

01. DECREASED SYSTEMIC VENOUS RETURN

The increase in intrathoracic pressure, caused by the pressure increase in the airways on insufflation, leads to a decrease in systemic venous return. This diminution is more marked when the intrathoracic pressure generated is high and the patient's volemia is low. According to Guyton's concept, this has long been attributed to a decrease in the pressure gradient of the venous return, defined as the difference between the mean systemic pressure and the right atrial pressure. However, positive-pressure ventilation does not seem to alter this gradient, suggesting that the decreased venous return results from a diminution in venous conductance due to the interposition of collapsible veins between the peripheral venous circulation and the right atrium.

This hypothesis has been demonstrated in spontaneously breathing patients during

serious acute asthma when inspiration can result in the collapse of the inferior vena cava.

Film no. 3 : TTE – Subcostal view

Spontaneously breathing patient hospitalized for serious acute asthma.
At each inspiration, the inferior vena cava collapses where it crosses the diaphragm
Recently, we have confirmed this during mechanical ventilation, but at the superior vena cava (SVC) which is subject to intrathoracic pressure. On clamping of the inferior vena cava for hepatectomy, we first observed the occurrence of inspiratory collapse of the SVC, responsible for marked inspiratory decrease in the right ventricular stroke volume.

Film no. 4 : TEE recorded during hepatectomy.

The longitudinal view of the superior vena cava (SVC) does not visualize respiratory variations in its size. Pulsed Doppler in the trunk of the pulmonary artery (PA) does not reveal variations in right ventricular ejection

Film no. 5

In the same patient, clamping of the inferior vena cava by the surgeon resulted in inspiratory collapse of the superior vena cava (SVC), thereby causing a marked inspiratory drop in right ventricular ejection (*).
This collapse, defined as an inspiratory reduction of 60% in SVC diameter occurs when the transmural pressure of the vessel (PSVC - Pintrathoracic) falls below its closing pressure. This physiological description has a major clinical impact, as we have since shown that collapse of the SVC is an excellent index of hypovolemia in patients mechanically ventilated for septic shock and can be used to predict the efficacy of volume expansion.

Film no. 6 : TEE – View of the vessels at the base of the heart. PA: pulmonary artery, Ao: aorta

Patient ventilated for septic shock following pneumopathy. The TEE was done because of the persistence of arterial hypotension.
At each inspiration, there was collapse of the superior vena cava (SVC), which caused a cyclic decrease in right ventricular ejection (*).
This appearance is suggestive of hypovolemia and calls for volume expansion.

Film no. 7

In the same patient, after volume expansion, the cyclic variations in size of the superior vena cava (SVC) and of right ventricular ejection disappear.

Lastly, its presence depends not only on the volemia of the patient but also on the ventilation parameters, allowing adjustment of the volemia to a given ventilation, or even the ventilation to a given hemodynamic state.

Film no. 8 : TEE – View of the vessels at the base of the heart. PA: pulmonary artery.
Patient mechanically ventilated following pneumopathy.
In ZEEP, there are no significant inspiratory variations in superior vena cava (SVC) diameter or in right ventricular ejection.

Film no. 9
In the same patient, the application of a PEEP of 5 cmH2O, needed to correct marked hypoxemia, led to inspiratory collapse of the superior vena cava (SVC), which in turn caused a drop in right ventricular ejection (*) and a lowering of blood pressure.

Media

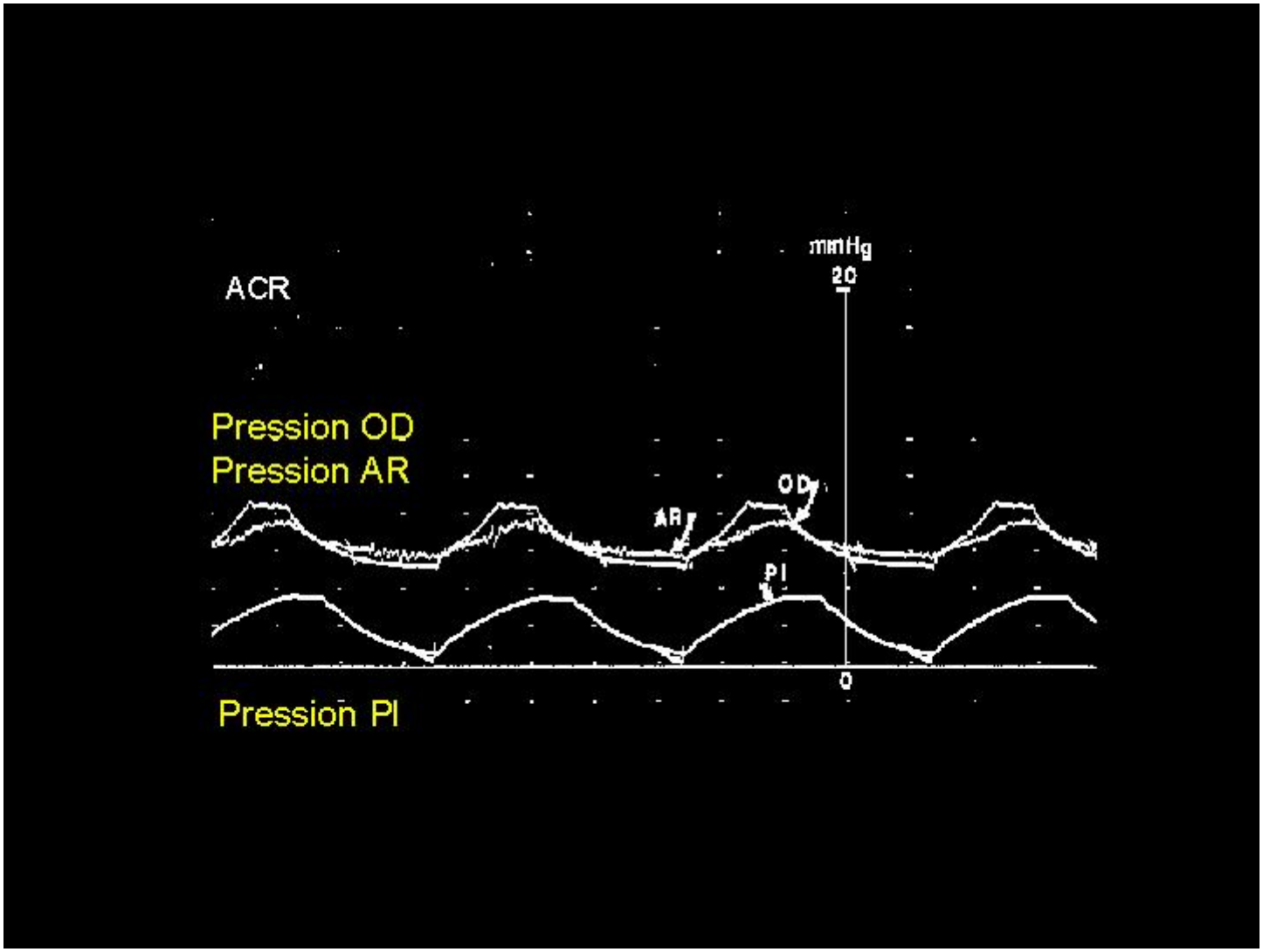


Figure 4 : Simultaneous recording of systemic arterial pressure (Pa), right atrial pressure (Pra) and pleural pressure (Ppl) in a mechanically ventilated patient, during cardiac arrest. The increase in pleural pressure, linked to the ventilation, is transmitted both to the systemic pressure and to the right atrial pressure.