



Chewing-induced asystole: a manifestation of the trigeminal cardiac reflex in stroke recovery

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Dear Editors,

The pathway to recovery from an acute ischemic infarct is unique for each patient. We present a 66-year-old woman with a history of hypertension and chronic obstructive pulmonary disease who suffered an acute right middle cerebral artery ischemic stroke confirmed by brain MRI. Recovery was complicated by brief asystolic events while eating. At presentation, she had a National Institutes of Health Stroke Scale (NIHSS) of 21, representative of left facial droop, left upper and lower extremity hemiparesis and ataxia, severe dysarthria and a reduced level of consciousness. Vital signs showed elevated blood pressure. The workup revealed 50–60% luminal narrowing of the proximal right internal carotid artery.

On the fourth hospital day, a swallow evaluation ruled out dysphagia, and she was provided oral nutrition. While chewing (under direct observation), cardiac telemetry showed bradycardia which progressed to asymptomatic asystole of 4-s duration with notable p-waves. She continued to experience a heart rate (HR) in the low 40s each time she ate. Initial evaluation by cardiology was unrevealing with a normal electrocardiogram. She continued to have bradycardic events while chewing, the majority of events progressing to asymptomatic asystole (Fig. 1). Ultimately, again while chewing, she experienced a 12-s asystole which resulted in loss of consciousness. Based on the stimulus-driven etiology of the patient's asystolic events, she was diagnosed with a pathological trigeminal cardiac reflex (TCR). A pacemaker

was placed for paroxysmal symptomatic asystole and she was discharged to an inpatient rehabilitation facility without further episodes. Of note, the patient had no bradycardia or asystole except while chewing. While glossopharyngeal neuralgia could also be considered in the differential diagnosis of a patient with bradycardia while chewing, our patient had no known history of paroxysmal glossopharyngeal pain, nor an identified lesion that would suggest glossopharyngeal nerve compression [1].

TCR is classically defined as a hemodynamic change such as drop in HR below 60 beats per min or mean arterial blood pressure (MAP) of more than 20% from baseline in response to a physical or chemical stimulus of the trigeminal nerve [2]. More recent literature defines the reflex in respect to the HR changes rather than MAP due to the multifactorial nature of MAP [2]. The literature available on TCR is still evolving as the reflex is further studied. A suggested diagnostic approach includes meeting two major criteria and two optional minor criteria. The two major criteria are, first, that the drop in HR is first biologically plausible and, second, reversible. The two minor criteria are that the events are (1) repeatable and (2) preventable [3]. In the case of this patient, the bradycardia and asystole occurred during episodes involving mastication, which relies on muscles controlled by trigeminal nerve activity, thus giving this case biological plausibility. Once mastication was complete, her HR would return to normal without any intervention (evidence for reversibility). Throughout her hospital stay she was fed multiple times with recorded bradycardic and asystolic events (evidence for repetitiveness). These events could be prevented by limiting oral intake. The progressively lengthening duration of each asystolic event eventually resulting in loss of consciousness led to the assumption that this reflex was not active in this patient prior to the stroke. It is also plausible that the TCR response is proportional to the trigeminal stimulation.

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Fig. 1 Asystole recorded during mastication

The TCR has been described during neurosurgical and maxillofacial surgical procedures and to our knowledge has never been reported in a post-stroke patient [2]. Recently, the TCR has been reported as a possible cause of sudden infant death syndrome (SIDS) due to brainstem nuclei dysfunction [4]. Additionally, the TCR has been suggested to link to bruxism and other sleep disorders [5].

We are not aware of a known pathophysiologic mechanism to directly link this patient's right middle cerebral artery ischemic stroke to manifestation of the TCR. While trigeminal–cortical connections are well-established in neuroanatomy, we think it would be speculative to try to directly anatomically link cortical ischemic stroke and the TCR at this time. It is possible that the patient had an unrecognized predisposition to the TCR which was provoked by masticatory movement as she re-learned how to chew and swallow during recovery.

This case broadens consideration of clinical scenarios in which a TCR can occur. The TCR may be under-recognized during stroke recovery, and may be a cause of sudden death in post-stroke patients. As such, there is a need for further study into the effect of ischemic brain injury on vagally mediated responses.

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Compliance with ethical standards

Conflict of interest All authors declare that they have no conflict of interest.

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