RESEARCH REPORT

EVALUATION OF EXERCISE-RESPIRATORY SYSTEM MODIFICATIONS AND INTEGRATION SCHEMES FOR PHYSIOLOGICAL SYSTEMS

by

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ABSTRACT

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Exercise subroutine modifications are implemented in an exercise-respiratory system model yielding improvement of system response to exercise forcings. A more physiologically desirable respiratory ventilation rate in addition to an improved regulation of arterial gas tensions and cerebral blood flow is observed. A respiratory frequency expression is proposed which would be appropriate as an interfacing element of the respiratory-pulsatile cardiovascular system.

Presentation of a circulatory-respiratory system integration scheme along with its computer program listing is given. The integrated system responds to exercise stimulation for both nonstressed and stressed physiological states. Other integration possibilities are discussed with respect to the respiratory, pulsatile cardiovascular, thermoregulatory, and the long-term circulatory systems.
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1. INTRODUCTION

1.1 Overview of Modeling Effort

The major objectives and emphasis of the overall physiological system modeling effort focus on the development of an effective whole-body algorithm as being the most significant feature. (1) Utilizing system theory, mathematical descriptions of physiological functions, appropriate empirical interrelationships between variables, and a computer implementation and display system complex physiological systems have been realized.

Physiological systems that were deemed important to the project's overall goals included the respiratory, fluid-electrolyte balance circulatory, pulsatile cardiovascular, and thermoregulatory systems. Proper evaluation, modification, and adaptation of selected models (2-5) have been made with their implementation coordinated with the project's goals. The indicated references provide the necessary details of the models. Therefore, only specific modifications will be discussed in this report.

The computer program for the integrated circulatory and respiratory control systems which will be presented in more detail in later sections is included in the Appendix. This program is comment laden and provides an adequate description of the involvement of each subroutine within itself and also its relationship to other subroutines in the program. Many of the comments in the respiratory system component refer to Grodins' basic model. (2) Block diagrams and explanatory material for each of the two major systems (circulatory and respiratory) are given by White (6) and Gallagher (7).
Since the primary thrust of the modeling revolves around the effects on parameters that are influenced by the space environment, variations in gravitational forces, external body pressures, inspired gas composition, and thermal conditions are some of the inputs utilized in determining dynamic physiological responses and associated hypotheses. Implementation and coordination of the simulations with major Skylab experiments provide the research-application impetus. Such a research effort demands a well-defined set of tasks which are fulfilled in sequence and/or concert. Some of the major milestones which are detailed by numerous specific tasks include

(a) evaluation and modification of major physiological subsystems,

(b) development and evaluation of hypotheses regarding physiological systems responses to stresses and altered environments,

(c) implementation of integrated system simulations and verification of responses, and

(d) implementation of supporting software systems for collecting, storing, and correlating simulated and experimental data.

With utilization of this complete whole-body simulation or its selected subsystems, revised techniques for remote medical care can be evaluated and implementation procedures established for future manned spaceflights. In addition, potential diagnostic and therapeutic capabilities may emerge from correlation of simulation and physiological testing during real-time medical monitoring. In keeping with the spirit of the project one should not lose sight of the important contribution that simulation provides in understanding physiological processes under the influence of normal and altered environments.

1.2 Exercise and the Respiratory System Model

The respiratory control system is an excellent example of a
physiological system that illustrates the influence of exercise upon its variables' responses. The magnitude and length of the exercise stimulus as well as the physical condition of the subject play integral roles in determination of the system's responses. One aspect of exercise simulation discussed in the following report involves the respiratory control system functioning as an independent system. Modifications of the exercise subroutine are evaluated illustrating improved control of ventilation rates and arterial and compartmental gas tensions.

1.3 Integration of Physiological System Models

There are several approaches which can be taken in the process of integrating the simulations of the respiratory and circulatory systems. Since both systems' dynamic responses are influenced by exercise and with exercise variations and their associated physiological forcing roles being major components of the manned spaceflight medical experiments, the integration scheme revolves around the implementation of exercise simulations. As implied in Section 3 there are extensions in the utility of these models for non-spaceflight experiments. (8)

In the following report emphasis is placed upon the interface component of the integrated model. For details concerning other variable formulations in the individual modes one should refer to the references. (2, 3, 6, 7). The interface allows for a minimal amount of alteration of each existing system while improving or supplementing the simulation responses of each system.

The integrated system can be described in terms of three functional components; stimuli, basic control systems, and the interfacing system as illustrated in Figure 1. Major categories of stimuli include exercise, parameter, and environmental variations.
Figure 1. Integration scheme for respiratory and circulatory control systems.
As previously mentioned emphasis is placed upon the exercise stimulus since several manned spaceflight experiments stress man's physiological performance under various exercise levels in an altered environment.

Since all of the basic control system's parameters are not common to both subsystems another stimulus includes the option of changing subsystem parameters and properties. This allows for investigation of the response of one system to a particular physiological change when that response is only indirectly related to that change. Therefore, another dimension of complexity has been added to physiological system modeling which has been previously unavailable.

Another stimulus which directly affects the respiratory system model is the gaseous composition of the environment. Responses to these environmental variations are not available in the existing formulation of the circulatory system model. The implications of providing an indirect environmental stimulus to the circulatory system via the respiratory system enhances the capabilities of the integrated system. Preliminary simulations of this classification of experiments have indicated a need for additional modifications of the circulatory system so that it is sensitive to the environmental stimuli.

1.4 Improved Respiratory Frequency Expression

Exercise presents the organism with the task of rapidly and optimally adapting to the requirements imposed upon it. One of these tasks is to increase ventilation in order to meet the accelerated metabolic demand for O_2 by the exercising muscle systems and the corresponding need to vent the CO_2 byproduct.

A ventilatory stimulus could be considered a physiological
variation which acts upon the respiratory centers and supplies information about the respiratory requirements of the body. There are two classifications of ventilatory stimuli: humoral and neural. Humoral stimuli involve modifications in the physical or chemical properties of the circulating blood that stimulate the respiratory centers either directly through the blood or via afferent nerve endings located in receptors in contact with the vessels. These are termed chemoreceptors and baroreceptors. The other stimuli are classified as neural stimuli. They originate in the brain or in cutaneous, mucosal, or deep peripheral receptors. Generally, these respond to localized conditions. Humoral and neural factors are postulated as controlling respiratory frequency.

The complexities of the interactions between neural and humoral control are manifested during exercise. The individual must increase ventilation to a level sufficient to provide the additional $O_2$ intake and $CO_2$ venting demanded by the body's high level of exercise metabolism. However, exercising subjects ventilate at a rate that exceeds the increases that would be dictated solely by arterial $PCO_2$, pH, and $PO_2$ alone. The difference between the humoral requirements and the actual ventilatory rate must then be a function of neural stimuli. This could be the result of neural pathways between the motor cortex and the respiratory centers and feedback from the forementioned proprioceptors. The neural factors assume even greater significance when ventilation is plotted as a function of exercise level and duration; however, there seems to be an upper limit of neural stimulation. It is noted that there is a rapid increase in perspiration immediately after the onset of exercise; this component has been postulated to be strictly neurological in nature.
An important question about ventilation, both for resting states and during exercise, concerns the actual frequency of respiration. It is obvious that identical ventilation requirements can be fulfilled either with a series of rapid, shallow inspirations or with long, deep ventilations.

The ventilation formulation in the respiratory system model is a satisfactory representation. The respiratory frequency expression is the one for which modifications are proposed. It is desirable to have a physiologically based expression for respiratory frequency since this could become an important interfacing component for the respiratory-pulsatile cardiovascular system model.

1.5 Acknowledgements

The author wishes to acknowledge the NASA-ASEE Summer Faculty Research Program for its support during particular phases of the study. Continuation of the research was supported in part by the General Electric Company (Purchase Order No. 036-E31001-T1494) and the Kansas State University Engineering Experiment Station. Special gratitude is extended to the personnel of the General Electric Company for their system programming and evaluation support in addition to Mr. John Schmalzel of Kansas State University.
2. EVALUATION OF EXERCISE SUBROUTINE MODIFICATIONS IN THE RESPIRATORY SYSTEM MODEL

2.1 Statement of Objectives
With the respiratory control system model functioning as an independent system, certain modifications of the exercise subroutine allow for a more realistic response to exercise stimulation. These modifications apply to both on- and off-transient responses. Evaluation of these modifications which preserve the neural and humoral control of ventilation is desired.

2.2 System Response Before Implementation of Modifications
The discussion in this section refers to the respiratory system functioning independently with all modifications relating to the computer program as listed in the Appendix of Gallagher's report. Some of the modifications are included in the integrated circulatory-respiratory system model in addition to the modifications required for the interfacing of the two systems.

For implementation and evaluation of exercise subroutine modifications for the respiratory control system simulations, a spectrum of exercise levels were simulated. Appropriate magnitudes and lengths of exercise levels were implemented. These ranged in magnitude from the resting state to a submaximal exercise level of 250 watts and in durations of 2.5 to 12.0 minutes depending upon the exercise increment.

Although the simulation provides approximately 60 physiologically oriented output variables, the following variables were closely monitored so as to determine the exercise simulation deficiencies.

(a) inspired ventilation rate ($V_I$, l/min)
Figures 2 and 3 illustrate the type of responses generated by the system before the modifications were introduced. In general, the greater the exercise level the more pronounced are the variables' transient responses. If one would superimpose a variable's response for a wide range of exercise levels a family of curves illustrating trends in the variable's response to exercise stimulation would be evident. After careful examination of the system's responses to each exercise level the 200 watt level was chosen as the base run from which exercise subroutine modifications were evaluated.

For the following discussion of the simulation deficiencies refer to Figure 3. Inspired ventilation, \( V_I \), contains a reasonable neurological component as illustrated by the immediate on-set of ventilation when exercise is initiated.

This is followed by a slowly rising \( V_I \) response, the humoral component. Sufficient ventilation is not available in order to properly regulate the arterial \( O_2 \) and \( CO_2 \) tensions as demonstrated by these responses. The inadequate blow-off of \( CO_2 \) is reflected in the increased concentration of the \( H^+ \) ion in the CSF compartment. Since the CSF compartment's \( H^+ \) ion is an important regulator of

- \( c \) cerebrospinal fluid \( H^+ \) concentration \( (C_{CSF}(H^+)) \), nanomoles/l CSF
- \( d \) arterial \( O_2 \) tension \( (P_a(O_2)) \), mm Hg
- \( e \) tissue \( O_2 \) metabolic rate \( (MRT(O_2)) \), l/min
- \( f \) tissue \( CO_2 \) metabolic rate \( (MRT(CO_2)) \), l/min
- \( g \) alveolar respiratory quotient \( (Alv \, RQ) \)
- \( h \) cardiac output \( (Q,L/min) \)
- \( i \) brain blood flow \( (Q_B \, L/min) \)
Figure 2. Selected variables from the respiratory system simulation of an exercise (work load) excitation of 150 watts prior to exercise subroutine modifications.
Figure 3. Selected variables from the respiratory system simulation of an exercise (work load) excitation of 200 watts prior to exercise subroutine modifications.
ventilation one would expect to see an increase in the ventilation rate; however, this regulatory component is of minor importance in exercise simulations with normal environmental conditions, i.e. physiologically compatible atmospheric gaseous mixtures.

Also evidenced from Figure 3 is the simulation's inadequate regulation of cerebral blood flow. This response correlates with an insufficient ventilation rate.

The off-transient exercise response provides a ventilation rate that exceeds the necessary rate. In other words, the ventilation rate causes an increase in arterial $O_2$ tension and a decrease in arterial $CO_2$ tension which could be smoothed with proper modification of the exercise subroutine. In addition, the piece-wise linear off-transient $V_I$ response is not physiologically justifiable.

2.3 System Response After Implementation of Modifications

Three basic modifications were implemented in the respiratory system program yielding improved simulations. These changes are reflected in the system responses for the respiratory control system independent of the circulatory system simulation. Consequently, these program changes are not contained in the program listing in the Appendix. Variations of some of these expressions with the integrated system are discussed in Section 3.

In compliance with Astrand and Rodahl (9) a change in the functional relationship between steady-state $O_2$ requirement and exercise level ($SSO2W(WORK)$) was made. The functional discontinuities listed in the statements of subroutine $SSO2W(X)$ were removed yielding the relationship

$$SSO2W(X) = \begin{cases} 
(X/75.) + .215*(75.-X)/75., & 0 \leq X < 75 \\
- .072 + X/70., & 75 \leq X < 250 \\
3.5, & X > 250 
\end{cases}$$

for $X = WORK$  \hspace{1cm} (2-1)
For a 200-watt exercise level this change increased the steady-state $O_2$ requirement by 4.5% to 2.785 l/min and correspondingly increased steady-state ventilation approximately 6.4%. (10) Thus, this subroutine dictates the steady-state level of required $O_2$ for an exercise stimulus and can be easily altered to meet individual demands.

Another modification which improved the ventilation response and consequently the regulatory aspects of the system involved the exponential functional expression that describes the on- and off-transient ventilation responses. $VTIME$ is an expression in subroutine RC12 which indirectly describes the dependency of ventilation upon magnitude and duration of exercise levels. The $VTIME$ expression which was applicable for both the on-transient and off-transient responses to exercise stimulation was originally programmed as

$$VTIME = \frac{TCT \times (CXT - TIMEON)}{9.2}.$$  \hspace{1cm} (2-2)

Modification of this expression yielded for the on-transient case the expression

$$VTIME = 1.1 - 1.1 \times \exp\left(-\frac{TCT \times (CXT - TIMEON)}{1.92}\right)$$ \hspace{1cm} (2-3)

and for the off-transient case

$$VTIME = 1.1 - 1.1 \times \exp\left(-\frac{TCT \times (CXT - TIMEON)}{3.84}\right).$$ \hspace{1cm} (2-4)

Here,

1/TCT = time constant associated with the exponential functions related to exercise levels.

CXT = simulated time, and

TIMEON = time for initialization of new exercise level.
Figure 4 illustrates the responses utilizing the forementioned modifications. The modifications retain the neurological component of inspired ventilation, $V_I$, and allows steady-state ventilation to be approached much more swiftly with a slight positive derivative in the ventilation rate prevailing until exercise is terminated. The faster response in ventilation rate, which is more acceptable physiologically speaking, provides good regulation of the arterial $O_2$ and $CO_2$ tensions during the initial portion of the exercise stimulus in addition to good regulation of cerebral blood flow.

Although not a critical problem, the off-transient response needs some refinement. The build-up of $H^+$ concentration in the CSF compartment suggests further modifications in the metabolism formulations. Cerebral blood flow, although not unstable, possesses a response which should be smoother.

To further illustrate the capabilities of the system to respond to a variety of exercise levels a 40-minute simulation involving the following series of exercise levels was run.

<table>
<thead>
<tr>
<th>Exercise level (Work load, watts)</th>
<th>Duration (min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>0.5</td>
</tr>
<tr>
<td>40</td>
<td>2.0</td>
</tr>
<tr>
<td>100</td>
<td>3.0</td>
</tr>
<tr>
<td>150</td>
<td>5.0</td>
</tr>
<tr>
<td>0</td>
<td>7.0</td>
</tr>
<tr>
<td>150</td>
<td>5.0</td>
</tr>
<tr>
<td>250</td>
<td>5.0</td>
</tr>
<tr>
<td>0</td>
<td>12.5</td>
</tr>
</tbody>
</table>

The system variables, $V_I (l/min)$, $C_{CSF}(H^+)$ (nanomoles/lCSF), $Q_B (l/min)$, $P_a(CO_2)$ (mm Hg), and $P_a(O_2)$ (mm Hg) are shown in Figure 5. Justifiable regulation of $P_a(CO_2)$ and $P_a(O_2)$ is achieved. $Q_B$
Figure 4. Selected variables from the respiratory system simulation of an exercise (work load) excitation utilizing exercise subroutine modifications.
Figure 5. Selected variables from the respiratory system simulation of several exercise (work load) excitations utilizing exercise subroutine modifications.
is regulated during on-transient periods and for low to medium exercise levels; however, the off-transient of $Q_b$ corresponding to sub-maximal exercise levels exhibits poor control which is supportive of the statements made in the previous paragraphs.

The third modification which could be implemented without appreciable sacrifice in the simulation's fidelity involves the differential equation Subroutine RCI3. Since the system's variables are not rapidly varying as a function of time the thesis is the following. There is no justifiable requirement for a differential equation subroutine having the capabilities of a 4th order Runge-Kutta method and an Adams-Moulton predictor-corrector scheme. Using this criterion the following subroutine may be substituted for the forementioned method. Note that the program listing in the Appendix does not contain this substitution.

```fortran
C(35) = C(35) + C(36)
CALL RCI4
DO 1350 I = 1, M
1350 C(I) = C(I) + C(36)*DC(I)
RETURN
END
```

Here $C(35)$ = time,
$C(36)$ = time increment,
$C(I)$ = system variable, and
$DC(I)$ = derivative of system variable.

With preliminary evaluations of this modification there was demonstrated a small variation in the responses of the simulations using the two differential equation routines in the initial transient responses. Since the variables are changing most rapidly during this
time interval this phenomenon should be expected. With due consideration given to the variety of simulations that have been performed with the respiratory model it appears that this modification would present the most difficulties when simulating extreme variations in environmental gaseous concentrations. Simple exercise stimulus variations should be handled without difficulty.
3. INTEGRATION OF PHYSIOLOGICAL SYSTEMS

3.1 Statement of Objectives

One of the major objectives of the overall research effort is to identify appropriate variables and subsystem components that can be utilized in an integration scheme for the physiological system models. Specific concentration within the present research effort is with the circulatory and respiratory systems. Supporting the objective is the establishment of an evaluation procedure. With exercise being the primary stimulus the proposed integration scheme for the circulatory-respiratory system is evaluated for both normal and stressed physiological system states providing partial fulfillment of the objectives. Section 3.5 presents descriptive material aligned with the objectives of the integration plans involving other physiological systems.

3.2 Circulatory-Respiratory System Integration Scheme

In Section 1.3 a description, including a block diagram (Figure 1), of the integrated circulatory-respiratory system model was presented. The three stimuli - exercise, parameter, and environmental variations - were discussed with reference to their important forcing capabilities. This section contains a discussion of the interfacing component of the overall system. Refer to Figure 1 for the pictorial representation.

With regard to the exercise phenomenon it is important to consider both aerobic and anaerobic oxygen deficits and debts. As an improvement upon the formulation of the O₂ metabolic rate for the tissue compartment (humoral forcing component of inspired ventilation) in the respiratory system simulation the following interface
was established. The total \( O_2 \) metabolic rate for the body is given as

\[
\frac{(RMO + DOB)}{1000} = \frac{RMT(2) + C(26)}{1000} = \frac{MR_T(O_2) + MR_B(O_2)}{1000}
\]  

(3-1)

where

\[ RMO \] = rate of \( O_2 \) delivery to the muscle tissue, ml/min.,
\[ DOB \] = rate of \( O_2 \) delivery by the blood to the non-muscle tissue, ml/min.,
\[ RMT(2) \] = \( MR_T(O_2) \) = metabolic rate of \( O_2 \) in the tissue compartment, l/min., and
\[ C(26) \] = \( MR_B(O_2) \) = metabolic rate of \( O_2 \) in the brain compartment, l/min.

\( RMO \) and \( DOB \) taken from the circulatory system are functions of several physiological variables; thus yielding a more reasonable description of \( O_2 \) requirements during exercise. The calculation of the metabolic production rate of \( CO_2 \) in the tissue compartment is retained in the respiratory system simulation. Also, direct neurological control of ventilation related to exercise levels is retained in the respiratory model.

The total cardiac output is an important component of both systems. The circulatory system simulation describes cardiac output as a weighted expression of several physiological attributes.

Here

\[
QLO = (QLN)(LVM)(HSL)(AUH)(HMD)(HPL)
\]  

(3-2)

where

\[ QLO = \] left ventricle output, l/min
\[ QLN = \] output of left ventricle under normal conditions, l/min.,
LVM = effect of arterial pressure loading factor on left ventricle,
HSL = basic strength of left ventricle,
AUH = degree of autonomic stimulation of left ventricle,
HMD = degree of deterioration of left ventricle caused by low coronary blood flow, and
HPL = degree of hypertrophy of left ventricle.

Refer to Guyton (3) and White (6) for a detailed description of the terms in Eq. (3-2).

In the respiratory system model cardiac output, $Q = C(10)$, is described by a first-order differential equation and depends only on specific levels of arterial $O_2$ and $CO_2$ tensions. (2) Based upon the comparison of these two formulations and the complexity of altering each, the decision was made to allow the respiratory system component to receive cardiac output from the circulatory system component. The formulation of cerebral blood flow is retained in the respiratory system.

Another interfacing component involves the blood oxygen capacity. In the respiratory system this component is a constant ($Hb = C(17)$) alterable by an input data card. In the circulatory system this term is continuously calculated, thus it is provided to the respiratory system as

$$\frac{HM \cdot OSA}{200} = Hb = C(17')$$

(3-3)

where

$HM$ = hematocrit
OSA = arterial oxygen saturation, and
$Hb$ = arterial blood oxygen capacity, $1\text{ }O_2/1\text{ blood.}$
In turn, the oxygen volume attached to hemoglobin in the aortic blood is developed in the respiratory system. The circulatory system utilizes this term in the expression

\[ \text{OVA} = 1000 \cdot \text{CHBA} \]

where

\[ \text{CHBA} = C_a(HbO_2) = \text{arterial hemoglobin concentration}, \]

\[ 1 \text{ O}_2/1 \text{ blood}, \]

\[ \text{OVA} = \text{oxygen volume attached to hemoglobin in aortic blood}. \]

This latter interface relationship is important in providing a possible pathway to the circulatory system for an environmental stimulus (alterations in gaseous composition). Since the venous blood in the respiratory model is not correlated with any particular venous site in the circulatory model, the calculations of tissue venous hemoglobin concentration \( C_vT(HbO_2) \) is retained in the individual models. The forementioned features of the interface system contribute to a more physiologically reliable representation of the variables than formerly existed in the individual systems.

The complete computer program for the integrated system is listed in the Appendix. However, there are some modifications which should be mentioned in comparing the individual respiratory system program with the respiratory system component of the integrated program. The original format and namelist statements are changed to comment statements.

The following comment statements are listed at the beginning of the respiratory system program, SUBROUTINE GRODIN. These identify the flow of variables between the two systems.
FOLLOWING FROM GUYTON TO GRODIN.

<table>
<thead>
<tr>
<th>GUYTON</th>
<th>GRODIN</th>
</tr>
</thead>
<tbody>
<tr>
<td>QLO</td>
<td>C(10)</td>
</tr>
<tr>
<td>(RMO+DOB)/1000.</td>
<td>RMT(2)+C(26)</td>
</tr>
<tr>
<td>(HM*OSA)/200.</td>
<td>C(17)</td>
</tr>
<tr>
<td>URZ4</td>
<td>URZ4</td>
</tr>
<tr>
<td></td>
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<td></td>
<td></td>
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FOLLOWING FROM GRODIN TO GUYTON

<table>
<thead>
<tr>
<th>GUYTON</th>
<th>GRODIN</th>
</tr>
</thead>
<tbody>
<tr>
<td>OVA/1000.</td>
<td>CHBA</td>
</tr>
</tbody>
</table>

In Subroutine RC12 the Guyton segment of the program is sent the work load change.

In order for the circulatory and respiratory systems' programs to be compatible with their formulation of oxygen requirements for various exercise levels, modifications were made in Subroutines FUNCTION SS02W(X) and FUNCTION SSVENT(X). The original Subroutine FUNCTION SS02W(X) is presented below.
FUNCTION SS02W(X)
C CALCULATION OF STEADY-STATE OXYGEN REQUIREMENTS FOR VARIOUS LEVELS
C OF WORK LOAD (X = WATTS).
    IF(X.GT.250.) GO TO 1
    IF(X.LT.75.) GO TO 2
    SS02W = -.072 + X/70.
RETURN
1 SS02W = 3.5
RETURN
2 SS02W = (X/75.) + .215*(75.-X)/75.
RETURN
END

The modification of the above subroutine yields

FUNCTION SS02W(X)
C CALCUALTION OF STEADY-STATE OXYGEN REQUIREMENTS FOR VARIOUS LEVELS
C OF WORK LOAD (X = WATTS).
    SS02W = .195 + (X/84.15)
    IF (X.GT.210.) SS02W = 2.7
RETURN
END

This modification produces a reduced level of steady-state oxygen requirement compared to the original respiratory program.

The original Subroutine FUNCTION SSVENT(X) is presented below.

FUNCTION SSVENT(X)
C CALCULATION OF STEADY-STATE VENTILATION RATE AS A FUNCTION
C OF TISSUE 'OXYGEN METABOLIC RATE
    IF (X.LE..215) SSVENT = 5.398
IF((X.GT.215).AND.(X.LT.2))SSVENT = 25.*X
IF(X.GE.2.)SSVENT = 50.+50.*(X-2.)
RETURN
END

The modification of the above subroutine yields

FUNCTION SSVENT(X)
C CALCULATION OF STEADY-STATE VENTILATION RATE AS A FUNCTION
C OF TISSUE OXYGEN METABOLIC RATE
IF(X.LE.195)SSVENT = 5.398
IF(X.GE.2.)SSVENT = 55.36 + 50.*(X-2.)
IF((X.GT.195).AND.(X.LT.2))SSVENT = 27.68*X
RETURN
END

A necessary condition that must be fulfilled for the integrated system to function properly is that both subsystems must be operating with the same steady-state values prior to application of an exercise stimulus. After steady-state conditions are established for any particular type of normal or abnormal environmental or physiological condition, the integrated system components will function in concert for all parameter or stimuli disturbances. One key used in determining when steady-state conditions are reached is the observance of a minimal change in variable values $C(1) - C(14)$ of the respiratory system model. These variables are related to the compartmental gaseous concentrations, cardiac output, and cerebral blood flow.

Documentation of initial physiological variable values are necessary so that both of the systems are functioning under compatible initial conditions for all levels of exercise. As an
example of the type of documentation required, steady-state resting cardiac output is adjusted to 5.12 l/min in both models. All other variables which are dependent upon this variable are adjusted accordingly. Model modifications such as these are deemed necessary if both models are going to function in concert.

The printed output data combines the forms of both individual systems. In addition to the tabular form of the respiratory system model's output, the following circulatory system variables are printed.

SECS = seconds
PA = arterial pressure, mm Hg,
QLO = left ventricular output, l/min.,
PLA = left atrial pressure, mm Hg,
PRA = right atrial pressure, mm Hg,
VP = plasma volume, l,
VPF = free fluid in interstitial spaces of lungs, l,
VTS = total interstitial fluid volume, l,
VUD = urinary output, l/min.,
RMO = rate of oxygen delivery to muscle tissue, ml/min., and
DOB = rate of oxygen delivery to non-muscle tissue, ml/min.

Also, special plotting routines are available and provide for excellent qualitative analysis and indicate variable trend setting phenomena. This feature of the simulation set-up is very useful when one is observing trends in a variable's response to a family of excitations, i.e. exercise levels.

3.3 Exercise Response for Nonstressed Physiological System States

As described in the previous section, in order to establish a base run, appropriate initial conditions have to be obtained for the respiratory system component. Basically, cardiac output and
metabolic rates must be compatible between the two systems.

The input data as shown in Appendix 6.1, Table 2 of the reference by Gallagher (7) was utilized with the following modifications.

<table>
<thead>
<tr>
<th>Input Data Card No.</th>
<th>Variable</th>
<th>New Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>Q</td>
<td>5.1200</td>
</tr>
<tr>
<td>25</td>
<td>MRB(CO2)</td>
<td>.0450</td>
</tr>
<tr>
<td>26</td>
<td>MRB(O2)</td>
<td>.0450</td>
</tr>
<tr>
<td>31</td>
<td>FI(CO2)</td>
<td>.0004</td>
</tr>
<tr>
<td>32</td>
<td>FI(O2)</td>
<td>.2096</td>
</tr>
<tr>
<td>33</td>
<td>FI(N2)</td>
<td>.7900</td>
</tr>
<tr>
<td>39</td>
<td>PRINT AL TIM</td>
<td>.2000</td>
</tr>
<tr>
<td>45</td>
<td>RMT(CO2)</td>
<td>.1716</td>
</tr>
<tr>
<td>46</td>
<td>RMT(O2)</td>
<td>.1950</td>
</tr>
</tbody>
</table>

Table 1. Input data cards reflecting changes in cardiac output and metabolic changes under normal environmental gaseous conditions.

This simulation was allowed to run until steady-state conditions were reached, i.e. changes in C(1) - C(14) were minimal. These values for C(1) - C(14) were then established as the input data for normal environmental conditions. The variable values obtained for the steady-state conditions are listed in Table 2.

<table>
<thead>
<tr>
<th>Input Data Card No.</th>
<th>Variable</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>FA(CO2)</td>
<td>.1767</td>
</tr>
<tr>
<td>2</td>
<td>FA(O2)</td>
<td>.5338</td>
</tr>
<tr>
<td>3</td>
<td>FA(N2)</td>
<td>.2895</td>
</tr>
<tr>
<td>4</td>
<td>CB(CO2)</td>
<td>.6345</td>
</tr>
<tr>
<td>5</td>
<td>CB(O2)</td>
<td>.0012</td>
</tr>
<tr>
<td>6</td>
<td>CB(N2)</td>
<td>.0011</td>
</tr>
<tr>
<td>7</td>
<td>CT(CO2)</td>
<td>.6142</td>
</tr>
</tbody>
</table>
Table 2 Continued

<table>
<thead>
<tr>
<th>Card No.</th>
<th>Variable</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>8</td>
<td>CT(02)</td>
<td>0.0014</td>
</tr>
<tr>
<td>9</td>
<td>CT(N2)</td>
<td>0.0013</td>
</tr>
<tr>
<td>10</td>
<td>Q</td>
<td>5.1554*</td>
</tr>
<tr>
<td>11</td>
<td>QB</td>
<td>0.7391</td>
</tr>
<tr>
<td>12</td>
<td>PCSF(CO2)</td>
<td>46.3498</td>
</tr>
<tr>
<td>13</td>
<td>PCSF(O2)</td>
<td>38.4441</td>
</tr>
<tr>
<td>14</td>
<td>PCSF(N2)</td>
<td>70.6931</td>
</tr>
</tbody>
</table>

*Perhaps Q = 5.12 should be used for complete compatibility with the circulatory system.

Table 2. Initial conditions for physiological variables under normal environmental conditions.

Included here is a brief description of a simulation run for the nonstressed physiological system which correlated very well with a simulation performed with the individual respiratory system. Refer to Figure 5. The stimuli were a series of exercise levels with specific durations as shown here.

<table>
<thead>
<tr>
<th>Exercise Level (EXC)</th>
<th>Work load, watts</th>
<th>Duration (min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.000</td>
<td>0</td>
<td>0.5</td>
</tr>
<tr>
<td>12.900</td>
<td>40</td>
<td>3.0</td>
</tr>
<tr>
<td>38.500</td>
<td>100</td>
<td>3.0</td>
</tr>
<tr>
<td>65.880</td>
<td>150</td>
<td>5.0</td>
</tr>
<tr>
<td>1.000</td>
<td>0</td>
<td>9.0</td>
</tr>
</tbody>
</table>

For each of the exercise levels and their corresponding transient and steady-state responses the integrated system provided a
slightly increased cardiac output, inspired and expired ventilation rates, alveolar RQ, tissue $P_O_2$, and decreased arterial and tissue $P_CO_2$, arterial $P_O_2$, and cerebral blood flow. All other physiological variables were altered accordingly. Although no definite trend was established, the VI-VE difference was altered when compared to the independently functioning respiratory system.

Similar types of unstressed physiological system simulations were performed under altered environmental conditions. To evaluate the initial conditions for the respiratory system model the input data as shown in Appendix 6.1, Table 2 of the reference by Gallagher (7) was used with the following modifications.

<table>
<thead>
<tr>
<th>Input Data Card No.</th>
<th>Variable</th>
<th>New Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>Q</td>
<td>5.1200</td>
</tr>
<tr>
<td>25</td>
<td>MRB(CO2)</td>
<td>.0450</td>
</tr>
<tr>
<td>26</td>
<td>MRB(O2)</td>
<td>.0450</td>
</tr>
<tr>
<td>30</td>
<td>B</td>
<td>260.0000</td>
</tr>
<tr>
<td>31</td>
<td>FI(CO2)</td>
<td>.0192</td>
</tr>
<tr>
<td>32</td>
<td>FI(O2)</td>
<td>.7000</td>
</tr>
<tr>
<td>33</td>
<td>FI(N2)</td>
<td>.2808</td>
</tr>
<tr>
<td>39</td>
<td>PRINT AL TIM</td>
<td>.2000</td>
</tr>
<tr>
<td>45</td>
<td>RMT(CO2)</td>
<td>.1716</td>
</tr>
<tr>
<td>46</td>
<td>RMT(O2)</td>
<td>.1950</td>
</tr>
</tbody>
</table>

Table 3. Input data cards reflecting changes in cardiac output and metabolic changes under altered environmental conditions.

This simulation was allowed to run until steady-state conditions were established. Input data cards C(1) - C(14) shown below correspond to steady-state conditions for the altered environmental conditions.
As illustrated by the values of Table 4 the combination of barometric pressure and volumetric gas fractions of the altered environment provided a physiological condition similar to that experienced at sea level. An interesting experiment involved implementing the conditions as illustrated in Tables 3 and 4 and observing the effects of exercise upon the system. On- and off-transient exercise conditions were simulated for levels of 50, 100, 150, and 200 watts. Each level was of a duration that allowed for the physiological variables to attain or approach their steady-state conditions. Results were similar to those obtained for the normal environmental conditions using the integrated system or for the respiratory system functioning independently.
3.4 Exercise Response for Stressed Physiological System States

A significant benefit is achieved in using the integrated system as a means of determining respiratory system response to exercise when the circulatory system is not optimally functioning. In this way one can demonstrate the dependency of the respiratory system, in particular the ventilation rates, upon abnormal (stressed) functioning of the circulatory system. Thus, by observing easily monitored physiological variables noninvasive evaluation of circulatory system malfunctioning is achieved.

The simulations involving exercise stimulation and the stressed circulatory system are implemented in the following manner. Under the influence of normal or physiologically compatible environmental conditions the circulatory system is stressed (parameter variation). Examples of stressful situations include malfunctioning of the renal system, excessive fluid intake, unilateral or bilateral heart failure, regional volume loading, anemia, or hematocrit level variations. All of these are situations that could have been encountered in recent spaceflights or while performing normal routine earth-bound tasks; consequently, interest is keen on such simulations. Steady-state conditions are then established for the stressed condition with the appropriate variable changes realized for the respiratory system. The exercise stimulus is then applied to the integrated system.

Justification for the above sequence of steps is stated in the following manner. Two types of systems are involved - a long-term model (circulatory) and a short-term model (respiratory). For a given stressed circulatory system state several hours or days of simulated time might be required for the system to approach steady-state conditions. Simulation with the integrated system is not
necessary for this period of time. Only after stressed steady-state conditions are reached is it necessary for the integrated system to become operative. Exercise stimulation is associated with rapid transient responses. It is applied for short periods of time; therefore, the short-term model is compatible for this time segment.

Examples of variable responses for a stressed circulatory system under the influence of an exercise stimulus are shown in Figures 6-8. The stressed circulatory state was a bilateral heart failure. The pumping capabilities of both the left and right ventricles were reduced, simulating a mild heart attack. A simulated recovery period of approximately 2 weeks established the stressed steady-state conditions. Responses to various levels of exercise were observed with a comparison of 100 and 150 watt exercise stimuli illustrated in Figures 6-8. The exercise stimulus was applied for 5 minutes followed by a 5 minute period of no exercise. Included in these illustrations are only a few of the more significant variable responses. Direct comparison of the responses for the two exercise levels illustrates the dependency of each variable upon exercise stimulation for the particular stressed state. A more detailed comparison may be observed from the tabular output data. Many details need to be refined before this particular system can be faithfully used in a quantitative predictive and/or diagnostic role. However, preliminary studies have been encouraging.

3.5 Interfacing Possibilities for Physiological Systems' Models

This section contains a brief summary of some possible integration schemes for the respiratory, cardiovascular, thermoregulatory, and the circulatory (long-term) system models. Even
Figure 6. Arterial pressure and inspired ventilation rate for 100 watt (---) and 150 watt (-----) exercise levels.
Figure 7. Cardiac output and tissue oxygen pressure for 100 watt (— — —) and 150 watt (——) exercise levels.
Figure 8. Rate of oxygen delivery by the blood to muscle (RMO) and non-muscle (DOB) tissues for 100 watt (---) and 150 watt (-----) exercise levels.
though there remains considerable uncertainty involving the de-
tails of the interfacing plans the overall goal has some defini-
tive aspects. The objective is a whole-body algorithm which sim-
ulates physiological system responses to specific conditions and
excitations directly related to those encountered in recent man-
ned spaceflights (Skylab).

Since there is a logical separation in the classification of
models, long-term and short-term, it seems advisable to mold a
total system which minimizes the interfacing structure and uses
the aspect of simulated time to its best advantage. Thus, a
plan which seems most encouraging at this time utilizes a separ-
ation between short-term and long-term models. Each of the short-
term models will interact with the other short-term models yielding
appropriate transient responses for the stimuli.

The respiratory and pulsatile cardiovascular system models' inter-
face relies heavily upon compartmental blood flows and blood
gas (O₂ and CO₂) concentrations. The respiratory model would re-
ceive the following variables: cardiac output, cerebral blood
flow, pulmonary vascular volume, and metabolic rate. The role of
metabolic rate as a function of exercise would be handled in the
respiratory system in a similar manner as described in the inter-
face of the circulatory-respiratory system (Section 3.2).

The respiratory model would be responsive to environmental
conditions. It would supply the cardiovascular system with a
physiologically justified description of respiratory frequency,
arterial CO₂ and O₂ concentrations, and an intrathoracic pressure
variable. Some of the weaker features of the respiratory system
model will be enhanced by inputs from the cardiovascular system.
A similar statement applies for transferal of information in the
opposite direction. The two models must be made compatible for the resting steady-state case. To allow for this compatibility to exist it is necessary to establish the empirical a-v $O_2$ difference curve for a range of exercise levels as formulated by the individual respiratory program.

A minimal degree of compartment redefining seems necessary with the above approach. If additional compartmentalization is deemed necessary the tissue compartment of the respiratory system could be subdivided to correspond to the legs, abdominal cavity, thoracic cavity, and upper extremities as described in the cardiovascular system model. A restructuring of all blood flows and metabolic formulations would be necessary. With minor modifications the other two compartments, brain and lungs, and their associated variables could be related to the head and pulmonary segments in the cardiovascular system.

The void of a compatible definition for compartmental blood flow and the dependency of compartments on the exercise phenomenon are two major obstacles in the integration of the respiratory and thermoregulatory systems. A more refined definition of blood flow may be achieved in the thermal model if a transition is made from lumped core regions to distributed core regions. However, this change would not yield itself to an easily implemented interface since all of the core, muscle, fat, and skin are lumped into a tissue compartment in the respiratory system.

Under the influence of exercise it is reasonable to assume that the muscle compartment plays the dominating role as far as $O_2$ and $CO_2$ functions are concerned. Thus, the interrelationships among blood level parameters could be defined as follows. Arterial $O_2$ and $CO_2$ tensions could be supplied to the muscle model along
with a fractional supply of total cardiac output. In return the muscle compartment would supply a description of O₂ and CO₂ metabolic rates, and O₂ and CO₂ gas tensions which would be interpreted as tissue compartment variables in the respiratory system.

The importance of the effects of the environmental temperature should be included in the respiratory component of the respiratory-thermoregulatory system. Temperature effects on total cardiac output (related to skin blood flow), ventilation rates, and metabolic rates are important. Additional terms or functions which are proportional to selected temperature deviations might be added to the calculation of cardiac output and the ventilatory controller equation to compensate for environmental temperature variations.

Sections 3.2 - 3.4 presented an integration of the respiratory and circulatory systems. It allowed for a minimal reorganization of the existing systems and yet simulated with reasonable accuracy the influence of the exercise stimulus. Further compartmentalization of the respiratory system would no doubt be a part of the next level of complexity if the integration effort between these two systems were extended. With emphasis on the exercise stimulus the tissue compartment in the respiratory system would be subdivided into (1) muscle, (2) renal, and (3) non-muscle, non-renal, and fat compartments. This partitioning would be in agreement with the major compartments of the circulatory system model. Blood flow and metabolism for each of the compartments would be developed as functions of O₂ requirements and CO₂ production in addition to normal basal levels and neural control. The combined blood flow of these compartments plus the cerebral blood flow would total the cardiac output. In comparison to the interface system
described in Section 3, total cardiac output might be handled in the same manner.
4. RESPIRATORY FREQUENCY FORMULATION

4.1 Statement of Objectives

Respiratory frequency is a term which can be related to the establishment of a sufficient \( \text{O}_2 \) supply and the proper venting of \( \text{CO}_2 \). It is also an important driving force in the pulsatile cardiovascular system thus possessing the potential use as an interfacing component for the respiratory and pulsatile cardiovascular system model. Consequently, a physiologically compatible expression for respiratory frequency is desired.

4.2 Proposed Respiratory Frequency Expression

Separation of the factors causing ventilation rate, respiratory frequency, and the different pulmonary lung volumes is practically impossible. Some humoral and neural control mechanisms have been experimentally justified while many others remain undefined.

Carbon dioxide can stimulate the respiratory centers directly and indirectly through the carotid and aortic chemoreceptors. For example, hyperventilation which decreases \( \text{P}_{\text{ACO}_2} \) by several millimeters of Hg results in a decrease in ventilation. Variations in \( \text{CO}_2 \) concentration necessarily involve changes in blood pH due to the combination of \( \text{CO}_2 \) with \( \text{H}_2\text{O} \) to form carbonic acid, and the subsequent dissociation to form \( \text{H}^+ \) ions. This confounds efforts to distinguish \( \text{CO}_2 \) from the \( \text{H}^+ \) ion effects. There are small chemosensitive respiratory areas on the surface of each side of the medulla oblongata near the entry point of the 8th, 9th and 10th cranial nerves. This region is very sensitive to changes in \( \text{H}^+ \) ion concentration in the cerebrospinal fluid (CSF). Because \( \text{CO}_2 \) diffuses from the
bloodstream into the CSF compartment much more rapidly than do free H⁺ ions, the stimulus to the medullary centers is primarily due to CO₂. The impact of the CSF compartment's H⁺ ion concentration is discussed in detail by Gallagher (7) including changes in its weighting in the ventilation controller equation.

Changes in arterial O₂ concentration to levels below normal also have a strong stimulating effect on the chemoreceptors located bilaterally in the bifurcations of the common carotid arteries (carotid bodies) and along the arch of the aorta (aortic bodies). Their afferent neurons pass to the medulla through Hering's nerves to the glossopharyngeal nerves and through the vagi respectively.

Understanding neural control of respiration is more elusive. In addition to neural pathways in the medulla that seem to have inherent inspiratory and expiratory patterns of rhythmicity, there are also other neural factors that influence respiration. Stretch receptors located in the visceral pleura and throughout the lungs give rise to the "Hering-Breuer" stretch reflexes which act through the vagus nerve to inhibit continued inspiration (or expiration) to certain limits of expansion or contraction. The Hering-Breuer reflex also contributes to cyclic respiratory rhythmicity.

There are also other stretch receptors located throughout the body in joints and muscle tissues that give proprioceptive feedback. The immediate emotional state of the organism also acts to modify respiratory patterns. Speech, fear, rage and other stresses serve to alter respiration.

One approach to the establishment of a respiratory frequency expression involves a work formulation. In this context work is associated with respiration itself. Otis, et al. (11) found that for low levels of ventilation the frequency of breathing could be fairly
accurately predicted by utilizing a minimum rate of work criterion. The problem definition assumed a sinusoidal airflow pattern, passive expiration, a linear elastic element and nonlinear resistance. Differential work was defined as

\[ dW = A(t) + B(t) + C(t) \]

where

\[ dW = KVdV + K^1 a^2 \sin^2 bt \, dt + K^1 a^3 \sin^3 bt \, dt \quad (4-1) \]

By graphing elastic, viscous, turbulent, and total work as functions of respiration frequency there emerged a frequency that minimized work for a constant alveolar ventilation. Also, it was determined that for too low a frequency a greater amount of elastic work was required to produce the large tidal volumes. In addition, for high frequencies, work was wasted in ventilating the dead space with each breath.

Yamashiro and Grodins (12), assuming negligible turbulent resistance \((C(t) \rightarrow 0)\) and noting that the terms in the series expansion of \(B(t)\) were orthogonal with respect to the period \(T\), minimized the rate of work done in breathing by using the equation

\[ \frac{dW}{dt} = W_f = KfV_T^2/2 + K^1 \sum_{i=1}^{\infty} a_i^2 / 4 \quad (4-2) \]

and the constant alveolar ventilation and dead space constraint,

\[ V_T = V_D + V_A/f. \quad (4-3) \]

Terminology used in Equations 4-2 and 4-3 follows:
\[ f = \text{respiratory frequency, bpm} \]
\[ V_T = \text{tidal volume, l} \]
\[ K^1 = \text{total respiratory resistance} \]
\[ a_i = i^{th} \text{ Fourier coefficient of airflow infinite series} \]
\[ V_D = \text{dead space volume, l} \]
\[ V_A = \text{minute alveolar ventilation, l/min} \]

Utilizing an optimization procedure with Equations 4-2 and 4-3 an optimal frequency was obtained as

\[ f_{\text{opt}} = \left( \frac{1 + 32RCV_A/V_D}{4GRC} \right)^{1/2} - 1 \]

with the same terminology as before in addition to

\[ G = \text{time constant associated with resistance and elastance of the tissues. (Experimentally determined to be .015 min)} \]

Yamashiro and Grodins (12) stated that since the airflow pattern was assumed periodic with no specific describing function the optimal work was achieved with a constant flow rate during inspiration and a decreasing exponential flow rate during expiration. This was associated with passive breathing.

Examination of predicted optimal respiratory frequencies for optimal and sinusoidal airflow patterns indicate that sinusoidal airflow can require up to a 11.7% increase in work cost when compared to the optimal airflows. The situation is altered if active expiration is considered. For active expiration the optimal airflow pattern assumes the shape of a square wave with maximum and minimum values of \( \pm 2(V_D f + V_A) \). Comparison with sinusoidal airflow indicates a 23.4% work cost increase if the pattern is sinusoidal.

Research involving active expiration seems to provide the
transition to the study of airflow patterns during exercise. Yamashiro and Grodins (13) examined the minimization of work during exercise when functional residual capacity (FRC), the airflow pattern, and respiratory frequency were included as controlled variables. The result of their derivation yielded the optimal frequency expression

\[ f_{opt} = \left( (1+32((1+a)/a)RCA/V_D)^{1/2}-1 \right) /16((1+a)/a)RC. \] (4-5)

The only term which has not been previously defined is "a". Here "a" is a ratio of inspiratory elastance \( (K_I) \) to expiratory elastance \( (K_E) \).

When correlated with experimentally determined curves, a time constant \( (RC) \) of 0.015 min. was obtained. This compared with a similar value of 0.011 min. in research reported by Mead. (14) The value of "a" was assumed to be 1.95 based on data taken by Rahn, et al. (15)

At levels of ventilation near rest, the experimental data appears consistent with the expression for respiratory frequency associated with a sinusoidal airflow pattern and constant expiratory reserve volume (ERV). This is Equation 4-4. However, at greater respiratory rates, the expression which accounts for FRC (Equation 4-5) provides the best fit with experimental data.

Based upon the previous discussion and the importance of exercise in the simulations associated with the project's goals a recommendation to implement Equation 4-5 in the respiratory program appears reasonable. This formulation would be substituted in Subroutine RC12 for the expression

\[ FREQ = 8.1 + 7.815w(RMT(2) + C(26)) \] (4-6)
which gives an empirical representation of respiratory frequency as a function of $O_2$ utilization. This change would require a modification in the calculation of dead space ventilation. Using the discussions of the cited references an empirical formulation for dead space ventilation as a function of exercise level could be established. Upon implementation of the appropriate variable designations an improvement in the calculation of respiratory frequency is proposed.
5. CONCLUSIONS AND RECOMMENDATIONS

Modifications of the individual respiratory system model have produced a system which simulates the effects of exercise in a manner which is acceptable with regard to regulation of $O_2$ and $CO_2$. The neural and humoral control of ventilation during on- and off-transient exercise stimulation is justified. Likewise, other variables are altered according to the regulation of gas tensions and ventilation rates.

Improvement of physiological system models is always possible. Such is the case with the respiratory frequency expression. With projected plans for using the respiratory frequency as an interfacing variable between the cardiovascular and respiratory systems it is desirable that the expression be valid for the types of simulations performed. The proposed expression has met with success with various levels of exercise as discussed in the references of Section 4. However, there is no sound documentation as to the percentage contribution of humoral and neural control as given by this frequency expression. This constitutes another level of complexity in both experimental and theoretical respiratory research.

To fulfill the goals of the research effort, future work should emphasize the integration of the short-term and long-term models so that an adequate whole-body algorithm is realized. There are many problems that will arise as the integrations of the interacting physiological systems are achieved.

Results of this study are encouraging since a minimal amount of individual system modification was necessary for the integration of the circulatory and respiratory systems. Perhaps features of this approach can be applied to other integration endeavors.
Continued improvement of the integrated systems will help establish a better understanding of the interrelated physiological processes, especially those which are influenced by altered environments or stressed physiological states. Obviously, the continued development of appropriate data integration and display routines should parallel the modeling phases.

The next phase of the project will undoubtedly be an enlightening experience with significant gains made in the establishment of techniques and useful procedures for integrating complex physiological systems. As an additional benefit of the modeling effort it is hoped that correlation of simulated and experimental data will provide potential noninvasive diagnostic and therapeutic capabilities for both stressed and nonstressed physiological states.
6. APPENDIX

6.1 Integrated Circulatory-Respiratory System
* 100 CALL PUTOUT (URZ4, URZ5)

C

CALL GRODIN (URZ1, URZ2, URZ3, URZ4, URZ5, URZ6)

C

CALL FUND (FUN1, FUN2, FUN3, FUN4)

C

CALL FUND (FUN5, FUN6, FUN7, FUN8)

C

CALL FUND (FUN9, FUN10, FUN11, FUN12, FUN13, FUN14, FUN15, FUN16, FUN17, FUN18, FUN19, FUN20, FUN21, FUN22, FUN23, FUN24, FUN25, FUN26, FUN27, FUN28, FUN29, FUN30, FUN31, FUN32, FUN33, FUN34, FUN35, FUN36, FUN37, FUN38, FUN39, FUN40, FUN41, FUN42, FUN43, FUN44, FUN45, FUN46, FUN47, FUN48, FUN49, FUN50, FUN51, FUN52, FUN53, FUN54, FUN55, FUN56, FUN57, FUN58, FUN59, FUN60, FUN61, FUN62, FUN63, FUN64, FUN65, FUN66, FUN67, FUN68, FUN69, FUN70, FUN71, FUN72, FUN73, FUN74, FUN75, FUN76, FUN77, FUN78, FUN79, FUN80, FUN81, FUN82, FUN83, FUN84, FUN85, FUN86, FUN87, FUN88, FUN89, FUN90, FUN91, FUN92, FUN93, FUN94, FUN95, FUN96, FUN97, FUN98, FUN99, FUN100)

C

CALL GRODIN (URZ1, URZ2, URZ3, URZ4, URZ5)

C

CALL GRODIN (URZ1, URZ2, URZ3, URZ4, URZ5, URZ6)

C

CALL FUND (FUN1, FUN2, FUN3, FUN4)

C

CALL FUND (FUN5, FUN6, FUN7, FUN8)

C

CALL FUND (FUN9, FUN10, FUN11, FUN12, FUN13, FUN14, FUN15, FUN16, FUN17, FUN18, FUN19, FUN20, FUN21, FUN22, FUN23, FUN24, FUN25, FUN26, FUN27, FUN28, FUN29, FUN30, FUN31, FUN32, FUN33, FUN34, FUN35, FUN36, FUN37, FUN38, FUN39, FUN40, FUN41, FUN42, FUN43, FUN44, FUN45, FUN46, FUN47, FUN48, FUN49, FUN50, FUN51, FUN52, FUN53, FUN54, FUN55, FUN56, FUN57, FUN58, FUN59, FUN60, FUN61, FUN62, FUN63, FUN64, FUN65, FUN66, FUN67, FUN68, FUN69, FUN70, FUN71, FUN72, FUN73, FUN74, FUN75, FUN76, FUN77, FUN78, FUN79, FUN80, FUN81, FUN82, FUN83, FUN84, FUN85, FUN86, FUN87, FUN88, FUN89, FUN90, FUN91, FUN92, FUN93, FUN94, FUN95, FUN96, FUN97, FUN98, FUN99, FUN100)

C

CALL GRODIN (URZ1, URZ2, URZ3, URZ4, URZ5, URZ6)

C

CALL GRODIN (URZ1, URZ2, URZ3, URZ4, URZ5)

C

CALL FUND (FUN1, FUN2, FUN3, FUN4)

C

CALL FUND (FUN5, FUN6, FUN7, FUN8)

C

CALL FUND (FUN9, FUN10, FUN11, FUN12, FUN13, FUN14, FUN15, FUN16, FUN17, FUN18, FUN19, FUN20, FUN21, FUN22, FUN23, FUN24, FUN25, FUN26, FUN27, FUN28, FUN29, FUN30, FUN31, FUN32, FUN33, FUN34, FUN35, FUN36, FUN37, FUN38, FUN39, FUN40, FUN41, FUN42, FUN43, FUN44, FUN45, FUN46, FUN47, FUN48, FUN49, FUN50, FUN51, FUN52, FUN53, FUN54, FUN55, FUN56, FUN57, FUN58, FUN59, FUN60, FUN61, FUN62, FUN63, FUN64, FUN65, FUN66, FUN67, FUN68, FUN69, FUN70, FUN71, FUN72, FUN73, FUN74, FUN75, FUN76, FUN77, FUN78, FUN79, FUN80, FUN81, FUN82, FUN83, FUN84, FUN85, FUN86, FUN87, FUN88, FUN89, FUN90, FUN91, FUN92, FUN93, FUN94, FUN95, FUN96, FUN97, FUN98, FUN99, FUN100)
IF (ABS(QA)-QRO).GT.4) GC TO 100

CALL NCRMEN

CALL BLOOD

CALL MUSCLE

CALL AUTORS

CALL ADM

CALL BIOSCI

CALL HEART

CALL CAPHOD

CALL PULMON

CALL MISC2

CALL PROBEN

CALL KIDNEY

CALL ICNS

CALL GELFLO

CALL TO 100

SUBROUTINE PUTIN
COMMON/ARRAY/A(400), TITLE(400), COL(20), ALPHA(20)

COMMON/NUMERO/NC(20), C(20)

COMMON/STORE/NGIN3?, NC, NOUT, NGSNG6, NGRNG9, OTTLPTNPNOD

COMMON/TAPE/TOTAL

DATA SECS/ISECS/, IMINMS/I

DATA ALL/ALL/-/, BLANK/''

DIMENSION WATEXC(13, 2)

DATA WATEXC/I, 5, 10., 20., 30., 40., 50., 60., 70., 80., 90., 100., 120.,
1 0., 16., 32., 64., 128., 256., 512.

T=A(1)

ICCNVT IS FLG
1=CONVERT EXC (A(3 9)) TO WATTS FOR GRODIN.
0=DO NOT CONVERT EXC TO WATTS.

ICCNVT = 0
IF(URZ4 .EQ. 1 ) ICCNVT = 1

NTIME = T/1440.

IF(UNITS .EQ. SECS) NTIME = T/60.

IF(UNITS .EQ. MIN) NTIME = T.

IF(UNITS .EQ. HOUR) NTIME = T/60.

IF(INTIME .NE. NTIME) GO TO 65

H=I IF IC PRINT.
6 IF(URZ4 .NE. 1) URZ4 = 2
IF(ALPHA(I) NE. ALL) GO TO 7
WRITE(6, 71) NTIME,UNITS,(TITLE(J), A(I), J=1, MAXNO)
GO TO 51
7 DO 20 I = 1, K

K = A(I)

COL(I) = A(I)

20 CONTINUE
IF(K .EQ. 10) GO TO 70
WRITE(6, 71) UNITS, (ALPHA(J), J=1, K)
21 FORMAT(44/54X,A4,...15X,A4,* = '4,F10.4,4X)
WRITE(6, 31) NTIME,(COL(J), J=1, K)
31 FORMAT(1 I10,2X,F10.4,9(1X,F10.4))
GO TO 51
70 WRITE(6, 71) NTIME,UNITS, (ALPHA(J), J=1, K)
71 FORMAT(56X,15X,A4,...15X,A4,* = '4,F10.4,4X)
51 NTIME = NTIME + IPNEXT
C SEE IF TIME TO STOP PPSEQV TIME STEP.
53 IF(ITIMEC.LT.NTIMEC) GO TO 65
54 READ(15,400) NTIMEC,CUNITS,IPNEXT,SYM3L,CVALUE
400 FORMAT(10,A6,I2,A4,E13.6)
C
C BLANK TIME STEP CARD ENDS RUN.
57 IF(SYM3L.EQ.CITYC) GO TO 86
58 IF(CUNITS.EQ.BLANK) GO TO 59
59 IF(A(2).LT.5) A(2)=5
450 DO 55 MN=1,4XANO
55 CONTINUE
57 FORMAT(15E6,51) NTIME,CUNITS,SYM3L,A(MN),CVALUE
55 FORMAT(15X,F15.15,1X,A4,E13.6)
C
C SET FLG.-INDICATION CHANGED WORK LOAD IF EXC INPUT.
C IF(A(I).EQ.319) ICONVT = 1
GO TO 54
59 UNITS = CUNITS
NTIMEP = T / 1440. + IPNEXT
IF(UNITS .EQ. SFCS) NTIMEP = T * 60. + IPNEXT
IF(UNITS .EQ. THIN) NTIMEP = T + IPNEXT
IF(UNITS .EQ. HOUR) NTIMEP = T / 60. + IPNEXT
C
NGT CNTIMECushing NTIMEC - IPNEXT
C
C CONVERT FLX TO WATTS FOR GRODIN IF HEAT ALREADY.
65 IF(ICONVT .EQ. 0) GO TO 650
C
U125 = 0.
605 CONTINUE
605 IF(A(319).LE.1.) GO TO 650
40 DO 605 JJ = 2,13
JJ2 = JJ
60 CONTINUE
GO TO 140
120 POL=TAB(JJ2-1,1)+(((A(319)-WATEXCIJJ2-1,1))/
1 (WATEXCIJJ2-1,1)-WATEXCIJJ2-1,1))/
2 WATEXCIJJ2-1,2))
C
650 RETURN
C
HERE IF DETECTED END OF RUN(BLANK TIME STEP CARD).
66 UP14 = 3
RETURN
END
SUBROUTINE FUNCTNTH,POL,TAB
D1=SY3VTAB(N1)
N1=14
DO 110 I=1,N,2
IF(IATB(1)-TH) 110,120,110
110 CONTINUE
GO TO 140
120 POL=TAB(I+1)
130 RETURN
140 N=32-2
V90 = VPA + VPC - VVS - VAS - VLA - VPA - VRA
VVS = VVS + VPA * 12 + VBD * 1.55
VAS = VAS + VAS * 12 + VBD * 2.51
VLA = VLA + VLA * 12 + VBD * 1.28
VRA = VRA + VRA * 12 + VBD * 0.574
VAF = VAS - 4.95
PA = VAS / 0.355

IF (PA.LT.0.) PA = 0.001
PA2 = PA / PA
CALL FUNCTN (PA2, LVMFUN1)

VP = VPA - 1
PRA = VPA / 0.05
CALL FUNCTN (PRA, QRMFUN2)

VPF = VPA - 3.0625
PPA = VPA / 0.048
PPI = 0.028 * PPA
IF (PPI.LT.10.) PPI = 10.**(-12)
RPA = PP1 * (1., 5)
PP2 = PPA / AUH
IF (PP2.LT.10.) PP2 = 0.001
CALL FUNCTN (PP2, RVMFUN3)

VLE = VLA - 4
PLA = VLA / 0.1
CALL FUNCTN (PLA, QLMFUN4)
PPY = 1. / (PLA + 20.) / 0.357
RHY = PYP + HPA
PGT = PPA - PLA
QMM = PLG / PPT

AUW = A'M
IF (AUW.LT.0.) AUW = 0
VVK = VVK - (AUW - 1.) * ANZ
VVK = VVK - VVT
IF (VVK6.LT.0.001) VVK6 = 0.001
PV5 = VVK5 / CV
P5 = PRA
IF (PRA.LT.0.) PRI = 0
RVG = 2.7375

CV0 = CV0 - VCG
C'V = C'V + ((C'V - 1.) * CN1 + 1.) * CN2 - CN3)

AVE = (AVU - 1.) * AUY + 1.
RV5 = AUY * (1. + CN1) * VIN1 * (ANU - 1.) * ANZ + 1.
PS5 = PPA - PVS
PS5 = PPA - AUH / AUH + VHS / VIM * RVS / 1.79

AFN = GS / 6.5
K5N = AUH / PV + AVU / AUH + AHF
QF = P5 / PS
QCA = QK + APRF / (PA - PRA) * FIS
QL = LVMFUN1 / (AUH + HSL + HMD + HPL)
QP = QRQ + (1. + QRF) * ANU + RVM + HS + HMD + HP + HQR + QLO / QLN
QP = QL + (QO - QLO) * U
QVR = QG + C'QD + QGD / U
QMS = QAC - QVG
PA = QK - QPG
SUBROUTINE AUTO (AU, AUB, AUCA, AUH, AUI, AIUK, AUL, AUN, AUG, AUP, AUS, *AIa, AUSA, AUV, AUEX, EXC, EXE, *FXI, I2, PA, PA1, PQO, P0T, P0U, STA, VVR, VV9, Y, Z, *AIi, O, 0)
END

SUBROUTINE HORMON (AM, AMC, AMR, AMTAN, AMK, AGK, A2, A3)
END
IF (AQR.LT.O.) Avq = 0.
CALL FUNCTN (PA, AMP, FUTAN)
AM = AM + [1.75 - 1(A(N-1) - ANF)ANF#1] + AMT
AMF = ANF(AMF - ANF)ANF + ANF#1
AMT = AMT + [1.75 - 1(A(N-1) - ANF)ANF#1]

ANG [0 TSIN CONTROL BLOCK

CNE = 52 - C
IF (CNE.LT.L.) CCNE = L.
A'f = (1.75 - 1(GCF*CNAH*AGK + 1d)*REK)

PC = ANP + ANW
IF (PC.GT.100) PC = 100.
IF (PC.LT.0) PC = 0.

VIB = VP + VR
SUBROUTINE BLOOD PK: FMK, FMK, FMK, FMK, FMK, FMK, FMK, FMK, FMK, FMK

C RED CELLS AND VISCOSITY BLOCK

C BLOOD VISCOSITY

C----------------------------------------
C RED PLCCD CELLS

C MUSCLE BLOOD FLOW CONTROL AND P02 BLOCK

C MUSCLE: FMK, FMK, FMK, FMK, FMK, FMK, FMK, FMK, FMK, FMK

180 OSA = ALG - VWF5 + 5
C RUZI IS OXYGEN VOL. IN AORTIC BLOOD (HBA) FROM GRODIN.
OVA = RUZI * 1000.
SUBROUTINE AUTORS(AU, ARM, AR1, AR2, AR3, AK1, AK2, AK3, BFN, DOB, HM, 1, 2, 3, 4, 5, 6, 7)  

C  OXYGEN DELIVERY BLOCK
C  AND NON-MUSCLE LOCAL BLOOD FLOW CONTROL BLOCK
C  ----------------------------------------
C  AUTOREGULATION, RAPID
C  ----------------------------------------
C  CSV = OSV + (DFN*VA-D'-OI/HM/5./BFN-OVS)/Z7
PVO = OSV*Z7.14
POV = POT**3.
IF (POC.LT.50.) RPO = 50.
G3 = (PVO*PCT)*2866.5/RDO
M2 = (1-PVO*PCT)*2866.5/RDO
G5 = (1-PVO*PCT)**3.5/3.12.
C3 = (PVO-PCT)**3.

C  AUTOREGULATION, INTERFACIAL
C  ----------------------------------------
C  PQA = PAF*PO1*POO+1.-POA/Z
IF (PQA.LT.5) PQA = 5
AR2 = AR2+(PQA-AR2)*(1.-EXP(-I/AlK))
RETURN
END
C-mean circulatory pressures

PM=(VAE+VVe+VAE+VPE+YLe)/11
PM=(VAE+VVe+VRe)/.09375
PM=(VPe+YLe)/.01625

C-HEART RATE AND STRIKE VOLUME BLOCK AND TOTAL PERIPHERAL RESISTANCE

HR=(32+H1*AUR+PRA*2)*((HM-.5)*.542.)
PTP=(PA-PRA)/QA
SVC=QLG/HR
RETURN
END

SUBROUTINE CAPPBD(BFNCFC.CPICPDPDFP,
*,IFPPC,PDC,PIFPLOPPCI
*,P{P,PTC,PTS,PTTPVGPVS,RVSTVDVG ,VIDVIFVP,
*,VPDVTC,VTD,VTL,VTS.VUO}ZLFUN6)
DI*ENSIN
FUN6(14)
REAL I,IFP

C-capillary caperane dynamics block

PTT=(VTS/2)^2.
VIF=VTS-VG
CALL FUNCTN (VIF,PTS,FUN6)
PIF=PTT-PTS
CPl=I*P/VP
PTC=.25*CPI
CPP=PRP/VP
PPC=.44*PP
PVG=VPSL*7.3*9F
PC=PVG*PVS
PCPC=PTC-PPC-PIF
VTG=VTC+(CFCPCD-VTC/2)
PLD=2.2*PIF-PTT
VTL=VTL+1.004*PLD-VTL)/Z
IF(VTL.LT.0.)VTL=0.
VTG=VTC-VTL=VTD
VTS=VTS+1.0D1
VP=VPD+VTD+VTL-VUO-DFP-VPD)/Z
RETURN
END

SUBROUTINE PULMNC(CPFP,CPN,DPF,PLA,PPA,PDDP,FPC,PPC,VP
VPD,VPE
REAL I

C-pulmonary dynamics and fluids block

VP=VP+VPD*7/23

200 PCC=.45*PPA+.55*PLA
PPT=2.32*VVF
VCP=PPV/VPF
Pp0=CPN*.4
PLF=PPI+11
PP0=(CPP-CPN)*.0003
PP=PLF*C
P0M=(CPP-CPN)*.000225
PPA=PP1+(PPB-PPB)*(PP1-PP)
PFI=PP-PPI+POS-PPCI*CPF
DFP=DFP4(PF1-PLF-DFP)Z
IF(VPF<CPF)-.001.LT.0.DFP=1.001-VF;I
VPF=VPF+DFP#I
PP=PP+PPD#I
PP=PP#I

SUBROUTINE HISC2 (HPL,HPH,HSL,HSH,T,PE,PPA,POT,STH,ZI0,ZI1,I3)
REAL I
C
C**********************************************************************
C HEART HYPERTROPHY OR CATERIORATION BLOCK
C**********************************************************************
HPL=HPL+((PA+100./HSL)*Z13)-HPL#1./S600.
HSH=HSH+((PPA+1./HSH)*Z13)-HPR#1./S600.
C**********************************************************************
C TISSUE EFFECT ON THIRST AND SALT INTAKE
C**********************************************************************
STH=(Z10-PCT)*Z11
IF(STH<1.)STH=1.
IF(STH>8.)STH=8.
RETURN
END

SUBROUTINE PROTNICKRY,CPP,CPI,CPK,CPP,CPR,CPD,DP1,DPC,DP1,DPL,
* 
DPC,DPP,GPI,GRPI1,IPP,LPK,PC,PCE,PVG ,
* 
VTLZ ,PPD)
REAL I
C
C TISSUE FLUIDS,PRESSURIF AND GEL BLOCK
C**********************************************************************
C PLASMA AND TISSUE FLUID PROTEIN
C**********************************************************************
135
DPL=DPL+((VTL1-CPD)*DPL)/Z
IF (PC.LT.0.)PC=0.
CPC=(CPP*(CPP+CP1)+PC*PCE-DPC)/Z
DPL=DPL*Z
DPL=DPL*(CPP-CPP)
DPL=(CPP+CPP)Z=6*OLZ
DPL=OL+1(LZ-OL)/Z
PRP=PPB*(CP1-PRP)+PL-PCE-PPD#I
C
C GEL PROTEIN DYNAMICS
C**********************************************************************
C
141
PGG=CPAY*2*01332*CPP*CGP
GDP=GDP#1.0005*CP1-PGZ*VG-GPD#I
GDP=GDP*GPD#I
IFP=IF#(GPI-GPD)#I
SUBROUTINE KIDNEY(AAR,AMH,AM,APD,ARF,AIM,CNE,CNX,CNY,GFL,GFN,GFR,
               GF2,GF3,GF4,GPL,IM,NAE,NED,NID,NIF,NOZ,PA,PAR,
               PFL,PPC,RFK,RFN,RR,STH,TRR,VIM,VUD,Z)
REAL 1,NAF,NED,NID,NOZ

C KIDNEY DYNAMICS AND EXCRETION BLOCK
142 GF3 = (GFN/1.125-1.)*GF4 + 1.
     IF (GF3.GT.15.)GF3 = 15.
     IF (GF3.LT.4)GF3 = 4.
     AP = 3.67*VIM + (AIM*ARF+1.-ARF)*GF3
     PR = AAR + 51.66*VIN
     PMA = P2-CBL
     PNN = PAR/PR
     RARF = RFK*PNN
150 GFV = AAR*GFN
     GPL = PAR-APD
     PFL = GLP-PPC-18.
     GF1 = GFN
     GF2 = GFN*(PFL/0.007FL-GFN) + GF2/Z
     IF (A8*GFN-GF1).GT.002)GO TO 142
     GFH = GFN+REK
     TPI = 0.9*GFH + O25*RFK-0.01*RFK/AM/ANM
     VUD = VUD + (GFR-TRR-VUD)/Z
     IF (VUD.LT.0002) VUD = 0.0002

C KIDNEY SALT OUTPUT AND SALT INTAKE
C (SEE ALSO ELECTROLYTES AND CELL WATER BLOCK)
C------------------- - - --
NOZ = 1000.*VUD/AH/CNX/CNE+CNY
NOD = NID*(NOZ-NOD)/Z
NED = NID*STH-NOD
NAF = NAE+NID
RETURN

SUBROUTINE IONS (AM,CCC,CKE,CKI,CH3I,KCD,KF,KKI,KID,KIE,4
                 KIR,KDD,NAE,REK,VEC,VIC,VID,VP,VPF,VTS,Z)
REAL 1,KCD,KF,KI,KKI,KID,KIE,KIR,KDD,NAE

C ELECTROLYTES AND CELL WATER BLOCK
C------------------- - - --
VFC = VTS+VP+VPF
CKE = KF/VEC
KDD = (0.0042*CKE+G0014*AM*CKE)*REK
KIR = 2450+140.*CKE
KCI = KI+KCI
KCD = KCD+K1P-0.01*REK/KI
KCI = KI+KCI
KID = KID+KDD
KE = KE+KID
CKI = KI/VEC
CNA = NAE/VEC
VID = VID4+0.01*CCD-VID)/Z
VIC = VIC+VID
RETURN
SUBROUTINE GELFDIC (CHY, GPG, CPI, PGR, HY1, IFF, PGC, PHG, PGP, PGRX, PIF,
   4, PRR, PTC, PTS, PTT, VG, VGO, VIF, VRS, VTS, V20, FUN6)
   REAL IFF

C GEL FLUID DYNAMICS
C
140 CHY=HYL/VG
  PRR=5.9*CHY*VZ4.2
  GPG=0.2*VG
  PGC=0.2*PGP
  PGX=1.2*CHY
  CALL #UNCTN (VIFPTS,FUN6)
  PIF=PTT-PTS
  CPI=IFF/VIF
  PTC=0.2*PICALC
  PGH=PIF+PTS+PRM
  VGO=V20*(PIF+PGC-PTC-PGH)
  VIF=TS-VG
  IF(VG-LT-0.) VGO.
  IF(.OL2.LT.ABS(VGO)) GOTO 140
  RETURN
 END

SUBROUTINE GRODIN (URZ1, URZ2, URZ3, URZ4, URZ5, RUZ1)

C FOLLOWING FROM GUYTON TO GRODIN-----
C GUYTON GRODIN
C
QLC  (URZ1) CARDIAC OUTPUT.
C(RHOG+DUB)/1000. (URZ2) TOTAL METABOLIC RATE OF BODY
C(HV05A)/200. (URZ3) BLOOD OXYGEN CAPACITY.
C (URZ4) (URZ4) FLG 1=1ST TIME GRODIN CALLED.
C (URZ5) (URZ5) WORK LEVEL (WATTS).
C
C FOLLOWING FROM GRODIN TO GUYTON-----
C GUYTON GRODIN
C (URZ1) OXYGEN VOL. IN AORTIC BLOOD.
C
C(140)
C ALVEOLAR VCL GAS FUNCTIONS
C 1 FA1(C2)
C 2 FA1(02)
C 3 FA1(N2)
C
C GAS CONCENTRATIONS IN ORGAN.
C 4 C01(C2)
C 5 C01(02)
C 6 C01(N2)
C
C GAS CONCENTRATIONS IN TISSUE.
C 7 C11(CO2)
C 8 C11(02)
C 9 CT(N2)
C CARDIAC OUTPUT.
C 10 Q
C CEREBRAL BLOOD FLOW.
C 11 QB
C GAS TENSION IN CSF.
C 12 PCSF(CO2)
C 13 PCSF(C2)
C 14 PCSF(N2)
C C LENGTH OF SIMULATION RUN.
C 15 TMAX
C WEIGHTING OF H+ CONC IN CSF VERSUS VENOUS BLOOD OF BRAIN.
C 16 CENTRAL SENSITIVITY PARTITION
C BLOOD OXYGEN CAPACITY
C 17 (HB)
C TIME CONSTANTS IN CARDIAC OUTPUT AND CEREBRAL BLOOD FLOW RESPONSES.
C 18 Pi
C 19 *Q2
C CONTROLLER EQUATION SENSITIVITY WEIGHTINGS.
C 20 CENTRAL SENSITIVITY COEFFICIENT
C 21 CAROTID BODY SENSITIVITY COEFFICIENT
C VOLUMES OF LUNG, BRAIN, AND TISSUE
C 22 KL
C 23 KB
C 24 KT
C BRAIN METABOLIC RATE OF CO2 PRODUCTION.
C 25 MBRCO2
C BRAIN METABOLIC RATE OF O2 CONSUMPTION.
C 26 MBRO2
C GAS DIFFUSION COEFF. FOR BLOOD-BRAIN-BARRIER.
C 27 KCCO2
C 28 KNO2
C 29 KCN2
C PARACETAMOL PRESSURE.
C 30 E
C VOLUME FRACTION OF INSPIRED GAS.
C 31 FI(N2)
C 32 FI(O2)
C 33 FI(N2)
C VOLUME OF CSF.
C 34 KCSF
C INITIAL TIME.
C 35 T
C COMPUTER TIME STEP.
C 36 H
C CONTROLLER EQUATION CONSTANT (MAINTAINS RESTING PACO2 APPROX. 40).
VENOUS GAS CONCENTRATIONS AT BRAIN EXIT.

VENOUS GAS CONCENTRATIONS AT TISSUE EXIT.

CARPIAC OUTPUT.

TISSUE ALKALO FLW.

ARTERIAL H+ CONCENTRATION.

ARTERIAL O2 TENSION.

TOTAL GAS CONCENTRATIONS AT BRAIN EXIT.

TOTAL GAS CONCENTRATIONS AT TISSUE EXIT.

VENT.

VENOUS BRAIN GAS CONCENTRATION AT LUNG ENTRANCE.

VENOUS TISSUE GAS CONCENTRATION AT LUNG ENTRANCE.

ARTERIAL GAS CONCENTRATIONS AT TISSUE ENTRANCE.

ARTERIAL H+ CONCENTRATION AT CAROTID BODIES' SITE.

ARTERIAL O2 TENSION AT CAROTID BODIES' SITE.

ARTERIAL H+ CONCENTRATION AT BRAIN ENTRANCE.

TOTAL GAS CONCENTRATION FROM BRAIN AT LUNG ENTRANCE.
COMPARTMENTAL GAS TENSIONS AND CONCENTRATIONS.
   1  PA(02)
   2  K AC(02) PA(02)
   3  P0(02)
   4  K AC(02) P0(02)
   5  PT(02)
   6  K AC(02) PT(02)
   7  PA(02)
   8  PA(02)
   9  CA(02)
  10  CA(02)
  11  CA(02) + CA(02) + CA(02)
  12  CVH(02)
  13  CVH(02)

PRODUCT OF DIFFUSION COEFFS. AND GAS DIFFERENTIALS ACROSS BLOOD-GRAIN BARRIER.
   14  UCC2 (P0(02) - P0SF(02))
   15  NO2 (PA(02) - P0SF(02))
   16  N2 (PA(02) - P0SF(02))
   17  PE(02)
   18  PE(02)

INTEGER U44
DIMENSION C(40), XN(40,2), SW(18,50), VRAN(18), RK(14,41),
   1 SC(14,5), CC(14), A(6), B(13), F(20), VOL(10), RMT(2),
   2 BC(4), OF(6), TAU(5), CC(3), CHB(3), CH(4), CPB(3),
   3 D0(4)
DIMENSION XN(84,2), D0J(4), DB(6), ID(12)
COMMON/C, XN, SV, VRAN, RK, SC, A, D, F, VOL, RMT, BC, OF,
   1 TAU, CC, CHB, CH, CPB, DQ, VE, VI, CPB, CP, CDBK, X, DT,
   2 X, LOC, INDEX, 1, J, 4, N
COMMON/P, KH, KS, C, CT, U, DUM, DUM, DUM, DUM, DUM, DUM, DUM, DUM, DUM,
IF THIS 1ST TIME GRODIN CALLED.
IF (URZ4 .NE. 1 ) GO TO 370
100
HERE IF THIS 1ST TIME GRODIN CALLED.

DATA FOR INITIAL CONDITIONS
WRITE (6,9)
9 FORMAT(6X,15X,F15.8)
1000 CONTINUE
WRITE(6,90)
90 FORMAT(3X, "RESPIRATORY CHEMOSTAT -- INPUT DATA")

DATA FOR INITIAL CONDITIONS
ON 10 I = 1,40
READ(5,190) C(I), (XNB(I,J), J=1,2)
10 CONTINUE

ESTABLISH COMPUTATION STEP INDEPENDENT OF INPUT DATA.
FOLLOWING TIME STEP TO MAKE GRODIN COMPATABLE TO GUYTON.
(130) = 0.003
(150) = URZ1
(170) = UPZ2 - C(26)

OUTPUT INPUT + ABOVE 3 VARIABLES FROM GUYTON.
J = 1
ON 30 I = 1,8
JJ = J+4
WRITE(6,92) J, (C(I),I=J,JJ)
92 FORMAT(6X,12X,5F9.4)
J = J + 5
30 CONTINUE
WRITE(6,92) J, (BC(I),I=1,4)
J = 49
93 WRITE(6,92) J, RMT(1), RMT(2), DJ(1), DJ(2)
C 10(C2)
DUM1=C(31)
C 10(C2)
DUM2=C(32)
C 10(H2)
DUM3=C(33)
W1R=0.
W3PZ=0.
C METABOLIC RATE OF C2 CONSUMPTION IN TISSUE.
RMTB=RMT(2)
RMTQJ2RMT(2)

C

XHS=0.

XH= 10. * C(36)/.003

MHS=0

201 CONTINUE

XOS=XOS+XH

IF(XHS=EQ.1)XOS=XOS+C(36)

XH=1

C(35)=C.

C(40)=0.

C

INITIAL GUESSES FOR ITERATIVE LOOPS

C MATERIAL CONCENTRATION OF CO2.

CC(1) = 0.6

C BRAIN CONCENTRATION OF CO2.

CC(2) = C(4)

C BRAIN C^2 TENSION.

CPH = 50.0

C TISSUE CO^2 TENSION.

CPT = 50.0

IF(XDS.GT.XMH) GOTO202

C SETS VARIOUS CONSTANTS AND AGGREGATES OF CONSTANTS.

C TMAX.

C(15) = C(15) * .0001

C PRINT ALL TIME.

C(39) = C(39) * .001

C2 200 I = 27.29

C FACTOR OF 1-E-7 MULTIPLYING DIFFUSION COEFFICIENTS.

C(11) = C(11) * 1.E-7

200 CONTINUE

202 CONTINUE

IPK = 1

M = 14

N = 9

I(11) = C

VOLUMILITY COEFFICIENTS.

C A(1)= (ALPHA)CO^2, A(2)= (ALPHA)O^2, A(3)= (ALPHA)N^2,

C A(4)= (ALPHA)CO^2, A(5)= (ALPHA)O^2, A(6)= (ALPHA)N^2

A(1) = 0.51

A(2) = 0.024

A(3) = 0.013

A(4) = 0.51

A(5) = 0.024

A(6) = 0.013

CYW/CHG CONVERSION FACTOR.

SK = 0.00132

C CARPOIC ACID DISSOCIATION CONSTANT.

CADK = 795.0

C V(1)=V(11) = VOLUMES USED IN CALCULATION OF VARIABLE TIME DELAYS.

V(1) = 0.045

VCL(2) = 1.062

VCL(3) = 0.188

VCL(4) = 0.06

VCL(5) = 0.188

VCL(6) = 2.96

VCL(7) = 0.735

VCL(8) = 1.062
C

70 CALL PC14
  UU = AMOOC(35), D(14)
  IF (UU .LT. .0001, UU .GT. .015) GO TO 50

C
C HEPE WHEN GUYTON SAID END OF RUN.
  20 IF (C(37) .GT. 1.0E-5) GO TO 250

220 CTERM = 0.0
     IF (VTRAN(I4) .LT. 0.040) 230, 240, 240

230 CTERM = (23.6E-9) * ((0.040 - VTRAN(I4))**4/4)

240 C(37) = C(20) * C(14) * VTRAN(I4) + (1.0 - C(16)) * CH(I)

C
C HERE WHEN GUYTON SAID END OF RUN.
  30 IF (C(37) .GT. 1.0E-5) GO TO 250

300 CONTINUE

STOP
192 FORMAT (I3, 2X, F15.5, 5X, 2A8)
194 FORMAT (111)

C

SURROUNDING RC

DIMENSION C(40), VN(40,20), SV(18,50), VTRAN(18), KK(14,4),
  1 : SC(14,5), DC(14), A(5), D(I1), F(I20), WDL(I10), RMT(I2),
  2 : QQ(I4), QF(I2), TAU(5), CC(I3), CH(I4), CH(I5), CPH(3),
  3 : QQ(I4)

COMMON /I2/ C, VN, SV, VTRAN, RK, SC, DC, A, D, F, VDL, RMT, BC, QQ,
  1 : TAI, CC, CHA, CHB, DO, WE, VL, CPB, CPT, CADK, X, DT,
  2 : TP, LOC, ITFRX, INDEX, I, J, K, N

C

CALCULATES TRANSPORT TIMES

C

EQUATIONS 8.10 THRU 8.14

C6969 DO 870 I = 1,5
     DT = C(35) - SV(I18,1)
     ND = 1
     GO TO 810

810 NC = 11
     NA = 10
     GO TO 820

812 NC = 10
     NA = 11
     GO TO 820

814 NC = 10
     NA = 12
     GO TO 820

816 NC = 12
     NA = 10
     CA = QF(1)
     GO TO 822

820 QA = C(NC)
     822 DO 860 J = 12
     GO TO (834, 824), J

824 NC = NA
     ND = K + 1
     IF (K) 825, 826, 832

826 IF (NC - 12) 830, 828, 830

C

STOP

FORMAT (11H7SUB RC8)

C6969 DO 870 I = 1,5
     DT = C(35) - SV(I18,1)
     ND = 1
     GO TO 810

810 NC = 11
     NA = 10
     GO TO 820

812 NC = 10
     NA = 11
     GO TO 820

814 NC = 10
     NA = 12
     GO TO 820

816 NC = 12
     NA = 10
     CA = QF(1)
     GO TO 822

820 QA = C(NC)
     822 DO 860 J = 12
     GO TO (834, 824), J

824 NC = NA
     ND = K + 1
     IF (K) 825, 826, 832

826 IF (NC - 12) 830, 828, 830
029 QA = SV(NC,1) - (SV(NC,1) - OF(1))/DT'(C(35) - SV(I8,1))
GO TO 634
030 QA = SV(NC,1) - (SV(NC,1) - C(NC))DT'(C(35) - SV(I8,1))
GO TO 634
032 QA = SV(NC,ND) - (SV(NC,K) - SV(NC,NH))DT'/D(14)
034 JJ = 2*I + J - 2
AA = VOL(IJ)'C(36)/.0078125
D3 836 K = ND.49,
IF (AA - AB) GO TO 836, 834, 840
036 AA = AA + C(36)*[SV(NC,K) + SV(NC,K+1)]
038 CONTINUE
IF (K) WRITE [6,090] 1
860 AA = AA - AB
K = K - 1
IF (K) GO TO 862, 842, 846
862 DV = SV(NC,1) - QA
IF (DV) GO TO 864, 840, 860
844 DT = QA/QA
GO TO 860
866 DV = SV(NC,K+1) - SV(NC,K)
IF (DV) GO TO 850, 850, 860
850 DT = (D/SV(NC,K))
GO TO 860
860 DT = (SV(NC,K+1) - SQRT (SV(NC,K+1)/*2 - DV*DA/C(36))/DV/D(14))
860 CONTINUE
TAU(1) = C(159) - SV(I8,K+1) - DT
870 CONTINUE
CONTINUE
RETURN
890 FORMAT (5X27HSV ARRAY EXCEEDED ON CYC.LE 12)
END

SUBROUTINE RCI2(URZ4,URZ5)

INTEGRURZ4
DIMENSION C(40), IN(40,2), SV(I8,50), VTRAN(I8), RK(14,4),
1 SC(14,5), DC(14), A(16), D(13), F(20), VOL(10), RMT(2),
2 BG(4), OF(61), TAU(5), CC(3), CH(13), CH(4), CPH(3),
3 DO(6),

COMMON/CY,XT,TV,RA,SC,DC,AD,F,VOL,RMT,DC,OF,
1 TAU,CC,CMO,CH,CPH,DQ,VE,V1,CPB,CPT,CADK,X,S,T,
2 HX,LOC, IENN, INDEX, I, J, N,

C(35)=(DEADVT*C(21)+VE*DUM3)/C(32)
C(33)=(VOL*C(3)+VF*DUM3)/C(DEADVT+VE)
C MINUTE VCLUMF.

H8ATE=3.8*(RMT(2)+C(26))+S4.5
C SEE IF WORKLOAD HAS CHANGED.
IF(URZ4 .NE. 0, GO TO 101
1 WORK = WORK2
MARKER = 0
C SYSTEM RESPONSES: TIME CONSTANTS FOR WORK LOAD LEVELS (INCREASING):
IF(WORK = .LE. 0.5) GO TO 500
 IF(WORK = .GE. 50.0) ICT = 2.3/(12.*WORK/200.)
 IF(WORK = .LT. 50.) ICT = 4.6
C TISSUE O2 METABOLIC RATE.
MT(2) COMES FROM GUYTON.
VTIME = 1.1 - 1.1*EXP(-ICT*(CXT-TIME)/1.92)
C TERM USED IN VT THAT IS A COMPONENT OF TRANSIENT RESPONSE RELATED
C TO WORK LOAD.
RMLIN = S502(WORK) - SSO2(WORK) + MT(2) * (1. - VTIME)
 IF(VTIME .GE. 0.4) RMLIN = S502(WORK)
C TISSUE CO2 METABOLIC RATE.
PTM(1) = 0.9*MT(2)
 IF(VTIME .GT. 0.7) PTM(1) = (VT1M+40.7)**2/86.5
 IF(URZ4 = .EQ. 0) GO TO 1230
 WRITE (6,333) PTM(1), RMLIN(2)
333 FORMAT ( '0.1F6.25CHANGE IN METABOLIC RATES, 5X,7HMCO2 = ',F10.4,
  1 5X,7MMRO2 = ',F10.4,/) 
2 CONTINUE
C IF(URZ4 .EQ. 0) GO TO 1230
C HERE IF GIVING TO PRINTOUT.
C ARTERIAL H2 TENSION.
1210 PAN2 = C1(1)*C1(3)
C TISSUE H2 TENSION.
PTM2 = C(2)/C(3)
C TISSUE H2 TENSION.
PIN2 = C(9)/C(4)
C CEREBRAL SPINAL FLUID pH, EQUATION 6.2.
PHCF = 9. - PCF1(CM(44))
C VENOUS BRAIN H+ CONCENTRATION, EQUATION 4.7.
HVB = CANKF(4)/C(C2) - F(4))
C VENOUS BRAIN PH, EQUATION 4.6.
PIVO = 9. - PCF1(HVP)
C VENOUS TISSUE H+ CONCENTRATION, EQUATION 5.7.
HTV = CANKF(5)/C(C3) - F(6))
C VENOUS TISSUE PH, EQUATION 5.6.
PHTV = 9. - PCF1(HTV)
C RESPIRATORY QUOTIENT (ALVEOLAR).
PF = (C(11)*VT4PA/4) + QF(1)*VT4PA/1/C10) - C(I1))/
1 IF(7) = IC(11)*VT4PA/5 + QF(1)*VT4PA/1/C10))
QF(5) = OF(6) - RW
C WRITE (6,1910) CXT, AQ, OF(5)
C WRITE (6,1915) C(11), I = 1,3, C(11), I = 1,3, F(7), F(11),
1 PAN2
WRITE (6,1820) C1(1), F(9), F(10), F(7), F(11), PAN2, C1(1),
1 CPH1, C1H1)
WRITE (6,1825) C(11), I = 4,6, C(11), I = 4,6, CP6, F(17),
1 F(11), C1H2, CPH2)
WRITE (6,1830) C(11), I = 7,9, D(11), I = 7,9, CPT, PT02,
SUBROUTINE QC3
DIMENSION C(40), XN(40,2), SV(19,50), VTRAN(18), RK(14,4),
1 SC(14,5), CC(14), A(16), D(11), F(20), VOL(10), RMT(2),
2 NC(4), OF(16), Tau(5), CC(13), CH(13), CH(4), CP(13),
3 GO(4)
COMMON/C, XN, SV, VTRAN, RK, SC, D, A, D, F, VOL, RMT, BC, OF,
1 Tau, CC, CB, CH, CH, D, D, VE, VE, CPB, CPT, CADV, X, DT,
2 TR, LCG, INDEX, I, J, I, N
C6960 PRINT(TH,MSUB QC3)
C SETS TIME-DEPENDENT EXPRESSIONS
C TISSUE BLOOD FLOW,
QF(1) = C(11) - C(11)
C ARTERIAL CO2 TENSION,
F(1) = D(11) - C(11)
C ARTERIAL CO2 CONCENTRATION,
F(2) = D(11) - C(11)
C BRAIN 12 CONCENTRATION / (CONV.FACT*SOLUBILITY COEFF.FOR O2)
F(3) = C(11)/10
C (CONV.FACT*SOLUBILITY COEFF.FOR CO2) = BRAIN CO2 TENSION.
F(4) = D(11) - C(11)
C TISSUE 12 CONCENTRATION / (CONV.FACT*SOLUBILITY COEFF.FOR O2)
F(5) = C(11)/10
C (CONV.FACT*SOLUBILITY COEFF.FOR CO2) = TISSUE CO2 TENSION.
F(6) = N(11) - C(11)
C ARTERIAL CO2 TENSION.
F(7) = D(11) - C(11)
C ARTERIAL O2 TENSION.
F(8) = D(11) - C(11)
RETURN END
SUBROUTINE RC4
DIMENSION C(40), XN(40,2), SV(19,50), VTRAN(18), RK(14,4),
1 SC(14,5), CC(14), A(16), D(11), F(20), VOL(10), RMT(2),
2 NC(4), OF(16), Tau(5), CC(13), CH(13), CH(4), CP(13),
3 GO(4)
COMMON/C, XN, SV, VTRAN, RK, SC, D, A, D, F, VOL, RMT, BC, OF,
1 Tau, CC, CB, CH, CH, D, D, VE, VE, CPB, CPT, CADV, X, DT,
2 TR, LCG, INDEX, I, J, I, N
C ITERATES FOR CC(1), ARTERIAL CO2 CONCENTRATION
C6960 PRINT(TH,MSUB RC4)
410 CALL RC21 (CHB(1), F(1), F(2), CC(1), CH(1), CP(1))
X = (CC(1) - F(1))/10.01 + F(1)
X = RC(1)
C SEE EQUATION 3.1, X = CALCO2
X = RC(1) + 0.375*{CC(17) - CHB(1)} + 2.4 + 0.8*{X - 0.14}
C CC(1) = CALCO2
CALL RC6 (CC(1))
CC(1) = CC(1) + 2.0*{X - CC(1)}1/3.0
C3000 PRINT(TH,SHCC(1),X,VE,F16.6)
IF (ITERX) 420, 410, 420
420 RETURN END
SUBROUTINE RC5 (CP, FB, CC, BHC)
DIMENSION C(40), XN(40,2), SV(19,50), VTRAN(18), RK(14,4),
1 SC(14,5), CC(14), A(16), D(11), F(20), VOL(10), RMT(2),
2 NC(4), OF(16), Tau(5), CC(13), CH(13), CH(4), CP(13),
IPM "I
I
C C1M4Mr)NZ/

C, XN, SV,

VTRAN, RK,

SC, :1., A,

0, F, VGL, RMT, BC, QF,

1

TAU, CC, CHH, CH, CPH, DU, VE, VI, CPB, CPT, CADK, X, DT,

2

IRK, LOC, ITFRX, INDEX, F, J, I, H, N

C ITERATES FOR BRAIN AND TISSUE PCO2

C6669 FORMMAT(1H 7HSUB RC5)

510 X = (CCD - FB)/V0.14*CP

X = RCFIX(X)

C SEE EQUATION 4.1, X = PB(CP2) .

X = (XH + CCD + 0.10)*X - 0.14)/0 2

C CP = PRECC2 :

CALL RCFIX (CP)

CP = CP + (X - CP)/10.0

C CEREBRAL BLOOD FLOW

FX = N(2)*CP

C3000 FORMMAT(1H ,4CP)= E16.6,4HF8= E16.6,5HCCB= E16.6,5HABC= E16.6)

IF (ITERX) 520, 510, 520

520 RETURN

END

SUBROUTINE RCF (Y)

DIMENSION C(40), XN(40,22), SV(18,50), VTRAN(18), RK(14,4),

1 SC(14,5), CC(14), A(16), D(15), F(20), VOL(10), RMT(2),

2 BC(14), QP(16), TAU(5), CC(3) CHH(3), CH(4), CPH(3),

3 DO(4)

COMMON/2/ C, XH, SV, VTRAN, RK, SC, LI, A, D, F, VOL, RMT, BC, QF,

1 TAU, CC, CHH, CH, CPH, DU, VE, VI, CPB, CPT, CADK, X, DT,

2 IPK, LOC, ITFRX, INDEX, F, J, I, H, N

C CHECKS CONVERGENCE OF ITERATIVE PROCEDURES

C RC4 : X=CA(CP2), Y=CC(C11) -

RC5 : X=PB(CP2), Y=CP .

C RC9 : X=CV(CP2), Y=CV .

C6669 FORMMAT(1H 7HSUB RC6)

ITERX = 0

DIFF = ABS ((X - Y)/Y)

IF (DIFF = 1.0F-5) 620, 620, 630

620 ITERX = 1

630 RETURN

END

SUBROUTINE RCF7

DIMENSION C(40), XN(40,2), SV(18,50), VTRAN(18), RK(14,4),

1 SC(14,5), DC(14), A(16), D(15), F(20), VOL(10), RMT(2),

2 BC(14), QP(16), TAU(5), CC(3) CHH(3), CH(4), CPH(3),

3 DO(4)

COMMON/2/ C, XH, SV, VTRAN, RK, SC, LI, A, D, F, VOL, RMT, BC, QF,

1 TAU, CC, CHH, CH, CPH, DU, VE, VI, CPB, CPT, CADK, X, DT,

2 IPK, LOC, ITFRX, INDEX, F, J, I, H, N

C FILLS SV ARRAY WITH INITIAL CONDITION!

CALL RC16

CALL RC18.GOTO725

720 CONTINUE

725 CONTINUE

2 CONTINUE
DO 730 J = 2,50
SV(I,J) = SV(I,J-1) - D(I,J)
730 CONTINUE
C3000 FORMAT(1H,12HESV S D(I,J),G(3X,E16.5)/1H",G(3X,E16.6)/1H",G(3X,E1)
C C6.6)
RETURN
END

SUBROUTINE RC9

CIMESIN C(401, X(N,2), SV(10,5), VTRAN(18), RK(14,4),
G(C(144), CC(14), A(6), D15), F(20), VOL(10), RMT(21),
B6(4), QF(6), TAU(13), CH(13), CH(14), CH(15),
3 COMMON/2 C, X, SV, VTRAN, RK, SS, A6, D, F, VGL, RMT, BC, OF,
1 TAU, CC, CH(14), CH, QF, VE, VI, CBP, TPI, CADK, X, OF,
2 X, LHC, ITM, INDEX, I, J, 4, N
C SETS VALUES IN VTRAN ARRAY
C6969 FORMAT(1H7SUB RC9)
DO 9001 = 1,5
TAU = TAU(I) - (C(35) - SV(I,1))
LOC = TAU/C141
IF (LOC - 49) 904, 904, 902
902 WRITE (6,S90) I,LOC
LOC = 49
904 XLOC = LOC
T8 = XLOC(I,4)
DT = T8 - DT
GO TO (910,920,930,940,950), I
910 ON 914 J = 1,3
C LUNG TO BRAIN CO2,O2,N2 TIME DELAYED ARTERIAL CONCENTRATIONS.
VTRAN(I) = RCF3(I)
914 CONTINUE
C LUNG TO BRAIN H+ TIME DELAYED ARTERIAL CONCENTRATION.
VTRAN(I) = RCF3(I)
GO TO 960
920 ON 924 J = 4,6
C BRAIN TO LUNG CO2,O2,N2 TIME DELAYED VENOUS CONCENTRATIONS.
VTRAN(I) = RCF3(I)
924 CONTINUE
C BRAIN TO LUNG COMBINED CO2,O2,N2 TIME DELAYED VENOUS CONCENTRATIONS.
VTRAN(I) = RCF3(I)
GO TO 960
930 ON 934 J = 7,9
C TISSUE T LUNG CO2,O2,N2 TIME DELAYED VENOUS CONCENTRATIONS.
VTRAN(I) = RCF3(I)
936 CONTINUE
C TISSUE T LUNG COMBINED CO2,O2,N2 TIME DELAYED VENOUS CONCENTRATIONS.
VTRAN(I) = RCF3(I)
GO TO 960
940 ON 944 J = 1,3
C LUNG TO TISSUE CO2,O2,N2 TIME DELAYED ARTERIAL CONCENTRATIONS.
VTRAN(I) = RCF3(I)
944 CONTINUE
GO TO 960
C LUNG TO CAROTID SITE H+ TIME DELAYED ARTERIAL CONCENTRATION.
950 VTRAN(I) = RCF3(I)
C LUNG TO CAROTID SITE O2 TIME DELAYED ARTERIAL TENSION.
VTRAN(I) = RCF3(I)
960 CONTINUE
C NAMELIST/INM/VTRAN
SUBROUTINE RC10
DIMENSION C(40), XN(40,2), SV(18,50), VTRAN(18), RK(14,4),
1 SC(14,5), CC(14), A(6), D(15), F(20), VOL(10), RMT(2),
2 BC(4), OP(6), TAU(15), CC(3), CH(3), CH(4), CP(3),
3 DO(4)
COMMON/C, XN, SV, VTRAN, RK, SC, CC, A, D, F, VOL, RMT, BC, OP,
1 TAU, CC, CH, CH, CP, OP, VE, VI, CPB, CPT, CADK, X, DT,
2 TRX, LOC, ITERX, INDEX, J, K, N
C
C Computes empirical functions for acr;iac output and brain blood
C flow differential equations
C
C FORMAT (5X27H5V ARRAY EXCEEDED IN CYCLE 12,12H WITH LOC = 14)
C FORMAT (H,5V)
C RCII (C(40), XN(40,2), SV(18,50), VTRAN(18), RK(14,4),
1 SC(14,5), CC(14), A(6), D(15), F(20), VOL(10), RMT(2),
2 BC(4), OP(6), TAU(15), CC(3), CH(3), CH(4), CP(3),
3 DO(4)
C COMMON/C, XN, SV, VTRAN, RK, SC, CC, A, D, F, VOL, RMT, BC, OP,
1 TAU, CC, CH, CH, CP, OP, VE, VI, CPB, CPT, CADK, X, DT,
2 TRX, LOC, ITERX, INDEX, J, K, N
C
C CALL RCII
C
C

DEPENDANCE OF CARDIAC OUTPUT ON TISSUE
C UTILIZATION OF OXYGEN.
XAB=5.5 *(RMT(2)-.215)*6.-(C10)
IF((RMT(2).GT..215).AND.(XAB.GT.0.))DC(10)=DC(10)+XAB/.010
C
C EQUATION 7.7
DC(11) = (-C11) + 0.75 + QF(2) + DQ(11)/C(11)
C EQUATION 1.10
DC(12) = F(11)/C(34)*D(11)
C EQUATION 1.11
DC(13) = F(15)/C(34)*D(12)
C EQUATION 1.12
DC(14) = F(16)/C(34)*D(13)
C
SUBROUTINE RC13
DIMENSION C(10), XN(40,2), SV(18,50), VTRAN(18), RK(14,4),
1 SG(14,5), DC(14), A(4), D(151), F(20), VOL(10), RMT(2),
2 P(14), OF(16), TAU(5), CC(3), CH(3), CH(4), CPH(3),
3 ND(4)
COM444/8 C, XN, SV, VTRAN, RK, SC, SC, A, D, F, VOL, RMT, SC, QF,
1 TAU, CC, CH, CH, CPH, PQ, VE, VI, CPB, CPT, CADK, X, DT,
2 IRK, LIG, INEX, INDEX, I, J, N, N
C6940.
FORMAT(IN UC) SUB RC13
C SOLVES DIFFERENTIAL EQUATIONS BY FIFTH-ORDER RUNGE-KUTTA AND
C Adams-Moulton: PREDICTOR-CORRECTOR METHODS
C NAMELIST/RF/CD,SC
1 IF ((IRK = 4) .OR. 1304, 1356, 1356
1304 DO 1302 INDEX = 1,4
1305 DO 1304 INDEX = 1,1,
1306 IF (((INDEX) = SG(1))
1307 CONTINUE
GO TO (1312, 1320, 1328, 1340), INDEX
1312 DO 1316 I = 1,4
1313 IF (INDEX) = SG(1,
1314 CONTINUE
1315 CONTINUE
TI = C(35)
1320 C(35) = TI + C(94)/2.0
DO 1324 I = 1,4
1325 RETURN
FNO
C(I) = SC(I,IRK+1) + C(36)*RK(1,INDEX) / 2.0
1324 CONTINUE
GO TO 1336
1326 C(35) = T1 + C(36)
GO 1332 I = I + 1
C(I) = SC(I,IRK+1) + C(36)*RK(1,INDEX)
1332 CONTINUE
1336 CALL GC14
GO TO 1332
1340 DO 1344 I = 1,N
C(I) = SC(I,IRK+1) + C(36)*RK(1,INDEX) + 2.0*RK(1,2) + 2.0*RK(1,3)
1 + RK(I,4))/6.0
1344 CONTINUE
IRK = IRK + 1
1352 CONTINUE
RETURN
1356 DO 1360 I = 1,N
SC(1,4) = C(1)
SC(1,4) = NC(1)
C(1) = SC(1,5) + C(36)*SC(1,4) - 59.0*SC(1,3) + 37.0*SC(1,2)
1 - 9.0*SC(1,1))/24.0
1360 CONTINUE
C(35) = C(35) + C(36)
NC35 = NC(35)/C(36) + 1
C(35) = NC(35)*NC35
1364 CALL GC14
DO 1368 I = 1,N
SC(1,1) = C(1)
C(1) = SC(1,5) + C(36)*SC(1,4) + 10.0*SC(1,6) - 5.0*SC(1,3)
1 + SC(1,2))/24.0
1368 CONTINUE
DO 1372 I = 1,N
IF (ABS (C(I) - SC(I,1)) < 1.0E-3) :372, 1372, 1364
1372 CONTINUE
DO 1376 I = 1,N
DO 1376 J = 1,3
SC(I,J) = SC(I,J+1)
1376 CONTINUE
RETURN
END
SUBROUTINE GC14
DIMENSION C(14), XN(40,2), SV(10,50), VTRAN(10), RK(14,4),
1 SC(14,5), OC(14), A(6), D(15), F(20), VOL(10), RMT(2),
2 NC(4), OF(6), TAU(15), CC(3), CH(13), CH1(4), CPH(3),
3 DC(4)
COMMON/Z/ C, XN, SV, HT, SC, CL, A, O, F, VOL, RMT, B, QA, QC,
1 TAU, CG, CHA, CH, CPH, DB, VE, VI, CPB, CPT, CADK, X, DT,
2 IRK, LOG, ITER, INDEX, J, J+, N
C CALLS OTHER SUBROUTINES IN C BLOCK
C6960 FORMAT(IH BSUB RC14)
CALL RC9
CALL RC8
CALL RC7
CALL RC6
CALL RC5
CALL RC4
CALL RC3
CALL RC2
CALL RC1
CALL RC0
CALL RC19 (CPT, CHB(3), CC(3), BC(1), F(6))
CALL RC10
CALL RC20
CALL RC11
RETURN
END

SUBROUTINE RC15
DIMENSION C(40), XN(40,2), SV(18,50), VTRAN(40), RK(14,4),
1 SC(14,5), GC(14), A(6), DI(15), F(20), VOL(10), RMT(2),
2 BC(4), OF(6), TAU(5), CC(3), CHB(3), CH(4), CPH(3),
3 DO(4)
COMMON/Z/ C, XN, SV, VTRAN, RK, SC, GC, A, DI, F, VOL, RMT, BC, OF,
1 TAU, CC, CH, CP, DO, VE, VE, CPB, CPT, CARK, X, DI,
2 IRK, LOC, ITERX, INDEX, I, J, N
C6969 FORMAT(1H 8HSUB RC15)
C NAMELIST/ECN/SV
C SHIFTS VALUES IN SV ARRAY
DO 1530 I = 1,18
DM 1520 J = 1,49
JM = 51 - J
JM4 = JM - 1
SV(I,JM) = SV(I,JM4)
1520 CONTINUE
1530 CONTINUE
RETURN
END

SUBROUTINE RC16
DIMENSION C(40), XN(40,2), SV(18,50), VTRAN(40), RK(14,4),
1 SC(14,5), GC(14), A(6), DI(15), F(20), VOL(10), RMT(2),
2 BC(4), OF(6), TAU(5), CC(3), CHB(3), CH(4), CPH(3),
3 DO(4)
COMMON/Z/ C, XN, SV, VTRAN, RK, SC, GC, A, DI, F, VOL, RMT, BC, OF,
1 TAU, CC, CH, CP, DO, VE, VE, CPB, CPT, CARK, X, DI,
2 IRK, LOC, ITERX, INDEX, I, J, N
COMMON/PP/ XDS, XH, XG, XH, XG, VOL, RMT, BC, OF,
1 rmkin
C6969 FORMAT(1H 8HSUB RC16)
C SETS VALUES FOR SV ARRAY
C ARTIFICIAL CO2 CONCENTRATION.
SV(1,1) = CC(1)
C ARTIFICIAL O2 CONCENTRATION.
SV(2,1) = F(9)
C BRAIN VENOUS CO2 CONCENTRATION.
SV(4,1) = CC(2)
C ARTIFICIAL N2 CONCENTRATION.
SV(3,1) = F(10)
C BRAIN VENOUS O2 CONCENTRATION.
SV(5,1) = F(12)
C BRAIN VENOUS N2 CONCENTRATION.
SV(6,1) = C(6)
C TISSUE VENOUS CO2 CONCENTRATION.
SV(7,1) = CC(3)
C TISSUE VENOUS O2 CONCENTRATION.
SV(8,1) = F(13)
C TISSUE VENOUS N2 CONCENTRATION.
SV(9,1) = C(9)
C CARDIAC OUTPUT.
SV(10,1) = C(10)
SUBROUTINE RC17
DIMENSION C(40), XM(40,2), SV(18,50), VTRAN(18), RK(14,4),
1 NC(4), TAU(5), CC(3), CH(3), CH(4), CPH(3),
2 Q(6), TAU(5), CC(3), CH(3), CH(4), CPH(3),
3 DO(4)
COMON/X/ C, XM, SV, VTRAN, KC, CC, A, D, F, VL, RMT, BC, QF,
1 TAU, CC, CH, CH, CH, DO, VE, VI, CPB, CPT, CAK, X, GT,
2 IRK, LOC, ITPFX, INDEX, I, J, K, N
COMON/X/ XOS, XMHCXT, RMTBRMTB, TIL$EOF
C NAMELIST/BA/CH(14),CP(12),C(11),C(12),C(13),C(14),C(15),C(16),C(17),C(18),C(19),C(20),C(21),C(22),C(23),C(24),C(25),C(26),C(27),C(28),C(29),C(30),C(31),C(32),C(33),C(34),C(35),C(36),C(37),C(38),C(39),C(40)
C TERMINAL VENTILATION EQUATION 6.1.
CH(14) = C(11)*C(12)/BC(14)
1.0175/CH(14) GT. 1.0E-5 GO TO 1700
1700 VI = C(138)
GO TO 1710
1701 TERM = 0.0
C DECISION ON ARTIFICAL O2 TENSION AT CARID BODIES SITE.
1.0401 > RT(14) - 1.0 GO TO 1710, 1720
1.0170 - 1.0 GO TO 1710, 1720
1710 V = C(120)*C(149)+VTRAN(15)+I,0.0 - C(116)*C(149)
C CONTROLLER EQUATION AS A FUNCTION OF HUMID TREATMENTS.
1720 VI = C(201)*C(141)*VTRAN(15)+I,0.0 - C(116)*C(141)
C INCLUSION OF NATURAL COMPONENT AS A FUNCTION OF WORK LOAD.
SVNT2 = SVNT2*SVNT2*W/SO2HWORK) - VI
1.0F(SVNT2GT.0.0) AND (SVNT2LE.15.0) VI = VI + SVNT2
IF(SVNT2GT.15.0) VI = VI - 15.0
C DESCRIPTION OF TRANSIENT VENTILATION RESPONSE.
SVNT = SVNT*(1.0 + 0.75*SVNT)
C EXPIRED VENTILATION RATE, EQUATION 11.1.
1730 VE = VI + D(0)+C(11)*VTRAN(16)+Q(1)+VTRAN(17)- C(110)*F(111)
IF VI + LT. 0.0 OR VE LT. 0.0) GO TO 1740
RETURN
1740 VE = 0.0
VE = 0.0
RETURN
END

SUBROUTINE PC19 (CPA, CVHBA, CVC, BH4, FC)
DIMENSION C(40), XN(40,2), SV(10,50), VTRAN(19), RK(14,4),
1 SC(14,5), DC(15), A(16), D(15), F(20), VOL(10), RMT(2),
2 BC(4), OF(6), TAU(5), CC(13), CHB(13), CH(4), CPH(3),
3 NO(4)
COMMON/Z, C, XN, SV, VTRAN, RK, SC, D, A, D, F, VOL, RMT, BC, OF,
1 TAU, CC, CHB, CH, CP, DQ, VE, VI, CPB, CPT, CDAK, X, BT,
2 IRK, LUC, ITRX, INDEX, I, J, N
C NAMELIST/DFK/CPA,CVHBA,CVC,BH4,FC
C6969 FORMAT(1H BMSUB RC19)
C ITERATES FOR VENOUS BRAIN AND VENOUS TISSUE CO2 CONCENTRATION
C TERM USED IN EQUATION 4.2
1910 X = (CVC - FC) / (0.01 * CPA)
C LOGARITHM SUPPLEMENTARY
2100 X = RC6 (CVC)
CVC = CVC + 2.00 * (X - CVCI/3.0)
IF (ITERX) 1920, 1910, 1920
1920 CONTINUE
RETURN
END

SUBROUTINE PC20
DIMENSION C(40), XN(40,2), SV(10,50), VTRAN(19), RK(14,4),
1 SC(14,5), DC(15), A(16), D(15), F(20), VOL(10), RMT(2),
2 BC(4), OF(6), TAU(5), CC(13), CHB(13), CH(4), CPH(3),
3 NO(4)
COMMON/Z, C, XN, SV, VTRAN, RK, SC, D, A, D, F, VOL, RMT, BC, OF,
1 TAU, CC, CHB, CH, CP, DQ, VE, VI, CPB, CPT, CDAK, X, BT,
2 IRK, LUC, ITRX, INDEX, I, J, N
C NAMELIST/DFK/CPA,CVHBA,CVC,BH4,FC
C6969 FORMAT(1H BMSUB RC20)
C SETS TIME DEPENDENT EXPRESSIONS
C ARTERIAL OXYGEN CONCENTRATION INCLUDING EFFECTS OF HEMOGLOBIN.
F(9) = D(9) * C(2) + CHB(11)
C ARTERIAL NITROGEN CONCENTRATION.
F(10) = D(10) * C(3)
C TOTAL ARTERIAL GAS CONCENTRATION AT LUNG EXIT.
F(11) = CC(11) + F(9) + F(10)
C VENOUS BRAIN OXYGEN CONCENTRATION INCLUDING EFFECTS OF HEMOGLOBIN.
F(12) = C(5) + CB(2)
C VENOUS TISSUE OXYGEN CONCENTRATION INCLUDING EFFECTS OF HEMOGLOBIN.
F(13) = C(6) + CHB(3)
C OXYGEN TENSION IN BLOOD.
F(17) = C(51) / D(13)
C NITROGEN TENSION IN BRAIN.
F(18) = C(6) / D(14)
C PRODUCT OF DIFFUSION COEFFS. AND DIFFERENTIAL BRAIN - CSF GAS TENSIONS
F(14) = C(27) * (CPB - G(12))
F(15) = C(28) * (F(17) - G(13))
F(16) = C(29) * (F(17) - G(14))
C RETURN
END
SUBROUTINE PC21 (CHA, FA, FD, CCA, CPHA)
DIMENSION C(40), XN(40,2), SV(18,50), VTRAN(18), RK(14,4),
1 SC(14,5), DC(14), A(6), D(5), F(20), VOL(10), RHT(2),
2 BC(4), QF(6), TAU(5), CC(3) + CHB(3), CH(4), CPH(3),
3 DQ(4)
COMMON/Z/C, XN, SV, VTRAN, RK, SG, HC, A, B, D, F, VOL, RMT, BG, QF,
1 TAU, CC, CH, CH, CHP, DO, VE, VI, CPB, CPH, CADK, X, DT,
2 IRK, LOC, ITERX, INDEX, I, J, M, N
C6669 FORMAT(IH BHSU RC21)
C NAMELIST/PCC/CHA,FA,F0,CCACHACPHAC COMPUTES H+ ION, PH, AND OXYHEMOGLOBIN N
C ARTERIAL H+ CONCENTRATION.
CHA = CADK*FD/(CCA - FD)
C ARTERIAL PH.
CPHA = 9.0 - RCF(CHA)
C DEVELOPMENT OF EXPRESSION USED IN CALCULATION OF ARTERIAL OXYHEMOGLOBIN SATURATION.
X = RCF2(CPHA)
X = -X * FA
X = iy - EXP (X)**2
X = ABS (X)
C ARTERIAL OXYHEMOGLOBIN CONCENTRATION.
CHBA = X*C(17)
RETURN
P40
FUNCTION RCFA(W)
DIMENSION C(40), XN(40,2), SV(18,50), VTRAN(18), RK(14,4),
1 SC(14,5), DC(14), A(6), D(5), F(20), VOL(10), RHT(2),
2 BC(4), QF(6), TAU(5), CC(3) + CHB(3), CH(4), CPH(3),
3 DQ(4)
COMMON/Z/C, XN, SV, VTRAN, RK, SG, OC, A, B, D, F, VOL, RMT, BG, QF,
1 TAU, CC, CH, CH, CHP, DO, VE, VI, CPB, CPH, CADK, X, DT,
2 IRK, LOC, ITERX, INDEX, I, J, M, N
C LOGARITHM TO BASE 10
RCFA = 0.4342948 * ALOG(W)
RETURN
END
FUNCTION RCF2(ZZ)
DIMENSION C(40), XN(40,2), SV(18,50), VTRAN(18), RK(14,4),
1 SC(14,5), DC(14), A(6), D(5), F(20), VOL(10), RHT(2),
2 BC(4), QF(6), TAU(5), CC(3) + CHB(3), CH(4), CPH(3),
3 DQ(4)
COMMON/Z/C, XN, SV, VTRAN, RK, SG, X, A, B, D, F, VOL, RMT, BG, QF,
1 TAU, CC, CH, CH, CHP, DO, VE, VI, CPB, CPH, CADK, X, DT,
2 IRK, LOC, ITERX, INDEX, I, J, M, N
C OXYHEMOGLOBIN - PH EPPINSICAL FUNCTION.
C EQUATION 3.4
PCF2 = (1.00066815*ZZ -0.10098)*ZZ + 0.44921*ZZ -0.454
RETURN
END
FUNCTION RCF3(KK)
DIMENSION C(40), XN(40,2), SV(18,50), VTRAN(18), RK(14,4),
1 SC(14,5), DC(14), A(6), D(5), F(20), VOL(10), RHT(2),
2 BC(4), QF(6), TAU(5), CC(3) + CHB(3), CH(4), CPH(3),
3 DQ(4)
FUNCTION SVTRAN
C VARIABLES WITH TIME DELAYS USED IN EQUATIONS 8.1-8.1 -
RCF3 = SV(KK,LOC) + SV(KK,LOC + 1) - SV(KK,LOC))*DT/D(14)
RETURN END

FUNCTION SSS2W(X)
C CALCULATION OF STEADY-STATE OXYGEN REQUIREMENTS FOR VARIOUS LEVELS C CF hork LCAO IX=WATTS) -
S502W = .195 * (X/84.15)
IF(X.GT. 210.) S502W = 2.7
RETURN END

FUNCTION SSVENT(X)
C CALCULATION OF STEADY-STATE VENTILATION RATE AS A FUNCTION OF TISSUE C OXYGEN METABOLIC RATE -
IF(X.LT.10.) SSVENT=5.398
IF(X.GE.2.) SSVENT=55.36+50.*(X-2.)
IF(X.GT.195).AND.(X.LT.2.) SSVENT=27.035X
RETURN END
7. BIBLIOGRAPHY


