

[2007] [125] Effective arterial elastance as an index of pulmonary arterial load

**P. Morimont, B. Lambermont, A. Ghuysen, P. Lancellotti, V. Tchana-Sato, P. Kolh, V. D'orio, L.A. Pierard.**  
*CHU Sart Tilman, Cardiology Department, Liege, Belgium*

**Purpose:** This study tested whether the simple ratio of right ventricular (RV) end-systolic pressure to stroke volume, known as the effective arterial elastance (Ea), provides a valid measure of pulmonary arterial load in case of pulmonary hypertension induced by acute pulmonary embolism (PE) or septic shock (SS).

**Methods:** Ventricular pressure-volume data (obtained with conductance catheters) and invasive pulmonary artery pressure and flow were simultaneously determined in 2 groups of 6 pigs. Measurements were obtained at baseline, and each 30 minutes after injection of autologous blood clots (0.3g/kg) in the superior vena cava in the PE group and endotoxin infusion in the SS group. Two methods of calculation of arterial load were compared. One was derived from three-element Windkessel model of the pulmonary arterial system Ea(WK), and the other one was more simply measured as the ratio of RV end-systolic pressure to stroke volume [Ea(PV)].

**Results:** Mean pulmonary artery pressure increased from  $10.81 \pm 0.29$  to  $23.01 \pm 0.29$  mmHg ( $p < 0.001$ ) one hour after PE and from  $14.72 \pm 0.23$  to  $24.73 \pm 0.24$  mmHg ( $p < 0.001$ ) one hour after endotoxin infusion. Ea(WK) raised from  $0.33 \pm 0.03$  to  $0.78 \pm 0.03$  mmHg/ml ( $p < 0.001$ ) one hour after PE and from  $0.44 \pm 0.02$  to  $1.26 \pm 0.03$  mmHg/ml ( $p < 0.001$ ) one hour after endotoxin infusion. Similarly, Ea(PV) raised from  $0.57 \pm 0.03$  to  $0.96 \pm 0.03$  mmHg/ml ( $p < 0.001$ ) and from  $0.80 \pm 0.02$  to  $1.57 \pm 0.03$  mmHg/ml ( $p < 0.001$ ) one hour after PE and endotoxin infusion respectively. Combining all the data in each group and despite the simplifying assumptions of Ea(PV), the correlation between Ea(PV) and Ea(WK) remained excellent over a broad range of altered conditions. The linear relations were nearly identical in both groups and given by  $Ea(PV) = 0.27 + 0.95 Ea(WK)$  ( $r^2 = 0.96$ ,  $n = 58$ ,  $SEE = 0.07$ ,  $p < 0.0001$ ) in the PE group and  $Ea(PV) = 0.37 + 0.90 Ea(WK)$  ( $r^2 = 0.97$ ,  $n = 56$ ,  $SEE = 0.20$ ,  $p < 0.0001$ ) in the SS group. Ea(PV) was somewhat higher than Ea(WK) with mean difference being  $0.25 \pm 0.07$  mmHg/ml in the PE group and  $0.28 \pm 0.11$  mmHg/ml in the SS group, corresponding to a good agreement between the two methods in each group.

**Conclusions:** Ea(PV) provides a convenient, useful and easier method to assess pulmonary arterial load and its impact on the right ventricle. Unlike classical mean pulmonary resistance that ignores waves reflections, Ea includes the pulsatile nature of the load that can be prominent in pulmonary hypertension and lead to right heart failure.

**Citation:** European Journal of Heart Failure Supplements 2007; Vol. 6(1), page 31

**Date:** Sunday, June 10, 2007

**Session Info:** Pulmonary hypertension