of the teeth, thereby arming themselves with another weapon with which to combat pain and suffering. In future lectures, I shall hope to have opportunities of showing you examples of those direct and indirect results of dental lesion to which I have alluded to-day, whilst I shall especially demonstrate to you those operations necessary to alleviate pain or to arrest disease, whether temporarily or permanently, by more complicated means. My task will be a labour of love, if I can impress upon you the importance of my subject when considered in its highest *rappports*; whilst I shall feel that, in disseminating amongst you what little knowledge I possess, I shall be contributing in some small way towards the lightening of those burdens, and the assuagement of those many ills to which flesh is heir.

CASE OF GRAVE LESION OF BOTH CORPORA STRIATA;

RECOVERY FROM HEMIPLEGIA; EXTENSIVE CEREBRAL DEGENERATION; DEMENTIA; DEATH TWELVE DAYS AFTER RUPTURE OF INTRATHORACIC ANEURISM.

By W. JULIUS MICKLE, M.D.,
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E. B., a private in the 19th Regiment, was admitted into Grove Hall Asylum on May 10th, 1873, having previously been under treatment in India and at the Royal Victoria Hospital, Netley. He was single, aged 37; had seen eighteen years' service, sixteen of which had been passed in India. The attack of insanity for which he was admitted was stated to be the first, to be of about six months' duration, and was supposed to be the result of hereditary predisposition and tropical climate. A brother had been insane, and formerly under care in the lunatic wards at Netley. While still serving in India, E. B. suffered from an attack of hemiplegia in March 1872, but was said to have "recovered" from that, and to have subsequently become the subject, in succession, of rheumatism, of debility, and of mental derangement. Previously to his admission here, he had been fussy, childish, forgetful, demented, and, at times, confused.

On admission, his height was 5 ft. 5 in., his weight about 10½ stones. He was much bronzed by exposure to sun; the skin of the face was of a greasy parchment-like appearance; his hair was short, thin, and streaked with grey. The pupils were slightly irregular, the ocular fundus hazy. There was faintly marked right facial paralysis, and occasional very slight dragging of the right heel in walking; the grasp of the hands was about equal. His speech was low, mumbling, indistinct, but not stammering or tremulous. There was the scar of an old bubo in the right groin. The mental symptoms enumerated above still continued; there was great loss of memory, and the manner was childish and silly. He did not at all know how long he had been here, stated that he was forty-five years of age, and that his father had died two years before at the age of forty-six, and so on.

Double ophthalmoma appeared during the earlier period of his residence here; but, after a few months, the traces of hemiplegia became even still less, and he seemed rather brighter, took exercise regularly, played interminable games of cribbage, etc., with his intimate companions; and his general health seemed to improve for a time under tonics. He made no special complaint of pain, or of any chest trouble, or of any symptom which might give rise to a suspicion of aneurism; but, on December 24th, 1874, it appears to have been noted that there was some cough, that the vesicular murmur generally was defective, that laterally there were large sonorous *râles*, and posteriorly some dulness. He was as usual until December 29th, when sudden profuse hæmoptysis occurred after he had retired to bed. The pulse was rapid, and there was irritable hacking cough. Absolute quietude was ordered; and ergot, gallic acid, and small doses of Dover's powder were administered. Profuse hæmoptysis recurred the same night at 3 A.M.; and, next day, there were dulness on percussion, distant respiration, and indistinct crepitation over the back of the left lung, and, in front, dry *râles* and feeble respiratory murmur, followed on the ensuing day by cooing expiratory *râles* at the front and side, and distinct crepitation and dulness posteriorly. On January 2nd, decided pleuritic friction-sound replaced the latter sign, and the dulness about the base and back was increased. Pleuritic creaking and percussion dulness continued to increase over nearly the whole posterior and lateral surfaces of the left lung, where, also, vocal resonance and fremitus were diminished or lost. But not only was there large effusion evidently present in the left pleural cavity; much of the lung, also, was consolidated; in various parts over the upper and anterior portions were increased vocal

resonance and fremitus, blowing, bronchial, or tubular breathing, with occasional large sonorous *râles*. There were exaggerated respiration and compensatory expansion on the right side; and, throughout, the measurements of the halves of the thorax were about equal. The urine became dark and smoky; dyspnoea came on, and gradually increased; the temperature was high throughout, the sweating profuse, the mind of its usual degree of clearness. The respiratory symptoms became more urgent day by day, and he gradually sank and died on January 10th, 1875. During this illness, the pulse and respiration on the successive days were: Pulse, 92, 100, 105, 114, 100, 108, 110, 120, 120, 128; respirations, 22, 25, 25, 30, 34, 32, 44, 42, 54, 60; temperature (third day), 102 deg.; last two days, 102.8 deg., and 103.6 deg.

NECROPSY, twenty-one hours after death.—*Head*: The vessels at the base were healthy. Much serum drained away during removal, etc., of the brain. The arachnoid and pia mater were thickened; the latter was oedematous. There was a considerable amount of sub-arachnoid serum over the frontal and parietal lobes, especially on the left hemisphere, filling up the wide and rounded sulci, and more particularly collected about the sulcus præcentralis, sulcus interparietalis, and fissure of Rolando. The membranes were but very slightly opaque, stripped off readily, and left scarcely any shreds in the sulci. There was moderate atrophy of the frontal and of the parietal gyri, with grooved indentation, and some wasting, of the inferior surface of the right occipital lobe. The cortical grey matter was rather thin, and of ordinary consistence and colour; its stratification was fairly marked, and its minute vessels unusually obvious. The medullary substance had a cribriform appearance from numerous dilated vascular channels; its consistence and sanguineous points were ordinary. The specific gravity of many of the convolutions was tested; but it need only be stated here that the departure from health in this respect was in two directions: firstly, the specific gravity, generally speaking, was .001 to .002 lower in the left than in the right hemisphere; and, secondly, the specific gravity of the posterior region more nearly approximated that of the middle and anterior than is usual in the healthy brain. The fornix was firm. The ventricles contained a moderate amount of serum; their ependyma was thick and firm. The choroid plexus and velum interpositum were adherent to the optic thalami; the third ventricle was dilated; the grey commissure absent. Certain areas of the intraventricular aspect of both corpora striata were softened, collapsed, shrunken, and of a drab yellowish or greyish tint. This change was more extensive in the left corpus striatum, the upper surface of which presented a patch of it, coarsely triangular in shape, of about the size of a sixpence, commencing a quarter of an inch behind the anterior border of the corpus; behind this, again, was a rounded and smaller area of *ramollissement*. There was, also, a patch of the same change on the intraventricular aspect of the right corpus striatum, similar in extent to the larger of those just described in the left ganglion, but placed more posteriorly and externally. On section, the softening and disintegration in the left corpus striatum were found to extend throughout the whole length of that body, and passed half an inch behind the level of the posterior border of the obvious change upon the superior surface; the two portions of softened, atrophied, and discoloured tissue, above described, jutting upwards, as it were, from the main body of the lesion, and cropping upon the intraventricular surface. On transverse section, the depth of the naked-eye change was found to be half an inch in the inner and middle thirds, and a quarter of an inch in the outer third. On section of the right corpus striatum, a similar lesion was seen, half an inch in depth, but less extensive, and situated slightly further back than that of the left side. In both, there appeared to be many vascular connective tissue-bands, the interstices of which were filled with disfluent material, which exuded on pressure. The optic thalami were slightly shrivelled. There were two small, firm-walled, cavities in the medullary substance, containing fluid *débris*, and traversed by minute bands, the larger of which lay immediately above the body of the left lateral ventricle, and the smaller above the anterior horn of the right. The average specific gravity of right corpus striatum was 1032½. The weight of the entire encephalon (*i.e.*, with two innies tunics, etc.) was 43 ounces; of the right cerebrum, 18½ ounces; of the left, 17¾ ounces; of the cerebellum, 4½ ounces; of the pons Varolii and medulla oblongata, ¾ ounce.

Microscopical Examination.—The vessels of the right optic thalamus, examined when fresh, were fatty, and the right corpus striatum was strewn with oil-molecules and large granular cells; there were granular and fatty deposits on its vessels. Prepared sections of the left corpus striatum exhibited fatty molecules, amyloid bodies, granular masses, and *débris*, twisted and broken nerve-fibres, fatty vessels, and points of much wavy connective tissue. In other sections, fatty-like molecules were still more prominent, and occurred either in aggregations, or widely strewn, or in fat-laden cells. The vessels of this corpus striatum pre-

sented a variety of alterations; such as the walls thickened, or covered by fatty molecules, or with dark granular intramural deposits. In places, there was bulging of the walls where they were thickened or replaced by the granular or the fatty deposits. The lumen of some was blocked by *débris*, others were tortuous, or kinked, or presented deposits at the bifurcations. In the fresh cortical substance, the capillaries were thickened, and dark granular masses were apparent in their walls; their outlines were irregular, and, at points, very slightly moniliform. In prepared sections from the left ascending parietal convolution, were rounded compound granular masses. The pyramidal nerve-cells were small, some granular; their fine angular outlines had disappeared, and their nuclei were rounded. There were also small round or oval cells containing granular matter, and some of the vessels were fatty or tortuous. In the first left frontal gyrus, were granular degeneration of the nerve-cells; granular collections on, and fatty like degeneration of, the walls of some of the vessels, whose outlines were thereby rendered irregular; some dilated perivascular spaces and points where shrunken vessels lay within their sheaths. In the tip of the left occipital lobe, the nerve-cells were small and granular; the vessels tortuous, thickened, or their walls bulging with deposits, mainly of a granular character.

Thorax.—The feature of great interest here was an aneurism of the descending aorta, where its coats were extensively atheromatous. The aneurism was of the size of a large orange, was situated just below and behind the root of the left lung, and had grown forwards and to the left side; it had ruptured and still contained loosened masses of firm laminated fibrine covering its walls, and projecting at the rent. The sac was adherent to the spine, to the root and internal surface of the left lung, and was surrounded by large dark clots. The lower half of the internal surface of the lung was hepatised, and was deeply burrowed into, and irregularly excavated, by the aneurism and by the extravasated blood, which latter had passed through the disintegrated pulmonary tissue, and opened into some of the bronchial ramifications. The rest of this lung was inflamed; it was hepatised in parts, and was gorged with blood, and hæmorrhagic nodules were found here and there throughout its substance. It weighed $35\frac{1}{2}$ ounces, and rode upon blood-clot. Thirty-four ounces of fluid blood and of clot, mingled with some lymph, from acute pleurisy set up by the extravasation, were removed from the left pleural cavity, the walls of which were also thickened by old pleuritic membranes. The bronchi were deeply congested. The right lung weighed 17 ounces; there were emphysema and some old pleuritic adhesions; two-and-a-half ounces of fluid were contained in the pleural cavity. The heart weighed $10\frac{1}{2}$ ounces; the left ventricle was contracted; the mitral valves and aortic arch slightly atheromatous.

Abdomen.—The cones of the kidneys were deeply injected; the cortices granular and mottled; the capsules adherent. The right kidney weighed $4\frac{3}{4}$ ounces; the left, $5\frac{1}{4}$ ounces. The spleen was firm, pale, and weighed nine ounces. The liver weighed 65 ounces; it was pale and flabby; there were a cicatrix on the upper surface, and starred depressions on the upper and lower surfaces near the anterior border, connected with each other by septa of firm white fibrous bands traversing the gland.

REMARKS.—1. The absence of any noteworthy symptoms of aneurism was due to the partial filling of the sac with laminated firm clot, and to the situation of the tumour at a point of the descending aorta, where it did not implicate the pulmonary plexus to any marked extent. If, however, perception and sensation had not been blunted, the relation of the sac to the bronchus and vessels would probably have induced E. B. to complain of his chest. The patient survived twelve days after the rupture of his thoracic aneurism. The lung immediately adjoining the sac received the first brunt of the hæmorrhage; it was extensively inflamed, and the hepatised and engorged tissue was made ragged by the burrowing blood, which had found its way into the bronchi, on the one hand, and had caused profuse hæmoptysis; and, on the other had escaped into the left pleural cavity, and had set up acute pleurisy, with fibrinous effusion. The cause of the aortic atheroma was not clear. How far the influences of tropical climate, strain of the circulatory organs arising from the exigencies of a soldier's life, or syphilis, were engaged in producing it, cannot now be ascertained. Certain it is that the chronic cirrhosis of the kidneys found after death was only of moderate degree, had given rise to no special symptoms during life, and had not induced cardiac hypertrophy. The weight of the heart was quite within the normal range for a healthy man of his size and time of life, and was about one ounce less than the average found in cases of similar brain-disease in males of a corresponding age. (Burman, *West Riding Reports*, 1873, p. 232.) It is true the hair was prematurely thinned and tinged with grey, the features withered, and of parchmenty hue; and there was arcus senilis; but these indications are not characteristic

of renal degeneration alone. (Fothergill, *BRITISH MEDICAL JOURNAL*, January 9th, 1875.)

2. From the existence of atheroma and of aortic aneurism, one was led to expect that probably the small miliary aneurisms described by Bouchard and Charcot existed in the minute vessels of the brain; and that the attack of hemiplegia was caused by a blood clot, resulting from the rupture of one or several of these little aneurismal dilations, which are not infrequently found in cases resembling the one described above. But this anticipation was not verified by the necropsy; the large vessels at the base appeared to be healthy; and numerous sections, from various parts of the brain, presented, under the microscope, no change of the character specified by the above writers, notwithstanding that the minute cerebral vessels were extensively diseased, and that certain changes on, or in, their walls were revealed; such as granular and fatty molecules, thickening, bulging, or twisting from intramural deposits, occlusion, etc. The microscopical appearances of the vessels resembled rather those observed by Major in senile atrophy of the brain (*West Riding Reports*, 1873 and 1874) than in chronic brain-wasting (*Ibid.*, 1873), differing from the latter mainly in the absence of any marked proliferation of the nuclei of the vascular walls. The changes in the minute vessels and cells were, in some respects, not unlike those found in certain cases of general paralysis. L. Meyer has described alterations analogous to some of those present in the above case, as being merely "characteristic of retrograde metamorphosis of tissues". (Abstract in *Journal of Mental Science*, October 1872.)

The cerebral degeneration was, in all probability, largely due to the abnormal state of the nutrient vessels, aided by the influence of the lesions of the corpora striata. No doubt the hereditary inclination to nervous disease made the cerebral tissues the more ready prey to the general departure from health, and to the premature senility; and there was the further stress of a long residence in the east. Degeneration had advanced to a greater degree in the left than in the right cerebral hemisphere; the weight of the left was the lesser, and the specific gravity of its cortex was lower than that of the cortex on the right side. Moreover, the under surface of the right occipital lobe was rather wasted, and there was not the normal difference of .002 to .003 between the specific gravity of the posterior and of the antero-median convolutional regions. (Bastian, *Journal of Mental Science*, January 1866, p. 493.)

3. But the principal gross lesions found in the brain of E. B. were those affecting both corpora striata. It has been seen how extensive was that of the left corpus, how the greater portion of this ganglion was implicated, especially in its deeper aspects; and how the tissue in the area of morbid change was completely broken down and almost reduced to mere *débris*; while a portion of the medullary substance immediately above the body of the lateral ventricle was converted into a minute cavity containing turbid fluid detritus. It has been seen, also, how, in the right corpus striatum, the complete *ramollissement* was confined to more narrow limits, especially in front, and, as on the left side, was associated with a minute cavity in the medullary substance—in this instance, situated above the anterior horn of the ventricle. Though one could not positively assert the contrary, there seemed to be no proof that the patches of *ramollissement* marked the sites of former hæmorrhages from ruptured vessels. Judging from the vascular condition, the portions of limited softening were probably due to thrombosis of the minute degenerated vessels supplying these regions; thrombosis which occurred long before he came under care here, and of which no noticeable trace might remain in the vessels after the lapse of so long a period. The portions of softened tissue were those mainly supplied by twigs from the middle cerebral artery to the corpora striata; and, on the right side, by those twigs, also, which pass onwards to the anterior and external portion of the optic thalamus. There was no history of the mode of onset of the attack of hemiplegia, from which he virtually recovered; and, therefore, no data for a clinical diagnosis of the particular lesion which produced it primarily. The average specific gravity of the various portions of the striate body was $1032\frac{1}{2}$, or considerably below the standard, which is about 1038 to 1039.

It was a feature of interest that, while so much of both his corpora striata were destroyed for all practical purposes, this man's hemiplegic symptoms were only slight when he was admitted, and scarcely continued beyond the earlier months of his residence in this asylum. True he was inert, indisposed to move, and walked slowly; but this, though partly from feebleness, was mainly the result of his mental condition. Part of the routine of his life here was to fall into the ranks, and march in the grounds with his fellow-patients. The only history of palsy to be obtained was that of an attack of hemiplegia about three years before his death—of which side not stated, the patient himself said it was of the right—and slight dextral hemiplegia existed when he was admitted. No history of symptoms due to the lesion in the right corpus striatum was known, and no symptoms of this character were observed while he

was here, unless, indeed, it aided in causing his inaptitude for exertion.

Finally, there was no aphasia. Speech was low and mumbling, he was disinclined to converse, his replies were usually nearly monosyllabic; but, when urged, he could, and often would, speak more fully to the point, and used his limited vocabulary without hesitation or mistake. The use of language was partly restricted by his mental condition; the impairment of articulation was merely of a paretic nature, and the essential characters of aphasia proper were absent.

HYDATID DISEASE :

AS ILLUSTRATED BY SPECIMENS CONTAINED IN THE PATHOLOGICAL MUSEUMS OF THE METROPOLIS AND IN OTHERS ELSEWHERE.

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IV.

BEFORE concluding my summary of the human hydatids contained in the metropolitan and certain other museums, there is in this connection an interesting literary contribution that I cannot pass unnoticed. In the November number of the *Indian Medical Gazette* for 1870, an article occurs in which it is stated that the Calcutta Medical College Museum contains eighteen specimens of hydatid cysts of the liver. This fact was, it seems, originally adduced to show not the frequency, but rather the rarity, of the occurrence of hydatids in India. However, from a valuable communication by Dr. James Cleghorn, which was published in the same periodical for the following March, it appears that hydatids of the liver are much more common in India than is generally supposed. This, according to Cleghorn, is owing to the circumstance that many of the so-called cases of tropical abscess are neither more nor less than examples of hydatid cysts that have suppurated.

I now turn to a phase of the subject from which much practical instruction may be gathered. The consideration of the pathological phenomena of hydatid disease as it affects the lower animals is of high interest, and no prejudice should induce any medical man from accepting such useful data as may be gathered from this source. The facts of hydatid parasitism in animals, though often peculiar, are, for the most part, of an order similar to those presented in the human subject. If any practitioner think it beneath his dignity to study the pathology of the lower animals, the conduct of John Hunter in this respect is a standing protest against such narrowness.

The museum of the Royal College of Surgeons of England contains some of the finest specimens of hydatids from the lower animals that are to be seen anywhere; the very choicest of them having been selected by Hunter himself. That distinguished man sought information from every available source; and hydatids were for him of almost equal interest, whether found in the body of a human being or in the carcass of an ox or an ass. Now, at all events, neither pathologists nor sanitarians can well afford to neglect comparative pathology; and, for myself, I am free to say that the yearly exposition to the students of the Royal Veterinary College of the phenomena of parasitic life amongst animals has brought with it an ever increasing knowledge of the most curious and often unlooked for information. Some of the data thus supplied are quite remarkable. Let me also add that my studies of the entozoa of wild animals have put me in possession of particulars of the highest value in regard to the larger question of the origin of epidemics. Beasts, birds, reptiles, and fishes of every description are liable to succumb to internal parasites, and there is practically no end to the variety of useful information to be obtained from this source. I have collected materials for a large treatise on this department of the subject, but I fear I shall never have either the means or opportunity of giving the facts due publicity. Here, for obvious reasons, I restrict myself to the hydatids, more or less properly so-called.

Referring, in the first instance, to those true hydatids of animals that have the same mode of origin, and that exhibit the same general characteristics as those found in man, I notice that four of the museums exhibit nine examples of liver echinococci. The Hunterian collection shows specimens of this kind from the pig, monkey, zebra, and lion. The museum at St. Bartholomew's Hospital contains two examples from the pig and one from a cow; whilst the animal liver-hydatids preserved in the King's College and Guy's Museums, respectively, are from the pig and sheep. That from the latter is partly calcified. Respecting animal hydatids affecting the lung, the Cambridge Museum exhibits a simple acephalocyst from a monkey, and the Guy's Hospital Museum shows a pulmonary hydatid from the kangaroo. In the museum at Oxford, Dr. Acland's (pathological) department shows a preparation of

"one large echinococcus cyst from the abdomen of a baboon", whilst Dr. Rolleston's department (anatomical) displays the echinococcus itself from the "cavity of the abdomen of the same animal". The collection also contains a variety of other bladder worms from different animals. The Hunterian Museum, Lincoln's Inn, exhibits four or five alleged examples of hydatids from the kidney of the sheep, besides another from the spleen. Some of these are very doubtful in character. A cystic kidney from the sheep, preserved in the London Hospital Museum, and originally supposed to have been due to hydatids, is (as hinted in the MS. catalogue) certainly not of parasitic origin. In regard to the occurrence of hydatids in the heart of animals, the Hunterian series shows two good examples from cattle, whilst the collection at University College exhibits one taken from the wall of the left ventricle of a sow. This was presented by Dr. Elliotson.

In the museum of the Royal Veterinary College, Camden Town, there are a number of excellent preparations of true hydatids taken from various animals, especially from cattle, swine, and sheep; and there are also many kinds of bladder-worms which, though often called hydatid by veterinarians, have a totally different origin from that of the true echinococci. The so-called gid-hydatids (*canuri*) and slender necked hydatids (*cysticercus tenuicollis*) are of this description. Specimens of the polyccephalous brain-hydatid, or *conurus*, also exist in the museums connected with St. Bartholomew's, Guy's, and St. Thomas's Hospital Medical Colleges, as well as in both the anatomical and pathological departments of the Oxford Museum. Specimens of large *conuri* occurring in the soft parts of rabbits may be seen in the Guy's Museum (presented by Mr. Carpenter). Similar characteristic specimens exist in the Oxford collection, labelled *C. cuniculi*, obtained from the "maseter and infrapinatus" muscles of a rabbit. My private collection also contains a recent addition of this remarkable hydatid, sent to me by Mr. Alston from Ayrshire. It is the only one I have seen from Scotland. Three examples of the slender necked hydatid (from a monkey and two sheep, respectively) may be seen in the Guy's and University College collections, and several are preserved at the Royal Veterinary College.

I cannot go out of my way to speak of other bladder-worms, except so far as to call attention to the heart of a bear preserved in the museum at Guy's, the walls of which are crowded with cysticerci. That unique preparation ought to be more carefully examined and described. Lastly, in this immediate connection, I have only to call attention to the fact that the Hunterian Museum contains two really magnificent specimens of hydatids affecting the bones of cattle. In the one case, a large solitary vesicle occupies the shaft of the humerus; whilst in the other, several "acephalocysts" have taken up their residence within the cancellous structure of the ilium. These facts are of high interest when taken along with certain others of a like character, already adduced in connection with hydatid parasitism as it affects the osseous system of man.

INJECTION OF PERCHLORIDE OF IRON IN POST PARTUM HÆMORRHAGE RESULTING IN RECOVERY.

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IN considering cases in which death has followed the injection of perchloride of iron in *post partum* hæmorrhage, we should be careful not to attribute more of the result to the perchloride, *per se*, than actually belongs to it. In the case reported in the JOURNAL by Mr. Boddy, I cannot avoid the conviction that the manner in which the iron was used was the cause of death. I observe that from the commencement there was decided contraction of the os uteri, requiring "dilatation for the passage of the tube", and also that subsequently "the uterus was so firmly contracted around the tube that some little force was required for its withdrawal". It is evident, therefore, that there could not have been sufficient passage for the free exit of the solution—a point of very great importance. The pipe being introduced to the fundus, the nurse worked the syringe, and sixteen ounces were forced in. The question immediately suggests itself, what became of this fluid? Doubtless, it dilated the uterus, and at the same time escaped at the weakest points. Mr. Boddy contends that fluid could not pass through a contracted uterus, but it certainly might through the placental site of a forcibly distended one, especially if introduced in the jerking manner essential to the working of an "ordinary enema-apparatus". Then, again, why should not some of the solution have escaped through the Fallopian tubes?

In my experience, the two most essential points in the use of the perchloride of iron are—first, to see that there is a free passage