

Unilateral High Vagal Paralysis: Relationship of the Severity of Swallowing Disturbance and Types of Injuries

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Objective: To evaluate the prognoses of high vagal lesions and their association with their clinical presentation, and seek to determine the best strategy of management for each patient.

Study Design: Retrospective case series with chart review.

Materials and Methods: Patients following laryngoplasty from 2001 to 2008 at a tertiary referral voice and swallowing center in Taiwan were reviewed. They were divided into two groups according to the etiologies of high vagal trauma. The pre- and posttreatment voice and swallowing functions were reviewed; swallowing status was compared between patient groups.

Results: Of 186 consecutive patients who underwent laryngoplasty, 8 females and 9 males were diagnosed of unilateral high vagal damage. One group of 11 patients suffered vagal damaging suddenly from skull base trauma, cerebrovascular accidents, and surgical complications; symptoms of vagal paralysis occurred immediately after those events. The second group was comprised of six patients who lost their high vagal functions gradually from a skull base tumor or mass compression. Feeding tube status was significantly different between the groups. All except one patient in group 2 recovered their voice and swallowing abilities after appropriate laryngoplasty.

Conclusions: High vagal nerve damage from skull base surgery or trauma leads to a higher incidence of feeding tube dependency than that from skull base tumor compression. Patients can be treated successfully with an appropriate injection or medialization thyroplasty. Immediate laryngoplasty is suggested for cases right after skull base surgery or trauma.

Key Words: Unilateral high vagal paralysis, vocal cord paralysis, dysphagia, laryngoplasty.

Level of evidence: 2a.

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INTRODUCTION

Unilateral vocal cord paralysis (UVCP) may be caused by lower laryngeal nerve damage or high vagal lesions. The incidence of high vagal lesions is much lower than that of lower laryngeal nerve damage.¹ In Taiwan, around 15% of UVCP cases are caused by high vagal lesions. Most of these are a result of complicated surgery above the skull base and from the cerebellopontine (CP) angle or from skull base tumors.¹

The laryngopharynx is innervated by the vagus nerve's sensory and motor branches. Patients with unilateral high vagal lesions would suffer from breathy dysphonia and aspiration because of impaired vocal cord

movement. Due to the loss of sensation of the laryngopharynx, patients might also suffer severe dysphagia related to the poor coordination of the oro- and hypopharyngeal phases of swallowing.

An impact on quality of life has been proven in patients with UVCP and with adequate management, this condition can be improved.² The goal of management for UVCP is to decrease the glottal gap and restore phonation and the swallowing function. Medialization thyroplasty and injection laryngoplasty are the most common surgical treatments for cases of uncompensated UVCP. Pou et al.³ reported that laryngeal framework surgery can successfully control aspiration. But some cases were reported that still required revised treatment of voice problems and 17% remained feeding tube dependent.

Symptoms of high vagal paralysis range greatly from etiology and when they happened, thus, the required treatment also differed greatly. However, the relationship between etiologies and treatment modalities has not been discussed in previous publications. The present study reviews the prognoses of high vagal lesions and their association with their clinical presentation, and seeks to determine the best strategy of management for each patient.

MATERIALS AND METHODS

This was a retrospective study. All cases of UVCP with voice and swallowing disturbances that received laryngoplasty

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TABLE I.
Patient Characteristics of Unilateral High Vagal Paralysis in the Present Study.

No.	Age at Treatment	Sex	Time Before Diagnosis	Side of Vagal Paralysis	Cause of Vagal Paralysis	Surgical Modality	Feeding Tube History	Group	Dysphagia at Presentation
1	51	F	x	R	Right PICA aneurysm and SAH s/p	Li	Y	1	Mild
2	72	M	x	R	Right CP angle acoustic neuroma Schwannoma s/p	Li	Y	1	Mild
3	35	F	x	L	Left CP angle AVM rupture with SAH	Li	Y	1	Mild
4	54	M	x	R	Right skull base fracture with traumatic SAH	Mt	Y	1	Severe
5	61	M	x	L	left skull base glomus tumor s/p	Li	Y	1	Mild
6	71	M	x	L	left middle fossa trigeminal neuroma s/p	Li	Y	1	Mild
7	37	F	x	L	Left skull base fracture	Mt	Y	1	Moderate
8	57	M	x	R	Right skull base meningioma s/p	Li	Y	1	Mild
9	56	F	6 months	R	Type I Chiari malformation type I with syringomyelia and syringobulbia	Li	N	2	Mild
10	55	M	x	R	Traumatic SAH and right cerebellar hemorrhage s/p	Mt	Y	1	Severe
11	55	M	x	L	Traumatic SAH involving bilateral frontotemporal region and left cerebellar tentorium	Li	Y	1	Mild
12	43	F	x	R	Right CP angle acoustic neuroma s/p	Mt	Y	1	Severe
13	17	M	2 years	R	Right skull base glomus tumor	Li	N	2	Mild
14	74	M	5 months	L	Left skull base glomus tumor	Mt	N	2	Mild
15	54	F	x	R,	Right skull base glomus tumor	Li / Mt*	N	2	Mild
16	77	F	x	R	Bilateral skull base glomus tumor	Mt	Y	2	Severe
17	56	F	4 years	L	Bilateral skull base glomus tumor	Mt	N	2	Mild

Group definition is in text.

Li = lipoinjection; Mt = medialization thyroplasty.

*Initial lipoinjection and medialization thyroplasty 5 years thereafter.

from 2001 to 2008 at Chang Gung Memorial Hospital were reviewed. Surgical techniques applied, including injection laryngoplasty and medialization thyroplasty, were described in previous publications.⁴⁻⁶ Of the 186 consecutive laryngoplasty patients in our database, the 17 patients with high vagal causes were reviewed. Diagnosis was based on serial video stroboscopic findings consistent with UVCP. A determined lesion in the area where the vagus nerve originates can be traced in each patient.

Using the onset pattern and causes of the high vagal paralysis, we divided the patients into two groups. Group 1, or sudden onset group, included those with skull base trauma, cerebrovascular accidents, or surgical complications. Symptoms of vagal paralysis occurred immediately after those events. Group 2, or gradual onset group, lost their high vagal functions because of a skull base tumor or mass. The durations from symptoms to final diagnosis were recorded. Clinical data, such as age, sex, symptoms, side of vocal cord paralysis, history and interval of feeding tube dependency as well as causes of high vagal lesions, were compared.

Voice Laboratory Measures

Voice analysis was performed pre- and postoperatively. Patients were asked to read a standard passage, and pronounce and sustain the vowel /a/. From the stable midportion segment of the vowel voice, a voice sample was captured. Acoustic characteristics of the voice were digitalized and measured using a Kay Elemetrics Computerized Speech Lab system (model CSL4300B, software version 5.02). From the recorded sample data, fundamental frequency (F_0), jitter (perturbation of frequency), shimmer (perturbation of amplitude), and harmonic-to-noise ratio (HN) values were tabulated. The time sustaining production of the vowel/a/ was recorded as maximal phonation time (MPT). Patients were also asked to project the consonants

/s/ and /z/. MPTs and ratio of s to z (s/z) were calculated with the help of speech language pathologists.

Dysphagia Grading

The swallowing disturbance was graded as mild, moderate, and severe. Mild dysphagia was defined as episodic aspiration while drinking, but without aspiration pneumonia episodes or limitations in oral intake; moderate was defined as patients who had aspiration pneumonia episodes but still able to take modified diet through mouth, whereas severe was defined as being unable to take anything through the mouth except by feeding tube. All patients were treated by either lipoinjection or medialization thyroplasty according to their clinical status.

Statistical Analysis

All analyses were performed using the Statistical Package for the Social Sciences software, version 11.05 (SPSS, Inc., Chicago, IL). The differences between the voice analysis data were analyzed by Student *t*-test. Differences between noncontinuous variables were tested by means of a nonparametric Mann-Whitney *U* test and Chi-square test. A two-sided *P*-value less than .05 was considered statistically significant.

RESULTS

Patient demographics are listed in Table I. The mean age was 54 years old (ranging from 17 to 77 years old); eight patients were female and nine were male. Among them, 10 suffered right-side paralysis and 7 suffered left-side paralysis. From reviewing the patients' histories, we learned there were 12 patients who had

TABLE II.
Serial Voice Analysis Data.

	Pre-OP	Post-OP 1 M	Post-OP 3 M	Post-OP 6 M
Maximal phonation time (sec)	3.7 ± 2.0	8.7 ± 3.6*	7.8 ± 3.1*	9.4 ± 3.5*
s:z ratio	2.6 ± 1.9	1.2 ± 0.5*	1.2 ± 0.4*	1.4 ± 0.6*
Fundamental frequency (Hz)	174.7 ± 38.1	173.7 ± 35.6	173.4 ± 38.3	169.5 ± 44.5
Jitter (%)	3.9 ± 1.8	1.4 ± 0.6*	1.5 ± 1.5*	1.1 ± 0.4*
Shimmer (dB)	1.1 ± 0.7	1.3 ± 1.2	1.0 ± 0.9	1.0 ± 1.0
Harmonic-to-noise ratio (dB)	-0.6 ± 7.2	9.3 ± 6.1*	9.6 ± 7.1*	11.3 ± 2.9*

**P* < .05 compared to preoperative evaluation.

used feeding tubes for various intervals after being diagnosed with high vagal lesions. Eight patients could change to oral feeding without surgical procedures. Four patients were still feeding tube dependent in the meantime when we performed laryngoplasty.

There were six patients whose vagal nerve was damaged after surgery of the skull base. Four patients had suffered skull base fractures with associated hemorrhaging, and one patient suffered a cerebrovascular accident. These 11 patients who had no swallowing complaints prior to attacked events but got complete vagal paralysis suddenly thereafter are assigned to Group 1 in our study.

Group 2 included six patients who loss their vagal function gradually. Five of them had a skull base neoplasm and one suffered from a type I Chiari malformation; the lapse of time from initial symptoms of UVCP to the confirmation diagnosis was from 5 months to 4 year.

There were 10 skull base tumor patients in this series. All the pathologies showed benign in nature. Five of them accepted a curative intent brain surgery and the other five received radiation therapy. In the surgical group, the tumors of greatest diameter ranged from 25 to 63 mm (median 35mm), while they were 35 to 50 mm (median 40 mm) in the radiation therapy group. All five patients who had accepted excision surgery needed tube feeding after surgery from 6 days to 8 months. One (case No. 12) patient was still feeding tube dependent before laryngoplasty but was removed thereafter. Another patient (case No. 16), who had radiation for her tumor, was not able to return to oral intake even after medialization thyroplasty.

There were 18 surgical procedures: 10 intracordal injections of autologous fat, and 8 medialization thyroplasties with silastic implants performed on the patients. One patient underwent medialization thyroplasty 5 years after an autologous fat intracordal injection due to progressive hoarseness.

All the patients accepted definite laryngoplasty surgeries at least 6 months later after symptoms occurred. In Group 1, based on their symptom severity, seven patients had lipoinjections and four underwent medialization thyroplasties. Three lipoinjection and three medialization thyroplasties were performed in Group 2.

The results of the voice analysis are shown in Table II. The postoperative voice showed a significant improve-

ment within 6 months. The feeding tube-dependent rates improved from 27% (4 of 17) preoperatively to 5.9% (1 of 17) postoperatively.

The incidence of ever tube feeding differed significantly between the two groups (Table III). Patients in Group 1 all had histories using the feeding tube, whereas only one patient had such a history in Group 2.

DISCUSSION

The laryngopharynx is a conduit for breathing and swallowing. It is also the most important organ for voice communication. Its motor and sensory innervation is mostly derived from the vagus nerve and branches into the superior and recurrent laryngeal nerve as it descends.

UVCP caused by a high vagal lesion is rare. The incidence of high vagal paralysis is not well established, but is estimated to be around 10% of unilateral vocal paralysis cases in Taiwan.¹ The causes of high vagal lesions may result from cerebrovascular accidents, skull base tumors or trauma, cervical spinal cord damage, or complications from operations on them. In addition to motor dysfunction, sensations of the larynx are also impaired by high vagal lesions. Thus, managing UVCP of high vagal origins should differ considerably from managing UVCP caused by recurrent laryngeal nerve damage.

The pharyngeal wall is also controlled by the plexus from the vagus nerve. Presentation of patients with high vagal UVCP may range from slight aspirations with breathy dysphonia to an inability to swallow through the mouth. Failure to relax the cricopharyngeus muscle combining vocal incompetence because of vagal injury results in laryngeal penetration or aspiration. Severe life-threatening symptoms may be induced. These patients may acquire repeated aspiration pneumonia

TABLE III.
In Patients of Sudden Onset High Vagal Nerve Damage, the Feeding Tube Usage Rate Was Higher Than That of Gradual Damage Cases.

	Group 1 (n = 11)	Group 2 (n = 6)	
Feeding tube history			<i>P</i> = .000
(+)	11	1	
(-)	0	5	

Chi-square test significance *P* < .05.

Mann-Whitney *U*-test, significant *P* < .05.

Li = lipoinjection; Mt = medialization thyroplasty.

UVCP with high vagal lesions

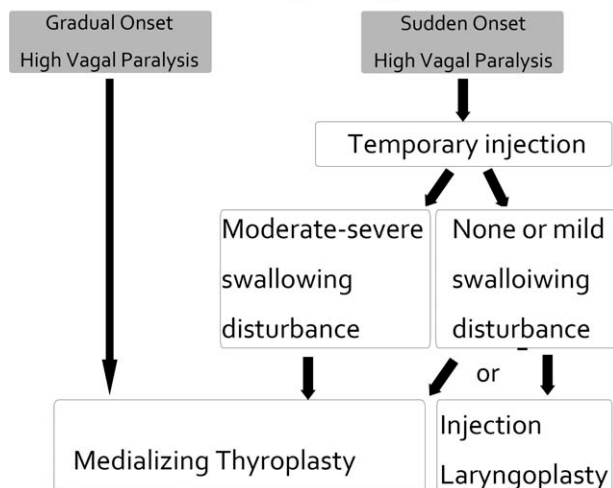


Fig. 1. Suggestions in managing high vagal paralysis.

and be dependent on feeding tubes because of their poor swallowing coordination.

Dysphagia due to high vagal paralysis is usually caused by aspiration or failure in food bolus propulsion. In our series, in the course of laryngoplastic surgery, which was performed after more than 6 months after symptom occurrence, there was still 24% (4 of 17) who were feeding tube dependent. Of these four, two resulted from direct brain injury, one case came from skull base surgery, and one case derived from chronic compression by a bilateral skull base glomus tumor. Because of prior radiation and major depressive mood disease, this last case was also the only one that failed to regain the ability for oral food intake after the surgery.

Acute nerve function damage from head injury or brain surgery leads to significant differences in swallowing disability for sudden and gradual vagal nerve damage. We noticed sudden nerve damage from skull base surgery or trauma led to higher tube feeding rate. Such patients lost nerve function suddenly, so did not have time to learn to modify their swallowing movements. Some might restore their swallowing by learning to coordinate their oropharyngeal motion, but most required surgery to regain their normal swallowing ability.³ In these patients, we suggested early laryngoplasty intervention in repairing their voice and swallowing functions. On the other hand, patients of tumor compression lose nerve function gradually. They seldom need feeding tubes because they are able to modify their swallowing movements by themselves.

There are 10 skull-base tumors associated high vagal paralysis patients that were reviewed in the study. Patients of surgical treatment lost their swallowing and vocal functions suddenly, and all of them needed tube feeding right after skull base surgery, whereas only one needed tube feeding as a nonsurgical patient. Unfortunately, none restored their vocal function after skull base tumor treatment, no matter how brief the symptom had occurred. A radical resection at the jugular foramen could

result in multiple cranial nerve paralysis and worsen the vocal and swallowing status. A long-term review demonstrated a slow tumor growth nature, but grave outcomes with interventions that were too aggressive.^{7,8} Because laryngoplastic surgery can satisfactorily restore voice and swallowing problems, when considering the patients' quality of life, we agree with Arts and Fagan,⁹ who suggested a conservative tumor resection or postponing tumor resection until the cranial nerve impairment becomes evident or when other vital structures are threatened. Although laryngoplasty can help most patients in their voice and swallowing, the one with severe swallowing problems by tumor compression could not regain the ability of oral intake. This 77-year-old lady gradual lost not only high vagal but also other cranial nerves functions within 25 years. She received benefit of vocal function but not swallowing from medialization thyroplasty surgery. Our unsuccessful experience may imply laryngoplasty is not expected to help swallowing in the type of gradual onset by multiple cranial nerve loss in patients.

Although medialization thyroplasty was considered a standard procedure for UVCP, the neck scar that is left behind discourages some patients. There were 10 patients who accepted autologous fat intracordal injections instead of medialization thyroplasty. One received a salvage medialization thyroplasty 5 years after lipoinjection. All except one patient gained voice and swallowing benefits from this treatment. We suggest injection laryngoplasty for patients with only voice disability and without dysphagia. This can achieve similar outcomes to medialization thyroplasty. For cases of aspiration or dysphagia other than voice problems, medialization thyroplasty is preferred.³

Although some reports^{3,10} suggested an immediate medialization thyroplasty for patients accepting skull base surgery, measuring the deficit needed for correction is difficult. We suggest injecting a short-term substance right after skull base surgery for temporary control. A determined surgery is suggested later on when the cord position is constant.

In our series, 33% of cases of skull base glomus tumor were noticed bilaterally. Considering this bilateral tendency and probable subsequent contralateral vocal cord paralysis, performing an irreversible procedure such as injecting a long-lasting substance for laryngoplasty is not suggested. Instead, a medialization thyroplasty for such patients should be considered because of its benefit in easily monitoring airway impact while the surgery is being performed. Figure 1 shows the suggestions in the management of patients of unilateral high vagal paralysis.

Four of our cases (Nos. 9, 13, 14, and 17) were labeled as idiopathic UVCP initially. The interval between hoarseness occurred through definite diagnosis of high vagal lesions ranging from 5 months to 4 years. Three of these cases had final diagnoses of a glomus tumor, and one case was diagnosed as a type I Chiari malformation. Most of the skull base lesions were benign and slow growth in nature. In our opinion, radiographic evaluation of the skull base and cervical spinal cord for patients of UVCP without known causes should not be overemphasized.

Although there was great improvement in vocal and swallowing performance after laryngoplasty surgery, nasal airflow leakage resulting in hypernasality was a problem for communication. Hypernasality and nasal regurgitation have been reported to be successfully managed using a palatal adhesion technique.¹¹ However, whether laryngeal function would be affected by the intubation required after palatal surgery requires further study.

CONCLUSIONS

1. High vagal nerve damage from skull base surgery or trauma leads to a higher incidence of tube feeding than damage from skull base tumor compression.
2. Most unilateral high vagal paralysis patients can get benefit from laryngoplasty in their voice and swallowing performances.
3. For cases of chronic high vagal lesion by tumor compression, considering its bilateral tendency, a reversible procedure like medialization thyroplasty is preferred to the injection of long-lasting substances.

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