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Breathing pattern and additional work of breathing in spontaneously breathing patients with different ventilatory demands during inspiratory pressure support and automatic tube compensation

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Abstract *Objective:* We designed a new ventilatory mode to support spontaneously breathing, intubated patients and to improve weaning from mechanical ventilation. This mode, named Automatic Tube Compensation (ATC), compensates for the flow-dependent pressure drop across the endotracheal tube (ETT) and controls tracheal pressure to a constant value. In this study, we compared ATC with conventional patient-triggered inspiratory pressure support (IPS).
Design: A prospective, interventional study.
Setting: A medical intensive care unit (ICU) and an ICU for heart and thoracic surgery in a university hospital.
Patients: We investigated two groups of intubated, spontaneously breathing patients: ten postoperative patients without lung injury, who had a normal minute ventilation (V_E) of 7.6 ± 1.7 l/min, and six critically ill patients who showed increased ventilatory demand (V_E = 16.8 ± 3.0 l/min).
Interventions: We measured the breathing pattern [V_E, tidal volume (V_T), and respiratory rate (RR)] and additional work of breathing (WOB_{add}) due to ETT resistance and demand valve resistance. Measurements were performed under IPS of 5, 10, and 15 mbar and under ATC.
Results: The response of V_T, RR, and WOB_{add} to different ventilatory modes was different in both patient groups, whereas V_E remained unchanged. In postoperative patients, ATC, IPS of 10 mbar, and IPS of 15 mbar were sufficient to compensate for WOB_{add}. In contrast, WOB_{add} under IPS was greatly increased in patients with increased ventilatory demand, and only ATC was able to compensate for WOB_{add}.
Conclusions: The breathing pattern response to IPS and ATC is different in patients with differing ventilatory demand. ATC, in contrast to IPS, is a suitable mode to compensate for WOB_{add} in patients with increased ventilatory demand. When WOB_{add} was avoided using ATC, the patients did not need additional pressure support.

Key words Respiration, artificial, methods · Respiratory insufficiency · Respiration, physiology · Ventilator weaning

Introduction

It is common practice to allow, if possible, the critically ill and intubated patient to breathe spontaneously under a pressure supported mode of ventilation rather than using a controlled mode. This is done mainly to avoid the negative side effects of controlled mechanical ventilation and to wean the patient from the ventilator. Under every pressure supported mode, patient and ventilator share the total work of breathing (WOB):

$$WOB_{tot} = WOB_{patient} + WOB_{ventilator} \quad (1)$$

In order to adjust the pressure support to the appropriate level and decide whether the patient can be extubated, the patient's WOB should be known [1]. The only direct way to assess $WOB_{patient}$ is to measure the oxygen consumption of the ventilatory muscles. As this is a cumbersome method [2], $WOB_{patient}$ must be indirectly assessed by determining WOB_{tot} and $WOB_{ventilator}$.

WOB_{tot} consists of two major parts: elastic work (to overcome the elastic recoil pressure of the lung and chest wall) and resistive work (to overcome airway and tissue resistance) [3]. If total respiratory system mechanics (compliance and resistance) are known, assessment of WOB_{tot} is possible using Eq. 2:

$$\begin{aligned} WOB_{tot} &= \int \Delta P \, dV = \int \Delta P_{elastic} \, dV + \int \Delta P_{resistive} \, dV \\ &= \int \frac{V}{C_{rs}} \, dV + \int R_{rs} \cdot \dot{V} \, dV \end{aligned} \quad (2)$$

with V = inspired volume above functional residual capacity, $\dot{V}$ = gas flow, C_{rs} = compliance of the respiratory system and R_{rs} = resistance of the respiratory system. From Eq. 2 it is obvious that WOB_{tot} depends not only on the respiratory mechanics, but also on the breathing pattern during inspiration. Less obvious from Eq. 2 is the following: when incomplete expiration [or intrinsic positive end-expiratory pressure (IPEEP)] occurs, V in Eq. 2 starts not at zero but at the volume retained in the lungs due to incomplete expiration. Thus, WOB_{tot} is also dependent on the breathing pattern during expiration.

In intubated, spontaneously breathing patients, the flow-dependent resistance of the endotracheal tube (ETT) can increase respiratory system resistance several fold [4], especially at high gas flow. ETT resistance limits expiratory gas flow and can cause or increase IPEEP, which can increase WOB_{tot}.

For reasons which are discussed later, we attribute the inspiratory ETT resistance to the ventilator. The ETT resistance consumes part of the pressure support, thus decreasing $WOB_{ventilator}$, dependent on the inspiratory gas flow [5, 6]. In addition to ETT resistance, the resistance and delay in the ventilator's gas flow delivery system further decreases $WOB_{ventilator}$ [7, 8].

It is widely accepted that the patient's WOB caused by ETT resistance and the resistance and delay in gas flow delivery must be compensated for by using appropriate pressure support [9, 10]. This is not a simple task, as the pressure support delivered depends on the inspiratory trigger criterion, the pressure rise time, and the inspiratory termination criterion, which again cause the pressure support delivered to be dependent on the patient's breathing pattern [11, 12]. Moreover, not only is $WOB_{ventilator}$ dependent on the patient's breathing pattern, but the breathing pattern changes when $WOB_{ventilator}$ is altered due to a change in pressure support [11]. It therefore seems unlikely that a simple general rule for adjusting the pressure support necessary to compensate for ETT resistance (as published in Brochard et al. [9] for example) is applicable to all patients. In addition, it is not even clear whether patient-triggered pressure support is a suitable mode to compensate for the WOB caused by the ETT resistance, as the time course of the pressure drop across the ETT and the time course of pressure support are not correlated.

A pressure support which is equal to the pressure drop across the ETT (ΔP_{ETT}) at any time could compensate for the patient's WOB arising from ETT resistance. We developed a ventilator which delivers such a pressure support mode, called the Automatic Tube Compensation (ATC) [13]. Under this mode, the ventilator controls the patient's tracheal pressure at a constant value during inspiration and expiration:

$$P_{trach} = PEEP \quad (3)$$

thus avoiding both an increase of the patient's WOB and expiratory flow limitation caused by ETT resistance. The pressure support delivered by the ventilator [which is equal to the airway pressure (P_{aw}), as the ventilator is connected to the outer end of the ETT] is not predefined and not triggered, but adapts automatically and immediately to the inspiratory effort of the patient:

$$P_{aw} = PEEP + \Delta P_{ETT} \quad (4)$$

We hypothesized that ATC can always compensate for WOB caused by ETT resistance, whereas conventional patient-triggered inspiratory pressure support (IPS) cannot. Furthermore, we expected that the patient's breathing pattern under ATC is different to that under IPS due to the absence of ETT-related WOB and expiratory flow limitation under ATC.

The objective of our study was to compare our new ventilatory mode, ATC, to conventional patient-triggered IPS in patients with different ventilatory demand in terms of breathing pattern and additional work of breathing.

Patients and methods

Patients

We investigated ten patients after open heart surgery and six patients who were ventilated for several days due to acute respiratory insufficiency. Patient characteristics are listed in Table 1. All patients were able to maintain sufficient gas exchange under IPS of 15 mbar or less.

Patients were ventilated in four modes: patient-triggered IPS of 5 mbar, 10 mbar, and 15 mbar, and under ATC. Patients were breathing in each mode for 15 min. The four modes were chosen in randomized chronological order to avoid time-dependent bias; other parameters were not changed during the investigation. The

Table 1 Details of two groups of patients studied ($n = 16$) (PaO_2/FIO_2 partial pressure of oxygen in arterial blood/fractional inspired oxygen, *ARI* acute respiratory insufficiency, *ARDS* adult respiratory distress syndrome, *CABG* coronary artery bypass graft, *CAD* coronary artery disease, *COPD* chronic obstructive pulmonary disease)

Age (years)	Sex	Tube ID (mm)	Reason for intubation	Relevant disease	PaO_2/FIO_2 (kPa/frac)	Duration of intubation[a] (days)	Extubation[b]
Patients with respiratory insufficiency							
38	F	7.5	ARDS	Septic toxic shock syndrome	54	25	x
51	M	8.0	ARDS after pneumonia	Pneumonia, septic toxic shock syndrome; multiple myeloma	18	10	–
31	F	7.0	ARI due to pneumonia	Cystic fibrosis	39	11	x
82	M	8.0	ARI due to pneumonia	–	15	5	–
43	M	8.5	Acute hepatic insufficiency	Alcoholic hepatitis with gastro-intestinal bleeding	45		x
60	F	8.0	ARI due to pneumonia and metabolic acidosis	CAD; acute myocardial infarction	44	19	–
51 ± 18 (mean ± SD)					36 ± 16 (mean ± SD)	14 ± 8 (mean ± SD)	
Postoperative cardiac patients							
70	F	7.5	CABG	CAD	58	< 1	x
59	M	8.5	CABG	CAD	21	< 1	x
54	M	8.5	CABG	CAD	40	< 1	x
65	M	8.5	CABG	CAD	42	< 1	x
63	M	8.5	CABG	CAD	40	< 1	x
62	M	8.5	CABG	CAD	37	< 1	x
64	M	8.5	CABG	CAD; COPD	46	< 1	x
54	F	7.5	CABG	CAD; COPD	60	< 1	x
66	F	7.5	CABG	CAD	57	< 1	x
53	F	7.5	Valve replacement	Severe aortic stenosis	58	< 1	x
61 ± 6 (mean ± SD)					46 ± 12 (mean ± SD)		

[a] Prior to investigation
[b] Immediately after investigation

investigation was conducted according to the ethical standards set out in the Declaration of Helsinki, approved by the ethical committee of our institution, and informed consent was obtained from the patients or from close relatives.

Equipment

In the IPS mode we used an EVITA-1 ventilator (Drägerwerk, Lübeck, Germany). In the ATC mode we used a modified EVITA-1 ventilator as described in Fabry et al. [13]. Briefly, the pneumatic piece of equipment was taken from an EVITA-1 ventilator in which we replaced certain electronic parts with our own control unit. The unit was used to control the inspiratory and expiratory valves of the ventilator in such a way that tracheal pressure remained constant. Further, the control unit calculated the tracheal pressure from the measured airway pressure, gas flow, and the tube resistance. Direct measurement of tracheal pressure [14] rather than its calculation would have been simpler; however, a reliable, long-term, stable measurement of tracheal pressure is so far not available. First, the control unit calculated the flow-dependent ΔP_{ETT} with a combination of a linear and a quadratic approximation:

$$\Delta P_{ETT} = K_1 \cdot \dot{V} + K_2 \cdot \dot{V}^2 \tag{5}$$

where K_1 and K_2 are the tube coefficients [4] and $\dot{V}$ is the gas flow. As the tube resistance depends on the direction of the gas flow, we used two different sets of tube coefficients for inspiration and expiration. Second, the control unit determined tracheal pressure (P_{trach}) from P_{aw} and the calculated pressure drop by simply subtracting ΔP_{ETT} from P_{aw}:

$$P_{trach} = P_{aw} - \Delta P_{ETT} \tag{6}$$

This calculation is sufficiently reliable [4]. Third, the control unit adjusted the inspiratory and expiratory pneumatic valves of the modified ventilator. To understand the adjustments and modifications, a knowledge of the unmodified ventilator is necessary.

The unmodified ventilator measures P_{aw} in the cavity of the expiratory valve, and regulates this pressure at a target pressure ($P_{aw,targ}$) by controlling the gas flow as a function of the deviation of P_{aw} from $P_{aw,targ}$ and time. Under IPS, $P_{aw,targ}$ is equal to the desired pressure support. The ventilator increases gas flow if P_{aw} falls below $P_{aw,targ}$, and decreases gas flow if P_{aw} exceeds $P_{aw,targ}$. The ventilator has a pneumatic expiratory valve which opens when P_{aw} exceeds the servo pressure (P_{servo}) applied to the valve membrane. P_{servo} is produced by a PEEP valve which generates a pressure proportional to a current I_{PEEP}. P_{servo} is generated relative to a reference pressure P_{ref}, which is atmospheric pressure. The ventilator adjusts I_{PEEP} so that P_{servo} is equal to $P_{aw,targ}$. The pressure difference between P_{aw} and $P_{aw,targ}$ is measured with a differential pressure transducer connected to the servo pressure ($P_{servo} = P_{aw,targ}$) and the cavity of the expiratory valve ($= P_{aw}$). This pressure difference is represented by a voltage U_{diff}.

In our modified ventilator we made use of the fact that the function of the ventilator is completely determined by the two electrical parameters U_{diff} and I_{PEEP}. The mode ATC can be generated by producing a U_{diff} which is proportional to the deviation of the tracheal pressure from PEEP so that

$$U_{diff} \sim P_{trach} - PEEP = P_{aw} - \Delta P_{ETT} - PEEP \quad (7)$$

During inspiration we controlled the expiratory valve by generating an I_{PEEP} so that P_{servo} is equal to $P_{aw,targ}$. Under ATC, $P_{aw,targ}$ is the sum of PEEP and ΔP_{ETT} (see Eq. 4):

$$I_{PEEP} \sim P_{aw,targ} = PEEP + \Delta P_{ETT} \quad (8)$$

During expiration, the pressure drop across the expiratory branch (ΔP_{eb}), which is caused by the ventilator tubing and the expiratory valve resistance, must be taken into account so that

$$I_{PEEP} \sim P_{aw,targ} = PEEP + \Delta P_{ETT} + \Delta P_{eb} \quad (9)$$

ΔP_{eb} can be calculated using Eq. 5 and the EVITA-1-specific coefficients $K_1 = 1.1$ mbar*s/l and $K_2 = 1.4$ mbar*s^2/l^2

To ensure complete tube compensation during expiration, the ventilator must be able to generate a subatmospheric P_{aw}, especially at low PEEP levels, high expiratory gas flow, or high ETT resistance. A small brushless blower with teflon bearings (RCBP/VS 203 CCW: EG & G Rotron, Woodstock, N. Y., USA) was fitted at the end of the expiratory branch to produce a subatmospheric pressure of approximately – 20 mbar, dependent on the expiratory gas flow. The reference input of the PEEP valve (P_{ref}) was connected to this negative pressure source, and we measured P_{ref} with a differential pressure transducer (32NA-005D: ICsensor, Milpitas, Calif., USA) to correct I_{PEEP} for changes of P_{ref}:

$$I_{PEEP} \sim P_{aw,targ} - P_{ref} \quad (10)$$

Figure 1 illustrates the function principle of the unmodified and the modified ventilator.

$\dot{V}$ was measured using a Fleisch No. 2 pneumotachograph (Metabo, Epalinges, Switzerland), which was placed at the outer end of the ETT. P_{aw} was measured between the pneumotachograph and the outer end of the tube. Differential pressure transducers for measuring P_{aw} (32NA-005D: ICsensor, Milpitas, Calif., USA) and $\dot{V}$ (CPS 10: Hoffrichter, Schwerin, Germany) were placed 20 cm from the patient in order to achieve good signal quality and short response time. The external control unit sampled the flow and pressure signals at a rate of 500 Hz and calculated the tracheal pressure and the control parameters U_{diff} and I_{PEEP} at the same rate.

The ability of the ventilator under ATC to control tracheal pressure to a constant value is comparable to the ability of the unmodified ventilator under continuous positive airway pressure (CPAP) to control airway pressure to a constant value. The ventilator's maximum gas flow delivery rate, however, is restricted to 2 l/s. As the ETT does not act any longer as a flow limitation in the ATC mode, some patients achieve an inspiratory gas flow limit of 2 l/s. P_{trach} can deviate considerably from a constant value in these patients. Figure 2 shows an example of P_{aw} and P_{trach} curves under ATC.

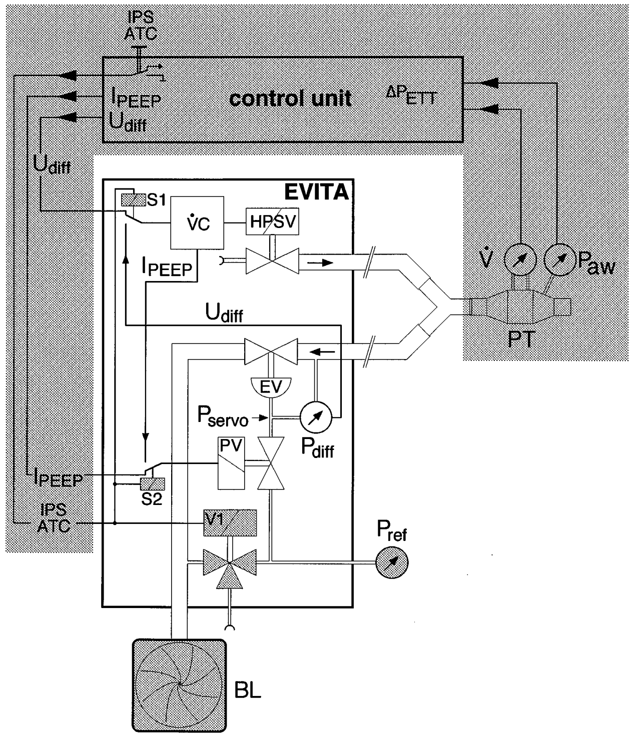

Fig. 1 Schematic diagram of the ventilator. Unmodified state: *unshaded area* the PEEP valve *PV* produces a servo pressure P_{servo} that regulates the expiratory valve *EV*. P_{servo} is regulated to the target airway pressure ($P_{aw,targ}$). The deviation of the airway pressure P_{aw} from $P_{aw,targ}$ can be measured by a differential pressure transducer which generates a voltage U_{diff}. Dependent on U_{diff}, the demand-flow controller of the ventilator $\dot{V}C$ controls the two high-pressure servo valves *HPSV* for oxygen and compressed air (only one valve shown) for the inspiratory gas flow. The blower *BL* is not in use, the expiratory limb is open to atmospheric pressure. The valve *V1* connects the reference entry of the PEEP valve with atmospheric pressure. Modified state (all modifications and added parts are shaded *gray*): the gas flow produced by the HPSV is controlled by the voltage U_{diff} that is fed into the demand-flow controller of the ventilator from an external control unit. The same unit produces a current I_{PEEP}, which controls P_{servo} to the value of $P_{aw,targ}$. The blower *BL* produces a subatmospheric pressure of – 20 mbar, which is connected to the expiratory valve *EV* and also acts as a reference pressure P_{ref} for the PEEP valve. $\dot{V}$ and P_{aw} are measured at the outer end of the ETT. The two relays *S1* and *S2* and the valve *V1* are used to switch from the modified to the unmodified state whenever desired. (Reproduced with permission from Fabry et al. [13])

Measurement of WOB in intubated, spontaneously breathing patients

Equation 2 does not offer a practical method for measuring WOB_{tot}, because it is difficult to determine the total respiratory mechanics of the spontaneously breathing patient. In addition, total respiratory mechanics depends on, among other factors, lung volume (nonlinearities of compliance) and gas flow (nonlinearities of airway resistance) [15, 16]. As an alternative to determining respiratory mechanics, many investigators measure the esophageal pressure amplitude during inspiration to calculate the ventilatory work necessary to overcome lung compliance, airway resistance, and lung tissue resistance [2, 9, 10]. To assess the work necessary to overcome chest wall compliance (C_w), an anthropometric value of C_w taken from published data [17] is often used. WOB can then be calculated using Eq. 11:

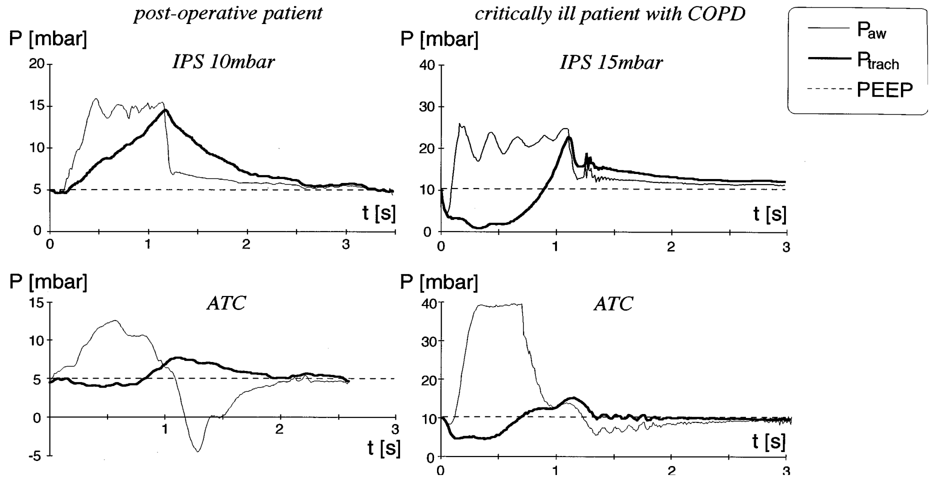

Fig. 2 Airway pressure and tracheal pressure curves under IPS *top* and ATC *bottom* in a patient after open heart surgery *left* and a critically ill patient with chronic obstructive pulmonary disease *COPD, right.* Note, although the ventilator lowers P_{aw} during expiration to subatmospheric pressure *bottom left,* controlling the expiratory valve ensures that P_{trach} is above or equal to PEEP. The patient with acute respiratory insufficiency under ATC generates an inspiratory gas flow of greater than 2 l/s *bottom right,* which accounts for part of the deviation between P_{trach} and PEEP

$$WOB_{patient} = \int \Delta P_{es}\, dV + \int \frac{V}{C_w}\, dV \qquad (11)$$

ΔP_{es} is the esophageal pressure amplitude (end-expiratory esophageal pressure minus actual esophageal pressure). Figure 3 a illustrates this calculation.

$WOB_{patient}$ obtained from Eq. 11 is independent of the quality of $WOB_{ventilator}$, as explained below. Equation 11 should, therefore, not be used to compare the effect of different ventilatory modes, ventilators, trigger mechanisms, endotracheal tube sizes, etc., on $WOB_{patient}$, even though it has been used (for example, in Banner et al. [1], Brochard et al. [9], and Fiastro et al. [10]).

An analysis of $WOB_{ventilator}$ offers a practical approach to investigate the influence of different ventilatory modes on $WOB_{patient}$: a change in $WOB_{ventilator}$ will cause an inverse change in $WOB_{patient}$ according to Eq. 1. This holds true only if the breathing pattern, and therefore WOB_{tot}, remains approximately unchanged. To correct $WOB_{ventilator}$ for a changed tidal volume, we normalized WOB per litre tidal volume (i. e., we divided WOB by tidal volume).

$WOB_{ventilator}$ can be subdivided into two components: at the onset of the patient's inspiration, P_{aw} normally falls below PEEP level, which causes additional (or imposed) work of breathing for the patient. Once pressure support has been triggered, P_{aw} increases. When P_{aw} rises above PEEP level, the ventilator reduces the work for the patient. The terms "additional work of breathing (WOB_{add})" and "reduced work of breathing (WOB_{red})" were first introduced by Katz et al. [7] and Viale et al. [8] to compare various CPAP delivery systems. Figure 3 b illustrates WOB_{add} and WOB_{red}. WOB_{add} and WOB_{red} can also be described by the relationship

$$WOB_{add} = \int_{P_{aw} < PEEP} (PEEP - P_{aw})\, dV \qquad (12\,a)$$

$$WOB_{red} = \int_{P_{aw} > PEEP} (P_{aw} - PEEP)\, dV \qquad (12\,b)$$

For use in a clinical situation, however, Eq. 12 must be adapted, as not only the demand-flow delivery system but also the endotracheal tube cause additional work [5, 6]. The adaptation can be easily done by replacing P_{aw} with P_{trach}, as the difference between P_{aw} and P_{trach} equals the pressure drop across the ETT:

$$WOB_{add} = \int_{P_{trach} < PEEP} (PEEP - P_{trach})\, dV \qquad (13\,a)$$

$$WOB_{red} = \int_{P_{trach} > PEEP} (P_{trach} - PEEP)\, dV \qquad (13\,b)$$

This is illustrated in Fig. 3 c.

WOB_{add} is equivalent to a certain amount of biological work due to muscular effort, whereas WOB_{red} is mechanical work delivered by a machine and has no biological equivalent. Once the patient has performed WOB_{add}, it cannot be compensated for by WOB_{red} or a pressure support as proposed by Fiastro et al. in their concept of "optimal pressure support" [10]. The ability of a ventilator to minimize WOB_{add} is therefore of the greatest importance. Consequently, only WOB_{add} is used to compare ATC with IPS.

To investigate the patient's breathing pattern and work of breathing, we measured P_{aw} and gas flow, as described above, and P_{trach}. P_{trach} was measured by introducing a 2.5-mm o. d. catheter with two holes on the side and no end hole (K-31: Baxter, Trieste, Italy) into the ETT. The tip of the catheter was 2 cm outside the tip of the ETT [4].

Data analysis

The signals for P_{aw}, $\dot{V}$, and P_{trach} were digitized with 12-bit resolution and stored at a rate of 100 Hz in a personal computer for further analysis. The first 5 min of each data section were excluded from our analysis. We calculated tidal volume (V_T), respiratory rate (RR), minute ventilation (V_E), and additional work of breathing on a breath-by-breath basis. V_T was calculated by numerical integration of $\dot{V}$. To calculate WOB_{add} according to Eq. 13 a, we did not use the adjusted PEEP value but we measured PEEP on a breath-by-breath basis as the mean value of tracheal pressure during the last 30 ml of expiration. All breath-by-breath values were averaged over at least 3 min and up to 10 min of undisturbed breathing (no coughing).

Statistical analysis

Values are expressed as mean ± standard deviation (SD). Statistical analysis was performed using a two-way analysis of variance for repeated measures and Bonferroni's test for multiple comparison. A value of $p < 0.05$ was taken as the level of statistical significance. We performed three statistical tests. (1) To analyse differences be-

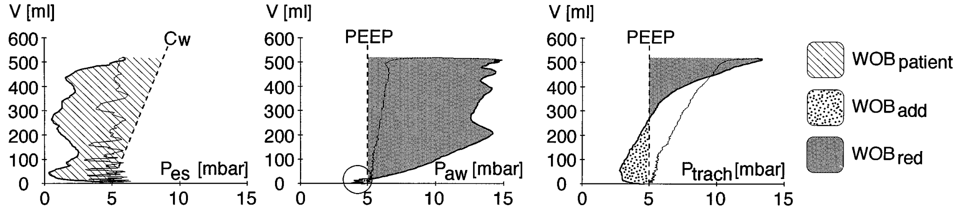

Fig. 3 Pressure-volume loops and work of breathing in a postoperative patient under patient-triggered inspiratory pressure support (IPS = 10 mbar). **a** The esophageal pressure-volume loop. The *striped* area bounded by the inspiratory esophageal pressure and the recoil pressure of the chest wall *broken line* corresponds to WOB as calculated in Eq. 11. **b** The airway pressure-volume loop of the same breath. The *dotted* area (indicated by the *circle*) corresponds to WOB_{add} as calculated in Eq. 12a and is due to the ventilator's demand-flow characteristics and trigger delay (in this case negligible). The *shaded* area corresponds to WOB_{red} as calculated in Eq. 12b and is due to pressure support. **c** The tracheal pressure-volume loop of the same breath. WOB_{add} *dotted area* as calculated in Eq. 13a is now related to tracheal pressure and arises mainly due to endotracheal tube resistance. WOB_{red} *shaded area* as calculated in Eq. 13b corresponds to the effective pressure support delivered to the patient

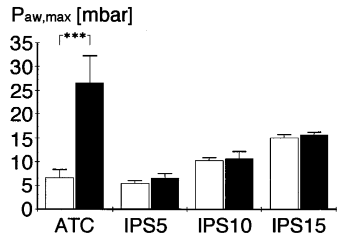

Fig. 4 Inspiratory peak airway pressure above PEEP (mean and SD) under ATC and IPS in postoperative patients *white bars* and critically ill patients *black bars*. The inspiratory peak airway pressure in ATC is automatically chosen by the ventilator and correlates to the maximum pressure drop across the ETT in inspiration. $^{***}p < 0.001$

tween the two patient groups, we compared absolute values of WOB_{add}, V_E, V_T, RR, and $P_{aw,max}$, separately for each mode. (2) To analyze the patient's response of breathing pattern and WOB_{add} to ATC and IPS, we compared relative changes of WOB_{add}, V_E, V_T, and RR between ATC and each IPS mode, separately for both groups. (3) To analyze whether the response of breathing pattern and WOB_{add} to ATC and IPS was different in both patient groups, we compared relative values of WOB_{add}, V_E, V_T, and RR (referring to ATC) in both groups, separately for each IPS level.

Results

Pressure support

Figure 4 shows inspiratory peak airway pressure ($P_{aw,max}$) (above measured PEEP) under the four different modes for the two patient groups. We analyzed $P_{aw,max}$ to ensure that the adjusted pressure support is delivered to the patient independently of the inspiratory effort. Under the ATC mode, $P_{aw,max}$ cannot be manually adjusted but is automatically chosen by the ventilator for complete tube compensation. Figure 6 shows that $P_{aw,max}$ is low (6.5 ± 1.8 mbar) in postoperative patients but high (26.5 ± 5.7 mbar) in critically ill patients, indicating a low inspiratory peak flow in postoperative patients and a high inspiratory peak flow in critically ill patients. Note that the modified EVITA-1 ventilator cannot produce a pressure support higher than 40 mbar and a gas flow higher than 2 l/s.

Breathing pattern

Figure 5 shows V_T, RR, and V_E in the two patient groups. V_E was 7.6 ± 1.7 l/min in postoperative patients and 16.8 ± 3.0 l/min in critically ill patients. V_E was independent of the ventilatory mode. In postoperative patients, V_T was lowest under ATC and increased with increasing pressure support. RR was highest under ATC and decreased with increasing pressure support. Critically ill patients showed different behavior: RR and V_T were nearly identical between ATC and IPS of 15 mbar. The increase in V_T (1.3% per mbar IPS) and decrease in RR (1.2% per mbar IPS) with IPS is less marked than in postoperative patients (V_T: 3.0% per mbar IPS; RR: 1.8% per mbar IPS).

Additional work of breathing

Figure 6 shows WOB_{add} in mJ per litre ventilation (which is equivalent to a pressure in Pa) in both patient groups under IPS and ATC. WOB_{add} is almost negligible in postoperative patients and only slightly increased at a pressure support of 5 mbar. ATC, as well as a pressure support of 10 or 15 mbar, almost completely compensated for WOB_{add}. In critically ill patients, however, only the ATC mode compensated for WOB_{add}, and IPS of 5, 10, or 15 mbar clearly could not.

Discussion

Postoperative patients without lung injury do not have to perform major additional work of breathing. WOB_{add}

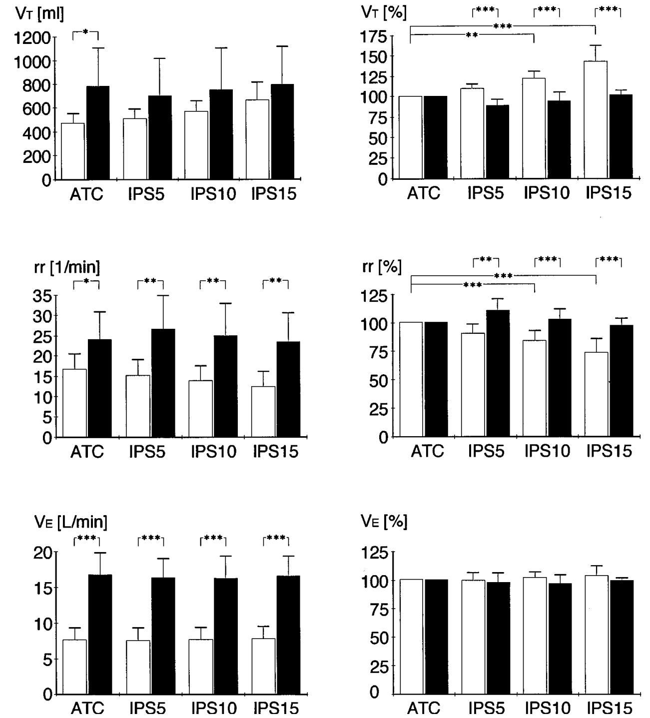

Fig. 5 Breathing pattern (tidal volume V_T, respiratory rate *rr*, and minute ventilation V_E; mean and SD) under ATC and IPS in postoperative patients *white bars* and critically ill patients *black bars*. Diagrams on the *left* show absolute values, diagrams on the *right* show the relative changes in breathing pattern in relation to ATC (values under ATC are set to 100 %). $^{*}p < 0.05$; $^{**}p < 0.01$; $^{***}p < 0.001$

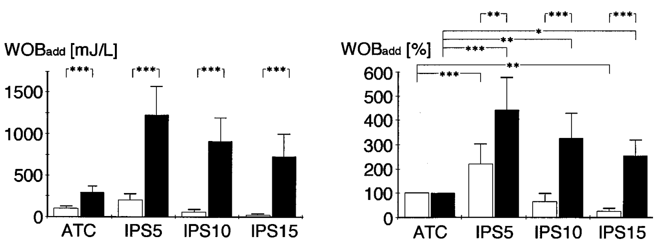

Fig. 6 WOB_{add} (mean and SD) under ATC and IPS in postoperative patients *white bars* and critically ill patients *black bars*. The diagram on the *left* shows absolute values, the diagram on the *right* shows relative changes in breathing pattern in relation to ATC (values under ATC are set to 100 %). $^{*}p < 0.05$; $^{**}p < 0.01$; $^{***}p < 0.001$

under ATC arises mainly from the demand-flow characteristics of the ventilator. Under IPS of 5 mbar, WOB_{add} is slightly, but significantly, increased due to ETT resistance, and IPS of 10 mbar can compensate for this work. This result corresponds with other findings [9] and is confirmed by the fact that peak airway pressure under ATC (i.e., the maximum pressure for tube compensation) is 6.5 ± 1.8 mbar.

In patients with increased ventilatory demand, a pressure support of even 15 mbar cannot compensate for the ETT resistance. Theoretically, WOB_{add} can be decreased with higher levels of pressure support. This is not always a practical solution, as it can cause overdistention of the respiratory system due to higher pressure loads being delivered at the end of inspiration. Additionally, high levels of pressure support can produce desynchronization between the patient's spontaneous breathing and the ventilator [12]. Further, it becomes more difficult with higher levels of pressure support to determine whether the ventilator only compensates for the ETT resistance or whether the ventilator effectively augments the patient's ventilation.

In contrast to IPS, WOB_{add} under ATC is only slightly increased. ATC does not produce an unnecessary pressure load at the end of inspiration. The high P_{aw} of 26.5 ± 5.7 mbar above PEEP in critically ill patients (see Fig. 3) only occurred during high gas flow, i.e., around the middle of inspiration. At the end of inspiration, P_{aw} is automatically lowered to PEEP level. Consequently, the P_{trach} remains fairly constant during the breathing cycle and this avoids the risk of barotrauma. The peak P_{aw} of 26.5 ± 5.7 mbar under ATC correlates to the pressure support in the IPS mode which would be necessary to compensate for the ETT resistance. This value is even higher than the pressure support of 18 ± 4 mbar that was necessary for nearly complete compensation of WOB, as reported by Alberti et al. [18]. We would not, however, recommend the use of such high IPS levels without consideration of the possibility of barotrauma or patient-ventilator desynchronization.

The patient's breathing pattern under ATC is barely influenced by additional or reduced WOB. ATC allows the patient to breathe at his or her inherent breathing pattern, with the exception that the patient is breathing with a PEEP, the resistance of the upper airways is replaced by the ETT, and the breathing pattern can be influenced by local irritations due to the ETT. Importantly, ATC also allows the physician to predict the patient's breathing pattern after extubation [19]. ATC is therefore a suitable mode for comparing the patient's inherent breathing pattern with the breathing pattern induced by the ETT and pressure support. We have found that IPS has a different effect on the breathing pattern in patients with different ventilatory demands. V_T was lowest under ATC and increased significantly with increasing pressure support in postoperative patients; however, in critically ill patients, V_T was similar under ATC and IPS of 15 mbar, but lower under IPS of 5 and 10 mbar (the differences were not significant).

A possible explanation for the distinctive behavior of V_T and RR in both patient groups is the following. In postoperative patients, V_E and, consequently, gas flow is low. The ventilatory mechanics are, therefore, mainly determined by the elastic properties of the lung and chest wall. Pressure support delivered will cause an increase in V_T due to the passive mechanical properties of the respiratory system. In patients with increased ventilatory demand and, therefore, increased gas flow, the resistive properties of the respiratory system become more dominant, mainly due to the flow-dependent ETT resistance. Figure 3 shows that the peak pressure drop across the ETT (which is equivalent to the peak P_{aw} above PEEP under ATC) can exceed 30 mbar. IPS of 5 or 10 mbar leads, therefore, to a decrease in V_T compared to that obtained under ATC.

The gas flow-dependent (and, consequently, breathing pattern-dependent) ETT resistance makes it difficult to distinguish what percentage of the pressure support augments the patient's ventilation and what percentage only compensates for ETT resistance. A patient showing obvious signs of fatigue under IPS of 10 mbar could, perhaps, breath without support when extubated. In fact, three of the six critically ill patients in this study who (1) did not need a PEEP higher than 5 mbar, (2) did not need a fractional inspired oxygen higher than 0.5, (3) who had sufficient cough and swallow reflexes, and (4) were able to cough up their tracheal secretions were successfully extubated following the investigation, although none of these patients was considered to be supported with IPS of 10 mbar or less by the ICU medical team.

In conclusion, the breathing pattern response to different levels of pressure support (IPS of 5, 10, 15 mbar, and no tracheal pressure support under the Automatic Tube Compensation mode) is different in patients with differing ventilatory demand. A pressure support of more than 5 mbar and ATC can compensate for the additional work caused by ETT in patients with low V_E. ATC, in contrast to IPS, is a suitable mode to compensate for WOB_{add} in patients with increased ventilatory demand. When avoiding WOB_{add} using ATC, the postoperative and critically ill patients we investigated did not need additional pressure support.

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