VAGUS AND GLOSSOPHARYNGEAL NERVE PARALYSIS: CASE REPORT[1]

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DISTURBANCES of cranial nerves following general anaesthesia are rare and, of all the cranial nerves, the vagus is least frequently involved (5). The following case appears, therefore, to be worth reporting since it concerns extensive bilateral involvement of the ninth and tenth nerves in all their aspects, sensory, motor, and autonomic

CASE REPORT

A 35-year-old well-nourished male entered the neurosurgical service of the hospital with a five-year history of headache Several years before he had a questionable cerebrovascular accident which cleared up completely Angiographic study, carried out uneventfully under general anaesthesia, revealed a "left frontal space-occupying lesion." History and laboratory findings were otherwise non-contributory

Craniotomy was scheduled for the day following angiography. Anaesthetic induction and intubation were uneventful with thiopental and suxamethonium, producing no change in systolic blood pressure (150 mm Hg) and pulse rate (100/min.). Respirations were manually controlled Twenty-five minutes after the start of anaesthesia, the patient was turned on his side for placement of a spinal needle. During this manipulation he coughed lightly twice Anaesthesia was immediately deepened with more thiopental It was noted that the systolic blood pressure rose to 170 while the pulse rate dropped to 70. Installation was completed 50 min. after starting the anaesthesia and preparation of the surgical field was started About 60 cc. of a 0 4 per cent dibucaine solution with 3 drops of adrenalin 1 1,000 to 100 cc. were injected into the scalp. Simultaneously, the patient was connected to a mechanical respirator and was given 100 mg gallamine tri-iodide intravenously Vital signs were again checked as soon as installation was completed and the patient's condition was found to be radically changed The blood pressure rose and soon reached 240 mm Hg and the pulse rate mounted from 65–70 to 180/min in less than 10 min The surgeons were alerted and surgery stopped as the skin flap was raised. Twenty minutes after the onset of the hypertensive episode, his blood pressure started to drop sharply with the pulse rate remaining at 170/min. Vasopressors and whole blood under positive pressure were administered as the systolic pressure dropped below 100 mm Hg and the patient was given 100 per cent oxygen via the respirator His colour remained excellent and his skin warm and dry, but, in spite of all efforts, his pulse disappeared 30 min. after the onset of difficulties Some time was lost convincing ourselves that cardiac arrest had occurred because his colour was still excellent When finally a skin incision was made over the thorax, bleeding was noted from the wound and a few seconds later the pulse returned, making it unnecessary to open the chest The pressure soon stabilized around 80 mm Hg and the pulse rate at 120/min. The chest incision was closed and

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the patient was observed for a time. Since there was a possibility that the brain had herniated, it was finally decided to carry on The brain was first visualized about 1 hr. after the circulatory arrest and showed none of the usual signs of herniation, though ventricular pressure was higher than normal In the course of a 5½-hour operation, a cystic meningoblastoma was removed from the left fronto-parietal region The anaesthetic course was without further incidents The patient was extubated 10 min after the end of surgery. at that time he obeyed spoken commands and answered questions. On the evening of the same day he recognized people whom he had only met just before surgery He was quite hoarse The following day his voice became worse and he was somewhat confused Then, 48 hrs after surgery, it was first noticed that the patient was unable to swallow and aspirated everything he took by mouth Laryngological examination revealed bilateral motor paralysis of the ninth and tenth nerves, complete anaesthesia of the root of the tongue and the larynx, with diminished sensation o- the bronchial mucosa Additional findings were a right Horner's syndrome, "jelly"-nystagmus and left hemianopia, left facial nerve paresis, and persistent tachycardia, between 120 and 140/min Diarrhoea was not noted at any time In view of the fact that bronchial aspiration was easily performed in this patient, he was put on tube feeding and tracheotomy was delayed until almost three weeks after surgery when there appeared no trace of recovery of the laryngeal reflexes His general course was otherwise quite satisfactory and he was discharged from the hospi al four weeks after surgery. At the time he was still unable to swallow, had to be tube-fed, and his pulse remained around 100/min at rest

The patient started to regain control of his pharynx and larynx about two weeks after his discharge At present (12 months after surgery), his tracheotomy is closed, his laryngeal sensations have returned, and he swallows well With a still paralyzed left vocal cord and left pharyngeal musculature, he has made a full functional recovery He has slight rotatory nystagmus and almost complete left hemianopia which have been stationary for the past six months His pulse is now in the high normal range and his exercise tolerance is excellent He has returned to his previous job in a brokerage firm

Comment

The case history suggests a brain-stem lesion involving mostly the nuclei of the ninth and tenth nerves The lesion was initially bilateral and receded in the course of a few months until it now appears permanent and sharply localized

In animal experiments high section of both vagi causes pulmonary oedema and is rapidly fatal The resistance of such animals against adrenalin is sharply reduced (9).

In humans (1, 2, 3, 4) the transected vagus nerves were usually diseased and in only one instance (2, 4) were both vagi cut Tachycardia is a constant feature in these reports, subsiding some time after surgery except where both nerves are cut. Hypertension has not been noted For the sake of completeness, one should also mention that there is good experimental evidence for sensory representation of the larynx in the reticular substance and the cortex (6, 7). Some authors also postulate a higher laryngeal centre in the same sense as the oculomotor system (8, 10).

Our patient evidently suffered a transient and unusually severe haemodynamic upset following a rather banal incident during installation. He had a peculiar involvement of the cranial nerves which lasted for several weeks and he now

apparently has permanent residual lesions which can be localized to the left nucleus ambiguus, the oculomotor system, and the left visual cortex. Chances are overwhelmingly against the possibility that these lesions occurred independently of each other The haemianopia is certainly due to anoxia, which in turn was brought on by the tachycardic episode. What explanation can be offered for the latter?

Surgical trauma cannot be considered more than an aggravating factor because the difficulties started before surgery got under way. Thus, the initial insult during installation was most probably caused by anoxia of a small cluster of cells and hypoxia of a larger surrounding area. This precarious equilibrium was prolonged for several days owing to the inevitable cerebral oedema after brain surgery It caused sufficient anatomical damage to require relearning of reflex pathways and compensation for the paralysed muscles by the remaining active elements. This would account for the unusually long interval between injury and recovery Alternatively, the prolonged recovery may be explained by damage to the supranuclear pathways and centres during surgery added to the anatomical lesion produced during installation.

On this basis the following might have been the sequence of events At the time of positioning, the rostral portion of the left nucleus ambiguus and the left solitary tract nucleus became either compressed or otherwise deprived of their blood supply It is impossible to say what rendered this usually well-protected area vulnerable in our patient. The break in the cardio-regulatory servo-loop, involving these synapses and the left carotid sinus, suddenly abolished the cardio-inhibitor efferent vagal impulses. Simultaneously adrenalin was injected into the thus sensitized (9) patient The resulting tachycardia and hypertension could explain the ensuing circulatory failure through sheer myocardial exhaustion. During the postoperative stage cerebral oedema increased the size of the brain-stem portion where the blood supply was marginal. This lamellar area now involved the contralateral ninth and tenth nuclei as well, while the twelfth nerve nuclei (located between the opposite ninth and tenth nuclei but in more dorsal plane) remained intact. As the cerebral oedema subsided and the blood supply of the area stabilized, a reintegration of laryngeal and pharyngeal functions began, leading in a few months to full compensation for the permanently paralysed elements.

It is appreciated that it is not conclusively proven that vagal or glossopharyngeal paralysis was complete at any time in our patient. Nevertheless the scarcity of reports about the paralysis of these nerves in humans and the peculiarities of the postoperative course in this subject might justify its recording.

Summary

Following essentially normal induction and installation for craniotomy, a 35-year-old male developed paroxysmal hypertension and tachycardia which culminated in cardiac standstill. Prompt initial recovery was followed postoperatively by complete motor and sensory paralysis of the larynx and pharynx as

well as persistent tachycardia. These symptoms continued for six weeks and the eventual full functional recovery took several months. The accident is tentatively explained in terms of paralysis of the ninth and tenth nerves and the pertinent literature is reviewed.

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