A677 - A reappraisal of the effects of fluid administration on left ventricular loading conditions in critically ill patients

M Jozwiak \(^1\); S Millasseau \(^2\); C Richard \(^3\); X Monnet \(^3\); P Mercado \(^3\); F Dépret \(^3\); JE Alphonsine \(^3\); JL Teboul \(^3\); D Chemla \(^4\)

\(^1\)Hôpitaux universitaires Paris-Sud, Hôpital de Bicêtre, APHP, service de réanimation médicale; Inserm UMR S_999, Univ Paris-Sud, service de réanimation médicale, Le Kremlin-Bicêtre, France, \(^2\)Pulse Wave Consulting, Saint Leu La Foret, France, \(^3\)Hôpitaux universitaires Paris-Sud, Hôpital de Bicêtre, APHP, service de réanimation médicale; Inserm UMR S_999, Univ Paris-Sud, Le Kremlin-bicêtre, France, \(^4\)Hôpitaux universitaires Paris-Sud, Hôpital de Bicêtre, APHP, service de physiologie; Inserm UMR S_999, Univ Paris-Sud, Le Kremlin-Bicêtre, France

**Introduction:**
Understanding the effects of therapeutics on the left ventricular (LV) loading conditions is of utmost importance in critically ill patients. The effective arterial elastance (Ea=ESP/SV, where ESP is aortic end-systolic pressure and SV stroke volume) is a lumped parameter of arterial load that has been proposed as an index of LV afterload. We aimed at comparing the effects of fluid administration on ESP (i.e., the LV afterload in the pressure-volume phase-plane according to the classic “cardiocentric” framework) and on Ea.

**Methods:**
In 30 mechanically ventilated patients, we recorded Ea from the femoral peripheral systolic arterial pressure SAP (Ea=(0.9×femoral SAP)/SV) before and after the infusion of 500-mL of saline. Patients in whom fluid administration induced an increase in cardiac index (PICCO-2) ≥15% were defined as “responders”.

**Results:**
At baseline Ea (1.92±0.98 mmHg/mL) was positively related to total arterial stiffness TAS (r=0.95), to systemic vascular resistance SVR (r=0.89) and to heart rate HR (r=0.37) (each P<0.05). Fluid administration increased ESP (from 103±20 to 117±23 mmHg) and SV (from 64±26 to 73±24 mL) (each P<0.05). This resulted in a non-significant decrease in Ea (1.79±0.73 mmHg/mL). The changes in Ea were positively related to changes in TAS and SVR (each r=0.88; P<0.05) but not in HR. Most patients (90%) increased their ESP and the concordance rate between changes in ESP and Ea was 57%. In fluid responders, ESP increased and Ea decreased (each P<0.05). In fluid non responders, ESP increased (P<0.05) while Ea remained unchanged.

**Conclusion:**
Fluid administration increased LV afterload (ESP) if one relies on the classic “cardiocentric” framework, and this challenged the opposite conclusions supported recently on the basis of the slight decreases in Ea (“arterial load” framework). Ea taken in isolation is not an index of LV afterload in critically ill patients.