

The stress of chess players as a model to study the effects of psychological stimuli on physiological responses: an example of substrate oxidation and heart rate variability in man

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Abstract We have studied the physiological consequences of the tension caused by playing chess in 20 male chess players, by following heart rate, heart rate variability, and respiratory variables. We observed significant increase in the heart rate (75–86 beats/min), in the ratio low frequency (LF)/high frequency (HF) of heart rate variability (1.3–3.0) and also a decrease in mean heart rate variability with no changes in HF throughout the game. These results suggest a stimulation of the sympathetic nervous system with no changes in the parasympathetic system. The respiratory exchange ratio was rather elevated (over 0.89) at the start and significantly decreased during the game (0.75 at the end), indicating that energy expenditure progressively switched from carbohydrate to lipid oxidation. The changes in substrate oxidation and the sympathetic system seem to be due to high cognitive demands and bring new insight into adaptations to mental strain.

Keywords Chess · Heart rate variability · Indirect calorimetry · Psychological crossover concept · Psychological stress · Substrate oxidation

Introduction

Traditionally, the effects of psychological stress in man have been investigated using two different kinds of situations: laboratory experiments and real-life stressors. Laboratory tests (e.g. speech tasks, arithmetic tasks, Stroop test, etc.) consist of a stimulus of short duration and of limited potency. Investigations using real-life stressors can be divided into those using chronic stressors (e.g. bereavement, unemployment, divorce, caring for patients with Alzheimer's disease, etc.) and acute stressors (e.g. academic exams). One advantage of using arranged stress situations (laboratory tests and real-life stressors) is that one can easily use each individual as his or her own control. However, because of ethical and legal considerations, it is difficult to experimentally induce pure psychological stress. Also, it is difficult to avoid unnatural and artificial arrangements (Dugué et al. 1992, 2001).

Stress is often defined as a threat (real or implied) to homeostasis (McEwen and Wingfield 2003) and is known to stimulate the autonomic nervous system, which in turn stimulates the cardiovascular system and the metabolism. Mental effort induces the mobilization of energy for cognitive purposes and induces a compensatory strategy to protect performance in the presence of augmented request tasks and psychological stressors (Gaillard 1993, 2001). Though it is well known that glucose is used as a primary fuel for energy generation in the brain during psychological stress or mental effort (Rao et al. 2006; Fairclough and Houston 2004; Sourkes 2006), very little information is available concerning the global metabolism of substrates during mental strain. This lack of exploration may be the result of drawbacks to the available techniques that disturb the subject during investigation, of difficulties in applying these techniques, and of continuously monitoring subjects

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through a mental challenge. However, with the development of new generation of portable metabolic systems, it is now possible to monitor cardio-respiratory variables during a challenging situation with only minor disturbances.

A chess game represents a legitimate psychological stress. It is a strategic and high cognitive demand task. Players have to think through a wide range of move sequences to find the best choice. This very challenging and interesting situation has not been thoroughly explored, and to date, most of the reports have concentrated on psychological measurements and not on physiological outcomes. The only available study in that field was the one of Schwarz et al. (2003).

Accordingly, we studied the effect of the tension caused by playing a chess game on heart rate, heart rate variability, and respiratory variables. Using indirect calorimetry, we deduced the oxidation rate of lipids and carbohydrates and also total energy expenditure during the challenge. Moreover, we investigated the influences of the expertise of the players on the physiological responses in this atypical sport.

Subjects and methods

Subjects

A total of 20 male chess competitors of national and international level (age: 42 ± 13 years; height: 178 ± 4 cm; weight: 76 ± 9 kg; BMI: 24 ± 3 kg m⁻²) participated in our study. They were subjectively healthy, and none were on medication. The experiment was conducted in accordance with the Declaration of Helsinki, and all the procedures were carried out with written informed consent of the participants. Volunteers were classified according their ELO score (official rating of the International Chess Association). The group of our subjects had an ELO (mean and extreme values) of 1,757 (1,250–2,170).

Study design

The experiment started with the lunch (standardized) of the participants, which was taken between 12.00 and 13.00. Then at 16.30, the participants started their chess session. Before the start of the experiment, instructions were given to the participants about the procedures and protocol requirements during the test. In order to make the participants comfortable, they were asked to relax in a supine position for 15 min. Moreover, all participants underwent a familiarization period with the equipment required for testing. The experimental room was calm (no more than 2 observers and the player) and light and temperature (18°C) were continuously regulated.

Chess game

The participants played a chess game against a computer with software that mimed a level of expertise which was similar to the player's level (Chessmaster 9th edition, 2004). This software was used because of the ability to select a wide range of ELO levels. In fact, without telling the participants, we set the program at a slightly higher level (+100 ELO points) than the level of the player. A laboratory assistant who was familiar with chess was always present during experiments. He performed the moves indicated by the computer on the chess board, and he also served as the human presence in front of the participants. One hour was allotted for each player (an electronic clock was used to control the time). Cardio-respiratory variables were continuously recorded until the chess game were over (approximately 90 min, the computer also needed some time before indicating its move).

Psychological measurements

PANAS

This test presents 20 adjectives, 10 that assess positive affectivity (e.g., excited) and 10 that assess negative affectivity (e.g., upset) (Watson et al. 1988). These adjectives describe different feelings and emotions. Participants described their present feelings on a five point scale, ranging from “very slightly” (1) to “extremely” (5).

Brief COPE (dispositional version of trait anxiety)

This test is the abridged version of the COPE inventory (Carver et al. 1989) and presents 14 scales that assess different coping dimensions: active coping, planning, using instrumental support, using emotional support, venting, behavioral disengagement, self-distraction, self-blame, positive reframing, humor, denial, acceptance, religion, and substance use. Each seven point scale, ranging from “never” (1) to “always” (7), contains two items (28 items altogether).

Both questionnaires were presented before the start of the experiment.

Physiological measurements

A portable metabolic measurement system (Vmax ST; Sensor Medics, Germany) and a heart rate monitor (Polar S810, Finland) were used to measure the cardio-respiratory responses and heart rate variability. The gas analyzer system was calibrated before each subject session using gases of known concentrations and used thereafter as previously described (Brehm et al. 2004; Laurent et al. 2008). The following variables were recorded throughout the game:

heart rate (HR), ventilatory flow (VF), tidal volume (Vt), breath frequency (bF), O₂ consumption ($\dot{V}O_2$), CO₂ production ($\dot{V}CO_2$), respiratory exchange ratio (RER), and the following indices were analyzed after the test: mean R–R, low frequency (LF), high frequency (HF)—an index of the activity of the parasympathetic branch of the autonomic nervous system, LF/HF ratio (LF/HF)—an index of the activity of the sympathetic branch of the autonomic nervous system, carbohydrate oxidation rate (CHO), lipid oxidation rate (FAT), and energy expenditure (EE). The baseline measurements were carried out before the beginning of the match in sitting posture during 3 min.

Data processing

The R–R intervals were recorded (Polar S810, Finland) at a frequency of 1,000 Hz (Ruha et al. 1997) and saved in a computer for further analysis of HR variability from the R–R interval tachogram with Heart Signal software (Kempele, Finland). All the R–R intervals were edited by visual inspection, to exclude all the undesirable beats, which accounted for <1% in every subject. The details of this analysis and the filtering technique have been described previously (Huikuri et al. 1992, 1996). The mean R–R and the standard deviation of all R–R intervals were used as time domain analysis methods. An autoregressive model (model order 20) was used to estimate the power spectrum densities of heart rate variability. The power spectra were quantified by measuring the area under the whole frequency band (total power) and under two frequency bands: LF power, from 0.04 to 0.15 Hz; and high-frequency power (HF), from 0.15 to 0.4 Hz. We also calculated LF and HF in normalized units (n.u) with the same algorithm.

Series of 300 s or approximately 256 consecutive R–R intervals (which is a minimal time requirement) were extracted 5 min before the game, at the beginning, at the middle, and at the end of each game. The same periods were analyzed for respiratory measurements. Indirect calorimetry was used to calculate the carbohydrate (CHO) and lipid oxidation (FAT), and total energy expenditure during the match. We used stoichiometric equations and appropriate caloric equivalents (Peronnet and Massicotte 1991), with the assumption that the nitrogen excretion rate was 135 $\mu\text{g kg}^{-1} \text{min}^{-1}$ (Romijn et al. 1993). The equations were:

$$\begin{aligned} \text{CHO rate oxidation (g min}^{-1}\text{)} \\ = (4.585 \times \dot{V}CO_2) - (3.226 \times \dot{V}O_2) \end{aligned}$$

$$\begin{aligned} \text{Fat rate oxidation (g min}^{-1}\text{)} = (1.695 \times \dot{V}O_2) \\ - (1.701 \times \dot{V}CO_2). \end{aligned}$$

Mass was expressed in grams per minute and gas volume in liters per minute. $\dot{V}O_2$ and $\dot{V}CO_2$ values were averaged every minute.

$$\begin{aligned} \text{Total energy expenditure} = [(\% \text{ CHO}/100) \times \dot{V}O_2 \\ \times 5.05 \text{ kcal L}^{-1}] \\ + [(\% \text{ Fat}/100) \times \dot{V}O_2 \\ \times 4.7 \text{ kcal L}^{-1}] \end{aligned}$$

The percentages of carbohydrates and lipid oxidations were calculated by using the following equations:

$$\% \text{ CHO} = [(\text{RER} - 0.71)/0.29] \times 100$$

$$\% \text{ Fat} = [(1 - \text{RER})/0.29] \times 100.$$

Statistics

All the statistical analyses were completed using Statistica 5.5 software. Results are presented as their mean \pm SD or extreme values. ANOVA for repeated measurements and Tukey post hoc tests were used to analyze the data. Spearman correlation test was also used. Significance was set at $P < 0.05$.

Results

The PANAS gave a positive affect of 3.2 (2.3–4.3) and a negative affect of 1.3 (1–1.8). The brief COPE showed an active coping score of 3 (1–4), planning of 2.9 (1–4), positive reframing of 2.8 (1.5–3.5), acceptance of 2.7 (1–4), self-distraction of 2.6 (1–4), using instrumental support of 2.4 (1–4), middle level of humor of 2.2 (1–4), using emotional support of 2.2 (1–4), self-blame of 2.1 (1.5–3), venting of 2 (1–3), low level of behavioral disengagement of 1.3 (1–2.5), denial of 1.3 (1–3.5), substance use of 1.3 (1–3), and religion of 1.2 (1–2).

We observed a significant increase in heart rate right at the beginning of the contest and the rate stayed elevated until the end of the game (Fig. 1). Significant increases both in LF and in the LF/HF ratio (Fig. 1) and a significant decrease in the mean R–R were also observed (Table 1). No changes in HF and HF n.u. were found. The CO₂ release and the respiratory exchange ratio significantly decreased during the game (Fig. 2). When calculating the oxidation rate, lipid oxidation was found to significantly increase, whereas glucose oxidation significantly decreased (Fig. 2). At the 25 min time point, the two oxidation curves crossed each other. No significant changes were noted in $\dot{V}O_2$, EE, VF, Vt, and bF.

In addition, no significant correlation was found between the outcome of the PANAS and brief COPE tests and any of the physiological variables. All participants lost their game and reported that their level of effort was similar to a tiring, serious match. All of the data concerning physiological variables before and during chess play are presented in Table 1. The overall energy expenditure in our

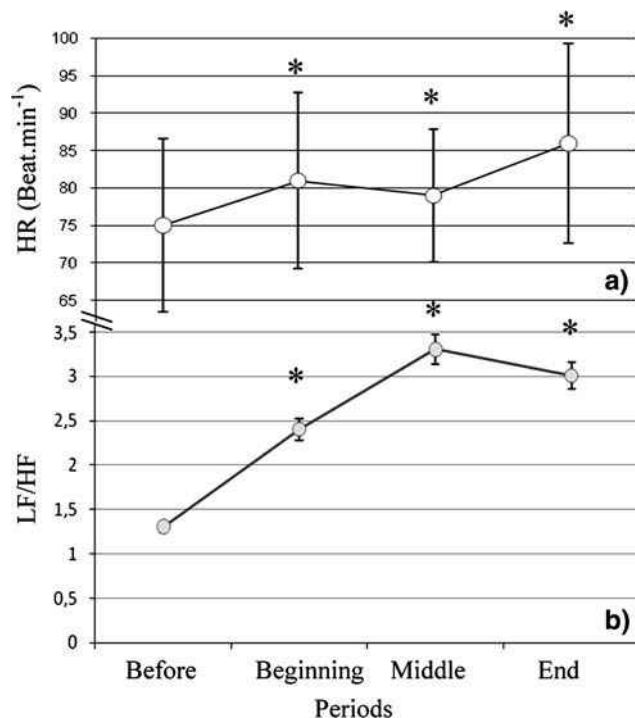


Fig. 1 Indexes of sympathetic involvement before and during a chess game: **a** Heart rate; **b** LF/HF ratio (number of subject = 20); * $P < 0.05$. LF/HF Ratio between the low and high frequency band

players during the entire game was of 138 kcal (extreme values 102–198 kcal).

Discussion

We have studied the effects of playing chess in chess competitors (national and international level) on a series of cardio-respiratory and metabolic variables.

The evaluation of our subjects through the PANAS and the Brief COPE tests before the chess contest showed that their basal amount of perceived stress was rather low and they were psychologically healthy. Also, all of our subjects reported that the contest was as challenging and tiring as a regular match. Therefore, artifacts due to specific psychological state or trait do not seem to generate bias in the stress-induced physiological responses of our subjects.

Before the beginning of the contest, the heart rate was rather elevated. Such observations indicate that our participants were reacting to the start of the game via anticipatory mechanisms (Wirtz et al. 2006). At the start of the game, both heart rate and RER slightly increased. Such elevated levels could indicate that substrate oxidation mainly involved carbohydrates. However, the transient increase in RER may also reflect changes in respiratory phenomena. Only a slight increase (not significant) in the ventilation rate was observed at the beginning of the game.

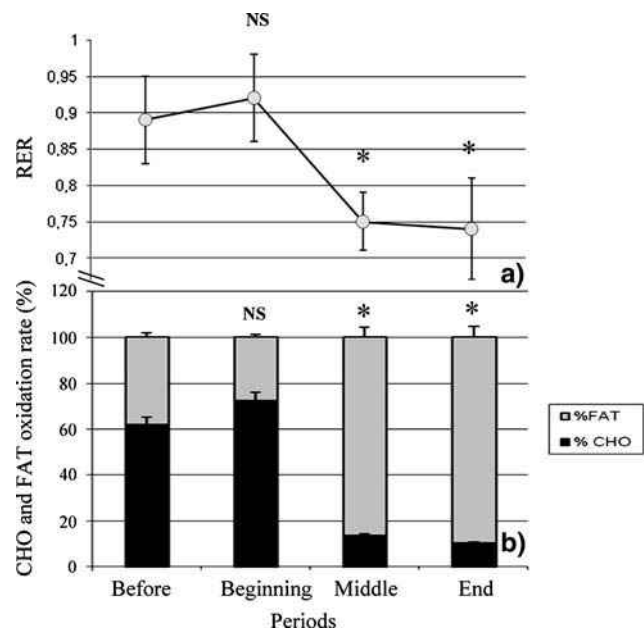


Fig. 2 Indexes of substrate utilization before and during a chess game: **a** RER; **b** CHO and FAT oxidation rate (%) (number of subject = 20); * $P < 0.05$. RER Respiratory exchange ratio; CHO carbohydrate oxidation rate; FAT lipid oxidation rate

In any case, this indicates that the players were very reactive. Such changes are certainly under sympathetic control, and may be related to unspecific stress responses (Selye 1951). Many studies in humans have documented an increase in SNS activation during mental stress (e.g. Seematter et al. 2000; Garde et al. 2002). In fact, the heart rate variability measurements in our participants at the beginning of the game revealed a significant increase both in LF and in the LF/HF ratio, a decrease in mean R–R and no changes in HF, indicating an activation of the sympathetic system (Montano et al. 1994; Pagani et al. 1991) with no changes in the parasympathetic system (Hayano et al. 1991). It is well known that Vt and bF have a large impact on the HRV indices, especially in the vagally mediated HF spectral band (Hirsch and Bishop 1981). Both Vt and bF were measured continuously during the study, and we did not observe any significant changes in breathing pattern. These results indicate that the changes in the spectral component of the R–R intervals are due to increased sympathetic stimulation rather than to changes in respiration pattern or in vagal activation. During the game, we initially observed stabilization in the heart rate, followed by a significant increase through the end of the game. During that time, the CO₂ release significantly decreased, whereas oxygen consumption stayed stable. This led to a significant decrease in the RER. One possible explanation could again be hyperventilation, which could eventually lead to a relative lowering in CO₂ production (hypocapnia) related to O₂ consumption. However, no hyperventilation

Table 1 Heart rate, gas and metabolic variations during the chess match in experienced chess players ($n = 20$)

Parameters	Units	Before	Beginning	Middle	End
HR	beats min^{-1}	75 (60–101)	81 (59–108)*	79 (65–95)	86 (65–120)*
Mean R–R	ms	877 (585–1,210)	819 (666–1,022)*	779 (639–930)*	766 (556–953)*
LF	$\ln \text{ms}^2$	6.3 (4.2–8.0)	6.7 (5.2–8.1)*	6.8 (4.9–8.2)*	6.6 (4.0–8.2)*
HF	$\ln \text{ms}^2$	5.7 (3.8–7.3)	6.1 (3.6–7.3)	5.8 (3.6–7.6)	5.7 (2.1–7.4)
HF	n.u	33.5 (22–44.6)	32.3 (18.9–51.5)	30.8 (13.6–44.3)	31.4 (13.1–50.2)
LF/HF		1.3 (0.2–5.1)	2.4 (0.5–5.1)*	3.3 (0.8–8.1)*	3.0 (1.0–7.0)*
Vt	ml min^{-1}	0.59 (0.40–0.79)	0.63 (0.44–0.82)	0.55 (0.38–0.80)	0.58 (0.43–0.80)
bF	cycle min^{-1}	15.99 (10.97–20.40)	15.94 (10.86–22.20)	15.94 (10.50–21.40)	16.29 (10.70–22.30)
VF	l min^{-1}	9.61 (6.87–13.38)	10.32 (6.62–13.55)	9.06 (5.95–13.15)	9.52 (5.59–13.30)
$\dot{V}O_2$	ml min kg^{-1}	4.18 (2.36–6.71)	4.51 (2.59–5.80)	4.32 (2.78–5.89)	4.41 (2.59–6.34)
$\dot{V}CO_2$	ml min kg^{-1}	3.57 (2.37–5.45)	3.94 (2.23–5.27)	3.04 (1.95–4.37)*	3.11 (1.72–4.65)*
RER		0.89 (0.79–1.03)	0.92 (0.81–1.07)	0.75 (0.67–0.81)*	0.74 (0.65–0.89)*
CHO	g min^{-1}	0.22 (0.10–0.51)	0.27 (0.11–0.74)	0.01 (0.00–0.16)*	0.002 (0.00–0.32) *
FAT	g min^{-1}	0.08 (0.04–0.21)	0.07 (0.02–0.15)	0.16 (0.09–0.22)*	0.16 (0.07–0.24) *
EE	kcal min^{-1}	1.53 (1.14–2.00)	1.67 (1.18–2.20)	1.53 (1.17–2.01)	1.55 (1.14–2.04)

Results are expressed as mean and extreme values in *brackets*

HR Heart rate; *Mean R–R* mean values of heart rate variability; *LF* low frequency; *HF* high frequency; *LF/HF* ratio between the low and high frequency band; $\dot{V}O_2$ O_2 consumption; $\dot{V}CO_2$ CO_2 production; *RER* respiratory exchange ratio; *bF* respiratory frequency; *VF* ventilatory flow; *Vt* tidal volume; *CHO* carbohydrate oxidation rate; *FAT* lipid oxidation rate; *EE* energy expenditure

* $P < 0.05$ compared to the data obtained before the contest

was noticed during the course of our study. Therefore, a significant increase in lipid oxidation and a dramatic decrease in carbohydrate metabolism may have occurred during the match.

At the end of the game, the oxidation of carbohydrates was very low, and the participants were almost exclusively oxidizing lipids. During the whole game, the total energy expenditure remained constant, but was more elevated than during resting time (Levine et al. 2000; Levine 2005). However, we cannot exclude that we might have observed a transient increase in carbohydrate consumption at the beginning of the game and then a return to resting condition. During the game, the levels of LF and the LF/HF ratio were elevated and were significantly higher than before the start of the game. The HF level did not change during the game. Therefore, the sympathetic system seems to be stimulated during the course of the contest, with no changes in the parasympathetic system. However, it has previously been demonstrated that brief exposure to psychological stressors may lead to both an increase in LF and in the LF/HF ratio with a reduction of the HF level, indicating increased sympathetic activity along with withdrawal of the parasympathetic system (e.g. Delaney and Brodie 2000). Somehow, our results with respect to the absence of any parasympathetic withdrawal during the course of the game are at variance with those reported in the literature. However, the nature of the stimulus was different, and the mental workload might have been lower

in our study as our volunteers were well trained in handling such stimuli. Furthermore, the interplay between the sympathetic and vagal regulation of HR is not always organized in a reciprocal fashion, wherein increased activity in one system is accompanied by decreased activity in the other. A simultaneous sympathetic and vagal activation has been observed during cold face immersion (Tulppo et al. 2005), and also in other circumstances in healthy subjects (Mourot et al. 2007). It is highly possible that increased sympathetic activation occurs without any change in vagal outflow during a demanding mental stress situation without exercise or body position changes, as in the present study.

All of our observations indicated that the chess players were very stimulated during the game, but the nature of the stimuli might have been different during the contest compared to the stimuli that occurred before and at the beginning of the game. Interestingly, the two oxidation curves (lipid and carbohydrate) crossed each other 25 min after the start of the contest. Such changes observed during the game may reflect an adjusted response to the real demands of the task. After the more acute response that was observed right at the beginning of the game, the subject may have been adjusting to a longer lasting effort.

It is not clear why there could be a switch in substrate oxidation during a lasting mental challenge. Numerous studies have shown that different kinds of stressors (examination, environmental stressors, and laboratory-

based stressors) may influence metabolism and the concentration of lipids in blood (Stoney et al. 1997, 1999; Niaura et al. 1992). However, not much is known concerning the use of substrates during mental challenge.

Interestingly, similar kinds of adaptations have been reported in exercise physiology. Moderate physical exercise is generally associated with the preponderant utilization of lipids, whereas acute and intensive physical exercises are under carbohydrate metabolism. The shift in substrate oxidation has been described as the “crossover concept” and depends on the relative intensity of the exercise and therefore on the endurance training of the subjects (Brooks and Mercier 1994). Similarly, this may apply to psychological stress physiology. However, to validate such a psychological crossover concept, one should be able to quantify the intensity of psychological stress and relate it to substrate oxidation. Another interesting feature in this context is that training (endurance training?) of the subject could influence substrate oxidation during a mental challenge. One could therefore speculate that specific training could be designed for chess players. However, to validate such a new approach, further investigations are required.

In summary, we herein described an interesting real-life stressor that seems to be a useful stress model in that it had significant effects on heart rate variability and metabolism.

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