# Sleep disturbances and cardiac arrhythmia after treatment of a craniopharyngioma

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The hypothalamus has a fundamental role in regulation of visceral functions and behaviour, so the consequences of injury are many and diverse[1,2].

## CASE HISTORY

A man aged 36 was referred to the neurosurgeons complaining of frontal headaches, listlessness and fatigue, decreased libido, lack of concentration and somnolence, all of three month's duration. Magnetic resonance (MR) imaging showed a 4 cm suprasellar lesion encroaching upon the anterior portion of the third ventricle, with a major extension into the interpeduncular cistern down to the upper surface of the pons. Hormonal profile was normal apart from low plasma testosterone at 6.8 nmol/L (normal 14–28) and plasma cortisol $<30$ nmol/L at 1400 h (50–410). The patient underwent a right frontal craniotomy with excision of a craniopharyngioma, followed by external radiotherapy. Postoperatively he was started on replacement hydrocortisone (20 mg morning, 10 mg evening), thyroxine (150 $\mu$g/day) and a testosterone 600 mg subcutaneous implant four-monthly.

Two months postoperatively he was referred to the department of endocrinology because of confusion and hallucinations. His mother reported that at times he appeared very frightened and disoriented. Rectal temperature was 33 °C, blood pressure 78/36 mmHg, pulse rate 88/min. Repeat MR imaging showed that resection of the craniopharyngioma was complete but the lateral and third ventricles were dilated and cortical markings were prominent. Thyroid function tests, cortisol day curves and testosterone levels were in the normal range. Further dynamic pituitary function tests were thought unwarranted in view of the extensive surgery and radiotherapy. Three months later he was readmitted with progressive drowsiness and was observed to fall asleep in mid-conversation. Rectal temperature was again 33 °C. Because of concern over his hypothermia he was started on ephedrine but his core temperature and somnolence seemed unaffected. After a further two months he had a new symptom, excessive salivation; and in the seven months since the operation he had gained 14 kg.

Six months later (thirteen months postoperatively) he was readmitted after the onset of episodes of apnoea. Rectal temperature was unchanged at 33 °C. He was thrombocytopenic ($37 \times 10^9$/L) and hypernatraemic (plasma sodium 155 mmol/L). An electroencephalogram (EEG) demonstrated 20 cycles of wakefulness and sleep over the 44 minutes of the examination. The average length of wakefulness was 41 seconds (longest period awake 105 seconds) and the average length of sleep was 84 seconds (sleep period 320 seconds). It was observed that the patient went from being alert to suddenly asleep. On three occasions during quiet sleep he had a 10 second period of sinus bradycardia, 20 beats per minute, during which the PR interval became abnormally long and an atrial ectopic escape rhythm developed. During these episodes the patient would become cyanosed and would awaken suddenly, with EEG features of the fully alert state, and his heart rate reverted to normal. No periods of apnoea were noted. A VVI pacemaker was inserted but the patient died a few weeks later at his home. A necropsy was not performed.

## COMMENT

The most striking features in this case are the disturbances of sleep and the episodes of bradycardia. After hypothalamic injury[1,2] psychiatric abnormalities are common, including attacks of rage, laughing and crying, excessive sexuality, antisocial behaviour and hallucinations[3]. The role of the hypothalamus in sleep regulation is still unclear, but somnolence (with posterior lesions) or pathological wakefulness (with anterior lesions) may occur and sleep-like coma has been reported with lesions of the posterior hypothalamus[4]. In this patient the sleep EEG findings were abnormal in that, although the patient appeared to be in stage one of sleep, there were no vertex sharp waves and sometimes when he appeared to be in stage two there were no sleep spindles. In addition, his eye movements and muscle tone suggested that REM sleep was not occurring. To our knowledge this pattern of sleep disturbance has not been reported before.

Hypernatraemia, as observed in this patient, is a recognized complication of hypothalamic damage[5]. It is assumed to arise from disturbances of antidiuretic hormone

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secretion, the thirst centre and osmoreceptors in the hypothalamus. The observed thrombocytopenia may have been related to the patient's chronic hypothermia; in hypothermic animals, platelets become sequestrated in the liver and possibly the spleen[6]. What of the cardiac arrhythmia? A well-documented consequence of hypothalamic injuries, it may have been the cause of this patient's sudden death. The excessive salivation may be relevant. This is an unusual feature of hypothalamic damage and has been reported in a patient with a third ventricle tumour[7]. In rats, lesions of the lateral hypothalamus are followed by excessive salivation[8]. Since salivation is a parasympathetic response, it may reflect high vagal tone, a cause of bradycardia. Excessive salivation and bradycardia in a patient with hypothalamic damage may therefore warrant early insertion of a pacemaker. In this case it is possible that hypothermia contributed to the cardiac arrhythmia.

## REFERENCES


1 Plum FC, Uitert RV. Nonendocrine diseases and disorders of the hypothalamus. In: Reichlin S, Baldessarini RJ, Martin JB, eds. *The Hypothalamus*, Vol 56. New York: Raven, 1978:415–74

2 Bauer HG. Endocrine and other clinical manifestations of hypothalamic disease: A survey of 60 cases with autopsies. *J Clin Endocrinol* 1954;**14**:13–31

3 Morgane PJ, Panksepp J. The handbook of the hypothalamus. *Behavioral Studies of the Hypothalamus*, Vol 3 Parts A and B. New York: Marcel Dekker, 1980, 1981

4 Plum F, Posner JB. *Diagnosis of Stupor and Coma*, 3rd edn. Philadelphia: Davis, 1977

5 d'Avella D, Zuccarello M, Scanarini M, Andrioli GC, Joh I, Trincia G. Neurogenic hypernatraemia: case report. *Acta Neurochim* 1979;**46**:151–7

6 Hessel EA, Schmer G, Dillard DH. Platelet kinetics during deep hypothermia. *J Surg Res* 1980;**28**:23–34

7 Penfold W. *Arch Neurol Psychiatr* 1929;**22**:358–74

8 Schallert T, Leach LR, Braun JJ. Saliva hypersecretion during aphagia following lateral hypothalamic lesions. *Physiol Behav* 1978;**21**:461–3


# Repair of a massive inguinal hernia

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Massive inguinal hernias are uncommon in the western world today. Their repair can be difficult and preoperative induction of pneumoperitoneum has been advocated. However, conventional methods can be successful.

## CASE HISTORY

An otherwise fit man aged 60 sought treatment for a large irreducible right inguinal hernia of over 40 years' duration. He had continually declined surgical correction because of a hospital phobia, and wore oversized trousers and carried a coat over his right arm to hide the bulge. Over the past 5 years it had caused difficulty in walking and problems with micturition due to effective disappearance of the penis into the scrotum. His wife had eventually persuaded him to seek repair. On physical examination he had a massive inguinal hernia reaching the knees (Figure 1); preoperative respiratory function tests (arterial blood gases and spirometry) were normal.

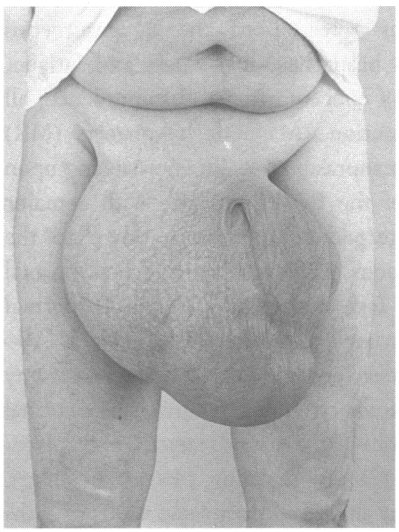

*Figure 1* **Massive inguinal hernia**

At operation, the hernia was found to contain all of the small and large bowel and omentum. The inferior epigastric artery and posterior wall of the inguinal canal were divided to facilitate reduction. A 2-layer double-breasted nylon repair was made and reinforced with polypropylene mesh. The right testis was not atrophic but later had to be removed because of venous infarction. The patient declined secondary cosmetic reduction of the scrotal sac. He recovered uneventfully and was discharged on the tenth postoperative day. Six months later there was no evidence of recurrence or of lymphoedema.

## COMMENT

When massive inguinal hernias are repaired there is a hazard that restoration of viscera to the 'empty' abdominal cavity

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