

CLINICAL USE OF OXYGEN STORES:

PRE-OXYGENATION AND APNEIC OXYGENATION

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INTRODUCTION

During states of respiratory arrest the human oxygen stores may be used therapeutically, regardless of the origin, i.e. either prior to the routinely induced apnea for endotracheal intubation or as an emergency measure in any other case of apnea. The present considerations focus on the clinical use of the oxygen stores available, applying

- * pre-oxygenation
and
- * apneic oxygenation.

OXYGEN STORES

Under physiological conditions, the total O₂ store of the human body (65 kgBW) contains about

- 300 ml O₂ physically dissolved or bound to myoglobin,
- 800 ml O₂ bound to hemoglobin (750g Hb, 1.39 ml/g, 15% arterial blood with sO₂ = 100%, 85% venous blood with a mean sO₂ of 75%),
- 400 ml O₂ within the functional residual capacity (3000 ml x 0.135),
- 1500 ml O₂ TOTAL (for comp. see [Nunn, 1987]).

During respiratory arrest, this total O₂ store theoretically could guarantee sufficient O₂ supply for approx. 3 minutes. In this case the arterial sO₂ decreases to 50% (pO₂ 25 mmHg), the mean venous sO₂ to 25% (i.e. an arterial plus venous sO₂ reduction of 50% = 500 ml O₂), simultaneously the alveolar pO₂ (pA_{O₂}) from 100 to 25 mmHg (i.e. = 300 ml O₂).

Consequently, the normal O₂ consumption of 750 ml (3 min x 250 ml/min) is guaranteed by the above calculated 800 ml O₂.

PRE-OXYGENATION

Following optimal pre-oxygenation with total denitrogenation, the pAO_2 increases to 673 mmHg, corresponding to 88.6% O_2 (pB 760 mmHg, $pACO_2$ 40 mmHg, pAH_2O 47 mmHg). In this case the intrapulmonary O_2 store amounts to 88.6% O_2 within the FRC of 3000 ml and an intrapulmonary oxygen store of 2650 ml O_2 is achieved.

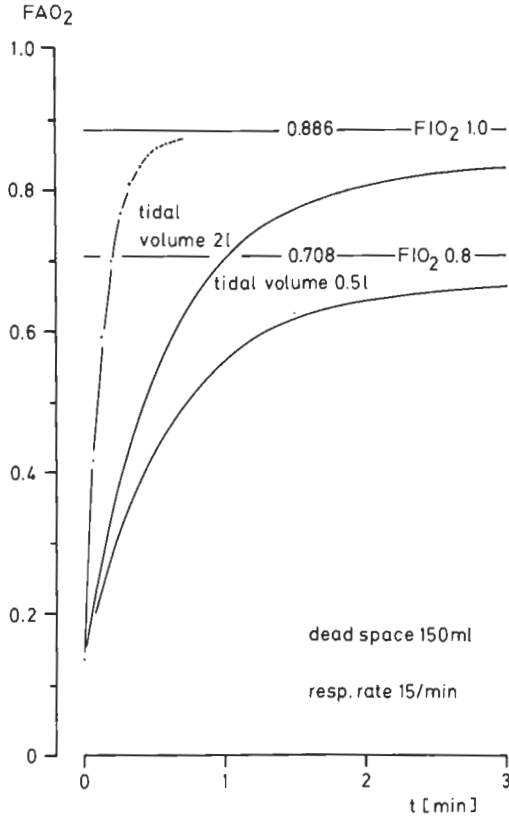


Fig. 1. Estimation of alveolar oxygen fraction (FAO_2) as a function of time during pre-oxygenation of a patient with pure O_2 . With normal ventilation (resp. rate 15/min, tidal volume 500 ml, dead space 150 ml) at FIO_2 1.0, the maximal FAO_2 of 0.886 is reached to 95% within about 3 min. When the inspired O_2 is diluted by ambient air (mask not tight, FIO_2 0.8), about 95% of the maximal FAO_2 of 0.708 are achieved within the same time. Hyperventilation with 10 breaths of 2 l O_2 shortens the time of pre-oxygenation to about 30 seconds.

Oxygen supply obviously is safely guaranteed for about 10 minutes among patients at rest (normal oxygen consumption of 250 ml/min), using this intrapulmonary O₂ store of 2650 ml alone.

However, pre-oxygenation per se is

- time consuming, and requires
- optimal systems for applying 100% O₂ (FIO₂ 1.0), i.e. for elimination of alveolar nitrogen.

Under these conditions only, pre-oxygenation could be successfully performed within 3 min breathing spontaneously or, within only 1 min when increasing tidal volume. Corresponding estimations are shown in figure 1.

The plotted data are in good agreement with published proposals: For instance, Gold et al. (1981) propose pre-oxygenation by four deep breaths during 30 sec, whereas Braun et al. (1980) or Berthoud et al. (1983) recommend 3 min of pre-oxygenation during normoventilation and using gas-tight fits of face masks.

APNEIC OXYGENATION

In case of subsequent apnea all gases present (N₂, CO₂, O₂) will equilibrate between alveolar space and capillary blood. Within 10 min of apnea the following diffusion rates between alveolar space and capillary blood can be estimated:

1) Nitrogen

The N₂ diffusion from the blood into the alveolar space may be calculated using different data:

a) Under extreme assumptions, i.e. total elimination of N₂ from mixed venous blood (pN₂ 626 mmHg: 760 mmHg - pO₂ 40 mmHg - pCO₂ 47 mmHg - pH₂O 47 mmHg), 5 l of blood (cardiac output) may release ca. 50 ml N₂/min (solubility = 0.012 ml/ml/atm). Therefore, the maximal and unrealistic value amounts to 500 ml N₂ per 10 min of apnea.

b) Compared to the total N₂ pool of 800 ml (400 ml dissolved in each body water and body fat [Farhi, 1964]), within 10 min only 25% of the total pool are assumed to be eliminated, i.e. only 200 ml N₂.

c) Miles et al. (1956), in contrary, have determined a N₂ elimination rate by the lungs of 500 ml during 30 min following optimal pre-oxygenation. This corresponds to ca. 170 ml N₂ for a 10 min period of apnea.

d) However, according to Farhi (1964), the pN₂ in mixed-venous blood decreases to about 10% of the initial value, i.e. 60 mmHg, after 10 min of pre-oxygenation under optimal conditions. Thus, the maximum amount of N₂ diffusion would be only 50 ml per 10 minutes.

In conclusion, it can be predicted that, during a 10 min apnea not more than 200 ml of N₂ diffuse from blood into the alveolar space under the condition of an optimal pre-oxygenation.

2) Carbon dioxide

During apnea the pCO₂ of mixed-venous (p \bar{v} CO₂) blood comes into equilibrium with the alveolar pCO₂ (pACO₂) as well as with the arterial pCO₂ (paCO₂). Within the first minute, the paCO₂ increases by about 10 - 13 mmHg corresponding to the three following factors:

- equilibration of the paCO₂ (40 mmHg) by the p \bar{v} CO₂ (47 mmHg),
- increase of paCO₂ by about 3 mmHg above p \bar{v} CO₂ as a consequence of the Christiansen-Douglas-Haldane effect, i.e. O₂ uptake into the blood without CO₂ release [Mertzlufft et al., 1989], and
- increase of paCO₂ by about 3 mmHg resulting from CO₂ production.

In practice, an increase of about 3-4 mmHg/min from the 2nd to the 10th minute has been measured by various authors. The corresponding data of paCO₂ increase in humans during hyperoxic hypercapnia are listed in figure 2, the resulting increase for a 10 min apnea is shown in figure 3.

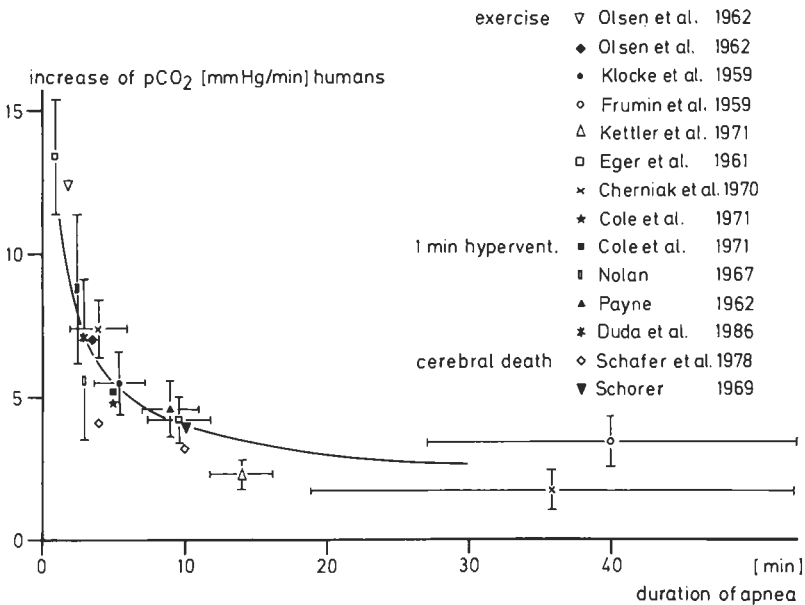


Fig. 2. Increase of arterial or alveolar pCO₂ (mmHg/min) during apnea in humans, i.e. hyperoxic hypercapnia, according to various authors.

The $p\text{CO}_2$ increase of 40 mmHg as shown in figure 3 for a 10 min period of apnea, corresponds to a 5.3% CO_2 increase within the alveolar space and, therefore, to only 160 ml CO_2 (FRC 3000 ml) entering the FRC.

The remaining 90% of the metabolically produced CO_2 are stored within the extracellular space of the body.

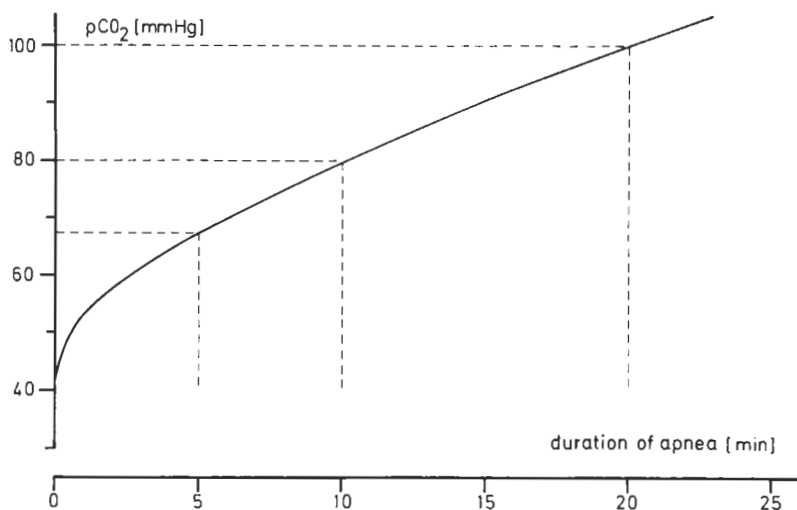


Fig. 3. Increase of the arterial $p\text{CO}_2$ in the human under "normal" conditions of hyperoxic hypercapnia during apnea.

Values of 160 - 170 mmHg of $p\text{aCO}_2$ [Payne, 1962; Birt et al., 1965] up to 200 - 250 mmHg [Ellison et al., 1955; Frumin et al., 1959] have been measured in humans during hyperoxic hypercapnia without any ill effects.

3) Oxygen

The Oxygen consumption of 250 ml/min under normal conditions will be reduced to about 200 ml/min under anaesthesia. Thus, 2000 ml O_2 will have to diffuse from the alveolar space into the arterial blood. This is achieved by the steep alveolar-capillary $p\text{O}_2$ gradient ($p\text{AO}_2$ 673 mmHg) due to optimal pre-oxygenation.

Patent airways provided, in the present case of a 10 min apnea the diffusion balance of all three gases in question causes a gas uptake by mass-movement as described by Volhard (1908) and Draper et al. (1944).

This mass-movement amounts to either

- 1640 ml during administration of pure oxygen (2000 ml O₂ - 160 ml CO₂ - 200 ml N₂) or to
- 1840 ml during the exposition to ambient air (2000 ml O₂ - 160 ml CO₂).

Ambient air

If a patient is exposed to ambient air instead of pure oxygen, the mass-movement will be increased to 1840 ml, as the N₂ of ambient air stops the N₂ diffusion from capillary blood into the alveolar space.

These 1840 ml of ambient air (21% O₂) entering the alveolar space by mass-movement, finally only contain 19.7% oxygen due to humidification (pH₂O of 47 mmHg) within the dead space.

Therefore, within 10 min of apnea only 2000 ml O₂ from the initial FRC store of 2650 ml O₂ are consumed, while 360 ml O₂ are replaced by mass-movement (1840 ml x 0.197).

The remaining 1010 ml O₂ (2650 ml O₂ - 2000 ml O₂ + 360 ml O₂) correspond to a FAO₂ of 0.336 or to a pAO₂ of 256 mmHg. Thus, the pAO₂ has been decreased from 673 mmHg to 256 mmHg, i.e. a decrease of 417 mmHg during the 10 min apnea or a decrease of about 40 mmHg per minute.

Pure oxygen

If the patient is connected to a source supplying pure oxygen, the mass-movement as compared to ambient air is only 1640 ml per 10 min but, containing 100% oxygen (corresponding to an O₂ concentration reduced to 93.8% after humidification).

Starting with a total FRC store of 2650 ml O₂ following pre-oxygenation, the patient consumes 2000 ml O₂ while 1540 ml O₂ are being replaced by 1640 ml mass-movement (1640 ml x 0.938, after humidification). Thus, 2190 ml O₂ remain within the FRC store (2650 ml O₂ store - 2000 ml O₂ consumption + 1540 ml O₂ replacement).

The remaining 2190 ml O₂ correspond to a pAO₂ of 555 mmHg. As the result, the pAO₂ has only slightly been decreased from 673 to 555 mmHg after a 10 min period of apnea.

The described mass-movement is the basic mechanism of the so-called apneic oxygenation, formerly termed diffusion respiration.

The possibility to achieve adequate oxygenation with apneic oxygenation has been demonstrated in both animals and man, in man for up to 40 min of apnea. For details see e.g. Nunn (1987).

SUMMARY

Optimal pre-oxygenation prior to apnea plus administration of pure O₂ during apnea provide adequate O₂ supply for patients with very high security, if required for at least 30 minutes.

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