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SIMULATIONS OF NEAR-DROWNING AND DECOMPRESSION SICKNESS: A PRELIMINARY STUDY

Chris Acott and David J Doolette

Key Words

Decompression illness, near drowning, oxygen, physiology, simulations, treatment.

Abstract

Theoretically near-drowning should decrease inert gas elimination from tissues by a reduction in cardiac output and increased intrapulmonary shunting. A delay in inert gas elimination may prolong tissue supersaturation and so

increase the risk of decompression sickness (DCS). However, there are no data on inert gas elimination or the incidence of decompression sickness in near-drowned compressed air divers. Resuscitation might also retard inert gas elimination because of the adverse cardiovascular effects of intermittent positive pressure ventilation (IPPV) and positive end expiratory pressure (PEEP).

Decompression modelling, using Linear-exponential kinetics, of near-drowning scuba dive accident scenarios have shown an increased risk of DCS for no-stop dives to above the acceptable level of risk of 2.3% used by the United States Navy. Modelling of resuscitation following near-drowning demonstrated that there is no further increase in DCS risk provided the cardiac output was normal before IPPV and PEEP were instituted.

All compressed air divers, who have near-drowned, except those who have a minimum disturbance of shunt and cardiac output, should be carefully assessed with regard to decompression risk and treated appropriately. Divers who had been resuscitated from a cardiac arrest or are severely shocked at presentation should be recompressed because of the risk of decompression sickness is increased to between 25 and 52%.

Introduction

Near-drowning should theoretically decrease inert gas elimination from a reduction in cardiac output, leading to reduced tissue perfusion, and an increase in pulmonary areas of low ventilation perfusion ratios, increased anatomical shunt, or a combination of both (collectively intrapulmonary shunt).[1,2] A delay in inert gas elimination would be expected to prolong tissue supersaturation and so increase the risk of decompression sickness (DCS). However, there are no data on inert gas elimination nor the incidence of decompression sickness in near-drowned compressed air divers.

Anecdotal reports indicate there is a decrease in cardiac output and an increased intrapulmonary shunt following near-drowning in humans but there are no reliable published data. Some victims may suffer a cardiac arrest but respond to cardiopulmonary resuscitation and will have cardiac output as low as 30%. There are also limited data on the magnitude of increase in shunt in near-drowned victims, however, there is one report that the shunt could increase to 75%.[3] Experimental studies in animals indicate decreased cardiac output and increased shunt following near-drowning.[4,5]

Resuscitation may initially retard inert gas elimination in a patient with compromised cardiovascular and respiratory systems. Intermittent positive pressure ventilation (IPPV) and positive end expiratory pressure (PEEP) are associated with a decrease in cardiac output and blood pressure due to an impaired venous return, decreased ventricular filling, increased pulmonary vascular resistance and altered configuration and compliance of the right and left ventricles even in patients without significant pulmonary pathology.[6,7]

Recompression and hyperbaric oxygen may also initially depress inert gas elimination if applied in a patient with decreased left ventricular function due to an increase in systemic vascular resistance, decrease in left ventricular contractility, increased after load, centralisation of blood volume and an imbalance between right and left heart function worsening pulmonary oedema.[8]

Since there are no experimental data on incidence of DCS in near-drowning we have made a theoretical evaluation on the risk of DCS using probabilistic decompression modelling. Probabilistic decompression modelling involves the "fitting" of a decompression model to a large data set of dive profiles (depth/time/breathing gas history) with known outcomes (DCS or no-DCS) using a non-linear regression procedure. Such a probabilistic decompression model assigns a probability of DCS to each dive profile. This probability is a function of the duration and the degree of tissue supersaturation.[9-11] These models present a theoretical framework for organising decompression experience, but are not an accurate description of the physiological and pathophysiological pathways of DCS. The most successful statistical decompression algorithm for air or nitrox mixtures up to 40% oxygen to date is the USN linear-exponential model (LE1). In this model 2,383 dives with an incidence of 131 cases of decompression sickness, and 75 "marginal cases", are used. In this paper we modified the USN LE1 model by combining it with a model of the cardio-pulmonary system which allowed arterial nitrogen tension to be modified by cardiac output and pulmonary shunt and used the resulting model to examine the effects of hypothetical near-drowning scenarios on probability of DCS.[9,12]

Definitions

We have defined **drowning** as death by submersion in a fluid with or without aspiration of fluid. **Near-drowning** is defined as survival following aspiration of large quantities of fluid or survival following unconsciousness while submerged in a fluid.

Method

Three square and 2 multi-level dive profiles were chosen because 2 South Australian dive sites near Adelaide provide such diving. The no-decompression times were derived from the Canadian Defence and Civil Institute of Environmental Medicine (DCIEM) decompression tables for the multi-level dives (D and E) and two (A and B) of the

square dives and from the United States Navy (USN) decompression tables for the other (C) square dive. All profiles were calculated for no-stop ocean diving.

A 18 m for 50 minutes (DCIEM).
B 30 m for 15 minutes (DCIEM).
C 30 m for 25 minutes (USN).
D a multi-level dive of 18 m for 50 minutes followed by 12 m for 30 minutes and then 6 m for 30 minutes.
E a multi-level dive of 18 m for 50 minutes followed by 12 m for 30 minutes and then 6 m for 5 minutes.

The resuscitation simulations were based on the clinical experience of near-drowning victims and diving accidents of one of the authors (CJA).

Intrapulmonary shunt of 5% (normal), 50% and 70% and cardiac output of 100% (normal), 50% and 30% were simulated for the different dive profiles and near drowning scenarios.

For the simulation it was assumed that the diver near-drowned at the end of the dive.

a After 15 minutes or 45 minutes, representing retrieval to the boat and to oxygen, on-site resuscitation was started using of 0.6 bar oxygen.
b During transfer from the boat to the hospital 2 hours from the end of the dive, the victim then breathed 0.6 bar of oxygen.
c After admission to the Intensive Care Unit (ICU) 1.0 bar oxygen was administered for another 10 hours. We assumed that ICU management was successful and that the patient improved steadily. By the end of this time the victim's cardiac output and pulmonary function had returned to normal.

Another resuscitation simulation involved the patient receiving 0.6 bar oxygen from 15 minutes after surfacing from dive A until hospital was reached 2 hours later. The victim's cardiac output remained normal during this time but intrapulmonary shunt was 70%. In hospital intermittent positive ventilation (IPPV) and positive end expiratory pressure (PEEP) were applied with 1 bar oxygen resulting in a transient fall in cardiac output to either 50 or 30% of normal followed by a slow return to normal over an hour. This simulation was used to see if the effect of a sudden fall in cardiac output with the use of PEEP and IPPV increased the risk of decompression sickness during resuscitation.

The probability of DCS for simulated dive profiles was calculated using a modified USN linear-exponential algorithm (LE1). In the LE1 algorithm the probability of DCS associated with any diving exposure is calculated by tracking gas tensions in arterial blood and in 3 parallel perfusion limited compartments with different rate constants. Uptake of gas into the compartments is exponential and elimination is either exponential or linear. Linear kinetics and positive instantaneous risk may occur during and after decompression if compartment dissolved gas tensions exceed a threshold value above ambient pressure. Probability of DCS results from the time integral of the instantaneous risk in the 3 compartments.

The algorithm used in the present report similarly calculates probability of DCS from 3 compartment linear-exponential model using parameters described in 1997 by Thalmann et al.[10] and an implementation similar to that described by Gerth and Vann in 1997,[11] but differs in how arterial nitrogen tension is calculated.[12] Both algorithms calculate inspired nitrogen partial pressure from the depth/time/breathing gas history. The original LE1 algorithm assumes that arterial nitrogen tension equals alveolar nitrogen partial pressure and predicts the latter from inspired nitrogen partial pressure using the alveolar gas equation and assuming a respiratory quotient of 1.[13] The current algorithm incorporates a model of the cardio-pulmonary system to calculate arterial nitrogen tension.[12] In this model inspired gas, alveolar gas, pulmonary blood and the body are in series, the latter composed of 4 parallel compartments representing vessel rich, muscle, fat, and vessel poor tissue groups with blood flows and compartment volumes based on the standard 70 kg man.[14,15] The model uses nitrogen tissue/gas partition coefficients of 0.015 (blood), 0.015 (lean), 0.075 (fat) and lung nitrogen diffusing capacity of 0.15 L/min/kPa.[14-16] Pulmonary blood flow (cardiac output) and intrapulmonary shunt can be manipulated. Arterial nitrogen tension is therefore not only a function of depth/time/breathing gas history but also distribution of nitrogen into the body tissues, cardiac output, and pulmonary shunt. Tissue compartment blood flows (and therefore rate constants) in both the cardio-pulmonary model and the linear-exponential risk model components of the algorithm were considered to vary in proportion to cardiac output. Simulations were performed using Scientist for Windows version 2.01 (Micromath Inc.).

Results

The risk of DCS was calculated for the (non-drowning) profiles using Linear-exponential kinetics. The calculated risk for each of these dives is: (A) is 1.7%, (B) is 2.1%, (C) is 2.1%, (D) and (E) 2.7%. The results for the near-drowning scenarios are shown in tables 1 to 7. The "normal" values reflect the risk if oxygen is breathed as described above for 12 hours from the end of the dive with no changes in intrapulmonary shunt or cardiac output.

The resuscitation simulation (Dive A with 0.6 bar oxygen after 15 minutes with a normal cardiac output and a 70% shunt followed by 1 bar oxygen with IPPV and PEEP when a hospital is reached) did not change the risk compare to that shown in Table 1.

Table 1

Risk of DCS associated with Dive A. 18 m for 50 minutes (Within DCIEM no-stop limits). No oxygen for 15 minutes after surfacing.

	Normal	50% Shunt	70% Shunt
Normal	1.5%	2.5%	3.8%
70% Cardiac Output		3.5%	6.1%
50% Cardiac output	2.3%	4.9%	13.6%
30% Cardiac output	3.3%	9.3%	52.6%

Table 2

Risk of DCS associated with Dive A. 18 m for 50 minutes (Within DCIEM no-stop limits). No oxygen for 45 minutes after surfacing

	Normal	50% Shunt	70% Shunt
Normal	1.5%	3.0%	5.3%
50% Cardiac output	2.5%	7.4%	18.9%
30% Cardiac output	3.4%	14.2%	57.4%

Table 3

Risk of DCS associated with Dive B. 30 m for 15 minutes (Within DCIEM no-stop limits). No oxygen for 15 minutes.

	Normal	50% Shunt	70% Shunt
Normal	1.5%	1.9%	2.5%
50% Cardiac output	2.0%	3.3%	6%
30% Cardiac output	2.7%	5.7%	25.7%

Table 4

Risk of DCS associated with Dive B. 30 m for 15 minutes (Within DCIEM no-stop limits). No oxygen for 45 minutes.

	Normal	50% Shunt	70% Shunt
Normal	1.5%	1.9%	2.5%
50% Cardiac output	2.1%	3.4%	7.8%
30% Cardiac output	2.8%	6.3%	32.6%

Table 5

Risk of DCS associated with Dive C. 30 msw for 25 minutes (Within USN no-stop limits). No oxygen for 15 minutes.

	Normal	50% Shunt	70% Shunt
Normal	1.6%		3.1%
50% Cardiac output		3.9%	
30% Cardiac output	3.2%		35.2%

Table 6

Risk of DCS associated with Dive D. Multi-level 18 m for 50 minutes, followed by 12 m for 30 minutes and 6 m for 30 minutes. No oxygen for 15 minutes.

	Normal	50% Shunt	70% Shunt
Normal	1.3%		
50% Cardiac output		4.1%	9.6%
30% Cardiac output			48.2.7%

Table 7

Risk of DCS associated with Dive E. Multi-level 18 m dive for 50 minutes, followed by 12 m for 30 minutes and 6 m for 5 mintues. No oxygen for 45 minutes.

	Normal	50% Shunt	70% Shunt
Normal	1.3%		
50% Cardiac output		4.4%	11.9%
30% Cardiac output			52.0%

Discussion

Probability of DCS increased in all the near-drowning scenarios. The USN uses 2.3% risk from their LE1 model to define acceptable dive profiles.[17] This was exceeded in the majority of scenarios modelled. In the worst scenarios the risk of DCS increased to between 25 to 58%.

This model demonstrated that, in isolation, increasing either the shunt fraction or decreasing the cardiac output increase the DCS risk by similar amount. When these two are combined the increased DCS risk is greatly enhanced. There are few published measurements of these parameters following near-drowning in humans. However, animal data supports the range of values chosen in this study. Experimental models of near-drowning in pigs and dogs have shown increase in pulmonary shunt to near 70% and depression of cardiac output to 75%.[4,5] The combination of the 70% increase in shunt fraction and reduction of the cardiac output to 30% would be represented by a severely shocked patient or one who has responded to CPR following a cardiac arrest.

In the simulated dives the DCS risk incidence is greater in the shallower longer dives. These longer dive profiles would allow a greater uptake of inert gas in slower tissues resulting in a prolonged supersaturation and therefore

greater risk. However, extending the shallower dive into a multi-level dive did not greatly increase the risk following near-drowning. We have not systematically investigated the reasons for this.

During the resuscitation phase the increased DCS risk varied with the timing of oxygen administration. This effect was noted more in the longer shallower dive.

The early administration of oxygen attenuated the risk. This was consistent in all the dives modelled. This underlines the importance of early intervention with oxygen during first aid and resuscitation of diving accident victims.

Conclusion

This model demonstrates that in near-drowning:

1 there is an increased risk of decompression sickness;
2 the risk increases the longer the dive, particularly in square dive profiles;
3 this increased risk is reduced if oxygen is used early in resuscitation but may not decrease it below the USN chosen maximum risk of 2.3%;
4 recompression should be considered in all near-drowned divers;
5 a sudden decrease in cardiac output associated with hospital resuscitation (IPPV and PEEP) does not increase the already increased decompression sickness risk provided the patient's cardiac output was normal prior to resuscitation commencing;
6 in 18 m dives the risk does not change with a multi-level dive conducted in accordance with the DCIEM tables; and
7 all divers who are severely shocked at the initial presentation or who have been resuscitated from a cardiac arrest need recompression because the risk of DCS is between 25 and 58%. However, the optimal timing of recompression therapy has not been determined and will be the subject of a future study.

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