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CORRELATION OF EYE-LEVEL BLOOD FLOW VELOCITY AND BLOOD PRESSURE DURING +GZ ACCELERATION

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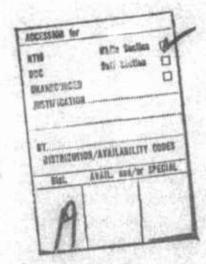
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CORRELATION OF EYE-LEVEL BLOOD FLOW VELOCITY AND

BLOOD PRESSURE DURING +GZ ACCELERATION

INTRODUCTION

The need for a reliable objective means for monitoring $+G_Z$ tolerance has long been recognized by acceleration physiologists. Loss of eye-level arterial pressure has been correlated with cessation of blood flow to the retina (1) and remains the most reliable indication of cardiovascular status during exposure to $+G_Z$ acceleration. This technique, however, has the distinct disadvantage of being invasive, requiring cannulation of a radial artery.

This study was designed to examine the efficacy of a transcutaneous Doppler flowmeter (2) monitoring eye-level (temporal artery) blood flow velocity during $+G_z$ acceleration and to correlate results with direct arterial pressure referenced to eye level.

This noninvasive technique would quantitate man's ability to withstand G without using invasive monitoring techniques, and thus altering his normal response to G stress because of the instrumentation.

METHODS

Seven healthy male volunteer subjects (age range 21 to 25) were studied. All had recently passed a USAF Class II flying physical examination and had extensive centrifuge experience.

Subjects were instrumented with miniature 8 MHz Doppler sensors (2 x 1 x 0.5 cm) secured to the skin above the maximum palpable impulse from both the right and left frontal branches of the temporal afteries to detect blood flow velocity from back-scattered ultrasound. A directional signal processor was used with one sensor while a nondirectional processor was used with the other. With this arrangement, retrograde flow was graphically portrayed. Audio recordings of the unprocessed Doppler-shift were also made. The right radial artery was cannulated, and eyelevel arterial blood pressure was measured using a Statham (P-37) miniature strain gauge transducer mounted at eye level. Mean arterial blood pressure was obtained by electronically damping the arterial pressure wave with appropriate filtering.

EKG was continuously monitored, and audiovisual communication was maintained with the subject at all times.

Both rapid onset runs (ROR, 1 G/sec) and gradual onset runs (GOR, 0.1 G/sec) were used to stress subjects to the point of visual failure on the USAFSAM human centrifuge. RORs were begun at peak 2.5 G for 15 sec and increased in increments of 0.5 G until peripheral light loss (PLL) occurred; at this time, runs were increased by 0.2 G until the endpoint of blackout (50% loss of central light). Subjects then underwent a GOR to blackout. Adequate time was allowed between runs for the subject to return to a normal physiologic state.

RESULTS

When blackout $+G_z$ level (range 2.7 to 4.6 G) was approached during rapid onset runs, eye-level arterial blood pressure began to fall concomitant with the occurrence of retrograde flow in the temporal artery during diastole (Fig. 1). This occurrence of retrograde flow has been verified using both directional and nondirectional Doppler systems (3). It can be easily recognized with audio recordings of the Doppler-shift. Zero forward temporal artery flow (\dot{Q}_{ta}) was determined by both graphic and audio recordings 6 sec prior to blackout (range 4-9 sec). Eye-level mean arterial pressure (\ddot{P}_a) decreased to 20 mm IIg when zero forward \dot{Q}_{ta} was recorded. Arterial pressure and \dot{Q}_{ta} increased simultaneously during centrifuge deceleration with a characteristic increase in arterial pressure and flow occurring postrun when compared to prerun values.

The correlation of mean arterial pressure, temporal artery blood flow, and visual symptoms recorded during rapid onset runs was not duplicated to the same degree during gradual onset runs. Although changes in mean arterial pressure and \hat{Q}_{ta} occurred simultaneously, sustained zero forward flow was recorded in only 2 subjects prior to blackout. In these subjects, the onset of sustained zero forward \hat{Q}_{ta} occurred simultaneously with a decrease in mean arterial pressure to 20 mm Hg and occurred 5 sec prior to blackout (Fig. 2).

In a third subject, mean arterial pressure was sustained at 20 mm Hg and below for 9 sec prior to blackout, and zero net forward flow occurred for the same duration; the nondirectional flowmeter was not functioning. In the four remaining subjects, zero sustained forward flow was never attained and mean arterial pressure was maintained at higher levels; nevertheless, the retrograde flow portion of the \dot{Q}_{ta} wave had become progressively dominant.

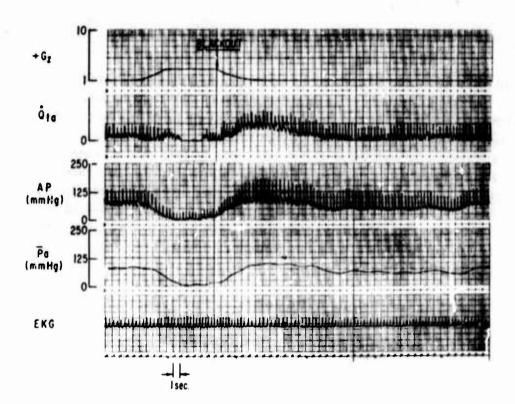


Figure 1. Eye-level arterial pressure and blood flow responses during ROR (1 G/sec).

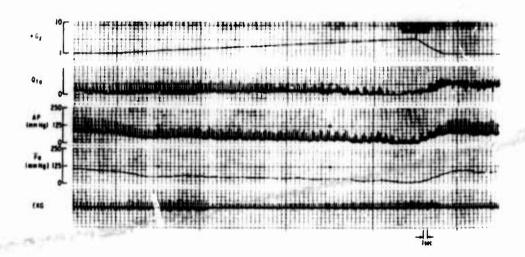


Figure 2. Eye-level arterial pressure and blood flow responses during GOR (0.1 G/sec).

DISCUSSION

Coburn (3), as recently as 1970, voiced his concern regarding the "reliability and repeatability of a large portion of the data reported in the literature." Many of his objections to subjective endpoints have been verified by other acceleration physiologists. An inexperienced centrifuge subject may terminate a run because of fear or a misunderstanding of the desired endpoint. Even an experienced, highly motivated subject will terminate a run prematurely if unduly fatigued from previous runs. It was the purpose of this study to determine if a correlation could be established between the objective measurements of temporal artery blood flow velocity, mean arterial pressure, and subjective blackout in highly trained, experienced centrifuge subjects.

During the rapid onset runs, zero forward flow and a mean arterial pressure of 20 mm Hg occurred 6 sec prior to blackout. It is assumed that at this eye-level arterial pressure, the critical closing pressure of branches of the temporal artery is approached (4).

Duane (5) using direct ophthalmoscopic observations and Leverett and Newsom (1) using retinal photography and fluorescence angiography found that subjective blackout coincides with cessation of flow in the retinal circulation. They also found that visual failure at blackout occurred when head-level arterial pressure had fallen below 20 mm Hg. Our findings using transcutaneous ultrasound suggest that blood flow velocity changes in the retinal circulation are reflected in flow velocity changes in frontal branches of the temporal arteries during rapid onset $+G_Z$ acceleration.

During the gradual onset runs, blackout occurred before a sustained zero forward flow and mean pressure of 20 mm Hg had been reached in 4 of 7 subjects. This could, in part, be attributed to a combination of hypoxic hypoxia, resulting from reduced oxygenation of the blood during these prolonged $+G_z$ exposures (6) and stagnant hypoxia, since the retrograde component of the flow wave gradually increased until blackout was reached.

CONCLUSIONS

The transcutaneous Doppler ultrasonic flowmeter monitoring \dot{Q}_{ta} appears to be a reliable tool for measuring cardiovascular status and predicting visual failure during rapid onset $+G_z$ acceleration. Cardiovascular status as indicated by mean arterial pressure is well correlated with changes in temporal artery blood flow and visual symptoms reported by experienced centrifuge subjects. Cardiovascular status during gradual onset runs also appears to be reflected by simultaneous changes in temporal artery blood flow and mean arterial pressure; however, the accurate prediction of visual failure during these prolonged runs requires further investigation. Work is continuing using this new noninvasive technique to assess cardiovascular status during high, sustained $+G_z$ acceleration and in the evaluation of new $+G_z$ protective devices and techniques.

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