CI, 1.014–1.032; $P < 0.001$) remained significant. Percentage predicted is not superior to absolute 6MWD as a predictor of mortality in PAH.

Climbing Kilimanjaro: time-course changes in pulmonary pressures and gas-exchange during altitude exposure in 27 non-acclimatized climbers

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High altitude exposure results in hypoxic pulmonary vasoconstriction (HPV) and increased pulmonary pressure. This study examined the relationship between acute changes in resting pulmonary pressure and pulmonary gas-exchange measures during exercise in a group of non-acclimatized climbers summiting Mount Kilimanjaro. Twenty-seven climbers (age, 44 + 15 years) completed the study. Exercise testing (4-min step test with gas-exchange) and echocardiographic measurements were completed at four different altitudes: 8801 m (barometric pressure, PB = 690 mmHg); 3500 m (PB = 505 mmHg); 4600 m (PB = 428 mmHg) and on return to 880 m (8802 m). Right ventricular systolic pressure (RVSP, mmHg) progressively increased as PB fell during the ascent (8801 m: 19 + 4; 3500 m: 27 + 8; 4600 m: 33 + 8 mmHg). During exercise, the fall in PB was associated with a decrease ($P < 0.01$) in oxygen saturation (nadir SpO2(%): 8801 m: 96 + 2; 3500 m: 82 + 3; 4600 m: 73 + 4). The time-course changes in gas exchange tracked changes in RVSP during ascent with a progressive decrease ($P < 0.01$) in SpO2 and end tidal carbon dioxide production (end-exercise PETCO2, mmHg: 8801 m: 37.1 + 3.6; 3500 m: 28.5 + 2.6; 4600 m: 20.8 + 1.9) and an increase in breathing efficiency (VE/VCO2: 8801 m: 28.5 + 2.9; 3500 m: 35.8 + 4.6; 4600 m: 50.7 + 5.8). However, while SpO2 and RVSP normalized on return to lower altitude (8802 m), gas-exchange measures remained altered (PETCO2, mmHg: 31.2 + 3.0; VE/VCO2: 32.8 + 3.2, $P < 0.01$ versus 8801 m). With high altitude exposure, there is an increased ventilatory drive characterized by altered gas exchange and associated changes in SpO2 and pulmonary pressures. However, following high altitude exposure and once SpO2 and pulmonary pressures are normalized, gas-exchange measures remain altered suggesting that HPV is no longer a potential stimulus for an increased ventilatory drive.

The National Echo Database Australia (NEDA) and pulmonary hypertension

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We have previously demonstrated that pulmonary hypertension (PHT), identified using echocardiography (echo) is common and that left heart disease accounts for the majority of PHT. Echo measurements of left heart disease may be helpful in predicting the cause of PHT. The aim of this study is to examine prevalence of PHT within NEDA, and uncover left heart predictors of PHT. NEDA utilizes novel database engineering to combine individual databases into a single database. A total of 307,656 echocardiograms from two laboratories have been included in this analysis. We defined PHT as a right ventricular systolic pressure (RVSP) over 40 mmHg. In total, 180,374 echos (59%) had a measurable tricuspid regurgitation (TR) velocity profile from which an RVSP could be calculated. PHT from any cause was identified in 39,699 (22%) echos. Of those in which PHT was identified, the mean RVSP was 51 ± 11 mmHg, compared with 29.5 ± 5.8 in those without PHT ($P < 0.0001$). These patients were older than the overall average for NEDA (mean age, 74.9 ± 12.1 years versus 62.9 ± 16.6, $P < 0.0001$). The ejection fraction (EF) was similar but significantly different between those with PHT and those without (58.1 ± 13.7 versus 61.9 ± 8.7%, $P < 0.0001$). Measures of diastolic function were markedly different (E:E' ratio 17.1 ± 8.5 versus 11.3 ± 5.5, $P < 0.0001$). PHT is common, representing 22% of those with a measurable RVSP in a large echo cohort (over 300,000 echos). Overall, the EF was similar in PHT compared to those without PHT, whereas surrogate markers of filling pressure such as E:E' ratio were markedly different, underpinning the importance of measuring diastolic function in the evaluation of PHT.