ANAESTHESIA FOR TRANSSPHENOIDAL SURGERY IN A PATIENT WITH EXTREME GIGANTISM

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True gigantism is a rare endocrine disorder caused by the excessive secretion of growth hormone from an anterior pituitary tumour before pubertal closure of the epiphyses. This results in exaggerated yet proportional bone growth, unlike acromegaly which has the same origin but occurs after puberty and is associated with disproportionate enlargement of the face and acral parts of the hands and feet. Treatment, whether medical or surgical, is aimed at restoring the production of growth hormone to normal so as to curb the progressive organ and soft tissue hypertrophy.

Although the anaesthetic management of acromegalic patients has been reviewed from a number of perspectives [1, 2], there has been no previous description of experience with pituitary giants. This case report illustrates some unique anaesthetic problems relevant to such a patient undergoing transsphenoidal surgery for the removal of a pituitary adenoma.

CASE REPORT

The patient was a 29-yr-old female with an established diagnosis of gigantism. Excessive growth had been noted at age 3 yr, and by the time she was 12 years old, she was more than 6 ft (1.80 m) tall. Treatment was declined until she was aged 22 yr and more than 7 ft (2.10 m) tall in stature when, at another centre, craniotomy to remove a pituitary adenoma was carried out. Seven years later, recurrence of an increase in growth hormone concentration brought her to this hospital for surgical intervention in an attempt to curb the progression of acromegalic disease.

Records available pertaining to the first operation indicated that general anaesthesia was uneventful. Previous surgery also included two knee operations in the remote past, and thyroidectomy 3 yr before the current admission. Although tracheal intubation had been performed without incident in all procedures, difficulty in maintaining optimal ventilation was encountered throughout the thyroid surgery using manually assisted ventilation with a Mapleson D (Bain) system, and episodes of ventricular bigeminy had occurred in association with hypercarbia.

Other relevant history indicated that she smoked 45 cigarettes daily. She had severely limited exercise tolerance attributable to her enormous body mass and osteoarthritis affecting her knees and lumbar spine. She had no symptoms of upper airway obstruction. Current hormone replacement consisted of thyroxine and cortisone acetate, and bromocriptine was being given to suppress the production of growth hormone.

On physical examination the patient was extremely large, measuring 232 cm (7 ft 7.25 in) in height and weighing 186 kg (410 lb). She occupied three hospital beds pushed together in a two-
TRANSSPHENOIDAL HYPOPHYSECTOMY

FIG. 1. The patient. Note the coarse facial features, progonatism and enlarged extremities.

patient room. The classical stigmata of advanced acromegaly were evident (fig. 1).

Respiratory and cardiovascular systems were normal clinically. Examination of the upper airway showed macroglossia, with some hypertrophy of the oral mucosa. Temporo-mandibular and neck mobility were normal, these findings being no different from those observed 3 yr previously. Haematocrit, chest x-ray and arterial blood-gas tensions were normal. Her tidal and minute volumes were 1.3 litre and 12.5 litre, respectively, with a total lung volume of 16.6 litre. Further spirometric studies showed FEV₁/FVC to be 54 % and moderate airflow reduction at low lung volumes. The flow-volume curve showed normal maximum inspiratory and expiratory flow rates with no evidence of extrathoracic upper airway obstruction. Endocrine studies demonstrated an increased serum growth hormone concentration of 30 ng ml⁻¹ (normal < 10 ng ml⁻¹) and a deficient cortisol response to insulin-induced hypoglycaemia. Computerized axial tomogram indicated no extrasellar extension of the pituitary tumour.

Preoperative preparation included chest physiotherapy and salbutamol by inhalation. The only immediate preoperative premedication was hydrocortisone succinate 100 mg i.v. Upon arrival in the operating room the patient was able to transfer herself to a specially reconstructed operating table.

After pre-oxygenation, and precurarization with pancuronium 1.5 mg, anaesthesia was induced with thiopentone given slowly i.v. until loss of eyelid reflex (700 mg) and suxamethonium 200 mg (1 mg kg⁻¹) was administered to facilitate intubation of the trachea. Upon direct laryngoscopy with a No. 4 MacIntosh blade, the pharynx and epiglottis were easily visualized, but the glottic opening was not seen because of its extreme inferior and anterior position. Intubation of the trachea with a No. 8 armoured tube was accomplished by blind per-oral insertion, during which supplementary thiopentone and suxamethonium were given. Anaesthesia was maintained with nitrous oxide and enflurane in oxygen; fentanyl and pancuronium were given i.v. as required. Ventilation was controlled using an Airshields Ventimeter ventilator, delivering a tidal volume of 1.3 litre, 12 b.p.m. (a total minute volume of 16 litre min⁻¹) via a circle system. A total fresh gas flow of 14 litre min⁻¹ of nitrous oxide and oxygen with an F¹O₂ of 0.4, maintained intra-operative PaCO₂ at 4 kPa (30 mm Hg) and PaO₂ at 18.7 kPa (140 mm Hg). Monitoring included end-tidal PEco₂, direct measurements of arterial and central venous pressures, peripheral nerve stimulator and Foley catheter.

Selective transsphenoidal resection of an anterior pituitary adenoma was accomplished in 6 h. After surgery the trachea was extubated in the operating room, and after 48 h of observation in the recovery room, the patient was transferred to the surgical ward where she subsequently made an uneventful recovery.

DISCUSSION

In a patient such as this, a number of difficulties may be anticipated by the anaesthetist. Difficulty in airway management as a result of increased
length of the hypopharynx and encroachment of pharyngeal and laryngeal mucosa in the acromegalic patient has been well recognized since 1896 [3]. Messick, Cucchiara and Faust [4] studied 94 patients, in 39 of whom there was difficulty in intubation: either visualization of the glottic opening was obscured by hypertrophic oral, pharyngeal and laryngeal soft tissue, or there was glottic stenosis or distortion. Airway obstruction from thyroid enlargement may also occur [5]. Southwick and Katz [6] defined four grades of airway involvement:

1. No involvement
2. Nasal or pharyngeal mucosal hypertrophy
3. Glottic stenosis
4. Combination of 2 and 3.

Elective tracheotomy was recommended for grade 3 and 4 patients, but other authors [7] have suggested that fibreoptic laryngoscopy with orotracheal intubation is a safe alternative.

Pre-anaesthetic assessment should note any symptoms and signs of airway involvement such as exertional dyspnoea, hoarseness, stridor, macroglossia or oropharyngeal mucosal hypertrophy. More objective assessment by indirect laryngoscopy, soft tissue x-rays of the neck [8] and inspiratory and expiratory flow-volume studies may be helpful [9]. Our patient had a mild degree of macroglossia, and some hypertrophy of the oral mucosa, with no observable changes in facial features or respiratory function since her last surgery; serious airway problems were, therefore, not anticipated.

Once a secure airway has been established, maintenance of adequate respiratory exchange should not normally pose a special problem in the acromegalic patient. However, in this case of gigantism, enormous respiratory volumes were required in order to maintain $Pa_{CO_2}$ at recommended values [10], and the need for specially adapted ventilatory equipment was considered.

Other problems which may be encountered in the acromegalic patient are hypertension, an idiopathic cardiomyopathy [11, 12] and diabetes mellitus; no evidence of these were found in this patient. Increased intracranial pressure may occur in the presence of extrasellar extension of the tumour, in which case volatile anaesthetic agents should be used with caution [4]. In this case there was no evidence of increased intracranial pressure and enflurane was used without incident. Sleep apnoea is also a recognized complication [13], daytime somnolence and excessive snoring being suggestive of the diagnosis. In such patients there is risk of postoperative hypoxaemia when the residual effects of the general anaesthetic agent may depress central nervous system arousal centres, obtund airway reflexes and delay secretion.
clearing in the larynx. Although our patient exhibited none of these symptoms, it was felt that extra vigilance with regard to postoperative respiratory status would be a wise precaution.

A very real problem in this patient, however, was posed by the mechanics involved both in the safe transportation of such very large subject to and from the operating area, and in positioning her on a surgical table. For this purpose a standard operating table was extensively modified with regards to length, width and supporting structures (fig. 2). Because suitable padding was used, it also served the patient as a recovery room bed during her immediate convalescence (fig. 3).

The major problem encountered in the practical management of this patient was that of difficulty in tracheal intubation after the induction of anaesthesia. This was unexpected in view of two previously-documented uneventful intubations. At the time of her first craniotomy, the concentration of growth hormone was much higher, and at the time of this surgery, no symptoms or signs of upper airway obstruction were identified. Glottic visualization was difficult, not because of mucosal hypertrophy, but rather because the larynx was extremely anterior; possibly the trachea had been drawn forward by post-thyroidectomy scarring. In retrospect (and we would recommend this in other such patients), awake indirect laryngoscopy would ideally have been carried out before operation; the hazard might then have been anticipated and intubation managed by elective fibreoptic laryngoscopy. Blind intubation, although successful in this case, should be discouraged, because mucosal trauma may induce post-anaesthetic airway obstruction.

The potential difficulty of providing adequate ventilation proved unfounded in this case. The patient's normal tidal volume was 1300 ml; the Airshields ventilators used routinely in our hospitals can deliver a maximum volume of only 1400 ml, leaving little reserve for any unexpected increase in ventilatory requirement. In the event of such increased requirement, it was planned to ventilate the patient either manually with a special 5-litre reservoir bag or automatically with an M.A.I. volume respirator having the potential of delivering a maximal tidal volume of 2200 ml. We elected to substitute a circle system with optional in-line carbon dioxide absorber for the Bain circuit usually used in our hospital, since to ensure adequate respiratory exchange with the latter might necessitate fresh gas flows in excess of the capability of our flowmeters [14]. This arrangement provided adequate ventilation for the duration of the procedure without using the carbon
dioxide absorber, and at the same time preserved the facility of using the nitrous oxide and enflurane supplementation.

Our patient was kept under close surveillance in the post-anaesthetic recovery room for 48 h, where her recovery was uneventful. The only complication was a transient period of diabetes insipidus, easily managed by administration of desmopressin.

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REFERENCES