Significance of tricuspid valve dysfunction as a consequence of one-lung ventilation

Editor—Chance echocardiographic observations of tricuspid valve dysfunction link lung separation devices and alveolar-capillary membrane damage. A hypothesis for a mechanism, which is independent of oxygen flux, is proposed.

Twenty-nine patients [17 male/12 females: 21–82 (mean 66) yr; 45–87 (mean 65) kg], recruited and fully consented, were investigated. Only one patient was rated ASA I, 21 (72%) rated ASA II, and 7 (24%) ASA III. Two were video-scopic procedures—the remainder, open thoracotomy. We used transoesophageal echocardiography (TOE) to define pulmonary vascular resistance changes, as first observed in a patient with alveolar proteinosis. In 23 (79%) cases, tricuspid regurgitation was noted during two-lung ventilation with an \( F_\text{IO2} \) 1.0 with patients in the supine position. The application of a one-lung ventilation test by clamping the tracheal lumen of a double lumen tube resulted in the observation being noted in 25 (86%) cases, with evidence of worsening in some. Observations were recorded variously as trivial or moderate, or as a gradient from 6.7 to 35.3 mm Hg. Only in four cases (13%) was no regurgitation evident.

The observed valvular changes are physiological rather than measurement artifact. Dynamics are quantifiable, but as the research design and echocardiographic axes were directed to contour analysis of the right ventricle and not valvular mechanics, an accurate measure of the degree of regurgitation could not be made. As the records are mixed, we are unable to use standard statistical analyses. We are uncertain of any patho-physiological significance of these observations, particularly as the clinical process was not influenced by the information and there were no haemodynamic (HR, BP, and \( S_P\text{ao2} \)) consequences.

It is evident that the observations are a part record of a progression set in motion by intubation. Double-lumen tubes—as opposed to other lung separation devices—not only induce pressor responses on insertion, but also can generate intrinsic PEEP sufficient to cause dynamic hyperinflation.

Tricuspid regurgitation is a common observation with a TOE probe. However, observations tend to have been viewed uncritically. The incidence in this group suggests that tricuspid valve changes may be the norm with a double-lumen tube. Therefore, it is difficult to define a population against which to judge observations on a thoracic surgical case mix which has much pulmonary vascular co-morbidity. That the cause may be a pulmonary pressor response to foreign body insertion (TOE probes, tracheal tubes, etc.) has to be considered as some of the other study data on systolic pulmonary artery pressure and contour analysis are in keeping with increases in right ventricular afterload at associated points.

The finding that the tricuspid regurgitation worsened in some on the induction of one-lung ventilation also is considered significant. Tricuspid valve dysfunction is common in heart transplantation. Two-lung to one-lung ventilation change, by virtue of the intrathoracic pressure shifts brought about by positive pressure and applying hemithoracic ventilation, parallel the cardiac displacement and pericardium dynamic effects on valvular hysteresis in the transplanted heart.

Pulmonary vasoconstriction is a normal physiological stress response, part of ‘fight and flight’. Inserting and deploying a lung separation device causes pulmonary vasoconstriction with a pressor response and afterload increase. As our research was conducted with 100% oxygen, we contend that these effects, similar to those ascribed to hypoxic stimuli, are independent in origin and precede change in oxygen flux.

The right heart governs pressure and flow through the pulmonary circulation. Tricuspid regurgitation, as a normal reaction to the pressor response, would protect the alveolar capillary membrane from the kind of haemodynamic surge that may damage its integrity. If the integrity of alveolar-capillary membrane was breached by failure of or overload of such a mechanism, for instance, in extreme environments or during thoracic anaesthesia and surgery, this could be part of the pathogenesis of acute lung injury such as that of high altitude or post-pulmonary resection.

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