AD NUMBER: AD0471880

LIMITATION CHANGES

TO:

Approved for public release; distribution is unlimited.

FROM:

Distribution authorized to U.S. Government agencies and their contractors; Administrative/Operational Use; 31 DEC 1960. Other requests shall be referred to Office of Naval Research, Arlington, VA 22203.

AUTHORITY

USAFSAM ltr dtd 20 Apr 1967

SECURITY MARKING

The classified or limited status of this report applies to each page, unless otherwise marked.
Separate page printouts MUST be marked accordingly.

THIS DOCUMENT CONTAINS INFORMATION AFFECTING THE NATIONAL DEFENSE OF THE UNITED STATES WITHIN THE MEANING OF THE ESPIONAGE LAWS, TITLE 18, U.S.C., SECTIONS 793 AND 794. THE TRANSMISSION OR THE REVELATION OF ITS CONTENTS IN ANY MANNER TO AN UNAUTHORIZED PERSON IS PROHIBITED BY LAW.

NOTICE: When government or other drawings, specifications or other data are used for any purpose other than in connection with a definitely related government procurement operation, the U. S. Government thereby incurs no responsibility, nor any obligation whatsoever; and the fact that the Government may have formulated, furnished, or in any way supplied the said drawings, specifications, or other data is not to be regarded by implication or otherwise as in any manner licensing the holder or any other person or corporation, or conveying any rights or permission to manufacture, use or sell any patented invention that may in any way be related thereto.

SAM-TR-65-42

MONITORING PSYCHOMOTOR RESPONSE TO STRESS BY EVOKED AUDITORY RESPONSES

J. A. FREEMAN, Captain, USAF, MC

May 1965

USAF School of Aerospace Medicine
Aerospace Medical Division (AFSC)
Brooks Air Force Base, Texas

MONITORING PSYCHOMOTOR RESPONSE TO STRESS BY EVOKED AUDITORY RESPONSES

J. A. FREEMAN, Captain, USAF, MC

FOREWORD

This report was prepared in the Biodynamics Branch under Task No. 793003. It was submitted for publication on 14 September 1964.

The technical assistance of the following personnel is gratefully acknowledged: C. F. Martin, G. A. Rex, Staff Sergeant S. E. Howard, Airman First Class O. Siahaya, and Airman First Class M. B. Kardon.

This report has been reviewed and is approved.

Harold V. Ellingson

HAROLD V. ELLINGSON
Colonel, USAF, MC
Commander

ABSTRACT

A sensitive central nervous system (CNS) monitoring technic that can be correlated with behavior and with changes in the surrounding environment during aerospace flight is desirable to the flight surgeon interested in the early detection of possible adverse effects of the flight on the subject, to the neurophysiologist concerned with basic cerebral mechanisms occurring during the unique conditions of space flight, and to the systems engineer interested in any redundant indirect measurement of environmental parameters which serve to enhance the total system reliability.

In this study, a special-purpose digital computer was used to obtain average EEG responses evoked from human subjects by repetitive, nondistracting clicks during sedentary activity, mildly symptomatic hyperventilation, hypoxia, and 2.5 $+G_z$ acceleration on the SAM human centrifuge and in an NF-100 aircraft.

The waveforms obtained were qualitatively distinct for each group. No appreciable alteration of the relative amplitudes or latencies of the individual response components was caused by distraction, habituation, or variations in ambient noise. No significant effects were detectable in the corresponding EEG's. This preliminary investigation suggests that average evoked responses may be useful and sensitive indicators of CNS activity during aerospace flight.

MONITORING PSYCHOMOTOR RESPONSE TO STRESS BY EVOKED AUDITORY RESPONSES

I. INTRODUCTION

Often in certain aerospace flight, research, and clinical situations in which a human being is subjected to stresses which alter or impair the activity of his central nervous system (CNS), a sensitive measure of his psychomotor and CNS response is desired. The EEG and GSR (galvanic skin response) are the only externally recorded indicators of CNS activity which have received extensive investigation, and both are of limited usefulness as monitors of subtle changes. The GSR is a good indicator of the state of CNS arousal and alertness, but it is too nonspecific to differentiate between different stress factors altering one's state of alertness. The EEG, which Rosenblith (1) has called "the muffled polyneuronal roar that manages to make itself heard through man's thick skull," possesses a wide spectrum of biologic information, but it is notoriously difficult to interpret or even to recognize in the EEG the small variations that might be caused by a relatively minor variation in the activity of the brain. Various investigators have reported EEG changes during acceleration in jet aircraft (2) and in the human centrifuge (3), but these changes were clearly discernible only at high levels of acceleration and were not readily quantifiable.

Deliberately applied external sensory stimuli produce minuscule variations in the EEG called evoked responses. These are characterized by their amplitude, waveform, and latency (time from stimulus to occurrence of response). The specific components of the evoked response lend themselves readily to both quantitative, as well as qualitative, analysis.

The development of the technic of averaging evoked responses—pioneered by Dawson (4), Barlow (5), and Rosenblith (6)—has given access to a great wealth of information contained in the EEG. The possible utility of average evoked responses as a monitor of subtle responses to stress is apparent. They are simple to record, and the several discriminating features of which they are composed can be correlated with specific external events, such as stresses and psychomotor performance. In this study average evoked acoustic responses (AEAR's) to clicks were used to monitor the following stresses: hyperventilation, hypoxia, and acceleration.

There is some uncertainty regarding the origin of AEAR's. Geisler et al. (7) originally suggested that they are of cortical origin; however, Bickford et al. (8) contend that the early components of the AEAR represent the myogenic response to impulses traveling over a vestibulo-spinal-myogenic pathway. The validity of using AEAR's as a monitoring technic does not depend on an exact knowledge of the cerebral mechanisms causing them. In fact, the AEAR may be thought of as a signal which carries information about the state of a portion of the brain and possibly of the overlying musculature, and which varies according to the alterations of this state induced by stressful changes in the environment. Viewed in this way, the AEAR may be useful in monitoring human brain function and performance and, indirectly, in monitoring the environment.

II. SUMMARY

This study was undertaken to determine whether average evoked acoustic responses (AEAR's) might serve as a useful technic to monitor the psychomotor response and, in-

directly, the nervous system response to these stresses: hyperventilation, hypoxia, and $+G_z$ acceleration.

AEAR's evoked by clicks were recorded from five male subjects during quiescent control periods and during moderately symptomatic hyperventilation, mildly symptomatic hypoxia, and 2.5 $+G_z$ acceleration. During each stress, changes occurred in the amplitude, latency, and waveform of the AEAR's. These changes were consistently reproducible, clearly recognizable, and statistically significant. Control AEAR's taken during a four-hour period showed little variability. No diagnostically significant changes appeared in the corresponding EEG's.

The magnitude of the changes in the AEAR is correlated with the severity of the psychomotor response to the stress. Given that a subject is in one of these four psychomotor states—e.g., quiescent, hyperventilated, hypoxic, or acceleratory—it is possible to tell both the type and the severity of the subject's psychomotor state. A more complete catalog of responses to other sorts of stresses would clearly extend the usefulness of the AEAR as a monitoring technic.

The cause for these changes has not yet been determined. It is probable that the changes represent an alteration in the excitability and synchrony of the neuronal and muscular processes generating the AEAR.

It was anticipated that the chief disadvantages to the use of AEAR's in monitoring central nervous system activity would be: (1) the variability in AEAR's caused by changes in auditory input (extraneous noise and variation of effective stimulus intensity) and head position; and (2) habituation to prolonged stimulation. These problems appear to be negligible when stimuli are averaged for approximately 2 minutes for each AEAR. Since quite a considerable change in one's psychomotor state can occur in this time, shorter averaging periods must be used if rapidly occurring changes are to be monitored. Thus, an inevitable compromise must be established between the clarity of the signal extracted from the noise of the EEG, and the time necessary to extract it.

We have recorded AEAR's via telemetry from subjects flying in the SAM NF-100 jet fighter aircraft. Changes during hyperventilation and $+G_z$ acceleration are similar to those observed on the ground. In addition, distinct changes in the AEAR, EEG, and subjective auditory acuity were observed during weightlessness, simulated by free-falling parabolic flight.

III. TECHNIC

Five men, ages 25 to 31, with normal EEG's and auditory and vestibular function, were experimental subjects. Auditory stimuli consisted of rarefaction clicks of 0.5 msec. duration and 50 μsec. rise-time from a unijunction transistor oscillator. They were delivered bilaterally, at a frequency of 7.9/sec. and an intensity of approximately 50 db above subjective threshold, to a snug-fitting regulation Air Force headset and flying helmet (type H79/A1C). There was no significant stimulus artifact.

EEG electrodes constructed of stainless steel wire mesh set in nylon discs were filled with electrolyte paste and attached to the scalp with Eastman 9-10 cement. Interelectrode d.c. resistance was less than 3,000 ohms; microphonic electrode noise was less than 0.4 μv. rms. The reference electrode was placed at the vertex; exploring electrodes were placed over the temporal bone 2 cm. above the ear, and on the inion. The signal was amplified with a Wilson Greatbatch differential amplifier with a bandwidth of 0.1 to 5,000 cps, and common-mode noise rejection greater than 100 db. The amplified EEG, clicks, and voice were recorded on a Sangamo model 4700 tape recorder with wow and flutter compensation.

Averaging was done with a CAT model 400B special-purpose digital computer with an analysis time of 125 msec. and 0-delay. Averaging was done on-line and the AEAR's were

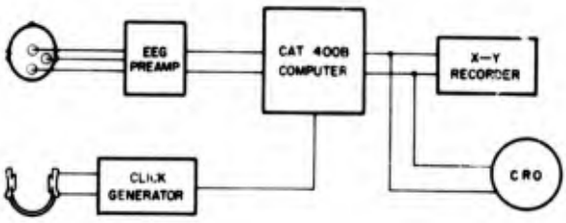


FIGURE 1

Block diagram of recording system.

plotted with an X-Y recorder with sensitivity set so that a true average was displayed (see fig. 1).

The CAT computes this sum:

$$A(t_i) = \sum_{j=1}^{N} V(t_{ij})$$

$i = 0, 1, 2 \ldots, 399.$
N = Number of clicks added.
$V(t_{ij})$ = Amplitude of the EEG in the "i^{th}" interval of the "j^{th}" click.

The X-Y recorder plots this average:

$$\frac{I}{N} A(t_i)$$

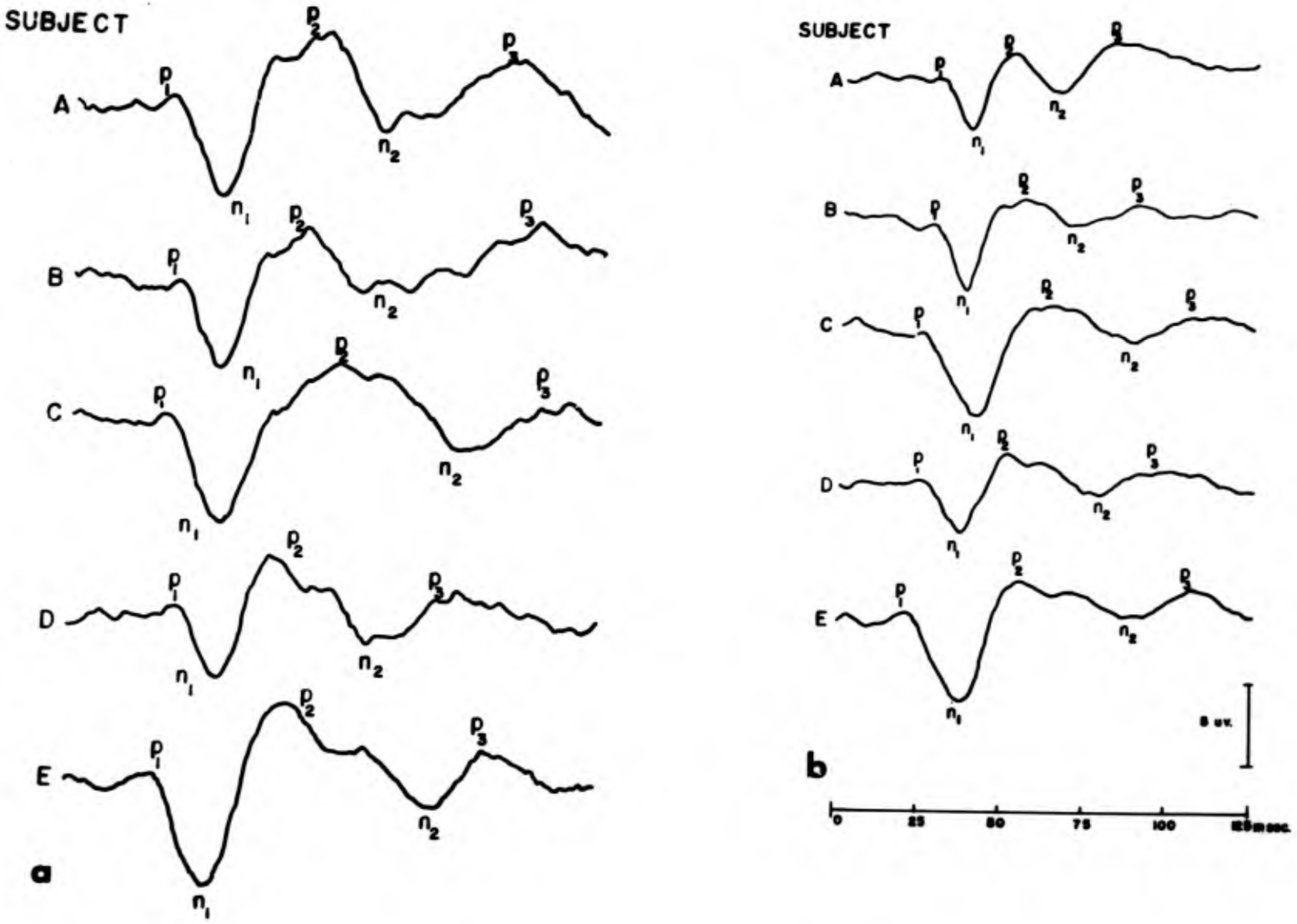
A typical experiment consisted of the following: While seated in an upright position, the subject listened to clicks during a 15-minute control period and then hyperventilated until symptoms of moderate severity developed (sweating, mild paresthesia, incoordination, trismus, and carpal stiffness). These usually appeared after 2 to 3 minutes and took 5 to 7 minutes to subside completely. Evoked response data were gathered during this mildly symptomatic period. When the AEAR returned to the prehyperventilation waveform, a 15-minute control period was observed with the subject breathing room air. Then the subject breathed a hypoxic mixture of 9.6% O_2 and 90.4% N_2. Each person was experienced in detecting his own early subjective symptoms of hypoxia. He was instructed to continue breathing the mixture until these symptoms were followed by the objective signs of hypoxia—i.e., constriction of peripheral visual field, decreased visual field, decreased visual acuity, and mild cyanosis of the oral mucosa and nailbeds. This symptomatic period typically lasted 6 to 7 minutes. After cessation of hypoxic symptoms, return of the AEAR to its original waveform, and a 15-minute control period, the subject entered the SAM human centrifuge and was exposed to 2.5 $+G_z$ acceleration for a total of 6 minutes. A final 15-minute control record was then obtained. No attempt was made to suppress the ambient noise level.

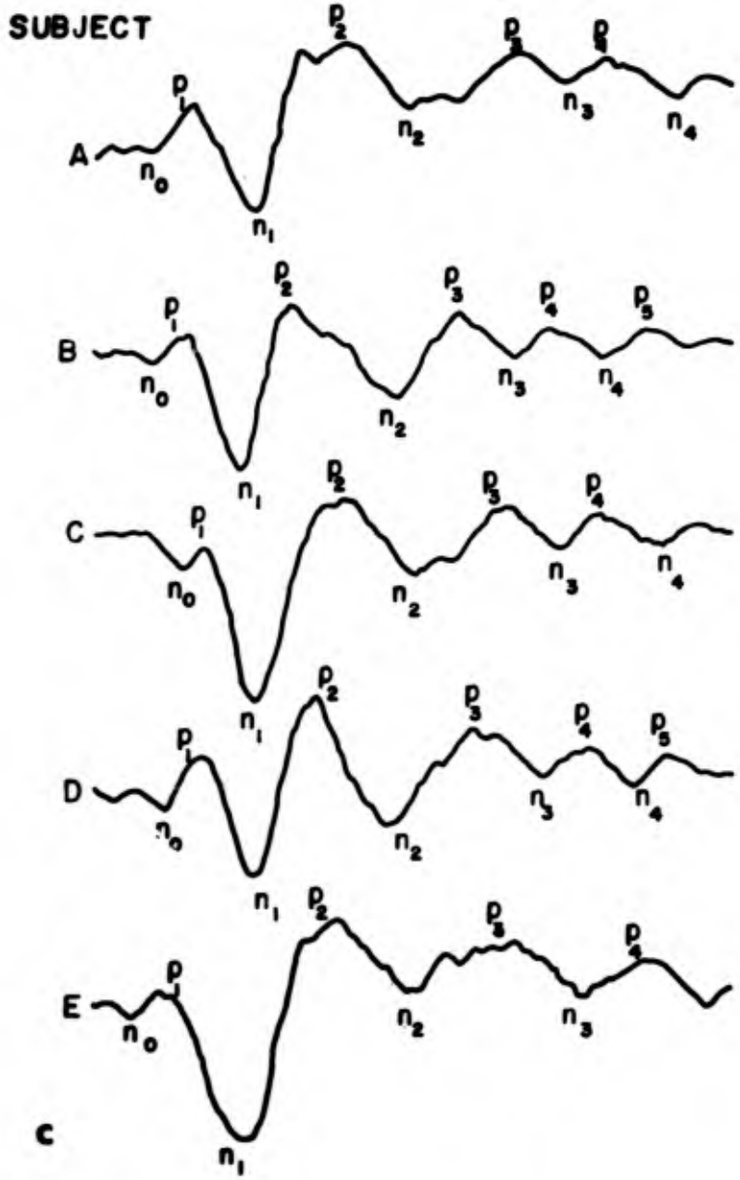
IV. RESULTS

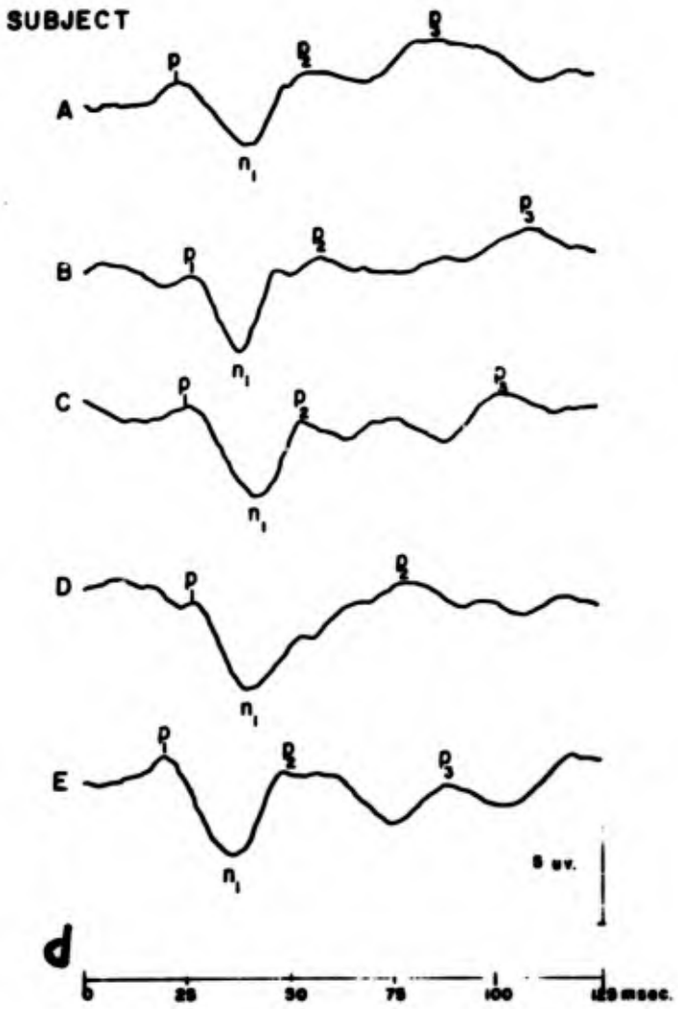
Each tracing in figures 2 and 3 represents the average of 1,000 clicks obtained during the last one-third of each experimental procedure. An upward deviation represents positivity of the occipital electrode with respect to the temporal electrode. The stimulus occurs at the beginning of each trace. In the control group several prominent components are labeled: p_1 = first positive peak; n_1 = first negative peak, etc. Each control response has several consistent features: an initial low-amplitude component, p_1, followed by a high amplitude negative component, n_1, with a latency of 30 to 35 msec.; a second positive component, p_2, with an amplitude consistently greater than p_1 and a latency that varies from 45 to 60 msec.; a second negative component, n_2; and a third positive component, p_3.

FIGURE 2

Each tracing represents the average evoked auditory response (AEAR) of the EEG of subjects A through E to 1,000 repetitive clicks. An upward deflection signifies positivity of the occipital electrode with respect to the temporal electrode. The clicks occur at the beginning of each tracing. Variations of the n_1 latencies and $p_2 - p_1$ amplitudes between the (a) control periods and the stress periods (b) hypoxia, (c) hyperventilation, and (d) acceleration are apparent.


SUBJECT
A
B
C
D
E
a
SUBJECT
A
B
C
D
E
b
0
25
50
75
100


SUBJECT
A
B
C
D
E
c


SUBJECT
A
B
C
D
E
5 uv.
d
0
25
50
75
100
125 msec.

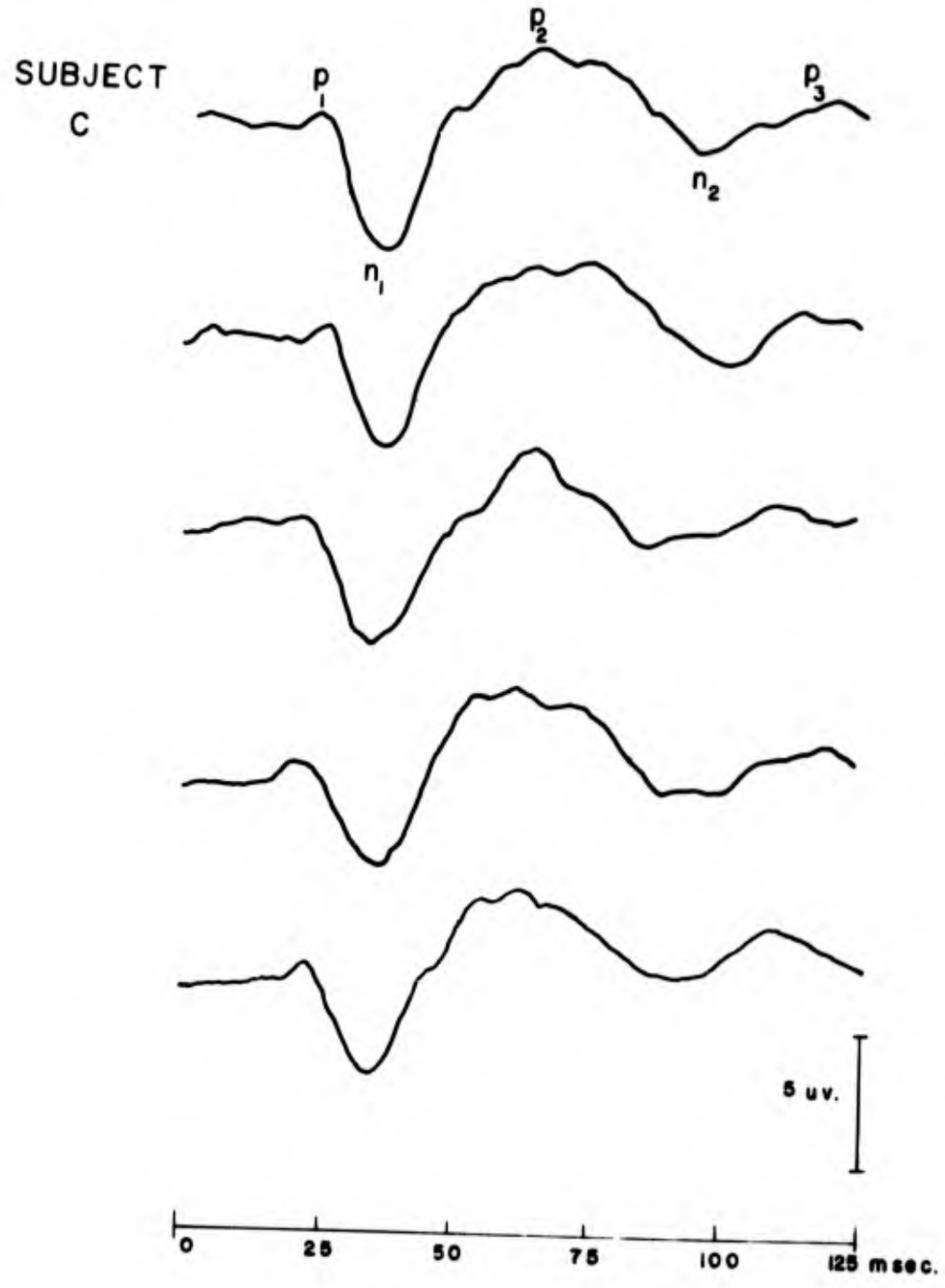


FIGURE 3

Each tracing is the average evoked auditory response of subject "C" to 1,000 clicks. The top tracing was obtained at time "zero" and each tracing below was obtained at successive hourly intervals. There is very little variation in the AEAR seen after 4 hours.

In the hyperventilation group the initial positive waves, p_1, are preceded in each case by a low amplitude negative wave, n_0. The latencies of n_1 have decreased to 28 to 30 msec., and the p_2 waves are followed by a number of distinct waves of decreasing amplitude.

In the hypoxic group the initial negative waves, n_0, seen in the hyperventilation group are of greatly diminished amplitude, and the p_1 waves are of less amplitude than those in the control group. The latencies of the n_1 waves have increased to 40 to 45 msec.; the amplitudes of the p_2 waves have decreased; and the same general configuration of the n_2 and p_3 waves seen in the control group is again present.

In the acceleration group no initial n_0 waves are present, the p_1 and p_2 waves have been remarkably reduced in amplitude, the latencies of the n_1 waves are from 35 to 42 msec., and there are no consistently occurring components after p_2. Moreover, the p_2 waves have a flattened appearance.

V. DISCUSSION

To promote mutual understanding, we shall define the AEAR as the average voltage variation in the EEG occurring during the intervals between successive clicks. This definition includes the entirety of each waveform shown in figures 2 and 3 and does not designate any specific complex.

A three-way analysis of variances was performed on each group of data shown in figure 2, with the experimental stress used as the treatment variable (see tables I and II). There was no statistically significant difference between the variances in the latencies of n_1, or of the amplitudes of p_2-p_1 in the four control trials of each of the five subjects. Likewise, there was no statistically significant difference in either of these two measurements between the records in the hyperventilation group, which included two trials on each subject. There was a significant decrease in the latency of n_1 ($P < .001$) and an increase in the amplitudes of $p_2 - p_1$ ($P < .001$) in the hyperventilation group as compared with the control group. In the hyperventilation group the after-potentials closely resemble a form of damped oscillatory activity, suggesting that one effect of hyperventilation is to alter the system "damping coefficient."

TABLE I

Mean values of the latencies of the n_1 peaks and the amplitudes of $p_2 - p_1$ for measurements made on the five subjects during the experimental periods

Stress	Number of measurements	Mean latency of n_1 (msec.)	Mean amplitude of p_2-p_1 (μv.)
Control	20	33.8	6.5
Hyperventilation	10	30.5	5.1
Hypoxia	5	37.9	3.8
Acceleration	55	38.1	0.34

There was no statistically significant variation ($P < .05$) between the latencies of the n_1 peaks or of the p_2-p_1 amplitudes of the four control measurements of each of the five subjects.

TABLE II

Significance level of the difference observed between the mean n_1 latencies and the mean $p_2 - p_1$ amplitudes of the control group as compared with the experimental stress groups

Comparison of stress groups	Latency of n_1	Amplitude of p_2-p_1
Control vs. hyperventilation	$P < .001$	$P < .020$
Control vs. hypoxia	$P < .001$	$P < .001$
Control vs. acceleration	$P < .001$	$P < .001$

For a monitoring technic to accurately reflect a subject's psychomotor response to a given stress, several criteria must be met. The output of the monitoring device should be time-invariant when no stress is applied. Changes in the output associated with a given stress ideally should be reproducible, easily recognized,

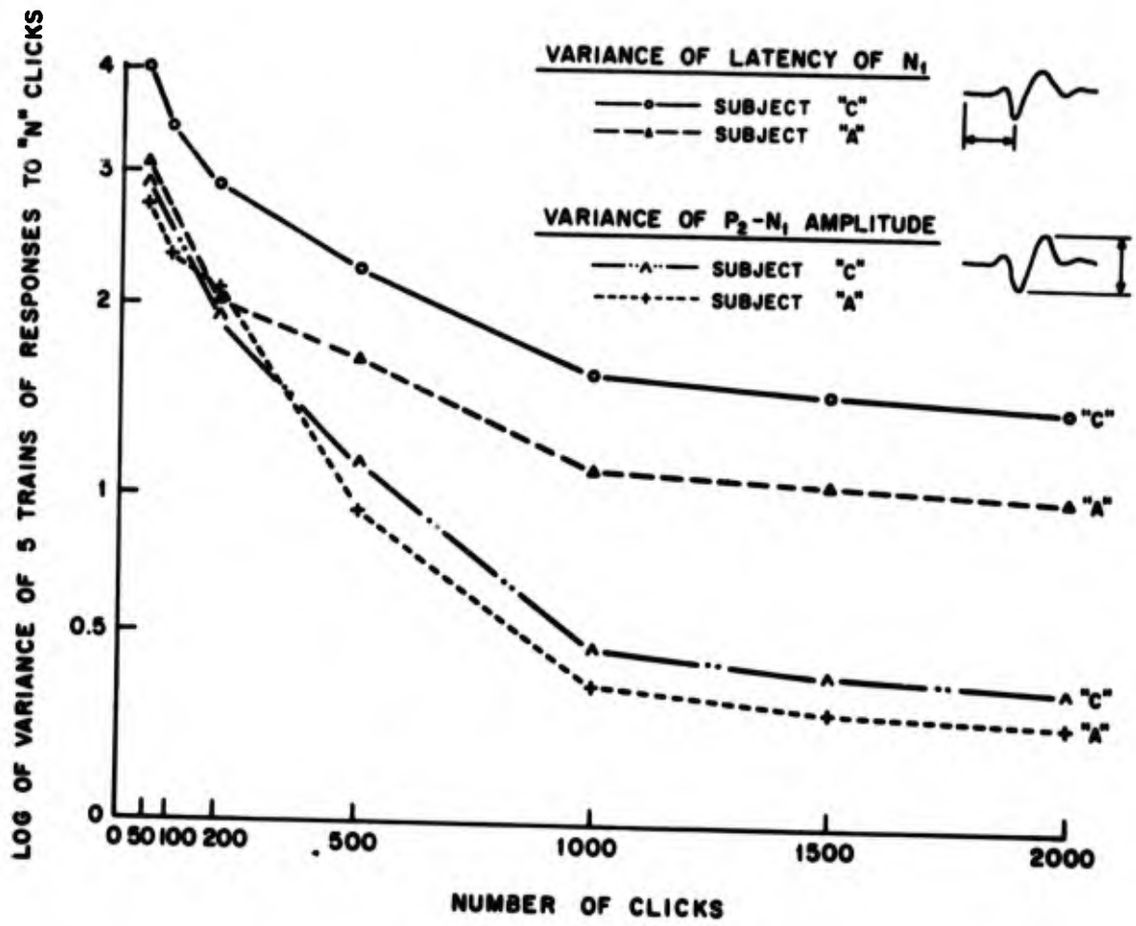


FIGURE 4

This graph demonstrates that the log of the variance of the n_1 latency and of the p_2-n_1 amplitude of subjects "C" and "A" diminishes as the number of clicks included in the average increases. The variances were computed from five separate trials of "n" clicks each. For "n" greater than 1,000 the log of the variance diminishes slowly.

sensitive, and free of artifact. Moveover, the records obtained for similar psychomotor states should show minimal variability.

Each subject's AEAR did vary during similar treatment periods, depending on electrode position, stimulus rate and intensity, and the number of stimuli averaged. Variability was minimized when the AEAR's were recorded with the vertex-occiput-inion electrode configuration, and as increasingly more stimuli were averaged. Figure 4 shows that the variance of the control AEAR's of subjects "A" and "C" approached a minimum value which remained sensibly constant for 1,000 clicks and more. Stated precisely:

$$\sigma^2(f) = \lim_{N\to\infty} \sum_{i=1}^{N} \frac{(x_i - \bar{x})^2}{N-1}$$

$\bar{x}$ = Mean value of component.
x_i = "i^{th}" value of that component.
N = Number of stimuli averaged.

Because the subject's state might vary during the presentation of a long train of clicks, the number of clicks averaged was limited to 1,000.

At a stimulus frequency of 7.9 clicks/sec., this yielded an averaging time of 126.5 seconds.

In sixteen experiments on subject "C," listening to clicks continuously for four hours produced only a slight decrease in the amplitudes of p_1 and p_2 and no significant change in the latency of n_1 (fig. 3). This is in contradistinction to the finding by Hernandez-Peon and Jouvet (9) that the AEAR in cats diminishes in time, supposedly because of an habituation effect caused by interruption of sensory transmission in the reticular formation.

Distraction caused by purposefully concentrating on a visual task (reading) or on an auditory task (conversation or music) produced no significant changes, whereas increasing the ambient noise diminished the relative amplitudes of each component but did not alter the latencies during any 1,000-click interval. These findings, while not conclusive, suggest that the AEAR is stationary for use in monitoring for periods up to four hours.

It is enlightening to compare the alteration in the AEAR during hyperventilation with the EEG, which showed no noticeable change (fig. 5). According to Dawson and Greville (10), "there is no evidence that the intensity of symptoms caused by hyperventilation is in any way correlated with any feature of the EEG." The severity of symptoms is correlated with the degree of hypocarbia caused by hyperventilation, but the CO_2 tension and pH of active cerebral cortex vary independently of changes in the arterial blood (10, p. 162). Hence, the EEG is an unsatisfactory indicator of the presence of symptoms or of impaired psychomotor performance caused by hyperventilation, whereas the AEAR does seem to be a satisfactory indicator of such changes.

In the hypoxia group there is a significant increase in the latencies of n_1 ($P < .001$) and a significant decrease in the amplitudes of $p_2 - p_1$ ($P < .005$). Again, it is interesting to note that there are no perceptible changes in the corresponding hypoxic EEG records. This may be a consequence of the mildness of the hypoxia produced in this study; for frequency analyses of the EEG have shown that hypoxia tends to produce slow waves whose amplitudes diminished with increasing hypoxia.

The acceleration records show a significant increase in the latencies of n_1 ($P < .001$) and a decrease in the amplitudes of $p_2 - p_1$ ($P < .001$) as compared with the control group, whereas a comparison between the hypoxia and acceleration groups shows that there is a small significant difference ($P < .05$) between the $p_2 - p_1$ amplitudes and no significant difference between the n_1 latencies. The similarity between acceleration and hypoxic AEAR's suggests that acceleration might produce hypoxia of that portion of the auditory system responsible for the AEAR. There was no subjective decrease in hearing acuity (subjective intensity of clicks) of any of the subjects during either the hypoxic or the acceleration trials; therefore, it appears that the AEAR is quite sensitive to changes that occur before any subjective symptoms develop. The apparent hypoxia in the auditory system during $+G_z$ acceleration may be produced by venous stagnation, by decreased cerebral perfusion secondary to a fall in head-level arterial pressure, and more probably, by a combination of the two.

Several possible sources of artifact deserve comment. The amplitude of the AEAR has been found to vary with head position, presumably because of variation of the myogenic contamination produced by tension on the neck muscles (11). Since a monitoring system should impose minimal restriction on the activity of the subject, we allowed the subjects to move their heads freely. The variability of the AEAR during forced hyperventilation, when the neck tends to flex and extend with respiration, and during acceleration, when the head tends to be compressed on the neck, was surprisingly little when averaging was performed for longer than 50 seconds. Extraneous noise, reduced though by no means abolished by the snug-fitting headset and helmet-assembly, remains a possible source of error. The intensity of the stimulus probably varies a small amount because of movements of the headsets relative to the tympanic membranes. Worden and coworkers (12) have shown in cats that the

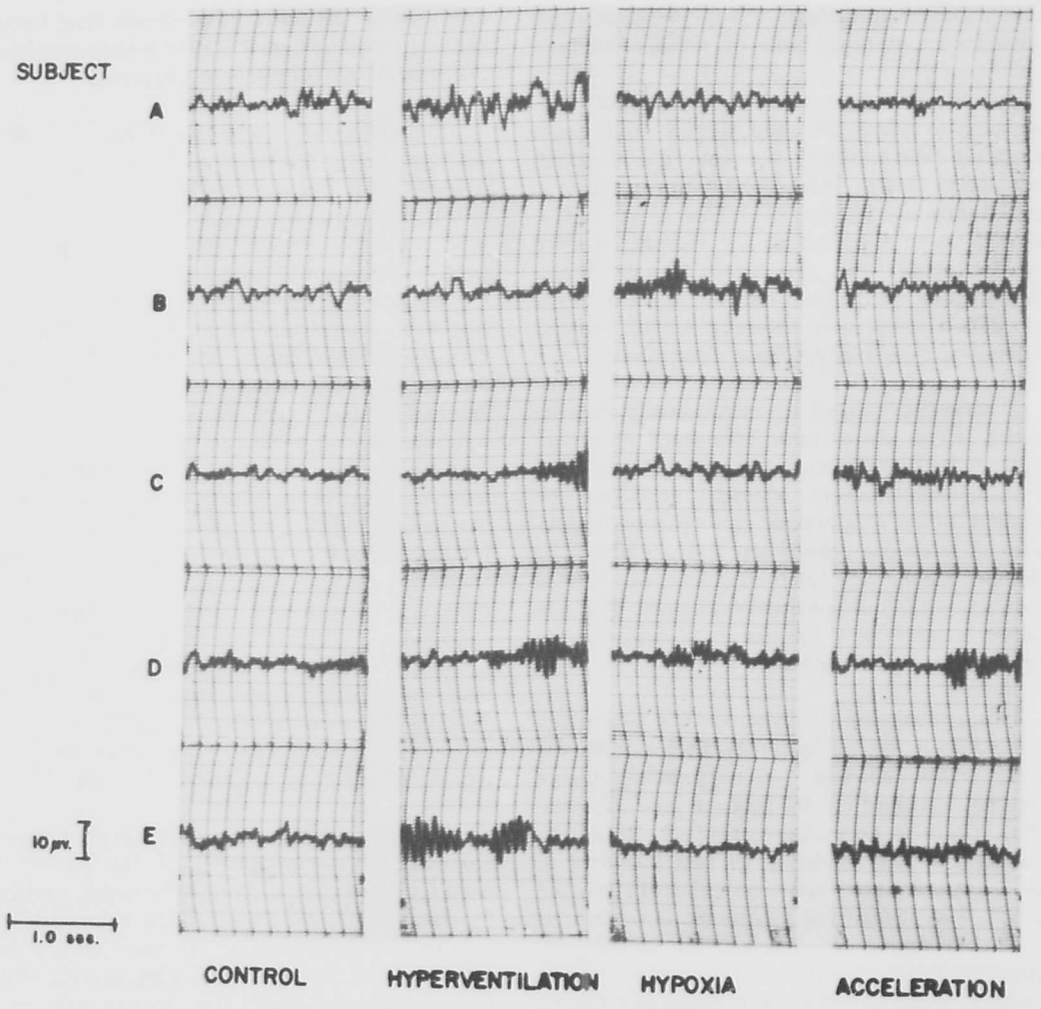


FIGURE 5

Representative EEG's of the subjects during the control and stress periods. No characteristic changes were noted in the EEG's during stress.

variability of the cortical AEAR due to such movement is minimal. Again, by averaging for sufficient length of time, variability of the AEAR caused either by extraneous noise or by movement of headsets was negligible.

Some artifact may be caused in the acceleration records by a change in the resistance and time constant of the electrode-scalp junction as a consequence of the acceleratory forces acting on them. Should such changes occur, we would expect, at worst, only amplitude or phase distortion and no significant change in peak latency.

The centrifuge cab rotates approximately 87° during the onset of acceleration until it is aligned in the plane of rotation of the centrifuge arm. It was anticipated that this small, angular acceleration component normal to the

positive G_z vector might induce a vestibular response interfering with the AEAR. To test this question, the rate of onset of acceleration was varied so that the rate of cab alignment varied from 1° to 40° per second, yet no significant differences appeared in the AEAR.

None of these possible sources of artifact seems to preclude the use of average evoked responses in measuring psychomotor response to stress; in fact, AEAR's give more information about psychomotor response than either the EEG or the GSR.

REFERENCES

1. Rosenblith, W. A. *In* Processing neuroelectric data, by Communications Biophysics Group of Research Laboratory of Electronics, and W. M. Seibert. Technical Report 351, MIT Press, 1959.
2. Sem-Jacobsen, C. W. Electroencephalographic study of pilot stresses in flight. Aerospace Med. 30:797-801 (1959).
3. Adey, W. R., J. D. French, R. T. Kado, D. F. Lindsley, D. O. Walter, R. Wendt, and W. D. Winters. EEG records from cortical and deep brain structures during centrifugal and vibrational accelerations in cats and monkeys. IRE Trans. Biomed. Electronics 8:182-188 (1961).
4. Dawson, G. D. A summation technique for the detection of small evoked potentials. Electroenceph. Clin. Neurophysiol. 6:65-84 (1954).
5. Barlow, J. S. An electronic method for detecting evoked responses of the brain and for reproducing their average waveforms. Electroenceph. Clin. Neurophysiol. 9:340-343 (1957).
6. Rosenblith, W. A. Some quantifiable aspects of the electrical activity of the nervous system (with emphasis upon responses to sensory stimuli). Rev. Mod. Phys. 31:532-545 (1959).
7. Geisler, C. D., L. S. Frishkopf, and W. A. Rosenblith. Extracranial responses to acoustic clicks in man. Science 128:1210-1211 (1958).
8. Bickford, R. G., J. L. Jacobson, D. Thane, and R. Cody. Nature of average evoked potentials to sound and other stimuli in man. *In* Katzman, R. (ed.). Sensory evoked response in man. Ann. N. Y. Acad. Sci. 112:204-224 (1964).
9. Hernandez-Peon, R., and M. Jouvet. Mécanismes neurophysiologiques concernant l'habituation, l'attention et le conditionnement. Electroenceph. Clin. Neurophysiol. Suppl. 6, pp. 39-49 (1957).
10. Dawson, M. E., and G. D. Greville. *In* Hill, D., and G. Parr (eds.). Electroencephalography: A symposium on its various aspects, pp. 147-193. New York: Macmillan Co., 1963.
11. Geisler, C. D. Average responses to clicks in man recorded by scalp electrodes. Technical Report 380, MIT Press, 1960.
12. Worden, F. G., J. T. Marsh, F. D. Abraham, and J. B. Whittlesey. Variability of evoked auditory potentials and acoustic input control. Electroenceph. Clin. Neurophysiol. 17:524-531 (1964).

Unclassified

Security Classification

DOCUMENT CONTROL DATA - R&D

(Security classification of title, body of abstract and indexing annotation must be entered when the overall report is classified)

1. ORIGINATING ACTIVITY *(Corporate author)*

USAF School of Aerospace Medicine
Aerospace Medical Division (AFSC)
Brooks Air Force Base, Texas

2a. REPORT SECURITY CLASSIFICATION

Unclassified

2b GROUP

3. REPORT TITLE

MONITORING PSYCHOMOTOR RESPONSE TO STRESS BY EVOKED AUDITORY RESPONSES

4. DESCRIPTIVE NOTES *(Type of report and inclusive dates)*

5. AUTHOR(S) *(Last name, first name, initial)*

Freeman, J. A., Captain, USAF, MC

6. REPORT DATE	7a. TOTAL NO. OF PAGES	7b. NO. OF REFS
May 65	11	12

8a. CONTRACT OR GRANT NO.	9a. ORIGINATOR'S REPORT NUMBER(S)
b. PROJECT NO.	SAM-TR-65-42
c. Task No. 793003	9b. OTHER REPORT NO(S) *(Any other numbers that may be assigned this report)*
d.	

10. AVAILABILITY/LIMITATION NOTICES

Qualified requesters may obtain copies of this report from DDC.

11. SUPPLEMENTARY NOTES	12. SPONSORING MILITARY ACTIVITY
	USAF School of Aerospace Medicine Aerospace Medical Division (AFSC) Brooks Air Force Base, Texas

13. ABSTRACT

A sensitive central nervous system (CNS) monitoring technic that can be correlated with behavior and with changes in the surrounding environment during aerospace flight is desirable to the flight surgeon interested in the early detection of possible adverse effects of the flight on the subject, to the neurophysiologist concerned with basic cerebral mechanisms occurring during the unique conditions of space flight, and to the systems engineer interested in any redundant indirect measurement of environmental parameters which serve to enhance the total system reliability.

In this study, a special-purpose digital computer was used to obtain average EEG responses evoked from human subjects by repetitive, nondistracting clicks during sedentary activity, mildly symptomatic hyperventilation, hypoxia, and 2.5 $+G_z$ acceleration on the SAM human centrifuge and in an NF-100 aircraft.

The waveforms obtained were qualitatively distinct for each group. No appreciable alteration of the relative amplitudes or latencies of the individual response components was caused by distraction, habituation, or variations in ambient noise. No significant effects were detectable in the corresponding EEG's. This preliminary investigation suggests that average evoked responses may be useful and sensitive indicators of CNS activity during aerospace flight.

DD FORM 1 JAN 64 1473

Unclassified

Security Classification

Unclassified

Security Classification

14. KEY WORDS	LINK A		LINK B		LINK C	
	ROLE	WT	ROLE	WT	ROLE	WT
Auditory response to stress Central nervous system monitoring Psychomotor response to stress Flight stress						

INSTRUCTIONS

1. ORIGINATING ACTIVITY: Enter the name and address of the contractor, subcontractor, grantee, Department of Defense activity or other organization *(corporate author)* issuing the report.

2a. REPORT SECURITY CLASSIFICATION: Enter the overall security classification of the report. Indicate whether "Restricted Data" is included. Marking is to be in accordance with appropriate security regulations.

2b. GROUP: Automatic downgrading is specified in DoD Directive 5200.10 and Armed Forces Industrial Manual. Enter the group number. Also, when applicable, show that optional markings have been used for Group 3 and Group 4 as authorized.

3. REPORT TITLE: Enter the complete report title in all capital letters. Titles in all cases should be unclassified. If a meaningful title cannot be selected without classification, show title classification in all capitals in parenthesis immediately following the title.

4. DESCRIPTIVE NOTES: If appropriate, enter the type of report, e.g., interim, progress, summary, annual, or final. Give the inclusive dates when a specific reporting period is covered.

5. AUTHOR(S): Enter the name(s) of author(s) as shown on or in the report. Enter last name, first name, middle initial. If military, show rank and branch of service. The name of the principal author is an absolute minimum requirement.

6. REPORT DATE: Enter the date of the report as day, month, year; or month, year. If more than one date appears on the report, use date of publication.

7a. TOTAL NUMBER OF PAGES: The total page count should follow normal pagination procedures, i.e., enter the number of pages containing information.

7b. NUMBER OF REFERENCES: Enter the total number of references cited in the report.

8a. CONTRACT OR GRANT NUMBER: If appropriate, enter the applicable number of the contract or grant under which the report was written.

8b, 8c, & 8d. PROJECT NUMBER: Enter the appropriate military department identification, such as project number, subproject number, system numbers, task number, etc.

9a. ORIGINATOR'S REPORT NUMBER(S): Enter the official report number by which the document will be identified and controlled by the originating activity. This number must be unique to this report.

9b. OTHER REPORT NUMBER(S): If the report has been assigned any other report numbers *(either by the originator or by the sponsor)*, also enter this number(s).

10. AVAILABILITY/LIMITATION NOTICES: Enter any limitations on further dissemination of the report, other than those imposed by security classification, using standard statements such as:

(1) "Qualified requesters may obtain copies of this report from DDC."

(2) "Foreign announcement and dissemination of this report by DDC is not authorized."

(3) "U. S. Government agencies may obtain copies of this report directly from DDC. Other qualified DDC users shall request through

_____."

(4) "U. S. military agencies may obtain copies of this report directly from DDC. Other qualified users shall request through

_____."

(5) "All distribution of this report is controlled. Qualified DDC users shall request through

_____."

If the report has been furnished to the Office of Technical Services, Department of Commerce, for sale to the public, indicate this fact and enter the price, if known.

11. SUPPLEMENTARY NOTES: Use for additional explanatory notes.

12. SPONSORING MILITARY ACTIVITY: Enter the name of the departmental project office or laboratory sponsoring *(paying for)* the research and development. Include address.

13. ABSTRACT: Enter an abstract giving a brief and factual summary of the document indicative of the report, even though it may also appear elsewhere in the body of the technical report. If additional space is required, a continuation sheet shall be attached.

It is highly desirable that the abstract of classified reports be unclassified. Each paragraph of the abstract shall end with an indication of the military security classification of the information in the paragraph, represented as *(TS), (S), (C), or (U)*.

There is no limitation on the length of the abstract. However, the suggested length is from 150 to 225 words.

14. KEY WORDS: Key words are technically meaningful terms or short phrases that characterize a report and may be used as index entries for cataloging the report. Key words must be selected so that no security classification is required. Identifiers, such as equipment model designation, trade name, military project code name, geographic location, may be used as key words but will be followed by an indication of technical context. The assignment of links, rules, and weights is optional.

Unclassified

Security Classification