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A MODEL OF CEREBRAL BLOOD FLOW DURING SUSTAINED ACCELERATION

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ABSTRACT

Rationale: Radial accelerations generated in modern combat aircraft maneuvers (Gz) may result in impaired vision or loss of consciousness (G-LOC). We are interested in developing mathematical models of cerebral blood flow during exposure to Gz. Our previous model [1] showed that intracranial vascular resistance does not change with Gz since the vessels are protected from collapse by the cerebrospinal fluid and that reduction of the blood flow to the brain is mainly due to the increased vascular resistance of the large extracranial veins.

Methods: Based on the previous results, we propose a model with simplified presentation of the arteries and intracranial vessels and a more detailed description of the jugular veins. The extracranial arteries are accounted for by the hydrostatic pressure drop from the heart to the head level. The intracranial vessels are represented by a resistance independent of the mechanical effects of Gz. However, a model of cerebral autoregulation is incorporated, which involves active change in the cranial vascular resistance in reaction to the change in blood pressure at the head level. The jugular veins are modeled using one dimensional equations of fluid dynamics and a non-linear relation between the transmural (blood minus external) pressure and the local vessel cross-sectional area. The central arterial and venous pressures are taken to be 105 $mmHg$ and 5 $mmHg$ respectively and Gz was varied from -5 to +10. To simulate the effects of positive pressure breathing, blood pressures at the arterial and venous ends of the model were elevated by the same amount, so that the perfusion pressure was always maintained at 100 $mmHg$.

Results and conclusions: The model is successful in reproducing the drop in cerebral blood flow with +Gz. This reinforces our belief that the elevated venous resistance plays a significant role in G-LOC. The autoregulation has a positive impact at moderate +Gz but is ineffective at higher +Gz. This is mainly due to the fact that the venous resistance becomes absolutely dominant at high +Gz and a further decrease in the cranial vascular resistance makes little difference. The model predicts an increase in the blood flow in the case when the central venous and arterial pressures are elevated. We attribute this to the fact that an elevated central venous pressure prevents the venous collapse and maintains the extracranial veins patent.

1 INTRODUCTION

It is known that exposure to $+Gz$ acceleration can cause inadequate perfusion of the retina and brain leading to loss of vision and/or loss of consciousness [2]. When Gz is greater than the normal gravitational acceleration of the earth (+1 Gz), the weight of the blood is increased, and at approximately +5 Gz blood pressure at the level of the head can be expected to be zero. However, in a closed system with no net change in potential energy, such as the circulatory system, an increased hydrostatic gradient is not sufficient to explain the decrease in cerebral blood flow. If the blood vessels were rigid the cerebral perfusion would not be affected by Gz force, since the hydrostatic gradients on the arterial and venous side are equal and of the opposite sign, and the vascular resistance is independent of Gz stress. Blood vessels, however, have elastic walls and their cross-sectional area is a function of the transmural (internal minus external) pressure. In particular, thin-walled compliant vessels, such as veins, have tendency to collapse when subjected to negative transmural pressure. Therefore, in the case when vessels are compliant the vascular resistance is affected by the hydrostatic gradient, central arterial and venous pressures, and the external pressure acting on the vessels.

We have used opened loop mathematical models of the cerebral vascular system to model cerebral perfusion under Gz stress. Figure 1 shows a resistive network of compliant vessels representing the cerebral vascular tree. The extracranial portion of the network is represented by carotid and vertebral arteries and by jugular veins, which extend vertically, from the heart level to the cranium. The intracranial vessels are enclosed in a rigid container representing skull and surrounded by the cerebrospinal fluid (CSF). The results from the network suggest that the drop in cerebral blood flow dur-

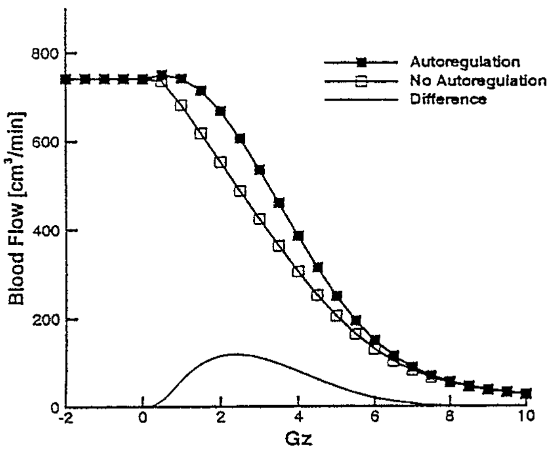

Figure 6: Cerebral Blood flow predicted by the model for Gz ranging from -2 to 10. Solid squares: Model with autoregulation. Hollow squares: Model without autoregulation. Faint line without symbols: Difference.

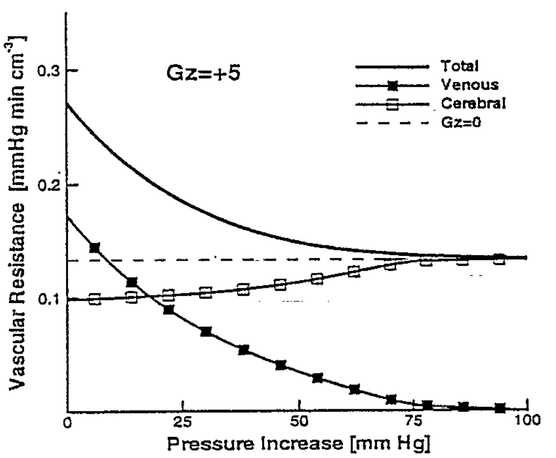

Figure 7: Vascular resistance at $+5$ Gz as a function of blood pressure increase. Thick line without symbols: Total cerebral resistance. Solid squares: Venous resistance. Hollow squares: Cranial vascular resistance. Broken line shows the normal value of cerebral resistance.

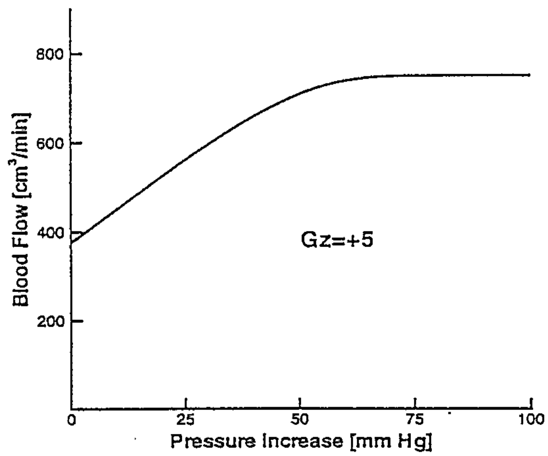

Figure 8: Cerebral blood flow at $+5$ Gz as a function of blood pressure increase.

The effect of the elevated blood pressure is to return vascular resistance to its normal value. Once the normal resistance is restored, further pressure increase has no effect. This is also reflected in the flow curve. Blood flow initialy increases with elevated blood pressure and then it levels at the normal value.

4 DISCUSSION

Vascular resistance and blood flow

This study indicates that even if the normal perfusion pressure is maintained, the increase in venous resistance caused by gravitational stress may lead to inadequate cerebral perfusion. The effect of the Gz-dependent jugular resistance can be discussed in terms of the extent to which pressure recovers as the blood descends towards the heart. If the veins were rigid (siphon), R_V would have been constant and negligible and the blood flow would be

$$Q_{MAX} = \frac{P_{AH} - P_{VH}}{R_C} \tag{6}$$

The other extreme case is the one in which viscous losses in jugular veins exactly equal the hydrostatic pressure component (waterfall). In this case blood pressure is P_{VH} everywhere in jugular veins, and blood flow is given by .

$$Q_{MIN} = \frac{(P_{AH} - P_{VH}) - \rho Gz \Delta H}{R_C} \tag{7}$$

In other words, blood flow is possible only if the perfusion pressure is higher than the hydrostatic pressure of the blood column going from the heart to the head, meaning that it would be zero at approximately +4.5 Gz. The flow rate predicted by the model is somewhere between the "siphon" and the "waterfall", meaning that some pressure recovery is always present as the bloods descend towards the heart.

Elevated blood pressure has positive effect on cerebral blood flow since it reduces the narrowing of the jugular vein. If the pressure increase is sufficiently high, jugular veins cease to be collapsed and the normal blood flow is restored. Further increase in blood pressure has no effect since, at that point, the venous resistance is very low.

Autoregulation significantly improves cerebral perfusion at moderate $+Gz$ but is ineffective at higher $+Gz$. There are two reasons for this. First, at very high $+Gz$ the jugular resistance dominates and a reduction of the cranial vascular resistance has little effect on the blood flow. Second, the autoregulation mechanism itself becomes less effective for very small cerebral perfusion pressures (see the Appendix) which is the case for high $+Gz$.

CSF pressure

Rushmer *et al* [6] measured blood and CSF pressure in cats exposed to positive and negative Gz and concluded that P_{CSF} and venous pressure always stay roughly the same. Our model leads to the same conclusions using the assumption that the cranial blood volume is conserved, meaning that any increase of the arterial blood volume must be matched by an equivalent decrease of the venous blood volume. If P_{CSF} is to produce exactly the same volume change on the arterial and venous side, it should be much closer to venous than to arterial blood pressure, since the venous compliance is much higher than the arterial. Therefore P_{CSF} is only several millimeters of Hg higher than the cranial venous pressure.

5 CONCLUSIONS

The results show that a reduction of the cerebral blood flow during Gz stress may be caused by an increased vascular resistance on the venous side, outside of the skull. Though the extracranial veins are collapsed for $+Gz$, the gravitational effects play a role as the blood descends towards the heart. When the central blood pressures are elevated sufficiently, normal blood flow can be restored even at substantially high $+Gz$. The cerebral autoregulation is effective only at very moderate $+Gz$.

The cerebrospinal fluid pressure is a consequence of the cranial volume conservation and is directly influenced by the central venous pressure and the venous vascular resistance.

6 ACKNOWLEDGMENTS

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Appendix

Steady Inertial Flow in the Jugular Vein

We consider steady 1-D flow in a vertical elastic vessel. The vessel properties and the external pressure are spatially uniform. The 1-D steady state governing equations are:

$$Q = AU \tag{8}$$

$$U\frac{dU}{dx} + \frac{1}{\rho}\frac{dP}{dx} - G_z = -\frac{\mathcal{R}(A)Q}{\rho} \tag{9}$$

$$P_t = K_p\mathcal{P}(\alpha) \tag{10}$$

where

$$P_t = P - P_e \tag{11}$$

$$\alpha = \frac{A}{A_0} \tag{12}$$

The momentum equation (9) can be rewritten using equation (8) and the wave speed c as:

$$\frac{d\alpha}{dx} = \frac{\alpha[\rho G_z - \mathcal{R}(A)Q]}{\rho[c^2 - U^2]} \tag{13}$$

$$c^2 = \frac{A}{\rho}\frac{dP}{dA} \tag{14}$$

Next, we introduce functions for $\mathcal{P}$, c, and $\mathcal{R}$ following [4].

$$\mathcal{P}(\alpha) = \alpha^{20} - \alpha^{-3/2} \tag{15}$$

$$c^2 = \frac{\alpha K_p}{\rho}\frac{d\mathcal{P}}{d\alpha}$$

$$= \frac{K_p}{\rho}(20\alpha^{20} + \frac{3}{2}\alpha^{-3/2}) \tag{16}$$

$$\mathcal{R} = \frac{8\pi\mu A_0^{1/2}}{A^{5/2}} = \frac{8\pi\mu}{A_0^2\alpha^{5/2}} \tag{17}$$

Substituting equations (15-17) into equation (15) and using $U^2 = Q^2/A^2 = Q^2/(A_0^2\alpha^2)$, we have

$$\frac{d\alpha}{dx} = \frac{\rho G_z\alpha - (8\pi\mu/A_0^2)Q\alpha^{-5/2}}{K_p(20\alpha^{20} + 3/2\alpha^{-3/2}) - (\rho Q^2/A_0^2)\alpha^{-2}} \tag{18}$$

This is a first order ordinary differential equation in terms of α. It can be solved if one boundary condition and the flow rate Q are known. Note that the area derivative is infinite for $U = c$. Consequently, a smooth transition to supercritical flow is only possible where the gravitational and dissipation effects cancel. Typically, the transition back to sub-critical flow occurs via an abrupt change in the area. This is analogous to the normal shock in gas dynamics.

Cerebral Autoregulation

The mathematical model of the cerebral autoregulation is taken from [9]. The model is based on the assumption that cranial resistance responds to the changes in cerebral perfusion pressure (cranial arterial minus cranial venous) and is described by the following equations:

$$\frac{dy(t)}{dt} = -\frac{y(t)}{\tau} + \frac{1}{\tau}\left[\frac{P_{AC} - P_{VC}}{(P_{AC} - P_{VC})_n} - 1\right] \tag{19}$$

$$\frac{1}{Rc} = \frac{1}{Rc_n}\left[1 - \frac{1}{\pi}tan^{-1}(y(t)\pi)\right] \tag{20}$$

where τ is a time constant and subscript n denotes normal values (zero G_z).

The solution to equation (19) is

$$y(t) = \left[\frac{P_{AC} - P_{VC}}{(P_{AC} - P_{VC})_n} - 1\right] + K\exp(-\frac{t}{\tau}) \tag{21}$$

where K is a constant of integration. For the steady state

$$\lim_{t\to\infty} y(t) = \frac{P_{AC} - P_{VC}}{(P_{AC} - P_{VC})_n} - 1 \tag{22}$$

Substituting equation (22) into (20) and using $P_{AC} - P_{VC} = RcQ$ we have

$$\frac{1}{Rc} = \frac{1}{Rc_n}\{1 - \frac{1}{\pi}tan^{-1}[(\frac{QRc}{(P_{AC} - P_{VC})_n} - 1)\pi]\} \tag{23}$$

This is a nonlinear algebraic equation in terms of Rc which can be solved if Q is known. The relation between cerebral perfusion pressure and cranial resistance, normalized with respect to their respective normal values, is shown in figure 9.

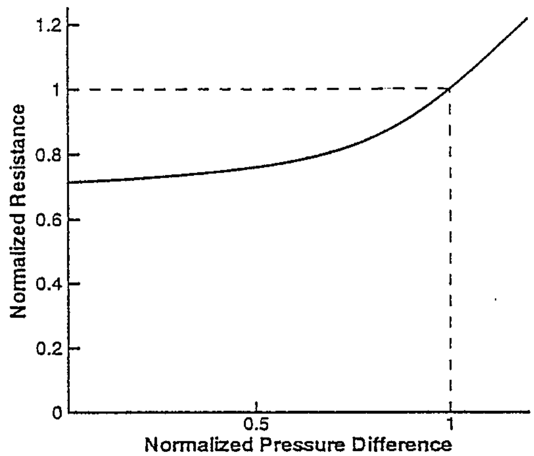

Figure 9: Normalized cranial resistance as a function of normalized cerebral perfusion pressure.

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