Estimates of $N_2$ narcosis and $O_2$ toxicity during submarine escapes from 600 to 1,000 fsw

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Abstract
The U.S. Navy recommends submarine escape for depths down to 600 fsw, with deeper escapes entailing the risks of decompression sickness, nitrogen ($N_2$) narcosis and CNS oxygen ($O_2$) toxicity. However, the escape equipment, including the submarine escape and immersion equipment and the escape trunk, could probably function even at 1,000 fsw. Here we report a theoretical analysis of the risks of both $N_2$ narcosis and CNS $O_2$ toxicity for different escape profiles from 600 to 1,000 fsw.

The effect of $N_2$ narcosis, calculated as a function of $N_2$ pressure in the brain using Gas Man® software, was expressed as equivalent narcosis depth (END), corresponding to the depth at which the same pressure of $N_2$ would be produced in the brain after five minutes of scuba diving with air. The risk of $O_2$-induced convulsions was estimated using the model developed by Arieli et al. Different dwell times (DTs) at maximal pressure in the escape trunk (from 0 to 60 s) and lungs-to-brain circulation times (10 to 30 s) were included in our analysis. When DT in the escape trunk is very short (e.g., 10 s), the risk of either incapacitating $N_2$ narcosis and/or $O_2$-induced convulsions occurring in the trunk is low, even during escapes from 1,000 fsw.

INTRODUCTION
In the U.S. Navy, submarine escape is currently recommended for depths down to 600 fsw (1). This depth limit corresponds to the maximal depths of three successful trial escapes performed at sea in 1970 from 176 m (keel depth: 182 m, 600 fsw) from HMS Osiris. On that occasion, the three escapers had “no difficulty carrying out the simple escape procedures” (2). In 1987, two submariners aboard HMS Otus escaped from a depth of 183 m, which is still the deepest submarine escape by humans ever recorded (Cf. 3).

On the other hand, in studies performed by Burgess and Hempleman in the 1970s with animals breathing oxygen, experimental escapes were successful from depths of both 300 m (984 fsw, with goats) and 400 m (1,320 fsw, with rats) (Cf. 2). In the U.S. Navy, deeper escape have been discouraged mainly because of concern for decompression sickness. However, two other risks associated with submarine escapes from depths greater than 600 fsw are nitrogen ($N_2$) narcosis and CNS oxygen ($O_2$) toxicity, both addressed here. An additional potential risk for these deep escapes is high-pressure nervous syndrome (HPNS), which occurs during diving to depths greater than 600 fsw (4). Nevertheless, the risks faced by submariners in such deep escapes may be acceptable if rescue cannot be undertaken before conditions in the disabled submarine become life-threatening. U.S. Navy submarines are equipped with submarine escape and immersion equipment (SEIE) that is rated to 1,000 fsw, and a submarine escape trunk, rated to 750 fsw. Therefore, it is reasonable to suppose that both of them could function for escapes deeper than 600 fsw, and possibly down to 1,000 fsw. Here we report a theoretical analysis of the risks of both $N_2$ narcosis and CNS $O_2$ toxicity that submariners could face during escapes from 600 to 1000 fsw.

METHODS
Depth-time profiles for escapes from a disabled submarine from depths ranging from 600 to 1,000 fsw were calculated, and examples of these profiles are shown in Figure 1. The initial curve of each depth-time profile describes the period of compression in the escape trunk. Sea water is allowed to enter and fill the
trunk, causing the air remaining within the trunk to be compressed to equilibrate with the water pressure outside. The pressure in the escape trunk rises exponentially; for practical reasons, here we use a pressure doubling time of 5 s (2). Once the pressure in the trunk has equalized with the pressure of the surrounding water, the submariner can open the escape hatch.

Correspondingly, there is a pressure plateau that here we call dwell time (DT). This is the length of time that a submariner must spend in the escape trunk at maximal pressure (that is, after pressurization has been completed), while the escape hatch is being opened. The profiles shown in Figure 1 are calculated with a DT of 10 s in the escape trunk, followed by ascent at 510 fsw/minute, which is fixed and determined by the SEIE (Cf. 5).

**Nitrogen Narcosis**

The effect of N₂ narcosis while within the escape trunk was calculated for escape profiles having DTs ranging from 10 to 60 s and for depths ranging from 600 to 1,000 fsw. Our analysis was performed using Gas Man® software (6; Appendix 1) in which the environment and the body are represented as a sequence of connected physiologic compartments, as shown in Figure 2. The most important of these physiological compartments is the vessel-rich group (VRG), which represents the combined properties of the well-perfused body tissues such as the brain, liver and kidneys.

The circulation time from the alveoli to the end organs, including the VRG, was allowed to vary from 0 to 30 s. The effect on consciousness of exposure to a narcotizing gas can then be estimated by determining the time course of its pressure within the VRG. Therefore, we calculated the pressure of N₂ present in the VRG for each escape profile. These N₂ pressures in the VRG, obtained with Gas Man®, were subsequently converted to an equivalent depth of sea water at which the same pressure of N₂ would be produced within the VRG after five minutes of scuba diving with air.

As N₂ is a relatively insoluble gas, five minutes of exposure is sufficient to come asymptotically close to equilibration. We performed this conversion because it is much easier to think of the magnitude of a narcotic effect when it is expressed as the depth of an equivalent air dive, rather than as an N₂ pressure within a body tissue. Therefore, we describe the narcotizing effect of N₂ that would be experienced by a submariner in the escape trunk as an equivalent narcosis depth (END) in fsw.

**Oxygen Toxicity**

The risk of CNS oxygen toxicity (in particular, convulsions) for different escape profiles was estimated using the model developed by Arieli et al. (7; Appendix 2). Their model describes a conversion from a time-course of partial pressure of oxygen into a probability variable \( z \), such that:

\[
    z = \frac{1}{\sigma} \left( \ln \left[ \frac{1}{\sigma} \left( \frac{P_{O_2}(t)}{101.3} \right)^{\sqrt{2}} \right] - 0.5 \ln K_c \right) \tag{1}
\]

from which the probability of oxygen toxicity from this time-course of exposure, \( P_{tox} \), is given by:

\[
    P_{tox} = \Phi(z) = \frac{1}{2} \left( 1 + \frac{\text{erf}(z)}{\sqrt{2}} \right) \tag{2}
\]

where \( \Phi \) is the standard normal cumulative distribution function and:

\[
    \text{erf}(z) = \frac{2}{\sqrt{\pi}} \int_0^z e^{-t^2} \, dt \tag{3}
\]

The appropriate values of the model parameters \( \sigma, c \) and \( K_c \) as used in Appendix 2 were derived by Arieli et al. (7) by fitting against toxicity risk from 100% O₂ as measured both at lower pressures, in the onset of symptoms of oxygen toxicity in human divers, and also at high pressures, in time to first seizure in anesthetized rodents. Variable DTs are introduced into this calculation by appropriately lengthening or shortening the time of exposure to the maximum pressure of oxygen in the time-course of the pressure \( P_{O_2}(t) \).

**RESULTS**

Table 1A shows ENDs for escapes ranging from 600 to 1,000 fsw, with different DTs in the escape trunk (0 s: end of compression; 10, 20, 30, 45 and 60 s, but no circulatory delay from lungs to the brain).

Tables 1B to 1D show ENDs with the same range of DTs but different circulation times, respectively 10, 20 and 30 s. For example, according to our calculations, a submariner with a circulation time of 10 s, leaving
the escape trunk after a DT of 10 seconds at 600 fsw, would be experiencing a level of N2 narcosis equivalent to a five-minute air dive to 139 fsw (Table 1B).

Figure 3 shows the probability of CNS oxygen toxicity (%) before leaving the trunk as a function of both depth and DT in the escape trunk. Figure 4 shows the overall probability of CNS oxygen toxicity (%) during escape, including ascent at 510 fsw/minute, as a function of both depth and DT in the escape trunk.

**DISCUSSION**

Here we report estimates of risk of both N2 narcosis and CNS O2 toxicity during submarine escapes from depths ranging from 600 to 1000 fsw, with DTs in the escape trunk varying from 0 to 60 s and lungs-to-brain circulation times ranging from 0 to 30 s. Our main finding is that, when the DT in the submarine escape trunk is very short, the risk of either incapacitating N2 narcosis and/or O2-induced convulsions occurring in the trunk is low, even during escapes from 1,000 fsw.

For example, with a DT of 10 s at this depth, the probability of convulsions is less than 1% (see Figure 3); with regard to N2 narcosis, the escaping submariner would probably feel as he were scuba diving at a depth of about 230 fsw (Table 1B). This END is based on a 10 s circulation time from the lungs to the brain, which is 3.7 seconds shorter than the average circulation time from the pulmonary gas-exchange region to central chemoreceptor areas, estimated by Bellville et al. in seven normal subjects (8).

The circulation time in escapers is likely to be shortened due to hyperdynamic cardiac function secondary to anxiety. However, hyperoxia in the escape trunk after pressurization may lead to a prolonged circulation time to the brain because of cerebral vasoconstriction.

The design of the escape trunk provides room for two submariners, with only one at a time being able to leave through the upper escape hatch. We found that the predicted likelihood of N2 narcosis and CNS O2 toxicity increases rapidly with DT in the escape trunk. Consequently, these problems are more likely to be experienced by a second escaper, as his DT will necessarily be longer than that of the first escaper.

Our calculations do not take into account some important physiological conditions that may alter escapers’ susceptibility to both N2 narcosis and CNS O2 toxicity, and may either worsen or improve their performance. Such conditions include hypercapnia during pressurization and hypocapnia during ascent, secondary to major changes in alveolar ventilation (9); possible CNS interactions between increased N2 and O2 pressures (10) and even HPNS during escapes from depths greater than 600 fsw (4). Therefore, animal and human experimental escapes from depths down to 1,000 fsw should be conducted prior to recommending changes in current submarine escape guidelines.

In summary, if single- or two-man escapes are performed with a DT of 10 s, it is unlikely that the escapers will face either incapacitating N2 narcosis and/or O2-induced convulsions in the trunk. On the other hand, because of the hemodynamic delay due to circulation time from the lungs to the brain, these problems may occur during ascent in the water, when escapers do not have to perform any essential tasks (except for keeping their glottis open, which does not appear to be a problem, at least in animals convulsing during simulated escapes; Cf. 11) and drowning should be prevented by the SEIE.

**ACKNOWLEDGMENTS**

This study was supported by a grant from the Naval Sea System Commands.

**References**


FIGURE 1: Depth-time profiles of escapes from depths ranging from 600 to 1,000 fsw, with a DT of 10 s.
**FIGURE 2:** Physiological compartments used to represent the uptake of N₂ from the environment into the body.

**FIGURE 3:** Probability (%) of O₂-induced convulsions with different DTs (listed in the small box), before leaving the escape trunk. This probability is dependent on depth of escape and DT.
FIGURE 4: Overall probability of oxygen-induced convulsions (%) during escapes, including ascent, with different DTs (listed in the small box) in the escape trunk. This probability is dependent on depth of escape, DT and ascent rate (assumed to be 510 fsw/min).

TABLE 1A: Equivalent narcosis depths (fsw) with no circulatory delay and DTs varying from 0 to 60 s. TET: total escape time.

<table>
<thead>
<tr>
<th>Depth of escape (fsw)</th>
<th>End Compression DT: 0 s TET: 25 s</th>
<th>DT: 10 s TET: 35 s</th>
<th>DT: 20 s TET: 45 s</th>
<th>DT: 30 s TET: 55 s</th>
<th>DT: 45 s TET: 70 s</th>
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### TABLE 1B: Equivalent narcosis depths (fsw) with a circulatory delay of 10 s and DTs varying from 0 to 60 s. TET: total escape time.

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<th>Depth of escape (fsw)</th>
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<th>DT: 20 s TET: 45 s</th>
<th>DT: 30 s TET: 55 s</th>
<th>DT: 45 s TET: 70 s</th>
<th>DT: 60 s TET: 85 s</th>
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### TABLE 1C: Equivalent narcosis depths (fsw) with a circulatory delay of 20 s and DTs varying from 0 to 60 s. TET: total escape time.

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**APPENDIX 1**

Gas Man® is a computer program available from the nonprofit corporation:
Med Man Simulations, Inc.,
P.O. Box 67-160, Chestnut Hill MA 02467,
Tel: (617) 277-2117, gasman@gasmanweb.com,

Gas Man® is a physiologically-based model of inhaled anesthetic uptake and distribution. It is a commercially available computer program used for education, and it can accurately predict expired anesthetic concentrations during induction and emergence from anesthesia.

Gas Man® makes several assumptions. Inhaled anesthetic kinetics may be described with a flow-limited 4-compartment mammillary model (lungs, VRG, muscle group [MG], fat group [FG]) attached to another compartment, the breathing circuit. Each compartment equilibrates instantly with the anesthetic brought to it, and, except for the concentration effect, the equilibration follows first-order kinetics.

Solubility in blood and tissues, gas and blood flows, and compartment volumes determine the rate of equilibration. The model allows the user to fix the essential elements: anesthetic solubility in blood and tissues, gas and blood flows, and tissue volumes. It does not correct for inter-tissue diffusion of anesthetics, anesthetic metabolism, or ventilation/perfusion abnormalities. The particular (Euler) integration used stabilizes the behavior of the model under extreme conditions of fresh gas flow, ventilation and cardiac output.

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**TABLE 1D:** Equivalent narcosis depths (fsw) with circulatory delay of 30 s and DTs varying from 0 to 60 s.

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APPENDIX 2

The following code, written in MATLAB implements the described oxygen toxicity model.

```matlab
depth=(600:20:1000);
dwell=(60:-5:5);

p=zeros(length(depth),length(dwell));
m='';

for j=1:length(dwell)
    for i=1:length(depth)
        p(i,j)=pOXTox(makeProfile(depth(i),5,dwell(j),510));
    end
    m= strvcat(m, sprintf('%i seconds', dwell(j)));
end

plot(depth,p);
legend(m,2);
xlabel('Escape depth (fsw)')
ylabel('Probability of CNS O_2 toxicity (%)')
title('CNS O_2 toxicity, dependent on escape depth and dwell time, assuming 510 ft min^-^ vortex ascent rate');
grid on;

function p=pOXTox(profile)
% Returns percentage probability of an oxygen toxicity event for the given
% ascent profile.

profile=[profile fswToPO2(profile(:,2))];
sigma=2.02; c=6.8; Rc=2.31e8;

rk=trapz(profile(:,1), (profile(:,3)/101.3).^(c/2));
z=(log(rk)-0.5*log(Rc))/sigma;
p=100*normcdf(z);

function profile=makeProfile(depth,a,dwell,r)
% depth: depth from which escape takes place
% a: pressure doubling time for trunk compression in seconds
% dwell: time in seconds between end of compression and starting ascent
% r: rate of ascent in feet/min

rhoSW=1027; g=9.81; one_foot=0.3048;
f=0:20:depth; if f(end)<depth, f=[f depth]; end

t=zeros(size(f));

for i=1:length(f)
    t(i)=(a/(60*log(2)))*log(1+(rhoSW*g*one_foot*f(i))/101.3e3);
end
astart=t(end)+(dwell/60);
profile=[t' f'; astart+(depth-flipud(f'))/r flipud(f')];

function PO2=fswToPO2(fsw)
% For a given depth of sea water, returns the PO2 in kPa.

FlO2=0.21; rhoSW=1027; g=9.81; one_foot=0.3048;
PO2 = FlO2 * (101.3 + (rhoSW*g*one_foot*fsw)/1000);
```