MYOELECTRIC FEEDBACK CONTROL OF ACCELERATION INDUCED VISUAL SCENE DIMMING IN AIRCRAFT TRAINING SIMULATORS

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SUMMARY

Visual dimming experienced during aircraft maneuvering accelerations is an important information source for the simulator pilot. A method for integration of the man with the simulator is demonstrated using computer-based physiologic models and readily measured electromyographic signals arising from the straining maneuver. This technique forces active participation and energy expenditure by the simulator pilot similar to the requirements of the aircraft pilot undergoing acceleration.

Simulation models are developed which relate the effects of the G suit, straining techniques and the cardiovacsular response system to a single protection variable (PV). The PV signal drives a predictive visual field model derived from analysis of the retinal circulation in the eye. Allowances are made for subject variation and cockpit seating configurations.

The model produces accurate predictions of short-term G_z tolerance in its present form and with slight modifications can be adapted to include energetic costs for long-term accelerations.

INTRODUCTION

The aircraft pilot in causing his plane to change its velocity vector places himself in a changing acceleration environment. At certain levels, durations and directions of acceleration the visual field of the pilot is diminished, possibly to the point of blackout. At greater levels and durations unconsciousness may occur. The greatest level at which the pilot still has vision is referred to as his acceleration tolerance. In flight, he has both protective measures and devices which improve this acceleration tolerance. Straining and grunting during tight turns were practiced by German pilots prior to World War II as a means of improving tolerance. This practice led to the development of the M-1 maneuver. Other techniques using posture changes, restraint systems, and body position have been well explored. Present G protective techniques (principally the G suit) attempt to prevent the consequences of the peripheral pooling of the blood during acceleration by the application of a suitable counter-pressure.

The simulator pilot does not have to contend with the actual acceleration forces in his ground maneuvers. The G suit and the G seat provide a tactile feel of the acceleration, however the internal visual field of the simulator pilot is not impaired nor does he need to strain. The muscular straining requirements can be included in the simulation scenario so that the pilot experiences a more realistic training situation in the simulator. Thus, it is possible to create an accurate representation of visual field dimming and fatigue onset for the simulator pilot. The G effects can be simulated realistically by integrating the man with the simulator system. This integration can be made by a combination computer-based dynamic physiologic models and easily instrumented myoelectric feedback.

PHYSIOLOGIC FACTORS OF ACCELERATION TOLERANCE

The physiologic changes caused by acceleration have been reported on extensively in research literature. The two widely recognized factors which contribute to pilot impairment in maneuvering aircraft are fatigue and visual dimming. These two factors can be included in a simulation model system to enhance the reality of simulated air combat.

The cardiovascular system has received the greatest attention in acceleration stress studies as it provides an easily measured set of symptoms. The limiting factor in the cardiovascular system from the viewpoint of combat maneuvering stress is the reduction of blood pressure at eye level and subsequent loss of vision. The pressure gradient changes caused by the G loading and blood pooling are, of course, the major contributing factors which reduce the pressure available at the eye and in more severe cases at the brain. Counteracting the reduced pressure is the primary purpose of straining maneuvers, the M-1 and L-1. Blood pooling in the lower body is reduced by use of the G suit. The factors then, which define an operating limit in the cardiovascular system, are related directly to the blood pressure response characteristics in the 3-15 second time frame.

Another area of interest in the physiologic response to G stress is the time period beyond 15 seconds. Once the pilot has passed a G stress level which represents his relaxed tolerance, he must expend energy in straining to increase blood pressure and, thus, maintain vision. The amount of energy expended in the blood pressure maintenance task is a factor of his straining efficiency and the magnitude of the G difference between his relaxed tolerance and the current G stress level. It can be speculated that the time endurance limit is a factor of the individuals available energy pool minus the energy used in visual maintenance. When the energy pool is diminished to a certain level, the fatigue limit is reached. One purpose of the proposed simulation system is to cause the simulator pilot to be energetically loaded in the same manner as the aircraft pilot.

SYSTEM STRUCTURE

The system presented on the following pages represents the results of partitioning the complex physiologic system into linear sub models. Each sub model is explored and developed in detail based on a common protection variable PV which is related physiologically to system blood pressure. The blood pressure models are then finally combined in a systematic paradigm which provides the driving values for the visual field response model. There are four seperable models which result from the partitioning process; the cardiovascular model, the straining model, the G suit model, and the visual field model.

The cardiovascular response model is represented by a dynamic linear transfer function which corresponds with the response of the pilots blood pressure to acceleration. The governing factor in pilot response to acceleration is the onset of greyout and blackout. These visual problems are directly related to the available blood pressure at eye level. This model output provides a dynamically responding signal which is equivalent to nominal eye level blood pressure values for a human undergoing the equivalent acceleration, G(t), profile.

The straining simulation model accounts for the G tolerance enhancement which is afforded by a properly executed M-1 maneuver. The purpose of the straining or M-1 maneuver is to increase the blood pressure delivered to the eye. Proper performance of the maneuver requires that the abdominal and upper torso muscles be tensed isometrically and that expirations should be made against a closed glottis. The result is an increased intrathoracic pressure and increased blood pressure at the eye. Proper application of the straining maneuver results in the appearance of myoelectric signals on the skin surface. These biologically derived signals are processed by the model to generate a straining protection variable PVs which represents the increased blood pressure due to the M-1.

The pressurized G suit is an important protective garment used to increase the individuals tolerance to $+G_z$. The suit uses pressurized bladders to press against the legs and lower abdomen. The external pressure inhibits displacement of the blood volume to the lower extremities thus insuring a better supply to the heart during acceleration. The suit must be inflated by the G valve to a predetermined level to be effective. The simulation model accounts for the required pressure level and uses the actual suit to provide the necessary dynamics. The suit pressure is compared with the required schedule and a protection value is generated by the model.

The dynamic visual field model is developed to be readily implemented in a

simulation system. The visual field model reacts to PV level inputs from an external source and produces a dynamically responsive signal which predicts the expected visual field of a pilot undergoing the identical G profile.

The required dynamic model of pilot visual response to G_z assembled as a superposition of the separate models. The completed system algorithms provide the means for implementation in aircraft training simulators. The integrated system is structured according to Figure 1.

Pilot commands (1) thru the simulator aircraft dynamics cause changes in the aircraft velocity vector. Vector changes which result in a linear component of $+G_z(t)$ are used to drive the cardiovascular model (3). In addition the $G_z(t)$ signal causes the pilots G suit (4) to inflate according to a preselected schedule. The effect of the proper suit inflation is to increase the value of the protective variable within certain limits. The sum of these signals (5) is added with a random signal related to individual variation. The resulting PV signal drives the visual field model. The instrument panel and the view screen are then dimmed according to the output of the model (6). The pilot senses the size and brightness changes and has the option of reducing his aircraft maneuver intensity or increasing his G tolerance level by performing an M-1 maneuver. If he initiates the M-1 maneuver, the straining model processes the myoelectric activity from his skin surface and modifies it with a signal related to the straining interval (7). If he performs the appropriate action, the model responds with an additive value of PV (8) and his visual scene is enhanced. The separate models are described in the following sections.

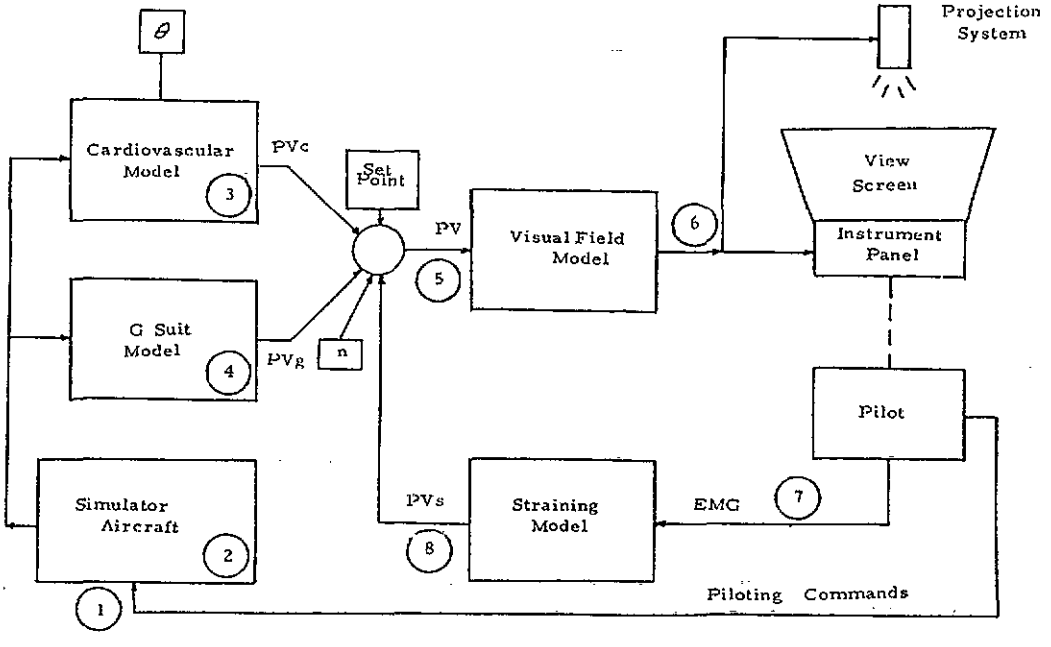

Figure 1. System Diagram

THE CARDIOVASCULAR MODEL

Human tolerance to long-term $+G_z$ acceleration is normally measured in terms of visual loss (blackout) and unconsciousness. Both of these tolerance end points are related to the ability of the cardiovascular system to deliver oxygenated blood at adequate pressure to the retinal and cerebral regions. The distribution of the blood in the body also changes as the acceleration pools blood in the lower parts of the body and lungs. There is, therefore, less available blood to circulate and a lower oxygen content because the lungs do not operate as efficiently.

There are two cardiovascular systems which are dynamically involved in the process of blood pressure maintenance while the human is undergoing $+G_z$ acceleration. The hydrostatic system which is related to classical fluid mechanics is responsible for the reduced retinal perfusion pressure at the eye and eventual loss of pressure at the cerebral level. The orthostatic system is related to blood pooling in the lower body with a concomitant reduction to venous return to the heart. The cardiovascular system has self-regulatory feedback systems which are affected by blood volume and pressure. The feedback mechanisms attempt to regulate the pressure and flow characteristics of the cardiovascular system.

Both animal analogs (principally canine) and human experimentation have led to the current knowledge about the blood pressure response to $+G_z$. Canine blood pressure response curves have been derived by Knapp with maximum gain in the 30 to 60 mHz range. Koushanpour et al. show a first order transfer function with a 20 second time constant. Levison has proposed a second order system for the canine carotid reflex with a natural frequency of 42 mHz. Examination of Gillinghams data as a Bode plot indicates that a single zero double pole transfer function can be applied with reasonable results. This study has adopted a like function to represent the Pressure-G_z acceleration (P-G) transfer function. The frequency range of interest is restricted to f 200 mHz as the physiologic responses of interest to long-term maneuvering accelerations falls within this range. The generalized transfer function is:

$$P(s) = \frac{K_1(1+a_1 s)}{1 + b_1 s + b_2 s^2}$$

Values are selected for a_1, b_1 and b_2 as compromise values from the literature and Gillinghams response curves. The system lead term is selected at f_z = 30 mHz and the system response is selected as a complex pole at fp = 70 mHz and $\xi = 0.7$. For these breakpoints the values of $a_1 = 5.31$, $b_1 = 3.23$ and $b_2 = 5.17$.

The blood pressure transfer function gain at eye level is directly affected by the seat back angle, the direction of the local vertical G axis and the anatomic offset of the eye. The static pressure at eye level is calculated from

$$Pae = Pa - .77\ he \cos\theta\ G$$

where Pae and Pa represent pressure at the eye and heart level respectively, and the effect of the seat angle offset is implemented by a modification of the cardiovascular transfer function gain $K_1(\theta)$. The transfer function gain is then given as

$$\frac{Pae - Pa}{G} = K_1(\theta) = -.77\ he \cos\theta.$$

The offset angles are shown in Figure 2 where the seat back is defined as coincident with the physiologic z axis P_z.

For normal operation the transfer function from G to blood pressure takes the form

$$P(s) = -21.4 \cos\theta \frac{1 + 5.31s}{1 + 3.23s + 5.17s^2}.$$

THE G SUIT MODEL

The history of G suits dates from World War II and is well reviewed in recent monographs. The wraparound CSU-3/P cutaway-type of anti-G suit, presently used by the U.S. Air Force, improves blackout tolerance in the $+G_z$ vector by about 2 G above resting tolerance.

The relationship between the G suit pressurization and effective blood pressure

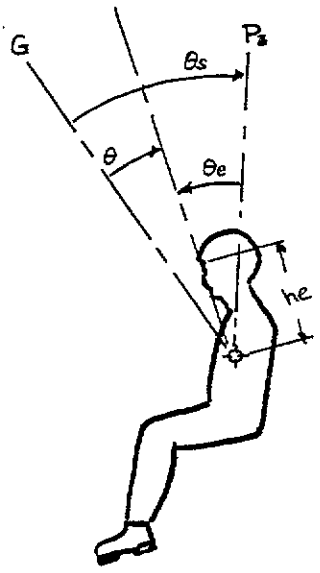

Figure 2. Physiological Axes with Anatomical Offsets

θs - seat angle θe - retinal angle
Gz - local gravity Pz - Physiologic z axis
he - eye height above aorta θ - G axis offset

increase at eye level is highly complex. The physiological mechanism of action of anti-G suits was originally established on the Toronto centrifuge and at the Mayo Clinic and has been summarized by Wood and Lambert. They show that inflation of a G suit at 1G produces an initial increase in arterial pressure, followed by an almost immediate decrease in heart rate probably due to a depressor reflex originating in the carotid sinus and aortic areas.

McCally has shown that the G suit does not provide protection unless the suit is inflated to at least 80 mmHg. In effect, the pressure difference between the suit bladder and the hydrostatic blood pressure in lower body must be such that the suit pressure is greater to effect a protection function. The same reasoning is used in developing the simulation model. The suit pressure must be within acceptable pressure tolerance or it will afford no protection value.

The protective G suit garment contains air tight bladders which are filled with pressurized air delivered from a G sensitive mechanical valve. The pressure delivered by the valve is a function of the current G level.

G suit inflation does begin when the valve has reached a level of 1.5 - 1.7 G. After this point the pressure output is a linear function of G with a value of approximately 75 mmHg per G. The air bladders, the suit air feed hose and the containing garment represent a dead space which introduces a time delay into the pressure system.

The blood pressure G suit model assumes a nominal 2G or equivalent 42.8 mmHg increase for a properly inflated suit. For a suit with lower pressure than designated the increased G protection and equivalent blood pressure value decay linearly to 0. The model is driven by a delta P (ΔP) representing the pressure difference between the standard suit pressure curve and the actual suit pressure. When suit pressure is equal to or greater than the standard curve value, full protection is assumed. When the suit pressure is less than required by the standard curve, the protection value is lowered. When the pressure differential is greater than 80 mmHg with the suit pressure below required, there is no protection afforded by the simulation model.

Where ΔP = Suit Pressure - Standard Pressure

For $\Delta P \leq -80$ Protection Value 0

For $-80 < \Delta P < 0$ $PV = \frac{42.8}{80} P = 42.8$

For $0 \leq \Delta P$ $PV = 42.8$

The G suit dynamic response characteristics are included in the simulation automatically as the G suit pressurization for the simulator pilot provides the driving signal to determine the protection value. Figure 3 is a block diagram representation of the G suit model.

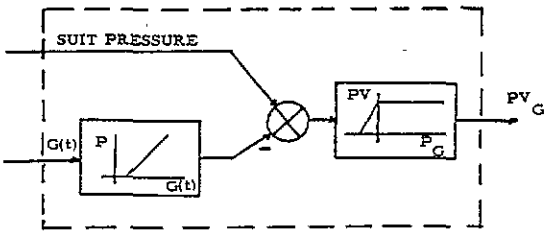

Figure 3. G Suit Model

THE STRAINING MODEL

Since the early days of flying, the value of muscular straining as protection against the effects of $+G_z$ acceleration have been recognized. Pilots observed that blackout or greyout could be postponed if they grunted or screamed and tensed the skeletal musculature during acceleration. The M-1 maneuver, defined as muscular straining with expiration against a partially closed glottis, is a most effective voluntary protection against the circulatory effects of $+G_z$ acceleration. The physiologic basis for this protection has been ascribed to its effect on increasing the arterial pressure at eye during acceleration. As forced expiration is instituted, the resulting increase in intrathoracic pressure is transmitted directly to the aorta and a like increase is felt at eye level.

Skeletal muscle is controlled by signals which are transmitted to selected motor units of a muscle through the motor neurons. The force generated by the muscle is the result of both the frequency of firing of motor units and the number of motor units which are recruited. The muscle tension is accompanied by electrical signals which can be detected by suitable electrodes on the skin surface. The electrical signal exhibits the characteristics of its primary source in that it represents a weighted sum of motor unit activations. There is, therefore, a correspondence between the EMG and muscle force.

The muscle straining maneuvers such as the M-1 and L-1 require a general tensing of skeletal muscle along with contraction of abdominal and peripheral muscles. The muscle straining is accompanied by the expiration of air through a partially closed glottis for the M-1. Both can cause increased intrathoracic pressure of 50 to 100 mmHg and consequently increase arterial blood pressure by a like amount.

This simulator model requires the presence of two signals to provide the maximum straining protection value, PVs. The signals represent the prime factors which are present in a properly executed straining maneuver. The EMG signal is generated by the straining subject and processed to provide and intermediate protection value PVm. The protection afforded by muscle straining is then modified by the timing of the straining maneuver.

The thoracic cavity is modelled as a flexible cylinder surrounded by a muscle girdle. The muscle straining signal representing muscle force around the thoracic volume is, therefore, assumed to bear a predictable relation to the internal pressure increase at low straining levels. At higher levels a maximum pressure is achieved and increases in muscles straining are no longer effective. The pressure straining relationship used in the model is a curve with a saturation level related to the maximum pressure rise in the thoracic cavity.

The effectiveness of the straining maneuver is also governed by the repetition rate. The straining protection value $K_2(t_r)$ is, therefore, modified by a function dependent upon maneuver repetition rate. Experimentally, $K_2(t_r)$ is maximum at 3-5 seconds according to Gillingham. Shorter time periods do not allow sufficient time for the internal pressure to rise and longer time periods involve countering responses due to baroreceptor feedback and reduced venous return to the heart. The generalized straining simulation block diagram is shown in Figure 4.

VISUAL LIMITS MODEL

The effect that acceleration has on the visual apparatus is observed in terms of tunnel vision, greyout, and blackout. During the periods when vision is impaired there are also decreases in visual activity; and

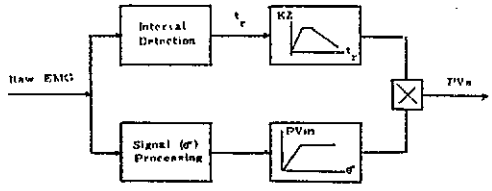

Figure 4. Straining Model

brightness contract detection ability. Although there are multiple factors related to the anatomy, psychology and physiology of the human which are responsible for these changes in visual perception, the structure of the eye is a primary factor and provides the basis for a useable model.

Consider the monocular field with the visual center located at the fovea, and the arterial supply offset and entering the retinal surface through the optic disk. The arterio lar branches which perfuse the retina spread out over the retina terminating in the capillaries, Figure 5. The major arterial

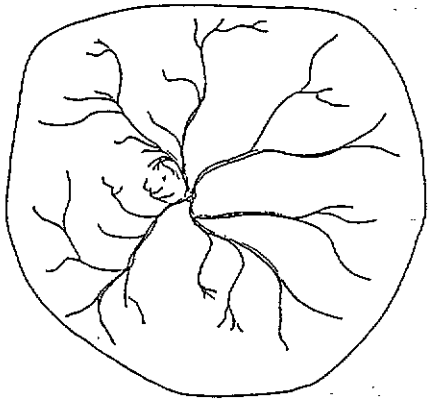

Figure 5. Retinal Artery Supply on Right Eye Field

branches course across the retina to form a general network which normally provides an adequate 0_2 blood supply to the retina. The arterial supply is formed so that a nominal pressure drop is encountered as the distance from the optic nerve entrance is increased, Figure 6.

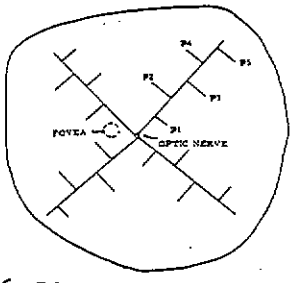

Figure 6. Linearized Representation of Blood to Retina

When the pressure drops below a critical value, the more distal areas begin to feel the effects of blood and oxygen depletion. When the pressure at the retinal artery drops below the interocular pressure blood flow ceases and the retina is no longer capable of transmitting neural signals due to light detection in the rods and cones.

As an initial approximation the retinal supply network is assumed to be a linear network with the supply pressure distributed linearly across the network. That is at any point along the supply

$$\frac{dP}{dx} = K_i$$

Where dx is the distance along the arteriolar bed dP is the corresponding pressure change along dx and K_i is a function of the central supply. At some critical pressure blood flow ceases with the peripheral areas experiencing the initial blood flow shut off. The supply of blood and oxygen then decays inwardly toward the supply point and the visual sensitivity of the system decays in a like manner.

The visual field model can now be extended to a binocular field. Figure 7 is a binocular field map showing the coincident foveal areas as the visual center, and the optic disc for each eye in a manner that depicts the observed field from inside out. The outer lines depict the outer edges of the peripheral vision. Lowered blood pressure supply in each of the modeled retinas causes the field to collapse as concentric circles

with centers at each of the optic discs as shown by the dashed lines. Thus the visual field collapses toward a somewhat ellipsoidal shape with the vertical field having a smaller visual angle than the horizontal field.

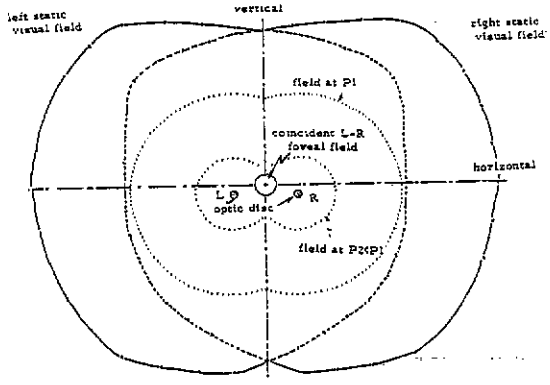

Figure 7. Binocular Visual Field Decay Due to Reduced Blood Supply Pressure

CONCLUSIONS AND RECOMMENDATIONS

The models presented in this study are designed for implementation in real-time simulation. As a consequence many simplifying assumptions have been made. The models do not represent the final stage of myoelectric integration but do provide a logical structure from which refinement can advance. There are gaps in the information available which must be filled to further refine the models and increase simulation fidelity.

Reevaluation of the visual model indicates that a dynamic model of the blood distribution system across the retina may be very valuable. A model analog can be seen in transmission line theory and could provide a single model for both peripheral and central visual fields.

A promising extension of the visual model would include a dynamic representation of oxygen transport at the synapse junctions in accordance with Miller and Green. Reduction of oxygen concentration levels due to impaired pulmonary function or exercise would play an important role in this extended model. The expected outcome would yield zonal gradients of contrast and acuity limits as a function of circulatory distance from the retinal supply. In the extended model both the available oxygen in the blood and the blood pressure would be important factors in defining the visual field response.

The extended model could provide definition of simulator display requirements by establishing maximum acuity and minimum brightness requirements for optimal scene projection fidelity based on visual capability. The extended model would also allow accurate manipulation of the generated scene in terms of both acuity, contrast and relative brightness.

Considerable benefit would result from an experimental procedure to determine the dynamic relationship between EMG and elevated blood pressure.

1) Experimental verification of the derived transfer function would provide the basis for refinement of the postulated model.

2) An evaluation of the energetic costs of the straining protective maneuvers could be derived from such a study and when incorporated into the current simulation model would provide a new method for evaluation of acceleration protection equipment.

Further exploration of electrode placement should include the possibility of using seat pan electrodes (recommended by K. Gillingham). This method would obviate the need for separate electrode preparation for each subject and provide the maximum in environmental fidelity. The simplifying assumptions used in linearizing the complex physiologic systems are both scientifically and pragmatically motivated. The reduction of assumption to fact for these models will require a wide range of experimental efforts. However, the system points in the new directions, provides the rationale and establishes the feasibility of biologic integration in training systems.

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