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Effects of emotional excitement on heart rate and blood pressure dynamics in patients with coronary artery disease

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ABSTRACT

The incidence of adverse cardiovascular events is higher among spectators of exciting sports events, but the mechanistic link between the events is not known. We assessed the heart rate (HR) and blood pressure (BP) dynamics of enthusiastic male ice hockey spectators (60 ± 9 years) with coronary artery disease (CAD) during Finnish national league ice hockey play-off final matches. Twenty-four-hour ambulatory ECG ($n = 55$) and BP ($n = 17$) were recorded at the time of the match and on a control day. Beat-to-beat R–R intervals and BP were recorded during the match and a bicycle exercise at equal HR levels ($n = 21$). Systolic and diastolic BP were significantly higher 1 h before, during, and 1 h after the match than on the control day, e.g., the highest systolic BP was 180 ± 14 vs. 145 ± 15 and diastolic 103 ± 13 vs. 82 ± 11 mmHg ($p<0.001$ for both). HR was higher throughout the match ($p<0.05$) and remained elevated 2 h after the match ($p<0001$), and measures of HR variability were decreased during the match ($p<0.01$). Low-frequency variability in BP was higher during the match than during the exercise test ($p<0.01$). The results show that cardiac vagal outflow is attenuated and vasomotor sympathetic activity elevated during exciting sports events and BP dynamics differ from those occurring during physical exercise at equal HRs. The autonomic reactions may partly explain the vulnerability to cardiovascular events caused by this type of leisure-time emotional excitement.

1. Introduction

Environmental stress has been shown to be an important risk factor for cardiovascular events (Strike and Steptoe, 2003; Steptoe and Brydon, 2009). Epidemiological data show earthquakes, war, and sporting events cause a peak in the incidence of cardiovascular events (Meisel et al., 1991; Leor et al., 1996; Serra Grima et al., 2005). Recently, Wilbert-Lampen et al. showed that a stressful soccer game more than doubled the risk of acute cardiovascular events, including acute coronary syndrome and symptomatic cardiac arrhythmia, during a World Cup Soccer match held in Germany in 2006 (Wilbert-Lampen et al., 2008). On those days, the highest average incidence of events was observed during the first 2 h after the beginning of each match. Carroll et al., 2002 have shown an increase in the incidence of acute myocardial infarction after the national team lost a penalty shoot-out (Carroll et al., 2002). Previous studies have also shown that triggering is more common in patients with coronary artery disease than without it (Leor et al., 1996; Strike et al., 2006a,b; Tofler and Muller, 2006). Some studies have also found that most of the additional cardiac emergencies occur in men, explained by sex-specific pathophysiological differences and/or interest in sports or vulnerability to emotional triggers (Tofler et al., 1990; Witte et al., 2000; Culic et al., 2003).

The mechanisms by which emotional excitement, such as watching sports events, increases vulnerability to untoward cardiac events is not well known. At the moment, ice hockey is the most intensively followed sport in Finland, and national play-off finals are of great significance to a large proportion of the population, especially males. The aim of this study was to evaluate changes in autonomic regulation measured by heart rate (HR) variability and ambulatory blood pressure (BP) in enthusiastic ice hockey fans with coronary artery disease (CAD) during play-off ice hockey matches in Oulu, Finland, played by the Oulun Kärpät team in the spring of 2008 and 2009. The changes related to the hockey match were compared with control day measurements without any sports events. To further elucidate the autonomic responses to emotional excitement compared with physical exercise, simultaneous beat-to-beat BP, R–R intervals, and respiration were measured in a subgroup of subjects at baseline controlled conditions, during the ice hockey match, and during a bicycle exercise test.

2. Methods

2.1. Study protocol

The study included patients with stable coronary artery disease (CAD) who participated as spectators in the ice hockey play-off finals

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of the Finnish National League played in the city of Oulu, Finland, during 2008 and 2009. The Oulun Kärpät team won the championship of the Finnish National Ice Hockey League in 2008 and ranked second in 2009. The measurements were performed in the ice hockey hall during all the play-offs, inside the full ice hockey hall among ~6000 other spectators, played in the city of Oulu ("home matches") in 2008 and 2009 (6 matches in 2008 and in 3 matches in 2009). The average number of monitored subjects in each match was 4±1 in 2008 and 10±1 in 2009, resulting in a total of 55 patients. The Oulun Kärpät team lost only one home match and 3 subjects where monitored during that match. Therefore, we were not able to study the effects of the result of the match on autonomic responses.

Twenty-four-hour ECG recording was performed for all the patients during the match day and during a control day within a one-week interval. Ambulatory BP measurements were performed for 17 patients during the match and the control day in 2009. Beat-to-beat BP, ECG, and respiration frequency were measured in 21 patients in Oulu University Hospital located next to ice hockey hall on the morning of the match day and during the match in the ice hockey hall in 2009. The next day the same recordings were performed (Oulu University Hospital) during a dynamic bicycle exercise test targeting HR at the same level as that measured during the match.

2.2. Patient selection

The CAD patients were selected from the ARTEMIS (Innovation to Reduce Cardiovascular Complications of Diabetes at the Intersection) study database. In the ARTEMIS study, patients with CAD have undergone a thorough evaluation of their cardiovascular status, including, e.g., a baseline examination done by a cardiologist, tissue Doppler echocardiography, an exercise stress test, and 24-h Holter recordings. CAD and its severity had been assessed by coronary angiography at Oulu University Hospital within six months of inclusion. To maximize the likelihood of emotional arousal during the ice hockey match, the candidates were interviewed by telephone according to a flow chart presented in Fig. 1. The characteristics of the patients are presented in Table 1. The study was performed according to the Declaration of Helsinki, and the local committee of research ethics of the Northern Ostrobothnia Hospital District approved the protocol, and all the subjects gave written informed consent.

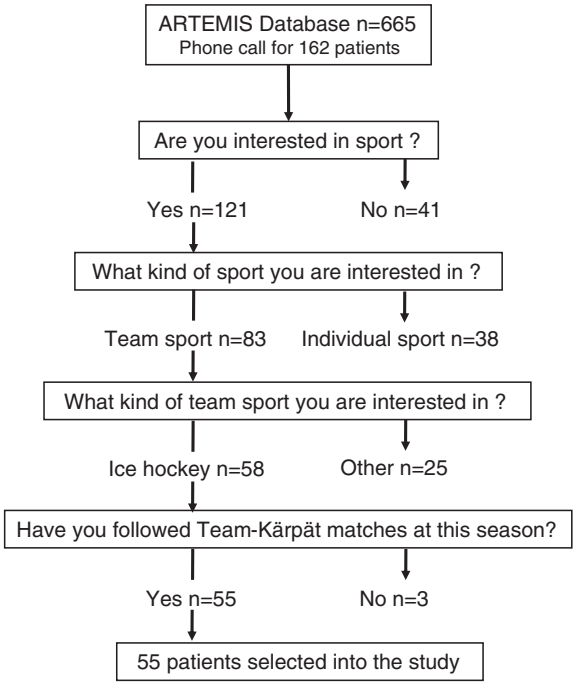

Fig. 1. The patient's selection protocol from ARTEMIS database.

Table 1
Characteristics of the study group.

	All	Ambulatory ECG	Ambulatory blood pressure	Beat-to-beat blood pressure
	$n=55$	$n=40$	$n=14$	$n=21$
Sex/male	55 (100%)	40 (100%)	14 (100%)	21 (100%)
Age (years)	60±10	60±9	60±9	58±11
BMI	29±4	29±4	28±4	29±4
Diabetes	23 (42%)	18 (46%)	7 (50%)	8 (42%)
Current smokers	3 (5%)	2(5%)	2 (14%)	2 (10%)
Sleep apnea	5 (9%)	4 (10%)	0	2 (10%)
Hypertension	33 (60%)	24 (60%)	7 (50%)	14 (67%)
History of AMI	26 (47%)	17 (43%)	7 (50%)	10 (48%)
Revascularized	47 (85%)	34 (85%)	13 (93%)	17 (81%)
CABG	19 (35%)	16 (40%)	7 (50%)	4 (19%)
PCI	31 (56%)	20 (50%)	7 (50%)	11 (52%)
Angina pectoris				
CCS Class 1	46 (83%)	35 (88%)	11 (79%)	15 (71%)
CCS Class 2	8 (15%)	4 (10%)	2 (14%)	5 (24%)
CCS Class 3	1 (2%)	1 (2%)	1 (7%)	1 (5%)
1-vessel CAD	18 (33%)	13 (33%)	2 (14%)	7 (33%)
2-vessel CAD	14 (25%)	7 (18%)	4 (28%)	5 (24%)
3-vessel CAD	23 (42%)	20 (50%)	8 (56%)	9 (43%)
EF	65±7.7	64±7	65±10	64±9
E´/E	10±4.1	11±4.3	9.2±2.5	9.1±4.3
LVH (echo)	19 (35%)	14 (35%)	5 (36%)	6 (29%)
Bicycle stress test				
max load(W)	169±40	166±36	160±38	172±43
METS	6,8±1.7	6,8±1.6	6.7±1.7	7.0±1.8
Max ST depression>1 mm	19 (33%)	15 (38%)	4 (29%)	5 (24%)
Max heart rate	135±20	135±20	135±22	132±19
Medication				
Aspirin	53 (96%)	38 (95%)	14 (100%)	21 (100%)
Clopidogrel	21 (38%)	12 (30%)	6 (43%)	8 (38%)
Warfarin	1 (2%)	1 (3%)	0	0
Beta Blockers	52 (95%)	38 (95%)	14 (100%)	21 (100%)
Bisoprolol	35 (67%)	24 (63%)	7 (50%)	15 (71%)
Metoprolol	16 (31%)	14 (37%)	7 (50%)	5 (24%)
Atenolol	1 (2%)	0	0	1 (5%)
Calcium antagonists	12 (22%)	8(20%)	1 (7%)	6 (29%)
ACEI/ARB	37 (67%)	28 (70%)	12 (86%)	18 (86%)
Diuretics	15 (27%)	11 (28%)	2 (14%)	5 (24%)
Statin	55 (100%)	40 (100%)	14 (100%)	21 (100%)
Insulines	6 (11%)	4 (10%)	1 (7%)	2 (10%)
Oral antidiabetics	16 (29%)	11 (28%)	3 (21%)	4 (19%)
Nitrates	14 (25%)	9 (23%)	5 (36%)	2 (10%)

Values are means ± SD; BMI; body mass index, diabetes (all were type 2 diabetes), AMI; acute myocardial infarction, revascularized; the patients who had at least one of the procedures (CABG coronary artery by-bass grafting or PCI percutaneus coronary intervention) EF; Ejection Fraction; E´/E basal septal E`with tissue Doppler ratio to mitral inflow E wave, LVH left ventricular hypertrophy with ASE guidelines, CCS Canadian cardiology society functional class, CAD coronary artery disease; 1-, 2- and 3-CAD; angiografically evaluated proximal coronary arteries with more than 50% stenosis; ACEI angiotensin conversion enzymes inhibitor; ARB angiotensin receptor blocker; ACEI/ARB the patients using at least one of them.

2.3. Ambulatory 24-h ECG

Ambulatory ECG was recorded with a digital Holter recorder (Medilog AR12, Huntleigh Healtcare, UK) with an accuracy of 1 ms and saved on a computer for further analysis with custom-made software, as described earlier in detail (Huikuri et al., 1990, 1994) (HEARTS software Heart Signal Co, Kempele, Finland). First, HR variability was analyzed over the entire ice hockey match (3 h) and during the control measurement at the corresponding time of day (3 h). Second, HR variability indices that showed significant differences between the match and control measurements were analyzed in 1-h periods before, during, and after the match and on the control days to reveal more accurate temporal changes in these indices. Seven

patients were excluded from the analysis of HR variability due to technical artifacts during the Holter recordings, four due to non-arrival to the agreed measurements, and four due to episodes of atrial fibrillation during either the match or the control measurements.

To exclude all undesirable beats, which accounted for <2% of the data in each included subject, the R–R intervals were edited by visual inspection. We used the deletion method, which simply removes the edited R–R intervals and replaces each removed R–R interval with the following normal R–R interval (Salo et al., 2001). Mean heart rate and SDNNi were used as time-domain measures of HRV. The values of low-frequency power (LF, from 0.04 to 0.15 Hz) and high-frequency power (HF, from 0.15 to 0.4 Hz) were calculated from blocks of 512 beats. Heart rate, SDNNi, and very-low-frequency power (VLF, from 0.0033 to 0.04 Hz) were analyzed over the entire analysis period.

2.4. Ambulatory BP

BP was analyzed automatically every 15 min (SunTech Medical, USA) and average values were calculated over the entire match and during the control day. Average values of BP were also analyzed in 1-h periods before, during, and after the match and at a corresponding time on the control day. The maximum and minimum BP values were also analyzed during the match and the control day. One patient was excluded from the final analysis due to a change in antihypertensive medication, one due to atrial fibrillation, and one due to denial from the control recording.

2.5. Beat-to-beat R–R interval, BPs and respiration

Five-minute recordings of beat-to-beat signals and respiration frequency at the baseline (temperature 20° ± 1 ° C) and during the match (temperature 20° ± 1 ° C) were measured in a sitting position (spontaneous breathing) without any confounding factors such as body movements, breathe holding, eating, or drinking. The patients were instrumented and measured inside a private balcony in the hockey hall during the match with an excellent view of the playground. Standard lead-II ECG, continuous finger blood pressure (Finapres, Ohmeda, USA and Finometer, Finapres Medical Systems, The Netherlands), and breathing by plethysmography (Pneumotrace, ADInstruments, Australia) were measured and collected using a PowerLab data acquisition system (PowerLab/8SP, ADInstruments, Australia) with a sampling frequency of 1000 Hz. Blood pressure was also measured with an automatic BP device by Dimensional K-Sound Analysis (Tango, SunTech, USA) two times during the baseline and the match. The Finapres values were corrected by Tango measurement (Imholz et al., 1998). R–R intervals and beat-to-beat systolic BP values were extracted from the continuous ECG and BP recordings as discrete event series that were then interpolated at 2 Hz (Tiinanen et al., 2008). In order to have time-synchronous signals, respiration was down-sampled at 2 Hz, as well.

The power spectral analyses of R–R intervals and systolic BP variability were performed with customized software using an autoregressive model (Burg's algorithm) (Tiinanen et al., 2008). The Akaike information criterion was used to determine the model order, validated by testing the whiteness of prediction error, separately for the R–R intervals and the systolic blood pressure data. The power spectrum densities of the high- (HF, 0.15–0.04 Hz) and low-frequency (LF, 0.04–0.15 Hz) oscillations in the R–R intervals and systolic BP were calculated (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). The spectral powers are presented as absolute units (ms^2, $mmHg^2$). Baroreflex sensitivity (BRS) was analyzed using alpha (Pagani et al., 1988) and sequence methods (Bertinieri et al., 1985). BRS for the LF spectral band was also calculated after extracting the respiratory component ($BRS_{LF\text{-}filtered}$) using an LMS adaptive filter described previously (Tiinanen et al., 2008).

2.6. Bicycle exercise test

Exercising was started at an intensity of 30 w and increased by 10 w every minute until an average HR equal to the average HR during the ice hockey match was achieved (a difference<5 bpm compared with emotional excitement during the match) in each individual. This steady-state HR was maintained for a 5-min period without increasing the workload. The beat-to-beat R–R intervals, BP, and respiration were recorded as described above from the steady-state portion of the exercise test. Two subjects were excluded because the same steady-state HR could not be maintained during the exercise (>5 bpm compared with the average HR during the match), one subject due to a marked number of ectopic beats during the exercise, and two subjects because coherence between the R–R interval and BP was <0.5 and therefore the BRS values could not be calculated.

2.7. Statistical analyses

Standard statistical methods were used to calculate means and standard deviations. Normal Gaussian distribution of the data was verified by the Kolmogorov-Smirnov goodness-of-fit test (z value<1.0). Spectrum values of R–R interval data were not normally distributed and therefore a logarithmic transformation to the natural base was performed. The differences in HR variability and BP variables analyzed over the entire match and during the control measurement were assessed using an independent t-test (2-tailed). Analysis of variance for repeated measures with time and interaction followed by post-hoc comparison (independent 2-tailed t-test) was used in hour-by-hour analysis of HR variability and BP indices between the match and the control day. The differences in maximum and minimum HR, HR variability, and BP were analyzed by independent 2-tailed t-test. The differences in beat-to-beat data were analyzed by paired t-test (HR and HR variability) or by Wilcoxon Signed Rank Test (BRS). All the statistical calculations were performed with SPSS 17.0 for Windows.

3. Results

3.1. Ambulatory heart rate and HRV

The average HR and HR variability values analyzed over the match and the corresponding time of day during the control measurements are shown in Table 2. The average HR was higher during the match than during the control day (76 ± 13 vs. 68 ± 11 bpm, $p<0.01$). SDNNi was 65 ± 22 ms during the match and 77 ± 26 ms during the control day ($p<0.05$) and the HF power of R–R intervals was 4.5 ± 0.9 ms^2 during the match and 5.0 ± 1.0 ms^2 during the control day ($p<0.05$).

Temporal changes in HR and HR variability in the 1-h periods are shown in Fig. 2. Two hours before the match, HR was at a level equal to the match and the control day (70 ± 10 vs. 70 ± 12 bpm, respectively, p = ns). HR was significantly higher 1 h before the match than during

Table 2
Average values of HR variability and blood pressure analyzed over the entire ice hockey play-off final (3 h) and during the control measurement at the corresponding time of day (3 h).

	Control	Match	p-level
HR, bpm	68 ± 11	76 ± 13	$p=0.005$
SDNNi, ms	77 ± 26	65 ± 22	$p=0.027$
HF power, ms^2	5.0 ± 1.0	4.5 ± 0.9	$p=0.031$
LF power, ms^2	5.8 ± 0.9	5.5 ± 0.8	$p=0.285$
VLF power, ms^2	7.0 ± 0.6	6.8 ± 0.7	$p=0.108$
LF/HF ratio	2.9 ± 1.8	3.7 ± 2.0	$p=0.076$
BPsys mmHg	130 ± 11	153 ± 11	$p<0.001$
BPdia, mmHg	74 ± 8	87 ± 7	$p<0.001$

Values are means ± SD. HR; heart rate, SDNNi; standard deviation of normal-to-normal R–R intervals, HF; high frequency power of R–R intervals, LF; low frequency power of R–R intervals, VLF very low frequency power of R–R intervals, BP; blood pressure.

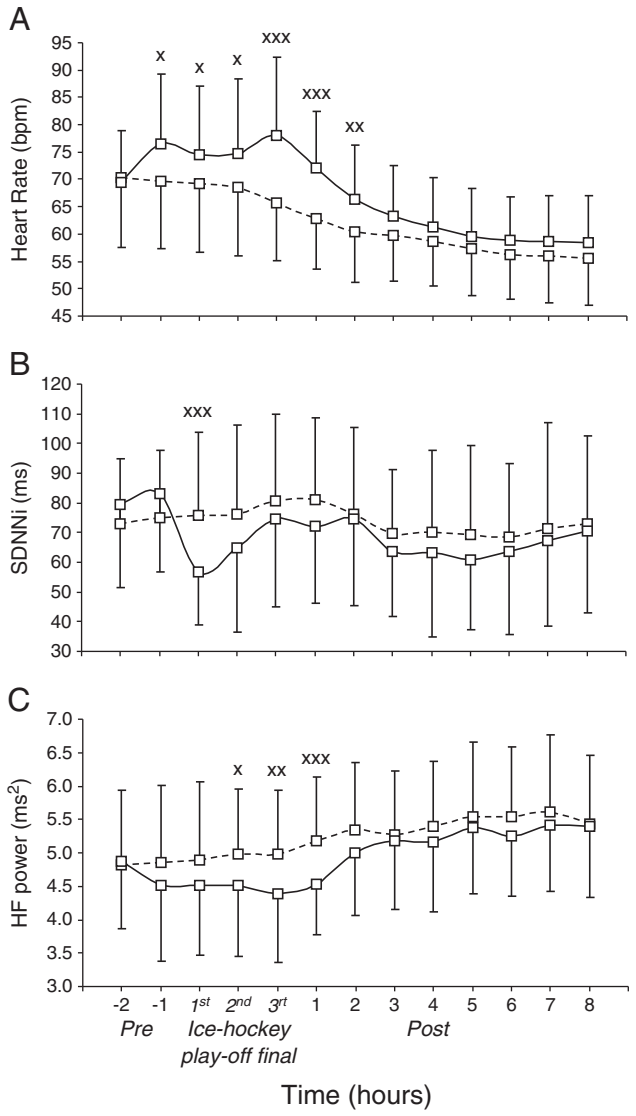

Fig. 2. Heart rate (A), standard deviation of normal-to-normal R–R intervals (SDNNi) (B), and the high-frequency power of R–R intervals (C) values before (pre), during, and after (post) the ice hockey play-off final ($n=40$). x: $p<0.05$, xx: $p<0.01$, and xxx: $p<0.001$ compared with control day values.

the control measurement (77 ± 12 vs. 69 ± 12 bpm, $p<0.05$) and remained significantly elevated during the entire duration of the match, e.g., 78 ± 14 vs. 65 ± 11 bpm ($p<0.001$) during the last hour of the match compared with the control period (Fig. 2A). HR was still elevated 2 h after the match compared with the control day (66 ± 9 vs. 60 ± 60 bpm, $p<0.01$). SDNNi was depressed during the first hour of the match compared with the control day and returned to the control level thereafter (Fig. 2B). The HF power of R–R intervals was at the same level before the match as on the control day but was significantly depressed during the match, e.g., 4.4 ± 1.0 vs. 5.0 ± 0.9 ms^2 during the last hour of the match vs. the control day ($p<0.01$), and remained attenuated 1 h after the match, 4.5 ± 0.7 vs. 5.2 ± 1.0 ms^2, $p<0.001$, respectively (Fig. 2C).

3.2. Ambulatory BP

The average values of systolic BP were 153 ± 11 and 130 ± 11 mmHg during the match and the control measurements, respectively ($p<0.001$). The corresponding diastolic BP values were 87 ± 7 and 74 ± 8 mmHg ($p<0.001$). Analysis of BP in 1-h periods revealed that BP was at the same level 2 h before the match as on the control day, e.g., systolic BP 134 ± 19 vs. 127 ± 16 mmHg, respectively, $p=$ ns. Systolic BP was significantly higher 1 h before the match than during the control period day and remained elevated until 1 h after the match, e.g., 157 ± 11 vs. 130 ± 19 mmHg at the beginning of the match compared with the control day, respectively, $p<0.001$ (Fig. 3A). Similarly, diastolic BP was significantly higher 1 h before the match and remained elevated until 1 h after the match, e.g., 90 ± 8 vs. 72 ± 10 mmHg during the first hour of the match compared with the control day, respectively, $p<0.001$ (Fig. 3B). The maximum values in both systolic (180 ± 14 vs. 145 ± 15 mmHg, $p<0.001$) and diastolic (103 ± 13 vs. 82 ± 11 mmHg, $p<0.001$) BP measured at any time were significantly higher during the match than during the control day. Examples of diastolic and systolic BP for one subject over 24 h during the match and control days are shown in Fig. 4.

3.3. Continuous cardiovascular signals

Beat-to-beat data during the controlled laboratory conditions, during the match, and during the exercise test are shown in Table 3. HR was significantly higher during the match compared with the control measurement (71 ± 15 vs. 61 ± 10 bpm $p<0.001$). SDNNi was at the same level in both condition (31 ± 13 vs. 31 ± 16 ms, $p=$ ns) but the HF power was lower (4.33 ± 1.38 vs. 5.03 ± 1.17 ms^2, $p<0.05$) and the LF/HF ratio was higher (2.43 ± 2.00 vs. 1.39 ± 1.27, $p<0.05$) during the match, when measured from the 5-min recordings.

Mean systolic BP was 157 ± 24 mmHg during the match and 132 ± 14 mmHg during the control measurement ($p<0.001$) and the corresponding maximum values were 179 ± 34 vs. 145 ± 18 mmHg ($p<0.001$). The mean diastolic BP values were 86 ± 13 mmHg and 76 ± 6 mmHg ($p<0.01$) during the match and control measurements, respectively, and the corresponding maximum values were 100 ± 15 vs. 82 ± 6 mmHg ($p<0.001$). The standard deviation of beat-to-beat systolic BP oscillation was 8.0 ± 4.0 mmHg during the match and 5.0 ± 2.1 mmHg during the control measurement ($p<0.01$). Both the HF

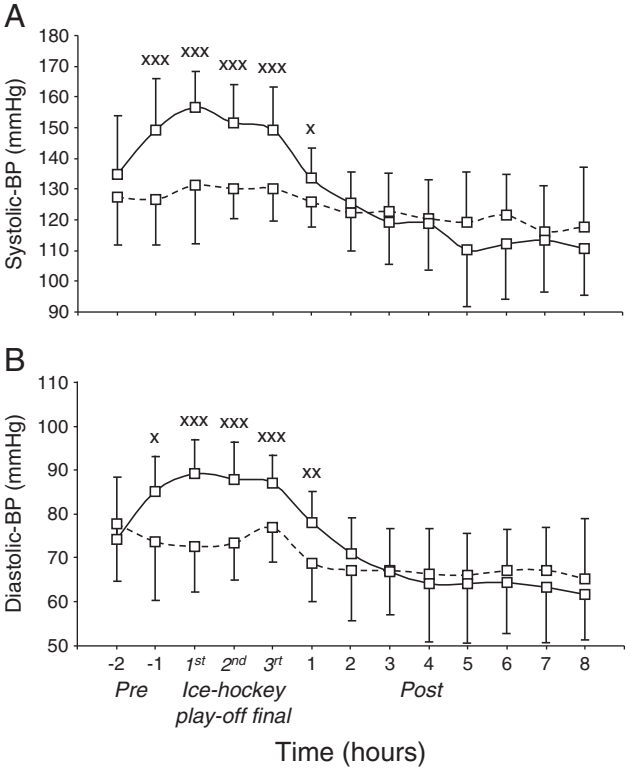

Fig. 3. Hour-by-hour ambulatory systolic (A) and diastolic (B) blood pressure values before (pre), during, and after (post) the ice hockey play-off final ($n=14$). x: $p<0.05$, xx: $p<0.01$, and xxx: $p<0.001$ compared with control day values.

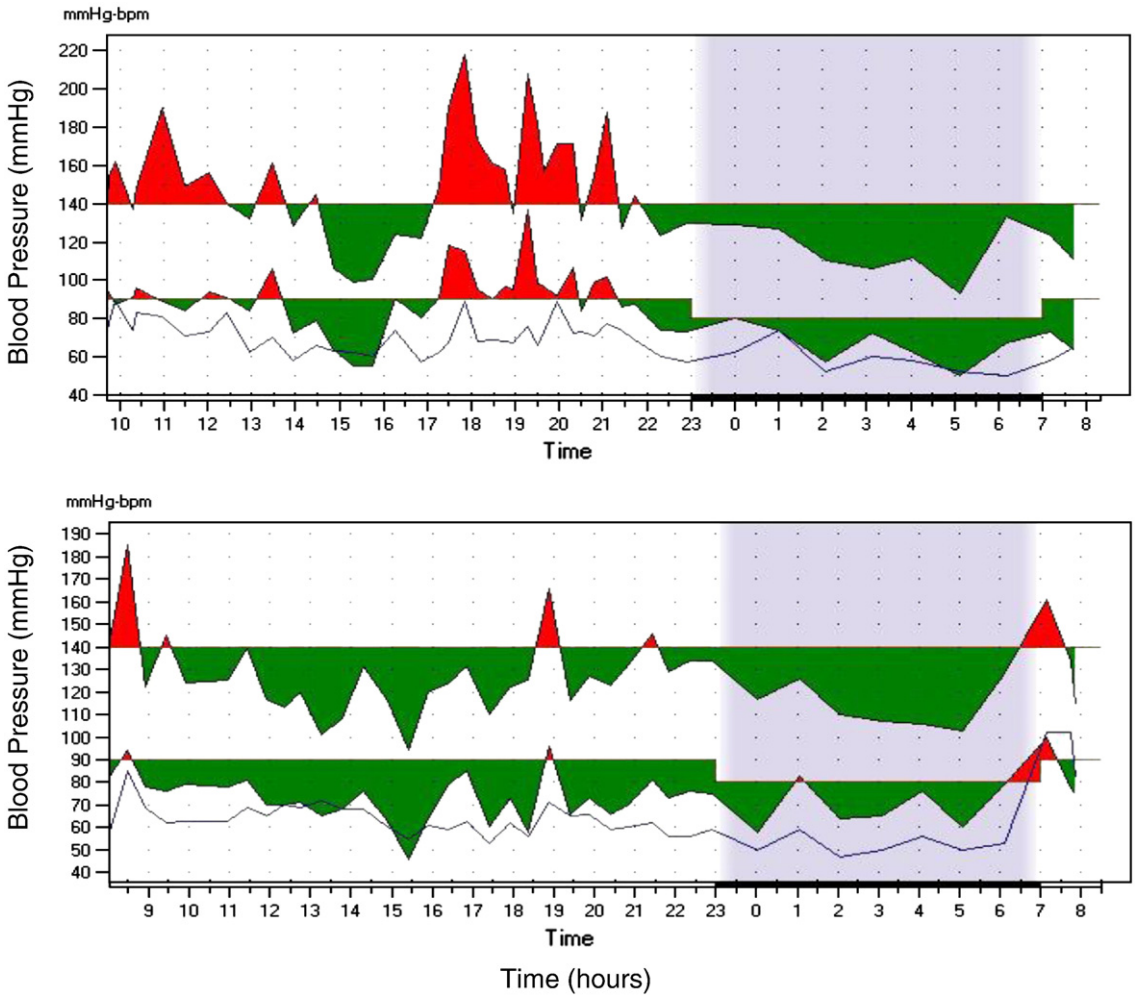

Fig. 4. Examples of systolic and diastolic blood pressure during the ice hockey play-off final day (upper panel) and during the control day (lower panel). The game started at 18:30 and ended at 21:30. The highest blood pressure measured during the match was 208/137 mmHg.

Table 3
Heart rate variability, blood pressure variability, and baroreflex sensitivity.

	Control	Match	Exercise	Match
	$n=21$	$n=21$	$n=16$	$n=16$
HR, bpm	61 ± 10	71 ± 15 ‡	77 ± 13	76 ± 14
SDNNi, ms	31 ± 13	31 ± 16	17 ± 6	28 ± 12 ‡
HF_{RR} power ln (ms^2)	5.03 ± 1.17	4.33 ± 1.38 *	3.91 ± 1.29	4.12 ± 1.09
LF_{RR} power ln (ms^2)	4.90 ± 1.08	4.84 ± 0.94	3.93 ± 0.99	4.75 ± 0.81 ‡
LF/HF	1.39 ± 1.27	2.43 ± 2.00 *	1.47 ± 1.61	2.56 ± 1.88 *
Mean BP_{sys} (mmHg)	132 ± 14	157 ± 24 ‡	155 ± 22	164 ± 22
Max BP_{sys} (mmHg)	145 ± 18	179 ± 34 ‡	168 ± 29	190 ± 34 *
SD BP_{sys} (mmHg)	5.0 ± 2.1	8.0 ± 4.0 †	4.9 ± 2.5	8.4 ± 4.3 ‡
$HF_{BP\text{-}sys}$ power ($mmHg^2$)	4.01 ± 3.66	5.32 ± 4.13 *	5.05 ± 6.04	6.03 ± 4.35
$LF_{BP\text{-}sys}$ power ($mmHg^2$)	5.37 ± 3.39	10.6 ± 6.8 †	9.0 ± 6.1	12.0 ± 6.7 †
Mean BP_{dia} (mmHg)	76 ± 6	86 ± 13 †	82 ± 10	88 ± 13 *
Max BP_{dia} (mmHg)	82 ± 6	100 ± 15 ‡	89 ± 10	104 ± 13 ‡
SD BP_{dia} (mmHg)	2.4 ± 0.7	3.7 ± 1.7 †	2.3 ± 0.9	3.9 ± 1.8 †
BRS_{HF}, (ms/mmHg)	9.4 ± 6.9	5.7 ± 4.4 †	5.5 ± 3.9	4.5 ± 3.7
BRS_{LF} (ms/mmHg)	6.8 ± 3.9	5.3 ± 4.2 *	3.1 ± 1.8	4.2 ± 3.3
$BRS_{LF\text{-}filtered}$ (ms/mmHg)	6.8 ± 3.9	5.0 ± 4.1*	3.0 ± 1.7	4.1 ± 3.4
BRS_{mean}, (ms/mmHg)	8.1 ± 4.5	5.5 ± 3.8 †	4.3 ± 2.6	4.4 ± 2.7
BRSseq (ms/mmHg)	8.7 ± 6.5	5.2 ± 4.5 †	4.2 ± 1.9	5.5 ± 3.9
Respiration (Hz)	0.23 ± 0.05	0.29 ± 0.05 ‡	0.29 ± 0.04	0.29 ± 0.05

Values are means ± SD, * $p<0.05$, † $p<0.01$, ‡ $p<0.001$. HR; heart rate, SDNNi; standard deviation of normal-to-normal R–R intervals, HF; high frequency power of R–R intervals, LF; low frequency power of R–R intervals, VLF very low frequency power of R–R intervals, BP; blood pressure, BRS; baroreflex sensitivity.

(5.32 ± 4.13 vs. 4.01 ± 3.66 $mmHg^2$, $p<0.05$) and LF (10.6 ± 6.8 vs. 5.37 ± 3.37 $mmHg^2$, $p<0.01$) power of systolic BP oscillation were significantly higher during the match than during the control measurements. Similarly, the standard deviation of beat-to-beat diastolic BP oscillation was elevated during the match compared with the control measurement (3.7 ± 1.7 vs. 2.4 ± 0.7 mmHg, $p<0.01$). All the BRS values were attenuated during the match compared with the control measurements, e.g., BRS analysed from the LF band by spectral techniques after extracting the respiratory component ($BRS_{LF\text{-}filtered}$) was 5.3 ± 4.1 ms/mmHg during the match and 6.8 ± 3.9 ms/mmHg during the control measurements ($p<0.05$). The frequency of respiration was higher during the match compared with the control period.

3.4. Continuous cardiovascular signals during the match vs. exercise

The average HR was titrated to an equal level during the match and the exercise (76 ± 14 vs. 77 ± 13 bpm). SDNNi (28 ± 12 vs. 17 ± 6 ms, $p<0.001$), the LF power of R–R intervals (4.75 ± 0.81 vs. 3.93 ± 0.99 ms^2, $p<0.001$), and the LF/HF ratio (2.56 ± 1.88 vs. 1.47 ± 1.61, $p<0.05$) were significantly higher during the match compared with the values during the exercise. The mean systolic BP did not differ significantly between the match and the exercise (164 ± 22 vs. 155 ± 22 mmHg, p = ns) but mean diastolic BP was significantly higher during the match than the exercise (88 ± 13 vs. 82 ± 10 mmHg, $p<0.05$). Both maximum systolic (190 ± 34 vs. 168 ± 29 mmHg, $p<0.05$) and maximum diastolic (104 ± 13 vs. 89 ± 10 mmHg,

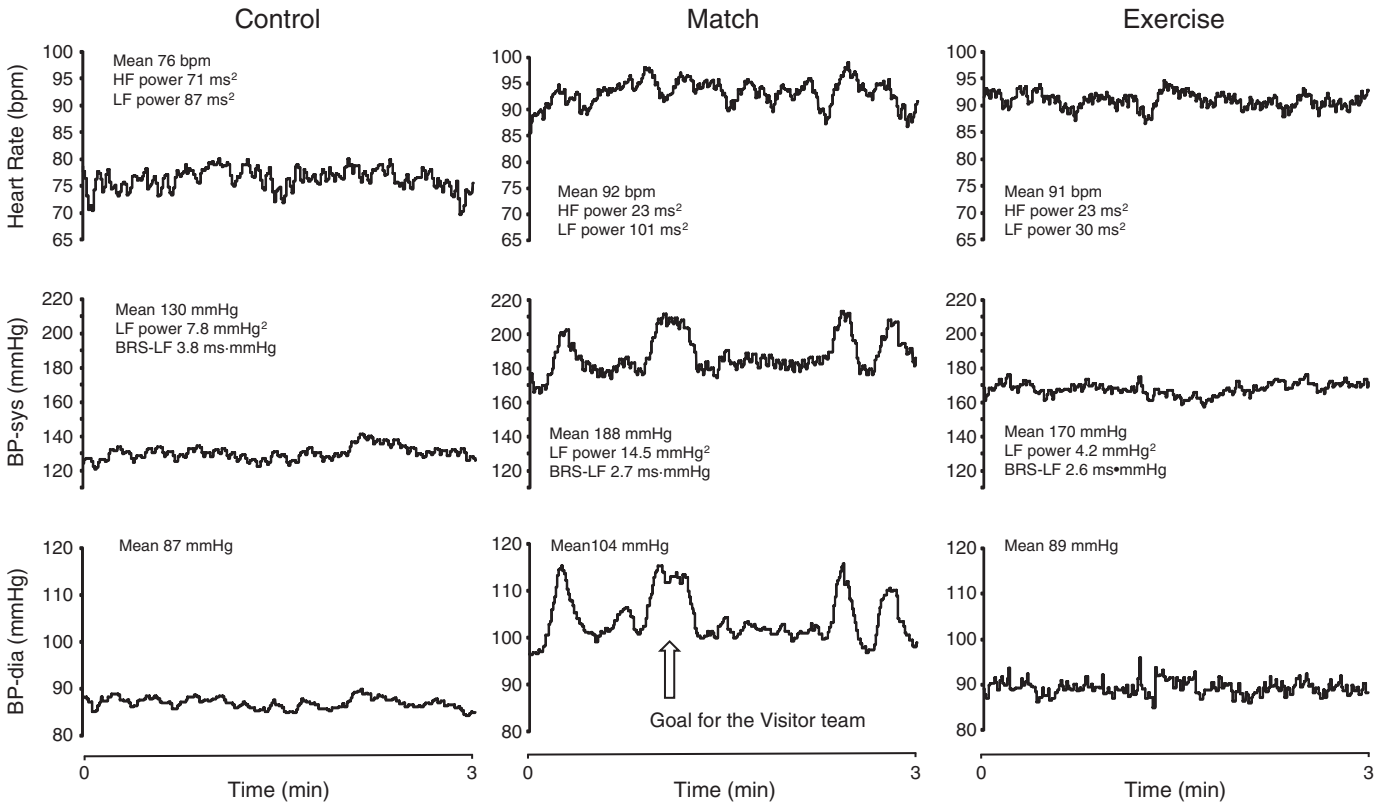

Fig. 5. R–R intervals (upper panel), beat-to-beat diastolic (middle panel) and systolic (lower panel) blood pressure at the baseline (control), during the ice hockey play-off final (match), and during the bicycle exercise (exercise).

$p<0.001$) BP were higher during the match compared with the exercise.

BP variability was higher during the match than during the exercise. Especially, the LF component of systolic blood pressure oscillation was elevated during the match compared with the exercise (12.0 ± 6.7 vs. 9.0 ± 6.1 mmHg^2, $p<0.01$). Similarly, the standard deviation of beat-to-beat diastolic BP was elevated during the match compared with the exercise (3.9 ± 1.8 vs. 2.3 ± 0.9 mmHg, $p<0.01$). BRS was depressed similarly during both emotional and exercise stress compared with the baseline measurements. The R–R intervals and corresponding beat-to-beat systolic and diastolic blood pressure data during the baseline (*left*), during the match (*middle*), and during the exercise (*right*) for one subject are shown in Fig. 5. Note the rapid elevations in both systolic and diastolic blood pressure during an exciting period of the match (goal for the visitors), which results in markedly elevated LF oscillation of BP (Fig. 5).

4. Discussion

There are several new observations regarding autonomic responses to leisure-time emotional excitement in cardiac patients in this study. In addition to an increase in systolic BP and HR, diastolic BP increased markedly during emotional excitement caused by a thrilling sports event. Many patients reached high temporary BP values despite appropriate beta-blocking and other cardiac medication. Secondly, an increase in diastolic BP was opposite to that which occurred during the physical exercise, which caused a subtle decrease or no change in diastolic BP. Thirdly, the elevation in BP occurred rapidly during the exciting periods of the match without any body movements or other confounding factors and resulted in markedly increased oscillations of both diastolic and systolic BP. Finally, the BP oscillations and HR variability responses during emotional excitement were different from those that occurred during physical exercise, despite similar average HR and respiration rate. Emotional excitement resulted in more unpredictable and more extensive variability in BP documented by differences in the spectral and time domain analyses of BP variability between these conditions. BRS was similarly attenuated during the emotional excitement and physical exercise.

4.1. Methodological considerations

Previous reports have concentrated on investigating experimentally induced mental stress, such as a frustrating computer task, a stressful interview, or arithmetic tests in a laboratory environment, in cardiac patients (Brydon et al., 2010; Pagani et al., 1991a,b; Strike et al., 2004, 2006a,b) and healthy subjects (Pagani et al., 1991a,b). The effects of public speech on autonomic regulation have also been studied (Jiang et al., 1993; Goldberg et al., 1996; Strike et al., 2006a,b). However, there are several fundamental methodological differences between previous studies and the present study design. First, previous studies designs have mostly focused on stressful tasks that are relatively short (~5–10 min). Secondly, the all of the previous studies have been performed in a laboratory-like environment and therefore do not represent the effects of real-life emotional excitement on autonomic regulation. Thirdly, the spectators of the sport events are *willing* to participate to the event and therefore are most probably waiting for *positive* emotions during the events. On the contrary, the subjects who are participating to the metal stress tasks in the laboratory condition are rather *waiting* for *negative* emotions during the tests.

In this study, we performed the long-lasting ambulatory HR and BP measurements before, during and after the real life emotional excitement. Secondly, beat-to-beat cardiovascular signals and respiration were measured at sitting position during real-life emotional excitement to avoid possible confounding effects of body movements and eating/drinking during the match. Thirdly, our study population was very carefully selected and all were strongly interested in ice-hockey. Furthermore, control measurements were performed for all

the patients during the day without any exciting sports events in the Oulu region. Taken together, our experimental study design cannot compare to previous studies.

4.2. Ambulatory measurements

Despite the epidemiological data on cardiac events during a sports event or other long-term emotional stress like earthquakes (Leor et al., 1996) or acute anger (Mittleman et al., 1995; Moller et al., 1999), there is little information on the autonomic, HR, and BP responses to this type of naturally occurring emotional stress in cardiac patients. Spectral analysis of R–R intervals has shown that the HF power of HR variability decreases and the LF/HF ratio increases, suggesting reduced cardiac vagal outflow and altered sympatho-vagal balance after terrorist attacks and after earthquakes in subjects without documented cardiac disease (Lin et al., 2001; Lampert et al., 2002). Also, both systolic and diastolic BP increased during the Wenchuan earthquake in China in a small group of hypertensive or suspected hypertensive patients (Chen et al., 2009). Concurrent with the results of previous research studying hemodynamic responses to emotional stress in healthy subjects, both systolic and diastolic BP and HR increased and the HF power of R–R intervals decreased during the emotional excitement (ice hockey match) in stable CAD patients who were using their normal cardiac medication in the present study. A more comprehensive temporal analysis revealed that these changes occurred already 1 h before the match and remained altered 1 h thereafter. The number of cardiovascular events is also increased not only during a thrilling sports event, but also one to 2 h before and after the match (Wilbert-Lampen et al., 2008). Furthermore, the maximum values of BP were even more markedly elevated than the average values during the match compared with the maximum values during the control day. Regardless of the relatively few measuring points of ambulatory BP, these findings suggest that abrupt and unpredictable elevations in BP occur during this type of emotional excitement, probably during the thrilling periods of the match.

4.3. Continuous cardiovascular signals

To elucidate further the autonomic response to leisure-time emotional excitement, we measured simultaneous beat-to-beat R–R intervals, BP, and respiration during the ice hockey match and compared the results with the data obtained from a standardized laboratory environment. Similarly to ambulatory measurements, HR was elevated, the HF power of R–R intervals was reduced, and the LF/HF ratio increased during the match compared with the baseline condition. Interestingly, all the BP variability indices of both systolic and diastolic BP were markedly elevated and the BRS indices were depressed during the match. Similar findings have been observed also in a laboratory environment during mental stress in healthy subjects (Pagani et al., 1988, 1991a,b) and in CAD patients (Jiang et al., 1993).

Emotional excitement resulted in different HR and BP variability responses than physical exercise at the same average HR level and respiration rate. In addition to more marked elevation of diastolic BP, increased oscillations of both systolic and diastolic BP were observed during the ice hockey match compared with exercise stress. Also, the LF power of R–R interval variability and the LF/HF ratio were at a higher level during the match than during the exercise. Increased LF power of R–R intervals is probably a result of higher LF oscillations of BP causing increased afferent input to the vasomotor center and a consequent increase in efferent outflow to the sinus node. The HF power of R–R intervals was depressed similarly during emotional excitement and exercise stress compared with the baseline values, revealing a withdrawal of vagal outflow in both conditions (Tulppo et al., 1996, 1998). Furthermore, BRS was impaired in both emotional excitement and exercise stress compared with the baseline condition, as documented in earlier studies (Pagani et al., 1991a,b; Iellamo et al., 1997; Lucini et al., 2002; Heffernan et al., 2007; Niemela et al., 2008). All these changes together provide evidence of increased momentary sympathetic activity at the time of long-lasting withdrawal of vagal outflow during leisure-time emotional excitement.

4.4. Limitations

The sample size of this study was based only on the availability of the play-off finals in the city of Oulu during 2008 and 2009. A control group of healthy subjects was planned to be monitored in 2010, but the team did not qualify for the play-off finals. The ambulatory BP recordings were not performed for all subjects due to the limited number of measurement devices. Similarly, the number of the patients was limited in the beat-to-beat study due to practical reasons, since it was possible to measure only one subject at a time. Despite these practical limitations in this type of study design, the results were concordant, with little inter-subject variability in the main findings. Medication may also have influenced the observed results. We were not able to stop, e.g., the beta-blocking medication because of ethical reasons and a well-known withdrawal effect of beta-blocking cessation. However, we feel the observed results have more practical implications when the analyses are performed at a time when the patients are on their normal medication. Finally, HR and BP variability techniques are not direct measures of autonomic function and these techniques has been criticized recently (Parati et al., 2006). However, direct measures of autonomic activity for marked number of subjects like measurements of muscle sympathetic nervous activity directly from peroneus nerve during the match is very difficult due to the practical reasons.

4.5. Conclusion

Participation as a spectator in a thrilling sports event results in extensive and abrupt elevations in both systolic and diastolic blood pressure, increased BP variability, altered HR variability, and attenuated BRS in CAD patients. The BP and HR variability responses during leisure-time excitement differ from those occurring during physical exercise at the same HR level and respiration rate. These changes were consistent with decreased cardiac vagal outflow combined with abrupt sympathetic activation (Lambert et al., 2010). The autonomic reactions may partly explain the vulnerability to cardiovascular events caused by this type of leisure-time emotional excitement. The marked BP reactions occurred despite beta-blocking medication. Therefore, more targeted medication, e.g., extra dosing of beta-blocking medication before the match or combined alpha- and beta-blocking medication, should perhaps be considered for enthusiastic sports fans with CAD during exciting sports events.

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