RESPIRATORY MECHANICS AND SURVIVAL IN PATIENTS WITH ACUTE CARDIOGENIC PULMONARY EDEMA

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BACKGROUND: The importance of respiratory mechanics (RM) as prognostic factor has been stated in patients with ARDS, and recently, in patients with severe COPD. However, in patients with cardiogenic pulmonary edema (CPE) treated with mechanical ventilation only cardiovascular factors have been related to final prognostic (1), but RM has not been systematically studied.

OBJECTIVE: To study RM in patients with CPE and to determine if there is any difference between survivor and nonsurvivor.

METHODS: 25 patients ventilated for acute CPE and 10 postsurgical without cardio-pulmonary pathology (selected for the control group) were studied. In every patient we obtained the static inflation compliance of the total respiratory sistem (Cst,rs), maximum respiratory resistance (Rrsmax) and intrinsic PEEP (PEEPi); we also calculated the work of chest inflation (Wi) performed by the ventilator and its resistive (Waw) and elastic (Wst,rs and W_{PEEPi}) components by simple geometric analysis (2,3). Data collected included patient demographics, SAPS-II score, causes of CPE, blood pressure, vasopressor and vasodilation medication and arterial blood gas on mechanical ventilation.

RESULTS: Patients with CPE had higher Rrsmax, PEEPi and Wi, but lower Cst,rs than patients of the control group (p<0.05). 12 patients with CPE died (48%) (group I) and 13 patients achieved hospital discharge (group II). Patient demographics, SAPS-II score and blood gas were similar in both groups. Patients from group II had higher PEEPi, Rrsmax, Wi, WPEEPi and Waw than patients from group I (p<0.05); Cst,rs was similar in both groups. Incidence of shock and treatment with vasopressor at the moment of tracheal intubation were higher in patientes from group I (p<0.05).

CONCLUSIONS: 1) Chest inflation impedance (Wi) was higher in patients with CPE. 2) Patients with CPE and higher Wi had better prognostic. 3) Patients with CPE who died had higher incidence of shock (83% vs 23%) and lower Wi; the cause of respiratory failure in these patients was probably due to pulmonary edema and perhaps to some component of muscular respiratory fatigue because of shock.

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