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Letter to the Editor

Unforeseen clinical outcome for laryngeal adductor reflex loss during intraaxial brainstem surgery

We read a Letter to the Editor by Satomaa et al. with great interest (Satomaa et al., 2019). The authors present an excellent account of surgery of an intraaxial brain stem tumor monitored with the laryngeal adductor reflex (LAR) and other cranial nerves recordings. Their LAR recordings suggest that this new methodology can be successfully applied in this group of patients with intraaxial brain stem tumors.

We were initially conflicted by their suggestion that loss of LAR had no impact on the patient's functional voice and swallowing outcomes given that prior studies (Kaneoka et al., 2018; Sinclair et al., 2018; Borders et al., 2019) have found the opposite. After consideration, it seems that the findings from their study open up two possible scenarios for the results observed:

In the first scenario, a stimulation fault may have been present on the right side from the start of the case as suggested by the left contralateral R1 being of very low amplitude. In the methods section, the authors mention that the LAR was elicited by mucosal stimulation using endotracheal tube electrodes, with *contralateral* R1 responses being recorded by hookwire electrodes. There is no mention of baseline ipsilateral R1 responses having been taken. It would be useful in this regard to know whether pre-incision ipsilateral right sided LAR traces (i.e., in response to right-sided mucosal stimulation) were performed and what the results have been. Alternatively, but less likely, there may have been a recording issue on the right side due to electrodes having been initially placed asymmetrically into each muscle (suspected by the asymmetry in LAR amplitudes at opening), with the right-sided electrode dislodged when the right LAR was lost.

In the second and more exciting scenario, as alluded to in their article, LAR interneuron damage inside the brainstem could have occurred to cause the right-sided contralateral R1 loss. Selective and unilateral interneuron damage would not be expected to significantly affect voluntary speech or swallowing due to bilateral nucleus ambiguous innervation for these functions as shown when unilateral stimulation of the motor cortical larynx area yields bilateral vocal fold movement. Also, automatic functions such as swallowing, coughing, and aspiration have been demonstrated to occur through separate pathways than voluntary functions such as speech (Iwatsubo et al., 1990; Jürgens, 2009). Therefore, postoperative inability to elicit a laryngeal closure reflex to supraglottic stimulation, rather than speech, would be the main deficit noted if LAR interneuron damage had occurred and would be impossible to diagnose clinically, needing specific stimulation of the supraglottic mucosa to display the deficit.

Finally, we wish to emphasize another issue with this manuscript that relates to outcome measures. Subjective voice evaluation as a predictor of vocal fold paralysis has been demonstrated in many studies to have low sensitivity between 30 and 60%. As such, when reporting laryngeal outcomes, a formal endoscopic laryngeal examination is required. The authors did not explain how voice and swallowing were assessed in this case report, either pre- or postoperatively. To state that the patient's laryngeal function was normal postoperatively, the patient should have had an examination involving direct laryngeal visualization during vocal and swallowing tasks. It would be appropriate for the authors to elaborate on how voice and swallowing functions were assessed. However, as noted above, if LAR interneuron damage were the cause of the observed EMG changes, voluntary speech would have been normal and deficits would have been noted only by the inability to elicit a bilateral LAR response to supraglottic mucosal stimulation.

Overall, damage to vagal LAR nuclei or the LAR brainstem interneuron network undoubtedly has different and more complex effects on the LAR compared with direct peripheral cranial nerve injury. In peripheral neck or extraaxial brainstem surgeries, LAR loss occurs due to damage to the peripheral motor 'final' pathway (vagus nerve or its recurrent branch) and renders the intrinsic laryngeal muscles entirely paralytic. This injury results in immediate postoperative findings and symptoms, including vocal fold paralysis ± dysphagia and aspiration. However, as demonstrated in the current case report, assuming no technical issues with LAR stimulation or recording, complete unilateral LAR loss to supraglottic stimulation with retention of mapping response during intra-axial brainstem surgery may occur without the patient exhibiting significant deficits in voluntary speech and swallowing functions.

Declaration of Competing Interest

None of the authors have potential conflicts of interest to be disclosed.

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