output. We predict that end organ fatigue of at least some inspiratory muscles might occur prior to or coincident with termination of some types of sustained, very high-intensity exercise in some highly fit subjects; we doubt if this would be a universal finding, but rather one highly dependent upon very special circumstances of sufficient ventilatory demand over sufficiently long periods.

**SUMMARY**

In most circumstances in health, efficient alveolar ventilation and alveolar-to-arterial exchange of O$_2$ and CO$_2$ are among the strongest of links in the gas-transfer chain during maximal exercise. Indeed, in most instances, the metabolic cost of ventilation represents the only significant contribution of the pulmonary system to the limitation of O$_2$ transport to locomotor muscles and thus to the limitation of maximum performance. Of the "weaknesses" inherent in the healthy pulmonary system response to exercise, the most serious one may well be its absence of structural adaptability to physical training or to the trained state. Thus, the lung's diffusion capacity and pulmonary capillary blood volume remain unaltered in the highly trained human or horse, while maximum pulmonary blood flow rises linearly with the enhanced max VO$_2$. Similarly, ventilatory requirement rises markedly, with no alteration in the capability of the airways to produce higher flow rates or of the lung parenchyma to stretch to higher tidal volumes, and little or no change in the pressure-generating capability of inspiratory muscles. The case of the elderly athlete who remains capable of achieving high maximum pulmonary blood flows and ventilatory requirements and whose lung undergoes a normal aging process underscores the importance of deficits (from "normal") on the capacity end of this continuum of cost versus capacity in the pulmonary system. The asthmatic athlete may represent another such example of limited flow-generating capacity; and the healthy, young, highly fit athlete who shows marked reductions in SaO$_2$ and in max VO$_2$ at even moderately high altitudes demonstrates that, in many situations, precious little room can be added to the demand side or removed from the capacity side before signs of failure can be seen.

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**REFERENCES**

15. Dempsey JA. Demand versus capacity in the pulmonary system response to exercise. (Bouchard, C ed.), in press

**Right Ventricular Pressure/Volume Relationship at Rest and during Exercise in Patients with Chronic Lung Disease**

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To evaluate right heart function during a passive and an active maneuver, we studied 26 patients with chronic lung disease: 11 with chronic bronchitis, 11 with chronic bronchitis and lung distention, and 4 with pneumoconiosis with normal hemodynamic values; 31% of these patients

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had previously suffered from right heart failure but were in stable clinical condition at the time of the study. Besides the standard hemodynamic measurements, right ventricular ejection fraction (RVEF), end-diastolic (RVEDV) and end-systolic (RVESV) volumes were determined with a fast-response thermistor catheter. The measurements were performed at rest supine (R), with the feet on the pedals of the ergometer (LR), and during exercise at two loads: first 0 or 20 W (E), then 20 or 40 W (E) according to the patients' ability to perform exercise in ventilatory steady state.

The patients were divided into 3 groups according to pulmonary vascular resistance (PVR), computed as 80 (PAP-Paw)/Q: group 1 consisted of 9 patients with PVR below 150 dyn.s.cm\(^{-5}\), group 2 of 12 patients with PVR between 150 and 250, and group 3 of 5 patients with PVR exceeding 250 dyn.s.cm\(^{-5}\). Spirometry was close to normal in group 1; group 2 showed airway obstruction, which was worse in group 3 (FEV1/VC: 41 ± 4%, mean ± SEM). PaO2 was 71 ± 2 mm Hg in group 1, 58 ± 2 mm Hg in group 2, 50 ± 3 mm Hg in group 3. It did not drop with exercise in any of the groups.

RVEF was normal at rest in group 1 on average (0.42 ± 0.03) and increased to 0.47 ± 0.03 at E. It was lower (0.38 ± 0.03) in group 2 but increased to 0.43 ± 0.03 at E. In group 3, however, it was significantly lower at rest (0.25 ± 0.03, p<0.001) and did not change with exercise.

The right ventricular systolic pressure/volume relationship was differentially influenced by LR in the 3 groups: in group 1, pressure increased by 4 mm Hg, and RVESV was unchanged; in group 2, pressure increased by 8 mm Hg, and RVESV by 13 ml; in group 3, pressure increased by 4 mm Hg and RVESV by 33 ml. The effect of exercise was not different from LR in groups 1 and 2, but the slope definitely increased in group 3, showing that the depressed cardiac contractility was enhanced, presumably by the sympathetic stimulation occurring during exercise. The relationship between stroke volume and RVEDV showed similar results, with a depressed cardiac function during the passive maneuver (LR) in group 3, but improvement during exercise.

These results show that, in patients with high PVR, but able to exercise, right heart function was depressed but could still be enhanced during exercise.

**Interaction of O\(_2\) and CO\(_2\) on Respiratory Drive (RD) in Severe Chronic Obstructive Pulmonary Disease (COPD)**

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Studies of respiratory drive (RD) tend to isolate O\(_2\) and CO\(_2\) as chemical stimuli and to isolate the mechanisms responsible for regulating PaO\(_2\) and PaCO\(_2\). Yet complex interactions between O\(_2\) and CO\(_2\) appear to be necessary for normal ventilatory control. We measured the RD response to hypoxia at 2 constant levels of end-tidal P\(_{CO2}\) in 16 men with COPD (FEV\(_1\) 0.7±0.1 Lit.) and 13 normal subjects. Airway occlusion pressure, measured 0.1 s after the onset of inspiration (P\(_{aw}\)), was the index of RD. The slope of the regression line relating increase in P\(_{aw}\) to SaO\(_2\) was used as the RD response to hypoxia, expressed as ΔP\(_{aw}\) cm H\(_2\)O/ΔSaO\(_2\).% The RD response to hypoxia was weak during eucapnic hypoxia in both COPD and normals (slope −0.16±0.1 vs −0.041±0.05). With hypercapnia, the RD response to hypoxia was much greater, and the mean slope of the COPD patients was higher than that in normals (−0.72±0.48 vs −0.54±0.4, p<0.001). These data show a multiplicative interaction of hypoxia and hypercapnia on RD.

We next studied the interdependence of PaO\(_2\) and PaCO\(_2\) regulation during exercise in 7 men with COPD (mean FEV\(_1\) 0.77±0.14 Lit). Subjects did 2 progressive-load exercise studies on a bicycle ergometer, once on room air and once on 0.5 FIO\(_2\). Most subjects quit at levels of 33–50 watts because of severe dyspnea, which was related to a large increase in RD as measured by the P\(_{aw}\). Strikingly, most subjects maintained PaCO\(_2\) at their resting levels during exercise on room air (mean resting PaCO\(_2\) 41.7±5.0 mm Hg vs mean end-exercise PaCO\(_2\) 41.5±6.0 mm Hg, p<0.01). In contrast, the end-exercise PaCO\(_2\) rose in all patients on 0.5 FIO\(_2\) (mean end-exercise PaCO\(_2\) 48.5±7.3 mm Hg, p<0.005). End-tidal P\(_{CO2}\) recordings showed that the rise occurred during exercise. The exercise data show that hypercapnia impairs the ability to fine-tune the PaCO\(_2\) during exercise. Together, these studies illustrate the complexity of respiratory control in COPD.

**A Possible Role for Mixed Venous Blood Changes in the Hyperventilation of Exercise in Heart-Lung Transplant Recipients**

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The initial rise in ventilation at the onset of exercise remains unexplained (Wasserman et al. J Appl Physiol 1975;39:354). Heart-lung transplant (HLT) patients differ from normals in 2 respects: they have denervated lungs and a disparity between their cardiac and ventilatory responses to exercise, probably a result of denervation of the heart, giving a poorer cardiac response. Six healthy HLT recipients were studied at rest and after 6 minutes of 50-W supine exercise. Thermodilution measurement of cardiac output and mixed venous blood gas changes were obtained with a right heart catheter. Metabolic gas exchange was continuously recorded by mixed expired gas analysis, using a mass spectrometer. Six normal subjects were also exercised, but without catheter. Results are shown in Table 1.

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