CLINICAL LECTURE ON TUBERCULAR MENINGITIS.

Delivered at St. Bartholomew's Hospital.

BY REGINALD SOUTHEY, M.D., F.R.C.P.,
Physician to the Hospital.

BEFORE narrating the cases which I propose to bring before you to-day, I wish to direct your attention to the statistical table placed upon the slate, which records, as I believe, with fair accuracy, the prevalence of this disease at different ages. It is compiled partly from Rilliet and Barthez, and from cases collected by Von Hesser and Dr. Seitz and myself. It would not have been difficult to collect more cases from our own and other hospital records; but the numbers are enough to show the relative frequency of this affection at different periods of life, and to enable you to see that adults share with children a proclivity to a particularly fatal localisation of tubercle.

Ages.	No. of Cases.	Ages.	No. of Cases.
0 to 5 years ..	79	35 to 40 years ..	13
6 to 10 " ..	46	41 to 45 " ..	12
11 to 15 " ..	7	46 to 50 " ..	4
16 to 20 " ..	31	51 to 55 " ..	3
21 to 25 " ..	31	56 to 60 " ..	2
26 to 30 " ..	23		
31 to 35 " ..	16		

267

What I desire to point out to you is, that the disease is apt to be more insidious in its approach and more difficult of diagnosis in the adult than it is in the child. Its clinical symptoms are less precise, almost in the direct ratio of the advancing years of the individual in whom meningeal tuberculosis occurs. To show this, I will select from my case-book some good illustrative cases, certain of which have been quite recently under my care.

CASE I. Tubercular Meningitis following upon Chronic Disease of the Hip-joint.—Eliza Jackson, aged 5 years. The duration of hip-disease was two years. She was a fair, thin, careworn child, entirely confined to bed for more than a year. The symptoms of the present illness dated back only one week; they were vomiting, frequent complaint of headache, listlessness, and constipated bowels.

On admission, her eyes looked heavy and hollow; the abdomen was sunken; both knees were drawn up. The head was not hot. Temperature 99; pulse 64; respirations 32. Examination of the thorax elicited nothing abnormal. The urine was in fair quantity, of specific gravity 1022; no albumen; phosphates were precipitated on boiling. The child understood what was said; answered questions; played with her toys; moved as she was directed. Her vision was good; the pupils were large and equal; the tongue clean and moist. She was restless, rather fidgety; but that was all. There was no high temperature; no irregularity of pulse, only a rather slow one; no twitching or palsy of facial muscles; no starting at night; no account of the sharp peculiar cry that often stamps the disease. Three days later, the symptoms were more unequivocally marked. Her nights had been restless; she was constantly tossing about, boring the pillow with her head, shrieking out whenever she was disturbed to take food. At other times, she would lie drowsy, grinding her teeth; and, although she could feed herself, the tremor of the hands and the uncertainty of her movements made her clutch the cup she carried to her lips with undue effort. The temperature had risen to 100.6; the pulse to 100, and it was intermittent every sixth beat.

The note of November 16th, the twelfth of her illness, reports sickness, bowels not open for two days, and an evening temperature of 101.4. The following night, she had several attacks of convulsions, and passed on to a state of coma, from which she never recovered consciousness, but lay with both pupils contracted; both eyeballs being frequently spasmodically directed to the left side; the eyelids frequently flicking; and her fingers tremulously plucking at the sheet, or rubbing her forehead, or picking the caruncle or outer corners of the eyes. The sphincters were relaxed, and the lips covered with sordes. On the fourteenth day of her acute illness, she died.

The case was very clear during life; and the necropsy showed all the ordinary and characteristic appearances: matting together of the vessels; granular aspect of the membranes; no purulent exudation at the base of the brain; tubercles upon the choroid plexus; excess of fluid in the ventricles; special distension of the fourth ventricle; opalescent

lymph in some of the subarachnoid spaces upon the convexity; tubercular peritonitis; special thickness and tubercular infiltration of the vermiform appendage.

The pathological series of events in this case were as straightforward as they are usually found to be: 1. Some prostrating chronic disease—the hip-joint disease; 2. Inflammation about the vermiform appendage, followed by thickening and tubercular infiltration, tubercular peritonitis, and meningitis.

CASE II. Tubercular Meningitis complicated by Diabetes.—Ellen Rankman, aged 13, a fair-complexioned girl, with blue eyes, ruddy face, well nourished, fat, and looking more like sixteen than her real age, was brought by her aunt from Victoria Park to the hospital on August 2nd. Her illness only dated back one fortnight, and began by sudden vomiting, unprovoked by pain or previous indigestion. The sickness was very persistent after nearly all food that she took, lasting for a week; and then, but not till then, she complained of severe pain in her stomach, and giddiness in her head, so that she could not hold her head up. I do not attach importance to the pain in the stomach. If a person have been vomiting frequently, the abdominal muscles become fatigued and ache, and the stomach itself has cramp-like pains. Her bowels were somewhat confined, so that, five days before admission, she took a dose of Epsom salts, with the effect of producing diarrhoea with increased colicky pain.

Previous Health.—She had had all infantile disorders—measles, scarlet fever, whooping-cough. Two years ago, she fell and hurt her head, and was liable to attacks of giddiness ever since, but never had a fit. Five weeks ago, she had a bad cough, and spat up some blood. A few days before her present illness, she had a chill through being wet through.

Family History.—Two sisters had had cerebral affections. One died of what was called inflammation of the brain; the other, now living, has a large hydrocephalic head, and is very dull and stupid.

Present Condition.—*Subjective.* She was dull and apathetic, sick and giddy on moving. Light hurt her eyes. She said she saw objects either indistinctly or double. She complained of pain in the head, and on the left side of the thorax on taking a deep breath.—*Objective.* Both pupils were dilated, but contracted pretty readily to light. No paralysis was noticed. Temperature 101.8; pulse 72; respirations 28. The tongue was coated, and inclined to dry. The urine was in fair quantity, 1020, acid; it contained no albumen. The heart-sounds were clear and loud. The thoracic resonance was tolerably good; breathing coarse generally; there was some dullness at the left base (epigastric tenderness).

August 3rd. She moaned, and complained of her head. There was slight strabismus. Temperature 102.2; pulse 74; respirations 32.

August 4th. She was moaning, restless, and delirious during the night, getting out of bed. She still complained of the back of her head. She took very little food. Temperature 102.8; pulse 100, slightly irregular. She had slight paralysis of the right eyelid.

August 5th. She was delirious at night, but herself by day, answering questions correctly. She said she felt more pain in the head. The bowels had acted well; the motions were scybalous. Temperature 101.4; pulse 120, very irregular.

August 6th. After a very delirious night, in which she twice got out of bed, she passed into a drowsy state. There was a little twitching of the feet, but no distinct convulsion. There was slight paralysis of the left external rectus, and congestion of the right conjunctiva. Temperature 101.8; pulse 137, very irregular.

August 7th. She passed a rather quieter night, still moaning, and occasionally crying out. The pupils were, as before, dilated. She was, perhaps, a little more conscious, and could be roused to perform the more automatic acts. Temperature 101.6; pulse 120; respirations 30.

August 8th. She was very noisy all night up to 6 A.M.; quieter since. She put her tongue out when asked, but had to be told to put it in. There was much congestion of the right eye, of which the pupil acted less than that of the left. There was slight ptosis of the right eyelid. The bowels were not open; no vomiting. The urine was strongly acid; some was passed in bed; it was of specific gravity 1044; it was not tested for sugar. Respirations 28 to 38; temperature 101.6; pulse 120.

August 9th. She was still noisy, but slightly more conscious. There was more ptosis of the right eyelid. The right eye was quite immobile; both pupils were dilated. The urine was of specific gravity 1051, and was found loaded with sugar. Temperature 101.2 to 101.6; respirations very irregular, 38; pulse 120 to 132, irregular.

August 10th. Drowsiness persisted; but she was, perhaps, more easily roused, although remaining generally moaning, and crying out when disturbed. It was doubtful if she saw with either eye at all.

Urine was passed under her. Temperature 102.2; pulse 136; respirations 40.

August 11th. She started and grasped at imaginary objects; otherwise was drowsy as before. There were some *râles* in the throat. Some urine was obtained; it was of specific gravity 1045, faintly acid, and still loaded with sugar. Temperature 101.8; pulse 164; respirations 48.

August 12th. She was more comatose. Her face was of a cyanotic aspect, reddish purple (*violacé*); the ears and lips livid; yet she roused up and appeared to know her mother's voice. Temperature 101.4; pulse 172. The mucous *râles* increased, and she died at 7 P.M.

Post mortem examination of the head alone was allowed. There was general venous turgescence. Lymph was effused, principally on the convexity of the brain and upon the left side. The vessels of the pia mater in the fissure of Sylvius were especially matted together. The ordinary nodular appearances were seen upon the coats of the vessels. There was considerable thickening of the membranes, with opacity at the base of the brain; with scattered tubercular deposits, especially noticed on the optic tract. Nothing abnormal was observed about the floor of the fourth ventricle.

REMARKS.—The entire pathology of this case remains obscure. There was a strong hereditary predisposition to cerebral meningitis; but the case was a fairly typical one, and, from its history and symptoms, easy of diagnosis. Still, let me call your attention to the fact that here was a well-nourished and forwardly developed girl who was suddenly attacked with meningeal tubercle.

CASE III. *Tubercular Meningitis*.—Henry R., aged 16, a Londoner, who came of a fairly healthy stock, was admitted under my care on July 18th, 1867, on account of pain in his back and nearly complete paralysis of motion of his right leg. He was a lantern-jawed, emaciated, sallow lad, who looked as if he had but recently got up from some long or serious illness. Up to May of that year, he had enjoyed uninterrupted good health; he then sickened with some fever, and was some weeks in bed, and part of the time delirious. There were no facts to establish the exact nature of this illness; but he was so feeble when he left his bed, that he attended the Devonshire Square Hospital as an out-patient; but at the end of a fortnight the loss of power in his right leg increased, so that he could not stand upon the leg, and he applied at St. Bartholomew's for admission.

Had he had typhoid fever? and was his illness that form of local paralysis which does in some rare cases follow this complaint? Or was that whole indefinite attack of fever part history of an acute tuberculosis, which albeit rarely does admit of temporary remissions, and exhibits sometimes fictitious convulsions? The sequel stamps this as the more likely interpretation.

Condition of Patient on Admission.—His skin was cool, rather harsh and dry; no rash. Pulse 92; respirations 24; temperature normal, or below. He had perfect sensation in the right leg, but was unable to move or even lift it. He moved it beneath the bedclothes with both his hands. There was no other evidence of palsy. Other functions were normal. There were some dulness and deficient respiratory sounds over the left thorax. He was ordered half meat-diet, porter, quinine, and tincture of nux vomica.

After a few days in bed, with occasional baths, he seemed better; and on July 22nd he was able to draw up and lift the leg as he lay in bed. But on July 23rd, five days only after his admission, he was not so well. He had been sick, vomiting several times, until nothing but green bile was thus ejected. His skin was hot. Temperature 101.4; pulse 92; respirations 28. The bowels had not been open for two days. The tongue was slightly furred. Still I was told that he slept well. I ordered some castor-oil and fluid nourishment, regarding his attack as biliousness or overfeeding under diminished digestive powers. But the next day's note records a graver condition. The sickness persisted, the vomiting keeping him awake nearly all night. The fluids vomited were bile-stained as before. The castor-oil did not act. Temperature 102.5; pulse 108, feeble, and a little irregular. There was no abdominal fulness or tenderness; and the tongue was moist, and only very slightly furred. Urine was passed in fair quantity, high coloured, of specific gravity 1023, containing no albumen. As to mental condition, there was slow apprehension, but perfect clearness of intellect. He answered questions correctly, and remembered what he had said.

* M. Beau has written a memoir upon acute general paralysis after typhoid fever; and we know of paralysis, general and partial, after diphtheria: still, although I have heard of it, and have often seen such extreme prostration and protracted debility following typhoid fever, as might be mistaken for actual paralysis, I cannot record a single case of what I could fairly call spinal or cerebral paralysis in the convalescence from this disease—a paralysis due to actual nerve-lesion. This palsy, indeed, appears to me merely an exaggeration of the muscular atrophy which follows attacks of fever and blood-poisoning—typhus, much more than typhoid. I women, Dr. Murchison notices (p. 285) incontinence of urine persisting after typhus.

The pupils were regular and equal. I directed brain-symptoms to be watched for; and this because sickness of a persistent kind, with clean tongue and soft and empty abdomen, and loss of power in a limb or one side of a patient, is usually a symptom of disease in the brain, rather than of disorder of the stomach. The treatment was directed to alleviate: ice and milk, a little brandy, citrate of potash and chloroform.

In the progress of this case, I will not weary you with every detailed symptom. I desire to point out the prominent and most diagnostic features as these appeared.

Two days later, the vomiting, high temperature (101.4), and constipation, still persisted. The pulse fell from 108 to 64 somewhat suddenly, but still retained its previously noted irregularity. The tongue obtained a brownish fur, and the lad complained of acute pain over his right eye. For two days this pain lasted, quite unaffected by some opium which he was taking; and the tongue became drier. The urine daily became more scanty and high-coloured. The pulse remained between 60 and 72, rather fuller, but always irregular.

The sickness ceased entirely from 6 P.M. on July 28th. It was reported that he slept well that night; and, having been perfectly clear in his head up to that date, talked in a rambling, slightly delirious manner. On the morning of the 29th, towards midday, there was well-marked converging strabismus of both eyes, but mostly of the left eye.

From this date (July 29) until August 2nd, a few hours before his death, he remained in an unconscious state, lying upon his back low down in bed, picking the bedclothes a little, muttering to himself, occasionally moaning, passing his urine and motions involuntarily under him. He was apparently quite blind, with dilated pupils, rolling the eyeballs slowly from side to side, with what are called oscillatory movements, whenever the eyelids were raised. He did not recognise his mother's voice, or appear to understand anything said to him. Sometimes he would swallow spoonfuls of fluid put into his mouth; but more often he would retain these a long while, and then spit them out.

On August 1st, the temperature remained at 101; the pulse rose up to 138 or upwards, and the respirations to 50 per minute. The *alæ nasi* were dilated; the mouth and teeth were covered with sordes. He was manifestly sinking; but, when I saw him the following day, a very remarkable change had taken place in his mental state—a waking up from delirium such as I have seen once before only, and then too in tubercular meningitis. He spoke to his mother; put out his tongue when I asked him to do so; answered a few questions I put to him, slowly, but correctly; swallowed what nourishment was given him with ease.

This rallying is very difficult to understand, when the actual brain-lesions which the *post mortem* examination discovered are considered. It is a rather painful episode, too, in these illnesses. Those who are earnestly longing for the recovery of the patient raise their hopes at it; and doctors themselves, who know that the more usual course of events is by gradually deepening coma to death, are apt to be deceived by it. It led me to prognosticate death as very close at hand, because I had seen the same thing once before; and the sequel proved the correctness of my forecast, for the lad only survived this return of consciousness a few hours, quickly afterwards becoming comatose again. The condition, however, is one clinically, from its exceptional rarity, well worth your remembering.

The *post mortem* examination discovered all those appearances most characteristic of tubercular meningitis; but there was much less mischief at the base of the brain about the issuing nerves, the pons and optic commissure, than is usual; whereas the membranes within the longitudinal fissure were much matted together, with a layer of firm yellowish lymph interposed, this lymph lying apparently beneath the pia mater, and dipping along with this into the sulci between each convolution for a short distance. The sides of the Sylvian fissure were on both sides very adherent, and microscopic examination of the vessels revealed abundant evidence of tubercle elsewhere. The brain-substance was congested, but there was little or no flattening of the convolutions. The evidence of tuberculosis was abundant, and its universality quite remarkable. There was general tubercular peritonitis: tubercle in the spleen, liver, kidneys, and in lungs, both old cretified nodules and recent discrete patches. The bronchial glands were enlarged and caseous.

The remarks I have interspersed with this case are to my mind sufficient. The axiom I may attach to it is not unimportant: that, when the pathological changes are confined more to the upper surface—the convexity rather than the base of the brain—paralytic symptoms of the limbs are apt to be forthcoming; and the paralysis is wont to be transitory or permanent according to the extent of the lesion. When

the chief mischief is at the base, about the issuing nerves, the organ of vision and its motor apparatus usually give clinical evidence of it. Lastly, the intellectual operations may be impaired or disordered by any circumstances which interfere with the nutrition of the cerebral hemispheres; but the functions of the brain proper are only entirely abrogated by pressure. Such pressure is wontedly signified by drowsiness, stupor, or deep coma.

I will give you two more illustrations of tubercular meningitis beginning very insidiously by symptoms of cerebral paralysis. They are published by Rondou (*Recherches sur les Paralysies*, p. 142), and quoted from Rodier (*Thèse de Paris*, 1871).

A robust young soldier, aged 23, awoke one morning with cramp and sense of formication upon the right side of his body. He next noticed a little weakness or halting in his gait on the same side. Five days later, although still able to get about, his power of locomotion was further impaired; the flexors of his right foot were partly paralysed, and he was unable to straighten it. For nine days longer, up to the fourteenth day of his illness, his foot continued to inconvenience and pain him; then vomiting, headache, and drowsiness supervened, followed by temporary aphasia and progressive paralysis both of motion and sensation. Finally, there was complete right hemiplegia. Death happened on the twenty-fourth day from the first appearance of the symptoms.

NECROPSY.—There was but little mischief at the base of the brain, or in the fissure of Sylvius; although close to the latter, on the left side, there was red softening of some convolutions of the left hemisphere of the brain, in connection with a mass of tubercular granulations upon the surface; while over the convexities of both hemispheres, especially the left, the pia mater exhibited some opacities from puriform exudation, and fine granulations could be detected in the sheaths of the vessels.

Hemiplegia or general paralysis is no unusual event late in the course of tubercular meningitis, arising in the stage of coma, or three or four days before death; but, as an early symptom, it is certainly rare. After a patient has been comatose for a while, slight spasmodic twitches sometimes invade the facial muscles, followed by transitory rigidity or slight spasmodic contractions—clonic spasms, as they are called, of one or other extremity; and then, a little later, the parts may be found limp and powerless, and giving no reflex response to electric or other stimuli. But, when the paralysis is an early symptom, its *début* is usually gradual, and its progress steady, as in the case above narrated, and also in the following illustrative case from Rondou.

Marie Clabru, aged 12, was admitted into hospital on September 18th, with the physical signs of pulmonary tuberculosis well established. Four days later, she became a little drowsy; the drowsiness increased, headache came on, and the pulse was noticeably slowed. On September 23rd, she was less drowsy; still much headache; indistinct vision, with photophobia. The left arm was motionless; the left leg moved with apparent difficulty, sensation on the left side was maintained, but less than upon the right. The same night, the left hemiplegia was complete. Her eyes were fixed, pulse very slow.

September 24th. She had coma and blindness, with dilated pupils; slight occasional contractions of the limbs on the right side, with habitually maintained rigidity. The left side was entirely hemiplegic. At night time, the right arm was still rigid, the right leg was completely flaccid and paralysed. On September 25th, complete paralysis; tracheal *râles*; and death.

POST MORTEM EXAMINATION.—In addition to the ordinary appearances of tubercular meningitis, there was a thickened patch about the size of a *centime* upon the membranes at the posterior inferior portion of the right occipital lobe of the cerebrum, which rather resembled a mass of Pacchionian glands, and there was a similar but smaller granular looking mass over the corpora quadrigemina. Beneath the first of these patches, there was slight bloody extravasation or infiltration into the substance of the brain.

The following case, as an insidious one which led to an important error in diagnosis, is too interesting for me to omit here.

A soldier, who had been dull and out of spirits for some few days, although able to attend his drills, was seized one morning with aphasia quite suddenly. The signs which he made to signify pain in the nape of his neck, and difficulty in swallowing, led his medical attendant to conclude that something had stuck in his œsophagus, and a probang was passed, discovering of course nothing. Eight days later, right hemiplegia became gradually but rapidly established; his pupils were now dilated, and he was drowsy, and his cutaneous sensibility was diminished. The sphincters were relaxed. The respiration became slow. The pulse small and obscure, and he died in deep coma. The necropsy revealed general tuberculosis of the lungs and pericardium,

disseminated tubercle along the vessels of the pia mater, with some small capillary apoplectic effusions on the left side of the brain on the level of the island, or lobulus intermedius, as well as puriform softening of the corresponding left optic tract.

These tiny capillary burstings leading to small apoplectic extravasations, as well as small patches of red softening from stagnation of the current of the circulation in the cerebral centres, are brought about by the tubercle growths pressing on and occluding the current of the blood, and, as one of the pathological events in the course of meningeal tubercle, have hardly received the notice they deserve. These occurrences may, I think, happen both early and late in the course of the disease, and will certainly lend features of great variety to its ordinary clinical symptoms.

I am warned, however, that I have occupied your time already too long. I have many more cases to bring before your notice, but they will illustrate other insidious phases of this disease, its mental peculiarities, and the disorders of the intellectual faculties to which it occasionally gives rise; these I must reserve for another day.

In summary, let me add; that some grade of paralysis of certain sets or groups of muscles, a paralysis of central or brain origin, although not an invariable symptom, is one that usually, sooner or later, supervenes in tubercular meningitis. This paralysis is less well marked in very young children, and better marked as life advances.

It is a paralysis both of motion and sensibility—of motion first, of sensibility later; it is one that rarely invades one side of the body in its entirety at once, but is gradual and progressive, impairing first certain sets of muscles which are ordinarily co-ordinated in action, and which can no longer be moved at will, although the will power is retained. In a large proportion of the cases observed, the upper and lower extremities upon one side of the body are affected, while the muscles of the face upon the same side are not implicated.

The first thing noticed in a good many cases collected by Dr. Seitz and Dr. Rodier, was the inertness of an arm, the slowness and apparent difficulty with which certain movements were executed; but, after a while, the first limb, arm, or leg affected, becomes either rigid, or completely flaccid and powerless. In my own case of Henry Reynolds, the right leg was first lost, and subsequently slightly recovered power. The paralysis is occasionally transitory, when it is so, it has usually succeeded some previous convulsive spasm, tonic or clonic, of the paralysed limb. When the paralysis is transitory, reflex irritability is retained, or even increased; when the paralysis is permanent, reflex irritability is usually lost. The paralysis is very rarely alternate, involving the arm on one side, the leg on the other; it is sometimes cross in this way, the face on one side, the upper extremity upon the other. (*Comptes Rendus*, Case XXVI, p. 129.)

The parts of the face which chiefly exhibit paralytic implication are the eyes, the eyelids, and the corners of the mouth. The muscles paralysed are chiefly those supplied by the oculo-motor nerves and the facial. Dilatation of one pupil, or of both, is often an early event which attracts the attention of an observer intent on discovering some sign of central paralysis; another is the drooping of one or other eyelid; a third is slightly converging strabismus; a fourth, blindness or double vision. Again, the movements of the mouth may be paralysed on one side, from paralysis of the orbicularis oris.

Lastly, as evidence of central nerve-lesion, I must not omit mention of a peculiar stiffness—tonic rigidity, often slightly, sometimes strongly expressed, of the muscles at the back of the neck, which lends a peculiar aspect and posture to the patient, derived from a, so to speak, limited opisthotonos. It was exceedingly well marked in a patient quite recently under my care, who died of tubercular meningitis, and whose case I shall have occasion to detail to you in my next clinical lecture. It is most likely due to some implication of the upper part of the spinal cord or medulla oblongata; but its exact pathology I cannot venture to infer from the evidence at present before me.

ON ABSORPTION BY THE HUMAN DIAPHRAGM.—Dr. Arcadius Rajewsky of St. Petersburg has made experiments with the human diaphragm, by which he obtained the following results. 1. The normal diaphragm absorbs fluids and such substances as there are dissolved in these. 2. If the diaphragm be inflamed, it allows fluids coming into contact with it to pass its system of vessels more easily. 3. When an inflamed diaphragm becomes injected, it will be seen that the intercellular spaces are connected with the lymphatic capillaries, and that they form peculiar canaliculi in the connective tissue. 4. After the removal of the epithelium of the serous membrane, there are opened new avenues for the transportation of fluids, by the intercellular canaliculi commencing on the surface of the membrane.—*Virchow's Archiv*, vol. lxi, July 1875.