others containing bacilli of a crescentic form. The white corpuscles are greatly increased in number and there are also seen numerous giant white corpuscles with patches of black colouring matter in them. The pathology of this I take to be: (1) the crescentic bacilli of malaria invade the red corpuscles and proceed to absorb the hæmoglobin, converting it into melanin, and they also multiply in number (these facts I have demonstrated by examining the blood of a patient subject to malarial fever, but about three hours before an attack of rigors was expected); and (2) after a variable time the red corpuscles burst, setting free crowds of bacilli (this process I have found in blood taken at the period of rigors); the giant white corpuscles absorb the bacilli and apparently digest them, leaving only the melanin which remains as black patches in the corpuscles (this process may be traced by examining the blood of a patient at intervals during a typical attack of fever). The effect of the malarial poison shows itself differently in different individuals, the most usual form being that of high fever, the period of incubation being in most cases fourteen days, during which time the patient has a great feeling of *bien aise*. Towards the end of the incubation he becomes irritable and chilly, generally suffering from nausea, sometimes actual vomiting. At this period he begins to have a succession of rigors, which, however, gradually cease and he becomes feverish, with a full, bounding pulse. Without drugs this stage lasts from six to eight hours, when he begins to perspire, the temperature falls, and he sleeps for several hours, awakening without fever. After a length of time, varying from twenty-four hours to a week, if the person has not been placed under the influence of drugs the fever occurs again in exact similitude to the initial attack and this may recur at intervals for months. There is also a variety of malarial poisoning without much elevation of temperature, but with marked cerebral symptoms resembling meningitis. It is a most fatal variety and post mortem no signs of inflammation of the meninges can be seen.

Residents in Rhodesia are also, I find, liable to attacks of dysenteric diarrhœa which will not yield to opium or ipecacuanha, but rapidly gives way to tincture of perchloride of iron with quinine and arsenic. I have also seen what were apparently syphilitic ulcers, especially on the legs and thighs, which proved most obstinate to the usual specific treatment, but got well at once with the iron, arsenic, and quinine mixture, with a dusting powder of quinine locally. On healing these ulcers leave a permanent brown stain on the skin. I consider both diarrhœa and ulcers due to malarial poison and the brown discolouration of the skin is possibly the local deposit of melanin from the bacilli.

The sequelæ of malarial fever are only what may be naturally expected, the first and most important being the intense anæmia which is so often found and which requires lengthy treatment by iron and arsenic with, if possible, change of air. This anæmia causes anasarca of the most dependent parts—viz., legs and feet—but this cures itself as the anæmia decreases, as also does the intense weakness, amounting in some cases to temporary paralysis; whilst another result of the poverty of red-blood corpuscles is shown in the dizziness, partial blindness, and flashes of light experienced by some on recovering from a severe attack.

With regard to treatment every treatise on medicine and also the so-called popular handbooks describe quinine as a "sheet anchor" in malarial fever, but my experience tells me there is no greater mistake. The result is that nearly everyone coming to Africa brings enormous quantities of quinine tabloids, which they take *ad lib.*, and also dose their friends, independently of any advice from a medical man, and they fully believe they are rendering themselves proof against fever. These large doses of quinine—and it is not uncommon to find persons taking from two to three drachms a day—produce an utter rottenness of the constitution. There is a distinct quinine cachexia, easily recognised by those who see it often, and when one of these individuals falls a victim to an attack of fever he generally has it badly. I have found the most successful treatment to be absolute rest in bed, a 15 gr. dose of antipyrin to begin with, followed in an hour by the following mixture: 10 minims of tincture of perchloride of iron, 5 minims of hydrochloric solution of arsenic, with water to 1 oz. 1 oz. to be taken every four hours with a 5 gr. tabloid of sulphate of quinine. At bedtime I generally give about 5 gr. of calomel, followed in the morning by a dose of some effervescent saline. In most ordinary cases of fever this treatment cuts short an attack under twenty-four hours and then I follow up with a tonic course of Easton's syrup in 1 dr. doses with 1 oz. of water three times a day at meals for a month, and I find that most individuals will thus enjoy an immunity from malarial fever for some months at least, although living in an unhealthy district.

With regard to food stimulants are required, especially champagne. This will often stop the obstinate vomiting when even a mustard poultice at the pit of the stomach fails. During an attack I order milk and hot water in equal parts, and in some cases milk with a tablespoonful of brandy to each pint. When the temperature is normal and the paroxysm is over I give any food the patient fancies, and I find this answers well. I have one patient who after an attack of fever eats ravenously of cheese, enjoys it, and, what is more to the point, digests it.

In the "cerebral apyrexial form" as I term that variety with low temperature and head symptoms, which is very fatal, I find, so far, that the best treatment is calomel and sulphate of magnesia in full doses, producing free purgation, giving at the same time the quinine, iron and arsenic as in an ordinary attack. It is happily a rare form. I have only seen 6 cases, of which 1 was fatal; the rest recovered, 3 completely and 2 with some mental aberration, which I am in hopes will prove only temporary.

In Africa "ice-bags" are an impossibility, and yet how often one longs for them. Here, where medical men are rare, being often from 85 to 100 miles apart, it stands to reason that there are many farmers and traders who when they get fever cannot obtain medical advice. For these Messrs. Burroughs, Wellcome, and Co. have prepared cases for me containing five bottles and a thermometer. These contain 25 (5 gr.) tabloids of antipyrin; 100 (5 gr.) tabloids of sulphate of quinine; 100 "Iron and Arsenic Co." tabloids; and 200 (1 dr.) tabloids of Easton's syrup. I order possessors of these when "shakes" begin to go to bed and take three 5 gr. tabloids of antipyrin and one hour afterwards to take one each of quinine and iron and arsenic tabloids, repeated every three hours until the temperature is normal; then to begin with the Easton syrup tabloids, taking one three times a day for a month, and in every case I have been told that these drugs and directions have proved a boon; and whereas the traders, &c., at outlying stations have previously suffered all the wet season from malarial ague, now the initial attack is all they experience for the season.

To sum up, it must be remembered in every case that the malarial parasite destroys the red blood corpuscles, and one method of treatment is to replace them and so raise the vitality of the tissues that they may resist further invasions of the poison.

Enkeldoorn, Rhodesia.

DISSECTING ANEURYSM.[1]

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THE term "dissecting aneurysm" is applied to cases in which a partial rupture of the inner tunics of an artery allows the blood to become diffused between the coats of the vessel. Having regard to the difference of opinion which exists as to the author of the original description of this very remarkable affection the following extract from Peacock's[2] "Report on Cases of Dissecting Aneurysm" may be of interest: "It has generally been supposed that Laennec was the first writer who applied the term 'dissecting' to this form of aneurysm, but it has been recently shown by Broca that Maunoir employed the same designation and clearly described the formation of aneurysms of this kind in his work published in 1802. A characteristic case is also related by Allan Burns in his work on 'Diseases of the Heart and Aneurysm,' published in 1809." Shekelton[3] published two cases of dissecting aneurysm in 1822, and the preparations are to be seen in the museum of the Royal College of Surgeons in Ireland. Erichsen, in the eighth

[1] A paper read before the Medical Section of the Royal Academy of Medicine in Ireland, May 20th, 1898.
[2] Transactions of the Pathological Society of London, vol. xiv., 1863.
[3] Dublin Hospital Reports, vol. iii., 1822.

edition of his "Science and Art of Surgery," incorrectly refers to this variety of aneurysm "as originally described by Shekelton," and the use of the name "Shekelton's aneurysm" as a synonym for "dissecting aneurysm" is to be deprecated. Peacock, in the paper to which I have already referred, collected 80 instances in which the affection appeared in well-marked form, including 5 Dublin cases published by Shekelton,[4] R. W. Smith,[5] Kirkpatrick,[6] Lees,[7] and MacDonnell respectively. An interesting specimen was exhibited by Connolly Norman at a meeting of the Pathological Section of the Royal Academy of Medicine in Ireland a few months ago. In the earlier published cases of dissecting aneurysm it was supposed that the separation of the tunics of the artery took place between the middle and external tunic; but modern pathologists agree that the blood almost invariably makes its way between the laminæ of the middle coat, so that the outer wall of the aneurysm is formed of the outer strata of the media together with the adventitia. In the vast majority of cases the rupture of the inner tunics is due to their excessive lacerability, the result of atheromatous degeneration. Indeed, out of 60 of Peacock's cases the artery was found healthy in only two instances. Ziegler[8] suggests that in the rare absence of morbid changes in the coats of the vessels, traumatic injury to, or defective development of, the vessel wall is the primary cause of the lesion, and Walshe[9] mentions 2 cases in which dissection of the aortic coats on a limited scale had been abruptly effected as the result of a railway concussion. On the other hand, Rokitansky[10] supposes that in some cases a morbid condition of the external coat deprives the inner coat of support and so predisposes to their rupture.

Dissecting aneurysm is practically confined to the aorta and its large branches, although Ziegler[11] states—on what authority I do not know—that the small arteries of the brain are a usual seat of the affection. The internal rupture is usually situated at the origin of the aorta or in the ascending aorta (it was so in 55 out of 73 of Peacock's cases) but it has been found as low as the end of the abdominal aorta, as happened in Shekelton's cases. The direction of the rupture is generally transverse when it is near the aortic valves, whilst it is more frequently vertical at the beginning of the descending aorta. Ordinarily from one-half to two-thirds of the vessel is torn asunder, although the whole circumference of the artery is occasionally separated. The longitudinal extent of the separation of the tunics varies considerably. As a rule the aneurysm does not extend beyond the ascending aorta, though it is occasionally found to extend into the iliac arteries. In my case the separation of the coats begins at the arch and ends in the left femoral, and in a very remarkable case recorded by Tessier[12] the separation extended from the commencement of the ascending aorta to the left popliteal artery. The dissection not unfrequently is carried along the primary branches of the aorta; for instance, in MacDonnell's[13] and Tessier's[14] cases it extended along the innominate and its branches to the internal carotid; in another case[15] it followed the course of the coronary arteries; in Laennec's[16] case it passed along the cœliac artery and in my case along the right renal artery. Some of the intercostal and lumbar arteries are frequently found to be cut across by the stream of blood "so that a probe introduced into the aortic orifice of one of those little branches passes directly into the aneurysmal canal and traverses this before entering the artery itself, which thus appears to arise directly from the aneurysm."[17] In cases which have ended fatally within a short time there has been only a single opening leading from the aorta into the arterial coats, death having been usually due to the subsequent giving way of the external wall of the sac. This is the common form, comprising 73 out of Peacock's 80 cases. My case is also an instance of it. On the other hand, when life is prolonged for any considerable time the blood is found to have forced its way from the subadventitious sac back again to the blood-stream at some point further on in the course of the vessel. In such chronic cases the new blood-channel acquires an endothelial lining. Shekelton's[18] and Fagge's[19] cases were examples of this variety. The disease is specially common in persons of advanced age, and though it probably occurs more frequently in men than in women there is not the excessive preponderance in males which is the rule in ordinary aneurysms. Indeed, some writers say that it is more common in women. The case which I now relate and the specimen which I exhibit typically exemplify the symptoms and pathology of the early stage of the fully formed dissecting aneurysm.

A man, aged sixty-five years, a house-painter by occupation, was admitted to the Whitworth Hospital under my care on Jan. 13th, 1898, complaining of severe pain in his back and of loss of power in his legs. On the morning of his admission to hospital he was on a ladder papering a room when he was suddenly seized with a violent pain in the lower part of his back and in his left hip. The pain appeared to begin in the lumbar region and to shoot up into his chest and downwards towards his left thigh; it was so intense that, to use his own expression, he "bellowed with agony." He felt faint but did not lose consciousness, and he was able with difficulty to walk across the room to a chair. Within a quarter of an hour his lower extremities were completely paralysed and his left leg was anæsthetic. He was sent to the hospital in a few hours. Beyond the fact that he was a painter there was nothing noteworthy in his personal history and he came of a healthy family. On admission to the hospital he was seen to be a well-nourished, grey-haired old man, but rather anæmic. He was unable to walk, but he could move his legs slightly. He still suffered from severe pain in his back and everything he ate or drank was vomited. He had no longer anæsthesia in his left leg. His arteries, in which there was visible pulsation, were atheromatous and his pulse was high-tensioned. His heart was hypertrophied; no murmurs were audible, but the aortic second sound was greatly accentuated. No sign of aneurysm could be detected, although the patient was carefully examined for it. There was marked arcus senilis and he had a "lead-line" on his gums. His urine was feebly alkaline; its specific gravity was 1014. It was clear and contained 0·2 per cent. albumin and a few hyaline and granular casts; neither sugar nor blood was present. The quantity of urine passed was about the average amount, and he had perfect control over the sphincters of the bladder and rectum. The pain in his back ceased after 12 hours and next day he felt much better and he was able to move his legs freely. The knee-jerks and plantar reflexes were absent. On the following morning he was able to walk a few steps and he expressed himself as feeling almost well. At this period I showed him to the hospital class and diagnosed the case as plumbism and chronic interstitial nephritis with the usual cardio-vascular changes. I pointed out that the history of the attack clearly indicated some vascular lesion of the spinal cord and that the sudden onset, wide diffusion, and intensity of the pain, together with the transient duration of the paraplegia, appeared to justify the further diagnosis of spinal meningeal hæmorrhage. The patient was apparently well about 4 P.M. when he took a drink of milk. Five minutes afterwards he was found dead in his bed, his death having thus occurred about 52 hours subsequent to the onset of the symptoms.

Necropsy.—The post-mortem examination, which was made within 24 hours of death, showed the right pleural cavity to be filled with blood. The pericardium contained a little clear serous fluid. The heart was rather large, there being hypertrophy of the left ventricle. The arch and upper part of the descending aorta were distended, forming a tumour-like mass from which the blood had escaped by a small rent into the right pleura. On opening the aorta its inner aspect was covered with atheromatous plaques and calcareous plates and a dissecting aneurysm extended from the arch at the level of the innominate artery and terminated in the left femoral artery about an inch below Poupart's ligament. A large mass of freshly coagulated blood was effused between the tissues at the level of the arch and the upper portion of the descending thoracic artery, and here the separation of the tunics took place around nearly the whole circumference of the vessel

[4] Ibid.
[5] Dublin Journal of Medical Science, vol. ix., 1836.
[6] Ibid., vol. xiv., 1844.
[7] Ibid., vol. xv., 1844.
[8] Special Path. Anat.
[9] Diseases of the Heart.
[10] Manual Path. Anat.
[11] Special Path. Anat.
[12] Giornale della Scienza Mediche, 1842.
[13] Dublin Journal of Medical Science, vol. xvi., 1845.
[14] Giornale della Scienza Mediche, 1842.
[15] Boyd: THE LANCET, Jan. 23rd, 1841.
[16] Traité de l'Auscultation Mediate, 2me. ed., 1826.
[17] Fagge: Transactions of the Royal Medical and Chirurgical Society, vol. lii.
[18] Dublin Hospital Reports, vol. iii., 1822.
[19] Transactions of the Royal Medical and Chirurgical Society, vol. lii.

wall. From the abdominal aorta through the left common iliac, external iliac, and femoral artery the coats of the vessels were separated by a thin layer of coagulum, but no palpable thickening of the arteries was so produced. In the lower part of the aneurysm the dissection of the arterial coats did not extend much more than half-way around the circumference of the vessels.

The microscopic sections which I exhibit are from the left common iliac artery and they show that the blood is effused in the substance of the middle tissue of the artery, so that the external wall of the aneurysmal canal is composed of the outer strata of the media together with the adventitia. The dissection was carried along the coats of the right renal artery, which was also occluded by a thrombus. The lumbar arteries were cut across and their orifices likewise contained thrombi. A large coagulum was adherent to the posterior wall of the aorta at the level of the renal and lumbar arteries. I have not been able to satisfy myself as to the position of the internal rupture of the aorta. There are many degenerated spots in the inner coat of the vessel through which it might have occurred, or possibly it took place at the origin of one of the primary branches of the aorta. The capsule of the left kidney was adherent, the organ being cirrhotic; the right kidney was in a similar condition and was, in addition, cystic; it was extremely engorged with blood and the microscopic sections (which were prepared by Dr. Dargan in Dr. MacWeeney's laboratory) show that a universal hæmorrhagic infarction was present. There was no hæmorrhage in the spinal cord or its membrances. In the light of the necropsy it is not difficult to account for the symptoms of this case. As predisposing causes of dissecting aneurysm the patient had extensive atheroma of the inner coats of his aorta, which was consequently unduly lacerable, and in addition he had hypertrophy of the left ventricle with high arterial tension. The intense pain at the onset of the symptoms was due to the primary rupture and separation of the coats of the aorta by the blood. The paraplegia was caused by the interference with the arterial supply to the lumbar region of the cord, partly from thrombi in, partly from rupture of, the origin of the lumbar arteries. The sudden death ensued on the rupture of the external wall of the aneurysm and the consequent escape of blood into the right pleural cavity. It is rather remarkable that the urine contained no blood and that the amount passed was not noticeably diminished, for the functions of the right kidney must have been completely arrested.

Other cases of dissecting aneurysm have been recorded in which a remarkable series of symptoms was due to the arrest of vascular supply to the brain, kidneys, or cord. Tessier's[20] patient died with symptoms of apoplexy, and a dissecting aneurysm was found extending from the arch of the aorta along the innominate artery and its branches to the internal carotid. Todd's case[21] was characterised by hemiplegia and suppression of urine, and the post-mortem examination showed softening of the brain due to obstruction in the carotids, and the renal arteries were also obstructed. Sainet[22] records a case in which the patient became rapidly paraplegic. In Dickenson's[23] case a policeman after seven hours on his beat was seized with loss of power over his lower extremities, followed by pain, collapse, and death in twenty-four hours. Swaine's[24] case was diagnosed during life, the patient having been suddenly seized with pain in his chest followed by paraplegia. Where the symptoms are liable to such variation it is evident that the diagnosis of dissecting aneurysm must be always difficult and uncertain. In reference to treatment Walshe[25] remarks: "Were the practitioner fortunate enough to divine the occurrence of acute separation of the coats of the aorta it does not appear that, in the present state of our knowledge, the management of the case would be materially improved by his sagacity. Did he fail to diagnose the occurrence, his aim would be to restore the patient from the first shock of the accident, control excited arterial action, and relieve the symptoms as they arose. And it does not appear that art could do more than this were the anatomical nature of the affection understood from the first."

Dublin.

[20] Giornale della Scienza Mediche, 1842.
[21] Todd: Transactions of the Royal Medical and Chirurgical Society, vol. xxvii., 1844.
[22] Bulletin de la Société d'Anatomie, 1851.
[23] Transactions of the Pathological Society of London, vol. xiii.
[24] Ibid., vol. vii.
[25] Diseases of the Heart.

A CASE OF DOUBLE LOBAR PNEUMONIA COMPLICATING INFLUENZA DURING THE COURSE OF ACUTE MANIA.

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THE following is a case of some interest, showing as it does the possibility of recovery from double pneumonia complicating influenza under conditions of such special difficulty as to render the prognosis almost certainly fatal.

The patient, a woman, aged thirty-seven years, was admitted into the Coppice Hospital, Nottingham, on March 18th, 1898, suffering from acute mania of three weeks' duration, her leading mental symptoms being extreme restlessness, hallucinations of sight and hearing, and obstinate refusal of food. She professed inability to walk and had to be carried to her room. On examination there were no signs of organic disease except at the apex of the right lung, where there was evidence of consolidation, which from the history was presumably tuberculous in character. The pulse was 84 and the respirations were 22 per minute, while the temperature was normal. The pupils were equal and reacted (though somewhat sluggishly) to light. The muscular power generally was fair, but the knee-jerks were greatly exaggerated. The patient was said to be of a highly excitable disposition and for several years had been subject to attacks of *petit mal*. After admission she was very troublesome in the matter of food, being only induced to take it after much persuasion, while the use of the nasal tube was necessary on one or two occasions. On the 22nd the patient had a slight cough and on the following evening the temperature was found to be 101·4° F., but the rate of respiration was not accelerated; the pulse was 92 per minute and no abnormal signs were found in the lungs apart from the condition at the right apex. On the following morning the temperature had fallen to 99·4° and in the evening it was normal; during the night, however, the breathing became very rapid and the face cyanosed, and the night nurse, becoming alarmed at the patient's condition, sent for the medical officer, when the respiration was found to be 60 per minute, the temperature 103·4°, and the pulse very rapid (160 per minute) and of so feeble a quality that a hypodermic injection of 15 minims of ether with $\frac{1}{20}$ gr. strychnine was given, upon which the patient rallied a little. She was then put on strychnine ($\frac{1}{20}$ gr. hypodermically every six hours) and fed four times daily through the nasal tube as it was impossible to get her to take food otherwise and the bowels were acting so frequently that rectal feeding would have been useless. At 6 P.M. the temperature had risen to 105° and the pulse was 168. Signs of consolidation were now observed over the lower hand's breadth of the right lung behind, the previous examination having given negative results. The patient's condition now appeared to be very grave and death was looked upon as imminent. She was fed, however, and 10 gr. of quinine were given with the food, the temperature dropping in the course of an hour to 103·2°, while the pulse had greatly improved. During the whole of the day the patient had been trying to throw the bedclothes off and had passed urine and fæces under her. On the 25th the temperature at 8 A.M. was 102·4°, and at 6 P.M. was 105·4°. The same treatment was adopted as on the previous day and the result was similar. On the 26th there was evidence of consolidation over the whole of the right lower lobe and a patch of a similar character at the left base. The temperature rose as on the last two evenings and fell under the influence of the same treatment. 5 gr. of quinine were now ordered three times a day. The feeding and injections of strychnine were to be continued as before. On the 27th signs of improvement began to manifest themselves, the temperature being 101·4° at 8 A.M. and 103° at 6 P.M. The case now progressed favourably, the injections of strychnine being discontinued on the 29th and the temperature falling to normal for the first time on the 30th. A slight recrudescence of fever which occurred on April 4th was attributed to the regurgitation and inhalation of a portion of her food during her feeding on the previous evening. After this the temperature dropped steadily to normal, the recovery being uninterrupted, while the signs of consolidation which had involved the whole of