Medicine®

CLINICAL CASE REPORT

OPEN

Severe Bradycardia During Neurosurgical Procedure: Depth of Anesthesia Matters and Leads to a New Surrogate Model of the Trigeminocardiac Reflex

A Case Report

Tumul Chowdhury, MD, DM, FRCPC, Nitin Ahuja, MD, FCARCSI, and Bernhard Schaller, MD, PhD, DSc

Abstract: Hemodynamic alterations are observed in various neurosurgical procedures and commonly related to different neurogenic mechanisms. However, anesthetic influences on causation of these perturbations or management are rarely investigated and therefore our present knowledge is still limited.

In this case of 43-old Caucasian male, propofol boluses aborted the trigeminal cardiac reflex (TCR) induced severe bradycardia during dural manipulation. There is a correlation of severity of bradycardia and slightness of anesthesia.

In the light of the larger distribution of the TCR all over the world, we see more and more aborted TCR, as seen in the present case; then the neuro-anesthesists more and more recognize the TCR at its very onset. A surrogate model for the daily use is present to underline the clinical needs.

We have therefore developed, for the first time, a surrogate model that helps in daily practice to recognize and prevent TCR episodes.

(*Medicine* 94(49):e2118)

Abbreviations: BMI = body mass index, BP = blood pressure, CSI = cerebral state index, HR = heart rate, OSA = obstructive sleep apnea, TCR = trigemino-cardiac reflex.

INTRODUCTION

Hemodynamic alterations are observed in various neurosurgical procedures and commonly related to different neurogenic mechanisms. However, anesthetic influences on causation of these perturbations or management are rarely investigated and therefore our present knowledge is still limited.[1] In the clinical context, it seems that the light plane of anesthesia leads to increased episodes of trigeminal cardiac reflex (TCR), a well-known brainstem reflex in skull base and maxilla-facial surgery.[1] Whether or not the type of anesthetics influences on such occurrences is still a matter of investigation[2,3] and no model exist that reflects the clinical needs. Here, we present an elective neurosurgical case with a phenomenon of sudden and profound bradycardia, its possible explanation, and different management strategies for such an event.

CASE REPORT

A 43-year-old, 101 kg, body mass index (BMI) 31.2, Caucasian male was scheduled for an elective right frontal craniotomy for a meningioma excision. The patient presented with right-sided headache (no signs of raised intracranial pressure), hypothyroidism, and mild obstructive sleep apnea (OSA). All laboratory investigations were unremarkable. Patient's preop vitals were HR 60 beats/min, BP 112/73, and 98% oxygen saturation on room air. Electrocardiogram (ECG) showed a normal sinus rhythm with a baseline heart rate of 50 beats per minute. All the standard monitors were attached and the preinduction invasive arterial line was also inserted. The patient was induced with remifentanil 1 mcg/kg bolus, propofol 200 mg, and rocuronium 60 mg intravenously, and tracheal was intubated with 8 mm cuffed endotracheal tube. Anesthesia was maintained on propofol infusion at 100 to 150 mcg/kg/min. This was supplemented with remifentanil 0.05 to 0.15 mcg/kg/min and desflurane at 3% end tidal concentration in oxygen and air (1:1). During the time of dural separation from tumor, the patient developed 2 episodes of severe bradycardia. Each time the heart rate went down to 35 to 37 beats/min lasting around 36 s; however, the blood pressure was maintained at the baseline. Surgeon was notified; however, there was no other option left except to continue separating dura from the tumor. We therefore decided to increase the depth of anesthesia. Each episode of bradycardia was terminated with a bolus of 50 mg of propofol intravenously. At the end of the procedure the muscle relaxant was reversed with neostigmine 2.5 mg and 0.4 mg glycopyrrolate. The patient was successfully extubated and transferred to postanesthesia care unit for further observation. The further clinical follow-up was uneventful.

DISCUSSION

This case highlights the unique neurogenic mechanism that is known as TCR and that manifests as severe bradycardia (and/or arterial hypotension) during stimulation of fifth nerve innervated dural covering.[4,5] There is a correlation of severity of bradycardia and slightness of anesthesia.[5] In the light of the larger distribution of the TCR all over the world, we see more

Editor: Kazuo Hanaoka.
Received: August 23, 2015; revised: October 13, 2015; accepted: October 26, 2015.
From the Department of Anesthesiology and Perioperative Medicine, University of Manitoba, Winnipeg, Manitoba (TC, NA); and Department of Research, University of Southampton, Southampton, UK (BS).
Correspondence: Tumul Chowdhury, Department of Anesthesiology and Perioperative Medicine, University of Manitoba, Winnipeg, Manitoba (e-mail: tumulthunder@gmail.com).
Written informed consent has been taken from the patient.
Both Tumul Chowdhury and Bernhard Schaller are academic editors for this journal.
The authors have no funding and conflicts of interest to disclose.

ISSN: 0025-7974
DOI: 10.1097/MD.0000000000002118

and more aborted TCR, as seen in the present case, then the neuroanesthesists more and more recognize the TCR at its very beginning. This complexity has substantial challenges on the definition of the TCR.[5,6]

As these TCR episodes were aborted by boluses of propofol, a surrogate concept can be derived regarding the depth of anesthesia related to the TCR occurrence. As during this case, we were not using any depth of anesthesia monitor, what is a clinical reality, so we can only associate plausibility by the sequence of events, by repetitive and reversible related causative mechanisms and by the prevention of the whole TCR occurrence by propofol.[1] Additionally, the current case confirms, for the first time for the central TCR, what is already know for the TCR in the meta-area of the Ganglion Gasseri,[7,8] that arrhythmias seems to be the more susceptible symptoms of the TCR occurrence. The arrhythmia is therefore a first and important surrogate model for the TCR on the one side and for the depth of anesthesia on the other side. The development of such a surrogate model was necessary as the TCR has reached the level that nowadays often the TCR is partly or fully abolished what reflects not at least in decreasing prevalence in different meta-areas.[7]

Whether or not, increasing depth with volatile agents would result similar abolition of reflex as seen in this case with propofol is matter of further investigation. However, in neurosurgical patients, increasing the depth of anesthesia by volatile agents does not seem to be a reasonable choice. Should the use of anticholinergics be the primary mode of management in such case, is debatable. It not only reduces the chances of detection of neurogenic reflexes, but also may cause significant hemodynamic alterations and is finally best known—because of pharmacological mechanism of action—to be unable to fully abolish the TCR.[1] In special cases, the preventive topical application of lidocaine could be a solution.[9,10]

CONCLUSION

We present here a TCR case that necessitates us to overthink the theoretical cerebral state index (CSI) model in TCR and the clinical definition of the TCR.[1] We have therefore developed, for the first the time, a surrogate model that helps in daily practice to recognize and prevent TCR episodes.

REFERENCES

1. Meuwly C, Chowdhury T, Sandu N, et al. Anesthetic influence on occurrence and treatment of the trigemino-cardiac reflex: A systematic literature review. *Medicine.* 2015;94:e807.
2. Jin YH, Zhang Z, Mendelowitz D, et al. Presynaptic actions of propofol enhance inhibitory synaptic transmission in isolated solitary tract nucleus neurons. *Brain Res.* 2009;1286:75–83.
3. Wang X, Gorini C, Sharp D, et al. Anaesthetics differentially modulate the trigeminocardiac reflex excitatory synaptic pathway in the brainstem. *J Physiol.* 2011;589:5431–5442.
4. Chowdhury T, Mendelowitz D, Golanov E, et al. Trigeminocardiac reflex: the current clinical and physiological knowledge. *J Neurosurg Anesthesiol.* 2015;27:136–147.
5. Meuwly C, Golanov E, Chowdhury T, et al. Trigeminal cardiac reflex: new thinking model about the definition based on a literature review. *Medicine.* 2015;94:e484.
6. Chowdhury T, Sandu N, Meuwly C, et al. Trigeminocardiac reflex: different behaviour and risk factors in the course of the trigeminal nerve. *Future Neurol.* 2014;9:41–47.
7. Meuwly C, Chowdhury T, Sandu N, Schaller BJ. Meta-areas of the trigeminocardiac reflex within the skull base: A neuroanatomical thinking model. J Neurosurg Anesthesiol 2015 in press. [publication ahead]
8. Chen CY, Luo CF, Hsu YC, et al. Comparison of the effects of atropine and labetalol on trigeminocardiac reflex-induced hemodynamic alterations during percutaneous microballoon compression of the trigeminal ganglion. *Acta Anesthesiol Taiwan.* 2012;50:153–158.
9. Meuwly C, Chowdhury T, Schaller B. Topical lidocaine to suppress trigemino-cardiac reflex. *Br J Anaesth.* 2013;111:302.
10. Chowdhury T, Nöthen C, Filis A, et al. Functional outcome changes in surgery for pituitary adenomas after intra-operative occurrence of the trigeminocardiac reflex: first description in a retrospective observational study. *Medicine 20151.* 2015;94:e1463.