous circulation of the limb unaided, and without causing oedema—for the swelling of the leg was not due to serous effusion into the subcutaneous tissue, but to the distension of the deep veins with clot, and exudation into the muscles and their areolar investments.

The original injury had evidently caused rupture of some small vessels in the popliteal space, which, being followed by subacute phlebitis, led to the formation of coagula in the veins of the leg and thigh, and, finally, to their complete obstruction. The exertion of climbing the steep flight of stairs at the railway station detached a portion of the clot in the femoral vein, which, after passing through the larger trunks and the right cavities of the heart, was arrested at the branches of the pulmonary arteries just as they enter the lungs, and brought about the fatal