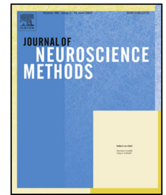
Journal of Neuroscience Methods 236 (2014) 40–43

Contents lists available at ScienceDirect

Journal of Neuroscience Methods

journal homepage: www.elsevier.com/locate/jneumeth



Basic Neuroscience

Cost-efficient method and device for the study of stationary tissular gas bubble formation in the mechanisms of decompression sickness

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HIGHLIGHTS

- Unique method to study the role of stationary tissular gas bubbles in decompression sickness.
- Evidence of cell injury induced by stationary tissular gas bubble formation in the rat brain.
- Method with potential for performing tissue compartment research.

ARTICLE INFO

Article history:
Received 30 May 2014
Received in revised form 14 July 2014
Accepted 15 July 2014
Available online 23 July 2014

Keywords:
Decompression sickness
Gas bubbles
Diving accident
Excitotoxic cell injury

ABSTRACT

Background: Current *in vivo* methods cannot distinguish between the roles of vascular and stationary tissular gas bubbles in the mechanisms of decompression sickness (DCS).
New method: To answer this question, we designed a normobaric–hyperbaric chamber for studying specifically the contribution of stationary tissular gas bubbles in the mechanisms of DCS in individually-superfused tissue samples. For validating our method, we investigated in rat brain slices exposed to 0.4 MPa air absolute pressure whether fast decompression rate – the most important cause of cerebral DCS – may induce an increase of lactate dehydrogenase (LDH), a marker of cell injury, compared to slow decompression rate.
Results: We provide a technical description of our pressure chamber and show that fast decompression rate of 0.3 MPa min^{-1} induced a rapid and sustained increase of LDH release compared to slow compression rate of 0.01 MPa min^{-1} ($P < 0.0001$).
Comparison with existing methods: There is no current method for studying stationary tissular gas bubbles.
Conclusions: This report describes the first method for studying specifically in tissue samples the role of stationary tissular gas bubbles in the mechanisms of DCS. Advantageously, according to this method (i) biological markers other than LDH could be easily studied; (ii) tissue samples could be taken not only from the brain but also from any part of the animal's body known of interest in DCS research, allowing performing tissue compartment research, a major question in the physics and theory of decompression research; and (iii) histological studies could be performed from the tissue samples.
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1. Introduction

Decompression sickness (DCS) is an environmental hazard that can occur when a subject – mainly underwater divers, caisson workers, and hyperbaric chamber and aircraft crews – is decompressed from a given ambient pressure to a lower pressure. Neurologic DCS is predominant. In open water divers, the spinal cord is the most affected area, but the brain can be affected too (Vann et al., 2011). The clinical picture patterns vary from

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http://dx.doi.org/10.1016/j.jneumeth.2014.07.010
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minimal sensory abnormalities to severe sensory, motor and/or urinary disorders (Francis and Mitchell, 2003a). Cerebral damage may also occur particularly following an unusual rapid decompression. In contrast, in aviators, the brain is more commonly affected than the spinal cord (McGuire et al., 2012). Then, the clinical picture mainly includes visual disturbances, vertigo, altered high cognitive function and speech, hemiparesis and unconsciousness (Francis and Mitchell, 2003a; McGuire et al., 2012). In both cases, the pathophysiological mechanisms of neurologic DCS are thought to result from an excessive venous or arterial gas bubble embolization, and from the occurrence of stationary gas bubbles originating from the inert gases dissolved within the spinal cord or the brain parenchyma (Francis and Mitchell, 2003b). Although the consequence of an excessive vascular bubble formation has been quite well documented in animal studies by showing gas bubble-induced vascular ischemia and subsequent ischemia-induced thrombin generation, blood platelet aggregation and coagulation, and immune-inflammatory responses (Ersson et al., 1998; Martin and Thom, 2002; Nyquist et al., 2004; Bigley et al., 2008), the effects of stationary gas bubble formation within the tissues, while being of possible critical importance in the production of mechanically-induced excitotoxic processes and subsequent neuronal injury (as this occurs in traumatic injury), still remains to be demonstrated.

Despite state-of-the-art hyperbaric oxygen treatment in hyperbaric medical units, about 30% of patients suffering neurologic DCS exhibit incomplete recovery (Blatteau et al., 2011). Clearly this indicates that further research is needed to increase our knowledge and understanding of the basic mechanisms of DCS in order to improve clinical therapeutic treatment. However, basic DCS research as usually investigated using the *in vivo* pressure chambers and methods currently available to date suffers major limitations that include the cost of the pressure chambers, which increases dramatically with size and pressure, the high level of safety and technology constraints required for operating these chambers, the limited number of animals (generally one or two) and laboratory devices that can be used and set up in the chambers' closed space, and importantly, the inability of the *in vivo* methods to distinguish between the roles of vascular and stationary tissular gas bubble formation in the mechanisms of DCS. To resolve the latter, we designed a cost-efficient normobaric–hyperbaric versatile chamber that allows investigating specifically the effects of stationary tissular gas bubble formation in individually-superfused tissue samples. Here, in addition of providing a comprehensive technical description of our pressure chamber, we report new data that validate its usefulness for the study of stationary tissular gas bubble formation and provide evidence for gas bubble-induced cell injury in the mechanisms of DCS.

2. Materials and methods

The pressure chamber was made from a poly-hexamethylene-adipamide (nylon) block. Briefly, after machining, the chamber includes a main body that comprises sixteen built-in wells to welcome individually-superfused tissue samples, a gas input connected to a micrometric valve that allows regulating gas inflow in the chamber, and two versatile caps that allow distributing the gas to the tissue samples through microtubing in normobaric conditions or by increasing the gas pressure in the chamber in hyperbaric conditions. A detailed technical description of the chamber is given in Fig. 1 (chamber equipped with cap for normobaric studies) and Fig. 2 (chamber equipped with cap for hyperbaric studies).

Then, as a necessary condition for validating our vessel and method, we investigated in rat brain slices whether fast decompression rate – the most important cause of cerebral DCS – may

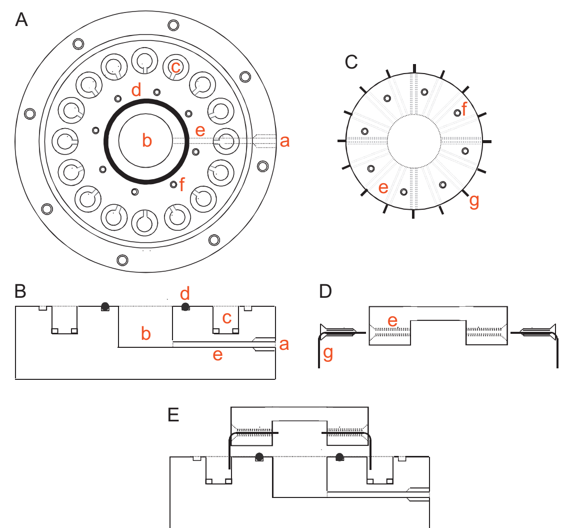

Fig. 1. Chamber mounted with normobaric cap. (A) Top view of main body. (B) Sectional front view of main body. (C) Top view of normobaric cap. (D) Sectional front view of normobaric cap. (E) Assembling of main body and normobaric cap. In (A)–(D): (a) gas input with screw thread for assembling a micrometer valve to the main body for regulating gas flow; (b) built-in main well; (c) built-in tissue samples' wells; (d) O-ring; (e) built-in pipes; (f) screw thread for assembling normobaric cap and main body; (g) microtubing for distributing gas to the tissue samples.

induce an increase of lactate dehydrogenase (LDH) used as a marker of cell injury (David et al., 2008) as compared to slow decompression rate. All experiments were performed in accordance with the declaration of Helsinki and the framework of the French legislation for the use of animals in biomedical experimentation. Brain slices were drawn from male adult Sprague-Dawley rats (Janvier, Le Genest Saint-Isle, France) weighing 250–280 g as follows: (i) rats were killed by decapitation, and the brains were carefully removed and placed in ice-cold freshly prepared artificial cerebrospinal fluid (aCSF) containing 120 mM NaCl, 2 mM KCl, 2 mM $CaCl_2$, 26 mM

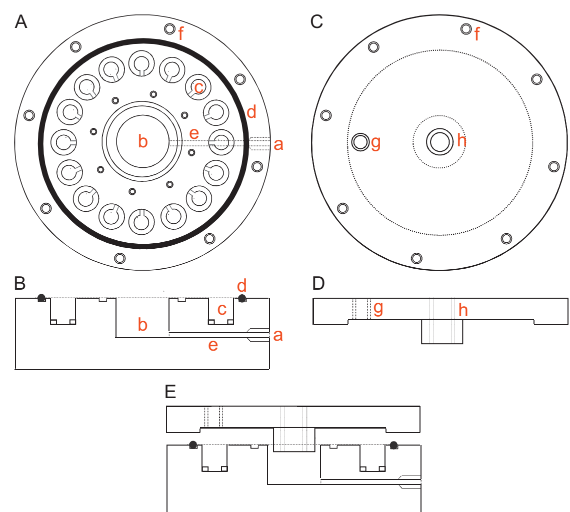

Fig. 2. Chamber mounted with hyperbaric cap. (A) Top view of main body. (B) Sectional front view of main body. (C) Top view of hyperbaric cap. (D) Sectional front view of hyperbaric cap. (E) Assembling of main body and hyperbaric cap. In (A)–(D): (a) gas input with screw thread for assembling a micrometer valve to the main body for regulating gas flow; (b) built-in main well; (c) built-in tissue sample's wells; (d) O-ring; (e) built-in pipe; (f) screw thread for assembling hyperbaric cap and main body; (g) screw thread for assembling a temperature probe to the hyperbaric cap (the temperature probe is placed instead of a brain slice in a built-in tissue sample's well to control aCSF temperature); (h) screw thread for assembling a pressure gauge to the hyperbaric cap.

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