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Miosis with dexmedetomidine: every little helps, every picture tells a story

Editor—Guedel used ocular signs as part of his classic description of the four stages of ether anaesthesia in 1937. These were deemed to be relevant clinical tools when ether, cyclopropane, and chloroform were in use, but with newer drugs and advanced monitoring, the eye signs gradually faded into obscurity.

We report a case of off-label use of dexmedetomidine with bupivacaine for epidural anaesthesia that led to deep sedation and bilateral miosis.

A 62-yr-old female patient, ASA I, was to undergo vaginal hysterectomy. Under strict asepsis, an 18 G epidural catheter was placed in the L3–4 space. A test dose of 3 ml of 2% lidocaine with epinephrine 5 μg ml^{-1} was given after which 12 ml of 0.5% bupivacaine with dexmedetomidine 1 μg kg^{-1} was administered through the epidural catheter. After an initial increase to 190/110 mm Hg, arterial pressure decreased to 90/40 mm Hg and heart rate to 46 beats min^{-1} over the next 5 min. The patient was breathing normally with 99% oxygen saturation but was deeply sedated and was not responding even to painful stimulus (Ramsay score 6). Rapid infusion of i.v. fluids was started and i.v. atropine 0.6 mg was given. Oxygen was delivered by a facemask and a quick re-check was done to exclude any medication error. The pupils were pinpoint in ambient light and a possibility of pontine haemorrhage was also considered. However, the patient started to stabilize gradually and the sedation score improved to 3 after 30 min. The sensory block was adequate and the surgery could be performed as planned. The rest of the perioperative period was uneventful without any neurological sequelae.

While a plethora of information exists about the clinical effects of dexmedetomidine in the anaesthesia journals, little has been said about its effects on the pupils. We examined the current medical literature for its mechanism of action and the effect on reflex pupillary reaction in humans.[1–4]

The regulation of sedation, autonomic function, and pupillary reaction are all inter-related and controlled centrally at the locus coeruleus (LC) due to stimulation of presynaptic α_{2A}-adrenergic receptors by dexmedetomidine. The LC is the largest group of noradrenergic neurones in the central nervous system and gives rise to fibres innervating most structures of the neuraxis. Any pharmacological alteration to this neuronal circuitry would affect the activity at the LC and clinically result in changes in the level of sedation, heart rate, arterial pressure, and pupil size.

There is a biphasic response with dexmedetomidine, with an initial transient sympathomimetic action (peripheral post-synaptic action leading to hypertension) followed by a persistent sympatholytic effect (hypotension and bradycardia). The central sympatholytic action also causes a reduction in pupil diameter due to attenuation of the activity of the coeruleo-spinal pathway and reduction in noradrenergically mediated inhibition of the Edinger–Westphal nucleus. This action is known to predominate in comparison with the peripheral post-synaptic α_2-adrenoceptors activation in humans.[1]

This insight into the mechanism of action unravels the fact that constriction of pupils, sedation, and a decrease in heart rate and arterial pressure are possibly correlated. Given the hypothesis then, that pupillary size and autonomic effects may possibly be a measure of the depth of sedation with dexmedetomidine, perhaps it would be clinically useful in the assessment of the level of sedation in the intensive care unit and a study designed to prove it would be worthwhile. Are we on the threshold of reviving an elementary clinical sign that would make the science of sedation an art?

Declaration of interest

None declared.

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Ventilation with the Ventrain through a small lumen catheter in the failed paediatric airway: two case reports

Editor—We would like to report two cases of ventilation through small lumen intubating and tube exchange catheters to manage critical paediatric airways using the Ventrain, a manually operated, flow-controlled ejector ventilator for emergency use.[1]

A 2.1 kg premature baby was undergoing cryocoagulation therapy of the eyes. Following repeated intubation attempts

on the neonatal intensive care unit, a size 1 laryngeal mask was placed and the baby transferred to the operating theatre (OT). During transport bag ventilation became insufficient. In the OT one additional attempt of direct laryngoscopy was made. With a Cormack–Lehane grade I view, an oedematous glottis was noted. But even a 2.5 mm ID tube could not be advanced into the trachea. Bag mask ventilation was restarted. Then the onset of severe desaturation (Sp_{O_2} below 40%) and bradycardia ($\sim$40 beats min^{-1}) demanded immediate intervention. A 35 cm long Frova intubating catheter [FIC; 8 Fr$=$2.66 mm outer diameter (OD), 5 Fr$=$1.66 mm inner diameter (ID); Cook Medical, Bloomington, IN, USA] was advanced into the trachea, despite some resistance. The Ventrain, connected to the flow regulator of a 2 litre oxygen tank, was attached to the Luer-lock connector of the FIC. Ventilation with active expiration was then started at an oxygen flow of initially 4 increasing to 6 litre min^{-1} with a frequency of $\sim$100 min^{-1} at an *I:E* ratio of about 1:1 while continuously observing chest movements. Within 1 min sufficient oxygenation could be re-established with an Sp_{O_2} above 90% and haemodynamics stabilized. To exchange the FIC for an orotracheal 2.0 mm ID and subsequently a 2.5 mm ID tube, a soft tip guide wire was threaded through the FIC to guide the tracheal tube without any risk of losing the airway. A post-operative X-chest showed no signs of pneumothorax or atelectasis.

In the second case tracheal intubation failed in a 4.3 kg baby undergoing abdominal surgery. After induction of general anaesthesia there was a Cormack–Lehane grade I view using video-laryngoscopy, but two attempts of intubation with a 3.0 and 2.5 mm ID tube, respectively, failed. As bag mask ventilation became difficult, a size 1 laryngeal mask was placed for tracheal intubation using a flexible fibrescope. Although its tip could be successfully placed in the trachea, a tracheal tube could not be advanced. Because of repeated decreases in oxygen saturation, a 45 cm long tube exchange catheter (TEC; 8 Fr$=$2.66 mm OD, 5 Fr$=$1.66 mm ID; Cook Medical) was carefully inserted into the trachea against slight resistance. The Ventrain was connected to the TEC and an oxygen flow of 6 litre min^{-1} was set. Instantaneously ventilation was started at a rate of $\sim$40 min^{-1} at an *I:E* ratio of about 1:2. During ventilation chest movements were continuously monitored and, after observing a considerable chest rise, active expiration was initiated. Sp_{O_2} immediately increased to 96%. A 3.0 mm ID uncuffed tracheal tube was loaded on the TEC and carefully advanced through the glottis under video-laryngoscopic view (Fig. 1). The following day the tracheal tube could be removed without any complications.

Obstruction of the paediatric airway can easily turn into a disaster. Insertion of a small lumen intubating or tube exchange catheter is a promising option. However, conventional positive pressure ventilation through such catheters is hardly possible due to high flow resistance limiting passive backflow even if lung compliance is as low as in neonates. For the same reason jet ventilation can easily lead to gas trapping and bears a high risk of barotrauma and haemodynamic instability or even circulatory collapse.

In the Ventrain, although driven by oxygen coming from a high pressure source, the insufflation pressures are (flow-

Fig 1 Ventilation through a 45 cm long, 1.66 mm ID tube exchange catheter using the Ventrain while advancing a tracheal tube in a difficult airway situation.

dependent) remarkably lower compared to jet ventilation as high pressure is turned into high flow velocity by a jet nozzle.[1] Furthermore, insufflated gas volume can easily be calculated.

Using Bernoulli's principle, the Ventrain provides active expiration by jet-flow generated suction and allows to actively lower increased intrathoracic pressure. Thus, in case of a highly or even completely obstructed airway, the Ventrain is not only capable of re-establishing sufficient oxygenation and efficiently ventilating through a 'straw',[2] but can also help restoring and maintaining adequate circulation.

The use of the Ventrain in combination with small lumen intubating or tube exchange catheters is a new alternative in paediatric airway emergencies. Because of the lack of continuous intrathoracic/intrapulmonary pressure monitoring, it is mandatory to closely observe chest movements and to adjust the *I:E* ratio properly to avoid lung injury.

Declaration of interest

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