Empirical Models For Use  
In Designing Decompression Procedures for Space Operations

Johnny Conkin, Benjamin F. Edwards,  
James M. Waligora, and David J. Horrigan, Jr.

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EMPIRICAL MODELS FOR USE IN DESIGNING
DECOMPRESSION PROCEDURES FOR SPACE OPERATIONS

ABSTRACT

Empirical models for predicting the incidence of Type I altitude decompression sickness (DCS) and venous gas emboli (VGE) during space extravehicular activity (EVA), and for use in designing safe denitrogenation-decompression procedures are developed. The parameters of the models are estimated using DCS and VGE incidence data from 26 NASA and USAF manned altitude chamber decompression tests involving 607 male and female subject tests.

These models, and procedures for their use, consist of (1) an exponential relaxation model and procedure for computing tissue nitrogen partial pressure resulting from a specified prebreathing and stepped decompression sequence; (2) a formula for calculating Tissue Ratio (TR), a tissue decompression stress index; (3) linear and Hill equation models for predicting the total incidence of VGE and DCS attendant with a particular TR; (4) graphs of cumulative DCS and VGE incidence (risk) versus EVA exposure time at any specified TR; and (5) two equations for calculating the average delay period (latency time) for the initial detection of VGE or indication of Type I DCS in a group after a specific denitrogenation-decompression procedure. Several examples of realistic EVA preparations are provided to demonstrate the use of the predictive models and interpretation of the results.

INTRODUCTION

This report documents empirical models based on NASA and USAF altitude chamber experiments for predicting the incidence (risk) of Type I altitude decompression sickness (DCS) and venous gas emboli (VGE) during space EVA operations, and for use in designing safe denitrogenation-decompression procedures consistent with an acceptable level of DCS and VGE risk. The models enable predictions, with a quantitative level of confidence and accuracy of the incidence of DCS and VGE to be expected within a group as a result of a particular prebreathe-decompression procedure.

Earlier guidelines for predicting DCS were based on data collected from a literature survey on past altitude DCS research. No data on VGE are available from those reports since the instrumentation for in vivo gas phase detection had not been developed. In addition, the type, intensity, duration of exercise, and altitude exposure time in those studies were not consistent with NASA's current EVA procedures.

The mathematical models described consist of (1) a time-dependent exponential model for computing tissue nitrogen partial pressure under stepped prebreathe and decompression conditions, (2) a formula for computing tissue nitrogen decompression stress or Tissue Ratio (TR), and (3) empirical linear and Hill equation models for predicting the incidence of Type I DCS, VGE, and Type I DCS Grade 3 (symptoms that forced early test termination or that reappeared after the test).
The predictive models are obtained by mathematically fitting the linear and Hill equations to the paired TR and % DCS or % VGE data obtained from 26 unique NASA and USAF manned altitude chamber tests that simulated EVA work profiles. In total, 607 male and female exposures were performed from 1982 to 1986 under controlled conditions at the Johnson Space Center, Houston, Texas, and Brooks Air Force Base, San Antonio, Texas.

DEVELOPMENT OF TISSUE DECOMPRESSION STRESS (TISSUE RATIO) MODEL

Nitrogen Uptake and Washout Exponential Model

All body tissues in equilibrium with ambient nitrogen will respond to increased or decreased nitrogen partial pressure in the breathing medium by absorbing or eliminating the gas that is dissolved in tissues. The rate of elimination/absorption is determined by the rate constant of the tissue in question and the nitrogen partial pressure difference between the tissue and ambient nitrogen, i.e.,

\[ \frac{dP}{dt} = k \left( P_a - P \right) \]  

where

\[ \frac{dP}{dt} = \text{time rate of change of tissue nitrogen partial pressure}, \]

\[ P = \text{tissue nitrogen partial pressure at any time } t, \]

\[ P_a = \text{ambient nitrogen partial pressure}, \]

\[ k = \text{tissue nitrogen partial pressure rate constant}. \]

The rate constant (k) of a given tissue is physiologically determined by the blood perfusion rate through the tissue, the diffusion rate of the inert gas through the tissue, and the solubility characteristics of the gas in the specific tissue.

The tissue rate constant (k) is related to the tissue nitrogen half-time (t_{1/2}) through the equation,

\[ k = \frac{(\ln 2)}{t_{1/2}} = .693/t_{1/2} \]

where

\[ t_{1/2} = \text{tissue nitrogen partial pressure half-time} \]
The body can be characterized by a spectrum of theoretical tissue types that represent "fast" and "slow" tissues. A theoretical tissue type is characterized by the tissue half-time (t₁/₂): the time required for a tissue to respond to a change in ambient nitrogen partial pressure by giving off or absorbing nitrogen until the initial difference between the tissue nitrogen partial pressure and ambient nitrogen partial pressure is reduced by one-half.

Since t₁/₂ characterizes the nitrogen perfusion-diffusion properties of the tissue, k is also a characteristic of the tissue-nitrogen relationship. It can be seen from equation 1 that, for a given (Pₐ - P) difference, a tissue having a greater k-value will absorb or eliminate nitrogen more rapidly than one with a lesser k-value. A "fast" tissue such as blood may have a half-time of only a few minutes while a "slow" tissue half-time may be several hours.

Following a rapid change in nitrogen partial pressure in the breathing medium, the nitrogen partial pressure which is reached in a designated "tissue type" after a specific time is described by equation 2, the solution of equation 1. This equation describes either nitrogen uptake or elimination in a specific theoretical tissue type depending on the initial tissue nitrogen partial pressure and the partial pressure of nitrogen in the ambient breathing mixture.

\[
P_t = P_o + (P_a - P_o) \left(1 - e^{-kt}\right)
\]

where

\[
P_t = \text{the nitrogen partial pressure in the tissue after exposure for } t \text{ minutes},
\]

\[
P_o = \text{initial tissue nitrogen partial pressure},
\]

\[
P_a = \text{ambient nitrogen partial pressure in breathing medium},
\]

\[
e = \text{base of natural logarithm},
\]

\[
t = \text{exposure time in minutes, and}
\]

\[
k = \text{tissue nitrogen partial pressure rate constant}.
\]

The exponential decay (relaxation) model (eq.2) is generally used to describe the final inert gas (in this case nitrogen) partial pressure within a specific theoretical body tissue type after a particular prebreathe procedure. Examples of its use are found in references 1 and 2.

Nitrogen Supersaturation and the Tissue Ratio Concept

Tissue nitrogen supersaturation can be defined in two ways. The second definition is used in this report in constructing models that predict the incidence of DCS and VGE.
A person breathes 100% oxygen from a mask. This procedure is intended to reduce the total dissolved nitrogen in the body by eliminating the nitrogen concentration in the breathing mixture. This allows the body to “wash” nitrogen from its tissues through perfusion and gaseous diffusion processes. The tissues are saturated with nitrogen in relation to the breathing medium and nitrogen diffuses from the tissues to the lungs due to an imbalance in the nitrogen partial pressure. No DCS or VGE result from this tissue nitrogen saturation since ambient pressure is not reduced. The nitrogen remains dissolved in tissues and body fluids.

Supersaturation exists when a system is in a metastable state and there is a potential for spontaneous change in phase of some constituent. This can occur when a stable equilibrium state is disturbed by a change in pressure, volume, temperature, or chemical constituent. Decompression (decrease in ambient pressure) as experienced by divers or aviators produces a transient metastable state in the body tissues such that the rate of normal, random micronucleation events increase as well as the size of the gas nuclei. Two definitions of tissue nitrogen supersaturation are presented.

(1) A tissue is supersaturated with dissolved nitrogen when the nitrogen partial pressure in the tissue is greater than ambient pressure.

With this definition of tissue nitrogen supersaturation, ambient pressure is reduced to a point where it is less than the partial pressure of nitrogen in a particular tissue. The tissue is not said to be supersaturated until the ambient pressure is less than the nitrogen partial pressure in the tissue. This implies and assumes that no DCS symptoms or VGE production will result as long as the decrease in ambient pressure is not greater than the nitrogen partial pressure in a particular tissue, and that nitrogen is the only gas that needs to be considered in nucleation events.

(2) A tissue is supersaturated with dissolved nitrogen and has the potential to form a gas phase whenever ambient pressure is reduced.

This implies that the potential to form gas emboli exist whenever ambient pressure is reduced regardless of the magnitude of pressure reduction. This definition does not assume that a finite degree of decompression can be tolerated. It assumes that all dissolved gases are potentially involved in nucleation events.

It is intuitively evident that the greater the reduction in ambient pressure, the greater the potential for decompression sickness. It is possible to establish an index of decompression stress that relates tissue nitrogen partial pressure and ambient pressure changes by developing the concept of Tissue Ratio.

The Tissue Ratio (TR) concept, a measure of tissue nitrogen supersaturation or decompression stress, was developed by Haldane for divers and later modified for aviators. He observed that the human body could be rapidly decompressed from 2 to 1 atmosphere or from sea level (1 atmosphere) to approximately 20,000 feet (1/2 atmosphere) without symptoms of decompression sickness. Thus, it appeared to be safe to decompress aviators on a schedule which never allowed the total gas partial pressure in the tissues to exceed twice the ambient atmospheric pressure, a TR of 2.0. These observations implied that the body could tolerate a degree of nitrogen supersaturation without overt manifestations of decompression sickness.
Haldane's concept has been modified over the years as a more complete understanding of tissue gas exchange dynamics has been achieved and ultrasonic detection equipment has been developed to identify venous gas emboli (VGE) in situ. Using this technology, VGE have been detected in experimental human subjects decompressed to a pressure equivalent of 13,120 feet (Haldane's TR = 1.65) and in animals decompressed to only 6,560 feet (Haldane's TR = 1.26)\(^{13}\). Type I DCS symptoms have been reported from as little as 12,000 feet (Haldane's TR = 1.57)\(^{19}\). Thus, it is clear that Haldane TR's of less than 2.0 can evolve nitrogen bubbles (VGE), and produce signs and symptoms of decompression sickness.

The calculation of the TR in equation 3 for a tissue of specific half-time uses the final tissue nitrogen partial pressure from one or more sequential applications of equation 2.

\[
\text{TR} = \frac{\text{Final Tissue } p_{N_2} \text{ (abs)}}{\text{Ambient Pressure (abs)}}
\]

The TR calculation used in equation 3 is different from Haldane's TR in that the numerator is the partial pressure of dissolved nitrogen in the tissues prior to decompression rather than the total pressure. Initial equilibrium tissue \(p_{N_2}\) is taken to be ambient nitrogen \(p_{N_2}\) at 14.7 psia \((P_a = 11.6 \text{ psia})\).

The resulting TR value for any theoretical tissue half-time type can be expressed as a single number and is unique for a particular prebreathe procedure regardless of the complexity of that procedure. This TR is associated (correlated) with the incidence of VGE and/or DCS occurring during the subsequent decompression to establish a statistical relationship between a particular prebreathe procedure and the resulting VGE and DCS at that altitude.

The potential to form a gas phase exists whenever ambient pressure is reduced. The second definition of tissue nitrogen supersaturation is therefore used in this report. Under equilibrium conditions at sea level pressure and gas composition the TR that represents no potential for DCS symptoms or VGE production is \(11.6/14.7 = 0.78\). Any reduction in ambient pressure will increase this TR if nitrogen washout procedures have not been initiated prior to the decompression.

**Example of Equation 2 and Tissue Ratio Application**

A simple example will provide a better understanding of how the exponential decay model and TR are used in the eventual development of the equations that predict DCS and VGE incidence in a population that performs a specific prebreathe procedure.

In this example, a person performs a 4.0 hour pure oxygen prebreathe while at 14.7 psia. Since no other prebreathe procedure preceded this event, all body tissues are initially in equilibrium with the ambient nitrogen partial pressure, \(P_a = 11.6 \text{ psia}\) at the onset of the prebreathe procedure. Alveolar nitrogen partial pressure \((P_{alv} = 11.0 \text{ psia})\) is not used as the initial equilibrium tissue nitrogen partial pressure to avoid the added complexity of employing the Alveolar Oxygen Equation to estimate alveolar nitrogen partial pressure during intermediate altitude exposures where breathing gas composition may have been modified. The final nitrogen partial pressure \((P_f)\) in any theoretical
tissue type is calculated with equation 2. Ambient nitrogen partial pressure is used to achieve operational simplicity in the predictive models.

In this example the 360-minute half-time theoretical tissue type is selected. The initial equilibrium tissue nitrogen partial pressure \( P_o \) is 11.6 psia. The prebreathe medium (pure oxygen) contains no nitrogen so the pressure difference \( P_a - P_o \) for the removal of nitrogen is maximal. How rapidly the nitrogen is removed from the tissue by this pressure difference from the 360-minute half-time theoretical tissue type is determined by \( k \), the tissue rate constant in equation 2.

After 4.0 hours of breathing pure oxygen, a final tissue \( pN_2 (P_f) \) for the 360-minute half-time tissue type is 7.3 psia. In theory, this individual can now decompress from 14.7 psia to 7.3 psia (18,000 feet) without supersaturating this theoretical tissue type. If, however, this individual is decompressed to 4.3 psia the 360-minute TR becomes

\[
TR = \frac{7.3 \text{ psia}}{4.3 \text{ psia}} = 1.70
\]

If a large group was exposed to 4.3 psia after a 4.0-hour oxygen prebreathe, the true incidence of DCS and VGE would eventually be expressed. We can now pair a theoretically derived TR based on a specific prebreathe procedure with an experimentally determined percentage of VGE or DCS to obtain a data point.

A different prebreathe procedure, analyzed in the same way, using the 360-minute theoretical half-time tissue type, will produce a different TR. This TR matched with VGE or DCS incidence obtained from testing the new decompression sickness prevention procedure provides another data point. If enough procedures are tested, providing a range of TRs for a specific theoretical half-time tissue, an equation can be fitted to the data points thus forming the basis for predicting the outcome of any prebreathe procedure where a TR is calculated. In this way, the TR serves as an empirical value associated with some DCS and VGE incidence rather than an absolute value that must not be exceeded.

**DEVELOPMENT OF THE % DCS AND % VGE PREDICTIVE EQUATIONS**

**Basic Approach**

To develop the predictive equations it is necessary to determine the theoretical half-time tissue type that provides the most precise estimator of subsequent DCS and/or VGE incidence. This is accomplished by accepting the theoretical half-time tissue type that provides the greatest correlation coefficient after a linear regression is fitted through the DCS or VGE incidence data paired to the calculated TRs from a selected theoretical half-time tissue type. A FORTRAN program* that calculates

*B. F. Edwards, Technology Inc., Houston, Texas, 1986*
the TRs for a spectrum of theoretical half-time tissue types is available to facilitate this process. It pairs the TR data with the actual DCS and VGE incidence produced under specific prebreathe procedures, and performs a linear regression and correlation coefficient analysis of the data. The 26 prebreathe procedures evaluated by this analysis are outlined in Appendix A. The DCS and VGE incidence results from testing the prebreathe procedures are in table 1.

When a column of TRs from any theoretical half-time tissue type is plotted against DCS or VGE incidence, the resulting scattergram could indicate a nonlinear relationship between the TRs and the % VGE or % DCS data. To develop a mathematical model relating the incidence of VGE and DCS to TR two questions had to be answered: (1) What is the best tissue half-time for calculating TRs?, and (2) What is the best functional form for the mathematical model? Analysis in the next two sections of this report address the questions.

**Fitting Linear Equations to % DCS and % VGE Data**

The first step in developing the predictive equations was to fit a linear function to DCS and VGE incidence data paired to calculated TRs from selected theoretical half-time tissue types. A measure of the "goodness of fit" of the function to the data was provided by the correlation coefficient for the regression. An analysis of the correlation data showed that the linear function (straight line) through the data provided an acceptable mathematical fit to the data. Figure 1 shows the correlation coefficients (r) derived from a linear analysis plotted against the spectrum of theoretical half-time tissue types covering from 60 to 960 minutes.

The point on the curve that indicates the maximum correlation between the % VGE or % DCS and the TR data was used to identify the theoretical half-time tissue type that could be used in predictive equations. Table 1 shows the theoretical half-time tissue types, and their TR's paired with actual % DCS and % VGE data. The 360-minute theoretical half-time tissue type TRs show the best linear correlation to the % VGE data, while the 480-minute theoretical half-time tissue type TRs show the best linear correlation to the % DCS data.

In the operational use of the models, both DCS and VGE TRs are standardized on the 360-minute theoretical half-time tissue type. The predictive linear equations obtained by regressing experimental DCS and VGE incidence onto computed TR's, standardized on the 360-minute theoretical half-time tissue are

\[
\begin{align*}
\% \text{VGE} &= 95.5 \times \text{Tissue Ratio (360')} - 101.1 \\
\% \text{DCS} &= 57.9 \times \text{Tissue Ratio (360')} - 74.1
\end{align*}
\]

The predictions from equations 4 and 5 are based on EVA's up to 6.0 hours in duration. Since exposure time at a final suit pressure is a critical factor in determining DCS or VGE outcome, these equations are useful in predicting results from EVA's of 3.0 to 6.0 hours in duration. The section entitled "Time to Onset of DCS and VGE During Simulated EVA" addresses DCS and VGE incidence as a function of EVA exposure time.
TABLE 1. % DCS AND % VGE DATA AND CORRESPONDING TISSUE RATIO VALUES COMPUTED AT THE OPTIMUM THEORETICAL TISSUE NITROGEN HALF-TIME FOR 34 ALTITUDE DECOMPRESSION EXPERIMENTS

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N = 607

*See Appendix A for description of each Prebreathe Procedure.
Figure 1. - Plots of the linear correlation coefficient versus specific theoretical half-time tissue types for % DCS and % VGE data.
Equations 4 and 5 were derived from a subset of the data from table 1. Not all the data on \% DCS and \% VGE contained in table 1 were used to develop these equations. The reason for excluding some data is that prebreathe procedures that provided complete bends protection also produced zero incidence of DCS and VGE. TRs derived from an analysis of these prebreathe procedures are also very low. As a result, incorporating these zero \% DCS and \% VGE data will artificially shift the slope of the linear equation. Excluding some zero data eliminates shifting the slope of the line to include prebreathe conditions that were extremely conservative. If some zero incidence data points are excluded, the predictive equations provide a better data fit.

The excluded zero data consisted of those zero percent DCS and VGE data points that had 360-min-ute theoretical half-time TRs that were less than the TR that first produced some DCS or VGE incidence. Using this method, the \% DCS data set included 26 data points and the \% VGE data consisted of 31 points (see table 1).

Figures 2, 3 and 4 show the linear plots of \% DCS and \% VGE against the TRs calculated from the 360-minute theoretical half-time tissue type. The 95\% confidence band for figures 2 and 3 is included to quantify the variability in the DCS and VGE data.

These equations can predict the best estimate of DCS or VGE incidence within specified confidence limits. The \% DCS linear regression indicates a zero incidence of Type I DCS when a TR of 1.28 is achieved. The \% VGE linear regression indicates a zero incidence of VGE when a TR of 1.06 is achieved. However; the incidence of DCS and VGE in figures 2 and 3 indicate that the incidence of DCS and VGE at these TRs can be greater than zero. To more accurately describe the relationship between TR and \% DCS and \% VGE when the TR is very low (<1.2) or very high (>1.8) the Hill equation is employed.

**Fitting the Hill Equation to \% DCS and \% VGE Data**

Since the relationship of both the incidence of DCS and VGE to TR are bounded at 0\% and 100\%, a model having sigmoidal characteristics is suggested. The two parameter Hill equation provides a model for such a sigmoidal relationship. The Hill equation, a probabilistic dose-response function, is frequently used in biology and pharmacology to quantify the dose-response behavior of subjects to some stress, medication, or treatment. The Hill equation has the form

\[
P = \frac{D^n}{D^n + D_{50}^n}
\]  

(6)

where \( D \) is the dose, \( P \) is the probability of a positive response in a dichotomous (yes/no) outcome set, and \( D_{50} \) is the dose at which \( P \) equals 0.5 (50\%). The exponent \( n \) and \( D_{50} \) are parameters to be estimated from the experimental data. Equation 6 is sigmoidal in shape, having a value of zero for zero dose, a value of 0.5 at \( D_{50} \) and, approaches unity as the certainty of a positive response increases with increasing dosage. The exponent \( n \), the order of the Hill equation, controls the slope of the central portion of the curve as it passes through the 50\% point.
Based on 607 man decompressions simulating EVA activity

\[ \% \text{DCS} = 57.9 \times \text{Tissue Ratio (360')} - 74.1 \quad r = 0.78 \quad n = 26 \]

Figure 2: Linear plot of DCS incidence using 360-minute theoretical half-time tissue and its Tissue Ratios with the 95% confidence interval.
Figure 3.- Linear plot of VGE incidence using 360-minute theoretical half-time tissue and its Tissue Ratios with the 95\% confidence interval.

\[ \% \text{VGE} = 95.5 \times \text{Tissue Ratio (360')} - 101.1 \quad r = 0.88 \quad n = 31 \]
% Symptoms Resulting in Test Termination plus Delayed Symptoms = 10.5 * Tissue Ratio (360') - 13.1  \( r = .32 \)  \( n = 15 \)

Figure 4 - Linear plot of DCS symptoms that resulted in test termination or symptoms that reoccurred after the test.
For probabilistic modeling of DCS, VGE, or the more serious DCS symptoms where TR quantifies the dose, the Hill equation is modified to reflect an incidence probability of zero at a TR of 0.78. This is required since the original definition of TR developed in equation 3 calculates a TR of 0.78 under equilibrium conditions at sea level. A TR of 0.78 becomes the starting-point or base line from which to construct the Hill equation. Therefore, the more appropriate form of the Hill equation in this application is

\[ P = \frac{(TR - .78)^n}{\left[ (TR - .78)^n + (TR_{50} - .78)^n \right]} \]  

(7)

The parameter \( n \) and \( TR_{50} \), and the correlation coefficient \( r \) of the fit are estimated using the experimental data set \( \{(TR, P)\} \) found in table 1, and a proprietary non-linear estimation program\(^{14}\). Unlike the zero exclusion procedure used in the linear regressions, the Hill equation utilizes all data from table 1.

When fitted to the experimental data the Hill equations for predicting \% VGE, \% DCS, and \% DCS Refractory Symptoms are

\[ \% VGE = \frac{(TR - .78)^{3.08}}{\left[ (TR - .78)^{3.08} + 0.47 \right]} \times 100 \quad r = .91 \]  

(8)

\[ \% DCS = \frac{(TR - .78)^{4.24}}{\left[ (TR - .78)^{4.24} + 2.16 \right]} \times 100 \quad r = .84 \]  

(9)

and

\[ \% \text{Refractory Symptoms} = \frac{(TR - .78)^{2.50}}{\left[ (TR - .78)^{2.50} + 17.61 \right]} \times 100 \quad r = .53 \]  

(10)

where TR is the Tissue Ratio for the 360-minute theoretical half-time tissue, and \% Refractory Symptoms (Grade 3) are those DCS symptoms that resulted in test termination or symptoms that reoccurred after the test.

Figures 5, 6 and 7 display the Hill equations with the experimental data points and the 95% confidence interval. Figure 8 shows a composite of the three curves. Figures 9 and 10 show each curve without the 95% confidence interval. These curves, in addition to the curve in figure 7, are currently the accepted predictors of DCS and VGE incidence based on all available data that can be applied to current Shuttle EVA operations.
Hill's Equation:
% DCS = (TR - .78)^4.24 / ( (TR - .78)^4.24 + 2.16 ) * 100

r = 0.84  n = 34

Based on 607 human decompressors with EVA work simulation

Figure 5.- Hill plot of DCS incidence using 360-minute theoretical half-time tissue and its Tissue Ratios with the 95% confidence interval.
Hill's Equation:

\[ \text{% VGE} = \frac{(\text{TR} - .78)^{3.08}}{[(\text{TR} - .78)^{3.08} + .47] * 100} \]

\[ r = 0.91 \quad n = 34 \]

Based on 607 human decompressions with EVA work simulation.

Figure 6: Hill plot of VGE incidence using 360-minute theoretical half-time tissue and its Tissue Ratios with the 95% confidence interval.
Hill's Equation:
\[ \text{Symptoms} = \left( T_R - .78 \right)^{2.50} \frac{\left( \left( T_R - .78 \right)^{2.50} + 17.61 \right)}{100} \]

\[ r = 0.53 \quad n = 31 \]

Based on 568 human decompressions with EVA work simulation

Figure 7: Hill plot of DCS symptoms that resulted in test termination or symptoms that reoccurred after the test.
Figure 8. - Hill plot of DCS and VGE incidence using 360-minute theoretical half-time tissue and its Tissue Ratios.
Hill's Equation:

\[
\% \text{ DCS} = \frac{(\text{TR} - .78)^{4.24}}{((\text{TR} - .78)^{4.24} + 2.16)}
\]

\[r = 0.84 \quad n = 34\]

Based on 607 human decompressions with EVA work simulation

Figure 9. - Hill plot of DCS incidence using 360-minute theoretical half-time tissue and its Tissue Ratios.
Hill's Equation:
\[ \% \text{VGE} = \frac{(\text{TR} - .78)^{3.08}}{[(\text{TR} - .78)^{3.08} + .47]} \times 100 \]
\[ r = 0.91 \quad n = 34 \]

Based on 607 human decompressions with EVA work simulation

Figure 10.- Hill plot of VGE incidence using 360-minute theoretical half-time tissue and its Tissue Ratios.
Confidence Intervals for the Predictive Linear and Hill Equations

Associated with each prediction of % DCS or % VGE is a degree of variability related to the scatter of real data about the linear regression line. A measure of this scatter is the error variance for the regression. From Ostle\(^5\), the error variance \(s_e^2\) is given by

\[
s_e^2 = \frac{1}{n-2} \sum (Y - \hat{Y})^2
\]

where \(Y\) is the measured DCS or VGE percentage value, \(\hat{Y}\) is the predicted value (a function of the TR and provided by the predictive model), and \(n\) is the number of data points in the real data set.

The estimated variance \(s_y^2\) for a predicted group outcome for a TR value \(x\) is

\[
s_y^2 = s_e^2 \left[ 1 + (1/n) + \left( x - \bar{x} \right)^2 / \left( x_i - x \right)^2 \right]
\]

where the \(x_i\) are the TR data set, and \(\bar{x}\) is the mean TR for the data set.

Finally the confidence interval (CI) for the DCS or VGE incidence estimate at TR \(x\) is given by

\[
CI = \hat{Y} \pm t_{(1 - \alpha/2)} \left( \frac{s_y}{\sqrt{n}} \right)
\]

where \(t\) is the specified t-statistic, and \((1 - \alpha)\) is the desired level of confidence, i.e., 95%.

For an example, the linear equation 4 predicts a 42.0% incidence of VGE for a TR of 1.50 calculated from the 360 minute theoretical half-time tissue (fig. 3). The 95% confidence interval calculated for this prediction is 42.0% \(\pm\) 20.0%. Since the true VGE incidence at a TR of 1.50 is a fixed value rather than a random variable, an interpretation of the confidence interval is that one can be 95% certain that it will contain the true VGE incidence. Alternately, 95% of the confidence intervals computed from similar data samples will contain the true VGE incidence.

Equation 5 predicts a 13.0% incidence of DCS with a TR of 1.50 calculated from the 360-minute theoretical half-time tissue type. The 95% confidence interval for this prediction produces an upper limit of 30.0% and a lower limit of 0.0% incidence within a tested group.

This same procedure can be applied to any prediction of % DCS or % VGE when using a spectrum of TRs. Figures 2 and 3 show a 95% confidence band about the linear regression line of % DCS and % VGE against their respective TRs. Thus, given a specific TR, a range of predicted % VGE or % DCS incidence can be determined.

The Hill equations (eq. 8 and 9) are analyzed in the same fashion. Figures 5 and 6 show the 95% confidence interval about the sigmoidal functions. In figure 5, a TR of 1.50 would predict a 10% DCS...
incidence. The 95% confidence interval calculated for this prediction is \(10\% \pm 14\%\). In figure 6, the same TR predicts 44% VGE with a 95% confidence interval of 44% \(\pm 20\%\).

Since the data pairs that comprise all the figures were from group results, it must be stressed that the 95% confidence interval applies to the percent incidence you would expect if a group of individuals, randomly chosen from a general population, were tested. For a group, the \textit{a priori} expected incidence and the \textit{a posteriori} incidence should be close, i.e., the \textit{a posteriori} incidence should be within the confidence interval. For an individual, an \textit{a priori} level of risk can be predicted, however; the \textit{a posteriori} incidence will be 0% or 100%.

**Accuracy and Precision of Predictive Linear and Hill Equations**

Accuracy assessment of equations 4, 5, 8, and 9 for predicting the incidence of DCS and VGE is accomplished using a cross validation procedure. The DCS and VGE data set is randomly divided into two equal subsets. One subset is designated the training set, and is used to generate a new linear and Hill predictive equations. The remaining data for this cross validation test is the test set.

Substituting the test set TR values into the training set predictive equations provides a set of \% VGE and \% DCS values and residuals which are statistically compared to the observed percentage values and residuals of the training set. Residuals are the differences between observed values and the regression equation predicted values.

Table 2 contains the training set and test set statistics for both the linear and Hill equation data, i.e., the average \(\bar{\theta}\) and standard deviation \(\sigma_\theta\) of the unsigned residuals, the mean error of the fit \(\bar{\epsilon}\), the 95% confidence upper bound for the test set deviation, and the critical and computed F-statistics for testing the hypothesis that the mean square residuals of the training set and the average squared residuals of the test set are equal.

From table 2, F-statistic comparisons provide no basis for rejecting the null hypothesis that the precision with which the training set predictive equations represent the test set is significantly different from that with which the predictive equation represents the training set. However, the mean deviations of the test sets are larger than those of the training sets except for the \% DCS Hill equation. In regard to the average deviation upper bound, loosely interpreted, one can be 95% confident that the incidence of VGE or DCS will be no more than approximately 20% greater than that predicted by equations 4, 5, 8, and 9. This is the degree of precision possible for any \% VGE or \% DCS prediction. Finally, in regard to accuracy, the average error \(\bar{\epsilon}\) for each test fit is small, 1.0% or less, indicating the central location of the predictive equations with respect to both training and test sets. The exception is the \% VGE Hill equation.

**TIME TO ONSET OF DCS AND VGE DURING SIMULATED EVA**

In the preceding analysis, consideration was given to the occurrence of DCS or VGE without regard to the time of onset or detection after the beginning of the simulated EVA. In this section the combined Air Force\(^3,4,17,18\) and NASA data\(^7,8,9,10,11,15,16\) are analyzed to provide graphs for predicting
### TABLE 2: CROSS VALIDATION TRAINING AND TEST STATISTICS

**Linear Equations**

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<th>( \bar{e} )</th>
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**Hill Equations**

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<th>( s_d )</th>
<th>( df )</th>
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<th>( \bar{e} )</th>
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23
the incidence of DCS and VGE as a function of the time into the EVA at a specified TR. The data in Table 1 consists of 111 tests for 3 hours of simulated EVA exposure, 45 tests for 4 hours of exposure, and 451 tests for 6 hours of EVA exposure.

Even though the expected DCS or VGE incidence may be quite high at certain TRs, the risk can be considerably lessened if the EVA time is shortened. Figures 11 and 12 present the cumulative incidence of DCS and VGE as linear functions of TR at various simulated EVA times up to 360 minutes.

The time lines in figures 11 and 12 are structured as follows

- Equations 4 and 5 are solved for the TR intercepts to obtain

  \[ \% \text{VGE} = 0 \text{ at } TR = 1.057, \text{ and} \]

  \[ \% \text{DCS} = 0 \text{ at } TR = 1.279 \]

- Assuming that all cumulative DCS or VGE time lines pass through the same respective TR intercept, the cumulative DCS and VGE incidence and TR data at the specified time are fitted to respective linear functions constrained to pass through the corresponding TR intercept. Figures 11 and 12 constitute families of cumulative percent incidence versus TR lines that have time as a variable parameter.

Figures 13 and 14 are families of lines for DCS and VGE incidence versus time with TR as a variable parameter. These TR lines are obtained by solving each of the family of DCS and VGE time lines for the incidence at a given TR and drawing a smooth curve through these calculated data points. Both figures show the expected incidence given a particular 360 minute theoretical half-time TR and a planned EVA duration. They express the increasing risk of DCS and VGE given a particular TR as EVA duration is increased.

Table 3 contains the calculated linear equations that can be applied to predict \% DCS or \% VGE for a specified EVA duration or if an emergency EVA of limited duration is needed.

**DCS and VGE Latency Times**

Table 4 contains data on the average time to report Type I DCS symptoms or to detect VGE after a particular prebreathe procedure. Figure 15 displays the DCS latency times against the calculated TR from the 360 minute theoretical half-time tissue type. A nonlinear fitting function was applied to the data. Figure 16 displays the VGE latency times plotted against the calculated TR. In both cases, the data indicates that lower TRs will delay the initial appearance of VGE and complaints of Type I DCS.
Figure 11.- Selected linear equations to describe DCS incidence as a function of simulated EVA exposure time.
Figure 12.- Selected linear equations to describe VGE incidence as a function of simulated EVA exposure time.
Figure 13.- Calculated DCS incidence with variable simulated EVA exposure time using the 360-minute theoretical half-time tissue type.
Figure 14.- Calculated VGE incidence with variable simulated EVA exposure time using the 360-minute theoretical half-time tissue type.
TABLE 3.- DCS AND VGE INCIDENCE AS A FUNCTION OF EVA DURATION

<table>
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<td>31</td>
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<td>60</td>
<td>%DCS = 21.9 * Tissue Ratio (360') - 29.9</td>
<td>.74</td>
<td>23</td>
</tr>
<tr>
<td></td>
<td>%VGE = 58.5 * Tissue Ratio (360') - 72.5</td>
<td>.77</td>
<td>31</td>
</tr>
<tr>
<td>90</td>
<td>%DCS = 49.1 * Tissue Ratio (360') - 68.0</td>
<td>.82</td>
<td>23</td>
</tr>
<tr>
<td></td>
<td>%VGE = 76.5 * Tissue Ratio (360') - 91.2</td>
<td>.74</td>
<td>31</td>
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<tr>
<td>120</td>
<td>%DCS = 54.6 * Tissue Ratio (360') - 74.6</td>
<td>.80</td>
<td>23</td>
</tr>
<tr>
<td></td>
<td>%VGE = 88.8 * Tissue Ratio (360') - 104.0</td>
<td>.79</td>
<td>31</td>
</tr>
<tr>
<td>150</td>
<td>%DCS = 47.0 * Tissue Ratio (360') - 62.4</td>
<td>.77</td>
<td>26</td>
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<tr>
<td></td>
<td>%VGE = 103.0 * Tissue Ratio (360') - 120.0</td>
<td>.87</td>
<td>31</td>
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<tr>
<td>180</td>
<td>%DCS = 55.7 * Tissue Ratio (360') - 73.7</td>
<td>.79</td>
<td>26</td>
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<tr>
<td></td>
<td>%VGE = 104.0 * Tissue Ratio (360') - 119.0</td>
<td>.87</td>
<td>31</td>
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<td>210</td>
<td>%DCS = 41.7 * Tissue Ratio (360') - 54.3</td>
<td>.71</td>
<td>23</td>
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<td></td>
<td>%VGE = 94.2 * Tissue Ratio (360') - 105.0</td>
<td>.83</td>
<td>28</td>
</tr>
<tr>
<td>240</td>
<td>%DCS = 43.0 * Tissue Ratio (360') - 54.6</td>
<td>.66</td>
<td>23</td>
</tr>
<tr>
<td></td>
<td>%VGE = 93.5 * Tissue Ratio (360') - 102.0</td>
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<td>28</td>
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<tr>
<td>270</td>
<td>%DCS = 29.5 * Tissue Ratio (360') - 34.8</td>
<td>.43</td>
<td>18</td>
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<tr>
<td></td>
<td>%VGE = 109.0 * Tissue Ratio (360') - 124.0</td>
<td>.80</td>
<td>22</td>
</tr>
<tr>
<td>300</td>
<td>%DCS = 32.2 * Tissue Ratio (360') - 38.4</td>
<td>.45</td>
<td>18</td>
</tr>
<tr>
<td></td>
<td>%VGE = 109.0 * Tissue Ratio (360') - 122.0</td>
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<td>22</td>
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<tr>
<td>330</td>
<td>%DCS = 36.0 * Tissue Ratio (360') - 43.6</td>
<td>.49</td>
<td>18</td>
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<tr>
<td></td>
<td>%VGE = 107.0 * Tissue Ratio (360') - 119.0</td>
<td>.83</td>
<td>22</td>
</tr>
<tr>
<td>360</td>
<td>%DCS = 34.0 * Tissue Ratio (360') - 40.3</td>
<td>.48</td>
<td>18</td>
</tr>
<tr>
<td></td>
<td>%VGE = 104.0 * Tissue Ratio (360') - 114.0</td>
<td>.82</td>
<td>22</td>
</tr>
</tbody>
</table>
### TABLE 4.- DCS AND VGE LATENCY TIMES

<table>
<thead>
<tr>
<th>Prebreathe Procedure Number</th>
<th>360 Final Exposure TR Pressure (psia)</th>
<th>Average VGE Latency Time (minutes) ± (SD) N</th>
<th>Average DCS Latency Time (minutes) ± (SD) N</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>1.80 4.3</td>
<td>54 (23) 7</td>
<td>61 (15) 4</td>
</tr>
<tr>
<td>2</td>
<td>1.83 4.3</td>
<td>47 (28) 14</td>
<td>82 (52) 5</td>
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<td>3</td>
<td>1.67 4.3</td>
<td>48 (31) 7</td>
<td>109 (37) 4</td>
</tr>
<tr>
<td>4</td>
<td>1.70 4.3</td>
<td>120 (62) 15</td>
<td>105 (60) 7</td>
</tr>
<tr>
<td>5</td>
<td>1.75 4.3</td>
<td>43 (40) 10</td>
<td>134 (68) 6</td>
</tr>
<tr>
<td>6</td>
<td>1.60 4.3</td>
<td>60 (41) 13</td>
<td>132 (84) 6</td>
</tr>
<tr>
<td>7</td>
<td>1.45 4.3</td>
<td>140 (58) 5</td>
<td>165 (81) 7</td>
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<tr>
<td>8</td>
<td>1.68 4.3</td>
<td>124 (70) 20</td>
<td>173 (85) 3</td>
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<td>9</td>
<td>1.43 4.3</td>
<td>121 (27) 5</td>
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<td>10</td>
<td>1.68 4.3</td>
<td>105 (43) 7</td>
<td>24 (--) 1*</td>
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<td>11</td>
<td>1.12 4.3</td>
<td>45 (17) 2*</td>
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<td>12</td>
<td>1.37 4.3</td>
<td>88 (45) 4</td>
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<td>14</td>
<td>1.35 4.3</td>
<td>66 (58) 4</td>
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<td>16/16a</td>
<td>1.49 7.8</td>
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<td>17/17a</td>
<td>1.43 7.8</td>
<td>122 (63) 25</td>
<td></td>
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<tr>
<td>18/18a</td>
<td>1.42 7.8</td>
<td>101 (62) 24</td>
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<td>19</td>
<td>1.29 9.0</td>
<td>96 (38) 7</td>
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<td>20</td>
<td>1.16 10.0</td>
<td>242 (6) 2</td>
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<tr>
<td>21</td>
<td>1.36 8.5</td>
<td>195 (81) 3</td>
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<td>22/22a</td>
<td>1.22 9.5</td>
<td>293 (44) 4</td>
<td></td>
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<td>23/23a</td>
<td>1.32 4.3</td>
<td>211 (99) 11</td>
<td>142 (68) 4</td>
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<tr>
<td>25/25a</td>
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<td>150 (59) 3</td>
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<tr>
<td>26/26a</td>
<td>1.40 8.3</td>
<td>118 (98) 8</td>
<td></td>
</tr>
</tbody>
</table>

*Data omitted from mathematical analysis shown in figures 15 and 16.
Figure 15.- Latency time for initial DCS symptoms versus 360-minute theoretical half-time Tissue Ratios.

Avg. DCS Latency Time = \( \frac{111}{(TR - 0.78)^{0.88}} \)

\( r = 0.61 \quad n = 10 \)

- Based on 48 human decompressions simulating EVA Activity
Figure 16.- Latency time for initial VGE detection versus 360-minute theoretical half-time Tissue Ratios.

Avg. VGE Latency Time = \( \frac{64}{(TR - 0.78)^{1.40}} \)

- Based on 226 human decompressions simulating EVA Activity

- \( r = 0.76 \)
- \( n = 22 \)
SUMMARY OF DCS AND VGE PREDICTIVE EQUATIONS

Tissue Nitrogen Partial Pressure Relaxation Equation

\[ P_t = P_o + \left(P_a - P_o\right)\left(1 - e^{-kt}\right) \]

where

- \( P_t \) = the nitrogen partial pressure in the tissue after exposure for \( t \) minutes,
- \( P_o \) = initial tissue nitrogen partial pressure,
- \( P_a \) = ambient nitrogen partial pressure in breathing medium,
- \( e \) = base of natural logarithm,
- \( t \) = exposure time in minutes, and
- \( k \) = tissue nitrogen partial pressure rate constant.

The tissue rate constant \( (k) \) is related to the tissue nitrogen half-time \((t_{1/2})\) through the equation,

\[ k = \frac{\ln 2}{t_{1/2}} = \frac{.693}{t_{1/2}} \]

where

- \( t_{1/2} \) = tissue nitrogen partial pressure half-time

Tissue Ratio

\[ TR = \frac{\text{Final Tissue pN}_2 \text{ (abs)}}{\text{Ambient Pressure (abs)}} \]
Linear DCS and VGE Predictive Equations

\[ \% \text{VGE} = 95.5 \times \text{Tissue Ratio (360')} - 101.1 \quad r = .88 \]

\[ \% \text{VGE} = 57.9 \times \text{Tissue Ratio (360')} - 74.1 \quad r = .78 \]

Hill DCS and VGE Predictive Equations

\[ \% \text{VGE} = \left( \text{TR} - .78 \right)^{3.08} / \left[ \left( \text{TR} - .78 \right)^{3.08} + 0.47 \right] \times 100 \quad r = .91 \]

\[ \% \text{DCS} = \left( \text{TR} - .78 \right)^{4.24} / \left[ \left( \text{TR} - .78 \right)^{4.24} + 2.16 \right] \times 100 \quad r = .84 \]

\[ \% \text{Refractory Symptoms} = \left( \text{TR} - .78 \right)^{2.50} / \left[ \left( \text{TR} - .78 \right)^{2.50} + 17.61 \right] \times 100 \quad r = .53 \]

where TR is the Tissue Ratio of the 360 minute theoretical half-time tissue.
USER SECTION

Predicting the Incidence of DCS and VGE

In order to properly calculate TRs for subsequent use in equations 8,9, and 10 the user must follow the exact procedures for calculating TRs that were used in the development of the predictive equations. A simple example of the use of equations 2 and 3 was given previously. A more complex example is presented to acquaint the user with three important rules that must be understood when calculating the 360 minute theoretical TR. A second example on the proper use of the models during a simulated emergency EVA is presented to demonstrate how to design a prebreathe procedure that takes advantage of a short EVA duration.

Example 1

Problem Calculate the risk of Type I DCS, VGE, and DCS symptoms that could result in early EVA termination given the following prebreathe procedure:

Event 1 Two crewmen prebreathe 100% oxygen for 60 minutes while at 14.7 psia in the Shuttle.

Event 2 The Shuttle atmospheric pressure is reduced to 10.2 psia within 10 minutes, while the EVA crewmen are still breathing 100% oxygen. Breathing gas composition at 10.2 psia is adjusted to 27% oxygen, 73% nitrogen. The entire crew spends 24 hours in this new pressure/gas environment.

Event 3 A 40 minute, 100% oxygen prebreathe is performed in the suit while still at 10.2 psia cabin pressure. This is followed by a 30 minute decompression to 4.3 psia.

Problem Calculate the resulting 360 minute theoretical half-time TR using equations 2 and 3 for the first EVA. Use the calculated TR in conjunction with equations 8,9, and 10 to calculate the risk of DCS and VGE for the first EVA.

Event 4 4.0 hours of EVA are performed while breathing 100% oxygen at 4.3 psia. The crew then returns to the Shuttle for a 2.0 hour lunch. Shuttle atmosphere is at 10.2 psia with 27% oxygen, 73% nitrogen.

Event 5 Once again, a 40-minute, 100% oxygen prebreathe is performed in the suit prior to a 30-minute decompression to 4.3 psia for a 3.0 hour EVA.

Problem Calculate the resulting 360 minute theoretical half-time TR using equations 2 and 3 for the second EVA. Use the calculated TR in conjunction with equations 8,9, and 10 to calculate the risk of DCS and VGE on the second EVA.
Example 1 Calculations

Initial Comments

(1) The 360 minute theoretical half-time tissue is to be used in all applications of equation 2.
(2) Time is expressed in minutes.
(3) Pressure is expressed in psia.

Rule 1

Always use the ambient nitrogen partial pressure ($P_a$) in the breathing mixture as the driving pressure in equation 2. Do not use alveolar nitrogen partial pressure. Initial tissue nitrogen partial pressure ($P_o$) is equal to 11.6 psia if the crewman has been exposed to sea level pressure and gas composition for over 24 hours.

Step 1

Two crewmen prebreathe 100% oxygen for 60 minutes while at 14.7 psia in the Shuttle. In equation 2, $P_o = 11.6$ psia, while $P_a = 0.0$ psia since 100% oxygen contains no nitrogen. Equation 2 calculates a final tissue nitrogen partial pressure ($P_f$) of 10.33 psia for the 360-minute theoretical half-time tissue after 60 minutes of prebreathe.

Rule 2

Any ambient pressure transition (decompression or recompression) that exceeds 10 minutes in duration must be accounted for in the prebreathe procedure. Since crewmen are usually breathing 100% oxygen during these transitions, the time from one ambient pressure to another must be included as part of the 100% oxygen prebreathe period if it exceeds 10 minutes.

Step 2

The Shuttle atmospheric pressure is reduced to 10.2 psia within 10 minutes while the EVA crewmen are still breathing 100% oxygen. Breathing gas composition is adjusted to 27% oxygen and 73% nitrogen. The entire crew spends 24 hours in this new pressure/gas environment.

Since the decompression time from 14.7 psia to 10.2 psia was less than 10 minutes, the additional oxygen prebreathe time is not added to the initial 60 minute prebreathe. The final tissue nitrogen pressure is still 10.33 psia upon reaching 10.2 psia.

Equation 2 is used again to establish the final tissue nitrogen partial pressure after 24 hours (1440 minutes) of exposure to 73% nitrogen. In this case

- $P_o = P_f$ from the previous application of equation 2: 10.33 psia.
- $P_a = 73\%$ nitrogen $\times 10.2$ psia $= 7.44$ psia nitrogen.
- $P_f$ after 1440 minutes in this environment $= 7.62$ psia nitrogen.
Step 3  A 40-minute, 100% oxygen prebreathe is performed in the suit followed by a 30-minute decompression to 4.3 psia.

Rule 2 requires that the 30 minutes of oxygen prebreathing during the decompression from 10.2 psia to 4.3 psia be included in the oxygen prebreathe period. Total oxygen prebreathe = 70 minutes.

Equation 2 is used again to establish the final tissue nitrogen partial pressure after the 70-minute exposure to 0.0 psia nitrogen. In this case

- \( P_0 = P_t \) from the previous application of equation 2: 7.62 psia.
- \( P_a = 0\% \) nitrogen \times 10.2 psia = 0.0 psia nitrogen.
- \( P_t \) after 70 minutes in this environment = 6.66 psia nitrogen.

For the first EVA, the TR is calculated by using equation 3

\[
TR = \frac{P_t}{\text{Ambient Pressure (suit pressure)}} = \frac{6.66 \text{ psia}}{4.3 \text{ psia}} = 1.55
\]

Substituting this TR into equations 8, 9, and 10 results in a 49% risk of VGE, 13% risk of Type I DCS, and a 3% risk of DCS symptoms that may force a crewman to end his EVA. These predictions are based on EVA durations up to 6.0 hours.

Rule 3  During multiple exposures to low ambient pressures separated by hours or days at higher nitrogen pressures, one must account for the nitrogen removed during the 100% oxygen prebreathe while at low pressures. In other words, the time during EVA must be used as part of the prebreathe period if additional EVAs are to be planned within 24 hours.

Step 4  4.0 hours of EVA are performed while breathing 100% oxygen at 4.3 psia, and the crew returns to the Shuttle for a 2.0 hour lunch.

Equation 2 is used again to establish the final tissue nitrogen partial pressure after 4.0 hours (240 minutes) of exposure to 0.0 psia nitrogen during the EVA. In this case

- \( P_0 = P_t \) from the previous application of equation 2: 6.66 psia nitrogen
- \( P_a = 0.0 \text{ psia nitrogen} \)
- \( P_t \) after 240 minutes in this environment = 4.20 psia nitrogen.

The crew returns to the Shuttle environment where \( P_a = 7.44 \) psia for 2.0 hours (120 minutes). \( P_0 = P_t = 4.20 \) psia nitrogen and equation 2 calculates that \( P_t \) after 2.0 hours in the Shuttle is 4.86 psia nitrogen.
Step 5  Once again, a 40-minute oxygen prebreathe is performed in the suit prior to a 30 minute decompression to 4.3 psia for a 3.0-hour EVA.

Rule 2 also applies in this situation. Seventy minutes of 100% oxygen prebreathe must be used in the calculation.

Equation 2 is used again to establish the final tissue nitrogen partial pressure after 70 minutes of exposure to 0.0 psia nitrogen. In this case

- \( P_0 = P_t \) from the previous application of equation 2: 4.86 psia nitrogen
- \( P_a = 0.0 \) psia nitrogen
- \( P_t \) after 70 minutes in this environment = 4.25 psia nitrogen.

For the second EVA, TR is calculated using equation 3

\[
TR = \frac{P_t}{\text{Ambient Pressure (suit pressure)}}
\]

\[
= \frac{4.25 \text{ psia}}{4.3 \text{ psia}} = 0.98
\]

Substituting this TR into equations 8, 9, and 10 results in a 2% risk of VGE, 0% risk of Type I DCS, and a 0% risk of DCS symptoms that could terminate an EVA.

Example 2

Problem  Shuttle crew members are at 14.7 psia breathing 21% oxygen and 79% nitrogen atmosphere. A life-threatening failure in the cargo bay occurs that requires an EVA for a repair mission. EVA duration should not exceed 2.0 hours for this repair task. Calculate the minimum time of 100% oxygen prebreathe that would allow a safe 2.0-hour EVA. In this emergency, a safe EVA is defined as one that does not expose EVA crewman to a Type I DCS risk greater than 25%.

From equation 9, a TR of 1.7 would predict a 25% risk of Type I DCS. Equations 4, 5, 8, 9, and 10 were derived from the cumulative incidence of DCS and VGE from simulated EVAs up to 6.0 hours. The incidence of DCS and VGE they predict reflect the influence of long duration EVAs. In this simulated emergency situation where EVA duration is planned for 2.0 hours, a higher TR can theoretically be used that provides the same DCS risk if the EVA exposure is limited. Figure 13 graphically shows that a TR of 1.9 could be tolerated for 2.0 hours and still predict a 25% risk of Type I DCS. In addition, Figure 15 shows that the average time to detect DCS with a 1.9 TR is 110 minutes. This would indicate a 2.0 hour EVA would present an acceptable risk for this emergency. Equation 2 can now be evaluated such that 100% oxygen prebreathe time becomes the variable to calculate, while the final TR becomes the fixed value. In this situation, the least amount of oxygen prebreathe prior to decompression to 4.3 psia is 182 minutes.
If the emergency EVA condition warrants less prebreathe time, then the risk of Type I DCS (higher TR) could be increased, or the planned EVA duration could be shortened. This flexibility allows the user options in the event of emergency EVA conditions.

Assumptions in and Limitations of The Models

Scientists and engineers attempt to maximize and utilize man's ability to adapt to low pressure environments. Space suits operating at a pressure lower than cabin pressure are desirable for many reasons and have been successfully used throughout the U.S. space program. The need to properly manage denitrogenation-decompression procedures prior to a reduction in ambient pressure is critical in the one atmosphere Shuttle and Space Station environments. The models developed in this report are useful to that end, but their limitations are discussed.

Any empirical model is limited in its ability to predict. It is limited to the data used to derive the model. Prebreathe conditions, possibly dictated by emergency conditions, that deviate dramatically from those tested may produce outcomes not predicted by the models. Denitrogen-decompression procedures that fall outside the range of the tests used to define these models or which deviate in any of the parameters that are known or suspected to effect the incidence of DCS may produce outcomes not predicted by the models. In such cases it is critical to perform verification testing prior to accepting the results from the models.

One of the environmental parameters that is different in space flight than in the altitude chamber test programs is the lack of gravity, and the physiological adaptations it induces. It is possible that microgravity could have some influence on the incidence of DCS or VGE. At the present time there is no positive or negative evidence of a zero-g influence on altitude decompression sickness. Experiments being planned for Space Station may provide evidence of a microgravity effect on DCS or VGE incidence during EVA.
REFERENCES


APPENDIX A
OUTLINE OF 26 SEPARATE PREBREATHE PROCEDURES
USED IN 607 MANNED TESTS
CONDUCTED AT THE JOHNSON SPACE CENTER
AND
BROOKS AIR FORCE BASE (1982 - 1986)

<table>
<thead>
<tr>
<th>No.</th>
<th>Prebreathe Procedure</th>
</tr>
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<tbody>
<tr>
<td>1.</td>
<td>3.5 hours oxygen prebreathe at 14.7 psia prior to 3.0 hour exposure to 4.3 psia. Decompression was rapid. Exercise stressed lower body. N = 11</td>
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<tr>
<td>2.</td>
<td>12.0 hours at 10.2 psia plus a 40 minute oxygen prebreathe prior to a 3.0 hour exposure to 4.3 psia. Decompression was rapid. Exercise stressed lower body. Gas composition at 10.2 psia was 26.5% O₂ - 73.5% N₂. N = 16</td>
</tr>
<tr>
<td>3.</td>
<td>12.0 hours at 10.2 plus a 90 minute oxygen prebreathe prior to a 3.0 hour exposure to 4.3 psia. Decompression was rapid. Exercise stressed lower body. Gas composition at 10.2 psia was 26.5% O₂ - 73.5% N₂. N = 12</td>
</tr>
<tr>
<td>4.</td>
<td>3.5 hours oxygen prebreathe at 14.7 prior to a 4.0 hour exposure to 4.3 psia. Decompression was gradual and allowed 30 minutes of additional oxygen prebreathe prior to reaching 4.3 psia. Exercise stressed upper body. N = 23</td>
</tr>
<tr>
<td>5.</td>
<td>12.0 hours at 10.2 psia plus a 40 minute oxygen prebreathe prior to a 4.0 hour exposure to 4.3 psia. Decompression was gradual and allowed 25 minutes of additional oxygen prebreathe prior to reaching 4.3 psia. Exercise stressed upper body. Gas composition at 10.2 psia was 26.5% O₂ - 73.5% N₂. N = 22</td>
</tr>
<tr>
<td>6.</td>
<td>4.0 hours oxygen prebreathing at 14.7 psia prior to a 6.0 hour exposure to 4.3 psia. Decompression was gradual and allowed 30 minutes of additional oxygen prebreathe prior to reaching 4.3 psia. Exercise stressed upper body. N = 28</td>
</tr>
<tr>
<td>7.</td>
<td>Same procedure as #6 except crew returned to 14.7 psia for 17.0 hours. Second EVA began after 4.0 hours oxygen prebreathe at 14.7 psia prior to second 6.0 hour exposure to 4.3 psia. Decompression was gradual and allowed 30 minutes of additional oxygen prebreathe prior to reaching 4.3 psia. Exercise stressed upper body. N = 14</td>
</tr>
<tr>
<td>8.</td>
<td>60 minutes oxygen prebreathe at 14.7 psia followed by 12.0 hours at 10.2 psia plus an additional 40 minute oxygen prebreathe prior to a 6.0 hour exposure to 4.3 psia. Decompression was gradual and allowed 25 minutes of additional oxygen prebreathe prior to reaching 4.3 psia. Exercise stressed upper body. Gas composition at 10.2 psia was 26.5% O₂ - 73.5% N₂. N = 35</td>
</tr>
</tbody>
</table>
9. Same procedure as #8 except crew returned to 10.2 psia for 17.0 hours. Second EVA began after 40 minute oxygen prebreathe at 10.2 prior to a second 6.0 hour exposure to 4.3 psia. Decompression was gradual and allowed 25 minutes of additional oxygen prebreathe prior to reaching 4.3 psia. Exercise stressed upper body. N = 12

10. 60 minutes oxygen prebreathe at 14.7 psia followed by 12.0 hours at 10.2 psia plus an additional 40 minute oxygen prebreathe prior to a 3.0 hour exposure to 4.3 psia. This was the first of two exposures in the same day. Decompression was gradual and allowed 25 minutes of additional oxygen prebreathe prior to reaching 4.3 psia. Gas composition at 10.2 psia was 26.5% O₂ - 73.5% N₂. N = 12

11. Same procedure as #10. Crew then returned to 10.2 psia for 80 minutes. A 40 minute oxygen prebreathe was then performed prior to a second 3.0 hour exposure to 4.3 psia in the same day. Decompression was gradual and allowed 25 minutes of additional oxygen prebreathe prior to reaching 4.3 psia. N = 12

12. Same procedure as #10 plus #11 except crew returned to 10.2 psia for 14.0 hours. First EVA of second day began with a 40 minute oxygen prebreathe prior to a 3.0 hour exposure to 4.3 psia. Decompression was gradual and allowed 25 minutes of additional oxygen prebreathe prior to reaching 4.3 psia. N = 12

13. Same procedure as #8 plus #11 plus #12. Crew then returned to 10.2 psia for 80 minutes. A 40 minute oxygen prebreathe was then performed prior to a second 3.0 hour exposure of the second day EVA to 4.3 psia. Decompression was gradual and allowed 25 minutes of additional oxygen prebreathe prior to reaching 4.3 psia. N = 12

14. Same procedure as #10 plus #11 plus #12 plus #13 except crew returned to 10.2 psia for 14.0 hours. First EVA of third day began with a 40 minute oxygen prebreathe prior to a 3.0 hour exposure to 4.3 psia. Decompression was gradual and allowed 25 minutes of additional oxygen prebreathe prior to reaching 4.3 psia. N = 12

15. Same procedure as #10 plus #11 plus #12 plus #13 plus #14. Crew then returned to 10.2 psia for 80 minutes. A 40 minute oxygen prebreathe was then performed prior to a second 3.0 hour exposure of the third day EVA to 4.3 psia. Decompression was gradual and allowed 25 minutes of additional oxygen prebreathe prior to reaching 4.3 psia. N = 12

16. Exposure to 7.8 psia for 6.0 hours using 50% O₂ - 50% N₂ mixture without prior oxygen prebreathe. Decompression required 10 minutes. Exercise stressed upper body. This was the first of a multiple exposure series separated by 18.0 hours at sea level conditions. N = 32

16a. Same as #16 except females were tested. N = 32

17. Same as #16 except crew returned to 14.7 psia for 18.0 hours prior to their second 6.0 hour exposure to 7.8 psia. No prebreathe prior to the 10 minute decompression. N = 31
17a. Same as #17 except females were tested. N = 31

18. Same as #16 plus #17 except crew returned to 14.7 psia for 18.0 hours prior to their third 6.0 hour exposure to 7.8 psia. N = 31

18a. Same as #18 except females were tested. N = 29

19. Exposure to 9.0 psia for 6.0 hours without prior oxygen prebreathe. Decompression required 10 minutes. Exercise stressed upper body and 50% O₂ - 50% N₂ was used. Pilot study using susceptible subjects. % VGE data in these tests were reduced by 1/2 since the tested group was selected for its susceptibility to develop VGE. A randomly selected group would probably have developed the listed % VGE with the given prebreathe procedure. N = 16

20. Exposure to 10.0 psia for 6.0 hours without prior oxygen prebreathe. Decompression required 10 minutes. Exercise stressed upper body and 50% O₂ - 50% N₂ was used. Pilot study using susceptible subjects. % VGE data in these tests were reduced by 1/2 since the tested group was selected for its susceptibility to develop VGE. A randomly selected group would probably have developed the listed % VGE with the given prebreathe procedure. N = 8

21. Exposure to 8.5 psia for 6.0 hours without prior oxygen prebreathe. Decompression required 10 minutes. Exercise stressed upper body. Pilot study using susceptible subjects. % VGE data in these tests were reduced by 1/2 since the tested group was selected for its susceptibility to develop VGE. A randomly selected group would probably have developed the listed % VGE with the given prebreathe procedure. N = 9

22. Exposure to 9.5 psia for 6.0 hours using 50% O₂ - 50% N₂ mixture without prior oxygen prebreathe. Decompression required 10 minutes. Exercise stressed upper body. Pilot study using susceptible subjects. % VGE data in these tests were reduced by 1/2 since the tested group was selected for its susceptibility to develop VGE. A randomly selected group would probably have developed the listed % VGE with the given prebreathe procedure. N = 6

22a. Same as #22 except females were tested. N = 11

22b. Same as #22 except males were tested. (extended test) N = 20

23. 6.0 hours oxygen prebreathe at 14.7 psia prior to 6.0 hour exposure to 4.3 psia. Decompression required 10 minutes. Exercise stressed upper body. N = 19

23a. Same as #23 except females were tested. N = 19

24. 8.0 hours oxygen prebreathe at 14.7 psia prior to 6.0 hour exposure to 4.3 psia. Decompression required 10 minutes. Exercise stressed upper body. N = 8
25. 2.0 hours oxygen prebreathe at 14.7 psia prior to 24.0 hours at 10.2 psia. 15 minute de-
compression from 14.7 psia to 10.2 psia was included as a portion of the 2.0 hour oxygen
prebreathe. 10 minute decompression from 10.2 psia to 6.0 psia after 24.0 hours. Subjects
exercised 6.0 hours while breathing 60% O\textsubscript{2} - 40% N\textsubscript{2} mixture. Exercise stressed upper body.
Gas composition at 10.2 psia was 28% O\textsubscript{2} - 82% N\textsubscript{2}. N = 15

25a. Same as #25 except females were tested. N = 14

26. Exposure to 8.3 psia for 6.0 hours without prior oxygen prebreathe. Decompression re-
quired 10 minutes. Exercise stressed upper body and 50% oxygen and 50% nitrogen
was used. N = 20

26a. Same as #26 except females were tested. N = 11
Empirical models for predicting the incidence of Type I altitude decompression sickness (DCS) and venous gas emboli (VGE) during space extravehicular activity (EVA), and for use in designing safe denitrogenation-decompression procedures are developed. The models are parameterized using DCS and VGE incidence data from 26 NASA and USAF manned altitude chamber decompression tests using 607 male and female subject tests.

These models, and procedures for their use, consist of: (1) an exponential relaxation model and procedure for computing tissue nitrogen partial pressure resulting from a specified prebreathing and stepped decompression sequence; (2) a formula for calculating Tissue Ratio (TR), a tissue decompression stress index; (3) linear and Hill equation models for predicting the total incidence of VGE and DCS attendant with a particular TR; (4) graphs of cumulative DCS and VGE incidence (risk) versus EVA exposure time at any specified TR; and (5) two equations for calculating the average delay period (latency time) for the initial detection of VGE or indication of Type I DCS in a group after a specific denitrogenation-decompression procedure. Several examples of realistic EVA preparations are provided to demonstrate the use of the predictive models and interpretation of the results.