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A CASE OF LANDRY'S PARALYSIS.[1]

By JOHN COWAN, M.D., A. J. BALLANTYNE, M.D., and D. MACDONALD, M.B.

Mrs. B., æt. 33, dressmaker, was admitted into Ward II on 24th September, 1907, complaining of weakness of the limbs of two weeks' duration.

On admission she was quite acute mentally, and stated that her illness had commenced a fortnight before. During the last fortnight of August she was on holiday in Fife, and for a week after her return home felt in excellent health, although she had undertaken an extra amount of housework. On 10th September, however, she noticed that her legs were weak, and the calves and her back were painful; and the weakness rapidly increased, so that on the 14th she was unable to rise from her bed. On 17th September her arms began to get weak, and by 21st September she was practically helpless, and unable to lift her head from the pillow, or to move her legs or arms. Her neck was painful on movement, she sweated profusely at night, and she had a slight cough and expectoration. For some days before admission to hospital she had "seen double," and her friends had noticed that she squinted.

Her previous health had been good, save for a rheumatic attack at the age of 19. Though married for six years she had never become pregnant. Her circumstances were comfortable, but she had hard work and long hours from her occupation as a dressmaker.

She was found to be stout and well coloured, though her muscles were flabby. She lay without discomfort flat on her back, but was absolutely helpless and unable to lift her head or limbs. The voice was a little husky and rather weak, but

[1] Read at a meeting of the Glasgow Medico-Chirurgical Society held on 11th December, 1908.

there was no respiratory embarrassment. Deglutition was difficult and slow, but there was no nasal regurgitation. There was complete retention of urine. The tongue was coated, and the fauces congested. She had a slight catarrh in the chest, and some muco-purulent spit. The heart seemed normal. The pulse was regular, moderately full, and easily obliterated, and numbered 96. The other viscera seemed normal.

The muscles generally were of fair size, though flabby, and were only very slightly tender on deep palpation. She was able to move her toes slightly, but otherwise could not move her legs. She could not lift her arms, but was able to move them at all the joints if the influence of gravity was not active. The abdominal muscles were powerless, and the diaphragm inactive. The thoracic movements were full and perhaps exaggerated, save at the left base, where they were apparently somewhat defective. There was evident weakness in the right facial muscles. The tongue could be protruded in the middle line, and the soft palate was elevated voluntarily. The mouth could be shut, but the "bite" was feeble and painless. The right pupil was larger than the left, and movements of the right eye outwards, upwards, and inwards were imperfect, though there was no squint during rest.

Electrical examination showed that the anterior tibial muscles did not react to the faradic current, but the galvanic response was brisk and active, and K.C.C. was greater than A.C.C. The reactions of the opponens pollicis were normal. The extensors of the index and little fingers reacted poorly to the faradic current, but normally to galvanism.

Examination of the reflexes showed a small response on stimulation of the right sole. The sphincter ani was active, and the urine was retained. The left pupil contracted slightly in near vision; otherwise no pupillary reflexes were obtained. Sensation of all kinds seemed normal and acute. There were no trophic or vasomotor phenomena.

For two days after admission the difficulty in deglutition and defective cough gave rise to much anxiety, but on 26th September they were both distinctly improved, and progress afterwards was rapid. On 27th September the power of the thoracic, abdominal, and arm muscles was considerably improved, and the "bite" was painful. On 30th September the diaphragm was active. On 2nd October mastication of solid food was possible, and the urine was voided naturally. On 5th October, eleven days after

admission, the legs were moved on the bed. On 10th October all the reflexes had returned, and power generally was considerable. She was allowed out of bed on 8th November, and was walking fairly well by the middle of the month.

The larynx, for obvious reasons, was not examined until 3rd October, but on that date Dr. Harrington reported that the cords were moving freely.

The case is interesting from several points of view. The symptoms were entirely motor, save for the involvement of the second nerve, and affected more or less the whole of the central nervous system. The lower neuron was involved, but no atrophy of muscles or R.D. occurred. The onset was subacute, the maximum palsy being attained in fourteen days, and recovery was rapid and complete. The sphincters were not involved, and sensation was everywhere normal. These phenomena negative a diagnosis of peripheral neuritis or acute anterior poliomyelitis, and seem to index the case as one of Landry's paralysis.

The onset of the symptoms in this disease is usually more acute than was the case with Mrs. B., and the result is usually less fortunate. Dr. Buzzard considers that these cases are the result of a lymphatic intoxication, and treatment was conducted upon this supposition. Elimination was assisted, while injections of strychnine, as well as diffusible stimulants, were given freely. The most careful examination failed, however, to reveal any evident cause for the intoxication. The possibility of an infective condition was negatived by the help of the Sanitary Office; and the only apparent lesion in the viscera was a by no means severe catarrh of the respiratory passages.

Landry's paralysis seems to be rare in Glasgow, as only one case is reported in the Society's *Transactions*, namely, that recorded by Dr. George Buchanan (1906, vol. vi, p. 82), who reviewed the literature on the subject up to date.

Dr. Buzzard[1] has recently made a further communication upon the subject. He states that he has found in three cases of this disease chromatolytic changes in the cells of the anterior cornua and of Clarke's column, and occasionally excentration of the nucleus, the changes being best marked in the lumbar region, while a little fatty degeneration was present in the nerve fibres of the cord and peripheral nerves. There was no definite evidence of neuroglial or vascular changes. He considers that the pathological lesions point to a spinal intoxication of lymphatic distribution; but the cause of the intoxication is still obscure.

[1] *Brain*, 1907, vol. xxx, p. 1.

NOTE ON THE OCULAR SYMPTOMS.

History.—The patient stated that before the onset of her illness she could read without glasses, and carried on a dress-making business without any visual difficulty. About six days before her admission, that is, a few days after the onset of her illness, she began to see double, and her friends noticed that she squinted. Up till the time of her admission she had not observed any defect in her visual acuity.

Examination.—When the first complete examination of the eyes was made on 28th September (four days after admission), the general condition of the patient had already begun to improve. The conditions found were as follows:—

The right-sided facial paresis involved the orbicularis palpebrarum, causing slight inequality of the palpebral fissures (right fissure larger than left), and practical absence of winking on the right side. Voluntary closure of the lids was possible on both sides, but was not so firm on the right side as on the left. The absence of winking in the right eye had led to lachrymation and irritation. The right pupil was dilated to a slight extent, and did not contract either on illumination or in near vision. The left pupil was also inactive to light, but contracted in near vision.

Movements of the right eye were markedly impaired, the most obvious defect being a limitation of dextrorotation, but deficiency of lævorotation, elevation, and depression were also noted. There was also limitation of lævorotation in the left eye. These facts, considered in conjunction with the fields of diplopia, apparently indicated paresis of the four recti muscles on the right side (especially of the rectus externus), and of the rectus externus on the left side.

The faculty of convergence was unimpaired. Accommodation was active throughout. Visual acuity was somewhat defective in the left eye ($\frac{5}{12}$), and still more so in the right ($\frac{5}{60}$).

The ophthalmoscopic examination showed relative pallor of the right optic disc. There was also slight pigmentary disturbance round the disc and in the neighbourhood of the macula, but these changes had apparently no connection with the visual defect, since they persisted after the latter had gone.

From the date of this first examination there was a steady improvement in the eye symptoms. Three days later up and down movements of the right eye were normal. On the 8th October the only affected muscle was the right external rectus, and this also steadily improved, although it was still slightly defective at the time of her dismissal on 6th December.

When she was seen three months later all trace of the muscular defect had disappeared.

The visual defect improved rapidly. By 8th October vision was $\frac{5}{5}$ in the left eye and $\frac{5}{15}$ in the right, and on 23rd October there was full vision in both eyes.

Her general condition put examination of the field of vision with the perimeter out of the question, and it was not till 10th October that a rough test was made. This showed some constriction of the right field as compared with the left, but no sector or hemiopic defect. Central V.A. was then $\frac{5}{10}$ in the right eye and $\frac{5}{5}$ in the left. There was no central scotoma.

Slight inequality of the palpebral fissures persisted up till the time of her dismissal, but had disappeared when she returned to report herself three months later.

The right pupil gradually recovered its reflex activity in near vision. When she was seen in March, 1908, both pupils contracted well in near vision, but the right pupil was fractionally larger than the left, and both were quite inactive to light.

On returning to work after leaving hospital, the patient experienced some visual difficulty in sewing. The refraction was estimated, and glasses gave the following result:—

R. $+1{\cdot}5$ D. sph. $\bigcirc$ $+0{\cdot}75$ D. cyl. axis $70° = \frac{5}{5}$
L. $+1$ D. sph. $\bigcirc$ $+0{\cdot}5$ D. cyl. axis $90° = \frac{5}{5}$

and these glasses enabled her to read Jaeger No. 1 at her working distance. The patient was seen again quite recently (21st January, 1909), and it was found that the "Argyll Robertson" pupil still persists. The right pupil remains very slightly larger than the left, and has a slightly oval form. The feeling of eyestrain at her work has disappeared.

Remarks.—All writers on this disease agree that involvement of the cranial nerves, and especially of those related to the eye, is rare.

Dr. F. Buzzard (*loc. cit.*) makes no reference to eye symptoms in his cases. Some authors, for example, Allan Starr and H. Charlton Bastian, speak of eye symptoms as occurring in the later phases of the fatal cases. A few text-books mention in general terms the occurrence of such symptoms as inequality of the pupils, loss of the pupil light reflex, paralysis of accommodation, and paralysis of extrinsic muscles of the eyes.

It is probable that ocular symptoms are even more rare in Landry's paralysis than these few references would suggest, for we have to bear in mind the tendency, to which Buzzard alludes, to confuse Landry's paralysis with such conditions as

acute poliomyelitis and acute polyneuritis, in consequence of which symptoms may be credited to Landry's disease which in reality belong to one of the other related affections. Thus, for example, Dr. T. H. Weisenberg (Posey and Spiller *The Eye and the Nervous System*) alludes to three cases, in which there was ptosis, with more or less involvement of the ocular muscles. All three cases were fatal, and the pathological examination revealed a diffuse myelitis in the medulla.

The present case seems to conform to the strict definition of Landry's acute ascending paralysis suggested by F. Buzzard. If so, the presence of such well-marked eye symptoms adds to its interest. Most of them can be consistently attributed to a lesion of the third nerve nucleus. The visual defect is of special interest, since such a symptom has not, so far as one can discover, been hitherto described. Indeed, sensory defects of any kind are very rare in this disease. The nature of the visual defect limits us to a lesion in front of the chiasma, and if we assume that the disease results from an intoxication of ganglion cells, the cause of the amblyopia in this case is probably to be looked for in the retina itself. The persistence of the "Argyll Robertson" pupil phenomenon suggests the possibility of a specific element in the case.