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Review

Anaesthesia for pituitary surgery

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Abstract

The anaesthetic care of patients undergoing pituitary surgery involves an understanding of the varied presentations of pituitary disease and their implications for the patient's perioperative condition and management. The neuroanaesthetist must also have an appreciation of the issues relevant to the surgical approach (either transsphenoidal or, less commonly, transcranial) and be able to anticipate and manage them accordingly.

Keywords: Anaesthesia; Neurosurgery; Pituitary

1. Introduction

The anaesthetic care of patients undergoing pituitary surgery involves principles common to all intracranial neurosurgical procedures, as well as an understanding of the issues specific to pituitary surgery. Knowledge of tumour anatomy and pathophysiology is required, because pituitary hormonal hyper- or hyposecretion and its consequences, and tumour mass effect, cause problems relevant to anaesthesia.[1] In addition, specific issues arise according to the surgical approach, either transsphenoidal or, less commonly, transcranial.[2] This review aims to present the challenges facing the neuroanaesthetist in these patients.

2. Preoperative assessment

Pituitary lesions may present with hormonal hyper- or hyposecretion, mass effect, or as incidental findings.[3] Thus, a thorough pre-anaesthetic assessment relevant to a neurosurgical patient should be performed, as well as an assessment for features of hormonal hyper- or hyposecretion syndromes, raised intracranial pressure (ICP), and secondary comorbidities. It is imperative that these patients are evaluated pre-operatively by an endocrinologist and undergo a full endocrine workup, visual fields and acuity assessment and magnetic resonance imaging (MRI) scanning.

2.1. Growth hormone excess: Acromegaly

Acromegalic patients are among the most challenging for the anaesthetist. The patient's preoperative condition and the airway can be particularly problematic. Although the treatment of choice is surgery,[1] some patients may be on medical therapy such as dopamine agonists, analogues of somatostatin (e.g. octreotide) or the growth hormone receptor antagonist pegvisomant.[4] Pretreatment with these drugs generally improves the more florid manifestations of acromegaly.

2.1.1. Cardiovascular

Most commonly, systemic hypertension,[5] but also cardiomegaly, accelerated coronary artery disease, aortic and mitral valve disease,[6] cardiomyopathy, conduction defects,[7–9] congestive cardiac failure and arrhythmias may be found.[5,6,9] Patients with chronic obstructive sleep apnoea may also develop right heart failure from pulmonary hypertension.

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2.1.2. Endocrine

Diabetes mellitus may also be associated with acromegaly.[10] A sliding insulin scale may be needed to manage the fluctuating endocrine status postoperatively.

2.1.3. Airway problems

Growth hormone excess produces hypertrophy of the soft tissues of the mouth, nose, tongue, turbinates, soft palate, epiglottis and aryepiglottic folds. Hoarseness should alert the anaesthetist to the possibility of laryngeal stenosis.[2] The concomitant coarsening of facial features with bony proliferation results in prognathism with malocclusion.[11] Furthermore, 25% of acromegalic patients have a coexisting goitre that may cause tracheal compression.[1,2] Therefore, mask ventilation and tracheal intubation may be difficult in acromegalic patients. A careful airway assessment using conventional criteria should be performed.[12] Indirect laryngoscopy, soft tissue X-rays of the neck, and flow volume studies may also be helpful.[10,13]

Obstructive sleep apnoea is an associated feature in acromegalic patients and may be a factor in perioperative airway compromise.[1,2,10] A history of loud snoring, apnoeic periods and daytime somnolence should be sought[1,12] and postoperative care in a monitored environment may be warranted.

2.2. Adrenocorticotropic hormone excess: Cushing's disease

The features of Cushing's disease relevant to the anaesthetist are cardiovascular disease, diabetes mellitus, immunosuppression and coexisting infection, skin fragility with easy bruising (which can make venous cannulation difficult), and osteoporosis (which increases the risk of fractures during patient positioning). Up to 85% of Cushingoid patients are hypertensive; ischaemic heart disease and left ventricular hypertrophy are also common and are the main causes of perioperative mortality in Cushing's patients.[2] Cushingoid features such as skin fragility with bruising[1–3,14] and osteoporosis make handling these patients more complicated. Perioperative haemorrhage is more common and wound healing is poor. Truncal obesity[14] may be associated with difficult airway control. Obesity and gastro-oesophageal reflux may also be present,[1] compromising tracheal intubation. The myopathy common in Cushingoid patients[3,14] may make postoperative ventilation inadequate or difficult. Preoperative treatment with metyrapone greatly reduces the floridity of typical Cushingoid symptoms. In our institution, these patients are nursed in a neurosurgical high-dependency unit postoperatively.

2.3. Prolactinomas

Patients with prolactinomas usually do not have systemic disease relevant to anaesthesia.[12] In women, hyperprolactinaemia causes galactorrhoea and menstrual dysfunction. In men, it causes secondary hypogonadism, reduced libido and erectile dysfunction. Dopamine agonist drugs (bromocriptine or cabergoline) are first-line agents in the treatment of both micro- and macradenomas.[3]

2.4. Hypopituitarism

Hypopituitarism should be suspected when peripheral hormone levels are low without elevation of corresponding pituitary tropic hormone.[3] Rarely, non-functioning tumours may present with panhypopituitarism secondary to pituitary apoplexy. This syndrome can present with sudden headache, loss of vision, loss of consciousness and panhypopituitarism, requiring urgent surgery. Patients with panhypopituitarism are susceptible to water intoxication and hypoglycaemia and are sensitive to central nervous system depressants, such as general anaesthetics.[15] Deficient adrenocorticotropic hormone (ACTH) secretion requires glucocorticoid replacement to reduce pressure on the surrounding neural tissue and prevent a hypoadrenal crisis.[2] Thyroxine replacement is also required (50–150 μg daily). Perioperatively, these patients are extremely sensitive to anaesthetic agents, and pressor agents may be needed to maintain blood pressure.

2.5. Mass effect

Mass effect of an expanding lesion are more likely with non-functioning macroadenomas greater than 1 cm in diameter. Structures adjacent to the pituitary most commonly compressed are the optic nerves and chiasm, resulting in bitemporal hemianopia. Occasionally, other cranial nerve palsies occur. Large tumours can also result in hydrocephalus and raised ICP.[1,3]

2.6. Non-secreting tumours

Pituitary tumours found unexpectedly during investigation for unrelated conditions ('incidentalomas') can be demonstrated in greater than 10% of patients undergoing cranial imaging.[1]

3. Intraoperative issues

The general aims of anaesthesia for pituitary surgery should encompass all aspects of neuroanaesthesia, including optimisation of cerebral oxygenation, maintenance of haemodynamic stability, provision of conditions that facilitate surgical exposure, prevention and management of intraoperative complications, and rapid, smooth emergence. Specific issues for this surgery include:

3.1. Airway management

In acromegalic and Cushing's patients, bag and mask ventilation may not be straightforward, and an oral airway is often required. Southwick and Katz[16] defined four grades of airway involvement in acromegalic patients:

1. No involvement.
2. Nasal or pharyngeal mucosal hypertrophy but normal vocal cords and glottis.
3. Glottic stenosis or vocal cord paresis.
4. A combination of 2 and 3.

They recommended elective tracheostomy for grades 3 and 4, but others have suggested that fibreoptic laryngoscopy with orotracheal intubation is an equally safe alternative.[17] Our approach for a suspected difficult intubation is to perform fibreoptic orotracheal intubation, with the patient either awake or asleep (if mask ventilation is easy).

3.2. Preparation of nasal mucosa

To minimise bleeding from the nasal mucosa, infiltration prior to transsphenoidal surgery is performed using cocaine, lignocaine with adrenaline, another agent, or a combination of these. However, the use of cocaine continues to be associated with the risk of arrhythmias and myocardial infarction, presumably from coronary artery spasm,[2,18] leading many to abandon nasal cocaine paste in anaesthesia. Xylometazoline, a sympathomimetic amine acting at alpha-adrenergic receptors, may be a safer alternative and is as effective as cocaine in producing vasoconstriction.[19] A higher concentration of lignocaine (1%) with adrenaline (1:200 000) has been shown to provide more effective anaesthesia than a lower concentration of lignocaine (0.5%) with adrenaline (1:200 000), and may produce a more stable haemodynamic profile, possibly by counteracting the hypertensive effects of adrenaline.[2] Adrenaline reduces the systemic absorption of local anaesthetics and prolongs the duration of their action. There may be a significant hypertensive response to nasal infiltration, secondary to the unopposed action of adrenaline.[20] This may be further exaggerated in Cushingoid patients. Prompt treatment with labetolol, alpha-antagonists (such as phentolamine), beta-blockers or vasodilators (such as nitroglycerin or sodium nitroprusside) is advocated.[21] Alternatively, deepening anaesthesia or bolusing a short-acting, potent opioid (such as alfentanil or remifentanil) is also effective.[2] Bilateral maxillary nerve blocks have also been described as a useful adjunct to general anaesthesia in suppressing the haemodynamic response to nasal infiltration with adrenaline-containing solutions, nasal dissection, and application of nasal speculum, but are rarely used in our region.[22] Our preferred regime is cophenylcaine (lignocaine/phenylephrine) nasal spray (maximum dose of five sprays per nostril) followed by nasal mucosal injection of 2% lignocaine with adrenaline (1:200 000; maximum dose 7 mg/kg).

3.3. Optimising surgical exposure

3.3.1. Lumbar cerebrospinal fluid catheter

If the tumour has significant suprasellar extension, a lumbar cerebrospinal fluid (CSF) drain may be required. This is usually inserted at the L3–4 interspace, and 10 cm of the catheter should be introduced in a cephalad direction.[12] This allows 5–10-mL boluses of 0.9% sodium chlorine or air, producing prolapse of the suprasellar portion of the tumour into the intrasellar operative field, ensuring a more complete excision of the tumour when using a transsphenoidal approach.[2] The intrathecal injection of fluid, gas or air must be performed under strict sterile conditions. If no lumbar drain is present, a forced Valsalva or Queckenstedt manoeuvre can often suffice.[23] If the arachnoid membrane is breached intraoperatively creating a CSF fistula that requires repair, the drain can be left in situ to promote resolution of the leak postoperatively.[1,2,12]

3.3.2. Hypercapnia

Controlled hypercapnia (to a maximum $PaCO_2$ of 60 mmHg) has been described as an effective and simple method of temporarily raising ICP to displace the suprasellar portion of a tumour down into the sella.[24] Limiting $PaCO_2$ to 60 mmHg minimises the deleterious side-effects of hypertension, tachycardia and increased myocardial work. In our institution, however, we aim for high-normocapnia (40–45 mmHg).

3.4. Perioperative steroid management

In deciding which patients undergoing pituitary surgery require routine glucocorticoid replacement perioperatively, an 0800 hours cortisol and short ACTH 1–24 (synacthen) test should first be performed. If test results are normal (cortisol >550 nmol/L) no perioperative glucocorticoid cover is required. If test results are abnormal, the patient should be given supraphysiological glucocorticoid cover for 48 h (hydrocortisone 50 mg i.v. 8-hourly on day 0, 25 mg i.v. 8-hourly on day 1, 25 mg i.v. at 0800 hours on day 2). All patients with Cushing's disease require glucocorticoid cover.

In the postoperative period, ongoing glucocorticoid replacement should be decided based on 0800 hours plasma cortisol levels, measured on days 1–3 for those patients with normal synacthen test results, and on days 3–5 for those with abnormal synacthen test results. The approach used depends then on the cortisol level:

- <100 nmol/L: maintenance therapy with 15–30 mg/day hydrocortisone; no further definitive tests of hypothalamic–pituitary axis (HPA) required as these patients are ACTH-deficient.
- 100–250 nmol/L: 10–20 mg hydrocortisone in a single morning dose; further testing of HPA is required.
- 250–450 nmol/L: these patients only require glucocorticoid replacement during times of stress and require definitive testing of HPA.
- >450 nmol/L: no glucocorticoid replacement required; these patients are not ACTH-deficient and do not require further testing.[25]

3.5. Anaesthetic technique

Regardless of the method of endotracheal intubation, a reinforced orotracheal tube is recommended, and is positioned in the left corner of the mouth. A throat pack is then inserted, and to ensure that it is removed prior to extubation, a throat pack label should be placed on the patient and the pack should be included in the surgical count sheet. Transsphenoidal surgery is characterised by intense surgical stimulation from nasal injection and dissection to the tumour, whereas a transcranial approach is associated with much less surgical stimulation. However, in both situations, meticulous anaesthetic technique is generally more important than the use of specific drugs.[26] The inhaled agents sevoflurane, desflurane and isoflurane have all been shown to increase lumbar CSF pressure in normocapnic patients undergoing transsphenoidal pituitary surgery;[27,28] although this effect can be desirable, their use is controversial and is avoided in our institution. Whether an inhalational or intravenous technique is employed, short-acting agents should be utilised to facilitate rapid recovery, because postoperative airway maintenance is an issue. In the presence of raised ICP, nitrous oxide is usually avoided, and anaesthesia is maintained with intravenous agents. Hypocapnia should be avoided, because reduction of brain bulk makes access to any suprasellar extension more difficult. Short-acting, potent opioids, such as remifentanil, should be titrated against blood pressure. Remifentanil is effective in controlling autonomic responses in neurosurgical patients[29] and has been shown to provide rapid emergence from anaesthesia while avoiding the risk of postoperative respiratory depression.[30] Postoperative analgesia is provided by small doses of a longer-acting opioid administered towards the end of surgery to ensure that patients do not awaken with pain. The administration of perioperative paracetamol is also recommended due to its highly favourable risk : benefit ratio[31] and opioid-sparing effect. However, paracetamol administered alone is known to be inadequate analgesia for post craniotomy pain.[32] Intravenous paracetamol may be more efficacious, but has yet to be studied. Non-steroidal anti-inflammatory drugs have been linked to postoperative haematoma formation,[33] but no large-scale studies in intracranial neurosurgical procedures have been performed. Their use in neurosurgery remains controversial. Tramadol has been reported to be less effective and to cause more sedation and postoperative nausea and vomiting than codeine.[34] It is not recommended after this type of surgery.

3.6. Monitoring

Routine monitoring, including electrocardiography, pulse oximetry and gas analysis are applied. In addition, invasive arterial blood pressure and temperature monitoring are usually instituted. Central venous pressure monitoring is only indicated if warranted by the patient's clinical condition. If the patient is in a semi-sitting position, a multi-orifice air aspiration catheter can be placed, but this is not routinely done at our institution. Visual evoked potentials (VEP) have been recommended for tumour surgery near the visual pathways, but the high incidence of false-positive and false-negative results and corneal abrasions has meant that few use these for monitoring the optic nerves.[35]

3.7. Positioning for transsphenoidal procedures

The position for a right-handed neurosurgeon is shown (Fig. 1). The patient is supine with the upper torso and head elevated so as to position the operative field above the level of the heart. This optimises venous drainage, minimising bleeding. The patient should also be positioned closer to the right hand side of the table with the neck tilted laterally to the left, slightly extended and secured in a Mayfield clamp. This has the advantage of immobilising the patient's head and facilitating application of the frameless stereotactic reference frame, allowing the neurosurgeon a face-on midline approach. For elderly patients with more rigid necks, the table can be tilted laterally towards the surgeon to obtain the face-on position. The anaesthetic circuit is brought down the left-hand side of the body, allowing access to the left peri-umbilical region for harvesting of a fat graft.

3.8. Operative complications

Venous air embolism has the potential to cause significant morbidity and mortality, but despite the semi-sitting position, it is uncommon during transsphenoidal surgery. Prompt diagnosis and treatment, including aspiration of air from a multi-orifice air aspiration catheter (if in situ), administration of 100% oxygen, application of internal jugular vein pressure bilaterally, saline irrigation of the wound, and haemostasis of open vessels are crucial.[36] Haemorrhage from carotid artery damage is a rare but potentially serious complication. It is initially controlled with packing of the sella, sphenoid sinus and nostrils. If long periods of carotid artery compression are required, cerebral protective drugs should be considered. These patients should not be extubated unless adequate tamponade has been attained. Pseudo-aneurysm and carotid-cavernous fistula formation are complications that can be seen after carotid artery injury, so postoperative angiography should be performed.

3.9. Emergence from anaesthesia

A rapid, smooth emergence from anaesthesia facilitates prompt neurosurgical assessment. Prior to extubation, the pharynx should be suctioned under direct vision, the throat pack removed and the patient's laryngeal reflexes should have returned. It is important to ensure that the nasal packs do not become dislodged during extubation because they may cause post-operative airway obstruction. To

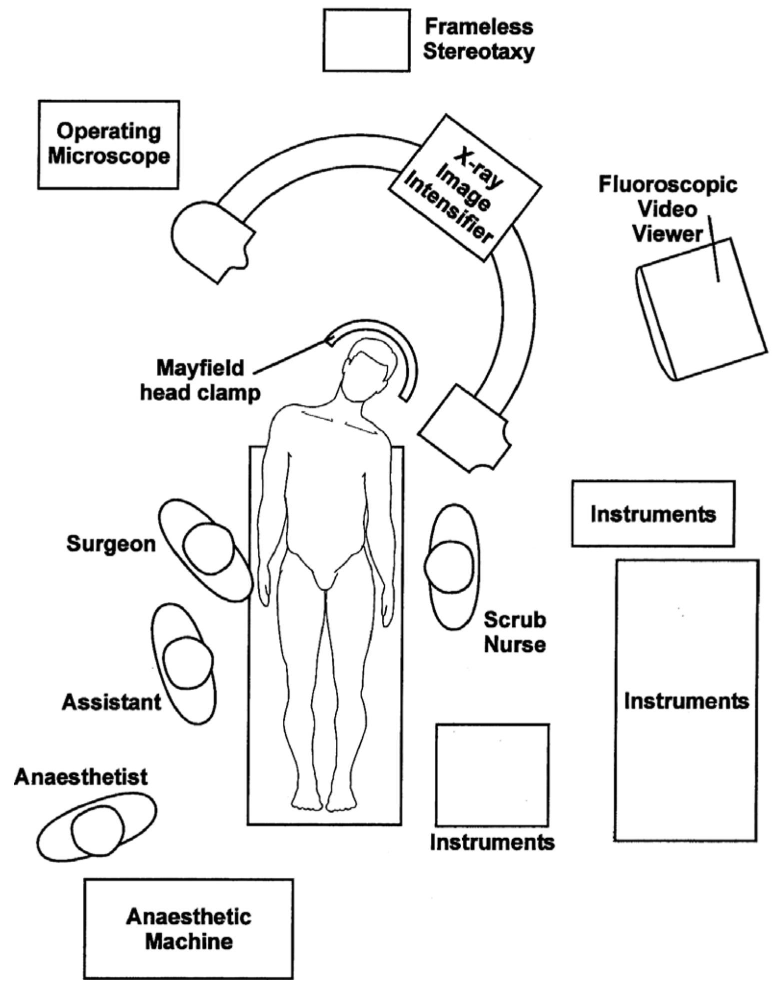

Fig. 1. Operating room arrangement for transsphenoidal pituitary procedures, showing positions of the team members and equipment.

avoid obligatory mouth breathing, we use Merocel nasal packs, which are atraumatic, expanding, soft foam packs that incorporate a ventilating tube to minimise airway obstruction. In these patients, positive pressure ventilation with a mask should be avoided, as there is a risk of tension pneumocephalus, venous air embolism and introduction of bacteria into the subarachnoid space.[37,38]

4. Postoperative issues

Airway compromise is a particular concern in the immediate postoperative period, due to the presence of blood in the naso- and oropharynx, nasal packs, and the predisposition of acromegalic and Cushingoid patients to obstruct. Acromegalic and Cushingoid patients with sleep apnoea are at even greater risk, and should be monitored in a high-dependency unit. Application of nasal continuous positive airway pressure is contraindicated after transsphenoidal surgery. Vital signs, conscious state, and visual fields and acuity should be tested frequently. Any deterioration should be discussed with the neurosurgeon concerned and radiological investigation and/or re-exploration considered. Diabetes insipidus usually develops within the first 24 h; a combination of increased plasma osmolality (>295 mosmol/kg), hypotonic urine (<300 mosmol/kg) and high urine output (>2 mL/kg per h) are the criteria on which the diagnosis can be made.[39] Early diagnosis is important to avoid hypernatraemia and dehydration. Diabetes insipidus is easily treated with parental DDAVP (desmopressin) or intranasal dosing if it is persistent. Hyponatraemia can be caused by excess DDAVP administration or, rarely, syndrome of inappropriate antidiuretic hormone (ADH) secretion. It is managed with fluid restriction (500–1000 mL/day); sodium correction must occur over 24–48 h (<1 mmol/h) to avoid central pontine myelinolysis.

5. Conclusion

Patients undergoing pituitary surgery can present a host of anaesthetic challenges. The neuroanaesthetist must therefore have a good appreciation of the varied presentations of pituitary disease and their implications for the patient's perioperative state. Furthermore, the transsphenoidal approach is associated with specific issues the neuroanaesthetist must anticipate and manage. Good communication and teamwork between the neurosurgeon, anaesthetist, neuroendocrine service, pathologist, radiologist, resident and nursing staff is fundamental to the successful management of patients undergoing pituitary surgery.

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