Intensive Care Med (1999) 25: 1092–1099

ORIGINAL

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Effects of open heart surgery on respiratory mechanics

Received: 14 January 1999
Accepted: 1 July 1999

Supported by Grant 407, from Telethon, Rome, Italy

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Abstract *Objective:* To investigate the changes in respiratory mechanics in patients undergoing cardiac surgery before and after the operation.
Design: Prospective physiological study.
Setting: Operating theatre of the Institute of Cardiac Surgery, Verona, Italy.
Patients: 8 patients needing heart surgery because of a coronary bypass or mitral valve replacement.
Measurements and results: We measured respiratory mechanics before and immediately after the surgical procedure with two techniques: (1) the rapid airway occlusion technique during constant flow inflation at different lung volumes, and (2) the negative expiratory pressure (NEP) technique. We found that static and dynamic elastance (15.3 ± 3.3 and 19.0 ± 5.5 cmH_2O/l, respectively) and respiratory resistance, both airway and total flow resistance (5.8 ± 2.5 and 10.3 ± 4 $cmH_2O \cdot l^{-1} \cdot s$, respectively) before surgery were slightly higher than in normal anaesthetised subjects. In all patients, the static inflation V-P curves fitted the power function and exhibited a slight upward concavity towards the volume axis (a = 16.9 ± 3.5, b = 0.74 ± 0.07), indicating that elastance decreased with inflating volume. Whereas elastance increased by 30 %, neither intrinsic positive end-expiratory pressure, which was small, averaging 1.5 ± 1.2 cmH_2O, nor flow resistance changed after surgery. With the NEP technique, four patients exhibited expiratory flow limitation during the tidal expiration, for about 67 % of the exhaled volume, without changes after surgery. Arterial carbon dioxide tension (32 ± 4 mm Hg) and pH (7.46 ± 0.07) did not change, whereas arterial oxygen tension (PaO_2) (242 ± 34 mm Hg) decreased significantly by about 70 mm Hg, on average, with a constant fractional inspired oxygen (0.50).
Conclusions: This study shows that (1) respiratory mechanics can be abnormal in patients undergoing cardiac surgery, including expiratory flow limitation; (2) elastance increases and PaO_2 decreases after surgery; (3) simple, noninvasive techniques are available to measure respiratory mechanics in the operating theatre.

Key words Cardiac surgery · Respiratory mechanics · Flow limitation · Interrupter

Introduction

It has been shown that patients undergoing cardiac surgery for both coronary bypass and valve replacement can have abnormal respiratory mechanics, such as increased elastance and resistance which may further deteriorate after surgery. In general, respiratory elastance increases, following both valvular and coronary intervention [1, 2], while conflicting results have been reported for changes in airflow resistance. Barnas et al. [2] found that lung resistance was increased following cardiac surgery, whereas Auler et al. [3] and Zin et al. [1] observed that pulmonary resistance decreased postoperatively. The different results obtained in different studies could be due to differences in the population of patients, for example the stage of the disease at the time of surgery, although some variance related to the techniques used for measurement cannot be ruled out. In particular, possible nonlinearity of the volume-pressure and pressure-flow relationships, the presence of expiratory flow limitation, and the influence of ventilator settings were not taken into account in those studies. Since measurement of respiratory mechanics in patients undergoing heart surgery is important to understand the consequences of surgery on the lungs (e.g. pulmonary oedema [2]), and to prevent postoperative complications, the purpose of this study was to perform a more complete analysis of respiratory mechanics in patients undergoing cardiac surgery before and after the operation.

Patients and methods

The investigate protocol was approved by the Institutional Ethics Authorities. Informed consent was obtained from the patients before enrolment in this study.

Subjects

Eight consecutive patients (five males) undergoing heart surgery in the Institute of Cardiac Surgery of the University of Verona in Verona General Hospital (Italy) were recruited for this study. The diagnosis for surgery was the need for a coronary bypass in four patients and of a mitral valve replacement because of mitral insufficiency in the other four patients. All patients were smokers, but all quitted smoking from 6 to 12 months before surgery. However, none of the patients had either a history or clinical evidence of chronic respiratory disease. This was confirmed by preoperative routine spirometry in six of the eight patients. Characteristics of the patients are shown in Table 1.

Measurements

Lung volumes were measured by means of a Collins type 13-l bell spirometer (Biomedin, Padova, Italy), according to standardized procedures, a few days before the operation. Flow (V′) was measured by means of a heated pneumotachograph Fleisch no. 1 (Fleisch, Lausanne, Switzerland) inserted between the proximal tip of the endotracheal tube and the "Y" of the ventilator tubings, and connected to a Hewlett-Packard 47304A flow transducer (Hewlett-Packard, Cuppertino, Calif., USA). Volume (V) was determined by numerical integration of the flow signal. Pressure at the airway opening (Pao) was sampled proximal to the pneumotachograph using a differential pressure transducer (143PC01D, Honeywell, Freeport, Ill., USA). All physiological variables were fed into a personal computer (CPU 80486 DX2 66 MHz) via a 12-bit analog-to-digital converter (Data Translation DT2801/A, Marlborough, Mass., USA) at a sampling rate of 200 Hz and stored on 3.5-inch floppy disks for subsequent data analysis with ANADAT software (version 5.1; RHT Infodat, Montreal, Canada).

Respiratory mechanics was measured before and after surgery by means of the rapid occlusion technique at the airway opening during constant lung inflation [4, 5]. We also used the negative expiratory pressure (NEP) technique to assess expiratory flow limitation [6]. Any measurement of respiratory mechanics was preceded by a few regular mechanical inflations to ensure steady-state and similar volume history.

Airway occlusion during constant flow inflation

By means of a rapid occlusion valve (closing time < 20 ms) (Rapid valve, AeroMech Devices, Ontario, Canada) inserted between the endotracheal tube and the pneumotachograph, the airway opening was occluded during constant lung inflation under different conditions. (1) With the standard setting of the ventilator decided by the attending anaesthetist, the end-inspiratory occlusion was performed by operating the valve at the end of inspiration. (2) In different ventilatory cycles (test breaths), the airway was occluded at different inflation volumes, in a range between 0.1 and 0.8 l, during constant flow inflation [7]; in each test breath only one airway occlusion was made. (3) For one breath, the frequency was decreased by keeping the same minute ventilation to obtain a greater inflation volume with an increase of about 50% of tidal volume, and the airway was occluded immediately before the end of the inflation. (4) By keeping the tidal volume and ventilatory frequency steady, the constant inspiratory flow rate was changed just for one breath such that the airway occlusion was performed at different values of the constant inspiratory flow [8–10]. In the Servo 900B, the lower and higher flow were obtained by increasing and reducing, respectively, the inspiratory time to 50 and 25% of the total ventilator cycle, plus the brief end-inspiratory pause (10%) which was maintained. For both different volumes and flows, the test breaths were separated by at least five regular breaths. The technique of rapid airway occlusion during constant flow inflation is described in details elsewhere [11, 12]. Briefly, after the airway occlusion, there was an immediate drop in airway pressure from the peak airway pressure to a lower value (P1), followed by a gradual decrease to an apparent plateau (P2) which was achieved in less than 5 s in all instances. P2 represents the elastic recoil pressure of the total respiratory system at end-inspiration. Then the occlusion was released. When airway occlusion was performed at end-inspiration, the patients were disconnected from the ventilator during the occlusion, such that the following relaxed expiration occurred through the pneumotacograph until the expiratory flow became nil to indicate that the elastic equilibrium volume of the total respiratory system (Vr) was reached [13].

End-expiratory airway occlusion

At the end of the tidal expiration, with standard ventilator settings, the airway opening was occluded by means of the rapid occlusion valve for direct measurement of the static end-expiratory recoil

Table 1 Patients' characteristics

Patient	Diagnosis	Sex	Age (years)	Height (cm)	Weight (kg)	FEV_1		FVC		FEV_1/FVC
						(l)	(% pred.)	(l)	(% pred.)	
1	Mitral insufficiency	M	67	171	54	3.26	112	3.45	92	94
2	Coronary bypass	F	73	163	50	2.66	132	3.27	135	81
3	Coronary bypass	M	67	170	74	3.33	116	4.13	111	81
4	Mitral insufficiency	M	58	157	62	2.06	80	2.74	83	75
5	Coronary bypass	M	60	165	83	3.18	111	4.31	120	74
6	Mitral insufficiency	F	74	160	63	–	–	–	–	–
7	Mitral insufficiency	F	68	165	59	–	–	–	–	–
8	Coronary bypass	M	66	172	80	3.49	117	4.75	124	73
	Mean		67	165	66	3.00	111	3.78	111	80
	SD		6	5	12	0.54	17	0.75	20	8

pressure [i.e. the intrinsic positive end-expiratory pressure ($PEEP_i$) if any] [14].

NEP method

On the expiratory limb of the ventilator, proximal to the expiratory valve, we connected a 5-inch radial ejector (NEP system, Aero-Mech Devices, Ontario, Canada) driven by a gas source (0 to 80 PSI), able to generate a negative pressure on the expiratory circuit. The pressure driving the device could be adjusted to obtain the target pressure that was a negative Pao of – 5 cm H_2O [6]. Signals were recorded for several breaths to ensure steady-state, then the NEP system was activated during inflation while the expiratory valve of the Servo 900 was still closed. Once expiration started after the opening of the expiratory valve, the patient was subjected to a negative airway pressure for all expiratory time. The manoeuvre was repeated at least five times, leaving a few breaths in between, and in different test breaths in which the rapid occlusion method was applied.

Procedure

A standard set of ventilator tubings supplied with the machine for adult patients was used and the humidifier was omitted from the inspiratory line during the experimental procedure, in order to reduce the effects of the compliance of the system connecting the patients to the ventilator. Special care was taken to avoid gas leaks in the equipment, particularly around the endotracheal cuff, which was checked frequently. All patients had a small polyethylene catheter inserted in the radial artery prior to the surgical procedure to sample arterial blood gases for clinical purposes. After induction of anaesthesia (diazepam and fentanyl), each subject was paralysed (pancuronium bromide) and intubated (Portex cuffed endotracheal tube with an internal diameter ranging from 7.5 to 9 mm) to be mechanically ventilated in the controlled mode with a constant inspiratory flow delivered by a Servo 900B Siemens ventilator (Siemens, Elema, Sweden). The ventilator settings were prescribed by the primary physicians according to their clinical judgement and remained essentially unaltered throughout the procedure. The tidal volume (V_T) was set at about 10 ml/kg, ventilatory frequency (f) at 12–15 beats/min, and the inspiratory time (TI) amounted to 33 % of the total ventilator cycle duration (TTOT). A brief end-inspiratory pause (10 %) occurred in all instances upon the decision of the attending anaesthetists, such that the complete TI amounted to 41 % of the TTOT. A catheter was positioned in a central vein for continuous measurements of central venous pressure, as it is routine practice in that institution. Electrocardiography, systemic arterial blood pressure, and arterial oxygen saturation were monitored throughout the study. The fractional inspired oxygen (FIO2) was 0.5 in all patients and was maintained constant until the end of any surgical and experimental procedure. All patients were ventilated on zero end-expiratory pressure at the time of measurements. During cardiopulmonary bypass, lung inflation was maintained with the same FIO2 at a PEEP of 5 cm H_2O [15].

The patients were studied twice, in the supine position, in the operating room: 30 min after sedation and intubation, during controlled mechanical ventilation, before any surgical intervention, and then within 2 h after the end of the entire surgical procedure. During this latter period the patients were recovering from anaesthesia and were still under the effects of anaesthetic drugs and the muscle paralysing agent as well as of inotropic vasoactive drugs. A physician not involved in the procedure was always present for patient care. Both before and after surgery the complete set of respiratory mechanics measurements was performed in the following order: (1) end-expiratory occlusion and end-inspiratory occlusion with standard ventilator settings, the latter followed by a complete relaxed expiration; (2) occlusion at different inflation volumes; (3) end-inspiratory occlusion at different inspiratory flow rate; (4) NEP techniques.

Data analysis

$PEEP_i$ was measured as the difference between the plateau in Pao during the end-expiratory occlusion and atmospheric pressure [14]. The amount of dynamic hyperinflation was measured as the difference between the total exhaled volume from end-inspiration and the V_T [11]. Dynamic elastance was obtained as the ratio of the difference in Pao between the two points of zero flow over V_T [16]. The static elastance of the total respiratory system (Est,rs) was obtained by means of two technique. Firstly, the end-inspiratory occlusion techniques [1, 17], according to the formula:

$$Est,rs = (P2 - PEEPt)/V_T \quad (1)$$

Since there was no PEEP set by the ventilator, PEEPt, measured by means of the end-expiratory occlusion technique, represented basically $PEEP_i$. Secondly, the static inspiratory volume-pressure relationship of the total respiratory system was obtained by plotting the different volumes in different breaths, during airway occlusion, against the corresponding values of occluded Pao, at 3–5 s after airway occlusion, which represented the static recoil pressure

of the respiratory system (Pst,rs) at that occluded lung volume. Changes in volume were related to Vr, whereas changes in Pao were referred to atmosphere. The experimental points were fitted to a power function of the type [4]:

$$\Delta Pst = a \cdot \Delta V^b \quad (2)$$

where coefficient *a* represents static elastance of the relevant respiratory component at ΔV of 1 l and coefficient *b* is a dimensionless number that indicates the variation of elastance with inflating volume [11]. Values of coefficient $b = 1$ indicate that elastance is constant in the experimental volume range and that, therefore, the V-P relationship is linear rather than curvilinear [1]. For values of coefficient $b < 1$, elastance decreases with inflating volume, while it increases for values of coefficient $b > 1$, respectively.

Flow resistance was computed by means of the end-inspiratory occlusion during constant flow inflation, as previously described [8, 11, 18, 19]. The total resistance of the respiratory system (Rrs) and interrupter resistance (Rint) were computed by dividing both (Pmax – P2) and (Pmax – P1) by the V′ immediately preceding the airway occlusion and subtracting estimated resistance of the endotracheal tube [16, 20]. We also computed the difference between Rrs and Rint – that is, ΔR. As previously mentioned, Rrs, Rint, and ΔR were computed at least at two different values of constant flow inflation.

For detection of expiratory flow limitation, as indicated by Valta and colleagues [6], the expiratory limb of the V_T flow loops of the NEP breaths was compared by superimposition with those obtained during the immediately preceding breath. The flow signal was corrected for any offset based on the assumption that inspired and expired volumes of the preceding breaths were essentially identical [6]. The portion of the tidal expiration over which there was no change in flow with NEP (i.e. the portion of the tidal expiration over which the expiratory flows were identical) was considered as flow limited. This portion was quantified as the percentage of the inspired control volume.

Statistical analysis was performed using the Friedman two-way analysis of variance, when needed, and then the Wilcoxon rank test for paired observation. The coefficient of correlation *R* was also calculated to see whether the model was appropriate for the data. A $p < 0.05$ was accepted as significant.

Results

The average values of minute ventilation ($\dot{V}_E$), V_T, f, and T_I/T_{TOT} set by the ventilator, before and after heart surgery, are shown in Table 2. In spite of the experimental protocol, for which the ventilator settings had to remain constant, we observed a slight increase in f after the surgery. In Table 2, the mean values of respiratory mechanics, obtained with the standard ventilator settings, are also shown. On average, both dynamic (Edyn) and static (Est) elastances increased after surgery by approximately 25 and 35 %, respectively. In contrast, $PEEP_i$ and dynamic hyperinflation, averaging 1.5 ± 1.2 cmH_2O and 0.1 ± 0.05 l, respectively, remained unaltered. Figure 1 shows a representative example of the static inspiratory volume-pressure relationship (V-P) in one patient before and after surgery: the slope of the V-P relationship was steeper before than after surgery, indicating that static elastance increased after the surgery. This was the case for all eight patients, as shown by the individual data reported in Table 3, according to Eq. 2. The coefficient of correlation ranged between 0.84 and 1.00 being highly significant ($p < 0.001$) in any instance (Table 3). In all patients, the static inflation V-P curves exhibited a convexity towards the pressure axis, indicating that elastance decreased slightly with inflating volume. The values of *a*, i.e. Est,rs at 1 l of inflation volume, were not significantly different

Table 2 Ventilator setting and respiratory mechanics ($\dot{V}_E$ minute ventilation, V_T tidal volume, *f* respiratory frequency, *TI/TTOT* ventilator duty cycle, *Edyn* dynamic elastance of total respiratory system, *Est* static elastance of total respiratory system measured by end-inspiratory occlusion, *Rrs* resistance of total respiratory system, *Rint* airway resistance, Δ*R* respiratory resistance due to viscoelastic phenomena)

	Before		After	
	Mean	SD	Mean	SD
$\dot{V}_E$ (l/min)	6.59	1.25	6.94	0.76
V_T (l)	0.53	0.08	0.51	0.05
f (breaths/min)	12.5	1.7	13.5*	0.4
T_i/T_{TOT}	0.41	0.05	0.42	0.05
Edyn (cmH_2O/l)	19.0	5.5	23.7*	5.2
Est (cmH_2O/l)	15.3	3.3	20.6*	5.2
Rrs ($cmH_2O \cdot l^{-1} \cdot s$)	10.3	4	11.1	3.8
Rint ($cmH_2O \cdot l^{-1} \cdot s$)	5.8	2.5	6.6	6.6
ΔR ($cmH_2O \cdot l^{-1} \cdot s$)	4.5	2.3	4.5	4.5

* $p < 0.05$

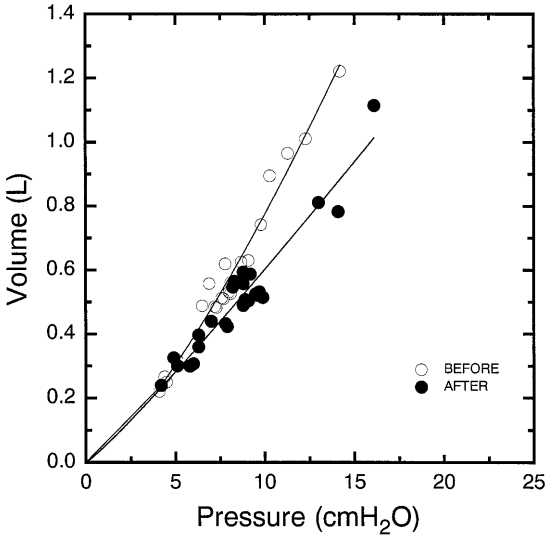

Fig. 1 Static volume-pressure relationship of the total respiratory system in one representative patient. *Circles* are single occlusion points before and after surgery. The *lines* represent the fit of the experimental points to a power function of the type $\Delta Pst = a \cdot \Delta V^b$. The static inflation V-P curves exhibited a convexity towards the pressure axis, with a coefficient $b < 1$, indicating that elastance decreased slightly with inflating volume

Table 3 Coefficients a and b of the static volume-pressure relationship of the total respiratory system fitted to a power equation of the type $\Delta Pst = a.V^b$ (*a* static elastance of the relevant respiratory component at delta volume of 1 l, *b* dimensionless number that indicates the variation of elastance with inflating volume)

Patient	Before			After		
	a ($cmH_2O \cdot l^{-1}$)	b ($cmH_2O \cdot l^{-1}$)	*r*	a ($cmH_2O \cdot l^{-1}$)	b ($cmH_2O \cdot l^{-1}$)	*r*
1	13.6	0.86	0.91	14.8	0.84	0.93
2	14.3	0.80	0.94	21.2	0.76	0.92
3	16.4	0.77	0.97	21.9	0.97	0.84
4	21.3	0.70	0.97	26.7	0.72	0.95
5	16.9	0.66	0.87	17.6	0.61	1.00
6	19.4	0.67	0.91	20.1	0.48	0.92
7	21.2	0.70	0.93	29.3	0.88	0.95
8	12.1	0.73	0.86	15.1	0.84	0.93
Mean	16.9	0.74	0.92	20.8	0.76	0.93
SD	3.5	0.07	0.04	5.2	0.16	0.04

from the values of Est,rs measured by means of the end-inspiratory occlusion technique and exhibited a similar increase after surgery. On average, coefficient *b* did not change. Flow resistance (Rrs, Rint, and ΔR) did not change after surgery. In Fig. 2, the mean values of Rrs and Rint at the lowest and highest flow before and after the surgical procedure are shown. Although there was no significant difference, on average, the values of Rint tended to increase with increasing flow, whereas the values of Rrs tended to decrease with increasing flow.

As illustrated in Fig. 3, four of the eight patients exhibited expiratory flow limitation during the tidal expiration, detected by the NEP technique. The expiratory flows during NEP coincided with the baseline expiratory flows for about 67 % of the exhaled volume, with a range from 50 to 80 %, without changes after surgery.

Table 4 shows that, on average, arterial carbon dioxide and pH did not change, whereas arterial oxygen tension (PaO_2) and the PaO_2/FiO_2 ratio decreased significantly after surgery. All the patients recovered fully from the operation and were successfully discharged from the hospital in the subsequent days.

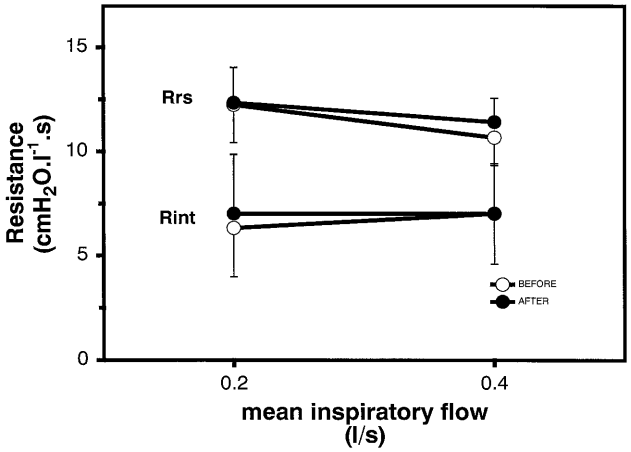

Fig. 2 Mean values *circles* and standard deviations *bars* of the total resistance of the respiratory system *Rrs* and interrupter resistance *Rint* before and after surgery

Discussion

The data from this study show that: (1) respiratory mechanics may be abnormal in patients with cardiac diseases, under anaesthesia, when they undergo heart surgery; (2) in addition to increased flow resistance, expiratory flow limitation can be found without previous evidence of chronic airway diseases; (3) after the surgical procedure, elastance increases whereas resistance and expiratory flow limitation remain essentially the same. We also show in this study that simple noninvasive techniques can be used for a detailed analysis of anaesthetised patients' respiratory mechanics in the operating theatre [12] to gather information which is potentially useful to prevent postoperative complications and to improve recovery from the surgery and anaesthesia.

Measurements before surgery

In the patients in this study, Est,rs obtained by means of the end-inspiratory occlusion technique (Table 2) was only slightly greater than the values obtained by D'Angelo et al. [8] and Pesenti et al. [21] (14.5 ± 2.1 and 15.3 ± 3.3 cmH_2O/l, respectively) with the same technique in anaesthetised subjects without known pulmonary disease undergoing minor surgery. With the end-inspiratory occlusion method, Auler et al. [3] found mean values of Est,rs (15.6 ± 1.8 cmH_2O/l) similar to ours in patients undergoing coronary bypass surgery, whereas higher values (22.4 ± 7.2 cmH_2O/l) were found in patients undergoing valve replacement. This may depend on different stages of the disease at the time of surgery with respect to the patients examined by Auler et al. [3]. The static volume-pressure relationship (Fig. 1) provided, on average, a value of *a* (i. e. Est,rs at 1 l of inflation volume) only slightly greater (Table 3) than that reported by D'Angelo et al. [8], but the value of *b* was low-

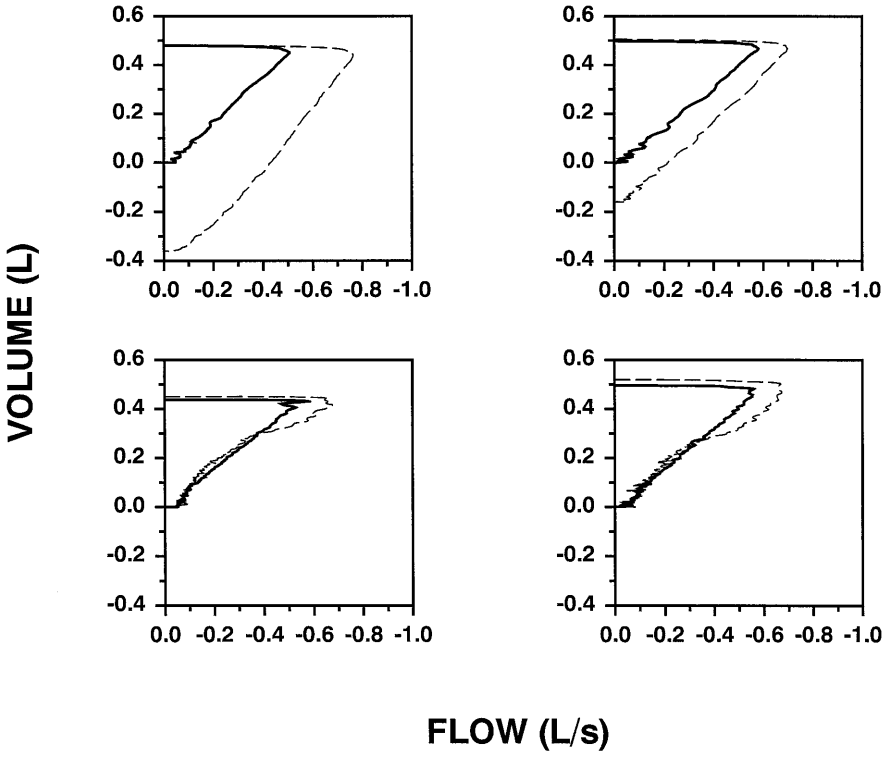

Fig. 3 Flow-volume curves of one patient without flow limitation *upper panels* and with flow limitation *lower panels* during control inflation *continuous line* and NEP application *dashed line,* before *left panels* and after *right panels* surgery

er, on average 0.74 and 0.95, respectively, in our study and their study. A $b < 1$ in Eq. 2 indicates that the V-P relationship is nonlinear and that Est,rs decreases with increasing volume (Fig. 1). This is in agreement with the results from Valta et al. [15], who found a significant recruitment following the use of PEEP in postcardiac surgery patients. In our study, Edyn,rs was significantly greater than Est,rs, the Est,rs/Edyn,rs ratio averaging 0.81. Following the analysis by Milic-Emili et al. [12], the difference between Edyn,rs and Est,rs represents the tissue viscoelastic component of elastance. Whether this results also from the effects of vascular congestion and interstitial edema, due to the cardiac disease, on pulmonary tissue mechanics remains to be established [22].

Although spirometric data reported in Table 1 are within normal limits, our patients had abnormal respiratory resistance. Rint, Rrs, and ΔR were higher than values reported in normal anaesthetised subjects [21, 23] for both flow rates examined in this study (Fig. 2). Similar data were obtained with the end-inspiratory occlusion technique by Auler et al. [3] in anaesthetised cardiac patients, although mean ΔR was slightly greater in our patients. According to the model analysis by Milic-Emili et al. [12], Rint reflects mainly airway resistance with a negligible contribution from the chest wall, while ΔR represents the additional tissue impedance due to viscoelastic properties of the respiratory tissue and to time constant inhomogeneity within the lungs [11]. The abnormal Rint could be due to the ignored effect of smoking habits, since all the patients were ex-smokers. An additional explanation might come from the combined effect of different factors. Firstly, anaesthetised patients in the supine position may have increased airway resistance because supine posture and anaesthesia decrease functional residual capacity and bronchial diameter [24–27]. A further factor affecting resistance in patients undergoing heart surgery might be decreased airway patency due to bronchial vascular congestion and reflex bronchoconstriction, while bronchial compression due to interstitial edema was ruled out [28]. Finally, it should be considered that abnormal lung mechanics could reflect changes in haemodynamics and in particular in left atrial pressure. However, the patients did not have a Swan-Ganz catheter and haemodynamics measurements were not obtained in this study. This may represent a limitation in the interpretation of the data.

Table 4 Arterial blood gases

	Before		After	
	Mean	SD	Mean	SD
pH	7.46	0.07	7.44	0.04
$PaCO_2$ (mm Hg)	32	4	33	3
PaO_2 (mm Hg)	242	34	163*	50
PaO_2/FIO_2 (mm Hg)	480	67	326*	100

* $p < 0.05$

In addition to abnormal flow resistance, half of the patients in this study had expiratory flow limitation, as disclosed by the NEP technique. Expiratory flow limitation is a well recognised event in patients with advanced chronic obstructive pulmonary disease (COPD) [29]. To our knowledge, this is the first demonstration of expiratory flow limitation during tidal expiration in mechanically ventilated, anaesthetised patients without evidence of COPD. The mechanisms underlying flow limitation are similar to those causing abnormal flow resistance, such as supine position, thicker bronchial wall, bronchoconstriction. An additional factor could be the advanced age of our patients whose mean age was 67 ± 6 years. In fact, it is known that maximum expiratory flow decreases with aging due to the decrease of lung elastic recoil such that tidal flows are physiologically closer to maximum flows than in younger subjects [30].

Measurements after surgery

The major modification after surgery was the increase in both dynamic and static respiratory elastance, which averaged, respectively, + 25 and + 35 %. This is in agreement with the finding by Auler et al. [3], who showed that the greater respiratory elastance after cardiac surgery was consequent to a significant increase in lung elastance, chest wall elastance remaining unchanged. An increase of extravascular lung water [31] and capillary volume [32] and alveolar collapse [33] were invoked as mechanisms underlying the change in elastance. Recent data from Gattinoni et al. [34] emphasized the role of abnormal chest wall mechanics and, in particular, of abdominal distension as a cause of increased respiratory elastance in mechanically ventilated patients with acute respiratory failure. We did not place an oesophageal balloon in our patients and hence we cannot distinguish between the lung and the chest wall contribution to the increased elastance in our patients. However, they were not in acute respiratory failure and can be considered more similar to the patients studied by Auler et al. [3], who had normal chest wall elastance. In our patients resistance did not change, whereas the patients examined by Auler et al. [3] also exhibited a slight decrease in respiratory resistance. The reasons for that change remained uncertain, perhaps attributable to release of smooth muscle active substances, or enlargement of the airways related to a greater pull on their walls due to the increased elastance. In our work, not only did resistance not change, but patients with expiratory flow limitation, detected with the NEP technique, remained flow-limited for the same fraction of the tidal expiration, despite the greater elastance. This discrepancy in the assessment of airway function between this study and Auler et al.'s study might well reflect some differences in the characteristics of the patient population.

Clinical implication

Firstly, it appears from our work that noninvasive measurement of respiratory mechanics in the operating theatre, even immediately before surgery, may disclose mechanical abnormalities of the respiratory system which were not revealed by conventional spirometry, although it should be taken into account that spirometric tests are performed in the standing position while our measurements were taken in the supine position under anaesthesia. The data from this study may be important in explaining the unexpected drop in a patient's oxygenation during or after the procedure and the need to treat it promptly, for example by adjusting the fractional inspired oxygen or the ventilator setting and the rate of fluid infusion. Unfortunately, we could not perform measurements of cardiovascular function to see if there is a relationship with lung mechanics. However, knowing about abnormal respiratory mechanics may help to treat it, for example with bronchodilators, and to prevent undue complications in the postoperative period (e. g. excessive work of breathing due to increased elastance and resistance and unexpected difficult weaning). In this connection it should be recalled that diaphragmatic paralysis due to a phrenic nerve lesion may be found in patients recovering from cardiac surgery [35]. Abnormal lung mechanics associated with a reduced diaphragmatic pressure-generating capacity may unduly prolong mechanical ventilation, exposing some patients to the well-known complications of this [36]. Since many modern microprocessor-equipped mechanical ventilators can provide easy measurement of respiratory mechanics, it seems reasonable to obtain these measurements in patients undergoing cardiac surgery, in addition to conventional spirometry.

In summary, the results of this study show that respiratory mechanics can be abnormal in patients undergoing cardiac surgery even in the presence of normal conventional lung function tests. In particular, expiratory flow limitation during tidal breathing may occur probably because of: (1) the decrease in functional residual capacity due to the supine position and anaesthesia and (2) the loss of lung recoil in the aging lung. Our work shows that simple and noninvasive techniques are available for bedside measurement of respiratory mechanics in the operating room.

Acknowledgements The authors wish to thank the medical nursing staff of the Institute of Cardiac Surgery of the University of Verona for their kind and skilful cooperation.

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