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Network model to study physiological processes of hypobaric decompression sickness: New numerical results

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ABSTRACT

We have studied decompression processes when pressure changes that take place, in blood and tissues using a technical numerical based in electrical analogy of the parameters that involved in the problem. The particular problem analyzed is the behavior dynamics of the extravascular bubbles formed in the intercellular cavities of a hypothetical tissue undergoing decompression. Numerical solutions are given for a system of equations to simulate gas exchanges of bubbles after decompression, with particular attention paid to the effect of bubble size, nitrogen tension, nitrogen diffusivity in the intercellular fluid and in the tissue cell layer in a radial direction, nitrogen solubility, ambient pressure and specific blood flow through the tissue over the different molar diffusion fluxes of nitrogen per time unit (through the bubble surface, between the intercellular fluid layer and blood and between the intercellular fluid layer and the tissue cell layer). The system of nonlinear equations is solved using the Network Simulation Method, where the electric analogy is applied to convert these equations into a network-electrical model, and a computer code (electric circuit simulator, Pspice). In this paper, numerical results new (together to a network model improved with interdisciplinary electrical analogies) are provided.

1. Introduction

Decompression sickness (DCS) describes a condition arising from dissolved gases coming out of solution into bubbles inside the body on depressurization. DCS most commonly refers to problems arising from underwater diving decompression (i.e., during ascent), but may be experienced in other depressurization events such as working in a caisson, flying in unpressurized aircraft, and extra-vehicular activity from spacecraft. DCS is a subset of decompression illness (DCI) which includes both DCS and arterial gas embolism (AGE). In more current applications, hypobaric decompression and the associated altitude decompression illness (DCI) are reported in high-altitude aviators and astronauts [1,2]. DCI may occur in response to acute reduction in ambient atmospheric pressure, for example, as experienced by astronauts during extravehicular activities (EVAs). Over the past decades, great strides have been made in the understanding of hypobaric physiology. Prevention of hypobaric or altitude DCI has involved oxygen prebreathing (before and during hypobaric exposure), staged decompression, and other modalities, although full protection against DCI has yet to be achieved [3].

The pathological processes linked to variations of pressure in hyperbaric or hypobaric conditions are closely associated with the conversion of the soluble gas absorbed in the tissues or components of the circulatory system to gas free phase [4]. The symptoms are initiated by the separation and expansion of the gas phase; therefore, hypothetically, an

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Nomenclature

C	sum of partial pressures of metabolic gases, kPa
D_1	nitrogen diffusivity in intercellular layer fluid, $cm^2\ min^{-1}$
D_2	nitrogen diffusivity in tissue cells layer, $cm^2\ min^{-1}$
F	force, N
G	volume (fluid) flow, m^3/s
I	electrical current, A
J	mass molar diffusion nitrogen flux, $\mu moles\ min^{-1}$
J_{B1}	mass molar nitrogen flux through the surface of the bubble, $\mu moles\ min^{-1}$
J_{12}	mass molar nitrogen flux between the intercellular fluid layer and in the tissue cell layer, $\mu moles\ min^{-1}$
J_{10}	mass molar nitrogen flux between the intercellular fluid and the blood, $\mu moles\ min^{-1}$
P	pressure, Pa
P_a	nitrogen tension in arterial blood, kPa
P_{Amb}	ambient pressure, kPa
P_B	partial pressure of nitrogen in a bubble, kPa
P_1	nitrogen tension at outer surface of an intercellular fluid layer, kPa
P_2	nitrogen tension at outer surface of a tissue cells layer, kPa
q	specific blood flow through a tissue, $ml_{blood}\ ml_{tissue}^{-1}\ min^{-1}$
$\dot{Q}$	heat flux, W
R	resistance
$\overline{R}$	universal gas constant, 8314 kJ/mol K
R_{Bi}	radius of a bubble seed, cm
R_B	current bubble radius, cm
R_1	current outer radius of a spherical layer of intercellular fluid adjacent to the bubble, cm
R_2	current outer radius of a spherical layer of tissue cells adjacent to the bubble, cm
R_{1i}	initial R_1 magnitude, cm
R_{2i}	initial R_2 magnitude, cm
T	torque, Nm
t	time, min
V	electrical voltage, V
v	velocity, m/s
$\Delta(P_1)_B$	Gradient of nitrogen tension at the bubble surface, $kPa\ cm^{-1}$
$\Delta(P_2)_B$	Gradient of nitrogen tension at the inner surface of tissue cells layers, $kPa\ cm^{-1}$
α_0	nitrogen solubility in blood, $ml_{gas}\ ml_{blood}^{-1}\ (101.3\ kPa)^{-1}$
α_1	nitrogen solubility in intercellular layer fluid, $ml_{gas}\ ml_{blood}^{-1}\ (101.3\ kPa)^{-1}$
α_2	nitrogen solubility in tissue cells layer, $ml_{gas}\ ml_{blood}^{-1}\ (101.3\ kPa)^{-1}$
θ	temperature, °C
γ	surface tension of intercellular fluid, kPa cm ($1\ dyna\ cm^{-1} = 10^{-4}\ kPa\ cm$)
ω	angular velocity, rad/s

index based on the growth dynamics of bubbles in the tissues would allow us to predict with greater accuracy the symptoms of DCS than the conventional index based on tissue oversaturation [5]. This author suggested that the slow bubble growth in tissues is the result of a diffusion barrier existing around a bubble that limits the intensity of gas flux into the bubble. His model of tissue bubble dynamics and estimations of decompression stress indices for a range of decompression procedures are consistent with DCS latency times, provided that the thickness of diffusion barrier surrounding a bubble is 3 μm and nitrogen diffusivity in this bubble shell equals $2 \times 10^{-8}\ cm^2\ s^{-1}$.

Boycott et al. studied the prevention of accidents in processes of decompression in 1908 [6]. They established the bases necessary to develop fast procedures and more accurate tables to carry out decompression, mainly for divers. The considerations that they took into account to elaborate those tables were: the composition of the gaseous mix and the criteria for the ascent. Based on the studies realized by Nikolaev [7], Foster et al. [8], Hyldergard and Madsen [9] and Webb and Pilmanis [10] in relation to the dynamic of gaseous bubbles in hypobaric conditions and those realized by Zueco and Rubio [11] and Zueco and Rubio-Hernández [12] using the Network Simulation Method (NSM) to obtain the numerical solutions of the cases studied here, we presented a network model based on electrical analog to the numerical simulation of the dynamics of the bubble size and the dynamics of nitrogen tension in tissue elements surroundings a bubble, varying tissue parameters final hypobaric pressure values.

The model describes the dynamics of extravascular bubbles formed in intercellular cavities of a hypothetical heterogeneous tissue submitted to a hyperbaric decompression process, with a supply of 100% oxygen, besides of the nitrogen tension distribution in the massive layers of cells surrounding the bubble (intercellular liquid, cellular layer of the adjacent tissue and blood flow system). Due to the high nitrogen diffusivity in the adjacent intercellular fluid, during the initial stage of growth a gas bubble is instantaneously formed to reach a size determined by the initial volume of the intercellular cavity, surface tension of the fluids, initial nitrogen tension in the tissues and final pressure level. The growth rate and the maximum size achieved by the new bubble depends on the nitrogen diffusivity value in the cellular layer, specific blood flow through a tissue, initial nitrogen tension and the final ambient pressure.

Conventional mathematical models of gas bubble dynamics in tissues are initially based on the simplification that each body tissue is a continuous homogeneous medium with a constant diffusion permeability for nitrogen or any other inert gas of the breathing mixture [13–15]. With assumed values in the order of $10^{-5}\ cm^2\ s^{-1}$ for nitrogen diffusivity in tissues, the models overestimate bubble size. In accordance with these models at this nitrogen diffusivity