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DD I e DD II



Journal of  
**Applied Physiology**<sup>®</sup>

Robert Boyle's landmark book of 1660 with the first experiments on rarified air

**John B. West**

Journal of Applied Physiology Published 1 January 2005

**Vol. 98 no. 31-39 DOI: 10.1152/jappphysiol.00759.2004**

NEW 1846  
EXPERIMENTS

*Physico-Mechanicall,*

Touching

The SPRING of the AIR,  
and its EFFECTS,

(Made, for the most part, in a New  
PNEUMATICAL ENGINE)

Written by way of LETTER

To the Right Honorable Charles,  
Lord Vicount of Dungarvan,  
Eldest Son to the EARL of CORKE.

By the Honorable Robert Boyle Esq;



August OXFORD:

Printed by H: Hall, Printer to the University,  
for Tho: Robinson. 1660.

2-67

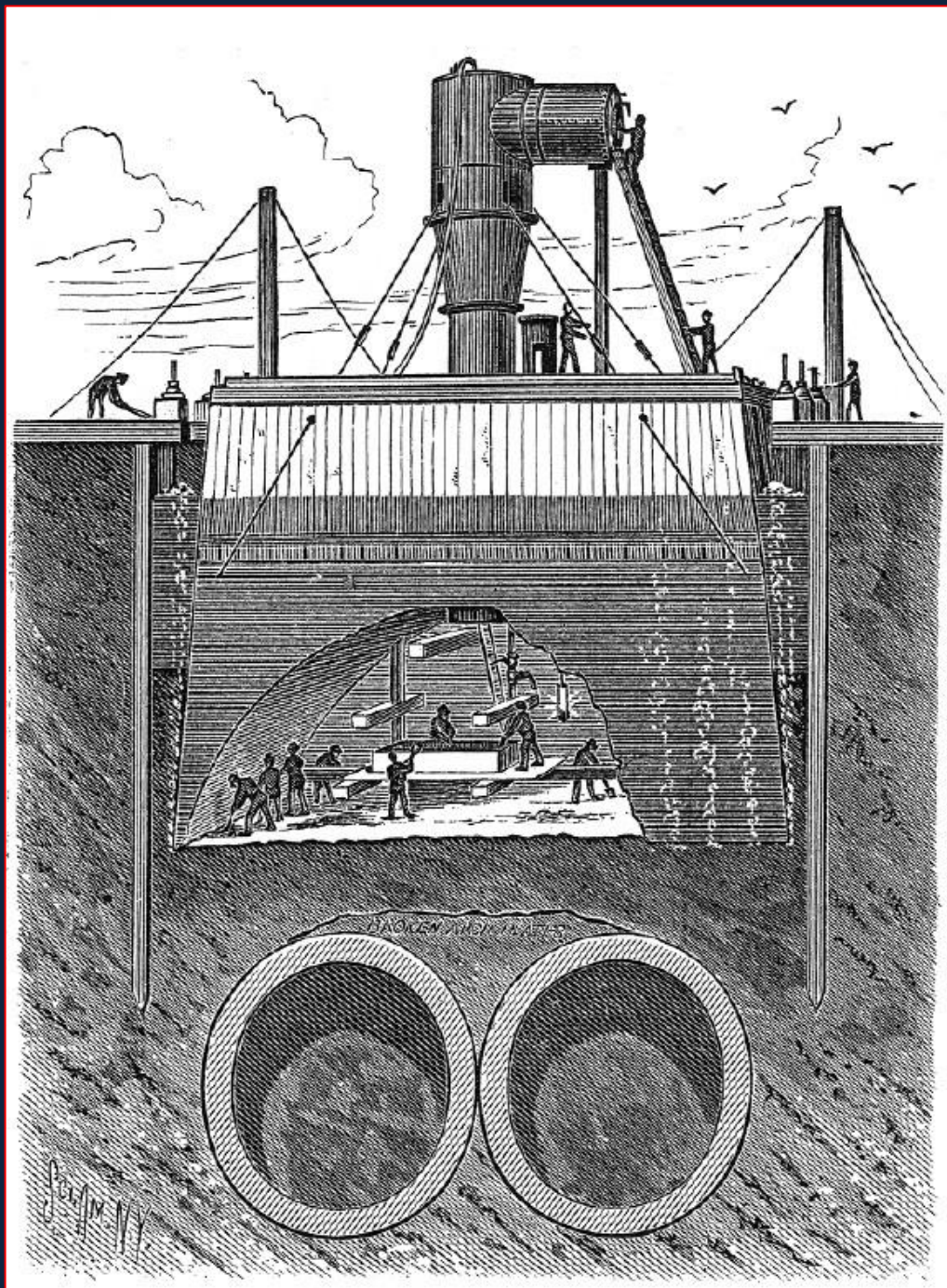




1867-1874: 352 casos / 13 óbitos

# TUBULÃO PNEUMÁTICO





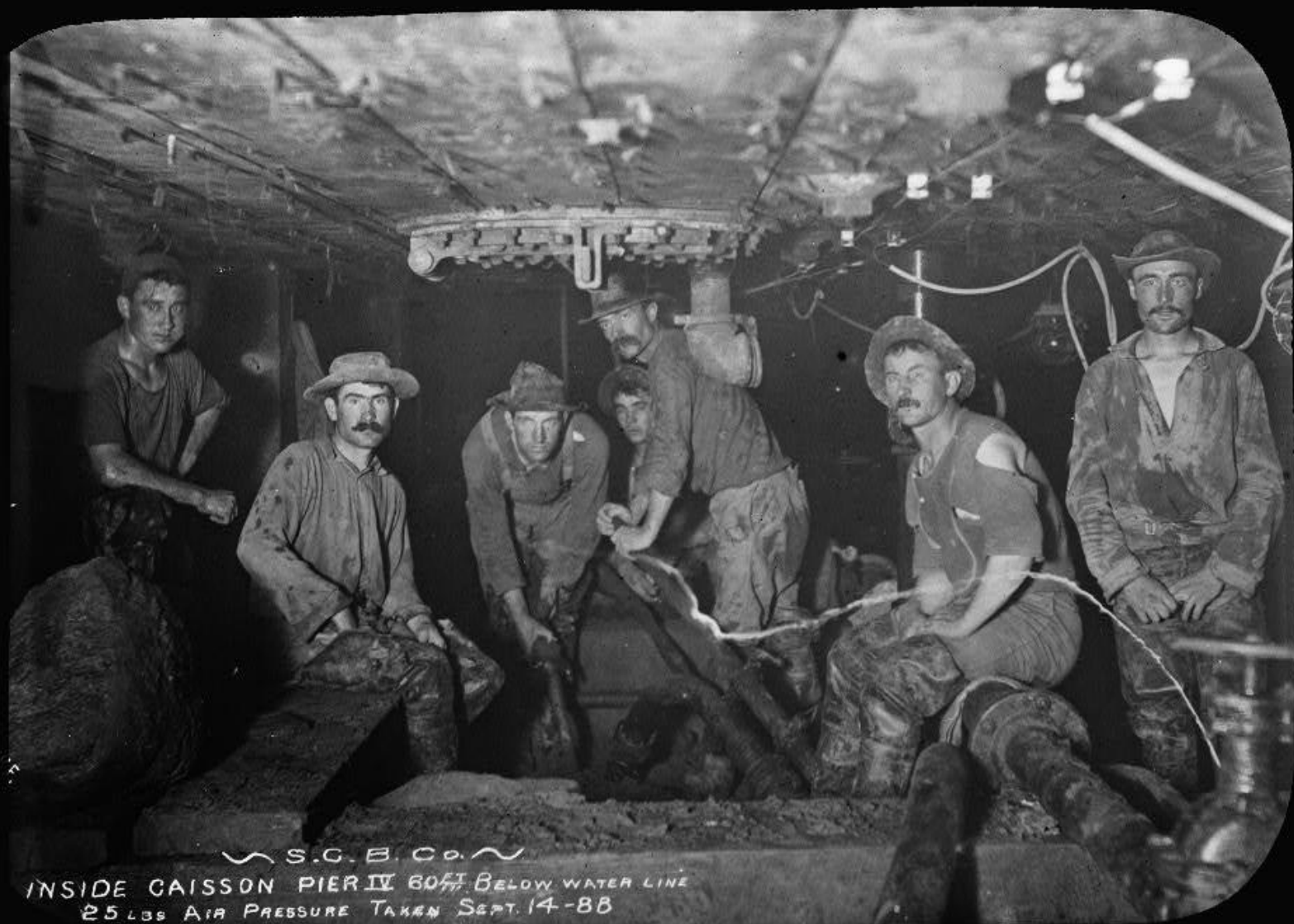


HAER No. 14-2-24

HAER No. 14-4-4

20'87

HAER No. ~~NS-2-4~~  
SD-1-4



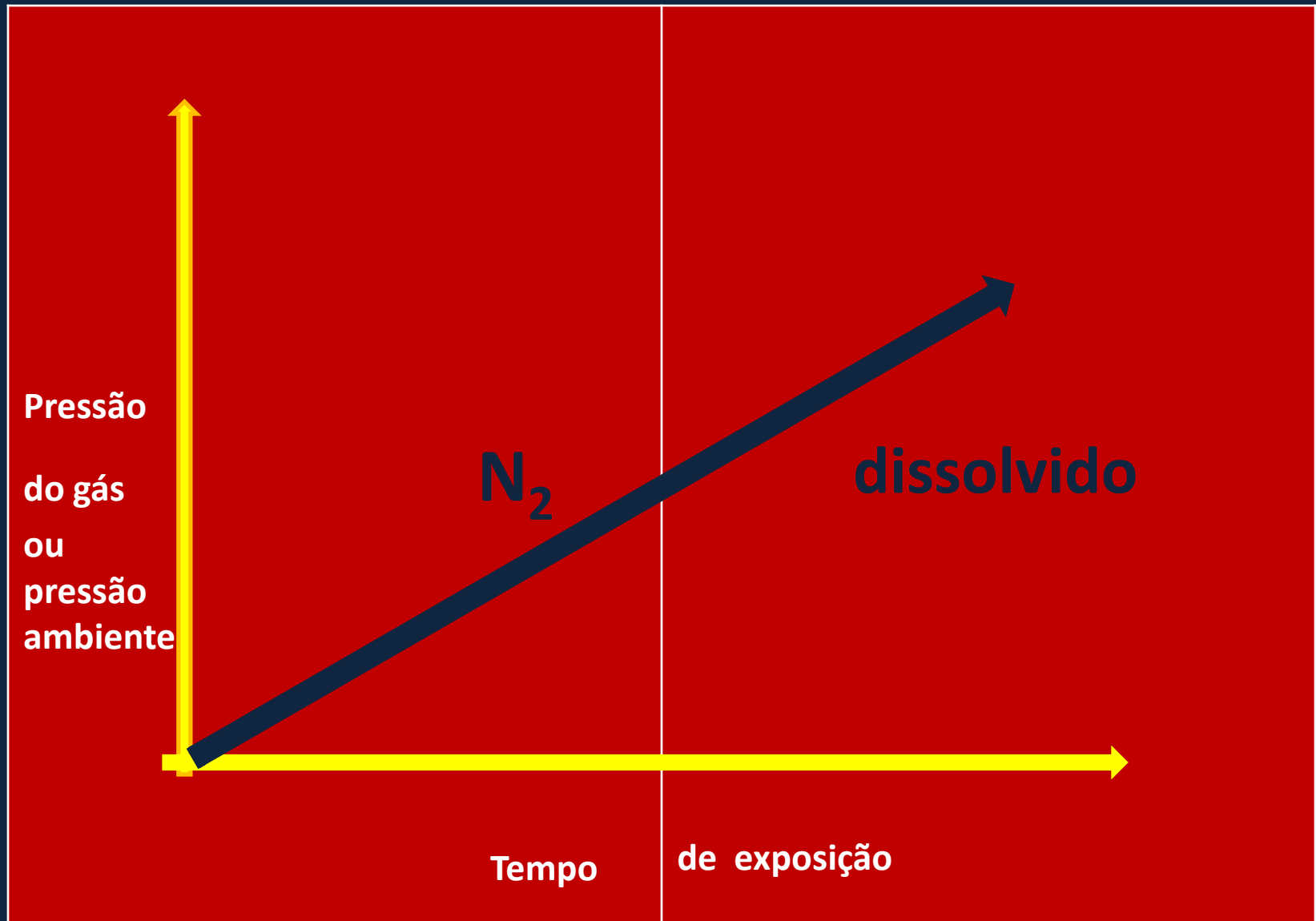
~ S. C. B. Co. ~  
INSIDE CAISSON PIER IV 60 FT. BELOW WATER LINE  
25 LBS AIR PRESSURE TAKEN SEPT. 14-88

# *Lei de Henry*

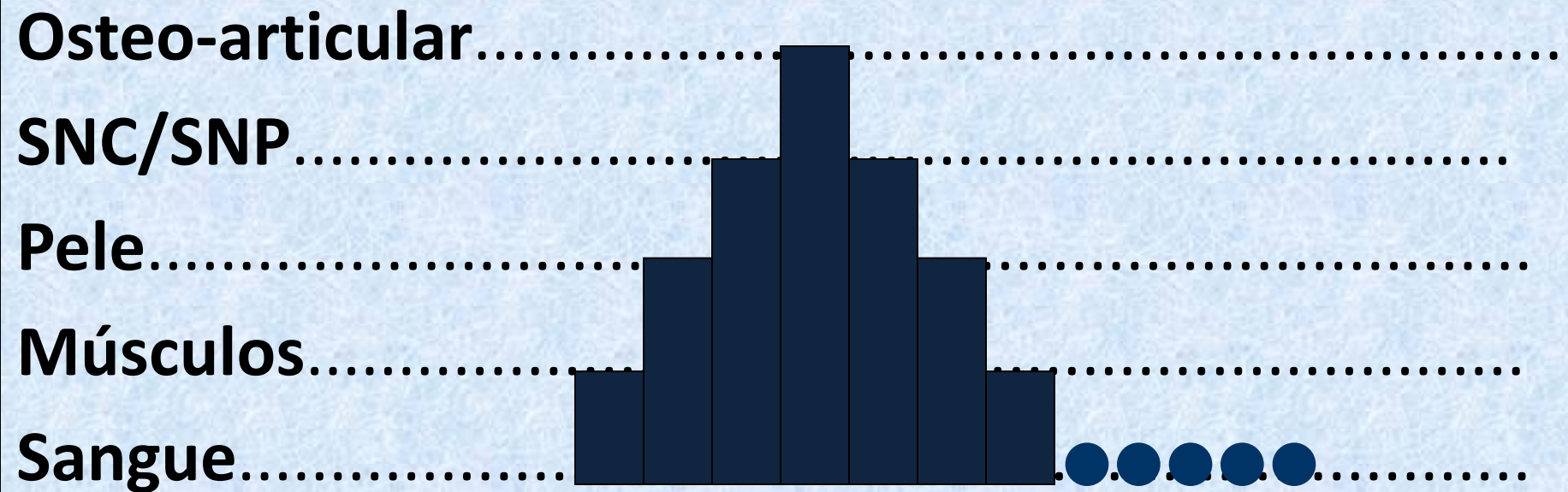
**“A quantidade de um gás que se dissolve em um meio líquido é diretamente proporcional à pressão exercida por este gás sobre este líquido”**

***“Quanto mais fundo ou mais longo o mergulho, mais gás ( $N_2$ ) se dissolverá no sangue e os tecidos”***

## Lei de Henry e a dissolução de gás nos tecidos



# Progressão de absorção/eliminação de $N_2$



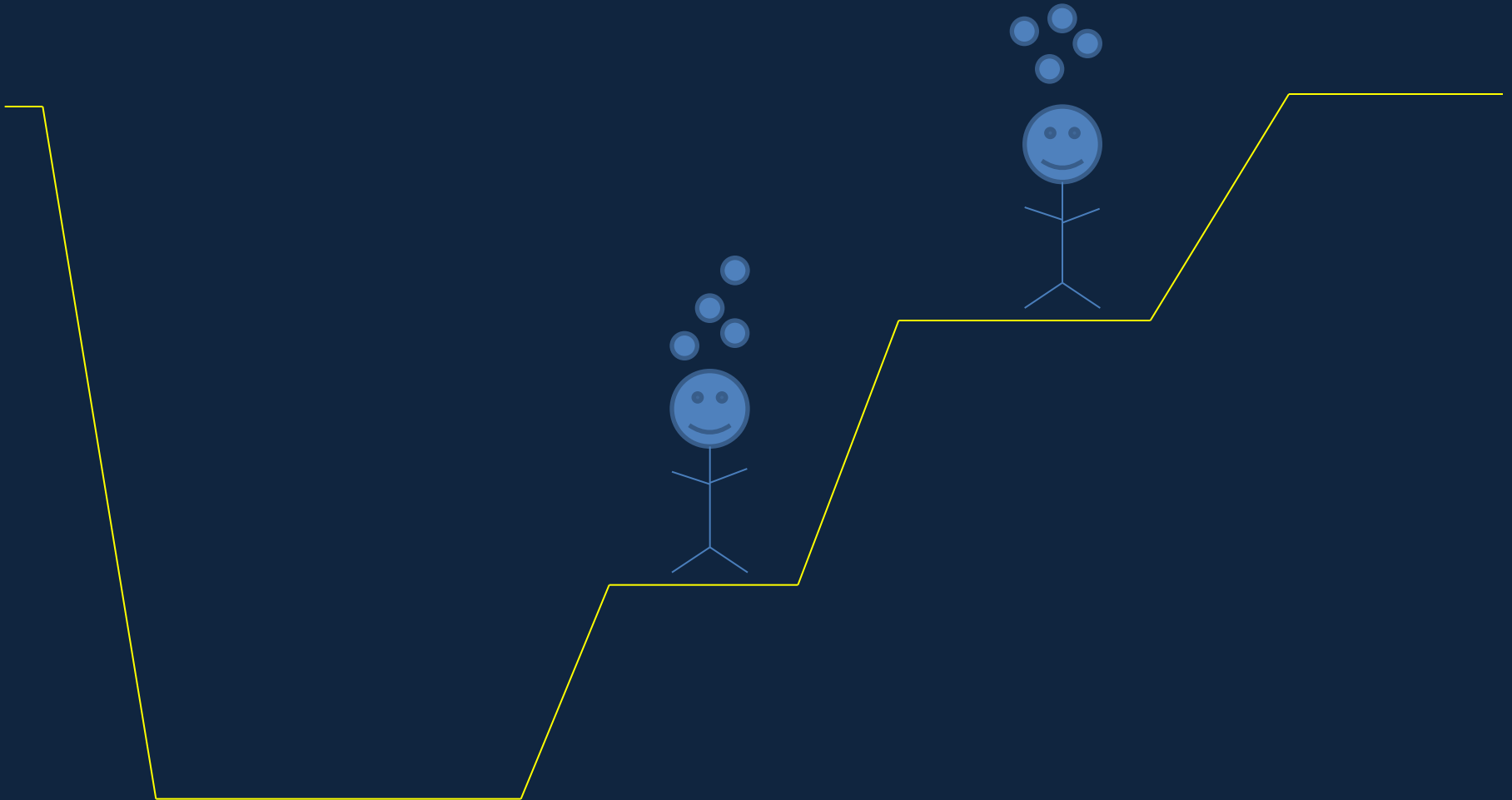
$N_2$  tissular: função de Pp X tempo X fluxo

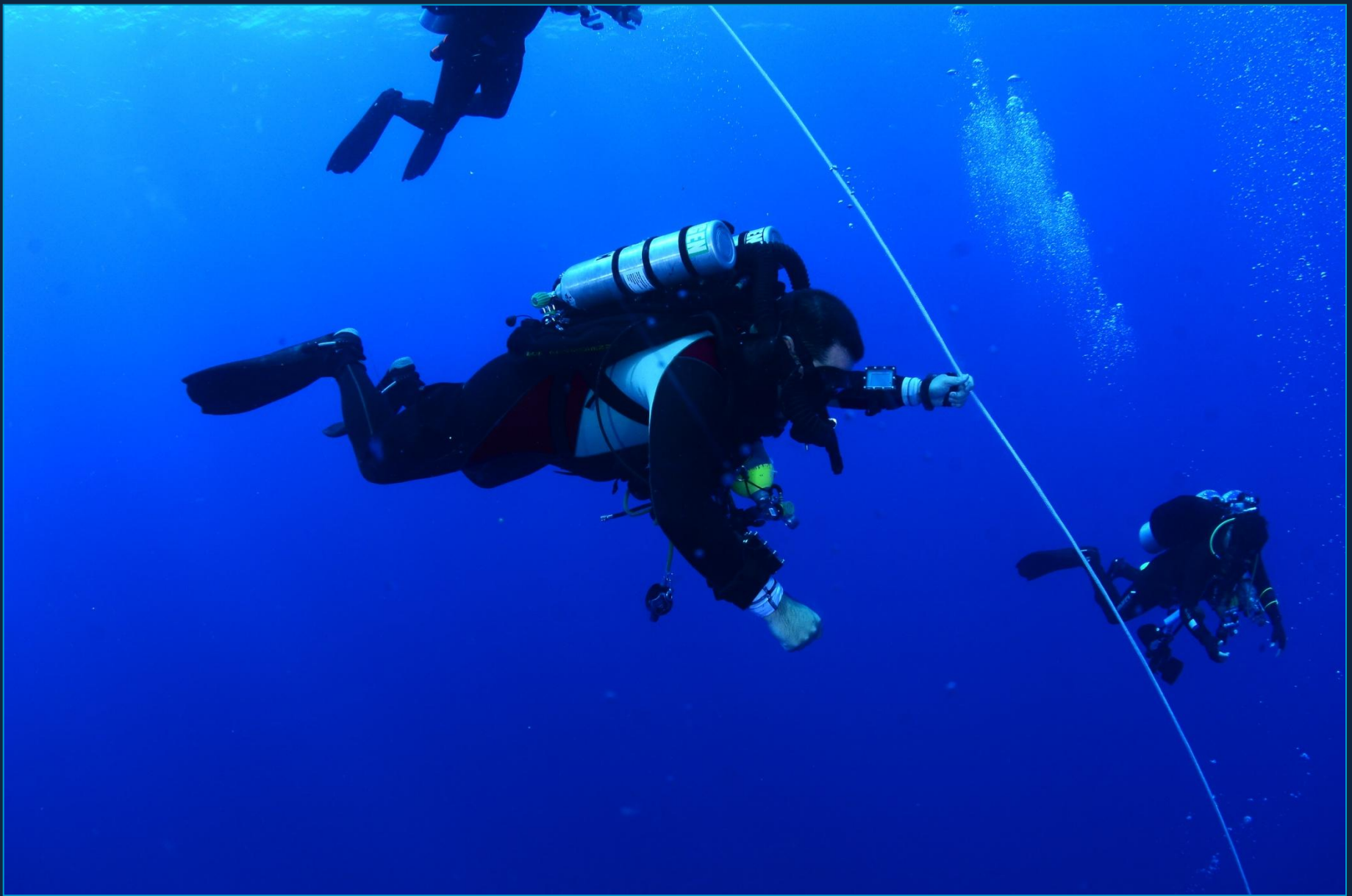
Obs.: (1)  $N_2$  residual e *IS* c/s  $O_2$

(2) Altitude e Vôo

# Tabela de Descompressão

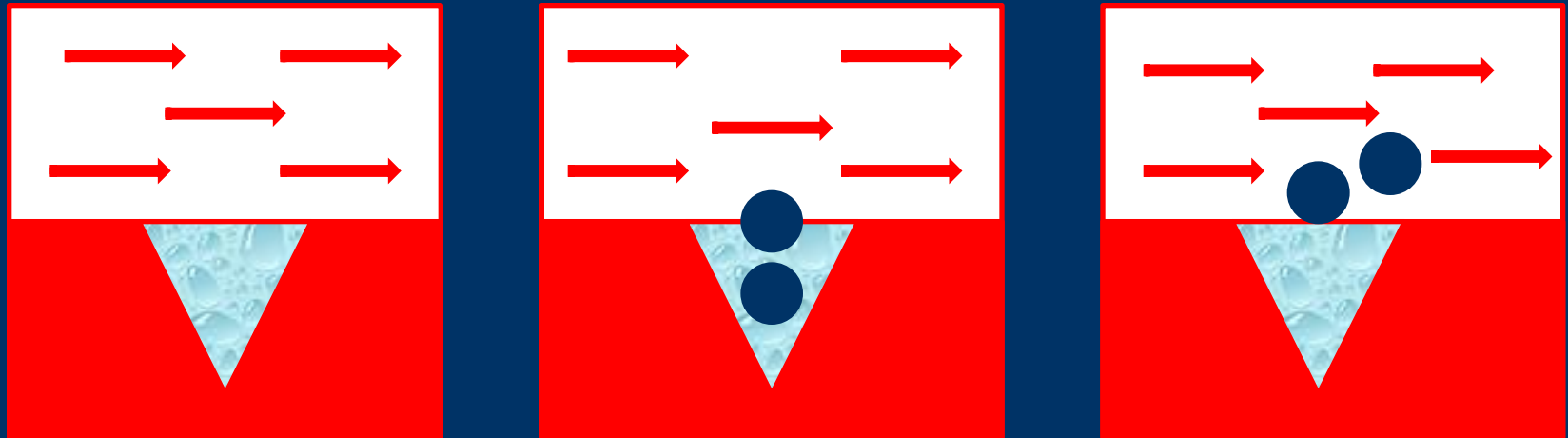
## Eliminação do Nitrogênio Dissolvido





$$P = 42 \quad T = 40 \quad D: V = 1,50' \text{ e } 2(9) - 16(6) - 26(3) = 46'20''$$

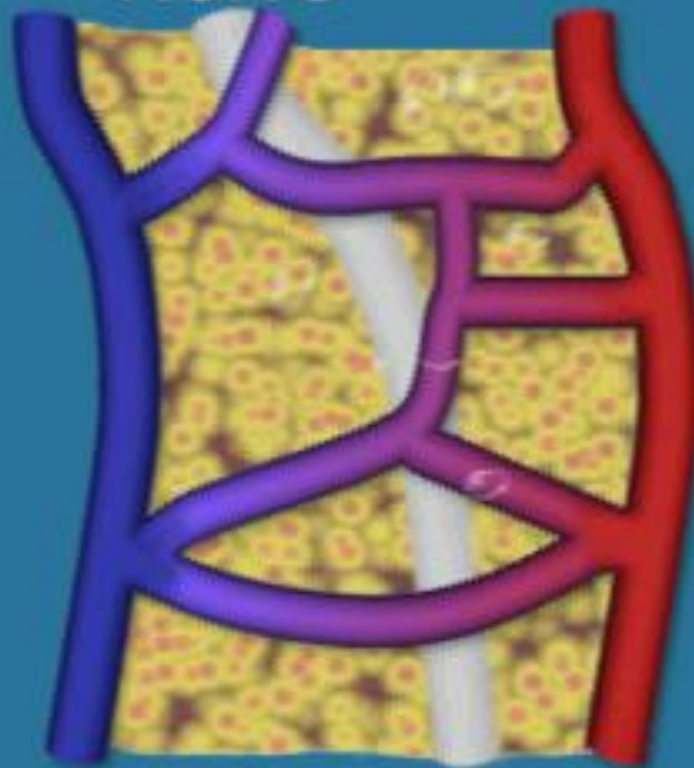
# O processo descompressivo



Formação do núcleo gasoso e embolização

(Obs.: temperatura da água)

Nerve



DD formação 1

$O_2$   
Deficient



Reação Antígeno-Anticorpo + Mediadores Inflamatórios + Lesão Endotelial  
(Citocinas / Leucotrienos / Tromboxane A )

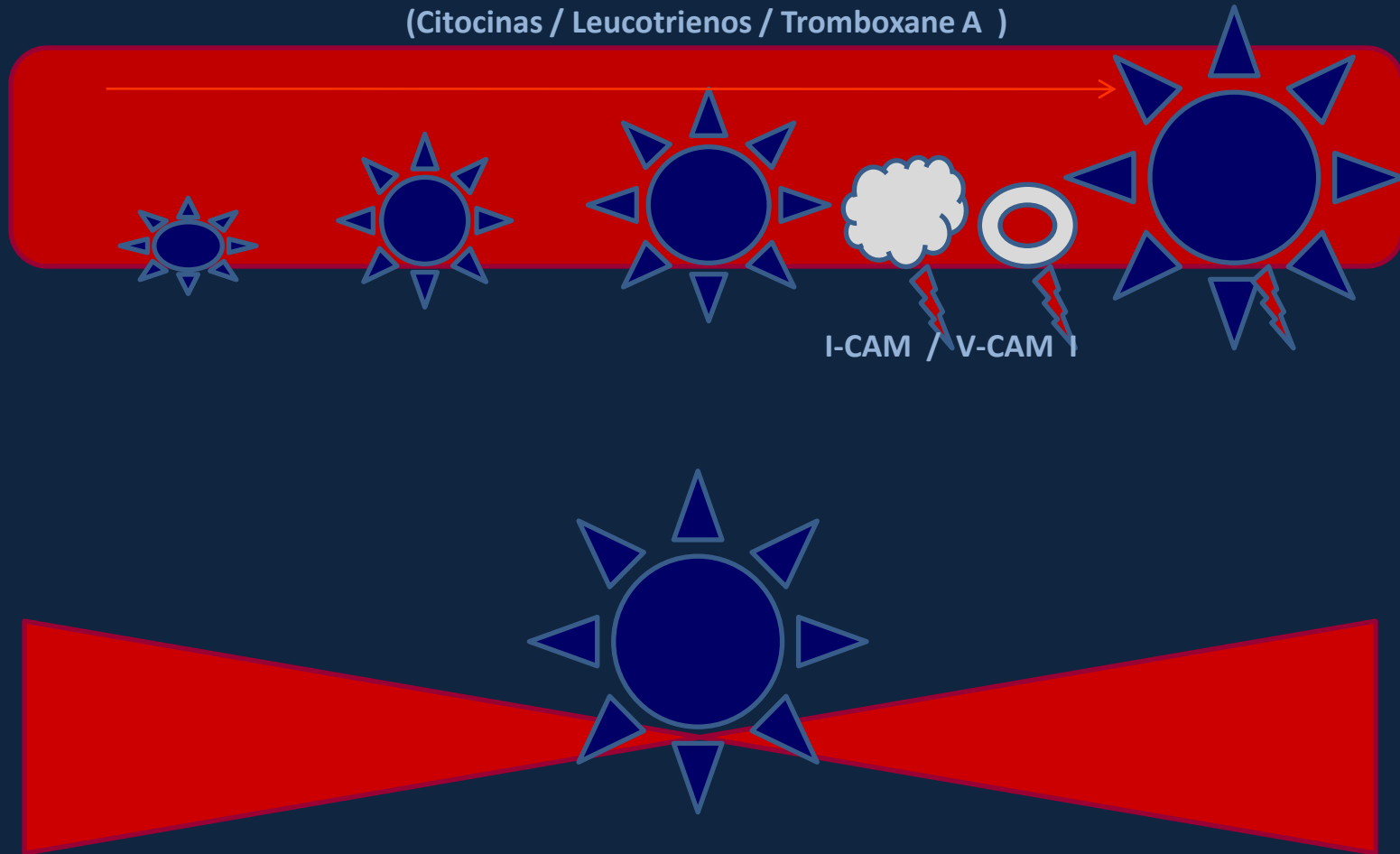


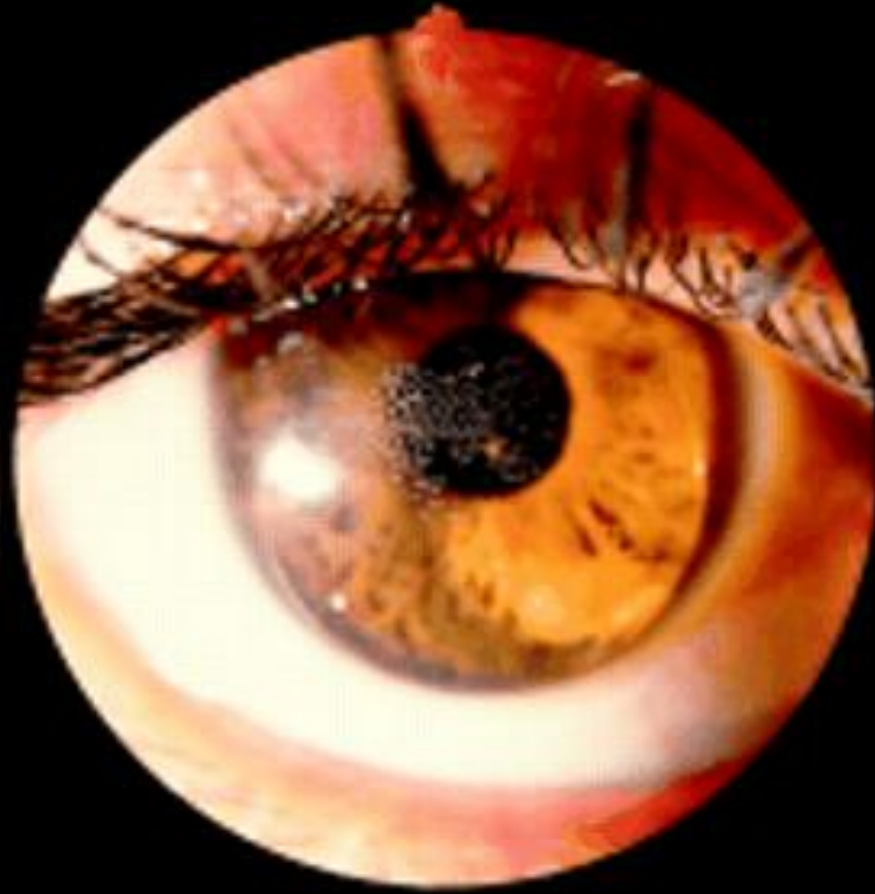
Table 12-1

## Frequency of Signs and Symptoms in 935 Cases of Decompression Sickness

Sign or Symptom	Number of Instances Within 935 Cases	Percentage of Instances Within 935 Cases	Number of Instances Manifested Initially	Percentage of Initial Manifestations
Localized pain	858	91.8	744	76.6
Numbness or paresthesia	199	21.2	41	4.3
Muscular weakness	193	20.6	8	0.8
Skin rash	140	14.9	42	4.4
Dizziness or vertigo	80	8.5	24	2.5
Nausea or vomiting	74	7.9	8	0.8
Visual disturbances	64	6.8	14	1.4
Paralysis	57	6.1	2	0.2
Headache	37	3.9	5	0.5
Unconsciousness	26	2.7	6	0.6
Urinary disturbances	24	2.5	0	—
Dyspnea ("chokes")	19	2.0	4	0.4
Personality change	15	1.6	0	—
Agitation or restlessness	13	1.3	0	—
Fatigue	12	1.2	2	0.2
Muscular twitching	12	1.2	0	—
Convulsions	11	1.1	0	—
Incoordination	9	0.9	0	—
Equilibrium disturbances	7	0.7	0	—
Localized edema	5	0.5	0	—
Intestinal disturbance	4	0.4	0	—
Auditory disturbance	3	0.3	0	—
Cranial nerve involvement	2	0.2	0	—
Aphasia	2	0.2	0	—
Hemoptysis	2	0.2	0	—
Emphysema—subcutaneous	1	0.1	0	—

From Rivera JC: Decompression sickness amongst divers: An analysis of 935 cases. *Milit Med* 129:314-334, 1963.





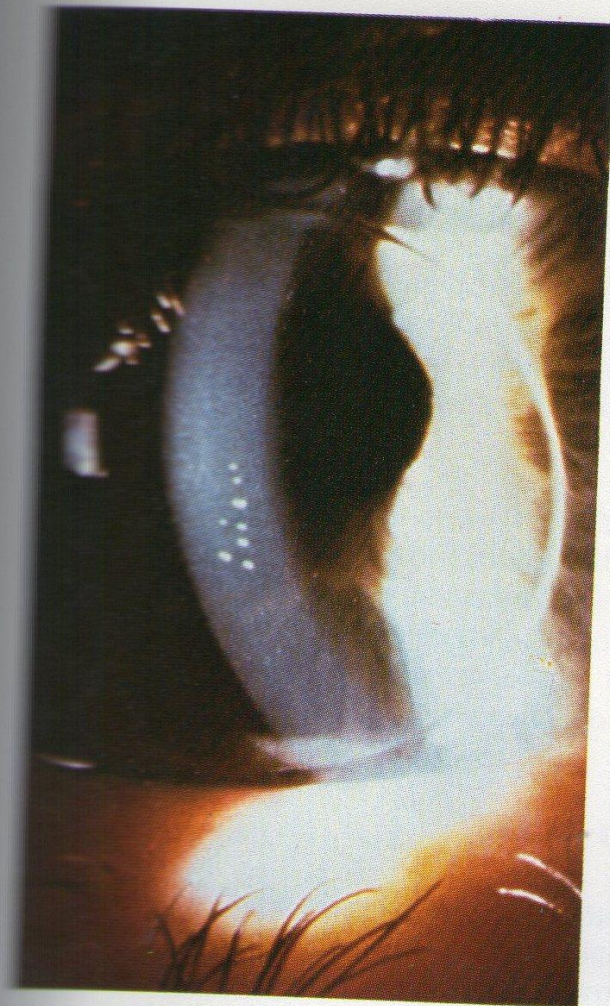


PLATE 6: (a) Bubbles developing between the hard contact lens and the cornea, decompression.

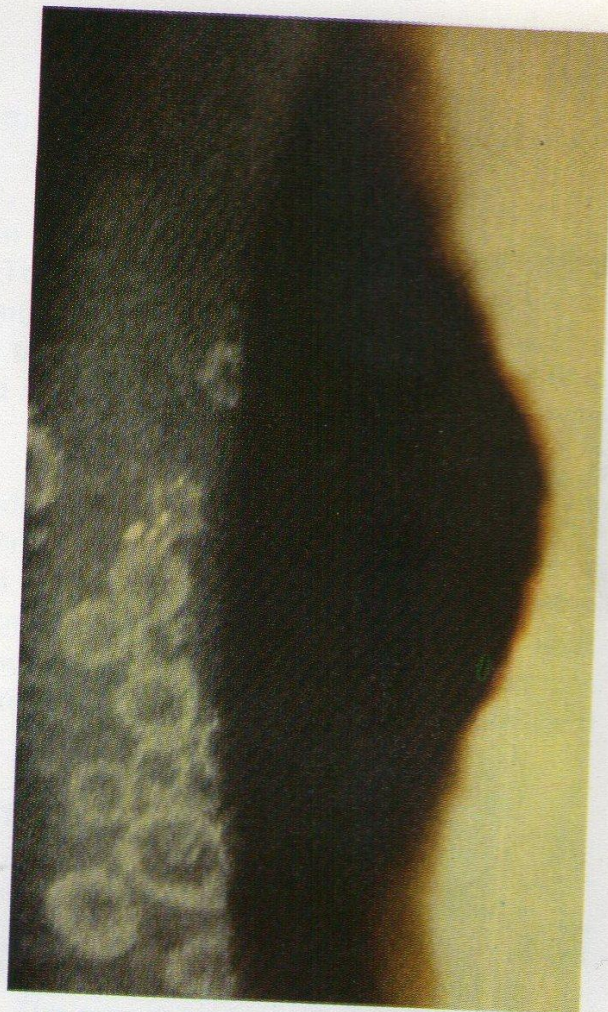
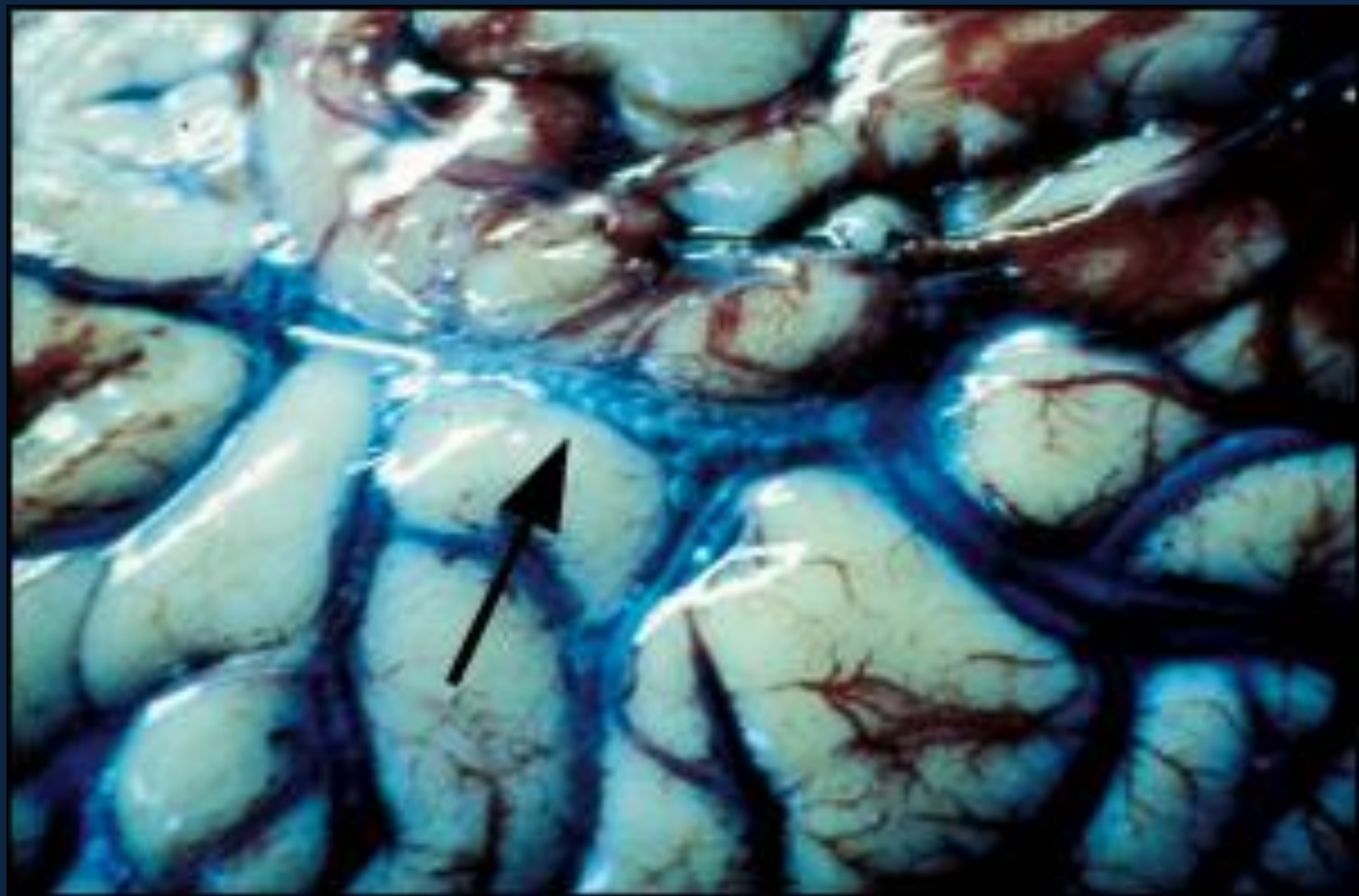
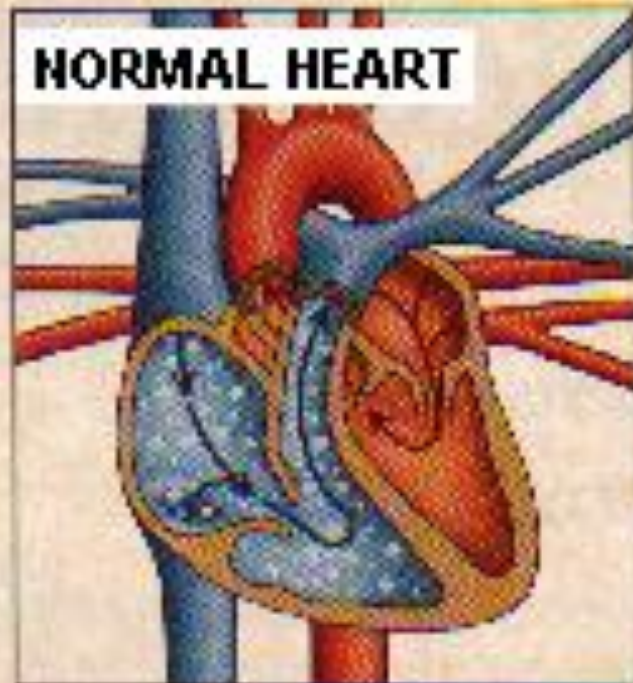


PLATE 6: (b) Nummular patches of corneal epithelial oedema, where the bubbles injure the cornea. (See Chapter 22).

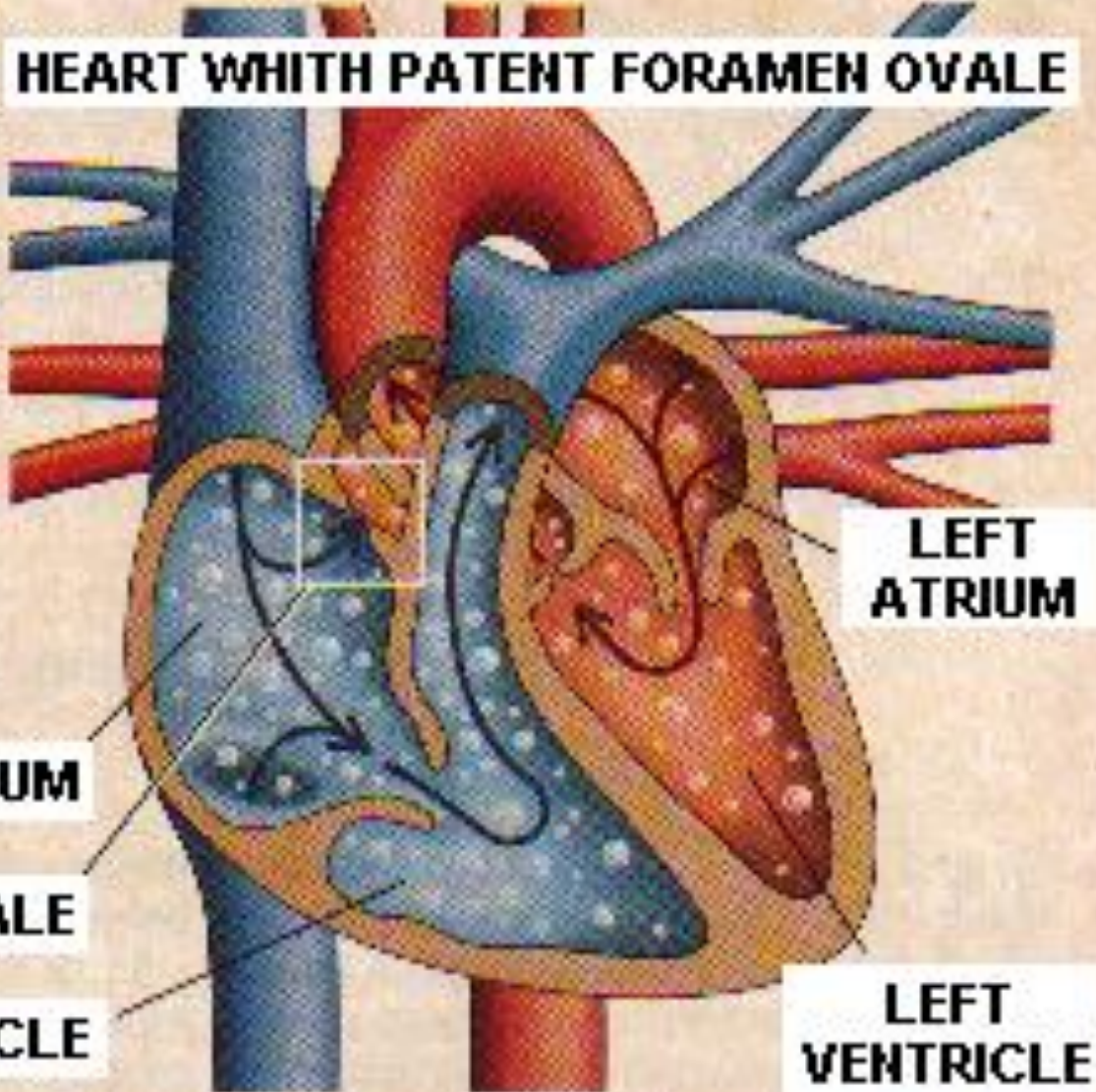
Photos: Courtesy of Drs. Mark E. Bradley & David Simon.



**NORMAL HEART**



**HEART WITH PATENT FORAMEN OVALE**



**RIGHT ATRIUM**

**PATENT FORAMEN OVALE**

**RIGHT VENTRICLE**

**LEFT  
ATRIUM**

**LEFT  
VENTRICLE**

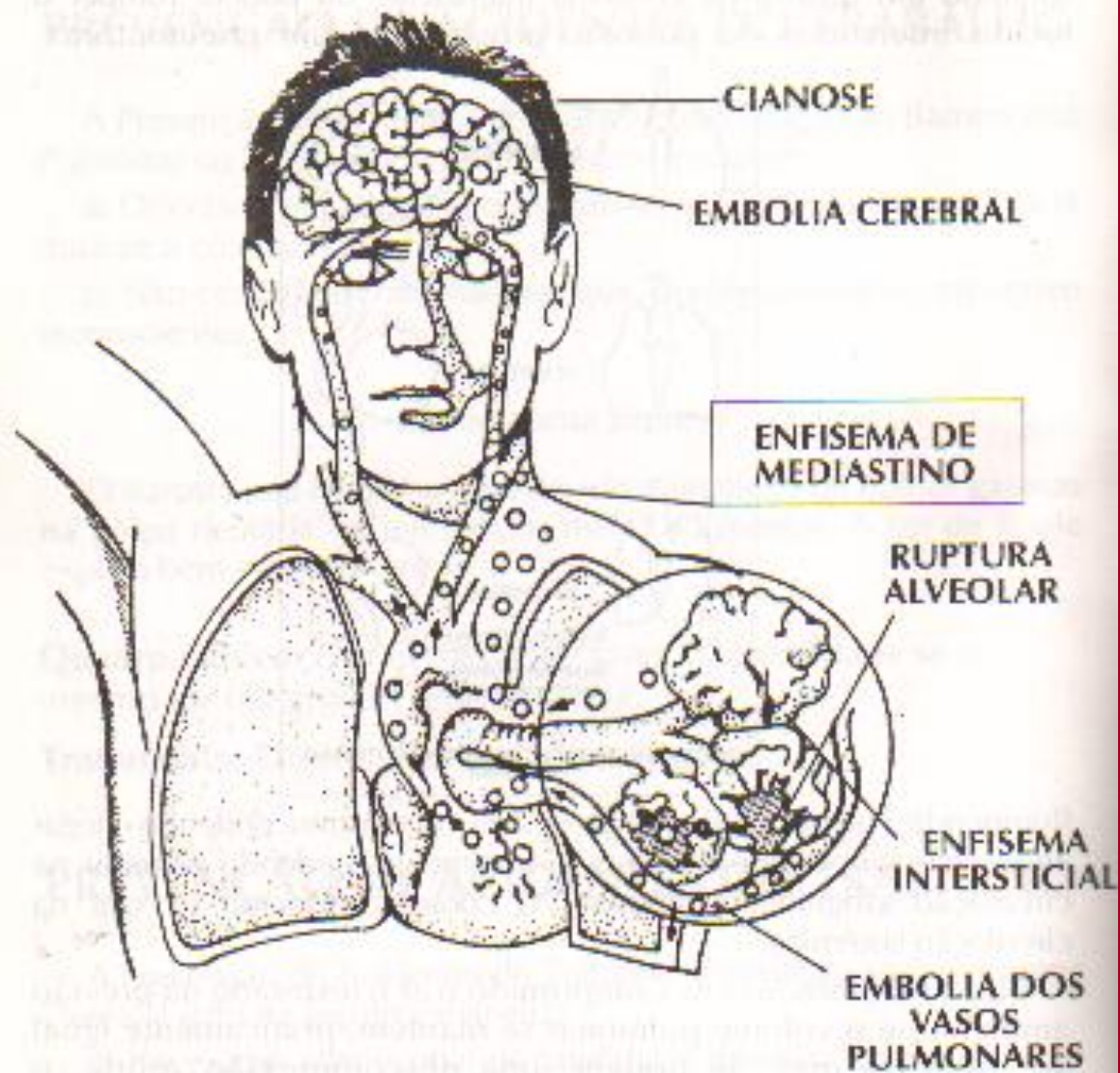




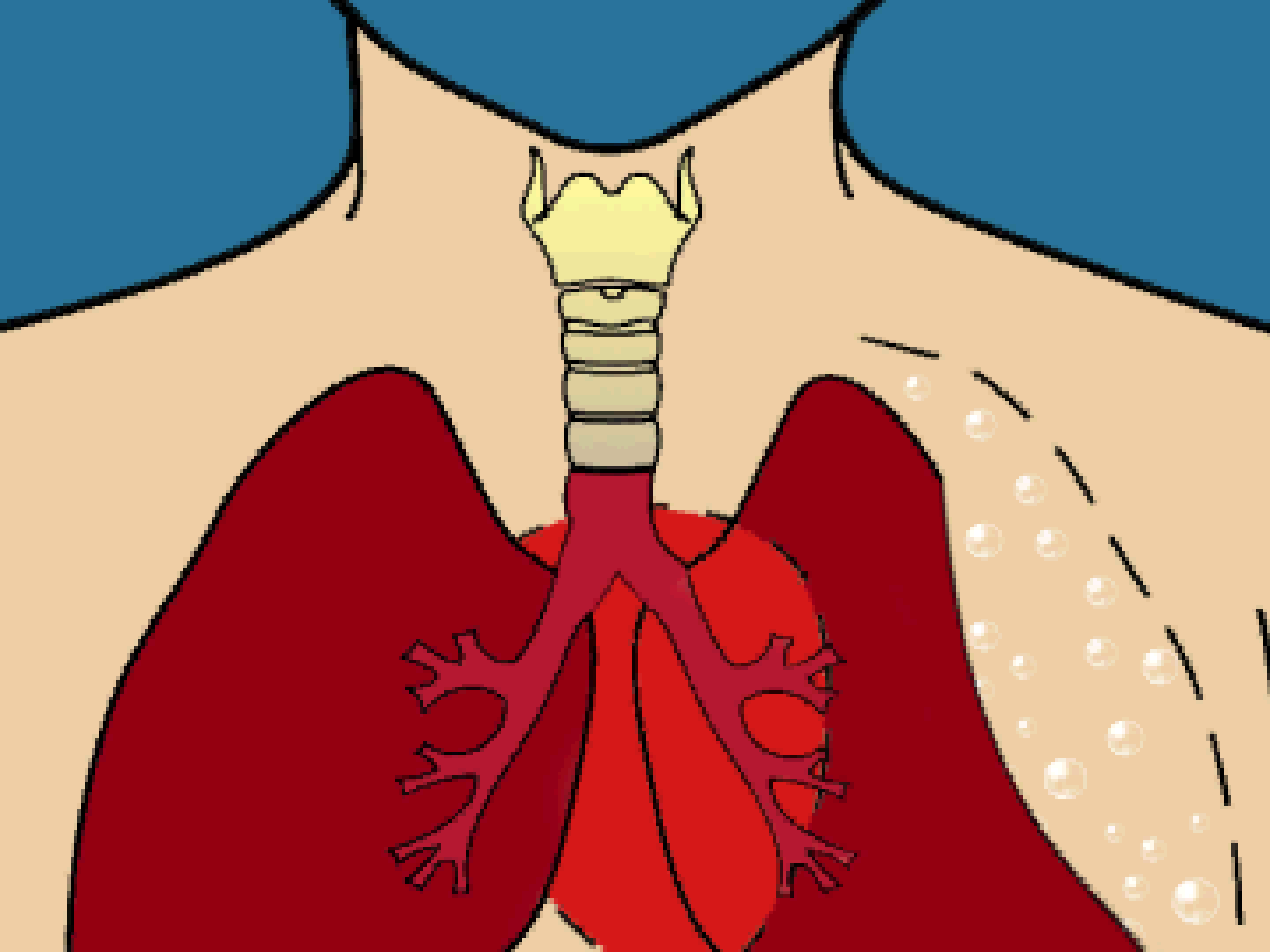
**Alvéolos**

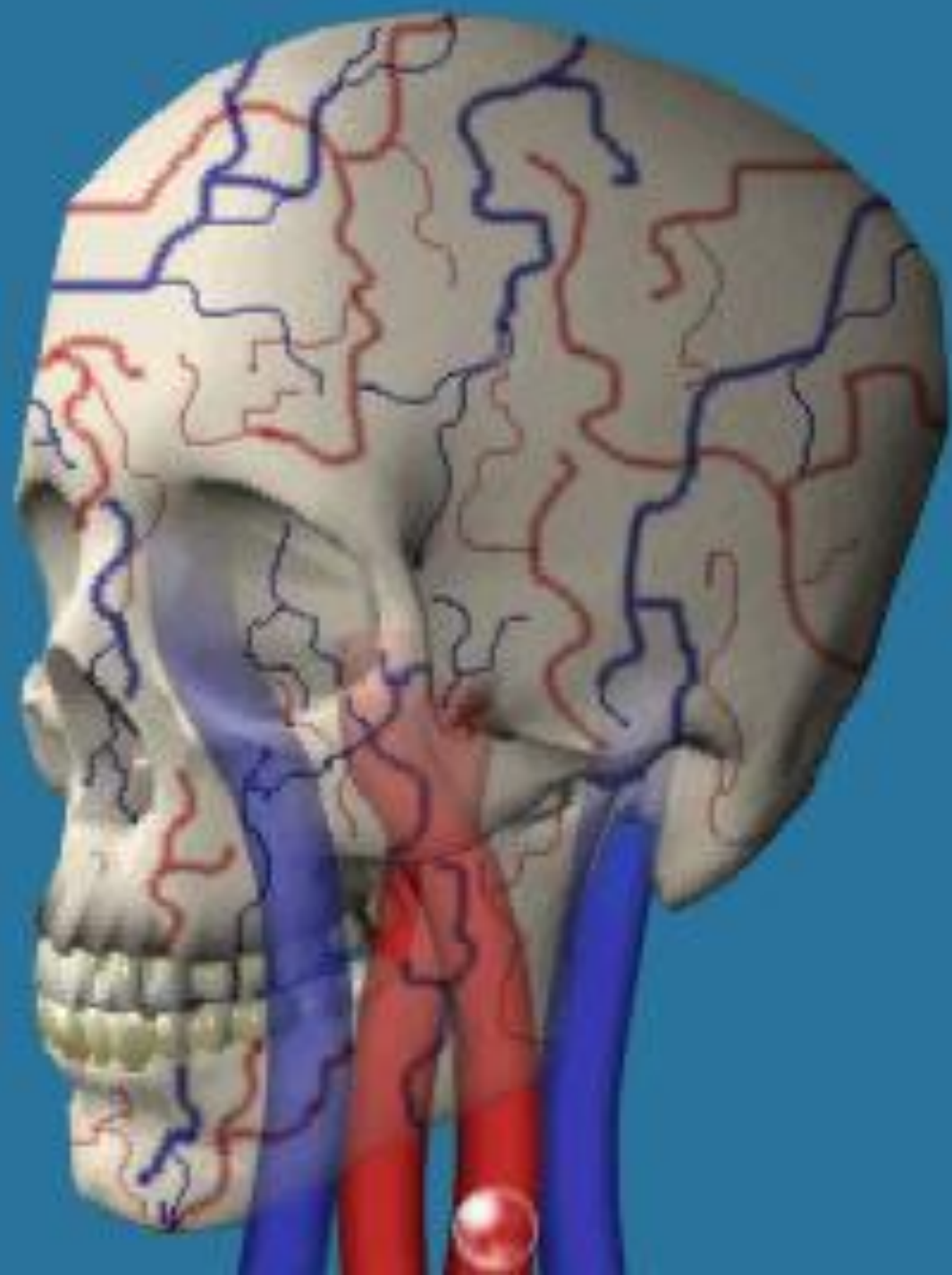
This diagram illustrates the human respiratory system. A central figure shows a human torso with the trachea and lungs highlighted in a glowing orange-yellow. A circular inset on the left provides a magnified view of the alveoli, showing their honeycomb-like structure and the network of blood vessels (arteries in red, veins in blue) that supply them. The inset also shows a cluster of alveoli that are collapsed and filled with blood, representing a pathological state.

**Alvéolos rompidos,  
inundados de sangue**



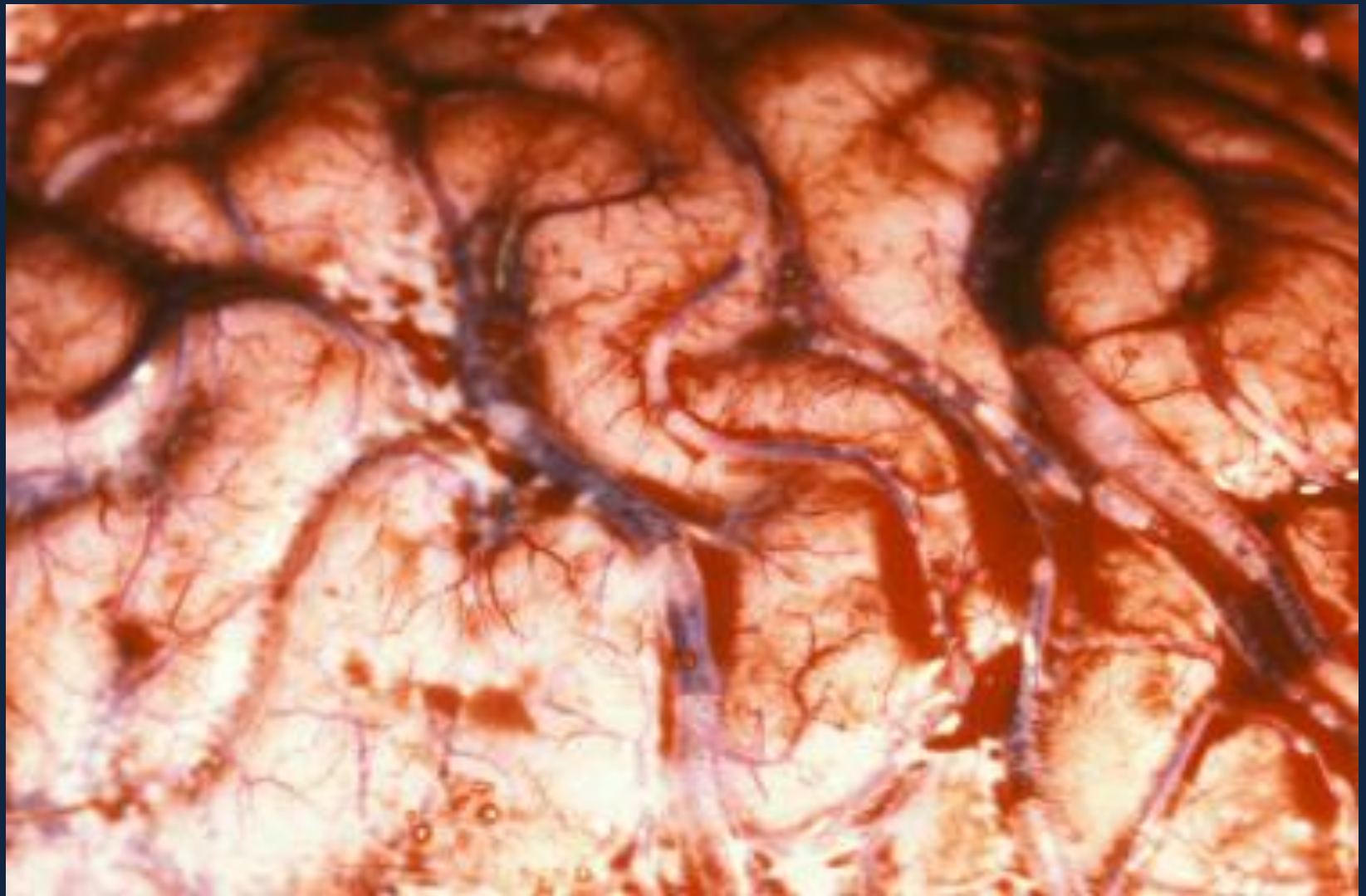
**TRAJETÓRIAS EXTRA-ALVEOLARES DO AR**

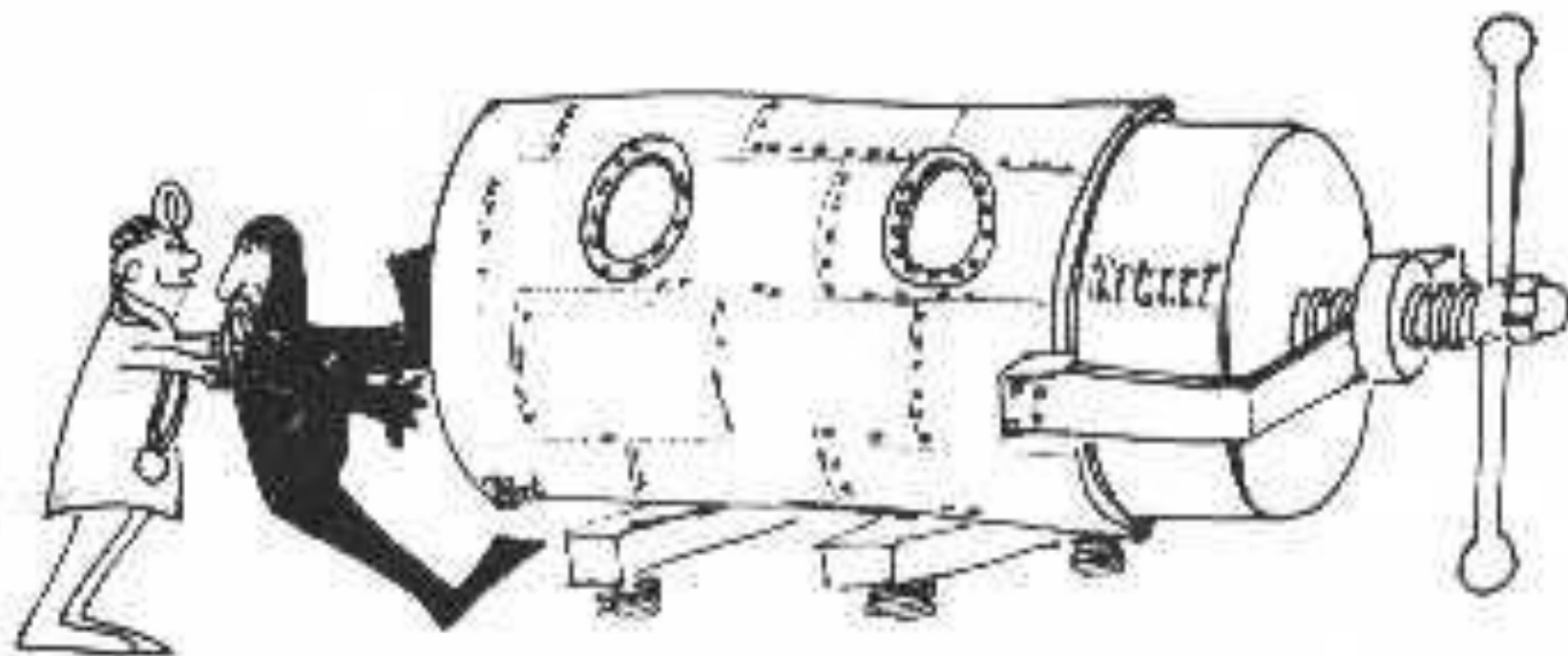












# Condutas em emergências subaquáticas

## 11) Doenças descompressivas (DD):

Urgência / Emergência em MH

Recompressão SEMPRE mesmo após remissão

Valorizar mesmo sinais / sintomas discretos

Inaptidão temporária ou definitiva

## 12) Embolia Traumática pelo Ar (ETA):

EMERGÊNCIA DAS EMERGÊNCIAS em MH

Drenar tórax ANTES de recomprimir

Pode estar associada à DD, barotraumas ou afogamento

Obs.: *Exame neurológico detalhado / documentado*

*Estar preparado para SBV / SAV*



## OXYGEN SYSTEM BASICS

Emergency oxygen systems come in various sizes and styles, but most incorporate the same basic elements: a high-pressure oxygen storage cylinder, a regulator/control valve assembly and a delivery mask.

The two primary types of emergency oxygen systems for divers are constant-flow systems and demand systems. Constant-flow systems provide a regulated, and in some cases adjustable, flow of oxygen to the delivery mask. Demand systems provide oxygen in response to the user's inhalation, thus delivering a higher percentage of oxygen and wasting less gas. Because treatment needs can vary from case to case, DAN recommends a multi-function system that can provide oxygen using both the demand and constant-flow options.







# Full Treatment

Emergency oxygen is only the first step to treating suspected cases of decompression illness.

BY MARTY McCAFFERTY



STEPHEN FRANK

## The Diver

The diver was a 44-year-old male recreational scuba diver, certified for more than 10 years, who was active in the sport and had no known medical conditions. During a two-week dive vacation in the South Pacific, he completed 36 dives on air over a 12-day period. His deepest dive in the series was to 100 feet, and his last dive was to 75 feet for 60 minutes. All dives went as planned, and he denied any history of rapid ascents, gear malfunctions or decompression obligations.

## The Incident

When he surfaced from his 36<sup>th</sup> and final dive, he lost feeling in his lower back and legs. His condition rapidly progressed to include loss of vision and lower-extremity muscular weakness. He required assistance getting into the boat and was immediately placed on high-concentration oxygen via a demand regulator. He had a brisk response to oxygen and reported feeling normal after 30 minutes of therapy, at which point oxygen treatment was discontinued. Physical examination revealed that he still had weakness and sensory deficits in his legs. Taking no chances, the boat crew arranged transport to the nearest medical facility for evaluation and recompression. The time from developing symptoms until the initiation of hyperbaric treatment was approximately two hours.

UTI HC

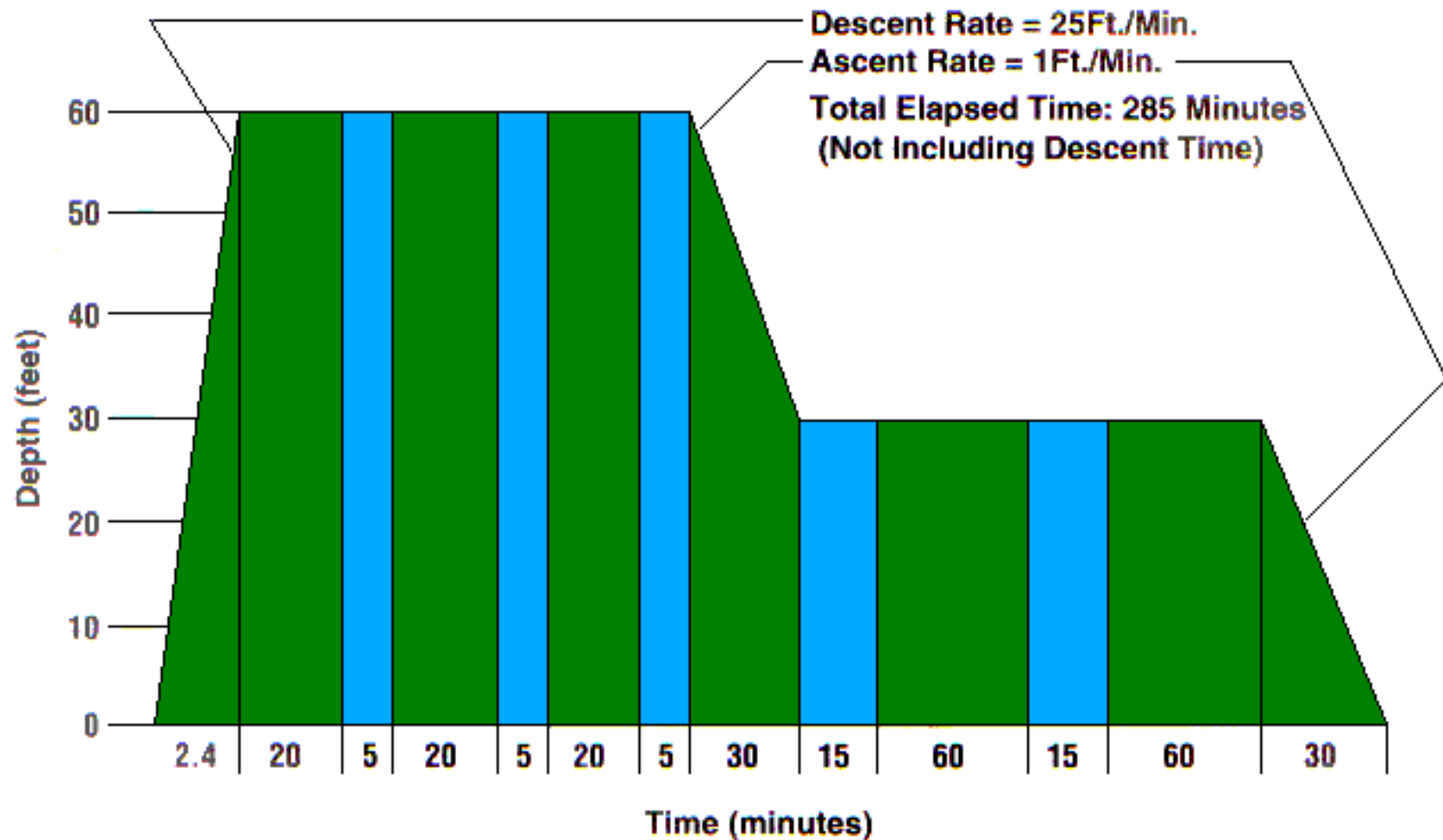
**Barotrauma**



**Pneumotórax**



**TABLE 6 DEPTH/TIME PROFILE**



*Obs.: existem tabelas com e sem oxigênio*

# Diagnósticos Diferenciais para DD e ETA

1- S. Guillain-Barré

2- Esclerose Múltipla

3- Paralisia Ulnar Progressiva

4- AVE

5- IAM

6- Embolia Pulmonar

7- Hipoglicemia

*Obs.: Dieta Vegetariana*

## **Decompression sickness in a vegetarian diver**

### **Are vegetarian divers at risk? A case report**

Robert A van Hulst<sup>1</sup>, Wim van der Kamp<sup>2</sup>

1 Diving Medical Center, Royal Netherlands Navy, The Netherlands

2 Department of Neurology, Sint Elisabeth Hospital, Willemstad, Curaçao

Corresponding author:

Dr. R.A. van Hulst – [ra.v.hulst@mindef.nl](mailto:ra.v.hulst@mindef.nl) + [ravhulst@planet.nl](mailto:ravhulst@planet.nl)

We present a case of a diver who suffered decompression sickness (DCS), but who also was a strict vegetarian for more than 10 years. He presented with symptoms of tingling of both feet and left hand, weakness in both legs and sensory deficits for vibration and proprioception after two deep dives with decompression. The initial clinical features of this case were most consistent with DCS, possibly because of a vulnerable spinal cord due to cobalamin deficiency neuropathy. This case illustrates the similarities between DCS and a clinically defined vitamin B12 deficiency. The pathophysiology of vitamin B12 deficiency and common pathology and symptoms of DCS are reviewed.









*Imagem by Valéria*













THE  
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