pea-like body on the extensor surface of the left forearm. The teeth had a tendency to a serrated condition of the lower border of the two upper incisors; the gums were inclined to bleed; the tongue clean. His appetite was fair; he was thirsty. His bowels were open four or five times a day. The stools were sometimes formed, at others were pulsaticeous; always of a light yellow colour. This had been the case, his mother said, for twelve months. He had frequently vomited blood.

His abdomen generally was distended, with marked prominence of the upper part. The liver-dulness in the right mammary line began at the sixth rib; in the right axillary line, at the eighth rib; in the vertical nipple-line, it extended three good fingers' breadth below the costal margin. The surface was smooth and hard; the margin slightly rounded. There was no tenderness on palpation. There was a distinct sulcus between the liver and a large tumour which occupied the left side of the abdomen. This tumour descended from below the left costal margin downwards and forwards, crossing the middle line just below the umbilicus. The lower border reached a point nearly an inch below a line drawn horizontally through the superior spinous processes of the ilium. The anterior edge was sharp and notched; the left border slightly rounded; the surface smooth. It was absolutely dull upon percussion, and descended nearly an inch and a half with a full inspiration. There was no evidence of fluid in his peritoneal cavity, neither was there any visible enlargement of the veins of his abdominal wall. His circulatory and respiratory systems were normal. His urine was amber-coloured. Gmelin's test showed the presence of bile-pigment, but not to any amount. There was no albumen nor sugar. From this time to the date of his death, on May 7th, he got gradually worse. His abdominal veins became enlarged, and fluid was poured out into his peritoneal cavity, his legs subsequently becoming anasarcaous. The ascites varied in amount from time to time, but never was sufficiently great as to embarrass his breathing.

Towards the close of his life, he occasionally complained of abdominal pain, but it was never severe. His jaundice was persistent throughout. His urine remained much the same as when first seen, specific gravity ranging between 1007 and 1010, of a deep yellow colour; bile-pigment was always present, but never to any great extent; never any albumen. On one occasion, I found a few granular casts. His midday temperature varied between 99 deg. Fahr. and 100 deg. Fahr.; generally 99½ deg. Fahr.; once it reached 100½ deg. Fahr. He never had any perspirations nor rigors. His pulse ranged between 80 and 120, but tended more frequently to the latter number towards the end of his life. Ecchymoses and petechial spots were occasionally visible on his skin; the small pea-like body upon his left forearm completely disappeared. He had repeated attacks of hæmatemesis. During the last two or three days of his life, he vomited large quantities of blood. For the last nine weeks, there was a systolic murmur audible all over his cardiac region, its point of maximum intensity being situated at the pulmonary orifice. During the last three weeks, his legs, especially the left, were affected by constant coarse tremor. He died comatose.

The necropsy was made thirty-six hours after death, on May 8th, in my presence, by Dr. Maunsell, then resident medical officer to the dispensary. The body was emaciated. Rigor mortis was absent. The peritoneal cavity contained nearly six pints of fluid. The liver was much enlarged; unfortunately there was no possibility of weighing the organ; it gave one the impression of an unusually big liver that was undergoing contraction, and the capsule here and there thickened; the general surface of the liver was finely granular. Upon the upper part of the right lobe, that portion which was up under the costal margin during life, were some rounded eminences about the size of a small pea. The tissue of the liver was tough and leathery. Upon section, it was deeply bile-stained, and presented the usual appearance of cirrhosis. There was no amyloid reaction. The gall-bladder contained bile; the ducts were patent and bile-stained. The portal vein was natural. The glands in the portal fissure were much enlarged and deeply pigmented. The spleen was enormously enlarged, gorged with blood, and somewhat friable. The kidneys were rather large; the capsules separated easily; the surface of each was lobulated; on section, bile-stained; the structure, to the naked eye, was normal. The pericardium was natural. The left ventricle was somewhat thickened; the cavities and valves of the heart were normal. The left lung was normal; the right contained a small spot of cretaceous matter at the apex. The mediastinal and bronchial glands were enlarged. The microscopic examination of the liver, spleen, heart, and kidneys was made by Mr. E. Lawrence. "Specimens were first hardened in spirit and water, afterwards in solution of chromic acid. The liver presented an abnormal amount of fibroid overgrowth, especially in the interlobular spaces, with more or less amorphous granular matter, some few cells only containing oily matter. The spleen was congested throughout. The Malpighian

corpuscles were somewhat increased in size; the trabeculae were rather more fibrous than usual. The heart had a very slight trace of fatty degeneration; the muscular fibres were inclined to break. There was no fat deposited between the muscular fibres. The kidney was congested, otherwise normal."

REMARKS.—When this lad first came under my notice, I felt in considerable doubt as to the nature of his hepatic and splenic enlargement, there being an absence of all the usual factors of cirrhosis. His large, painless, and seemingly smooth liver, when coupled with his unusually big dense spleen, rather inclined me to regard his case as one of lardaceous disease, with enlarged glands in his portal fissure; but against this view was the apparent integrity of his kidneys and other organs, and the fact of there having been none of the usual causes which give rise to lardaceous disease; besides, there was a slight amount of pyrexia present, which is not, according to Dr. Murchison, a symptom of lardaceous disease any more than are jaundice and ascites, unless there be enlarged glands in the portal fissure. The intense jaundice in the boy was infinitely more persistent than in any case of cirrhosis that I have hitherto met with, and was probably due, I imagine, to the pressure which must have been exercised during life upon his bile-duct by the enlarged glands; not sufficient, however, to entirely occlude it, as there was always a certain amount of pigment present in his motions; while, on the other hand, his urine never showed any great amount of the same. Dr. Fagge, in *Guy's Hospital Reports*, 1875, refers to a case of cirrhosis where there was persistent jaundice for seven years, the patient dying at last of hæmatemesis; and he remarks that, when cirrhosis is associated with jaundice, "the liver is not contracted, as a rule, but is increased in size". The point of paramount interest in connection with this case is its etiology. The boy was certainly not a drunkard, neither was there any history of congenital syphilis, or of his having received a chill. After his death, I was informed that, during the last two or three months, he had a "craving" for beer and porter, and I remember his asking me for some eight or nine days before he died. It appears to me rather unlikely that a boy of his age should evince such a "craving" for stimulants, unless he had previously indulged in them. His mother had been a chronic drinker for years, and, during the whole of the time that she was pregnant with him, and the year that she suckled him, she drank more or less to excess, frequently being intoxicated. Now, Gintrac remarks (Dr. Taquet on "Hereditary Alcoholism", *London Medical Record*, Jan. 15, 1878), that the children of female drunkards, if they survive their birth, are often "idiots, imbeciles, insane, or epileptic", the consequences of "congestion or hæmorrhage into the membrane or nervous substance". Why, therefore, may not cirrhosis in children be to some extent one of the manifestations of alcoholic heredity? For, as Dr. Taquet observes, "that alcohol carries into the composition of the fecundating material modifications of which we are ignorant, must be admitted". I believe, however, with Dr. Wickham Legg (*St. Bartholomew's Hospital Reports*, 1877: Cases bearing on Liver-Disease), that cirrhosis may occur independently of alcohol, and may be merely the local expression of some grave general disease. Apart from some of the recorded cases, such as the late Dr. Hillier's, Dr. Hayden's, and the case of a child aged three years, mentioned in Dr. Payne's edition of Drs. Jones and Sieveking's *Pathological Anatomy*, where there has been no history of alcohol, how can we otherwise account for this condition occurring in animals? for Dr. Walshe states (*Diseases of the Lungs*, fourth edition, Art. Cirrhosis) that "the liver of the cow exhibits as perfect specimens of cirrhosis as are ever seen in the human viscus".

ON SPINAL EPILEPSY.

By ALEXANDER M. McALDOWIE, M.B.,
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THE term "spinal epilepsy", applied by Brown-Séquard to paroxysmal convulsions limited to the lower extremities, with persistence of consciousness and sensibility, has been objected to by those authorities who regard epilepsy solely as an idiopathic affection, and who exclude all convulsions resulting from lesion of the cerebro-spinal centres, all excentric convulsions, and, in short, all convulsions occurring when any definite exciting cause can be shown to exist. By these authorities, epilepsy is classed amongst the purely functional diseases, along with hysteria, neuralgia, and some other nervous affections in which no lesions have as yet been detected; and, with them, included under the generic term *neuroses*. In this restricted sense, epilepsy could not correctly be applied to the convulsions described below. But with this limited meaning attached to it, the word becomes merely an expression of our ignorance of the nature of the affection; for we may fairly assume that some

lesion or exciting cause exists in every case of epilepsy. With reference to this, Professor Flint says (*Principles and Practice of Medicine*, page 20, 1873): "Doubtless, in all the so-called functional or dynamic deviations from health, there are either molecular or cellular, that is, anatomical changes, at present inappreciable, or at all events undiscovered, which may be hereafter ascertained by continued investigation with improved means of observation." We shall find, moreover, if we limit the meaning thus, our use of the term decrease as our knowledge of the pathology of the nervous system advances, until it will eventually become erased from our nomenclature.

Brown-Séquard strongly opposes the confining of the term to an idiopathic affection, and even the existence of an idiopathic epilepsy. "It cannot be doubted", he says (*Lectures on the Physiology and Pathology of the Central Nervous System*, pp. 179-80, 1860), "that a disease of the spinal cord or of its membranes, as well as an affection of any centripetal nerve in the human body, may be the primitive origin of a real epilepsy, quite similar to the erroneously-called idiopathic epilepsy." And again: "The spinal cord in animals may be the cause (I do not say seat) of an epileptic affection." An analysis of the history and symptoms of the case narrated below, leaves no doubt as to the spinal lesion being the cause of the convulsive seizures.

On December 24th, 1876, I was hastily sent for to see Ann S., aged 22, a ward-maid, who had fainted while performing her duties in the ward. Consciousness had returned before my arrival; she was then quite sensible, but feeling very sick and faint. Two hours afterwards, she complained of great pain in the right knee and in the lower part of the back, which she had hurt by her fall. Examination revealed a small angular curvature at the junction of the dorsal and lumbar regions, and the patient stated that she had suffered from spinal disease about four years antecedent to the date of examination.

During the next eight days, the patient continued low and faint, complaining of thirst, headache, and sickness. The pain in the back remained with the same severity as at first, although the pain in the knee had completely ceased the day after the fall. There was great tenderness on pressure over the affected portion of the spine, but neither swelling or discoloration could be detected. The temperature ranged between 98 deg. and 100 deg.; rising to 100 deg. on December 30th and 31st.

January 1st. The patient, who had been complaining of severe headache and sickness all day, had a convulsive seizure in the evening.

January 2nd. The patient expectorated about two drachms of blood early in the morning. During the afternoon, she had a fit similar to that on the previous day.

January 3rd. The patient again expectorated a small quantity of blood.

On January 6th, the patient was again seized with convulsions of the same character as on the two previous occasions, and with the same sequence—a slight attack of hæmoptysis on the following day.

These paroxysms—several of which I witnessed myself during the period which the patient was under treatment—varied in duration and severity, lasting, on an average, a little over a minute. At the commencement of an attack, the patient suddenly grasped both sides of the bed, keeping the arms quite stiff, while the back became arched and rigid. The lower extremities then became violently convulsed, remaining so for about a minute, when the spasm gradually subsided. The contractions were of a clonic character, and the inferior extremities were alone affected. The patient retained consciousness throughout the attack, the features wearing an expression of great suffering—indeed, she sometimes screamed on account of the intense pain in the back. With the exception of a slight embarrassment at the commencement, the breathing was unaffected during the paroxysms; they presented none of the three stages of interrupted, of violent, and of laborious respiration which are observed during an ordinary fit of epilepsy.

The occurrence of these attacks led to a more careful inquiry into the previous history of the case, and the following facts were elicited. About four years previously, the patient had experienced a sense of weakness in the back, followed by a dull aching pain, which was increased by exercise or by any sudden movement of the back. The pain—slight at first, and very gradual in its development—had existed for three months before she consulted a surgeon, who at once detected spinal disease, and ordered her to be kept entirely in the recumbent posture. After she had been confined to bed for some time, the curvature formed. She then began to suffer from severe cramps in the legs, which were followed by fits similar to those with which she was now afflicted. The fits were very slight at first, but gradually increased both in strength and frequency for more than two months, when they slowly declined. The fits, when at their height, were much more severe than the present attacks, and also more frequent, as on several occasions she had as many as three in one day. She stated that she sometimes lost

her consciousness during an attack and occasionally bit her tongue. The arms were never affected with the convulsions. She had had four or five fits yearly since, but her general health was very good and her back felt quite strong. Menstruation was regular. Physical examination detected no abnormal condition of any of the internal organs.

On January 9th, at the earnest solicitation of the patient, she was allowed to get out of bed for an hour or two, as she had been quite free from pain and spinal tenderness for two or three days. When out of bed, she thoughtlessly exerted herself moving a heavy box, and injured her spine a second time, so that she had to be assisted to bed. The same night, she had several severe convulsive attacks. Next evening, she had two more, followed on the succeeding day (the 11th) by a slight attack of hæmoptysis. The patient was confined to her room for about ten days; but she remained under observation for six weeks, during which period she regained health and strength and remained quite free from convulsive attacks. She also lost all spinal pain and weakness, although the curvature existed as before. She was then discharged from the hospital service.

There can be no doubt that the above convulsions were of an epileptic character. Although the patient occasionally manifested symptoms approximating hysteria, yet the short duration of the seizures, their intensity, and their limitation to the inferior extremities, are differential points which separate them from those of the hysterical type. The existence of a definite source of irritation—the spinal lesion—and the amount of pain produced by the muscular spasm, are also distinctive features in the case.

A noteworthy peculiarity of this affection is the absence of that cerebral palsy which is the most characteristic phenomenon of ordinary epilepsy. Spinal epilepsy is, in fact, a purely motor neurosis, affecting neither sensibility nor consciousness. Dr. Russell Reynolds classes it as a variety of *epilepsia abortiva*, although he is of opinion it is not a true epilepsy, and ought not to be included under that term. He places it last on his list of four varieties; *epilepsia mitior*, or *le petit mal*, occupying the first place in the group. "At the one extreme", he states, "we have attacks consisting of evident disturbance in regard of mind alone; at the other, paroxysms without notable loss of consciousness; and, between the two, a combination—in divers degrees of intensity—of general spasmodic contractions, with mental obscuration." (*Epilepsy: its Symptoms, Treatment, etc.*, page 81, 1861.) When we consider that both elements of the disease have a common origin; and that the loss of consciousness, during the first stage at least, is due merely to the middle coat of the arteries of the brain becoming involved in the general muscular spasm, diminishing the calibre of the vessels and arresting the cerebral circulation; we can readily conceive how a fit may be rendered incomplete by the absence of either convulsions or unconsciousness, according as the contraction is limited to the parts above or below the medulla oblongata. I have recently seen two cases of *le petit mal*, occurring after injury to the head. In one case, the only convulsive movements observed were slight tonic contraction of the right arm and twitching of the face, while the loss of consciousness was complete, although it lasted only a few seconds, the patient appearing quite stupid for a minute or two afterwards. The patient showed, in addition, symptoms of mental failure; both memory and apprehension being greatly impaired. The other case also presented complete insensibility, but there was entire absence of convulsions. A third case, which came under observation some months ago, was one of *epilepsia gravior*, occurring after fracture of the frontal bone in the right superciliary region. It had existed for nine years. The convulsions were well developed, but the loss of consciousness was extreme, the patient remaining in a state of profound coma for nearly twenty-four hours. It may be that, when an epileptic affection is produced by irritation of the nervous centres situated within the cranium, the loss of consciousness is more marked than the muscular contractions; while the reverse happens when the exciting stimulus affects the spinal cord, or any of the spinal nerves.*

Lesions of the cord, however, are but the remote cause of the convulsions in spinal epilepsy. The proximate cause, as in ordinary epilepsy, is to be referred to the medulla oblongata: the spinal cord probably acts only as a conductor, transmitting the impulses it receives from the brain to the muscles. In the well-known experiments of Brown-Séquard, when the disease was produced artificially by division of one-half of the spinal cord, or of the posterior columns, the epileptic symptoms did not occur until about three weeks after the operation. The inflammation produced by the wound at the inferior part of the spinal cord had created a condition of exalted sensibility, which had gradually

* Since this was written, other three cases of traumatic epilepsy have come under observation. In two cases it occurred after fracture of the frontal bone; in the third (complicated with hemiplegia), after fracture of the right parietal bone. In all these the loss of consciousness was much more marked than the convulsions.

extended up the whole of the cord, and eventually to the medulla oblongata. Schroeder van der Kolk (*On the Proximate Cause and Rational Treatment of Epilepsy*, Sydenham Society, 1859, page 223) has shown that the medulla oblongata contains, in addition to numerous groups of ganglia both for sensitive and motor nerves, special auxiliary ganglia, possessing the property of exciting, even in the healthy condition, on the application of any stimulus, numerous reflex phenomena in different groups of muscles. He affirms that, as a consequence of augmented irritability, this capacity for reflexion is greatly increased, and in epilepsy rises so high that the reflex phenomena manifest themselves spontaneously and without external irritation. "Moreover," he adds, "what is most important, these phenomena are not constant; but, as Brown-Séquard expressly observes, the fit does not last long, but is often repeated."

In reference to the nature of the spinal irritant, it is interesting to notice the result obtained by Kussmaul and Tenner, in their experiments on the production of epileptic convulsions by profuse hæmorrhage. Rapid abstraction of blood from the nervous centres situated within the cranium produced general convulsion; but the sudden production of anæmia of the spinal cord was followed by paralysis of the hinder part of the body. In only three of the animals experimented on, a slight tremor preceded the paraplegia, and in these the trembling movements lasted only for a few seconds, and were immediately followed by total paralysis of both hind legs. The fact that, in the case detailed above, the convulsions first manifested themselves eight days after the injury, points to some hyperæmic or inflammatory condition having been developed in the cord—a view which the experiments of Brown-Séquard favour, and the experiments of Kussmaul and Tenner tend to support.

ON THE TREATMENT OF DROPSY OF THE GALL-BLADDER BY OPERATION, WITH NOTES OF A SUCCESSFUL CASE.*

By GEORGE BROWN, M.R.C.S. ENG.

AT a meeting of the Medical Society of London, held on October 17th, 1859, Dr. Thudichum read a paper on the Pathology and Treatment of Gall-Stone, in which he suggested for the consideration of surgeons the advisability in appropriate cases of an operation for the removal of gall-stones through the abdominal walls.† From the report of the meeting (*Lancet*, October 22nd, 1859, page 421), the idea does not appear to have been received with much favour by the surgeons present, and Mr. Hilton, the President, said that he thought there were many difficulties in the way of such operation. He should like to hear from physicians the number, nature, and prospects of such cases in which the operation for extracting gall-stones could be thought of. The operation would be impossible in cases where the calculus was closely embraced by the bladder. But, from his knowledge, he thought it not impossible that cases fit for operative relief might present themselves; and, in cases of distended gall-bladder (which might occur with calculi), an operation such as the author had mentioned had actually been performed with success. I have looked through several medical journals, but have been unable to find any report of such a case. The only case at all like it of which I could find any notice is one which was brought before the Medical Society of London by the late Mr. Harvey, on January 29th, 1849, in which he tapped an abdominal tumour which had been diagnosed to be an ovarian cyst, but which ultimately proved to be connected with the liver, and drew off, it is stated, no less than sixteen quarts of fluid. The patient recovered from the operation; but some months after the cyst refilled, and the patient died from exhaustion. (*Vide Lancet*, vol. i, 1849, page 183.)

It is singular that, although nearly twenty years have passed since Dr. Thudichum proposed to cut down on the gall-bladder in suitable cases, the operation has scarcely occupied the attention of the profession (although its feasibility must have occurred to many surgeons) until the publication of Dr. J. Marion Sims' paper in the *BRITISH MEDICAL JOURNAL* of June 8th last. When Dr. Sims wrote that paper, he was under the impression that the operation was unique; but it will be seen, from the notes of the case which I have now the honour of bringing to the notice of this Association, that at the time when Dr. Sims operated on his patient, one on whom I had performed a somewhat similar operation was then convalescent.

The patient, Mrs. C., aged 45, wife of a carpenter residing in Isling-

ton, a tall, thin woman with very sallow complexion, consulted me, at the end of March 1877, on account of an abdominal tumour. I obtained from her the following history.

She was the mother of six children, all alive; the youngest seven years old. She had enjoyed very good health during the time of child-bearing; but, for about the last six years, had suffered from indigestion, pains of stomach, constipation, and occasional attacks of bilious colic. The catamenia were still regular. Her present illness dated from early in February 1877, when one day, after cleaning windows, she had felt pain in her right side below the liver. She took no particular notice of the pain at the time, concluding that she had strained the muscles in overreaching whilst cleaning the windows; but, on going to bed, she rubbed her side, with a view to relieve the pain, when she felt a hard lump. Next day, she called in a local practitioner, who told her she had a tumour of the side, which diagnosis was confirmed by his partner, who added that its nature and situation were such that nothing could be done for it by way of treatment except to endeavour to relieve pain. As she could obtain no information from these gentlemen beyond the assurance that she suffered from "tumour", she went to one of the metropolitan hospitals specially devoted to the treatment of diseases of women. The physician in attendance diagnosed "abdominal tumour", but would give no opinion as to its nature. He suggested, however, that he should attend her at home, where he would "puncture" the tumour to see if it contained fluid. She promised to consider the matter, but decided not to return to the hospital.

On examination, I found a tumour in the right hypochondriac region, extending from the lower border of the liver to about four inches below it. On percussion, its lateral boundaries were found to be limited by two lines—one drawn from the right nipple to the spine of the pubes, and the second just half an inch to the right of the umbilicus. It was pyriform in shape; the narrower part of the tumour was directed towards the liver, and it could be grasped through the abdominal walls. To the touch, it gave one the impression of being fully as wide at its widest part as a medium-sized lemon. It was free from the abdominal wall, and could be moved from side to side. On asking the patient to take a deep inspiration, it responded to the movements of the diaphragm, from which I concluded that it was attached to the liver, and I felt no doubt that the tumour was an enlarged gall-bladder. (The rough sketch Fig. 1 will give some idea of its size and relations.) Whether the enlargement was due to fluid or gall-stones, I was unable to deter-

Fig. 1.—Mrs. C. Dropsy of Gall-Bladder. March 1877.

Fig. 2.—Mrs. C. Dropsy of Gall-Bladder. January 1st, 1878. A. represents the point of Aspiration Puncture.

mine, as no fluctuation could be detected. As she was suffering from no constitutional disturbance, and felt equal to the management of her household affairs, I advised that nothing should be done for the present except to treat the constipation and biliousness. I kept her under observation for about nine months before any important alteration in her condition took place. During this time, she had two or three attacks of bilious colic (with vomiting of bilious fluid), which were relieved by poultices, turpentine stupes, and opiates. Meantime, the tumour slowly but very steadily increased in size. On the night of the 31st of December last, she was suddenly seized with rigors and extreme pain of the bowels. I saw her next morning, and ordered the usual remedies. She had had a green liquid motion. There was great tenderness over the tumour, which at this time measured about five inches in length and four inches and a half in width. Its right boundary

* Read in the Section of Medicine at the Annual Meeting of the British Medical Association in Bath, August 1878.

† Dr. Thudichum's paper was published in the *BRITISH MEDICAL JOURNAL* for November 19th, 1859.