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Impairment of chest wall mechanics and increased chest wall work of breathing cause postoperative respiratory failure in patients who have undergone radical esophagectomy

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Abstract
Purpose. We verified the hypothesis that impairment of chest wall mechanics would be related to the cause of postoperative respiratory failure in patients undergoing radical esophagectomy.
Methods. A total of 21 patients were studied. After management with mechanical ventilation to prevent respiratory failure for several days, trial weaning from the ventilator was performed. The patients were divided into a successful weaning group (S group) and an unsuccessful weaning group (US group), depending on the results of the weaning trial. We compared respiratory mechanics and the respiratory work of breathing during weaning from the ventilator between the two groups.
Results. In the US group, lung and chest wall compliance was significantly lower and the development of intrinsic positive end-expiratory pressure (PEEPi) was observed. The work of breathing and the oxygen cost of breathing were significantly higher in the US group. The increased respiratory energy work was due to a moderate increase in lung work and a significant increase in chest wall work.
Conclusion. Our results suggested that postoperative respiratory failure was related to increased respiratory energy expenditure, significantly deteriorated chest wall mechanics, and increased chest wall work, all of which are involved in the development of postoperative respiratory failure after radical esophagectomy.

Key words Chest wall mechanics · Esophageal cancer · Postoperative respiratory failure · Work of breathing

Introduction

Postoperative respiratory failure and other pulmonary complications, such as atelectasis and severe hypoxemia, occur frequently in patients who have undergone surgery for esophageal cancer. The surgery includes esophagectomy, anterior-mediasternal procedures, and upper abdominal surgery for reconstruction of the esophagus with a stomach tube, with extensive radical resection of the lymph nodes. The surgical procedures involve the abdominal to the thoracic cavity and the abdominal and thoracic wall, including the diaphragm, which all constitute structural components of the chest wall. Chest wall mechanics and diaphragmatic function are keys to respiratory function, along with lung mechanics. Thus, invasive surgical procedures could seriously impair chest wall respiratory function. The objective of this study was to verify the hypothesis that impairment of chest wall mechanics would be related to the cause of postoperative respiratory failure in patients who have undergone radical esophagectomy.

Patients and methods

Twenty-one consecutive patients who had undergone surgery for esophageal cancer were studied. The institutional ethics committee approved the investigative protocol, and informed consent was obtained from each patient. All of the patients underwent subtotal esophagectomy, retrosternal reconstruction of the esophagus, and extensive lymph node resection. All patients were managed with mechanical ventilation postoperatively in the intensive care unit (ICU) to prevent acute respiratory failure and postoperative pulmonary complications.

Apparatus

At the end of surgery, a thin polyethylene air-filled balloon catheter (length 95 mm, diamenter 9 mm, No. 700-3-100, Bicore, Irvine, CA, USA) was directly inserted into the pleural cavity at the intersection of the

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seventh intercostal and median axillary line, and advanced cephalad about 20 cm to measure intrapleural pressure. The balloon was positioned at the point of closest agreement between inspiratory fluctuations in the intrathoracic and airway pressures against airway occlusion [1]. A minimal volume of air (approximately 0.2 ml) was used to inflate the balloon to avoid geometric changes in the pleural surface. The appropriate placement of the balloon was later checked in the ICU by chest X-ray. The catheter was placed for several days until weaning from mechanical ventilation was successfully accomplished.

A flow-calibrated, heated pneumotachograph (ATD 145, Type AS, Minato Medical Science, Osaka, Japan) was placed between the endotracheal tube and the Y-piece of the ventilator circuit to measure respiratory flow. Integrated inspiratory volume was checked with a 1.5-l piston cylinder before measurement. Gas samples were continuously taken through a sampling tube with a port positioned at the connection of the endotracheal tube and the Y-piece. The oxygen and carbon dioxide analyzer (Medical gas analyzer, MG-360, Minato Medical Science) was calibrated before measurement with air and a gas mixture of 5% CO_2, 40% O_2 in N_2 balance. The pressure, flow, oxygen concentration, and carbon dioxide concentration were measured with a sampling time of 30 ms and processed breath-by-breath with a microcomputer system (Metabolic and Respiratory Monitoring System, RM-300, Minato Medical Science). Tidal volume (V_T), minute volume (MV), respiratory rate (RR), airway resistance (R), dynamic compliance, respiratory work, oxygen consumption, and carbon dioxide production were measured for 20 min, and the mean values were obtained. The pressure time product of intrathoracic pressure (PTP), $P_{0.1}$, and auto-positive end-expiratory pressure (PEEPi) were measured with a respiratory monitor (Bicore CP-100), and averages of 40 breaths were obtained.

Protocol

The patients were placed on controlled mechanical ventilation with the ventilator set at TV 10 ml·kg^{-1}, PEEP 5 cmH_2O, and the respiratory rate was adjusted to achieve $PaCO_2$ of 35–40 mmHg. The inspiratory flow rate was set at 60 l·min^{-1}, and the inspiratory pause was set at 0.2 s. Mechanical ventilation and respiratory management were continued for several days to prevent respiratory failure or respiratory complications. Weaning a patient from mechanical ventilation was decided at the ICU morning conference in which the staff discussed the patient's general condition, hemodynamic stability, chest X-ray findings, a PaO_2/FIO_2 ratio of more than 250, forced vital capacity over 10 ml·kg^{-1}, restoration of effective cough reflex, level of consciousness, and fluid balance. Data on respiratory mechanics used in this study were not considered in determining the initiation of weaning. Weaning was defined as the process of transition from controlled mechanical ventilation to spontaneous ventilation. Weaning was considered successful when the patient was able to breath spontaneously on 5 cmH_2O continuous positive airway pressure (CPAP) overnight without any signs to abort weaning from mechanical ventilation. We divided the patients into two groups depending on the results of the weaning trial: patients who were successfully weaned from mechanical ventilation (S group) and patients who were not (US group).

The synchronized intermittent mandatory ventilation mode (SIMV) with demand flow type was used for weaning, in which we decreased the rate of mandatory ventilation in four steps (0%, 25%, 50%, 75%, and 100% weaning). For example, the SIMV rate was adjusted to 12, 9, 6, 3, and 0 breaths·min^{-1} in a stepwise fashion. The IMV rate was held for 2 h at each step, and arterial blood gas analysis was performed. Measurements were taken during the last 20 min of each step until CPAP was achieved in successful cases, or until weaning was aborted in unsuccessful cases. The criteria for abortion of weaning from the ventilator were the following: the patient complained of dyspnea when the nurse or doctor asked him about respiratory conditions, or the patient had tachypnea ($\geq$40 breaths·min^{-1}) on respiratory monitoring, restlessness, marked perspiration, PaO_2 $\leq$80 mmHg, $PaCO_2$ $\geq$50 mmHg or $\leq$30 mmHg on arterial blood gas analysis, tachycardia ($\geq$120 bpm) and/or arrhythmia on electrocardiographic monitoring, or systolic pressure $\geq$180 mmHg on direct arterial pressure monitoring. Final measurements of the study parameters were taken for comparison. The study parameters were measured while the patient was receiving partially assisted ventilation for cases in which weaning failed (in some cases under spontaneous ventilation), and during spontaneous breathing for cases in which weaning succeeded.

Measurement of compliance

In all patients, total compliance (C_T), lung compliance (C_L), and chest wall compliance (C_{CW}) were measured during controlled mechanical ventilation by the zero-flow method [2]. To ensure the accuracy of the measurements of compliance and the contribution of respiratory muscle tone to compliance, C_T, C_L, and C_{CW} were measured both while the patient was awake with muscles relaxed and while the patient was anesthetized with buprenorphine 0.4 mg, diazepam 10 mg, and thiopental 100 mg, and with muscle relaxation with vecuronium 8 mg. The results were compared in seven patients who were selected at random from the latter half of the series.

Calculation of respiratory work

Respiratory work applied to the patient by the ventilator during assisted ventilation (Wassist). Wassist was calculated as joule·min^{-1} by the integral of airway pressure across the tidal volume during the inspiratory positive phase of airway pressure in the pressure-volume loop (Fig. 1).

Patient respiratory work of breathing (WOBT). Patient respiratory work was calculated using the method described by Marini et al. based on Campbell's diagram [3], where WOBT is the sum of the lung respiratory work (WOBL) and chest wall respiratory work (WOBCW). WOBL was determined by integrating the pleural pressure across tidal volume, which is the negative pressure portion of the pleural pressure-volume curve from the initiation to the termination of inspiration. This work contains minimal imposed work load (less than 0.2 joule·min^{-1} in our measurements) for ventilator circuit. WOBCW was calculated as $TV^2 \cdot (2Ccw)^{-1}$ on spontaneous ventilation. During assisted ventilation, WOBCW was derived as follows: first, the chest wall compliance pressure-volume line and the actual pressure-volume loop were overlaid on the pressure-volume diagram (Fig. 1); then the integral of pleural pressure across tidal volume during the positive pleural pressure phase was used to determine the work done by the ventilator to inflate the chest wall, and the remaining triangular area indicates the work done by the patient's effort to expand the chest wall.

Since respiratory work varied breath-by-breath during the IMV mode, WOBT was calculated for each breath, and the mean of a 20-min measurement was determined for respiratory work and expressed as joule·min^{-1}.

Calculation of other parameters

The inspiratory airway resistance (R) was measured during constant flow (60 l·min^{-1}) controlled mechanical ventilation. The calculation was as follows:

$$R = (\text{peak airway pressure} - \text{PEEP} - V_T/C_T)/\text{inspiratory flow}$$

Total oxygen consumption (Vo_2) and carbon dioxide production (Vco_2) were determined by the breath-by-breath method with the conventionally used formulas. The oxygen cost of breathing (OCB) was defined as a percentage of the difference between Vo_2 measured during assisted or spontaneous breathing and Vo_2 during controlled mechanical ventilation in the Vo_2 during controlled mechanical ventilation [4].

PTP was calculated as an integral of the area defined by pleural pressure over time from the onset of patient effort to the end of inspiratory flow. Respiratory drive was assessed with esophageal pressure change during the first 100 ms of the inspiratory phase ($P_{0.1}$). This change was obtained with the use of time delay occurring when the occluded demand valve of the ventilator opened. This pressure ($P_{0.1}$) was analogous to a $P_{0.1}$,

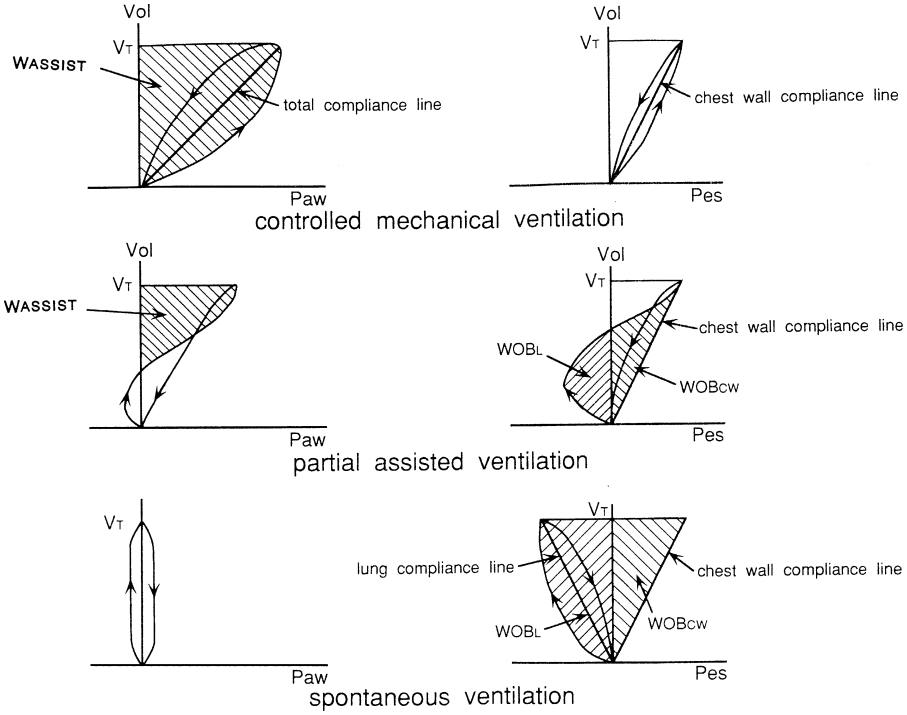

Fig. 1. Schema for calculation of respiratory work. *WASSIST* Respiratory work applied to the patients by ventilator during assisted ventilation; *WOBT*, total patient work of breathing; *WOBL*, patient lung work of breathing; *WOBCW*, patient chest wall work of breathing. *Shaded* areas in pleural pressure-volume diagram were WOBT in Campbell's diagram

mouth occlusion pressure used in the laboratory setting [5].

Auto-PEEP (PEEPi) was measured by the pleural pressure deflection method; the inspiratory pressure drop in pleural pressure that must occur in the pleural space to overcome auto-PEEP and initiate flow from a demand system was taken to equal auto-PEEP [6].

Statistical analysis

Values are shown as means ± standard deviation (SD). Analysis of variance for single measures and the Newman-Keuls test were applied for the comparison between the groups. A *P*-value less than 0.05 was considered significant.

Results

Weaning was successfully accomplished on postoperative day two to six in 14 patients, but weaning from mechanical ventilation failed in 7 patients. Among these patients, three complained of dyspnea, two showed marked perspiration and restlessness, and two had marked hypertension. Ventilator settings were returned to the earlier weaning steps or controlled mechanical ventilation in these patients.

Comparison of respiratory mechanics (Table 2)

C_T, C_L, C_{CW} in the US group was significantly lower than in the S group. There was no difference in R between the groups. PEEPi in the US group was significantly increased compared with that in the S group.

Comparison of patient's work of breathing, OCB, PTP, and metabolism

For the US group, WOB_T was 11.7 ± 4.9 joule·min^{-1}, which was significantly greater than that in the S group (6.7 ± 2.4 joule·min^{-1}). For further analysis, WOB_T was divided into WOB_L and WOB_{CW}. For the US group, WOB_{CW} was 5.1 ± 2.6 joule·min^{-1}, which was significantly greater than that in the S group (2.6 ± 1.2 joule·min^{-1}). There was no significant difference in WOB_L between the groups (Table 3 and Fig. 2). In the US group, OCB, PTP, and $P_{0.1}$ were significantly increased in comparison to the values in the S group (Table 3). There were no differences in V_{O_2} and V_{CO_2} between the groups before weaning, but after the weaning trial, V_{O_2} and V_{CO_2} were significantly increased in the US group compared with the S group (Table 3).

Table 2. Comparison of respiratory mechanics between patients who were successfully and unsuccessfully weaned from mechanical ventilation

Measurement	S group (n = 14)	US group (n = 7)
C_T (ml·cmH_2O^{-1})	51 ± 12	33 ± 8*
C_L (ml·cmH_2O^{-1})	140 ± 55	86 ± 21*
C_{CW} (ml·cmH_2O^{-1})	95 ± 25	53 ± 12*
R (cmH_2O·l^{-1}·s^{-1})	4.4 ± 2.3	4.7 ± 2.0
PEEPi (cmH_2O)	0.3 ± 0.3	2.8 ± 1.8*

S group, Patients who were successfully weaned from mechanical ventilations; US group, patients who were not successfully weaned from mechanical ventilation; C_T, C_L, C_{CW}, total, lung, chest wall dynamic compliance; R, respiratory resistance; PEEPi, auto PEEP. Data were obtained under controlled mechanical ventilation. PEEPi was measured under mechanically assisted ventilation or spontaneous ventilation. *Significant difference from S group ($P < 0.05$)

Table 1. Patient demography

Feature	S group (n = 14)	US group (n = 7)
Age (yr)	60 ± 12	60 ± 10
Sex	All male	All male
Height (cm)	160 ± 7	159 ± 4
Weight (kg)	52 ± 7	50 ± 11
Serum albumin (g·dl^{-1})	3.5 ± 0.4	3.3 ± 0.5
Preoperative %VC (%)	116 ± 17	97 ± 18
Preoperative $FEV_{1.0\%}$ (%)	101 ± 11	104 ± 25
Preoperative P_{O_2} (room air) (mmHg)	93 ± 16	82 ± 18
Operation time (min)	577 ± 70	628 ± 169
Blood loss during operation (ml)	894 ± 347	1282 ± 627
Fluid balance during operation (ml)	+3860 ± 680	+4510 ± 1533
VC before weaning (l)	1.0 ± 0.2	0.8 ± 0.3
MIP before weaning (cmH_2O)	37 ± 14	28 ± 10
P_{O_2}/F_{IO_2} ratio before weaning	327 ± 76	294 ± 74

S group, Patients who were successfully weaned from mechanical ventilation; US group, patients who were not successfully weaned from mechanical ventilation; VC, vital capacity; $FEV_{1.0}$, forced expiratory volume first 1 s; %VC, percent of VC measured in VC predicted; $FEV_{1.0\%}$, percent of $FEV_{1.0}$ measured in $FEV_{1.0}$ predicted; MIP, maximum inspiratory pressure; P_{O_2}/F_{IO_2} ratio, values of PO_2 in mmHg divided by F_{IO_2}. There were no significant differences between the groups

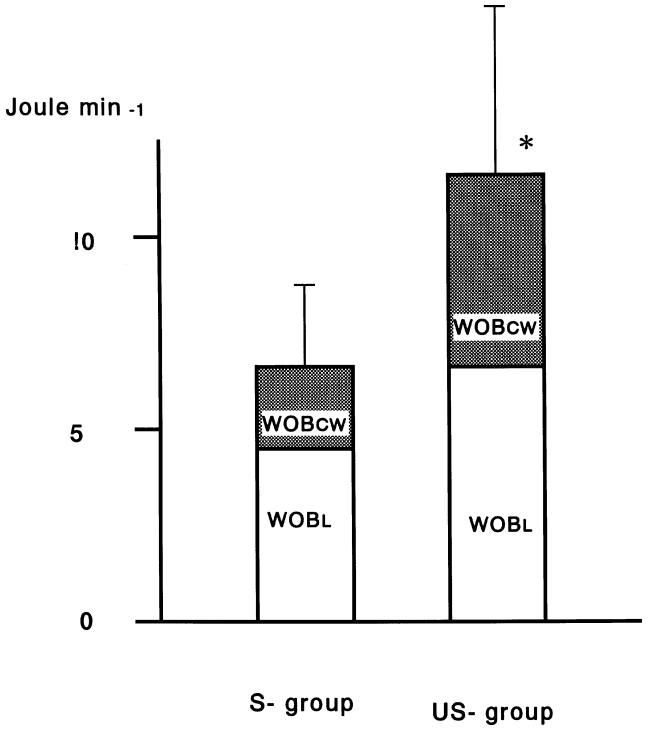

Fig. 2. Comparison of total work of breathing, lung, and chest wall work in patients successfully (S group) and unsuccessfully (US group) weaned from ventilator. Total work of breathing in US group was significantly increased. The causes of increased respiratory work were moderate increase of lung work and significant increase of chest wall work. *WOBL*, Lung work of breathing; WOBcw, chest wall work of breathing. *Significant difference from S group ($P < 0.05$)

Comparison of respiratory work in the same weaning steps

Wassist of both groups decreased in a stepwise fashion during weaning from mechanical ventilation. Wassist in the US group exhibited high values at the same weaning levels, but there were no significant differences between the groups. WOBT of the US group was significantly higher than that of the S group at 50% weaning. WOBcw of the US group was significantly higher than that of the S group at 25% and 50% weaning (Fig. 3).

Comparison of compliance during waking, anesthesia, and complete muscle relaxation

The differences in CT (31 ± 12, 33 ± 8, and 34 ± 7 ml cmH_2O^{-1}), CL (93 ± 46, 81 ± 35, and 83 ± 62 ml cmH_2O^{-1}), and Ccw (54 ± 23, 50 ± 15, and 50 ± 10 ml cmH_2O^{-1}) while the patient was awake with muscles relaxed, anesthetized, and anesthetized with muscle paralysis were not significant in seven patients.

Table 3. Comparison of work of breathing and other parameters for respiratory muscle energy demand between patients who were successfully and unsuccessfully weaning from mechanical ventilation

Measurement	S group ($n = 14$)	US group ($n = 7$)
IMV rate (min^{-1})	0	6.0 ± 3.4
Wassist (joule·min^{-1})	0	3.3 ± 2.5
WOBT (joule·min^{-1})	6.7 ± 2.4	11.7 ± 4.9*
WOBL (joule·min^{-1})	4.3 ± 1.7	6.7 ± 3.0
WOBcw (joule·min^{-1})	2.6 ± 1.2	5.1 ± 2.6*
OCB (%)	−11.2 ± 10.2	18.3 ± 16.9*
PTP (cmH_2O·s·min^{-1})	152 ± 44	241 ± 69*
$P_{0.1}$ (cmH_2O)	2.9 ± 1.0	4.9 ± 1.5*
VT (ml)	499 ± 94	458 ± 81
RR (min^{-1})	18 ± 4	26 ± 7*
MV (l·min^{-1})	8.6 ± 1.3	11.4 ± 2.8
V_{O_2} CMV (ml·min^{-1})	208 ± 51	302 ± 72*
V_{O_2} after weaning (ml·min^{-1})	241 ± 46	241 ± 66
V_{CO_2} CMV (ml·min^{-1})	194 ± 34	211 ± 37
V_{CO_2} after weaning (ml·min^{-1})	195 ± 45	271 ± 37*

S group, Patients who were successfully weaned from mechanical ventilation; US group, patients who were not successfully weaned from mechanical ventilation; IMV, intermittent mandatory ventilation; Wassist, respiratory work applied to patients by ventilator during assisted ventilation; WOBT, patient's total work of breathing; WOBL, patient's lung work of breathing; WOBcw, patient's chest wall work of breathing; OCB, oxygen cost of breathing; PTP, pressure time product of pleural pressure; $P_{0.1}$, inspiratory pleural pressure change in the first 100 ms; VT, tidal volume; RR, respiratory rate; MV, minute volume. Data were obtained under mechanically assisted ventilation in the US group and under spontaneous respiration in the S group. V_{O_2} CMV, Oxygen consumption before weaning; V_{O_2} after weaning, oxygen consumption after weaning; V_{CO_2} CMV, carbon dioxide production before weaning; V_{CO_2} after weaning, carbon dioxide production after weaning. *Significant difference from the S group ($P < 0.05$)

Discussion

We found that in patients with respiratory failure requiring mechanical ventilation, WOBT, OCB, and PTP increased significantly, suggesting that respiratory muscle energy expenditure increased after radical esophagectomy. The increased respiratory energy work was caused by a moderate increase in lung work and a significant increase in chest wall work, which were caused by impairment of respiratory mechanics. Postoperative respiratory failure was related to significantly increased chest wall work, which was caused by significant deterioration of chest wall mechanics.

Estimation of the average global intrapleural pressure changes is essential for measuring respiratory mechanics and the respiratory work of breathing. The esophageal balloon technique is commonly used and is acceptable for estimation of intrapleural pressure with careful placement of the balloon in esophagus with the occlusion test [1,7]. Measurement of pleural pressure

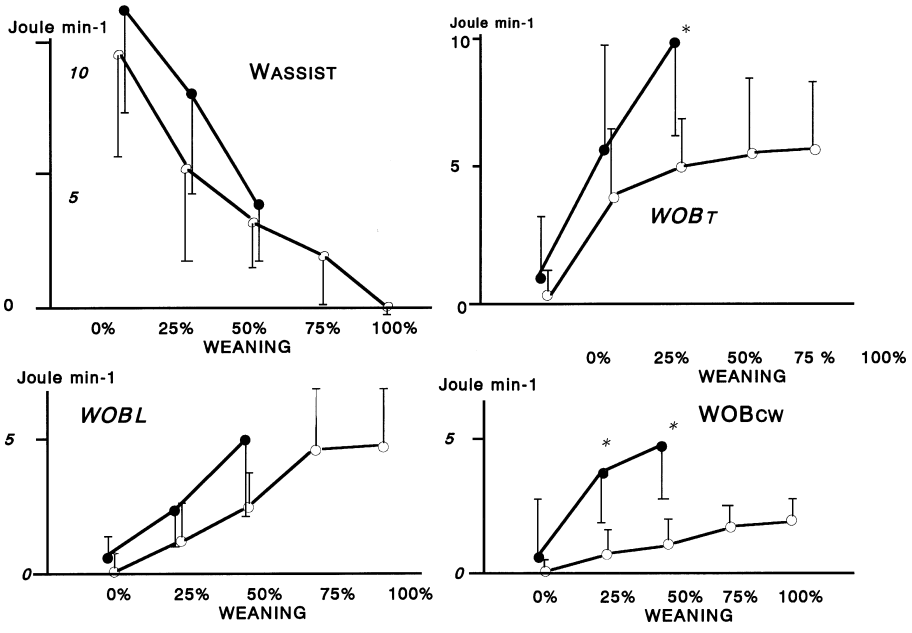

Fig. 3. Comparison of respiratory work during weaning from ventilator. WASSIST decreased in a stepwise fashion during weaning from ventilator in both groups. WOBT and WOBCW in US group increased significantly at 50% weaning compared with S group. *Wassist*, Respiratory work applied to the patients by ventilator during assisted ventilation; *WOBT*, total patient work of breathing; *WOBL*, patient lung work of breathing; *WOBCW*, patient chest wall work of breathing

with this technique does not reveal the absolute pleural pressure, because the mediastinal contents weigh on the lower esophagus. Although there were inaccuracies in measurement of absolute pleural pressure, fluctuational changes in esophageal pressure are acceptable [8]. The direct measurement technique also does not reveal global pleural pressure, because local pleural pressures are influenced by hydrostatics and geometric change around the balloon. D'Angelo et al. reported that tidal changes in local pleural pressure at different sites were similar to change in esophageal pressure during spontaneous breathing. The deformation of the potential pleural space by insertion of a balloon catheter into the pleural cavity also influences the absolute value of pleural pressure. However, such influences are negligible on tidal fluctuation of pleural pressure [9]. When a minimal balloon catheter is used to separate the lung and chest wall, uniformity, fidelity, and accuracy are restored [8].

Our study demonstrated that chest wall mechanics significantly deteriorate after radical esophagectomy. Few detailed studies on changes in chest wall mechanics associated with upper abdominal surgery have been done. Katz et al. reported that Ccw decreased after major abdominal surgery, such as surgery for abdominal aortic aneurysm, which caused postoperative respiratory failure. They suggested that the deteriorated chest wall mechanics were due to abdominal distension, chest wall edema, or pleural effusion [10]. We speculate that in our patients the causes of deteriorated chest wall mechanics may be marked endema of the chest wall, including both the abdomen and the thoracic wall, abdominal distension (ascites, organ edema, intestinal fluid, and gas retention), and pleural effusion resulting from esophagectomy and radical resection of lymph nodes. These changes were all due to the physiological reactions to surgical stress. The facts that the US group had longer operation time and much blood loss, or positive fluid balance, supported the speculation.

Mutoh et al. investigated the effects of a large intravenous volume infusion on respiratory function in pigs and reported that large quantities of intravenous infusion, resulting in edema of the abdominal organs, caused abdominal distension, markedly altering chest wall mechanics and markedly decreasing the functional residual capacity (FRC), although lung mechanics were not significantly affected. They concluded that decreased FRC was caused by the deterioration of chest wall mechanics. They also pointed out that this situation might occur after abdominal surgery [11]. These changes in chest wall mechanics were thought to be functional and to recover during the postoperative periods.

Another possibility was structural changes in the thoracic cavity caused by the surgical procedure. The esophagus was removed and reconstructed with a tubed stomach with a large quantity of peritoneum in the mediastinal space. Such a space-occupying mass can affect chest wall mechanics and the movement of the thoracic wall. The thoracic cage itself also may become stiff because of extensive surgical scarring, fixation of costal structures, and tissue fibrosis. These changes are considered permanent. Maeda reported long-term postoperative evaluation of respiratory function in 50 cases of esophageal cancer. In this report, significant restric-

tive respiratory dysfunction was observed after three months postoperatively; vital capacity was significantly decreased from a preoperative value of 2.1 ± 0.4 l/m^2 to 1.6 ± 0.3 l/m^2. No significant change was observed in FEV 1.0, and shallow quick respiration was observed on the ergometric loading test [12]. This report suggested that impairment of chest wall mechanics persists over the long term. The third possibility is the alteration of muscle tension in the abdominal and thoracic wall or the diaphragm. Pansard et al. reported that decreased abdominal compliance after upper abdominal surgery was related to abdominal muscle activities [13]. The hypothesis that increased tone of the abdominal muscles and diaphragm causes the decrease in Ccw is not supported by our finding that Ccw did not change after the administration of muscle relaxants. We believe that the decrease in Ccw was caused not by changes in the muscle tone but by the persistent structural changes and/or transient physiologic changes in the chest wall.

Deterioration of lung and chest wall mechanics increases respiratory workload, leading to respiratory muscle fatigue, and develops into so-called respiratory pump failure. In our study, parameters of respiratory muscle energy consumption, such as WOBt, OCB, and PTP, increased in all cases in which weaning from the ventilator failed, although the patient received high ventilator-assisted work of 3.3 to 6.1 joule·min^{-1}. In a study using a phrenic electromyogram, Brochard et al. reported that diaphragmatic fatigue occurred when the respiratory work of breathing exceeded 8 to 10 joule·min^{-1} [14]. In our study, the mean total respiratory work was 11.7 joule·min^{-1} for patients in the US group, even though the patients received mechanically assisted ventilation. However, the mean total respiratory work for the S group did not exceed 8 joule·min^{-1} even during spontaneous ventilation. These results suggest that weaning had to be aborted when respiratory work exceeded the diaphragmatic fatigue threshold.

Alie et al. reported that a marked decrease in vital capacity and FRC occurred as a result of severe restrictive pulmonary dysfunction after upper abdominal surgery and suggested that increased lung water, peripheral airway occlusion, elevation of the diaphragm due to abdominal distension, and decrease in lung and/or chest wall compliance were possible causes of the decrease in FRC [15]. Mankikian et al. hypothesized that the two main causes of restrictive respiratory dysfunction after upper abdominal surgery were alterations in chest wall mechanics or diaphragmatic dysfunction, both of which lead to deterioration of the functions that constitute respiratory movement [16]. Thus, deteriorated chest wall mechanics might be involved in decreased FRC after upper abdominal surgery.

In conclusion, after radical esophagectomy, a significant decrease in Ccw occurred, suggesting that chest wall mechanics had significantly deteriorated. Our results suggest that impairment of chest wall mechanics and increased chest wall workload are involved in the development of postoperative respiratory failure after surgery for esophageal cancer.

References

1. Baydur A, Behrakis PK, Zin WA, Jaeger M, Milic-Emili J (1981) A simple method for assessing the validity of the esophageal balloon technique. Am Rev Respir Dis 126:788–791
2. Nunn JF (1977) Principle of measurement of compliance. In: Applied respiratory physiology, 2nd edn. Butterworths, London, pp 91–93
3. Marini JJ, Smith TC, Lamb VJ (1988) External work output and force generation during synchronized intermittent mechanical ventilation. Am Rev Respir Dis 138:1169–1179
4. Annat J, Viale JP, Dereymez CP, Bouffard YM, Delafosse BX, Motin JP (1990) Oxygen cost of breathing and diaphragmeatic pressure-time index. Chest 98:411–414
5. Marini JJ, Rodriguez M, Lamb V (1986) The inspiratory workload of patient-initiated mechanical ventilation. Am Rev Respir Dis 134:902–909
6. Macintyre NL (1991) Intrinsic positive end-expiratory pressure. Prob Respir Care 4:44–51
7. Milic-Emili L, Mead J, Turner JM, Glauser EM (1963) Improved technique for estimating pleural pressure from esophageal balloon. J Appl Physiol 19:207–211
8. Marini JJ (1990) Bedside lung mechanics measurement. Respir Care 35:669–691
9. D'Angelo E, Sant'Ambrogio G, Agostoni E (1979) Effect of diaphragm of pleural pressure. J Appl Phsiol 37:311–315
10. Katz JA, Zinn SE, Ozanne GM, Fairley HB (1981) Pulmonary, chest wall, and lung-thorax elastance in acute respiratory failure. Chest 80:304–311
11. Mutoh T, Lamm WJE, Embree LJ, Hildebrandt J, Albert RK (1992) Volume infusion produces abdominal distension, lung compression, and chest wall stiffening in pigs. J Appl Physiol 72:575–582
12. Maeda F, Pulmonary function during exercise before and after esophagectomy for esophageal cancer (1997) Nippon-Kyobu-Geka-Gakkai-Zasshi 45:1–11
13. Pansard JL, Makikian B, Bertrand M (1993) Effects of thoracic extradural block on diaphragmentic electrical activity and contractility after upper abdominal surgery. Anesthesiology 78:63–71
14. Brochard L, Harf A, Lorino H, Lemaire F (1989) Inspiratory pressure support prevents diaphragmatic fatigue during weaning from mechanical ventilation. Am Rev Respir Dis 139:513–521
15. Alie J, Weisel RD, Layug AB, Kripke BJ, Hechtman HB (1974) Consequences of postoperative alterations in respiratory mechanics. Am J Surg 128:376–822
16. Mankikian B, Cantineau JP, Bertrand M, Kieffer E, Sartene R, Viars P (1988) Improvement of diaphragmatic function by a thoracic extradural block after upper abdominal surgery. Anesthesiology 68:379–386