The respiratory muscle pump in emphysema after single-lung transplantation

A. William Sheel1 and Jeremy D. Road2

1School of Human Kinetics and 2Faculty of Medicine, University of British Columbia, Vancouver, British Columbia, Canada

In this issue of the Journal of Applied Physiology, De Troyer et al. (5) provide an important advance in our understanding of how SLT could affect the mechanics of the diaphragm. It was hypothesized that the interpulmonary difference in ΔPao was the result of a different shortening of the two halves of the diaphragm. To address this they used single-lung inflation to simulate unilateral emphysema in anesthetized dogs. With animals positioned in a computed tomographic (CT) scanner, individual lungs were inflated and the phrenic nerves were stimulated while the endobronchial tubes were occluded. A major strength of this study was the CT-derived determination of muscle displacement and length of the two hemidiaphragms (midcostal region) and radius of curvature. They found that with unilateral lung inflation the pressures developed by the diaphragm are asymmetric. As such, it appears that single lung inflation (as a model of SLT) induces shortening of both hemidiaphragms, but the degree of shortening on the inflated side is much greater than the noninflated side. Said differently, the initial resting length is shortened by the inflation but the delta shortening during stimulation is less on the inflated side. Moreover, it was shown with CT that the asymmetry of contraction is largely explained by a differential effect of inflation on the length and curvature of the two hemidiaphragms.

As with most good studies, the generation of further questions is often as important as the main findings. The study by De Troyer et al. is no exception. It is, of course, difficult to extend the findings from acute animal studies to human SLT recipients, but some comment is warranted. Given the increased life expectancy of SLT recipients it stands to reason that they would be able to increase their participation in the activities of daily living (e.g., walking, climbing stairs) as well as structured exercise programs. What are the consequences of SLT on dynamic lung volumes and the mechanics of the diaphragm with exercise? There is some evidence that SLT patients develop expiratory flow limitation and dynamic hyperinflation with exercise. For example, Murciano et al. (12) found that during exercise SLT patients became flow limited and complained of dyspnea of progressively increasing severity. The native lung, which was probably flow limited at rest, presumably became severely hyperinflated at relatively low levels of exercise. So, during exercise in SLT there appears to be a high propensity for expiratory flow limitation and reduced expiratory time, leading to lung hyperinflation (via gas trapping) and consequent increases in end-expiratory lung volume (EELV). The rise in EELV places the patient on the less compliant region of the lung’s pressure-volume relationship, which in turn increases elastic and threshold loads on the inspiratory muscles leading to an increased work of breathing/sense of effort. The rise in EELV in the native lung during exercise would enhance the difference in length between the two hemidiaphragms. As such, the hyperinflation associated with SLT likely requires the inspiratory muscles to contract from a shorter length, meaning that the muscular force required to ventilate the lungs is substantially increased during exercise.

Address for reprint requests and other correspondence: A. W. Sheel, 6108 Thunderbird Blvd., Vancouver, BC, Canada, V6T-1Z3 (e-mail: bill.sheel@ubc.ca).
Does SLT and the associated shift in mediastinum have an effect on cardiac function? The heart and the lungs are both situated within the intrathoracic space and consequently the heart is exposed to respiratory-related changes in intrathoracic pressure ($P_{\text{ITP}}$). It is inevitable therefore that changes in the volume of one will influence the other. With each inspiratory descent of the diaphragm, $P_{\text{ITP}}$ is reduced and the lungs expand. This lowered $P_{\text{ITP}}$ is also transmitted across the walls of the right atrium, promoting right atrial filling and widening of the right atrial transmural pressure. This means that cardiac preload and afterload are a function of intracardiac pressure and also $P_{\text{ITP}}$ and lung surface-cardiac fossa pressure. The transmural wall stress across the blood vessels within the abdominal compartment and rib cage are also subjected to respiratory-induced swings in $P_{\text{ITP}}$. How do two lungs of different sizes and mechanical properties along with asymmetrical contraction of the diaphragm affect the functioning of the heart and vascular system under exercising conditions where both ventilation and cardiac output are obliged to increase simultaneously? This may be an important consideration as it is often underappreciated that the act of respiration has a significant effect of venous return (10, 11) and on the splanchnic circulation (1).

**DISCLOSURES**

No conflicts of interest, financial or otherwise, are declared by the author(s).

**REFERENCES**


