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MICROCOPY RESOLUTION TEST CHART
PULMONARY ADAPTATION TO HIGH ALTITUDE

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The findings in this report are not to be construed as an official Department of the Army position unless so designated by other authorized documents.
Three major aims of the contract were addressed in the past 6 months:

1. We completed our study of the relationship between hypoxia-induced periodic breathing in sleep and the occurrence of obstructive apneas. We used normal subjects and those who might be "susceptible" to upper airway closure, i.e., heavy snorers and even some patients with obstructive sleep apnea syndrome. As expected we found that administration of hypoxia caused immediate hypocapnia leading to a Cheyne-Stokes type of oscillatory breathing pattern which caused marked increases in airway resistance during the periods of low ventilatory drive. The surprising finding was that once full-blown periodic breathing developed—after about 5 mins of hypoxia—airway resistance was markedly reduced to levels < than those observed while awake and no evidence of occlusive apnea occurred. The conclusion is that hypoxia must have exerted a protective effect on the upper airway, by ensuring that as inspiratory drive increased toward the end of each apneic period, activity to the muscles controlling upper airway caliber was greater than and/or preceded that to the diaphragm and other inspiratory muscles of the chest wall. Further studies are now needed of the EMG activity of these upper airway and chest wall muscles to determine their relative activities during the apneic periods. This "protective" mechanism is central to the sojourner at high altitude—particularly the
heavy snorer—to guard against occlusive apnea and even greater nocturnal hypoxemia. (This work was presented at the recent American Thoracic Society Meeting.

2. The question of respiratory muscle fatigue during exercise in humans was studied in highly fit subjects performing high intensity exercise to exhaustion. First we found that a partial "unloading" of ventilatory work—by breathing low density He:O₂ gas mixtures—significantly increased exercise endurance time to exhaustion and reduced "perception" of effort. On the other hand our additional data did not implicate the mechanical work of breathing during exhaustive exercise as an important contribution to overall fatigue. We determined the pleural pressure wave form and magnitude generated each breath during exhaustive exercise. Then we mimicked this form and magnitude of pressure development at rest and found that the subject could tolerate this form of pressure development for much longer times than he could exercise. Work continues on this project in normoxic and hypoxic conditions. (Aaron et al., Med. Sci. Sport, 1985 (Abstract).

3. We have studied respiratory muscle recruitment during exercise and saw evidence of active expiration even in mild exercise. We documented this by measuring the change in FRC and in end-expiratory value and in esophageal and gastric pressures. We have also documented this change by recording abdominal muscle EMG activity even at light work loads. (see Sharatt, Med. Sci. Sport, 1985 Abstract) and Henke, Med. Sci Sport, 1985 Abstract).

4. Finally, we have presented the concept in two recent reviews and presentations (Am. J. Cardiology, 1985; and Wolfe Memorial Lecture, American College of Sports Medicine, May 1985) that the lung becomes a
"limiting" factor to exercise capacity as one proceeds from an untrained state to a highly trained state, because training causes adaptation of the locomotor muscles but not the respiratory muscles nor the lung. Eventually the capability for $O_2$ uptake by the locomotor muscles becomes greater than that afforded by the $O_2$ transport capabilities of the lung and chest wall. This selective effect of physical training contrasts markedly with that of the long-term resident of high altitude who shows true structural adaptation of the pulmonary system.
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Manuscripts Published or in Press


Abstracts


**Military Significance**

Our contract work is aimed at a better understanding of two physiological problems occurring in hypoxic environments which clearly affect the well-being and performance capabilities of the human sojourner at high altitudes. These problems are periodic breathing during sleep leading to loss of quality sleep and the resulting daytime hypsomnolence and fatigue; and the regulation of the ventilatory response and pulmonary gas exchange during exercise in hypoxia which are key determinants of exercise performance.

Our work on periodic breathing during hypoxic sleep provides the first comprehensive, quantitative description of this problem and provides the first definitive evidence detailing the major causes of periodicity and the reasons behind the beneficial effects of acute $O_2$ administration. Further, our more recent data suggests that acclimatization over a matter of a few days at high altitude may greatly alleviate periodic breathing during sleep. However, this remains a highly individual characteristic which we were unable to predict from available measurements. Indeed, the test of acute hypoxic ventilatory response—which is commonly used as a predictor of many facets of acclimatization—had no predictive value at all for the occurrence or severity of periodic breathing in hypoxic sleep.

Exercise capacity as determined by the pulmonary system in hypoxia and the debilitating symptoms of dyspnea which accompany exercise in hypoxia have been the subject of our investigations. Our work has detailed the critical limitations to oxygen transport presented by the failure of the lung's gas exchange and ventilatory control system and chest wall mechanics to respond adequately and/or efficiently to heavy work in hypoxic environments. Further, the baseline work in normoxic environments clearly shows the susceptibility of some highly fit individuals to these problems during exercise, thereby providing a basis for prediction of problems with high altitude exercise from measurements made at sea-level. We also showed the simple use of exercise tests in acute hypoxia—even using non-invasive measurements of arterial $O_2$ saturation—should provide excellent prediction of gas exchange "failure" at high altitudes. Our recent findings also strongly implicate a highly significant role for pulmonary and chest wall mechanics in the regulation of ventilation—and thus of gas exchange—during exercise—especially hypoxic exercise. We would predict with some confidence that the sea-level native with even "mild," asymptomatic airway disease (such as that due to chronic cigarette smoking or the mostly reversible airway disease of the otherwise healthy asthmatic) will have substantial problems in maintaining arterial oxygenation and/or avoiding extreme dyspnea during exercise at even mild elevations in altitude.

**Facilities and Personnel**

No changes were made in the past year.
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