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Hoarseness subsequent to cardiovascular surgery, intervention, maneuver and endotracheal intubation: The so-called iatrogenic Ortner's (cardiovocal) syndrome

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Abstract

Background: *The clinical characteristics and outcomes of hoarseness subsequent to cardiovascular surgery, intervention, maneuver and endotracheal intubation have not been systematically elucidated.*

Methods: *The literature of hoarseness following cardiovascular surgery, intervention, and maneuver and intubation published between 1980 and 2011 was comprehensively retrieved in the MEDLINE database and the Google and Highwire Press search engines.*

Results: *The so-called "iatrogenic Ortner's (cardiovocal) syndrome" developed 0–7 (2.33 ± ± 2.66) days following cardiovascular surgery, intervention, maneuver and endotracheal intubation with an incidence of 10.15%. The most common symptoms associated with hoarseness were stridor (49.45%) and aspiration (15.38%). Patent ductus arteriosus ligation and otherwise congenital heart disease repair were the two main causes leading to such a complication. Patients' hoarse voice spontaneously resolved in 70.52%, and persisted in 33.61% of the patients. Treatment of the hoarseness included gelfoam/teflon injection, intravenous steroid therapy, type 1 thyroplasty and arytenoid adduction. Hoarseness recovered in 46.67%, improved in 13.33%, and persisted in 40%.*

Conclusions: *The recurrent laryngeal nerve was often injured following cardiovascular surgery, intervention, maneuver and endotracheal intubation. Care must be taken during the manipulations in order to avoid the nerve injury. The so-called "iatrogenic Ortner's (cardiovocal) syndrome" was a wrong concept as it did not meet the satisfaction of a main element "cardiovascular disease as an underlying cause of hoarseness" of the definition of Ortner's (cardiovocal) syndrome defined by Ortner in 1897. It was actually an immediate vocal cord complication following cardiovascular manipulation.* (Cardiol J 2012; 19, 6: 560–566)

Key words: cardiovascular surgical procedures, hoarseness, intratracheal intubation, postoperative complications

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Introduction

Vocal cord paralysis may have various etiologies including inflammation, tumor and idiopathy [1]. It is predominantly complicated in thyroidectomy, a local lesion such as edema, rubor, hematoma and granuloma, or dysfunctional disorder without structural malformations [2]. The left vocal cord was more involved than the right, most commonly resulted from thoracic or cardiac surgery (53.1%) [3], cardiac surgery only (28.5%) and prolonged intubation/prematurity (16.7%) [4]. The underlying cause was neoplastic in 32%, surgical in 30%, idiopathic in 16%, traumatic in 11%, central in 8%, and infectious in 3% of the patients with unilateral vocal cord paralysis [5]. In USA, postoperative hoarseness has once been claimed for all surgical operations: general surgery (31%), cardiothoracic surgery (12%), neurosurgery (12%), otolaryngology (10%), and orthopedic surgery (5%). In the claims, the most common procedures were thyroid and parathyroid surgery (32%), cervical disc procedures (16%), thoracic procedures (12%), endarterectomy (9%), and open neck biopsy (6%). The most common claims cited were improper performance (61%), failure to recognize a complication (36%), and consent issues (19%) [6]. The functional recovery rates for different causes were surgery 31%, idiopathic 19%, traumatic 18%, and neoplastic 0% [5]. The diagnosis was based on a review of the history, a physical examination, and computed tomographic scan or magnetic resonance imaging [5].

Hoarseness was the most common presentation of vocal cord paralysis. Inada et al. [7] categorized the hoarseness according to a four-point scale. In their report, 32% (22/68) patients developed hoarseness: five (23%) were grade 1, 14 (64%) were grade 2, and three (14%) were grade 3 (aphonic). Hoarseness lasted 4 ± 3 (1–14) days, and an additional patient with left vocal cord paralysis lasting 60 days. Except for hoarseness, stridor, difficulty in expectoration, dysphagia, and aspiration were the common symptoms in such patients [8]. In infants, they often manifested stridor and feeding difficulty and both required feeding tubes [9]. The natural resting state of the vocal cords after paralysis is the paramedian or median position. These positions can cause obstruction of the airway at the level of the glottis and result in dyspnea, stridor, and aspiration risk for the pediatric patient [10]. A paramedian position of the left vocal cord may narrow the laryngeal entrance, while collapse of left supraglottic structures may cause airway obstruction. Left vocal cord paralysis may be associated with prolonged inspirium, wheezy respiration, or severe stridor during heavy exercise [11].

The clinical characteristics and outcomes of hoarseness subsequent to cardiovascular surgery, intervention, maneuver and endotracheal intubation have not been systematically elucidated in the medical literature so far. Moreover, the concept of "iatrogenic Ortner's (cardiovocal) syndrome" has been misused for the expression of such complicated conditions. Thus, a comprehensive review and thorough discussion are necessary.

Methods

Retrieval policies

The medical literature of hoarseness subsequent to cardiovascular surgery, intervention, and maneuver, and endotracheal intubation published between 1980 and 2011 was comprehensively retrieved in the MEDLINE database and the Google and Highwire Press search engines. Patients with left vocal cord paralysis who did not present with hoarseness or hoarseness were not mentioned as one of the symptoms in the reports were not included. Recurrent nerve palsy caused by cerebral and carotid arterial lesions were excluded. The search was ended on December 31, 2011.

Statistics

Data were expressed in mean ± standard deviation and student t test was used to evaluate intergroup differences. $P < 0.05$ was considered of statistical significance.

Results

Totally 50 reported were collected, including 19 (38%) case reports or case series [12–29], 1 (2%) technique [30], 17 (34%) retrospective [31–48] and 13 (26%) prospective reports [7, 11, 49–59].

The patient setting included 3,802 patients. Their age range was between neonate and 83 years old (median 51 year; mean 38.82 ± 28.65 years). Of the 3,003 patients whose genders were given in the reports, 1,921 were males and 1,082 were females with a male-to-female ratio of 1.78. The detailed information of cardiovascular surgery, intervention, maneuver and intubation that induced hoarseness was listed in Table 1.

Hoarseness developed 0–7 (2.33 ± 2.66) days postoperatively (n = 6). Of the 296 patients who had their recurrent nerve palsy recorded, 262 (88.51%) were left, 27 (9.12%) were right, and 7 (2.36%) were bilateral. The incidence of hoarse and/or low voice in the whole patient population receiving cardiovascular surgery, intervention,

Table 1. Cardiovascular surgery, intervention, maneuver and endotracheal intubation that induced hoarseness.

Cardiac operations:
Replacement of the aorta
Coronary artery bypass grafting
Valve replacement
Congenital heart disease repair
Patent ductus arteriosus ligation/clips/under video-assisted thoracic surgery
Heart transplantation
Cardiac interventions:
Endoluminal treatment of thoracic aortic aneurysm
Transcatheter coil closure of patent ductus arteriosus
Percutaneous embolization of a vagal paraganglioma
Catheter ablation of atrial fibrillation and flutter
Left pulmonary artery stenting
Transradial cardiac catheterization
Cardiac maneuvers:
Central venous catheterization
Cardioversion
Endotracheal intubation

Table 2. Symptoms associated with hoarseness.

Stridor	45 (49.45%)
Aspiration	14 (15.38%)
Respiratory distress/failure to extubate	9 (9.89%)
Poor feeding	8 (8.79%)
Difficulty weaning from nasal continuous positive airway pressure	6 (6.59%)
Dysphagia	5 (5.49%)
Dysphagia mainly to liquids	1 (1.10%)
Delayed swallowing	2 (2.20%)
Reduced laryngeal closure	1 (1.10%)

maneuver and intubation was 386/3,802 (10.15%). Based on the retrospective and prospective studies, the prevalence of hoarseness was 6.64% (46/693) in aorta replacement, 4.5% (98/2,192) in cardiovascular surgery, 19.5% (51/262) in congenital heart disease excluding patent ductus arteriosus (PDA) repair, 29.2% (150/513) in PDA ligation, and 1.82% (5/275) in PDA intervention, respectively.

The most common symptoms associated with hoarseness were stridor (49.45%) and aspiration (15.38%). Alternative symptoms included aspiration, respiratory distress, and poor feeding, etc. (Table 2).

Table 3. Hoarseness recovery without receiving a medical therapy.

Hoarse recovery	Case	Follow-up time (month)
Resolved	122 (70.52%)	4.47 ± 4.67 (0.07–14.8)
Improved	4 (2.31%)	6.33 ± 1.53 (5–8)
Right vocal cord compensation	6 (3.47%)	12
Persist	41 (33.61%)	10.75 ± 7.46 (1–24)*

*p = 0.012 in comparison with the hoarseness-resolved patients

Table 4. Cordal therapy in 15 patients with hoarse voice complication following cardiovascular operation, intervention and maneuver.

Therapy	Recovered	Improved	Persist
Gelfoam/teflon injection	4 (26.67%)	1 (6.67%)	1 (6.67%)
Intravenous steroid therapy	2 (13.33%)		
Type 1 thyroplasty	1 (6.67%)		
Speech therapy		1 (6.67%)	
Arytenoid adduction			5 (33.33%)
Total	7 (46.67%)	2 (13.33%)	6 (40%)

Without receiving treatment, patients' hoarse voice resolved in 70.52%, while persisted in 33.61%. The follow-up time interval of the patients with persist hoarseness was much longer than those with resolved hoarseness (p = 0.012) (Table 3).

In 14 patients with hoarse voice complication, the following managements were made: gelfoam/teflon injection in 6, intravenous steroid therapy in 2, type 1 thyroplasty in 1, and arytenoid adduction in 5. Hoarseness recovered in 7 (46.67%), improved in 2 (13.33%), and persist in 6 (40%) (Table 4).

Discussion

Incidence

As reported by Truong et al. [43], the prevalence of weak cry/weak voice was 59 (54%), and that of hoarseness was 23 (21%) after congenital heart defect (mainly PDA) repair. In extremely low body

weight baby, the incidence of hoarseness after PDA ligation was 88% [59]. The incidence of hoarseness was higher in valve replacements (2.2%) than in coronary artery bypass grafting (0.9%), with a total incidence of 1.2%. The position of the paralyzed cord was in the median, paramedian, and intermediate positions in 2, 13 and 2 patients, respectively [37]. In thoracic aortic aneurysms types I, II and III, the incidences of vocal ford paralysis were 64%, 25% and 21%, respectively. Hoarseness improved in 16% and persisted in 84% at a follow-up of 3 months [35]. Spanos et al. [57] reported that the incidence of iatrogenic vocal fold paralysis in patients with vascular clips (19%) was similar to the incidence in those with suture ligature (20%).

Etiology

Cardiovascular operation. The etiology of vocal cord dysfunction following open heart surgery remains unclear [8]. Shaw and Pierce [6] recommended that indirect injury by large double-lumen endotracheal tubes or transesophageal probes, phrenic or vague nerve injury associated with cooling and cardioplegic infusion, and excessive traction due to sternal retraction are the main causes of vocal fold paralysis subsequent cardiac surgery. Ishimoto et al. [37] proposed intraoperative low temperature may contribute to the recurrent laryngeal nerve paralysis. Dissection of the left internal mammary artery to its origin potentially jeopardizes the vagus nerve or its associated branches [12]. Injury from local cooling of the heart may be a possible etiology of hoarseness, but is more frequently associated with phrenic nerve palsy [60].

PDA ligation techniques included clips (52.9%), suture ligatures (41.2%), or both (5.9%) [32]. Among extremely low birth weight babies, the recurrent laryngeal nerve paralysis was more involved in the clip closure of PDA rather than by traction injury. The former was preferred in extremely low birth weight babies due to the advantage of less dissection, shorter operative time and safety to the great vessels. Nevertheless, less dissection may increase the risk of impingement of the recurrent laryngeal nerve by the clip [32].

Cardiovascular intervention. By coil closure of PDA, inappropriately implanted coil may distort the slender PDA with resultant angulation of the pliable PDA itself and precipitated the impingement on the left recurrent laryngeal nerve [22]. Hoarseness may be secondary to endoluminal treatment of a thoracic aortic aneurysm [26], transcatheter ablation of atrial fibrillation [21], and stenting of the left pulmonary artery [20] with analogous pathogenetic mechanisms.

Cardiovascular maneuver. The incidence of postoperative recurrent laryngeal nerve palsy was 7.8% in the patients with transesophageal echocardiographic (TEE) monitoring, but with significant longer durations of surgery, anesthesia, and cardiopulmonary bypass. These results suggest that placement of the TEE probe did not cause postoperative recurrent laryngeal nerve palsy, but the surgical manipulation itself and the durations of surgery, cardiopulmonary bypass, and tracheal intubation did [50].

Recurrent laryngeal nerve injury can be caused by traumatic injury from central venous line insertion [8]. The right internal jugular vein is usually used as a route for central venous catheterization as the anatomical continuity with the superior vena cava. The posterior route to the internal jugular vein was associated with the lowest complication rate. The commonest complication was inadvertent arterial puncture that may predispose a vagal trauma [61]. Recurrent laryngeal or vagus nerve is therefore predisposed to be injured by hematoma, thrombosis or fibrosis [62]. Similar complications have also been described during percutaneous interventional procedures, where compression of the anterior branch of the recurrent laryngeal nerve between the cuff of the endothracheal tube and the posterior part of the thyroid cartilage was the hypothesized mechanism [27].

Endotracheal intubation. Neuroparaxia of the recurrent laryngeal nerve has been described following endotracheal intubation without any local lesion, prolonged mechanical ventilation was a significant risk factor for vocal cord paralysis [63]. The inflated cuff of an endotracheal tube can compress the anterior branch of the anterior recurrent laryngeal nerve against the thyroid cartilage and cause paralysis [64]. In particular the anterior branches of the recurrent laryngeal nerve is vulnerable to damage by the endotracheal cuff [65, 66]. However, the authors who reported the complication did not mention whether the central venous catheterization was done. Therefore the role of the maneuver of endotracheal intubation in recurrent laryngeal nerve injury was suspected [15].

Diagnosis

The presentation of unilateral vocal cord paralysis is often a weak cry or low voice after extubation [67]. The diagnosis is best made by fiberoptic laryngoscopy, or barium swallow is best means of diagnosis of pharyngeal function [67]. Twenty-five (45%) of 55 patients demonstrated aspiration or laryngeal penetration with modified barium swallow [43]. The median time between cardiac surgery and

diagnosis of vocal fold paralysis was 20 days [43]. Postoperative vocal cord dysfunction was classified by laryngoscopy into 2: paresis and paralysis. Vocal cord paralysis is more common than paresis, and a paralyzed cord was more in the paramedian position than in the median position [68]. At a mean follow-up of 14.8 months, 77% patients had full recovery of vocal fold motion [47]. Clinical observations revealed patients demonstrated partial recovery, progression from paralysis to paresis, at 4 months, and then had full recovery when examined at 6–7 months. The minimum incidence of permanent vocal cord paralysis after cardiovascular surgery was only 3.5%, and the estimated incidence of permanent vocal fold paralysis was 5.3% [47].

Risk factors

Predictors of vocal cord paralysis were uncertain. The intubation time was an independent predictor of the severity of hoarseness [7]. Vocal cord paralysis after tracheal intubation may be attributed to ageing (aged 50 or above) and comorbidity (diabetes mellitus or hypertension), and longer intubation (3–6 h or > 6 h) [69]. Is has been reported that the prevalence of left vocal cord paralysis after PDA ligation was 4.2% of the whole patient population, and it was 8.0% in infants with low birth weights [32]. The intraoperative damage to the left recurrent laryngeal nerve along its course around the PDA during the dissection of the nerve might result in stretch injury [55]. Røksund et al. [11] found that infants with left vocal cord paralysis had a lower birth weight, longer duration of ventilation, and the greater proportion with bronchopulmonary dysplasia. Smith et al. [58] noted that infants < 30 weeks postmenstrual age and/or body weight < 1,250 g at PDA ligation were at the highest risk, with an incidence of left vocal cord paralysis of 25%. The risk of vocal cord paralysis increased with the operation time, and the risk was 5.6 times higher in patients receiving aortic surgery, 8.7 times higher in patients with descending aortic surgery [70]. In a multivariate logistic regression model, male gender, low ligation weight, and presence of laryngeal symptoms (stridor and dysphonia) were significant predictive factors [58]. There was no association between deep hypothermic circulatory arrest and the development of dysphagia or between length of TEE probe insertion time and development of dysphagia whether controlled for age of less than 1 year or not [53].

Management

Spontaneous recovery of hoarseness may take place within weeks, but in some cases, hoarseness would persist and the damage to the nerves remains permanent [8]. For prevention of nerve injury in the aortic aneurysm repair, "pull through technique", i.e., distal anastomosis to the prosthetic graft performed in the left pleural cavity, and the prosthetic graft pulled into the pericardial cavity through an aortic tunnel may be helpful [71]. Twenty-six percent of the patients required surgical intervention, including injection into the paralyzed fold and medialization thyroplasty [4]. Gelfoam, radiesse voice, and radiesse voice gel injections in patients with hoarseness led to improvement in 94% [48]. Joo et al. [47] reported that injection laryngoplasty or medialization thyroplasty were applied in all three of their patients with permanent vocal cord paralysis. Improvement could be achieved by speech therapy [26]. Truong et al. [43] found that 65% patients had persistent vocal fold paralysis with a median follow-up time of 16.4 months, 27% of the 109 patients underwent surgical intervention for their airway, feeding or voice. Vocal cord medialization rendered the median penetration-aspiration scale score improved from 4.0 to 3.0 [3]. After surgical treatment, arytenoid adduction or silicone injection into the vocal cord was performed, the maximum phonation time improved from 4 s before treatment to 10 s after treatment [35]. Infants with left vocal cord paralysis usually developed bronchopulmonary dysplasia, reactive airway disease and required a gastrostomy feeding tube due to abnormal swallow or aspiration with feedings [59].

In general, the so-called "iatrogenic Ortner's (cardiovocal) syndrome" often developed 0–7 (2.33 ± 2.66) days following cardiovascular surgery, intervention, maneuver and endotracheal intubation with an incidence of 10.15%. The most common symptoms associated with hoarseness were stridor (49.45%) and aspiration (15.38%). PDA ligation and otherwise congenital heart disease repair were the two main causes leading to such a complication. Patients' hoarse voice spontaneously resolved in 70.52%, and persisted in 33.61% of the patients. Treatment of hoarse voice complication included gelfoam/teflon injection, intravenous steroid therapy, type 1 thyroplasty and arytenoid adduction. Hoarseness recovered in 46.67%, improved in 13.33%, and persisted in 40%.

Conclusions

In conclusion, the recurrent laryngeal nerve was often injured following cardiovascular surgery, intervention, maneuver and endotracheal intubation. Care must be taken during the manipulations

in order to avoid the nerve injury. The so-called "iatrogenic Ortner's (cardiovocal) syndrome" was a wrong concept as it did not meet the satisfaction of a main element "cardiovascular disease as an underlying cause of hoarseness" of the definition of Ortner's (cardiovocal) syndrome defined by Ortner in 1897. It was actually an immediate vocal cord complication following cardiovascular manipulation.

Conflict of interest: none declared

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