

# Clinical Analysis of Prolonged Mechanical Ventilation >72 h Following Acute Type A Aortic Dissection Repair

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## Abstract

**Background:** Influencing factors for prolonged mechanical ventilation (PMV) following acute type A aortic dissection repair (AADR) were investigated.

**Material and methods:** 325 patients receiving AAADR were enrolled. They were divided into two groups based on the duration of mechanical ventilation; 72 h or less (Group A; n=250) and more than 72 h (Group B; n=75).

**Results:** Preoperative backgrounds showed % of those with COPD, redo operative cases, mal-perfusion to coronary arteries or lower limbs were significantly higher in Group B. Procedure related data revealed that operation time, cardio-pulmonary bypass time, aortic cross clamp time, and postoperative ICU stay were significantly longer in Group B. There were more intraoperative bleeding amounts identified in Group B. % of those complicated with postoperative acute renal failure were significantly higher in Group B. 30-day mortality was significantly higher in Group B. Multivariate analysis demonstrated that COPD, preoperative mal-perfusion to vital organs or lower limbs, operation time were significantly influencing factors of PMV.

**Conclusions:** COPD, mal-perfusion to vital organs or lower limbs, prolonged operation time were influencing factors for PMV. Identifying them could help to establish optimal perioperative management strategies following AAADR.

**Keywords:** Acute type A aortic dissection; Prolonged mechanical ventilation; Malperfusion; Limb ischemia; COPD.

**List of abbreviations:** PMV: Prolonged Mechanical Ventilation; AAAD: Acute Type A Aortic Dissection; AAADR: Acute Type A Aortic Dissection Repair; COPD: Chronic Obstructive Pulmonary Disease; ICU: Intensive Care Unit; CPB: Cardio Pulmonary Bypass; LOS: Low Cardiac Output Syndrome; TAR: Total Arch Replacement; ASCP: Antegrade Selective Cerebral Perfusion; PEEP: Positive End-Expiratory Pressure; OT: Operation Time; ACC: Aortic Cross Clamp; IBA: Intraoperative Bleeding Amount; ARF: Acute Renal Failure; CABG: Coronary Artery Bypass Grafting; PCI: Percutaneous Coronary Intervention

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## Introduction

Acute type A aortic dissection (AAAD) remains one of the most life-threatening cardiovascular disorders with high morbidity and mortality. Past reports regarding clinical outcomes of surgically treated AAAD revealed that in-hospital mortality ranged from 22-58% [1-4]. Under such critical conditions, prolonged mechanical ventilation (PMV) was necessary for some patients after surgery.

Compared to other cardiovascular surgical procedures, patients complicated with AAAD can suffer from hemodynamic instability on admission, mal-perfusion to vital organs, prolonged CPB time which can lead to low cardiac output syndrome (LOS) and uncontrollable bleeding during or after surgery, and neurological disorders [2]. All of these factors can create the need for PMV after AAAD repair (AAADR). In addition, systemic inflammatory response-related lung injuries caused by the acute aortic

dissection itself can accelerate PMV following AAADR [5-7]. Although there are several reports focusing on the contributing factors of PMV after cardiac surgery and the monetary costs on their clinical outcomes [8-17], little evidence is known regarding the contributing factors for PMV following AAADR. The aim of this study was to clarify influencing factors for PMV following AAADR on clinical outcomes.

## Methods

A total of 325 patients who underwent AAADR in our institute between 2009 and 2017 were enrolled. All underwent emergency surgery within 48 hours after admission. The diagnosis of aortic dissection was confirmed by enhanced computed tomography or trans-thoracic echocardiography. Patients were divided into two groups based on the duration of mechanical ventilation: 72 h or less (Group A, n=250) and more than 72 h (Group B, n=75).

### Operative technique

The primary entry tear was routinely resected. In the case where an intimal tear was located at the ascending aorta, replacement of the ascending aorta was performed. Also, when there was an intimal tear in the aortic root or the aortic root was dilated, the aortic root was replaced. If no initial tear was identified at the ascending aorta, Total Arch Replacement (TAR) with the elephant trunk technique was performed. Under median sternotomy in the supine position, CPB was initially established through the right axillary artery and femoral arteries for arterial cannulations and bicaval cannulations for venous drainage. In the case where the right axillary artery was not available due to its dissection, the isolated unilateral femoral artery was cannulated. A left ventricular vent tube was inserted through the right superior pulmonary vein. After bladder temperature decreased to 25°C, the ascending aorta was opened under circulatory arrest. Then, Antegrade Selective Cerebral Perfusion (ASCP) was initiated by clamping the brachiocephalic artery and inserting a 12F balloon tipped cannulas into the left common carotid and left subclavian artery, respectively. Myocardial protection was achieved with retrograde delivery of cold crystalloid solution. The aortic segment, including the intimal tear, was resected. When performing TAR, a sealed 4-branched graft (J graft SHIELD NEO., Japan Lifeline Co. Ltd., Shinagawa, Tokyo, Japan) was used. An elephant trunk graft was inserted into the distal aorta. The distal aortic stump was reinforced with felt strips followed by open distal anastomosis with continuous 4-0 monofilament sutures. Circulation of the lower body was restarted through the side branch of the branched graft after completion of open distal anastomosis. Systemic rewarming was initiated followed by the resection of the proximal aorta at 10 mm distal to the sino-tubular junction and reinforcement of the aortic stump was achieved with a pair of Teflon felt strips, both inside or outside of the aortic wall. Proximal anastomosis was performed with continuous 4-0 monofilament sutures. Finally, the left subclavian artery, the left common carotid artery, and the brachiocephalic artery were reconstructed step-by-step. On the other hand, in performing the replacement of the ascending aorta, a sealed single branched graft (J graft, Japan Lifeline, Tokyo) was used. Distal and proximal anastomosis was performed in a similar fashion as that of TAR under moderate hypothermic circulatory arrest with ASCP.

### Respirator discontinuation protocol

After surgery, mechanical ventilation in volume-control mode on the Servo-I (Maquet Critical Care, Solna, Sweden) at an appropriate inspired oxygen fraction (FiO<sub>2</sub>) was started at the time of being transferred to the Intensive Care Unit (ICU) to keep arterial oxygen saturation (SaO<sub>2</sub>) above 97%. Once hemodynamic stability without excessive bleeding, normothermia, normal acid-base balance, full recovery from anesthesia, and muscle relaxation were obtained, the discontinuation process was initiated in the synchronized intermittent mandatory ventilation plus the pressure support mode. The trachea was extubated when the patient's breathing was comfortable with a tidal volume >6 ml/kg, respiratory rate <25/min, vital capacity >15 ml/kg, arterial oxygen tension (PaO<sub>2</sub>/FiO<sub>2</sub>) >200, and arterial carbon dioxide tension (PaCO<sub>2</sub>) <50 mmHg under positive endo-expiratory pressure (PEEP) of 3-5 cm H<sub>2</sub>O without any signs of excessive pharyngeal and tracheal secretion. If the patient did not meet all of the discontinuation criteria or the extubation criteria, the process was suspended, and appropriate treatments under respirator support were continuously performed.

### Statistical analysis

Continuous data are presented as median and interquartile range. Normally distributed data were analyzed using 2-tailed t-tests, and non-normally distributed data were compared with the Mann-Whitney test, as appropriate. Categorical variables are given as a count and percentage of patients and compared using the  $\chi^2$  test. When any expected frequency was less than 1, or 20% of expected frequencies were less than or equal to 5, Fisher's exact test was used. Clinical outcomes between both groups and the influencing factors of PMV were analyzed by multivariate analysis of variance (MANOVA). A p-value of <0.05 was considered significant. All data were analyzed using the Statistical Analysis Systems software JMP 12.0 (SAS Institute Inc., Cary, NC, USA).

Patients received emergency surgery under the diagnosis of AAAD after written informed consent was obtained from all patients or their families. In proceeding with this study, approval from the Institutional Review Board was granted and for reporting patient information.

## Results

Preoperative backgrounds (**Table 1**), demonstrate the percentages of those with chronic obstructive pulmonary disease (COPD) (13% vs. 3%, p=0.002), redo operative cases (8% vs. 1%, p=0.006), mal-perfusion to heart (11% vs. 1%, p<0.001) or lower limbs (20% vs. 8%, p=0.004) were significantly higher in Group B than in Group A. Procedure related data in **Table 2A** revealed that operation time (OT) (485 ± 140 vs. 387 ± 97 min, p<0.001), CPB time (251 ± 78 vs. 212 ± 57 min, p<0.001), aortic cross clamp (ACC) time (156 ± 52 min vs. 132 ± 36 min, p<0.001), ventilation time (183.9 ± 129.0 h vs. 29.6 ± 22.2 h, p<0.01), and postoperative ICU stay (34 ± 20 vs. 27 ± 16 days, p<0.001) were significantly longer in group B than in group A. There were no significant differences in re-incubation rate within 24 h after extubation

**Table 1** Comparison between both groups in preoperative patient's backgrounds.

	Group A MV ≤ 72 h (n=250)	Group B MV>72 h (n=75)	p value
Age, mean (SD), years	69 ± 13	70 ± 12	0.5
Elderly (aged > 75 years) (%)	99 (40)	32 (43)	0.69
Male gender (%)	114 (46)	40 (75)	0.29
BSA, mean (SD), m2	1.6 ± 0.2	1.6 ± 0.2	0.4
Hypertension (%)	179 (72)	59 (79)	0,24
Hyperlipidemia (%)	61 (24)	23 (31)	0.29
Diabetes (%)	9 (19)	3 (4)	0.43
Past or current smoking (%)	90 (37)	23 (31)	0.41
COPD (%)	8 (3)	10 (13)	0.002
Serum creatinine, mean (SD), mg/dl	1.2 ± 1.7	1.3 ± 0.8	0.64
Chronic hemodialysis (%)	7 (3)	2 (3)	1
History of CVD(%)	24 (10)	7 (9)	1
History of IHD (%)	6 (2.5)	5 (7.5)	0.31
Previous cardiac surgery (%)	3 (1)	6 (8)	0.006
Marfan syndrome (%)	29 (12)	16 (21)	0.037
<b>* Malperfusion status</b>			
Brain	29 (12)	16 (21)	0.037
Heart	3 (1)	8 (11)	<0.001
Spinal cord	1 (0.4)	0 (0)	1
Bowels	7 (3)	5 (7)	0.16
Kidneys	5 (2)	4 (5)	0.22
Lower limbs	19 (8)	15 (20)	0.004

**Table 2A** Comparison between both groups in procedure related data.

	Group A MV ≤ 72h (n=250)	Group B MV>72 h (n=75)	p value
TAR (%)	47 (117/250)	47 (35/75)	1
OP time, mean (SD), min	389 ± 97	485 ± 140	<0.001
CPB time, mean (SD), min	212 ± 57	251 ± 78	<0.001
SCP time, mean (SD), min	71 ± 26	69 ± 35	0.81
ACC time, mean (SD), min	132 ± 36	156 ± 52	<0.001
CA time, mean (SD), min	52 ± 14	54 ± 14	0,26
IBA, mean (SD), ml	2093 ± 1314	3128 ± 1852	<0.001
BT, mean (SD), units	6.3 ± 4.8	8.3 ± 6.2	0.004
Ventilation Time (h)	29.6 ± 22.2	183.9 ± 129.0	<0.01
ICU stay, mean (SD), days	3.5 ± 2.1	10.0 ± 7.0	<0.001
Hospital stay, mean (SD), days	27 ± 16	34 ± 20	0.004
Renal failure (Serum Creatine >2.0mg/dl) (%)	3 (1)	13 (17)	<0.001
CVA (%)	2 (1)	5 (7)	0.008
Re-incubation within 24 h (%)	5 (2)	2 (2.6)	0.87
Tracheostomy (%)	2 (1)	5 (7)	0.008
SSI (%)	0 (0)	0 (0)	1
Operative mortality (%)	12 (5)	16 (19)	<0.001
In-hospital mortality (%)	12 (4.8)	7 (23)	<0.001

(2% vs. 2.6%, p=0.87) between both groups. There was more intraoperative bleeding amount (IBA) (3128 ± 1852 vs. 2093 ± 1314 ml, p<0.001) identified in Group B. The percentage of cases complicated with postoperative acute renal failure (ARF) (17% vs. 1%, p<0.001) were significantly higher in Group B. The reasons for PMV are demonstrated in **Table 2B**. The 30-day mortality was significantly higher in Group B than in

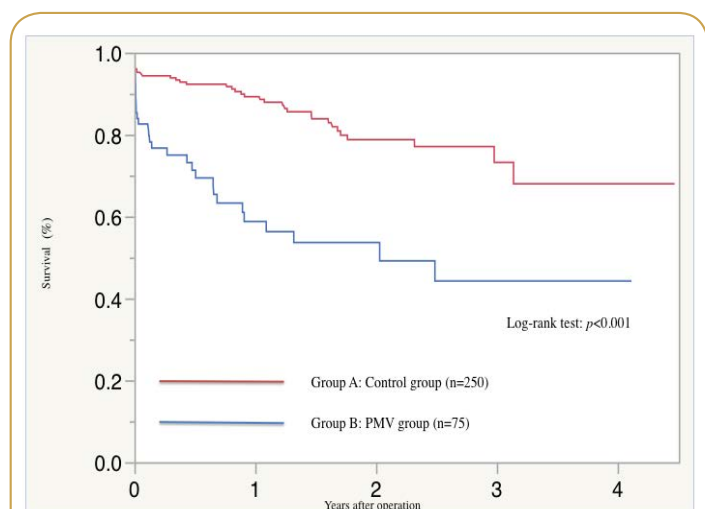
Group A (23% vs. 5%, p<0.001). Multivariate analysis in **Table 3** demonstrates that COPD, preoperative mal-perfusion to vital organs or lower limbs, and operation time were significant influencing factors for PMV. Kaplan-Meier survival curve is shown in **Figure 1**. The 5-year overall survival (44.2% vs. 68%, Log-rank test; p<0.001) was significantly worse in Group B than in Group A in this study.

**Table 2B** The breakdowns of the primary reasons for prolonged mechanical ventilation.

	Group B MV>72 h (n=75)
Acute Kidney Injuries (%)	24 (32)
Respiratory Failure (%)	22 (29)
Cerebro Vascular Accident (%)	24 (32)
Low Cardiac Output Syndrome (%)	3 (4)
Acute Liver Dysfunction (%)	1 (1.3)
Gastrointestinal Perforation (%)	1 (1.3)

**Table 3** The risk factors for prolonged mechanical ventilation on multivariate analysis.

	Odds Ratio	95%CI	p value
<b>COPD</b>	6.2	2.0-19.6	0.004
<b>Malperfusion</b>	2.4	1.3-4.4	0.009
<b>OP time</b>	1.04	1.02-1.08	0.003



**Figure 1** Kaplan-Meier curves for actuarial survival of patients with and without prolonged mechanical ventilation (PMV) after acute type A aortic dissection repair showing that the 5-year overall survival was significantly worse in Group B than in Group A.

## Discussion

There have been several reports of correlations between acute aortic dissection and impaired pulmonary oxygenation [5,18-21]. The previously reported studies mainly referred to patients receiving conservative medical treatments for acute aortic dissection, and exacerbated oxygenation was caused by systemic inflammatory reaction syndrome (SIRS) rather than pulmonary congestion [18-21]. From the viewpoint of pathological findings in the acute dissected aorta, infiltration of macrophages and leukocytes and increased expression of genes related to inflammatory processes, including interleukin-6 and interleukin-8, are frequently found [20,22]. These findings suggested that localized aortic injuries caused by acute aortic dissection have the potential to produce humoral factors that penetrate the pulmonary vasculature and activate both leukocytes and the pulmonary vascular endothelium. Also, Sugano noted that the powerful assault of intimal rupture and the

dissection propagation into the media might cause activation of the cellular and humoral inflammatory systems, which eventually led to oxygenation insufficiency. Kimura proposed that this inflammatory related lung injury might well be a major underlying mechanism of respiratory impairment following AAADR [23].

In the present study, 23% of patients who underwent emergency AAADR depended on PMV for more than 72 h. This percentage was significantly higher than the previously reported incidence of PMV following coronary artery bypass grafting (CABG), ranging from 2.6 to 6.5% [8-17]. Advanced age, female sex, obesity, COPD, smoking, history of stroke, acute kidney injury, preoperative shock, preoperative use of IABP, concomitant valve procedures, redo cardiac surgery, prolonged CPB time and ACC time, emergency surgery, and unstable angina were revealed as risk factors for PMV after adult cardiac surgery in the previously reported articles [8-10,12-17]. On the other hand, the risk factors for PMV for more than 48 h following AAADR, preoperative shock, malperfusion to coronary arteries requiring concomitant CABG and lower limbs, and preoperative renal failure were identified [23]. In the present study, COPD, malperfusion to vital organs, including brain, coronary arteries or lower limbs, longer OT, and IBA were shown as independent risk factors for PMV for more than 72 h after AAADR. In patients complicated with coronary ischemia, preoperative hemodynamic instability is quite common, and concomitant procedures, including percutaneous coronary intervention (PCI) prior to the establishment of CPB or CABG are frequently required. These additional treatments can lead to increased OT as well as CPB time [24,25].

As previously shown in the literature by Kimura [23], clinical analysis in the present study demonstrated that limb ischemia was another risk factor for PMV as well. The ratio of AAADR complicated with limb ischemia was 25-33% and was related to increased morbidity and mortality [26]. Although the relationships between limb ischemia and PMV following AAADR have not clearly been identified yet, rhabdomyolysis and acute kidney failure as a consequence of reperfusion injury from prolonged ischemic time to the lower limbs can have the potential to increase the duration of mechanical ventilation [23]. Therefore, we consider comprehensive treatment strategies to recover from malperfusions to visceral organs and lower limbs as promptly as possible during the AAADR are important in improving in-hospital mortality and long-term outcomes in Group B. In this regard, Hsu reported clinical efficacy of complete attachment (PETTICOAT) technique to facilitate distal aortic remodeling, including increase in diameter of true lumen and decrease in that of false lumen at the time of performing AAADR [27]. In treating those complicated with malperfusions to visceral organs and lower limbs, we consider it is imperative to transfer the patients to the operating room immediately after the definitive diagnosis of those disorders on enhanced CT followed by the establishment of CPB, including antegrade or retrograde arterial perfusions through the true lumen as soon as possible to shorten ischemic time of malperfusion status. We consider the PETTICOAT technique can be applied as an effective endovascular treatment in cases of persistent malperfusion status regardless of central repair, including intimal tear resection and elephant trunk insertion or frozen elephant trunk technique [28].

From the viewpoint of management in the intensive care unit (ICU) after surgery, multidisciplinary treatment approaches, which include cardiovascular surgeons, intensive care specialists, ICU-registered nurses, respiratory physical therapists, pharmacists, clinical engineers, registered nutritionists, and medical technologists are expected to promote inter-professional collaboration among the participating members stated above, which can significantly reduce the duration of PMV following AAADR [29,30].

This investigation has some limitations. First, compared to other studies of PMV after cardiovascular surgery, we concentrated solely on those receiving AAADR, yielding a relatively small number of enrolled cases. So, further investigations involving larger sample sizes are required. Secondly, due to the emergency nature of AAADR, reliable information concerning preoperative cardiac functions on echocardiography was not fully obtained. In some cases, there were no choices but to go directly to the operating room without echocardiographic evaluations because of hemodynamic instability. Thirdly, the presence of COPD was judged on only the patient's medical history and the oral medications prescribed. Therefore, we might have

underestimated the precise number of those suffering from COPD preoperatively.

## Conclusion

In the present study, 23% of those who underwent emergency AAADR depended on PMV for more than 72 h in the present study. PMV was significantly associated with preoperative malperfusion to vital organs or lower limbs, OT, and IBM. Our study revealed that managing these influencing factors perioperatively can contribute to improving surgical and long-term outcomes of AAADR.

## Disclosure Statement

All of the authors have nothing to disclose and also state no conflict of interest in the submission of this manuscript.

## Availability of Data and Materials

The datasets used and/or analyzed during the current study are available from the corresponding author on the reasonable request.

## References

1. Rampoldi V, Trimachi S, Eagle KA, Nienaber CA, Oh JK, et al. (2007) Simple risk models to predict surgical mortality in acute type A aortic dissection: The International Registry of Acute Aortic Dissection Score. *Ann Thorac Surg* 83: 55-61.
2. Chiappini B, Schepens M, Tan E, Dell' Amore A, Morshuis W, et al. (2004) Early and late outcomes of acute type A aortic dissection: analysis of risk factors in 487 consecutive patients. *Eur Heart J* 26: 180-186.
3. Kallenbach K, Ozelze T, Salcher R, Hagl C, Karck M, et al. (2004) Evolving strategies for treatment of acute aortic dissection type A. *Circulation* 110: II243-II249.
4. Centofanti P, Flocco R, Ceresa F, Attisani M, La Torre M, et al. (2006) Is surgery always mandatory for type A aortic dissection? *Ann Thorac Surg* 82: 1658-1664.
5. Hasegawa Y, Ishikawa S, Ohtaki A, Takahashi T, Sato Y, et al. (1999) Impaired lung oxygenation in acute aortic dissection. *J Cardiovasc Surg (Torino)* 40: 191-195.
6. Sugano Y, Anzai T, Yoshikawa T, Satoh T, Iwanaga S, et al. (2005) Serum C-reactive protein elevation predicts poor clinical outcome in patients with distal aortic dissection: Association with the occurrence of oxygenation impairment. *Int J Cardiol* 102: 39-45.
7. Schillinger M, Domanovits H, Bayegan K, Holzenbein T, Grabenwoger M, et al. (2002) C-reactive protein and mortality in patients with acute aortic disease. *Intensive Care Med* 28: 740-745.
8. Cohen AJ, Katz MG, Frankel G, Medalion B, Geva D, et al. (2000) Morbid results of prolonged intubation after coronary artery bypass surgery. *Chest* 118: 1724-1731.
9. Pappalardo F, Franco A, Landoni G, Gardano P, Zangrillo A, et al. (2004) Long-term outcome and quality of life of patients requiring prolonged mechanical ventilation after cardiac surgery. *Eur J Cardiothorac Surg* 25: 548-552.
10. Rajakaruna C, Rogers CA, Angelini G, Ascione R (2005) Risk factors for and economic implications of prolonged ventilation after cardiac surgery. *J Thorac Cardiovasc Surg* 130: 1270-1277.
11. Spivack SD, Shinozaki T, Albertini JJ, Deane R (1996) Preoperative prediction of postoperative respiratory outcome: Coronary artery bypass grafting. *Chest* 109: 1222-1230.
12. Branca P, McGaw P, Light R (2001) Factors associated with prolonged mechanical ventilation following coronary artery bypass surgery. *Chest* 119: 537-546.
13. Yende S, Wunderink R (2002) Causes of prolonged mechanical ventilation after coronary artery bypass surgery. *Chest* 122: 245-252.
14. Dunning J, Au J, Kalkat M, Levine A (2003) A validated rule for predicting patients who require prolonged ventilation post cardiac surgery. *Eur J Cardiothorac Surg* 24: 270-276.
15. Jin R, Grunkemeier GL, Furnary AP, Handy JR Jr (2005) Is obesity a risk factor for mortality in coronary artery bypass surgery? *Circulation* 111: 3359-3365.
16. Reddy SL, Grayson AD, Griffiths EM, Pullan DM, Rashid A (2007) Logistic risk model for prolonged ventilation after adult cardiac surgery. *Ann Thorac Surg* 84: 528-536.
17. Prapas SN, Panagiotopoulos IA, Hamed Abdelsalam A, Kotsis VN, Protogeros DA, et al. (2007) Predictors of prolonged mechanical ventilation following aorta no-touch off-pump coronary artery bypass surgery. *Eur J Cardiothorac Surg* 32: 488-492.
18. Hata M, Sezai A, Niino T, Yoda M, Wakui S, et al. (2007) Prognosis for patients with type B acute aortic dissection: Risk analysis of early death and requirement for elective surgery. *Circ J* 71: 1279-1282.
19. Sakakura K, Kubo N, Ako J, Ikeda N, Funayama H, et al. (2007) Determinants of in-hospital death and rupture in patients with a Stanford B aortic dissection. *Circ J* 71: 1521-1524.
20. Muller BT, Modlich O, Prissack HB, Bojar H, Schipke JD, et al. (2002) Gene expression profiles in the acutely dissected aorta. *Eur J Vasc Endovasc Surg* 24: 356-364.
21. Pararajasingam R, Nicholson ML, Bell PR, Sayers RD (1999) Non-cardiogenic pulmonary edema in vascular surgery *Eur J Vasc Endovasc Surg* 17: 93-105.

22. Ishii T, Asuwa N (2000) Collagen and elastin degradation by matrix metalloproteinases and tissue inhibitors of metalloproteinase in aortic dissection. *Hum Pathol* 31: 640-646.
23. Kimura N, Tanaka M, Kawahito K, Sanui M, Yamaguchi A, et al. (2008) Risk factors for prolonged mechanical ventilation following surgery for acute type A aortic dissection. *Circ J* 72: 1751-1757.
24. Kawahito K, Adachi H, Murata S, Yamaguchi A, Ino T (2003) Coronary malperfusion due to type A aortic dissection: mechanism and surgical management. *Ann Thorac Surg* 2003 76: 1471-1476.
25. Neri E, Toscano T, Papalia U, Frati G, Massetti M, et al. (2001) Proximal aortic dissection with coronary malperfusion: Presentation, management, and outcome. *J Thorac Cardiovasc Surg* 121: 552-560.
26. Fann JI, Sarris GE, Mitchell RS, Shumway NE, Stinson EB, et al. (1990) Treatment of patients with aortic dissection presenting with peripheral vascular complications. *Ann Surg* 212: 705-713.
27. Hsu HL, Chen YY, Huang CY, Huang JH, Chen JS (2016) The provisional extension to induce complete attachment (PETTICOAT) technique to promote distal aortic remodeling in repair of acute DeBakey type 1 aortic dissection: preliminary results. *Eur J Cardiothorac Surg* 50: 146-152.
28. Di Bartolomeo R, Pantaleo A, Berretta P, Murana G, Castrovinci S, et al. (2015) Frozen elephant trunk surgery in acute aortic dissection. *J Thorac Cardiovasc Surg* 149: S105-109.
29. Cox CE, White DB, Hough CL, Jones DM, Kahn JM, et al. (2019) Effects of personalized web-based decision aid for surrogates decision makers of patients with prolonged mechanical ventilation: A randomized clinical trial. *Ann Intern Med* 170: 285-297.
30. Black CJ, Kuper M, Bellingan GJ, Baston S, Matejowsky C, et al. (2012) A multidisciplinary team approach to weaning from prolonged mechanical ventilation. *Br J Hosp Med (Lond)* 73: 462-466.