SHARKS SUCK

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EXTENDED ABSTRACT ONLY – DO NOT CITE

Over the last 25 years, it has been all downhill for Schoenlein and Willem's (1894) idea that negative pericardial pressures established in a rigid pericardium are necessary for cardiac filling in elasmobranchs (see Lai et al., 1996). The lack of control of essential haemodynamic variables to inconsistencies in the shapes of recorded blood pressure and flow pulses, raises questions about the validity of some of these criticisms.

We looked at the effect of changing intrapericardial pressures on cardiac function in both unanaesthetized and anaesthetized dogfish (*Squalus acanthias*). The pericardial cavity was occlusively cannulated through the pericardio-peritoneal canal which allowed fine regulation of pericardial pressure by injection or withdrawal of dogfish Ringer solution. Cardinal sinus pressure, ventral aortic pressure and flow were measured in resting and swimming animals (up to 1 $m.sec^{-1}$) while pericardial cavity pressure was changed from $\pm$ 10 mm Hg. Profound changes in ventral aortic pressure and flow pulse shapes occurred when pericardial pressures were changed from – 2 to + 2 mm Hg (Fig. 1). Pressure and volume flow in the ventral aorta declined with increasing pericardial pressure.

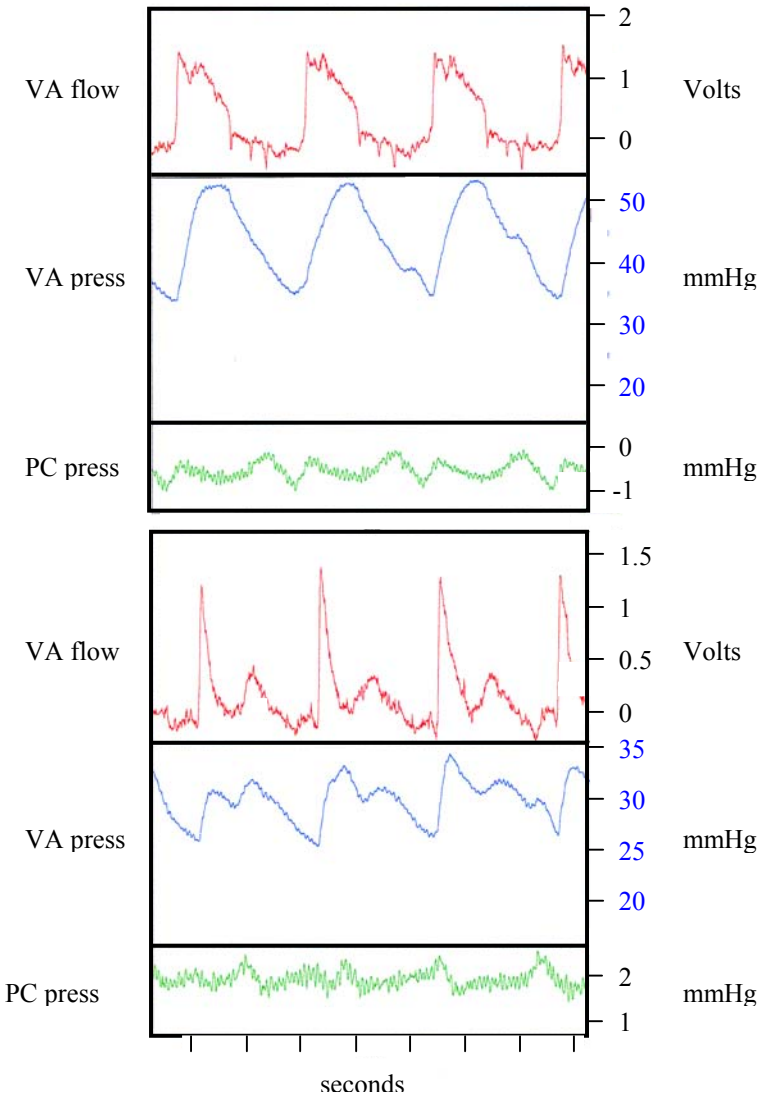
VA flow
2
1
0
Volts
VA press
50
40
30
20
mmHg
PC press
0
-1
mmHg
VA flow
1.5
1
0.5
0
Volts
VA press
35
30
25
20
mmHg
PC press
2
1
mmHg
seconds

Figure 1: Ventral aortic (VA) flow and pressure changes when pericardial cavity (PC) pressure was varied from –1 (upper panel) to +2 (lower panel) mmHg in a dogfish swimming at 1 m sec^{-1}.

In a further series of experiments on anaesthetized dogfish the heart was partially enclosed in a plastic pericardium. Cardinal sinus, atrial, ventricular and ventral aortic pressures were measured along with blood flow from atrium to ventricle and ventricle to ventral aorta. Sonomicrometry was simultaneously used to monitor changes in atrial and ventricular volumes. By changing pressure independently of volume and volume independently of pressure, we showed that pressure is the dominant force in cardiac filling. In a nutshell, atrial filling occured down a pressure gradient, that was greatly enhanced by negative intrapericardinal pressures. Ventricular pressure and ventral aortic flow varied with the degree of ventricular filling, which was solely achieved by atrial contraction. These data show that Schoenlein and Willem (1894) had in fact got to the heart of the matter.

References

Lai, N.C., J.B Graham, V. Bhargava and R. Shaetai. Mechanisms of venous return and ventricular filling in elasmobranch fish. {1996}. Am. J. Physiol. 270: H1766 – H1771.

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