Difficult intubation and brain-stem anaesthesia

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Purpose: To present a case of difficult intubation with brainstem anaesthesia after retrobulbar block with bupivacaine and lidocaine and sedation with midazolam and to point out that close monitoring and timely treatment is important in preventing an unfavourable outcome.

Clinical features: An 82-yr-old man with treated hypertension and stable angina was scheduled for cataract extraction. Physical examination revealed a class 2 airway. He had a retrobulbar block after topical tetracaine drops, with bupivacaine 0.5% and lidocaine 2% with hyaluronidase under sedation with 1 mg midazolam. Five minutes after the block, respiration slowed, he became unresponsive and oxygen saturation decreased to 80%. Immediate ventilation with mask without additional oxygen improved saturation. Attempted tracheal intubation failed: the epiglottis could not be visualized despite flaccid jaw and extremities. A laryngeal mask airway was placed which was leaking and adequate ventilation could not be achieved but a second laryngeal mask airway was placed successfully.

Conclusion: This case emphasizes the need for close monitoring and personnel capable of managing the difficult airway when intra-orbital anaesthesia is used.

Objectif : Présenter un cas d'intubation difficile avec anesthésie accidentelle du tronc cérébral consécutive à un bloc rétrobulbaire à la bupivacaine et à la lidocaïne avec sédation au midazolam en mettant l'accent sur l'importance du monitoring et d'un traitement opportun dans le but de prévenir une complication désastreuse.

Éléments cliniques : Un angineux stable de 82 ans et hypertendu sous traitement était programmé pour une extraction de cataracte. L'examen physique révélait des voies aériennes de classe 2. Après une anesthésie de contact à la tétracaïne, il recevait un bloc rétrobulbaire à la bupivacaine 0,5% et à la lidocaïne 2% associées à de l'hyaluronidase sous sédation avec 1 mg de midazolam. Cinq minutes après le block, la respiration ralentissait, il devenait inconscient et la saturation en oxygène tombait à 80%. La ventilation immédiate au masque sans oxygène permettait d'améliorer la saturation. Une tentative d'intubation échouait, l'épiglotte n'étant pas visualisée malgré la flaccidité de la mâchoire et des extrémités. Un masque laryngé était inséré mais à cause d'un manque d'étanchéité, il était impossible de ventiler. On réussissait finalement à insérer un autre masque laryngé fonctionnel.

Conclusion : Cette observation souligne la nécessité d'un monitoring rigoureux et d'un personnel capable de prendre charge de voies aériennes difficiles quand une anesthésie intraorbitale est administrée.

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BRAIN-STEM anaesthesia is an uncommon but well-recognized complication of intra-orbital regional anaesthesia.¹ We present such a case, where the anaesthetic management was complicated by a coincidental anatomical variation in the patient's airway anatomy rendering tracheal intubation difficult or impossible by conventional laryngoscopic methods. The case is presented to highlight choices in management, and to discuss its implications for safe anaesthetic practice.

Case report

An 82-yr-old man, weighing 62 kg, scheduled for cataract extraction and lens implantation under retrobulbar anaesthesia with monitored anaesthesia care, was seen in the pre-operative anaesthesia assessment clinic. Medical history revealed stable angina pectoris and treated hypertension. Medications included amlodipine, atenolol, and isosorbide dinitrate. Physical examination showed a blood pressure of 160/70 mmHg and heart rate of 78 bpm. Assessment of the airway, including modified Mallampati score,²⁻⁶ mouth opening,⁷ cervical spine mobility,⁸⁻¹¹ and thyromental distance^{5,12} revealed no abnormality. Quantitative assessment was only applied to the Samssoon modification of the Mallampati score (Class II), while the other variables were qualitatively unremarkable.

The patient arrived in the operating room, having taken his regular medications with less than 20 ml water three hours before surgery. Routine monitoring (automatic blood pressure cuff, ECG, pulse oximetry) was instituted. He was moderately anxious. The blood pressure was 160/90 mmHg, heart rate 78 bpm, and oxygen saturation 97%. An intravenous line was established, and two increments of 0.5 mg midazolam *iv* were administered. Ten minutes after arrival, the patient was alert but calm, and haemodynamically stable, with oxygen saturation of 96%. Retrobulbar block was performed by the surgeon using a mixture of 2 ml bupivacaine 0.5% and 2 ml lidocaine 2%, with 0.4 ml Wydase® (hyaluronidase) added. A further 6.6 ml of the same mixture were used for facial nerve block.

Over the next five minutes, the patient became progressively more obtunded, his respiratory rate initially slowed, then he progressed from exclusively diaphragmatic breathing to apnea and unconsciousness. Oxygen saturation decreased to 80%. Effective positive pressure ventilation was established by mask with $F_{I}O_2 = 1.0$, facilitated with an oropharyngeal airway. (SpO_2 increased immediately to 97%.) A laryngeal mask airway but the cuff proved to have a large leak and it was subsequently removed. Tracheal intubation

proved unexpectedly impossible, as only the very tip of the epiglottis could be visualized, despite the complete relaxation of the patient.

Another laryngeal mask was obtained and inserted, the cuff inflated, and ventilation established without difficulty. At this point, examination showed both pupils dilated and unresponsive to light. The blood pressure was 110/60 mmHg, heart rate 60 bpm and SpO_2 97%. Mechanical ventilation was continued for 20 min, until satisfactory spontaneous ventilation was re-established. After a further ten minutes, the patient was awake. With recovery of consciousness, contralateral pupillary reaction was restored. The patient had no lasting sequelae from his presumed brain-stem anaesthesia.

Discussion

This patient presented with progressive loss of consciousness and respiratory depression requiring ventilatory support. This was diagnosed as brain-stem anaesthesia and was complicated by difficult intubation.

The differential diagnosis included drug-induced unconsciousness and apnea. The small dose of midazolam, complete muscle relaxation and the presence of a contralateral dilated pupil (the blocked eye is invariably completely mydriatic) excluded systemic drug effect as the cause. The risk of brain stem anaesthesia with retrobulbar local anaesthesia has been estimated as 16 in 6000 (0.27%) by Nicoll *et al.*¹³ Brain stem anaesthesia is considered to be due to loss of brain stem function due to the presence of local anaesthetic in the cerebro-spinal fluid, hence the term "brain-stem anaesthesia."¹⁴⁻¹⁵

The mechanism by which intra-orbital local anaesthetic arrives in the cerebro-spinal fluid is not clear. Direct injection through the dural sheath surrounding the optic nerve, involving needle penetration of the sheath, has been the generally accepted mechanism.¹ For this reason, it has been suggested that avoidance of Atkinson's position (the eye gazing medially and upward) and the use of peri-bulbar rather than retrobulbar injections should minimize or eliminate the risk of sub-perineural injection of the optic nerve and consequent brain stem anaesthesia.¹⁶

However, Mandelcorn *et al.*,¹⁷ drawing the analogy with spinal epidural anaesthesia, suggested that any intra-orbital local anaesthetic may diffuse through the dural sleeve of the optic nerve and, hence, track centrally into the cerebro-spinal fluid causing brain stem anaesthesia even when the optic nerve has not been directly penetrated by the needle. The importance of this suggestion is that local anaesthetic techniques

such as peribulbar and sub-tenons injection although perhaps less likely to produce brain stem anaesthesia, give no guarantee of its avoidance.

The preoperative assessment of our patient, by two experienced practitioners, one in the pre-operative consultation clinic, the other in the immediate pre-operative period outside the operating room, failed to reveal a difficult intubation. This is not unprecedented, as no clinical test or system of tests has proven completely sensitive or specific for the difficult or impossible laryngoscopic intubation.^{18,19}

Ophthalmic surgery in our hospital is conducted in one of our standard operating rooms. Either monitored anaesthesia care during local anaesthesia, or general anaesthesia, is provided according to the Guidelines to the Practice of Anaesthesia of the Canadian Anaesthetists' Society, (1994). In particular, airway equipment includes laryngoscopes (with a variety of both straight and curved blades), endotracheal tubes, oropharyngeal and naso-pharyngeal airways, laryngeal mask airways, and malleable stylettes in each anaesthetic machine. Flexible fiberoptic bronchoscopes and "light-wands" are kept in a central location adjacent to our operating rooms.

In this case, there was careful pre-operative assessment, the operation was performed in an appropriately-supplied operating room, and the patient was closely monitored. The clinical diagnosis of brain-stem anaesthesia was made and initial treatment (support of ventilation and circulation as required)^{20,21} carried out immediately. Despite these precautions, the patient became hypoxaemic and, because of unanticipated difficult intubation, was exposed to the same risks as any apneic, anaesthetized patient whose trachea cannot be intubated laryngoscopically i.e., gastric distention, regurgitation and aspiration.²²

Ventilation by mask proved to be easy in our patient, and the readily available laryngeal mask airway was selected initially to allow positive pressure ventilation while the decision was made whether tracheal intubation would be necessary for the brief apnea that was expected for brain stem anaesthesia (20 min – several hours). The leaking cuff on the laryngeal mask airway encouraged a traditional method of positive pressure ventilation in the operating room – the endotracheal tube. However, the unsuspected airway anatomy, making laryngoscopic intubation impossible, shifted our judgement back to the relatively easy, rapid laryngeal mask airway. In the event, ventilation was easy and effective, with no evidence of leak, gastric distention or regurgitation on insertion of the second laryngeal mask airway. Since the patient was fasted, was not obese, and had no known gastro-oesophageal

pathology, and ventilatory pressures <20 cm H₂O were required, we considered it safe to continue using the laryngeal mask airway.²³

Our patient was adequately resuscitated and the lungs were ventilated by mask and oropharyngeal airway (or laryngeal mask airway). However, in our opinion, a patient who is iatrogenically apnoeic for "minutes to hours"²⁰ should not have to depend on mask ventilation, even if they have a "difficult airway." As personnel and equipment are required to manage rare complications of anaesthesia such as brain-stem anaesthesia as part of managed anaesthesia care, it should be recognised that these complications may also be associated with another rare complication – unpredicted difficult intubation.

The case highlights the importance of adherence to guidelines for MAC in ophthalmic anaesthesia. It demonstrates that safe anaesthetic practice during MAC requires readiness, not only for the management of apnea, but also for management of apnea in the presence of an unexpectedly difficult or impossible intubation.^{18,19} This may have implications for Monitored Anaesthesia Care in locations outside the hospital operating room (eg., endoscopy suites, emergency departments, office-based operating suites, etc.), where procedures are carried out under heavy sedation or general anaesthesia.²⁴

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