

Re: Measuring Contractility During Mechanical Circulatory Support Would Be a Strong Plus

To the Editor:

We have carefully read with great interest the letter to the editor entitled “Measuring Contractility on Mechanical Circulatory Support: If It Can Not Be Done, Does It Matter?” from McConnell. We definitely agree that end-systolic elastance (Ees) derived from end-systolic pressure–volume relationship (ESPVR) is a key parameter, which is difficult to precisely obtain outside experimental frameworks.¹ However, approximation of this parameter may be adequately used to analyze contractility changes in clinical settings; in this way, the aim of our report was to show discrepancy between left ventricular (LV) contractility and ejection fraction in heart failure.² As pointed out by McConnell, ESPVR is indeed curvilinear in extreme ranges.³ However, despite this curvilinearity, linear approximation is largely enough to make a decision and is commonly used in most clinical (and experimental) settings.^{4–8} Using veno-arterial extracorporeal membrane oxygenation (VA ECMO) flow to vary loading conditions is an easy way to assess interaction between LV contractility and arterial elastance. Indeed, if there is no significant aortic valve regurgitation, VA ECMO will change both pre- and after-load, impacting end-systolic pressure and volume and allowing to calculate at least two points of ESPVR. Of course, measurements must be performed during transient ramp-up (or -down). Otherwise, as clearly explained by McConnell, the heart will attempt to “recouple” by changing its contractility, but this is also the case in experimental settings if you impede venous return (or if you clamp the aorta). We do not claim that “noninvasive” Ees derived from aortic pressure combined with echocardiography can be used in place of Ees derived from ventricular pressure–volume loops. Of course, aortic pressure indirectly reflects LV pressure, but, again, this approximation is currently used in clinical settings.^{4–6,8} Finally, further studies should be performed to compare Ees derived from LV pressure–volume loops and “noninvasive”

Ees derived from aortic pressure combined with LV echocardiography obtained during transient changes in pump speed.

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References

1. McConnell PI, Sun BC: Pressure-volume analysis during axial flow ventricular assist device support. *J Heart Lung Transplant* 25: 256–257, 2006.
2. Morimont P, Lambermont B, Guiot J, et al: Ejection fraction may not reflect contractility. *ASAIO J*: 1, 2017.
3. Kass DA, Beyar R, Lankford E, Heard M, Maughan WL, Sagawa K: Influence of contractile state on curvilinearity of in situ end-systolic pressure-volume relations. *Circulation* 79: 167–178, 1989.
4. Guarracino F, Ferro B, Morelli A, Bertini P, Baldassarri R, Pinsky MR: Ventriculoarterial decoupling in human septic shock. *Crit Care* 18: R80, 2014.
5. Asanoi H, Sasayama S, Kameyama T: Ventriculoarterial coupling in normal and failing heart in humans. *Circ Res* 65: 483–493, 1989.
6. Aslanger E, Assous B, Bihry N, Beauvais F, Logeart D, Cohen-Solal A: Effects of cardiopulmonary exercise rehabilitation on left ventricular mechanical efficiency and ventricular-arterial coupling in patients with systolic heart failure. *J Am Heart Assoc* 4: e002084, 2015.
7. Kolh P, Lambermont B, Ghuysen A, et al: Alteration of left ventriculo-arterial coupling and mechanical efficiency during acute myocardial ischemia. *Int Angiol* 22: 148–158, 2003.
8. Guarracino F, Cariello C, Danella A, et al: Effect of levosimendan on ventriculo-arterial coupling in patients with ischemic cardiomyopathy. *Acta Anaesthesiol Scand* 51: 1217–1224, 2007.