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TITLE

Heart Rate and Blood Pressure Responses to +Gz Following Varied-Duration - Gz

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ORIGINAL RESEARCH

Heart Rate and Blood Pressure Responses to +Gz Following Varied-Duration −Gz

L. S. GOODMAN, PH.D., R. D. BANKS, M.D., B.ENG., J. D. GRISSETT, PH.D., AND P. L. SAUNDERS, M.SC.

GOODMAN LS, BANKS RD, GRISSETT JD, SAUNDERS PL. *Heart rate and blood pressure responses to +Gz following varied-duration −Gz.* **Aviat Space Environ Med 2000; 71:137–41.**

Background: The push-pull effect has been defined previously as decreased +Gz tolerance caused by previous baseline zero or −Gz exposure. Earlier work indicates that the delay in BP (BP) recovery during +Gz is a function of time at −Gz, and is due to the lengthened time-course of sympathetically mediated peripheral vasoconstriction. ***Hypothesis:*** The purpose of this study was to retrospectively determine whether heart rate (HR) varies with BP as duration at preceding −Gz increased. ***Methods:*** Continuous ECG R-R interval data from 15 s of +2.25Gz after preceding 2, 5, 10, or 15 s at −2Gz obtained from previous experiments were analyzed and compared with the previously reported BP data. Repeated measures ANOVA and regression analyses were used to compare +2.25Gz HR responses after the four −Gz conditions and one control +2.25Gz condition. ***Results:*** An initial rapid rise in HR was observed for all conditions with a consistent steady-state plateau achieved after the first 7 s of +2.25Gz. However, there were significant differences in mean HR attained during the +2.25Gz plateau for preceding 15 s −2.0 Gz vs. the control, 2, 5, and 10s −Gz conditions (109 ± 1.1 vs. 102 ± 1.8, 100 ± 2.0, 97 ± 1.1 and 101 ± 1.1, bpm, respectively; $p < 0.05$). ***Conclusions:*** HR, unlike BP, increases briskly across all preceding −Gz time conditions, adapting within the initial baroreflex-compensatory time frame typically expected for +Gz exposures. These results suggest there may be a threshold effect for HR response. Consequently, vasoconstrictor response is a critical adaptive mechanism during +Gz when preceded by long (>10 s) −Gz exposures.

Keywords: positive acceleration, negative acceleration, cardiovascular regulation, carotid-cardiac baroreflex, push-pull effect.

WHEN AIRCREW ARE EXPOSED to less than +1 Gz acceleration during tactical manouvering, hydrostatic forces increase in vessels above heart-level. The consequence, as reported elsewhere (2,3,6,19,22) is an immediate slowing of heart rate (HR) and reduction of arterial blood pressure (BP).

On an immediate transition to high-sustained +Gz levels, the time-course of BP recovery is prolonged to a greater extent vs. when +Gz exposure is attained from a +1 Gz baseline. It was recently reported that this delay, and the associated impairment of subsequent +Gz tolerance ("push-pull effect"), was directly related to the magnitude (3) and duration (2) of the preceding −Gz exposure.

However, the corresponding heart rate response data during these experiments has not been reported. Based on the BP response findings, we hypothesized that the HR response pattern to +Gz after previous −Gz would be directly related to the delay in BP rise. In other words, it seemed tenable that the underlying delay in vasoconstrictor tone, due to increasingly longer preceding −Gz exposures, would be compensated for by a more rapid and sustained increase in HR. Thus, the purpose of this study was to retrospectively examine the HR response to push-pull effect with respect to previously reported BP responses and duration of preceding −Gz exposure.

METHODS

The study and experimental protocol described here was undertaken previously on the U.S. Navy's NAMRL Coriolis Acceleration Platform (CAP), and has been described in greater detail (2,3). The CAP combines angular and linear motion in order to create −Gz and +Gz vectors in subjects placed in the supine seated position. This is achieved by the movement of the subject seated in a capsule, along a track to either the "feet-out" (+Gz) or the "head-out" (−Gz) positions while being rotated on the CAP. The magnitude of ±Gz is a function of distance of the subject from the center of rotation.

Experimental Design

Collected, but unreported electrocardiographic (ECG) data obtained from previous CAP experiments were analyzed in combination with the previously published BP data. ECG data from six subjects were examined under each experimental condition. Each run was separated by 30 s rest (**Fig. 1**). The first and last exposures were always control runs (15 s at +2.25 Gz from +1Gz baseline). The four experimental exposures consisted of 2, 5, 10, and 15 s −2 Gz baseline, followed by transition (at +1 Gz · s^{-1}) to +2.25 Gz for 15 s. The order of −2 Gz exposure times were balanced across subjects.

From the Defence and Civil Institute of Environmental Medicine, Toronto, Ontario, Canada.

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Address reprint requests to: Dr. L. Goodman, Defence and Civil Institute of Environmental Medicine, Aerospace Life Support Section, 1133 Sheppard Ave. W., Toronto, ON, Canada M3M 3B9; len.goodman@dciem.dnd.ca

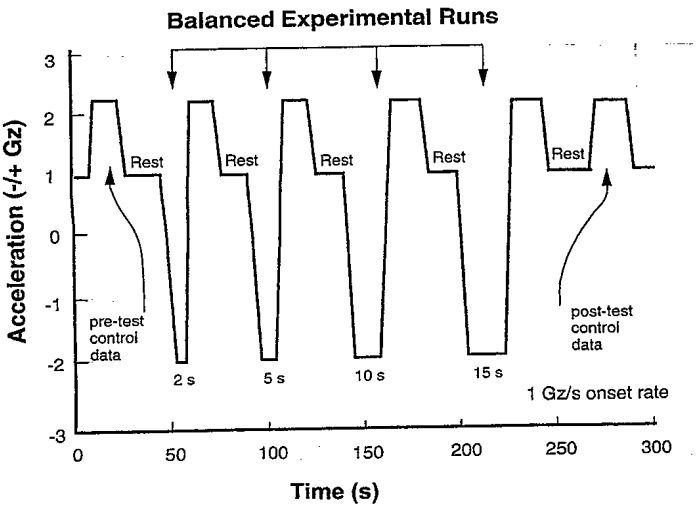

Fig. 1. Experimental Design. A control run of +2.25 Gz from a 1.0 Gz baseline was inserted before and after the experimental runs. The order of experimental runs was balanced across subjects. The Gz onset rate was 1 $Gz \cdot s^{-1}$.

Subjects remained relaxed during all runs, and were not wearing an anti-G suit. Subjects were in audio communication with the investigators, and were able to respond to the +Gz-induced light-loss by responding to a standard light-bar arrangement consisting of center red and peripheral green lights (2,3).

Physiological Measurements

A detailed description of subject and CAP instrumentation is described in detail elsewhere (2,3). Measurements of continuous BP were obtained using a Finapres (Model 2500, Ohmeda, Austell, GA). The finger/pressure cuff was placed at mid-clavicular level.

A single channel ECG was used to record HR. Individual R-R intervals from the analog ECG records were subsequently computed for each run, and transferred to a computer spreadsheet program (Excel, Microsoft Inc., Redmond, WA). These R-R data were transformed to HR values, and then transferred to a Macintosh IIfx running UNIX, where they were subjected to a custom natural cubic spline-transformation algorithm (8) for averaged interpolation to calculated HR at fixed time intervals (seconds of +Gz from 1–15 s).

Statistical Analysis

In order to test the hypothesis that exposure to increasingly longer duration −Gz resulted in amplified HR response pattern to subsequent +2.25Gz, a General Linear Models ANOVA regression model was used on the dependent variable HR (SuperAnova, Adobe Concepts Inc., Berkeley, CA). For the regression analysis, the regressors were −Gz time (2, 5, 10, and 15 s) and time at +2.25Gz (1–15 s). A repeated measures ANOVA was also used to test differences in mean HR over the final 7 s of +2.25 Gz. Analysis of differences between mean −Gz duration conditions were performed using the Duncan New Multiple Range test. For all results, statistical significance threshold was set at an α level of 0.05.

RESULTS

Heart rate fell significantly during exposure to −2 Gz, from a mean resting level of 78 bpm to mean of 49 bpm within 2 s for all −2 Gz exposures, and was maintained at this same level during all −Gz duration conditions (2, 5, 10 and 15 s) (**Fig. 2**). Transient periods of asystole and/or lengthened ECG PR intervals were experienced by 80% of the subjects during the −Gz exposures. There was only one case of premature ventricular contractions during the −Gz exposure.

On transition to +2.25 Gz, there was a rapid increase in HR within the first 5–7 s for all conditions, followed by a relative plateau for the remainder of the +Gz phase (**Fig. 3**). Results of the ANOVA regression analysis taking into account the entire 15 s of +2.25 Gz revealed a significant linear main effect of time at +2.25 Gz on HR, indicating an increase in HR during +Gz occurred, but with no differences in response patterns across −Gz duration conditions.

For all preceding −Gz duration conditions, a plateau in HR was established 7 s into the subsequent 15 s +2.25 Gz exposure. An ANOVA performed on mean HR values during the plateau phase (7–15 s) revealed that only for the 15 s −2 Gz condition, was mean HR significantly greater than the control, 2, 5, and 10 s −2 Gz conditions (109 ± 1.1 vs. 102 ± 1.8, 100 ± 2.0, 97 ± 1.1, and 101 ± 1.1 bpm, respectively).

DISCUSSION

The purpose of this study was to analyze HR response data from previously published CAP studies which reported only BP responses during the push-pull maneuver. Our results demonstrate significant amplification of compensatory HR response to +Gz occurs only during the most prolonged (15 s) periods of preceding −Gz. This finding is in contrast to the progressively impaired BP response pattern previously reported; it was found that compared with the control condition (+1Gz baseline), the increased duration of preceding −Gz resulted in a significantly depressed BP within the initial few seconds of +Gz. The resultant recovery of BP during the remainder of the +Gz exposure was thus directly related to the duration of preceding −Gz (2,3).

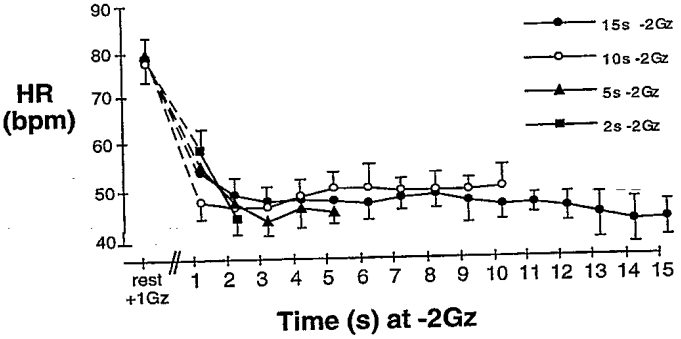

Fig. 2. Mean resting heart rate and mean −Gz heart rate during the 2, 5, 10, and 15 s of preceding −2.0 Gz experimental conditions. Data are expressed as mean HR (bpm) ± SEM.

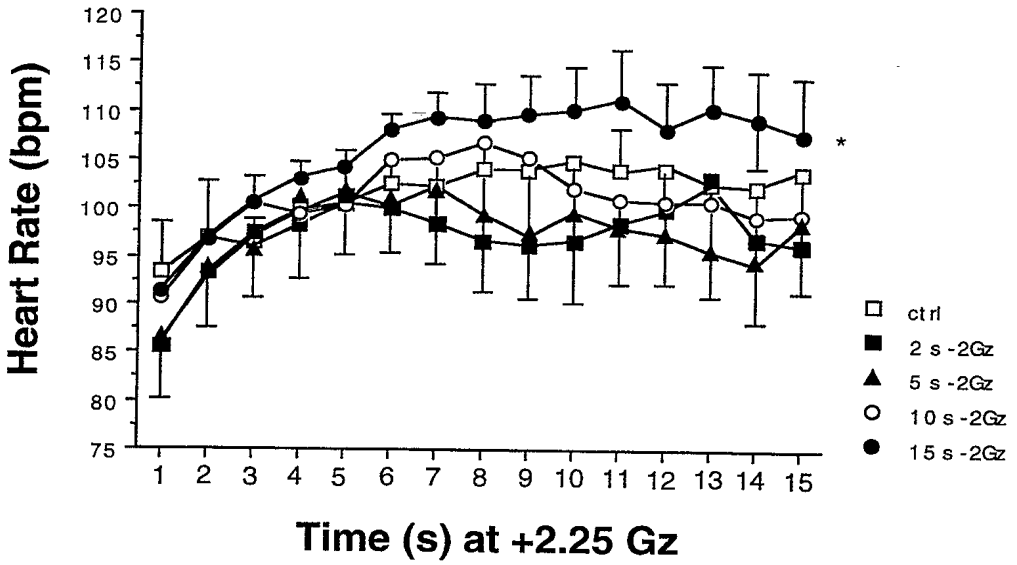

Fig. 3. Mean heart rate (bpm) during 15 s of subsequent +2.25 Gz for the +1 Gz control (0), 2, 5, 10, 15 s preceding −2.0 Gz experimental conditions. Data are expressed as mean ± SEM * 15 s +2.25 Gz response pattern curve is significantly different than control, 2-, 5-, and 10-s conditions.

HR Response to Preceding −Gz

In agreement with other reports, we demonstrated the rapidly evolving bradycardia and mild rhythm disturbances caused by −Gz exposure. Britton (7) found that during +Gz exposure, HR rise occurred within 3 s, vs. an instantaneous fall during exposure to −Gz. Kennealy et al. (15) also described a pronouced bradycardia with prolonged PR intervals during exposure to −2Gz. Prior (19) reported rapidly evolving bradycardia during −Gz exposure in an instrumented human subject during actual in-flight −Gz/+Gz exposures, as did others (18,22).

Rapidly evolving bradycardia has also been demonstrated in head-down tilt table studies. Green et al. (13) reported significant slowing of HR on tilt to −45°. In contrast, Armstrong (1) found that HR actually increased during −Gz, and Ryan et al. (20) reported that HR initially declined, but subsequently recovered during head-down tilt. A common finding in all these studies, is that the speed of HR slowing during head-down tilt or −Gz exposure is greater than the speed of HR rise during subsequent head-up tilt or +Gz exposure. This has been since confirmed in animal studies (9).

The slowing of HR is due to stimulation of the carotid sinus (5,11,17). During −Gz, the carotid baroreceptor firing increases by cephalid transfer of blood during −Gz, leading to increased transmural pressure in the carotid sinus. Afferent signals to the medullary cardiovascular control centers reflexly inhibit sympathetic tone, and cause enhanced parasympathetic tone by direct efferent signaling to the SA node.

The importance of the carotid baroreceptors in this response was clearly demonstrated earlier by Ernsting (12) and later by Eckberg (10), who showed that application of neck suction (i.e., increased carotid transmural pressure) resulted in a lengthened R-R interval. The speed of the response to carotid sinus stimulation, whether by direct hydrostatic forces such as −Gz, or by direct stimulation has been observed by others (4,5).

HR Responses to Subsequent +Gz

The fact that the HR response—unlike BP—was similar across most preceding −Gz conditions, implies that the carotid-cardiac baroreceptor response to +Gz was similar regardless of preceding −Gz. Only for the 15 s preceding −Gz condition was the HR response magnified—and only during the steady-state plateau phase (after 7 s of +2.25 Gz). Since real-life tactical aerial maneuvers rarely involve −Gz duration longer than 2–5 s, these results are important: HR response during the critical initial period of subsequent +Gz is not scaled to the preceding −Gz duration, and peripheral vasomotor reflexes contributing to activating pressor responses must act in the defense of head-level BP.

To examine HR response to subsequent +Gz in more detail, the change (Δ) in HR during the early baroreceptor-response phase of +Gz was analyzed, and is depicted **Fig. 4a**. It is evident that excluding the control condition (Δ 9 bpm), the Δ HR within the first 7 s of +2.25 Gz for all −Gz conditions was 15 bpm. Thus, the early carotid-cardiac response appears to be similar across preceding −Gz duration. However, during the latter plateau phase of +Gz (**Fig. 4b**), only the 15 s −Gz condition is distinguishable from the other −Gz duration conditions, implying a threshold phenomenon may exist for baseline −Gz duration when −Gz magnitude is constant. It is unclear, however, whether the relatively low +2.25 Gz in the present study is a factor in this response. It is indeed possible that excursion to higher, and more operationally realistic +Gz levels would evoke a more accentuated HR response pattern.

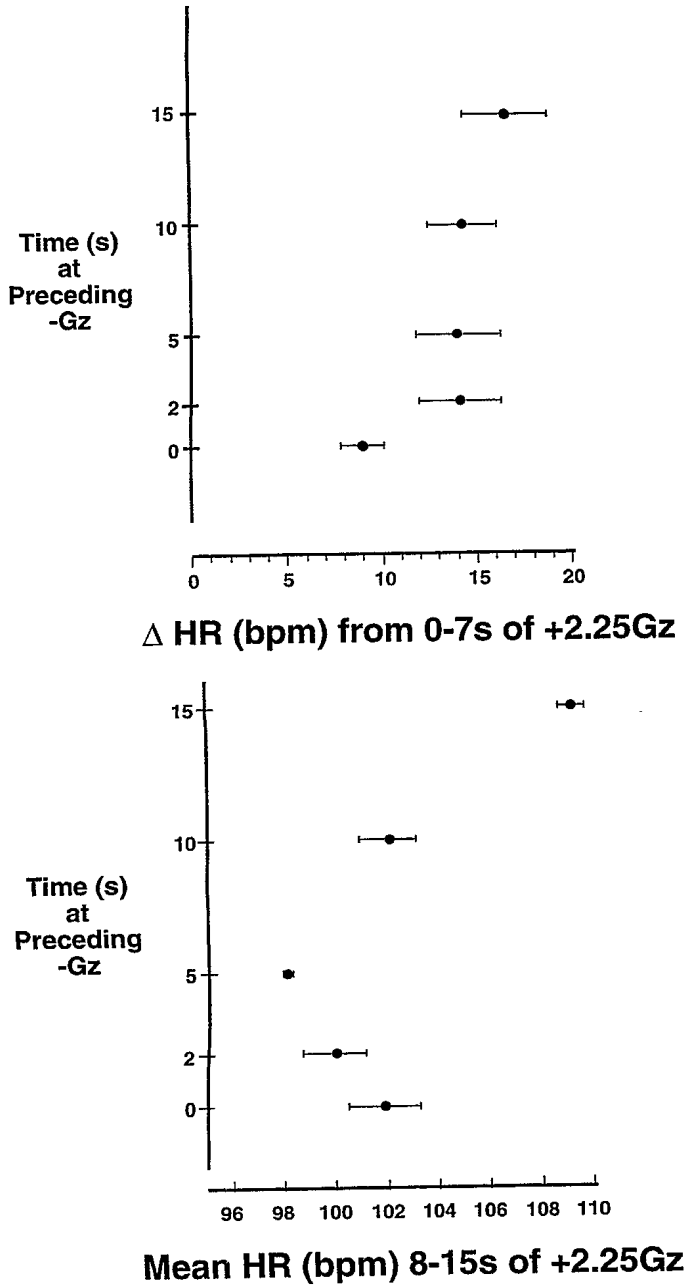

Fig. 4 (a) The mean change in heart rate (bpm) during the initial 0–7 s of +2 25 Gz after preceding 0 (+1Gz control), 2, 5, 10, and 15 s of −2.0 Gz. (b) Mean +2.25 Gz heart rate (bpm) during the plateau phase (8–15 s) of +2.25 Gz for the 0 (+1Gz control), 2, 5, 10, and 15 s preceding −2.0 Gz conditions. Data are expressed as mean ± SEM.

General Cardiovascular Responses to −Gz and Push Pull Effect

The cardiovascular response to sudden and large cephalid blood volume shifts is appropriately aimed at neutralizing sudden increases in head-level BP. This reflex operates in parallel with highly efficient cerebrovascular autoregulatory mechanisms. The resultant peripheral vasodilation and bradycardia is a poor physiologic baseline condition for successful cardiovascular accommodation to a subsequent high +Gz environment. Acute adaptation to +Gz involves a rapid, sympathetically mediated increase in HR, cardiac output (CO), and peripheral vascular resistance to counteract the hydrostatically mediated decline in head-level BP (16,21).

Doe and collegues (9) recently reported that the time to 75% maximal vasodilation in a push-pull effect dog model (open chest experimentally perfused) was shorter than for the vasoconstrictor response (6.7 vs. 12.5 s, respectively). Thus, when the −Gz duration increases, the time required to develop vasocontrictor tone during subsequent +Gz increases. Consequently, the resultant delay in baroreceptor-mediated vasoconstriction during +Gz may reflect progressively increased levels of arteriolar dilatation as preceding −Gz duration increases. This would necessitate a longer time-course for development of arteriolar vasoconstriction during the subsequent +Gz phase (2).

The first 8–10 s of subsequent +Gz represent the transient protective window where the retina and cerebral cortex use endogenous high energy metabolites. If peripheral vascular resistance and thus BP fails to increase at about the same time as endogenous metabolites are depleted, symptoms of +Gz intolerance will ensue rapidly, and at a greater rate than anticipated. If the cardiac-carotid baroreceptor-mediated HR response (and presumably augmentation of myocardial contractility) is fixed, other factors must compensate during subsequent +Gz.

Finally, it should be noted that variations in HR were observed during the +Gz plateau phase. These oscillations are most probably due to pressoreceptor-autonomic oscillations, first observed by Guyton and Harris (14). These waves were explained as random variations of over-and under-correction due to cyclic opposing reflexes initiated by the cardiopulmonary baroreceptor populations, and are due to differences in transmural pressure between the carotid sinus and aortic arch. This effect might be important in the severity of the push-pull effect from one exposure to the next, depending on the exact timing of −Gz/+Gz exposures.

CONCLUSIONS

This study demonstrates firstly, that the magnitude of bradycardia during −Gz exposure is the same, regardless of the duration of −Gz. This result strengthens the argument that even short baseline −Gz duration results in pronounced withdrawl of sympathetic tone, leading to potential impairment of subsequent +Gz tolerance. Secondly, the compensatory HR response during the critical early cardiovascular adaptive phase of +Gz is uniform across all operationally expected scenarios. This is in contrast to BP responses, which are systematically depressed as preceding −Gz time (and magnitude) increases. Thus, the determining physiologic factor/mechanism during push-pull maneuvers affecting pilot +Gz tolerance would appear to be time to increase systemic vascular resistance. Whether this is due to a delay in development of arteriolar smooth muscle tone, or a delay in cardiovascular central command efferent signaling, can only be determined by further experimental work.

Since +Gz in the fighter flight environment is mostly

rapid-onset in nature, outpacing the relatively slow +Gz baroreflex responses, the use of rapidly responding G-valves and/or a well executed anti-G straining maneuver to counter the latent vasodilated state of the peripheral circulation is paramount. Further investigation should be performed to determine whether this uniform HR response to subsequent +Gz occurs during high +Gz exposures after −Gz, and to elucidate in more detail, the physiological mechanisms involved in the push-pull maneuver.

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