ON A CASE OF RECOVERY FROM ORGANIC BRAIN-DISEASE.*

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THE case I have to bring before this society is one of great practical interest as regards both diagnosis and treatment. In one sense, it must be unsatisfactory, as there is no completion by necropsy. I do not ask anyone to believe that there is or has been local gross organic disease. We learn from the case, however, that a patient may recover from an exceedingly grave symptomatic condition which is often fatal, and such an one as usually does depend on local organic disease. Besides, although incomplete in one way, the case is complete in another, in the very pleasant way of recovery—temporary recovery, at all events. I speak of local gross organic disease. I add the adjective gross, as I do not believe that there is such a thing as a symptom independent of organic change, although of course I admit that the changes may be slight and recoverable, or that they may be undiscoverable *post mortem*.

I read a heading giving the most important symptoms of the case, mentioning some which may not be part of the ailment for which she was treated, and also certain symptoms conspicuous by their absence. The notes of the case were taken by Dr. J. R. Jones and Mr. Sheard.

CASE. *Supposed Hysteria. Double Optic Neuritis, with nearly complete Blindness; Severe Headache; no Vomiting; Loss of Smell; No Deafness; Reeling Gait; Absence of Patellar Tendon-Reflex; Doubtful Lightning Pains; Recovery under Iodide of Potassium and Mercurial Inunction, except for Anosmia and Absence of Patellar Tendon-Reflex.*—Annie L., aged 22, was admitted into the London Hospital, December 22nd, 1879. She was well nourished. She had no disease of thoracic, abdominal, or pelvic viscera; and, when she came round from the miserable state into which she had fallen up to the date of her admission, she was found to be very intelligent, and also cheerful. Her family history, so far as we could get to know it, presented no noteworthy features. Her menstruation was regular, and always had been. There was "a good constitution", an important thing in a case requiring "vigorous treatment". We obtained no evidence of syphilis, although no direct questions were asked her on this matter. The following is the history of the case.

History.—In August 1878, a butcher's cart ran over her, but her legs only were hurt, a little above the ankles; there was a wound of one of them, of which the scar persists. She went into domestic service in May 1879, being at that date quite well, except for what she called a weakness in her legs; she bandaged them up to her knees "for support". The so-called weakness slowly increased; two days before admission, she could not stand at all, and took to bed. During the week before admission, there was a weakness of the right arm, which would go and come; afterwards, "pins and needles" in this limb; and then weakness of her left arm set in. Her sight began to fail two or three weeks before admission. She was sent in for hysteria.

Symptomatology.—The following is the symptomatology of the case on the girl's admission, and for some time afterwards. The symptoms may be divided into, 1. Those which are the best evidence towards the diagnosis of gross local organic disease within the cranium; 2. Symptoms which localise.

There may appear, at first glance, some inconsistency in the wording of these last two remarks. There is in reality none. From certain symptoms—those in the first list—we often do conclude correctly that there is local gross organic disease, and yet are unable to say what the locality is. Then as to localising symptoms; we may feel sure that a certain symptom localises, although we may not know to what locality it points. If a man has had a hundred seizures of epilepsy, each beginning by a noise in his left ear, we may be confident that the "discharging lesion" is of some particular part of his central nervous system, although we may not know what that part is, and may not discover morbid change anywhere *post mortem* (it is true enough that, from Ferrier's experiments, we should, in the particular case instanced, have strong grounds for inference). If local symptoms do not imply local lesions, localisation is a dream.

1. *Symptoms of Local Gross Organic Disease.*—*a. Double Optic Neuritis* (with mere perception of light). *b. Intense Headache.* It is

important to note the absence of symptoms. She had not a third symptom, which often goes with optic neuritis and intense headache; there was no vomiting. An examination of her eyes was made by Mr. Couper, January 7th. They were examined by myself and my assistants; but Mr. Couper's examination is more valuable, as well as more authoritative, than mine. "*Left Eye.*—Great oedema and swelling of disc, with large blood-extravasations. The oedema extends some distance into the retina, adjoining to the disc and macula, giving to the retina there an opaque, yellowish-grey, mottled look. The macula stands out as a deep blood-red patch, the surface of which is obviously cupped as regards the adjoining retina. The macula is emmetropic, the most prominent part of the disc hypermetropic. Although the swelling of the right disc is more than that of the left, the oedema of the adjoining retina is much less; the natural colour of the choroid is seen all round the disc. The chief alteration at the macula is the atrophy of its pigment, which makes it difficult to recognise by its pigmentation; and this difficulty in finding the macula is increased by the patient being blind. The sides of the disc form an abrupt declivity towards the retina. There are numerous hæmorrhages, somewhat radially arranged. The retinal veins are large and tortuous, and the tortuous arrangement is traced far out towards the equator (similar changes, although less in degree, are traceable in the veins of the left disc and retina)." *c. Anosmia.*—On her recovery, we ascertained that smell was entirely wanting; she averred that she used to smell things well before her illness. I do not know whether anosmia is in her case a localising symptom or not. I do not know whether it may have been owing to pressure, direct or indirect, on the olfactory bulbs, or, what is quite hypothetical, to an olfactory neuritis.

To anticipate for one moment: it seems most likely that in this case there was, and may be still, tumour of the cerebellum. In a few cases of tumour of the middle lobe of the cerebellum, I have found loss of smell; but in those cases there was enormous increase of intracranial pressure by effusion of fluid in the lateral ventricles, and this is not likely to have been so here. Although she said she could smell well before her illness, we must not be sure that she could. We know that some patients are unaware of any such loss; they often think it is their taste which is defective.

2. *Localising Symptoms.* *a. Absence of Patellar Tendon-Reflex.* This is an exceedingly important symptom, especially so since her gait was abnormal, and yet not ataxic, nor, I may say at once, paraplegic either. We examined into this matter with very great care, as did also my colleague Dr. Stephen Mackenzie. I put it in the list of localising symptoms, because (since the researches of Westphal and Erb) I believe it to be in many cases a very valuable help to the diagnosis of sclerosis of the posterior columns—the pathological condition for *tabes dorsalis*, or, as it is commonly and yet inconveniently called, *locomotor ataxy*. This reflex was still absent when she left the hospital (March 23rd). It may have been absent long before she had the other symptoms. Of this matter, by the nature of the symptom, we could, of course, know nothing. *b. Pains in the Limbs.* At one spot a little above the left knee, she had occasionally a dart of sharp pain, followed by dull aching; it would come and go. She had no severe pain except in the head. We inquired about pains in her limbs, on account of the absence of patellar tendon-reflex; but I cannot affirm that the pains were the characteristic pains of *tabes dorsalis*. It is well to mention, as Pierret and Buzzard have pointed out, that there may be "lightning pains" about the head in *tabes*. Her head-pain was not of this character. *c. Reeling Gait.* We noted her gait most carefully. On admission, she could scarcely walk at all, and not without help. Her gait, then, so far as it could be observed, was not like that of *locomotor ataxy*. As she improved a little, we could analyse it better; it was evidently a reel—a drunkenlike walk. She swayed from side to side; tended to fall forwards or backwards; one leg would cross in front of the other. Shutting her eyes at these trials could make no difference, as her sight was almost *nil*. She had apparently good power in her legs. Using popular and, as I think, inexact language, there was disorder of co-ordination without paralysis. *d. Other Symptoms.* There was some slight weakness of the left arm; only a very slightly feeble grasp. These symptoms were very slightly marked indeed. There was some tenderness of the lower part of the spine. No very local tender spinal spot was revealed by a hot or a cold sponge, or by manipulation; there was no spinal curvature. There was no atrophy of any muscles, and no defect of sensation. There was no vesical trouble of any kind.

Progress: Rapid Recovery.—We prescribed at first five grains of iodide of potassium three times a day; but on January 4th mercurial inunction was ordered in addition. (The dose of iodide of potassium was smaller than I usually give in such cases.) Under this mixed treatment, she rapidly improved. From what was practically blind-

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ness, she became able to read, on January 26th, No. 1 of Snellen, the smallest of test-types, at six inches, with her left eye; but with the right only No. 3. On the day she left the hospital (March 23rd) she could read No. 1 very easily with the left eye, and could read it with but slight difficulty with the right. Practically, her sight was quite good. She felt quite well; and, except that patellar tendon-reflex was still absent, and that she could smell nothing, we considered her to be entirely free from symptoms. A second ophthalmoscopic examination by Mr. Couper (January 31st) showed remarkable clearing up of her optic discs. "*Left Eye.* No swelling of disc; vessels of normal size; sheaths much thickened both on disc and on adjoining retina; choroidal margin of disc indefinite; disc pale and intranslucent. These appearances are consistent with great increase of connective tissue. The pigmentation of the macula is generally good, except that there is an irregular circlet of minute pale punctiform patches there. There are other similar pale patches between the macula and disc, and all of these are probably produced by partial choroidal atrophy. *Right Eye.* Disc as pale as left, and with probably more connective tissue change. Sheaths of all large vessels much thickened. The positions of the finer vessels are concealed as they dip backwards into the substance of the disc. The connective tissue change spreads a little way into the retina, giving a 'woolly' edge to the disc, and obscuring its choroidal boundary. There is some atrophy of pigment at the macula; the groups of minute punctiform patches are more numerous; and there is more general defect of pigmentation. The rest of fundus in each eye normal." Mr. Couper adds: "There is nothing pathognomonic of specific disease in the changes now seen in each fundus. At the same time, the circular shape of the little atrophic patches resembles that often seen in specific disease of the choroid."

I have already pointed the symptoms in two groups, but, in remarking further on the case, I shall consider some of them in other particular relations. By doing so, I shall have to consider the same symptom several times, and yet from a different standpoint each time. I also speak of some collateral topics necessary to be considered in scientific diagnosis.

Remarks on Symptoms.—Hysteria.—Importance of Routine Use of the Ophthalmoscope in Cases of Brain-disease.—This patient was sent in for hysteria. I say nothing in blame here. Such a diagnosis has been made in the early stages of serious, and sometimes fatal, cases of brain-disease, by the best clinical observers. Diagnosis in the earliest stage of severe intracranial disease is often very difficult indeed. In this case, the affection of sight might possibly, until it became lost, have been put down to hysteria. The ophthalmoscope would have shown optic neuritis doubtless long before sight failed, and thus that the case was, if the girl were hysterical, something more than one of hysteria. Several times have I known the diagnosis of hysteria refuted by the discovery of optic neuritis. I have several times been helped to a correct diagnosis by finding double optic neuritis in patients whom, before ophthalmoscopic examination, I had supposed to be either hysterical or hypochondriacal. We must never forget that this condition in the fundus oculi may exist for a long time without any defect of sight. Although optic neuritis is spoken of as a symptom, it is really a visible pathological condition, with which there may or may not be the symptom defect or loss of sight.

I may at this juncture most conveniently mention the case of a young member of our own profession, who, some years ago, consulted me for severe headache and double optic neuritis (he had caught syphilis at a midwifery case). Later on, he attended a meeting of this society, seeing perfectly well. He had still double optic neuritis, and was examined by several people. He was examined by Mr. Brudenell Carter, Dr. Buzzard, and Dr. Broadbent; all agreed that this patient, who could see well, had well-marked double optic neuritis. Later, he became paraplegic; ultimately he recovered, and now feels and looks perfectly well. He never had any defect of sight of any kind. This I believe to be a case of recovery from organic brain and spinal disease of syphilitic nature. My impression is that optic neuritis is nearly always present in a very marked degree before sight fails, and as this medical man's case shows well it may not fail at all. In the girl's case, the subject of this paper, neuritis might, I believe, have been discovered long before sight even failed. Indeed, it is commoner for us to see in the medical wards, optic neuritis with good vision, than with even impaired vision. Very rarely do I, a physician, see cases of loss of sight with optic neuritis, almost never, if those patients sent to me by ophthalmic surgeons be excepted. If we find optic neuritis either with or without defect of sight, we may be certain that the case is not hysteria—not hysteria only I mean—for plainly, a woman with symptoms of organic brain-disease may be hysterical.

Optic Neuritis and Headache.—I have so often spoken of these symptoms that I will say little now. They are the best evidence of local gross organic disease within the cranium. This statement implies that

they are not decisive evidence of such disease. I have published many cases of intracranial tumour with optic neuritis, and I have published a case of optic neuritis without any sort of local gross organic disease inside the head. My colleague, Dr. Stephen Mackenzie, has recently published such a case.

Tabes Dorsalis?—This diagnosis was in no way tenable, at any stage in which we saw the patient, as an explanation of the whole case. The comparatively rapid onset of the difficulty in walking, the quickly reached climax, inability to stand, and prompt recovery on treatment, show that this was not a case of tabes dorsalis. Again, the gait was not that of such disease. Moreover, the sex was not masculine, as it is in most cases of tabes dorsalis. Further, whilst defect of sight from simple atrophy of the optic nerves is not very uncommon in tabes dorsalis, optic neuritis does not, so far as I yet know, occur as part of the process of that disease. Atrophy follows neuritis in some cases, but nearly always it is ophthalmoscopically to be distinguished from that occurring in locomotor ataxy; moreover, while sight does not return in atrophy of the optic nerve in tabes, it came back under treatment in this case. Nevertheless this woman had one symptom which is often of great value in helping us to the diagnosis of tabes dorsalis. There was no patellar tendon-reflex. This is the only thing pointing to disease of her posterior columns, except the somewhat indefinite account of pains in one part of one of her thighs. The absence of this reflex is a very important symptom. We owe a deep debt of gratitude to Westphal and Erb for their discovery of its significance, and for further researches on the matter to Grainger Stewart, Buzzard, Russell, Byrom Bramwell, and Gowers; and here I gladly acknowledge that I have learned much about this symptom directly from my colleague, Dr. Buzzard. So very rarely is this reflex present in tabes, either in the developed form of the disease, or in cases of lightning pains, or in simple atrophy of the optic nerve before gait is abnormal, that I think it very likely that this woman's posterior columns are diseased, although, as I say, I do not think the other symptoms are attributable to any pathological change there seated; at any rate there must be some break in the loop betwixt her patellar tendon and her quadriceps; for as a certain kind of reflex action is absent, there must be a flaw in at least some one of the parts subserving it—tendon, afferent nerve, centre, efferent nerve, or muscle to be moved. There is an omission to be acknowledged, I can find no note of the condition of the pupils. It is known that in many, if not in most cases of tabes, the pupil does not act to light, and yet usually acts somewhat during accommodation (Argyll Robertson's phenomenon). Buzzard and Erb have suggested that the loss of reaction of the pupil to light is analogous to loss of tendon reflex.

Atrophy of the Optic Nerves and Absent Patellar Tendon-Reflex.—This woman's optic nerves are doubtless a little damaged by the past neuritis; a few fibres are no doubt lost, although the defect of sight is insignificant. We cannot suppose that the severe pathological condition of the nerves observed by Mr. Couper, on January 7th, did no permanent damage at all. But if there remained great defect of sight, and if there were conspicuous atrophy, the changes in the disc, as Mr. Couper describes them on his second inspection, are those after a neuritis; any one would infer that, who knew nothing more of the case than from reading his examination of January 31st. I have now seen a number of cases of atrophy of the optic nerves with absent patellar tendon-reflex, and with lightning pains in the legs, but this is the first time I have known that reflex absent with optic neuritis, or with atrophy after it. The atrophy which may precede, go with, or occur late in cases of tabes dorsalis, is simple, is non-inflammatory; there is, at any rate, nothing usually called inflammatory. I asserted that the optic atrophy in cases of tabes dorsalis differed from that after optic neuritis fifteen years ago (as others abroad had done before me), and was met with some contradiction.*

Reeling Gait.—My opinion is, that the abnormal gait was owing to disease of the cerebellum. It is not, however, of much use speaking of localisation of disease in a case with the complications mentioned, where there is no opportunity of verification or disproof of diagnosis. I do not underrate the symptom, because I do not here comment on it. I read a paper last session describing the case of a patient who reeled, and who died of cerebellar tumour (*Lancet*, January 24th, 1880).

Nature of the Disease and Effect of Treatment.—Whatever the pathology of the case may be, it is quite certain that the patient had the symptoms which are the best, although not decisive, evidence of gross disease of some part of the encephalon. She had another symptom—the reel—which, in chronic cases without ear-disease, nearly always, if not always, points to disease of some part of the cerebellum. It may be asserted that, since antisyphilitic treatment was followed by re-

* See *Lancet*, June 10th and August 26th, 1865; and *Medical Times and Gazette*, September 1st, 1866.

covery, the disease was syphilitic. This inference is by no means valid. There was no evidence of syphilis, except that of which Mr. Couper speaks doubtfully after making his second ophthalmoscopic examination. I do not say that there was not syphilitic disease. All I deny—and I deny it strongly—is the assertion that recovery from such symptoms after antisyphilitic treatment points necessarily to recovery from syphilis. It is certain that recovery for a time from grave conditions, with intense headache and double optic neuritis, occurs when subsequent *post mortem* examination shows local gross organic disease of other kinds than syphilis. I treated her energetically for syphilis, partly on the chance of syphilis being present. I should, however, have adopted the same treatment could I have been certain that there was no syphilis. For the optic neuritis in this case, and, no doubt, the pathological condition for some other symptoms, as in cases of undoubted local gross disease of the encephalon, are secondary changes; optic neuritis is, I think, amenable to treatment, whatever kind of gross local organic disease leads to it.

Here, urging the thing *ad nauseam*, as I have been doing for very many years, I repeat that optic neuritis may exist with good sight; and my impression is that, if found in an early stage, iodides or mercurials would prevent sight from failing. I know that the changes often clear up under treatment. That they would not without I do not know, as I never run the risk of not using either the iodide of potassium or mercury, or both. Nevertheless, my impression is that there would be fewer blind people if, by routine use of the ophthalmoscope in cases of severe headache, optic neuritis was discovered early and treated promptly.

Everybody knows that there may be an abscess or a tumour in the brain of a patient who has no symptoms which lead him to seek medical advice, and that we sometimes find local organic brain-disease, which, during life, we never suspected. It is certain, too, that a patient may have an acute illness from local organic brain-disease, and recover—usually, but not always, with some incapacity, mostly defective sight. (Children with atrophy of the optic nerves are brought to us, with the statement that they became blind during “bilious” or “gastric” fever.) These recoveries, partial or complete, or recovery to good general health, are sometimes put down as so much recovery from meningitis. Without denying that meningitis is the cause in some, I believe they are more often recoveries from acute illness dependent on local gross organic disease. I know some of them to be so. We had a man under care in the London Hospital for severe headache and optic neuritis, who got quite well to all appearance; his sight had never been affected. He was about the wards, and read the newspaper regularly. One day he had hæmorrhage from a brain-tumour, and died. Very many years ago, there was under my care a boy who had an acute illness, with severe headache and optic neuritis, who recovered, except that the sight of one eye was much impaired. He was well enough to attend as an out-patient in the ophthalmic department at Guy’s; but later on, brain-symptoms appeared again. At this juncture the case diagnosed itself: a tumour, cancerous, opened its way through the base of the skull into the mouth; we saw the rest of it inside the head *post mortem*.

The statement that there could not be tumour in a case of recovery, at least temporary recovery, from the symptoms alluded to, is a theoretical one. The fact is, that recovery does follow when there is tumour. Whilst we may not like to affirm that the girl whose case I have related has still intracranial tumour, still, he would, I submit, be a very rash man who would say she had not.

I will mention, too, that tumour may exist for years from the first symptom. A patient of mine had epileptiform seizure limited to the right arm for about six years. He had then no headache, and no neuritis. These symptoms come on later; he died, and tumour of the brain was found. I have seen a boy about seven years before he died with tumour of the middle lobe of the cerebellum for double optic neuritis (with good vision) and reeling gait. In fact, the boy was killed; he had meningeal hæmorrhage from a fall. I may mention that a correct diagnosis was made in this case at the very first by my friend Dr. Young of Aldershot.

Absence of Deafness.—It is very remarkable that, whilst blindness is common in intracranial disease, deafness is rare. (We are not speaking of cases where deafness depends on organic disease of the ear, and in which the ear-disease causes cerebral or cerebellar abscess or meningitis. Most of them are cases of “bone-disease” causing intracranial disease, not of deafness resulting from intracranial disease.) I think that persistent deafness of nervous origin, in a case of intracranial tumour, is a localising symptom; that it points to disease involving or compressing, directly or indirectly, the auditory nerve. I would not deny, however, that disease of Ferrier’s auditory (cerebral) centre on both sides might entail some deafness. With this exception—and I have yet not seen such a case—deafness of nervous origin has an entirely different diagnostic

value from amaurosis from optic neuritis. The former may be of localising value, the latter never is. The former, by itself, is of no value as to kind of brain-disease; the latter, by itself, is of much value towards the diagnosis of local gross disease.

ON ANTHRAX AND ANTHRACÆMIA IN WOOL-SORTERS, HEIFERS, AND SHEEP.*

By JOHN H. BELL, M.D., Bradford.

FOR nearly forty years it has been known in the worsted district around Bradford, that sorters of alpaca and mohair not unfrequently died from a peculiar and rapidly fatal disease, the cause and nature of which were not understood. Although unaccountable, the deaths were generally certified as from ordinary forms of disease. Many deaths from this disease were not recognised as such, and, when they were, single deaths with some weeks’ interval were not much noticed; but, when three or four occurred within as many weeks, of men working in the same room or for the same firm, the sorters became alarmed; public interest was excited; *post mortem* examinations, investigations, reports, and recommendations followed; but all ended without any satisfactory result. The number of deaths increased in proportion to the quantity and quality of these materials which were used. It appears strange that, during all this time, the knowledge of the existence of this disease should scarcely have passed beyond the immediate neighbourhood of Bradford; or that it has not been detected in other towns, where similar wools and hairs are used. I believe it will be found, if looked for, not only among hair- and wool-sorters, but also among other workpeople who are known to suffer occasionally from anthrax or malignant pustule, which is the external form of this disease.

My attention was first directed to this disease three years ago, in consequence of a man whom I knew well dying within seventeen hours from the apparent commencement of his illness. He was at work in the morning; but, feeling weak, left during the forenoon, walked two miles and a half to his home, went to bed, had no rigor, cough, pain, vomiting, or purging—only a feeling of exhaustion, with quickened breathing, low external temperature, and collapse, terminating in death the same night. Since then, in consequence of repeated and positive statements in the local papers to the effect that these deaths might easily be prevented, several inquests have been held on some who have fallen victims to the disease; juries have made recommendations to employers; public attention has been aroused; numerous paragraphs and cases have appeared in the medical periodicals; the Local Government Board have sent down Mr. Spear to investigate and report upon it; the Bradford Medico-Chirurgical Society have appointed a commission to inquire into the causes, nature, prevention, and treatment of this and other allied affections; and Professor Greenfield is doing important work in his special department; so that by-and-by we expect to have full and reliable knowledge respecting it.

During the last few months, I have seen several cases of external anthrax in persons who have come into contact with mohair or dry wools. This has not previously been associated with “wool-sorters’ disease”; it is, however, the local or external form of this disease, and is caused by the introduction of the poison at the part affected. The constitutional form of anthrax, as it affects woolsorters, is not generally attended by external or internal pustule. It is a general blood-disease, caused by the introduction into the circulation of the spores of the *bacillus anthracis* derived from the fleeces of animals which have died from this disease. As there is no anthrax, I have called the disease anthracæmia. It is sometimes epizootic in the countries where these hairs and wools are grown. It affects persons who come into contact with the animals; but their illness and rapid death are not attributed to this cause. The blood of a person suffering from woolsorters’ disease or anthracæmia, when injected under the skin of a rabbit or other animal, produces death in two or three days; and the blood of these, when examined shortly after death, sometimes swarms with the *bacillus anthracis*.

As illustrations of different forms of woolsorters’ disease, I will mention very briefly some cases which have come under my notice during the last two months.

CASE I. *External Anthrax without Constitutional Symptoms.*—J. G., aged 36, for three months had been sorting Persian and Bokhara wools. On Tuesday, June 15th, he noticed a small pimple or heat-spot on the outer surface of the left arm, over the elbow-joint. On the fourth day, it was inflamed and painful. On the seventh day, there

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