Canadian Science Publishing

ARTICLE

Cardiac-autonomic and hemodynamic responses to a hypertonic, sugar-sweetened sports beverage in physically active men

Mark Christiani, Gregory J. Grosicki, and Andrew A. Flatt

Abstract: Hydration practices may confound heart rate variability (HRV) measurements when collected in the pre-training period. We aimed to determine the effects of ingesting a hypertonic, sugar-sweetened sports beverage on HRV and hemodynamic parameters in physically active young men. Fifteen subjects consumed 591 mL of Gatorade (6% carbohydrate, ~330 mOsmol/kg), 591 mL water, or 10 mL water (control) in random order on separate days following overnight fasting. HRV and hemodynamics were evaluated in 5-min windows immediately before (T1) and 5–10 min (T2), 25–30 min (T3), 40–45 min (T4), and 55–60 min (T5) post-drinking. Root-mean square of successive differences and the standard deviation of normal RR intervals increased post-water intake at all time-points relative to T1 ($P < 0.05$). No increases were observed post-Gatorade intake, though small effect sizes were noted at T2 and T3 ($P > 0.05$, ES = 0.27–0.32). Systemic vascular resistance increased at T2 post-Gatorade intake and at T2 and T3 post-water intake ($P < 0.05$). No interactions were observed for blood pressure measures, stroke volume, or cardiac output. Gatorade does not evoke cardiovascular adjustments to the same magnitude as water. Practitioners should wait at least 45 min to record HRV post-Gatorade intake and >60 min post-water intake.

Novelty:

- Equal volumes of cold water and Gatorade produce inequivalent cardiac-autonomic and hemodynamic responses.
- HRV responses of greater amplitude and duration were observed following intake of water versus Gatorade.
- Failure to account for recent fluid intake may result in misinterpretation of autonomic status.

Key words: Gatorade, heart rate variability, parasympathetic, vagal.

Résumé : Les pratiques d'hydratation peuvent confondre les mesures de variabilité de la fréquence cardiaque (« HRV ») lorsqu'elles sont prises au cours de la période de pré-entraînement. Cette étude a pour objectif de déterminer les effets de la prise d'une boisson sportive hypertonique sucrée sur l'HRV et les variables hémodynamiques chez de jeunes hommes physiquement actifs. Après un jeûne nocturne, 15 sujets prennent dans un ordre aléatoire en des jours distincts 591 mL de Gatorade (6 % de glucides, ~330 mOsmol/kg), 591 mL d'eau ou 10 mL d'eau (contrôle). L'HRV et l'hémodynamique sont évaluées par segments de 5 min immédiatement avant (« T1 ») et 5 à 10 min (« T2 »), 25 à 30 min (« T3 »), 40 à 45 min (« T4 ») et 55 à 60 min (« T5 ») après avoir pris la boisson. La moyenne quadratique des différences successives et l'écart type des intervalles RR normaux augmentent après la prise d'eau à tous les moments comparativement à T1 ($p < 0,05$). On ne note aucune augmentation après la prise de Gatorade bien que de petites ampleurs de l'effet soient enregistrées à T2 et T3 ($p > 0,05$, ES = 0,27–0,32). La résistance vasculaire systémique est plus grande à T2 après la prise de Gatorade et à T2 et T3 après la prise d'eau ($p < 0,05$). Aucune interaction n'est observée à propos des valeurs de pression artérielle, du volume systolique ou du débit cardiaque. La boisson de Gatorade n'engendre pas d'ajustements cardiovasculaires de même ampleur que l'eau. Les praticiens doivent attendre au moins 45 minutes pour enregistrer l'HRV après la prise de Gatorade et plus de 60 minutes après la prise d'eau. [Traduit par la Rédaction]

Les nouveautés :

- Des volumes égaux d'eau froide et de Gatorade produisent d'inégales réponses cardio-autonomes et hémodynamiques.
- Comparativement à la prise de Gatorade, on enregistre après la prise d'eau des ajustements plus importants de l'amplitude et de la durée de l'HRV.
- Le fait de ne pas tenir compte de l'apport hydrique récent peut entraîner une mauvaise interprétation de l'état autonome.

Mots-clés : Gatorade, variabilité de la fréquence cardiaque, parasympathique, vagal.

Received 14 February 2021. Accepted 21 March 2021.

M. Christiani, G.J. Grosicki, and A.A. Flatt. Biodynamics and Human Performance Center, Department of Health Sciences and Kinesiology, Georgia Southern University (Armstrong Campus), Savannah, Georgia, USA.

Corresponding author: Andrew A. Flatt (email: aflatt@georgiasouthern.edu).

Introduction

Heart rate variability (HRV) refers to the variation in time between successive heart beats and reflects activity of the autonomic nervous system (Malik et al. 1996). Greater HRV is indicative of increased cardiac-parasympathetic modulation (Malik et al. 1996) and is associated with superior health (Dekker et al. 2000) and stress resilience (Souza et al. 2013). Lower HRV is suggestive of vagal withdrawal and has been linked to a variety of disease states and fatigue (Laborde et al. 2018). Due to its time efficient, inexpensive, and readily accessible nature, there has been profound interest as of recent in the application of HRV in a variety of settings ranging from clinical care to sports medicine. In the field of sport and exercise science, HRV has become an increasingly popular tool to monitor training adaptations and to tailor exercise prescription among general (da Silva et al. 2019), athletic (Javaloyes et al. 2018), and clinical populations (Behrens et al. 2015).

Intrinsic (e.g., age, sex, ethnicity) and extrinsic (e.g., stimulants, chronobiology, medications) factors may influence HRV (Laborde et al. 2018). Food and beverage consumption are also widely recognized to affect HRV parameters (Monrroy et al. 2019), yet experimental control for these variables is ignored by more than half of published studies in the field (Heathers 2014). Self-performed post-waking HRV recordings limit the effects of confounding factors, but maintaining long-term compliance from athletes is challenging (Plews et al. 2014). Thus, standardized HRV acquisition supervised by team staff at the training facility is becoming more common. For instance, pre-training HRV measures have been used to assess autonomic status in futsal (Nakamura et al. 2015), rugby (Nakamura et al. 2017), soccer (Proietti et al. 2017), and American football teams (Flatt et al. 2020). Though a convenient alternative to post-waking measures, these procedures are potentially problematic since athletes are encouraged to hydrate with water or a sports beverage in the hours preceding physical conditioning or competition (Sawka et al. 2007).

Fluid ingestion is a recognized confound to HRV measurement (Heathers 2014) and previous studies have helped to elucidate the underlying physiological adjustments to water ingestion. Water drinking significantly and rapidly raises vasoconstrictor activity via the pressor response, leading to an acute but profound rise in arterial blood pressure (Jordan et al. 2000; Routledge et al. 2002). Subsequent stimulation of arterial baroreceptors triggers an increase in vagal discharge to lower blood pressure via a reduction in cardiac output, consequently increasing HRV (Brown et al. 2005; Heathers et al. 2018). Thus, HRV assessment shortly following fluid intake will capture its physiological effects rather than a resting state, resulting in misinterpretation of autonomic status. For example, staff may misinterpret fluid-induced elevations in HRV to reflect favorable recovery from physical exertion (Stanley et al. 2013) or sport-related concussion (Flatt et al. 2019). Subsequent training prescription would therefore be unmatched to the athlete's true functional state and may carry potential risk in clinical exercise settings (Behrens et al. 2015; Senthinathan et al. 2017). Intriguingly, cardiovascular responses to water intake are not observed following ingestion of an equivalent dose of physiological saline (0.9%) (Brown et al. 2005), portending to a role of fluid osmolality in shaping autonomic and hemodynamic adjustments. Meanwhile, insulin infusion and glucose challenge lower HRV, interpreted to reflect autonomic shifting in the sympathetic direction (Chapman et al. 2021; Perpiñan et al. 2019; Rowe et al. 1981). Hypertonic, sugar-sweetened sports beverages (e.g., Gatorade) are particularly popular among athletes in whom HRV measures may be of interest; however, the cardiac-autonomic and hemodynamic repercussions of ingesting these beverages are unclear.

The primary purpose of the present study was to characterize the cardiac-autonomic and hemodynamic responses to a commercially available, hypertonic, sugar-sweetened sports beverage. We also aimed to examine the time-course of cardiovascular changes following sports beverage ingestion to provide team staff, clinicians, and researchers with an understanding of the persistence of these effects to guide HRV acquisition methodology conducted beyond the immediate post-waking period. To this end, we monitored HRV and hemodynamic adjustments for 60 min following ingestion of 591 mL of Gatorade (6% carbohydrates, ~330 mOsmol/kg) (Rowlands et al. 2011) and compared them with those following ingestion of an equivalent volume of water and a control (10 mL water). We hypothesized that Gatorade would induce cardiac-autonomic and hemodynamic adjustments of a magnitude comparable to or less than what is seen with water.

Materials and methods

Participants

Fifteen recreationally active (≥150 min/week) men with >2 years resistance training experience (age = 24.9 ± 3.5 years, height = 181.6 ± 8.2 cm, weight = 83.6 ± 13.6 kg) were recruited to represent athletic populations using pre-training HRV assessment (Flatt et al. 2020; Nakamura et al. 2015, 2017; Proietti et al. 2017). All participants were free from acute or chronic illness (cardiac, pulmonary, liver, or kidney abnormalities, cancer, uncontrolled hypertension, insulin- or non-insulin dependent diabetes or other known metabolic disorders), free from orthopedic limitations, not taking any heart rate-altering medications (e.g., β-blockers or β_2 agonists), and they did not smoke or participate in other forms of tobacco use. The study was approved by the affiliated University's Institutional Review Board and prior to participation all study procedures, risks, and benefits were explained to the subject and written informed consent was obtained.

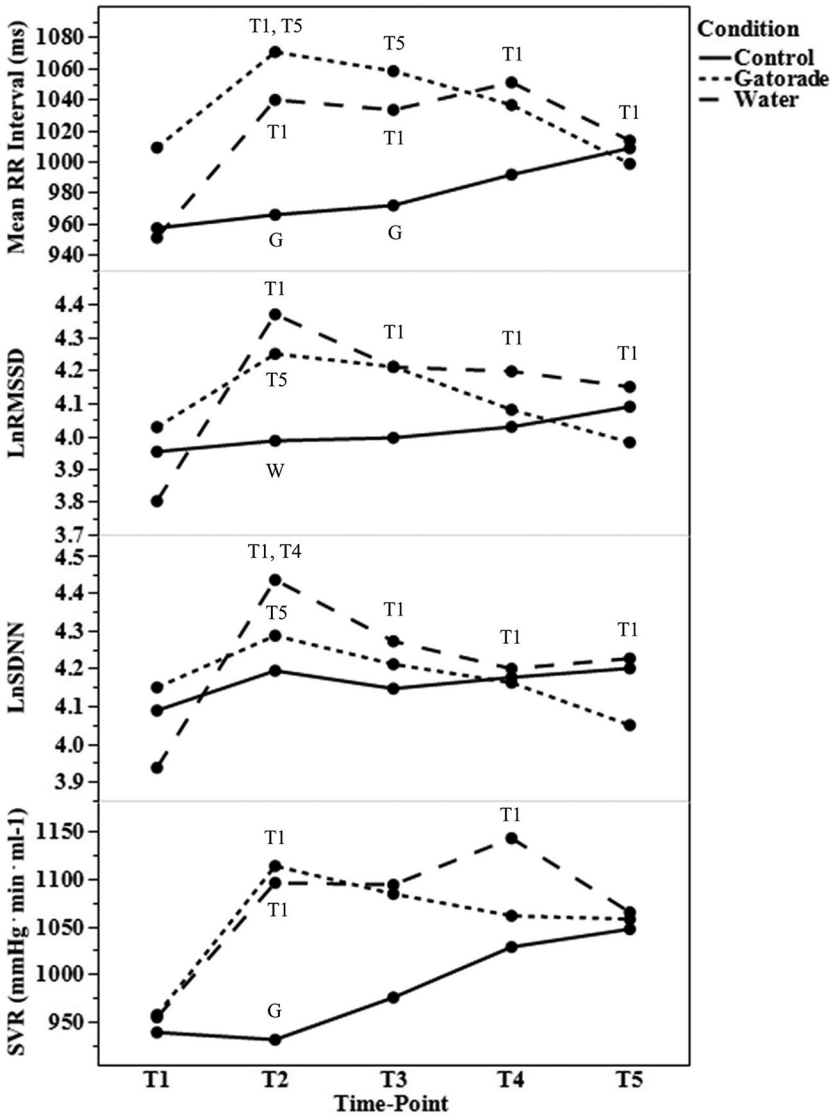
Study design

Using a crossover design, participants reported to the laboratory for 3 trials, each of which was separated by a minimum of 24 h and all of which were completed within 14 days. On the day of each trial, participants were asked to consume (1) 591 mL of bottled water, (2) 591 mL of a hypertonic sports beverage (orange flavored Gatorade: 6% carbohydrates, ~20 mmol/L Na^+, ~330 mOsmol/kg), or (3) 10 mL of bottled water (control), the order of which was counterbalanced. Subjects consumed fluids at their own pace within a 2-min window. Beverage bottles were refrigerated for 24 h prior to consumption in a commercial refrigerator (1.6 °C) for ecological validity (athletes are served beverages from coolers and refrigerators) and to control for the known effects of temperature on autonomic modulation (Girona et al. 2014). HRV and hemodynamic variables were assessed for 10 min immediately before and 60 min after fluid ingestion. All visits were scheduled during morning hours (0600–1000 hours) and participants were asked to arrive in a fasted state, having abstained from all food or drink for at least 8 hours. Vigorous physical activity and the use of caffeine or alcohol was discouraged 24 hours prior to each testing visit.

Cardiovascular measures

Upon arrival, participants were taken to a quiet, temperature-controlled room (21 °C) where they were outfitted with a clinically validated single-lead electrocardiographic (ECG) sensor (Bittium Faros 180, 1000 Hz, Oulu, Finland) (Vandecasteele et al. 2017). RR interval recordings were then performed while subjects were seated comfortably in a back-supported chair. Throughout data collection, participants remained quiet, still, and breathed spontaneously. RR intervals were extracted from ECG recordings in 5-min segments (Malik et al. 1996) immediately before fluid ingestion (T1, following a 5-min stabilization period) and then again 5–10 min (T2), 25–30 min (T3), 40–45 min (T4), and 55–60 min (T5) following the intervention. All subjects urinated in a nearby restroom following T4, after which they returned to the

Fig. 1. Condition × time interaction plots for mean RR interval, natural logarithm (Ln) of the root-mean square of successive RR interval differences (RMSSD), Ln of the standard deviation of the normal-to-normal RR intervals (SDNN), and systemic vascular resistance (SVR). Standard deviations are not displayed for clarity. Within-condition significant effects are noted by time-point. Between-condition significant effects are noted by condition. G = Gatorade, W = water.



seated position at least 5 min before T5. RR data were exported to Kubios software (University of Kuopio, Finland) for manual inspection and processing of artifact and ectopic beats with a low pass filter. HRV parameters recorded for this study were the mean RR interval, the root-mean square of successive RR interval differences (RMSSD), and the standard deviation of normal-to-normal RR intervals (SDNN). RMSSD reflects parasympathetic modulation and is commonly used for monitoring athletic training status (Buchheit 2014). SDNN reflects global variability from sympathetic and parasympathetic influences and has widespread interest as a marker of cardiovascular health (Malik et al. 1996).

Concomitant with ECG recordings, finger plethysmography was used for beat-to-beat blood pressure measurement using an automated continuous non-invasive arterial pressure (CNAP) 500 Monitor HD (CNSystems, Graz, Austria). The CNAP device uses a volume clamp method based on the Penaz principle (Penaz 1973). Participants placed their left index and middle fingers into the double finger cuff and the vascular unloading technique was used to generate an arterial pressure waveform (Smolle et al. 2015). Changes in arterial diameter and wall tension were subsequently detrended using concentrically interlocking control loops (Fortin et al. 2006) and the VERIFI-algorithm ("Vasomotoric Elimination and Reconstructed IdentiFication of the Initial set-point") (Fortin et al. 2013). Finger cuff-derived arterial pressure measures were calibrated to oscillometric brachial blood pressure measurements on the ipsilateral arm. Pulse contour analysis of the arterial pressure waveform was then used to estimate stroke volume (SV), cardiac output (CO), and systemic vascular resistance (SVR, mean arterial pressure – central venous pressure/cardiac output) using a proprietary algorithm taking into consideration the participant's sex, age, height, and body mass (Wagner et al. 2018). Hemodynamic measures were refreshed and recorded in 20-second increments and 5-min averages of these values were used at the pre- and post-time points (Watso et al. 2020). Automated brachial systolic and diastolic blood pressures were recorded immediately before each 5-min time point.

Statistical analyses

Natural logarithm (Ln) transformations were applied to the non-normally distributed RMSSD and SDNN values (Shapiro–Wilks tests, $P < 0.05$). Linear mixed models were used to evaluate changes in RR interval, LnRMSSD, LnSDNN, systolic and diastolic blood pressure, SV, CO, and SVR across time and between conditions. Condition (water vs. Gatorade vs. control) was included as a fixed effect, time (T1 vs. T2 vs. T3 vs. T4 vs. T5) as a repeated fixed effect, the condition × time interaction as a fixed effect, and subject identification as a random effect. Tukey's post hoc tests were used for follow-up assessment. Hedges' *g* effect sizes (ES) were calculated to determine standardized differences between mean values (Lininger and Riemann 2016). ES were qualitatively interpreted as follows: <0.20 = trivial, <0.60 = small, <1.20 = moderate, <2.0 = large, and >2.0 = very large (Hopkins et al. 2009). All between-condition comparisons were assessed at the same time-points only. Statistical significance was set at $P < 0.05$. Procedures were carried out using JMP 13 (SAS Institute., Cary, North Carolina, USA).

Results

A condition × time interaction was observed for RR interval ($P < 0.0001$, Fig. 1). Within-condition effects showed that RR interval increased following water ingestion at all time-points relative to T1 ($P < 0.05$, ES = 0.36–0.51). RR interval increased following Gatorade ingestion at T2 relative to T1 ($P < 0.05$, ES = 0.35) and decreased at T5 relative to T2 ($P < 0.05$, ES = −0.39) and T3 ($P < 0.05$, ES = −0.32). Between-condition effects showed that RR interval at T2 ($P < 0.05$, ES = 0.58) and T3 ($P < 0.05$, ES = 0.48) was greater following Gatorade versus control.

A condition × time interaction was observed for LnRMSSD ($P < 0.0001$, Fig. 1). Within-condition effects showed that LnRMSSD increased following water ingestion at all time-points relative to T1 ($P < 0.05$, ES = 0.51–0.83). Though non-significant ($P > 0.05$), small ES increases in LnRMSSD that may be practically relevant were observed following Gatorade ingestion at T2 (ES = 0.32) and T3 (ES = 0.27). In addition, LnRMSSD decreased following Gatorade ingestion at T5 relative to T2 ($P < 0.05$, ES = −0.42). Between-condition effects showed that LnRMSSD following water ingestion was greater at T2 versus control ($P < 0.05$, ES = 0.40).

A condition × time interaction was observed for LnSDNN ($P < 0.0001$). Within-condition effects showed that LnSDNN increased following water ingestion at all time-points relative to T1 ($P < 0.05$, ES = 0.53 – 0.98) and that T2 was greater than T4 ($P < 0.05$, ES = 0.47). Though non-significant ($P > 0.05$), a small ES increase in LnSDNN that may be practically relevant was observed following Gatorade ingestion at T2 (ES = 0.28). LnSDNN decreased following Gatorade ingestion at T5 relative to T2 ($P < 0.05$, ES = −0.47). No between-condition effects were observed.

No main effects were observed for systolic blood pressure (P = 0.392–0.653). A main effect of time was observed for diastolic blood pressure (P = 0.009). Diastolic blood pressure at T4 was greater than T1 (73.6 ± 7.5 vs. 70.5 ± 7.5 mm Hg, P = 0.004, ES = 0.36).

A main effect of time was observed for SV (P = 0.004). SV at T1 (109.0 ± 17.9 mL, $P < 0.05$, ES = 0.28 – 0.29) and T2 (109.0 ± 16.3 mL) were greater than T5 (104.3 ± 14.7 mL) ($P < 0.05$, ES = 0.28). Main effects of condition (P = 0.009) and time ($P < 0.0001$) were observed for CO. CO for control (6.7 ± 1.1 L·min^{-1}) was significantly greater than water (6.2 ± 1.0 L·min^{-1}, P = 0.01, ES = 0.46) and Gatorade (6.3 ± 1.1 L·min^{-1}, P = 0.039, ES = 0.35). Additionally, CO at T1 (6.8 ± 1.0 L·min^{-1}) was significantly higher (P = 0.0002–0.0008, ES = 0.28–0.56) than T2 (6.5 ± 1.0 L·min^{-1}) T3 (6.2 ± 1.1 L·min^{-1}), T4 (6.2 ± 1.2 L·min^{-1}), and T5 (6.3 ± 1.1 L·min^{-1}).

A condition × time interaction was observed for SVR ($P < 0.05$). Within-condition effects showed that SVR for water at T1 (954.8 ± 202 mm Hg·min·mL^{-1}) was lower than T2 (1095.5 ± 202 mm Hg·min·mL^{-1}, $P < 0.05$, ES = −0.57) and T4 (1142.3 ± 202 mm Hg·min·mL^{-1}, $P < 0.05$, ES = −0.80). SVR for Gatorade at T1 (957.3 ± 202 mm Hg·min·mL^{-1}) was lower than T2 (1113.2 ± 202 mm Hg·min·mL^{-1}) ($P < 0.05$, ES = −0.71). Between-condition effects showed that SVR at T2 for Gatorade was greater than control (931.4 ± 157.6 mm Hg·min·mL^{-1}) ($P < 0.05$, ES = 0.96).

All within- and between-condition ES are presented in Tables 1 and 2. Group and individual within-condition changes (%) in HRV parameters relative to T1 are displayed in Fig. 2.

Table 1. Within-condition effect sizes for heart rate variability parameters.

	RR Interval (ms)	LnRMSSD	LnSDNN
Control T1 vs.			—
T2	0.05	0.05	0.19
T3	0.08	0.06	0.10
T4	0.20	0.09	0.15
T5	0.30	0.20	0.19
Water T1 vs.			—
T2	0.51	0.83	0.98
T3	0.47	0.59	0.64
T4	0.58	0.58	0.53
T5	0.36	0.51	0.59
Gatorade T1 vs.			—
T2	0.35	0.32	0.28
T3	0.28	0.27	0.11
T4	0.16	0.08	0.02
T5	−0.06	−0.07	−0.17

Note: Ln = natural logarithm, RMSSD = root-mean square of successive RR interval differences, SDNN = standard deviation of normal-to-normal RR intervals, T = time-point.

Table 2. Between-condition effect sizes for heart rate variability parameters.

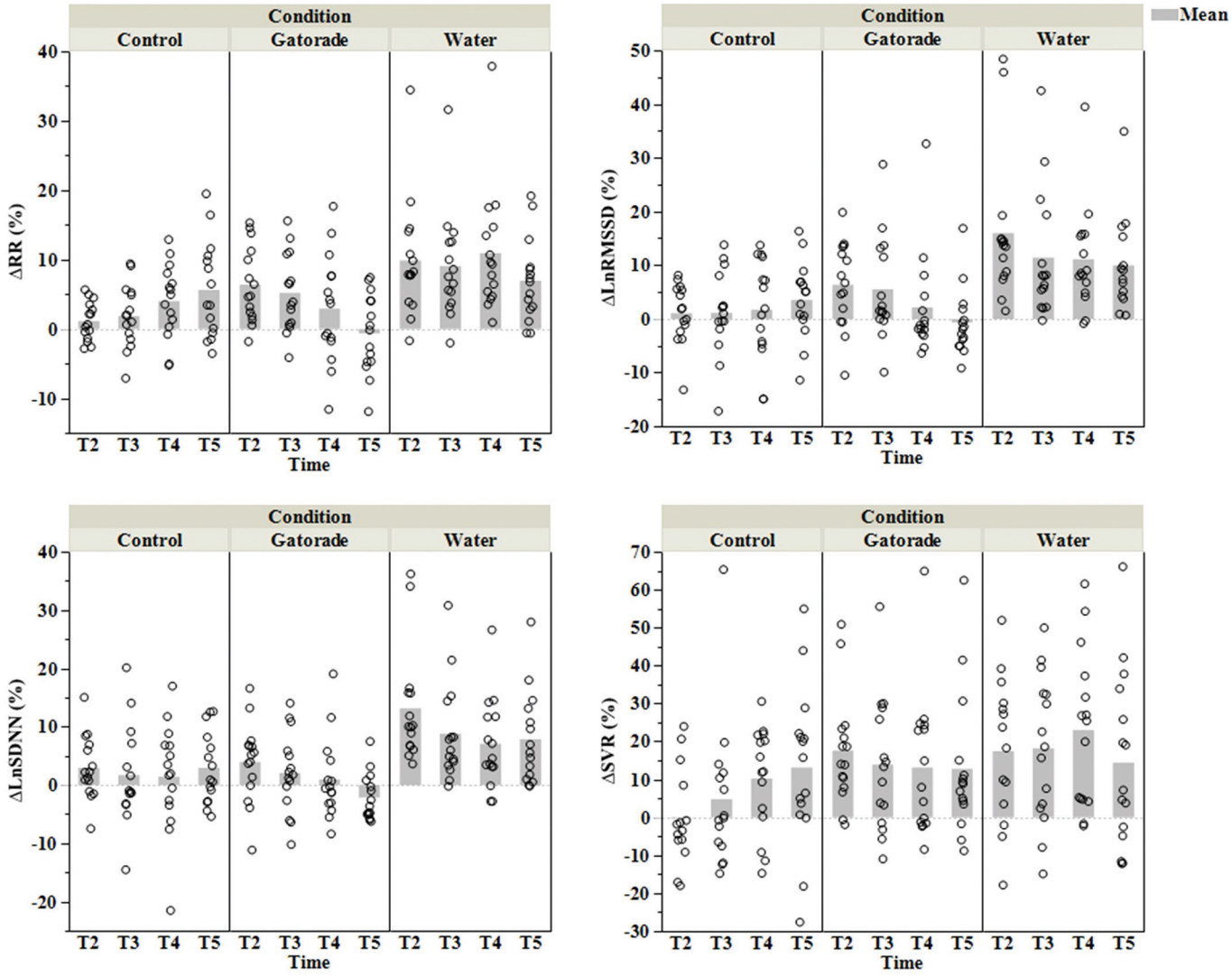
	T1	T2	T3	T4	T5
RR interval					
Gatorade vs. control	0.28	0.58	0.48	0.24	−0.06
Gatorade vs. water	0.33	0.18	0.14	−0.08	−0.09
Water vs. control	−0.04	0.47	0.38	0.37	0.03
LnRMSSD					
Gatorade vs. control	0.10	0.40	0.31	0.08	−0.15
Gatorade vs. water	0.32	−0.19	0.00	−0.18	−0.27
Water vs. control	−0.21	0.57	0.29	0.25	0.09
LnSDNN					
Gatorade vs. control	0.10	0.18	0.12	−0.03	−0.27
Gatorade vs. water	0.38	−0.31	−0.12	−0.08	−0.35
Water vs. control	−0.27	0.47	0.22	0.04	0.05

Note: Ln = natural logarithm, RMSSD = root-mean square of successive RR interval differences, SDNN = standard deviation of normal-to-normal RR intervals, T = time-point.

Discussion

The main finding of our study was that drinking a hypertonic, sugar-sweetened sports beverage (i.e., Gatorade) did not evoke the rapid and prolonged cardiac-autonomic and hemodynamic responses associated with water ingestion. In line with previous research (Routledge et al. 2002), shortly following water ingestion we observed an increase in SVR and RR interval with a corresponding increase in HRV parameters. Meanwhile, changes in SVR and RR interval following Gatorade were qualitatively *small* and short-lived (i.e., 5–10 min post), and HRV parameters were unaltered relative to baseline. Taken together, and interpreted in the context of previous literature (Brown et al. 2005), these

Fig. 2. Condition × time group and individual changes (Δ) relative to T1 for mean RR interval, natural logarithm (Ln) of the root-mean square of successive RR interval differences (RMSSD), Ln of the standard deviation of the normal-to-normal RR intervals (SDNN), and systemic vascular resistance (SVR).



findings suggest that the hypertonic nature of Gatorade seems to blunt the cardiac-autonomic and hemodynamic adjustments associated with hypo-osmotic fluid ingestion, despite the added sympathetic stimulus of a glucose challenge.

The dearth of cardiac-autonomic modulation following sports beverage ingestion supports our hypothesis, which was partly based on an anticipated glucose- and insulin-mediated sympathetic outflow (Rowe et al. 1981) reflected by a reduction in HRV indices, or an attenuated increase. Our findings are consistent with those of Prasertsri et al. (2019), who observed no significant changes in heart rate or HRV in young healthy individuals following ingestion of a comparable fluid volume of glucose-fructose solution or passion fruit juice. However, our findings are in contrast to observations showing that glucose administration during an oral glucose tolerance test (OGTT; 300 mL fluid volume) is associated with a significant increase in the ratio of low and high frequency spectral power (LF/HF) in apparently healthy young individuals (Paolisso et al. 1997) and reduced RMSSD in type 1 diabetics (Eckstein et al. 2021). Comparably, a recent investigation reported reductions in RMSSD and cardiovagal baroreflex sensitivity in healthy young individuals following sugar-sweetened, caffeinated (~75 mg) soft drink consumption (Chapman et al. 2021). Notably, cardiac-autonomic responses (LF/HF) to glucose ingestion (Paolisso et al. 1997) and insulin infusion are shown to be dose-dependent and inversely related to body fat (Paolisso et al. 1999). Given our purposeful inclusion of only healthy and active young individuals, it seems unlikely that impairments in insulin sensitivity were responsible for the lack of observed cardiac-autonomic responses to Gatorade. Rather, we attribute this finding to the relatively low glucose load in the sports beverage (36 g), which was approximately half of what was administered in previous studies showing cardiac-autonomic adjustments following consumption of a sugar-sweetened (~61 g), caffeinated soft drink (Chapman et al. 2021), or in response to an OGTT (75 g) (Eckstein et al. 2021; Paolisso et al. 1997).

Vagal enhancement following water drinking, evidenced by unidirectional changes in HRV parameters, is consistently observed in healthy young individuals (Girona et al. 2014; Heathers et al. 2018; Routledge et al. 2002). This finding is attributable to an up-regulation of parasympathetic modulation to decrease CO as a means of compensating for pressor-mediated sympathetic activation (Jordan et al. 2000). We observed a peak in HRV 5–10 min after water drinking (i.e., T2) in agreement with Girona et al.

(2014), which is faster than what has been observed previously (~20-min) (Brown et al. 2005; Heathers et al. 2018; Routledge et al. 2002). This more rapid up-regulation of cardiac-vagal activity may be explained by the administration of colder fluids in the present study (1.6 vs. 4–22 °C), as temperature is known to influence cardiovascular responses to fluid ingestion (Girona et al. 2014). A relatively novel aspect of the present investigation was the longer time-course of the cardiovascular adjustments to fluid ingestion compared with what has been studied previously (i.e., 60 vs. 45 min) (Heathers et al. 2018; Routledge et al. 2002), allowing us to obtain unique insight into the persistence of these adaptations. Though some studies included follow-up of 60–90 min post-fluid ingestion, lack of a control condition makes it difficult to determine if alterations in heart rate were due to persisting effects of fluid, or natural variation (Girona et al. 2014; Grasser 2020). Relative to baseline in the current study, HRV values were elevated following water drinking throughout the observation period (Figs. 1 and 2). However, comparison of HRV following water consumption with measures made at the same time-points in the control condition provide evidence for a pronounced diminution of the effects of water drinking on cardiovagal activity at T5 (no significant difference and trivial ES, Table 2). We also note that *small* or nearly *small* ES increments (ES = 0.19–0.30) in HRV parameters were detected at T5 for the control condition relative to T1 (Table 1), which were similar in magnitude to the ES observed for water at T5 relative to T1. This is an important finding suggesting that accurate resting HRV parameters may be safely obtained 60-min after water drinking, a point that should be carefully considered in research studies and applied settings where day-to-day tracking of HRV is of interest.

Owing to functional baroreflex buffering, we observed no changes in systolic blood pressure following water or Gatorade ingestion. This finding is in contrast with autonomic failure patients in whom profound systolic blood pressure increases are observed after drinking a lower volume of water (480 mL) (Jordan et al. 2000). However, we did observe a rise in SVR shortly following (i.e., 5–10 min, T2) ingestion of both water and Gatorade, though this change was only sustained in the water condition. Comparably, Brown et al. (2005) observed a gradual rise in total peripheral resistance after water drinking, but no change after ingestion of isotonic saline (0.9%). These findings are in contrast with those of Rontoyanni et al. (2015) who reported a drop in SVR, explained by an increase in CO and SV following a 400-mL carbohydrate beverage (57 g). These increases in CO and SV following beverage ingestion, which was not observed in our study, may be related to greater inotropic influence of insulin (Klein and Visser 2010) following administration of a higher carbohydrate beverage (37 vs. 57 g). Interestingly, hyperosmotic solutions have been demonstrated to exhibit a negative inotropic influence on cardiac muscle (Wildenthal et al. 1975), and thus it is possible that the hyperosmotic content of Gatorade may have blunted increases in cardiac contractility due to insulin alone.

Strengths of the present study include our use of sensitive and reliable techniques for constant monitoring of cardiovascular and hemodynamic variables for 60 min post-beverage ingestion and our consideration of physical activity level in subject enrollment. Failure to measure and thus our inability to account for baseline hydration status, insulin and glucose levels, and plasma osmolality in our participants can be viewed as limitations of the present investigation, as is inclusion of only men. Measures of blood pressure variability and cardiovagal baroreflex sensitivity, and exploration of the relationship between changes in these variables with changes in HRV among men and women would provide a more comprehensive understanding as to the effects of sports beverage ingestion on cardiovascular control, and should be considered in the future. We note that sex-related differences have been reported in HRV responses to post-exercise fluid ingestion (Matias et al. 2019). The applicability of the current findings is therefore limited to healthy young men. Finally, data collection occurred following an overnight fast. Thus, whether responses would be of different magnitude following shorter periods (e.g., between meals) is unclear.

In conclusion, ingestion of a hypertonic, sugar-sweetened sports beverage does not evoke the rapid and sustained cardiac-autonomic and hemodynamic adjustments associated with water drinking. While water consumption should unequivocally be considered a confounder to accurate HRV measurement, a hypertonic sports beverage seems to have less of an effect on resting HRV despite an associated glucose perturbation. To avoid fluid-induced inflation of HRV parameters in physically active young men, HRV should be obtained at least 45 min following 591 mL of cold Gatorade ingestion and 60 min following an equal volume of cold water.

Conflict of interest statement

The authors declare no conflicts of interest and no specific funding for this work.

Acknowledgements

The authors thank volunteers for participating in this investigation.
Author contributions: M.C. spearheaded data collection, assisted with data analysis, and authored the initial draft. G.J.G. co-authored and edited the final draft. A.A.F. developed the research question and methodology, supervised data collection and analysis, and co-authored and edited the final draft.

References

Behrens, K., Hottenrott, K., Weippert, M., Montanus, H., Kreuzfeld, S., Rieger, A., et al. 2015. Individualization of exercise load control for inpatient cardiac rehabilitation. Development and evaluation of a HRV-based intervention program for patients with ischemic heart failure. Herz, **40**(1): 61–69. doi:10.1007/s00059-013-4037-2.

Brown, C.M., Barberini, L., Dulloo, A.G., and Montani, J.P. 2005. Cardiovascular responses to water drinking: does osmolality play a role? Am. J. Physiol. Regul. Integr. Comp. Physiol. **289**(6): R1687–R1692. doi:10.1152/ajpregu.00205.2005.

Buchheit, M. 2014. Monitoring training status with HR measures: do all roads lead to Rome? Front. Physiol. **5**:73. doi:10.3389/fphys.2014.00073.

Chapman, C.L., Reed, E.L., Worley, M.L., Pietrafesa, L.D., Kueck, P.J., Bloomfield, A.C., et al. 2021. Sugar-sweetened soft drink consumption acutely decreases spontaneous baroreflex sensitivity and heart rate variability. Am. J. Physiol. Regul. Integr. Comp. Physiol. **320**(5): R641–R652. doi:10.1152/ajpregu.00310.2020.

da Silva, D.F., Ferraro, Z.M., Adamo, K.B., and Machado, F.A. 2019. Endurance running training individually guided by HRV in untrained women. J. Strength Cond. Res. **33**(3): 736–746. doi:10.1519/JSC.0000000000002001. PMID:28570494.

Dekker, J.M., Crow, R.S., Folsom, A.R., Hannan, P.J., Liao, D., Swenne, C.A., and Schouten, E.G. 2000. Low heart rate variability in a 2-minute rhythm strip predicts risk of coronary heart disease and mortality from several causes: the ARIC Study. Circulation, **102**(11): 1239–1244. doi:10.1161/01.CIR.102.11.1239. PMID:10982537.

Eckstein, M.L., Moser, O., Tripolt, N.J., Pferschy, P.N., Obermayer, A.A.M., Kojzar, H., et al. 2021. Rapid glucose rise reduces heart rate variability in adults with type 1 diabetes: A prospective secondary outcome analysis. Diabetes Obes. Metab. **23**(7): 1681–1684. doi:10.1111/dom.14287. PMID:33283963.

Flatt, A.A., Wilkerson, G.B., Allen, J.R., Keith, C.M., and Esco, M.R. 2019. Daily heart rate variability before and after concussion in an American college football player. Sports, **7**(5): 97. doi:10.3390/sports7050097. PMID:31035597.

Flatt, A., Allen, J., Bragg, A., Keith, C., Earley, R., and Esco, M. 2020. Heart rate variability in college football players throughout preseason camp in the heat. Int. J. Sports Med. **41**(9): 589–595. doi:10.1055/a-1145-3754. PMID:32353883.

Fortin, J., Marte, W., Grüllenberger, R., Hacker, A., Habenbacher, W., Heller, A., et al. 2006. Continuous non-invasive blood pressure monitoring using concentrically interlocking control loops. Comput. Biol. Med. **36**(9): 941–957. doi:10.1016/j.compbiomed.2005.04.003.

Fortin, J., Wellisch, A., and Maier, K. 2013. CNAP - Evolution of continuous non-invasive arterial blood pressure monitoring. Biomed. Tech. **58**(Suppl. 1): bmt-2013-4179. doi:10.1515/bmt-2013-4179. PMID:24042825.

Girona, M., Grasser, E.K., Dulloo, A.G., and Montani, J.P. 2014. Cardiovascular and metabolic responses to tap water ingestion in young humans:

does the water temperature matter? Acta Physiol. **211**(2): 358–370. doi:10.1111/apha.12290. PMID:24684853.

Grasser, E.K. 2020. Dose-dependent heart rate responses to drinking water: a randomized crossover study in young, non-obese males. Clin. Auton. Res. **30**(6): 567–570. doi:10.1007/s10286-020-00673-6. PMID:32078090.

Heathers, J., Quintana, D., Angus, D., Krygier, J., Kemp, A., and de Rosnay, M. 2018. Water consumption as a source of error in the measurement of heart rate variability. Open Science Framework, Charlottesville, VA, USA. pp. 1–40. doi:10.31219/osf.io/83exy.

Heathers, J.A. 2014. Everything Hertz: methodological issues in short-term frequency-domain HRV. Front. Physiol. **5**: 177. doi:10.3389/fphys.2014.00177. PMID:24847279.

Hopkins, W., Marshall, S., Batterham, A., and Hanin, J. 2009. Progressive statistics for studies in sports medicine and exercise science. Med. Sci. Sports Exerc. **41**(1): 3–13. doi:10.1249/MSS.0b013e31818cb278. PMID:19092709.

Javaloyes, A., Sarabia, J.M., Lamberts, R.P., and Moya-Ramon, M. 2018. Training prescription guided by heart rate variability in cycling. Int. J. Sports Physiol. Perform. **14**(1): 23–32. doi:10.1123/ijspp.2018-0122. PMID:29809080.

Jordan, J., Shannon, J.R., Black, B.K., Ali, Y., Farley, M., Costa, F., et al. 2000. The pressor response to water drinking in humans: a sympathetic reflex? Circulation, **101**(5): 504–509. doi:10.1161/01.CIR.101.5.504. PMID:10662747.

Klein, L.J., and Visser, F.C. 2010. The effect of insulin on the heart: Part 1: Effects on metabolism and function. Neth. Heart J. **18**(4): 197–201. doi:10.1007/BF03091761. PMID:20428418.

Laborde, S., Mosley, E., and Mertgen, A. 2018. A unifying conceptual framework of factors associated to cardiac vagal control. Heliyon, **4**(12): e01002. doi:10.1016/j.heliyon.2018.e01002.

Lininger, M., and Riemann, B.L. 2016. Statistical primer for athletic trainers: using confidence intervals and effect sizes to evaluate clinical meaningfulness. J. Athl. Train. **51**(12): 1045–1048. doi:10.4085/1062-6050-51.12.14. PMID:28107049.

Malik, M., Bigger, J.T., Camm, A.J., Kleiger, R.E., Malliani, A., Moss, A.J., et al. 1996. Heart rate variability: standards of measurement, physiological interpretation and clinical use. Eur. Heart J. **17**(3): 354–381. doi:10.1093/oxfordjournals.eurheartj.a014868.

Matias, A., Dudar, M., Kauzlaric, J., Frederick, K.A., Fitzpatrick, S., and Ives, S.J. 2019. Rehydrating efficacy of maple water after exercise-induced dehydration. J. Int. Soc. Sports Nutr. **16**(1): 5. doi:10.1186/s12970-019-0273-z. PMID:30744654.

Monrroy, H., Borghi, G., Pribic, T., Galan, C., Nieto, A., Amigo, N., et al. 2019. Biological response to meal ingestion: Gender differences. Nutrients, **11**(3): 702. doi:10.3390/nu11030702. PMID:30917512.

Nakamura, F.Y., Flatt, A.A., Pereira, L.A., Ramirez-Campillo, R., Loturco, I., and Esco, M.R. 2015. Ultra-short-term heart rate variability is sensitive to training effects in team sports players. J. Sports Sci. Med. **14**(3): 602–605. PMID:26336347.

Nakamura, F.Y., Pereira, L.A., Esco, M.R., Flatt, A.A., Moraes, J.E., Abad, C.C.C., et al. 2017. Intraday and interday reliability of ultra-short-term heart rate variability in rugby union players. J. Strength Cond. Res. **31**(2): 548–551. doi:10.1519/JSC.0000000000001514. PMID:27243917.

Paolisso, G., Manzella, D., Ferrara, N., Gambardella, A., Abete, P., Tagliamonte, M.R., et al. 1997. Glucose ingestion affects cardiac ANS in healthy subjects with different amounts of body fat. Am. J. Physiol. **273**(3): E471–E478. doi:10.1152/ajpendo.1997.273.3.E471. PMID:9316435.

Paolisso, G., Manzella, D., Tagliamonte, M.R., Rizzo, M.R., Gambardella, A., and Varricchio, M. 1999. Effects of different insulin infusion rates on heart rate variability in lean and obese subjects. Metabolism, **48**(6): 755–762. doi:10.1016/S0026-0495(99)90176-2. PMID:10381151.

Penaz, J. 1973. Photoelectric measurement of blood pressure, volume and flow in the finger. *In* Digest of the 10th International Conference on Medical and Biological Engineering, Dresden. 104 pp.

Perpiñan, G., Severeyn, E., Wong, S., and Altuve, M. 2019. Cardiac autonomic modulation in response to a glucose stimulus. Med. Biol. Eng. Comput. **57**(3): 667–676. doi:10.1007/s11517-018-1913-1. PMID:30349959.

Plews, D.J., Laursen, P.B., Meur, Y.L., Hausswirth, C., Kilding, A.E., and Buchheit, M. 2014. Monitoring training with heart-rate variability: How much compliance is needed for valid assessment? Int. J. Sports Physiol. Perform. **9**(5): 783–790. doi:10.1123/ijspp.2013-0455. PMID:24334285.

Prasertsri, P., Booranasuksakul, U., Naravoratham, K., and Trongtosak, P. 2019. Acute effects of passion fruit juice supplementation on cardiac autonomic function and blood glucose in healthy subjects. Prev. Nutr. Food Sci. **24**(3): 245–253. doi:10.3746/pnf.2019.24.3.245. PMID:31608249.

Proietti, R., di Fronso, S., Pereira, L.A., Bortoli, L., Robazza, C., Nakamura, F.Y., et al. 2017. Heart rate variability discriminates competitive levels in professional soccer players. J. Strength Cond. Res. **31**(6): 1719–1725. doi:10.1519/JSC.0000000000001795. PMID:28538325.

Rontoyanni, V.G., Werner, K., Sanders, T.A., and Hall, W.L. 2015. Differential acute effects of carbohydrate- and protein-rich drinks compared with water on cardiac output during rest and exercise in healthy young men. Appl. Physiol. Nutr. Metab. **40**(8): 803–810. doi:10.1139/apnm-2014-0358. PMID:26244599.

Routledge, H.C., Chowdhary, S., Coote, J.H., and Townend, J.N. 2002. Cardiac vagal response to water ingestion in normal human subjects. Clin. Sci. **103**(2): 157–162. doi:10.1042/cs1030157. PMID:12149107.

Rowe, J.W., Young, J.B., Minaker, K.L., Stevens, A.L., Pallotta, J., and Landsberg, L. 1981. Effect of insulin and glucose infusions on sympathetic nervous system activity in normal man. Diabetes, **30**(3): 219–225. doi:10.2337/diab.30.3.219. PMID:7009270.

Rowlands, D.S., Bonetti, D.L., and Hopkins, W.G. 2011. Unilateral fluid absorption and effects on peak power after ingestion of commercially available hypotonic, isotonic, and hypertonic sports drinks. Int. J. Sport Nutr. Exerc. Metab. **21**(6): 480–491. doi:10.1123/ijsnem.21.6.480. PMID:22089303.

Sawka, M., Burke, L., Eichner, E., Maughan, R., Montain, S., and Stachenfeld, N. 2007. American College of Sports Medicine position stand. Exercise and fluid replacement. Med. Sci. Sports Exerc. **39**(2): 377–390. doi:10.1249/mss.0b013e31802ca597. PMID:17277604.

Senthinathan, A., Mainwaring, L.M., and Hutchison, M. 2017. Heart rate variability of athletes across concussion recovery milestones: a preliminary study. Clin. J. Sports. Med. **27**(3): 288–295. doi:10.1097/JSM.0000000000000337. PMID:27379659.

Smolle, K.H., Schmid, M., Prettenthaler, H., and Weger, C. 2015. The accuracy of the CNAP® device compared with invasive radial artery measurements for providing continuous noninvasive arterial blood pressure readings at a medical intensive care unit: a method-comparison study. Anesth. Analg. **121**(6): 1508–1516. doi:10.1213/ANE.0000000000000965. PMID:26496367.

Souza, G.G., Magalhães, L.N., Cruz, T.A., Mendonça-De-Souza, A.C., Duarte, A.F., Fischer, N.L., et al. 2013. Resting vagal control and resilience as predictors of cardiovascular allostasis in peacekeepers. Stress, **16**(4): 377–383. doi:10.3109/10253890.2013.767326. PMID:23327672.

Stanley, J., Peake, J.M., and Buchheit, M. 2013. Cardiac parasympathetic reactivation following exercise: implications for training prescription. Sports Med. **43**(12): 1259–1277. doi:10.1007/s40279-013-0083-4. PMID:23912805.

Vandecasteele, K., De Cooman, T., Gu, Y., Cleeren, E., Claes, K., Paesschen, W.V., et al. 2017. Automated epileptic seizure detection based on wearable ecg and ppg in a hospital environment. Sensors, **17**(10): 2338. doi:10.3390/s17102338. PMID:29027928.

Wagner, J.Y., Körner, A., Schulte-Uentrop, L., Kubik, M., Reichenspurner, H., Kluge, S., et al. 2018. A comparison of volume clamp method-based continuous noninvasive cardiac output (CNCO) measurement versus intermittent pulmonary artery thermodilution in postoperative cardiothoracic surgery patients. J. Clin. Monit. Comput. **32**(2): 235–244. doi:10.1007/s10877-017-0027-x. PMID:28540614.

Watso, J.C., Robinson, A.T., Babcock, M.C., Migdal, K.U., Wenner, M.M., Stocker, S.D., et al. 2020. Short-term water deprivation does not increase blood pressure variability or impair neurovascular function in healthy young adults. Am. J. Physiol. Regul. Integr. Comp. Physiol. **318**(1): R112–R121. doi:10.1152/ajpregu.00149.2019. PMID:31617739.

Wildenthal, K., Adcock, R.C., Crie, J.S., Templeton, G.H., and Willerson, J.T. 1975. Negative inotropic influence of hyperosmotic solutions on cardiac muscle. Am. J. Physiol. **229**(6): 1505–1509. doi:10.1152/ajplegacy.1975.229.6.1505. PMID:1211482.