

Physics of compression chambers and physiological physics of hyperbaric environments

A comprehensive treatise of well known and under-exposed effects

Part 2. Implications of the gas laws for chamber residents and divers

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Voorwoord

Dit deel behandelt de implicaties van de gas-wetten en de wetten voor gastransport door luchtpijpjes bij mensen onder hyperbare druk. Het is onder duikers algemeen bekend dat bij het oppersen van gasflessen de flessen warm worden en daarom vaak op een iets hogere druk dan de standaard van 200 bar worden afgeperst. Het proces van oppersen is derhalve deels adiabatisch. Leerboeken geven geen uitsluitsel over de vraag of in het lichaam ook adiabatische processen plaatsvinden, een reden om dit hier te behandelen. Weinig leerboeken beschrijven het mechanisme van de inerte gas narcose terwijl in elke handleiding voor de leek de stikstof narcose behandeld wordt. Een ander onderwerp dat men in de (inleidende) standaardwerken nauwelijks behandeld ziet is de longmechanica onder druk.

Deel 2 begint met het memoreren van het belang van de gaswetten voor de duikerziekten en doet uit de doeken waarom luchtbehandelingen van deco-ziekte op 30 en 50 m op fysische gronden te ontraden zijn. Hierna volgen adiabatische verschijnselen in het lichaam. Daarna volgt een uiteenzetting over zware fysieke inspanning in een droge hyperbare kamer. De vraag wordt beantwoord wanneer warmte stuwung optreedt en hoe dit te bestrijden is, een zeer relevant onderwerp voor bijvoorbeeld caissonwerkers.

Na de fysica van de gasdiffusie in vloeistoffen wordt de theorie van de hyperbare inerte gasnarcose en het HPN-syndroom behandeld. Hierna volgt een kleine uitwijding over de theorie van hypercapnie en tenslotte wordt veel aandacht besteed aan de hyperbare longmechanica waarbij gepoogd wordt na te gaan wat de theoretische diepte-grens van de gas-uitwisseling is.

See for a preface of this part the preface of Part 1, The physics of peopled compression chambers.

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Notes:

- In this manuscript SI units or their derivatives are preferably used.
- In Figures 2.3 and 2.4 are modified from van Grol (1977).

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2.1. Barotraumatata explained by Boyle's Law

These traumata have all to do with pressurizing or depressurizing gas filled cavities in the body, which are reigned by Boyle's Law. These disorders are extensively discussed in all types of textbooks at the various levels (Bennett and Eliot, 1978; Edmunds et al., 1992, Lawrence, 1997). Therefore, they are not discussed.

2.2 Effects related to law of Gay-Lussac and law of Dalton

Between the lungs and the ambient gas there is a temperature difference. In the lungs, the inspired air will be heated and therefore during the breath hold phase of a breathing cycle the lungs will expands. Although the effect is small, it may be relevant in calculations. Knowing the temperatures, the expansion factor can be calculated, as illustrated in the following examples.

For the chamber resident: $V_{\text{inspiration, lung}}/V_{\text{inspiration, chamber}} = T_{\text{body}}/T_{\text{chamber}} = 310/298 = 1.040$

For the (ascending!) diver: $V_{\text{inspiration, lung}}/V_{\text{inspiration, bottle}} = T_{\text{body}}/T_{\text{water}} = 308/278 = 1.108$

In (very) cold water, breath holding during a small Scuba ascent has more risk since the $V_{\text{inspiration}}$ is larger than in warm water. Under certain conditions the combination of pressure and temperature effect may be risky. Suppose an ascent during breath holding is made from 6 to 4 m depth, but due to waves actually the ascent is from 8 to 4 meters. This yields a factor of expansion of 1.286. Since the inspired air is also vaporised there is a third factor of expansion, being 1.031 (1+0.062/1.8) (see Table 2.1). Together this yields an expansion of the lungs during the breath-hold of $1.108 \times 1.286 \times 1.031 = 1.46$. An inspiration of more than, say, 70% of vital capacity is risky, since a part of the expansion of the lung cannot be realized without damage.

Summarizing, the often forgotten effects of heating and vaporising of the inspired air is relevant for physiological calculations and may be of importance in diving practise.

Dalton's law applied to the composition of inspired and end-tidal air yields Table 2.1.

Table 2.1

components of inspired air		N ₂	O ₂	CO ₂	H ₂ O	remainder					
Dalton's law:	p_{bar}	=	p_{N_2}	+	p_{O_2}	+	p_{CO_2}	+	$p_{\text{H}_2\text{O}}$	+	p_{rem}
in Pa	101325	=	78852	+	21153	+	37	+	304	+	979
in vol%:	100.000	=	77.821	+	20.876	+	0.037	+	0.3	+	0.966
components of end-tidal ¹ air		N ₂	O ₂	CO ₂	H ₂ O	remainder					
Dalton's law:	p_{bar}	=	p_{N_2}	+	p_{O_2}	+	p_{CO_2}	+	$p_{\text{H}_2\text{O}}$	+	p_{rem}
in Pa	101325	=	74423	+	15826	+	3850	+	6262	+	963
in vol%:	100.00	=	73.45	+	15.62	+	3.8	+	6.18	+	0.95

In calculations of respiratory (clinical) physiology the next measures are often practised:

STPD is Standard Temperature & Pressure Dry, so at 1 atm, 0 °C and $p_{\text{H}_2\text{O}}=0$.

1 litre STPD is abbreviated as 1 l_n.

BTPS is Body Temperature & Pressure Saturated, defined at 37 °C, ambient pressure minus $p_{\text{H}_2\text{O}} = 6.3$ kPa (47 mm Hg).

In this paper the abbreviated STPD and BTPS are avoided. More common are:

RMV / Respiratory Minute Volume;

V_{O_2} / minute respiratory oxygen uptake in STPD;

V_{CO_2} / minute carbon dioxide production in STPD.

V_{O_2} and V_{CO_2} are never given with BTPS conditions.

2.3 Do adiabatic phenomena occur?

The question arises how much the alveolar gas is heated when a skin diver dives with a velocity of 0.5 m/s. At 0.5 m depth, the temperature is expected to increase to 4.35 K ($1.05^{0.4/1.4} \times 310$, see Part 1, §1.2.2). The deeper, the less the temperature rises per meter. From 10 to 10.5 m it is 2.19 K. When the effects over the whole diving depth are cumulative, the skin diver would "burn" his alveoli. This clearly not happens, since the heat transfer to the surrounding tissue (total alveolar surface is about 10 m² with a blood flow of 150 ml/s) is sufficient to prevent heating.

Next, the question arises what happens when somebody dives from the 10 m diving board within 0.5 s from the surface to a depth of 3 m. Now, the increase is 24.1 K. However, this will not happen. The compression of the lungs is delayed due to the properties of inertia of the chest wall. Therefore, the pressure of 1.3 bar is reached after a larger interval of time (some 2 s). So, the temperature effect is smaller since heat transfer directly starts to occur.

¹ End-tidal gas is the gas expired at the end of the expiration

This probably limits the temperature increase below 45 °C. I suppose that such temperatures are not harmful during short exposures, since ambient air temperatures of about 50 °C can be inspired for hours.

2.4 Non-barotrauma explained by Daltons and Henry's law

2.4.1 Decompression sickness and the discredit of the therapeutic air tables

Decompression sickness occurs after ascent and is based on Henry's law (see §2.2.2). This law gives the relation between the amount of dissolved gas in a liquid (e.g. N₂ in blood) and the partial pressure of this gas in the ambient gas mixture (alveolar gas), which is in equilibrium with the liquid. In other words, the liquid is saturated with the gas. When the partial pressure in the gas is suddenly decreased (by e.g. reducing the total pressure, as happens during ascent of a diver), the liquid becomes supersaturated and small gas bubbles will appear.

In the past decades therapeutic air tables starting at 50 and 30 m for treatment of DC were used in addition to oxygen-air tables. In the last decade the frequency of application of the air tables was strongly diminished. This change in practise was mainly evidence based. The question arises whether the new practise has also a medical-physical basis. Below, the answer is given.

Suppose that spherical bubbles are present at 1 bar, then the change of bubble volume V and diameter D can be calculated (by Boyle's law) under the assumption that there is no diffusion from the gas to the liquid phase of the blood (as holds rather well for a surface recompression) and that the shape remains spherical. The volume V_d of a spherical bubble decreases linear with the pressure. So, $V_d = V_o/(1+d/10)$ with V_o is the volume at depth d (d in meter). The change of volume as a function of depth is illustrated by the curve (\square) of Fig. 2.1 However, bubble diameter changes with the cubic root, being $(1/(1+10/d))^{1/3}$. This change is visualized in the upper curve of Fig. 2.1 (small \circ) and the upper equation of the inset gives the relation between diameter and volume, irrespective pressure. Increasing pressure from 1 to 6 bar gives a reduction to 0.55 (45%) of sphere diameter, which decreases with the cubic root of the pressure.

Actually, in the blood, the bubbles are generally not spherical but more or less elliptical. The middle curve (mediate \circ) of Fig. 2.1 gives the reduction for an ellipsoid bubble with a long/short axis ratio L/D (is length/diameter; both short axes are identical) of 2, irrespective the depth. At 1 bar the volume of sphere and ellipsoid are the same (normalised at 1). Although the diameter is 0.79 times that of the sphere, the relative change of D of the sphere and ellipsoid is the same as function of pressure. The middle equation of the inset gives the relation between D and D_{sphere} , which holds for all depths.

Actually, in the blood, constancy of shape does not happen since in the small arterioles at 1 bar many bubbles are long shaped and at 6 bar they are much more spherical. The lower curve (large \circ) gives a model of this behaviour. To understand the behaviour of the diameter it is assumed that at 1 bar L/D is 4 and at 6 bar 2. Here, the L/D ratio reduces linear with d . At one bar, the volume of this elongated spheroid is also normalized at 1. The bottom equation of the inset presents D as function of d expressed in D_{sphere} at the given d . In the figure it can be seen that the diameter-change of this bubble changes much less. At 0 m, D of the long bubble is $4^{-1/3} = 0.63$ and at 50 m $2^{-1/3} \times 0.55 = 0.44$ (the same as for the elliptic bubble). Consequently, the long bubble is reduced in size only 31%.

Of course, D is the relevant parameter for the occurrence of DCS. Therefore, we have to conclude that on the basics of the bubble physics in small arterioles:

treatment with Table 1A, 2A, 3 and the mixed tables 6A and 4 starting with air is highly inadequate if not dangerous.

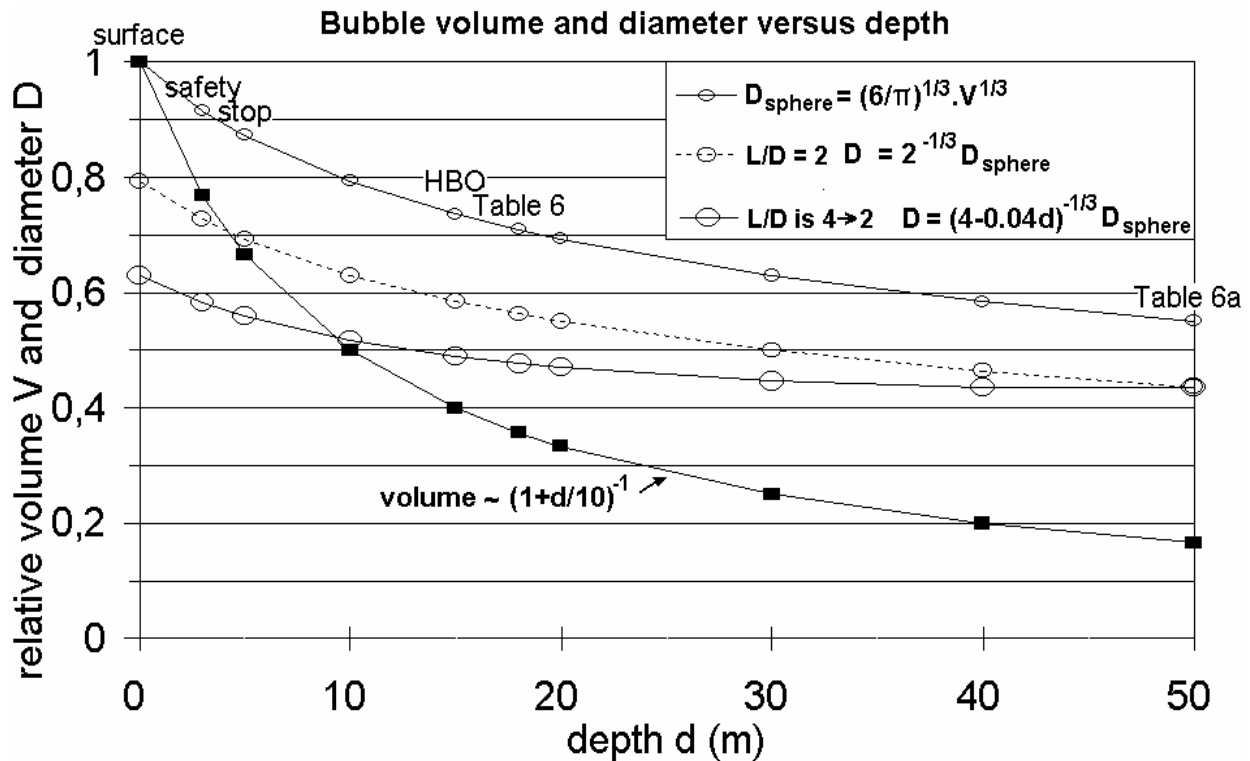


Fig. 2.1 Influence of bubble shape on bubble diameter as function of depth. Specific depth are indicated above the upper curve (surface at 0 m, safety stop at 3-6 m, HBO treatment at 15 m, Table 6 treatment at 18 m and Table 6a treatment at 50 m). The curves with open symbols present diameters. At 0 m the volumes of the three types of bubbles are the same.

When nevertheless applied, it would be interesting to apply a Heliox mixture at 30 and 50 m, since the water solubility of He is about 30% lower than that of N₂, but its diffusion coefficient is about 2½ times larger (see §2.2.2).

2.2 Gas diffusion and absorption

A liquid is incompressible since the molecules are very tightly packed. The distance between water molecules is ca. 0.31 nm, resulting in a free space of 0.05 nm. They attract each other substantially and collide continuously with each other and the molecular velocity is much lower than in the gas phase.

When the liquid is free of gas molecules, they penetrate massively the liquid via the free space (holes). Finally, in equilibrium per unit of time as many as molecules, e.g. N₂ molecules of the gas mixture, penetrate and leave the water. Then pN₂ below the gas-liquid interface, which is proportional with the number of absorbed N₂ molecules, is the same as above the interface. The time course of the amount of gas molecules just below the liquid interface is exponential as illustrated in Fig. 2.2. At some cm below the surface the curve is sigmoidal. For O₂ the same story holds, but equilibrium is reached slightly later since O₂ penetrates more slowly. In the gas as well as liquid phase Dalton's law hold.

The next question is how absorption of gas occurs in the tissues of an HBO patient or diver

when pressure is (step-wise) increased? At “sea-level”, the body is saturated with 0.79 bar of N_2 . At the instant of pressure increase, N_2 molecules penetrate the alveolar-capillary wall and dissolve in the blood. Via the capillaries, the tissues are supplied with N_2 molecules and consequently pN_2 increases. The distance between the blood capillaries is of the order of 50 μm . Therefore, the increase of the amount of absorbed gas in a watery tissue can be described rather well with an exponential curve as illustrated in Fig. 2.3. The curve is however delayed with respect of the curve in the arterial blood.

The velocity of the diffusion process is given by diffusion constant $D_{\text{gas,liquid}}$ (D is gas, liquid and temperature dependent). D increases with temperature, due to the higher molecular velocity. Generally speaking, D decreases with molecular diameter. For a mono-atomic gas, D is well correlated (negatively) with the diameter.

The amount of dissolved gas C is given by the gas absorption coefficient q_v times the partial gas pressure p :

$$\text{Henry's Law: } C = q \cdot p. \quad (2.1)$$

As holds for D , the gas absorption coefficient is also dependent on the gas, the liquid and the temperature. The higher the temperature, the lower the constant. This is due to the higher velocity of the liquid as well as the gas molecules, resulting in less availability of the holes. It can be expressed in mass/volume liquid, C_m , or gas-volume/volume, C_v (then e.g. as l_n/l).

The rate of diffusion and the amount of absorbed gas are completely different physical parameters. The first one has as a constant of proportionality the diffusion constant D and the second one on the absorption coefficient q . Values of both are given in Table 2.2.

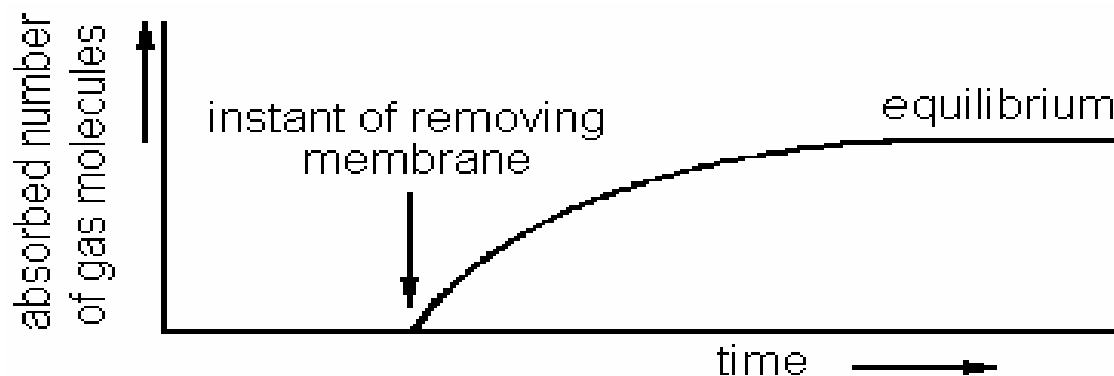


Fig. 2.2 Time course of gas diffusion just below the gas-liquid interface. Originally, the liquid-surface was covered by a gas-impermeable membrane. The instant of removal is indicated.

Table 2.2 Absorption and diffusion coefficients

gas	Absorption-coefficient in water (Bunsen) q_v ($\text{ml}_n \cdot \text{l}^{-1} \cdot \text{bar}^{-1}$) #			Diffusion constant in mouse muscle & $D_{\text{gas,water}}$ ($10^{-10} \text{ m}^2 \cdot \text{s}^{-1} \cdot \text{bar}^{-1}$)	dynamic viscosity coefficient η • $\text{Pa} \cdot \text{s} \cdot 10^{-6}$ (273 K)
	EC	0	#		
H ₂	-	-	-	24	8.4
He	-	-	0.008	4	18.6
N ₂	0.024	0.014	0.012	14*	17
O ₂	0.049	0.028	0.024	15	18.9
CO ₂	-	-	0.570	11	13.9
CO	-	-	0.018		

The q_v of fatty tissue is about 5 times that of watery tissues.

These values hold for blood and watery tissues. The values for fatty tissues are about 5 times larger.
& Kawashiro, 1975.

* Estimate of NS.

• PTZ, 1993.

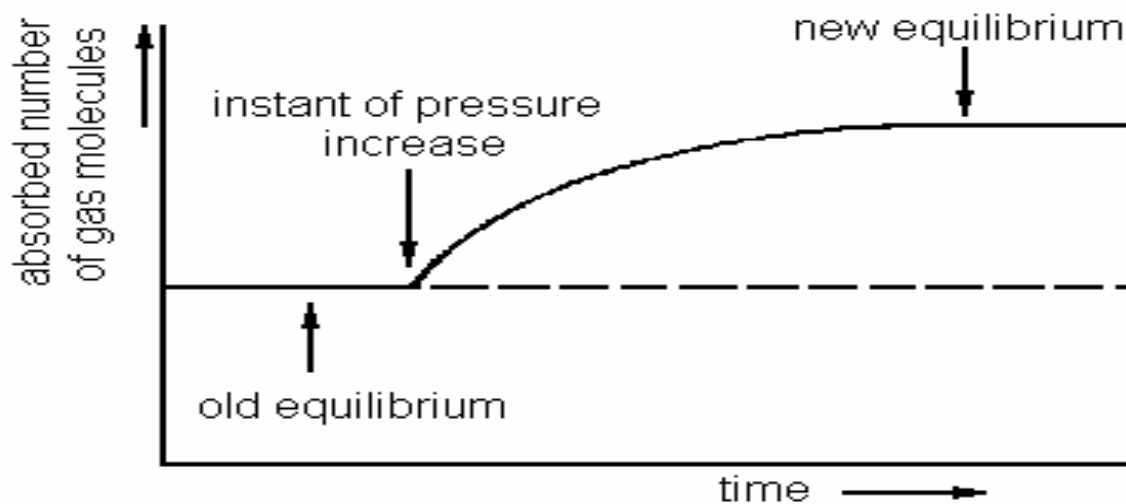


Fig. 2.3 Increase of partial pressure in the non-blood watery tissues.

Due to Henry's law, during HBO treatment plasma-dissolved O₂ is about 1/3 of the O₂ bound to Hb, as illustrated in Fig. 2.4.

Knowing q_v of N₂ and O₂, the amount of nitrogen and oxygen in a diver can be estimated.

Suppose, that the total body volume is 75 l with 80% watery tissues and 20% fatty tissues (20 years old male), and the mean body temperature is 35 °C.

At 1 bar pO₂ and pN₂ are 0.195 and 0.79 bar respectively.

For nitrogen holds:

watery tissues $60 \times 0.79 \times 0.0143 = 0.678 \text{ l}_n$;

fatty tissues $15 \times 0.79 \times (0.0143 \times 5) = 0.846 \text{ l}_n$;

in total ca. 1.5 l_n .

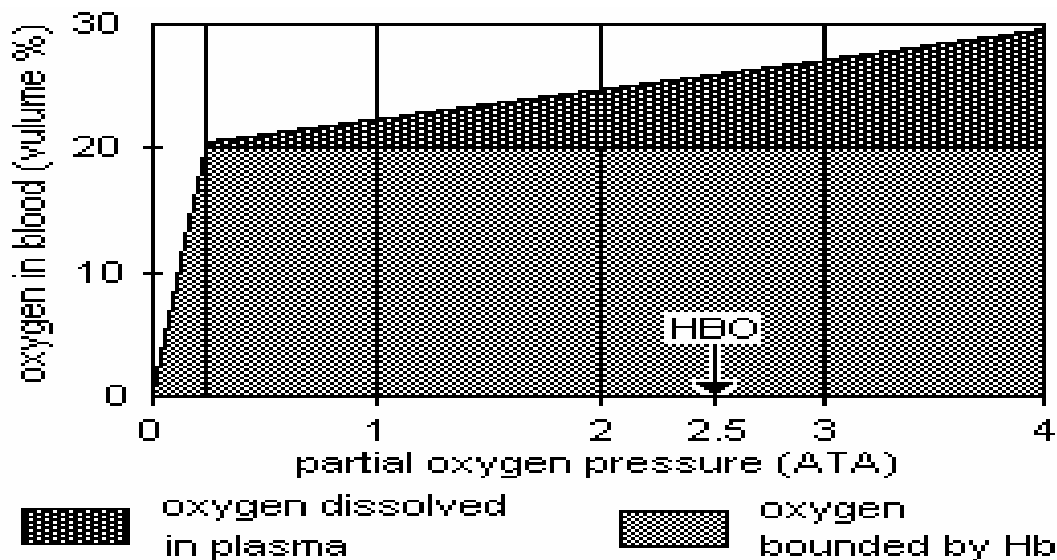


Fig. 2.4 Relation between ambient p_{O_2} and dissolved oxygen in plasma

At a depth of d meter, these values should be multiplied with the factor $(d/10+1)$. This results in the rule of thumb that:

FOR ASCENDING TO SURFACE, A SATURATED DIVER SHOULD RELEASE ABOUT 1.5 L_N OF N_2 FOR EACH 10 M OF DEPTH.

For a saturation dive at 30 m, the volume of absorbed N_2 (relative to 0 m) is $3 \times 0.678 l_n$ in the watery tissues (see below; diver 75 kg). For a bottom time of 20 min this is about $0.5 \times 3 \times 0.678 = 1.02 l_n$. A very rapid surface-ascent produces millions of bubbles. Under the assumption that this volume completely becomes gaseous at the surface and is transformed to bubbles with a diameter of 0.5 mm, 15.6 million bubbles are produced. So, 5 litre of blood will comprise 1.3 million bubbles. This will evoke serious *neurological disorders* and possible *heart failure*.

2.4.3 Non-barotrauma explained by membrane biophysics: state of the art of the biophysics of hyperbaric inert gas narcosis

Nitrogen narcosis is an *acute* pN_2 effect (Dalton) and is comparable with inert gas narcosis by N_2O and SF_6 , gases practised for decades in the surgical theatre.

The narcotic effect of an inert gas is well correlated with the olive oil/gas partition coefficient and not related to the hydrate dissociation pressure (Östlund et al., 1994). Therefore, the inert gas is supposed to exert its effect in the phospholipid bi-layer and not in a watery phase. The narcotic action of the dissolved inert gas is probably due to conformational changes in the lipid bi-layer or at some other hydrophobic site in excitable membranes (see Östlund et al., 1994), which disturbs the neurophysiological function of axons, dendrites (propagation) and (or) synapses (transmission). However, the molecular mechanism of inert gas narcosis is still enigmatic. Some decades ago, Miller and co-workers (1977) made an important contribution to the understanding of the mechanism with his critical volume theory, which will be discussed in short below.

Box 2.1 The expansion can be expressed by the simplified equation:

$$E = V_{\text{gas}} \cdot x \cdot p_{\text{gas}} / V_m \cdot \beta \cdot p_{\text{gas}}, \quad (2.2)$$

with V_{gas} the partial molar volume of the gas dissolved in the partial molar volume V_m of the membrane (fluid), x the partial fraction solubility at 1 bar, and β the compressibility of the membrane.

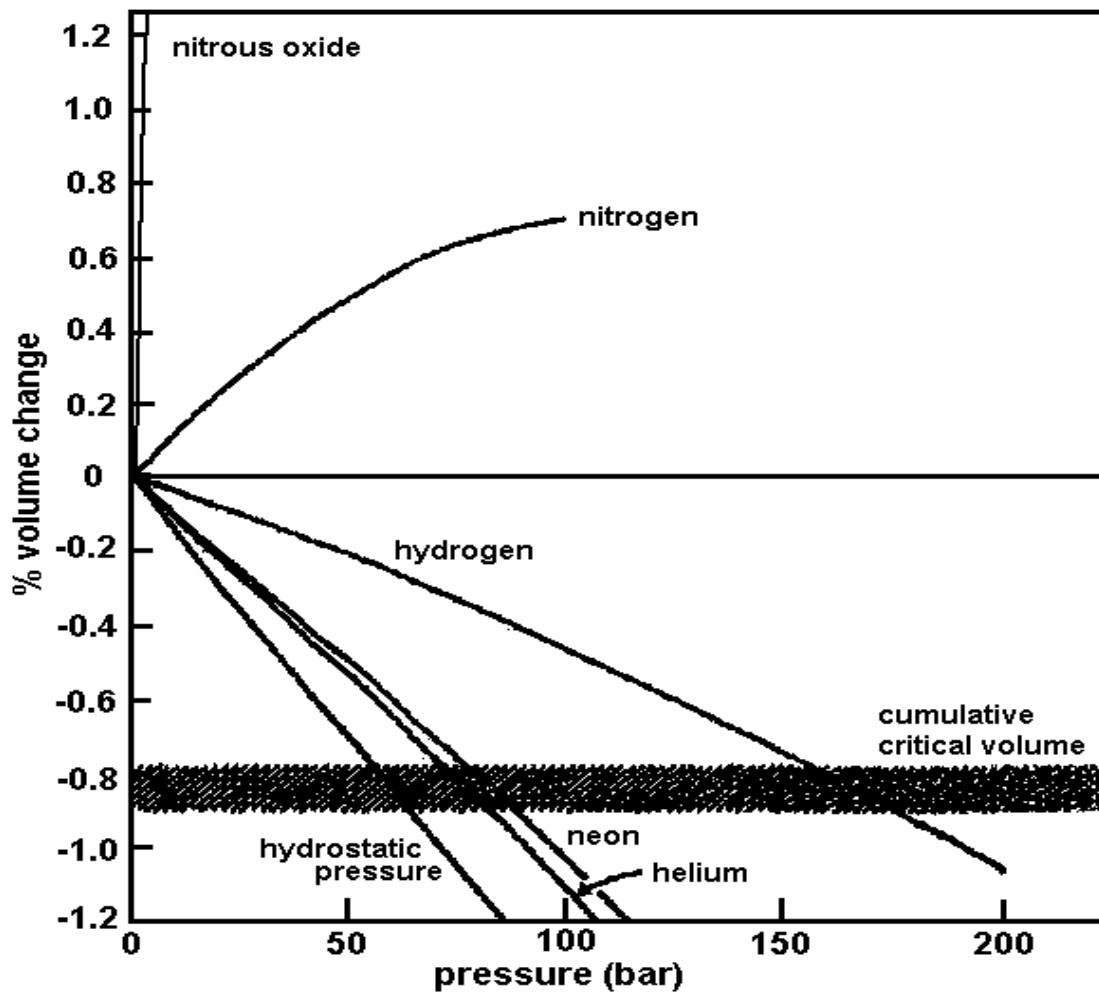


Fig. 2.5 Critical volume hypothesis with the effect of the various inert gases. (After Miller, 1977a.)

Water is, although very slightly, compressible. At low pressure and 35 °C the compressibility coefficient is $0.044 \times 10^{-3}/\text{bar}$ (HP&C, 1972). The phospholipid-like membranes have a coefficient of about $0.1 \times 10^{-3}/\text{bar}$ (see Aldridge and Bruner, 1985; Beney et al., 1997). The critical volume theory distinguishes two effects. The compressibility itself causes a strong neurological effect, starting with hyperexcitability resulting in tremor of extremities. When the compression reaches a critical value, convulsions occur. These disorders are mentioned the hyperpressure nervous syndrome (HPNS). From animal experiments it appeared that only extreme pressures, above 200 bar, finally result in paralysis

and death (Miller, 1977). All inert gases, dissolved in the membrane cause a volume expansion of the membrane, which could be established by manipulating the partial pressures of mixtures comprising two inert gases and oxygen.

The compressibility of the membrane causes a capacity change, but this is too small to be relevant for a neural effect. I hypothesize that a possible candidate for the effect is the observed decrease of the carrier mediated K^+ -conductance with pressures up to 1000 bar (Aldridge and Bruner, 1985).

In contrast to the exciting effect of pressure, the inert gases give a depressive effect. Anaesthesia and analgesia occur when the partial pressure is high enough. Both effects counteract, which is dependent on the type of inert gas, the partial pressures and the total pressure. Fig. 2.5 shows the net effect of the pressure dependent compression and the narcotic effect (Miller et al, 1977). Nitrogen has a nearly 10 times higher fat-solubility than He, which causes the membrane to increase more in volume, which is thought to cause the narcotic effects. In a more advanced version of the critical volume hypothesis there are two sites of action of an inert molecule, one in a lipid-like phase with high solubility and low compressibility with a high narcotic effect, and the other in much more compressible phase with a slightly lower solubility and an anti-hyperexcitability effect (Miller et al, 1978?).

The solubility of He in the membranes is very low. Therefore at pressures of some 20 bar with Heliox the narcotic effects are small. The helium tremor is caused by the pressure rather than by the helium. The figure suggests that a Heliox mixture (1 bar oxygen) with about 7.5% of nitrogen is optimal. The nitrogen will counteract the pressure effect.

2.2.4 Non-barotrauma explained by gas rheology and blood (bio)chemistry: hypercapnia

Although the CO_2 level in the blood is unchanged by an increase in the ambient pressure (i.e., the depth) per se, an inadequate ventilatory release of CO_2 can easily result in hypercapnia (high $[CO_2]$ in blood). The expired gas shows a decreased $[CO_2]$. When the level is high enough it can cause CO_2 toxicity, which can lead to shortness of breath, headache, confusion and, when diving, to drowning.

Hypercapnia can easily occur during:

- HBO;
- compressed-air-working;
- scuba diving;
- breath-hold diving.

Hypercapnia is caused by three reasons.

1. The density of the inspired gas increases with pressure. For a perfectly laminar flow the resistance does not increase and this is independent of the density of the gas (see §2.6). However, this does not hold. at bifurcations of bronchi, when the wall is not smooth, and when there are stenoses. The increased resistance results in an increase in ventilation labour and often it causes shallow breaths. This causes a less efficient release of CO_2 , which results in a moderate hypercapnia.
2. Decreased CO_2 release due to impaired venous transport of HCO_3^- .
3. Decreased binding capacity of CO_2 to the alpha chain of Hb.

As noticed, shallow breathing promotes hypercapnia, but also increases heat loss (see Part 1 §1.2.3) and ventilatory work since RMV is increased (§2.6). When diving, a $V_{\text{inspiration}}$ 3 times that in rest is rather optimal, and during the safety stop 2 times (see §2.2).

Conclusion: increase your tidal volume when under pressure.

Some hypercapnia related items

Exertion Heavy work or deep diving give rise to a higher CO₂ production, which causes shallow breathing and consequently a higher blood [CO₂] (CO₂ toxicity).

Saving air Some experienced divers practice "skip" breathing, which is holding the breath in order to conserve air. This might save air but also leads to CO₂ build-up.

Influence at vessel diameter Hypercapnia causes vaso-dilatation in brain vessels. This is counter-acted by the vaso-constriction due to hyperoxia. During HBO treatment of (neurological) patients, the net effect is not known.

2.3. Thermal stress of compressed air workers

Although the heat-loss calculations of Part 1 §1.2.3 may be interesting, their relevance for the practise of operating chambers is limited. However, it is a good starting point to discuss the problem of hyperthermia of compressed air workers. This subject can be found in Parsons (1994). Work classified as heavy to very heavy asks for a V_{O₂} of 2.57 l/min (the standard subject of 75 kg), 8 times the V_{O₂} at rest (Lanphier, 1975). This is equivalent to a RMV_{ins} of 44.8 l/min and it results in a production of 727 W. Muscular efficiency is about 25% (Bernards and Bouwman, Ch. 15, 1988), resulting in a heat production of 545 W. At 2.5 bar, the work load mentioned above can be maintained for some hours. At this pressure the RMV_{ins} of 44.8 l/min is about half the maximal value (Lanphier, 1975). Compressed air work is often done with ambient temperatures well above 30 °C. This means that there is hardly any heat loss by radiation, by heating the inspired gas and by convection. So, what remains is heat loss by evaporation via the expired gas (E_{res}) and perspiration from the skin (E_{skin}). At 2.5 bar E_{res} of the worker becomes 75.2 W to evaporate 104 ml water/hour (calculated according to 1.2.3b). The remaining 470 W should be dissipated by perspiration of 696 ml water/hour ((470/14)A(0.5/24), see the calculation of E_{skin} in Part 1 §1.2.3). The fluid loss by perspiration and evaporation together should amount of 0.800 l/hour. However, compressed air work is mostly done in hot chambers with a very high humidity. This obstructs the process of perspiration. This can be compensated by a substantial sweat production, which does partly evaporate (air current). However, most of the sweat will wet the clothing and will drip off. To avoid an increase of core temperature, the fluid intake is supposed to be at least 1.2 l/hour. During heavy work and heavy running the fluid loss is about 1.6 l/h (own observations and see v.d. Putten, 1996 for reference). Within some hours, a too low fluid intake will result in severe hyperthermia and other serious hazards like dehydration, hypercapnia, cardiovascular stress etc. In a previous study dehydration of caisson workers (work load not reported) was estimated on 0.2 to 1.8 l in 2 hour without supplementary fluid intake (v.d. Putten, 1996). From sports medicine it is well known that long lasting heavy exertion can result in a disturbance of the mineral balance and exhaustion. Therefore, fluid intake must be accompanied by mineral and sucrose intake. A mineral-sucrose fluid with a slight hypertonic excess of NaCl speeds up the intestinal uptake of sucrose and water, since Na⁺ facilitated the sucrose membrane-transport. The uptake of NaCl and sucrose causes a fast osmotic uptake of water by the intestinal epithelial cells.

Conclusion. It can be stated that for the health of compressed air workers (and for a good physical performance) regular and sufficient fluid (with especially NaCl and sucrose) intake is an absolute conditio sine qua non.

2.6. Lung mechanics and hyperbaric pressure

2.6.1 Theory of gas flows in tubes

From the rheology of pipes it is well known that with laminar flow the flow-resistance is dependent on the viscosity of the gas and not on its density. Consequently, it is not dependent on pressure. It is also known from diving practise that at, say, 50 m the work of respiration (and therefore the resistance) is much larger than at the surface. This apparently means that the flow in the airways is not laminar and the question arises how this is possible. When this question is answered the next question is whether for a given gas mixture it is possible to calculate the maximal depth of breathing, such that a minimal but stationary oxygen and Carbon dioxide exchange is possible to maintain a moderate work load.

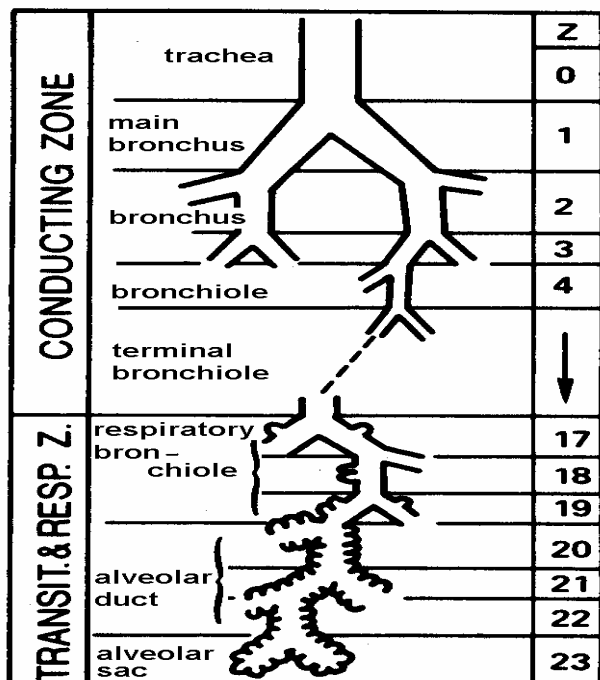


Fig. 2.6 Weibel model of airways with its generations indicated (see below). The function of the conduction zone is limited to transport. In the transition zone transport and gas exchange occur. In the respiratory zone (alveoli) only exchange occurs (Drawn up to generation 23; modified from Weibel and Gil, 1977).

Before discussing lung mechanics and pulmonary gas physiology under pressure, it is helpful to have some knowledge of gas streams in the airways, which can be described as a system of many-fold bifurcating tubes as illustrated in Fig. 2.6.

Streams of gas and liquid in pipes behave on a gliding scale from orderly to chaotic. Orderly behaviour is laminar (top line Fig. 2.7) and chaotic is turbulent (middle line Fig. 2.7). The various types are classified according to the number of Reynolds, Re . For low Reynolds numbers the flow is laminar and for much higher ones the flow is turbulent. A laminar flow has a velocity increasing with the distance from the wall of the pipe. This increase is parabolic with the maximum of the parabola in the centre of the pipe and consequently the velocity is there maximal (Fig. 2.7).

Box 2.2 The Reynolds number is defined as:

$$\text{Re} = v \cdot D \cdot \Delta / \eta, \quad (2.3)$$

with v the mean velocity (m/s), D the diameter of the pipe (m), Δ the density ($\text{kg} \cdot \text{m}^{-3}$) and η the viscosity ($\text{Pa} \cdot \text{s}$). Dependent on the Reynolds number or better five states of flow can be distinguished. When $\text{Re} < 2000$, than the flow is laminar and when $\text{Re} > 10000$ the flow is turbulent. In between are two transitional states. The 5th one occurs at the orifice of a pipe in some cavity. Laminar flow velocity is dependent on the viscosity of the gas, but not on its density.

Consequently, it is pressure independent.

We will start with an attempt to calculate RMV on the basis of the law of Poiseuille:

$$\dot{V}_s = \pi \cdot \Delta p \cdot r^4 / (8 \cdot \eta \cdot L), \quad (2.4a)$$

with \dot{V}_s is the stationary volume flow (m^3/s), Δp (Pa) the pressure difference between the both ends of an ideal tube with radius r and length l , and η the dynamic viscosity of air ($17.1 \times 10^{-6} \text{ Pa} \cdot \text{s}$). The resistance of the tube is given by $\Delta p = \dot{V}_s \cdot R_{\text{tube}}$, analogue to the electric law of Ohm ($V = i \cdot R$). Consequently,

$$R_{\text{tube}} = 8 \cdot \eta \cdot L / (\pi \cdot r^4). \quad (2.4b)$$

The airway system can be described by a bifurcating tree with many levels of bifurcation. The trachea is called the 0th generation, the bifurcation of the trachea is of the 1st order and the two main bronchi are the 1st generation. At generation 17 the first alveoli appear what goes on to the 26th generation. For RMV up to 180 l/min no one of the generation exceeds $\text{Re} = 10000$ at 1 bar. But many generations have intermediate flow, which means a higher resistance than with laminar flow. Before discussing the implications of this phenomenon under pressure, first Poiseuille's law (see also Box 2.2) and other complications will be discussed.

Poiseuille's law The driving force of volume flow² is the pressure difference Δp . For the respiratory system this is the transpulmonal pressure, the difference between the tissue surrounding the lung and the mouth cavity. Generally it is measured in the oesophagus proximal of an intra-oesophageal inflated balloon. This difference for the whole respiratory system varies from 200 Pa = 2 cmH₂O; RVM about 6 l/min) to 800 Pa (high flows) and under special conditions it can go up to 4000 Pa. The pressure difference Δp is distributed over the whole bifurcating network from mouth up to the alveoli. When the flow-velocity v increases, then Δp increases, such that the $\Delta p - v$ relation is linear, as shown in the small top-middle graph of Fig. 2.7. The gas density ρ does not influence the flow. In lung mechanics generally for flow \dot{V} is practised which is the flow \dot{V} per second (in physical calculations $\dot{V} = 2 \cdot \text{RMV}/60$ in l/s and in physiological calculations the factor 2 is often already processed in the equations).

² For simplicity flow is considered as stationary in stead of (by approximation) sinusoidally. This means that the effect of inertia is not considered in this chapter.

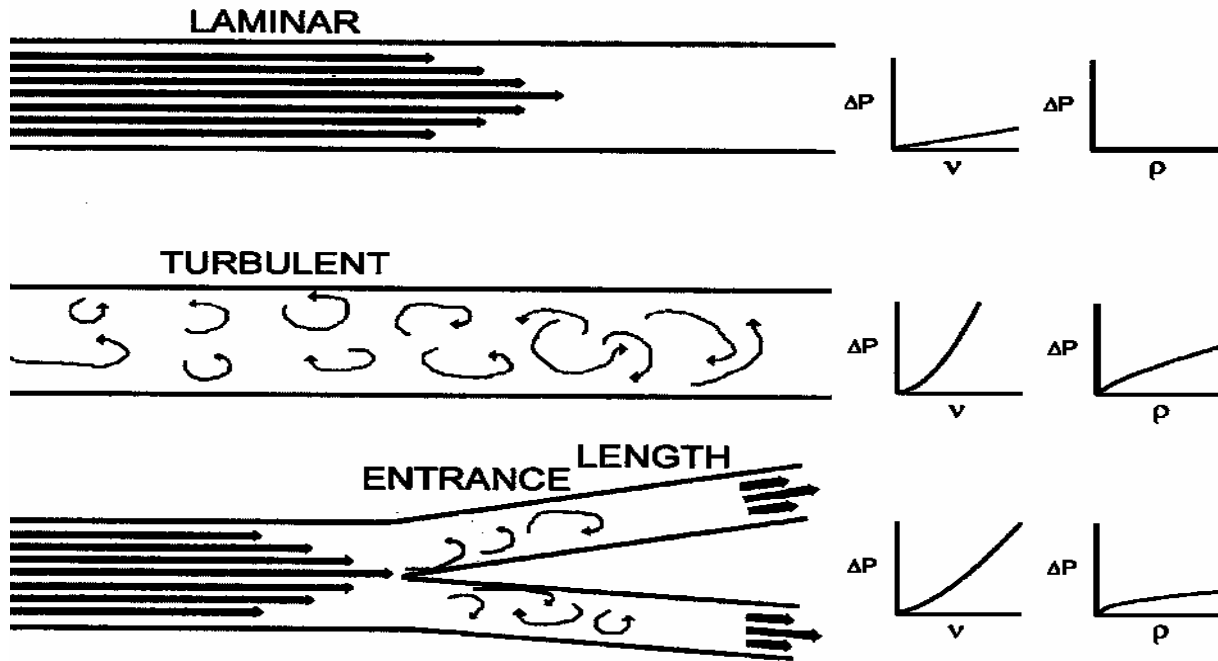


Fig. 2.7 The effect of bifurcation at laminar flow (after Clarke and Flook, 1999). For the same ΔP the laminar flow velocity is most fast and that of the turbulent flow most slow.

For given dimensions of a generation and a given pressure drop along its length, and a supposed laminar flow, Poiseuille's law yields \dot{V} . This law (see last box) applied to the trachea, with a length $L=0.12$ m (the effective length between the distal part of the oesophagus and the first bifurcation at the end of the trachea), $r=8.8 \times 10^{-3}$ m (the effective radius) and an assumed (non realistic) pressure drop of only 1 Pa, a \dot{V}_{tr} of 1.16 l/s (69.8 l/min) is found.

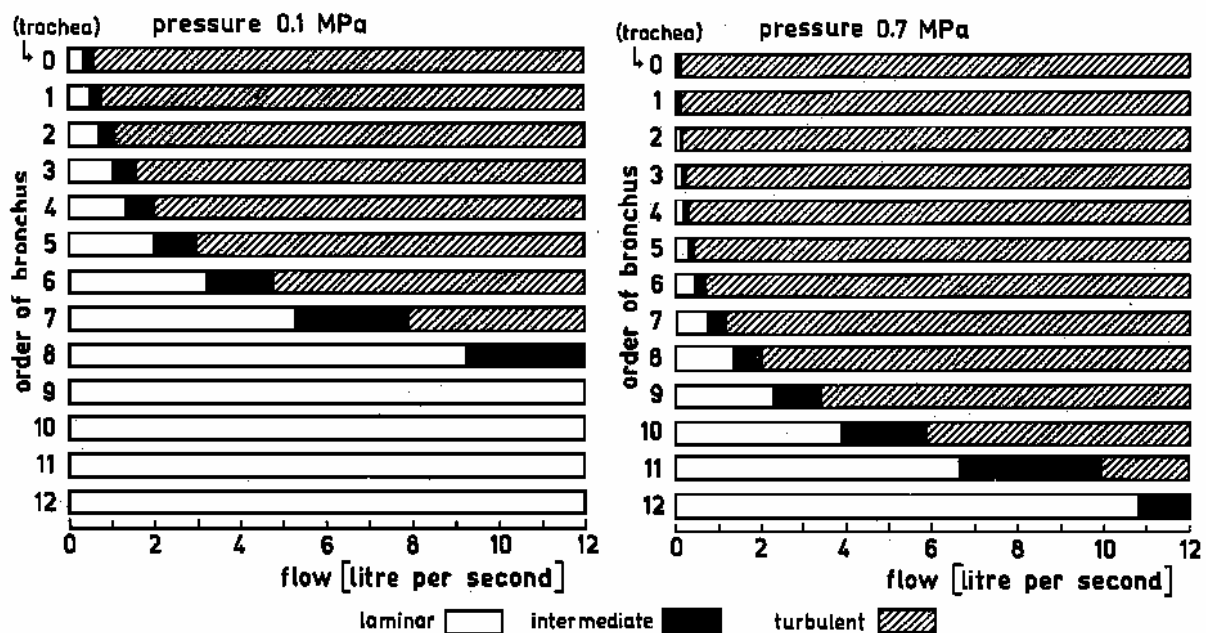


Fig. 2.8 Types of flow in the airway generations at 1 (left) and 7 bar (= 0.7 MPa, right; after Hrnčíř, 1996). In the model, entrance effects are not considered. Their effect can roughly be accounted for by increasing the generation (order) numbers at 1 bar by 10 and at 7 bar by 14 (see Clarke and Flook, 1999).

Physiologically, this flow is certainly too high for such a small pressure drop. The question arises whether this flow has still a laminar profile, which means that $Re < 2000$. It can be calculated that $v = 4.8$ m/s, which results in $Re = 590$. This means that the flow is doubtless laminar. Consequently, there should be other causes why \dot{V}_{tr} is too high. One cause is that for application of Poiseuille's law the wall should be perfectly smooth, which is certainly not the case. Another one is, that the diameter varies a little. However, the most important complication is that the air stream just passed the constriction of the glottis, which causes a very substantial pressure loss and non-laminarity. The non-laminar behaviour implies that $\Delta p \propto \dot{V}^n$ with $1 < n < 2$. In conclusion, even for moderate flows ($\dot{V} = 0.5$ l/s) Poiseuille's law is not valid.

With turbulent flow, a higher Δp is needed to obtain the same velocity (graph in middle column, middle line Fig. 2.7). Now, there is also density dependency: a lower density (e.g. He) implies a lower Δp (right graph, middle line Fig. 2.7).

Also at the bifurcations, the laminar profile is disturbed (bottom line Fig. 2.7) and the Δp - v relation is non-linear (be it less deviating from linear than with turbulent flow), and the same holds for the Δp - ρ relation. At a bifurcation, a laminar flow becomes non-laminar, but after some length in the daughter branch the flow regains a laminar profile. This distance, called the entrance length is linear dependent on the diameter of the daughter branch and its Reynolds number. Subsequent orders of bifurcations are so close together that for the lower generations the current fails to be laminar in a generation before the next bifurcation is reached. However, from a certain, critical generation laminar flow is realised. For $\frac{1}{4}$ l/s this is at generation 16 (see Clarke and Flook, 1999). At the same time, (very) high flows are not laminar, dependent on the Reynolds number. Both effect, the entrance phenomenon and flow-profile dependency on Re results in a critical generation number that is dependent on the flow. Of course, this critical generation has a higher number the higher the flow.

In the first halve of last century it was generally accepted among physiologists that the airways have a mixed laminar and turbulent behaviour, yielding the equation:

$$\Delta p = K_1 \cdot \dot{V} + K_2 \cdot \dot{V}^2, \quad (2.5a)$$

with the laminar term linear with \dot{V} and turbulent term quadratic (Milic-Emili and D'Angelo, 1997). The factor K_1 presents the flow-resistance of the airways, which comprises the viscosity of the gas, and the dimensions of the airways and K_2 accounts for the extra resistance due to irregularities of the walls of bronchi etc., and constrictions and bifurcations. The constants hold for mouth breathing, which are lower than breathing via the nose. The behaviour of the last compartment of the respiratory tree, constituted by the alveoli, is not yet incorporated. The epithelium of the alveoli is covered by a thin film of water. Since the alveoli are so small, cohesive forces in this water film (due to the surface tension according to Pascal's law) would collapse them. Fortunately, this force is strongly reduced by the epithelial surfactant. However, when by inspiration the vesicles are inflated, the force to collapse increases. In addition to this, the tissue of alveoli and bronchioli also contribute in the elastic E of the system. Of course, this elastic term should also be incorporated in the equation. The final, semi-theoretical approximation is given by the equation:

$$\Delta p = E \cdot V + K_1 \cdot \dot{V} + K_2 \cdot \dot{V}^2, \quad (2.5b)$$

with V the inspired volume. An obvious drawback is that (2.5) does not account for intermediate flow which is described by the relation $\Delta p \propto \dot{V}^n$ with $1 < n < 2$. With known values of the laminar resistive constant K_1 ($=1.4 \text{ cmH}_2\text{O} \cdot \text{l}^{-1} \cdot \text{s}^1$), the turbulent resistive constant K_2 ($=$

$0.3 \text{ cmH}_2\text{O}\cdot\text{l}^{-2}\cdot\text{s}^2$; as K_1 from Milic-Emili and D'Angelo, 1997), V ($=0.7 \text{ l}$) and \dot{V} ($=0.093 \text{ l/s}$) of our standard subject, and the static elasticity E of bronchioli and alveoli, the pressure difference from mouth to alveoli Δp can be calculated. Various values of E can be found in literature (Gillissen et al., 1990, $4 \text{ cmH}_2\text{O}\cdot\text{l}^{-1}\cdot\text{s}^{-1}$; Jonson et al., 1993; $16.7 \text{ cmH}_2\text{O}\cdot\text{l}^{-1}\cdot\text{s}^{-1}$). With $E = 4 \text{ cmH}_2\text{O}\cdot\text{l}^{-1}\cdot\text{s}^{-1}$, Δp appears to be equal $2.9 \text{ cmH}_2\text{O}$, a realistic value. However with a higher value of E the result is not realistic. This makes (2.4b) very sensitive for the value of E and inappropriate for hyperbaric application.

2.6.2 Lung mechanics with hyperbaric pressures

Laminar flow is dependent on the dynamic gas viscosity η , (which is pressure independent (e.g. Kronig 1966) and not on the gas density ρ , which is linear proportional with pressure p . However the turbulent resistance is proportional with $\rho^{0.75}$ (Clarke and Flook, 1999). At extreme depth the respiratory system has to cope in first place with CO_2 retention (see § 2.4) and in the second place with hypoxia due to the low efficacy of the gas flow. To maintain gas exchange, it is supposed that RVM should be 14 l/min^3 f 0.1 Hz and p the high value of $8 \text{ cmH}_2\text{O}$. To calculate the maximal density of the breathing gas the right hand term of (2.5b) is multiplied by $(\rho_{1 \text{ bar}}\cdot p)^{0.75}$ (p in bar; $\rho = \rho_{1 \text{ bar}}\cdot p$), the only unknown parameter in (2.5b). The solutions for p is 56 bar , equivalent with a Trimix composition of He, 2% O_2 and 2% N_2 this is equivalent to 318 bar , rather unrealistic values. The problem with (2.4b) is that K_1 and K_2 themselves are ρ dependent. Therefore, another approach was attempted. When $p > 7 \text{ bar}$, all generations have turbulent flow because of cumulative entrance effects with intermediate flow. Then the tube resistance R_{tube} is (Clarke and Flook, 1999):

$$R_{\text{tube}} = 2^{5/2} \cdot \pi^{-7/4} \cdot L \cdot D^{-15/4} \cdot \mu^{1/4} \cdot \dot{V}^{3/4} \cdot (\rho_{1 \text{ bar}}\cdot p)^{3/4} \quad (2.6)$$

with L and D (diameter) in cm, μ the dynamic viscosity (in Poise), \dot{V} in l/s and ρ in g/l. In all generations there is some pressure drop Δp_{gn} . For the higher generations Poiseuille' law practically holds for $p = 1 \text{ bar}$ and low flows. With high pressures, R_{tube} of the higher orders change from a Poiseuille resistance to resistance according to (2.6). This is a substantial increase. With equation 2.4b of Box 2.2 it can be calculated that the increase is 1000-10000 times. For a given \dot{V} , the R_{tube} of all generations and so of the whole system can be calculated with p as the only unknown parameter. For $\text{RMV} = 14 \text{ l/min}$, R is $0.28 \text{ cm H}_2\text{O}\cdot\text{l}^{-1}\cdot\text{s}$ times $p^{0.75}$ (dimensionless). By comparing this result (which is in accordance with literature) with experimental data, it appears that this calculated R is about 2.5 times lower than actually measured R 's. The reason is not clear, since it is not likely that the non-implied boundary layer effects and smoothness factor can account for the factor 2.5. For the airways (without alveoli) with constant flows (and effects of compliance and inertia ignored), analogue to the electric law of Ohm ($V = i \cdot R$), it holds that:

$$\Delta p = R \cdot \dot{V} \quad (2.7a)$$

At pressure p holds that $\Delta p = p^{3/4} \cdot R_{\text{airways},1 \text{ bar}} \cdot \dot{V}$. Defining a new R_{airways} ' without the \dot{V} dependency, (2.7) becomes $\Delta p = R_{\text{airways}} \cdot \dot{V}^{1.75}$. Using R is $0.28 \text{ cm H}_2\text{O}\cdot\text{l}^{-1}\cdot\text{s}$ and the experimentally found factor of discrepancy, arbitrarily assumed to be 5 (to restrict the maximal p to be calculated), the final equation is:

$$\Delta p = 1.95 \cdot p^{3/4} \cdot \dot{V}^{7/4} \quad (2.7b)$$

³ 14 l/min produces a metabolic power of about 200 W , which means that effectively about $120/4=30 \text{ W}$ is available for mechanical work. This is enough for slowly swimming with a Scuba.

For a given \dot{V} (RMV is 14 l/min, so $\dot{V} = 2 \cdot 14/60$ and $\dot{V}_{in} = 0.5 \dot{V}$) and $\Delta p = 8 \text{ cmH}_2\text{O}$, p can be calculated with equation 2.7b. This results in $p = 38.5 \text{ bar}$, a rather high value, equivalent to 218 bar Trimix. From experiments with mice it is known that at 2000 bar (with Trimix) that the animals were able to breath, despite suffering from HPNS. Further, the calculated pressures lack theoretical and experimental support. Therefore, it was tried to give some validation from the results of other approaches.

Experimentally, it has been found that at peak ventilatory performance:

$$\text{RMV}_{p,\text{max}} = \text{RMV}_{1 \text{ bar,max}} \cdot (\rho_{1 \text{ bar}} \cdot p)^{-0.45}, \quad (2.8)$$

with $\text{RMV}_{1 \text{ bar,max}}$ the maximal RMV at 1 bar (Hesser et al, 1989). In literature, values of $\text{RVM}_{1 \text{ bar,max}}$ of 180-300 l/min are found. For a pressure of 38.5 bar, $\text{RMV}_{p,\text{max}}$ is between 35 and 58, and consequently more than twice 14 l/min. Therefore the RVM of 14 l/min is well feasible.

Another approach is to see whether the work of breathing under the calculated extreme pressure can be supplied by the system. Therefore, first respiratory work under normal conditions has to be calculated.

2.6.3 Cost of hyperbaric breathing

The work of breathing with strict laminar conditions is $P_{\text{res}} = \Delta p \cdot \dot{V}$. For the airways system, the classical approach is to substitute Δp by $E \cdot V + K_1 \cdot \dot{V}_{in} + K_2 \cdot \dot{V}_{in}^2$ (equation 2.5b), and doubling the cost due to the expiration. With a low tidal volume V , the “elastic energy” stored during inspiration in the alveoli is used during the expiration. So, the term with the E in (2.5b) is positive during inspiration and negative during expiration. After some more calculation P_{res} becomes (see Milic-Emili and D’Angelo, 1997):

$$P_{\text{res}} = 4.8K_1 \cdot \dot{V}_{in}^2 + 9.6K_2 \cdot \dot{V}_{in}^3 \quad (\text{W}) \quad (2.9)$$

With \dot{V}_{in} is 0.23 l/s (14 l/min) P_{res} appears to be only 0.37 W. Adding the work to overcome chest wall resistance ($R_{\text{wall}} = 0.4 \text{ cmH}_2\text{O} \cdot \text{l}^{-1} \cdot \text{s}^1$; Milic-Emili and D’Angelo, 1997), P_{res} is about 0.57 W. When K_2 shows a pressure dependency with a factor of $p^{0.75}$, breathing cost at 38.5 bar including the 0.13 W of the chest wall becomes 1.1 W. This is very unlikely and therefore an approach via (2.9) is rejected.

Breathing cost can also be calculated with a more theoretically based model which also implies the tissues surrounding the lung (Tammeling and Quanjer, 1978). When a volume, e.g. a spirometer or a plastic back is filled normobaric and resistance-free with air, the work is $E = p \cdot V$ (in Joule). E has a viscosity part of E_{vis} , given by the viscosity of the gas, and an elastic part E_{el} . For the respiratory system, there is a part of the thoracal wall E_{W} and the airways system, E_{L} . Total work E for the inspiration together then becomes:

$$E_{\text{ins}} = E_{\text{W,vis}} + E_{\text{W,el}} + E_{\text{L,vis}} + E_{\text{L,el}} \quad (2.10)$$

The instantaneous tidal filling of the lung is $V(t)$, which is a function of Δp , as illustrated in of Fig. 2.9. The dynamic elasticity of the lung is expressed as compliance C , the reciprocal of elasticity. The compliance is defined as $C_{\text{L,dyn}} = V_{\text{in}}/\Delta p$ and:

$$E_{\text{W,el}} = \frac{1}{2} \cdot V_{\text{in}} \cdot \Delta p = \frac{1}{2} \cdot C_{\text{L,dyn}} \Delta p^2 \quad (2.8)$$

In rest, Δp amounts 0.4 kPa and the tidal volume V about 0.6 l. For 0.4 and 0.6 respectively and a respiratory frequency $f = 0.17$ Hz $P_{L,el}$ is 0.2 W. Since V_{exp} is about 4% higher than V_{ins} (depending on the RQ and assuming the same temperature), there is some net (but negligible) effect between inspiration and expiration. $E_{W,el}$ is regained when the expiration takes place. The same holds for $E_{L,el}$, which mainly comprises the compliance of the alveoli. What remains are both viscous parts. At 1 bar $E_{W,vis}/E_{L,vis}$ is about 2 (which is not consistent with Milic-Emili and D'Angelo, 1997). $E_{L,vis}$ is the area of the hysteresis loop of Fig. 2.9. $E_{L,vis}$ can be approximated when the $V(t)$ - p_{tp} relation is elliptic:

$$E_{L,vis} = \frac{1}{4} \cdot \pi \cdot k \cdot p \cdot (V_{in}^2 + \Delta p^2)^{0.5} \quad (2.11)$$

with constant k the dimension-less short-axis/ Δp ratio (the square root has the dimension of litre). Constant k is 0.6 in rest and for low to moderate frequencies and V values.

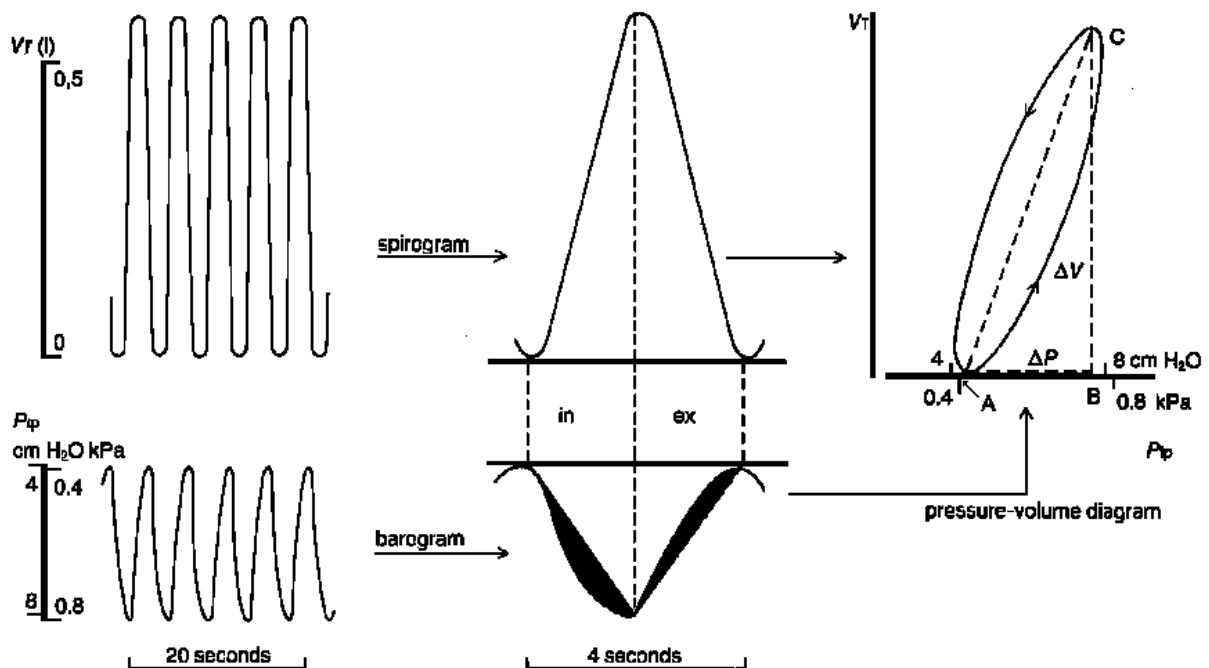


Fig. 2.9 V-time and p-time registration of normal breathing cycle (left and middle), and p-V diagram (right).

Extrapolating data of Clarke and Flook (1997; their Fig. 19) the maximal \dot{V}_{exp} (and so \dot{V}_{in}) is about 1.0 l/s at 38.5 bar. This justifies our choice of $V_{in}=1.0$ litre with $f = 0.23$ Hz. Then, for $\Delta p=0.4$ (kPa), $P_{L,vis}$ is 0.193 W. For the expiration the same amount of power is dissipated, so in total the loss in the airways is 0.386 W. Including $P_{W,vis}$ total cost P_{res} is about 1.2 W, three times more than the value calculate with equation 2.9. For very large flows the cost of the $P_{L,el}$ of inspiration and that of expiration don't longer cancel; there is a net loss which increases progressively with V and the same holds for $P_{W,el}$. Moreover, other costs to overcome other "resistances" in the chest wall (viscoelastic and plasto-elastic) become relevant, which extent the number of terms in equation (2.7).

The behaviour of equation (2.11) under pressure is not easy to derive. Equation (2.11) assumes an approximately elliptic $\Delta V/\Delta p$. Also the dependency of k on p is not known. Therefore, unfortunately this approach does not result in a validation of the maximal pressure.

An approach via (2.7b) seems more realistic:

$$P_{\text{res}} = \Delta p \cdot \dot{V} \cdot p^{0.75} \text{ (W)}, \quad (2.12)$$

with Δp in Pa/m², \dot{V} in m³/s and p dimensionless and numerically in bar. Now, a P_{res} of 5.77 W is found. Is this a reasonable physiological value? Work of respiration under pressure has been measured experimentally for short lasting peak-exercise, and maximally forced breathing (Hesser et al., 1981). Breathing work increases with about $p^{0.18}$ and for short lasting maximally forced breathing with $p^{0.23}$ (values calculated from data of Hesser et al., 1981). Supposing that the maximal work of breathing which can be maintained for a few hours is proportional to $0.5 \cdot p^{0.2}$ times P_{res} at 1 bar, obtained from short lasting peak-exercise and maximally forced breathing (i.e. 18 W). The result is 4.33 W, which means that the approach of (2.10) is slightly high but the best at present available.

Deep diving generally implies that the temperature of the breathing mixture is lower than with shallow depths. This results in a higher airway resistance (see Flynn, 1997) and so reduces the maximal attainable depth.

Conclusion Considering the results of this paragraph it seems save to conclude that the maximal attainable pressure is some 30 bar. Taking this value the maximal attainable pressure p_{max} can be calculated from (2.7b) for any \dot{V} and any mixture with known $\rho_{1 \text{ bar}} \cdot p$ at $T=310 \text{ K}$:

$$p_{\text{max}} = 0.37 \cdot \Delta p^{4/3} \cdot \dot{V}^{-7/3} \cdot \rho_{1 \text{ bar}}^{-4/3}. \quad (2.13)$$

Evaluating this result in relation to the HPNS technique it becomes clear that not the hyperbaric lung mechanics but HPNS limits the maximal attainable depth. The critical volume barrier in Fig. 2.5 indicates that a Heliox mixture can be used up to about 700 bar, whereas the lung mechanics allows 417 bar. With a 75% He, 25% N₂ mixture with 1 bar O₂, volume compression is just above the critical volume compression and the attainable pressure is 123 bar. By replacing He by H₂ the pressure can be maximized to 190 bar (20% N₂) with a tissue compression well above the critical level.

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Abbreviations, constants and variables (preferably mKsA system)

A	area (m ²)
atm	1 atmosphere=101325 Pa
β	compressibility
C	compliance
C _v	amount of dissolved gas per volume of liquid
(C _p /C _v ratio (ratio of heat capacity with constant p and constant V)
D	diffusion constant, diameter
E	energy (J)
E	static elasticity
0	dynamic viscosity coefficient (Pa.s)
FE	specific fraction of expired air
FI	specific fraction of inspired air
H	height (m)
)H	specific evaporation heat (kJ/kg)
k,K	constant
K	Kelvin; 273.15 K / 0 °C
l,L	length (m)
l _n	1 liter at 1 bar and 0 °C
m	molecular mass
m	mass (kg)
μ	dynamic viscosity coefficient (Poise)
Mw	molecular weight (kg)
n	N/m ³
n	number of kmoles
p	pressure (bar, ATA or Pa)
P	power (W)
P	poise, a measure of viscosity (=0.1 Pa.s)
Pa	Pascal/N/m ² = 1 ATA / 1 bar /10 ⁵ Pa
r	radius (cm, m)
R	gas constant
R	resistance
R	Heat transport by radiation (w)

RMV	respiratory minute volume
Δ	specific density at 0 °C (kg/m ³)
q	Bunsen's absorption coefficient (l _n /l, g/l)
t	temperature in °C
T	temperature in Kelvin
v	velocity (m/s)
V	volume (m ³ or l)
\dot{V}	volume flow (l/s)
W	weight (kg)
x	the partial fraction solubility at 1 bar.