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Original Article

Hemodynamic effects of liquid food ingestion in mid-thoracic paraplegia: is supine postprandial hypotension related to thoracic spinal cord damage?

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Background: Postprandial hypotension (PPH) appears in various conditions with autonomic failure and was symptomatic in a patient with thoracic paraplegia, but was not remarkable in patients with tetraplegia.
Objective: To determine whether the pathology causing PPH may include a thoracic but not a cervical spinal cord lesion (SCL).
Design: An experimental controlled study.
Setting: The spinal research laboratory, Loewenstein Hospital, Raanana, Israel.
Subjects: Thirteen healthy subjects, 10 patients with traumatic T_4–T_6 paraplegia, and 11 patients with traumatic C_4–C_7 tetraplegia.
Main outcome measures: Heart rate (HR), blood pressure (BP), HR and BP spectral components (LF, HF, LF/HF), cerebral blood flow velocity (CBFV), and cerebrovascular resistance index (CVRi).
Methods: The effects of a standard liquid meal on the outcome measures were compared between the three subject groups monitored for HR, BP, and CBFV, from 55 min before to 45 min after the start of the meal. The recorded signals were digitized online and analyzed off-line in the time and frequency domains.
Results: After meal, BP decreased only in the paraplegia group ($P<0.01$), HR increased more prominently in this group ($P<0.01$), CVRi tended to decrease only in the paraplegia group, CBFV did not change significantly in any group, and HR LF/HF increased ($P<0.001$) in all groups but tended to increase more in paraplegia.
Conclusions: Patients with mid-thoracic SCL may develop PPH. The pathology causing PPH can include a thoracic but not a cervical SCL. The normal hemodynamic reaction to liquid meal ingestion is mediated through the mid-thoracic spinal cord. The sympathovagal balance increases after food ingestion, more prominently in patients with PPH, and cerebrovascular resistance changes during PPH may help maintain the cerebral circulation.
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Keywords: postprandial hypotension; spinal cord lesions; hemodynamic changes; spectral analysis; cerebral blood flow velocity; cerebrovascular resistance

Introduction

After food ingestion, systemic blood pressure (BP) remains virtually unchanged in normal subjects but falls in people with certain pathological states. Whether symptomatic or asymptomatic BP fall after a meal is defined as postprandial hypotension (PPH). PPH has been described in people with various conditions associated with autonomic failure, including old age, diabetes mellitus, Parkinson's disease, and Shy–Drager syndrome.[1–3] In these conditions, PPH is attributed to the lack of normal sympathetic response to the decrease in splanchnic vascular resistance, which presumably follows food ingestion.[1]

Surprisingly, marked PPH could not be demonstrated in patients with severe cervical spinal cord lesions (SCL),

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in either the supine or tilted position, despite their obvious autonomic dysfunction.[1,4] However, PPH was demonstrated, in a single case report, after thoracic SCL with complete neurological deficit below the T_3 segment. In that case, the hypotension was symptomatic when food ingestion was followed by head-up tilting (HUT).[5]

The demonstration of significant PPH in a patient with thoracic paraplegia but not in tetraplegia raised the hypothesis that the pathology causing PPH includes a thoracic but not a cervical SCL. To verify this hypothesis, we studied responses to food ingestion in a group of individuals with thoracic SCL and compared them with those of patients with cervical SCL and of healthy participants.

Methods

Subjects

Thirteen healthy control subjects and 21 patients with traumatic SCL of 3 months to 41 years duration, 10 with T_4–T_6 paraplegia, and 11 with C_4–C_7 tetraplegia were included in the study. The control subjects were nine men and four women, 34 ± 13 years old. The patients with paraplegia were eight men and two women, 38 ± 13 years old, with ASIA grade[6] A. The patients with tetraplegia were 11 men, 42 ± 8 years old, eight with ASIA grade A and, three with ASIA grade B. The age differences between the groups were nonsignificant ($P = 0.28$). Patients with medical conditions that might affect the results, such as febrile disease, heart failure, renal failure, diabetes mellitus, or an additional neurological impairment, were not included.

Procedure

The study was approved by the ethical committee of Loewenstein Rehabilitation Hospital, and all participants signed an informed consent. In the morning, after a 12-h fast (allowing free drinking of water before the meal), each subject lay supine in a relatively quiet hospital environment, with about 22°C ambient temperature. All subjects were continuously monitored for heart rate (HR) and BP, from 55 min before a standard liquid meal, which lasted a few minutes, to 45 min after the start of the meal. The meal consisted of 60 g Isocal powder (Mead Johnson, The Netherlands) and 55 g Polycose powder (Ross Products Division, Abbott laboratories, USA) in 300 ml of milk. The meal contained 0.35 g Na, 102.1 g carbohydrates, 14.8 g fat, 18 g protein, amounting to 614 kcal. Cerebral blood flow velocity (CBFV) was also continuously monitored, but only before the meal and 40–45 min after the meal start, because of patient sensitivity to prolonged pressure of the measurement probe.

This was one in a series of experiments performed on the same subjects, and it was preceded by a 10-min 35° HUT and followed by a second HUT and a cold-pressor test (described elsewhere). Food ingestion started 15 min after the end of the first HUT.

Recording and analysis

For HR recording, continuous ECG traces were obtained using surface electrodes and a preamplifier A/D system (BIOPAC Systems, USA). The Finapres (Ohmeda, USA) system was employed for noninvasive continuous BP recording by means of a cuff applied to the subject's thumb. The ECG and the thumb arterial pressure signals were simultaneously sampled online at 500 Hz. The digitized ECG signal was converted off-line into an HR signal, and the digitized arterial pressure signal was low-pass filtered and resampled off-line at 10 Hz.

The processed HR and BP signals underwent time and frequency analyses. The frequency analysis (a spectral analysis of BP and HR variations) was performed after a median filtering of 251 samples length. It used a discrete fourier transform (DFT) combined with a Welch Periodogram method to compute the power (amplitude squared) of the sampled signal fluctuations (HR and BP) as a function of their frequency.[7] The integrals of the power values between 0 and 0.17 Hz (LF) and between 0.17 and 0.5 Hz (HF) were calculated to obtain the low- and high-frequency components of the power spectrum.

A transcranial Doppler (TCD) ultrasonograph (Smartlite, Rimed, Israel) was used to record CBFV. The TCD probe, applied to the subject's right temple, transmitted a 2 MHz pulsed wave through the ultrasonic window in the temporal bone. The Doppler frequency shift of the reflected wave was recorded by the device to compute the flow speed in a proximal segment of the middle cerebral artery (MCA).[8] The CBFV signals were digitized online at a 2 Hz rate and submitted to an off-line time-dependent analysis.

The cerebrovascular resistance index (CVRi) was obtained by dividing the BP by the mean CBFV (mCBFV) (Figures 1 and 2).

Statistical analysis

The mean values of HR, BP, HR or BP LF, HF and LF/HF, mCBFV, and CVRi were calculated for four time intervals: 40–30 min before the meal (at supine rest before the preceding HUT), and at 0–15, 15–30, and 30–45 min after the meal start. By analysis of variance with repeated measurements, we examined the effects of the liquid meal on the hemodynamic-dependent variables within and between groups (paraplegia, tetraplegia, and healthy participants), and the effects of the groups themselves on the variables. Correlations between changes in variables were examined by Pearson's correlation test. Before the statistical analysis, the spectral components were subjected to a square root transformation and their ratios were subjected to a natural logarithm (ln) transformation, to approach normal distributions. Data were analyzed by SPSS for Windows, version 11 (SPSS Inc., USA).

Results

Heart rate

Before the meal, the difference in HR values between groups was nonsignificant ($P > 0.16$). The liquid meal

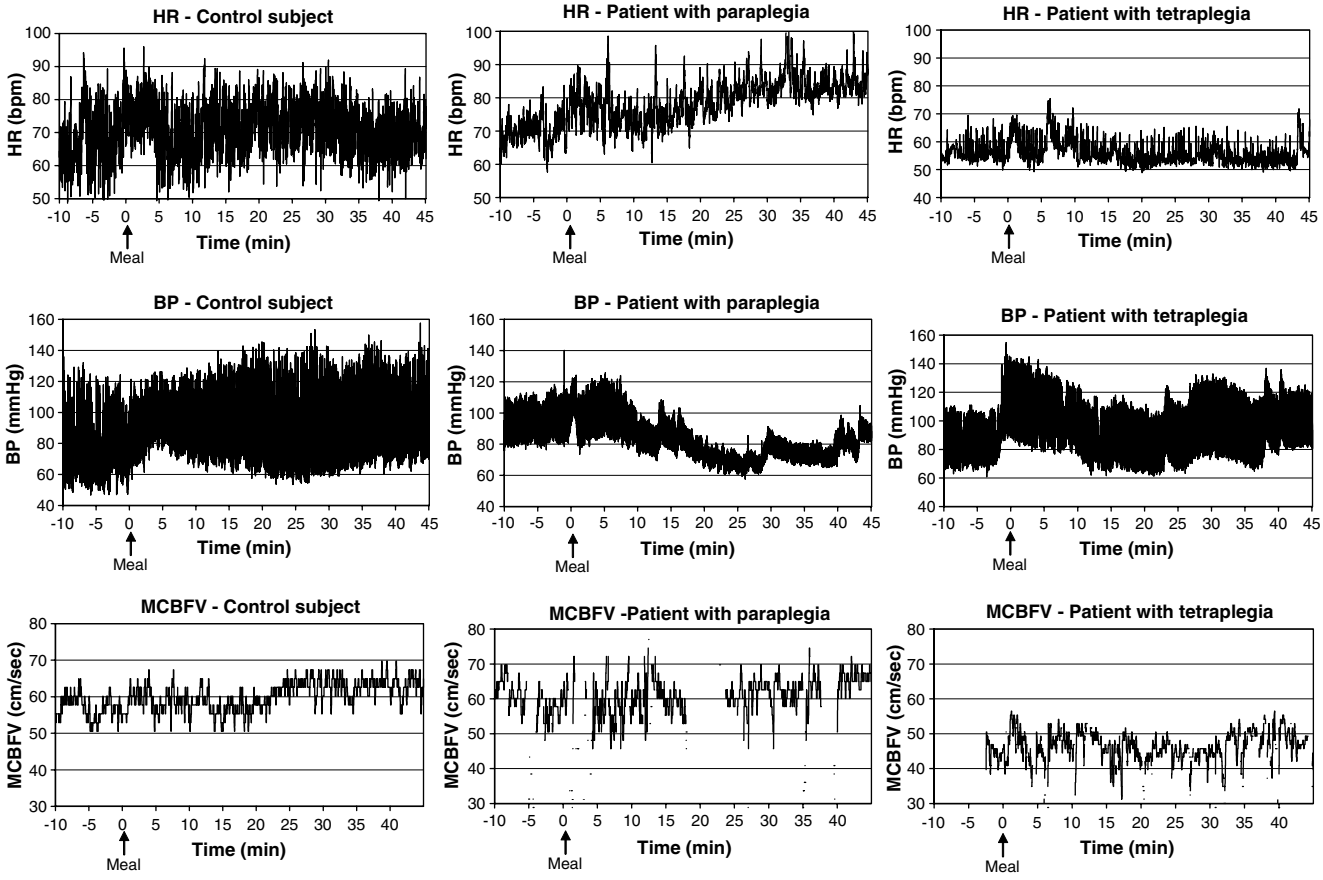

Figure 1 Examples of HR, BP, and mCBFV recordings in a control subject and in patients with paraplegia or tetraplegia, before and after the meal

significantly affected HR ($P<0.001$): after the meal HR increased in all subject groups. The increase was significantly affected by the group ($P=0.003$): it was highest in the patients with paraplegia (Table 1a).

HR power spectrum LF component
The liquid meal significantly affected HR LF ($P<0.001$), which increased soon after the meal but returned within 15 min after the meal start to slightly above the pre-meal value (Table 1b), irrespective of group ($P>0.8$).

HR power spectrum HF component
The meal also had a significant effect on the HR HF ($P=0.001$), which also increased soon after the meal, but returned within 15 min after the meal start to the pre-meal value or below (Table 1c), irrespective of group ($P>0.45$).

HR power spectrum LF/HF
The meal had a significant effect on ln HR LF/HF as well ($P<0.001$). The ratio steadily increased after meal, irrespective of group ($P>0.25$), but its mean values increased more in the patients with paraplegia (Table 1d). The increase in HR LF/HF was significantly correlated with HR increase after meal ($r=0.736$; $P=0.006$) in the control group, but not in the patients. No significant group effect on ln LF/HF was found before or after the meal ($P>0.3$).

Blood pressure
Before the meal, mean BP values were similar in the three groups ($P>0.07$). The liquid meal affected BP differently in the various groups ($P=0.005$). BP increased after the meal in the control group, showed fluctuations in the tetraplegia group, but decreased in the paraplegia group (Table 2a). Group effect on the values of BP was also significant after the liquid meal ($P<0.01$): BP was lower in the patients, mainly in the tetraplegia group, than in the control group.

BP power spectrum LF component
The liquid meal affected BP LF, which significantly increased after the meal start ($P=0.001$) irrespective of group ($P>0.8$). However, BP LF returned within 15 min after the meal start to below the pre-meal value and then to slightly above the pre-meal value in the

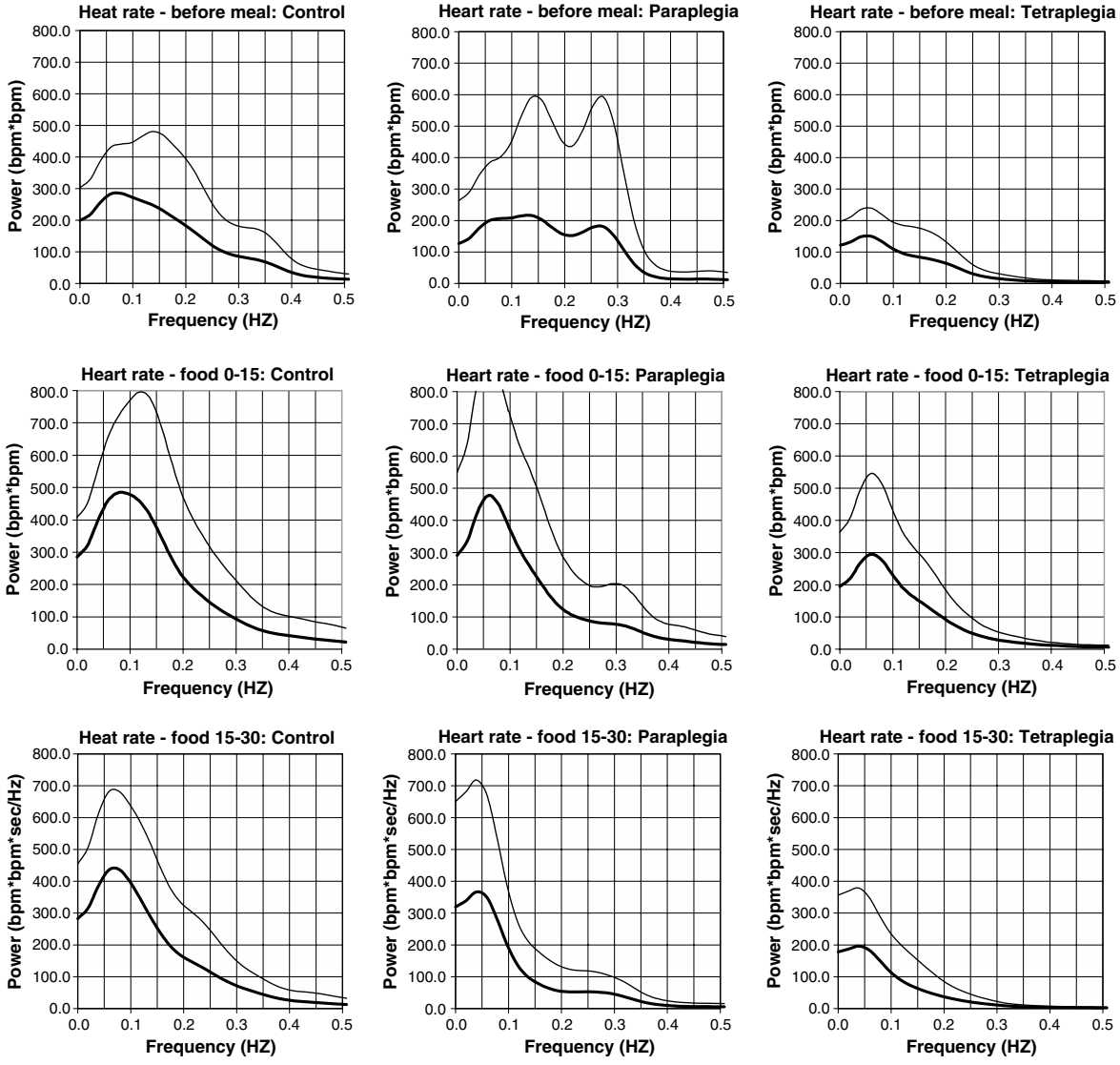

Figure 2 HR power spectrum: before, 0–15 min after, and 15–30 min after meal start. The power (amplitude squared of HR fluctuations) values are the average (lower curve) and average + SD (upper curve) of each group at each fluctuation frequency

paraplegia group, and to slightly above the pre-meal value in the tetraplegia group (Table 2b). BP LF values were lower in the patients than in the control group ($P<0.001$).

BP power spectrum HF component
The meal had a significant effect on BP HF as well ($P=0.004$). BP HF increased soon after the meal irrespective of group ($P>0.19$) (Table 2c). This increase was maximal within the first 15 min after the meal. The BP HF values were not significantly different in the various subject groups ($P>0.06$).

BP power spectrum LF/HF
Meal and group effects on ln BP LF/HF were not significant.

Cerebral blood flow velocity
An ultrasonic window was found in the subjects' temporal bone and CBFV signals were obtained in all the TCD examinations. mCBFV values did not differ significantly before and after the meal in any of the groups ($P=0.13$). Both before and after the meal, CBFV values were significantly affected by the group ($P=0.02$): they were highest in the healthy participants and lowest in patients with tetraplegia (Table 3a).

Cerebrovascular resistance
The CVRi mean values increased after the meal in the control and the tetraplegia groups, but decreased in patients with paraplegia (Table 3b). However, the difference between means was only near statistical significance ($P=0.08$), and when all the groups were considered, significant differences were not found

Table 1 Meal effect on (a) HR and its (b–d) spectral components in the three subject groups

	Mean ± SD		
	Control	*Paraplegia*	*Tetraplegia*
(a) HR (b.p.m.)			
Before meal	63.17 ± 7.47	65.07 ± 8.75	59.99 ± 9.36
0–15 min after meal start	68.51 ± 6.45	77.15 ± 11.99	64.24 ± 10.82
15–30 min after meal start	69.57 ± 8.51	81.78 ± 12.38	68.83 ± 12.63
30–45 min after meal start	72.28 ± 10.79	83.43 ± 10.12	69.73 ± 12.91
(b) Square root HR LF (b.p.m.)			
Before meal	1.86 ± 0.65	1.34 ± 0.46	1.25 ± 0.51
0–15 min after meal start	2.32 ± 0.71	1.91 ± 0.66	1.75 ± 0.75
15–30 min after meal start	2.09 ± 0.71	1.55 ± 0.55	1.40 ± 0.62
30–45 min after meal start	2.01 ± 0.71	1.75 ± 0.60	1.35 ± 0.57
(c) Square root HR HF (b.p.m.)			
Before meal	1.39 ± 0.91	0.82 ± 0.43	0.77 ± 0.45
0–15 min after meal start	1.44 ± 0.85	0.91 ± 0.46	1.04 ± 0.51
15–30 min after meal start	1.31 ± 0.81	0.72 ± 0.39	0.72 ± 0.48
30–45 min after meal start	1.23 ± 0.78	0.83 ± 0.59	0.67 ± 0.34
(d) Ln HR LF/HF			
Before meal	0.85 ± 0.86	1.15 ± 0.72	1.15 ± 0.55
0–15 min after meal start	1.19 ± 0.72	1.64 ± 0.72	1.12 ± 0.48
15–30 min after meal start	1.16 ± 0.83	1.66 ± 0.54	1.57 ± 0.84
30–45 min after meal start	1.20 ± 0.96	1.82 ± 0.82	1.45 ± 0.55

Table 2 Meal effect on (a) BP and its (b–c) spectral components in the three subject groups

	Mean ± SD		
	Control	*Paraplegia*	*Tetraplegia*
(a) BP (mmHg)			
Before meal	89.48 ± 10.17	92.68 ± 14.64	84.15 ± 13.87
0–15 min after meal start	99.15 ± 15.27	89.71 ± 16.38	86.66 ± 12.81
15–30 min after meal start	99.23 ± 17.78	80.56 ± 15.49	80.68 ± 17.05
30–45 min after meal start	100.10 ± 17.05	83.33 ± 14.82	84.52 ± 19.29
(b) Square root BP LF (mmHg)			
Before meal	1.89 ± 0.50	1.32 ± 0.31	1.20 ± 0.74
0–15 min after meal start	2.42 ± 0.49	1.80 ± 0.46	1.96 ± 0.70
15–30 min after meal start	2.51 ± 0.59	1.14 ± 0.17	1.48 ± 0.62
30–45 min after meal start	2.51 ± 0.93	1.59 ± 0.30	1.41 ± 0.55
(c) Square root BP HF (mmHg)			
Before meal	0.87 ± 0.38	0.84 ± 0.40	0.60 ± 0.25
0–15 min after meal start	1.15 ± 0.31	0.87 ± 0.36	0.85 ± 0.22
15–30 min after meal start	1.04 ± 0.27	0.79 ± 0.40	0.76 ± 0.25
30–45 min after meal start	0.98 ± 0.27	1.02 ± 0.46	0.79 ± 0.28

between the values of the right MCA segment CVRi before and after the meal ($P = 0.697$).

Discussion

The thoracic spinal cord and PPH

The findings of the experiment show that PPH can occur in patients with thoracic paraplegia but not with tetraplegia, and support the hypothesis that the pathology causing PPH includes a thoracic but not a cervical SCL.

BP decreased following meal in the paraplegia subjects but not in the others. However, prominent tachycardia, and probably a decrease in cerebrovascular resistance allowed preservation of the pre-meal CBFV in the paraplegia patients despite the BP decrease, and PPH in the supine position was asymptomatic.

Table 3 Meal effect on (a) CBFV and (b) CVRi in the three subject groups

	Mean ± SD		
	Control	*Paraplegia*	*Tetraplegia*
(a) Rt mCBFV (cm/s)			
Before meal	66.31 ± 10.47	61.36 ± 13.53	53.44 ± 9.18
40–45 min after meal start	70.86 ± 9.34	62.19 ± 13.96	53.94 ± 9.86
(b) Rt CVRi (mmHg/cm/s)			
Before meal	1.46 ± 0.28	1.64 ± 0.46	1.58 ± 0.36
40–45 min after meal start	1.49 ± 0.31	1.48 ± 0.39	1.81 ± 0.49

Hemodynamic changes following SCL are customarily attributed to disruption of the sympathetic signals that travel from the brainstem through the spinal cord to target organs.[9] However, the findings in paraplegia patients in this experiment cannot be explained merely by such a disruption. If they were a consequence of sympathetic disruption, they should have also appeared after a cervical SCL. Therefore, it is plausible that the normal reaction to liquid meal ingestion requires an intact mid-thoracic spinal cord that conveys hemodynamic regulatory signals other than those descending through the cervical spinal cord.

The notion of an alternative source of hemodynamic regulatory signals is supported by spectral analysis. The HR high-frequency fluctuations, which are considered to be vagally mediated,[10] increased in all groups, indicating an increase in vagal activity immediately after the meal start. Therefore, the change in vagal transmission cannot explain the HR increase soon after meal start. An increase in sympathetic activity, indicated by the increase in low-frequency HR fluctuations, which are presumably of brainstem vasomotor center and baroreceptor origin,[10] cannot explain the HR increase after the meal either, because sympathetic signals could not have been transmitted from the brainstem to the heart in patients with severe cervical SCL. Therefore, it is plausible that signals that do not require the descending cervical spinal cord pathways and may reside in the mid-thoracic spinal cord induced the low-frequency HR variations following the liquid meal ingestion.

These findings, and the inability to demonstrate in the patient groups the significant correlation found in the control group between HR and HR LF/HF increases following the meal, imply that hemodynamic changes following a meal are controlled by at least two neural mechanisms, with different signal generators: one in the brainstem vasomotor center, which can affect the heart without an intact spinal cord, and another, perhaps in the thoracic spinal cord, which cannot.

Postprandial vagal and sympathetic responses

The HR LF/HF, which represents the sympathovagal balance,[10] significantly increased after the meal irrespective of group, as well as in healthy young subjects in a previous study,[11] and in healthy elderly subjects in another study.[12] However, in the last study, young subjects showed no postprandial HR LF/HF changes. HR LF did not change significantly after meal in these studies, and HR HF decreased significantly only in the first previous study.

Whereas these previous studies analyzed spectral changes over periods of 30 min, we observed an increase in HR LF, HR HF, and HR LF/HF during the first 15 min after the meal start. After that period, HR LF and HR HF decreased, HR HF more than HR LF, and HR LF/HF remained higher than before the meal, as in the other studies. These results imply that sympathetic activity, and to a lesser extent vagal activity, increase immediately after the meal. Then both, but mainly the vagal activity fade and the sympathetic activity remains dominant. The increase in sympathovagal balance, represented by HR LF/HF, tended to be more prominent in the paraplegia patients, who exhibited PPH, as in the older subjects in the second previous study, who also suffered from PPH.[12] These phenomena probably represent the dynamics of the compensatory response of the sympathetic and parasympathetic autonomic limbs to food ingestion, and its associated circulatory decompensation.

Changes in BP LF were similar to those in HR LF, but the interpretation of BP HF and LF/HF is not clear because they depend on breathing, which was not controlled.

Postprandial cerebral blood flow velocity and cerebrovascular resistance

Food ingestion with or without PPH did not change CBFV in the present study, nor did it in previous ones.[13,14] In this study, cerebrovascular resistance tended to decrease in the group with paraplegia and PPH, but it increased in a previous study, in elderly subjects with PPH.[14] This difference may indicate that the pathology causing PPH is different in paraplegia and in old age, and that cerebrovascular resistance changes during PPH may contribute to cerebral ischemia in certain circumstances,[14] but may assist in maintaining the cerebral circulation in others.

Study limitations

The study design imposed a few limitations having to do with the completeness of the SCL, the multiplicity of tests and analyses, the non-continuity of the CBFV measurement, and the potential effects of fluid intake.

As in most human spinal cord studies, the exact anatomical extent and boundaries of the lesions were not known, although all of them were complete or almost complete according to clinical definitions (using ASIA grading). This does not exclude sparing of autonomic nerve fibers at the lesions sites, but the probability of interference of such sparing with the findings in groups with almost-complete lesions is small.

The experiment presented in this study was preceded and followed by additional tests on the same participants; multiple analyses were performed on the results of these tests. The additional tests were performed to prevent losing important information that can be obtained only from patients who match the inclusion criteria and who are difficult to recruit. These tests included the preceding 10 min HUT, which could have confounded the supine postprandial findings, although the 15-min interval between the HUT and the meal start was likely sufficient to eliminate this effect.

HUT-induced renin–angiotensin elevation may have contributed to the prevention of BP fall in tetraplegia soon after the meal start.[1] However, this effect was probably negligible as renin half-life in the circulation is 15–30 min,[15] and plasma renin activity measurements (to be presented in detail elsewhere) were not significantly different in patients with tetraplegia at supine rest before the preceding tilt or 15 min after meal start. The negligibility of this effect is further supported by a previous study,[4] in which renin activity and pre-meal BP were not affected by HUT that ended 10 min before a similar meal.

Atrial natriuretic hormone (ANH) plasma levels, which previous studies showed to be elevated in patients with paraplegia, and even more in those with tetraplegia,[16] could have also been affected by the preceding tilt and could have confounded the postprandial findings. But ANH half-life is only a few minutes, and it probably did not change the effect of the meal on BP either.

The multiplicity of analyses increased the chance of incidental findings. Therefore, only P-values <0.01 or <0.001 were considered significant for the main inferences.

The noncontinuous CBFV measurement after the meal might cause some CBFV measurement error, but its effect on group data is probably minimal.

Free drinking of water was allowed before the meal to minimize the potential effect of hypovolemia on measurements. The amount of water intake before the meal was not controlled, assuming that subjects would drink according to their volume requirements. This might have resulted in different effects on each group because the drinking of water may have had hemodynamic effects (it increases cardiac output and supine BP in healthy subjects[17,18] and attenuates PPH in patients with autonomic failure).[19] However, all the subjects actually drank less than one glass of water over 90 min before the meal, which minimizes this potential problem.

Gastric distension by the volume of the fluid-meal, which increases BP in healthy people,[20] more so in older than in younger ones,[21] could also have attenuated the drop in BP in tetraplegia. However, this effect was manifested with a 600 ml but not with a 200 ml fluid load,[20] and the 300 ml fluid load of the meal in the present experiment is unlikely to have had different effects, if any, on the study groups.

Conclusions

This research demonstrates asymptomatic PPH in a group of patients with mid-thoracic SCL, shows that the pathology causing PPH can include a thoracic SCL but not a cervical one, and indicates that the normal hemodynamic reaction to a liquid meal is mediated through the mid-thoracic spinal cord.

The findings imply that sympathetic and vagal activities increase immediately after food ingestion and then fade, leaving the sympathetic activity dominant and the sympathovagal balance increased, more prominently in patients with PPH. The results also imply that the pathology underlying PPH is different in paraplegia and in old age and that cerebrovascular resistance changes during PPH may help maintain the cerebral circulation.

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