Trigeminocardiac reflex: Can the neuro-anaesthesiologist afford to be complacent?

Trigeminocardiac reflex (TCR) is a unique autonomic brainstem reflex that manifests as sudden cardiac dysrhythmia (bradycardia), hypotension and gastric hypermotility because of the stimulation of the trigeminal nerve or any of its branches.[1] Other possible presentations include apnoea, asystole without any preceding bradycardia and sometimes, hypertension and tachycardia. It is believed to be a protective reflex with purposeful functions but sometimes becomes too much exaggerated leading to catastrophic consequences. Even though some understanding of this phenomenon dates back well over 100 years, much research work still remains to fully elucidate the utility of this reflex in humans and its diverse and complex physiology. We have come a long way in our understanding of the TCR from the early pioneering works of Kratschmer[2] and Aschner[3] but are yet to identify all the variants with clarity, design tools to accurately identify the reflex and ascertain why inciting factors and clinical manifestations differ vastly from one individual to another.

There is a sizeable literature on oculocardiac reflex during ophthalmic surgeries, which is recognised as a peripheral and physiological subtype of TCR. However, anaesthesiologists practising neurosurgery and ophthalmic surgery encounter this clinical entity more frequently than the anaesthesiologists practising other subspecialities of anaesthesiology. Neurosurgery involving the trigeminal nerve or any of its branches such as microvascular decompression, posterior fossa surgery (cerebellopontine angle tumour) and skull base surgery is a potential trigger for this reflex.[4] Craniofacial surgeries frequently involve manipulation of soft tissues innervated by ophthalmic, maxillary and mandibular divisions of the trigeminal nerve. These procedures may also require osteotomies leading to TCR. The nerve blocks involving scalp and face may lead to a similar response. TCR is commonly encountered during the percutaneous procedures (balloon microcompression, retrogasserian glycerol rhizotomy and radiofrequency thermocoagulation) carried out for the management of pain in patients with trigeminal neuralgia. It has also been observed during neuroradiological interventions involving middle meningeal artery.[5] The incidence of TCR may range from 1% to 2% in craniofacial surgery and 8%–18% during skull base surgery to more than 90% in ophthalmic manipulations without anticholinergic administration.[4] An abrupt sustained manipulation or traction is more responsible for the TCR than gentle and smooth interventions. This phenomenon is probably underdiagnosed owing to the lack of a universal definition of TCR. The most widely cited diagnostic criteria of TCR are given by Schaller *et al*.[6] as a sudden drop in mean arterial pressure of 20% and/or a drop in heart rate by 20%. However, there are inherent problems with this definition. Hypertension can be present in peripheral forms of TCR. Occurrence of dysrhythmias such as ventricular bigeminy, nodal beats and premature ventricular beats and premature atrial contraction[7] without an accompanying haemodynamic (heart rate or blood pressure) change is not addressed in this definition. Moreover, it is not possible to diagnose apnoea and gastric hypermotility when the patient is under general anaesthesia. The resultant bradycardia and hypotension could be the result of complication after administration of an anaesthetic agent, direct vagus nerve stimulation or stimulation of cardiac centre during brainstem manipulation during posterior fossa surgeries without the involvement of the trigeminal nerve. A 'cause' (stimulation of trigeminal nerve) and 'effect' (heart rate and blood pressure changes) relationship may not always be conspicuous during the conduct of major neurosurgeries. This definition appears to be arbitrary and assumptive although better diagnostic criteria are not available at present to supplant it.

The resultant haemodynamic perturbations during TCR, most often, revert back to normal, instantaneously, after the cessation of the causative stimulus. The episodes are usually without any perioperative or neurologic complication. Thus, the neuro-anaesthesiologists may often be complacent when they encounter such a clinical reflex. However, the TCR may not necessarily be benign each time. Sustained reflex bradycardia may require anticholinergic medication (e.g., atropine) and may even require inotropes such as epinephrine if the condition is resistant to anticholinergics.[8] Even though the duration and size of the haemodynamic disturbance may vary from one patient to another, and also on the type and location of stimulus, catastrophic consequences may result from this seemingly benign condition.[6,9-12] Adequate precaution for the commonly accepted triggers of TCR such as hypercarbia, hypoxia, inadequate depth of anaesthesia, pain and acidosis is crucial. More importantly, a good communication between the neurosurgeon informing the neuro-anaesthesiologist of the anatomic proximity to vital structures (including the trigeminal nerve) and prompt reciprocation by the

neuro-anaesthesiologist in case any such response is evoked, cannot be overemphasised.

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