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NEUROLOGICAL INFLUENCE IN PUSH-PULL EFFECT

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NEUROLOGICAL INFLUENCE IN PUSH-PULL EFFECT. R. D. Banks. Defence and Civil Institute of Environmental Medicine, North York, Ontario, Canada, M3B 3B9.

INTRODUCTION. Recent reports indicate that tolerance to +Gz decreases more when preceding Gz becomes more negative, and when time exposed to preceding -Gz increases ("push-pull effect"). These results suggest involvement of two physiological mechanisms. NEUROLOGICAL MECHANISMS. Bradycardia induced by -Gz is caused by increased parasympathetic tone as a result of hydrostatic pressure acting on the upper thoracic and carotid baroreceptors. Other effects include decreased cardiac contractility and peripheral vasodilatation through inhibition of sympathetic influence. Tolerating subsequent +Gz depends on the speed of response of the heart and vascular system to increased sympathetic tone. It is known that full heart rate (HR) response to increased sympathetic tone takes 6-8 s. However, results from power spectral analysis of heart rate indicate that the vascular system responds more slowly than HR to autonomic nervous system influence. PREVIOUS OBSERVATIONS. Recovery of blood pressures (BP) measured during +Gz following exposures to -Gz for either 2s or 5s were very similiar, and matched the time course of HR recovery. Conversely, when exposure time to preceding -Gz increased to 10 s and 15 s durations, recovery of BP was delayed beyond the time course of HR recovery. This suggested that a slower responding mechanism was also involved. DISCUSSION. These observations suggest that HR is the primary mechanism of response in push-pull effect when exposures to -Gz are less than 5 s duration. The slower responding vascular system becomes an increasingly dominant influence as the time at -Gz increases beyond 5 s. By 15 s of exposure to -Gz, recovery of BP is primarily mediated by the speed of vasoconstriction. CONCLUSION. Designing adequate protection for pilots will require understanding these mechanisms. Research on the response of the heart and vascular bed to short-term autonomic influence is indicated.

NEUROLOGICAL INFLUENCE IN PUSH-PULL EFFECT

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INTRODUCTION

During straight and level flight, aircraft occupants are exposed to the normal acceleration force of gravity, that is +1 Gz. Evolutionary development of homeostatic control of blood pressure (BP) via neurological and cardio-vascular mechanisms occurred under the continuing influence of +1 Gz during walking or sitting. Thus, humans have not yet evolved mechanisms to compensate for significant excursions from +1 Gz. Aircraft occupants are frequently exposed to short duration accelerations other than +1 Gz (12). Air turbulence, for example, exposes occupants to short (up to 0.5s) exposures to ± Gz. Coordinated banked turns in jet aircraft expose occupants to sustained increases in +Gz. Nose-down, or 'bunt' maneuvers expose occupants to relative -Gz (that is, Gz that is less than +1 Gz).

Understanding of the effects of exposures to variations in Gz is incomplete. Because sustained increases in +Gz occur commonly in flight and can be studied in laboratories with the use of human centrifuges, much work has been accomplished. However, research conducted on human centrifuges commonly suffers from the bias of a starting baseline of +1.4 Gz, a condition that seldom exists in actual flight (3). Much less is known of the effects of increased +Gz when the starting condition is less than +1.4 Gz.

The few studies that report on G-tolerance when +Gz baseline varies are preliminary, but have demonstrated that tolerance to +Gz decreases more when preceding Gz is relatively more negative (4, 6, 27, 37). This effect, termed the "push-pull effect", increases with increased time of exposure to preceding relative -Gz (5, 27, 37). In-flight studies have shown that a proper anti-g straining maneuver (AGSM) and/or g-suit inflation only partially counters push-pull effect (37).

The recent fatal crash of a Canadian Forces CF-18 due to push-pull effect has highlighted the inadequacies of current protection strategies. Prevention of similar accidents requires completing our understanding of acceleration physiology. As a rationale for future research efforts aimed at achieving this understanding, this paper discusses the possible neuro-cardiovascular processes involved in push-pull effect.

NEUROLOGICAL MECHANISMS OF BP CONTROL AT +1 Gz

During straight and level +1 Gz flight, short term BP control occurs via the autonomic nervous system. The parasympathetic system is normally dominant in rest at +1 Gz, blunting sympathetically-mediated increases in BP sensed at the upper thoracic and carotid baroreceptors.

The baroreceptors are stretch receptors that sense change, and rate of change of transmural pressure (8, 26, 30, 32). A change in transmural pressure sensed at the baroreceptor results in a transmitted signal centrally to the medulla (1, 26, 30, 31). After central processing, parasympathetic efferent response occurs via the vagus nerve to the heart, and parasympathetic-mediated outflow to the peripheral circulatory system. Vagal parasympathetic influence results in rapid cardio-deceleration, and decreased cardiac contractility. Parasympathetic inhibition of efferent sympathetic nervous system-mediated vasoconstriction results in relaxation of vascular smooth muscle and vasodilation. BP is therefore reduced as a result of decreased cardiac output (secondary to reduced heart rate (HR), cardiac

filling, and cardiac contractility), as well as increased vascular conductance due to circulatory vasodilation.

Conversely, a decreased transmural pressure presented to the upper thoracic and carotid baroreceptors is similarly interpreted centrally as a reduction in BP, and increased sympathetic efferent activity results. Increased sympathetic influence to the myocardium occurs via the glossopharyngeal nerve and results in cardio-acceleration, but at a rate slower than parasympathetically influenced cardio-deceleration (4, 13) and, presumably, increased cardiac contractility. Sympathetic effector activity to vascular smooth muscle via efferent sympathetic nerves results in peripheral resistance vessel vasoconstriction (skeletal muscles and splanchnic beds). These reflexes consequently restore BP as a result of increased cardiac output (CO) due to a faster beating, greater contractile heart and increased peripheral resistance due to arteriolar vasoconstriction.

The net effect is that in health, BP is tightly regulated, such that mean BP varies little, even during reasonably rapid postural changes from supine to +1 Gz upright positions.

BLOOD PRESSURE REGULATION UNDER INCREASED +Gz

The role of the sympathetic nervous system in maintaining BP during increased +Gz is understood (28), and matches the model described above. Under increased +Gz, HR compensatory response is complete within 5-12 s (16) As BP decreases in the upper body under increased +Gz, sympathetic efferent mechanisms are activated by arterial and cardiopulmonary baroreceptors. Depending upon the extent and rate of +Gz onset, the reduction in the relative hydrostatic column between heart and head may be too great for these BP compensatory mechanisms to become effective. In this case, head (eye) level hypotension leads to "grey-out", "black-out", or G-induced loss of consciousness (G-LOC) (11, 28, 40, 44).

When cerebral ischemia develops under increased +Gz, intracellular substrates allow continued brain tissue function for 5-7 s before symptoms occur (40, 41, 42, 43). This period of substrate latency is extremely important since it defines the time available for compensatory mechanisms to be effective in preventing G-LOC. Also, protective countermeasures (e.g., G-suit inflation, AGSM) must be effective within this time period.

EVALUATING THE PHYSIOLOGICAL MECHANISMS IN PUSH-PULL EFFECT

Bradycardia during relative -Gz is caused by parasympathetic influence (4, 6, 14). This influence occurs because of increased transmural pressure on the upper thoracic and carotid baroreceptors due to the increased weight of the inverted hydrostatic column. As described above, other anticipated effects would be decreased cardiac contractility and peripheral vasodilatation. Tolerance to subsequent +Gz then depends on the ability of the body to reverse these conditions within the 5-7 s period of brain substrate latency. When evaluating transition from -Gz to +Gz, therefore, the following mechanisms must be effected rapidly in order to maintain cerebral perfusion:

1. bradycardia (-Gz) reverting to tachycardia (+Gz)

2. low cardiac contractility (-Gz) reverting to high contractility (+Gz)

3. excess atrial/ventricular preload (-Gz) reverting to normal or reduced atrial/ventricular preload (+Gz)

4. peripheral vascular vasodilation (-Gz) reverting to vasoconstriction (+Gz)

1. Bradycardia (-Gz) reverting to tachycardia (+Gz). Bradycardia occurs within 2-4 s under the influence of -Gz, although some recovery in HR may occur with sustained exposures to -Gz (9, 17, 24). Tachycardia during +Gz becomes established at a slower rate, taking 6-8 s (4). This point is illustrated in Figure 1, which is an example of HR recordings taken from two Canadian Forces pilots during flight. Since the 6-8 s recovery time is greater than the 5-7 s of substrate brain latency, it is likely that this mechanism contributes to symptoms experienced during push-pull effect.

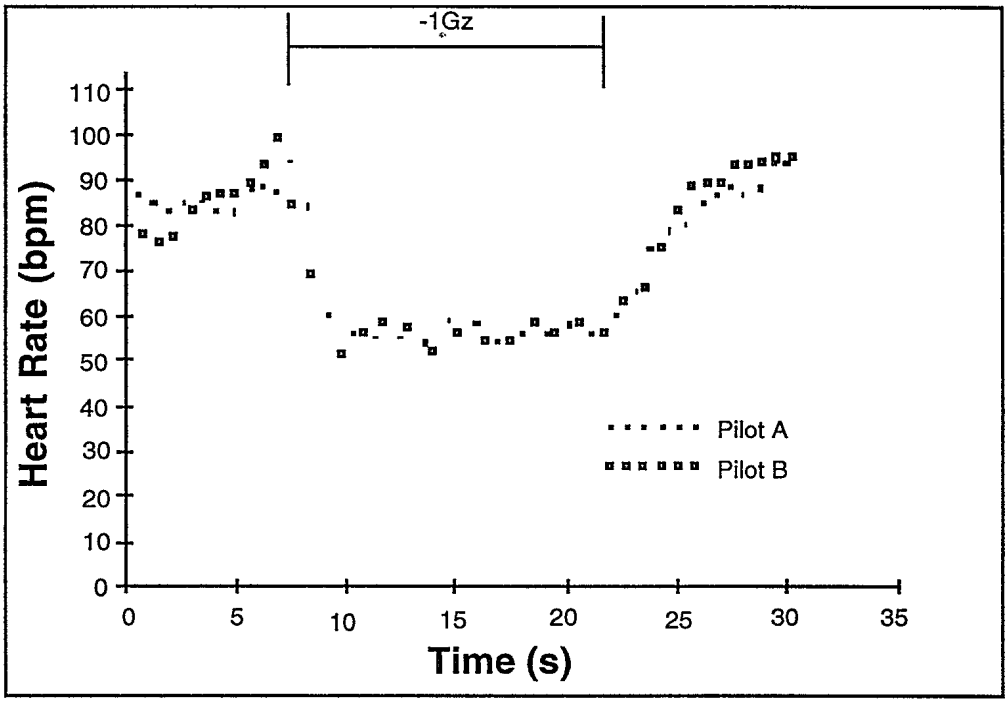

Figure 1. Heart rate recordings from two pilots. Rapid drop in HR occurs immediately on transition to -1 Gz from +1 Gz. Note stable bradycardia plateau, then slower recovery when flight is resumed at +1 Gz.

2. Low cardiac contractility (-Gz) reverting to high contractility (+Gz). Extrapolation of cardiac contractility changes under autonomic nervous system influence during ±Gz is based upon studies conducted at +1 Gz. Understanding the place of this mechanism in BP recovery from -Gz to +Gz transitions is complicated by the limited understanding of myocardial energetics during -Gz (33) and +Gz (29), as well as the opposite and competing influences of cardiac filling. During -Gz, high parasympathetic tone attempts to decrease cardiac contractility, however, this is superimposed upon increased atrial filling due to increased venous return. Conversely, during subsequent +Gz, contractility will increase under sympathetic influence, but the time course is difficult to predict and is currently unknown. Thus, the role of cardiac contractility in push-pull effect is currently undefined.

3. Excess cardiac diastolic filling (-Gz) reverting to normal filling (+Gz) The diastolic filling of the atria and ventricles (by increases in venous return) is known to greatly influence the heart's output (via Frank Starling Mechanism) (20). During -Gz exposure, right atrial filling will transiently increase markedly. In one +1 Gz study with high levels of positive pressure breathing, over-pressurization of a full-coverage G-suit was shown to translocate blood into the thorax, increase end-diastolic filling rate, and slow heart rate below resting conditions (19). It is probable that cardiac mechanoreceptors (7, 23) might also interact with cardiovascular centres (in concert with arterial baroreceptors) to slow heart rate in excessive venous return conditions. However, during subsequent +Gz, the ventricles are unloaded, and the normal BP rise is dependent upon a rising CO secondary to adequate venous return and diastolic filling.

4. Peripheral vasodilation (-Gz) reverting to vasoconstriction (+Gz). Total peripheral resistance (TPR) is normally derived as BP/CO and depends on measurements made over a steady-state period of time in excess of the 5-7 s period of brain substrate latency. Derived TPR in evaluation of push-pull effect is therefore not valid, and modifications of this classical expression must be developed for acceleration

physiology. Very little work has been done to directly measure the changes in either the venous or arterial vascular systems in response to parasympathetic or sympathetic stimuli. Recent work has, however, suggested that both peripheral arterial and capacitance vessels participate in ±Gz responses (38). The vascular walls are composed of smooth muscle, a typically slow responding end-organ (30, 31, 32). Increased sympathetic tone causes contraction of smooth muscle in the vessel wall; parasympathetic influence blocks sympathetic tone and allows relaxation of the vessel wall, and vasodilation. Of interest in consideration of push-pull effect is the time-response of this smooth muscle. Complicating features relate to locally-mediated vasodilation in skeletal muscle beds caused by prior AGSM, which could also induce further peripheral vasodilation during subsequent -Gz.

Clues to the time-response of the vascular bed can be found in several reports. Wallin and Eckberg (39) noted vasodilation periods of 9-15s during neck suction experiments that simulated -Gz type impulses. Gauer and Henry (18) demonstrated during -Gz that BP typically changed immediately, according to the expected hydrostatic column inversion. A further reduction which occurred secondarily was interpreted as continued peripheral vasodilation under -Gz influence. Peterson and colleagues (36) reported that peripheral resistance did not begin to increase until 5.8 s after exposure to +Gz, in dogs. Maximum vasoconstriction was not achieved until 20.4 s following onset on +Gz. Guyton (21) reported that the full pressor response in dogs took 13 s, with 7-10 s for the initial response and up to 15-20 s for full expression. Brown and colleagues (10) demonstrated that in humans exposed to lower body negative pressure (simulation of +Gz), total peripheral resistance was maximum after 14 s.

Additional clues to the time-response of the vascular bed may be found in the wave-length of vasomotor waves, otherwise known as Mayer waves or Trobe-Herring-Mayer (THM) waves. These waves are embedded within normal respiratory fluctuations in HR and BP tracings. Guyton and others (22, 35) described these in hemorrhaged dogs, and felt that they represented a measure of the speed of response of the baroreceptors. Similar waves have recently been identified in human subjects exposed to high +Gz in the DCIEM centrifuge (Buick, F. personal communication). Techniques such as spectral analysis have further refined our understanding of the period of these waves, and indicate an average of about 10 s for humans (2, 15, 34). Much work needs to be done to further define the meaning of these waves. They may represent a measure of the time-response of the smooth muscle of the vascular bed to pertebations in BP, and/or alternating competition between the high and low-pressure (cardiopulmonary) baroreceptors.

Of particular significance in consideration of push-pull effect are the recent findings of Doe and colleagues (13). Using the dog model, they have demonstrated that parasympathetically mediated vasodilation is a more rapid process than sympathetically mediated vasoconstriction. This finding suggests a time response that parallels HR response during -Gz. If true in the human, this difference in parasympathetic-sympathetic response times may further explain the occurrence of push-pull effect.

If relaxation-contraction of resistance vessel walls is a significant mechanism in push-pull effect, it follows that the effect will be lessened if any portion of the arterial tree becomes more rigid and therefore a less responsive end-organ to contraction-relaxation inputs. Since vessel wall rigidity increases with normal aging, due primarily to atherosclerosis (25), it also follows that older subjects might be less susceptible to push-pull acceleration stresses. During pilot laboratory studies on push-pull effect, two healthy males aged 48 and 58 yrs showed minimal or no susceptibility to push-pull effect (Banks, R.D. unpublished data). While not constituting scientific proof, these observations are consistent with the notion that the compliance and reactivity of the vascular bed plays an important role in the etiology of push-pull effect.

	+1Gz control	2s - 2 Gz	5s - 2 Gz	10s - 2 Gz	15s - 2 Gz	Total
Light loss	0	1	2	6	4	13
No light loss	12	5	4	6	2	29
Total	12	6	6	12	6	42

Table I. Subject's reported incidents of visual light loss during 15-s exposure to +2.25 Gz, following preceding flight at +1 Gz (normal flight), and -2 G for 2-, 5-, 10-, and 15 seconds.

CURRENT STUDIES

In consideration of the relative importance of each of these mechanisms, Figure 2 is presented. Figure 2 depicts the averaged systolic blood pressure (BPs) of groups of subjects exposed to +2.25 Gz for 15 s following exposure to +1 Gz, and exposures to -2 Gz for 2-, 5-, 10-, and 15 s (taken from (5)). The difference in each of these BP plots from the plots preceded by +1 Gz (baseline) can be considered a quantification of loss of +Gz tolerance. Table I (taken from (5) and (6)) consolidates the reports of visual light loss by subjects during these experimental conditions, and shows that subjective assessment of light loss correlated well with quantitative estimates of +Gz tolerance loss shown at Figure 2.

Evident from Figure 2 is the observation that the 2- and 5 s exposures to -2 Gz result in very similar plots. Also evident is the fact that BP recovery is complete within 6-8 s of exposure. This recovery period matches the recovery time period of heart rate following -Gz (4). For purposes of comparison, Figure 3 depicts the 2 and 5 s exposures to -2 Gz with superimposed heart rate recovery data recovered from pilots exposed to -1 Gz in flight.

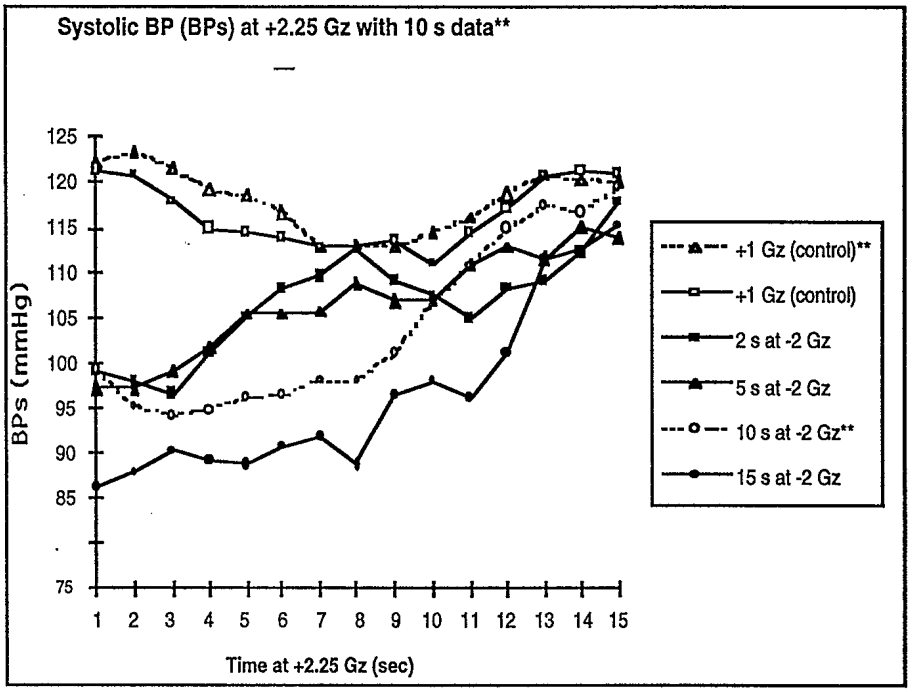

Figure 2. Systolic BP of subjects taken from two studies (5,6). BP was recorded while subjects experienced +2.25 Gz following exposure to -2 Gz for various periods of time.

Further inspection of Figure 2 indicates that 10- and 15 s exposures to -2 Gz result in greater loss of Gz tolerance, and slower recovery. By implication, a mechanism other than HR rate recovery must be responsible for continued BP recovery beyond HR recovery.

DISCUSSION

The BP recovery time at +Gz following short duration (less than 5 s) exposures to -Gz matches the time recovery period for HR. As the time of exposure to -Gz increases beyond 5 s, BP recovery time increases beyond the time for full HR recovery. This increase becomes greater with more time at -Gz. Figure 4 shows the 10 and 15 s BP recovery plots with HR recovery data superimposed. In this case, the longer duration spent at -Gz (10 and 15 s) preceding immediate exposure to +2.25 Gz results in a more prolonged recovery of BP than for the 2 and 5s -Gz exposures.

These observations illustrate that HR increase may be the predominant mechanism of BP recovery for short exposures to -Gz (less than 5 s). When -Gz time exposures exceed 5 s, HR recovery alone is insufficient to effect full BP recovery. This suggests that vascular bed reactivity plays an increasingly important role in BP recovery for longer exposures to -Gz. Using clues to the speed of reactivity of the vascular bed, we might be able to assume a period of either full vasodilation, or full vasoconstriction of 7-15 s in humans, noting in the process that vasodilation may be the faster of the two processes (13).

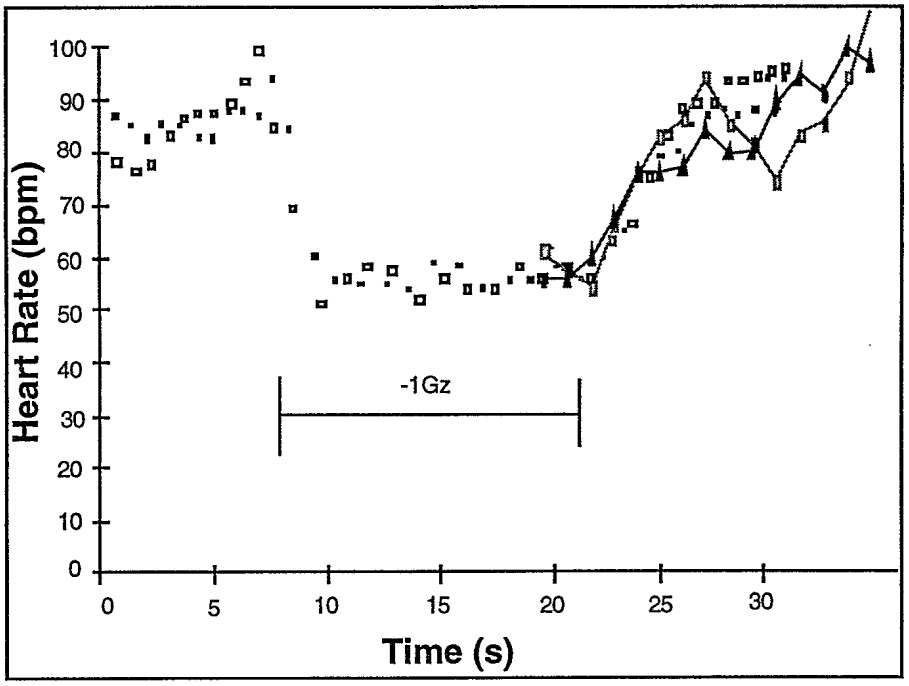

Figure 3. Recovery of blood pressure at +1 Gz after exposure to -Gz. Closed and open boxes are in-flight heart rate data for pilots A and B(from figure 1), respectively during maneuvers from +1 Gz, to -1 Gz, and to +1 Gz; heavy superimposed symbol line is mean blood pressure laboratory data during +2.25 Gz after 5 s at -2 Gz; light superimposed symbol line is the mean laboratory data for the same subjects during +2.25 Gz, after 2 s at -2 Gz.

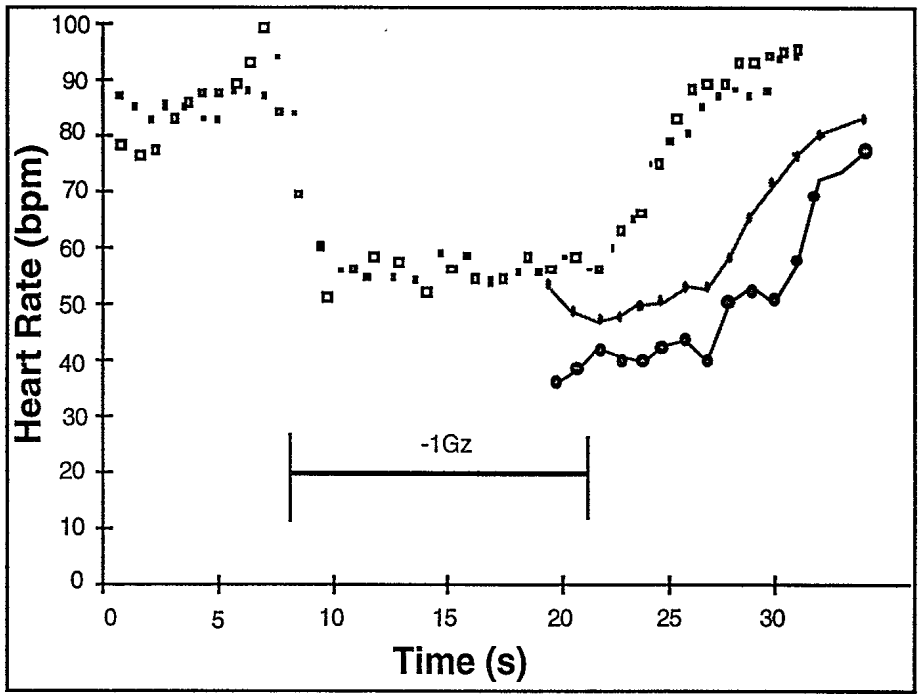

Figure 4. Recovery of blood pressure at +1 Gz after exposure to -Gz. Closed and open boxes are in-flight heart rate data for pilots A and B (from figure 1), respectively during maneuvers from +1 Gz, to -1 Gz, and to +1 Gz; light superimposed symbol line is mean blood pressure laboratory data during +2.25 Gz, after recovery from 10 s at -2 Gz; heavy superimposed symbol line is the mean laboratory data for the same subjects during +2.25 Gz, after recovery from 15 s at -2 Gz.

If these estimates are valid, exposures to -Gz less than 5 s would not allow time for full peripheral splanchnic and skeletal muscle vasodilation to occur. Under this circumstance, partial vasodilation results in a moderate increase in vascular volume space leading to a BP drop during +Gz, but not enough to overwhelm the HR-based compensation that occurs during +Gz.

When exposures to -Gz approach the postulated time to full vasodilation of 7-15 s, full relaxation of the peripheral circulatory bed results. Full vasodilation results in a greatly increased volume of vascular space, and profound drop in BP that can not be compensated for by increased HR alone. Full BP recovery is then dependent on the relatively slow vasoconstriction of the vascular system.

As mentioned earlier, two of the experimental subjects were older, and these two exhibited the least susceptibility to push-pull effects. If we assume that they had the normally expected decrease in arterial compliance (i.e., arterial stiffening) due to aging, then neither vasodilation nor vasoconstriction would have been prominent mechanisms in dealing with push-pull stresses. Rather, HR would have been the primary mechanism of recovery. With less ability to vasodilate, there would have been less BP liability during subsequent +Gz, and HR increase might have effectively ensured BP recovery. This may explain the apparent lack of push-pull effect in these two older subjects. Future aged-based analysis of experimental data is planned.

The role of cardiac contractility however, is unknown and difficult to postulate. Since BP maintenance is also dictated by CO (dependent upon cardiac filling), this issue will be explored in future studies using a new miniaturized nuclear cardiology biotechnology during simulated ±Gz.

CONCLUSION AND RECOMMENDATIONS

This analysis suggests that HR is the primary mechanism of response in push-pull effect when exposures to -Gz are less than 5 s duration. The slower responding vascular system becomes an increasingly dominant influence as the time at -Gz increases beyond 5 s. By 15 s of exposure to -Gz, recovery of BP is primarily determined by the speed of vasoconstriction of peripheral vascular beds.

Designing adequate protection for pilots will require understanding these mechanisms. Research on the response of the heart and vascular bed to short-term autonomic influence is indicated.

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