



THE TRANSPLANTED LUNG

by Jim Harvey MS, RPFT, RCP

At certain times we have access to a unique population of patients who share a specific medical condition. It is then reasonable to ask how their lung function is affected by that medical condition. By the mid 80's, at Stanford Medical Center, where the first lung transplant had been performed in 1981, there was a small population of surviving patients who had been surgically given combination heart and lung transplants.

It seemed reasonable to ask how their recipient lungs functioned after being transplanted. I was involved, as a co-author, in a series of pulmonary function research projects with this special patient population. After combination heart and lung transplantation in humans, all afferent and efferent nerves to the heart and lungs are cut and do not regenerate, so that there is no CNS control or modulation of the nerves remaining within both heart and lung. Certain cough reflexes remain partially intact as the nerves run through to the end of the intact trachea. In the transplanted lung, we had found that chronic rejection takes the form of bronchiolitis obliterans as commonly indicated by measuring the FEV1 and FEF25-75%. Also with the transplanted lung there is a disruption of bronchial circulation and pulmonary lymphatics. We thus asked how the lack of CNS control or feedback affects different pulmonary function parameters.

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The first study was to look at the exercise response in these patients. Studies were performed measuring parameters of respiratory and circulatory function at rest and during maximum tolerable constant work rates using a treadmill in sixteen clinically well patients who had undergone heart lung transplantation for end-stage primary pulmonary hypertension. (Primary pulmonary hypertension is now treatable.) Follow up exercise studies were done at one and two years post transplantation. The results were that the heart-lung transplant patients demonstrated normal exercise dynamics and gas exchange even at higher levels of exercise. The transplanted patients had normal VO₂ Max and normal anaerobic threshold points and minute ventilation. There were minimal circulatory limitations from the new heart, but overall, it is evident that denervation of the heart and lungs does not effect exercise tolerance.

Another study was to look at the elastic behavior of the transplanted lung. When lung transplantation is performed, the donor lung may not be the exact size of the patient's original lung and if larger, will have to compress to some extent into the recipient's chest cavity and if smaller, will have room for expansion. If the donor lung is larger or smaller, what will be the effect on elastic recoil pressures or compliance? With twelve heart-lung transplant patients, we passed esophageal balloons and measured static transpulmonary pressure as the difference between mouth and esophageal pressure during multiple, interrupted deflations from TLC to RC, while corresponding vital capacities were measured with a pressure differential pneumotach at the mouth. Interruptions to expiratory flow were accomplished by a mouthpiece shutter. Pressure-volume points were graphed and analyzed and compliance calculated at a point above FRC. Using body plethysmography, it had been found that the mean TLC of the group was 80% of predicted. For the group as a whole, compliance was essentially normal at 91% of predicted, indicating that the ventilatory defect of 80% was not due to an intrinsic abnormality of the elastic properties of the transplanted lung. However, indices of respiratory muscle function, PIMax, were low, suggesting that the decreased inspiratory force, due to breathing muscle weakness, perhaps from surgical affects, may have been instrumental in the inability of some patient to achieve a normal TLC.

It is technically difficult to transplant lungs that are larger than the recipient's thoracic cavity and donors are usually chosen whose lungs are smaller than the recipient. Since the post transplant TLC is relatively normal, it is evident that the donor lung, irrespective of their size, ultimately assumes the volume of the recipient chest cavity. There are several factors which can predispose the recipient's lung to a restrictive defect such as the age of the donor lung, possible past exposure to particulates or chemicals. Another problem may be that we have computed predicted lung volume values based on the recipient instead upon the donor. Overall, it was concluded that the overall elastic properties of the transplanted lung were within normal range.

And finally, a study was done to test the bronchial hyperresponsiveness in the transplanted lung. It was initially evident that heart-lung transplant recipients often exhibit marked bronchial hyperresponsiveness to methacholine, which was thought to be due to the denervation of muscarinic receptors. Muscarinic recep-

tors are found in the parasympathetic nervous system. In smooth muscle they regulate cardiac contractions, gut motility, and bronchial constriction. Muscarinic antagonists include atropine, scopolamine, ipratropium, and darifenacin, which promotes urinary retention. Specific airway conductance (SGaw) improved significantly with both albuterol and ipratropium bromide but FEV1 did not. The transplanted lung's response to methacholine was demonstrated using eight heart-lung transplant patients. The mean PC20 FEV1 was 0.43 mg, cumulative, of methacholine which demonstrates marked hyperresponsiveness. Then patients were given ipratropium bromide before further methacholine trials, this pretreatment with inhaled ipratropium bromide blocked the response to a standard methacholine challenge. Serial methacholine provocation tests were performed in seven long term survivors and the results did not change.

It has been shown that following pulmonary autotransplantation and allotransplantation in dogs and in baboons, neural regeneration occurs by about six to eight weeks. But despite regeneration, neural function remains impaired as shown by the loss of the Hering-Breuer reflex. In humans this nerve regeneration has not been demonstrated. The response to inhaled ipratropium bromide is in keeping with the presence of intact airway ganglia and postganglionic parasympathetic efferents as demonstrated in animal models. These results support the concept of denervation hypersensitivity of intact muscarinic receptors within the lung. These nerves remain intact within the lung and function to some extent without direct CNS control.

If you ever run across a patient population having a unique characteristic, it might be a good opportunity to see how their lung function is affected by their medical condition. If you are interested in further details of the above studies, feel free to contact me.

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Obesity + Homecare Services... *Continued from page 34*

these items within a private residence. Many private residences for instance, must have doorways enlarged in order to use those wider bariatric mobility products. A thorough home evaluation will provide answers regarding which products will meet the patient's mobility needs within a private home and in the community as well. Bathroom products including shower chairs, bedside commodes and transfer benches can improve patient independence within the residence as well.

Insurance coverage for some bathroom and mobility products can vary widely between policies and providers. Many durable medical equipment carriers will contact a patient's insurance carrier to obtain information on which products will be reimbursed by their insurance carrier and at what level.

The respiratory care practitioner will continue to face many challenges as he or she works to meet not only the respiratory requirements but also the lifestyle needs of the bariatric patient. The home respiratory therapist must be educated on all the different products available to treat the bariatric patient so that all the needs of the patient can be met for the most positive outcome.

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