

Contributions of lower limb and abdominal compression to ventilation inhomogeneity in hypergravity

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Abstract

Gravito-inertial load in the head-to-foot direction (Gz) and compression of the lower body half by an anti-G suit (AGS) are both known to influence ventilation distribution in the lungs. To study the interaction of Gz and AGS and to assess the separate contributions from lower limbs and abdominal compressions to large and small-scale ventilation inhomogeneities nine males performed SF₆/He vital capacity (VC) single-breath washouts at 1, 2, and 3 Gz in a centrifuge, with abdominal and/or lower limbs compressions. SF₆/He and (SF₆-He) phase III slopes were used for determination of overall and small-scale ventilation inhomogeneity. Closing volume and phase IV height were used as measures of large-scale inhomogeneity.

VC decreased marginally with G-load but markedly with lower limbs compression. Small-scale ventilation inhomogeneity increased slightly with G-load, but substantially with AGS pressurization. Small-scale ventilation inhomogeneity increased with AGS pressurization. Large-scale inhomogeneity increased markedly with G-load.

Translocation of blood to the lungs might be the key determinant for changes in small-scale ventilation inhomogeneity when pressurizing an AGS.

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1. Introduction

The lung is a gas exchange organ, designed to efficiently distribute and match air and blood flows. Due to its design it is highly sensitive to external forces such

as gravity, which is an important determinant of both ventilation and perfusion distribution. Gravity exerts its effects on ventilation distribution mainly via the pleural pressure gradient, which to a large degree determines regional lung expansion and ventilation (Bryan et al., 1966). In the upright position, gravity leads to a greater expansion of apical than basal lung regions and consequently ventilation (V') is preferentially directed to basal regions (Bryan et al., 1966; Milic-Emili et al.,

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1966). The hydrostatic pressure gradient results in a similar but steeper apico-basal gradient of lung perfusion (Q') (Hughes et al., 1968; West et al., 1964). As a result, the V'/Q' ratio is high in apical and low in basal lung regions. When increasing the gravito-inertial (G) force (here termed hypergravity) the differential effects of gravity on ventilation and perfusion distribution are put to their extremes (Green, 1999).

From a physical perspective gas transport can occur only by convection or molecular diffusion. When an inert non-resident gas is inhaled it is rapidly transported and distributed by convection through the first lung airway generations. As a result of the exponential increase in total airway cross-sectional area the linear velocity of the gas decreases dramatically in the peripheral conducting airways. When finally reaching the end of the lung, i.e. the wall of an alveolus, the convective transport of the gas must be zero. As a result a time-independent concentration profile, a quasi stationary front, called the diffusion front will arise in the peripheral airways in a zone, where diffusion and convection contribute equally to gas mixing (Paiva, 1973). The location of the zone where this front occurs depends on the diffusion coefficient of the gas molecule and the convective flow. With respect to the diffusion front, the lung can be divided into a purely convective zone, a diffusive-convective zone, and a purely diffusive zone.

The rate of molecular diffusion for a gas is inversely proportional to the square root of its mass. The molecular mass for SF_6 is 146 and for He it is 4, which means that He will mix by diffusion six times faster than SF_6 . The depth in the airway tree where the diffusion front arises for a given pattern of breathing will therefore differ for these gases. While for He the diffusion front is believed to occur close to the entrance to the acini (Paiva and Engel, 1989) it will occur a few airway generations deeper in the acinus for SF_6 (Paiva et al., 1988; Paiva and Engel, 1984). During the course of a normal breath, diffusion is thought to completely even out any concentration differences within airways located distally in the acinus.

Convection affects gases of different molecular mass equally. Convection dependent ventilation inhomogeneity (CDI) can be defined as non-uniform distribution of ventilation between parallel lung units separated at branch points, proximally in the airway tree such that diffusion cannot even out concentration differences between them, i.e. mouthward to the

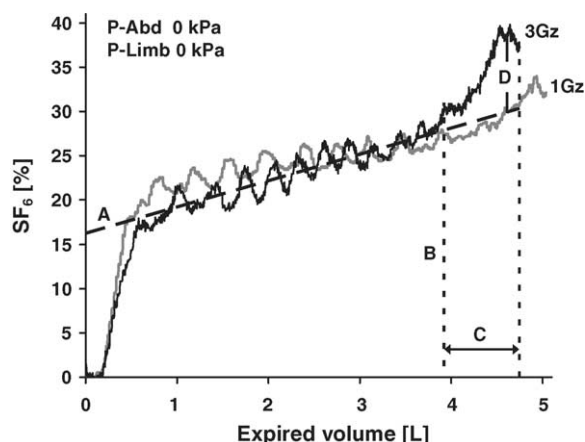


Fig. 1. Vital capacity single-breath washout curves from one test subject without pressurized anti-G suit. SF_6 recordings as a function of expired volumes from 1 (gray line) and 3 Gz (black line). Indications in the 3 Gz washout curve are: the phase III slope calculated between 25 and 75% of expired volume (A, broken line); the start of phase IV (B, vertical dashed line); closing volume (C, double headed arrow); and height of phase IV (D, vertical line).

diffusion front. If these units fill and empty sequentially, then CDI will result in a sloping alveolar phase (phase III) of the washout trace of an inert marker gas measured at the mouth (Engel et al., 1974; Engel and Paiva, 1981).

As a result of geometrical asymmetries at branch points where the diffusion front occurs (e.g. due to differences in subtended volumes or differences in cross-sectional areas at branch points) interaction between convection and diffusion can also give rise to a sloping phase III (Fig. 1) during expiration. This mechanism has been called diffusion interaction dependent inhomogeneity (DCDI), and has been shown to be the major contributing mechanism to the inhomogeneity (positive phase III slope) occurring during normal tidal breathing in healthy subjects (Crawford et al., 1985; Paiva et al., 1988; van Muylem et al., 1995).

The phase III slope of an inert marker gas is thus composed of the CDI and DCDI contributions. The CDI component may be due to the gravity-induced pleural pressure gradient causing differences in regional expansion between dependent and non-dependent lung regions, and may also result from other factors affecting regional differences in mechanical properties of the lung (Olson and Rodarte, 1984; Wilson et al., 1987; Lai-Fook and Rodarte, 1991). The

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