Interaction between Heart and Lung

Edited by Severin Daum

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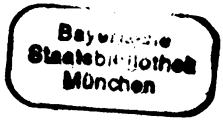

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"Paradoxic" Septal Motion during PEEP Ventilation

H. Forst, B. Zwißler, J. Racenberg, M. Menger, K. Meßmer

Introduction

Mechanical ventilation with positive endexpiratory pressure (PEEP) often is associated with a fall in cardiac output and stroke volume. Not all the mechanisms leading to this serious side-effect are well understood. While the reduction in venous return resulting from the elevated intrathoracic pressure is widely accepted as the predominant causative factor, the role of a direct tamponade of the heart by the lungs (1), and of the effects of increased pulmonary vascular resistance compromising right ventricular systolic function (2) remain to be elucidated. Furthermore, the importance of direct mechanical interaction of both ventricles, mediated by the commonly shared interventricular septum and enhanced by the non-compliant pericardium, has not been clarified yet (3, 4).

For this reason a study was designed in dogs to answer the following questions:

1. Does PEEP ventilation influence the dynamic geometry of the left ventricle as a consequence of ventricular interference?
2. Does the expanding lung alter the diastolic configuration of the heart by direct mechanical interaction (cardiac "tamponade")?

Material and Methods

The studies were performed in anesthetized dogs, using sonomicrometry and 2-dimensional transoesophageal echocardiography to assess the dynamic geometry of the heart. Pressures in both ventricles were measured by micromanometers, cardiac output was determined by the thermodilution technique. After a right-sided thoracotomy and after pericardiotomy a miniaturized piezoceramic transducer was inserted through the tract of a 16-gauge needle into the interventricular septum. Fcur corresponding transducers sutured to the epicardial surface of the right and left ventricular walls allowed continuous and simultaneous measurement of the septal-lateral (s.-l.) diameter of the right and left, and of the anterior-posterior (a.-p.) diameter of the left ventricle. Intrathoracic pressure was measured by a micromanometer fixed at the level of the right atrium on the pericardial surface. In all animals isovolemic hemodilution with dextran 60 was performed (mean het 29%) to obtain blood for volume expansion during PEEP. After closure of the pericardium and airtight closure of the chest the endexpiratory pressure was raised to 10, 15, and 20 cm H_2O and lowered to 10 and 0 H_2O respectively. PEEP levels 10, 20, and 0 were held for a 20 minutes period. To eliminate the well known effects of impaired venous return, the transmural right ventricular filling pressure (i.e. central venous pressure minus intrathoracic pressure) was kept constant by blood transfusion during the PEEP intervention. In a second series of experiments a short axis view of the heart was imaged by 2-D echocardiography prior and after induction of PEEP, with the probe inserted via the oesophagus and sutured

to the pericardium to avoid changes of the tomographic plane due to motions of the heart within the thorax.

Results

With stepwise increase of PEEP cardiac output (CO) and stroke volume (SV) decreased significantly (at PEEP 20: CO –21% and SV –31%) despite volume expansion (14 ml/kg body weight) and in presence of a constant transmural filling pressure. Thus, factors other than reduced preload have to be considered to explain the side effects of PEEP.

With increasing levels of PEEP right ventricular diameter increased (at PEEP 20: +6%), while left ventricular s.-l. diameter decreased concomittantly (–9%). The a.-p. diameter of the left ventricle remained unaffected. To demonstrate changes in ventricular dynamic geometry the ventricular pressure-diameter relationship was recorded at 2 ms intervals and pressure-diameter loops were constructed from single cardiac cycles. At control conditions the pressure-diameter loops of the s.-l. and the a.-p. axis of the left ventricle revealed a configuration, which is well known from pressure-volume loops (5).

During PEEP, however, the s.-l. loop was displaced to the left (indicating a decrease of the s.-l. axis) and revealed a marked deformation: the s.-l. diameter increased during early systole and decreased at the end of the isovolemic relaxation phase, thus suggesting "paradoxic" movement of the septum. The corresponding a.-p. loop did not show a comparable deformation: along this axis shortening during isovolemic contraction was even more pronounced with PEEP. From the pressure-diameter loops one can not differentiate conclusively, whether the rearrangement of dynamic geometry observed, was due to a paradoxic motion of the septum or of the left ventricular lateral wall. 2-D echocardiography verified diastolic flattening of the interventricular septum towards the left ventricle and abnormal systolic bulging of the septum, predominantly at the site of the right ventricular inflow tract when PEEP levels were greater than 10 cm H_2O.

Conclusion

These findings suggest that the shift of the interventricular septum to the left observed during diastole is caused by a dilatation of the right ventricle. There was no evidence for a tamponade of the left ventricle by the lung. Thus, PEEP induces a rearrangement of left ventricular dynamic geometry, characterized by a "paradoxic" motion of the septum, which could partly account for the hemodynamic side-effects of PEEP. Enthusiastic volume expansion during PEEP guided by pressure monitoring only, may aggravate these phenomena and result in a deterioration of cardiac performance.

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