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Alveolar recruitment of atelectasis under combined high-frequency jet ventilation: a computed tomography study

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Abstract *Objective:* To quantify the effect of superimposed high-frequency jet ventilation on lung recruitment in adult patients with acute lung injury. *Design and setting:* Prospective clinical study in the intensive care unit of a university teaching hospital. *Patients:* Eight adults suffering from acute lung injury with a mean lung injury score of 2.6 ± 0.6 and pronounced atelectasis in at least two lung quadrants. The cause was either pneumonia ($n=5$) or postoperative sepsis ($n=3$). *Interventions:* Superimposed high-frequency jet ventilation was initiated in patients following a mean of 4.4 ± 1.7 days of conventional ventilation. Before and 4 h after the start of superimposed high-frequency jet ventilation differential lung volumes were determined by volumetry using computed tomography. *Measurements and results:* Superimposed high-frequency jet ventilation significantly increased the lung volume of every patient due to alveolar recruitment. This was achieved despite lower peak inspiratory pressures and higher $\text{PaO}_2/\text{FIO}_2$

ratios than with conventional ventilation. *Conclusions:* Treatment with superimposed high-frequency jet ventilation for 4 h resulted in rapid alveolar recruitment in dependent lung areas, improved gas exchange, and better arterial oxygenation. It offers an effective and advantageous alternative to conventional ventilation for ventilatory management of respiratory insufficient patients.

Keywords Mechanical ventilation · Superimposed high-frequency jet ventilation · Respiratory insufficiency · Atelectasis · Computed tomography

Introduction

Acute lung injury and adult respiratory distress syndrome are characterized by early morphological changes within the lungs, including alveolar fading, associated with lung collapse and impaired gas exchange. However, the application of high peak inspiratory and end-expiratory pressures cannot always compensate poor gas ex-

change. In addition, long-term application of high pressures can also compromise lung architecture and cause ventilator induced lung injury [1]. Dorsobasal lung regions are at risk of developing atelectasis due to local compression by overly distended upper lobes [2, 3]. Application of plateau pressure, prone positioning, and recruitment maneuvers are essential to open atelectatic lung, while adequate positive end-expiratory pressure

(PEEP) is used to keep open. The new approach of Amato et al. [4] demonstrated improved gas exchange and lung recovery in patients with acute respiratory distress syndrome applying low-tidal volume ventilation with high PEEP.

High-frequency ventilation techniques have shown to achieve a sufficient gas exchange when conventional ventilation methods are not effective in ventilatory support of patients with pulmonary insufficiency [5, 6]. Especially, combined high-frequency ventilation techniques proved to be effective due to better oxygenation and mainly due to improved PaCO₂ elimination. The ventilation mode used in our study, superimposed high-frequency jet ventilation (SHFJV) [7, 8], is a special form of the combined high-frequency jet ventilation (CHFJV) [9, 10], provided by an electronically regulated jet ventilator. This jet technique superimposes a HF jet stream over a low-frequency jet (LF) stream to assure oxygenation and CO₂ elimination, respectively.

The aim of our study was to examine the effect of SHFJV on the alveolar recruitment in patients with acute lung injury by means of computed tomography (CT) and

to compare this method with the conventional ventilation technique.

Material and Methods

Patients

After the institutional ethics committee approved the study and informed consent was obtained from their patients' next of kin, eight patients were included in the study when meeting the following criteria; two quadrant opacities on bedside chest radiography, atelectasis on thoracic CT, PaO₂/FIO₂ ratio less than 110 mmHg peak inspiratory airway pressure (PIP) higher than 30 cmH₂O, and PEEP higher than 10 cmH₂O during conventional ventilation. History or clinical evidence of lung fibrosis, pneumoconiosis, left ventricular failure, or severe obstructive lung disease were exclusion criteria. Tables 1 and 2 show patient characteristics at study entry, causes of acute lung injury, duration of conventional ventilation, and clinical patient scores. All patients received a continuous infusion of sufentanil (0.01–0.015 mg/kg per hour), midazolam (0.1–0.15 mg/kg per hour) and neuromuscular blocking agents to achieve one or two twitches out of a train of four stimuli to the ulnar nerve.

Table 1 Clinical Characteristics of the Patients at Study Entry (*ALI* acute lung injury, *AP* aspiration pneumonia, *APACHE* Acute Physiology and Chronic Health Evaluation, *ASA* score of American Society of Anesthesiology, *BPN* nosocomial pneumonia, *clr*

colorectal, *CV* conventional ventilation, *LISS* Lung Injury Severity Score, *PSE* postoperative sepsis, *SHFJV* superimposed high-frequency jet ventilation, *u* urological)

Patient no.	Age (years)	Sex	Cause of ALI	APACHE II	ASA	LISS	CV days prior to SHFJV
1	42	M	AP	21	2	1.5	5
2	58	M	BPN	18	3	2.0	3
3	28	F	PSE (u)	22	2	3.5	2
4	25	M	PSE (u)	28	2	2.5	6
5	59	F	AP	23	3	1.7	3
6	37	M	BPN	17	2	2.0	4
7	25	M	PSE (clr)	22	2	3.0	5
8	33	M	BPN	15	2	2.5	7
Mean	38.4	–	–	20.8	2.3	2.6	4.4
SD	13.7	–	–	4.1	0.5	0.6	1.7

Table 2 Respiratory characteristics of the patients at study entry (*OI* oxygenation index, *PIP* peak inspiratory pressure, *P_{mean}* mean airway pressure, *PEEP* positive endexpiratory pressure)

Patient no.	Aerated lung volume (ml)			PaO ₂ /FIO ₂	OI	PIP	PEEP	P _{mean}	PaCO ₂
	Well	Poorly	Total						
1	3490	710	4530	102.3	31.4	35	14	24.5	65.8
2	2890	660	3840	124.7	30.1	34	13	23.5	79.2
3	3270	720	4300	79.9	26.3	29	12	20.5	62.4
4	2820	590	3695	110.6	30.1	33	14	23.5	67.9
5	2970	770	4040	89.6	24.7	33	15	24	59.3
6	3160	700	4170	95.3	28.6	33	12	22.5	61.6
7	2780	620	3690	102.4	30.8	35	12	22	70.2
8	3160	760	4240	97.7	34.0	37	16	26.5	68.5
Mean	3068	691	4063	100.3	29.5	33.6	13.5	23.4	66.9
SD	245	64	303	13.5	2.9	2.3	1.5	1.8	6.2

ALEXANDER 1

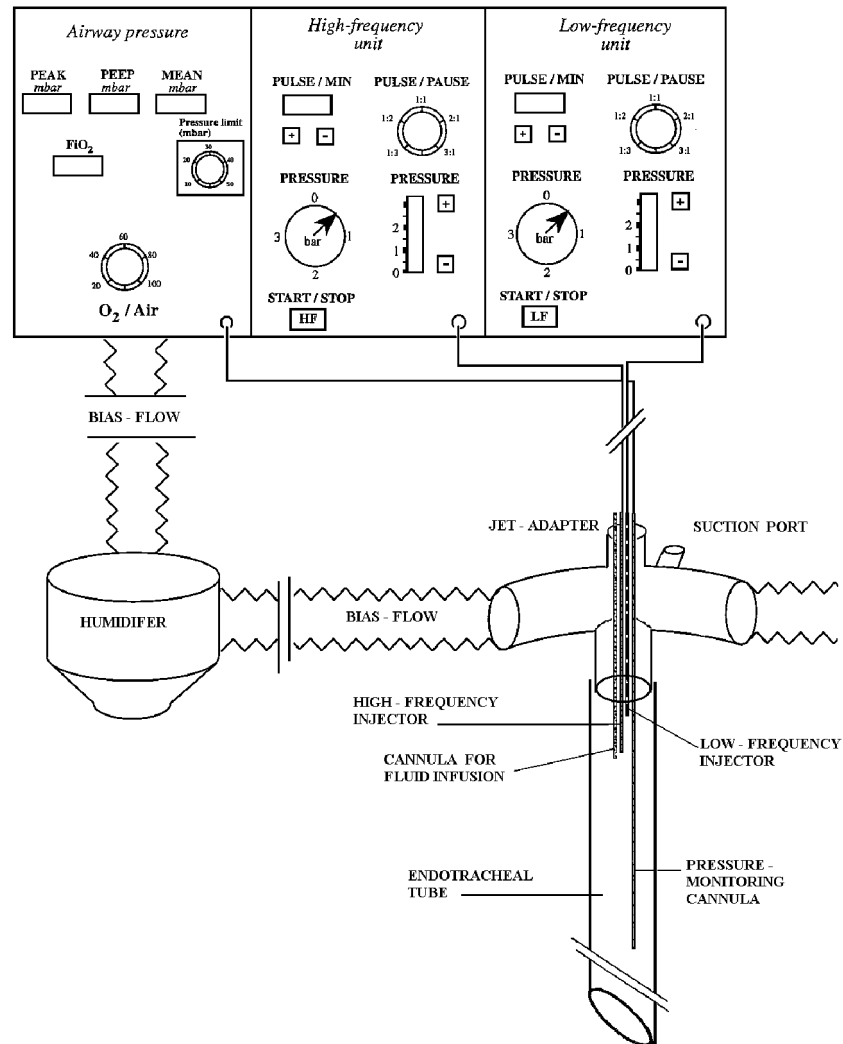


Fig. 1 Above The electronically regulated jet ventilator (Alexander 1). The respirator is split into three different sections. *Left* Airway pressure unit with the three displays (*Peak, PEEP, Mean*) for the airway pressures, the *FIO₂* display underneath, and the switch (*pressure limit mbar*) for the pressure limit allowed (*right*). At the button is the master switch for the *FIO₂* blender. *Center* High-frequency unit, with the display (*pulse/min*) for the high-frequency rate with its switches to increase or reduce underneath. *Right* The switch (*pulse/pause*) to set the inspiratory to expiratory ratio of the high-frequency jet stream. *Below* Display (*pressure*) for the high-frequency jet stream pressure with its switches on the side to increase or reduce. *To its left* Display of the high-frequency driving pressure showing the pressure from the wall outlet connector. At the button is the master switch (*start/stop*) for the high-frequency jet stream. *Right* Low-frequency unit, with the display (*pulse/min*)

for the low-frequency rate with its switches to increase or reduce underneath. *To the right* The switch (*pulse/pause*) to set the inspiratory to expiratory ratio of the low-frequency jet stream. *Below, right* Display (*pressure*) for the high-frequency jet stream pressure with its switches on the side to increase or reduce. *To its left* Display of the low-frequency driving pressure showing the pressure from the wall outlet connector. At the button is the master switch (*start/stop*) for the low-frequency jet stream. *Lower part, left* Hot water humidifier plus the flexible tube for the bios flow. *Right* Jet adapter with its four cannulas. One cannula is for the high-frequency injector. It is next to the cannula for the fluid infusion. This one is needed for the additional humidification system [21]. Another one, the shortest, is connected to the low-frequency injector. The fourth is for pressure monitoring. It is longer than the others and allows a pressure measurement right down in the tube

Hemodynamic profile

An arterial catheter (Arterial Cannula, REF 682245, Becton Dickinson, Swindon, UK) and a central venous line (Arrow-Howes Multi-Lumen Central Venous Catheterization Set, 7-F, Arrow International, Reading, Pa, USA) was in place in all patients. Seven out of eight patients had a pulmonary arterial catheter (Swan-

Ganz, EFV/OTD, CE 0123, Irvine, Calif., USA). Arterial monitoring was performed by Hewlett Packard Monitoring (Merlin, Hewlett Packard, Irvine, Calif., USA).

Before initiation of SHFJV and every 30 min thereafter throughout the 4-h study we measured the following parameters: arterial blood gas analysis, $\text{PaO}_2/\text{FIO}_2$ ratio, oxygenation index ($\text{P}_{\text{mean}} \times \text{FIO}_2 \times 100 / \text{PaO}_2$), peripheral oxygen saturation, heart rate,

systolic, diastolic, and mean arterial blood pressure, mean central venous pressure, pulmonary arterial pressure, cardiac index, oxygen transport index, and pulmonary capillary wedge pressure were recorded. All patients remained recumbent for the duration of the study avoiding prone position.

Ventilation management

Conventional ventilation protocol

Initially all patients were conventionally ventilated in a pressure limited mode with 35 mbar PIP and 15 mbar PEEP (Evita, Dräger, Lübeck, Germany). The VF was limited with 25 bpm. Inspiratory to expiratory time ratio was set at 1:1. Following adjustments of the airway pressures and the VF were set according to the PaCO₂ determined by arterial blood gas analyses. We accepted PaCO₂ values up to 59 mmHg. Higher values caused intervention in terms of increasing VF. The FIO₂ was set to keep the PaO₂ values above 75 mmHg and the arterial oxygen saturation above 89%.

High-frequency jet ventilation protocol

SHFJV was started with the same levels of FIO₂, LF ventilation, inspiratory to expiratory ratio, PIP, and PEEP (Fig. 1). The rate of the high-frequency jet stream (HF) was set to 600 bpm and remained unchanged throughout the study. The inspiratory to expiratory ratio of the HF was set at 1:1. Pressure limits were set as under conventional ventilation (PIP 35 mbar, PEEP 15 mbar); allowing PaCO₂ values up to 59 mmHg. Higher values were treated by increasing the low VF up to 25 bpm. PIP was reduced when PaCO₂ values decreased by more than 10%. FIO₂ adjustments were performed according to the PaO₂ and arterial oxygen saturation values. The study protocol included a FIO₂ setting to keep the PaO₂ values above 75 mmHg and the arterial oxygen saturation above 89%. Oxygenation variables and ventilatory parameters including PaO₂/FIO₂ ratio, oxygenation index, PIP, P_{mean}, and PEEP were recorded before initiation of SHFJV and every 30 min thereafter throughout the entire study period.

Superimposed high-frequency jet ventilation technique

The jet technique used in our study is a combination of a LF jet with a HF jet applied by a single jet ventilator. The LF generates an inspiratory pressure plateau within the lungs providing an upper pressure plateau (PIP). Variables of the LF ventilation are: LF (4–40 bpm), inspiration and expiration time ratio of the LF and its driving pressure, required for the LF gas to leave the jet nozzle. The HF generates the lower pressure plateau creating a pulsing PEEP. In addition, superposition of the HF jet over the LF jet results in oscillation during the LF inspiratory pressure plateau. Variables of the HF jet are: frequency (90–900 bpm), inspiration and expiration time of the jet impulse, and its driving pressure. This ventilation mode is pressure limited. The pressure limitation can be variably adjusted from 20–60 mbar.

Respirator

We performed SHFJV in all patients for the study period of 4 h using a prototype of an electronically regulated jet ventilator (Alexander 1, Carl Reiner, Vienna, Austria). This jet ventilator uses a time-regulated pressure-controlled technique with a decelerating flow and allows simultaneous delivery of two jet streams to the patient. For humidification a hot water humidifier (MR850AGU, Fischer&Paykel, Auckland, New Zealand) and fluid warmer (fluid warmer HL-90INT, Technologies, Rockland, Mass., USA) were used [11].

Tubing

The jet streams were applied via a jet-adaptor (Fig. 1, Jet-Adapter, CE 0124, Rüsch, Kernen, Germany), which was connected to a conventional endotracheal tube (Tubus, Rüsch, Kernen, Germany) [12]. This jet-adaptor is made of synthetic material (plastic) and contains four nozzles, welded at the proximal end. The HF injector, the LF injector, and the additional humidification system [11] were connected to the proximal nozzles. The forth nozzle is located 10 cm distal of the jet nozzles for airway pressure measurement inside the tube and is displayed on the respirator's monitor. The pressure information contains PIP, P_{mean}, and PEEP and is renewed ten times per second.

Imaging and volumetry

For volumetric analysis, a state-of-the art multislice spiral CT scanner (Volume Zoom, Siemens, Forchheim, Germany) was used [13, 14]. This made it possible to scan the chest within 25 s. During that time an end-inspiratory hold was applied. It allowed us to determine the absolute amount of air contained in each lung [15]. For volumetric analysis we first extracted the bronchial system from the three-dimensional data set and then identified either lung. Within these segmented lung volumes we determined the volume of hyperinflated lung, well-aerated, and poorly aerated lung using the CT density ranges proposed by Gattinoni et al. [16]. For detailed description of the technique please refer to the accompanying electronic supplementary material.

Statistics

The Wilcoxon rank test was performed with variables that were not normally distributed to determine whether PIP, PaO₂/FIO₂ ratio, PaCO₂, and oxygenation indices were significantly different after 4 h of SHFJV. Unless otherwise stated, data are expressed as the mean ±SD. A *p* value less than 0.05 was considered statistically significant.

Results

Hemodynamic effects

We observed no hemodynamic impairment associated with SHFJV (Table 3) and no changes in oxygen delivery index or and cardiac index during SHFJV. Furthermore, we neither observed any combined effects of sympathetic stimulation, increased heart rate or peripheral vasodilatation, nor an increase in wedge pressures at the beginning of SHFJV. In addition, we did not observe any increase in mean pulmonary arterial pressure or signs of right ventricular failure during our study.

Oxygenation and ventilation under SHFJV

As early as 30 min after SHFJV we observed significant improvement in arterial oxygenation compared to conventional ventilation. SHFJV increased the PaO₂/FIO₂ ratio (100.3±13.5 vs. 151.4±9.9 mmHg, *p*<0.05) and the oxygenation index (29.0±2.1 vs. 13.4±1.5, *p*<0.05) (elec-

Table 3 Hemodynamic profile of the patients during the study. Values are the means of the average values representing all measurements performed for each patient (P_{ART} systemic arterial pressure, P_{PA} pulmonary arterial pressure, P_{CWP} pulmonary capillary wedge pressure, DO_2I oxygen delivery index, VO_2I oxygen consumption index)

	CPPV		SHFJV (superimposed high-frequency jet ventilation)													
	0	30 min	60 min	90 min	120 min	150 min	180 min	210 min	240 min	210 min	180 min	150 min	120 min	90 min	60 min	30 min
Mean P_{ART} (mmHg)	77±15	73±13	78±11	73±22	81±12	79±17	81±16	76±17	75±13	76±17	79±17	81±16	81±12	73±22	78±11	73±13
Mean P_{PA} (mmHg)	23.1±2.3	25.1±2.9	24.6±2.1	23.8±2.2	24.1±2.1	24.9±2.6	23.9±2.2	23.1±2.1	24.5±2.2	23.1±2.1	24.9±2.6	23.9±2.2	24.1±2.1	23.8±2.2	24.6±2.1	25.1±2.9
$PCWP$ (mmHg)	9.1±1.5	10.6±1.3	12.1±1.3	11.4±0.8	9.8±1.2	10.3±1.1	9.6±1.0	10.5±1.2	11.1±1.4	10.5±1.2	10.3±1.1	9.6±1.0	9.8±1.2	11.4±0.8	12.1±1.3	10.6±1.3
Cardiac index ($l \text{ min}^{-1} \text{ m}^{-2}$)	4.92±0.53	5.16±0.49	4.80±0.43	5.17±0.33	5.41±0.46	5.20±0.45	5.32±0.37	4.96±0.39	5.12±0.37	4.96±0.39	5.20±0.45	5.32±0.37	5.41±0.46	5.17±0.33	4.80±0.43	5.16±0.49
Hemoglobin (g/dl)	11.6±0.9	11.9±1.2	11.1±1.2	11.3±0.9	11.9±1.1	11.4±1.0	11.6±0.9	11.7±0.9	11.8±1.2	11.7±0.9	11.4±1.0	11.6±0.9	11.9±1.1	11.3±0.9	11.1±1.2	11.9±1.2
DO_2I ($mlO_2 \text{ min}^{-1} \text{ m}^{-2}$)	731±83	773±94	782±111	839±78	826±72	791±69	755±74	811±98	793±114	811±98	791±69	755±74	826±72	839±78	782±111	773±94
VO_2I ($mlO_2 \text{ min}^{-1} \text{ m}^{-2}$)	158±24	171±13	176±27	164±24	169±15	173±18	178±23	169±15	164±14	169±15	173±18	178±23	169±15	164±24	176±27	171±13

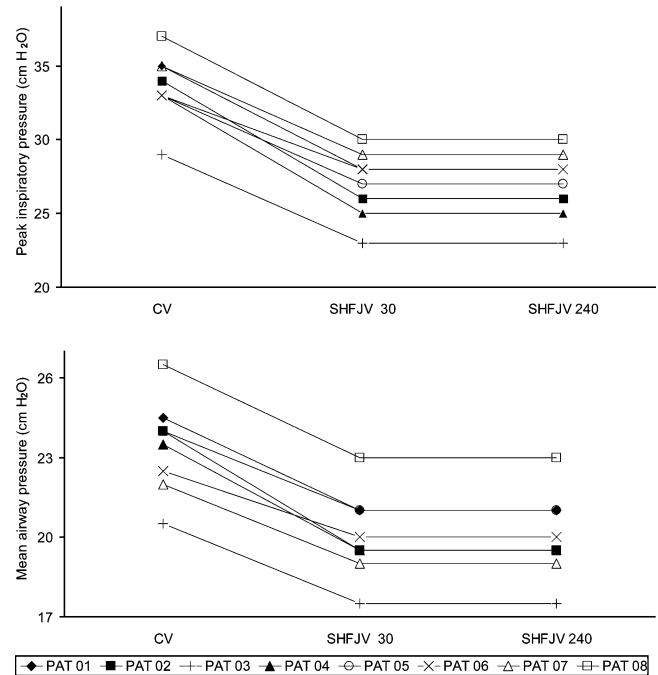


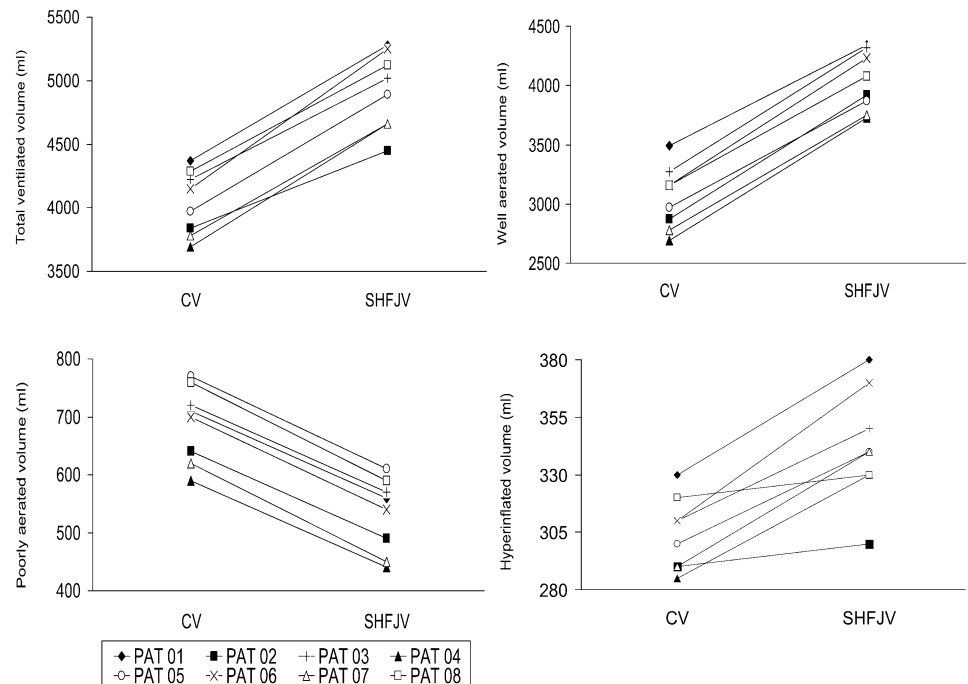
Fig. 2 The setting of the peak inspiratory pressure (PIP) and the resulting mean airway pressure (P_{mean}) over the study period of 4 h. As there was no other regulation performed after the 30 min decrease until the end of the study, no further time intervals are displayed in the figure

tronic supplementary material, Fig. 4). This was accompanied by a marked decrease in $PaCO_2$ (66.9 ± 6.2 vs. 55.1 ± 5.2 mmHg, $p < 0.05$) allowing for a reduction in PIP (33.6 ± 2.3 vs. 27.0 ± 2.3 cmH₂O, $p < 0.05$). The decrease in P_{mean} (23.4 ± 1.8 vs. 20.3 ± 1.6 cmH₂O, $p < 0.05$) was not associated with an impairment in oxygenation (Fig. 2). No further adjustments of ventilator settings had to be made.

Effective alveolar recruitment by SHFJV

Before initiation of SHFJV all patients presented with apical and dorsobasal atelectasis as demonstrated by chest radiography, and thoracic CT (electronic supplementary material, Picts. 1a, 1b, 2a, 2b). Atelectasis followed the known conventional ventilation-induced distribution pattern in recumbent patients, predominating in juxtadiaphragmatic regions. We quantified the total ventilated lung volumes of each patient before and after the 4-h period of SHFJV. Poorly aerated, well aerated, and hyperinflated lung volumes were differentiated, based on corresponding HU ranges. Treatment with SHFJV caused prominent recruitment of atelectasis in dependent lung regions. Total ventilated volume of each patient improved considerably under SHFJV (4063 ± 303 vs. 4908 ± 304 ml, $p < 0.05$). Particularly substantial incre-

Fig. 3 The changes in ventilated volume of all patients before and after 4 h of SHFJV. Total ventilated volume, well aerated volume, poorly aerated volume, and hyperinflated volume



ments were seen in the category of well aerated volumes (from 3068 ± 245 to 4036 ± 248 ml, $p < 0.05$), while poorly aerated volumes were found to be significantly reduced (691 ± 66 vs. 531 ± 64 ml, $p < 0.05$). Only minor changes in hyperinflated volumes were recorded (304 ± 16 vs. 340 ± 28 ml, $p > 0.2$; Fig. 3). No signs of pneumothorax, pneumopericardium, or pneumomediastinum were detected.

Discussion

Combined HF ventilation techniques have proven to be a sufficient respiratory therapy [17]. Further development in this field resulted in a modified form of combined HF ventilation using only one respirator [18, 19, 20]. This technique follows either a pneumatically regulated jet application (high-frequency percussive ventilation) or an electronically regulated jet application (SHFJV) as used in our study.

These respirators generate an upper and a lower pressure plateau by two separate jet streams, resulting in an oscillating PIP and PEEP. During SHFJV the gas is applied via the jet adapter [21, 22]. The disadvantage of every form of HF ventilation is the difficulty in measuring tidal volume, as the calculable amount of gas volume which the jet nozzle applies does not correspond to the tidal volume presented in the lung.

All patients reacted promptly to the switch from conventional ventilation to SHFJV as shown by a $\text{PaO}_2/\text{FIO}_2$ ratio increase. In addition, a marked decrease in PaCO_2 indicated ameliorated ventilation. Interestingly, even under attenuated PIP levels oxygenation remained improved until the end of the study period. We speculate

that apart from enhanced diffusion mechanisms which may contribute to these results the relatively fast recruitment per se is mainly responsible for the improvement in our patients [23, 24].

Although combined HF ventilation is used successfully in clinical routine, no CT-aided studies have yet been performed to examine its effect on atelectasis. For this reason we performed a volumetric analysis of lung parenchyma quantifying well aerated, poorly aerated, and hyperinflated lung regions. After 4 h of SHFJV we found a considerable increase in total ventilated volume in all patients. A similar outcome was found in well aerated volumes. Furthermore, the poorly aerated lung volumes significantly decreased, demonstrating successful alveolar recruitment by SHFJV. However, performing CT under a 25-s end-inspiratory hold can be regarded as a recruitment maneuver. Although the same maneuver was performed under conventional ventilation, it does represent a limitation of the study.

CT showed no signs of hyperinflation. In fact, volumetric determination of hyperinflated volumes showed only insignificant increments (less than 55 ml), located mainly in the upper lobes. Another SHFJV related concern is the influence of auto-PEEP on respiratory improvements and lung recruitment. As with any ventilation form, SHFJV produces auto-PEEP, especially at higher levels, where it can cause a significant increase in lung volume [25]. Primarily in patients suffering from chronic obstructive pulmonary disease such developments can be seen [26, 27]. Although no patient in this series suffered from chronic obstructive pulmonary disease, the possibility of auto-PEEP generation under SHFJV cannot be denied, particularly as the prototype

ventilator does not provide any method of measuring it. However, as the mechanism of lung recruitment under SHFJV cannot be explained by an increase in PIP it is subject of further investigations. We speculate that this effect can be a result of the pulsatile gas flow [28].

Conventional ventilation can also induce alveolar recruitment. Under this form of ventilation plateau pressures and PEEP recruit primarily cephalic nondependent areas, while dorsobasal lung regions remain atelectatic. Moreover, Gattinoni et al. [3] and Puybasset et al. [2] reported that PEEP levels of 10 cmH₂O increase the amount of noninflated tissue in the dependent parts of the lung. They suggested a PEEP-associated alveolar derecruitment due to local compression atelectasis by overdistended upper lobes. In addition, other studies reported that 30% of patients with acute lung injury do not respond to PEEP, or even deteriorate after PEEP implementation [29, 30], suggesting that the resting volume of the lower lobes markedly influences the response to PEEP. PEEP-associated alveolar derecruitment was not observed under SHFJV.

Amato and coworkers [4] showed several beneficial effects of the open lung approach using high PEEP and low tidal volumes in acute respiratory distress syndrome, including improved pulmonary compliance, increased PaO₂/FIO₂ ratio, decreased shunt fraction, shorter period of high FIO₂, and improved weaning rate. However, both Kolobow et al. [31] and Gattinoni et al. [32] demonstrated that mechanical ventilation of a diseased lung can be a major factor for further lung damage. Barotrauma has been attributed to high pressures. Attention has also been focused on volutrauma and on continuous reopening and collapsing (alveolar cycling) of lung parenchyma [33].

Gattinoni et al. [34, 35] described changes in density on CT of patients with acute lung injury. Regions of higher density were particularly pronounced in dependent lung areas and represented edema, compression atelectasis, consolidation, or a combination of the three [36, 37]. They further described a PEEP-induced density reduction, correlated with improvement in oxygenation. Their results provide important evidence for the benefits of applying PEEP in patients suffering from acute lung injury. Later reports show that an increased hydrostatic pressure along an anteroposterior gradient is responsible for compression atelectasis [38, 39]. Puybasset et al. [40] recently showed in a CT-based study that in addition to an anteroposterior gradient a cephalocaudal gradient influences the distribution of aerated and nonaerated areas.

In conclusion, our study demonstrated rapid improvement in oxygenation and ventilation in each patient during SHFJV. This improvement was associated with a substantial increase in ventilated lung volumes and a concomitant reduction in atelectasis, even in dorsobasal lung areas. Therefore our results suggest that SHFJV is an effective ventilatory strategy for patients with acute lung injury. However, during a pressure limited ventilation peak airway pressure may or may not represent peak alveolar pressure, depending on the flow value at end inspiration. In addition, reliability of peak pressure measurements at high frequencies depends very much on the frequency response of all the measurements chain. Therefore this represents something of a limitation of the study, especially under the lack of a plateau pressure measurement. Further clinical studies should examine the effect of recruitment maneuvers during SHFJV in severe acute respiratory distress syndrome.

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