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Spacecraft launch and re-entry: Effects of simulated +Gx acceleration on cardiorespiratory parameters

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ABSTRACT

Introduction: In the spaceflight, during launch and re-entry, the crew is exposed to acceleration ranging from +4Gx to +8Gx in nominal conditions. This study was conducted to assess the changes in cardiorespiratory parameters, namely, heart rate (HR), electrocardiogram (ECG), respiratory rate (RR), and SpO_2 on exposure to simulated +Gx acceleration.

Material and Methods: Fifteen randomly selected healthy male volunteers participated in the study. They were exposed to a simulated acceleration profile consisting of two peaks in the high-performance human centrifuge; first peak of +4Gx for 30 s and second peak of+8Gx for 30 s. The cardiorespiratory parameters were monitored and recorded during the acceleration exposure. The data were compiled and analyzed using one-way repeated measures ANOVA.

Results: Significant increase in HR was observed on exposure to +4Gx (110.4 ± 16.7 bpm; $P < 0.001$) in comparison to the baseline value (80.5 ± 7.5 bpm). However, the changes in the HR at +8Gx were not significant in comparison to baseline as well as +4Gx values. On the other hand, RR indicated a significant increase on exposure to +8Gx (25.2 ± 5.8 breaths/min) in comparison to the baseline (15.1 ± 1.6 breaths/min; $P = 0.001$) and +4Gx (19.0 ± 6.1 breaths/min; $P = 0.009$) values. SpO_2 showed a significant reduction at +8Gx (94.2 ± 3.8%) in comparison to baseline (98.9 ± 0.3%; $P = 0.004$) and +4Gx (96.9 ± 1.5%; $P = 0.003$). ECG did not show any evidence of arrhythmia during the exposure to +Gx acceleration.

Conclusion: The insignificant changes in the HR at peak of +8Gx indicate less pronounced effects on the smaller hydrostatic gradient in +Gx acceleration unlike +Gz acceleration. However, the findings of the study point towards a significant increase in respiratory rate and reduction in SpO_2 at +8Gx.

Keywords: +Gx acceleration, High-performance human centrifuge, Spacecraft launch and re-entry.

INTRODUCTION

Exposure to +Gx acceleration is a known entity experienced during spacecraft launch and re-entry.[1,2] Spacecraft such as Mercury and Gemini-Titan exposed the crew to a peak acceleration of +6Gx to +7Gx during launch[3,4] and up to a maximum of +11Gx in Mercury during re-entry.[5] The Russian spacecraft (Soyuz) exposes the crew to a peak acceleration of +4.3Gx during launch[6] and +4.2±0.1Gx during controlled automatic descent phase of re-entry. However, the transition to ballistic descent during re-entry exposes the crew to peak of +8Gx.[7] This necessitates indoctrination of space crew to simulated acceleration profiles experienced during launch and re-entry.

National Aeronautics and Space Administration (NASA) has specified standards to limit the exposure of +8Gx during re-entry for 30 s; exceeding which the risk of significant incapacitation increases.[8] The Russian Cosmonauts also receive systematic centrifuge training with exposure to +4Gx and +8Gx to facilitate the enhancement of G tolerance during actual spaceflight.[7] Understanding the physiological effects of exposure to +Gx acceleration under simulated condition are important in assessing tolerance of an individual and his functional capability during actual spaceflight. Rai and Gupta reported tachycardia, increased respiratory rate, and no significant arrhythmia in their study of exposure to +8Gx for 40 s on human centrifuge at Institute of Aerospace Medicine (IAM), Bengaluru, in 1984.[9]

The present study involved simulation of acceleration profiles in the high-performance human centrifuge (HPHC) likely to be encountered during the launch and re-entry, typically during nominal conditions. Physiological parameters, namely, heart rate (HR), respiratory rate (RR), oxygen saturation (SpO_2), and electrocardiogram (ECG) were monitored and analyzed to understand the effects of such +Gx exposures on important cardiorespiratory parameters mentioned above.

MATERIAL AND METHODS

Subjects

Fifteen randomly selected healthy male volunteers aged between 25 and 40 years participated in this study. The mean age, height, and weight were 32.1 ± 4.4 years, 173.4 ± 7.4 cm, and 74.6 ± 7.1 kg, respectively. None of them had any previous experience of +Gx acceleration. Participants were screened for their fitness to undergo centrifuge run. Subjects with any medical disability or history of cardiorespiratory illness were excluded from the study. A written informed consent was obtained from the volunteers before the study. The protocol was approved by the Institute Ethics Committee.

Equipment

The HPHC at the IAM, Indian Air Force manufactured by M/s AMST®, Austria, was used to simulate the acceleration profiles. Equivital Wireless Physiological Monitoring System (Equivital EQ02, Hidalgo, UK) was used to record the physiological parameters. Visual analog scale (VAS) was used for subjective assessment of discomfort/pain during the HPHC run.

Experimental protocol

The participants were advised to abstain from alcohol and to have adequate rest and sleep on the day before the experiment. On the day of experimentation, they reported to the department of Acceleration Physiology and Spatial Orientation (AP&SO) at 0800 h. After resting for 15 min, they were instrumented and their baseline physiological parameters were recorded. Thereafter, they were strapped up in the gondola of the HPHC and pre-run physiological parameters (HR, RR, and SpO_2) were recorded.

The HPHC was accelerated to baseline of +1.4Gz and thereafter to a peak of +4Gx at 0.1G/s and maintained for 30 s. Further, the acceleration stress was increased to +8Gx at 1G/s and sustained for 30 s. This was followed by descent from +8Gx at 1G/s. The simulated profile is depicted in Figure 1. HR, SpO_2, and RR were monitored and recorded throughout the exposure to +Gx acceleration and immediately after cessation of the run (post-run) till 5 min post-run to assess their recovery to baseline values. Blood pressure (BP) was only measured and compared at baseline and recovery levels. The subjective assessment of chest pain/discomfort was obtained using the VAS. The end points for HPHC run were (a) successful completion of +Gx profile, (b) evidence of sustained cardiac arrhythmias, (c) SpO_2 fall to <72%, and/or (d) voluntary termination due to subjective discomfort.

Statistical analysis

Normality of the data was confirmed using Shapiro–Wilk test. One-way repeated measures ANOVA was carried out to analyze the recorded HR, RR, and SpO_2 between baseline, pre-run, +4Gx, +8Gx, post-run, and recovery conditions. *Post hoc* analysis was carried out using Bonferroni test. The level of significance was kept at $P < 0.05$.

RESULTS

All the subjects could tolerate the exposure to +Gx acceleration. The mean HR, RR, and SpO_2 recorded during baseline, pre-run, +4Gx peak, +8Gx peak, post-run, and recovery conditions are shown in Table 1.

One-way repeated measures ANOVA showed that HR differed significantly across all the six conditions ($F = 14.297$, $P < 0.001$). However, *post hoc* analysis [Table 2] revealed that

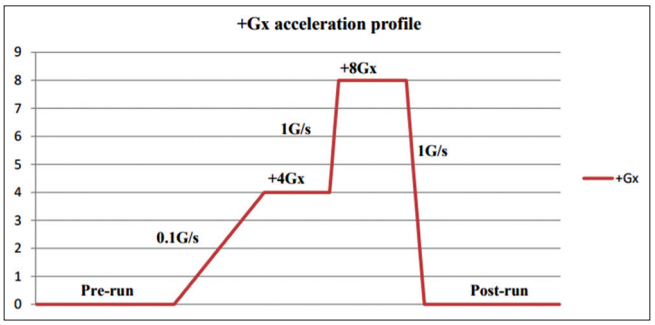

Figure 1: Simulated acceleration profile.

Table 1: Mean and SD values of HR, RR, and SpO_2 recorded at various stages of study protocol (n=15).

Variables	Baseline	Pre-run	+4Gx run	+8Gx run	Post-run	Recovery
HR (bpm)	80.5±7.5	102.9±20.5	110.4±16.7	97.3±19.9	99.3±16.3	78.4±7.9
RR (breaths/min)	15.1±1.6	16.6±4.4	19.0±6.1	25.2±5.9	16.8±3.5	15.7±1.6
SpO_2 (%)	98.9±0.3	98.7±1.0	96.9±1.5	94.2±3.8	96.6±2.3	98.7±0.6

HR: Hear rate, RR: Respiratory rate, SpO_2: Oxygen saturation

the mean HR recorded during pre-run was significantly more than the baseline HR (P = 0.008). Similarly, the HR showed a significant increase at +4Gx as compared to baseline values (P < 0.001). However, the HR recorded at +8Gx did not show any significant difference with that of baseline, +4Gx, and post-run conditions. The HR recorded post-run differed significantly from baseline (P = 0.01). The difference between mean HR recorded during recovery condition and baseline was not statistically significant (P = 1.00).

ECG of seven subjects showed sinus tachycardia during pre-run and +8Gx peak; 13 subjects showed sinus tachycardia at +4Gx peak. However, there was no evidence of any arrhythmia on exposure to peak +Gx acceleration. A paired t-test revealed that there was no significant difference in the systolic BP for baseline (125.1 ± 8.8 mmHg) and recovery (127.9 ± 9.3 mmHg) conditions (t = −0.833, P = 0.418). Similarly, no significant difference was observed in the diastolic BP for baseline (81.9 ± 7.1 mmHg) and recovery (81.9 ± 7.5 mmHg) conditions (t = 0.307, P = 0.762).

One-way repeated measures ANOVA revealed a significant difference in RR across the six conditions (F = 13.928, P < 0.001). Post hoc analysis [Table 3] revealed that the difference in the RR at baseline (15.1 ± 1.6 breaths/min) and +4Gx (19.0 ± 6.1 breaths/min) was not statistically significant (P = 0.58). However, the RR recorded at +8Gx (25.2 ± 5.9 breaths/min) increased significantly from baseline (P = 0.001) and +4Gx (P = 0.009). A significant decrement in RR (P = 0.001) was also observed between +8Gx and post-run RR (16.8 ± 3.5 breaths/min).

Mean SpO_2 differed significantly across the six conditions (F = 13.077, P < 0.001). Post hoc analysis [Table 4] revealed significant difference in the mean SpO2 between baseline and +4Gx (P = 0.003), +8Gx (P = 0.004), and post-run (P = 0.022), respectively. The mean post-run SpO_2 was not statistically significant from +8Gx (P = 0.939). Application of Pearson's correlation to the relation between RR and SpO_2 during exposure to +Gx acceleration revealed statistically significant strong negative correlation (r = −0.94, P = 0.006).

The subjective discomfort experienced during the +Gx run was obtained using VAS indicated highest score of 7 (pain in the right hypochondrium) and the most commonly reported score was 0 (no discomfort/pain) at +4Gx. On exposure to +8Gx, highest score reported was 8 (pain in the right hypochondrium) and the most common reported score was 4 (three experienced chest pain and one had upper backache).

Table 2: *Post hoc* analysis P values showing differences in mean HR recorded at various stages of study protocol.

	Baseline	Pre-run	+4Gx	+8Gx	Post-run	Recovery
Baseline	-	0.008	0.000	0.097	0.011	1.000
Pre-run	0.008	-	1.000	1.000	1.000	0.007
+4Gx	0.000	1.000	-	0.216	0.048	0.000
+8Gx	0.097	1.000	0.216	-	1.000	0.078
Post-run	0.011	1.000	0.048	1.000	-	0.002
Recovery	1.000	0.007	0.000	0.078	0.002	-

Post hoc analysis was carried out using Bonferroni test. Blue background denotes statistically significant difference. HR: Heart rate

Table 3: *Post hoc* analysis P values showing differences in mean RR recorded at various stages of study protocol.

	Baseline	Pre-run	+4Gx	+8Gx	Post-run	Recovery
Baseline	-	1.000	0.581	0.001	0.756	1.000
Pre-run	1.000	-	1.000	0.001	1.000	1.000
+4Gx	0.581	1.000	-	0.009	1.000	1.000
+8Gx	0.001	0.001	0.009	-	0.001	0.001
Post-run	0.756	1.000	1.000	0.001	-	1.000
Recovery	1.000	1.000	1.000	0.001	1.000	-

Post hoc analysis was carried out using Bonferroni test. Blue background denotes statistically significant difference. RR: Respiratory rate

Table 4: *Post hoc* analysis p values showing differences in mean SpO_2 recorded at various stages of study protocol.

	Baseline	Pre-run	+4Gx	+8Gx	Post-run	Recovery
Baseline	-	0.502	0.003	0.004	0.022	1.000
Pre-run	0.502	-	0.142	0.019	0.148	1.000
+4Gx	0.003	0.142	-	0.088	1.000	0.011
+8Gx	0.004	0.019	0.088	-	0.939	0.005
Post-run	0.022	0.148	1.000	0.939	-	0.050
Recovery	1.000	1.000	0.011	0.005	0.050	-

Post hoc analysis was carried out using Bonferroni test. Blue background denotes statistically significant difference. SpO_2: Oxygen saturation

DISCUSSION

Spaceflight exposes the crew to acceleration forces of approximately +3Gx to +4Gx acceleration during nominal launch and re-entry.[6,7,10] However, a ballistic descent or an abort could result in acceleration as high as +7Gx to +8 Gx.[7,11] Therefore, the acceleration profiles used in the present study included peaks of +4x and +8Gx. The selection of maximum of +8Gx for 30 s in the present study is also in accordance with the NASA's safe limits of acceleration sustained under nominal and off-nominal conditions.[8] It is well established that the baroreceptor response to the G stress sets in by 6–9 s, settles down by 15 s.[12] Therefore, the duration of peak acceleration was limited to 30 s at both +4Gx and +8Gx sufficient to monitor the changes in cardiovascular parameters.

Sinus tachycardia was observed before the commencement of the centrifuge run due to the anticipatory psychological stress in novice subjects. This can be attributed to "Anticipatory Tachycardia," which is a well-known entity in the centrifuge and has been documented by many researchers.[13-15] A slight rise in mean HR was observed on exposure to +4Gx in comparison to pre-run values. This could be attributed to initiation of Bainbridge and McDowall reflexes, due to the large increase of pressure in the venous side of circulation, especially in the right auricle.[16] This also indicates smaller hydrostatic pressure gradient produced by +Gx acceleration. This smaller hydrostatic pressure gradient could also explain why the difference between mean HR at +4Gx and +8Gx was not statistically significant.[1] The post-run mean HR differed significantly from the baseline value ($P < 0.05$) indicating that recovery was not complete immediately after cessation of exposure. However, the mean HR reached baseline values at approximately 5 min after the cessation of the HPHC run indicating that this much period would be required for complete recovery. The same was also collaborated by no significant difference in BP between before the run and that following recovery. Although literature review revealed occurrence of arrhythmia on exposure to +Gx acceleration above +6Gx to +8Gx,[1] no such events were observed in the present study. This may be due to limited duration of exposure to +8Gx of 30 s in our study. Rai and Gupta also did not find any significant arrhythmia on exposure to +8Gx for 40 s in their study.[9]

One of the primary difficulties experienced on exposure to +Gx acceleration is the difficulty encountered in breathing. The change in mean RR at +4Gx was not statistically significant from baseline/pre-run. However, the increase was significant at +8Gx from baseline. These findings are similar to other studies which reported that the respiratory rate increased in proportion linearly with the applied +Gx acceleration. This is possibly mediated by stretch receptors in the lung and chest wall through stretch or proprioceptive type of reflex.[17,18]

A significant reduction in SpO_2 was observed at +8Gx in the present study. This could have been a manifestation of "physiologic pulmonary arterial-venous shunts" due to increment in effective weight on the lung and pulmonary circulation. These shunts are likely to magnify the inequalities of the ventilation-perfusion ratio (V/Q); leading to increase in perfusion but poor ventilation in the dependent parts of the lungs causing marked reduction of SpO_2.[19-23] Mean SpO_2 recorded post-run (96.6 ± 2.3%) did not reach normality indicating that recovery was incomplete. However, mean SpO2 recorded during recovery (98.7 ± 0.59%) was statistically different from post +Gx run ($P < 0.05$). These findings signify slow though complete recovery without any residual complications such as acceleration atelectasis. Similar observation of slow recovery after cessation of +Gx exposure has also been documented in various studies.[19,24]

The increase in RR on exposure to escalating +Gx acceleration and fall in SpO_2 showed statistically significant strong inverse correlation ($r = -0.94$, $P = 0.006$). Increase in RR and reduction in SpO_2 was also observed in a study conducted by Zechman *et al*., wherein, better ventilation was achieved by increasing the amplitude or the rate of respiration or both on exposure to +Gx acceleration.[18] Hershgold documented that exposure to high +Gx acceleration resulted in causation of severe dyspnea due to reduction in oxygen exchange.[25]

Although BP is one of the important physiological parameters indicating cardiovascular health; the efforts put in to record the BP during acceleration exposure using Portapres BP monitoring system in HPHC were not successful due to the limitation of the equipment. Hence, BP was recorded before and after exposure to acceleration only. This is considered a limitation of the study.

CONCLUSION

It could be concluded from the study that HR rose significantly from baseline to +4Gx acceleration, did not show any significant changes at +8Gx, and recovered gradually after cessation of exposure. The ECG showed no evidence of rhythm disturbances/ectopic beats on exposure to peak +Gx acceleration. The major effects of +Gx acceleration were observed on respiratory system as noted by an increase in the respiratory rate and reduction in SpO_2.

Declaration of patient consent

The authors certify that they have obtained all appropriate consent from the participants.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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