12

Analysis of Time Course Changes in the Cardiovascular Response to Head-Up Tilt in Fighter Pilots

David G. Newman[1] and Robin Callister[2]
[1]*Aviation Discipline, Faculty of Engineering and Industrial Sciences, Swinburne University, Melbourne,*
[2]*Human Performance Laboratory, Faculty of Health, University of Newcastle,*
Australia

1. Introduction

Fighter pilots are exposed to significant levels of +Gz acceleration on a frequent occupational basis (Newman & Callister, 1999). There is an emerging body of experimental research that suggests that they physiologically adapt to this frequent +Gz exposure (Convertino, 1998; Newman & Callister, 2008, 2009; Newman et al, 1998, 2000). Our previous work has shown that fighter pilots are able to maintain their cardiovascular function to a much greater extent than non-pilots when exposed to an orthostatic stimulus such as head-up tilt (Newman & Callister, 2008, 2009; Newman et al, 1998, 2000).

To further examine the mechanisms underlying these differences in cardiovascular response to +Gz, a beat-to-beat analysis of the time course of dynamic cardiovascular responses to head-up tilt (HUT) was conducted. The hypothesis was that the time course of acute changes in mean arterial pressure (MAP), heart rate (HR), stroke volume (SV) and total peripheral resistance (TPR) in +Gz-adapted fighter pilots would be different from that of non-pilots. Such differences would provide further evidence of cardiovascular adaptation to repetitive high +Gz exposure, and help to further our understanding of how this adaptation is mediated.

2. Methods

The subjects were 20 male volunteers drawn from personnel of Royal Australian Air Force (RAAF) Base Williamtown. No female subjects were recruited as the RAAF did not have any female fighter pilots at the time of the study. The control group consisted of 12 non-pilots (NP). The second group consisted of 8 current operational jet fighter pilots (FP) from RAAF Base Williamtown.

The two groups were closely matched in terms of age, height, weight, aerobic fitness level, resting blood pressure and heart rate (Newman et al, 1998, 2000). All subjects gave their

written informed consent before being tested. The study was approved by both the Australian Defence Medical Ethics Committee and the Human Research Ethics Committee of the University of Newcastle. All subjects were asked to refrain from eating for 2 hours and from drinking caffeinated beverages for 4 hours prior to the test for standardisation purposes. Subjects were assigned an alpha-numeric code to maintain confidentiality.

Each subject was non-invasively instrumented for the beat-to-beat measurement of stroke volume via impedance cardiography. Four impedance cardiograph metallic band electrodes were applied to the thorax of the subject in the manner described previously (Newman et al, 1998, 2000). The leads were then attached to the impedance cardiography unit (Instrumentation for Medicine, Model 400, Greenwich, CT). Heart rate was determined via an electrocardiogram (ECG) signal generated by the impedance cardiography unit.

Data from the impedance cardiograph and other recording instruments were stored on video tape via a digital video cassette recorder (Vetter, Model 4000A, Rebersburg, PA). The video tape data were analysed using a MacLab/8s 8-channel digital chart recorder and analysis system (ADInstruments, Model ML 780, Castle Hill, Australia). MacLab Chart software (ADInstruments, Version 3.5.2/s, Castle Hill, Australia) was used to capture and analyse the digital video data.

Four cardiovascular parameters were examined in this analysis: MAP, HR, SV and TPR. MAP was calculated according to the formula MAP = DP + 1/3 (SP-DP). SV was determined using the Kubicek equation (Newman et al, 1998, 2000). TPR was calculated as MAP/(HR x SV).

The data were divided into Control (C), Anticipation (A) and Tilt (T) periods. C consisted of data from the beginning of recording until the start of A, which was defined as the 5 heart beats immediately prior to the tilting event. T consisted of the 30 heart beats from the onset of tilt. For the purposes of tracking changes across time, and for ease of description, the T period data were divided into 6 phases (I-VI) consisting of 5 heart beats each. The transition from the supine to the full $+75^0$ head-up tilt position occurred during Phase I.

Analysis of the data was performed using a statistical software package (SuperANOVA, Abacus Concepts, Inc., v1.1). Repeated measures analysis of variance with one within factor (time) and one between factor (group) was used as the test of statistical significance. An alpha level of $p<0.05$ was considered significant at the 95% confidence interval for all effects.

3. Results

Figure 1 shows the mean T period values (+ SEM) on a beat-to-beat basis for each of the four variables for both experimental groups. The mean values (+ SEM) for each group's C and A periods are shown as the first two data points. The data are divided into phases for ease of reference during description.

3.1 Responses to tilt

The NP data show an early rise (Phase I) in MAP, which then decreases to values significantly below control levels in Phase III. MAP then progressively rises to levels slightly above but not significantly different from C during the late part of the tilt (Phases IV to VI). In the FP group, MAP also rose initially during Phase I and decreases towards C values in

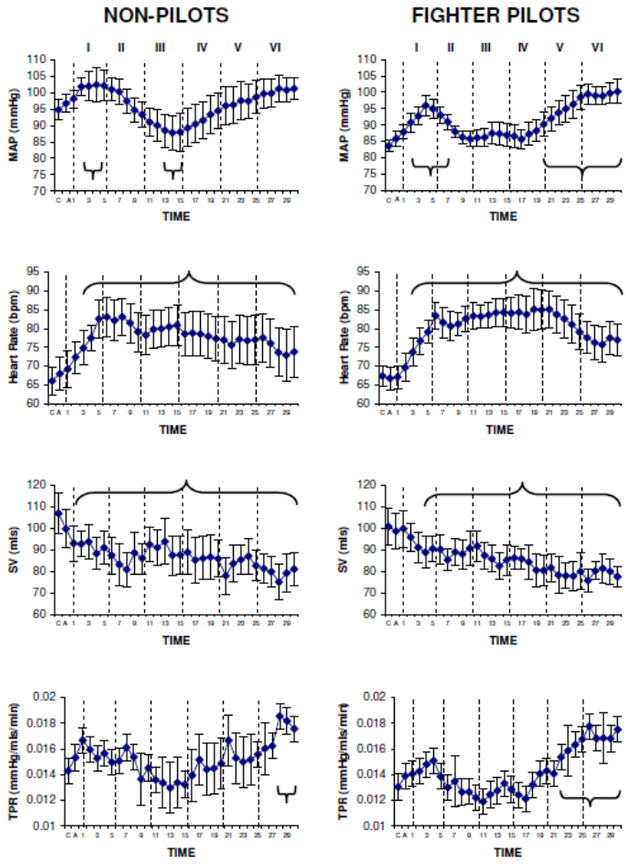


Fig. 1. Comparison of the time course of the non-pilot (left columns) and fighter pilot responses to 75^0 HUT across time. The first two data points on each plot are C and A values. The bracketed areas on each curve represent areas of significant difference ($p<0.05$) from C.

Phase II. Phase III of the FP MAP response is clearly different from that of the NP group, with MAP plateauing and never falling below the C level. Early in Phase IV, MAP rises progressively, reaching values in Phases V and VI significantly greater than the C level.

HR is elevated significantly above C in both groups within four heartbeats of tilt. In NP, HR rises immediately during Phase I, is sustained at this level during Phase II and then progressively decreases slowly towards C levels in Phases III to VI. HR is significantly different from C for most of the tilt period. In FP, HR also increases in Phase I, begins to decrease in the early part of Phase II but then increases again by the end of Phase II, and remains significantly elevated throughout Phases III and IV, reaching maximum elevation at the junction of Phases IV and V, then begins to decrease back towards C values.

In NP, SV falls precipitously at the onset of tilt, then increases slightly during the later part of Phase II and the early part of Phase III. SV then progressively decreases again, although at a slower rate in Phases III to VI. SV in the FP group falls in Phase I, but not as immediately or to the same extent as the NP group. It recovers a little in Phase II, then progressively decreases in later phases. Like the NP group, this late-phase decrease occurs at a slower rate than in Phase I.

In NP, TPR increases initially during Phase I, then decreases to levels below C values by the middle of Phase III. Phases IV to VI are marked by progressive increases in TPR to values significantly above C values by Phase VI. In FP, TPR rises during Phase I, then decreases below C values during Phase II. Phase III is marked by a small recovery in TPR, which is not evident in the same phase in the NP group. TPR increases throughout Phases IV to VI, becoming significantly different from C values earlier than in the NP group.

3.2 Group comparison

Figure 2 plots the T deviation from C values for each of the four cardiovascular variables, again divided into the same 6 phases. The analysis was performed on individual data points, although these and the error bars have been removed from the figure, purely for ease of visualising the comparison between the groups across time. This series of curves demonstrates the relative contribution of these variables to the observed time-based changes in cardiovascular dynamics.

The MAP curves show a similar overall pattern of response to tilt, although a significantly greater response is seen in the FP group to the same gravitational stimulus ($p<0.05$). The FP group maintains MAP above C values at all times, and in the second half of tilt MAP values are significantly higher than those of the NP group.

The HR curves are similar for each group, although in the later phases the group responses tend to diverge, with the FP group demonstrating a more sustained elevation. In this group, HR is maintained at its peak level until the early part of Phase V, when it begins to decrease. In the NP group HR begins to decrease in Phase II, although it remains elevated above control levels throughout tilt.

There is no statistically significant difference in the SV response to tilt of the two groups, although the initial rate and magnitude of decrease in SV appears less in the FP group.

The TPR curves show similar patterns, rising initially, then decreasing and rising again in Phase V in both groups. The FP group shows a more marked late-phase rise in TPR, which is of greater magnitude than that in the NP group, and coincides with the FP's fall in HR during Phase V. This rise in TPR becomes significantly different ($p<0.05$) from C values earlier in the FP group.

4. Discussion

The results of this analysis show similar overall patterns of response between the two groups. There are some key differences, however, in terms of the timing and magnitude of the responses. These are just sufficiently different that they produce statistically significant and physiologically meaningful differences in the MAP response between the two groups.

The NP response to HUT is the normal, well-documented human response to upright posture. On assuming the upright position, there is an initial, transient HR- and TPR-mediated rise in MAP, then both MAP and venous return fall in accordance with the applied hydrostatic force. The fall in these parameters activates the baroreceptors, both the high-pressure arterial baroreceptors and the low-pressure cardiopulmonary baroreceptors. This leads to activation of these negative feedback regulating systems and a subsequent restoration of MAP and venous return towards normal levels (Mancia & Mark, 1983).

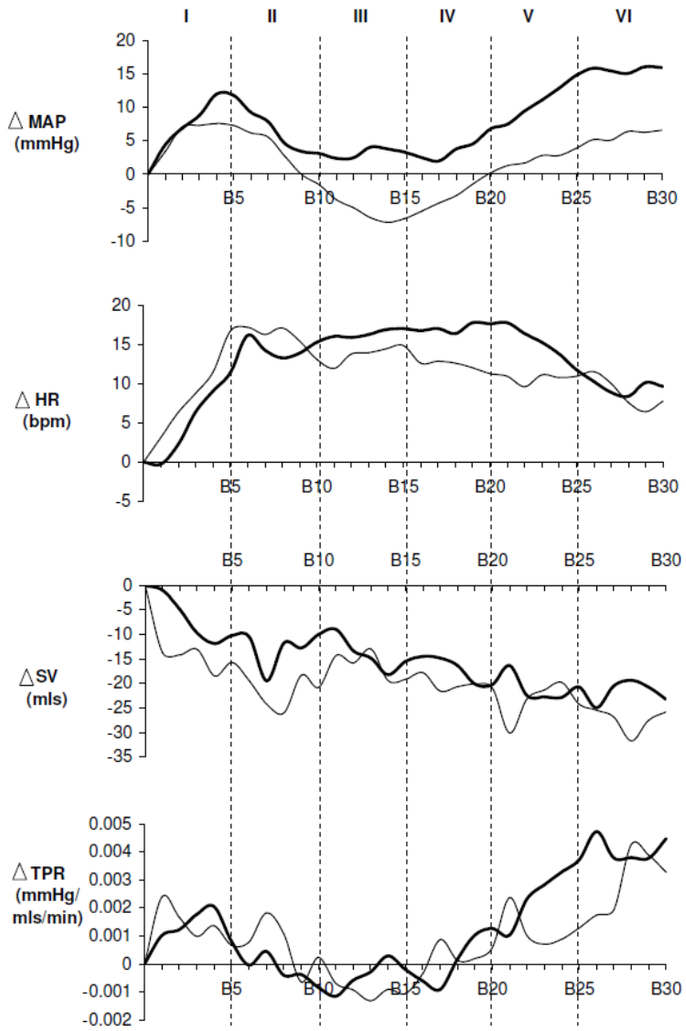


Fig. 2. Comparison of the change from control values across time of the non-pilots (thin line) and fighter pilots (thick line) in response to 75^0 HUT. Data are mean values. SEM bars have not been drawn. The time course has been divided into 6 phases of 5 beats each, labelled I to VI (details in text).

The FP response is an adapted or modified version of the NP response. Analysis of the different phases of the groups' responses to tilt provides important information as to the mechanisms that are active during the sequence of events. The focus of this discussion will be on the integration of cardiovascular control inputs.

4.1 Cardiovascular regulation

There are four possible inputs used in the regulation of the cardiovascular system under conditions of orthostatic stress such as HUT. Firstly, there may well be some cognitive or psychological input to the autonomic nervous system at the onset of a rapid tilt or postural change, and in anticipation of this impending event. This heightened sense of arousal or

alerting reaction would produce an increase in HR, vasodilation in some vascular beds (e.g., skeletal muscle) and vasoconstriction in others (e.g., gastrointestinal tract and kidneys). The rapid, almost immediate increase in HR (due to parasympathetic withdrawal) will shorten cardiac ejection time, which will in turn contribute to a fall in SV. These changes reflect an overall shift in the autonomic balance in favour of the sympathetic system. The net effect is an increase in arterial pressure (Mancia & Mark, 1983).

The arterial baroreceptors also have a well established influence on the cardiovascular system under orthostatic stress (Mancia & Mark, 1983). The overall effect is also a shift in the autonomic balance, with the sympathetic system becoming more dominant. HR increases due to parasympathetic withdrawal, while cardiac contractility and total peripheral resistance both increase due to greater sympathetic drive. A more forceful, rapid ejection of blood with higher vascular resistance results in an overall boost in mean arterial pressure. The time taken for cardiac contractility and vascular resistance to increase is much longer than that for HR, due to these sympathetically-innervated tissues taking longer to respond to neural command signals.

During HUT the aortic and carotid baroreceptors will be stimulated to different extents, based on their respective distances from the heart. In this experiment, arterial pressure was recorded effectively at aortic level, and as such does not reflect the changes occurring at the level of the carotid baroreceptors. HUT to $+75^0$ would lead to a decrease in carotid distending pressure providing a stimulus for cardiovascular compensation to drive up mean arterial pressure.

The third input source is from the cardiopulmonary baroreceptors, on the low-pressure side of the circulation. Changes in hydrostatic force will affect not only the arterial baroreflexes but also the cardiopulmonary reflexes. On standing (i.e., on exposure to the +Gz axis) central venous pressure, venous return, stroke volume and cardiac output all decrease. The drop in central venous pressure and venous return leads to activation of the cardiopulmonary baroreflexes, and subsequent reflex increases in HR and TPR. Again, HR changes will be rapid (within 1 to 2 seconds) while vascular resistance changes will take several seconds to become evident after the stimulus.

Fourthly, the vestibular system may also be involved in regulation of the cardiovascular system via the vestibulosympathetic reflex (Doba & Reis, 1974; Essandoh & Duprez, 1998; Ray et al, 1997; Shortt & Ray, 1997; Yates, 1992; Yates & Miller, 1998). The vestibular system will signal the dynamic postural change taking place, which may be supplemented by ocular inputs (the vestibulo-ocular reflex). The state of the cardiovascular system may then be altered by the action of the VSR, which may provide feed-forward adjustment of arterial pressure during dynamic postural change.

The efferent output of the vestibulosympathetic reflex will be reflected in changes in vascular resistance, based on experimental findings in animals and humans (Doba & Reis, 1974; Essandoh & Duprez, 1998; Ray et al, 1997; Shortt & Ray, 1997; Yates, 1992; Yates & Miller, 1998). The time course of changes in vascular resistance will be in the order of several seconds. A change in HR is not likely, given that this has not been reported as a feature of VSR activity.

4.2 Experimental findings

The phases seen in Figures 1 and 2 are in 5-beat intervals, which amount to approximately 4 to 6 seconds. Due to the inherent time lags in the tissue response to efferent signals of the neural control mechanisms responsible for cardiovascular regulation, the effect in a particular phase is generally a response to a stimulus that occurred in the previous one to two phases.

4.2.1 Anticipation period

During the 5-beat anticipation period, there may be changes occurring in the cardiovascular system due to an alerting response to the impending postural challenge. These changes will result in an increase in HR and changes in regional vascular resistance. While the HR change will occur rapidly, the changes in vascular resistance will take longer to develop. As such, changes in TPR due to arousal prior to tilt are likely to be seen in the tilt period phases rather than within the anticipation period itself.

4.2.2 Phase I

Phase I coincides with the dynamic phase of tilt, in which the postural change is made from 0^0 to $+75^0$. During this phase MAP rises almost immediately in both groups, and reaches a maximum at the conclusion of this phase. This rise in MAP is due to observed increases in both HR and TPR, since SV falls immediately in both groups during this phase.

Which of the four control inputs discussed above is responsible for driving the increase in HR during Phase I? An increase in arousal at the onset of HUT could account for this observed increase in HR, given that the temporal characteristics of this increase closely mirror the time taken to achieve the full HUT position (approximately 4 seconds). The HR changes seen in this early phase of HUT may be due to these arousal effects alone, and mediated by withdrawal of parasympathetic control. The fact that the FP group experienced a smaller increase in HR during Phase I could reflect a lower level of psychological arousal than in the NP group, due to the former's frequent exposure to a dynamic motion environment. This is supported by the FP group having little anticipatory rise in HR compared with the NP group, whose HR increased in anticipation of impending tilt.

The change in HR could be due to the action of the arterial or cardiopulmonary baroreflexes. However, these reflex arcs must be stimulated first, and as such some postural change must take place before baroreflex-mediated increases in HR occur. There is not likely to be a stimulus to the high- or low-pressure receptors until at least midway through this phase. Baroreflex-mediated HR increases are thus unlikely to be seen until the end of Phase I. HR increases immediately in both groups, well before the full head-up tilt position is reached, which suggests that other inputs such as arousal are responsible for the early Phase I HR increases.

Since there is no established connection between vestibular control of the cardiovascular system and HR changes, the action of the VSR is not likely to be responsible for the increase in HR.

The increase in TPR in this phase is interesting, given that changes in vascular resistance take time to occur after the initiating stimulus. The stimulus for this increase must be something that occurred prior to tilt, such as the alerting response to impending postural change.

This increase in TPR in both groups during Phase I is important, as it combines with the HR increase to boost MAP. There are several speculative explanations for this phenomenon. The first reflects the changes in vascular resistance effected by the increase in arousal during the anticipation period. Since these changes take time to develop, they may not be evident until Phase I. Vasoconstriction of some regional vascular beds (such as renal and splanchnic regions) occurs as a consequence of increased arousal. Due to the low level of skeletal muscle vasoconstrictor drive in the horizontal resting position of the anticipation period, there is likely to be little additional vasodilation occurring in these vascular beds as a result of arousal. The net result of these changes would be an increase in TPR due to the anticipatory stimulus, which is seen in Phase I.

The second explanation involves the vestibular system and its influence on the cardiovascular response to HUT. The activation of a vestibulosympathetic reflex due to the dynamic postural changes as HUT proceeds may facilitate the observed increases in TPR during Phase I. The vestibular system is in effect responding in a dynamic fashion to the postural change stimulus. The time course of this phenomenon is in accord with experimental findings that vestibular stimulation can evoke sympathetic discharges within 100 milliseconds (Yates, 1992). However, the response of vascular smooth muscle will take longer to occur, and changes in resistance values will take longer again (in the order of several seconds). The vestibular system could initiate vascular resistance changes, but these would probably not occur until late in Phase I at the earliest.

The third possible explanation may be a mechanical feature of the blood vessels themselves. As HUT proceeds, the hydrostatic force will progressively dump more blood into the dependent lower limb vessels. This sudden increase in vascular volume as HUT occurs may initiate a smooth muscle reflex in the blood vessels, in keeping with the length-tension relationship of muscle. Such a short-lived response may lead to the transient increase in TPR seen during Phase I.

The postural changes in Phase I will eventually lead to stimulation of the arterial and cardiopulmonary baroreceptors, particularly late in Phase I when the full HUT position is reached. However, the time interval involved during Phase I is too short for arterial and cardiopulmonary baroreceptor activity to have much effect in this phase. Efferent output from these baroreflexes will be seen in later phases.

What is responsible for the precipitous fall in SV during Phase I? In the NP group, SV falls in the anticipation period, reflecting a shortened ejection time as a consequence of increased HR. HR continues to increase throughout Phase I, which will exacerbate the fall in SV. As the tilt progresses, more hydrostatic force is generated. This is unlikely to be a significant input to the cardiovascular system until the second half of Phase I, and it is only at the end of the phase that it becomes maximal, once the full HUT position is achieved. The dramatic falls in SV observed in Phase I are thus due to the combination of HR changes due to the arousal effects from the anticipation period (early in Phase I) and progressive increases in hydrostatic force reducing venous return.

SV falls less in the FP group during Phase I than it does in the NP group. As a result, MAP reaches a higher peak value for the same effective increase in TPR as the NP group, while the increase in HR is slightly slower. What could account for this better SV performance in the FP group? There are two possibilities. The FP group did not have a significant fall in SV or a rise in HR during the anticipation period. As a group they begin Phase I in a better cardiovascular state. This would help defend SV against further falls due to a developing hydrostatic force. Another explanation may be an expanded circulating blood volume in the FP group. An expanded blood volume would also help to preserve SV in the face of an orthostatic challenge. There is emerging evidence that such blood volume expansion does occur in +Gz-trained individuals (Convertino, 1998). In the FP group, the important effect of even slightly improved SV performance is a greater value of MAP during this early dynamic postural change.

Therefore, it appears that the changes in HR and TPR seen in Phase I are due to the effects of a prior alerting reaction in anticipation of an impending postural change. Although the FP group has less HR rise during this phase, it is able to generate a higher level of MAP due to enhanced SV performance.

4.2.3 Phases II and III

Phase II begins with the full HUT position having been achieved. Phases II and III are marked by the progressive effects of the hydrostatic force on the cardiovascular system, and the system's attempts to compensate for these effects.

MAP falls in both groups from the peak value in Phase I towards C values. In the NP group it falls well below the C value, reaching a minimum in the late part of Phase III. In contrast, the FP response to tilt in these phases is clearly different from that of the NP group. MAP plateaus, and remains at or slightly above C levels during both phases. The difference in MAP response in Phase III is the most striking and fundamental difference between the responses of the two groups. During Phases II and III, the two groups' MAP responses diverge considerably from each other, whereas in Phase I they tracked relatively closely.

What is driving MAP down during these two phases? Heart rates in both groups during Phase II are similar, remaining at the elevated levels achieved in Phase I for most of Phase II. HR then tends to decrease during Phase III in the NP group, but increases slightly in the FP group during this phase. If the Phase I rise in HR was due to the autonomic effect of increased psychological arousal, the fact that HR tends to remain at the same elevated level during Phase II in both groups suggests that the arousal effect cannot increase HR any further. This is especially true given that arousal levels tend to be higher in the upright position compared with the supine or prone positions. HR presumably decreases towards C values in the NP group due to arousal no longer being the dominant stimulus to the cardiovascular system. The FP group, however, goes on to a further sustained HR increase during Phase III. What is responsible for this rise, which is quite different from the NP response? Further increases in HR may be due to the developing action of the arterial and cardiopulmonary baroreflexes, as a result of the ongoing effect of hydrostatic pressure. The fact that this occurs in the FP group and not in the NP group may well reflect a difference in the operating characteristics of the baroreflex in the FP group. This would suggest an enhanced level of baroreflex activity on modulation of HR.

SV continues to decrease in both groups during Phases II and III, despite a transient recovery in SV which occurs at a similar point in both groups, around the junction of Phases II and III. This temporary increase in SV may well reflect an increase in cardiac contractility, as a countermeasure against the orthostatic challenge of HUT. There is little difference in either the time course or magnitude of this contractility change between groups. This increase in contractility is mediated by the baroreflexes (arterial and cardiopulmonary). Assuming that the stimulus for this is the consequence of the full HUT position, the time course for this contractility increase would fit with the operating characteristics of cardiac tissue. Eventually, of course, this increase in contractility is unable to effectively counteract the ongoing deterioration in VR due to the upright position, and SV continues to fall.

TPR falls in both groups back to C values during Phase II after peaking in Phase I. It then effectively plateaus during Phase III. This is likely to be a reflection of the changes occurring due to the alerting reaction developed in the anticipation period. The lack of significant vasoconstrictor drive generated by the alerting reaction in the supine position is now being realised in Phases II and III. Although there may be a small contribution from vasodilation of skeletal muscle beds to this fall in TPR, it is the time lag in developing adequate vasoconstriction that is more likely to be responsible for this overall reduction in TPR. As vasoconstriction develops in Phase III, further decline in TPR is arrested. This considerable time lag between afferent input and efferent output is consistent with the operating characteristics of vascular resistance changes. The effect of arousal-induced changes in regional vascular resistance is the most likely explanation for the observed decline in TPR.

While the arterial and cardiopulmonary baroreceptors would clearly be stimulated by the decreases in MAP and VR, especially in the NP group, their ability to effect a change in vascular resistance is not evident for some time due to their inherent inertia and latency of operation. The baroreflexes are likely to contribute towards arresting further decline in both MAP and TPR and driving them up again by the very end of Phase III, but will exert their efferent effects predominantly in subsequent phases of tilt.

In both groups, the fall in MAP appears to be due to a decrease in TPR, despite the sustained increase in HR. TPR plateaus in both groups presumably due to the developing action of the baroreflexes that were initiated in Phase I. In the FP group, the fall in MAP that occurs during Phase II is arrested during Phase III by the combination of a sustained increase in HR and an increase in cardiac contractility. These increases compensate for any vasodilation-induced decrease in TPR and the ongoing deterioration in SV. Phase III demonstrates that the FP group is much better able to defend MAP against the fall in VR and SV caused by sudden exposure to an orthostatic challenge than the NP group.

4.2.4 Phases IV to VI

Phases IV to VI, the late stages of HUT, show a progressively stabilised picture, with no dynamic postural changes occurring. Hydrostatic force is constant, and the efferent outputs of all the stimulated control mechanisms are now operative. MAP rises in both groups throughout these three phases, largely due to increases in TPR. In the NP group, it is not until the end of Phase IV that MAP is restored to C levels, mediated largely by increases in TPR. In the FP group, MAP is boosted in mid-Phase IV via a combination of HR and TPR increases. HR reaches its maximum value in FP during Phase IV, but as these last three

phases progress, HR decreases. TPR increases significantly and as such assumes the dominant role in maintaining MAP.

The rise in TPR is almost certainly due to the activity of the arterial and cardiopulmonary baroreflexes. These reflexes were initiated during Phase I, with the onset of the dynamic postural change. Another reflex that will have been stimulated is the vestibulosympathetic reflex. The VSR is likely to respond to the dynamic inputs of postural change, as these may have cardiovascular consequences that the VSR is presumably designed to modulate and counter.

Clearly it has taken a long time for the vascular resistance changes to occur following this initial stimulation. This is consistent with what is known about the operating characteristics of the sympathetically-mediated vascular resistance changes. The FP group's rise in TPR occurs basically at the same time as that of the NP group. This suggests that any adaptation to +Gz does not extend to shortening the time lag involved in effecting a change in vascular resistance. This may reflect a mechanical limitation in the system. Indeed, this fact helps explain why fighter pilots continue to rely on the anti-G suit, which will boost peripheral resistance almost immediately after the onset of +Gz acceleration. The FP group's TPR rise is, however, steeper than the NP group, and reaches a maximum value earlier. This reflects an increased gain.

Another contributing factor to the increase in TPR may be the putative feed-forward function of the VSR. After detecting a postural change, the vestibular system may send an excitatory signal to the medullary vasomotor centre to effect a change in vascular resistance before the efferent arm of the arterial baroreflexes becomes fully active. Such a feed-forward mechanism would clearly be an advantage to the pilot operating in the high +Gz environment. A point worthy of note is that although the vestibular input has changed from the dynamic input of Phase I to a stable static input in the full HUT position, it is likely that this static input continues to act as a command signal for the vestibulosympathetic neural link.

While both groups in this experiment presumably had some vestibulosympathetic input, it is possible that the VSR in the FP group could adapt to the demands of the high +Gz environment (and its cardiovascular effects) leading to enhancement of this feed-forward mechanism. This phenomenon would better protect the pilot from circulatory compromise due to high +Gz, and may contribute to the gain increase in vascular resistance changes observed in the FP group.

The HR and TPR changes in the FP group are very closely related. The sustained elevation in HR is effectively switched off only when TPR begins to increase substantially. This effect is not seen in the NP group, with HR progressively decreasing well before TPR rises to any great extent. It seems reasonable to suggest that this pattern of response in the FP group indicates an adaptation strategy. The +Gz-adapted baroreflexes are able to increase HR and sustain it at higher levels until such time as the increase in TPR is sufficiently established for it to assume the dominant position. Knowing that TPR increases will take a finite amount of time, the only other protective option is to keep HR up. Only when the vascular resistance changes are safely underway will the increased HR be allowed to switch off. This effect is not seen in the NP group. As such, it is highly suggestive of enhanced baroreflex function as a result of adaptation to repetitive +Gz acceleration.

4.3 Significance of the findings

Previous studies have demonstrated the existence of a difference in the cardiovascular response to an applied +Gz load in the FP group compared with the NP group (Newman et al, 1998, 2000). MAP, SP and DP all increased significantly, with PP being maintained in the FP group, whereas in the NP group MAP and SP were unchanged, DP increased and PP fell dramatically. HR, SV and TPR all demonstrated some degree of enhanced performance in the FP group relative to the NP group. These findings suggested that the FP group had more effective activation of their baroreflexes in response to a given accelerative stimulus. The FP group appeared to have enhanced baroreflex function due to their frequent and repetitive exposure to high +Gz loads.

The findings in this time course analysis support these earlier results. Indeed, from this analysis it is apparent that in fact the time course of changes in the cardiovascular response to dynamic postural change is similar between the groups, but that adaptation to +Gz appears to lead to a greater magnitude of response. The +Gz-adapted pilot demonstrates increased sensitivity of the arterial and cardiopulmonary baroreflex arcs, which in turn reflects an increase in the gain of these reflexes. This enhanced function is demonstrated by a sustained increase in HR and a more marked increase in TPR relative to the NP group.

It is likely that both arterial and cardiopulmonary baroreflexes contribute to the rise in HR and TPR seen in both groups, and that their enhanced function in the FP group acts to drive HR up (and to sustain it for longer) and to increase TPR to a greater extent over a similar time course.

Both the arterial and cardiopulmonary baroreflexes have been shown to be capable of a certain degree of functional plasticity and altered function. The central fluid shifts accompanying long-duration spaceflight have been shown to cause attenuation of both cardiopulmonary and arterial baroreflexes (Billman et al, 1981; Bungo & Johnson, 1983; Fritsch-Yelle et al, 1994; Thompson et al, 1990). Significantly, changes in cardiovascular parameters with resultant orthostatic intolerance have been observed after only 5 hours exposure to the microgravity environment. Microgravity analogue experiments, such as 60 head-down bedrest studies, have produced similar results. These studies confirm that removal of the normal gravitational gradient results in impaired baroreflex function, with these important mechanisms becoming less sensitive and as such less effective in dealing with transient changes in arterial pressure (Convertino et al, 1990).

In contrast, the research reported in this paper involving increased levels of +Gz suggests an opposite effect, with the baroreflexes becoming more effective at reacting to transient changes in cardiovascular dynamics. Other researchers have also shown enhanced baroreflex function in different settings (Krieger, 1970). It seems logical to argue that if a) both low- and high-pressure baroreflexes can develop attenuated function, and b) high-pressure baroreflexes can develop enhanced function, then the low-pressure cardiopulmonary baroreflexes must also be capable of enhanced function. The findings in this analysis would tend to support this.

These results confirm the findings in previous studies that the cardiovascular response of fighter pilots to a mild accelerative stimulus is different from that of a group of non-pilots (Newman et al, 1998, 2000). Furthermore, this analysis shows that this difference is mediated

by differences in the magnitude-time course balance of the dynamic cardiovascular response to applied +Gz, specifically in terms of HR and TPR. These results provide some additional insight into the mechanisms involved in postural baroreflex adaptation to high +Gz in fighter pilots. In addition, this adaptation may not be limited to the arterial baroreflexes alone; the cardiopulmonary baroreflexes may similarly adapt to the same stimulus. Indeed, it seems likely that all reflex arcs involved in the regulation of arterial pressure undergo some form of adaptation to repetitive +Gz exposure.

The roles of the vestibular system in cardiovascular control in general and in adaptation to +Gz in particular have also been highlighted in this analysis. It is quite possible that the vestibular system also adapts to frequent exposure to high +Gz, by enhancing its normal feed-forward vestibulosympathetic action. The enhanced function of the baroreflexes may well be aided by earlier signals of changing hydrostatic force being sent via the vestibular system as a means of early alerting and correction of potentially deleterious postural changes. This certainly warrants further research attention.

5. Conclusion

The findings in this analysis support the results of previous studies, in that repetitive occupational exposure to the high +Gz environment is capable of inducing a degree of physiological adaptation. This adaptation appears to be due in part to enhanced arterial and cardiopulmonary baroreflex sensitivity, which in this analysis is illustrated by sustained rises in HR and more marked elevations in TPR. The effect of this magnitude-time course balance shift is to produce a more marked elevation in MAP in the +Gz-adapted pilot. The analysis also suggests that an increase in effective circulating blood volume may also make a contribution to the adaptation process. In addition, the results point indirectly to the possibility of a vestibulosympathetic input into the regulation of arterial pressure during an orthostatic challenge.

6. References

Billman GE, Dickey DT, Teoh KK, Stone HL. (1981). Effects of central venous blood volume shifts on arterial baroreflex control of heart rate. *Am J Physiol*, Vol. 241 (Heart Circ. Physiol. 10): pp. H571-H575.

Bungo MW, Johnson PJ. (1983). Cardiovascular examinations and observations of deconditioning during space shuttle orbital flight test program. *Aviat Space Environ Med,* Vol. 54, pp. 1001-4.

Convertino VA, Doerr DF, Eckberg DL, Fritch JM, Vernikos -Danellis J. (1990). Head-down bed rest impairs vagal baroreflex responses and provokes orthostatic hypotension. *J Appl Physiol*, Vol. 68, pp. 1458-64.

Convertino VA. (1998). High sustained +Gz acceleration: physiological adaptation to high-G tolerance. *J Grav Physiol,* Vol. 5, No. 1, pp. P51-4.

Doba N, Reis DJ. (1974). Role of the cerebellum and the vestibular apparatus in regulation of orthostatic reflexes in the cat. *Circ Res*, Vol. 34, pp. 9-18.

Essandoh LK, Duprez DA, Shepherd JT. (1998). Reflex constriction of human resistance vessels to head-down neck flexion. *J Appl Physiol*, Vol. 64, pp. 767-70.

Fritsch-Yelle JM, Charles JB, Jones MM, Beightol LA, Eckberg DL. (1994). Spaceflight alters autonomic regulation of arterial pressure in humans. *J Appl Physiol*, Vol. 77, pp. 1776-83.

Krieger, EM. (1970). Time course of baroreceptor resetting in acute hypertension. *Am J Physiol,* Vol. 218, p. 486.

Mancia G, Mark AL. (1983). Arterial baroreflexes in humans. In: *Handbook of Physiology. The Cardiovascular System*, Sect. 2, Vol III, Ch. 20, pp. 755-793, American Physiological Society, Bethesda, MD.

Newman DG, Callister R. (1999). Analysis of the +Gz environment during air combat manouevring in the F/A-18 fighter aircraft. *Aviat Space Environ Med*, Vol. 70, pp. 310-15.

Newman DG, Callister R. (2008). Cardiovascular training effects in fighter pilots induced by occupational high G exposure. *Aviat Space Environ Med*, Vol. 79, pp. 774-778.

Newman DG, Callister R. (2009). Flying experience and cardiovascular response to rapid head-up tilt in fighter pilots. *Aviat Space Environ Med,* Vol. 80, pp. 723-726.

Newman DG, White SW, Callister R. (1998). Evidence of baroreflex adaptation to repetitive +Gz in fighter pilots. *Aviat Space Environ Med,* Vol. 69, pp. 446-51.

Newman DG, White SW, Callister R. (2000). The effect of baroreflex adaptation on the dynamic cardiovascular response to head-up tilt. *Aviat Space Environ Med*, Vol. 71, pp. 255-259.

Ray CA, Hume KM, Shortt TL. (1997). Skin sympathetic outflow during head-down neck flexion in humans. *Am J Physiol*, Vol. 273 (Regulatory Integrative Comp. Physiol. 42), pp. 1142-46.

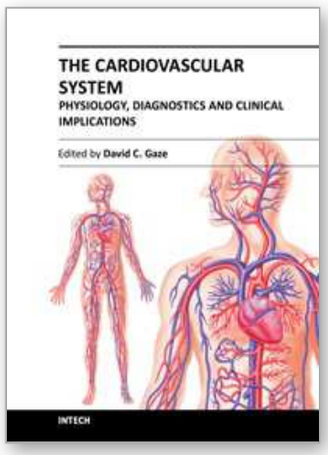
Shortt TL, Ray CA. (1997). Sympathetic and vascular responses to head-down neck flexion in humans. *Am J Physiol*, Vol. 272 (Heart Circ. Physiol. 41), pp. H1780-1784.

Thompson CA, Tatro DL, Ludwig DA, Convertino VA. (1990). Baroreflex responses to acute changes in blood volume in humans. *Am J Physiol*, Vol. 259 (Regulatory Integrative Comp. Physiol. 28), pp. R792-R798.

Yates BJ. (1992). Vestibular influences on the sympathetic nervous system. *Brain Res Rev*, Vol. 17, pp. 51-9.

Yates BJ, Miller AD. (1998). Physiological evidence that the vestibular system participates in autonomic and respiratory control. *J Vestibular Res*, Vol. 8, pp. 17-25.

Zoller RP, Mark AL, Abboud FM, Schmid PG, Heistad DD. (1972). The role of low pressure baroreceptors in reflex vasoconstrictor responses in man. *J Clin Invest,* Vol. 51, pp. 2967-2972.



The Cardiovascular System - Physiology, Diagnostics and Clinical Implications

Edited by Dr. David Gaze

ISBN 978-953-51-0534-3
Hard cover, 478 pages
Publisher InTech
Published online 25, April, 2012
Published in print edition April, 2012

The cardiovascular system includes the heart located centrally in the thorax and the vessels of the body which carry blood. The cardiovascular (or circulatory) system supplies oxygen from inspired air, via the lungs to the tissues around the body. It is also responsible for the removal of the waste product, carbon dioxide via air expired from the lungs. The cardiovascular system also transports nutrients such as electrolytes, amino acids, enzymes, hormones which are integral to cellular respiration, metabolism and immunity. This book is not meant to be an all encompassing text on cardiovascular physiology and pathology rather a selection of chapters from experts in the field who describe recent advances in basic and clinical sciences. As such, the text is divided into three main sections: Cardiovascular Physiology, Cardiovascular Diagnostics and lastly, Clinical Impact of Cardiovascular Physiology and Pathophysiology.

InTech Europe
University Campus STeP Ri
Slavka Krautzeka 83/A
51000 Rijeka, Croatia
Phone: +385 (51) 770 447
Fax: +385 (51) 686 166
www.intechopen.com

InTech China
Unit 405, Office Block, Hotel Equatorial Shanghai
No.65, Yan An Road (West), Shanghai, 200040, China
中国上海市延安西路65号上海国际贵都大饭店办公楼405单元
Phone: +86-21-62489820
Fax: +86-21-62489821

IntechOpen