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# Transient hypothalamic dysfunction causing episodic cardiac dysrhythmias

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EDITOR:
The hypothalamus plays an important role in regulation of the autonomic nervous system and includes an anterior group of parasympathetic nuclei and posterior group of sympathetic nuclei. Normally, both sympathetic and parasympathetic nuclei are continually active resulting in an optimal basal tone. Manipulation of either group of nuclei could lead to alteration of the basal tone leading to autonomic dysfunction. There are few cases of autonomic dysfunction as a result of intraoperative hypothalamic injury [1,2]. We report an unusual case of postoperative heart rate (HR) variations probably due to intraoperative hypothalamic insult.

## Case report

A 4-yr-old male child, weighing 15 kg, presenting with complaints of headache and diminution of vision for 1 yr, was admitted to our neurosurgical ward. His systemic examination and routine investigations were normal. Magnetic resonance imaging of head revealed a suprasellar lesion of $2.87 \times 3.37 \times 4.5$ cm size associated with mild hydrocephalus suggestive of hypothalamic glioma. For an elective pterional craniotomy and tumour resection, general anaesthesia was induced with fentanyl 30 μg and thiopentone 150 mg. Rocuronium 15 mg was given to facilitate tracheal intubation. Anaesthesia was maintained with isoflurane in a mixture of nitrous oxide and oxygen and intermittent boluses of fentanyl and rocuronium. A subtotal excision of tumour was performed. The child had an uneventful intraoperative course. Intraoperative arterial blood gas analysis was normal. The surgery lasted for about 5 h. At the end of surgery, anaesthesia was discontinued and neuromuscular block reversed with neostigmine and glycopyrrolate. The child became fully awake and responded to verbal commands. However, it was noted on the monitor that the patient had alternate episodes of bradycardia and tachycardia, each episode lasting for about 30 s. It was also noticed that there was dilatation of both pupils. The trachea was not extubated and the child was transferred to the neurosurgical ICU for ventilatory support. Six hours later, the cardiac dysrhythmias subsided and the pupils had returned to normal size. Mechanical ventilation was discontinued and the trachea extubated 12 h after surgery. A cardiac evaluation 12 h postoperatively was normal. The child was discharged 5 days later with a normal neurological function.

## Discussion

Many reports have discussed hypothalamic syndromes related to disruption of the hypothalamic pituitary axis, and various authors have also studied

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the role of the hypothalamus in producing cardiac dysrhythmias [3–5]. We believe that the cardiac manifestations in our patient were a result of transient anterior hypothalamic dysfunction. Two possibilities were considered. Firstly, there may have been some hypothalamic injury at the time of surgery causing oedema formation. Secondly, manipulation of blood vessels during surgical resection, which supply the hypothalamus, may have caused a transient dysfunction of the anterior hypothalamus, thereby altering the delicate basal sympathetic and parasympathetic tones. The first possibility is unlikely, as the symptoms subsided within 6 h. Resolution of oedema may take much longer. The initial event in our patient was bradycardia, which may have been a result of manipulation or spasm of blood vessels supplying the anterior hypothalamus. This, in turn, may have caused tachycardia due to unopposed stimulation of the posterior hypothalamic nuclei.

Normally, the HR is under the control of the hypothalamus, the medullary vasomotor centre and the autonomic system. It is likely that, in the presence of a non-functional parasympathetic centre in the hypothalamus, there was an absence of a background modulating tone to decrease the HR. Therefore, the medial portion of the vasomotor centre and baroreceptors in carotid and aortic sinus sensed the tachycardia. These impulses may have been sent to the dorsal nuclei of the vagus nerve, causing vagal stimulation to decrease the HR. This decrease in HR was again sensed by an intact posterior hypothalamus, causing an increase in HR, and hence an episodic tachycardia and bradycardia. We also observed that this episodic dysrhythmia was not seen when the patient was sedated and paralysed under anaesthesia. As the hypothalamus integrates responses to both internal and external afferent stimuli with the complex analysis of the world provided by the cerebral cortex, these changes were manifested only after the patient was awake. The common cause of pupillary dilatation in surgery for hypothalamic glioma is thought to be the intraoperative manipulation of the oculomotor nerve with its parasympathetic supply to the constrictor pupillae muscles via the ciliary ganglion. In our case, pupillary dilatation may also be attributed to unopposed sympathetic stimulation, due to intact posterior hypothalamus, causing contraction of the meridional fibres (radial muscle) of the iris.

Spirin and Lykoshina [1] reported a high incidence of sinus tachycardia and ventricular extrasystoles postoperatively in patients undergoing surgery for hypothalamic tumours, attributing it to the operative injury to the hypothalamo–diencephalic apparatus. Rehman and Atkin [2] reported cardiac arrhythmia and sleep disturbances postoperatively in a patient operated for a craniopharyngioma. Ishikawa and colleagues [6] reported life-threatening sinus bradycardia persisting for 3 weeks in their patient whose brain scan revealed a low-density area around the hypothalamus. The bradycardia was attributed to hypothalamic insufficiency. Rudelli and Duck [7] reported hypothalamic infarction due to avulsion of the optic chiasma and anterior perforating arteries resulting in cardiac arrhythmias. In our case, we believe that transient nature of the cardiac dysrhythmias were due to the vasospasm of vessels supplying the hypothalamus. The pupillary dilatation can be also attributed to manipulation of the third cranial nerve.

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