CrossTalk:  

Rebuttal from:  

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Word count: 397

Key words: heart failure, LVAD, pulsatility, blood pressure, blood flow
Cornwell et al. (2018) provide irrefutable arguments that the human circulation is able to survive on lower arterial oscillations than the normal arterial pulsatility. Thus, we agree that a key aim for future research will be to determine the *optimal* amount of pulsatility for each individual CF-LVAD patient. To ultimately achieve a personalized therapeutic target, the optimal balance between LVAD device settings (including its effects on native heart function) and the patient’s pre-existing peripheral arterial health will need to be considered. At present, the consistently low pulsatility in CF-LVAD patients is, in our opinion, still of concern. Despite an impressive improvement in CF-LVAD outcomes with the HeartMate 3 device (Mehra et al., 2017; Mehra et al., 2018), approximately 8-10% of patients still experience disabling stroke (Colombo et al., 2018). The precise role of arterial pulsatility in these patients is currently not known. Notwithstanding, our own observations suggest that patients with similar CF-LVAD settings can have different arterial pulsatility (Castagna et al., 2017), indicating the necessity to examine not just the local cerebral haemodynamics but also the transmission of energy across the whole circulation (Webb et al., 2012).

The consideration of comorbidities that may be partially responsible for stroke is indeed important. Taking on our opponents’ argument, we propose that some of those comorbidities could in fact be a strong indication of a wider, systemic cardiovascular problem, that may reflect inadequate pulsatility in multiple organs, not just the brain. Indeed, the prevalence of cortical microbleeds in 97% of CF-LVAD patients, macrovascular bleeding events in 40-50%, including gastrointestinal bleeding in >25%, suggests a common haemodynamic origin (Yoshioka et al., 2017; Mehra et al., 2018).

Perhaps most importantly, we think it is essential to extend the elegant previous findings based upon mean blood velocities and mean arterial pressure to the measurement of pulsatility (Cornwell
et al., 2014; Cornwell et al., 2015). For example, the same mean pressure and mean flow velocity can be underpinned by different pulsatility. These specific mechanical oscillations will stimulate the mechanoreceptors and possibly the baroreceptor, as well as the endothelium-derived release of nitric oxide (Nakano et al., 2000). Thus, while cerebral autoregulation provoked by a stressor that does not alter pulsatility might be preserved in CF-LVAD patients, it is conceivable that the chronic exposure to a low pulsatile load might narrow the range of adequate functional responses in the event of a stressor of pulsatile nature.

Acknowledgements

This project has received funding from the European Union’s Horizon 2020 research and innovation programme under the Marie Skłodowska-Curie grant agreement No 705219.

References


Study: A Randomized Controlled Trial of the HeartMate 3 Versus the HeartMate II Cardiac Pump. *Circulation* **In press**.


