Video-assisted thoracoscopic bilateral thoracic sympathectomy: An alternative to pharmaceutical disruption of sympathetic neural activity in patients with heart failure

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In the article of Zanoni and colleagues, the authors have devised a sound experimental model to investigate the effects of bilateral mechanical thoracic sympathectomy on postinfarction left ventricular modeling and function. We are well aware of the previous contention that cardiac function can be influenced by deregulation of neural sympathetic control of the myocardial cells, which ultimately leads to heart failure.

Exact physiologic changes associated with or caused by thoracic sympathectomy remain elusive. As seen in the recipients of heart transplant, a denervated donor heart clearly does not respond to the manipulations of the nervous system. Furthermore, total cardiac denervation impairs the response to changes in heart chamber filling pressures and alters the renin–angiotensin–aldosterone system. Increased resting heart rate for the cardiac transplant recipient is independent of hypovolemia, vasodilation, or exercise. Hence, this experimental design raises the possibility that denervation of the heart may not be a disadvantage for the injured myocardium. In fact, it may offer beneficial effects, as suggested in this article, in contrast to our previous knowledge based on the universally adopted clinical practice of using pharmaceutical inhibition for exaggerated sympathetic activity associated with failing myocardium.

After its initial description in 1942, thoracoscopic sympathectomy has remained as an infrequent surgical operation. With the advent of video-assisted thoracoscopic technology, it has been used mainly for the surgical treatment of primary hyperhidrosis with controversial indications and outcomes associated with distressing orthostatic hypotension. Because the surgical technology has become more user-friendly than before, it is now feasible to perform thoracoscopic bilateral mechanical thoracic sympathectomy in patients with severe ventricular dysfunction or end-stage heart failure. In a quest of better alternative therapeutic options that alleviate or curtail the progression of heart failure, this minimally invasive operation may be a welcome addition to our clinical surgical armamentarium.

This experimental model with its potential clinical implications certainly achieves its primary objective of enhancing our curiosity and skepticism for our current understanding and knowledge of unclear link between cardiac function and its consequential sympathetic control of the myocardial cells. With recognition of the real difficulty in overturning our common clinical belief, further experimental, possibly clinical, studies are needed to evaluate this provocative concept while investigating its mechanistic relationship and potential clinical benefits, if any.

References