Prone Positioning Unloads the Right Ventricle in Severe ARDS*

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Background: **Despite airway pressure limitation, acute cor pulmonale persists in a minority of ARDS patients. Insufficient airway pressure limitation, hypercapnia, or both may be responsible. Because prone positioning (PP) has been shown to be a safe way to reduce airway pressure and to improve alveolar ventilation, we decided to assess its effect on right ventricular (RV) pressure overload in ARDS patients.**

Methods: **Between January 1998 and December 2006, we studied 42 ARDS patients treated by PP to correct severe oxygenation impairment (Pa_{O_2}/fraction of inspired oxygen ratio, < 100 mm Hg). RV function was evaluated by bedside transesophageal echocardiography, before and after 18 h of prone-position ventilation. RV enlargement was measured by RV/left ventricular (LV) end-diastolic area ratio in the long axis. Septal dyskinesia was quantified by measuring short-axis systolic eccentricity of the LV.**

Results: **Before PP, 21 patients (50%) had acute cor pulmonale, defined by RV enlargement associated with septal dyskinesia (group 1), whereas 21 patients had a normal RV (group 2). PP was accompanied by a significant decrease in airway pressure and Pa_{CO_2}. In group 1, this produced a significant decrease in mean (± SD) RV enlargement (from 0.91 ± 0.22 to 0.61 ± 0.21) after 18 h of PP (p = 0.000) and a significant reduction in mean septal dyskinesia (from 1.5 ± 0.2 to 1.1 ± 0.1) after 18 h of PP (p = 0.000).**

Conclusion: **In the most severe forms of ARDS, PP was an efficient means of controlling RV pressure overload.** ***(CHEST 2007; 132:1440–1446)***

Key words: acute cor pulmonale; ARDS; echocardiography; prone position; right ventricle

Abbreviations: ED = end-diastole, diastolic; ES = end-systole, systolic; F_{IO_2} = fraction of inspired oxygen; LV = left ventricle, ventricular; LVEDA = left ventricular end-diastolic area; PEEP = positive end-expiratory pressure; PP = prone positioning; RV = right ventricle, ventricular; RVEDA = right ventricular end-diastolic area; TR = tricuspid regurgitation

From a physiologic point of view, there is a major mismatch between lung ventilation and lung perfusion in ARDS patients.[1] From a clinical point of view, extensive lung damage in ARDS patients produces severe hypoxemia, which is difficult to control by mechanical ventilation. In the supine position, this lung damage mainly concerns dependent areas of the lung.[2] Prone positioning (PP), a respiratory strategy employed for > 20 years,[3] has repeatedly proven to be an efficient way to improve oxygenation in ARDS patients. A possible mechanism of this improvement can be summarized as follows. In the supine position, the posterior part of the chest wall, lying on the plane of the bed, is less compliant, and ventilation is thus mainly distributed to the anterior, and nondependent, lung areas. At the same time, lung perfusion is affected by a vertical hydrostatic pressure gradient, favoring blood flow in the depen-

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dent lung areas. The net result of both combined is a tendency for the dependent areas to be underventilated and overperfused (shunt), whereas the nondependent areas have a tendency to be overventilated and underperfused (dead space). In the prone position, the compliance of the anterior part of the chest wall, lying now on the plane of the bed, is reduced, favoring a better distribution of the ventilation to the posterior areas of the lung, which are now nondependent. At the same time, the hydrostatic pressure gradient favors a better distribution of blood to the anterior areas of the lung, now dependent. The net effect of both combined is a better match between lung ventilation and lung perfusion, thus reducing both the shunt and dead space. Such matching improvement is expected to reduce resistance to blood flow in the lung, which is a major afterloading factor for the right ventricle (RV) and is functionally affected during the course of ARDS.[4,5]

In January 1998, we started a prospective study focused on patients with severe ARDS who were treated by PP to improve oxygenation. All of these patients were monitored daily by transesophageal echocardiography, our usual strategy in ARDS monitoring.[6] We have now recorded a complete data set, including respiratory and echocardiographic measurements, in 42 patients. This gives us the unique opportunity to report the short-term effect of PP on RV function in patients with severe ARDS. Both PP and bedside transesophageal echocardiography have been accepted as a routine strategy in ARDS patients by the Ethics Committee of the "Societé de Réanimation de Langue Française."

Materials and Methods

Within the ARDS population treated between January 1998 and December 2006, we selected those patients meeting the following inclusion criteria: (1) patients with "severe" ARDS, leading to a $Pa{O_2}$/fraction of inspired oxygen ($F{IO_2}$) ratio of < 100 mm Hg after 48 h of respiratory support with our "low-stretch" respiratory strategy; and (2) treatment by PP during the first week of respiratory support.

Mechanical Ventilation and PP

Our low-stretch strategy has been extensively described elsewhere.[7] Briefly, we use a controlled mode with a limited airway pressure (plateau pressure, < 30 cm H_2O), a low respiratory rate, and a positive end-expiratory pressure (PEEP) determined by the criteria of Suter et al.[8] Tidal volume, as the consequence of these choices, differed considerably from patient to patient (from 4 to 10 mL/kg measured body weight; average, 8 mL/kg). Patients were sedated with a continuous IV infusion of midazolam and sufentanil, and were paralyzed by cisatracurium besylate, which is our usual strategy during the first 48 h of respiratory support in ARDS patients.

With this low-stretch strategy, PP was systematically implemented after 48 h of respiratory support in patients with severe ARDS.[7] We used the method described by Chatte et al.[9] During PP, we never use thoracopelvic support, and the anterior walls of the thorax and the abdomen lie directly on the plane of the bed. Each PP session lasts 18 h per day and is repeated over 3 to 4 days, thus restoring supine ventilation at the end of the first week of respiratory support. In these selected cases, sedation and curarization were pursued throughout PP. In hypercapnic ARDS patients, we also systematically used a heated humidifier, instead of a heat and moisture exchanger.[10]

Echocardiographic Examination

During the first days of respiratory support, bedside echocardiographic evaluations of left and RV function by a transesophageal approach are performed daily in all ARDS patients who are receiving mechanical ventilation in our unit.[6] All echocardiographic studies were recorded on videotape, permitting computer-assisted measurements, and all recordings are kept in our videotape bank. In the present study, we compared echocardiographic measurements that were performed with patients in the supine position, just before the first PP session, which lasted 18 h, and was repeated just after; thus, also with the patient in supine position too.

We used a wide-angle, phased-array digital sector scanner (Corevision, model SSA-350A; Toshiba; Otawarashi, Japan; or Sequoia C 256; Siemens Medical Solutions; Malvern, PA), equipped with a multiplane transesophageal transducer. Using the signal from the respirator, airway pressure was displayed on the screen of the echo-Doppler device, accurately timing cardiac events during the respiratory cycle and permitting the selection of an end-expiratory beat for measurements.

During an echocardiographic study, we first examined a long-axis, four-chamber view of the cardiac chambers. From this view, left ventricular (LV) and RV areas were measured at end-diastole (ED) and at end-systole (ES). From these measurements, we calculated the RV ED area (RVEDA)/LV ED area (LVEDA) ratio. RV dilatation was defined by an RVEDA/LVEDA ratio of > 0.6. LV ED and LV ES long axes were measured as the distance from the apex to the midpoint of the mitral valve ring, and LV volumes were calculated using the single-plane, area-length formula. Ejection fraction was calculated as (LVEDV − LVESV)/LVEDV. During echocardiographic study by this approach, we also noted the absence or the presence of tricuspid regurgitation (TR) using color Doppler, and the severity of TR was graded from digital planimetry of the area of color-coded regurgitant flow. An area < 2 cm^2 was considered as mild TR (+), and an area between 2 and 4 cm^2 as moderate TR.[11]

A short-axis cross-sectional view of the LV at the mid-papillary muscle level was obtained secondarily by a transgastric approach. From this view, septal motion at ES/early diastole was carefully examined to detect any systolic septal dyskinesia, and this abnormality was quantified by measuring the eccentricity index of the LV chamber at ES.[12] Acute cor pulmonale was defined by the combination of RV enlargement with septal dyskinesia.[13] Finally, the measurement of aortic diameter and continuous Doppler recording of aortic flow by a specific approach were used to quantify cardiac output.[14]

Statistical Analysis

Statistical calculations were performed using a statistical software package (Statgraphics Plus package, version 5; Manugistics; Rockville, MD). The data are expressed as the mean ± SD. Paired comparison of quantitative variables were performed using a Wilcoxon signed rank test, whereas a χ^2 test was used to compare qualitative variables. A p value of < 0.05 was considered to be statistically significant.