

Dead space breathing in patients with cancer

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Eichkorn T. Dead space breathing in patients with cancer. *J. pulmonol.* 2022; 6(6):78-80.

ABSTRACT

Dyspnea from musculoskeletal, circulatory, and ventilatory origins is frequently experienced by cancer patients, and cardiopulmonary exercise testing is the best way to diagnose dyspnea (CPET). As part of this retrospective pilot investigation, we used CPET to assess patients with hematologic and solid malignancies to identify the main cause of their dyspnea. On a bike ergometer, the subjects worked out at progressively higher intensities. A number of variables were monitored during baseline and maximal activity, including minute ventilation, heart rate, breathing reserve, oxygen absorption ($\dot{V}O_2$), O_2 -pulse, and ventilatory equivalents for carbon dioxide and oxygen, respectively. All subjects had their slope and intercept for $\dot{V}E/\dot{V}CO_2$ calculated. Peak $\dot{V}O_2$ below

the expected level of 84% suggested a circulatory or ventilatory restriction. Complete clinical and physiological data were available for 36 individuals (M/F 20/16); 32 (89%) showed signs of circulatory or ventilatory restriction as indicated by a decreased peak $\dot{V}O_2$ and 10 subjects had normal physiological data. With a mean SD peak $\dot{V}O_2$ of 61% 17% anticipated, the pulmonary vascular group ($n = 18$) made up the biggest cohort. $\dot{V}O_2$ and spirometric measurements showed strong correlations. The highest peak values of $\dot{V}E/\dot{V}O_2$ and $\dot{V}E/\dot{V}CO_2$ were found in the circulatory and ventilatory cohorts, which is consistent with an increase in dead space breathing.

Key Words: *Pleural disease; Interventional pulmonology; Pneumococcus; Microorganism; Inflammatory reaction*

INTRODUCTION

In patients with cardiovascular dysfunction, the intercept of the $\dot{V}E/\dot{V}CO_2$ connection was lowest. Dead space breathing is common in dyspneic individuals with malignancies, and many of these patients show a circulatory source for exercise limitation with a clear pulmonary vascular component. The effects of chemotherapy and radiation therapy on the pulmonary vascular endothelium and cardiac function are potential contributing factors. Dyspnea and tiredness are frequently experienced by cancer patients. The aetiology of these symptoms may be musculoskeletal, cardiovascular, pulmonary vascular, or ventilatory. Ventilatory restriction may be brought on by a tumour directly affecting the respiratory system or by underlying lung and/or pleural illness. Cardiovascular restrictions may result from structural heart disease, tumour involvement in the heart, or side effects from chemotherapy. Pulmonary vascular restriction may be caused by drugs, intrinsic acute or chronic thromboembolic illness, or both. Anemia, muscular wasting, hunger,

discomfort, electrolyte problems, and depression are other conditions that might contribute to functional limitation and lower one's ability to carry out everyday activities [1]. Cardiopulmonary exercise testing (CPET), with a focus on the degree of decline in oxygen uptake at peak exertion ($\dot{V}O_2$ max), is the optimal tool for determining the cause of dyspnea. Additionally, decreases in the ventilatory reserve and oxygen-pulse (which represent stroke volume) signify a cardiovascular or ventilatory restriction, respectively [2-4]. A combination of circulatory and ventilatory limitations can cause many people to experience exercise limitation. Finally, an increase in dead space breathing due to lung parenchymal, cardiovascular, or pulmonary vascular damage is indicated by a failure to see a decrease in the ventilatory equivalents (efficiency) for oxygen and carbon dioxide ($\dot{V}E/\dot{V}O_2$ and $\dot{V}E/\dot{V}CO_2$, respectively) during exercise. As a result, the $\dot{V}E/\dot{V}CO_2$ slope has been used to differentiate between heart failure and COPD as a cause of exercise limitation. However, many patients with cardiovascular limitation display abnormalities in respiratory function, which may impede the $\dot{V}E/\dot{V}CO_2$ slope's capacity to discriminate. Recently, the

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Received: 03-Nov-2022, Manuscript No. *puljp-22-5344*; Editor assigned: 06-Nov-2022, PreQC No. *puljp-22-5344* (PQ); Reviewed: 18-Nov-2022, QC No. *puljp-22-5344* (Q); Revised: 24-Nov-2022, Manuscript No. *puljp-22-5344* (R); Published: 30-Nov-2022, DOI: 10.37532/puljp.2022.6(6).78-80.



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distinction between ventilatory and circulatory restriction has been further refined using the intercept derived from the $\dot{V}E$ - $\dot{V}CO_2$ relationship during exercise. With deteriorating disease, COPD patients exhibit a decreased $\dot{V}E/\dot{V}CO_2$ slope but an increased $\dot{V}E/\dot{V}CO_2$ intercept. In this pilot study, we assessed cancer patients who received CPET in a cancer hospital to assess their dyspnea [5]. The patients had varied malignancies. The primary goal was to detect the cardiorespiratory cause of the dyspnea in individuals whose symptoms could not be identified by clinical, imaging, or respiratory function data. Additionally, we tried to spot any individuals who had both circulatory and ventilatory limitations. A big cancer center's clinic evaluated patients with hematologic and solid malignancies for dyspnea in this retrospective pilot study. The Institutional Review Board of the University of Southern California Health Sciences Center gave the study their blessing (#HS-13-00759). Between August 2008 and March 2013, research was done. Through February 2019, patients were monitored. While undergoing therapy for their cancers, the patients were in a stable clinical state. Exclusion criteria were those with acute respiratory failure, acute heart failure, acute neuropathic, and acute myopathic disorders. Each patient got a clinical evaluation that included imaging, a lung function test, and a full blood count. Patients were contrasted with a group of healthy, non-smoking, unaffected by cardiorespiratory diseases participants. According to the recommendations of the American Thoracic Society and European Respiratory Society (ATS/ERS), spirometry was carried out while the patient was seated. Crapo et al. provided the FVC and FEV1 reference values. An FEV1/FVC ratio that is lower than 0.7 indicates chronic airflow limitation. An FEV1/FVC ratio 0.7 and an FVC 80% expected were considered signs of restrictive respiratory impairment. There were no plethysmographically assessed lung volumes [6-8]. The clinical and anthropometric characteristics of cancer patients undergoing CPET, adherence to international standards for CPET techniques, and the safety of CPET as measured by reported side events were all taken into account in the study. The exercise testing apparatus included a stationary cycle ergometer that was calibrated before and after each test (Med Graphics CPX Ultima system, Medical Graphics Corporation, St. Paul, MN). For this device, the mechanical dead space volume varied from 45 to 65 mL depending on the mouthpiece and connections employed. Prior to each test, gas concentrations were calibrated using primary standard gases and flow using a 3 L syringe. The same 3 exercise technologists with certification conducted each test. The day of the test and four hours prior to it, subjects were requested not to exercise and to abstain from eating or drinking anything with caffeine. An informed consent was acquired after each technique was explained. The stationary cycle ergometer and mouthpiece were demonstrated to the test subjects before they started, and they cycled on it for roughly 10 minutes. They were seated, breathing through a nose clipped mouthpiece. Following at least five minutes of resting measures, the Godfrey protocol was used to train the subjects on an ergometer with progressively heavier workloads at increments of 5 to 15 Watts. The patient's $\dot{V}O_2$ max plateau (average of the five highest consecutive $\dot{V}O_2$ readings near peak exercise) and a respiratory exchange ratio of at least 1.1 during peak activity were used to calculate the patient's maximum effort. Following exercise, data was collected for many minutes in order to monitor the ECG and collect gas samples.

Minute ventilation ($\dot{V}E$), inspired oxygen concentration, expired oxygen tension, inspired carbon dioxide output, oxygen absorption ($\dot{V}O_2$), and expired carbon dioxide output were all measured every 15 seconds. The V-slope approach was used to calculate anaerobic threshold (AT), which was then confirmed by the crossover of ventilatory equivalents for O_2 and CO_2 . Heart rate and rhythm were measured constantly during the investigation with the 12-lead ECG. Maximum voluntary ventilation (MVV), ventilatory reserve ($\dot{V}E/MVV$), respiratory exchange ratio (RER), oxygen pulse ($\dot{V}O_2/HR$), and ventilatory equivalents for carbon dioxide and

oxygen ($\dot{V}E/\dot{V}CO_2$ and $\dot{V}E/\dot{V}O_2$, respectively) were also derived. For these variables, the usual values. The following conditions had to be met in order to qualify as a maximal effort: (a) a steady plateau in $\dot{V}O_2$ (average of the five highest consecutive $\dot{V}O_2$ values near peak exercise with a variance of 150 mL/min); (b) achieving an RER of less than 1.05; and (c) a heart rate that was within 10 beats/min of the age-predicted maximum. When the link between $\dot{V}E$ and $\dot{V}CO_2$ clearly increased and when $\dot{V}E/\dot{V}O_2$ increased during gradually rising labour rate without a corresponding increase in $\dot{V}E/\dot{V}CO_2$, the anaerobic threshold had been reached. When symptoms such as unbearable dyspnea, chest pain, significant ST-segment depression on an ECG, a reduction in systolic blood pressure, or arterial oxygen saturation of less than 88% appeared, exercise testing was halted. Reviewing the data of 43 patients who had been referred to the cancer centre for a dyspnea examination revealed that 7 did not have cancer. The other 36 patients (males 20, females 16) had complete clinical and physiological data, of which 31 (86%) were diagnosed as having a ventilatory or circulatory restriction and one as having a musculoskeletal limitation based on clinical and physiological data. Ten individuals were classified as having normal exercise capacity because they showed normal lung function and CPET results. 22 patients had smoking histories available; ten had smoked before (including one person who was considered to be normal), with pack-years ranging from 10 to 66; the other 12 had denied using tobacco products. One patient, a former smoker, also disclosed a history of exposure to chemicals at work. Hemoglobin levels in three cases were under 10 g/dL.

32 individuals (89%) had solid malignancies, with breast, lung, and prostate being the most prevalent. 14 people had a history of multiple tumours, 12 of which were solid tumours, the most prevalent of which were lung and prostate. Hematopoietic malignancies, including non-Hodgkin lymphoma, Hodgkin disease, and leukaemia, were discovered in eight patients. Patients with solid tumours had two of the lymphomas (lung and breast, one each). Numerous cancer medications have negative cardiovascular side effects that put patients at risk for both diminished and preserved ejection fraction heart failure. Heart failure risk is increased by coexisting cardiovascular risk factors or cancer-related cardiometabolic consequences. To comprehend the prevalence of heart failure, especially that with maintained ejection fraction, more research is required. With the increased use of biologic drugs, such as checkpoint inhibitors, which were not given to any of our patients, this becomes even more crucial. Peak $\dot{V}O_2$ was substantially correlated with FVC and FEV1 across the cohort as a whole, but among the subcohorts, this link was most pronounced in the pulmonary vascular group, who showed a normal or mildly restrictive pattern on spirometry while having a normal ventilatory reserve. Because of smoking history and perivascular cuffing with edoema, airflow restriction is frequently linked to heart failure. Patients with ventilatory dysfunction had a weaker correlation, but they made up a smaller cohort. If the primary cardiovascular group had been larger, they most likely would have displayed a similar link. Patients with impaired cardiac output and/or increased pulmonary vascular resistance at peak exercise have decreased oxygen delivery, according to the Fick equation, while patients with chronic lung disease exhibit hypoxemia as a result of ventilation-perfusion mismatching and decreased gas transfer, which in turn increases pulmonary vascular resistance. Additionally, anaemia affects oxygen transmission since haemoglobin is necessary for oxygen content (1.34 gm carried in 100 mL blood). In the circulatory and ventilatory cohorts, the ventilation equivalents of O_2 and CO_2 increased. Spirometric volumes and ventilatory equivalents were correlated negatively, especially in the pulmonary vascular group. The ventilatory group showed a less clear connection with this. Patients with chronic heart failure, pulmonary hypertension, and the majority of lung disorders have been shown to have a drop in ventilatory efficiency in direct proportion to the decline in exercise capacity [9]. The side effects of medication,

hormonal, biologic, and radiation treatments most certainly further compromised gas exchange in the majority of our patients. Impaired cardiac output may be linked to increased peripheral ergoreceptor or chemoreceptor ventilation drive, which can result in excessive efferent muscle nerve activity and exercise hyperventilation.

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