Archives Italiennes de Biologie, 138: 93-97, 2000.

GRAVICEPTIVE CONTROL OF BLOOD PRESSURE IN MAN

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INTRODUCTION

The nausea, and feelings of fainting which frequently accompany vertigo are potent evidence that the labyrinth is intimately involved in autonomic functions. Hypothetically, the reason why the vestibular apparatus should be so important to autoregulation is that the otolith 'graviceptor' organs of the labyrinth, which signal tilt with respect to 'upright' (the gravitational vector) and linear accelerations, are well suited to inform the brain of rapid changes in posture which challenge the regulation of blood pressure (BP).

The mechanisms by which the labyrinth may affect BP have remained obscure until recent findings, in the cat, of fairly direct projections from the otolith to centres of cardiovascular regulation (4, 5). Hence, as a basis for investigating vaso-vagal symptoms in otological disease, we sought to demonstrate a rapidly acting graviceptive influence on arterial BP in normal man. The experimental requirement was to move the body in such a way that small changes in head orientation would allow comparison between the effects on BP of a minimal versus substantial otolithic stimulation, whilst other sensory inputs remained fairly similar.

MATERIALS, METHODS

Subjects were seated in an electric car (Fig. 1) and tasked with making head tilts (similar to a nod) in flexion-extension or vice versa and cued by an up-front visual display which the subject tracked with a helmet mounted pointer. Some head movements were accompanied, unexpectedly by an acceleration of the car, backwards or forwards, from rest, at $2m/_s^2$ for 1s, thereafter coasting gently to rest. During these acceleration pulses, the direction of the subjects inertial 'uprightness' (ie, vector sum of gravity plus acceleration pulse) would tilt by 12°: eg, for forwards acceleration of the car the upright would tilt forwards whereas the upright tilts backwards for backwards acceleration. *The head tracking was scaled so that the amplitude of head tilting approximated the magnitude of tilt of uprightness induced by car acceleration.* Thus, if the head tilted in *alignment* with this vector it remained inertially 'upright' giving little graviceptive stimulation (Fig. 1). If the head tilted in the opposite direction it was *misaligned* with the inertial upright by 24°. Misaligned tilts were similar to the passive 'jerk' of the head induced in car passengers by unexpected acceleration or braking: some readers may be familiar with a sickening 'lurch' provoked by such a manoeuvre.

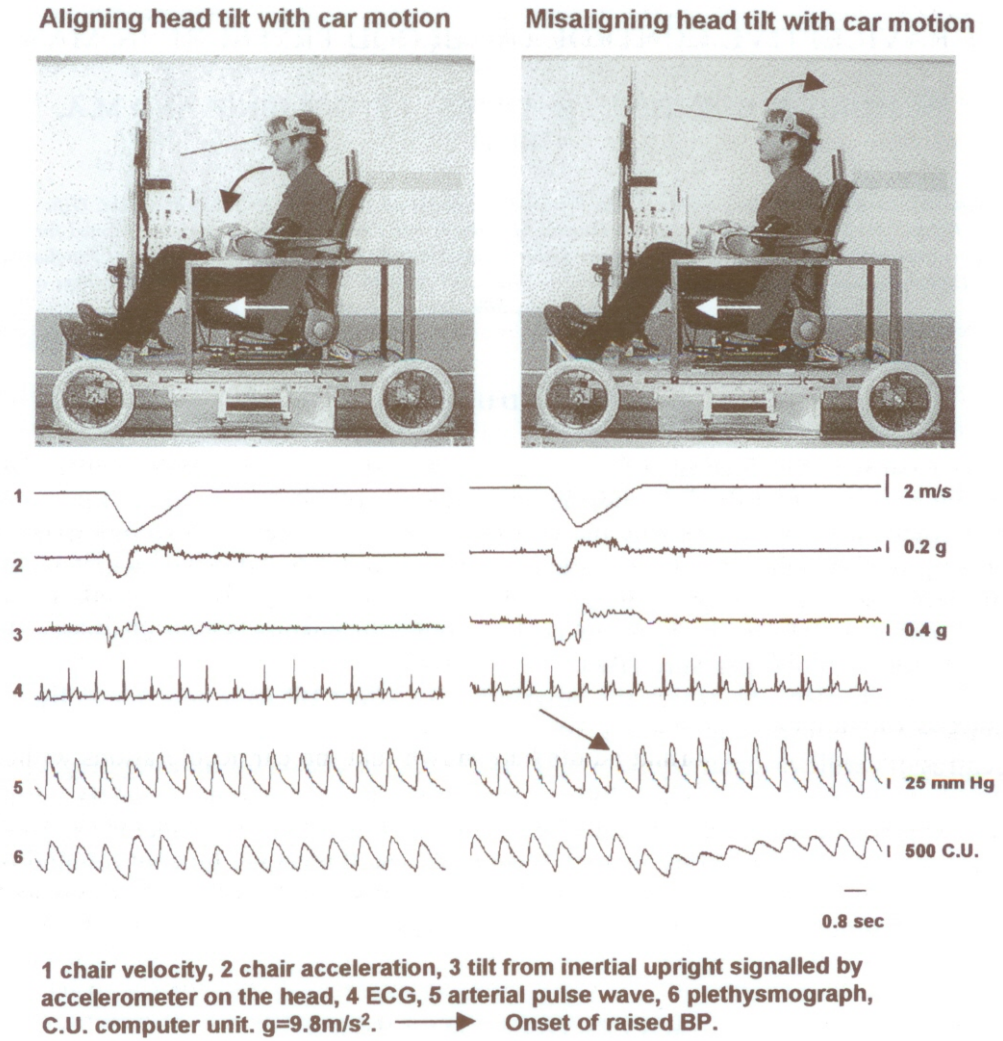

Fig. 1. - *Recording of autonomic response to aligned head tilt and misaligned head tilt with chair motion in the forward direction.*

The subjects were 8 normal males (age 27-52) who were initially trained in head movements and acclimatised to the car motion to minimise novelty or startle. Thereafter, in a balanced design, subjects performed head tilts, some of which occurred in isolation (control condition) and some of which were accompanied, without warning, by car accelerations in an unpredictable sequence. For 4 subjects the manoeuvres coupled with car motion were firstly aligned then misaligned. The order of aligned and misaligned conditions were inverted in the second 4 subjects. Within each condition of alignment, head tilts were executed in blocks of flexion or extension, in a balanced order, so that subjects would know what kind of head movement to make next and nod with precision. Each combination of head tilt and car motion was given 8 times to each subject. Approximately 40s elapsed between trials and subjects rested between aligned/misaligned manoeuvres to negate any possibility of motion sickness developing.

ECG and tonometric BP in the left radial artery were recorded with a Colin 508™. Infra red transmission plethysmography was obtained from the first finger of the right hand. The arms were restrained in adduction, oriented orthogonally to the direction of motion to minimise transduction artefacts from the passive redistribution of blood.

RESULTS

Inspection of the records showed that responses occurred within 10s of motion onset (Fig. 1) thus the peak values attained and means over 10s of the responses were assessed by comparison with means taken during a 10s baseline before stimulus onset.

Repeated measure ANOVAs on alignment x direction of head tilt x condition order showed a significant effect of alignment on systolic BP ($F31.4$; df 2, 5; $p = 0.001$). There was no effect of order on heart rate or BP but plethysmographic responses decreased throughout experimentation ($p = 0.02$).

Changes in BP with respect to baseline in the various conditions are given in Table 1. Isolated head movements caused a slight lowering of BP. Head tilts maintaining alignment with 'upright' provoked increases in BP of 4-6 mm Hg for only one or two heartbeats. Misaligned head tilts provoked highly significant peak increases in systolic (7.6-9.4 mm Hg) and diastolic (5-6 mm Hg) BP and the 10s average BP was raised significantly above baseline, particularly when the car surged forwards and the head tilted backwards.

A pilot study of 10 normal adults had shown that the car accelerations without head consistently raised systolic BP by an average of 7 mm Hg. In this upright posture the acceleration caused a misalignment of the head with respect to inertial upright of 12°. Accordingly, on the hypothesis that BP should rise more with head misalignment, paired T-tests were performed on the differences between the average pre-and post stimulus systolic BPs for the aligned and misaligned head tilts. This was significant for head tilting backwards with car accelerating forwards ($p = 0.003$) as compared with head forwards car forwards difference (ie an increase in BP specifically during misalignment). No significant difference was found for the comparison between head tilt backwards car motion backwards and head forwards-car forwards BPs; despite there being a significant rise in actual systolic pressures ($p = 0.05$, Tab. 1) with head tilt forwards-car motion backwards in comparison with baseline. BP changes were evident within the first 3 heartbeats but it would be hazardous to define a precise latency. Allowing for the mechanical delay between car and head motion and fluctuations in heart rate it is possible that the stimulus to the graviceptors could affect systole within 1 cardiac cycle

Heart rate was lowered slightly by all stimuli and all car movements reduced digital blood flow significantly (Tab. 1).

Three subjects reported a slight, transient malaise which occurred immediately after a misaligned tilt, despite the motion being gentle compared with that of an automobile. No subject rated particularly susceptible to motion sickness on a questionnaire (1).

Table 1. - *Averaged heart rate, blood pressures and plethysmographic responses recorded in baseline 10s before and during 10s after onset of car acceleration (or head motion).*

Each value shows average ± SE (8 trials in 8 subjects).

	Head tilt only		*Aligned head tilt with car motion*		*Misaligned head tilt with car motion*	
	HF	HB	HF-CF	HB-CB	HF-CB	HB-CF
Heart rate (bpm)	-1.4 ± 0.3**	-2.1 ± 0.5**	-1.8 ± 0.4**	-2.0 ± 0.5**	-0.2 ± 0.4	-1.7 ± 0.5**
Diastolic mmHg mean of 10s	-1.0 ± 0.3*	-1.5 ± 0.4*	0.44 ± 0.26	0.94 ± 0.91	0.9 ± 0.4	1.7 ± 0.6*
Peak value in 10s post Stimulus	1.0 ± 0.7	0.3 ± 0.5	5.0 ± 1.4*	4.1 ± 1.3*	5.0 ± 1.0**	6.0 ± 0.8**
Systolic (mmHg) mean of 10s	-0.3 ± 0.4	-1.1 ± 0.5	0.8 ± 1.26	0.2 ± 1.1	1.7 ± 0.6*	2.6 ± 0.6**
Peak value in 10s post Stimulus	2.6 ± 0.9*	2.1 ± 0.6*	5.6 ± 1.7*	5.8 ± 1.9*	7.6 ± 1.8*	9.4 ± 1.2**
Plethysmograph (CU)	-0.1 ± 3.9	2.2 ± 3.5	-31.7 ± 4.5**	-29.8 ± 5.2**	-39.7 ± 5.2**	-23.3 ± 4.4**

** $p < 0.002$, * $p < 0.05$ Significant differences from average of 10s pre-stimulus recording and peak values.
HF: head forwards, HB: head backwards, HF-CF: head forwards + chair forwards.
HB-CB: head backwards + chair backwards, CU: computer units.

DISCUSSION

The results demonstrate that acceleration of the body coupled with a misalignment of the head with inertial 'uprightness' provokes an increase in arterial BP, particularly when the neck extends during a surge forwards. The primary stimulus for the change in arterial BP during linear acceleration is most likely to be signal from the graviceptive organ since other sensory systems are stimulated similarly in alignment and misalignment. There is a proviso that the mechanism controlling BP must involve a graviceptive signal *in the context* of a whole body movement since head tilt alone had little effect on BP. Peripheral blood flow appears to have a less specific sensory control since plethysmographic responses were observed in all conditions.

Although adjustments of BP by the labyrinth are entirely appropriate during active movement, they become inappropriate when provoked by passive transport or disease. This susceptibility helps to explain how attacks of vertigo may have such distressing vaso-vagal consequences. Regulation of BP by the graviceptors may also be an important factor in motion sickness and, more specifically, explain why patients in autonomic distress are further compromised by riding in a conventional ambulance (2, 3). The accelerating and braking motion is likely to provoke rapid and frequent changes in BP through the acceleratory stimulation of graviceptors,

which overstress autoregulation. In contrast, a helicopter, the preferred transport for the critically ill, necessarily tilts forwards when taking off, and backwards when braking; manoeuvring which tends to maintain the alignment of passengers' heads with the inertial 'upright' and thus minimise changes in BP.

SUMMARY

The purpose of the study was to demonstrate a rapid 'graviceptive' influence on blood pressure in man. Subjects, sitting in an electrically powered car, made discrete head tilts, some of which were unpredictably accompanied by transient linear accelerations of the car *i*) with head tilting to align with the direction of the resulting inertial force vector (gravity + car acceleration) so that the graviceptors were not stimulated; *ii*) with head tilting in the opposite direction 'misaligning' which stimulated the graviceptors but otherwise maintained similarity of other sensory inputs. Stimuli were dispensed in a balanced, cross over, repeated measures design on 8 normal males. Recordings were made of arterial blood pressure in the left radial artery, the electrocardiogram and plethysmographic responses in the right hand first digit. Comparisons of 10s pre-stimulus baseline with 10s post stimulus responses. Misaligned head tilts provoked highly significant peak increases in systolic (7.6-9.4 mm Hg) and diastolic (5-6 mm Hg) BP and average BP over 10s was significantly raised. Head tilts maintaining alignment with the inertial force vector provoked raised systolic BP by 4-6 mm Hg for only one or two heartbeats. Head movements alone caused a slight lowering of BP. Effects were evident within 1-2 heartbeats of the acceleration onset. The results demonstrate that the graviceptors have a direct influence on BP in normal man. They also help to explain the profound vaso-vagal symptoms of patients with vertigo and why patients with autoregulatory impairment may be further compromised by uncontrolled accelerating and braking when they are transported in an ambulance.

Acknowledgement. - B.J. Yates was supported by a travelling fellowship from the The Ruth Margaret Dix Foundation.

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