CROSSTALK

Rebuttal from Daniel Beard and Eric Feigl

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Dr. Andrew's proposal (Andrew, 2013) to rename Guyton's venous return curve the 'venous pressure curve' seems unlikely to overcome the decades of misinterpretation and confusion that the Guyton model of the systemic circulation has engendered.

In Guyton's experiments venous return curves were produced with a pump connected from the right atrium to the pulmonary artery in an anaesthetized, open chest, horizontal, acute preparation. Paired data points of flow and right atrial pressure were obtained. Guyton fitted the data pairs with a four-parameter model, consisting of arterial and venous resistances and capacitances. The model fitted the data at different flow values *without a change in any of the parameter values.* The Guyton model with fixed parameter values does not pertain to the change in flow observed during heart failure where resistance and capacitance values do change. Thus in Dr Andrew's Fig. 1, heart failure does not proceed along a venous return curve with fixed parameters from point C to point D, but rather, over time from point C to point G. This is one reason that the inclusion of fixed parameter venous return curves in Dr Andrew's figure only adds confusion.

Compare the figure below with Dr Andrew's figure as an aid in teaching about heart failure.

The major cardiology texts (Topol, 2007; Crawford *et al.* 2010; Walsh *et al.* 2011; Bonow *et al.* 2012), and the leading medical student textbook (Lilly, 2011) do not utilize Guyton's venous return curves, indicating that Guyton's model is not important for an understanding of heart failure. This supports our thesis, that Guyton's model of the systemic circulation is unnecessary and confusing when teaching cardiovascular medicine (Beard & Feigl, 2011, 2013).

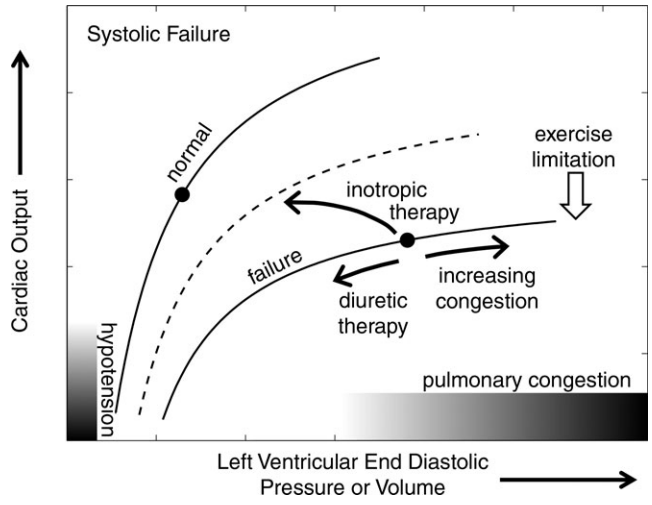

Figure 1
Idealized Sarnoff-Berglund ventricular function curves demonstrating the Frank-Starling property of cardiac muscle for normal and failing left ventricles. In systolic heart failure the curve is displaced downward and flattened. Therefore an increase in end-diastolic pressure during heart failure provides little improvement in cardiac output. Diuretic therapy will decrease pulmonary congestion but may not improve cardiac output. Inotropic therapy improves cardiac contractility, thus lessening pulmonary congestion and improving cardiac output. This simplified teaching scheme does not include the effects of changes in heart rate or systemic vascular resistance. (After Lilly, 2011.)

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