The Physiological Limitations of Performance During Acceleration

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A PILOT'S TOLERANCE to eyeballs-down acceleration can be readily assessed from the g stress levels at which he loses vision and then consciousness. With g stress applied at right angles to the spinal axis (eyeballs in or eyeballs out), however, the cardiovascular impairment is so much less severe that there is no comparably dramatic end point to mark tolerance. A crude evaluation of tolerance to transverse acceleration is possible from the period for which a pilot will endure the g stress in a centrifuge before he voluntarily terminates the run but this is subject to a large number of unmeasurable variables, such as motivation, competitiveness, and experience.

The acceleration stresses encountered by the crew of a space vehicle on reentry will vary widely with the lift-drag ratio of the vehicle and with the pilot's orientation relative to the direction of motion of the vehicle. Numerous centrifuge studies have shown that healthy men can remain conscious under sustained EBI and EBO accelerations of high magnitude [1, 9] but the adequate performance of control tasks during reentry clearly requires more than mere maintenance of consciousness. Therefore, the development and implementation of refined criteria of pilot tolerance to EBO and EBI accelerations are essential not only for the further basic investigation of the physiological effects of acceleration but also for the objective evaluation of personnel, restraint systems, and the level of control performance that can be reasonably expected of men under reentry conditions.

A continuing study conducted by the Ames Research Center is directed to this end. Experienced test pilots have performed sophisticated tracking tasks under acceleration and their performances were evaluated by the techniques described.[1, 2, 8] Concurrent physiological experiments have been conducted in an effort to correlate psychomotor performance with changes in physiological functions. The experiments have been chiefly concerned with three organ systems most acutely affected by acceleration stress, namely visual, circulatory, and respiratory.

A particularly important aspect of this program is that the development of a suitable restraint system [10, 11, 12] has permitted us to study pilots under acceleration in the EBO vector as well as in the EBI vector, the latter having been used for almost all previous experiments in the field. From the very beginning, it was clear there are interesting differences between the effects of EBI and EBO acceleration, and much of our subsequent work comprises a systematic comparison of the changes in various functions during acceleration in these two vectors. For example, early comparative experiments showed that there is a distinct ventilatory advantage to the EBO orientation, and that the majority of pilots found it subjectively more comfortable as well.[9] Later experiments [7] evaluated the ventilatory advantage of EBO acceleration more precisely.

The present report is concerned with our principal findings with respect to visual and cardiovascular functions, and some extensions of our previously reported respiratory data.

METHODS

The subjects in this study were experienced test pilots; some had had previous centrifuge experience and all could be considered expert in the tracking skills required by the performance tests and in their subjective evaluation of the difficulties encountered. Their motivation was uniformly excellent, and in some cases, they were highly competitive. It is appreciated that this is a select population but, nevertheless, one similar to that from which crew members for space flight missions are selected.

Most of the work was carried out in the human centrifuge at the Naval Air Development Center, Aviation Medical Acceleration Laboratory, at Johnsville, Pennsylvania. Some of the respiratory and the cardiovascular studies were conducted in the human centrifuge at the University of Southern California, Los Angeles. In addition to the shorter length of the centrifuge arm at the University of Southern California, there is an additional minor difference in that the subject's seat is fixed and consequently there is always a 1 g EBD component added to whatever stress the centrifuge rotation applies. The angles and the resultant g stresses actually applied to the subject are given in Table I. The gimballing of the Johnsville centrifuge enables the g stress to be applied exactly at right angles to the trunk.

Performance tests were undertaken simultaneously with the physiological studies in most of the runs at

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TABLE I. ACCELERATION LEVELS AND ANGLE APPLIED (∝) FROM TRUE TRANSVERSE DIRECTION (EBO, EBI) DUE TO 1 G EBD

Indicated g	Actual g	∝
1	1	0
4	4.12	14.5°
6	6.08	9.5°

Johnsville but not at Los Angeles. Furthermore, during the visual studies at Johnsville, the pilot's attention to the vision experiment necessarily precluded a concurrent performance task.

VISUAL STUDIES

A reduced scale Snellen chart 36 inches from the eye was used for simple studies of visual acuity. The chart was illuminated from behind to give a light intensity of approximately 30-foot candles. In addition, an astigmatic dial was mounted just below the Snellen chart.

A measurement of possible deformations of the cornea under acceleration was made by continuous photography of the reflection of concentric rings on the cornea. As shown in Figure 1, a Placido disk was mounted in front of the eye so that its rings were reflected by the cornea, and a motion picture camera behind the disk photographed the cornea through a hole in the center of the disk. This technique is described in detail by Morgan and Lester.[6]

Changes in the accommodating power of the eye were investigated by the arrangement shown in Figure 2. A phoropter was modified so that, while under acceleration, the pilot could rotate a series of lenses in front of the eye by means of a string attached to a finger. The pilot then read the Snellen chart, and his ability to accommodate to a series of lenses ranging from 0 to 1.50 diopters was compared with his accommodation at 1 g.

In a different experiment, the right eye orifice of the phoropter was darkened so that the subject could only see with the left eye. Then at peak g, the subject could pull the string which would replace the blacked out lens with a plano lens and permit vision with both eyes.

The pilots were also asked to complete questionnaires concerning their subjective sensations under acceleration, and this information was applied to mark sense IBM cards for analysis. Some of the information requested concerned their vision under acceleration. A sample card is shown in Figure 3.

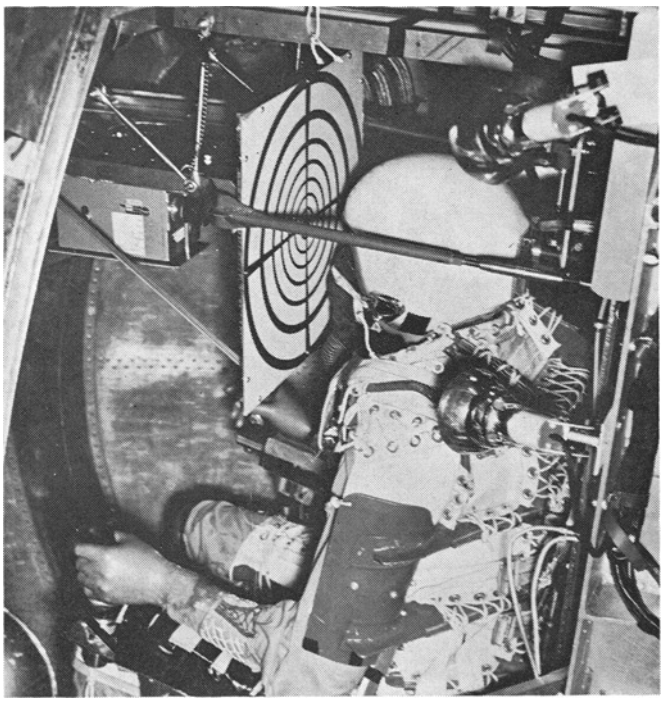

Fig. 1. Placido disk technique for measuring corneal deformation in the NADC centrifuge, Johnsville, Pa.

PHOROPTER INSTALLATION ON CENTRIFUGE

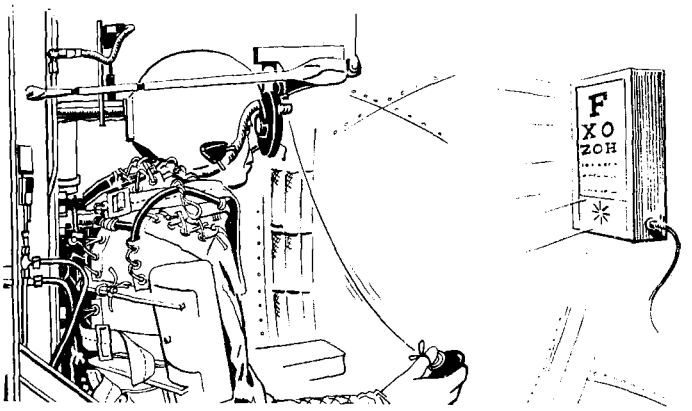

Fig. 2. Phoropter technique for measuring accommodating power of the eye in the NADC centrifuge, Johnsville, Pa.

CARDIOVASCULAR STUDIES

The pilots' blood pressures were measured with the Ames automatic blood pressure device; this has been described in detail by Holden, et al.[4] It comprises the usual arm-cuff, which is automatically inflated with nitrogen at intervals; then the pressure is immediately allowed to bleed down. Through a calibrated pressure transducer, the changing cuff pressure is recorded on one channel of an oscillograph. A small microphone placed over the brachial artery distal to the cuff picks up the sounds which are displayed as "blips" *superimposed* on the pressure tracing. The pressures at which the blips appear and then disappear are taken as the systolic and diastolic pressures, respectively. Since our experiments are concerned with *comparisons* of blood pressures at high accelerations with those at 1 g, the refinements of diastolic pressure measurements are relatively unimportant.

Pulse wave excursions were measured by a photocell device attached to the ear.

The subjects' electrocardiograms were recorded continuously from leads placed in the axilla and precordially.

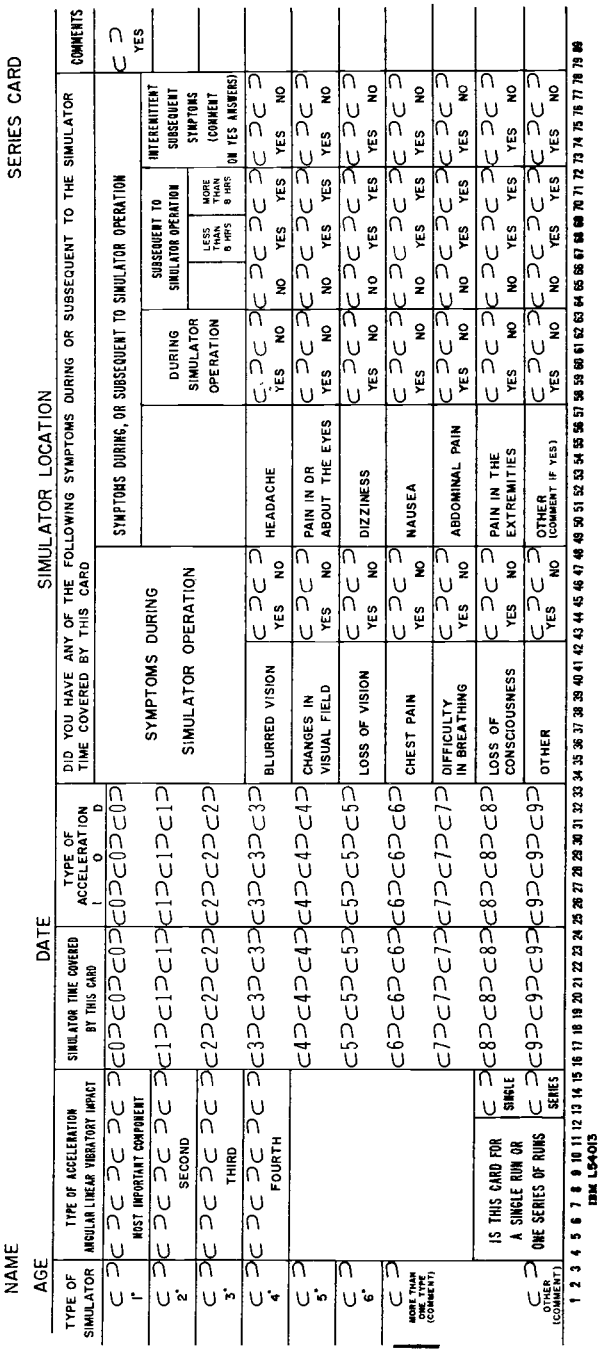

Fig. 3. Ames mark sense record card (series card).

Arterial hemoglobin-oxygen saturation was measured with the Water's ear oximeter operating in the range of 0.64 and 0.80 μ. The earpiece and the subject's ear were immobilized with adhesive tape, and with this small precaution the instrument was surprisingly stable under acceleration.

RESPIRATORY STUDIES

The Ames respiratory monitoring equipment has been described by Holden, et al.,[4] and is illustrated diagrammatically in Figure 4. In all experiments for the measurement of functional residual capacity and oxygen uptake, the facepiece of the pilot's helmet was modified

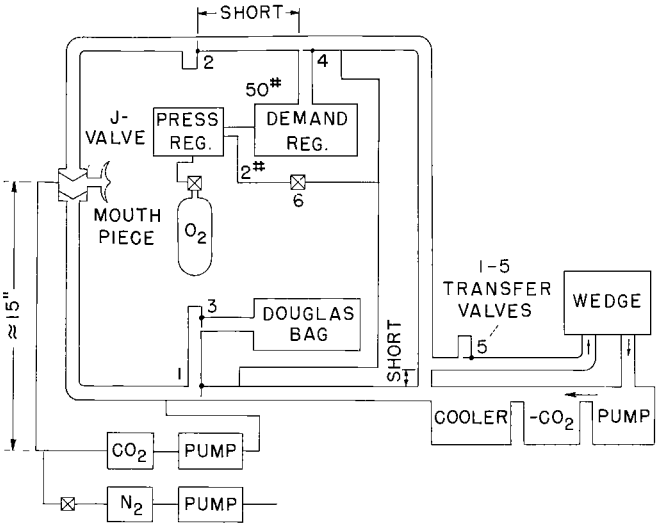

Fig. 4. Ames respiratory system.

so that he breathed through a bite mouthpiece rather than from a face mask. It had been noted that even individually molded face masks tended to leak slightly under acceleration.

The nitrogen content of the expired air (and of the total system at equilibrium) was determined by a nitrogen meter. Remotely controlled valves in the respiratory equipment permitted the instantaneous change from ambient air to closed-circuit breathing from an oxygen-filled wedge spirometer.

The CO_2 content of expired air was determined by a modified Beckman-Spinco LB-1 instrument. In some experiments this was placed about 1 meter distal to the pilot's mask so that it analyzed largely "mixed" expired air. In other experiments, the intake catheter was virtually in the bite mouthpiece, permitting the analysis of "end-expiratory" air.

RESULTS AND DISCUSSION

VISUAL

The concensus of the pilots' subjective impressions is that at accelerations up to 8 g, the main visual decrement is a blurring, and that this is most noticeable under EBO stress. At accelerations above 8 g, in both EBO and EBI, there is also a graying of vision which is similar to that encountered prior to blackout under EBD acceleration. This similarity, and some cardiovascular observations to be described later, suggests that the graying of vision at the higher levels of transverse acceleration results from retinal ischemia. On the other hand, the blurring of vision (which begins to appear between 4 and 6 g) might be reasonably attributed to physical deformation of the eye, concomitant with the general hydraulic displacement of the tissues. The well-supported crystalline lens and the retina are presumably less sensitive to mechanical displacement than the cornea; furthermore, the cornea and the aqueous humor of the anterior chamber account for about 65 per cent of the refraction. Therefore, a series of experiments was carried out to measure the extent

of corneal deformation with the Placido disk as described above and shown in Figure 1.

In a preliminary trial, photographs were taken (at 1 g) of the Placido disk reflection on the cornea of a subject with a pronounced astigamatism (3.5 diopters). Measurement of the intervals between the rings in an enlarged print permitted a characterization of the astigmatism which was in general agreement with that obtained by conventional ophthalmological techniques. Subsequently, motion pictures were taken of the reflections of the corneas of centrifuge subjects at 1, 4, 6, and 8 g, both EBI and EBO. Figure 5 is a photograph of the reflection of the Placido disk at 1 g. Careful examination of these photographs at 4, 6, and 8 g revealed no distortion of the Placido disk reflections. It is very significant, however, that pronounced tearing at 6 and 8 g EBO caused a different and gross distortion as shown in Figure 6.

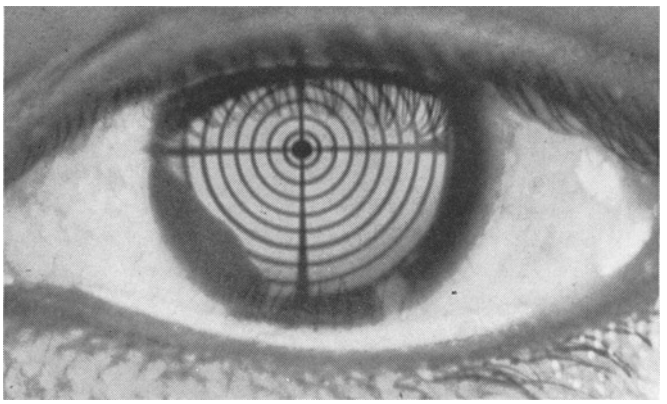

Fig. 5. Placido disk reflection on cornea at 1 g.

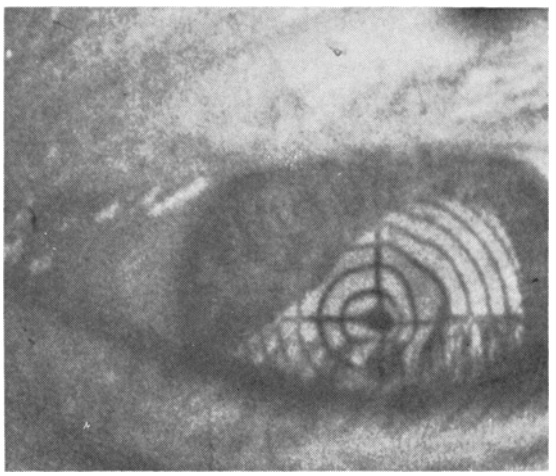

Fig. 6. Placido disk reflection on cornea at 8 g.

In a separate approach to the same problem, it was found that at accelerations up to 8 g, both EBO and EBI, the pilots' abilities to read the 20/15 line on the reduced-scale Snellen chart were virtually unimpaired, except when tearing occurred. Similarly, the pilots' impressions of the radii of the astigmatic chart were unchanged under acceleration except in a single instance.

The modified phoropter described under "Methods" was used to evaluate the ability of the eye to accommodate while under acceleration. The pilot was able to see only with the left eye as acceleration was applied; then, at peak g, by substituting a plano lens for the darkened one in the right eye orifice, he was able to see with both eyes. This invariably resulted in a double image which could not be fused while the acceleration stress persisted. We can only surmise that perhaps the extraocular muscles may be at a mechanical disadvantage during transverse acceleration.

In summary, we find that with the exception of the tendency to diplopia, none of our optical measurements account for the pilots' subjective reports of visual decrement. The pronounced blurring under EBO acceleration, however, can be largely attributed to the accumulation of tears on the cornea. This may be partly due to increased secretion, but the acceleration vector itself would cause tears to flow toward the center of the cornea and to accumulate there.

The blurring of vision (without tearing) under 6 g EBI acceleration may be a preliminary manifestation of the graying of vision at 8 g and above. It has been noted that this blurring worsens to the point of blackout as exposure to EBI acceleration is prolonged, again suggesting retinal ischemia as a cause.

CARDIOVASCULAR

The systolic and diastolic blood pressures (at heart level) increased invariably in both EBI and EBO accelerations. With experienced pilots, the increments were characteristically from 120/80 to 150/115 at 8 g, but in a few inexperienced subjects the diastolic pressures rose to 140 with the systolic beyond the range of the instrument. This emphasizes the important emotional component of this variable.

The experienced subjects showed an increase of heart rate during EBI acceleration, typically from 85 to 120, which is an unremarkable tachycardia considering the blood pressure changes. In EBO acceleration, however, three curiously different heart rate responses were observed (Figs. 7, 8, 9) and several subjects displayed all three (in separate runs). In some instances the heart rate increased, which is the response one would expect;

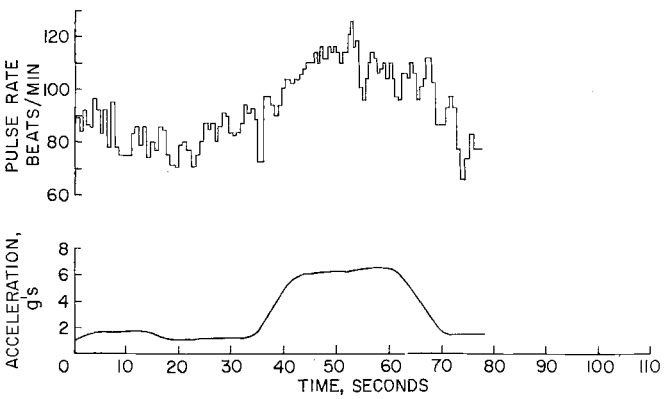

Fig. 7. Heart rate during 6 g EBO.

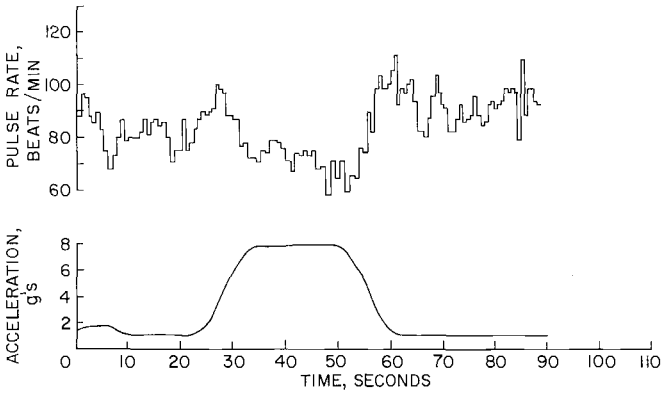

Fig. 8. Heart rate during 8 g EBO.

in other runs there was a modest bradycardia (declining from 85 to 70). It is conceivable that the hydraulic displacement of the thoracic viscera could mechanically stimulate the pressoreceptors in the arch of the aorta, but this would seem more likely to occur in EBI acceleration. The third response resembles an exaggerated sinus arrhythmia, and since it follows the respiratory pattern so closely, it is undoubtedly associated with the changing intrathoracic and intraabdominal pressures. We do not consider any of the heart rate changes in EBO acceleration as contributory to a decline in pilot performance during acceleration exposures of the duration we have studied.

Premature cardiac contractions were observed frequently during EBI accelerations but only on a few occasions with EBO acceleration. The greatest incidence was seen in those test pilots with the least

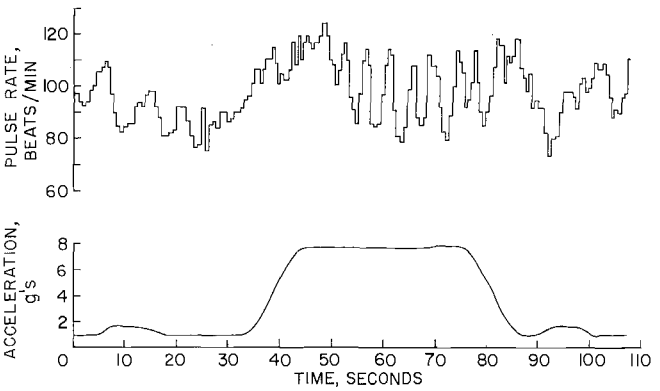

Fig. 9. Heart rate during 8 g EBO.

centrifuge experience (although they were otherwise professionally comparable to the others). One of the most dramatic differences between the effects of EBI and EBO acceleration was seen in the arterial hemoglobin saturation as measured by the Water's ear oximeter. The two records shown superimposed in Figure 10 are typical of eight pairs of runs made at 4 and 6 g EBI and EBO on the University of Southern California centrifuge. The significance of the rapidly diminishing saturation under EBI acceleration is discussed in the section on respiratory studies below.

In other experiments, the pulse amplitude at the

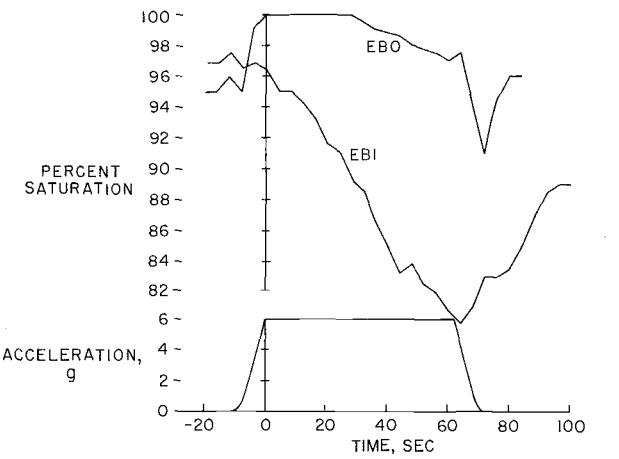

Fig. 10. Human arterial hemoglobin saturation during acceleration.

ear was measured semiquantitatively by a photocell device. This amplitude showed little change in either vector except at accelerations in excess of 8 g, and then the decrement in EBI was more pronounced.

RESPIRATORY STUDIES

With one exception, all of our subjects report dyspnea and difficulty on inspiration when subjected to EBI acceleration above 6 g, whereas they are able to breathe much more comfortably under EBO acceleration at each level of g stress.

Vital capacity and alveolar ventilation.—In a previous publication[7] we reported that at 6 g EBI, the vital capacity is reduced to 25 per cent of its value at 1 g and that the expiratory reserve was virtually abolished. At 6 g EBO, the vital capacity is still 85 per cent of the 1 g value. We have been unsuccessful in extending our vital capacity measurements during acceleration exposures in excess of 6 g. At the higher EBI g stresses, the maximal inspiratory and expiratory efforts nearly always induced paroxysmal coughing, causing the subject to terminate the run. In fact, uncontrollable coughing was the commonest single cause for premature termination of centrifuge runs.

Nevertheless, some measurements of minute ventilation have been extended to 14 g EBI and 10 g EBO. At the higher levels of EBI g stress, the tidal volume approaches the anatomical dead space volume, and so even with increased respiratory frequency, the alveolar ventilation is greatly diminished. In the plot shown in Figure 11, a very conservative dead space volume of 120 ml is arbitrarily assumed for each subject, and the minute alveolar ventilation is calculated in the usual fashion.

$$V_A = f(V_I - V_D)$$

For each pilot, the mean minute alveolar ventilation at each level of g stress was calculated as a percentage of that at 1 g prior to the centrifuge run. The means of these percentages are plotted in Figure 11.

The ventilatory difficulties of EBI acceleration stress are partly due to the displacement of the abdominal viscera against the diaphragm, causing not only a smaller

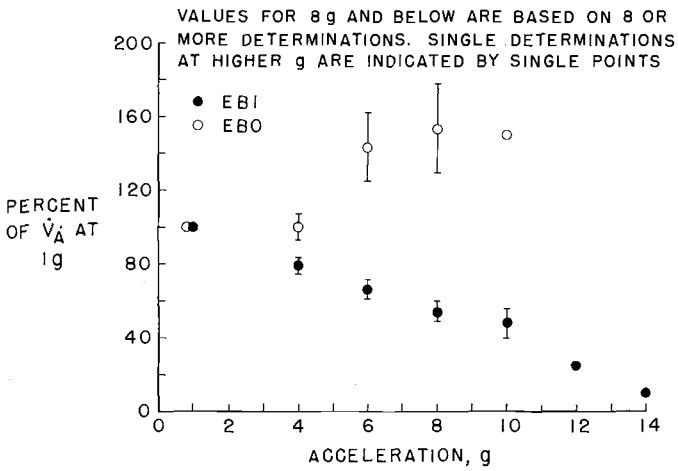

Fig. 11. Alveolar ventilation, per cent of mean minute alveolar ventilation (V_A) at 1 g.

relaxation volume of the thorax but also a greater resistance to the expansion of the intrathoracic volume. In EBO acceleration, the abdominal viscera do not crowd against the diaphragm in this fashion and the volume of the lungs at the end-tidal position may actually be greater than the 1 g normal.

Functional residual capacity.—The relative deflation of the lung under EBI acceleration at only 6 g is clearly shown in the upper spirometer record in Figure 12. Since the pilot is breathing in a closed circuit, the increased volume in the spirometer at the onset of g stress reflects a decrease in lung volume. The end-tidal pulmonary volume now imposed on the subject is one ordinarily achieved only by forced expiration, and, as discussed previously,[7] the consequent stimulation of deflation receptors is probably an important component of the acceleration dyspnea.

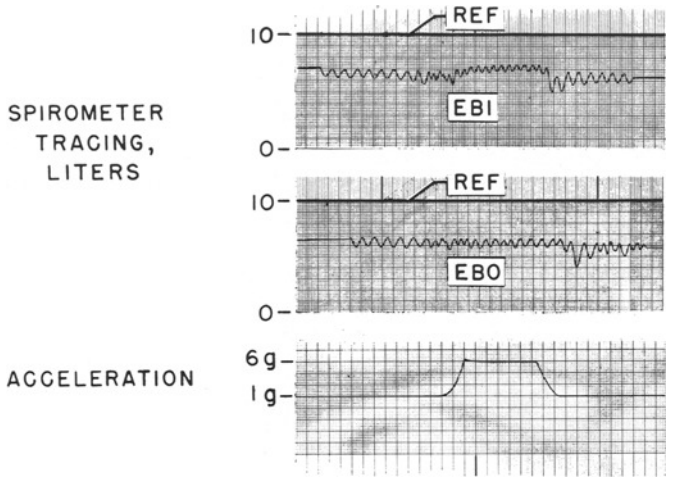

Fig. 12. Functional residual capacity changes during 6 g EBI and EBO.

Systematic measurements of the shifts in the end-tidal spirometer volume at the onset and cessation of g stress permitted the calculation of the changes in functional residual capacity induced by acceleration. The functional residual capacities of two of our subjects were first determined at 1 g by the closed-circuit nitrogen technique. The mean values obtained were 2.9 and 2.6 liters, respectively. The mean decrement during 4 g EBI was 0.6 liter in each pilot, and at 6 g EBI it was 0.9 liter in each. In EBO acceleration, however, there was no change at 4 g, and an increase of 0.4 liter at 6 g.

Nitrogen washout.—Observations of nitrogen washout during acceleration revealed the interesting phenomenon shown in Figure 13. Although the nitrogen content of the system appeared to have reached equilibrium during acceleration, the cessation of 4 or 6 g EBI stress was accompanied by a small but rapid rise in the closed-circuit nitrogen content; this was never seen after EBO acceleration.

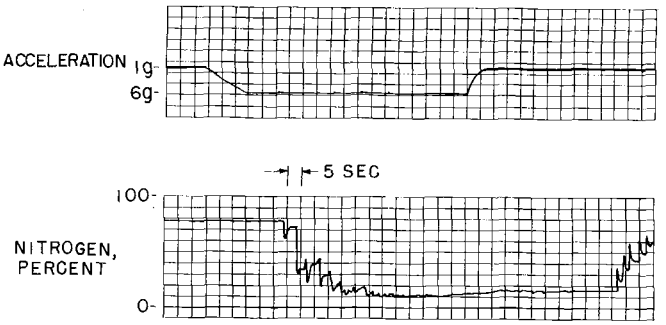

Fig. 13. Nitrogen washout during 6 g EBI acceleration.

Because of the fluctuations in the spirometer volume at this time, it is not clear whether this is due to the "dumping" of 100-150 ml of extra nitrogen, the rapid uptake of a similar volume of oxygen, or a combination of both.

If there is any diminution in venous return during EBI acceleration, the nitrogen content of the pooled blood would be in equilibrium with air. During the near washout of the lungs with oxygen (after the onset of g), the alveolar partial pressure of nitrogen falls to low levels, so that with the cessation of g stress, the return of the previously pooled venous blood to the pulmonary circulation would cause a rapid unloading of nitrogen consistent with that observed. The return of pooled blood with a substantially subnormal oxygen tension could similarly account for a sudden increase in oxygen uptake.

Another possibility to be considered, however, is that during EBI acceleration, the thoracic compression may occlude some portions of the lungs which still contain air but remain unventilated until the cessation of g stress. The subsequent release of air trapped in this fashion could also account for the rise in nitrogen content of the system.

Oxygen uptake.—The spirometer records were used to estimate oxygen uptake in two subjects during several runs at 1, 4, and 6 g EBI and EBO. It is recognized that even with the longer acceleration runs (2 to 3 minutes), the slope of the declining spirometer volume cannot be determined with a suitable degree of precision, and that, furthermore, the oxygen uptake during these short periods does not necessarily reflect the oxygen consumption. One pilot's mean oxygen uptake was 320

ml/m²/min at 1 g, and remained essentially unchanged at 4 and 6 g EBI; it increased to 360 ml/m²/min at 4 and 6 g EBO. The other pilot showed the same pattern in that his oxygen consumption was 240 ml/m²/min at 1 g and also at 4 and 6 g EBI. During EBO acceleration it increased to 264 ml/m²/min and 300 ml/m²/min at 4 and 6 g, respectively. These values may be compared with the normal basal rate of about 135 ml/m²/min. (All values S.T.P.)

It would be expected that during acceleration, the greater muscular tone and greater work of breathing would increase the pilot's oxygen requirements. The absence of an increased uptake during EBI acceleration is, however, consistent with our other findings and it is probably due to the pilot's incurring an oxygen debt, both in the usual sense and as a transient consequence of diminished venous return, as discussed above.

Hypercapnia and hypoxia.—For a pilot breathing pure oxygen, the diminished alveolar ventilation in EBI acceleration would be more likely to affect his psychomotor performance through hypercapnia than through hypoxia. For this reason we have paid particular attention to the elimination of CO_2 under acceleration.

Under EBI accelerations of 8 g or more, the carbon dioxide content of the mixed expired air fell to 1 or 2 per cent, which is to be expected, since each breath does little more than ventilate the dead space. After the conclusion of 1½ to 2 minutes of acceleration, the carbon dioxide content then showed a compensatory rise to 6 or 7 per cent, indicating some degree of hypercapnia. Carbon dioxide retention was not observed under EBO acceleration.

In some later runs in which the intake catheter of the carbon dioxide meter was located right at the pilot's mouth, however, the end-expiratory carbon dioxide content during 6 g EBI acceleration was elevated by only 0.5 per cent over the normal. It is appreciated that the end-expiratory gas composition does not accurately reflect alveolar gas composition under the circumstances of rapid shallow breathing and great turbulence, but this observation suggests, nevertheless, that the alveolar carbon dioxide does not increase markedly, and that venostasis and a diminshed pulmonary perfusion may be at least as important in causing the carbon dioxide retention as is the reduced alveolar ventilation.

In this same context, it is highly significant that the arterial hemoglobin saturation declined during EBI acceleration, whether the subject was breathing air or nearly pure oxygen. This is also strongly indicative of a diminished effective pulmonary blood flow.

Factors likely to diminish the effective pulmonary perfusion are: (1.) As suggested in connection with the nitrogen "dumping" immediately after acceleration, the EBI g stress may cause occlusion of airways so that part of the pulmonary blood flow perfuses unventilated portions of the lungs; and (2.) The roentgenographic studies by Hershgold[3] show a greatly decreased vascularity in the anterior portion of the lungs during EBI acceleration. As the same author points out, the pulmonary circulation is especially sensitive to the hydrostatic effects of increased gravity because of its normally low mean arterial pressure. Lindberg, et al.,[5] have shown that up to 5 g EBI, cardiac output even increases slightly, and so we can conclude that the total pulmonary blood flow is not diminished. Consequently, the posterior portions of the lungs must accept the whole of the normal pulmonary blood flow and also a pooling effect from the hydrostatic gradient. The vascular volume in the posterior portions is increased, therefore, at the expense of the alveolar volume, causing a less favorable ventilation-perfusion relationship.

The graying of vision and the decline in ear pulse amplitude during EBI acceleration in excess of 8 g suggest that at these higher levels, the increasing pulmonary resistance actually contributes to a diminution in cardiac output. It is almost certain that the reflex pulmonary hypertension is largely contributory to the dramatic increases in right atrial pressure reported by Lindberg, et al.,[5] and our own evidence of impeded venous return.

Without roentgenographic studies of the lungs under EBO acceleration, it is not practical to estimate the hydrostatic displacement of pulmonary blood flow in this acceleration vector. The increased alveolar ventilation during EBO g stress may be a compensation for impaired perfusion, but, on the other hand, it may be a simple response to an unusual sensation. The absence of carbon dioxide accumulation under EBO acceleration certainly suggests, however, that there is no significant impedance to venous return.

SUMMARY AND CONCLUSIONS

We find that alveolar ventilation and arterial hemoglobin saturation are severely diminished during EBI acceleration, and that the latter decrement is progressive. There is also indirect evidence that diminished venous return contributes to the progressive hypoxia and hypercapnia. The dyspnea reported by most pilots under EBI acceleration can be accounted for in part by the hypercapnia, but the substantial deflation of the lungs is probably the chief cause of this discomfort.

In contrast, our measurements show that under EBO acceleration, alveolar ventilation and arterial hemoglobin saturation are essentially normal. Furthermore, the ear pulse amplitude, blood pressure, and heart rate data all suggest that cephalic blood flow is at least as good as that during EBI acceleration and almost certainly better.

The tracking performances shown by the pilots under EBO acceleration, however, do not show the clear-cut superiority to be expected from the physiological advantages described above. Performance deteriorates in both acceleration vectors, and yet our physiological data suggest that only in EBI acceleration is there sufficient cerebral hypoxia to account for the deterioration. It would appear, then, that some other factor causes the decline in performance during EBO acceleration, and that its effects are fortuitously similar to the more obvious factors causing the deterioration during EBI acceleration. It is possible that during EBO acceleration, despite a generally adequate cephalic blood flow, some increase in intra-optic pressure may cause a strictly local retinal ischemia. This will be

investigated by cinephotography of the retina during acceleration. Despite the greater respiratory comfort, EBO acceleration is, nevertheless, a strange sensation even for experienced test pilots. It is likely that a subject's tracking performance suffers because a portion of his attention is inevitably channeled into concern for his restraint system and the unfamiliar sensations in this acceleration vector.

We consider that the most likely explanation of the seeming discrepancy, however, lies in the flow and *accumulation* of tears on the cornea during EBO acceleration. Tracking performance depends initially on the visual input, and if vision is blurred by tears, the performance will be affected just as adversely as if the visual decrement were of a more sinister etiology. This suggests that if the wavering refraction of the tears could be overcome by, for example, the pilot's wearing goggles filled with a physiologically compatible fluid of appropriate refractive index, then the major advantages of EBO acceleration would be reflected in an enhanced control facility.

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