Wallenberg (1895) first described a single case of this interesting but uncommon syndrome, under the title acute bulbar affection, and in Europe it is frequently referred to eponymously as Wallenberg's syndrome. The original patient survived for 6 years, and came to autopsy in 1901, when the original anatomical diagnosis was confirmed. Babinski and Nageotte (1902 a, b) described three cases of an apparently identical syndrome due to syphilis.

The posterior inferior cerebellar artery is a branch of the vertebral artery, and supplies the lateral region of the medulla from about the level of the middle of the seventh nerve nucleus above, to that of the twelfth nerve nucleus below, as well as parts of the cerebellar cortex. Classically, thrombosis of this artery has a dramatic mode of onset; consciousness is undisturbed, but as a result of involvement of the vestibular nucleus, the patient is seized with intense giddiness, and if standing, tends to fall to one side. Because of involvement of the dorsal nucleus and nucleus ambiguus of the vagus, dysphagia and dysarthria are present, and there may be nausea. Involvement of the descending root of the fifth nerve may cause pain on the side of the face, and involvement of the spinothalamic tract causes subjective numbness over the entire half of the body on the opposite side. Diplopia is common as a result of sixth nerve involvement, and there is gross ataxia of the cerebellar type, on the same side of the body as the lesion, through involvement of the cerebellar cortex and the dorsal and ventral spino-cerebellar tracts. Because of their central position neither the mesial fillets nor the pyramidal tracts are involved, and tactile sensibility, postural sense, and voluntary power are all unaffected.

This classical picture is not always seen, however; the damage may be less extensive, and one particular symptom or sign may dominate the scene.

My original interest in this syndrome was a very personal one, dating back to a day, nearly 20 years ago, when my grandfather, then a man in his late seventies, was seized with sudden intense giddiness accompanied by gross ataxia and dysarthria. Looking back, I think my unfamiliarity with the condition must have been painfully obvious, and I hastened to obtain a second opinion.

Professor Henry Cohen, now Lord Cohen of Birkenhead, was called in consultation, and had no difficulty in making a diagnosis from my account of the symptoms, even before seeing the patient. The prognosis was said to be good and my grandfather eventually made a full recovery (for which I was given undeserved credit), and like Wallenberg’s original patient, he lived a further 6 years before eventually succumbing to a further cerebral vascular catastrophe.

The two patients described below were both seen within a few weeks of each other early in 1952.

**Case Reports**

**Case 1, a marine engineer aged 38,** was a naturalized Pole, living in Liverpool. At 6.30 p.m. on September 8, 1951, while on board ship in S. American waters, the patient ate some salmon salad followed 2 hours later by a salmon sandwich. He then went ashore, and about midnight, had a cheese and ham sandwich and some olives. At 3 a.m. the following morning he awoke with stomach pain and vomited. He became dizzy, and had great difficulty getting back to his bunk, but subsequently fell asleep. On being called at 11.45 a.m. to go on watch, he felt very dizzy, had double vision, and was unable to get out of bed. He was given some milk of magnesia, and this was the last thing he was able to swallow for 3 weeks.

The following day he was admitted to hospital at Rio de Janeiro, and during the next 2 or 3 weeks remained very ill; he was quite unable to swallow and was fed intravenously. There was continuous right-sided headache, and the doctors noted right facial weakness, right-sided hemiplegia, weakness of the glottis, general hypotonia, hiccough, and vomiting.
No lumbar puncture was performed and no serological tests were undertaken, but on clinical grounds, the patient was diagnosed as suffering from Botulism, and on September 22 Botulinus Antitoxin, appropriately labelled "A Matter of Life and Death", was flown in from New York and administered to the patient. Should the attendant doctors have had any doubts as to the correctness of their diagnosis, these were no doubt dispelled when they happened to see an article on "Biological Warfare" in the American magazine Life (October 8, 1951), which described the symptomatology of botulism as double vision, difficulty in swallowing and breathing, and thirst and vomiting 12 to 36 hours after ingesting the toxin. Very thoughtfully, they included this extract with the report which accompanied the patient, when he eventually left the hospital.

A month after admission, he was once more able to swallow, and was allowed up. He still felt dizzy, and noted a tendency to fall to the right. He still complained of right-sided headache, and stiffness of his right hand. When he had a hot bath, the water seemed cold to his left leg and arm.

On November 19 he was discharged from hospital at Rio, and was seen 3 weeks later as an out-patient at the Liverpool Royal Infirmary. He still suffered from occasional right-sided headache, with slight difficulty in swallowing, and a tendency to fall to the right. He complained of stiffness in the right hand and forearm, and had difficulty in distinguishing hot from cold down the left side of the body. On a number of occasions he had burnt the fingers of the left hand without noticing it. There was no diplopia, but food and voice sounds went through his nose.

Neurological examination showed a small right pupil and right-sided ptosis. Both pupils reacted to light and accommodation. Eye movements were full, but there was nystagmus on looking to the right. Pain sensation was diminished on the right side of the face, and the palate and pharynx deviated to the left side. In the arms, the reflexes were brisk and equal, but whereas the right arm fell away when held horizontal and showed slight dysdiadochokinesia, and the finger-to-nose test was poorly performed, the left arm showed impaired pain and temperature sensation. Light touch was normal in both arms. Pain and temperature senses were impaired on the left side of the trunk and in the left leg. The gait was unsteady and broad-based, but there was no Rombergism. The knee and ankle jerks were brisk and equal, and the plantar responses flexor.

The correct diagnosis of a right-sided posterior inferior cerebellar artery thrombosis was now made. There had been an acute onset with vertigo and diplopia, and the patient now showed right-sided cerebellar signs, a right Horner’s syndrome, and involvement of the 5th, 9th, and 10th right cranial nerves with left-sided spinocerebellar signs. Chest and skull X rays were normal, and the only abnormal finding in the cerebrospinal fluid was a slightly raised protein content. The blood Wassermann and Meinicke reactions, however, were both strongly positive.

When I first saw this patient in January, 1952, the physical signs were substantially unchanged. There was a history of gonorrhoea in 1933, but no history of syphilis. After a short preliminary course of treatment with bismuth and iodides, he was given a course of 14-4 mega units P.A.M. By mid-March, on completion of this course, he was sufficiently fit to return to sea. He was given a further course of penicillin a year later.

In March, 1954, he left Liverpool for Manchester, where he subsequently attended the Manchester Royal Infirmary. In June, 1956, a letter from Dr. Watt informed me that this patient had been given a further course of injections in 1954, and was attending at regular intervals for surveillance. So far he has remained well and symptomless.

Case 2, an out-of-work newsvendor, aged 47, walked into the Casualty Department of the Liverpool Ear, Nose and Throat Infirmary in January, 1952, with a history of inability to swallow food or drink for 3 days. A foreign body was suspected, and he was admitted to hospital for X ray and oesophagoscopy, but no foreign body or obstruction was found in the oesophagus. The radiologist reported that there appeared to be difficulty in the actual act of deglutition. There was delay in the vallecula, but no obstruction. He added that the appearances would be consistent with a bulbar palsy. The chest X ray showed chronic pulmonary fibrosis and emphysema.

It was noted on admission that the pupils were unequal, and blood was taken for a Wassermann reaction; this was now reported as strongly positive, and a course of penicillin was started on January 30. During the first 5 days in hospital, fluids were given by intravenous drip, but on February 2nd, a Ryle’s tube was passed to the stomach after oesophagoscopy, and through this he was subsequently fed.

On February 5, 9 days after admission and 12 days after the onset of symptoms, the patient was transferred to my care at Newsham General Hospital. His general condition appeared to be satisfactory, and he was able to give a good account of himself. He lived on his own, and it was when attempting to drink a cup of cocoa, which he had made for himself before going to bed, that he found himself unable to swallow. Close questioning elicited no other symptoms whatsoever. The patient was seen by my Registrar 2 hours later, and complained of feeling tired; he was restless and unco-operative and examination resulted in considerable distress. He was clearly very ill. The pulse was weak, the heart sounds were completely obscured by rhonchi, and basal crepitations were also present. Despite lack of co-operation, an attempt was made to examine the central nervous system. The pupils were recorded as being equal and reacting to light, and eye movements as full. No palsy of the soft palate or tongue was noted. No sensory or motor loss was found in arms or legs. Biceps, knee, and ankle jerks were reported as being present and equal. Before his transfer to Newsham, the only neurological abnormalities noted were the unequal pupils, to which reference has already been made, and slight palatal weakness.
The patient's condition rapidly deteriorated, and he died later that day, some 10 hours after admission.

A post mortem examination performed 2 days later, showed that the immediate cause of death was bronchopneumonia. The brain stem was rather congested, but there was no meningitis. The arteries appeared, for the most part, healthy, but there was a thrombosis of the left posterior inferior cerebellar artery. Sections showed both thrombosis and peri-arteritis, and, together with perivascular haemorrhages, there was a small area of softening in the medulla on the left side, some 2–3 mm. from the floor of the fourth ventricle. Specimens of faeces, liver, and spleen, were examined for Clostridium botulinus, but neither the organism nor its toxin could be found.

Summary

Thrombosis of the posterior inferior cerebellar artery is described in two men suffering from syphilis.

REFERENCES

— — — (1901). Ibid., 34, 923.

Thrombose de l'artère postérieure inférieure du cervelet

Résumé

On décrit la thrombose de l'artère postérieure inférieure du cervelet chez deux hommes atteints de syphilis.