Autonomic Neuroscience: Basic and Clinical 185 (2014) 112–119

Contents lists available at ScienceDirect

Autonomic Neuroscience: Basic and Clinical

journal homepage: www.elsevier.com/locate/autneu

Quiet standing after carbohydrate ingestion induces sympathoexcitatory and pressor responses in young healthy males

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ARTICLE INFO

Article history:
Received 18 February 2014
Received in revised form 22 June 2014
Accepted 23 July 2014

Keywords:
Orthostatic stress
Meal ingestion
Blood pressure
Sympathetic nervous activity
Stroke volume

ABSTRACT

Objective: To investigate the role of the sympathetic nervous system in the cardiovascular response to quiet standing in the postprandial state.
Method: Following a 30 min pre-ingestion phase, 14 healthy young male subjects consumed a 600 kcal carbohydrate-rich meal. Arterial blood pressure (BP) and heart rate (HR) were recorded for a further 120 min. Measurements were obtained (Finometer) in both the supine (5 min) and standing (5 min) condition every 30 min. Power spectral analysis of RR-interval and BP variability was calculated, and heart rate responses to the baroreceptor reflex were calculated to estimate spontaneous baroreflex sensitivity (sBRS). Derived stroke volume (SV) was measured to track changes to postural stress postprandially.
Results: Quiet standing increased RR-interval low frequency power, ratio of RR-interval low frequency power/high frequency power (ratio of RR LF/HF), and systolic BP low frequency power (SBP LF power), and decreased RR HF power and sBRS before, and after eating. After meal ingestion, SBP LF power increased and sBRS decreased in lying and standing conditions. During quiet standing postprandially, DBP and the mean arterial pressure increased ($P < 0.01$). The increased BP is associated with increased SV ($P < 0.05$) early postprandially, and increased SBP LF power ($P < 0.01$) in the later postprandial phase. SBP LF power is inversely correlated with SV postprandially ($P < 0.001$, $R^2 = 0.96$).
Conclusion: The findings suggest a sympathetic activation mediated by baroreflex resetting. Quiet standing in the postprandial state enhances sympathetic outflow to the vasculature, increasing BP. SV may be a compensatory factor stabilising BP during quiet standing early postprandially.

1. Introduction

Meal ingestion induces an appreciable increase in blood flow in splanchnic organs and skeletal muscular tissue (Sidery et al., 1991; Someya et al., 2008), so as to facilitate nutrient delivery and metabolism (Baron, 1994). To meet the challenge of splanchnic hyperaemia, and increased peripheral blood flow, there is a rise in cardiac output (CO) to prevent a fall in blood pressure (Sidery et al., 1991; Waaler and Eriksen, 1992). The rise in CO is mostly attributable to increased stroke volume (SV), due to a considerable dilatation of the cardiac chambers (Kelbaek et al., 1989). In the postprandial state there is a peripheral vasodilation that is likely due to humoral, and other non-neural, factors. This vasodilatory effect lasts for at least 2 h, and is accompanied by a rise in CO (Waaler and Eriksen, 1992). The peripheral vasodilation is also associated with a graded, reflex, increase in sympathetic nerve activity (Berne et al., 1989; Fagius and Berne, 1994). Postprandial peripheral vasodilation and sympathetic activation are particularly prominent after high-carbohydrate food ingestion (Fagius and Berne, 1994; Sidery et al., 1991). Sympathetically-mediated vasoconstrictor effects are crucial in stabilising blood pressure (Anderson et al., 1991; Scherrer and Sartori, 1997). Failure of this neural compensatory mechanism may lead to postprandial hypotension (Fagius et al., 1996; Lipsitz et al., 1983, 1993).

Responses to assuming an upright posture in the initial stage, i.e. short-term quiet standing, is mainly controlled by autonomic nervous system (Smit et al., 1999). Short term standing unloads cardiopulmonary receptors and baroreceptors (Abboud et al., 1979; Taylor et al., 1995), and is associated with a reflex sympathetic activation (Cooke et al., 1999; Furlan et al., 2000) and regional vasoconstriction to counteract gravitation-induced venous pooling (Abboud et al., 1979); and also a reduction in parasympathetic activity with an increase in heart rate (Cooke et al., 1999; Taylor et al., 1995). Maintenance of normal arterial blood pressure is critical in order to ensure appropriate circulation of blood to different parts of the body as required. A failure to maintain normal arterial blood pressure can lead to postural

Abbreviations: LF, low frequency; HF, high frequency; nu, normalised units; sBRS, spontaneous baroreflex sensitivity; MAP, mean arterial pressure; BP, blood pressure; HR, heart rate; SV, stroke volume; CO, cardiac output; TPR, total peripheral resistance.

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http://dx.doi.org/10.1016/j.autneu.2014.07.007
1566-0702/

hypotension (Smit et al., 1999), or hypertension (Fessel and Robertson, 2006; Streeten et al., 1985), if counter-regulatory systems such as the baroreceptor reflex fail to operate correctly.

Individuals spend approximately 50% of the daytime in a postprandial state. In the modern Western world, meals rich in carbohydrates are common, and assuming an upright posture (including sitting or standing) after eating is a normal daily activity. However, the autonomic and haemodynamic effects of a brief loading with carbohydrate-rich food and its interaction with brief periods of short-term of standing are not clearly understood. Here we aimed to determine if there is an interaction between food intake and posture on arterial blood pressure and changes in sympathetic nerve activity and baroreflex function.

In addition, postprandial hypotension frequently occurs within ~30 min after eating (Lipsitz et al., 1983). Whether or not an impairment in cardiac filling contributes to postprandial hypotension is also unclear (Jansen and Lipsitz, 1995). Therefore, the current study also aimed to examine the role of stroke volume (cardiac filling) in maintaining BP after eating.

2. Methods

2.1. Study participants

Fourteen males were recruited from a population of post-graduate students. Subjects were non-smokers on no medication (age 31.4 ± 2.9 years; height 179.6 ± 7.9 cm; weight 75.9 ± 7.2 kg; body mass index (BMI) 23.6 ± 2.6 kg/m^2 (healthy weight range 18.50–24.99 kg/m^2)). Subjects were instructed not to drink water 1.5 h prior to experiments, and to abstain from caffeinated beverages and food for 12 h, alcohol for 24 h, and moderate or strenuous physical activity for 48 h prior to the experimental sessions. After receiving a detailed verbal and written explanation of the intended experimental protocol and measurements, each subject provided an informed consent. All experimental procedures and protocols were approved by the Human Ethics Committee of Macquarie University, Sydney, NSW, Australia.

2.2. Recording

Electrocardiogram (ECG) and BP (finger photoplethysmography; Finometer Pro, Ohmeda, Amsterdam, Holland) were measured continuously. ECG was sampled at 1000 Hz and stored for off-line analysis (LabChart 7.2 and Powerlab8/30, ADInstruments, Bella Vista, Sydney, NSW, Australia). BP files were recorded at a sampling rate of 200 Hz for further power spectral analysis. The subject's left arm was placed with the left hand (testing hand) at the level of the heart at all times (Imholz et al., 1998). Brachial arterial blood pressure was recorded from the right arm with an automated sphygmomanometer (Microlife A100 PLUS, AG, 9443 Widnau, Switzerland) to confirm the accuracy of the Finometer measurements of absolute blood pressure. All other haemodynamic variables were downloaded with Finolink and derived from Beatscope software (FMS, Finapres Medical Systems BV, Amsterdam, The Netherlands). Details of the methodology used by the Finometer software ("beatscope") to calculate different haemodynamic parameters are available from http://www.finapres.com/site/index.php (Wesseling et al., 1993). With this approach it is also possible to calculate CO, SV and total peripheral resistance (TPR) (Bogert and Van Lieshout, 2005).

Before starting the experiment, subjects were asked to maintain breathing frequency at 0.2 Hz by following a metronome until the subjects were confident and comfortable with this breathing pattern. To avoid mental stress, subjects were then asked to approximate this frequency and depth spontaneously during the experiment. Breathing was also carefully supervised by investigators, and adjusted if the breathing deviated excessively during recording in both supine and upright positions throughout the study (Bernardi et al., 2000; Bloomfield et al., 2001; Taylor et al., 1995).

2.3. Experimental protocol

All subjects were fasted overnight and studies were conducted from 0900 to 1230. The laboratory temperature is centrally air-conditioned and maintained at 23 °C throughout the experiment. To minimise stress, the study was undertaken in a semi-dark laboratory room and subjects were instructed not to read, listen to music or chat with the investigator during the experiment. Subjects were awake throughout the study.

Baseline measurements of all parameters (brachial BP, 5 min of ECG and finger blood pressure recording) were obtained in the lying state after a 20 minute rest period. Subjects then stood and after 2 min of haemodynamic equilibration data was recorded again.

Subjects were then fed a standard breakfast. Breakfast was an ordinary Australian meal, including 30 g Weet-Bix Bites (wild berry) (Sanitarium Health and Wellbeing, Australia), 100 ml Original Milk (Dairy Farmer Pty Ltd., Australia), 170 ml Low Fat Fruit Yogurt (Dairy Farmer Pty Ltd., Australia), 200 ml Orange Juice (The Daily Juice Company, Australia), and one medium-sized banana (all sourced from Woolworth Ltd., Australia). The food formula is a 600 kcal carbohydrate-rich mixed meal (semi-liquid): 118 g carbohydrate (including 85 g sugar) (78%), 20 g protein (13%), 6 g fat (8%), and sodium (300 mg). The carbohydrate-rich meal was consumed within 10–12 min (Cozzolino et al., 2010; Fagius and Berne, 1994; Lipsitz et al., 1993; Sidery et al., 1991; Someya et al., 2008). The first 30-minute *time point* postprandially was defined as starting from the first mouth of food intake (10–12 min of eating time), until 18–20 min after the meal with supine resting state. Recordings were repeated at each of the following 30-minute time-interval for a further 2 h. Each time-interval incorporates 12 min recording time (5 min lying and 7 min standing) followed by 18 min in the supine resting state (Fig. 1).

2.4. Data analysis

Power spectral analysis of RR interval was calculated with the HRV module in the commercial software of LabChart (LabChart 7.2, ADInstruments, Bella Vista, Sydney, NSW, Australia). Power spectrum analysis of systolic and diastolic blood pressure (SBP and DBP) was performed using custom written scripts with Spike2 software (Cambridge Electronic Design Limited, Cambridge, England). All analyses were performed from stable haemodynamic regions with a duration of 5 min that was free of ectopic beats and any technical artefacts.

Fast Fourier Transform (Hanning window; 512 block size) was applied to the R–R interval, SBP and DBP in time series to obtain power spectral estimates of HRV (Malik, 1996) and BPV (Cooke et al., 1999; Pagani et al., 1986). Low-frequency (LF) power refers to the area under curve amplitude between 0.04 and 0.15 Hz and high frequency power refers to the area under curve amplitude between 0.15 and 0.40 Hz. Signal powers of each band were calculated as integrals under the respective power spectral density functions and expressed in normalised unit for HRV and absolute units for BPV (mm Hg^2).

Spontaneous baroreceptor reflex sensitivity (sBRS) (RR interval to systolic BP) was evaluated with HemoLab software (http://haraldstauss.com/HemoLab/HemoLab.php), using the sequence

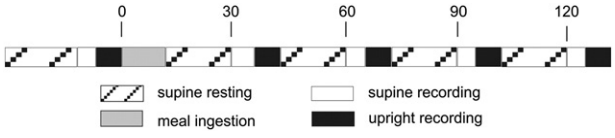

Fig. 1. Experimental protocol. Baseline (pre-ingestion) and postprandial measurements of short-term ECG and finger blood pressure (5 min lying followed by 5 min standing) were obtained at each 30 min time-interval. Prior to each recording ($5min_{lying} + 5min_{standing} = 10$ min), there is a 20 min supine resting period.