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A technique for the noninvasive monitoring of cerebral oxygen status was evaluated on volunteer subjects on the USAF School of Aerospace Medicine Centrifuge. By using multiwavelength near-infrared spectrophotometry, the instrumentation measured changes in the quantities of reduced and oxygenated hemoglobin (and their sum, an indicator of cerebral blood volume), and the quantity of oxidized cytochrome c oxidase within the forebrain. Test used acceleration of up 9 G with onset rates from 0.1 to 5.0 $G \cdot s^{-1}$, anti-G suits and straining maneuvers, and hyperoxic and hypoxic breathing mixtures. In general, +Gz acceleration produced a fall in blood volume within the cerebral microcirculation with a relative increase in the content of reduced hemoglobin and a tendency toward reduction of cytochrome c oxidase. These findings are discussed in relation to accepted changes in arterial blood pressure, cerebral blood flow, and arterial oxygen saturation caused by acceleration exposure.

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A Near-Infrared Spectrophotometric Method for Studying Brain O₂ Sufficiency in Man During +Gz Acceleration

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GLAISTER DH, JÖBSIS-VANDERVLIET FF. *A near-infrared spectrophotometric method for studying brain O₂ sufficiency in man during +Gz acceleration.* Aviat. Space Environ. Med. 1988; 59:199–207.

A technique for the noninvasive monitoring of cerebral oxygen status was evaluated on volunteer subjects on the USAF School of Aerospace Medicine centrifuge. By using multiwavelength near-infrared spectrophotometry, the instrumentation measured changes in the quantities of reduced and oxygenated hemoglobin (and their sum, an indicator of cerebral blood volume), and the quantity of oxidized cytochrome c oxidase within the forebrain. Tests used acceleration of up to 9 G with onset rates from 0.1 to 5.0 G·s⁻¹, anti-G suits and straining maneuvers, and hyperoxic and hypoxic breathing mixtures. In general, +Gz acceleration produced a fall in blood volume within the cerebral microcirculation with a relative increase in the content of reduced hemoglobin and a tendency towards reduction of cytochrome c oxidase. These findings are discussed in relation to accepted changes in arterial blood pressure, cerebral blood flow, and arterial oxygen saturation caused by acceleration exposure.

CEREBRAL OXYGEN sufficiency is of the utmost importance to the maintenance of performance in aircrew, yet may be compromised by several common flight stresses. While excessive +Gz acceleration is the most obvious cause for inadequate cerebral blood flow in fighter aircrew, G-induced right-to-left shunts, altitude hypoxia, hyperventilation, or toxic fumes may all reduce the oxygen supply to brain tissue. However, despite aeromedical relevance, cerebral blood flow and cerebral oxygen status have received scant attention, undoubtedly because of the hitherto complex, invasive, and hazardous procedures necessary

for their investigation. This state of affairs may be corrected by the application of a noninvasive technique utilizing multiwavelength near-infrared transmission spectrophotometry.

In the near-infrared (NIR) range of 700–1,300 nm, a significant amount of radiation can be transmitted through several centimeters of tissue. Fortuitously, both oxygenated and reduced hemoglobin (HbO₂ and Hb) exhibit weak absorption activity in this part of the electromagnetic spectrum, Hb having a peak at 760 nm which disappears upon oxygenation, while HbO₂ has a broad band of absorption around 900 nm (Fig. 1).

The final step in the chain of metabolic reactions which lead to the production of carbon dioxide and water occurs when cytochrome c oxidase (also known as cytochrome a,a₃) reacts directly with molecular oxygen. This reaction releases energy available to the tissue in the form of high energy phosphate bonds, and accounts for more than 90% of the tissue's oxygen uptake. Oxidized cytochrome c oxidase also has a weak absorption band in the near infrared (780–870 nm) which disappears upon reduction (Fig. 1). Thus, changes in absorption at selected NIR wavelengths can be used to determine the redox status of both hemoglobin and cytochrome c oxidase through several centimeters of tissue (8). While reduced cytochrome c oxidase does not have a specific absorption peak in the NIR region, its presence may be assumed from the disappearance of the oxidized cytochrome signal; for, unlike hemoglobin, this chromophore remains fixed in the tissue. Further details of the methodology are given elsewhere (10).

While cells in skin and bone contribute to the NIR absorption, bone contains relatively little blood and cytochrome; and cat studies have confirmed that the skull contributes none to the latter and no more than 5% to the blood signals (10). Again, cytochrome a₃ is practically
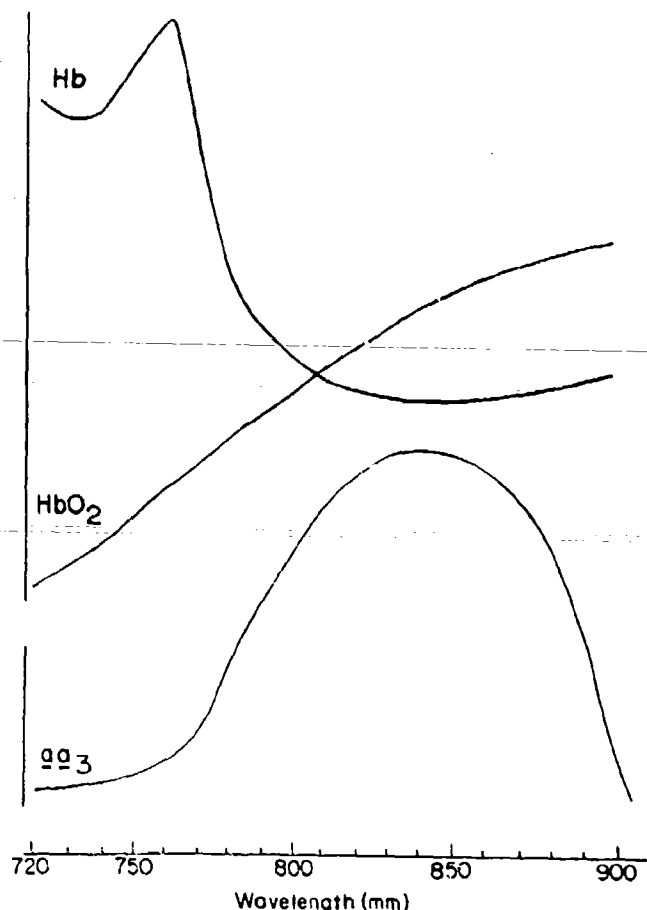
Fig. 1. Near-infrared absorption spectra for hemoglobin (Hb), oxyhemoglobin (HbO₂), and for oxidized minus reduced cytochrome ç oxidase (o,o,ₛ), (not to scale).

MATERIALS AND METHODS

The optics unit of the OMNI-4 produces four wavelengths of NIR light centered at 775, 815, 870, and 910 nm, using pulsed laser diodes. The emerging light is combined and fed to the subject's forehead in a single fiber optic bundle, 3.0 mm in diameter and 3.7 m in length. Energy levels applied to the forehead are less than for normal sunlight, but are effective because the energy is pulsed and has a narrow bandwidth. Light reflected from the skin at the entry optrode passes back through the bundle to a photodiode, the output of which is used to regulate the laser intensity and to become the reference signal for subsequent computation. NIR light transmitted through the skin, skull, and brain tissue is collected at an exit optrode and led through a 5-mm-diameter fiber optic bundle to a detector photomultiplier. The output of this device is preamplified, demodulated, and amplified, the four wavelength signals are then ratioed to their respective reference signals and expressed as logarithmic functions (optical density being logarithmically related to light transmission). Algorithms are then applied to yield signals which represent changes in the concentrations of Hb and HbO₂, their sum (which represents changes in blood volume in the tissue given that the hematocrit remains constant), and the redox status of cytochrome ç oxidase. Each complete cycle requires 1.024 ms; but a sufficient number of values are summed to give a smooth output, and further smoothing is applied through the use of a 1.0-s time constant. Outputs are displayed on digital voltmeters and recorded in analog form on a strip-chart recorder.

Because each of the output channels is based on weighted changes in optical density measured at each of the four NIR wavelengths, and the length of the intracerebral lightpath is unknown, the recordings cannot be scaled in absolute units, but only in terms of "variation in density" ($^v/_d$, commonly referred to as "Vanders") caused by changes in the concentrations of the relevant chromophores in the transilluminated tissue. One Vander represents a 10-fold change in the computed signal. Although not interpretable in absolute concentration terms, the observed changes are internally consistent, i.e. linearly related to concentration.

Initially, the optrodes were mounted in an aircrew protective helmet (type HGU-26). Subsequently, they were attached to the inside of the browband of a lightweight head harness using dental registration material (Express; 3M). The optrode centers were 43 mm apart on the forehead and between 25 and 35 mm superior to the upper margin of the orbit.

Acceleration exposures were conducted on USAFSAM's 6.1-m radius centrifuge, the OMNI-4 units being mounted on the forward racking while the subject sat in a conventional ejection seat with the back at an angle of 15° from the vertical. The OMNI-4 outputs were recorded via slip-rings, together with acceleration level, two channels of electrocardiogram, and beat-by-beat heart rate. Visual loss induced by +Gz acceleration was estimated by using a conventional horizontal light bar. The subject fixated on a central red light and observed changes in its brightness, and that of two peripheral green lights which subtended a visual angle of 50°. The voluntary informed consent of the subjects used in this research was obtained in accordance with AFR 169-3.

indetectable in skin (9) and the contribution from blood in the overlying scalp can be minimized by applying the fiber optic terminations, or "optrodes," with sufficient pressure to cause local blanching. Hence, the major part of the received signal comes from the brain cortex. Studies at Duke University Medical Center (unpublished observations) have shown that in the cat, and presumably also in man, the normal resting contribution to the blood signal constitutes approximately 75% Hb and 25% HbO₂. Thus, the technique appears to be biased towards the venous end of the cerebral microcirculation, so yielding information which should correlate well with the oxygen status of the brain cells rather than with that of the incoming arterial blood. This report considers the application of multiwavelength near-infrared transmission spectrophotometry to human subjects exposed to +Gz acceleration stress on the USAF School of Aerospace Medicine centrifuge. It is not intended as a comprehensive evaluation, but to demonstrate the potential of the technique as a tool for aerospace research. The device used was an OMNI-4 (Oxidative Metabolism Near Infrared using 4 wavelengths.)[1]

The possible sensitivity of the basic OMNI-4 instrumentation to inertial forces was evaluated by clamping the optrodes across a stack of white card discs in a light tight box to simulate the NIR absorption of skull and brain tissue. The number of discs was chosen so that the high voltage setting required for the photomultiplier was the same as that used for a human subject. Gain settings were 0.2 %/d for full-scale deflection of the pens. Centrifuge runs were carried out at up to 9 G with onset rates of 1 G·s⁻¹ and 30-s plateaus. No deflections related to G were noted in any of the OMNI-4 outputs.

With human subjects, and despite the use of a lightweight optrode mount, deflections were seen which were related to the onset and offset of G, and which could be reproduced by applying and releasing downward pressure to the optrode assembly. These motion artifacts were completely abolished when the optrode mount was braced by a rod attached to a dental bite plate. All the reported centrifuge results were obtained on three subjects fitted with this device.

RESULTS

Shown in Fig. 2 is the response of a subject to a gradual increase in acceleration (0.1 G·s⁻¹) terminated when he lost central vision (blackout). All three blood channels—Hb,

HbO₂, and cerebral blood volume (CBV)—show decreases with increasing acceleration, although HbO₂ falls more than does Hb. Return to 1 G led to recovery of all three measures. Cytochrome c oxidase shows a trend towards reduction starting at about +4Gz, and returns to baseline by way of a prolonged overshoot. Fig. 3 illustrates a similar run on another subject who did not blackout until +8.0Gz. The blood signals show a similar pattern of changes, though greater in degree. However, the cytochrome c oxidase changes were reversed with both Hb and CBV exhibiting pronounced overshoots before regaining their baselines.

Shown in Fig. 4 are runs using a rapid rate of G onset. Panel A is the response to a 30-s plateau of +5Gz, attained at 1 G·s⁻¹ with the subject unprotected and straining to the minimal extent required to maintain central vision. He lost 100% of the peripheral lights and 80% of the central red light. Panel B shows a similar run with the same subject wearing an inflated anti-G suit; only 50% of the peripheral lights was lost. In panel C he wore an anti-G suit and performed a maximal anti-G straining maneuver (AGSM); there were no visual symptoms. Finally, panel D shows the response to +5Gz attained at an onset rate of 5.0 G·s⁻¹, no anti-G suit was worn and only minimal straining was carried out. He lost 100% of both peripheral and central lights and

ACCELERATION

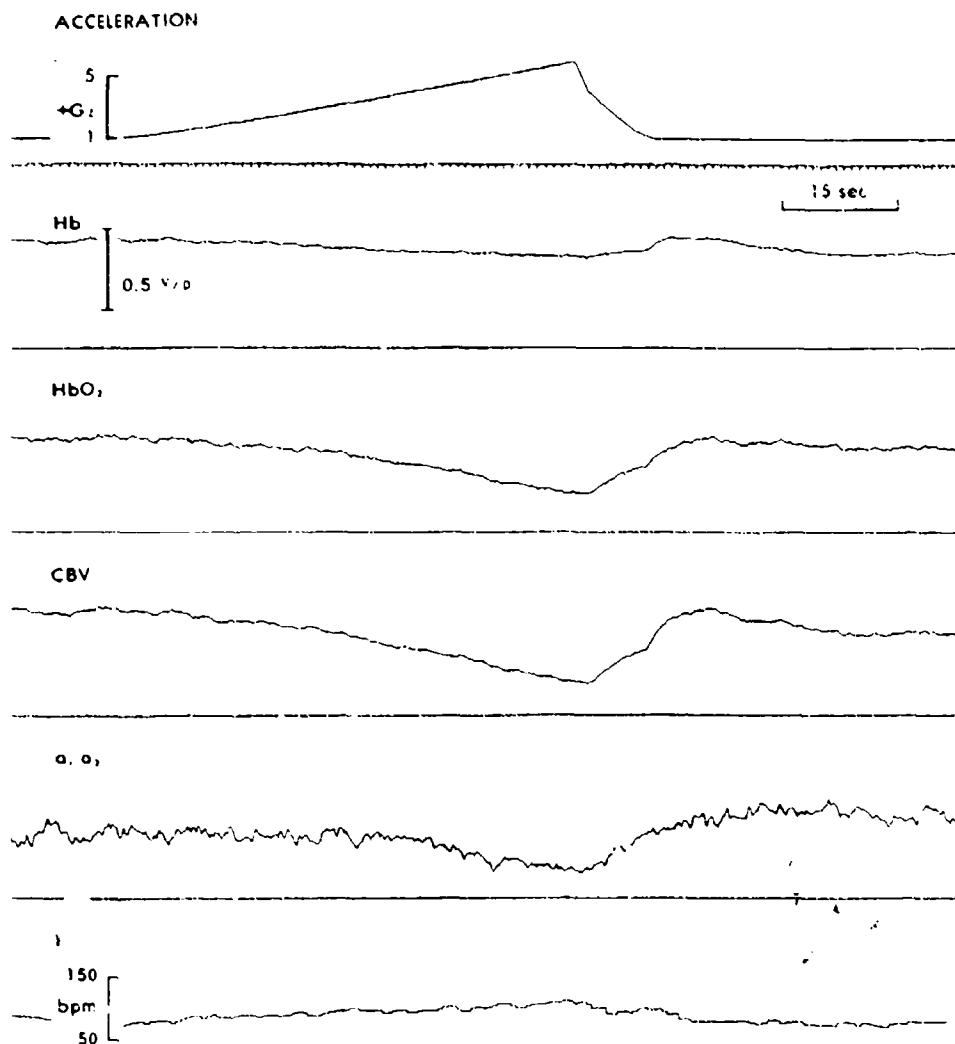Fig. 2. The effect of a gradual onset of acceleration (0.1 G·s⁻¹) on subject JW. From above, downwards, the records show applied acceleration, the quantities of Hb and HbO₂ in the forebrain, the regional blood volume (CBV), the quantity of oxidized cytochrome c oxidase (a,a₃), and heart rate (HR). The OMNI-4 signals are to the same scale.
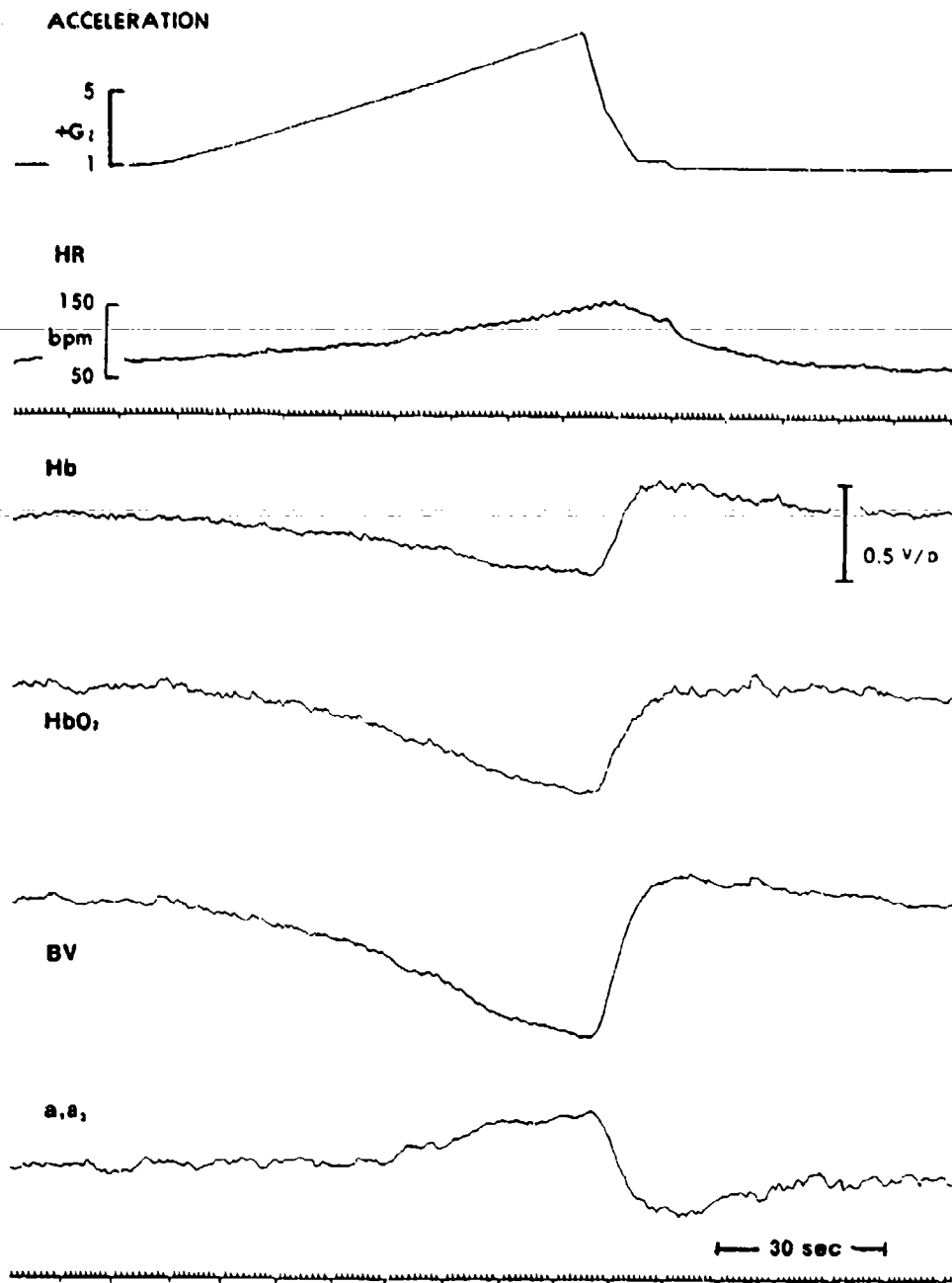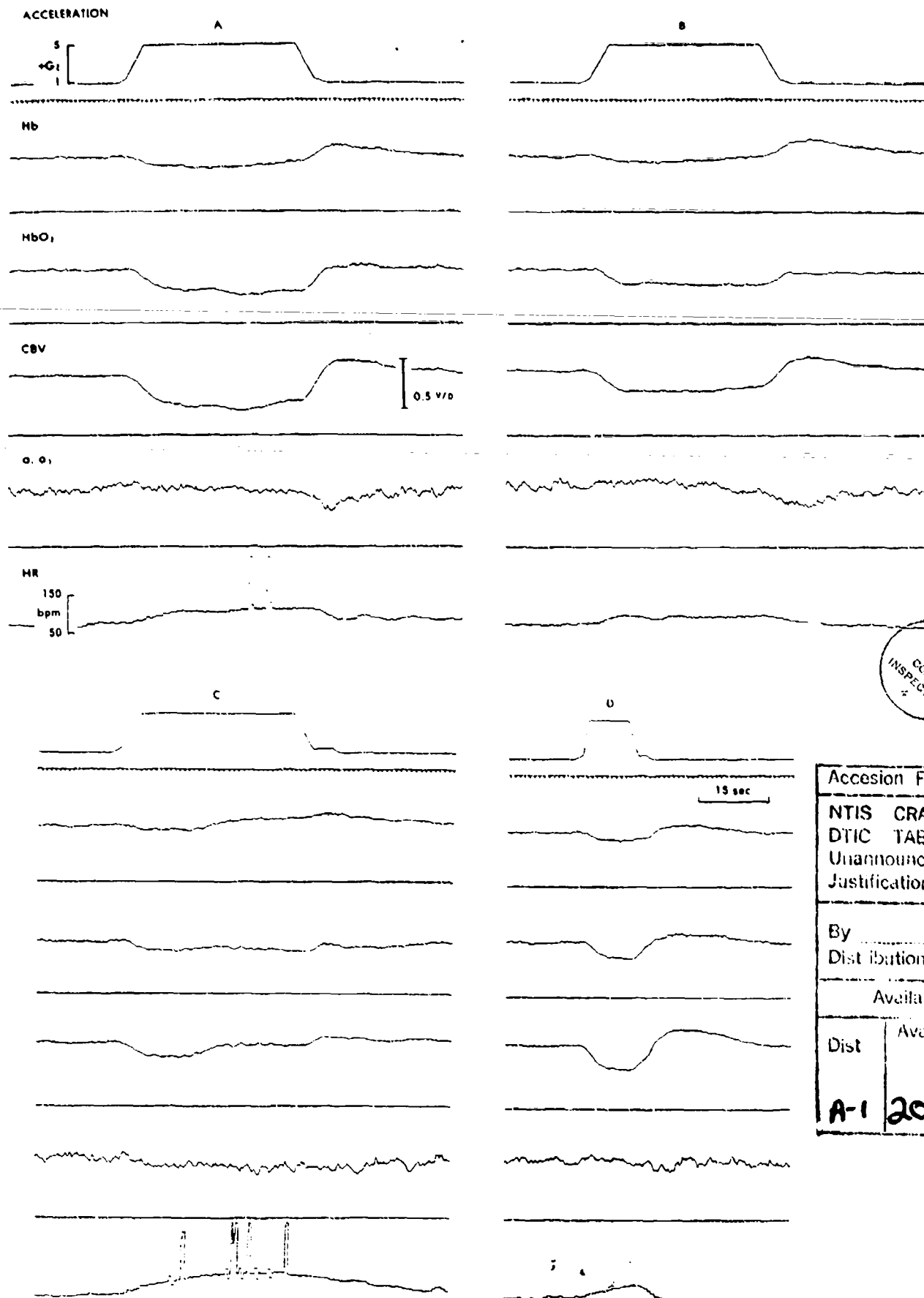
ACCELERATION

HR

Hb

HbO₂

BV

a,a₃

├── 30 sec ──┤

Fig. 3. The effect of a gradual onset of acceleration (0.1 G·s⁻¹) on subject DG. From above, downwards, the records show applied acceleration, heart rate (HR), the quantities of Hb and HbO₂ in the forebrain, the regional blood volume (BV), and the quantity of oxidized cytochrome ç oxidase (ç,ç₃). The OMNI-4 signals are to the same scale.

terminated the run when he felt close to losing consciousness. All acceleration exposures caused falls in the three blood channels which were greater for HbO₂ than for Hb; and return to 1 G was associated with overshoots for Hb and blood volume. However, the use of an anti-G suit reduced the magnitude of the blood changes by some 40%. The performance of an AGSM (panel C) led to even smaller changes in outputs, Hb actually rising above its control level during the latter half of the run, while blood volume approached its baseline value at this time. The overshoots in Hb and CBV following return to 1 G (panels A, B, and D) were virtually abolished by use of the AGSM. While not illustrated, a simultaneous recording of anti-G suit pressure allowed the inspiratory gasps of the AGSM to be timed, and this showed that the ripples seen on the cerebral blood

volume tracing (panel C) are of respiratory origin. Thus, cerebral blood volume increases during each strain, and starts to fall approximately 1 s following the onset of an inspiratory gasp. Cytochrome ç oxidase showed little or no change during the acceleration exposures, but there was a transient reduction upon return to normal gravity following the first two runs (panels A and B).

Illustrated in Fig. 5 is the response to a simulated aerial combat maneuver (SACM) in which plateau acceleration alternated, every 15 s, between 1.5Gₓ and +7.0Gₓ. The subject wore an anti-G suit and strained as necessary to maintain central vision. The record shows a stepwise increase in Hb and a similar fall in HbO₂, although cerebral blood volume had a tendency to recover and stabilize during the second plateau at +4.5Gₓ (arrow). The final +7Gₓ pla-

Fig. 4. Recordings as for Fig. 2, but rapid onset rate (1.0 G·s⁻¹) runs to + 5 G₂, subject DG. Panel A: unprotected subject with minimal tensing experienced 80% central light loss. Panel B: anti-G suit, minimal tensing; 50% peripheral light loss. Panel C: anti-G suit, anti-G straining maneuver; no light loss. Panel D: as for panel A, but onset rate increased to 5 G·s⁻¹; 100% central light loss and subject close to loss of consciousness.
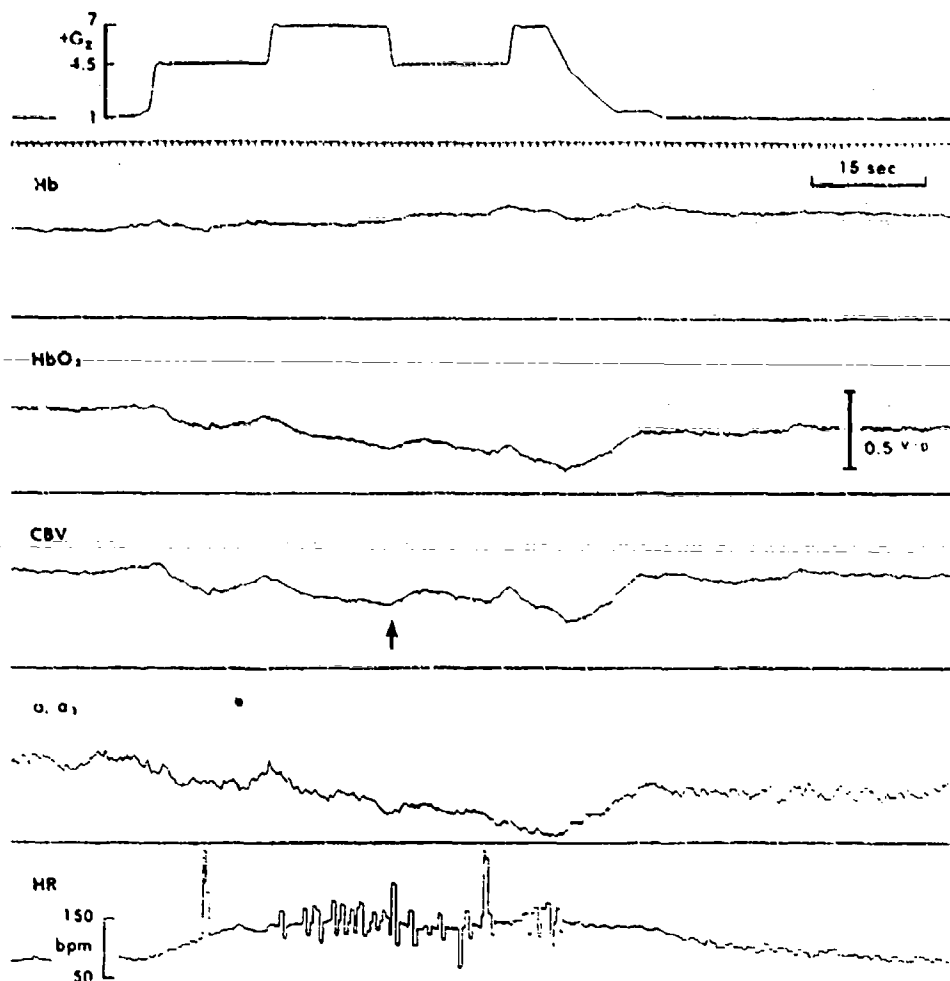
Fig. 5. Response to a simulated aerial combat maneuver, with subject JW wearing an anti-G suit and straining as necessary to maintain vision. Recordings as for Fig. 2.

teau produced a greater fall in brain blood content, and the subject terminated the run at this point as he could no longer maintain his central vision. despite straining. Cytochrome c oxidase also showed stepwise reductions, the lowest value being seen as the run was terminated. Heart rate, on the other hand, remained effectively constant from the end of the first $+4.5G_z$ plateau until the end of the run.

Subjects also performed Valsalva maneuvers while seated at rest, two such recordings being illustrated in Fig. 6. The left-hand panel illustrates a well sustained, but not very forceful, maneuver (subject MK). As intrathoracic pressure was raised, cerebral blood volume increased, the increase being contributed to by both Hb and HbO_2. No change was noted in cytochrome c oxidase. Subject DG, on the other hand, performed a very energetic, but brief maneuver (Fig. 6, right panel) in which Hb contributed the entire increment in blood volume, and cytochrome c oxidase moved towards reduction.

Finally, illustrated in Fig. 7, is the effect of breathing hypoxic (9% O_2, 91% N_2) and hyperoxic (95% O_2, 5% CO_2) gas mixtures. Hypoxia caused an increase in Hb, a fall in HbO_2, and a reduction in cytochrome c oxidase. These changes were reversed by hyperoxia, 5% CO_2 being added to prevent a cerebral vasoconstrictor response. The subject was on this mixture at the start of the recording, and no significant alteration in blood volume is seen until the final

change to breathing air. whereupon the cerebral blood volume falls. During hypoxic, or CO_2-induced. cerebral vasodilation, cardiac pulsations are apparent in the blood volume and HbO_2 traces (Fig. 7).

DISCUSSION

The OMNi-4 equipment was found to be easy to set up and operate and, motion artifacts excepted. gave no problems. Stabilizing the optrodes was found essential whenever a change in G vector occurred, as with head movement, or centrifugal acceleration. Obviously the scalp is freely mobile on the skull and offers inadequate stability; but, while the dental bite offered an efficient remedy, a more acceptable long-term alternative should be sought. Subjects also complained of discomfort from the optrodes, though the pressure needed to achieve blanching of the underlying skin was acceptable for up to an hour by our well motivated subjects.

Figs. 6 and 7 illustrate changes resulting from two well established procedures having predictable effects on cerebral hemodynamics. A Valsalva maneuver is known to cause an initial rise in systemic blood pressure which subsequently falls as venous return is impeded by the raised intrathoracic pressure. The raised arterial and venous back pressures will tend to increase the blood content of the brain as demonstrated. In the first subject illustrated, (Fig. 6, left panel).
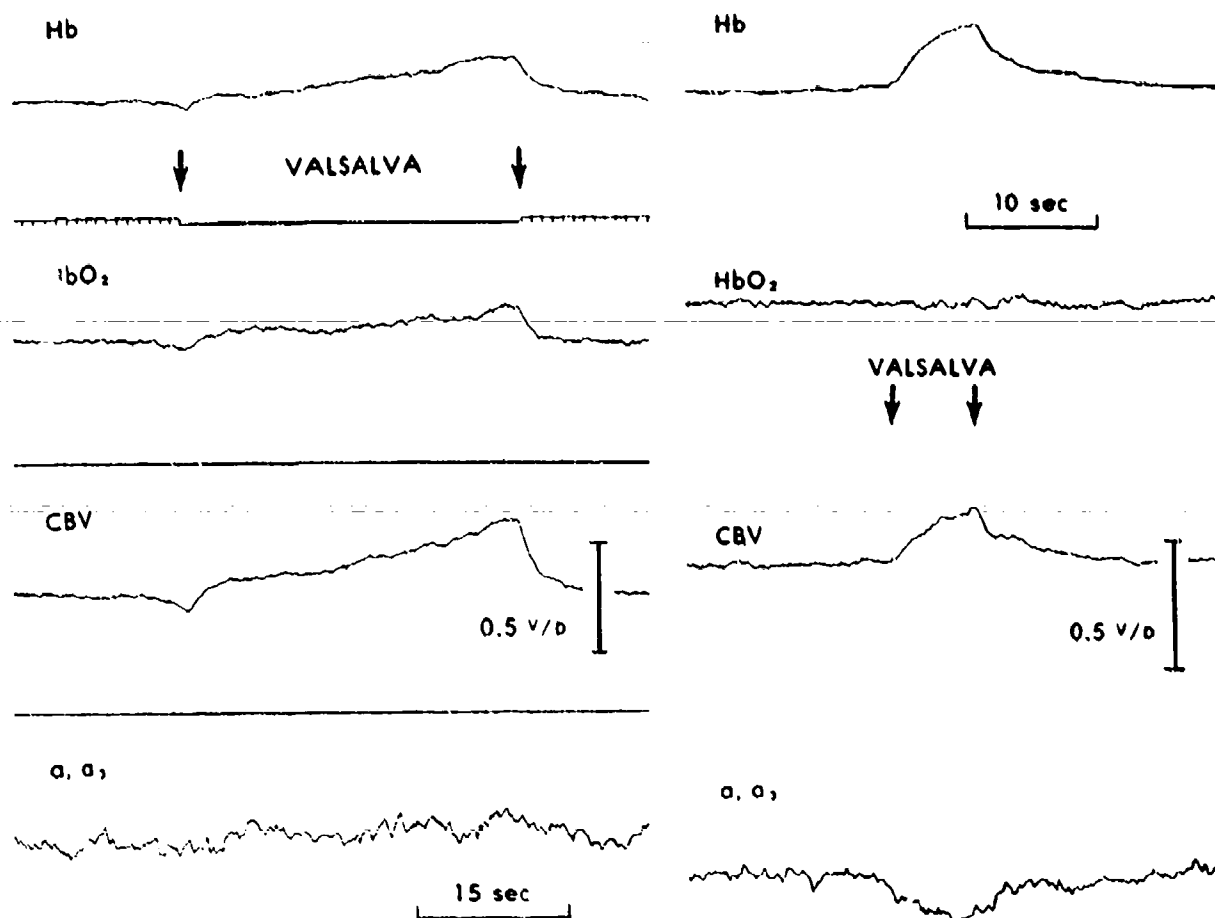
Fig. 6. Valsalva maneuvers at 1 G: subject MK, left panel; subject DG, right panel. The four signal channels are to the same scale and show changes in Hb, HbO₂, regional cerebral blood volume (CBV), and oxidized cytochrome c oxidase (a.a₃).

cerebral blood flow was not apparently compromised and the brain's oxygen sufficiency was maintained. With the more forceful maneuver exerted by the second subject, (Fig. 6, right panel), cerebral blood flow was presumably impaired. Thus, blood in the cerebral microcirculation became proportionately more reduced and the brain cytochrome c oxidase also moved towards reduction. As shown in Fig. 7, recorded from the same subject at the same amplification, the degree of reduction was comparable to that following a change from breathing room air to 9% O₂.

An increase in HbO₂ was seen when changing to an O₂ rich breathing mixture (Fig. 7), and also during a prolonged Valsalva maneuver (Fig. 6, left panel). The former increase must represent an overall rise in O₂ saturation as it was mirrored by a fall in Hb content, while the latter presumably reflects a rise in the volume of blood in the cerebral microcirculation due to increased venous back-pressure. Thus, Hb contributed more to the increase in cerebral blood volume than did HbO₂; so that, overall, there was a relative desaturation of blood in the transilluminated field. This effect was even more pronounced during the brief, but forceful, Valsalva maneuver illustrated in Fig. 6, right panel, during which the increase in cerebral blood volume was solely contributed to by Hb.

The pattern of blood and cytochrome c oxidase changes observed in the forebrain during centrifugation needs to be considered in relation to acceleration's known cardiovas-

cular and pulmonary effects. Exposure to +Gz causes a fall in arterial pressure at head level due, initially, to hydrostatic pressure differentials and, subsequently, to impaired venous return and cardiac output (2). Acting in the opposite sense are the baroreceptor reflexes, predominantly from the carotid sinuses, which induce compensatory vasoconstriction, tachycardia, and an increase in blood pressure at heart level. Vision is impaired first peripherally, then centrally, as arterial pressure at eye levels falls towards intraocular pressure, while cerebral blood flow appears to be rather well maintained until arterial pressure at brain level falls to near zero (7). In addition, G-induced inequalities in the distributions of blood and gas within the lungs cause arterial oxygen desaturation (5).

The most consistent finding of NIR spectrophotometry was the loss of HbO₂ from the cerebral microcirculation seen during each exposure to +Gz acceleration. The magnitude of loss was, on occasion, greater than that which followed a change in breathing gas from 95% O₂ and 5% CO₂, to 9% O₂ in N₂ (c.f. Figs. 3, 4 and 7, which were all recorded from the same subject). The magnitude of the HbO₂ loss was also affected by factors known to modify G tolerance and, presumably, the O₂ status of the cerebral tissues. Thus, the fall was lessened by wearing an anti-G suit (Fig. 4, panels A and B) and even more so by maximal straining at +5Gz (Fig. 4, panels A and C). The fall tended to be progressive during a simulated aerial combat maneu-
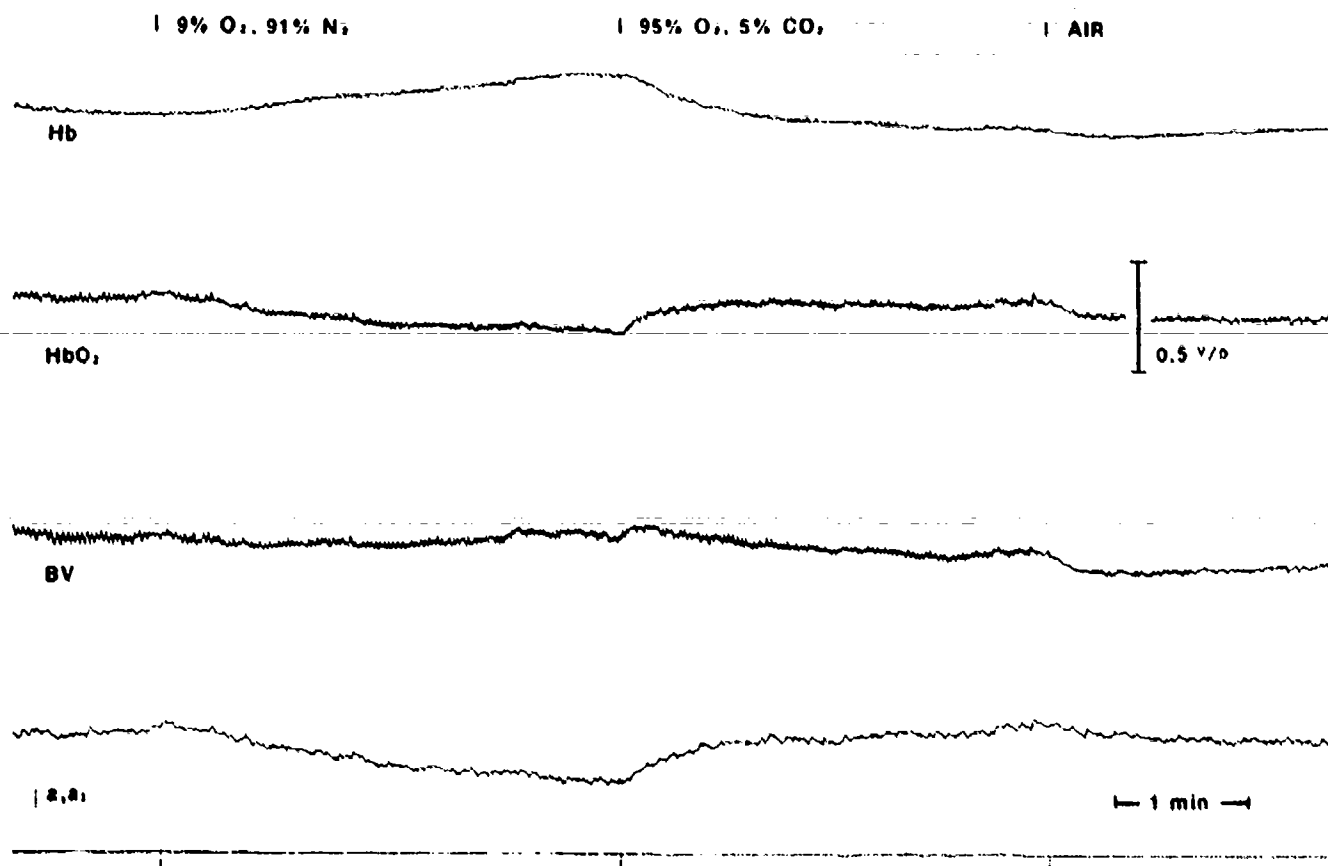
| 9% O₂, 91% N₂ | 95% O₂, 5% CO₂ | AIR

Hb

HbO₂

0.5 V/D

BV

| *,*| |— 1 min —|

Fig. 7. Response to hyperoxic and hypoxic breathing gas mixtures, subject DG. Recordings as for Fig. 6. The subject was breathing the hyperoxic mixture at the start of the recording.

ver (Fig. 5), despite a temporary relaxation in the G stress from +7.0 to +4.5G$_z$.

The blood volume detected within the cerebral microcirculation reflects the sum of changes in the Hb and HbO₂ contents, on the assumption that no significant short term changes occur in local hematocrit. Sustained +G$_z$ acceleration does cause hemoconcentration in man (4), but the effect is quite small—4 to 5% after 5 min at +4G$_z$—and so irrelevant in relation to the large and rapid changes in total hemoglobin content seen, for example, in Fig. 5. Such changes may, therefore, be taken to indicate changes in the volume of blood contained within the cerebral microcirculation.

With a gradual onset of acceleration (Figs. 2 and 3), cerebral blood volume first started to fall at about +1.5G$_z$ and, thereafter, fell at an increasing rate. The maximum fall seen was about 0.7 %, which represents a decrease to about one-third of the control value, though again it must be stressed that absolute figures could only be obtained by making control measurements following total exsanguination. This two-thirds reduction in blood volume is surprisingly large considering that a maximal degree of local vasodilation would be expected as cerebral blood flow became compromised; and, to some extent at least, the fall in transmural pressure would be compensated for by a decrease in cerebrospinal fluid pressure (11). That we are seeing a true physiological effect and not a G-induced artifact is confirmed by a number of observations. Blood volume was not simply related to G level, but was influenced in a

predictable way by several factors known to affect G tolerance: the degree of fall was reduced by inflation of an anti-G suit, and virtually abolished by an anti-G straining maneuver (Fig. 4, panel C); and swings in cerebral blood volume were seen during steady acceleration which were related to intrathoracic pressure (Fig. 4, panel C, and Fig. 6, both panels). Furthermore, cerebral blood volume could return to baseline when its components, Hb and HbO₂, were still considerably offset (Fig. 5). Finally, in runs in which the subject was blacked out and approaching loss of consciousness, return to 1 G was accompanied by an overshoot in blood volume indicative of a reactive hyperemia (Fig. 4, panel D). Such a response has been described in experimental animals after even brief obstruction of cerebral blood flow (6).

A differential effect of acceleration on the brain content of hemoglobin versus oxyhemoglobin was perceived in many of the recordings. A common feature was that both would be reduced immediately after the onset of acceleration, but Hb would then increase while HbO₂ continued to decline (Fig. 4, all four panels). Furthermore, return to 1 G would usually produce an overshoot in the quantity of Hb, while HbO₂ remained below its resting value for 30-60 s. This effect is seen in Fig. 4, panels B and C, and is clearly demonstrated after a simulated aerial combat maneuver (Fig. 5). It is not possible to estimate oxygen saturation from the recorded data since absolute values of Hb and HbO₂ are not known. A rise in Hb and a fall in HbO₂ at a time when blood volume is not changing is, however, most readily

accounted for by desaturation, for which two mechanisms are likely to coexist. Pulmonary shunting induced by acceleration causes a fall in the oxygen saturation of arterial blood (5); and a decrease in cerebral blood flow with maintained oxygen uptake will cause a fall in the oxygen saturation of capillary and venous blood within the brain. O_2 uptake may also be increased locally following a period of hypoxia. Since NIR spectrophotometry appears to be biased towards the venous end of the cerebral microcirculation (presumably because more venous than arterial blood is in the transilluminated field), any of these mechanisms could account for the observed changes. However, a slow return to normal of HbO_2 in the presence of a profound hyperemia (Fig. 4, panel B), and despite the near normal blood volume achieved by use of an anti-G straining maneuver (Fig. 4, panel C), suggests that arterial hypoxia probably plays a significant role. Following the simulated aerial combat maneuver (Fig. 5), HbO_2 had barely started to recover 50 s after return to 1 G, a known feature of the arterial desaturation provoked by shunting and acceleration atelectasis (5). This phenomenon is enhanced by the inflation of an anti-G suit (which reduces lung volumes and promotes closure of basal airways), so accounting for the different recovery patterns seen in panels A and B of Fig. 4.

Changes recorded in the concentration of oxidized cytochrome c oxidase are less easy to account for, as they were not consistent. This chromophore is more difficult to detect than the hemoglobins for several reasons. First, as can be seen from the recorded noise levels, a higher gain is needed for its detection (there being less cytochrome than Hb per unit volume of brain tissue, and its NIR absorption is weaker); and second, reduced cytochrome does not absorb in the NIR. While the quantity of cytochrome per unit volume of brain tissue must stay constant, the transilluminated field could enlarge as hemoglobin drains away and the tissue becomes more translucent. Furthermore, a small mechanical shift of the brain could occur under G which would also affect the cytochrome signal more profoundly than the blood signals. That the problem is related specifically to acceleration is suggested by the observation that a consistent physiological relationship between cytochrome redox status and the Hb/HbO_2 ratio was seen under other conditions, such as breathing of hypoxic or hyperoxic gas mixtures (Fig. 7, and Ref. 10). Furthermore, the use of similar NIR measurement techniques has proved satisfactory during anesthesia (3) and in pre-term infants (1), other situations in which the G vector would have remained constant.

The response of subject JW to an SACM acceleration profile (Fig. 5) is of particular interest, in that this exposure most nearly matches an operational environment; and there is some speculation as to what extent recovery in physiological function can take place during periods of lesser, although still raised, acceleration stress. While cerebral blood volume tended to recover when the stress decreased from +7 to +4.5G_z (Fig. 5, arrow), the returning blood contained more Hb than HbO_2 and cytochrome c oxidase continued to move towards reduction. Also at the conclusion of this

run, blood volume was rapidly restored; but, HbO_2 remained low and the oxidase reduced for a considerable period, presumably because of the development of acceleration atelectasis with maintained right-to-left shunting in dependent regions of the lung. Obviously, the technique of NIR spectrophotometry would be of great value in studying the response to other SACM profiles.

While loss of consciousness was clearly approached on one occasion (Fig. 4, panel D), no changes in OMNI-4 outputs were seen which would have differentiated this run from another (Fig. 4, panel A) in which the cerebral circulation was subjectively less compromised. This was the subject (DG) who failed to show consistent cytochrome c oxidase changes, however; and further studies are required to improve the instrumentation response to this chromophore. Nevertheless, the three blood channels not only furnish a great deal of information relevant to the oxygen sufficiency of the forebrain, but also provide a valuable noninvasive tool for future aeromedical research.

ACKNOWLEDGMENTS

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