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# Respiratory system mechanics in the early phase of acute respiratory failure due to severe kyphoscoliosis

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**Abstract** *Objective:* To evaluate respiratory mechanics in the early phase of decompensation in a group of seven patients with severe kyphoscoliosis (KS) (Cobb angle > 90°) requiring mechanical ventilatory support.
*Design:* Prospective clinical study with a control group.
*Setting:* General intensive care unit at University of Rome "La Sapienza".
*Patients:* Seven consecutive patients affected by severe KS in the early phase of acute decompensation and a control group of six ASA (American Society of Anesthesiology) 1 subjects who were mechanically ventilated during minor surgery.
*Measurements and results:* Respiratory mechanics were evaluated during constant flow-controlled mechanical ventilation at zero end-expiratory pressure with the end-inspiratory and end-expiratory occlusion technique. In five patients who showed increased ohmic resistance (RRSmin), we evaluated the possibility of reversing this increase with a charge dose of 6 mg/kg doxophylline i. v. In four KS patients, in whom a reliable esophageal pressure was confirmed by a positive occlusion test, we separated respiratory system data into lung and chest wall component. All KS patients showed reduced values of respiratory compliance (CRS) and increased respiratory resistance (RRS). The average basal values of CRS were $36 \pm 10$ vs $58 \pm 8.5$ $cmH_2O$ in control patients; RRSmax was $20 \pm 3.1$ vs. $4.5 \pm 1.2$ $cmH_2O$/l per s; RRSmin $6.2 \pm 1.2$ vs. $2 \pm 0.5$ $cmH_2O$/l per s: $\Delta$RRS $14 \pm 2.6$ $cmH_2O$ vs $2.4 \pm 0.7$ $cmH_2O$/l per s. All KS patients showed low values of intrinsic positive end-expiratory pressure (PEEPi) ($1.8 \pm 1.5$ $cmH_2O$). Separation of lung and chest-wall mechanics, performed only in four patients, showed a reduction in both lung ($66.7 \pm 7.2$ ml/$cmH_2O$) and chest wall values ($84 \pm 8.2$ ml/$cmH_2O$), while both $Rmax_L$ and $Rmax_{CW}$ were increased ($16.6 \pm 2$ and $2.8 \pm 0.4$ $cmH_2O$/l per s, respectively). Infusion of doxophylline did not significantly change respiratory mechanics when evaluated 15, 30, and 45 min after the infusion.
*Conclusions:* During acute decompensation, both lung and chest-wall compliance are severely reduced in KS patients: conversely, and, contrary to that in patients with chronic obstructive pulmonary disease, increases in airway resistance and PEEPi seem to play only a secondary role.

**Key words** Kyphoscoliosis · Mechanical ventilation · Respiratory mechanics

## Introduction

Kyphoscoliosis (KS) is defined as a deformation of the spine involving both lateral displacement (scoliosis) and anteroposterior angulation (kyphosis). The most common form of KS is idiopathic, although secondary KS may be observed as a consequence of various neuromuscular diseases. The severity of KS is usually calculated by the angle formed by both lines of the convex primary curvature, defined as the Cobb angle.

Severe KS is not a rare cause of respiratory failure, and KS patients are often referred to the intensive care unit (ICU) for non-invasive or conventional mechanical ventilatory support. Despite decades of clinical observations, many aspects of cardiorespiratory failure in KS still require further evaluation. In particular, the data on the mechanisms that cause progression to cardiorespiratory failure, the exact mechanisms of cardiac and respiratory failure, and the pattern of disordered respiratory mechanics in the early phases of decompensation are controversial [1]. If a marked decrease in respiratory compliance can be considered a hallmark of severe KS [2–6], nevertheless, little is known about other variables such as respiratory system resistance, airway resistance, and intrinsic positive end-expiratory pressure (PEEPi), particularly during the early phases of acute decompensation requiring mechanical ventilatory support.

The aim of our study was to evaluate respiratory system mechanics in the early phase of decompensation in a group of patients affected by severe KS (Cobb angle > 90°) requiring mechanical ventilatory support, using the end-inspiratory occlusion technique [7] during constant flow ventilation.

**Table 1** Physical characteristics of normal subjects and patients with kyphoscoliosis. Values are mean ± SD

| | Control patients | Patients with kyphoscoliosis |
|---|---|---|
| Sex (M/F) | 3/3 | 4/3 |
| Age (years) | 48 ± 15 | 52 ± 11 |
| Weight (kg) | 61 ± 11 | 47 ± 5 |
| Height (cm) | 166 ± 7 | Arm span 159 ± 8<br>Cobb angle 106 ± 6.3 |
| Residual volume (% teoric value) | 102 ± 7 | 92 ± 21 |
| Forced vital capacity (% teoric value) | 103 ± 5 | |
| Vital capacity (% teoric value) | | 31 ± 8 |

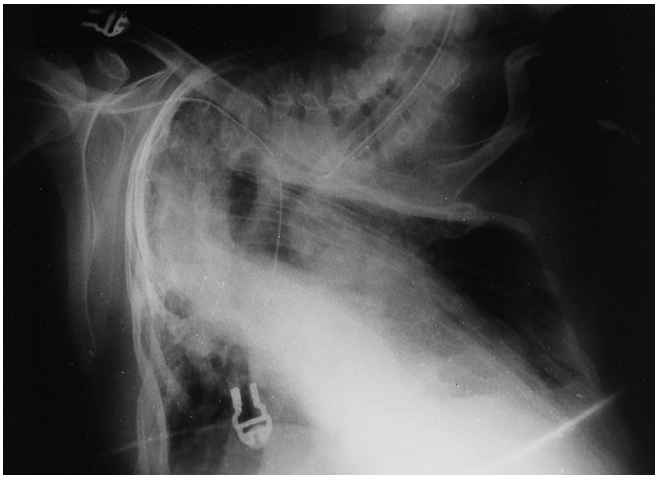

**Fig. 1** Radiologic evidence of the severe spinal deformity in one of our patients caused by KS

## Patients and methods

Seven consecutive patients with an acute exacerbation of chronic restrictive pulmonary disease due to severe idiopathic KS were enrolled in this study, after informed consent was obtained from the patients' legal guardian. Main patient data are reported in Tables 1 and 2. The study was approved by our institutional ethics committee.

The diagnosis of KS was confirmed on the basis of clinical, radiologic, and spirometric data showing a severe reduction of total lung capacity and vital capacity, with relatively well preserved values for residual volume. Figure 1 shows radiologic evidence of KS in one of our patients. All patients had a history of repeated respiratory tract infections in the decade preceding ICU admission; no patient had had a tracheostomy or received mechanical ventilation. Spine deformity was assessed by measuring on an anteroposterior chest radiograph the Cobb angle, which in all patients was > 90° (Table 1). In six of seven patients the cause of the acute exacerbation was a bronchopulmonary infection, while in one patient no clear cause was found.

After admission to the ICU, none of the patients improved after receiving standard treatment with oxygen, inotropic drugs, diuretics, and antibiotics; in view of the deterioration in gas exchanges values and the clinical condition, they were intubated tracheally via the nasal route (8–8.5 mm i.d.), and mechanically ventilated (Servo 900C, Solna Sweden) in control mode with the following setting: inspiratory:expiratory ratio 1:2, fractional inspired oxygen ($FIO_2$) 0.3–0.5, tidal volume ($V_T$) 10 ml/kg, respiratory rate (RR) 18 beats/min, square wave; no PEEP was administered until the end of the study.

The patients were studied between 3 and 5 h after intubation while in a semirecumbent position with the head in the median position. All patients were sedated (Ramsay score V class) and well adapted to the ventilator, without spontaneous triggering activity.

Six patients (ASA 1) undergoing minor abdominal surgery under general anesthesia with controlled mechanical ventilation ($V_t$: 10 ml/kg; $FIO_2$ 0.5; square wave; RR 15 beats/min; no PEEP) served as a control for the respiratory mechanics data. Respiratory mechanics was evaluated in stable conditions, 15 min after induction of anesthesia, with the same procedure as used in KS patients. Airflow was measured with an n.2 heated pneumotachograph (Metabo, Switzerland) connected to a differential pressure transducer (MP45, Validyne, Calif., USA) and connected between the Y-piece of the ventilator circuit and the patient connectors. Airway pressure was measured at tracheal level by a pressure transducer (Bentley Trantec, Irvine, Calif., USA) connected to a 1.5-mm catheter with multiple lateral holes inserted into the tracheal tube. The

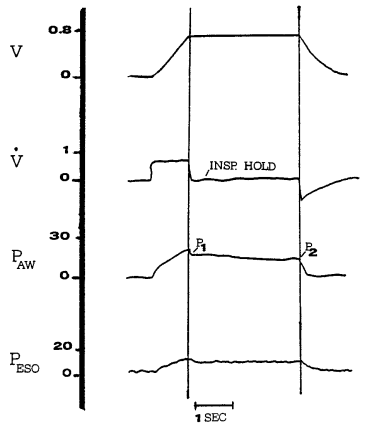


**Fig. 2** Representative tracing of flow $\dot{V}$ (l/s), volume $V$ (l), airway pressure $P_{AW}$ ($cmH_2O$), and esophageal pressure $P_{eso}$ ($cmH_2O$) changes during end-inspiratory airway occlusion (see explanation in patients and methods section) in a KS patient

catheter tip was positioned 2 cm from the carinal end of the tube, as described in a previous paper [8]. Volume was measured by integration of the airflow signal (Gould integrator, France).

In six of seven KS patients we attempted to measure esophageal pressure by inserting an esophageal balloon (8 cm long, 2.5 cm circumference) sealed over a 2-mm i.d. catheter and connected to a Spectramed pressure transducer. Probably because of the profound thoracic deformation, correct positioning of the balloon in the lower third of the esophagus was achieved in only five patients and was confirmed by an acceptable occlusion test [9] in four patients.

All signals were recorded with a multichannel Roche polygraph at a speed of 12.5 mm/s. During the study, we performed continuous electrocardiography and monitored heart rate, systemic arterial pressure, right atrial pressure (Siemens Sirecust, Sweden), and pulse oximetry (Ohmeda Biox, France).

After careful airway suctioning, during controlled mechanical ventilation the inspiration and expiration hold buttons on the ventilator were used to obtain a 3-s end-inspiratory and end-expiratory airway occlusion [7]. All corrections for the occlusion time of the Servo 900c scissor valves were made according to Kochi et al. [10]. End-inspiratory airway occlusion was followed, on airway pressure tracings, by a biphasic pressure drop from peak inspiratory pressure ($P_{max}$) to the elastic recoil pressure of the respiratory system (P2; Fig. 2); a pressure value at the end of the initial rapid pressure drop was observed (P1). The ohmic resistance of the respiratory system (RRSmin) was calculated as $(P_{max} - P1)/\dot{V}i$. No correction for endotracheal tube resistance was done, as airway pressure was measured 2 cm below the proximal end of the tube [8]. The total resistance of the respiratory system (RRSmax), including the sum of RRSmin and the additional respiratory impedance, generated by stress relaxation and time constant inhomogeneities, was calculated as $(P_{max} - P2)/\dot{V}i$. The difference between $RRS_{max}$ and $RRS_{min}$ (representing thoracic viscoelastic resistance) was defined DRRS.

End-expiratory airway occlusion was maintained for 3 s, obtaining a plateau: the expired $V_T$ was divided by the difference between end-inspiratory and end-expiratory occlusion airway pressure to calculate respiratory system compliance (CRS).

In the four patients in whom a positive occlusion test confirmed the reliability of esophageal pressure tracings, the elastance of the chest wall was determined as the difference between end-inspiratory and end-expiratory plateau esophageal pressure divided by the inspired $V_T$. By subtracting this value from respiratory system elastance, we evaluated lung elastance and its reciprocal, lung compliance (CL). The resistance of the chest wall (Rcw) was calculated as ($P_{max}$ eso – P2 eso/$\dot{V}i$). As no rapid pressure drop (i.e., P1) was identified in the Peso tracings after end-inspiratory occlusion, RRSmin was considered to reflect essentially $RRSmin_L$, and therefore $RRSmax_L$ was computed as the sum of RRSmin + $DRRS_L$. $DRRS_L$ was obtained by subtracting Rcw from DRRS.

After the basal measurements were obtained, in the five KS patients who showed at least a 50% increase in RRS min in comparison with the control group, we evaluated the reversibility of RRSmin after a methylxanthine infusion: doxophylline was infused at the dosage suggested by Poggi et al. [11] and validated in mechanically ventilated patients affected by chronic airway obstruction. A dose of 6 mg/kg doxophylline was injected over 10 min and respiratory mechanics data evaluated 15, 30, and 45 min after the infusion (Table 5).

Basal data and respiratory mechanics data obtained after the doxophylline infusion were compared with the Friedman nonparametric two-way ANOVA test for repeated measures. The Wilcoxon rank test was used to evaluate the difference between two sets of data; $p$ values $< 0.05$ were considered statistically significant.

**Table 2** Mean (SD) values of gas exchange variables and maximum inspiratory pressure *MIP* in patients with kyphoscoliosis before intubation ($PaO_2$, $PaCO_2$ partial pressure of $O_2$ and $CO_2$ in arterial blood, $FIO_2$ fractional inspired oxygen)

| $PaO_2/FIO_2$ | pH | $PaCO_2$ (mm Hg) | MIP ($cmH_2O$) |
|---|---|---|---|
| 199 ± 33 | 7.26 ± 0.4 | 50 ± 3.2 | 13 ± 3.4 |

**Table 3** Total respiratory system mechanics in control patients and in patients with kyphoscoliosis (*CRS* static compliance, *RRSmax* maximum resistance, *RRSmin* minimum resistance, *DRRS* gradient between RRSmax and min, *PEEPi* intrinsic PEEP)

| | Control patients | Patients with kyphoscoliosis |
|---|---|---|
| CRS ($ml/cmH_2O$) | 58 ± 8.5 | 36 ± 10.2* |
| RRSmax ($cmH_2O$/l per s) | 4.5 ± 1.2 | 20 ± 3.1* |
| RRSmin ($cmH_2O$/l per s) | 2 ± 0.5 | 6.2 ± 1.2* |
| DRRS ($cmH_2O$/l per s) | 2.4 ± 0.7 | 14 ± 2.6* |
| PEEPi ($cmH_2O$) | 0 | 1.8 ± 1.5 |

* $p < 0.01$

**Table 4** Mean values of chest wall *Ccw* and lung *CL* mechanics in four patients with kyphoscoliosis. Reference values from Pelosi et al. [12]

| | Patients with kyphoscoliosis | Control patients |
|---|---|---|
| Ccw ($ml/cmH_2O$) | 84 ± 8.2 | 178 ± 11 |
| CL ($ml/cmH_2O$) | 66 ± 7.2 | 107 ± 5.8 |
| $Rmax_L$ ($cmH_2O$/l per s) | 17 ± 2.1 | 3.2 ± 1.4 |
| $Rmax_{CW}$ ($cmH_2O$/l per s) | 2.8 ± 0.4 | 1 ± 0.4 |
| $DRRS_L$ ($cmH_2O$/l per s) | 11 ± 1.6 | 1 ± 0.6 |
| $RRSmin_L$ ($cmH_2O$/l per s) | 6 ± 1.3 | – |

**Table 5** Modifications of respiratory mechanics variables 15 min, 30 min, 45 min after the infusion of doxophylline in five patients with basally increased RRSmin

| | Basal | 15 min | 30 min | 45 min |
|---|---|---|---|---|
| CRS (ml/cm$H_2O$) | 36 ± 10 | 37 ± 8 | 36.5 ± 10.2 | 36 ± 9.5 |
| RRSmax (cm$H_2O$/l per s) | 23 ± 6.3 | 22 ± 7 | 23 ± 6.5 | 23 ± 7 |
| RRSmin (cm$H_2O$/l per s) | 6.6 ± 0.7 | 5.8 ± 1 | 5.7 ± 0.9 | 5.7 ± 1 |
| DRRS (cm$H_2O$/l per s) | 17 ± 6.5 | 17 ± 6 | 17.5 ± 5.8 | 17 ± 5.7 |
| PEEPi (cm$H_2O$) | 1.5 ± 1.2 | 1.2 ± 1 | 1.2 ± 1 | 1.2 ± 0.9 |

## Results

The mean values for respiratory system mechanics in KS and control patients under basal conditions are summarized in Table 3.

In the early phase of mechanical ventilatory support and before the administration of PEEP, KS patients showed an important reduction of CRS to values 30% lower than the control group. Both RRSmax and RRSmin were higher, suggesting an increase in both the ohmic and the viscoelastic resistive component of the respiratory system. All KS patients showed RRSmin values under basal conditions that were higher than control patients and the values for five out of seven patients were > 50% higher than reference values [12]. The mean values of basal RRSmax were also three or more times greater than the values in the control group and those reported in the literature [12].

PEEPi was present in six of seven KS patients, with values ranging from 1 to 4.5 cm$H_2O$ (mean 1.8 ± 1.5 cm$H_2O$). Only two patients exhibited residual expiratory flow at the beginning of the next inspiratory phase, suggesting the presence of a moderate flow limitation.

The mean ratio of the partial pressure of oxygen in arterial blood ($PaO_2$) and $FIO_2$ in KS patients was 199 ± 33. No significant correlation was found between RRSmin, RRSmax, CRS values, and the $PaO_2/FIO_2$ ratio. Infusion of doxophylline did not cause significant changes in $P_{max}$, PEEPi, RRSmin, RRSmax, and CRS values. Finally, no significant correlation was observed between the Cobb angle and vital capacity values and the respiratory mechanics variables. In the four KS patients in whom reliable data on chest wall mechanics were confirmed with an occlusion test [9], we observed a dramatic decrease of chest wall compliance (Table 4).

All except one patient were discharged in stable clinical conditions; two patients received mechanical ventilation at home.

## Discussion

The aim of the present study was to investigate the early changes in the mechanical properties of the respiratory system in a group of patients affected by severe KS requiring mechanical ventilation for acute respiratory failure. The patients were therefore evaluated during the first few hours of mechanical ventilation and before PEEP was started. A group of ASA1 patients was evaluated as a control group for normal respiratory mechanics data.

We found that (1) the acute exacerbation of KS was characterized by a dramatic decrease of CRS together with an increase in the resistance of the respiratory system (RRSmax, RRSmin); (2) PEEPi was present in all KS patients, but no patient showed values > 4.5 cm$H_2O$; (3) the administration of doxophylline at doses capable of producing a significant reduction in airway resistance in patients on mechanical ventilatory support for acute exacerbations of chronic airway obstruction [11] did not modify respiratory system mechanics. A decrease of CRS, causing an increase of the work of breathing, is considered a hallmark of severe KS in stable conditions, but few data are available from acute exacerbations requiring mechanical ventilatory support.

Sinha and Bergofsky [4] measured, in six patients (three affected by idiopathic and three by postparalytic KS), dynamic compliance and total resistance of the respiratory system. They reported decreased values for dynamic lung compliance but only slightly increased values for resistance; conversely, we found in our KS patients a dramatic increase of RRSmax. According to the studies of Bates et al. and D'Angelo et al. [13, 14], RRSmax represents the sum of airway resistance (RRSmin) and the resistive loads produced by stress relaxation of the respiratory system (including the chest wall) and the time constant inhomogeneities. However, separating RRSmax into RRSmin and DRRS suggests that the observed increase in the resistive load was mainly due to an increase of tissue resistance and only marginally to a reduction of airway caliber.

Concerning respiratory system tissue resistance (DRRS), we observed an important increase under basal conditions (14 ± 2.6 cm$H_2O$/l per s) to values six times greater than the control values and three times greater than the values that Pesenti et al. [8] observed in ARDS patients. Also, the values reported by Broseghini et al. in ARDS patients were lower (7.5 ± 1.9 cm$H_2O$/l per s) [15]. This dramatic increase in the viscoelastic load can probably be explained by the simultaneous reduction in both chest-wall compliance (due to kyphoscoliosis) and lung compliance (mainly attributable to the presence of atelectasis, edema, and infectious consolidation). Moreover, important phenomena of pendelluft between the lungs with different time constants can be also expected, given the severe anatomic alteration in the conductive airways and the parenchyma.

In this study, values for RRSmin, mainly representing airway resistance, were moderately increased; however, the administration of doxophylline did not modify RRS-

min or dynamic hyperinflation, despite infusion of doses capable of reducing these variables appreciably in patients affected by chronic airway obstruction. The complete lack of response to the infusion of a bronchodilator suggests that the increase in RRSmin in this group of KS patients may have been induced by irreversible mechanisms, such as the airway deformity, flooding of the airway with edematous fluid and secretions, and a reduction in the number of aerated areas in the lung due to atelectasis or consolidation. This can be confirmed by the presence in all our KS patients of radiologic evidence of a deformed airway architecture together with atelectasis and consolidation (see Fig. 1, for example). These data support the evidence that, in stable conditions as well, KS patients seldom show evidence of small airway disease [6].

As Lisboa et al. [6] have reported, the marked thoracic deformity of KS patients makes it difficult to assure adequate positioning of the esophageal balloon, and therefore we were able to obtain reliable chest-wall mechanics data in only four KS patients; however, these data confirm the importance of chest-wall deformation in causing an appreciable increase in the inspiratory workload. In particular, values for both chest-wall mechanics and lung mechanics showed a dramatic decrease. The mean chest-wall compliance in our KS patients was much lower than that reported by Pelosi et al. [12], both in normal anesthetized and paralyzed patients and in ARDS patients ($84 \pm 8.2$ ml/cm$H_2O$ vs $178 \pm 43$ ml/cm$H_2O$ and $101 \pm 47$ ml/cm$H_2O$, respectively) using the same technique we used in this study. Also, $Rmax_{CW}$ was increased in comparison to that reported by Pelosi et al. [12] in normal subjects ($2.8 \pm 0.4$ vs $1 \pm 0.4$ cm$H_2O$/l per s).

An important reduction of respiratory system compliance and both its components, lung compliance and chest-wall compliance, has been confirmed by Baydur et al. [16] in a group of young (mean age $24.7 \pm 2.1$ years) KS patients during spinal corrective surgery under general anaesthesia and mechanical ventilation. They reported values for CRS and lung and chest-wall compliance that were similar or slightly inferior to those we found in our KS patients. Although the patients in the Baydur study were not in acute respiratory failure, the similar level of thoracic mechanical alterations can be explained by the important decrease in functional residual capacity, commonly observed in patients spontaneously breathing during general anesthesia, which has been explained, at least in part, by a change in the pressure-volume characteristics of the chest-wall, together with dorsal atelectasis [17, 18]. Moreover, the Baydur data were obtained in patients spontaneously breathing at a tidal volume much lower than our patients ($V_T$ 4–6 vs $V_T$ 10 ml/kg, respectively).

Lisboa et al. [6] have shown that in stable patients with advanced KS, inspiratory muscle function is significantly impaired, this contributing with thoracic rigidity to the progression to respiratory failure. By measuring transdiaphragmatic pressure (Pdi) during quiet breathing and during maximal inspiratory efforts, Lisboa et al. observed a reduction in respiratory muscle strength and a correlation between Pdi and abnormalities in gas exchange. Our data suggest that the additional mechanical alterations, mainly caused by an alteration in the viscoelastic components of the respiratory system, produced by infection, atelectasis, and fluid overload, could play a role in the rapid clinical deterioration, leading to acute respiratory failure and mechanical ventilation. It is interesting to note that in one of our KS patients on home ventilation, whose condition was stable 2 months after ICU admission, there was a dramatic improvement both in CRS and in RRSmax and DRRS. Thus, contrary to the case in patients with chronic obstructive pulmonary disease, the increase of airway resistance with PEEPi and dynamic hyperinflation seem to play only a secondary role.

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