**Progress in Respiration Research**
Editor: H. Herzog, Basel

**Reprint**
Publishers: S. Karger, Basel
Printed in Switzerland

Prog. Resp. Res., vol. 21, pp. 117–120 (Karger, Basel 1986)

# Alveolar Plateau Caused by P-V Heterogeneity and Cardiogenic Oscillations Generated by Differential Effect of the Heart – A Model

*Ran Arieli, Fred Wiener*

Rappaport Family Institute for Research in the Medical Sciences and Department of Physiology and Biophysics, Faculty of Medicine, Technion-Israel Institute of Technology, Haifa, Israel

## *Introduction*

Inspired gas is distributed unevenly in the lung, and the single-breath $N_2$ washout and its modifications are probably the commonest tool to investigate this inhomogeneity. Recently, many studies have used computer models to investigate the distribution of gas to acini by the interaction of convection and diffusion [1]. Another approach to explain the sloping alveolar plateau is by sequential emptying, based on different pressure-volume (P-V) relations of the 2 lung units [2].

We describe a lung model for studying the effect of heterogeneity of P-V properties in the lung on the single-breath $N_2$ test where both instantaneous compliance and resistance are considered.

## *Methods*

The lung was pictured as consisting of 3 compartments, 2 which were affected by the pulsations of the heart, one of high compliance (HC) and another of low compliance (LC), plus a third, nonoscillatory compartment (NC). At FRC the volumes of HC, LC and NC were 39, 39 and 22%, respectively. Three sigmoid P-V curves were assigned to the 3 compartments, for both acini and airway (generation 11 to 23), so that total compliance summed up to 200 ml/cm $H_2O$. Bifurcation of NC was at generation 5/6 and that of HC and LC at generation 15/16 [3]. Resistance was calculated using Weibel's lung model and allowing turbulent flow in the upper airways. Nitrogen concentration at the mouth was calculated following $O_2$ inspiration from RV to TLC.

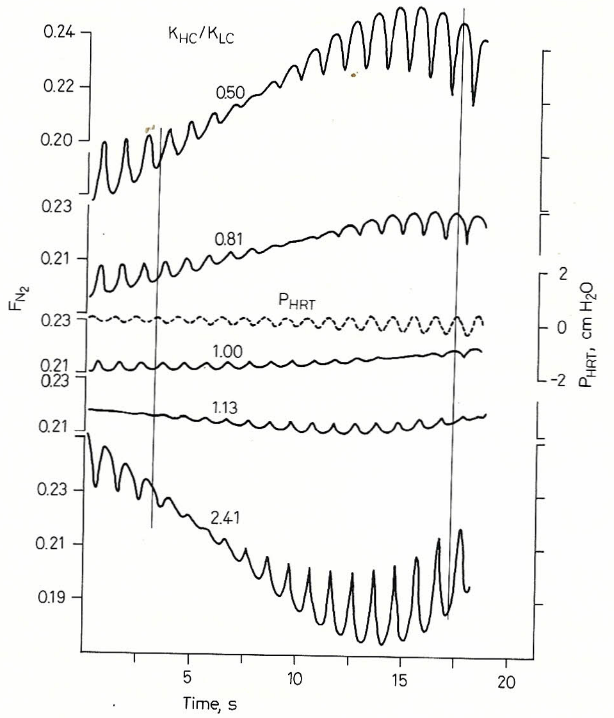


*Fig. 1.* Effect of ratio of the K's of P-V curves of HC and LC on expiration profile of $N_2$ ($F_{N_2}$) as function of time. Pressure pulsations produced by the heart ($P_{HRT}$) are shown and two vertical lines are drawn for comparison to CO. Expiration time was 19 s.

## *Results and Discussion*

Figure 1 shows the $N_2$ concentrations for different ratios of the P-V curve K's: HC/LC. As $K_{HC}/K_{LC}$ increased over 1.00, the $N_2$ profile became concave, and when $K_{HC}/K_{LC}$ was below 1.00, the $F_{N_2}$ profile was convex. At $K_{HC}/K_{LC}=1$, there was a moderate positive slope of the alveolar plateau. The cardiogenic oscillations (CO) were small at $K_{HC}/K_{LC}$ ratio of 1.00 and increased as $K_{HC}/K_{LC}$ became either smaller or larger than 1. At the ratio $K_{HC}/K_{LC} \leq 1.00$, there was a phase shift between cardiac pulsations and CO, as demonstrated by the 2 thin lines, which agrees with the experimental observations of Arieli [3]. The CO in the $N_2$ profile occurred when the flows from the oscillatory compartments (HC and LC) fluctuated widely with the cardiac pulsations while the flow from NC changed only

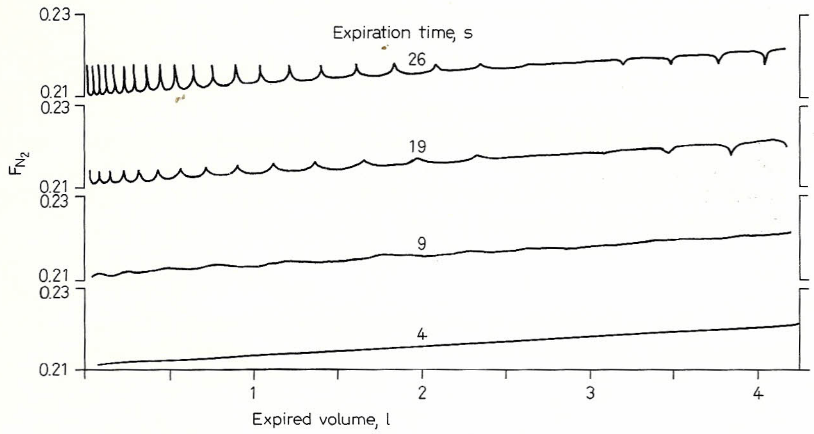


*Fig. 2.* Effect of expiration rate on profile of expired $N_2$.

slightly. Phase shift in the CO occurred when domination of flow from HC changed to domination of flow coming from LC. This phase shift occurred when expiratory $F_{N_2}$ equaled the $N_2$ concentration of NC. Increase in relative size of NC had a moderating effect, thus decreasing the concavity or steepness of the $N_2$ profile. Minor CO were seen when the volume of NC was zero. Only at late expiration was the difference in the time constants between HC and LC large enough to generate small but visible oscillations of $F_{N_2}$. Cardiogenic oscillations increased with size of NC up to 10% of the volume of FRC. Beyond 10% there was a very small difference in the amplitude of CO, but the spike-like wave became broader as the size of NC increased from 10 to 60% of the total lung volume.

Cardiogenic oscillations became smaller as the expiration was shortened (faster expiratory rate; see fig. 2). The amplitude of CO at 19 s expiration was one half the amplitude of 26 s expiration and 3 times the amplitude of 9 s expiration. The CO disappeared at 4 s expiration.

The disappearance of CO from the model in fast expiration is in accordance with experimental findings and agrees with the suggestion of Arieli [3] that when flow is resistance-dominated, CO should disappear.

We conclude that implementing 2 assumptions, (1) heterogeneity in P-V relations and (2) differential cardiac influence, one can reproduce different alveolar plateaus and cardiogenic oscillations which are similar to experimental findings.

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R. Arieli, PhD, Department of Physiology and Biophysics, Faculty of Medicine, Technion-Israel Institute of Technology, POB 9697, Haifa 31096 (Israel)