Review Article

Oculocardiac Reflex: Know-How?

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ABSTRACT

This review article presents an overview of the current literature concerning the Oculocardiac reflex (OCR). It's a reflex in which bradycardia or arrhythmia occurs due to stretching of the extraocular muscle or increase in the eye ball pressure. It is mostly encountered during squint surgery. Mechanism, cardiac effects and the various predisposing factors leading to OCR are described. The interesting association of OCR with postoperative nausea and vomiting (PONV) is also described. Last but not the least, chapter also presents a detailed guide to preventing and managing this phenomenon.

Key Words: Oculocardiac reflex, bradycardia, strabismus, vagus, extraocular muscles (EOM), autonomic nervous system (ANS)

History

The Oculocardiac reflex (OCR) is a physiological response of the heart to physical stimulation of the eye or the ocular adnexa, characterized by bradycardia or arrythmias, which sometimes leads to cardiac arrest. It was in 1908, both Bernard Aschner and Guiseppe Dagnini, independently reported cardiac slowing after pressure on the eyeball.[1,2]

Aschner demonstrated by animal investigations that the afferent impulse is carried via the ophthalmic division of the trigeminal nerve to the vagal centres, from where the impulses are relayed to heart via the vagus nerve.

The importance of OCR was brought to light when Sorenson and Gilmore in 1956 reported that traction on the medial rectus muscle caused cardiac arrest in a patient.[3] Following this fatal cardiac arrest as a response to the OCR been reported by Walton (1957), Kirsh and others (1957) Mallinson and Coombes (1960), and Smith, Douglas and Petruscak (1972).[4-7] Alexander reported 90% incidence of positive OCR in children under 15 years of age using the criteria of 10% change in the heart rate as a positive OCR.[8] This reflex is especially sensitive in neonates and children, as their vagal tone in higher.[9]

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However, it is also reported in adults. The overall incidence of OCR, both reflex bradycardia and dysrhythmias is higher with general anesthesia compared with regional anesthesia.[10]

Positive OCR is defined as dysrhythmia or bradycardia of 10% or more below relative baseline heart rate resulting from traction of an extraocular muscle.[11] Vrabec et al[12] and Eustis et al[13] defined it as a 10% decrease, while Karhunen et al[14] defined it as a 20% decrease in baseline heart rate. Other reports defined it as at least a decrease of heat rate 10 beats per minute at any time during surgery and persisting for more than 15 seconds as study criteria and OCR as slowing of heart rate by more than 20% or arrhythmia during traction irrespective of heart rate.[15]

Braun and his colleagues divided the haemodynamic response of OCR into two phases. The first phase is cholinergic, causes bradycardia and the second phase of the reflex, adrengeric, is described as counter-regulation (CR). The counter-regulatory process maintains the heart rate during traction at the extraocular muscles after the bradycardic reflex has been initiated.[16]

Mechanism

The trigeminocardiac reflex (TCR), previously known as the OCR or Aschner phenomenon, is a relatively well-known reflex that may arise through manipulation of the trigeminal nerve, via its pathway from the cranial base to its nerve endings on the mouth, jaws, and face.

The reflex is mediated by nerve connections between the trigeminal cranial nerve and the vagus nerve of the parasympathetic nervous system. The afferent tracts are derived mainly from the ophthalmic division of the trigeminal nerve. These afferents synapse with the visceral motor nucleus of the vagus nerve, located in the reticular formation of the brain stem. The efferent tract is carried by the vagus nerve from the cardiovascular center of the medulla to the heart, of which increased stimulation leads to decreased output of the sinoatrial node (figure 1).

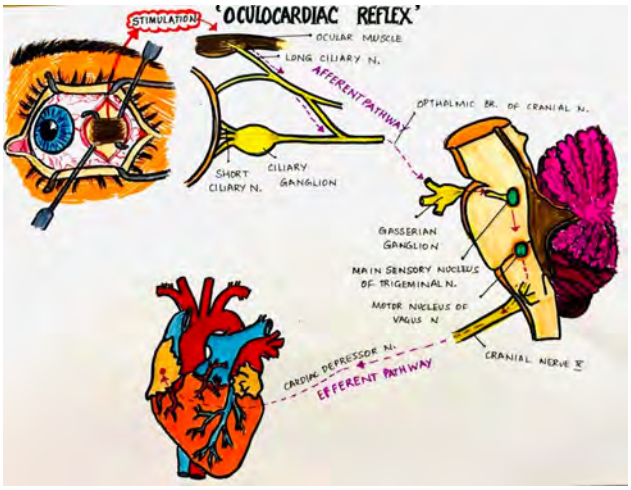

Figure 1. Afferent and efferent pathways of oculocardiac reflex

This reflex sets in due to stretching of the extraocular muscle or increase in the eye ball pressure. In clinical practice, OCR is most often encountered during squint surgery in paediatric age group. It can also occur in repair of detached retina,[10] especially during tagging of extraocular muscles, enucleation of eye,[17] intraorbital injection of local anaesthetics, following digital pressure to the eye, pinching of the conjunctiva with a forceps and post-operative ocular pressure due to bandage.[18]

The reflex is also evoked by contact lens,[19] repair of nasal fracture under general anaesthesia,[20] and by stimulation of eye lids (blepharocardiac reflex),[21] face and oral cavity.

Predisposing factors

The various factors that can increase the risk of OCR reviewed by Scott Lang and van der Val are hypercarbia, hypoxaemia, hypoventilation, acidosis, light anaesthesia, young age due to higher resting vagal tone, pharmacological agents such as potent narcotics (sufentanil, alfentanil), β blockers and calcium channel blockers, and the nature of the provoking stimulus—namely, strength of stimulus, and duration.[22] Though retrobulbar anaesthetic infiltration is a method of prophylaxis to prevent OCR (figure 1), yet still before the block has taken full effect, the reflex may be induced by stimulus of needle disturbance, retrobulbar bleeding, or even the anaesthetic solution (2.5 ml) which probably has effects on intraorbital pressure within a short time. Ocular compression following regional block was found to be the most common triggering event in precipitating the OCR.[23]

Cardiac effects

OCR may manifest as bradycardia, bigeminy, ectopic beats, junctional (nodal) rhythm with disappearance, inversion or shift of the P wave on the ECG, AV block and cardiac arrest.[7,24] Initial baseline heart rate has no influence on the incidence of OCR: tachycardia is therefore not protective.

Ohashi found that occurrence of reflex bradycardia was a graded phenomenon as a function of the tension applied to the extraocular muscles. In addition to the depth of the bradycardia, the period of time to reach the minimum heart rate also became shortened as the tension increased.[25]

Thus, Ohashi suggested that the depth and occurrence of bradycardia in the OCR were closely related to the strength of tension, and that the responses of extraocular muscles to stretch were quantitatively transmitted to the heart and then suppressed the heart rate. He also showed that there were differences of depth and threshold of the reflex as well as the incidence among the medial rectus, the inferior oblique, and the lateral rectus muscle. Stretch on the medial rectus and the inferior oblique evoked the reflex in all the patients tested, whereas, in the lateral rectus, the reflex could be induced only in about 50% of patients. The discrepancy in heart rate sensitivity between surgical extraocular muscle tension and ocular compression might be due to different sensory receptors and brain stem processing for the trigeminal mediated OCR.

Vagal escape

There is a tendency for the patient to adapt to the vagal tone with the traction of the extraocular muscle with the heart rate returning to the pre-traction rate.[26] The OCR normally fatigues with repetitive stimuli and thus lesser decrease in heart rate is observed when the second muscle is stimulated compared to first muscle traction.[27,28]

This was pointed out by Platen (1958).[27]

Association between OCR and Postoperative nausea and vomiting (PONV) in strabismus surgery

PONV is particularly common after strabismus surgery in children. In addition to the distress caused to child and parent, it is also the most frequent cause for unplanned admission after day-case surgery. Although many factors may influence the occurrence of PONV, its particular propensity for occurring after strabismus surgery has led to the theory that an oculo-emetic reflex is responsible.

This has been described as a reflex resetting of the vomiting centre in the medulla following stimulation of the ophthalmic division of the trigeminal nerve during extraocular muscle manipulation.[28] Allen et al found that there was a significant association between a positive intraoperative OCR and PONV. Children with a positive OCR were 2.6 times more likely to vomit than those without the reflex.

It is clearly desirable to reduce the incidence of both the OCR and PONV, and possible methods for prophylaxis include pharmacological inhibition of either the afferent or efferent pathways of the reflex arc. Although prophylactic anti-cholinergic agents such as atropine/glycopyrrolate prevent the bradycardia produced by the OCR there are conflicting reports as to whether they lower the incidence of post-operative emesis.

This may imply that the effect of the OCR on the vomiting center in the medulla may not be reliant purely on cholinergic pathways but on other neurotransmitters.

Local anaesthetic infiltration of the orbit has been shown to inhibit the OCR, by blocking the afferent limb of the reflex. Thus Allen et al, were trying to detect whether sub-tenon's infiltration before muscle traction can reduce the subsequent emesis in the postoperative period.[28]

Prevention of OCR

To avoid any untoward effect of OCR it is recommended to monitor ECG, pulse oximetry, encourage surgeon to be gentle and ensure adequate depth of anaesthesia during surgery.

A quick traction on extraocular muscle provoked a reflex in 87% of instances, whereas progressive traction did so in only 51% of instances. Thus, to reduce the incidence of OCR occurring during strabismus surgery, minimal and gentle manipulation of the extraocular muscles must always be employed by the surgeons.[29]

The regional eye block (peribulbar or retrobulbar) can block the afferent limb of the reflex, while intravenous injection of an anti-muscarinic acetylcholine (ACh) antagonist, such as atropine or glycopyrrolate can block or attenuate the efferent limb of the reflex.

Topical lignocaine applied to eye muscles also significantly attenuates the OCR.[30]

Grover and his colleagues have shown that local anaesthesia produces less bradycardia

and ectopic arrhythmia and is better than general anaesthesia for surgeries in which traction of extraocular muscle is required.[10]

Incidence of OCR was found to be lesser with induction of general anaesthesia with ketamine IV compared with induction using Pentothal sodium / propofol IV.[24] Ketamine by increasing the sympathetic tone, counteract the vagal stimulation during the first phase of the OCR.

Treatment

If bradycardia does occur, removal of the inciting stimulus is immediately indicated, and is essential for successful termination of OCR. The surgeon operating on the eye should be asked to cease their activity and release the applied pressure or traction on the eyeball. This often results in the restoration of normal sinus rhythm of the heart. If not, the use of atropine or glycopyrrolate will likely successfully treat the patient and permit continuation of the surgical procedure. If bradycardia persists or is worrisome, the patient may be given atropine (10 to 20 mcg/kg, IV) or glycopyrrolate (10 mcg/kg, IV). The initial effects of atropine should be evident within 20 seconds, and the maximal response is observed after 80 seconds.[16]

Conclusion

Prevention of OCR requires awareness of the anatomical structures involved, good understanding between the surgeon and anaesthesiologists, and knowledge of the pre-disposing factors known to favor or evoke these reflexes.

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Conflict of Interest:

Nil

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