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# Vocal Cord Paralysis Due to Post-Viral (COVID-19) Vagal Neuropathy Assessed by Transcranial Magnetic Stimulation

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## Introduction

Vocal cord paralysis, probably caused by post-viral vagal neuropathy resulting in dysfunctional vocal cord mobility, has been reported in patients with no history of intubation recovering from coronavirus disease (COVID-19).[1]

## Aim of study

The study's aim is to present a patient, with paralysis of the left vocal cord recovering from COVID-19, assessed with standard laryngoscopy and stroboscopic examination and with additional navigated transcranial magnetic stimulation technique.

## Case report

Here, we present a 39-year-old female, diagnosed with dysphonia and vocal fatigue complaint at the otolaryngology outpatient clinic. The patient had no known history of surgery, chronic disease, or trauma. Neurological examination and brain MRI revealed no signs of neurological deficits or other cranial/peripheral nerve involvement. A CT scan of the neck and chest showed no lesions or masses.

The patient suffered COVID-19 eleven months before admittance (presented with headache and loss of taste and smell with full spontaneous recovery) and suffered the second COVID-19 (an antibody test on the admittance proved detectable SARS-CoV-2 virus IgM antibodies). Three weeks before the admittance, the symptoms of upper respiratory tract infection were reported, followed by headache, burning sensation in the throat, and nasal secretion. Laryngeal symptoms (dysphonia) developed on the third day of the second COVID-19.

Video laryngoscopy and stroboscopic examination revealed paralysis of the left vocal cord with an intermediate (cadaveric) position.

Acoustic voice analysis showed pathological voice findings for jitter (1.15 %), shimmer (1.182 dB), and noise-to-harmonic ratio (NHR) (8 dB).

Control examination represents the period when the patient subjective symptoms subsided approximately six months after the diagnosis.

Navigated transcranial magnetic stimulation (TMS) (Nexstim Plc.) was applied to assess the corticobulbar tract integrity by recording the corticobulbar motor-evoked responses (MEPs) [2] from thyroarytenoid (TA) muscles at the hospital admittance and control examination.

The disposable paired hook-wire electrodes were inserted in the TA muscle using a transcutaneous approach via a cricothyroid membrane.

A single-pulse TMS was applied with a figure-of-eight coil. The subject's head was co-registered to the individual MRI using anatomical landmarks.

The primary motor cortex (M1) for the TA muscle was mapped to elicit contralateral corticobulbar MEPs in the TA muscle.[2] At least five out of 10-20 repeatable MEPs were collected.

Right hemispheric M1 stimulation for TA muscle representation elicited **MEPs from the left TA with peak-to-peak MEP amplitude significantly** (t (32)=-3.71, **p<0.001**) **lower at the admittance to the hospital** (mean 396.02 μV) **compared to the control examination** (mean 1298.36) (Fig 1, left).

Left M1 stimulation for TA muscle representation elicited MEPs in the right TA muscle significantly (t (53)=-2.16, p<0.03) lower at admittance to the hospital (mean 843.99 μV) compared to the control examination (mean 1002.28 μV) (Fig 1, right).

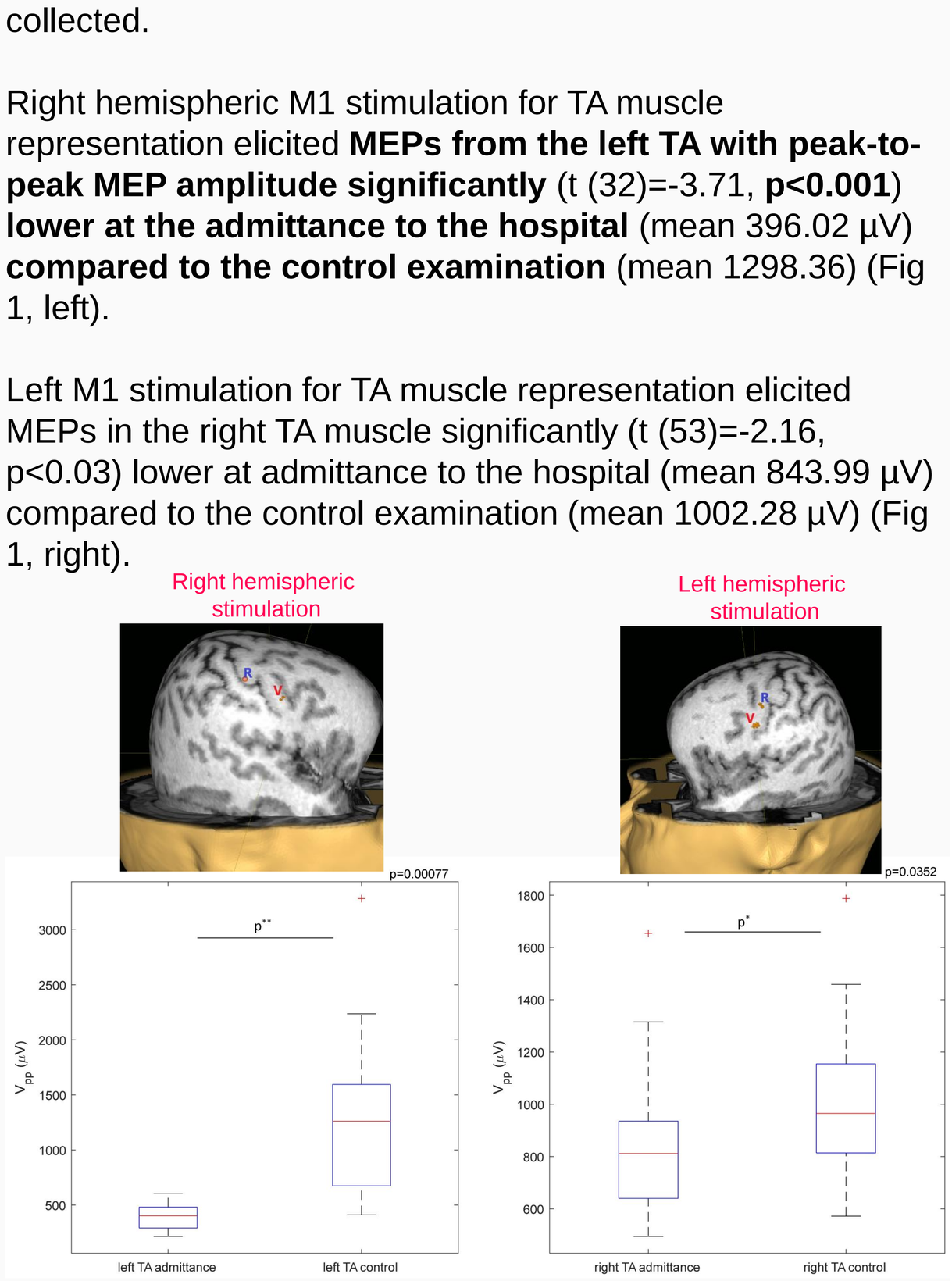


**Fig 1.** Graphical representation of corticobulbar motor evoked potentials (MEPs) peak-to-peak amplitude distributions from the left and right thyroarytenoid (TA) muscles, at the admittance and after six months, following full recovery (control examination).
*Note: On each box, the central red mark indicates the median, and the bottom and top edges of the box indicate the 25th and 75th percentiles, respectively. The whiskers represent data between the minimum and maximum values not considered outliers, and the outliers are plotted individually using the red '+' marker symbol. A custom-made Matlab script (R2021a) performed MEP amplitude estimation using an automatic algorithm. p** < 0.001, p*<0.05. 3D MRI presents "**R**" referent M1 spot for hand representation, and "**V**" represents M1 for vocal, TA muscle.*

Control laryngoscopy and stroboscopic examination showed completely consistent vocal cords mobility with complete adduction during phonation, and steady findings in acoustic analysis for jitter (0.428 %), shimmer (0.251 dB), and noise-to-harmonic ratio (NHR) (19.882 dB).

## Conclusion

This is a unique case report of a patient with vocal cord paresis most likely due to post-viral (COVID-19) vagal neuropathy, which was tested by navigated TMS, a non-standard laryngological technique that proved to be sensitive in testing the integrity of the corticobulbar pathway.

*[1] Rapoport SK, et a. Acute Vocal Fold Paresis and Paralysis After COVID-19 Infection: A Case Series. Ann Otol Rhinol Laryngol. 2022;131:1032-1035.*
*[2]Rogić Vidaković M, et al. Excitability of contralateral and ipsilateral projections of corticobulbar pathways recorded as corticobulbar motor evoked potentials of the cricothyroid muscles. Clin Neurophysiol. 2015;126:1570-7.*

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