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Physiological effects of decannulation in tracheostomized patients

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Abstract *Objective:* To evaluate the physiological effects of decannulation on breathing patterns and respiratory mechanics by comparing mouth breathing (MB) to tracheal breathing (TB) in tracheostomized patients. *Design and setting:* Prospective cross-over study in a critical and neuromuscular care unit. *Patients and methods:* Nine consecutive neuromuscular tracheostomized patients. Flow, esophageal pressure, gastric pressure, expiratory gas, and arterial blood gases were measured during MB and TB. *Results:* MB induced an increase in tidal volume (from 330±60 ml to 400±80 ml) without changing respiratory frequency, inspiratory time, or arterial CO_2 pressure. This ventilation increase was due to a significant increase in physiological dead space (from 156±67 to 230±82 ml) and was associated with significant increases in work of breathing (from 6.9±3.4 to 9.1±3.3 J/min), transdiaphragmatic pressure swing (from 10±4 to 12.5±7 cmH_2O), diaphragmatic pressure-time product per minute (from 214±100 to 271±92 $cmH_2O\ s^{-1}\ min^{-1}$), and oxygen uptake (from 206±30 to 229±34 ml/min). Upper airway resistance did not differ from in vitro tracheostomy tube resistance. In addition, total lung-airway resistance, dynamic pulmonary compliance, and intrinsic positive end-expiratory pressure were similar in both conditions. *Conclusions:* Decannulation resulted in a dead space increase with no other detectable additional loading. It increased work of breathing by more than 30%. Decannulation deserves special attention in patients with restrictive respiratory disease.

Keywords Upper airway · Dead space · Work of breathing · Tracheostomy · Decannulation

Introduction

Tracheostomy is widely used in patients with neuromuscular disorders who are dependent on a ventilator and do not respond adequately to noninvasive ventilation [1]. Decannulation is sometimes poorly tolerated [2]. This suggests that changing the breathing route may modify the loads imposed on the respiratory muscles. Indeed, tracheostomy may reduce the extrathoracic dead space [3, 4] and bypasses the upper airway resistance (R_{UA}) [5]. However, the resistance of the tracheostomy tube may induce a specific work of breathing (WOB) by reducing the tracheal lumen diameter [6]. Early studies of inspiratory effort parameters before and after tracheostomy produced conflicting results. Kim and Froed [7] reported that oxygen consumption V_{O2} was higher during tracheal breathing (TB) than mouth breathing (MB), whereas Cullen [8] found an increase in V_{O2} during MB.

Thus the effects of decannulation on breathing workload remain unclear. Moreover, despite weaning from tracheostomy has been advocated by some authors in ventilator-dependent neuromuscular patients [9] no studies have specifically investigated respiratory effort modifications induced by decannulation in this population.

Table 1 Characteristics of the study patients; all gas volumes expressed at body temperature and pressure (*VC* vital capacity, *pred* predicted, FEV_1 forced expiratory volume in 1 s, *TLC* total lung capacity, C_{Ldyn} dynamic lung compliance, *sniff Pdi* transdiaphragmatic pressure swing during sniff, *CVA* cerebrovascular accident, *CIDP* chronic inflammatory demyelinating polyradiculopathy, *CIN* critical illness neuropathy, *IBM with DP* inclusion body myopathy with diaphragmatic paralysis)

Case no.	Sex	Age (years)	Height (cm)	Weight (kg)	Diagnosis	Pa_{CO2} (mmHg)	Pa_{O2} (mmHg)	VC (l)	VC (% pred)	FEV_1/VC (% pred)	TLC (% pred)	C_{Ldyn} (ml/cmH_2O)	sniff Pdi (cmH_2O)
1	F	70	169	70	CIN	41	86	1.91	64	123	76	70	39
2	F	53	155	58	CVA	41	66	1.16	45	79	57	40	34
3	F	67	160	63	CIN	44	71	1.05	45	55	52	20	82
4	F	64	169	75	CIDP	40	63	1.01	34	76	49	50	24
5	F	70	160	44	Polymyositis	42	71	0.79	33	96	46	30	23
6	M	18	169	70	CIDP	41	53	1.22	27	91	59	30	23
7	F	34	168	60	Myelitis	39	81	2.32	64	60	78	80	37
8	M	29	181	70	IBM with DP	45	86	2.91	54	99	59	30	0
9	M	32	185	60	T11 paraplegia and rib cage injury	42	81	1.02	19	99	51	70	50

For these patients with reduced pulmonary volumes, even a small increase in dead space loading may be particularly harmful [10]. The purpose of this prospective study was to evaluate the effects of decannulation on respiratory pattern and WOB by comparing MB to TB in tracheostomized patients with neuromuscular disease. In addition, the study sought to elucidate the mechanism involved in WOB changes.

Materials and methods

Patients

The study protocol was approved by our institutional review board, and written informed consent was obtained from each patient. Nine consecutive tracheostomized patients were studied. Table 1 lists the main clinical and respiratory features in each patient. Mean age was 44±21 years, height was 170±10 cm, and weight was 62±10 kg. All the patients wore a Portex cuffed #7 or #8 (internal diameters in millimeters) tracheostomy tube. At the time of the study all the patients satisfied the following criteria: (a) restrictive respiratory disease according to the European community criteria [11], (b) successful breathing and eating with a plugged tube and a deflated cuff for longer than 24 h, (c) little or no airway secretions, and (d) no glottic or tracheal abnormalities detected by indirect laryngoscopy and fiberoptic bronchoscopy.

Experimental protocol

As shown in Fig. 1, respiratory flow, esophageal pressure, gastric pressure, tracheal pressure, O_2 consumption V_{O2}, CO_2 production V_{O2}, and arterial blood gas were measured during MB and TB to compare breathing patterns, physiological dead space (V_D^{phys}), and respiratory mechanics. Tracheostomy tubes were changed at 7–8 hours a.m.; the study began at 1–2 hours p.m. Each patient was studied in the seated position, in a quiet room, and after a fast of at least 8 h. The head and neck were kept in a neutral position, as changes in head and neck position can cause substantial variation in anatomical dead space [4] and R_{UA} [12]. Each patient was studied twice, once during MB and once during TB, in random order. Each test lasted at least 25 min. The time interval between the two tests was sufficiently long (20–30 min) to allow all respiratory parameters to return to their baseline values. Arterial samples for blood gas analysis were collected at the end of each test. During the TB test (Fig. 1A), the cuff was inflated sufficiently to prevent leaks around the tracheostomy tube. During MB (Fig. 1B), patients were asked to breathe through a mouthpiece (MPE1, Pall Newquay, Cornwall, UK) and were fitted with a nose clip. Then the tracheostomy tube was removed, and the stoma was sealed with a taped dressing (Tegaderm 3M Health Care, Borken, Germany). With this method leaks around the stoma are readily detected as air bubbles between the skin and the transparent taped dressing.

Measurements

Flow was measured using a Fleisch # 2 pneumotachograph (Lausanne, Switzerland) connected to a differential pressure transducer (Validyne MP 45±5 cmH_2O, Northridge, Calif., USA). The flow signal was integrated to yield tidal volume (V_T). Esophageal pressure (Pes) and gastric pressure (Pga) were recorded using a catheter-mounted transducer (Gaeltec, Dunvegan, UK). Appropriate placement of the esophageal transducer was verified using an occlusion test [13]. A pressure transducer (Validyne MP 45± 14 cmH_2O) was placed between the pneumotachograph and the patient to allow measurement of the pressure at airway entry (Paw). In addition, during the MB period the pressure drop in the upper airway (ΔP_{UA}) was recorded using a differential pressure transducer (Validyne MP 45±14 cmH_2O) connected to the stoma and to the circuit between the pneumotachograph and the patient (Fig. 1B). All signals were sampled and digitized at 128 Hz. An analogical/numerical system (MP100, Biopac System, Goleta, USA) was used to store data in a microcomputer for subsequent analysis.

During both tests the patients breathed through a low-resistance two-way valve (Hans Rudolph 2700L, Hans Rudolph, Kansas City, Mo., USA) whose expiratory end was connected to a metabolic monitoring device (Sensormedics Horizon System, Sensormedics, Anaheim, Calif., USA). The expired gas passed into a 2.6-l mixing chamber, from which continuous samples were drawn for analysis of mixed expired percentage oxygen and percentage carbon dioxide by rapid-response analyzers. To reduce the resistance and dead space of the circuit the pump of the metabolic monitoring device was activated and adjusted at 20 l min^{-1}. This

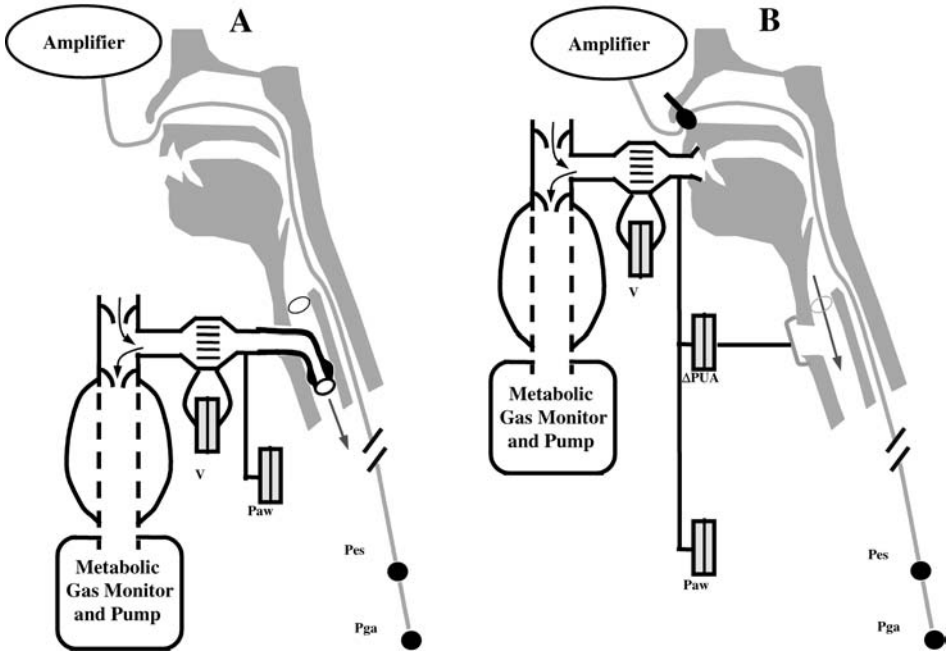

Fig. 1A, B Schematic representation of the two experimental conditions. **A** the patient breathed through a tracheostomy tube with the cuff inflated (tracheal breathing, *TB*). See text for details. **B** the patient breathed through a mouthpiece (MPE1, Pall Newquay, Cornwall, UK) and was fitted with a nose clip (mouth breathing, *MB*). See text for details

kept the total inspiratory and expiratory resistances of the circuitry (two-way valve included) under 0.8 $cmH_2O\ l^{-1}\ s^{-1}$ at 0.5 $l\ s^{-1}$. The dead space of the circuitry (V_D^{circ}) was 45 ml and similar during MB and TB. The metabolic monitoring device was calibrated before each measurement using various gas mixtures of known composition in the physiological range. It displayed oxygen uptake V_{O2} and carbon dioxide elimination V_{CO2} every minute. Arterial blood gas levels were measured in capillary samples taken from the radial artery after local anesthesia (lidocaine prilocaine, Emla, Astra) and analyzed immediately (Radiometer ABL 330, Tacussel, Copenhagen, Denmark).

Data analysis and assessment of effort to breathe

During each test data used for analysis were collected during a 5- min period of stability. Stability was defined as fluctuations in V_{O2} and V_{CO2} of no more than 5% during a 5-min period after the 15th min. Breathing pattern and minute-ventilation (V_e) were determined from flow tracings. Power of breathing (joule/min) was computed from Pes-volume loops [14]. Briefly, inspiratory WOB was calculated from a Campbell diagram by computing the area enclosed between the inspiratory esophageal pressure-tidal volume curve and the static esophageal pressure-volume curve of the chest wall. A theoretical chest wall compliance value of 4% of the predicted vital capacity per cmH_2O was used in this computation. Pes values at instants of zero flow were taken as the beginning and end of inspiration. Although use of a theoretical chest wall compliance value may be a source of error, this error, if present, was the same during MB and TB and consequently does not invalidate our comparison. The chest wall relaxation curve was superimposed on the Campbell diagram, assuming that the end-expiratory elastic recoil pressure of the chest wall was equivalent to the Pes level at the beginning of inspiratory effort. The beginning of the sharp negative deflection in the Pes curve was taken as the onset of the inspiratory effort. Dynamic intrinsic positive end-expiratory pressure ($PEEPI_{dyn}$) was measured as the pressure difference between Pes at inspiratory effort onset, as defined above, and Pes at inspiratory flow onset. The method developed by Lessard et al. [15] was used to correct this $PEEPI_{dyn}$ value for the presence of expiratory muscle activity during expiration as detected on Pga tracings.

WOB was partitioned into its elastic (W_{EL}) and resistive (W_{RE}) components. The line between the two Pes zero-flow points was used to separate these two components. The slope of this line yielded dynamic lung compliance (CL_{dyn}). Mean resistive pressure divided by mean inspiratory flow gave an estimate of total lung-airway resistance (R_L).

Average swing (Pdi=Pga-Pes) and pressure-time product for the diaphragm (PTPdi/breath, in $cmH_2O\ s^{-1}$] were measured from 30 consecutive breaths. PTPdi/breath was obtained by measuring the area under the Pdi signal from the beginning of the inspiratory deflection to the end of inspiratory flow [16]. PTPdi/breath was multiplied by respiratory frequency to obtain PTPdi/min ($cmH_2O\ s^{-1}\ min^{-1}$]

Physiological dead space (V_D^{phys}) was calculated using Enghoff's modification of the formula of Christian Bohr:

$$V_D^{phys}+V_D^{circ}=V_T\times(Pa_{CO2}-PE_{CO2}/Pa_{CO2})$$

where PE_{CO2} is the mixed expired carbon dioxide tension and Pa_{CO2} the arterial carbon dioxide tension inferred from the arterial blood gas level.

During MB, ΔP_{UA} was measured at various inspiratory flow levels (0.3, 0.4, and 0.5 $l\ s^{-1}$). In addition, R_{UA} (ΔP_{UA}/Flow) was calculated at 0.4 $l\ s^{-1}$ during inspiration. The static pressure-flow relationship of the tracheostomy tubes was determined in vitro as described previously [17]. Tracheostomy tube resistance (R_{TT}) was calculated at the same flow as R_{UA}, i.e., 0.4 $l\ s^{-1}$. All gas volumes were expressed at body temperature and pressure except V_{O2} and V_{CO2}, which were expressed at standard dry temperature and pressure.

Statistical analysis

Data are expressed as means ±SD. The Wilcoxon signed-rank test was used to compare data between MB and TB and to compare in vitro R_{TT} and in vivo R_{UA}. *P* values less than 0.05 were considered statistically significant.

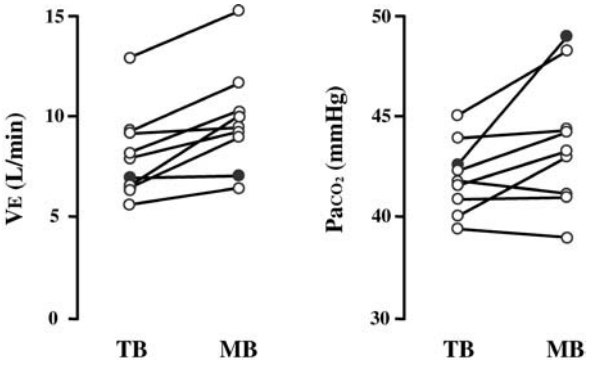

Fig. 2 Individual values for minute ventilation (V_E) and arterial carbon dioxide tension (Pa_{CO2}) during mouth breathing (*MB*) and tracheal breathing (*TB*). *Closed circles* Patient with a vital capacity less than 20%

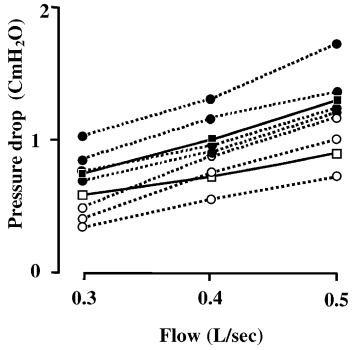

Fig. 3 In vitro flow-pressure relationship of the tracheostomy tubes (*solid lines with open and closed squares* representing TT #7 and TT #8) superimposed on in vivo flow-pressure relationships of the upper airways (*UA*) of the study patients (*dashed lines*). *Dashed lines with open circles* Patients wearing TT #7; *dashed lines with closed circles* patients wearing TT #8. The UA flow-pressure relationships were similar to the corresponding TT flow-pressure relationships. Then the pressure drop (y-axis) was either the upper airway pressure drop (ΔP_{UA}) or the tracheostomy tubes pressure drop determined in vitro as described previously [17]

Table 2 Ventilatory and arterial blood gas parameters during mouth breathing (*MB*) and tracheal breathing (*TB*); all gas volumes expressed at BTPS (V_T tidal volume, f_R respiratory frequency, V_E minute ventilation, T_I inspiratory time, T_{TOT} total respiratory time)

	TB	MB	*p*
V_T (ml)	330±60	400±80	<0.01
f_R (breaths/min)	24.3±4.9	24.5±4.4	NS
T_I (s)	1.11±0.23	1.13±0.17	NS
V_E (l/min)	8.0±2.1	9.6±2.4	<0.05
V_T/T_I (l/s)	0.31±0.08	0.36±0.09	<0.05
f_R/V_T (b.min^{-1}. l^{-1}	77±30	64±18	NS
T_I/T_{TOT}	0.45±0.06	0.45±0.07	NS
Pa_{O2} (mmHg)	73±11	70±10	NS
Pa_{CO2} (mmHg)	42±1	43±2	NS
pH	7.41±0.03	7.40±0.03	NS

[a] Wilcoxon test

Results

All nine patients tolerated both tests, and stability was consistently obtained by the 20th min of each test.

Respiratory pattern

The mean values of the main ventilatory parameters are presented in Table 2. MB significantly increased V_T by about 17.5% without changing respiratory frequency (f_R) or inspiratory time (T_I). V_E and V_T/T_I increased significantly, but Pa_{CO2} showed no significant changes. Individual values for V_E and Pa_{CO2} are shown in Fig. 2. The duty cycle, f_R/V_T, Pa_{O2}, and pH were similar during MB and TB, as depicted in Table 2. The V_E increase seen during MB in the absence of a Pa_{CO2} decrease was due to a significant increase in V_D^{phys}, from 156±67 ml during TB to 230±82 ml during MB. Individual values for V_E and Pa_{CO2} are shown in Fig. 2. Interestingly, in all the patients but one, Pa_{CO2} increased by no more than 2 mmHg during MB. The only patient who increased Pa_{CO2} by more than 2 mmHg during MB was the one with the most severe restrictive defect (vital capacity <20% of the predicted value).

Upper airway and tracheostomy tube resistance

ΔP_{UA} was measured in seven patients. The measurement failed for technical reasons in two patients (in one, connection between stoma and pressure transducer was occluded by secretions; in the other, leaks occurred around the stoma when the pressure transducer was connected). In vivo R_{UA} was similar to in vitro R_{TT} (2.28± 0.59 cmH_2O l^{-1} s^{-1} and 1.91±0.06 cmH_2O l^{-1} s^{-1}, respectively; NS). The flow-pressure relationships of #7 and #8 tracheostomy tubes used by the study patients superimposed on the curves of the patients' upper airways are shown in Fig. 3.

Respiratory mechanics in the study patients

Respiratory mechanics parameters are reported in Table 3. No expiratory activity was detected during MB or TB tests. R_L was similar during MB and TB. Neither $PEEPI_{dyn}$ nor CL_{dyn} were modified during MB compared to TB. Inspiratory effort parameters, namely, swing Pdi, and pressure-time product for the diaphragm (PTPdi), were significantly higher during MB than during TB, as depicted in Table 3. Individual values for WOB and V_{O2}

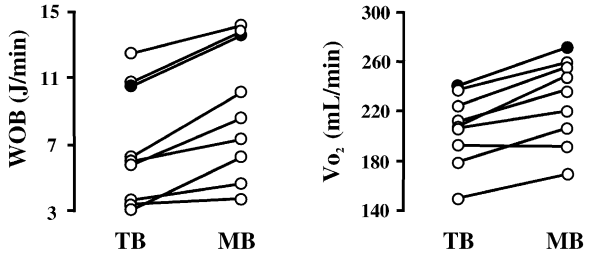

Fig. 4 Individual values for work of breathing (*WOB*) and oxygen uptake V_{O2} during mouth breathing (*MB*) and tracheal breathing (*TB*). *Closed circles* Patient with vital capacity less than 20% of predicted

Table 3 Respiratory mechanics during mouth breathing (MB) and tracheal breathing (TB); V_{O2} and V_{CO2} were expressed at standard dry temperature and pressure (*WOB* work of breathing, W_{EL} elastic work, W_{RES} resistive work, *Pdi* transdiaphragmatic pressure, *PTPdi* Diaphragmatic pressure time product, V_{O2} oxygen consumption, V_{CO2} carbon dioxide elimination, C_{Ldyn} dynamic lung compliance, $PEEPi_{dyn}$ dynamic intrinsic positive end-expiratory pressure, R_L total lung-airway resistance)

	TB	MB	*p*
R_L ($cmH_2O.l^{-1}.\ s^{-1}$)	8.7±3.8	8.8±7.4	NS
C_{Ldyn} (l/cmH_2O)	57±39	64±34	NS
$PEEPi_{dyn}$ (cmH_2O)	0.8±0.4	1.5±2	NS
WOB (J/min)	6.9±3.4	9.1±3.3	<0.01
W_{EL} (J/min)	4.6±2.1	6.5±2.7	<0.05
W_{RES} (J/min)	2.2±1.4	2.6±1.3	NS
Pdi swing (cmH_2O)	10±4	12.5±7	0.007
PTPdi ($cmH_2O\ s^{-1}\ min^{-1}$)	214±100	271±92	0.02
V_{O2} (ml/min)	206±30	229±34	<0.05
V_{CO2} (ml/min)	166±26	176±25	NS

[a] Wilcoxon test

are presented in Fig. 4. Significant increases in WOB and V_{O2} occurred during MB (Table 3). The elastic component of WOB increased significantly during MB, whereas the resistive component did not (Table 3).

Discussion

The present study demonstrates that tracheostomy decannulation induces an increase in dead space responsible for a substantial increase in V_E with no concomitant decrease in Pa_{CO2} in patients with neuromuscular restrictive lung disease. Moreover, elastic and resistive properties of the respiratory system remained unchanged after decannulation. The observed increase in inspiratory muscle activity after decannulation was due mainly to the increase in dead space.

Before discussing the implications of these data, several methodological issues need to be addressed. One of the limitations of this study was patient recruitment. However, this invasive physiological study required insertion of an esophageal pressure monitoring catheter in each patient, as well as monitoring of oxygen uptake during two periods, including the difficult period of decannulation. To our knowledge, no other study has attempted such extensive investigations in a similar setting. In addition, despite the small number of patients, significant differences were observed. Furthermore, all the patients exhibited the same behavior. Thus, increasing the number of patients would not have changed the results. It can also be argued that heterogeneity of the patients tested in this study was a major limitation. The patients had generalized neurological disease due to a variety of causes. However, as mentioned above, all our patients exhibited the same behavior (see Figs. 2, 3, and 4). Thus, our finding that an increase in dead space without any other detectable additional loading occurred consistently after decannulation despite the heterogeneity of the population strengthens our conclusion.

Using a mouthpiece can induce nonchemical stimulation of ventilation [18, 19]. However, inductive plethysmography [20], which does not require attachment of measurement devices to the airway, has not been validated in the literature as an accurate procedure for WOB assessment. Furthermore, in our study, Pa_{CO2} did not decrease during MB. Therefore it is reasonable to conclude that the ventilation increase observed during MB in our study was not caused by nonchemical stimulation related to the mouthpiece and/or nose clip.

We had three main reasons for comparing TB to MB rather than to nasal breathing. First, subglottic resistance is lowest during MB with a mouthpiece (mouth open) [18]. Therefore MB through a mouthpiece minimizes WOB. Second, nasal masks used during nasal breathing have been shown to cause a significant increase in dead space [21] and to alter the breathing pattern [22]. Third, nasal resistances can vary widely during the day and among patients [23]. This variability may have affected the results of our study.

Although we were careful to keep resistance and dead space as small as possible, our set-up probably increased both the ventilatory demand and the inspiratory activity in our patients. However, set-up characteristics were identical during MB and TB. Therefore it is reasonable to assume that the observed differences between TB and MB were due only to decannulation and not to the set-up.

Our data were collected over a short period after decannulation. This study does not provide information on changes in breathing pattern or WOB that may occur later. Upper airway resistance increases during sleep [24]. Thus, the increase in inspiratory activity seen during wakefulness in our study may be magnified during sleep. Furthermore, tracheostomy tube removal may be followed by transient bronchoconstriction. However, R_L was similar during MB and TB, and R_{UA} was similar to tracheostomy tube resistance. This suggests that bron-

chopulmonary airway resistance was not substantially different during MB and TB.

A first finding from our study is that decannulation was associated with an increase in total ventilation due primarily to an increase in dead space. Although this finding was perhaps predictable, to our knowledge it has not been reported before. Increasing the external dead space has been widely used as a means of challenging the respiratory system. The main breathing pattern changes after dead space loading are increases in V_T and V_E [19, 25, 26] whereas respiratory frequency remains unchanged. This increase in ventilation aims at maintaining the Pa_{CO2} level. Interestingly, all our patients but one maintained isocapnia after decannulation. The only patient who rapidly became hypercapnic after decannulation, despite an increase in ventilation, was the patient with the lowest vital capacity (<20% of predicted). We can speculate that his severe respiratory defect made him unable to cope with the additional dead space loading and to augment his ventilation sufficiently. However, the mechanisms underlying the hypercapnia in this patient remain unclear.

The second important finding from our study is that decannulation induced a 30% increase in WOB and a 10% increase in V_{O2}. Cullen [8] obtained comparable results in 14 emphysematous patients before and after tracheostomy and observed that the increase in ventilation was associated with a trend toward an increase in V_{O2} during MB. No data on WOB were available. To determine whether the WOB increase in our study was due only to changes in ventilatory demand related to the dead space increase or was caused by alterations in the mechanical properties of the lung and airway we measured resistance in various portions of the respiratory system and determined dynamic lung compliance. The upper airway, which is bypassed by tracheostomy, is a major source of respiratory system resistance [24]. We found no difference between in vivo R_{UA} and in vitro R_{TT}. Accordingly, total R_L as assessed on flow and esophageal pressure data was similar during MB and TB (Table 3). Neither did we find any significant differences in $PEEPI_{dyn}$ or dynamic lung compliance between MB and TB. These findings strongly suggest that dead space was the only respiratory loading feature that was different between MB and TB and that the WOB and V_{O2} increases found during MB were due only to the increase in ventilatory demand.

The difference in physiological dead space (V_D^{phys}) between MB and TB was about 75 ml. In a cadaver study by Nunn and colleagues [4] the extrathoracic dead space was 72±32 ml. Rohrer [3] estimated that the volumes of the nasal cavity, pharynx, and trachea were 15, 30, and 45 ml, respectively. Thus, the V_D^{phys} difference between MB and TB in our study was similar to the upper airway volume found in prior morphological studies [3]. This suggests that in our study alveolar dead space was not noticeably different between MB and TB. Our results differ from those of Mohr and colleagues [27], who studied 45 mechanically ventilated surgical patients before and after tracheostomy. Only minimal improvements in pulmonary mechanics occurred after tracheostomy. Moreover, changes in physiological dead space did not predict the outcome of weaning. Nevertheless, several differences in study population, methods, and end-point must be outlined. First, the study population was very different from ours. Second, Mohr and colleagues [27] compared pulmonary mechanics before and after tracheostomy, whereas our study compared TB to MB in tracheostomized patients. Third, Mohr and colleagues [27] compared two artificial airway conditions, both of which by-passed the upper airway, (i.e., tracheostomy vus. endotracheal tube). Thus the absence of a difference in dead space between these two conditions is not surprising. However, our study was not designed to evaluate factors that influence weaning from tracheostomy [2, 28, 29]. We can only speculate that the large increase in WOB due to decannulation evidenced in our study may be a mechanism of respiratory failure after decannulation, especially in patients with small tidal volumes. This should be taken into account when planning decannulation in patients with severe neurological restrictive disease. As noninvasive ventilation has been demonstrated to reduce WOB [30], we can also hypothesize that noninvasive ventilation may reduce WOB after decannulation. However, this needs to be evaluated in clinical studies.

Conclusion

This in vivo study provides the first clues needed to understand the major physiological changes that occur after tracheostomy decannulation. Ventilation was augmented primarily because of the addition of dead space. No other lung and airway mechanical properties were modified. The dead space loading resulted in a substantial increase in WOB and inspiratory effort. Further studies are needed to evaluate the clinical implications of this finding, especially in neuromuscular patients.

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