Effect of anesthesia in a patient with pre-existing anisocoria

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Abstract. – In this case report, we describe an accentuation of a pre-existing anisocoria shortly after tracheal intubation in a patient undergoing thyroidectomy.

A 45-yr-old female patient with unequal pupillary diameter (right 2 mm > than left) and decreased light reflex in the right eye – due to a previous eye trauma – was scheduled for thyroidectomy because of multinodular goiter. Anesthesia was induced with propofol 2.5 mg/kg, fentanyl 3 mcg/kg and cisatracurium 0.15 mcg/kg. Immediately after tracheal intubation, examination of the right eye revealed a markedly dilated pupil (8 mm) which was nonreactive to direct and consensual light reflex. The left pupil was 2 mm, and normally reactive to light. An increase in heart rate was also registered (> 20% of baseline) with spontaneous return to baseline within 2 minutes. The right pupil returned to preoperative size within approximately one hour after awakening.

From this case report, it emerges that a pre-existing anisocoria may be exacerbated during anesthesia probably due to incomplete abolition of response to painful stimulus, such as tracheal intubation, provided by anesthetic drugs in the affected eye. The main contributing factor for accentuation of anisocoria could be sympathetic dominance in the pupil with pre-existing mechanical interruption in compensatory parasympathetic mechanisms.

Key Words:
Anaesthesia, Anisocoria, Sympathetic system.

Introduction

Pupillary function is controlled by a balance of sympathetic (dilator) and parasympathetic (constrictor) neural pathways. Pharmacologic or mechanical disruption of these pathways may result in anisocoria. A variety of potential causes for anisocoria exists, ranging from normal to life threatening variation. Finding of anisocoria during anaesthesia is a disturbing and unusual sign and may indicate serious neurological conditions. Previous reports have documented unilateral mydriasis occurring as a result of accidental direct or indirect exposure of papillary muscles to mydriatic (alpha-adrenergic or anticholinergic) agents before or during surgery. Sometimes, ciliary ganglion may be unintentionally anaesthetized by the spread of local anaesthetics. Other possible causes may be mechanical ones, such as eye trauma during intubation or dislocation of an unstable cervical spine resulting in Horner’s syndrome, or asymmetric impaired venous return from head and neck resulting in anisocoria with concurrent exophthalmos. In case of hypertension after intubation, an acute intracranial hemorrhagic event (e.g. undiagnosed intracranial aneurysm) should be suspected.

Evidence supporting effects of anaesthetic drugs has been discussed, but cases of exacerbated pre-existing unilateral mydriasis in patients undergoing general anaesthesia have never been described.

In the present case report, we describe an accentuation of a pre-existing anisocoria shortly after tracheal intubation in a patient undergoing thyroidectomy.
Patient did not receive anaesthetic premedication. At the patient’s arrival in the operating room, electrocardiogram, peripheral oxygen saturation, non-invasive blood pressure, expired carbon dioxide and bispectral index were monitored. Anaesthesia was induced with propofol 2.5 mg/kg and fentanyl 3 mcg/kg. Cisatracurium 0.15 mcg/kg was administered to facilitate tracheal intubation. Immediately after tracheal intubation, examination of the right eye revealed a markedly dilated pupil (8 mm) which was non-reactive to direct and consensual light reflex. The left pupil was 2 mm, and normally reactive to light. Neither eye was oedematous or showed signs of pressure or traumatic injury. We also registered an increase in heart rate (>20% of baseline) with spontaneous return to baseline within 2 minutes. Systemic blood pressure was preserved. We felt that surgery could proceed because of her history of anisocoria.

Desflurane in an air-oxygen mixture was used for maintenance of anaesthesia. No supplementary doses of fentanyl or cisatracurium were required. Ventilation was adjusted to maintain normocapnia (end-Tidal partial pressure of carbon dioxide range, 35-40 mmHg).

At the end of the procedure, which lasted 50 minutes, neuromuscular blockade was reversed with neostigmine (2.5 mg) and atropine (1 mg). After establishing recovery of a spontaneous diaphragmatic breathing with adequate Tidal volume, the tracheal tube was removed. The right pupil returned to preoperative size within approximately one hour after awakening. The 24-hr postoperative course was uneventful.

**Discussion**

In the present case, we find an exacerbation of preexisting anisocoria in a patient undergoing general anaesthesia. In the first instance, we theorized that this phenomenon was due to the parasympathetic dominance caused by sympathicolysis as a result of concomitant use of propofol and fentanyl. However, the parasympathetic-dominant status would induce a rapid constriction in the normal eye, but much slower constriction in the tonic pupil, resulting in marked differences in the two pupillary diameters. In our patient, we did not find a constric-
From literature, it emerges that unilateral mydriasis under anaesthesia may reflect alterations in either sympathetic or parasympathetic autonomic tone, triggered by either a disturbance in cranial nerve function or a pharmacological alteration in autonomic nerve transmission. Anisocoria during anaesthesia has previously resulted from accidental direct or indirect eye exposure to phenylephrine, submucosal infiltration with epinephrine or lidocaine plus epinephrine or as a result of cocaine spread (nasociliary ganglion block). Anisocoria has also been described after airway topical anaesthesia with lidocaine and phenylephrine provided by an oxygen driven face mask nebulizer. Anisocoria may be also caused by impaired ocular blood flow. In a child showing anisocoria during renal transplantation probably due to a retinal hypoperfusion, mydriasis slowly developed on both eyes, but was more pronounced for the left pupil and was associated with concurrent bilateral conjunctival chemosis. Evidence of impaired ocular blood flow was obtained by cranial ultrasound. The pupil size returned to normal with administration of norepinephrine.

Therefore, the assessment of perioperative unilateral mydriasis should always include consideration of accidental exposure of mydriatic agents before or during surgery. As neurologic assessment by physical examination is often difficult under general anaesthesia, in absence of any history of anisocoria or certain causes of anisocoria, the diagnosis of any intracranial pathology to rapidly exclude life-threatening neurologic events requires a cranial computed tomography scan.

In conclusion, from this case report, it emerges that a pre-existing anisocoria may be exacerbated probably because of the uncompleted abolition of response to painful stimulus, such as tracheal intubation, provided by anaesthetic drugs in the affected eye. The main contributing factor could be sympathetic dominance in a pupil with pre-existing mechanical interruption in compensatory parasympathetic mechanisms.

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References