

ORIGINAL ARTICLE

Perioperative factors related to the severity of vocal cord paralysis after thoracic cardiovascular surgery

A retrospective review

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Background Vocal cord paralysis (VCP) is a rare complication of thoracic cardiovascular surgery. In severe cases, life-threatening airway obstruction may occur.

Objective To evaluate the incidence and severity of VCP among patients who underwent thoracic cardiovascular surgery and to identify possible risk factors.

Design Single-centre retrospective review of adult patients.

Setting Osaka University Hospital, Suita, Japan, from January 2013 to August 2015.

Patients We included 688 patients in the final analysis. Preoperative, intraoperative and postoperative data were collected from medical records. Patients with preoperative VCP or tracheostomy prior to extubation were excluded. The VCP severity in relation to functional recovery was graded using the following categories: absent; mild, remission at 6 months; moderate, partial or persistent VCP at 6 months; or severe, airway obstruction after extubation requiring reintubation. An otolaryngologist diagnosed all VCP cases.

Main outcome measures The incidence and severity of VCP after extubation.

Results The incidence (number) of VCP was 4.7% (32), with those of mild, moderate and severe VCP being 1.7% (12), 1.5% (10) and 1.5% (10), respectively. The ICU stay was significantly longer in patients with severe VCP than in patients without VCP [12.5 days (interquartile range 5.5 to 25.5) vs. 3 days (interquartile range 2 to 5), $P=0.0002$]. In our multivariable analysis, type 2 diabetes mellitus [odds ratio (OR) 1.853, $P=0.009$], intubation period (OR per 24 h 1.136, $P=0.014$), ascending aortic arch surgery with brachiocephalic artery reconstruction (OR 8.708, $P<0.001$) and ventricular assist device implantation (OR 3.460, $P=0.005$) were independent predictors for VCP.

Conclusion The identification of these risk factors may facilitate screening for VCP before extubation and possibly help anaesthesia personnel to be prepared to treat VCP-related airway obstruction should it occur.

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Introduction

Vocal cord paralysis (VCP) is a rare but well documented complication of thoracic surgery.^{1–3} Clinical symptoms of VCP include a characteristic breathy voice that is often accompanied by swallowing disabilities, shortness of breath and inefficient throat clearing which may cause aspiration and recurrent pneumonia.¹ The degree of VCP-related obstructive symptoms varies; however, in severe cases, stridor and airway obstruction may occur, resulting in extubation

failure.² Reintubation in the ICU is associated with increased morbidity and mortality, and therefore should be avoided.⁴

Currently, the exact mechanisms of VCP after thoracic cardiovascular surgery are unclear. Recurrent laryngeal nerve (RLN) and laryngeal injuries are probably involved and several patient and surgical-related risk factors have been proposed.^{4–7} Although the relationships among

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such risk factors and the incidence of VCP are clear, their effects on the severity of VCP with regards to airway obstruction and recovery have not been fully examined. Furthermore, the incidence and severity of VCP in high-risk patients undergoing implantable ventricular assist device (VAD) surgery or new, less invasive procedures including endovascular debranching repair are unknown.

Therefore, the aim of the present study was to evaluate the frequency and severity of VCP in patients who underwent several types of thoracic cardiovascular procedures at a single centre to update existing and/or identify new possible risk factors for VCP.

Methods

We performed a retrospective, record-based review of all adult patients who underwent thoracic cardiovascular surgery at our centre between January 2013 and August 2015. A comprehensive database was created from the patients' hospital records. These records included information from the time of surgery to outpatient visits to our hospital up to at least 1 year after surgery.

Ethical approval for this study (Ethical Committee Number 15412) was provided by the institutional ethical review board of Osaka University Hospital, Suita, Japan (Chairperson Professor T. Sobue) on 7 January 2016 to seek permission to publish the results. The necessity for specific informed consent from each patient was waived for this analysis because of the retrospective nature of the study.

Using the records, we examined the following patient and surgical characteristics: sex, age, BMI, preoperative hypertension (yes/no), type 2 diabetes mellitus (yes/no), New York Heart Association functional classification (1 to 4), transthoracic echocardiogram-measured ejection fraction (%), duration of intubation period (h), endotracheal tube (ETT) outer diameter, fixed depth of ETT, experience of the intubation provider (>4 years), surgery duration, internal thoracic artery harvest (right, left, bilateral, number) and deep hypothermic cardiac arrest (yes/no). The outer diameter was measured for both single and double-lumen ETTs. Cases with incomplete data, age less than 18 years, fatal cases and patients with confirmed preoperative VCP or tracheostomy prior to extubation were excluded from our study. In patients who underwent consecutive procedures, only data from the primary surgery were used.

Surgical procedures were classified as follows: ascending aortic arch surgery without brachiocephalic artery (BCA) reconstruction, ascending aortic arch surgery with BCA reconstruction, off-pump coronary artery bypass grafting, on-pump coronary artery bypass grafting, valve replacement, valve replacement with coronary artery bypass grafting, VAD implantation surgery; all other types of surgery were included in the 'other' group. Abdominal

vascular repair and congenital and transcatheter aortic valve replacement procedures were not included in our study.

All patients received anaesthesia by standard methods and the details were left to the attending, board-certified anaesthesiologist's discretion. Tracheal intubation was performed after complete muscle relaxation was achieved. Transoesophageal echocardiography was used in all open-heart surgery cases. An anaesthesiologist performed tracheal intubation with either a videolaryngoscope (McGRATH; Covidien, Dublin, Ireland) or a Macintosh laryngoscope. A standard ETT (TaperGuard Tracheal Tube with Stylet; Covidien) with a high-volume, low-pressure cuff was used. The attending anaesthesiologist selected the size of the ETT, which ranged from 6.5 to 8.5 mm internal diameter for all adult patients. The cuff was inflated until no leakage was present after intubation. Cuff pressure was maintained at 20 to 25 mmH₂O in the ICU with a cuff pressure monitor (Mallinckrodt Pressure Control; Covidien). A double-lumen tube (Mallinckrodt Endobronchial Tube; Covidien) was used for minimally invasive cardiac procedures and was changed to a single-lumen ETT at the end of surgery. Most patients were admitted to the ICU and extubation was performed by intensivists. After extubation, symptoms of dysphonia and dysphagia were sought daily by the ICU personnel until recovery was confirmed. If symptoms persisted, the attending physician was contacted and an otolaryngologist consulted. Following hospital discharge, all patients remained under the care of our university hospital physicians for indefinite periods. A typical follow-up for patients would be scheduled as twice per month for 2 months, then once every 2 months for 4 months, and once every 6 months thereafter. By including outpatient visit medical records in our database, we were able to follow patient recovery after hospital discharge for at least 1 year.

Our primary outcome measure was the severity of VCP. When symptoms for VCP were present, an otolaryngologist was contacted and a laryngeal examination was performed. The severity of VCP was assessed using a grading scale with the following categories: absent, no or slight dysphonia with no sequelae; mild VCP, dysphonia with remission within 6 months that did not interfere with respiration or swallowing; moderate VCP, partial (improvement from paralysis to paresis) or persistent VCP symptoms within 6 months; and severe VCP, airway obstruction after extubation that required reintubation. To exclude other causes of failed extubation such as laryngospasm, we only included cases where both of the following factors could be confirmed; airway obstruction symptoms prior to reintubation and a diagnosis of VCP by an otolaryngologist with fiberoptic laryngoscopy. Secondary outcomes included the duration to full recovery, requirement for tracheostomy, length of stay in the ICU and side of VCP.

Statistical analysis

Demographic and clinical characteristics were compared using the Wilcoxon rank-sum test for continuous variables and the χ^2 or Fisher's exact tests for categorical variables. In our multivariable analysis, estimates of effect were calculated using proportional odds logistic-regression models. This approach fits a uniform log cumulative odds of progression across our four categories of VCP severity as a function of the covariate risk factors and allowed us to compare multiple outcome categories. Based on a review of the literature, the risk factors of ascending arch procedures with BCA reconstruction, VAD implantation surgery, internal thoracic artery harvest, deep hypothermic cardiac arrest, duration of intubation (per 24 h), hypertension, type 2 diabetes mellitus, fixed depth of ETT (per cm), outer diameter of ETT, experience of the intubation provider and age (per decade) were included. The proportional odds assumption for our model was tested and confirmed that it met assumption requirements. Data for intubation periods were analysed with receiver operating characteristic curve and area under curve. A *P* value of less than 0.05 was considered statistically significant. Analyses were performed using Microsoft Excel (version 2010; Microsoft Corporation, Redmond, Washington, USA) and JMP Pro 11 software (SAS Institute, Cary, North Carolina, USA).

Results

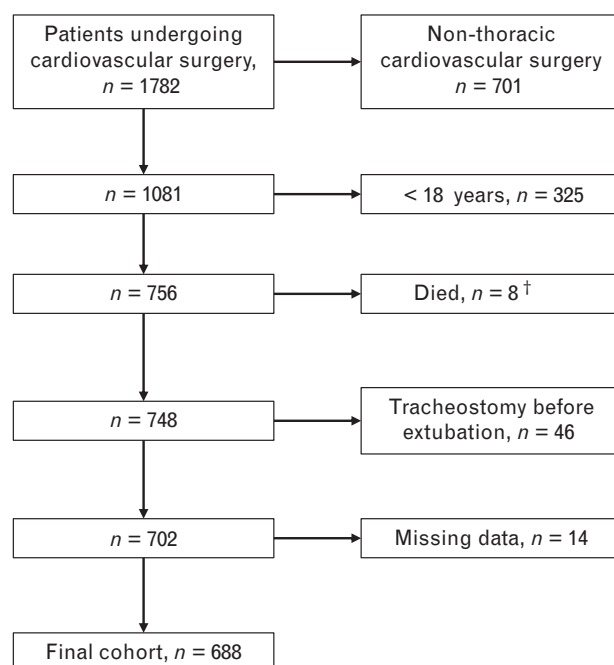
We included a total of 1782 patients during our 2-year study period. After excluding patients who did not meet

our criteria, 688 patients were included in the final analysis (Fig. 1). The patient characteristics, stratified by the severity of VCP, are summarised in Table 1.

The severity and clinical outcomes of VCP are shown in Table 2. VCP occurred in 32 (4.7%) patients, which included 12 mild (1.7%), 10 moderate (1.5%) and 10 severe (1.5%) cases. All 12 patients with mild VCP recovered fully within 6 months and the median recovery time was 9 weeks [interquartile range (IQR) 2.5 to 13.0]. Two patients in the moderate VCP group had full recovery after 36 and 50 weeks. The overall full recovery rate was 43.8% and the overall median recovery time for these patients was 9 weeks (IQR 3.5 to 20.8).

The types of surgery that were related to persistent VCP included VAD implantation (four cases), ascending aortic surgery with BCA reconstruction (two cases) and valve replacement surgery with coronary artery bypass grafting (one case). Of the 10 cases with severe VCP, reintubation was required in all of the patients because of upper airway obstruction after extubation, and seven of these patients later required tracheostomy. The length of ICU stay was significantly longer for patients with severe VCP [12.5 days (IQR 5.5 to 25.5)] than it was for patients without VCP [3 days (IQR 2 to 5), *P* = 0.0002]. Bilateral VCP was observed mainly in patients with severe VCP in whom airway obstruction was present. Unilateral VCP occurred on the right and left sides of the vocal cords throughout the groups (Table 2). The rates of pulmonary complications associated with VCP were aspiration 40.6% (13/

Fig. 1



Flow chart illustrating patient selection process. [†]Vocal cord paralysis was not directly related to cause of death in these cases.

Table 1 Clinical characteristics of the 688 patients and the severity of vocal cord paralysis

	None (n = 656)	Mild (n = 12)	Moderate (n = 10)	Severe (n = 10)
Female, n (%)	242 (36.9)	4 (33.3)	3 (30.0)	3 (30.0)
Age, years	70 (59.0 to 76.0)	67 (56.3 to 77.3)	59 (46.0 to 69.3)	56 (46.0 to 63.0)
BMI, kg m ⁻²	22.2 (20.1 to 24.7)	22.4 (19.7 to 24.2)	22.9 (18.7 to 24.6)	21.4 (18.7 to 24.8)
Hypertension	411 (62.7)	10 (83.3)	6 (60.0)	3 (30.0)
Type 2 diabetes mellitus	242 (36.9)	8 (66.7)	5 (50.0)	6 (60.0)
NYHA ≥3	125 (19.1)	8 (66.7)	6 (60.0)	6 (60.0)
Left ventricular ejection fraction, %	61.0 (47.0 to 69.8)	61 (30.3 to 72.3)	38.5 (12.0 to 68.8)	24.0 (14.5 to 67)
Duration of intubation, h	24.9 (15.1 to 49.5)	39.7 (21.9 to 220.0)	53.1 (22.3 to 269.0)	99.3 (29.6 to 158.2)
Outer diameter of endotracheal tube >10.8 mm	50 (7.6)	3 (25)	–	1 (10.0)
Fixed depth of the endotracheal tube, cm	23.0 (21.0 to 23.0)	22.5 (22.0 to 27.3)	22.5 (21.8 to 24.0)	23.0 (21.8 to 23.0)
Experience of intubation provider <4years	278 (42.4)	6 (50.0)	5 (50.0)	4 (40.0)
Type of surgery				
Ascending aortic arch surgery	38 (5.8)	–	–	–
Ascending aortic arch surgery with BCA reconstruction	44 (6.7)	3 (25.0)	4 (40.0)	2 (20.0)
OPCAB	158 (24.1)	1 (8.3)	–	1 (10.0)
On-CABG	32 (4.9)	1 (8.3)	–	–
VR	229 (34.9)	3 (25.0)	–	1 (10.0)
VR with CABG	66 (10.1)	–	–	1 (10.0)
VAD	37 (5.6)	2 (16.7)	6 (60.0)	5 (50.0)
Other ^a	52 (7.9)	2 (16.7)	–	–
Duration of surgery, h	5.1 (4.2 to 6.3)	5.6 (3.9 to 7.2)	6.9 (5.9 to 8.5)	6.5 (4.4 to 7.1)
ITA harvest	216 (32.9)	2 (16.7)	–	2 (20.0)
Deep hypothermic cardiac arrest	38 (5.8)	1 (8.3)	2 (20.0)	1 (10.0)

Values are presented as median (IQR) or number (%). ^aUncategorised types of surgery were included in this group. BCA, brachiocephalic artery; ITA, internal thoracic artery; NYHA, New York Heart Association functional classification; On-CABG, on-pump coronary artery bypass grafting; OPCAB, off-pump coronary artery bypass grafting; VAD, ventricular assist device surgery; VR, valve replacement.

32) and pneumonia 25% (8/32). The area under curve for intubation periods was 0.66 ($P < 0.01$).

After adjusting for potential confounding factors in our multivariable analysis, type 2 diabetes mellitus [odds ratio (OR) 1.853, $P = 0.009$], intubation period (OR per 24 h 1.136, $P = 0.014$), ascending aortic arch surgery with BCA reconstruction (OR 8.708, $P < 0.001$) and VAD implantation surgery (OR 3.460, $P = 0.005$) remained independent predictors for the severity of VCP (Table 3).

Discussion

In our single-centre retrospective study, we evaluated the frequency and severity of VCP in patients who underwent thoracic cardiovascular surgery to identify any

potential risk factors. We found that VAD surgery, ascending aortic arch surgery with BCA reconstruction, prolonged intubation periods and the presence of type 2 diabetes mellitus were independent risk factors for VCP. Other well established risk factors were not significant in our study.

In the present study, 4.7% of patients who underwent cardiovascular surgery experienced VCP to some degree, which is concerning for several reasons. In severe cases, VCP may cause life-threatening complications such as aspiration, stridor and airway obstruction. Each of these situations may require reintubation, resulting in longer ICU stays and morbidity.^{4–6} Glottic incompetence secondary to VCP impairs swallowing, which can delay oral

Table 2 Severity and outcome measurements of vocal cord paralysis

	Mild (n = 12)	Moderate (n = 10)	Severe (n = 10)
Frequency of VCP (%)	1.7	1.5	1.5
Functional recovery at 6 months			
full	12	2	–
partial (paresis)	–	4	7
no	–	4	3
Median time to full recovery (weeks)	9 (2.5 to 13.0)	43 (36 to 50)	–
Airway obstruction & reintubation	–	–	10
Tracheostomy	1	2	7
Median length of ICU stay (days)	3 (2 to 13.8)	8 (2 to 20.5)	12.5 (5.5 to 25.5) ^a
Side of VCP			
right	5	4	2
left	6	6	3
bilateral	1	–	5

Values are presented as median (IQR) or number. VCP, vocal cord paralysis. ^a $P = 0.0002$ c.f. VCP-absent patients.

Table 3 Results for independent predictors of vocal cord paralysis severity in postcardiovascular surgery patients

	Univariate analysis		Multivariate analysis	
	OR (CI)	P	OR (CI)	P
Female	0.881 (0.589 to 1.275)	0.516	1.048 (0.626 to 1.712)	0.855
Age (per decade)	0.769 (0.630 to 0.952)	0.012	0.821 (0.583 to 1.177)	0.256
BMI	0.961 (0.863 to 1.064)	0.461	0.944 (0.828 to 1.066)	0.359
Hypertension	0.680 (0.471 to 0.970)	0.034	0.709 (0.455 to 1.098)	0.130
Type 2 diabetes mellitus	1.236 (0.861 to 1.766)	0.243	1.853 (1.174 to 2.980)	0.009
Intubation duration (per 24 h)	1.231 (1.119 to 1.350)	<0.001	1.136 (1.025 to 1.275)	0.014
Outer diameter (>10.8 mm)	1.299 (0.697 to 2.122)	0.349	2.435 (0.932 to 5.750)	0.048
Depth (per cm)	1.083 (0.930 to 1.236)	0.272	1.002 (0.773 to 1.320)	0.991
Experience ≥4 years	0.915 (0.641 to 1.313)	0.624	0.933 (0.618 to 1.422)	0.742
Type of surgery				
Ascending aortic arch surgery with BCA reconstruction	2.318 (1.499 to 3.454)	<0.001	8.708 (3.164 to 29.057)	<0.001
OPCAB	0.459 (0.184 to 0.841)	0.034	0.737 (0.164 to 3.658)	0.705
On-CABG	0.785 (0.185 to 1.737)	0.642	1.071 (0.168 to 5.800)	0.938
VR	0.514 (0.279 to 0.827)	0.014	1.103 (0.456 to 3.129)	0.835
VR + CABG	0.540 (0.128 to 1.181)	0.227	0.857 (0.145 to 3.800)	0.847
VAD	3.479 (2.339 to 5.122)	<0.001	3.460 (1.589 to 9.624)	0.005
Other ^a	0.869 (0.347 to 1.611)	0.708	ref	ref
Duration of surgery (per h)	1.321 (1.114 to 1.555)	0.001	1.050 (0.852 to 1.284)	0.645
ITA harvest	0.540 (0.292 to 0.868)	0.023	1.533 (0.433 to 5.278)	0.523
Deep hypothermic cardiac arrest	1.527 (0.816 to 2.513)	0.130	0.591 (0.268 to 1.233)	0.173

Estimates of effect were calculated using proportional odds logistic-regression models. All variables are dichotomous except for age and length of intubation. Intubation period is defined as the time of endotracheal intubation to the time of extubation. Values are expressed as median (IQR). CI, confidence interval; OPCAB, off-pump coronary artery bypass grafting; On-CABG, on-pump coronary artery bypass grafting; OR, odds ratio; VR, valve replacement; BCA, brachiocephalic artery; VAD, ventricular assist device surgery; ITA, internal thoracic artery. ^a This group served as the reference group and included ascending arch procedures without brachiocephalic artery reconstruction and other uncategorised cardiovascular procedures.

feeding and bowel movement recovery.^{7–9} Although not life-threatening, iatrogenic dysphonia greatly affects the patient's quality of life and is susceptible to litigation where the anaesthesiologist is often named as the defendant.¹⁰

Moreover, the true incidence of VCP after cardiovascular surgery is difficult to establish, as laryngeal examinations are not routinely performed. Thus, the incidence varies, with reported values ranging from 0.67 to 23% for all types of thoracic cardiovascular procedures^{1,2,11,12} and from 7.3 to 32.3% for aortic arch surgery;^{3,13} our incidences of 3.9 and 9.9%, respectively, are within these previously reported ranges.

The recovery of patients from VCP after thoracic cardiovascular surgery has not been fully examined in the adult population. Joo *et al.*² reported that the time required for functional recovery was less than 6 months in 10 of the 13 adult patients they studied and that the estimated rate of persistent VCP was 3.5 to 5.3% after a mean follow-up time of 14.8 months. A systematic review of hoarseness after thoracic cardiovascular surgery, which included newborn and paediatric patients, suggested that the rate of persistent hoarseness was 33%.¹⁴ In our study, the overall rates of partial recovery and persistent VCP were 34.4 and 21.8%, respectively. Overall, only 43.8% of the patients had full recovery after follow-up at 1 year. The median time to recovery in these patients was 9 weeks (IQR 3.5 to 20.8). The low rate of full recovery in our cohort may be related to the inclusion of high-risk patients and shorter follow-up periods. We also found a significantly longer ICU stay in patients with severe VCP

compared with patients without VCP. The delayed recovery in patients with severe VCP highlights the importance of evaluating laryngeal function before extubation in high-risk patients.^{5,15,16} Although there were eight cases of mortality in our cohort, they were not directly related to VCP and we did not include these cases in our analysis.

A unique finding of our study is that even after adjusting for confounders such as ejection fraction, New York Heart Association status and intubation period, VAD implantation surgery remained a significant independent risk factor for VCP, which to our knowledge has not been reported previously. Furthermore, when VCP occurred, it often caused severe disabilities requiring tracheostomy. Particularly, four of the seven patients with persistent VCP underwent VAD implantation. The pathological mechanisms for VCP in patients who underwent VAD implantation are unclear but may be the result of several factors related to RLN and laryngeal injuries. Patients who require VAD implantation suffer from low cardiac output with enlarged left atrium and elevated pulmonary artery pressures secondary to high end-diastolic ventricular pressures. Left RLN injury may occur because of compression by such enlarged cardiovascular structures (Ortner's syndrome).^{17,18} Direct manipulation and retraction of the heart during VAD insertion may also cause RLN injuries.¹ Cardiovascular instability during the perioperative period may cause low perfusion and ischaemia in the laryngeal membrane, which can produce oedema and inflammation. The laryngeal membrane may be further

aggravated by physical trauma related to the ETT cuff in the postoperative period.

Previous studies have extensively demonstrated the association between aortic surgery and VCP, showing that the incidence is more frequent with para-aortic procedures and aortic procedures extending to the distal arch.^{12,13,19,20} In our cohort, we also found that the risk of VCP was significantly higher in patients who underwent thoracic aortic surgery with BCA reconstruction. Notably, VCP did not occur in patients who underwent aortic surgery without BCA reconstruction. However, the classifications of ascending aorta and arch or descending aorta used in prior reports may not reflect the likelihood of injury to the RLN owing to the development of less invasive endovascular surgical techniques in recent years; therefore, we chose to classify the aortic procedures according to the presence or absence of BCA reconstruction. Potential mechanisms for VCP in BCA reconstruction cases include surgical manipulation of the left common carotid artery during BCA reconstruction and microembolisation of the afferent arteries after artery clamping.

Our findings are in agreement with previous reports demonstrating that prolonged intubation periods and the presence of type 2 diabetes mellitus are risk factors for VCP.^{6,21–23} In our study, intubation periods longer than 97.9 h were related to the occurrence of VCP with 43.3% sensitivity and 33.4% specificity.

Other previously reported risk factors such as age, sex (female), ETT fixation depth, surgery duration, internal thoracic artery harvest, the use of large-diameter ETTs and deep hypothermic cardiac arrest were not significant in our study.^{1,11,13,24,25} Many of the studies that reported these factors also included noncardiac surgery patients and patients with undiagnosed VCP. As the leading cause of postsurgical VCP is thyroidectomy, these previous reports probably reflect the characteristics of this patient population, including advanced age and female sex.¹⁰

We have described several risk factors found after cardiovascular surgery and our results may help assist intensivists and anaesthetic staff in preparing for VCP after extubation. Nevertheless, detection of VCP and avoiding extubation failure is an essential part of postoperative management. It is important we do not miss the clinical symptoms of VCP even in patients without the risk factors described in our study.

We acknowledge the following potential limitations of our study. First, given that there were only a few cases of VCP in our single-centre study, our results should be interpreted with caution. The inherent biases involved in the retrospective design and data collection are also limitations of our study. Although risk factors for VCP were identified, the pathogenesis of VCP remains unclear and further studies are warranted. During the span of our study period, a videolaryngoscope was often used and the

difficulty and Cormack-Lehane classification could not be properly assessed for many cases. Therefore, we were unable to include these factors in our analysis. Moreover, we did not routinely treat patients with corticosteroids prior to or after extubation. This may have had an effect on the severity of VCP in the high-risk group. Finally, although we actively asked patients for symptoms of VCP after extubation we acknowledge that there was the possibility of missing the occurrence of VCP, especially for mild cases of dysphonia. This may have led to underestimation of the true occurrence rate of VCP.

Conclusion

In this retrospective study of thoracic cardiovascular surgery patients, the potential risk factors of VCP severity were type 2 diabetes mellitus, intubation period, ascending aortic arch surgery with BCA reconstruction and VAD implantation. Identifying such risk factors may allow us to screen for patients who are at risk of developing VCP after thoracic cardiovascular surgery and to assist anaesthesia personnel so that they can be prepared for VCP-related airway obstruction after extubation.

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Conflicts of interest: none.

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