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Human Tolerance to Prolonged Acceleration

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THE JET age of aircraft design and the rocket age which promises to follow have brought entirely new problems to aviation medicine. These aircraft, flying at supersonic speeds, require longer time periods to complete even simple maneuvers. The accompanying prolonged accelerations will create rapidly increasing demands on cardiovascular function and the nervous system. The problem of dealing with the ever growing demands on the human element within the weapons system assumes prime importance. One of two approaches must be selected. Man must either give way to electronic control systems, or the limits of his tolerance must be found and aircraft performance programmed to stay within his capabilities.

This study was undertaken to determine human tolerance to prolonged accelerations. A few examples will show how greater aircraft speed has increased cardiovascular and nervous

insults involved. At operational speeds prevalent during World War II, such as 300 mph, an aircraft could complete a 180° turn in fourteen seconds by pulling three *g*. Present day jet fighters traveling at 800 mph require thirty-eight seconds at three *g* to complete the same turn. It seems likely that aircraft in the foreseeable future will be capable of speeds of 2000 mph, or approximately Mach three. To make a 180° turn at this speed by pulling three *g* will take ninety-five seconds (Fig. 1).

Furthermore, a 180° turn at 2000 mph at three *g* requires fifty-two miles to complete. This may well mean that aircraft will be limited to a single interception pass or attack approach unless the radius of turn can be shortened. Figure 2 shows how the time required to complete a 180° turn at Mach three can be lessened by shortening the radius of turn, thereby pulling greater *g*. However, even at five *g* the time required is fifty-seven seconds, or approximately seven times that needed by World War II fighters,

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and two and one half times that of current aircraft similar g .

The solution offered by shortening the turn and increasing the g load is

signed to investigate man's tolerance to varying levels of g for prolonged periods. The general durations and g levels used were based on Figure 2.

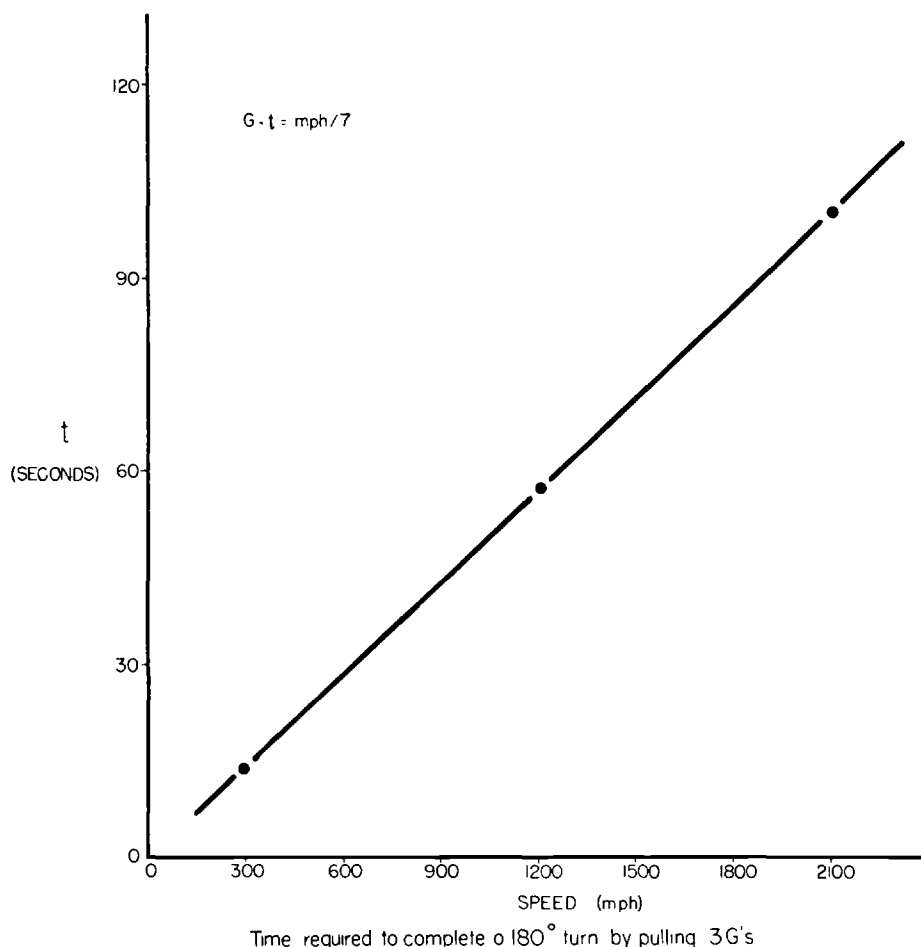


Fig. 1. The relationship between time required to complete a 180° turn and aircraft speed, the g load remains constant.

untried. While it might be feasible to endure three g for ninety-five seconds, five g for fifty-seven seconds might be completely intolerable. The present research program, therefore, was de-

APPROACH TO PROBLEM

To investigate man's tolerance to the stresses which will be imposed by the high speed aircraft of the future, a broad interdisciplinary approach is re-

quired. The principal focus was on cardiovascular changes and their effects under increasing g loads. The mere recording of changes in one sys-

system affected, or in another system, to compensate for the change. A state of fatigue or decompensation may be reached when changes are so severe

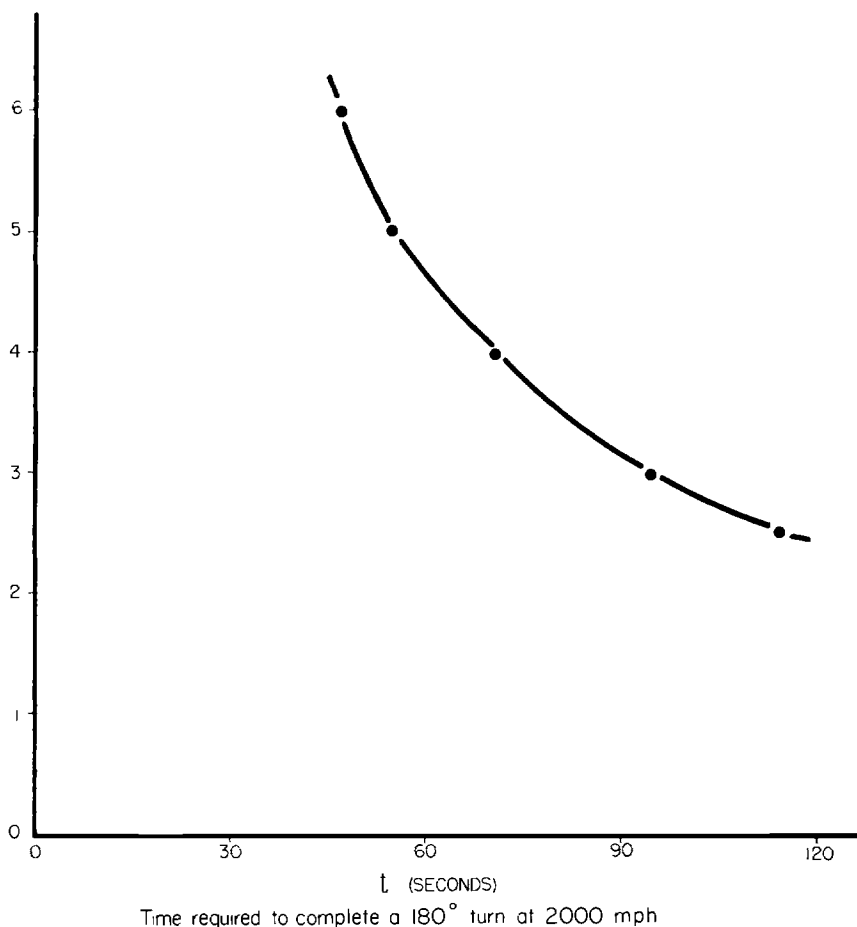


Fig. 2. Relationship between time required to complete a 180° turn at 2000 mph and the g load involved. Note that the turning time is lessened by shortening the radius and pulling greater g .

tem after the imposition of various stressors experimentally may fail to provide adequate information about the functioning of the total organism in an operational condition. Alterations in any system which upsets man's homeostasis require changes in the

that readjustments cannot be made by any system to overcome the effects of a stressor agent. For example, if a person is exposed to g forces there is a compensatory rise after an initial fall in blood pressure. However, the rise may not be adequate to keep the

cerebral blood flow at the level it was prior to the stress. This relatively small decrease in vascular supply does not, by itself, cause a decrease in cerebral functioning. Besides other cardiovascular compensatory changes, such as decreasing effective venous pressure, neurophysiologic changes may occur to help maintain cortical capacities. Physiologic and psychologic stimuli may bombard the reticular formation and cause an increase in level of arousal or altering of the person. The increased activation of the central nervous system may more than compensate for the decrease in blood and oxygen actually reaching the brain. One might anticipate that this would cause a progressive decrease in cortical functioning. Instead, more cortical units are activated so the person can maintain a relative state of alertness. However, if the vascular deficiency increases past a critical point, sudden decompensation may occur, and neurophysiologic activation can no longer adequately compensate for cardiovascular deficiencies.

We believe that failure to function in a stressful situation is not characterized by gradual deterioration due to progressive failure of any one system reflected by the results of measuring any one parameter. It is a sudden phenomenon representing the inability of the total organism to compensate for the effects of a stress imposed on it. To measure this multiple, simultaneous and continuous measures of several parameters are necessary.

PROFILE OF RUN

Using the hypothetical aircraft speed of Mach three, the g level and time

duration relationships were patterned after Figure 2. To obtain as much information as possible on physiologic tolerance and ability to perform useful tasks, the duration of the runs was increased by approximately 10 per cent over the amount shown in the figure. In this way a margin of error would be introduced, and more points would be covered on the curve below the point selected.

Blackout level was determined while wearing the standard Air Force g suit. Each subject underwent a series of nine runs, distributed in random order, consisting of three runs at 2.5 g for 115 seconds each, three at 4 g for eighty seconds each, and three at 0.4 g below his protected blackout level. These were denoted X G, and the duration of these runs was calculated for each specific case.

The physiologic effects of prolonged high gravitational stress were studied by three means. The pulse rate and cardiac integrity were followed by electrocardiography, utilizing the three standard limb leads. Arterial blood pressure was recorded by the use of an indirect method developed in this laboratory.⁶ Visual symptoms were observed by the subject's responses to the usual white center lights on a panel before him, and the status of his vision was recorded on a pen-writer.

The galvanic skin response was selected as a bioelectric measure,³ and index of vascular and nervous reactivity to psychologic stimuli and gravitational stress.

The use of a multidisciplinary approach was intended to furnish a many-sided picture of man's responses to prolonged stress. It was hoped that

psychologic testing and observations would reveal something of his ability to function usefully under these conditions.

Three types of psychologic data were collected. The subject reported his subjective feelings and attitudes before, during, and after each run. His performance was assayed by two methods. First, he continuously performed a "tracking" test, i.e., attempting to keep "zeroed" by means of a wrist control stick two dials into which were fed asynchronous sine waves. Second, at intervals a light was displayed and he was instructed to cut this off only when both tracking needles were on zero. Because it was important to know how well a subject could judge his own capabilities in the face of severe stress, he was asked to estimate his own level of performance following each run. This judgment was then compared with actual performance to determine if vascular or psychologic decompensation were accompanied by deterioration of judgment.

RESULTS

Visual Symptoms.—Because each subject was wearing an anti- g suit, his protected blackout level was determined immediately prior to the prolonged g runs. His peak g level was then determined by running him at 0.4 g lower than blackout. A central observer maintained contact with the subject by means of direct vision and the light performance task. The five subjects studied reported some degree of visual loss at higher g levels, i.e. at X g . Three reported slight dimming of vision at 4 g , and one noted slight dimming late in one 2.5 g ride.

In general, all noted the onset of visual loss after the run was well under way. In this respect, it differs from the usual blackout which characteristically begins after about six seconds at g . The mechanism responsible may be the same in both cases however, for under these circumstances we are dealing with what is probably borderline oxygen supply. The prolonged exposure may well cut into oxygen reserves, with resulting visual failure.

Blood Pressure and Pulse Rate.—Arterial blood pressure was recorded at twenty-second intervals by an indirect technique.⁶ This gave a minimum of three determinations during each prolonged run. All pressures were taken at heart level and show very similar characteristics. The vascular responses of the five subjects studied may be summarized as follows: The response to increasing prolonged gravitational stress is graded, i.e., higher g plateaus are accompanied by higher systolic and diastolic levels to overcome the increasing head of hydrostatic pressure in the arterial tree. The pressures tend to rise slightly during the course of the run, so that pressures late in the ride averaged from 10 to 15 mm. Hg. higher in both systolic and diastolic components than earlier determinations. This also suggests that fatigue of vascular smooth muscle probably does not play a role in the development of visual symptoms at high g . Pulse pressures tend to remain fairly constant for any one individual, although actual systolic and diastolic levels shifted as noted above.

Changes in pulse rate showed considerably variability from one subject

to another. In general, a graded cardiac acceleration was noted, and tachycardia was more pronounced at higher *g* levels. This, of course, is to be expected from the demands for increasing cardiac output.

Electrocardiogram.—All five subjects were monitored electrocardiographically during pre-run controls, the runs themselves, and post-run controls. The three standard leads were used and all conclusions stated are based on these records. Because of the nature of the arrhythmias observed, the EKG findings on each subject will be summarized.

Subject 1 (S. D. L.).—No significant change other than slight increase in pulse rate from eighty-five to ninety-five during three rides at 2.5 *g* for 115 seconds each. Three runs at 4 *g* for eighty seconds each were accompanied by an increase in rate from a control level of ninety to an average of 140 during the runs. Low voltage throughout, and flattening of T waves in standard leads one and two was noted. It was felt that this might be attributed to the tachycardia. Two runs at 4.6 *g* for eighty-two seconds each constituted his runs at X *g*. The third run was omitted because of a complaint of substernal pain radiating down the left arm. Electrocardiographic records revealed exactly the same changes seen at 4 *g*. No evidence for coronary insufficiency was obtained. Repeated EKG at intervals following the run, including a Master's test, failed to show any abnormalities.

Subject 2 (D. A. R.).—Two runs at 2.5 *g* for 115 seconds each failed to show any significant change other than a slight increase in rate. Two runs at 4 *g* for eighty seconds each showed an increase in pulse rate from a control of 80 to an average of 105. Sinus arrhythmia and lowered T waves were noted. An occasional premature ventricular contraction followed by a compensatory pause was noted in the second run. Two

runs at 5.4 *g* for sixty-three seconds each made up this subjects X *g* exposure. The first run showed a tachycardia of 130 and sinus arrhythmia or vagal stimulation. Frequent interpolated premature ventricular contractions were noted, as was evidence of early pulsus bigeminy emanating from multiple foci and showing no compensatory pauses. One configuration suggested either auricular or nodal premature ventricular contractions. The second run was aborted after five seconds at 5.4 *g*. Multiple premature ventricular contractions were noted and ventricular fibrillation was feared. The rhythm was irregular and auricular fibrillation was probably transiently present. Upon stopping the run the rhythm immediately reverted, and the EKG became normal. Post-run EKG's and a later Master's test revealed no abnormalities.

Subject 3 (J. B.).—Three runs at 2.5 *g* for 115 seconds each were uneventful except for one nodal premature contraction. A slight sinus tachycardia was noted. Three runs at 4 *g* for eighty seconds each had varied EKG tracings. One run was insignificant except for a tachycardia of 120, from control levels of ninety. A second run showed one nodal premature contraction with shortened PR interval and low voltage T waves. A third run showed frequent premature contractions of nodal or auricular origin. These were accompanied by shortened PR intervals, and were most marked near the end of the run. Three runs at 5 *g* for sixty-seven seconds each constituted this subject's X *g* exposure. A sinus tachycardia of from 120 to 130 was seen in all three but, except for low T wave voltage, no abnormalities were present.

Subject 4 (G. A.).—Three runs at 2.5 *g* for 115 seconds each showed a pulse rate increase from controls of seventy-six to an average of 100. Except for flattening of the T waves, possibly accompanying the tachycardia, no abnormality was seen. Three runs at 4 *g* for eighty seconds each failed to reveal any significant changes other than flattening of T waves and a pulse rate increase to an average of 115. Two runs at 5 *g* for sixty-seven seconds showed only a pulse rate increase to about 115 and flat-

tened T waves. A third run showed two interpolated premature ventricular contractions. Post run EKG's and a later Master's test revealed no electrocardiographic evidence of myocardial abnormalities.

Subject 5 (L. V.).—Three 2.5 *g* runs for 115 seconds each showed sinus arrhythmia, a pulse rate increase from control averages of sixty to run averages of eighty. The QRS and T waves were inverted in lead 2 and ST segments sagging here. The ST segments in leads one and three were elevated. Three runs at 4 *g* for eighty seconds each showed changes identical with those seen at 2.5 *g*, but of greater degree. The pulse rate averaged 100. Three runs at 5.6 *g* for sixty-one seconds each again showed the elevated ST segments in leads one and three and the reciprocal inversion of the QRS complex and T wave in lead two with sagging ST segments. The pulse rate averaged 100.

Following the last run while a control EKG was being taken the subject began auricular fibrillation. A tachycardia of 140 followed, and ten minutes later a sinus bradycardia of forty developed. This was replaced by a sinus arrest for one beat and subsequent frequent premature ventricular contractions. The subject was hospitalized, and after four hours his rhythm reverted to normal. Two subsequent episodes of auricular fibrillation have occurred, one while performing very light activity and one during a Master's test. A complete cardiac examination failed to reveal any underlying cardiac abnormalities. The diagnosis was reported simply as paroxysmal auricular fibrillation and tachycardia of unknown etiology. The patient has been maintained on quinidine for three months since the incident without further difficulty.

DISCUSSION

Four of five subjects experienced cardiac arrhythmias at higher *g*, and the fifth complained of substernal pain without EKG abnormality. All of the four subjects developing arrhythmias showed increasing myocardial irritability. Three had premature ventricular contractions and one had premature

contractions of nodal or auricular origin. Two of the arrhythmias were felt to be serious in nature and the other two were certainly potentially serious, with the danger of ventricular tachycardia and fibrillation. We believe that it is likely that they differ only in degree. The two more serious arrhythmias occurred in the subject's experiencing the highest *g*, i.e., 5.6 and 5.4 *g*, respectively. The potentially serious arrhythmias occurred at 5.0 and 4 *g*. The one subject without EKG changes experienced substernal pain at 4.6 *g*, but no others complained of chest or arm pain or untoward discomfort. It is therefore impossible to know subjectively when cardiac arrhythmias are imminent.

Gauer³ investigated the electrocardiographic changes in 200 subjects during brief exposures to acceleration. He noted changes which may be summarized as showing flattening, disappearance or negative T waves in leads two and three; the ST segment is deflected upward to join the T wave, and a right ventricular preponderance was suggested. Some persons showed arrhythmias which were thought to be due to a downward displacement of the pacemaker. This was attributed to the sudden recovery of strong vagal tonus as centrifugal force was decreased, and arrhythmias occurred as the centrifuge was slowing. In contradistinction to these observations three of the four arrhythmias observed in the present study occurred during the sustained *g* period. The fourth subject showed marked ST segment changes during the run and the arrhythmias began immediately following.

It is likely that relative myocardial anoxia is responsible for the increased cardiac irritability in the present study. Two factors deserve mention here. First, a tremendous increase in cardiac work is demanded. Coronary flow which may well be adequate for normal exercise requirements might fall short under such extreme demands, and a type of high output failure result. Second, as the hydrostatic pressure head within the arterial tree increases, the left ventricle must often develop pressures of greater than 170 mm. Hg. diastolic even to open the aortic valves. Under these extreme loads cardiac output may be falling off, thereby aggravating the already embarrassed myocardial supply to a laboring heart. This observation has been confirmed in animals wearing anti *g* suits by Salzman.⁴ He noted that at high *g* levels the pulse pressure fell off very rapidly when aortic pressure at heart level rose to a level at which left ventricular pressure was barely sufficient to open the aortic valves. It is likely that cardiac output began to fall off even before this sudden complete decompensation.

Bioelectric Measures.—The skin resistance has long been known as an indicator of responsivity to psychologic and physiologic stimuli. Its use has been hindered by deficiencies in instrumentation and the lack of specificity of the results of recordings. However, recent improvements in techniques using skin resistance meters (GSR machines) and in instrumentation have permitted its use in a more discriminating manner.¹

Transient, rapid drops in resistance

can be obtained by applying specific GSR responses. Furthermore, spontaneous fluctuations similar to the above are also seen, and because they cannot be attributed to any specific discernible stimuli are called nonspecific responses. It has been found that the level of alertness of subjects (varying from sleep to panic) can be evaluated by relating the amplitude of specific responses to the frequency of nonspecific response.¹ The frequency of nonspecific responses seems to be related to the level of arousal or central nervous system excitability of subjects. The amplitude of specific responses seemingly reflects the perceptual acuity of the subject and his responsivity to specific, external stimuli.

The amplitude of specific responses and number of spontaneous responses increase as subjects go from drowsiness and relaxation to alertness. However, as subjects become more alerted and begin to approach hyperexcitability and panic, the amplitude of specific responses drops but the number of nonspecifics continue to rise or stay at a high level (Fig. 3 top).

Figure 3 (bottom) shows the results from this series of runs. At 2.5 *g* the amplitude of specifics increases, as does the number of spontaneous fluctuations, indicating the subject is alert but not aroused to a point where his behavior and performance would be disorganized and his perceptions distorted. At *g* the amplitude of specifics drops slightly while the number of nonspecifics is elevated. Thus the subjects are quite aroused perhaps to the point where some decrement in coordination and goal directed activity

might be anticipated. At $X g$ the amplitude of specifics drops sharply while the nonspecifics rise, indicative of considerable central nervous sys-

tem activation and implying a decrease in perceptive, integrative ability, and perhaps an inability to react to tasks in a meaningful and accurate manner. Note how, after this ride, the level of specifics rise and number of nonspecifics falls but remains higher than

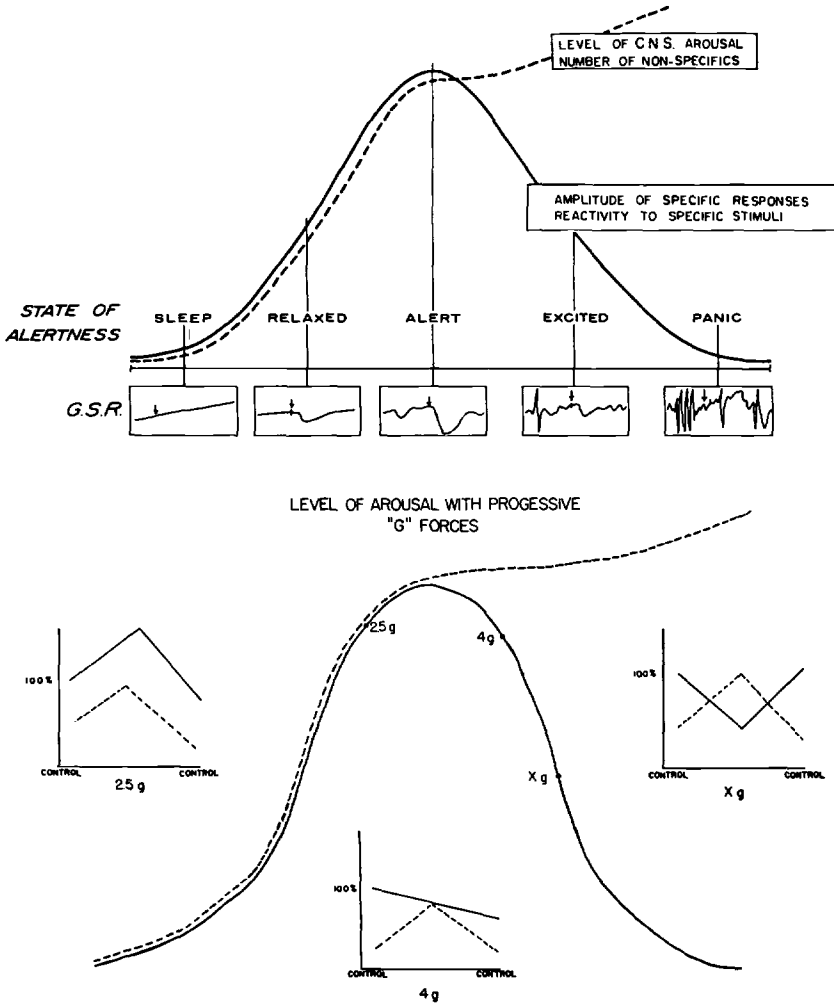


Fig. 3. (Above) Normal curve of state of alertness. Nonspecific responses (dotted) show level of central nervous system arousal. Specific responses (solid) show reactivity to specific stimuli. Boxes show GSR response on complete scale from sleep to panic. (Below) Points denote average position of subjects on alertness scale at 2.5 g, 4.0 g and X g respectively.

Solid lines indicate amplitude of specifics as per cent of control. Broken lines indicate number of nonspecifics per unit of time.

tem activation and implying a decrease in perceptive, integrative ability, and perhaps an inability to react to tasks

after runs at 2.5 and 4 g. This was interpreted to mean that following the 2.5 and 4 g runs the subjects returned

to a fairly relaxed state, while after the X g runs they remain in a fairly alerted condition. This again indicates the marked central nervous system activation occurring at X g.

as afford a correlation of physiologic and bioelectric changes with psychologic ones. Subjective awareness of decreasing physiologic capacity is notoriously poor in many situations and

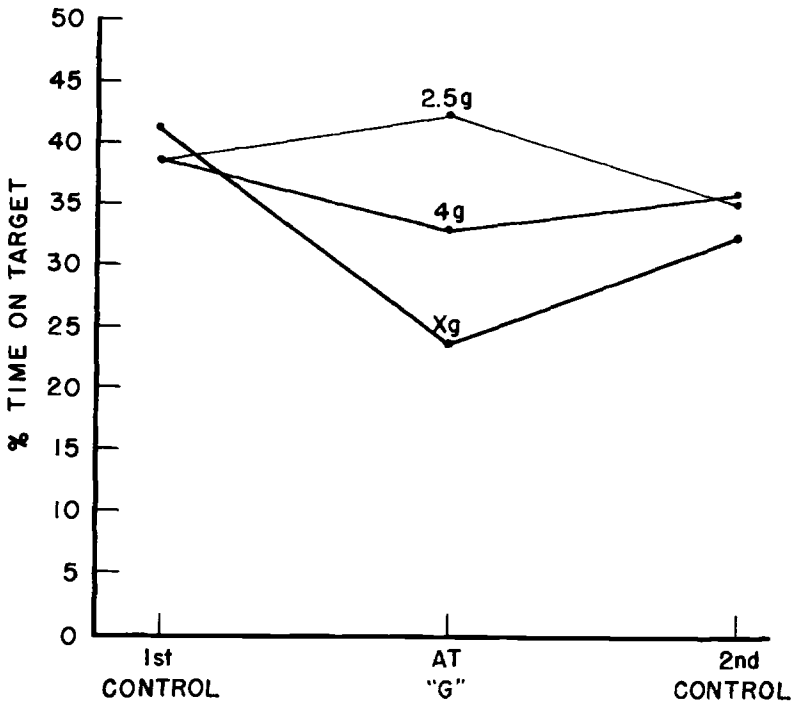


Fig. 4. Average performance of five subjects during pre-run control, period at g and post-run control respectively. Note slight increase in per cent time on target at 2.5 g, probably due to "alerting." Four g and X g shows progressive deterioration.

Psychologic Parameters.—Three areas were evaluated in an effort to get a picture of the subject's psychologic status: (1) Subjective report of feelings and attitudes before, during and after the runs; (2) performance during the runs; and (3) judgment of performance following each run.

It was thought that the information derived from these areas would help in judging the ability of the subjects to function at various g levels as well

may lead individuals, such as pilots, to make gross errors because of a failure to detect their decreasing capacities. Furthermore, it is of extreme importance in many flight operations that accurate judgments be given after stressful situations.

Figure 4 shows the time on target of the subjects at various g; after a slight increase at 2.5 g there is a progressive decrement of performance as the g load increases. The increase

at 2.5 *g* is compatible with previous observations that moderate anxiety, alerting or arousal will facilitate performance. The GSR response seemed to substantiate this impression. How-

of accuracy and length of time required to perform this task. This test differed from the tracking in that it was a periodic rather than continuous task. The results demonstrated that

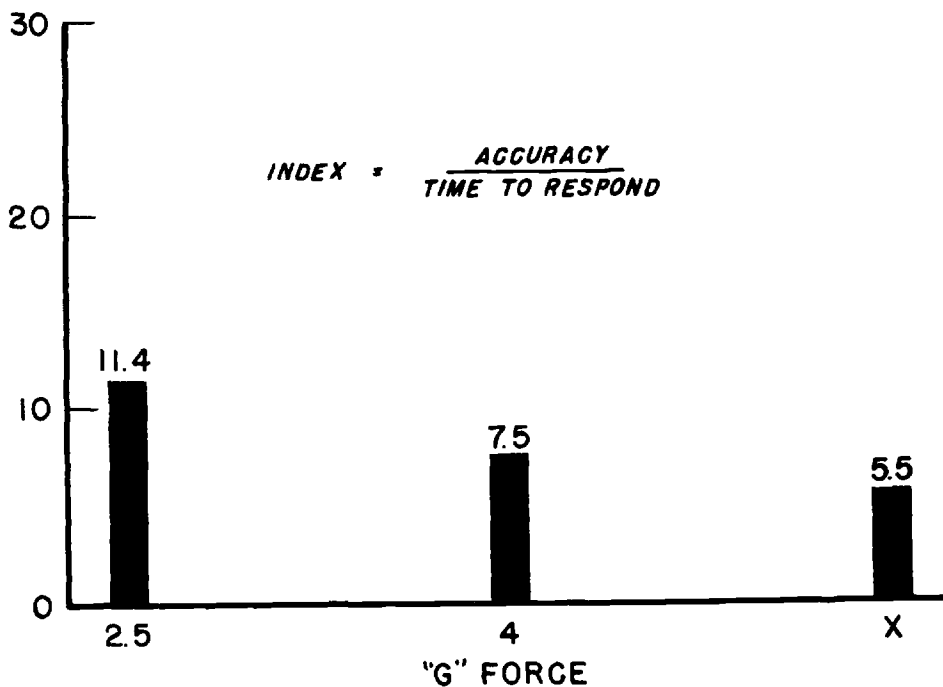


Fig. 5. Performance of trigger light task under increasing *g*. Note decrement of this discontinuous performance task at high *g* levels.

ever, past an optimal point, arousal may lead to disorganization of behavior and performance. One subject who showed marked cardiovascular changes at X *g* as well as marked arousal on the GSR, was on target only 4 per cent of the time during X *g*, having been on target 35 per cent at 2.5 *g* and 25 per cent at 4 *g*, thus indicating marked physiologic and psychologic decompensations.

Figure 5 shows the results of the trigger light task. The subject's performance is determined as a function

with increased *g* stress there is decreased ability to perform a discontinuous task accurately and quickly.

Subjective Reports.—All of the subjects reported many more symptoms and unpleasant feelings after the X *g* ride. In some, the discomfort was quite acute, and in contrast to rides at 2.5 and 4 *g*, the subjects felt so concerned they had difficulty focusing their attention on the performance tasks. Some stated they were quite anxious but the anxiety was not re-

lated to anything specific; it was diffuse and free-floating. This is a degree of psychologic arousal in which persons are unable to focus their attention on circumscribed tasks or respond effectively to tasks because of the extreme excitability of their central nervous system. This inability may of course, be due to physiologic or psychologic threats. Related work has shown that the level of anxiety correlates with biochemical and physiologic changes thus influencing the actual blackout level.²

It should again be noted that the subjects were not aware of the g loads to which they were being exposed, at any particular time, the rides being distributed by random.

Judgment.—The subject's judgment of his performance was given on a six-point scale after each run. If he thought his performance was as good as a "control" performance during a nonstressful time, he reported "1." However, if he felt his performance was extremely poor, he reported "6." A list of numbers and their meaning was kept in the cab so the subject could refer to it. The post-run judgments were fairly accurate at least as far as being able to compare performance at various g . Although marked physiologic and psychologic changes occurred during the X g run the subjects were able to judge their performance and discriminate differences between their performance at the various g levels. It should be pointed out that the subjects' responses may not have been integrative judgments of performance but rather judgments made from cues during the ride of the amount of

g stress imposed on them. Thus, the subjects would be describing their expected performances at various g rather than an independent judgment of the performance itself. However, this "cue" variable would imply the subjects were capable of making discriminations between the rides in a period following the imposition of stress, indicating that the capacity of intellectual functioning was not impaired. GSR findings using the arousal scale confirmed the observation of no excessive fatigue or hyperexcitability.

All subjects showed increasing stress response with increasing g loads in at least three areas: cardiovascular, performance, and bioelectric. However, there was no evidence of post-run fatigue or continued decompensation in these parameters. We believe then that X g was a very stressful situation and did produce considerable physiologic and psychologic difficulties which were reversible when the stressing stimulus was removed. Hence, the subject's judgment (a complex ability based on multiple psychophysiologic determinants) was maintained after the rides.

CONCLUSIONS

This report has described an experiment designed to evaluate man's tolerance to prolonged acceleration stress. The dramatic cardiovascular findings indicated the subjects were close to the limits of stress tolerance. The psychologic and bioelectric findings support this thesis. The findings are particularly interesting because some systems will maintain their over-all compensation at the expense of those which decompensate. During the max-

imal stress point in this experiment, all measured parameters showed decrements. We believe that increasing the stress exposure period or the intensity of the stress would have produced more prolonged decompensation, e.g., in the post-run periods. The results then indicated that man is approaching his psychophysiological limit using present protective devices. Future weapon systems must take this into account if man is to remain a precise computer who can function efficiently as part of this system.

SUMMARY

Using physiologic and psychologic measures, man's tolerance to graded prolonged accelerations was investigated. Dimming of vision occurred late in the higher g runs of all subjects despite the fact that they were protected by anti- g suits and running at 0.4 g below their predetermined blackout level. Blood pressures at heart level showed graded increases in both systolic and diastolic components under increasing g . Pulse pressure remained relatively constant. Four of five subjects showed arrhythmias at high g levels. This myocardial irritability may be attributed to a relative coronary insufficiency with maximum

coronary flow proving to be inadequate for a massive work load. High g loads produced excessive central nervous system excitability as reflected by skin resistance measures. This degree of excitability was not compatible with organized, goal-directed performance as demonstrated by decrements in continuous and discontinuous performance tasks. The higher levels of sustained g in this experiment approach man's physiologic and psychologic limits of tolerance.

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Sunset in the East

We have now reached the point in flight where we must consider the earth not merely as the good old "terra firma" of the ancients, but rather as a rotating planet. This is best demonstrated by the fact that if a craft flies in the middle latitudes in a westerly direction with supersonic speed, higher than the rotational speed found in this belt of the earth the sun will be left behind, and the passengers will encounter the strange spectacle of a "sunset in the East."—HUBERTUS STRUGHOLD: *International Record of Medicine and General Practice Clinics*, Sept. 1955.