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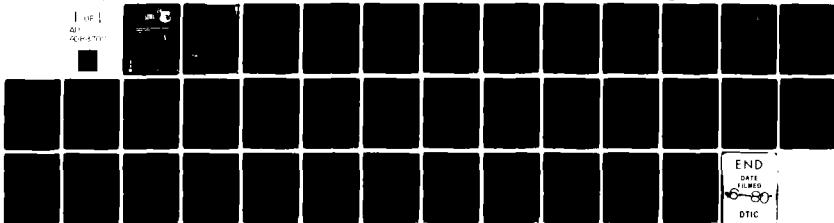
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ACCELERATION AND PERFORMANCE MODELING WORKSHOP, WASHINGTON, DC,--ETC(U)  
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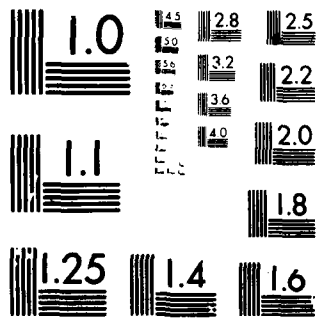
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20. ABSTRACT (Continue on reverse side if necessary and identify by block number) A workshop, sponsored by the Aerospace Medical Research Laboratory, was held to exchange current knowledge in the area of acceleration and performance modeling and to provide future guidance. Representatives from the Air Force, US Navy Medical Research Laboratories as well as distinguished academicians participated in this workshop. Two current modeling approaches were presented and provided the basis of later discussions. The participants responses to this effort in this area were favorable and future topics for another meeting were discussed.		

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PREFACE

One of the principal objectives of aerospace medicine is to develop and apply the technologies required to assess and quantify those attributes that correlate with excellence in systems control within the operational environments of the Air Force mission. The better we understand man's behavior in the process of controlling a machine, the better we can enhance or negate the effectiveness of such man-controlled systems. Such knowledge is also essential for adequate selection of aircrew as well as to the process of optimizing man-machine integration.

The complexity (and the cost) of the process of integrating controller and machine is highly dependent on the degree of communication and understanding between the people responsible for accomplishing the machine design and the people responsible for the man-machine integration and for the ultimate assessment of its effectiveness. The feasibility of tactical and strategic mission accomplishment is directly tied to available resources and state-of-the-art technology. Thus, manned weapon systems effectiveness plays a central role in the process of mission objective formulation. Consequently, effective communication and cooperation is essential between agencies and elements engaged in developing manned systems effectiveness metrics.

The purpose of this workshop was to review current modeling efforts of human performance under acceleration. The discussion focused on approaches to integrate physiological submodels with performance predictive models of aircrew members during acceleration. Participants of this workshop from the Air Force (Aerospace Medical Division, USAF School of Aerospace Medicine, the Air Force Aerospace Medical Research Laboratory, the Navy (Naval Air Development Center), and civilian academic community (University of California at Davis, University of Connecticut, John Hopkins University, and the University of New Hampshire), met on 16-17 May 1979, in Washington, DC, during the annual Aerospace Medical Association Meeting.

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## HISTORICAL BACKGROUND AND MISSION REQUIREMENTS

Roy L. DeHart, Colonel, USAF, MC

The Aerospace Medical Research Laboratory historically has been involved in studies of the physiological responses to sustained acceleration and seat design, performance, and a variety of other activities well before World War II. Today our charter in acceleration research is more limited. The USAF School of Aerospace Medicine (USAFSAM) has a very important role to contribute to the field and it is our effort to try to bring together some of the basic physiology and the performance metrics accomplished in our Laboratory.

For those of you who are not associated with the Laboratory, let me explain our organization. We are a part of the research and development effort of the Air Force Systems Command and we respond to the Surgeon, Systems Command. Our representation to the research and development of the Air Force is through the Aerospace Medical Division (AMD). There are two research organizations within the Aerospace Medical Division charged with responsibility of research; the School of Aerospace Medicine and the Aerospace Medical Research Laboratory (AMRL). Over the years there has been a transition in the programs between our Laboratory and our sister Laboratory in San Antonio. The transition has been one in which we no longer have the primary responsibility to do physiology, nor do we intend to sustain a large physiology program in our Laboratory. Our primary effort in the area of acceleration research is related to human performance. But we are also interested in the causes of the decrement of performance of aircrews in sustained acceleration and what can be done to enhance that performance. In the total biodynamic field for nearly 15 years we have been describing what happens with the human organism in these force fields. We have been reasonably successful, with Dr. von Gierke's leadership, in modeling in the impact arena. We have been less successful in modeling in the sustained acceleration field, although several of you who are here this afternoon have been playing a role as we initiate efforts in this regard.

The Laboratory, for the last several years, has been looking at various modeling techniques to describe man's performance in sustained acceleration. I don't think we can afford to lose the direction in which we are going, however, and this is basically in enabling our pilots to perform in high G fields where they are currently either compromised or unable to tolerate.

Until recently, the airplane could not perform in these high G fields. But today, the airplane can out-perform the man. In the 80's and on into the 90's this will become even more critical, for the airframe and the energy available to that airframe will be such that high, sustained G over prolonged periods of time can occur.

In the 90's, airplanes are going to be flying in an unconventional maneuvering environment unlike anything we have seen before. We are going to have airplanes that instead of banking can simply slip, move side to side, can be augmented in their thrust vectors forward and aft; not that they will stop, but that they can pulsate. In other words,



they could theoretically let somebody suddenly pass them "by putting on the brakes" and then come in on them. There is the possibility of suddenly popping up or dropping. How do we restrain and protect an aircrew member in that kind of environment? What kind of disorientation is he going to have? How does he control the forces on that particular aircraft? There is an enormous amount of work to be done. For once I would like to see the biotechnology community leading instead of following afterward like a little puppy dog.

The moderator for the session will be Dr. Rodriguez. He is the Vice Commander of our Laboratory, and also has the challenging position, currently, as Chief of the Manned-Systems Effectiveness Division. This is his idea principally to gather us together.

We have unique contributions. The School of Aerospace Medicine (USAFSAM) and the Naval Air Development Center (NADC) have unique contributions. I think together we can have a program that will be of value to the tri-services and not just to the Air Force per se. Dr. Rodriguez and Dr. von Gierke have brought you together to talk and to listen to one another and to find out what recommendations, thoughts and views you each have in the area of acceleration physiology modeling.

#### OPENING REMARKS

Enzor Rodriguez-Lopez, Colonel, USAF, MC

I will, in the next few minutes, give you a problem and a challenge. I will also be establishing some rules for the workshop. Our real objective is one of communication and I mean real communication: Finding out what we have in common in our different approaches to the problem. That is the challenge. The challenge then is finding a common denominator; the simplest terms through which we can communicate among ourselves. Around the table we have leading authorities in a number of disciplines that are required to approach the problem: Mathematics, physiology, medicine, engineering. It is difficult for physicians to communicate among themselves or for physiologists to do so. Trying to communicate across other disciplines, gentlemen, is a tough challenge. So, for the first rule of the workshop I will ask you in your remarks, to keep one thing in mind: Make them clear. Keep in mind that some of the other people around the table may not understand the terms that you are used to. Acronyms should be avoided, or at least explained. Present your ideas graphically; use the blackboard. The other rule I would like to suggest to you is focus. Obviously in the two hours left, we will not be getting anywhere unless we keep our discussions on target.

As a point of departure for our discussion we will use the paper by Witte et al, 1975. Let me highlight some objectives outlined in that paper, so that we can reexamine them and perhaps reconsider the strategy recommended by the authors. The technical report was prepared in 1975 and has not been reexamined by our Laboratory since. Some interesting remarks, made in the introduction to that paper, read as follows: "...AMRL is vitally concerned with understanding the relationship between acceleration stress and the resulting piloting performance decrements." "...Effective countermeasures to enhance performance

require a predictive model relating accelerative forces to cardiovascular responses, central nervous system oxygen supply, and the concomitant changes in performance." "...Specific links between altered physiology due to acceleration stress and performance changes have not been adequately developed." "...There is a lack of testable models that relate increased acceleration stress to alterations in physiology, and thence to changes in performance."

Thus, the objectives at that time were clear: To look for the links between performance under acceleration stress and its physiological determinants.

Let us now examine the conclusions and recommendations that were given in that paper: First, to carry out the program described in their chapter on Physiological Aspects of Model Development. Second, to incorporate the features indicated by experiment, suggesting that perhaps two systemic compartment models should be explored and other aspects, such as intrinsic cardiac mechanisms and autonomic nervous control mechanisms, also need attention. Then they go on to say that once we have accomplished that, we should start developing our model and at each step do a mathematical analysis and so on. Since our main concern was not with the physiological research per se, but with the performance end of this modeling effort, some work was done in those four years and I would like to present it to you. Or rather, I am going to ask Dr. Daniel Repperger to present that approach to you. Following him we will have Dr. Dana Rogers who will present another point of view of that effort and then we will open these papers to general discussion.

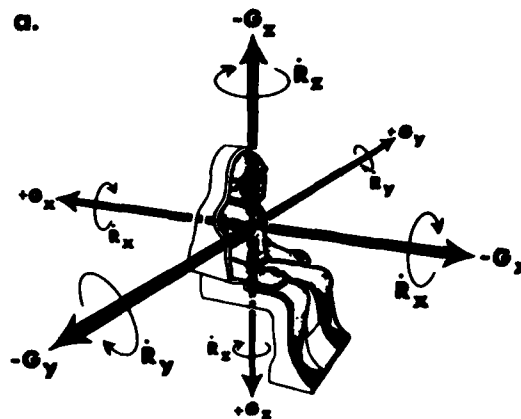
#### ACCELERATION OPTIMAL CONTROL MODEL

Daniel W. Repperger, Ph.D.

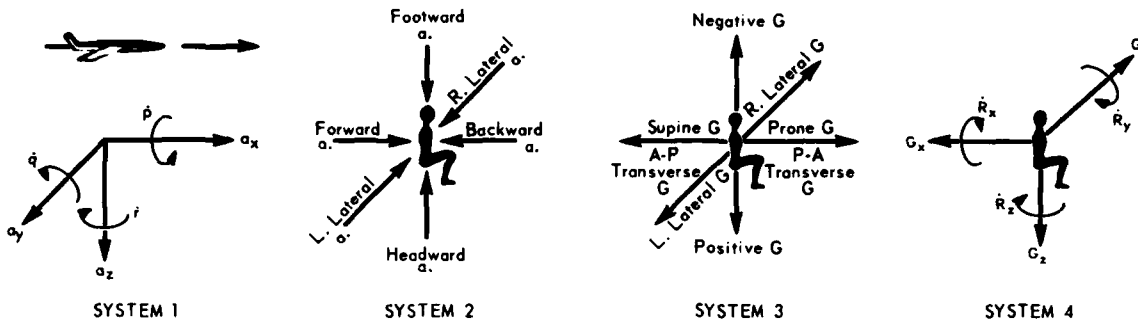
In response to the problems stated by Dr. Rodriguez, one method of obtaining performance correlates under acceleration can be developed by using the optimal control model originated by Kleinman et al, 1970. In a program funded by AMRL for the last couple of years the optimal control model was used to study air-to-air missions that were simulated on our centrifuge. The purpose of these programs was to relate human tracking performance decrements in a quantitative manner to the acceleration variables of interest. It was hoped that such a project could be used in several ways to better understand functionally the effects on a human of the  $+G_z$  stress (Fig 1). The current work involves a  $+G_z$  stress variable as indicated in the diagram (Fig 2) and the performance model called the optimal control model. The work up to the present time has used this model to predict closed loop performance. In the new work that will be proposed here, we suggest that the same  $+G_z$  stress be used and that a type of physiological model that will relate certain parameters in this optimal control model be developed. Through this procedure, going into a different model, we would have a performance prediction. We will explain in a minute how this will be done. In this way we can obtain a correlate between the physiological variable and performance. The reason why this method has a possibility to work is due to certain invariant rules in the optimal control model. These invariant rules are rules that occur in

Symbols and vectors used in this book are based on the direction a body organ (e.g., the heart) would be displaced by acceleration. Table II below--and in particular System 4, which is based on displacement of body fluids--explains the most commonly employed terms.

Source: Adapted from Gell.  
NASA SP-3006



b.



c.

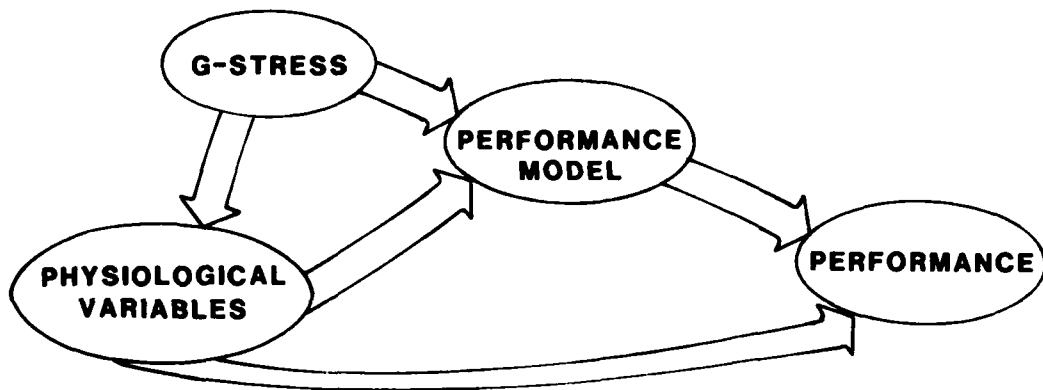
Linear Motion	Table I Direction of Acceleration		Table II Inertial Resultant of Body Acceleration		
	Aircraft Vector (System 1)	Acceleration Descriptive (System 2)	Physiological Descriptive (System 3)	Physiological Displacement (System 4)	Vernacular Descriptive (System 5)
Forward	$+a_x$	Forward accel.	Transverse A-P G* Supine G Chest to back G	$+G_x$	Eyeballs in
Backward	$-a_x$	Backward accel.	Transverse P-A G Prone G Back to chest G	$-G_x$	Eyeballs out
Upward	$+a_z$	Headward accel.	Positive G	$+G_z$	Eyeballs down
Downward	$+a_z$	Footward accel. (tailward)	Negative G	$-G_z$	Eyeballs up
To right	$+a_y$	R. lateral accel. (rightward)	Left lateral G	$+G_y$	Eyeballs left
To left	$-a_y$	L. lateral accel. (leftward)	Right lateral G	$-G_y$	Eyeballs right
<b>Angular Motion</b>					
Roll right	$+p$	cartwheel	Roll	$-R_x$	
Roll left	$-p$	cartwheel		$+R_x$	
Pitch up	$+q$	somersault	Pitch	$-R_y$	
Pitch down	$-q$	somersault		$+R_y$	
Yaw right	$+r$	pirouette	Yaw	$+R_z$	
Yaw left	$-r$	pirouette		$-R_z$	

\* A-P and P-A refer to Anterior-Posterior and Posterior-Anterior

Source: Adapted from Gell

Figure 1. ACCELERATION TERMINOLOGY

- TO DETERMINE PHYSIOLOGICAL CORRELATES, USE A PERFORMANCE PREDICTIVE MODEL SUCH AS THE OPTIMAL CONTROL MODEL



- OCM PARAMETERS ARE "INVARIANT" UNDER STATIC CONDITIONS; VALUES CHANGE WITH ENVIRONMENTAL STRESS

**Figure 2. ACCELERATION-OPTIMAL CONTROL MODEL**

psychophysics like Weber's Law and Fitt's Law\* and they are implicit in the model. From these invariant rules and the use of modeling identification we can gain some insight how to link performance changes to parameter changes and relate this to the physiological problems.

Dr. Green: You have been talking about closed loop performance, would you define what this is?

Dr. Repperger: Closed loop performance would be the correction of the tracking error in the loop; the tracking error is the difference between the desired output of what you are controlling and some reference trajectory. In other words, if there is an error you correct it.

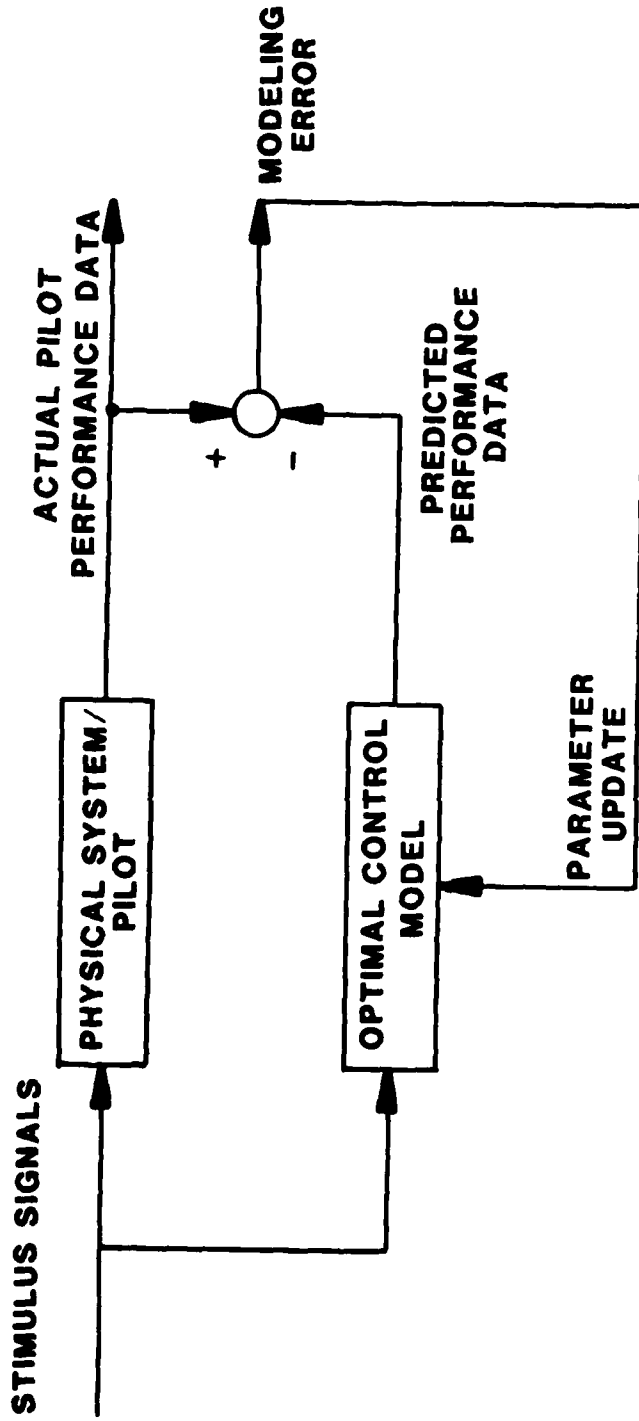
How do we accomplish this difficult task in matching the model to data? The key in accomplishing this task is in the model identification (Fig 3). We take a physical closed loop system involving a man-in-the-loop and excite the closed loop system with a stimulus signal in the static condition as well as in an acceleration stressed condition. This would be the target trajectory or the desired trajectory that you wish to track. This signal excites the closed loop man-machine system; as a result, some closed loop performance measures are obtained. In the identification phase parameters are obtained which describe the human in both the static and the stressed condition. If we compare the two sets of parameters in both these conditions, it is noted that the optimal control model has the same stimulus input signal as in the experiment and the model generates an estimate of the closed loop performance. The difference between the model's estimate of the performance and the true performance is called the modeling error. Ideally, if we modeled this situation exactly, the modeling error would be zero. In this way the model would be a perfect representation of a physical system. Due to randomness\*\* and noisy measurements and things of this nature, it never occurs. Generally the modeling error is a time series which has random properties. That is, there is no correlation in this time series. If the modeling error did not appear random in the time series, this indicates that the model has not captured the true correlation between the input and the output.

So what is done? First, we take the static condition with no stress and the man-in-the-loop is modeled with the

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\*These laws determine the relationship of speed and accuracy of visual observation of signals (Weber's) and of hand control (Fitt's). The product speed x accuracy is an invariant constant.

\*\*Randomness encompasses not only individual variability, but also errors in the measurement and recording of empirical data.



**PRIMARY OCM PARAMETER CHANGES:**

- INDIFFERENCE THRESHOLDS INCREASE WITH G.
- MOTOR NOISE STRONGLY DEPENDENT ON G  
(SEE VIBRATION RESEARCH).

**Figure 3. MODELING PHASE: MODEL IDENTIFICATION**

parameters updated in such a way that the modeling error has essentially no correlation. Then the stress condition is considered. In this case the stimulus signal into the loop includes not only the trajectory that has to be tracked but, in addition, the  $+G_z$  stress signal. In this case what has happened is that the model uses the old parameters, that is the parameters under the static condition, and when the model is fitted to the data, it turns out that now the modeling error has correlation. That is, something happens in this man-machine system between the stress and the non-stress condition. We now ask the question, what could cause this condition because the only difference between the stress and the non-stress condition is the man-in-the-loop, since the display is the same, the stick is the same, and its controlling element is the same? Therefore, the effect of the stress on the man manifests itself through the modeling error into different sets of parameters. So we see now that we have applied the modeling procedure twice. First in the static condition with no stress and have obtained one set of parameters. We applied it again in the stressed condition and obtained another set of parameters.

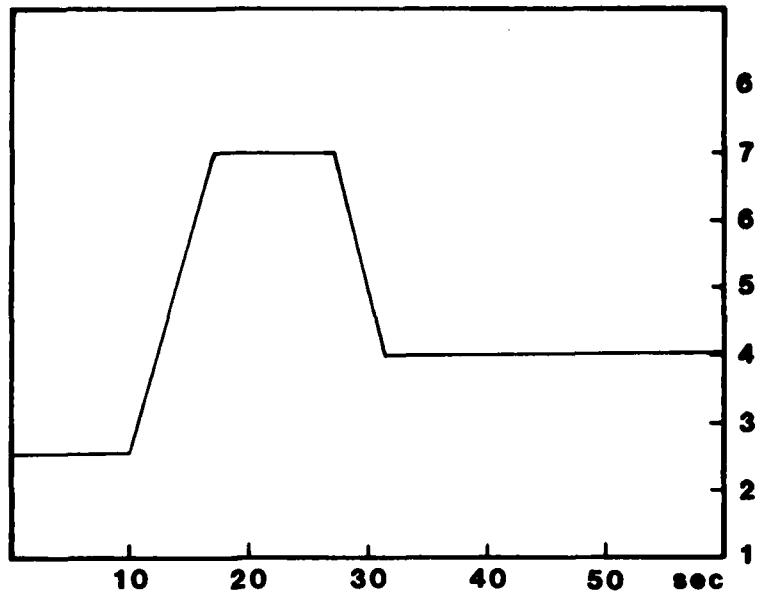
Now the question is asked: What makes those parameters change? Clearly, this must have some link to physiology. Some results so far on the work up to the present time, between the stressed and non-stressed condition, is that under the effects of  $G$ , the indifferent thresholds\* change with respect to the human. This means that the human will trade off pain for performance. That is, under high  $G$  stresses, the human has the tendency to let the error signal become larger than it normally does. A second result of the present research is that the motor noise depends on  $\dot{G}$ , where  $\dot{G}$  is the time rate of change of the  $G$  vector on the pilot. This now gives us some insight. Remember that we have a static and a dynamic condition and it is known that under the dynamic condition we have to change the parameters relating motor noise which is translated into a hand response. This tells us implicitly that in the model the human must have had an effect to change his hand motor dynamics. Therefore, this effect gives us insight as to which physiological model has been affected by the stress. In this case it's a hand or neuromotor type representation. So, now we can build a physiological model to correlate these two parameters of the hand between the stress and the non-stress condition. In this way the model has given us insight into which physiological system should be used under that stress condition.

Let us see how the model is used in a typical application. Here we have a target forcing function (Fig 4a). This is the trajectory that the pilot has to track.

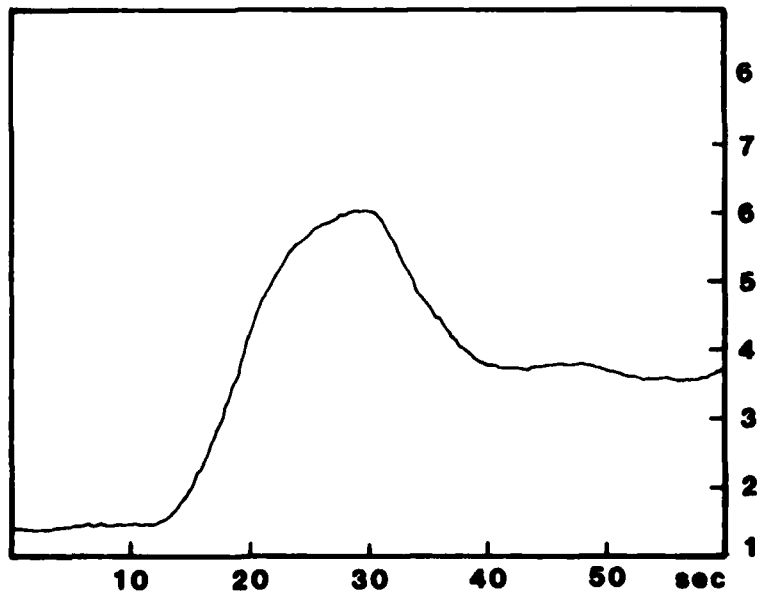
The Dynamic Environment Simulator (DES) having a large mass, doesn't always have the capability of turning those

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\*Indifferent thresholds. Refers to levels below which the human is indifferent to a stress (and above which the human is significantly affected by it).



**a. TARGET COMMAND G PROFILE**



**b. ATTACKER ATTAINED G PROFILE**

**Figure 4. a-b THE COMMAND AND ATTAINED ACCELERATION STRESS PROFILES**

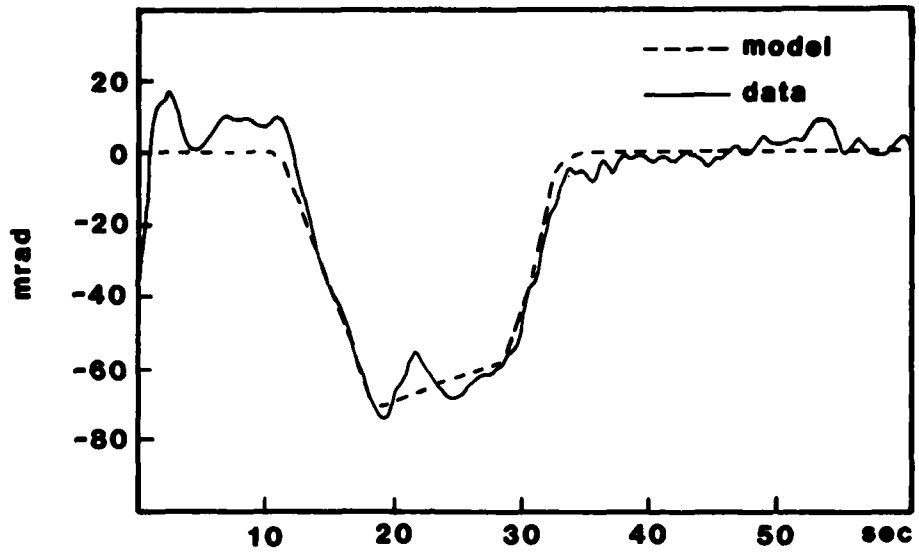


corners or following the G command profile in Fig 4. But we should like to point out that the DES has enough of a dynamic range that we can make some observations about certain characteristics of this type of experiment. Fig 4b illustrates the attained G profile as a result of the DES operation.

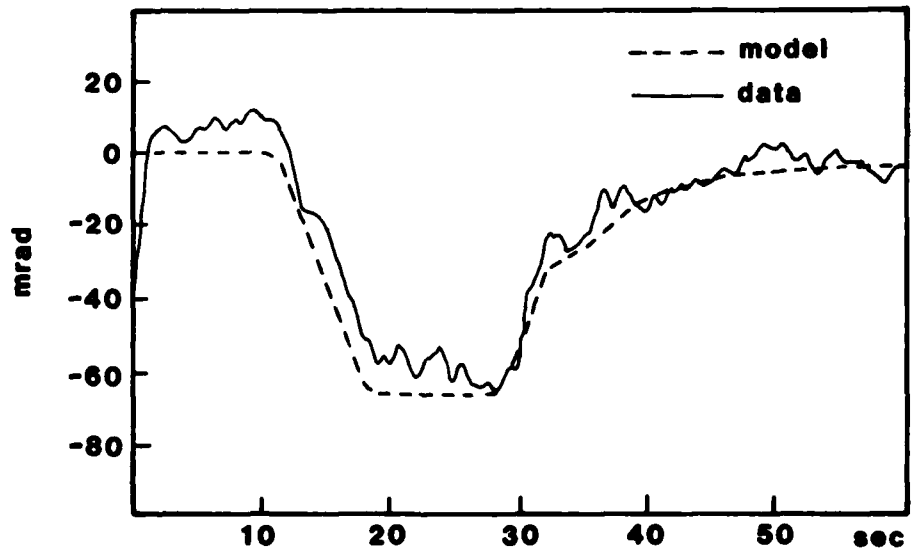
In the application of the model we take the static condition and in Fig 5a-b, we have a plot of the mean tracking error, which is the response of the performance of the human versus time for that particular trajectory illustrated on the last transparency (Fig 4). We notice that the solid line is the empirical data, which is averaged over replications and the dotted line is the model. Correspondingly, the model is fit using different parameters to the dynamic data (Fig 5b). Here we have the empirical tracking error ensemble mean represented by the dotted line. Now you notice that it is not obvious which parameters were adjusted for the modeling, but you can see correspondingly what the fits are. Now that we have an estimate of performance, we ask the question with what confidence do we have an estimate in this performance? For example, the model's estimate of this performance can be no better than the experimental data itself over replications. So we look at the second moment of this variable; the standard deviation (Fig 6). It is seen that the model has a certain confidence in this estimate of the performance over the replications indicated by the dotted line. The solid line is the average across replications. If the model were too conservative a guess on this type of estimate, it would have a large standard deviation. If it were too liberal in some sense, it would be the other way. But the model's estimate of the data can be no worse than the data. It should be no better either. So, in the static condition we would have it fit correspondingly in the dynamic condition. Therefore, we have two sets of parameters here. Another thing is noticed and that is the variance of the estimate seems to be larger in the dynamic condition versus the static condition. This is true of most of the stress experiments. The variability of your estimate of any parameter generally is greater in the stress conditions than in the non-stress conditions.

We said before that the model has certain laws of invariance. Using this same model, Professor Kleinman took those parameters from the static case and he changed the dynamics of the system, that is the aircraft dynamics, (what we call the system being controlled). In the last slide (Fig 5-6), the controlled element was the pitch axis; in Fig 7 the subject has to control the roll or lateral axis and Professor Kleinman inserted the dynamics from the lateral axis in the model simulation. We see plotted the results with the new system dynamics; the dotted line is what Professor Kleinman thought the response would be. Correspondingly we obtained some data that shows that the invariance of the model seems to show across tasks as well as other relationships.

How can we extend this work so that it can be used to study physiology? In the prior work which has been displayed so far, we have our  $G_z$  stress; we also have the nominal parameters obtained from the identification. We have two sets of parameters, the parameters in the static case and the parameters in the dynamic case. What we don't

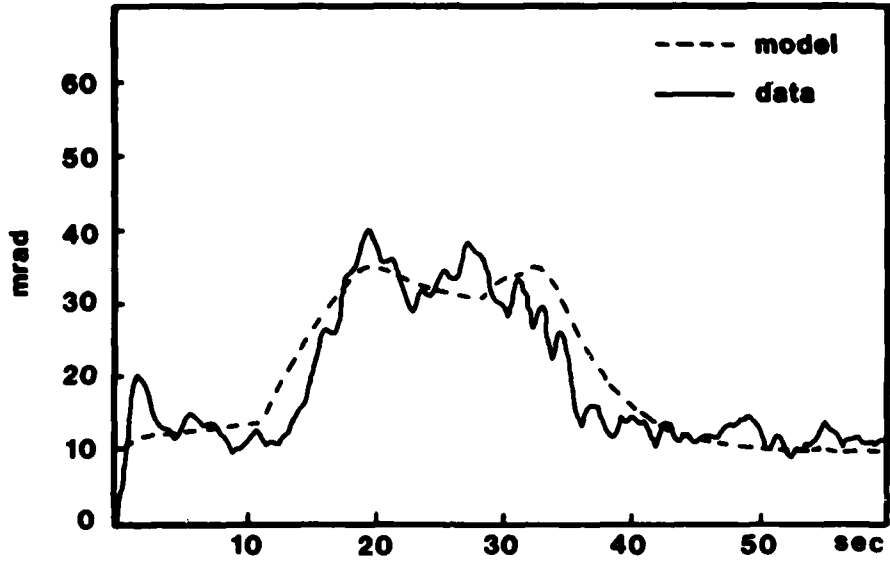


**a. MEAN PITCH TRACKING ERROR, STATIC G  
DATA-VS-MODEL COMPARISON**

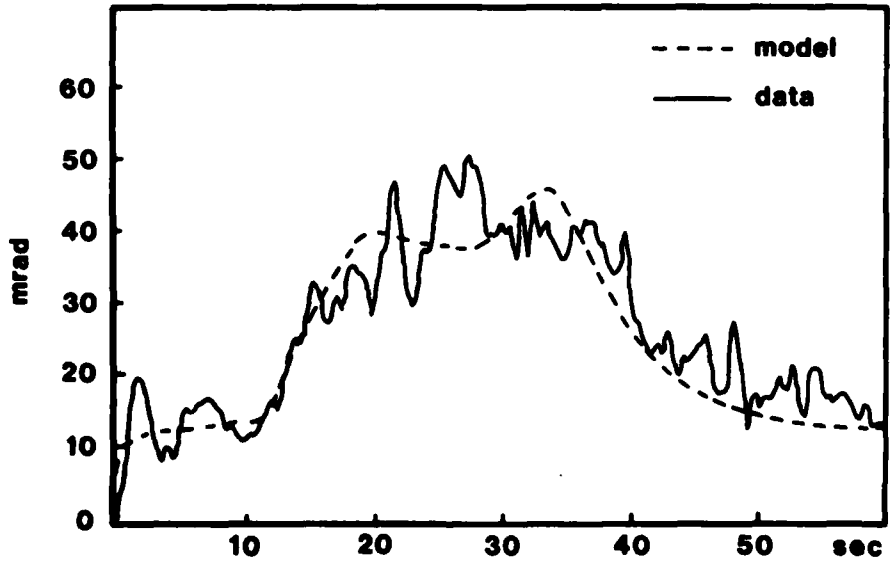


**b. MEAN PITCH TRACKING ERROR, G STRESS  
DATA-VS-MODEL COMPARISON**

**Figure 5. a-b COMPARISON OF MODEL TO  
EMPIRICAL TRACKING ERROR**

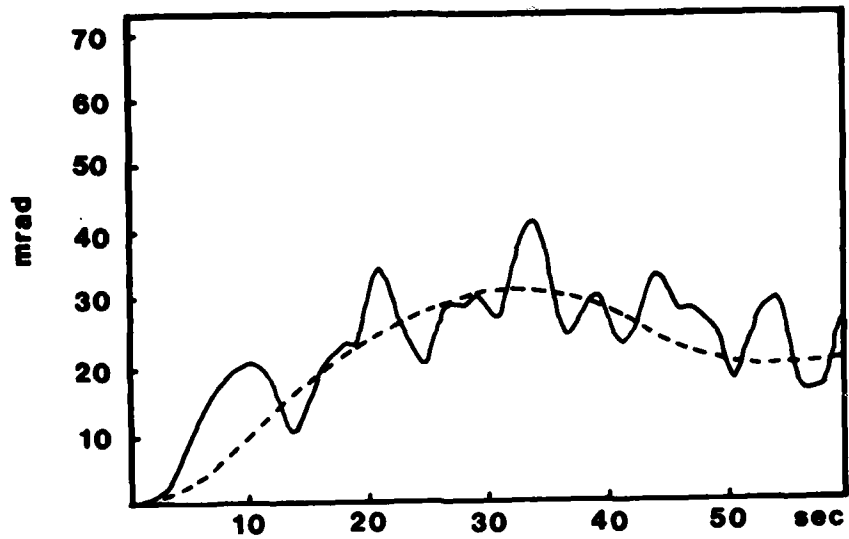


**a. STANDARD DEVIATION PITCH ERROR, STATIC G  
DATA-VS-MODEL COMPARISON**

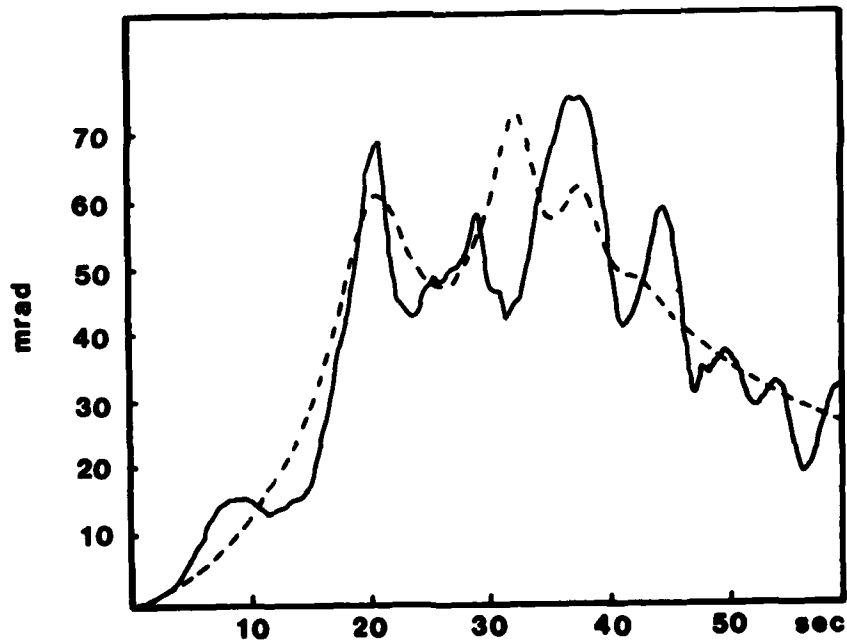


**b. STANDARD DEVIATION PITCH ERROR, G STRESS  
DATA-VS-MODEL COMPARISON**

**Figure 6. a-b VARIATION OF MODELS ESTIMATE**



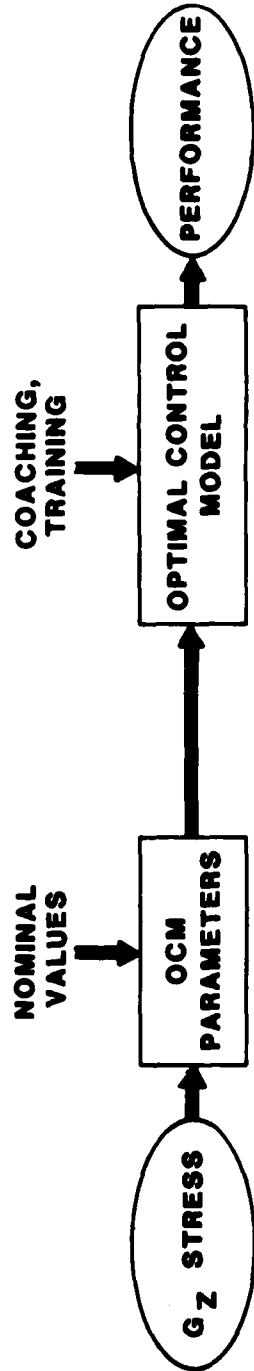
**a. LATERAL TRACKING ERROR-ENSEMBLE  
STANDARD DEVIATION  
STATIC G**



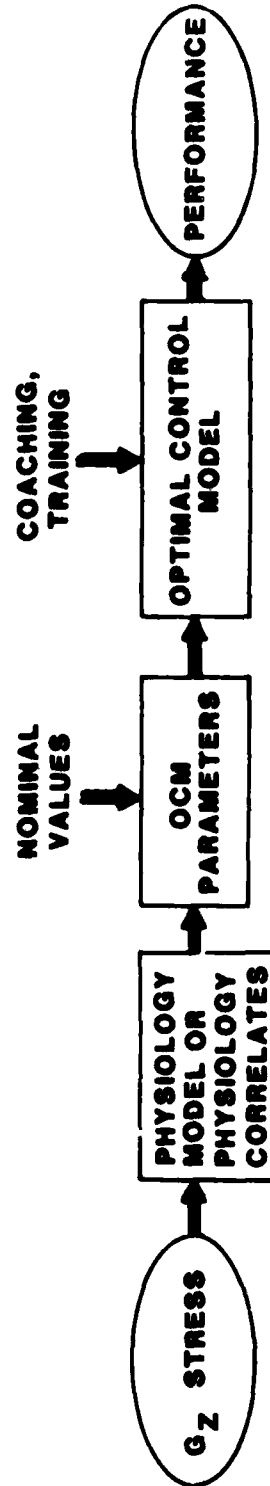
**b. LATERAL TRACKING ERROR-ENSEMBLE  
STANDARD DEVIATION  
DYNAMIC G**

**Figure 7. a-b LATERAL TRACKING ERROR AND MODEL ESTIMATES**

**PHASE 1:**



**PHASE 2:**



**Figure 8. EXTENSIONS OF THIS WORK TO STUDY PHYSIOLOGY**

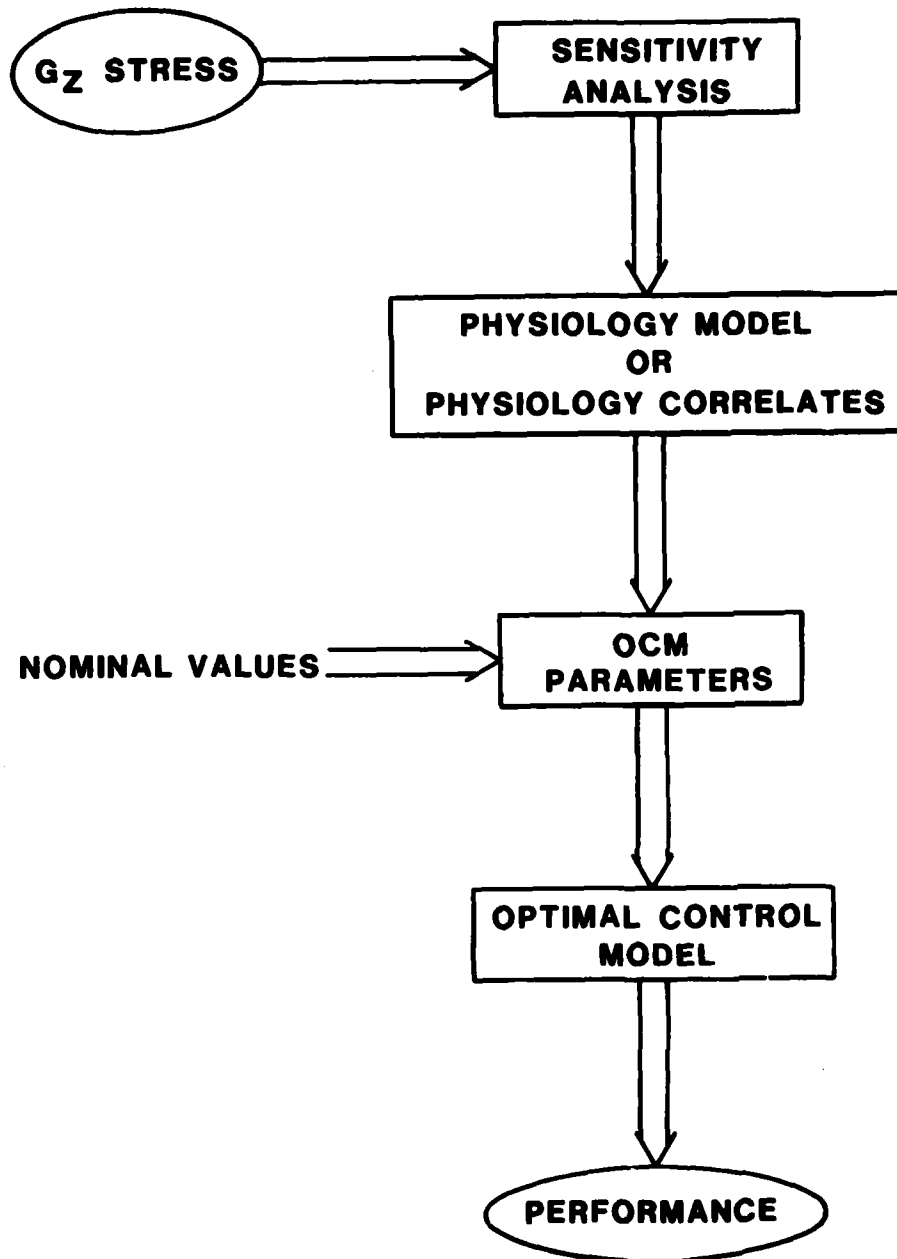
understand at this point is why are those parameters different. Why are they different from phase 1 and phase 2 in Fig 8? It turns out that in the first phase of this study we have different sets of parameters in describing the performance. The important point to be made now is that first it appears that "this modeling is independent of physiology." But it is observed that we have obtained different parameters under different physiological conditions. So, the linear dependence between the parameters must be implicitly described in the physiology effects that occur. The new work that is proposed here would be to take these different sets of parameters, static and dynamic, and in phase 2 to relate static and dynamic parameters to a single rule which becomes a physiological model which correlates these parameters. Therefore, the  $G_z$  stress now changes some physiological model which goes through our prior model and becomes a physiological correlate to performance. Another way to look at this approach is as follows: Let's use a term called sensitivity (Fig 9). We take our  $G_z$  stress vector and suppose we have two experimental conditions, static and dynamic, or non-stress and stress. We will look for certain parameters which describe the process. Between the two conditions, if a parameter changes a lot, we will say it's insensitive. So, by looking at which parameters change between static and dynamic cases, must reflect physiological correlates because that is the only variables that can change between the static and dynamic conditions. This is true because the two experimental conditions are essentially replications except for the man-in-the-loop. Sensitivity analysis tells us which parameters are sensitive and they are actually physiological correlates which relate these parameters in the performance model. Therefore, sensitivity implies physiological correlates.

In conclusion, we propose that the following factors can be used to obtain information on physiology.

- o HOW CAN A MODEL BE USED TO OBTAIN INFORMATION ON PHYSIOLOGY?
- o WHAT OCM PARAMETER IS IMPORTANT IN THE MODELING OF THE STRESS DATA?
- o EXTENSION OF THE MODEL (OCM) TO BETTER UNDERSTAND THE EMPIRICAL DATA.
- o PERFORMANCE PREDICTION UNDER DIFFERENT ENVIRONMENTS THAN CONSIDERED HERE.

It is only necessary to apply identification of parameters to static and dynamic conditions. By using this procedure, it is seen which parameters change and we study these parameters with a great deal of care which means the optimal control model can better be used to understand the data. Finally, when we have obtained a physiological model which can relate changes in parameters to changes in  $G_z$  stress, then we have enhanced the ability of the model to predict under wider varieties of environments. That is, we can then determine performance prediction under different  $G_z$  types of profiles by using these physiological rules or relationships.

**SENSITIVITY UNCOVERS IMPORTANT VARIABLES**



**Figure 9. SENSITIVITY USED TO DETERMINE PHYSIOLOGY**

A MODEL FOR THE ENERGETIC COST OF  
ACCELERATION STRESS PROTECTION IN THE HUMAN  
Dana B. Rogers, Ph.D.

Coming at the same problem from the other side of the house, I will first discuss a general stress performance model which encompasses the subsystems subject to the effects of stress and then, in turn, affect the parameters which are identified in the control model. I will present a generalized structure, then break this down into submodels which have defined linkages in terms of oxygen and pressure. Then I will reassemble parts into a test case of an energetic systems model and use portions of that energetic model to show you some of the predictive things that can be done and then go back again and tie these factors in to the control model. If we were to contemplate the general problems of physiologic and protection models and performance models this (Fig 10) might be one structure we could work with. The first grouping is that of protection models (left side of Fig 10). The protection models relate to supports and restraints, the configuration of the cockpit, the ability of the G suit to enhance the person's capability to withstand excessive acceleration and the models that, perhaps, relate the techniques of restraining the person in the acceleration environment. There are certain biodynamic and physiologic models driven by the protection models (center Column of Fig 10). There is the proprioception system, including the disturbance of the muscular systems, perhaps changes in spindle fiber output, and changes in the perceived weight of the muscle because of the acceleration force.

The cardiovascular system has been studied in many ways and, in certain cases, has provided some very good information to allow us to relate back to other thing . The ventilation system provides (together with the cardiovascular) the capability to intake oxygen and then distribute it to the muscle masses and to the cortex so that it can operate as a decision system. The proprioception and the cardiovascular and the ventilation models all feed into the perception models and the output models which are identified here as the performance models (the two columns on the right-hand side of Fig 10). Within the perception models we have the vestibular system, reacting directly to the acceleration changes; we have the vision system, reacting to the cardiovascular system and to the ventilation in that it takes both oxygen flow and adequate pressure to the visual system for operation. We have the decision system, located in the cortex with its influence on the outputs. Audition, another input to the decision system can't be overlooked at this point either. The output models which are determining performance are essentially tied to muscular systems, through manual control (hand and feet), and through speech, another muscular output.

In normal activities the pilot, who senses changes in the visual system, the acceleration vector, has to decide how to perform his tasks. He has the option of unloading his aircraft, that is reducing the acceleration vector in his aircraft; he has the option of straining in some manner to increase the pressure of the eye. Is he going to strain for three seconds, take a breath, strain for three seconds, take a breath? Is he going to strain for five seconds, take a breath? He has



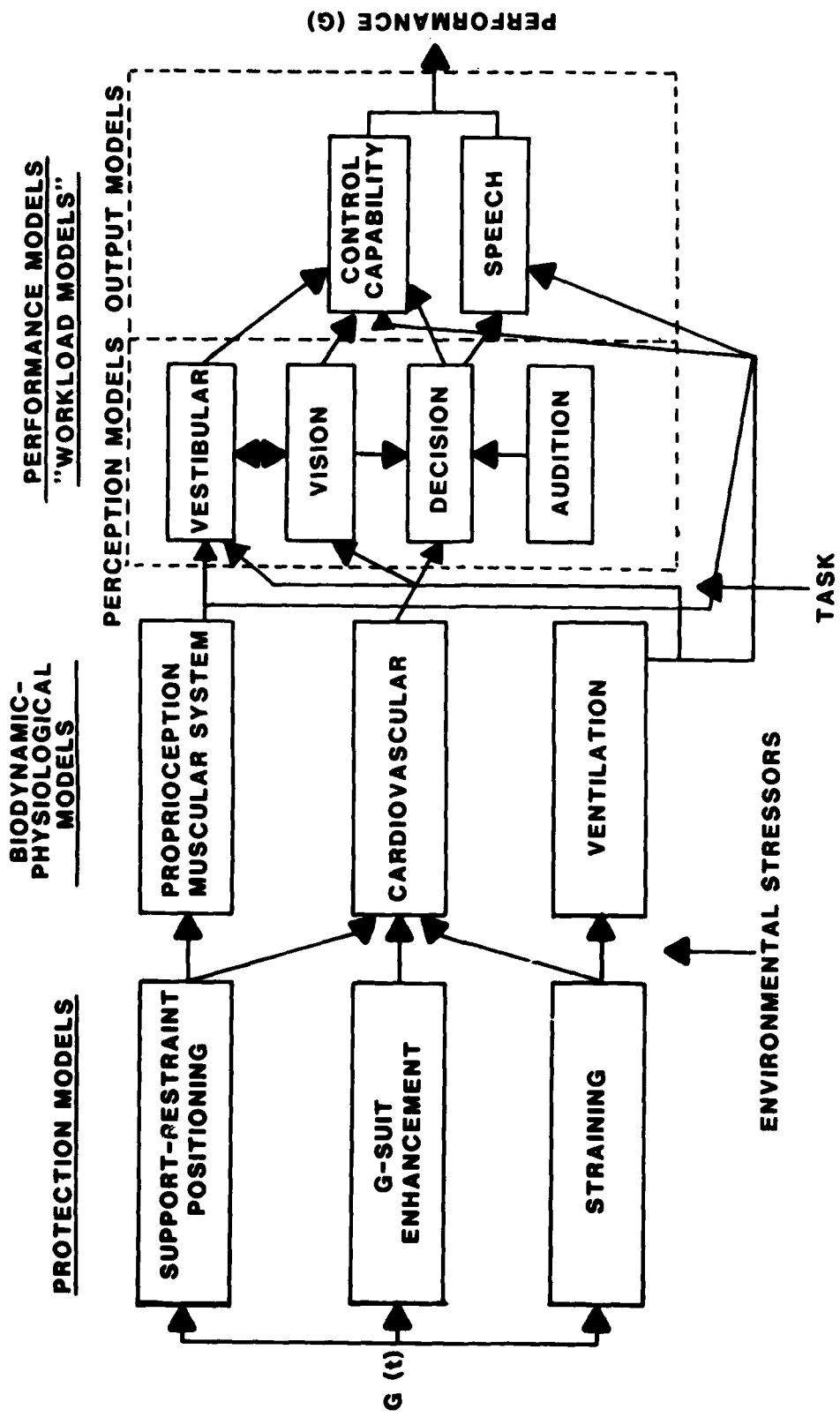


Figure 10. PERFORMANCE CAPABILITY UNDER ACCELERATION

to develop this effort strategy to the best of his ability, so that his protection system delivers the appropriate pressure and the appropriate oxygen into his visual system. His effort strategy, however, is causing him to lose energy in the terms of the amount of oxygen consumed in his system. When he strains his muscles he may be doing anaerobic exercise, but he has to replace those energy stores at some point. The ventilation system is affected because the blood shifts in the acceleration field. The lungs aren't able to perfuse oxygen across the interface and are unable, in an efficient manner, to establish a full supply of oxygen in the blood identified as a percentage of arterial oxygen available,  $PA_{O_2}$ . The vestibular system is affected by both the pressure changes and the lack of oxygen. The decision filter now is processing information from the visual system that is being affected by lowered perfusion pressure and lack of oxygen and it is also receiving inputs from the vestibular system. Now this decision filter has to establish a control strategy. It has to establish observations of what is going on. It has to pass information through this filter and therefore is affected by the visual system. It is easy to see, at this point, that all of these systems are tied closely together. It is difficult to linearize and separate a system which is obviously nonlinear. However, this is one approach that we can begin with so that we can establish preliminary models and then develop these models. In most cases, however, there is very little information available in the appropriate form.

Let us now look at these parameters as an energetic system. By energetic system we mean a system which looks at how much is this going to cost in terms of the energy expended and the intake of oxygen needed to replace it so that the subject can continue to go on. What kind of effort strategy is he going to define so that he can optimize his ability to not only withstand acceleration, but to be able to stand it for the longest period of time? Does he really want to extend the period in the acceleration or to establish a system which gives him the highest possible acceleration protection for a specific point in time? (See Editor's Note.)

#### DISCUSSION

Dr. Green: What concerns me the most is that many of these models may be based on inadequate basic physiological data. In recent years a tremendous amount of work related to the term "interdependence" of cardiopulmonary function has been accomplished. In essence this concept states that the chest wall interacts with the circulation. In other words, interdependence of systemic circulatory mechanics and pulmonary mechanics dictates the interdependence of vascular capacity in the pulmonary system and the vascular capacity of the systemic circulation. In addition, you have the interdependence of left ventricular function with right ventricular function. If you change pleural pressure you are changing the afterload of the ventricle.

EDITORS' NOTE: The full description of the models are described by Rogers (3).

These questions have not been adequately investigated and are just beginning to be addressed. I think that all this must be adequately addressed from a basic physiologic point of view before they can be firmly and adequately incorporated into any meaningful physiological model. One other comment that I have is related to a general philosophy of models. From a physiologic point of view, I tend to approach it differently; rather than taking a black box and putting it in terms of input and output and trying to get a transfer function, I prefer to begin asking what do I know about that black box? In terms of the cardiovascular system, we have an arterial system feeding into a venous system. The arterial vessels and venous vessels have certain hemodynamic functions which can be described as capacitive function and resistive function. Now if we look at the function in each serial section we know that the function is different. In other words, on the arterial side of the system we have primarily resistive functions whereas; on the venous side we have primarily capacitive function. Yet even within the vein the capacitance is distributed non-uniformly. It is at the level of the smaller venules that we have the primary areas of vascular capacity. We have, in the past, taken a very elementary and simplified view of this and put it into a single channel lumped parameter model of the systemic circulation. In this single channel model we described the return of blood back to the heart as the pressure gradient between the capacitance areas and the right atrium divided by the resistance to venous return. We further define the upstream pressure to venous return as simply that pressure within the small veins and venules (called the mean systemic pressure) which is also the ratio of the stressed volume of that area divided by the compliance of the small veins and venules. In the past, with such an approach, we have attempted to show how a very simplified model along these lines can go a long way toward providing an explanation for the changes in the pressures and flows which are observed in both dog and man under acceleration stress. We published a paper several years ago (Green and Miller, 1973), that came pretty close to predicting those responses. However, that paper was a very elementary approach and attempted only to show that by looking at the physiologic parameters from the basic hemodynamic point of view we can arrive at a box which has something in it which we can interpret.

Dr. Rodriguez has referred to the development of more sophisticated predictions. When we talked over the phone prior to coming to this meeting I began thinking again along these lines and began to realize that we could develop a two-compartment model which could be composed of an upper level vascular compartment and a more dependent vascular compartment. We can derive flow equations for each compartment essentially identical to what we did for the former compartment lumped parameter model. When you study such a system what you find is that there is an intravascular redistribution of volume from the upper level compartment to the lower level compartment. Thus, immediately upon applying an acceleration stress, the flow and the arterial pressure come down. Now, as the volume is distributed from the upper compartment to the lower compartment, the pressure in the lower compartment rises back towards normal, returning the flow and pressure, so you have an immediate drop in eye level arterial pressure which slowly over the next

few seconds returns to normal. This is a simplified approach assuming that vascular compliance is totally linear and independent of transmural pressure which we know not to be the case. In fact, what we know to be the case from isolated limb studies is that, as you increase the volume in a vessel you are going to decrease the compliance. With an incremental increase in volume the pressure is going to get larger and larger. If you were to apply this knowledge to the two-compartment model you would find that as the intravascular volume is being distributed to the inferior segments of the body an even more pronounced change occurs in cardiac output and arterial blood pressure. But there are an awful lot of questions that need to be answered. Some questions we just don't have answers for. For instance, exactly how are these model parameters a function of transmural pressure? What I said can be considered extrapolations from very basic isolated limb studies.

Another question is that the volume of the systemic circulation is not simply stressed volume, it is also unstressed volume. There is a lot of work in the literature lately that shows that the unstressed volume is capable of changing and indirectly altering flows and pressures. Another recent observation from our laboratory is that, when we produce pulmonary alucolar hypoxia, there is a tremendous shift in blood volume from the vascular bed of the lungs to the systemic circulation. I can go on and on in terms of the basic physiologic mechanisms we need to investigate before we can put basic principles into an adequate model. Many of those ideas were itemized in the Witte, et al. paper (1). I guess what I am trying to say is that I think that techniques of modeling are extremely powerful. Yet what we need is basic information to go into those models. Looking at the system from the physiologic point of view I don't see the information at hand that would allow us to adequately describe these systems. Another very important aspect of which we have become aware in recent years is what are the appropriate model parameters to use for man. There appears to be tremendous species dependency on model parameters. For instance, the animal dog model can be described very adequately by two-compartment model parameter. We can measure the various time constants in the various channels involved and come up with two parallel compartments. There are also other reasons why the dog is hemodynamically described by the two-compartment model. But that may not be a good description of man. To identify appropriate parameters and test the prediction of any models that we come up with, we would have to go back to the physiologic systems of that prediction. The question is what is the appropriate model to be used. This is another area of strong concern of mine.

Anyhow, if I can summarize what I have been thinking for the last hour: These are powerful tools. I think we need to build on those tools by establishing such programs not currently supported by either the Air Force or by NIH which are to investigate mechanical parameters and how the mechanics of the system behave. Another area that might require and might be good to investigate is the idea of interventions. Perhaps it might be appropriate to investigate the various effects of pharmacological agents. There are all sorts of drugs that we might use as an inhalant which, given in the appropriate time, might produce a dramatic response in physiological function. It might also be appropriate to use current techniques to investigate the physiologic

basis of the G-suit with the idea of possible redesigning this suit.

Dr. Ehrlich: First of all I believe that if we concentrate only on performance we won't get anywhere; we won't understand the performance problem if we don't resort to physiology. I am very impressed by your methods, but I feel that it is dangerous if at this stage your model can predict performance. This is dangerous because then we might think that we understand. I think Jerry is right; we don't understand very much at all. For example, say that the blood fails to reach the eye in sufficient amounts by virtue of the Laplace effect on the parcel of blood in the descending aorta and carotid artery. Is that all there is to it? If that is so, are we saying that the parcel of blood next to the aortic valve is not also being accelerated? But I think that when you have  $G_z$  acceleration that parcel of blood, which is being accelerated downward does have an effect by increasing the afterload on the ventricle. Dr. Rodriguez himself has shown the dramatic effects of sudden increases of afterload on the left heart. I think that effect may have been overlooked. That's just an example and it could be all wrong, but if it is not, that fact alone could decrease the cardiac output significantly, even before the effects of a decreased venous return came into play. What I'm saying is that we have to look at all the aspects of the circulation; not just peripheral effects; not just venous return. I don't see how one can look at portions of the system under the G stress and not others. Excuse me if I tend to focus on a narrow problem but I think that we don't have the strength to attack all the problems at once. It is better to solve one square in the black box at a time.

Dr. DeHart: In terms of our Laboratory and the Air Force I really don't care what happens in the black box. Perhaps, I am overstating this view. Our purpose is to increase the performance envelope for our aircrew. We have already been successful in doing that through a variety of ways without fully understanding the total circulation aspects of the human physiology. I don't expect our work or what we may subcontract will result in a solution to Guyton's or anyone else's model in terms of total circulation. We have been able to describe the metabolic pathways without fully understanding the Krebs cycle. Our Laboratory is not involved in physiology per se. We have been able to describe successfully and in some simple ways performance in terms of modeling. Dave Kleinman and others have been able to predict, to some degree, where it is starting into the predictive mode and that is what we want to be able to do. We may not understand everything that is going on, but if over a large number of subjects the models that are derived are predictive, that's what is important.

Dr. Rodriguez: The data for the models has to come from some place. Whether we do the physiology or somebody else collects the data, the information has to come from someone. I don't want to leave today without making a point that we have important sources of data within our laboratories. We are working towards providing any data we generate in the process of our performance studies at AMRL to the other laboratories. What I am really asking for is the pooling of that data from wherever it is obtained, to be shared by all.

Dr. von Gierke: I would like to clarify Dr. DeHart's statement when he says we don't care what is in the black box. I guess we don't care about it if we can't do anything about it. I guess that is really how all this modeling of control systems developed when we knew we cannot do much about it, let's get the input/output function and work with it in the human engineering and control environment. But, I think we all are reluctant to throw anything into the black box if it is a parameter we can do something about and we know how it is composed and how it can be modified and influenced. So I think having an overall control model input/output as a black box just doesn't make any sense to me if there are components in it like simple components such as position. The upright versus reclined positions and similar things where we know we can do something about it and we can introduce this particular subsystem very nicely and cleanly into the model. Coming back to the report we discussed and to the outline that Dr. Rodriguez has here, I think the biggest hole in this report and in this model is that the whole proprioceptive system, the position of the hand, and the changing of the manual capability without the increased G load is immersed in the model. Although we know a lot about it and as Dana Rogers and Dan Repperger indicated, we know from the vibration study what the transfer functions, the control capability of the hand/arm system are under G load. Probably some more experiments should be done on this particular control system alone and how it is modified. But I certainly would put this ahead of the final black box and study the subsystem because there is a lot we can do about it. We can change the position of the hand, we can change the control stick, we can change the support of the hand, the direction of the hand, all these things. If that is all in the black box we end up in an infinite number of transfer functions and parameters and it doesn't make much sense. The G suit problem is another subsystem which I think would be nice to take out of the black box. I agree with everything Dr. Green said about the physiology and the interrelationship of the biomechanical factors and the various types of hemodynamic factors and their relationship. I am not sure that all these details have to be known before we can make a useful submodel of this section of the overall model. At least with respect to the parameters which we can modify and we know we can modify. There is a G suit and there is pressure breathing and particularly for these two aspects there are beneficial effects which may have been shown and the technology can lead us to additional ways of modifying, time-wise, the time cost of applying the G suit and the positioning and the pressure breathing. I think modeling this subsystem outside of the black box will bring us great benefits. So I think it is completely wrong and absurd for me to talk about one or the other. I think that the black box approach is just admitting that I can't do anything about the black box and I had better use input/output as it is.

Question: Do you exclude the possibility of introducing some pharmacologic agent at certain times?

Answer: No.

Question: Do you, for example, change the breathing mixture to increase the concentration of CO<sub>2</sub>?

Answer: Right.

Dr. Gillingham: I would like to offer a different perspective on this subject. Perhaps it is a different version of the same perspective, but what has been bothering me about this whole discussion is that I am not sure that we know where we are going. What is it that we really want to get? I think that what you want to get is to be able to determine performance as a function of G. I want to know how strong the data base is in that area because I don't think there is any point in going anywhere without that data base. You can get into a detailed synthetic approach which is very, very expensive and very time consuming, leading you astray. It would be 1999 before anything happens in terms of obtaining a performing conceptual model. It would seem to me that the simplest model necessary to provide the required prediction is what you want; and if that simple model doesn't work, then you start looking at the physiologic parameters that are going to explain it at the level of resolution that you really need. You could ask what aspect of the physiology is necessary to explain satisfactorily the relation between the G stress and the performance, but we are operating backward if that is the case. I think that it is very appealing and satisfying from the scientist's standpoint if we go ahead and try to put together the various components of the total system; but it is also very expensive, and I am not sure that it is the best way to approach it. I also would really like to know where the data are that describe performance as a function of G. It seems to me that this should be studied almost exhaustively before getting into the complicated physiologic model. See what the defects in the simpler predictive models are, first.

Dr. von Gierke: The question is what do you call performance? That is the main question and I think that is well illustrated on that model which Dana has shown which has the inputs of G stress and the outputs of performance and the actual output a human being can give is control and speech. It also has information processing and noise. Now, this can all be modified by our human engineering. So performance is a very nebulous term and it is an infinite thing to study unless you have very specific tasks in mind.

Dr. Kleinman: I feel a little awkward here being among physiologists and being a control theorist, but I approached this performance modeling work maybe two years ago and the work that Dan Rapperger presented is essentially the work of one Ph.D. student and a little bit of my time in the summer. This is not a big effort. In our case performance is very well defined. It is very well defined in the subjects doing the task. It was a simulated, "air-to-air" combat task. Performance in any experiment that we derive is not going to be nebulous. These subjects must know precisely what they are to do and it will undoubtedly involve something to do with an airplane. It will involve decision-making, control, tracking, and the like. When I went into this work, I didn't know any physiology and we had fortunately some excellent data that was generated on the human centrifuge (DES). We had some very good control theoretic models that predicted, that is predicted, not replicated; we were able to predict with a set of input parameters that deal with such items as time delays, noise-to-signal

ratios, neuromotor time constants; in other words, the kinds of parameters that usually make sense to people when you talk about them in a generic way. They are for all intents and purposes invariant in the normal condition. They give pretty good predictions on what performance is in that case, as the results we saw with the no-stress case. Those results were essentially no work at all. Now you get something that works and I could lay out hundreds of cases that we have looked at with the optimal control model with the same parameter values that worked. Bolt, Beranek, and Newman has been doing this under the support of Dr. von Gierke on vibration and it has held up very well. Now the question is what happens when you put somebody under stress? Aside from the physiology, there is performance change. This is actually what the Laboratory wants to know. How does the performance change? Not how much blood is changing in the aorta. What we can do and what we have done is relate how the parameters that go into such an optimal control model change as a function of time. Not necessarily as a function of G effort, it is a function of time needed to match the results. When I have motor noise, it is a randomness in the motor response as a function of time and I say, gee, I notice that correlate has something to do with the stress. What should I correlate it with? Well, I don't know enough about physiology to sit down and correlate it with the PA<sub>02</sub> or heart rate or anything like that so I correlate it with the thing that I have available. G,  $\dot{G}$ , integral G or a few other things like that and I can come up with the moderate results you saw. They are pretty much independent across tasks. There is a vertical plane, pitch dynamics and a lateral, third order dynamics. Very different kind of dynamics. Same exact model, same parameters. Now the question, how can this be used to look at physiology? Physiology is ultimately paying attention and is what you want to get at because you want to say well what if I can change the G suit, what if I change the position? What is going to happen? Well, I can't tell you that because I don't have any of the physiological submodels feeding into what I can give. This is where Dana's work comes in. You take outputs--and I don't mind doing black box kind of work to the extent that we know of--and do progression steps or something like that and say, okay, let's not correlate motor noise function of time with G, correlate it with something that is a little more inherent as the output of a physiological model; PA<sub>02</sub> or whatever.

I am working the problem from the top down, Dana is working it from the bottom up and we are going to meet. That is really the gist of this. Dana was talking about building more sophisticated models.

Dr. Cohen: A problem with the models is that we can get very bogged down with details, elaborating our models physiologically with more and more assumptions, looking for more and more variables and never really looking for the essence of what it is that the models are trying to do. On the other hand we can be overly simplistic. We can only look, for example, at performance and correlate the performance with the environment and fail to find the underlying variables by which we can ameliorate the environmental effects on performance. The physiology provides us an approach--if we can understand the physiology--by which we can ameliorate the environmental effects on the performance. That's why we want to go to physiology.



An issue that the engineering approach does emphasize that I think is useful is that physiologists tend to look at the steady state. In the performance arena it is the deltas, it is the changes in the state that provide the many conditions that are critical for us to look at. Physiologists are now beginning to look at these. However, that change in emphasis came from a control engineering approach, really, and I think it is very important for us to look at these dynamic changes. Now, there, a model can be very useful because the model can point out the dynamic changes that are of interest and they can direct the physiologist where to look and where you get nice interplay.

The ultimate criterion for a model is the percentage of variance in the dependent variable that you are interested in that the model can explain. I think that criterion is good from the physiological point of view, from the behavioral point of view or any point of view. The trouble is that we can account for an awful lot of variance under one set of conditions, but if we change the conditions the model can be useless because we didn't have adequate parameters in the model to look at what is really going on. This plumbing model that has been presented as the cardiovascular system, with rigid non-collapsible tubes and everything coming up and down very nicely, does not represent reality. If the cardiovascular system were totally non-compressible, totally non-distensible, I could go for it. But the cardiovascular system is not totally non-compressible, non-distensible, non-distortable and therefore I don't think the cardiovascular response will follow many of these plumbing models, and that's why you (Dr. Green) brought out what you did. But you can get too complicated and get bogged down in details. We can't look for everything. So getting back, the percentage of variance that your models can explain under restrictive conditions you want to enhance; you want to maximize that. Where values are not adequate, then go back to the physiology to try to get explanations so you can improve the model. I think it is an interactive kind of process and I think that Dr. Rodriguez by bringing us all together, all the various disciplines, recognizes that is what is needed to look at this problem.

BREAKFAST SESSION - 17 May 1979

Dr. Rodriguez: In order to summarize our viewpoints, during our session yesterday and having had overnight to think some more about them, I would like to go around the table and invite your comments. As a guide I have written a few questions for you on the blackboard. Your opinions will also help structure a next meeting, if we agree on having one.

We have been basing our objectives on the recommendations of the Witte et al., report, which we took as point of departure for our discussion yesterday. In your opinion, are those recommendations still valid today? And, if so, are we "tracking" them well? If not, what adjustments would you recommend? Is this kind of meeting worthwhile in advancing your understanding of the issues you consider important?

Dr. Green: Are the objectives of the Witte report valid? I would say yes. Are you tracking well? I would say no. What adjustments need

to be made? I would say follow the recommendations of that report. Was this meeting worthwhile? I would say extremely so. Any time you can get a multi-disciplinary group to sit down and talk each other's language, I think it is a worthwhile situation. I would be happy to meet again and when and where as soon as possible.

Dr. Repperger: With respect to the question of objectives, I see two sets of objectives, alternatives, viewpoints, in doing this modeling. One is Dr. Kleinman's where we start from the stress to the performance and we find that the model fits. Then we ask questions why the model is not fitting every stress condition? The second method is the one way Dana Rogers has proposed where he has some knowledge on the physiology, he has some idea of what would go on in a system and he may hypothesize as certain physiological occurrences occur. Then he tries to fit that particular model to the data. So I see two points of view and like Dave mentioned yesterday, there can be a coming together. The insight from one could give information to the other. As far as the meeting being worthwhile, I learned some things while Professor Green was talking the other day. I thought he really got off the track. Then I discussed it with some other people and found out that what he has discussed was important and worthwhile, going far beyond anything I, as an engineer, had envisioned in the problem. I learned from that. I think if we meet again perhaps we could let each person have a limited amount of time like twenty or thirty minutes and present a different point of view and then maybe have an argument and pick a common goal and let each person present a different point of view on how they think they can solve it and then get together and discuss.

Dr. Rogers: First, looking at the questions about the objectives and are we tracking well. My answer to that is, moderately well. A different set of objectives which are perhaps further out than where we are right now were discussed at length in our recent visit to the Naval Air Development Center (NADC). One area that was discussed with personnel at the NADC was the integration of the man in the aircraft. Ultimately where we are headed reaches a point where sophistication or understanding of what is going to happen to that man's physiology in that flight regime and in his combat role is greatly extended. We could see a computer augmentation of the pilot; a computer which is not only being observed and controlled by the pilot to fly his plane but a computer which is also monitoring his physiologic parameters. In effect being able to generate flight profiles beyond which perhaps he can control himself, in terms of escape, in terms of restraint and in terms of biologic recovery. If the man is going to fly in a regime beyond his acceleration tolerance and beyond this energetic tolerance, then the computer may fly the profile and then turn the control back to the pilot after he has a recovery period. I think in the future there are going to be two kinds of aircraft systems. There will be the man who has a computer augmented symbiotic relationship with his aircraft and we will label him the survivor. The other pilot who is still flying by the seat of his pants and pulling G as hard as possible is going to be the loser. I think if we don't continue in this direction with information coming from the control engineers, the physiologist feeding information from the other side and, someone in the middle to integrate the two, we are going to lose.

Dr. Erickson: I think Dana Rogers has expressed some of my sentiments regarding the objectives. I would like to see a practical useful model that can be applied. Modeling, I think, is sometimes done just to model a system and it is very engineering and mathematically oriented and we don't see the results of it. So I think Dana has expressed my feelings here. I think we can improve our tracking. I think there is a lot of information that is available regarding G suits, G valves and benefits of straining maneuvers that could be incorporated into the work that Dana is doing and incorporate it into a useful model. I think the meeting certainly was worthwhile and we should try to meet again. I think another opportunity might be when the Air Force Office of Scientific Research (AFOSR) has their annual review. A group like this could get together. They usually have small working groups at that meeting. So that might be an early opportunity and then again at the Aerospace Meeting, next year.

Dr. Kleinman: A few comments on the objectives. You are talking about the objectives of that Witte report and in a way that was a sort of a rabbit pulled out of a hat and I was not under the impression that that was a point of departure with this meeting. I have only glanced at that report and I have some reservations about it and feel at this point it is to premature for me to say if the objectives are valid. Certainly some of the technical stuff, I feel, is not. But that is something that I think that I will lay aside. As far as, are we sort of heading in the right direction, I think if we head in a balanced direction we will head in the correct direction. What I mean by balanced direction is to not over-emphasize or put more effort in terms of manhours or whatever into the control performance models versus the physiological type of models. I think you want to balance the kind of work that goes into them because the ultimate modeling work and understanding you want to do is going to have to have both of those pieces there. I don't think any one of those two approaches, although it is possible to follow, will wind up as a product that is of any use to the Air Force. It may be very nice for an academic purpose but I don't think you are going to be able to use it without understanding the effects of different G suits, seat positions, and all that is added to it. On the other hand the physiological models have no way of understanding or bringing into it how the human controls the airplane. So I would like to make a plea for a balanced program in this and not stress one versus the other. Although this is saying these are two different approaches, they are not. They approach the same problem from opposite ends.

I came to this meeting with somewhat of a hesitation, I wasn't too sure at first. I am glad I came. It opened up some vision in me in areas that I had not really been into as an engineer. Not just in our discussions here but in some of the sessions. I think I have a better feeling for how to tailor and approach our own research and future research in this problem and also better understanding of what precisely the problems are and what the physiologists can contribute. The kind of meeting, as I mentioned last night to Jim, might be worthwhile along the lines of workshop sessions with very specific goals of two or three individual subgroups and then meeting in a common or combined task either the next day or afternoon getting together on a final working paper. That could possibly be a vehicle on that.

Dr. Rodriguez: Let me clarify something in terms of the objectives and the Witte report which was taken as a point of departure. The statements that I showed yesterday on the screen were from the report. That was the point of departure. That's the objective. That is our first problem to solve. The authors stated that effective countermeasures to enhance performance require a predictive model relating G forces to cardiovascular responses, oxygen supply, etc., and concomitant changes in performance. So I saw the Witte report as an excellent point around which we should orient our discussion because those were the objectives then, and I should submit, unless I hear differently, I think they still are.

Dr. Kleinman: I wasn't saying anything about that. I look at that report, not in terms of its Preface and Conclusions, which I can't argue with, I certainly think they are absolutely correct, but in terms of the center body where technical approach is put down in terms of details--analytic and technical modeling--that part of the report is where I am not ready to follow.

D. Ehrlich: We have had controversy, disagreement and so on but this is the first step of being able to collaborate. As much as I disagreed with Dr. Kleinman yesterday I am in much agreement of what he has just now stated.

If we meet again, we should focus on one, two, or three issues, so that there can be some real substantial discussion in depth.

Dr. von Gierke: I think we all agree that these things have to be done, should be done, that you have to attack the problem from all sides, but there is definitely a difference in the problems one can solve for twenty years from now and the problems we want to solve, have to solve, in the next two or three years and that somehow will determine our program and will be clear in the way we attack these things. Some of the problems alluded to, I think, are extremely important, but they are more in the basic research area. They will pay off probably after another five or ten years. Other problems, particularly with respect to integrating the man into the kind of fighter aircraft we envision in the next ten years, those we have to answer much sooner and for those, I think, as I said yesterday, we are in a position to take certain components, subsystems out of the black box and study them in more detail and give some specific answers we need. These are subsystems particularly the system for positioning, pressure suits, G suits and pressure breathing. I think they are extremely important because there are some opportunities to optimize the system in relatively short runs. Second, I think, and that may be my main disagreement with the Witte et al. report, that it leaves out all the proprioceptive input which is one of the major inputs into the manual control capability and similarly it leaves out, to a large extent, the vestibular system that we know a lot about and it does not indicate it. So coming back to the Witte report I think it was a good piece of work at the time it showed us or could have shown us where to go in a specific area, but I don't think that it addressed the whole problem. I think we know more today, quite a bit more, at this time. We know more in the acceleration physiology and G suit area, so I think we are quite a step around on what the report

recommended at that time. Another area is the performance end of Dana's slide and I think we have to see how to indicate short periods of G performance into the overall performance and workload aspects of the whole mission and that is why there are gains. The tracking alone and tracking for a short period is not the whole answer but we have to go to something like the energetic model or some kind of larger workload model to be able to model the whole mission and the whole mission stress. So I think it was a very fruitful and successful discussion and I am sure we should have and will have more discussions in the future. One of the best places to do it probably would be at the Laboratory at Wright-Patterson. We have the actual data and can go into the nitty-gritty of some of the problems.

Dr. Gillingham: Everybody has been so nice and so tactful, and I wish I had your skills along that line, Dr. von Gierke, but I have to call it the way I see it. As I said yesterday, you have to decide what it is you want. Do you want to study physiology or do you want to study man's performance during G? Now certainly, if you are interested in performance, you have to do experiments. The model is no substitute for experimentation. I think too many of us have been led down the path where the model is functioning before the data have been obtained. If you can just get on the wheel up there at Wright-Patterson and do your performance studies during G, it would give you the insights that you need to relate performance deficits to particular physiologic responses. I see that you have several possibilities. You have the possibility of cognitive breakdown, the possibility of judgmental breakdown, and the possibility of motor system breakdown. Certainly the model that Dana Rogers has put together would give us some insight into where to look for performance deficits once they are discovered. It may be that an elaborate model is not even required. Do the experiments that you need to do in order to get the data relating performance to G, find the defects in the performance, use the model to find the reason for the failure in performance. Now don't get me wrong, I am a physiologist and I am not trying to be hard on the physiologists; but I just think you have to get the performance questions asked first, then go ahead and find out how the physiological experimentation should be done to improve a model that will answer questions regarding performance. I certainly enjoy making mathematical physiologic models; it is a lot of fun, and it will have some use some day. But if you are interested in looking at a pilot's performance under G, there is no substitute for the experiments. You have the capability up there at AMRL to do beautiful experiments, but I don't believe that there has been much done since the days when Dana Rogers was up there and created that graph in which he showed performance decrements versus G at various seatback angles. Correct me if I am wrong about that. But every time we talk about performance at G, that is what we go back to, and I think it is time to improve on that data base.

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