Phonatory function following acoustic neuroma surgery

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Disclosure

The authors have no disclosures to report.
I acknowledge my continuing obligation to disclose to AANS/NREF/NPA, promptly and in writing, any change in my circumstances. I further acknowledge that if there is any case where my private interest conflict with the interests of AANS/NREF/NPA, I will indicate that I may have a conflict and abstain from any vote, speaking engagement, planning related to that issue.
Acoustic neuroma (AN) is a well-recognized cause of neurological morbidity, peripheral facial paralysis being one of the most prevalent. Phonatory disfunction in the late post-operative term has not been properly addressed thus far. Purpose: The objective of this study is to describe the phonatory function outcomes on the long-term postoperative follow-up of AN surgery and identify its prognostic factors.
Methods

This cohort study included patients submitted to AN surgery from 1999 to 2014, with a mean follow up of 6.4 ± 4.5 years. To evaluate the phonatory function, we performed a combination of noninvasive acoustic and aerodynamic measurements including vocal intensity and stability, maximum declination rate of the glottal airflow (MDR) and transglottal pressure scale (TP).
Results

101 patients were studied. 25(24.7%) presented a deficit in phonatory function. Women comprised 56% and the mean age was 42.4 ± 13.8 years (range 19-80). 100% presented reduced expiratory airflow capacity with excessive manifestation of the laryngeal musculature (TP>1.23 s; MDR /z/ ≥ MDR /s/).
Results

Dysphonic patients presented more neurofibromatosis II (NF II) (40% vs 12.7% p=0.002), large tumors (3.8 ±1.1 p=0.002) and needed less surgeries (≤2, 56.7% vs 74.6%, p=0.0073) in univariate analysis. NFII and tumor size were predictive of dysphonia according to multivariate analysis (NFII - OR 5.57, p=0.006; tumor size- each 1 cm, OR 1.68, p=0.062).
Conclusions and Summary Points

• The late postoperative prevalence of dysphonia corresponded to 24.7%.
• Dysphonia could be secondary to the hyperfunction of laryngeal musculature explained by the reduced expiratory airflow found in our patients.
• Tumor size and NF II were predictors for the occurrence of dysphonia in the present study.