



Case report

Cardiopulmonary exercise testing after laryngectomy: A connection conundrum

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ABSTRACT

A patient presents with a new bronchogenic carcinoma 5 years after laryngectomy for recurrent laryngeal tumor and 13 years after chemoradiation for concurrent lung cancer with synchronous base-of-tongue tumor. Due to his complex history and perceived limited respiratory reserve, he was felt high risk for the completion pneumonectomy needed for resection of this new tumor. The attending surgeon requested a full cardiopulmonary exercise test for risk assessment prior to surgery. We found that there was no commercially available connector that would allow our CPET equipment to reliably collect respiratory gases from a patient with tracheostomy stoma or tube. We report here a simple coupling devised "in house" that allowed for the performance of an interpretable test leading to a significant change in medical care.

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1. Introduction

Cardiopulmonary exercise testing (CPET) uses a patient generated workload to provide objective assessment of the integrated fitness of the cardiac, respiratory, and musculoskeletal systems [1]. In use for decades in the evaluation of enigmatic dyspnea, CPET has received increasing attention as a tool to determine readiness for major cardiovascular and cancer surgeries [2,3]. In addition, there is evidence that achieving certain milestones on CPET testing may help risk stratify patients undergoing evaluation for major thoracic resections [4,5]. A well-performed CPET that provides quality data for analysis requires a cooperative and motivated subject, well-trained technicians and supports staff, and properly calibrated equipment that allows for simultaneous measurement of a host of physiology parameters including collection of all inspired and expired air without leak [6].

Cigarette smoking is associated with an increased incidence of a host of chronic illnesses including coronary artery disease, chronic obstructive lung disease (COPD) and a variety of malignancies. Among the more common, and deadly, of these cancers include

neoplasms of the Head and Neck and Bronchogenic Cancer. United by the common exposure to chronic cigarette smoke, patients with Head and Neck cancers and Lung cancers often have coexistent COPD complicating their operative risk for curative resections. In addition, a history of heavy cigarette smoking increases the risk for second tumors even if the primary lesion is successfully treated.

2. Case description

Mr. T is a 67-year-old smoker (150 pack-years) who presented in 2002 with dysphagia. He was found to have a locally advanced base of tongue cancer (T₄N₀) and a right upper lobe lung nodule (T₂N₀), both biopsy-proven to be squamous cell carcinomas. As it was deemed impossible to distinguish a synchronous lung primary tumor from metastasis, he was treated with primary radiotherapy to both locations followed by a full course of cis-platinum doublet adjuvant chemotherapy with a complete response at both sites. He was disease free for 5 years and then lost to follow-up. He continued to smoke and presented in 2010 with hemoptysis. Pan endoscopy revealed a multifocal squamous cell neoplasm in the larynx. He was referred to our institution and underwent total laryngectomy without further adjuvant therapy; there was no apparent new disease in the chest. At this point, he quit smoking. A one-year surveillance positron emission tomography scan (PET) in

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2011 revealed no evidence for persistent or recurrent disease. In the fall of 2014, he noted increasing chest congestion and sputum production. There was no evidence for tracheoesophageal fistula, but chest imaging demonstrated a new right lung mass (Fig. 1A). PET scan revealed an FDG-avid lesion involving both the posterior segment of the right upper lobe and crossing the major fissure into the right lower lobe (Fig. 1B); there was no obvious nodal involvement or disease outside the chest. The mass appeared intimately associated with the bronchus intermedius. Bronchoscopy revealed endobronchial tumor in a subsegment of the posterior segment right upper lobe and bulging of the membranous portion of the bronchus intermedius. Endobronchial ultrasound confirmed a large homogeneous soft tissue mass posterior to the airway without an obvious tissue plane between the airway and tumor. Biopsy revealed poorly differentiated squamous cell cancer.

Mr. T. had a long history of chronic bronchitis without frequent exacerbations. He used nebulized combination short acting beta-agonist and anticholinergic bronchodilators two to four times daily. He had a daily cough productive of small amounts of thick white sputum with recent occasional blood streaking. He had not needed supplemental oxygen with any of his prior procedures. His CT scan showed upper lobe predominant centrilobular emphysema and he carried the diagnosis of COPD. Baseline PFT prior to his previous cancer treatments could not be located in an outside system. He noted dyspnea with walking a block on level ground or

with any incline; he was able to climb two flights of stairs slowly in Thoracic Surgery Clinic without finger oximetry desaturation, though he did appear short of breath. He had no known heart disease, no chronic edema, and no orthopnea.

3. Physiologic testing

Pulmonary function testing was performed using a standard soft rubber adapter than is held against the stoma to achieve a short-term seal for spirometry and diffusion capacity (DLCO) testing. Spirometry revealed a post-bronchodilator FEV₁ of 2.18 L (63% predicted), FVC of 3.24 (69%), and FEV₁/FVC of 0.67. DLCO was reduced at 44% predicted. MVV was reduced at 77 L/min ($>35 \times$ FEV₁). Flow volume loops were reproducible without evidence for leak on the Volume–Time plot. Quantitative Ventilation/Perfusion scanning (V/Q) revealed 16% perfusion right lung vs. 84% left lung. Resting room air ABG showed pO₂ = 87, pCO₂ = 34, and pH = 7.45.

Based on prior experience, we have found that the standard cycle-based CPET set-up in our lab is not compatible with the stoma interface adapter we use for PFT testing; the leak is large and variable. We contacted the manufacturer of our CPET equipment and were told there was not a readily available connecting device compatible with a stoma. We assumed we could place a tracheostomy tube with inflatable cuff, but similarly, there are no standard parts available to connect the 15 mm outside diameter (OD) tracheostomy tube fitting to the 30 mm OD CPET mass flow sensor. Any connector used would need to provide an airtight seal while flexible enough to allow for the movement inherent in a maximal effort exercise test. Using parts available within the Respiratory Therapy Department, we crafted a connector from the tracheostomy tube to the mass flow using a straight 15 mm–15 mm tube connector and a silicone rubber connector from Servo 900C ventilator parts (part #MCCO.6343420) (Fig. 2 A & B). After discussing options with the patient, a #8 cuffed plastic Shiley tracheostomy tube was placed using 2% lidocaine lubricating jelly. After allowing 15 min to acclimate, the balloon was inflated to the point of eliminating leak with Valsalva during tube occlusion (8–10 cc air). Repeat spirometry revealed nearly identical expired volumes (FEV₁ = 2.21, FVC = 3.42) and flow volume loop. Using this set up, the patient was able to breath comfortably through the circuit with reproducible breath-to-breath volumes and no evidence of leak.

The patient exercised on a cycle ergometer at a 30-Watt ramp for 4 min and 20 s stopping due to leg fatigue (Fig 3). The test was deemed near maximal as evidenced by achieving 83% of predicted target heart rate (134 bpm), a Respiratory Quotient (RQ) at peak exercise of 1.14, and signs of physical exhaustion. ABG revealed no hypoxemia or hypercarbia at peak exercise, and a drop in serum HCO₃⁻ of 3 mmol/L. There was no evidence for a ventilatory limitation to this level of exercise with a breathing reserve of 35% (VE max of 50 L/min with MVV of 76), a peak respiratory rate of only 34, and normal tidal volume recruitment (Vt/FVC going from 12% to 55%). Ventilation-perfusion parameters showed an elevated A–a DO₂ of 27 at rest that did not fall with exercise (A–a DO₂ of 32 at peak stress), but no desaturation noted. There was a normal drop in the arterial to end tidal PCO₂ gap and normal fall in VD/VT (41% at rest to 18% at peak VO₂), demonstrating the tube and connectors did not seem to introduce a meaningful dead space load. There was no apparent cardiac limitation with Anaerobic Threshold (AT) occurring normally at 54% of predicted VO₂ max (13 ml/kg), a normal rise in the O₂-pulse (a surrogate for stroke volume recruitment), and no ECG changes to suggest ischemia. At peak exercise, the patient's VO₂ max was 1375 ml/min (65% of predicted) or 15.7 ml/min/kg with patient 4 kg above ideal body weight

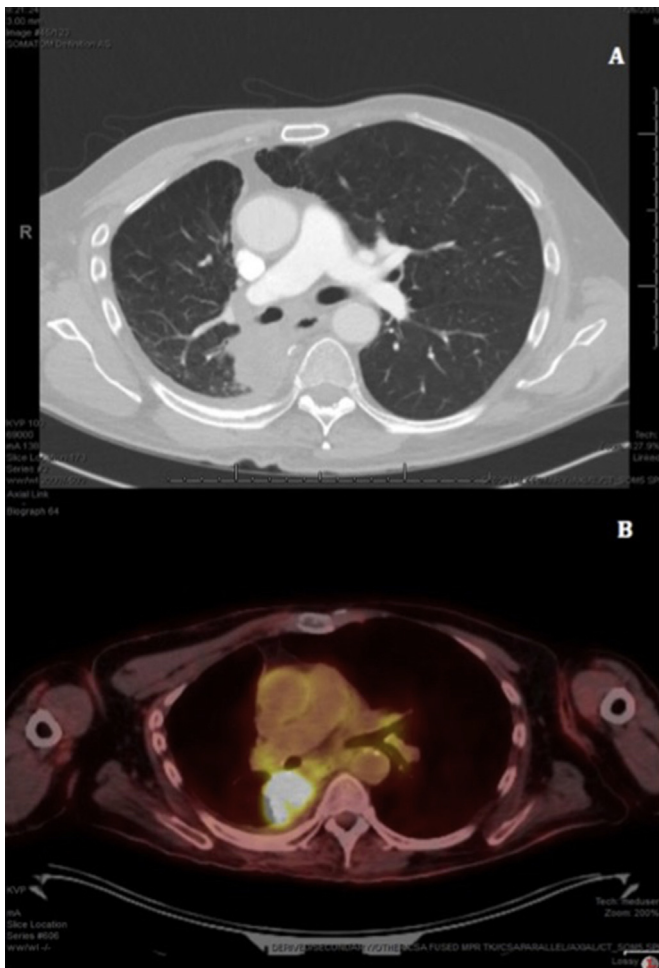


Fig. 1. A) CT imaging showing mass in the posterior RUL. B) PET-CT showing FDG-avid mass posterior to bronchus intermedius.

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