About the stability of phase-shifts between slow oscillations around 0.1 Hz in cardiovascular and cerebral systems

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***Abstract*—One important feature of the baroreflex loop is its strong preference for oscillations around 0.1 Hz. In the present study, we investigated heart rate intervals, arterial blood pressure (BP), and prefrontal oxyhemoglobin changes during 5 minutes rest and during brisk finger movements in 19 healthy subjects. We analyzed the phase-coupling around 0.1 Hz between cardiovascular and (de)oxyhemoglobin oscillations, using the cross spectral method. The analyses revealed phase-shifts for slow oscillations in BP and heart rate intervals between -10° to -118° (BP always leading). These phase-shifts increased significantly ($p < 0.01$) in the movement session. The coupling between cardiovascular and oxyhemoglobin oscillations was less clear. Only 12 subjects demonstrated a phase coupling ($COH^2 \geq 0.5$) between oxyhemoglobin and BP oscillations. This may be explained by an overwhelming proportion of nonlinearity in cardiovascular and hemodynamic systems.**

The phase-shifts between slow cardiovascular and hemodynamic oscillations are relatively stable subject-specific biometric features and could be of interest for person identification in addition to other biometric data. Slow BP-coupled oscillations in prefrontal oxyhemoglobin changes can seriously impair the detection of mentally induced hemodynamic changes in an optical brain-computer interface, a novel non-muscular communication system.

***Index Terms*—cardiovascular oscillations, heart rate, blood pressure, oxyhemoglobin oscillations, near-infrared spectroscopy**

I. Introduction

SLOW arterial blood pressure and heart rate oscillations have been addressed in many studies. Rhythmic fluctuations in the heart rate were described, for the first time, by A. von Haller [1]. Mayer [2] was the first who reported on slow waves in human blood pressure around 0.1 Hz (so-called Mayer waves; for review see Julien [3]). The involvement of the baroreceptor reflex in the genesis of slow oscillations around 0.1 Hz was proposed by Guyton and Harris in 1951 [4]. Important components of the baroreflex loop are the baroreceptors, the cardiopulmonary receptors, the cardiovascular nuclei in the brain stem and the heart. Several simulation studies predicting Mayer waves have been described [5]–[9] and showed that one important feature of the baroreflex loop is its strong preference for oscillations around 0.1 Hz, which can be seen as "resonance" or "eigenfrequency" of the loop, and another feature is the lead of blood pressure oscillations before beat-to-beat interval (inverse of heart rate) oscillations.

Slow 0.1-Hz oscillations have been reported not only in the blood pressure (BP) and heart rate (HR) [3], [7], [9]–[11] but also in different cerebral signals, such as oxygen availability of cortical tissue, cerebral blood flow velocity, oxyhemoglobin changes, full-band EEG and cerebrospinal fluid [12]–[21]. The relationship between intrinsic 0.1-Hz oscillations in the resting brain and in the cardiovascular system deserves special attention, because the identification of the mechanism underlying the generation of these slow oscillations in central and autonomous systems is still elusive. Two theories have been proposed to explain the generation of 0.1-Hz oscillations: First, the generation of slow oscillations as resonance phenomenon of the baroreflex loop [7], [9], [10] and second, independently of baroreceptor influences probably as result of central oscillators. From a series of observations in anesthetized animals, we know that central oscillators may contribute to the generations of oscillations around 0.1-Hz (pacemaker theory; for details see [3]).

Of interest is the mutual interaction between brain and heart. Recently, Thayer and Lane [22] made a review starting with the work of Claude Bernard over 150 years ago about the physiology of heart-brain connections and discussing the pathways via which the prefrontal cortex can control cardiac activity. From non-human animal research, we have evidence that cortical systems, especially including the medial-prefrontal cortex act, as a network together with subcortical systems (known as "central autonomic network") to initiate and represent cardiac autonomic adjustments [23], [24]. Further insight in the brain-heart connections may be obtained when the phase coupling between slow fluctuations in cardiovascular and central autonomic networks through analysing blood pressure time series and (de)oxyhemoglobin (Hb, HbO_2) recordings at optodes placed over the prefrontal cortex.

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We hypothesized that cyclic finger movements at the resonance frequency of the baroreflex loop (∼0.1 Hz) would have an impact on the strength of phase coupling between slow oscillations in cardiovascular (BP, beat-to-beat intervals) and hemodynamic (HbO_2) systems and involve linear or nonlinear system components. In the former case the coherence may have a large value as a measure of linearity; the opposite would be true in the latter case [8], [17], [25]–[27].

Cyclic brisk finger movements are accompanied by two responses, the heart rate response and the blood pressure response. First, "central commands" [22], [28], which impinge upon brainstem cardiovascular nuclei induce cardiac slowing [29], [30] and efferences from motor cortex innervate the somatic musculature. Second, the reafferent input from kinaesthetic receptors evoked by the movement elicits changes in blood pressure known as "exercise pressure response" [31] and accounts for a cardiac acceleration. Finger movements are also accompanied by changes in motor cortex excitability associated to pre-movement negative potential shifts [32] and pre-movement EEG desynchronisation [33] both of frontal cortex origin.

Knowledge about the incidence of slow fluctuations in cerebral oxy- and deoxyhemoglobin changes is very important for developing a near-infrared spectroscopy (NIRS)-based brain-computer interface (BCI) [34]–[36]. Such an optical BCI is a new non-muscular communication and control system that directly measures (de)oxyhemoglobin changes associated with the user's intent and transforms the recorded signal into a control signal [37], [38]. Mentally elicited (de)oxyhemoglobin changes (e.g. through mental arithmetic or motor imagery [39], [40]) have a small magnitude and are often hidden in slow spontaneous (de)oxyhemoglobin fluctuations. For classification approaches it is essential to improve the signal-to-noise ratio and to decrement false classifications which occur primarily due to misclassification of physiological noise [41]. Regardless of the improvement of the signal-to-noise ratio it is of general interest to know how the couplings are distributed in a normal population.

The most common biometric method of person identification is through finger prints. This method is however not always secured enough and therefore it becomes important to find other biometric methods to replace or augment the fingerprint technology as e.g. by recording and analysing signals from the brain (EEG) and heart. While bioelectric signals from the brain have already been used as biometric measures for person identification [42]–[47], the use of cardiovascular features is relatively novel. It has already been shown that electrocardiogram (ECG) traces exhibit features that are unique to an individual and may be suitable as biometric measure [48]–[50]. While these identification methods rely only on the ECG signal, it is of interest whether the common analyses of ECG and blood pressure signals reveal also biometric information, especially when phase-shifts of slow oscillations are considered. Reproducible and stable phase shift measures could be one additional feature in a battery of biometric measures from brain and heart signals.

The present study aims to answer the following questions:

- Does a difference exist in the strength of phase-coupling between beat-to-beat fluctuations of arterial blood pressure and heart rate intervals during rest and cyclic finger movements?
- How many subjects display slow arterial blood pressure-coupled prefrontal (de)oxyhemoglobin fluctuations around 0.1 Hz?
- Are phase-shifts measures between 0.1 Hz oscillations in beat-to-beat heart rate intervals, blood pressure and (de)oxyhemoglobin suitable as biometric measures?

II. METHODS

A. Sample

Twenty-six naive subjects (12 male, 14 female) aged 19 to 31 years (23 $\pm$ 2.8, mean $\pm$ SD) participated voluntarily in the present study. All subjects were right-handed (Edinburgh-Handedness-Inventory (EHI) [51]), had normal or corrected to normal vision and were seated in a comfortable armchair for the experiments. All experiments were in compliance with the World Medical Association Declaration of Helsinki. The protocol was approved by the Ethics committee of the Medical University of Graz and the subjects gave informed written consent before the experiments.

B. Experimental paradigm

The study consisted of different sessions, namely data recording during rest, during self-paced brisk finger movements, periodic cued finger movements and cued finger movements in random intervals. Part of the data about self-paced movements has been published recently [52]. Here we report data from initial 5-minute recording periods during rest (labelled "rest") and two sessions with 40 cued brisk finger movements, one with periodically presented cues (labelled "EF1") and one with randomly presented cues (labelled "EF2"). From the 5-min blood pressure recording during rest, the power spectrum was estimated. When one dominant peak was found in the power spectrum between 0.07 and 0.13 Hz, it was interpreted as the "eigenfrequency" of the cardiovascular system. In the case of periodical stimulation (EF1) the inter-stimulus was equivalent to the subject-specific peak frequency. The intervals between randomly presented cues (EF2) varied between 9 and 23 seconds. For further details see [52].

After inspecting the data, five subjects were eliminated due to high blood pressure, irregular respiration, extra ventricular contractions, or recording artefacts. Two of the remaining 21 subjects were eliminated because of optode problems during the NIRS recordings. The data from 19 subjects (aged 23 $\pm$ 2.9 years; mean $\pm$ SD) are reported. HR and BP data are summarized in Table I.

C. Data recording and processing

For this study, we analyzed the ECG recorded bipolarly at electrodes placed on the thorax (filter setup: 0.5-100 Hz), the BP and the respiration. All of these signals were sampled at a frequency of 500 Hz. For BP recording, a continuous non-invasive monitoring system (CNAP^{TM} Monitor 500, CNSystems, Austria) was used. Data were recorded from the proximal limb of the index or middle finger. The respiration patterns were obtained by using a respiratory sensor (Respiratory Effort Sensor, Pro-Tech Services Inc., filter setup: 0.1-100 Hz). (De)oxyhemoglobin fluctuations were recorded with a custom made one-channel, continuous wave method based NIRS system (for details see [34]). The sources and the detector were placed over the frontal cortex 1.5 cm to the left and right of position FP1 according to the international 10/20 system for EEG recording [53]. A fifth-order Butterworth filter with a cut-off frequency of 0.9 Hz was used to remove variability due to the cardiac cycle. After detection of the beat-to-beat intervals (RRI) in the ECG signal (based on an algorithm using a filter bank to decompose the ECG signal into various subbands), the intervals were linearly interpolated, resampled at 2 Hz, and displayed as RRI time series. From the arterial blood pressure recording, the systolic (BPsys) and diastolic (BPdia) pulse pressure amplitudes were extracted, linearly interpolated, resampled at 2 Hz, and displayed as BPsys and BPdia time series. Power spectra were estimated for all signals after subtraction of the mean over all samples. Recording time of all variables varied, depending on the task between 5 min (rest), 8 min (EF1, cyclic movements) and 15 min (EF2, randomized movements). For all spectra calculations, 1024 samples were used. The spectral values were smoothed using a 31-point triangular window, and cross-spectra were calculated between all variables ([54]; for details see De Boer et al. [10], [11]). After an automatic search for the largest peak in the cross-spectrum in the range 0.07 - 0.13 Hz, the corresponding coherence (COH^2) and phase-shift (PHA) values were determined. When no peak was found in the indicated range, the frequency was set to 0.1 Hz. Examples of time series and cross spectra (coherence and phase) from two representative subjects are displayed in Fig. 1.

D. Statistics

Statistical analysis contained a Shapiro-Wilk test for testing normal distribution of the data and paired sample t-tests for evaluating differences in phase shifts and time delays. All relevant data were normally distributed. Because of multiple comparisons, the Holm-Bonferroni method was used for alpha adjustment to reduce the probability of a Type I error. Pearson's product moment correlation was used for evaluating possible relations between phase shifts in the different conditions.

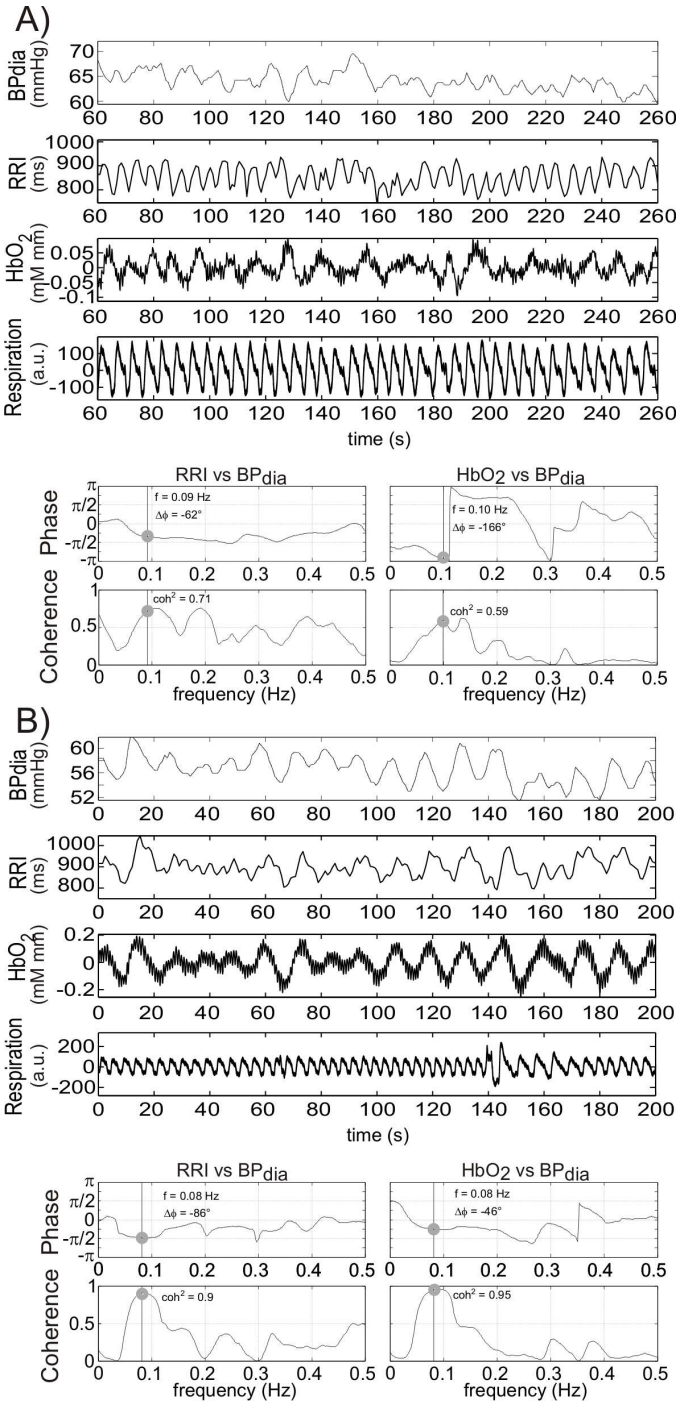

Fig. 1. Beat-to-beat time series (200 s) and spectra from 2 representative subjects (one with a large phase shift between blood pressure and HbO_2 (A) and one with a small phase shift (B)). Upper panels: Time series of BPdia (mmHg), RRI (ms), HbO_2 (mM*mm) and respiration (a.u.) during rest. Lower panels: Phase- and coherence spectra from RRI and BPdia time series (left side) and from HbO_2 and BPdia time series (right side). The automatically determined frequency, phase and coherence values are indicated by vertical lines.

III. RESULTS

A. Phase-shifts between slow BP and RRI oscillations around 0.1 Hz

Fig. 1 shows two representative examples of BPdia, RRI, HbO_2 and respiration signals recorded during rest (upper

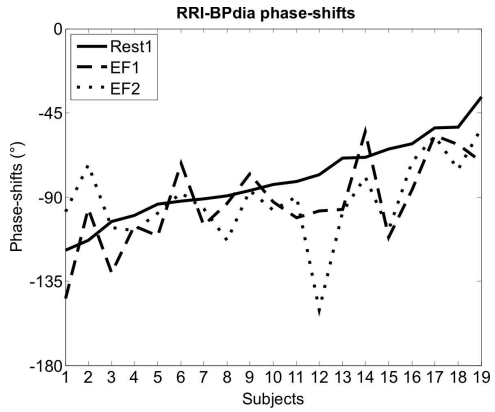

Fig. 2. Display of phase-shifts between slow RRI and BPdia oscillations (0° to -180°) for all 19 subjects for the sessions rest, cyclic (EF1) and randomized movements (EF2). Subjects are ordered according to the size of phase-shifts during rest.

panels) and the corresponding phase- and coherence spectra between RRI vs. BPdia and HbO_2 vs. BPdia (lower panels). One subject (Fig. 1A) displayed oscillations at 0.09 Hz with a large phase shift between HbO_2 and BPdia while the other subject (Fig. 1B) displayed oscillations with a frequency at 0.08 Hz and a small phase shift between HbO_2 and BPdia. Remarkable in Fig. 1 are the relatively similar phase shifts for cardiovascular oscillations in both subjects (between RRI and BPdia: -62° and -86°) and the quite different phase shifts (-166° and -46°) between HbO_2 and BPdia oscillations. The mean ± SD values for phase-coupling between arterial BP and RRI oscillations at ∼0.1 Hz for all 3 sessions are summarized in Table II. For example, a strong phase-coupling (COH^2 = 0.69 ± 0.17; mean ± SD) was found during rest not only for slow oscillations in BPdia and RRI (PHA = -82° ± 21°) but also for oscillations in BPsys and RRI (COH^2 = 0.68 ± 0.18; PHA = -35° ± 18°). Only one subject displayed a weak coupling ($COH^2 < 0.5$).

The analysis of phase shifts between slow blood pressure and RRI oscillations revealed not the expected significant stronger but only a tendency to a slightly stronger (p = 0.051) phase coupling during cyclic finger movements (COH^2 = 0.75) when compared to rest (COH^2 = 0.69). The phase shifts between RRI and BPdia, and between RRI and BPsys oscillations, were significantly larger ($p < 0.01$) in the cyclic movement session compared to rest (-93° to -81° and -51° to -35°, respectively). The phase-shift variations over all subjects are visualized in Fig. 2. Also, the time delays between RRI and BPsys oscillations were significantly longer ($p < 0.05$) during cyclic movement (-1.54 s) compared to the rest (-0.99 s). The correlation values across subjects between the different phase-shifts and sessions (rest, cyclic movement) are indicated in Fig. 3.

B. Phase-shifts between slow BP and prefrontal (de)oxyhemoglobin oscillations around 0.1 Hz

In detail, the phase coupling between following time series were estimated for the sessions rest, cyclic and randomized movements (in parenthesis, following the names of the two time series, the number of subjects with a $COH^2 \geq 0.5$ during rest are indicated): HbO_2 vs. BPdia (12 subjects), HbO_2 vs. RRI (6), HbO_2 vs. BPsys (5) HbO_2 vs. Hb (10), Hb vs. BPdia (6), Hb vs. RRI (7) and Hb vs. BPsys (5). The best results involved HbO_2 and BPdia: 12 out of 19 subjects showed a $COH^2 \geq 0.5$ during rest with COH^2 = 0.66 ± 0.15 (mean ± SD) and a relative variable phase-shift of PHA = -55° ± 46° documenting that the phase-shift between HbO_2 and BPdia (with BP leading) was varying between -1° and -167°

TABLE I
HR AND BP DATA

	HR (bpm)	BP_{sys} (mmHg)	BP_{dia} (mmHg)
Rest	71.9 ± 4.8	111.5 ± 4.3	70.2 ± 2.6
EF1	70.1 ± 4.4	112.5 ± 4.2	71.3 ± 3.1
EF2	70.2 ± 4.3	111.6 ± 4.5	70.1 ± 2.9

Mean values (± SD) of HR (bpm), BPsys (mmHg) and BPdia (mmHg) of 19 subjects for rest, periodic (EF1) and randomized movements (EF2).

TABLE II
PHASE-COUPLING BETWEEN RRI, ARTERIAL BP AND HbO_2

	RRI-BP_{sys}	RRI-BP_{dia}	HbO_2-BP_{dia}
	(N = 19)		*(N = 12)*
Rest			
Frequency (Hz)	0.10 ± 0.01	0.10 ± 0.02	0.10 ± 0.02
COH^2	0.68 ± 0.18	0.69 ± 0.17	0.66 ± 0.15
Time delay (s)	-0.99 ± 0.51	-2.35 ± 0.81	-1.49 ± 1.31
Phase shift (°)	-35 ± 18	-81 ± 21	-55 ± 46
EF1			
Frequency (Hz)	0.10 ± 0.01	0.10 ± 0.01	0.09 ± 0.01
COH^2	0.68 ± 0.23	0.75 ± 0.15	0.54 ± 0.25
Time delay (s)	-1.54 ± 0.88	-2.77 ± 0.92	-1.66 ± 1.37
Phase shift (°)	-51 ± 24	-93 ± 23	-56 ± 47
EF2			
Frequency (Hz)	0.10 ± 0.01	0.09 ± 0.02	0.10 ± 0.01
COH^2	0.64 ± 0.19	0.69 ± 0.16	0.52 ± 0.20
Time delay (s)	-1.51 ± 0.96	-2.88 ± 1.04	-1.62 ± 1.12
Phase shift (°)	-49 ± 25	-92 ± 22	-56 ± 36

Left: Frequency (Hz), coherence (COH^2), time delays (s) and phase shift (°) (mean ± SD) calculated for RRI and BPsys, and RRI and BPdia oscillations around 0.1 Hz for rest, EF1 and EF2.

Right: Frequency (Hz), coherence (COH^2), time delays (s) and phase shift (°) (mean ± SD) between HbO_2 and BPdia oscillations around 0.1 Hz for 12 selected subjects ($COH^2 \geq 0.5$) during rest.

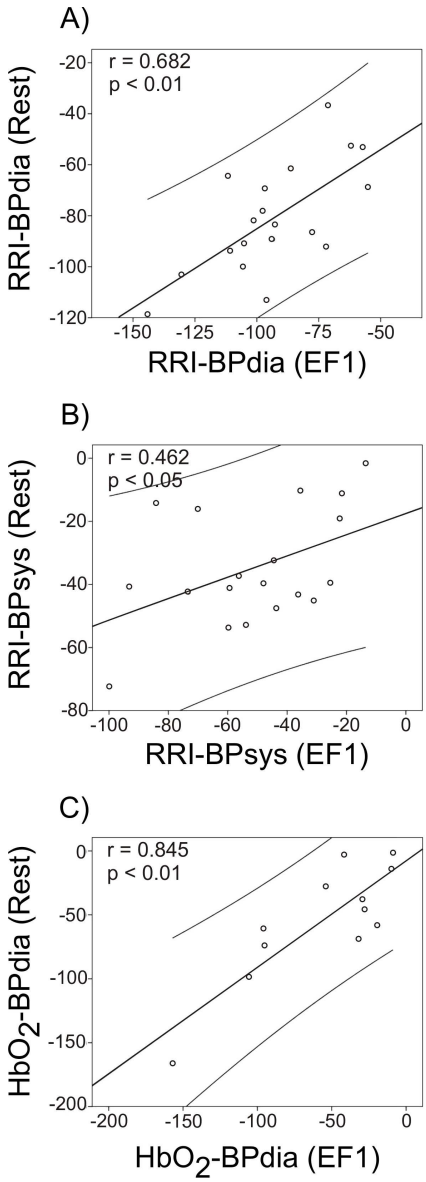

Fig. 3. Scatter plots displaying significant correlations between phase shifts during rest and during cyclic movements (EF1). The larger the phase shift between RRI and BPdia (A) respectively, RRI and BPsys (B) and RRI and HbO_2 (C) during rest, the larger the phase shift during cyclic movement, and vice versa. In addition, the line of best fit and a 95% confidence interval are plotted in the diagrams.

(see also Fig. 4). These phase-shifts were reproduced in the movement tasks. The following correlations were obtained: r = 0.85 ($p < 0.01$) for phase-shifts between rest and cyclic movements (Fig.3) and r = 0.91 ($p < 0.01$) between rest and randomized movements. These findings of the great intersubject variability of phase-shifts and the high correlation between recordings during rest and finger movement suggest that the phase-coupling between HbO_2 and BPdia may be one suitable feature for biometric identification. Of interest is also that 12 out of 19 subjects displayed a $COH^2 \geq 0.5$ for coupling between HbO_2 and BPdia time series, but only 5 subjects for coupling between HbO_2 and BPsys time series.

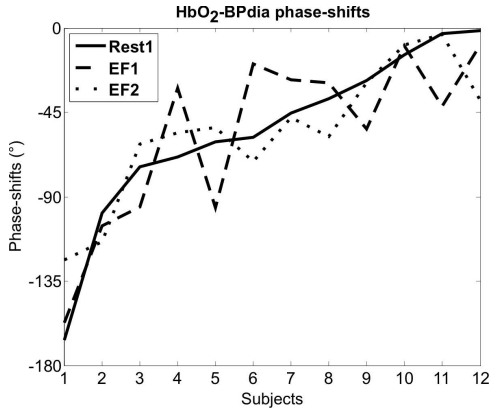

Fig. 4. Display of phase-shifts between HbO_2 and BPdia oscillations (0° to -180°) for 12 selected subjects ($COH^2 \geq 0.5$ during rest) for the sessions rest, EF1 and EF2. Subjects are ordered according to the size of phase-shifts during rest.

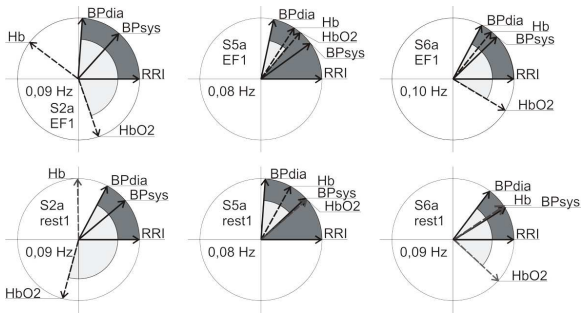

Fig. 5. Examples of vector diagrams from rest (lower diagrams) and movement sessions (upper diagrams) from 3 characteristic subjects. One with a large phase shift between BPdia and HbO_2 (left vector diagrams), one with a medium phase shift (right diagrams) and one with a small phase shift (middle diagrams). Phase-differences between BPdia and RRI are indicated by "dark grey" and between HbO_2 and BPdia by "bright grey". BPdia leads always RRI and Hb leads always HbO_2 oscillations around 0.1 Hz.

C. Visualization of phase-shifts in form of subject-specific vector diagrams

Vector diagrams were constructed for a better visualization of the individual phase-differences in the resting state and during cyclic finger movements. The vector diagrams of three different subjects are displayed in Fig. 5. Of interest are the different "resonance" frequencies between 0.08 Hz and 0.10 Hz, the difference in phase-shifts between subjects and the relative similarity of the phase-shift differences in the rest and movement sessions in all three subjects.

IV. DISCUSSION

A. Phase-coupling between slow arterial BP and RRI oscillations around 0.1 Hz

Abrupt decreases and increases in systolic arterial blood pressure produce baroreflex-mediated shortening and lengthening, respectively, of the beat-to-beat interval [55] and slow fluctuations in arterial pressure result in subsequent changes in beat-to-beat intervals in the same direction. The phase-shifts found during rest were -35°± 18° between RRI and BPsys oscillations and -81°± 21° between RRI and

BPdia oscillations around 0.1 Hz. Relationships between slow BP and RRI oscillations have been reported by cross spectral analyses [10], [11], [56]. The former reported a phase shift of 60° for the lead of systolic BP to RRI fluctuations and a phase shift of 90° for the lead of diastolic BP fluctuations and the latter a phase shift between -30° and -50° for BPsys and RRI oscillations in the range 0.07 - 0.13 Hz.

The expected higher phase coupling in all subjects during cyclic movement was not fulfilled. There was only a trend to a higher phase coupling but the increase of coherence reached not significance. In the case of a dominantly linear process, a coherence value close to 1.0 can be expected. Notably, during rest, no subject displayed a $COH^2 > 0.9$, while during cyclic movement, 4 subjects reached a $COH^2 > 0.9$. In some subjects, however, the coherence (actually COH^2) was also close to 0.5 during cyclic movement. This gives support to the notion that some components in the cardiovascular system are not linear [25], and these components vary from subject to subject. In contrast to the strength of phase coupling, the phase shift was significantly larger between slow oscillations in BPdia and RRI as well as BPsys and RRI during cyclic movement compared to rest. It is well-known that under such circumstances (cyclic movements), physiological oscillations may be entrained to behavioural responses and due to the latency (delay) of the BP response in the range of up to 6 s [57] and the pre-movement cardiac slowing [30], [52], [58] an additional time delay is introduced and the phase shift is enlarged.

B. Slow BP-coupled oscillations around 0.1Hz in prefrontal (de)oxyhemoglobin

The present study has used cross correlation technique in normal subjects to investigate whether slow HbO_2 oscillations around 0.1 Hz are related (phase-coupled) to arterial BP oscillations. In only 64% of the subjects, phase-coupled HbO_2 and BP oscillations were found. The mean coherence during rest was 0.66 $\pm$ 0.15 (mean $\pm$ SD), and the mean phase-shift was -55° $\pm$ 46°. Similar numbers were found during movement (see Table II). The high standard deviation around 40° in all sessions can be explained by the phase shifts varying between $\sim$zero (slow HbO_2 and BP oscillations are in-phase; for examples, see Fig. 1B and Fig. 5 middle panel) and $\sim$180° (slow oscillations are out-of-phase; for examples, see Fig.1A and Fig. 5 left panel) during rest but also in movement sessions. This is a novel finding and can be interpreted that in each subject a specific "wiring" exists within and between baroreflex loop and central autonomic networks. No significant phase shift changes were observed during cyclic or randomized movement compared to rest.

The reason for this relatively small number of phase-coupled HbO_2 and BP oscillations could be the overwhelming proportion of nonlinearity in cardiovascular and hemodynamic systems [8], [17], [25]–[27]. In all cases, the BP oscillations lead HbO_2 oscillations. These results suggest that, at least in some subjects, in addition to intrinsic hemodynamic oscillations, BP-coupled oscillations can also be found in the brain. Besides the NIRS technique, the transcranial Doppler (TCD) sonography technique can help to study the relationship between slow oscillations in the cerebral and cardiovascular systems. It is widely accepted that cerebral blood flow velocity (CBFV) as measured by TCD is proportional to the blood flow through insonated vessels and suitable to provide information about cerebral autoregulation [14]. Diehl et al. [59] reported a phase-shift between arterial BP and CBFV of 71° $\pm$ 30° (mean $\pm$ SD) in normal subjects. At this time it is not clear which relationship does exist between slow BP-coupled HbO_2 and CBFV oscillations. TCD measures the velocity (CBFV) of blood flow in the middle cerebral artery, whereas NIRS measures an averaged tissue concentration (HbO_2) in the illuminated region which consists of arterial, arteriolar, capillary and venous flow. An isolated increase of cerebral blood flow will lead to an increase in HbO_2 and a decrease in Hb, while an isolated increase in cerebral blood volume leads to an increase in both HbO_2 and Hb [60].

The stronger phase coupling between HbO_2 and BPdia on the one side, and the weaker coupling between HbO_2 and BPsys on the other, is unexpected and needs explanation. One reason could be that slow BPdia changes are clearly leading BPsys changes (see Table II and Fig. 5) and therefore also the BPdia may be seen as the driving force for slow HbO_2 oscillations. Another reason could be the physiologically plausible relationship between successive blood pressure values and RR intervals and respiratory variations: The diastolic pressure values depend mainly on the preceding diastolic pressure values and are minimally influenced by respiration; the BPsys is linearly related to RR intervals and strongly modulated by respiration (see models for the baroreflex loop [7], [11]). The more stable behaviour of diastolic pressure values could be also a reason for the stronger coupling between HbO_2 and BPdia compared to HbO_2 and BPsys.

V. CONCLUSION

Two points are of interest. First, in about 60% of the subjects, clear BP-coupled HbO_2 oscillations around 0.1 Hz were observed in the NIRS signal recorded over the prefrontal cortex. These oscillations can be seen as physiological noise [41] when an optical brain-computer interface (BCI) is realized. They can be removed e.g. by the use of a transfer function model [61]. The prefrontal cortex is of special interest because the optodes can be easily attached there [34], [35] and because the user's intent is accompanied by the activation of prefrontal networks [62]. Such optical BCIs with optodes placed over the prefrontal cortex might be suitable for use at home or work and can be realized as brain switch either alone or in combination with an EEG-based BCI [34], [35], [63], [64]. Second, phase-shifts between slow arterial blood pressure, heart rate beat-to-beat intervals and oxyhemoglobin oscillations are relatively stable, subject-specific and similar during rest and movement. This is an interesting observation, but it is still unclear if measures of the "eigenfrequency" of slow cardiovascular oscillations and phase shifts between cardiovascular and cerebral oscillations around 0.1 Hz are suitable biometric features for person

identification (even in combination with other biometric data). Further research is needed, especially to investigate the reproducibility and long-term stability of these parameters.

APPENDIX
LIST OF ABBREVIATIONS

BCI: Brain-computer interface
BP: Blood pressure (non-invasive recorded continuous signal)
BPsys: Systolic BP amplitude
BPdia: Diastolic BP amplitude
CBFV: Cerebral blood flow velocity
COH^2: Coherence squared
ECG: Electrocardiogram
EF1: Session with periodically presented cues
EF2: Session with randomly presented cues
EHI: Edinburgh- Handedness-Inventory
HR: Heart rate
Hb: Deoxyhemoglobin
HbO_2: Oxyhemoglobin
NIRS: Near-infrared spectroscopy
PHA: Phase-shift
RRI: Beat-to-beat interval
TCD: Transcranial Doppler sonography

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REFERENCES

[1] A. von Haller, *Elementa Physiologiae Corporis Humani Vol. II*. Lausanne, Swiss: Sumptibus M. M. Bousquet et Sociorum, 1760.
[2] S. Mayer, "Studien zur Physiologie des Herzens und der Blutgefässe. 6. Abhandlung: Über spontane Blutdruckschwankungen." in *Sitzungsberichte Akademie der Wissenschaften in Wien. Mathematisch-naturwissenschaftliche Klasse, Anatomie*, vol. 74, Wien, Austria, 1876, pp. 281–294.
[3] C. Julien, "The enigma of Mayer waves: Facts and models," *Cardiovasc Res.*, vol. 70(1), pp. 12–21, Apr. 2006.
[4] A. C. Guyton and J. W. Harris, "Pressoreceptor-autonomic oscillation; a probable cause of vasomotor waves," *Am J Physiol.*, vol. 165(1), pp. 158–166, Apr. 1951.
[5] R. DeBoer, J. Karemaker, and J. Strackee, "Hemodynamic fluctuations and baroreflex sensitivity in humans: a beat-to-beat model," *Am J Physiol.*, vol. 253, pp. H680–H689, 1987.
[6] J. Madwed, P. Albrecht, R. Mark, and R. Cohen, "Low-frequency oscillations in arterial pressure and heart rate: a simple computer model," *Am J Physiol.*, vol. 256(6), pp. H1573–H1579, Jun. 1989.
[7] A. M. VanRoon, L. J. Mulder, M. Althaus, and G. Mulder, "Introducing a baroreflex model for studying cardiovascular effects of mental workload," *Psychophysiology*, vol. 41(6), pp. 961–981, Nov. 2004.
[8] H. Seidel and H. Herzel, "Bifurcations in a nonlinear model of the baroreceptorcardiac reflex," *Physika D.*, vol. 115(1-2), pp. 145–160, Apr. 1998.
[9] K. Wesseling and J. Settels, "Baromodulation explains shortterm blood pressure variability," in *The Psychophysiology of Cardiovascular Control*, J. Orlebeke, G. Mulder, and L. VanDoornen, Eds. New York: Plenum Press, 1985, pp. 69–97.
[10] R. DeBoer, J. Karemaker, and J. Strackee, "Relationships between short-term blood-pressure fluctuations and heart-rate variability in resting subjects. I: A spectral analysis approach," *Med Biol Eng Comput.*, vol. 23(4), pp. 352–358, Jul. 1985.
[11] ——, "Relationships between short-term blood-pressure fluctuations and heart-rate variability in resting subjects. II: A simple model," *Med Biol Eng Comput.*, vol. 23(4), pp. 359–364, Jul. 1985.
[12] S. Vanhatalo, J. Palva, M. Holmes, J. Miller, J. Voipio, and K. Kaila, "Infraslow oscillations modulate excitability and interictal epileptic activity in the human cortex during sleep," *Proc Natl Acad Sci.*, vol. 101(14), pp. 5053–5057, Apr. 2004.
[13] R. Cooper, H. C. W. Walter, and A. Winter, "Regional control of cerebral vascular reactivity and oxygen supply in man," *Brain Res.*, vol. 3(2), pp. 174–191, Dec. 1966.
[14] R. Diehl, D. Linden, D. Lücke, and P. Berlit, "Spontaneous blood pressure oscillations and cerebral autoregulation," *Clin Auton Res.*, vol. 8(1), pp. 7–12, Dec. 1998.
[15] N. Lundberg, "Continuous recording and control of ventricular fluid pressure in neurosurgical practice," *Acta Psychiatr Scand Suppl.*, vol. 36(149), pp. 1–193, 1960.
[16] M. Schroeter, M. Bücheler, C. Preul, R. Scheid, O. Schmiedel, T. Guthke, and D. Y. von Cramonet, "Spontaneous slow hemodynamic oscillations are impaired in cerebral microangiopathy," *J Cereb Blood Flow Metab.*, vol. 25(12), pp. 1675–1684, Dec. 2005.
[17] G. Taga, Y. Konishi, A. Maki, T. Tachibana, M. Fujiwara, and H. Koizumi, "Spontaneous oscillation of oxy- and deoxy- hemoglobin changes with a phase difference throughout the occipital cortex of newborn infants observed using non-invasive optical topography," *Neurosci Lett.*, vol. 282(1-2), pp. 101–104, Mar. 2000.
[18] V. Toronov, M. Franceschini, M. Filiaci, S. Fantini, M. Wolf, A. Michalos, and E. Gratton, "Near-infrared study of fluctuations in cerebral hemodynamics during rest and motor stimulation: Temporal analysis and spatial mapping," *Med Phys.*, vol. 27(4), pp. 801–815, Apr. 2000.
[19] Y. Tzeng, S. Lucas, G. Atkinson, C. Willie, and P. Ainslie, "Fundamental relationships between arterial baroreflex sensitivity and dynamic cerebral autoregulation in humans," *J Appl Physiol.*, vol. 108(5), pp. 1162–1168, May 2010.
[20] R. Zhang, J. Zuckerman, C. Giller, and B. Levine, "Transfer function analysis of dynamic cerebral autoregulation in humans," *Am J Physiol.*, vol. 274(1 Pt 2), pp. H233–241, Jan. 1998.
[21] F. Zheng, A. Sassaroli, and S. Fantini, "Phasor representation of oxy- and deoxyhemoglobin concentrations: what is the meaning of out-of-phase oscillations as measured by near-infrared spectroscopy?" *J Biomed Opt.*, vol. 15(4), p. 040512, Aug. 2010.
[22] J. F. Thayer and R. D. Lane, "Claude bernard and the heart-brain connection: further elaboration of a model of neurovisceral integration," *Neurosci Biobehav Rev.*, vol. 33(2), pp. 81–88, Aug. 2008.
[23] E. Benarroch, "The central autonomic network: functional organization, dysfunction, and perspective," *Mayo Clin Proc.*, vol. 68(10), pp. 988–1001, Oct. 1993.
[24] L. Resstel and F. M. Correa, "Involvement of the medial prefrontal cortex in central cardiovascular modulation in the rat," *Auton Neurosci.*, vol. 126-127, pp. 130–138, Jun. 2006.
[25] C. Wagner and P. Persson, "Chaos in the cardiovascular system: an update," *Cardiovasc Res.*, vol. 40(2), pp. 257–264, Nov. 1998.
[26] X. Hu, V. Nenov, T. Glenn, L. Steiner, M. Czosnyka, M. Bergsneider, and N. Martin, "Nonlinear analysis of cerebral hemodynamics and intercranial pressure signals for characterization of autoregulation," *IEEE Trans Biomed Eng.*, vol. 53, pp. 195–209, 2006.
[27] C. A. Giller and M. Müller, "Linearity and non-linearity in cerebral hemodynamics," *Med Eng Phys.*, vol. 25, pp. 633–646, 2003.
[28] A. Verberne and N. Owens, "Cortical modulation of the cardiovascular system," *Prog Neurobiol.*, vol. 54(2), pp. 149–168, Feb. 1998.
[29] E. Damen and C. Brunia, "Changes in heart rate and slow brain potentials related to motor preparation and stimulus anticipation in a time estimation task," *Psychophysiol.*, vol. 24(6), pp. 700–713, Nov. 1987.
[30] D. Papakostopoulos, N. Banerji, and P. Pocock, "Performance, EMG, brain electrical potentials and heart rate change during a self-paced skilled motor task in parkinson's disease," *J Psychophysiol.*, vol. 4, pp. 163–183, 1990.
[31] B. Yates and S. Stocker, "Integration of somatic and visceral inputs by the brainstem: functional considerations," *Exp Brain Res.*, vol. 119(3), pp. 269–275, Apr. 1998.
[32] L. Deecke, B. Grözinger, and H. Kornhuber, "Voluntary finger movement in man: cerebral potentials and theory," *Biol Cybern.*, vol. 23(2), pp. 99–119, Apr. 1976.

[33] G. Pfurtscheller and F. L. da Silva, "Event-related EEG/MEG synchronization and desynchronization: basic principles," *Clin Neurophysiol.*, vol. 110(11), pp. 1842–1857, Nov. 1999.
[34] G. Bauernfeind, R. Leeb, S. Wriessnegger, and G. Pfurtscheller, "Development, set-up and first results of a one-channel near-infrared spectroscopy system," *Biomed Tech (Berl).*, vol. 53(1), pp. 36–43, 2008.
[35] S. Coyle, T. Ward, M. C., and G. McDarby, "On the suitability of near-infrared systems for next generation brain-computer interfaces." *Physiol Meas.*, vol. 25, pp. 815–822, Aug. 2004.
[36] F. Matthews, P. Pearlmutter, T. Ward, C. Soraghan, and C. Markham, "Hemodynamics for brain-computer interfaces: optical correlates of control signals." *IEEE Signal Process Mag.*, vol. 25(1), pp. 87–94, 2008.
[37] S. M. Coyle, T. Ward, and C. Markham, "Brain-computer interface using a simplified functional near-infrared spectroscopy system," *J Neural Eng.*, vol. 4(3), pp. 219–226, Apr. 2007.
[38] S. Luu and T. Chau, "Decoding subjective preference from single-trial near-infrared spectroscopy signals," *J Neural Eng.*, vol. 6(1), p. 016003, Apr. 2009.
[39] S. Wriessnegger, J. Kurzmann, and C. Neuper, "Spatio-temporal differences in brain oxygenation between movement execution and imagery: a multichannel near-infrared spectroscopy study," *Int J Psychophysiol.*, vol. 67(1), pp. 54–63, Jan. 2010.
[40] G. Pfurtscheller, G. Bauernfeind, S. Wriessnegger, and C. Neuper, "Focal frontal (de)oxyhemoglobin responses during simple arithmetic," *Int J Psychophysiol.*, vol. 76(3), pp. 186–192, Jun. 2010.
[41] S. Coyle, T. Ward, and C. Markham, "Physiological noise in near-infrared spectroscopy: implications for optical brain computer interfacing," *Conf Proc IEEE Eng Med Biol Soc.*, vol. 6, pp. 4540–4543, 2004.
[42] S. Marcel and J. R. Millán, "Person authentication using brainwaves (EEG) and maximum a posteriori model adaptation," *IEEE Trans Pattern Anal Mach Intell.*, vol. 29(4), pp. 743–752, Apr. 2007.
[43] M. Näpflin, M. Wildi, and J. Sarnthein, "Test-retest reliability of resting EEG spectra validates a statistical signature of persons," *Clin Neurophysiol.*, vol. 118(11), pp. 2519–2524, Nov. 2007.
[44] R. Palaniappan and D. P. Mandic, "Biometrics from brain electrical activity: a machine learning approach," *IEEE Trans Pattern Anal Mach Intell.*, vol. 29(4), pp. 738–742, Apr. 2007.
[45] R. Palaniappan and K. Ravi, "Improving visual evoked potential feature classification for person recognition using PCA and normalization," *Pattern Recognit Lett.*, vol. 27(7), pp. 726–733, May 2006.
[46] M. Poulos, M. Rangoussi, N. Alexandris, and A. Evangelou, "On the use of EEG features towards person identification via neural networks," *Med Inform Internet Med.*, vol. 26, pp. 35–48, Jan. 2001.
[47] L. Thie and E. Fried, "Acclairism: questioning biometric technology through an airport security clearance system," in *Conf. on Human Factors in Computing Systems, CHI '05 extended abstracts on Human factors in computing systems*, Portland, OR, USA: ACM, 2005, pp. 1148–1149.
[48] S. C. Fang and H. L. Chan, "Human identification by quantifying similarity and dissimilarity in electrocardiogram phase space," *Pattern Recognit.*, vol. 42(9), pp. 1824–1831, Sep. 2009.
[49] S. A. Israel, J. M. Irvine, A. Cheng, M. D. Wiederhold, and B. K. Wiederhold, "ECG to identify individuals," *Pattern Recognit.*, vol. 38, pp. 133–142, Jan. 2005.
[50] F. Sufi and I. Khalil, "Faster person identification using compressed ECG in time critical wireless telecardiology applications," *J Netw Comput Appl.*, 2010.
[51] R. Oldfield, "The assessment and analysis of handedness: the Edinburgh inventory," *Neuropsychologia.*, vol. 9, pp. 97–113, 1971.
[52] G. Pfurtscheller, R. Ortner, G. Bauernfeind, P. Linortner, and C. Neuper, "Does conscious intention to perform a motor act depend on slow cardiovascular rhythms?" *Neurosci Lett.*, vol. 468, pp. 46–50, Jan. 2010.
[53] H. Jasper, "The ten-twenty electrode system of the international federation." *Electroencephalogr Clin Neurophysiol.*, vol. 10, pp. 371–375, 1958.
[54] G. Jenkins and D. G. Watts, *Spectral analysis and its applications*. San Francisco, USA: Holden-Day, 1986.
[55] A. Steptoe and C. Vögele, "Cardiac baroreflex function during postural change assessed using non-invasive spontaneous sequence analysis in young men," *Cardiovasc Res.*, vol. 24(8), pp. 627–632, Aug. 1990.
[56] T. Kuusela, T. Kaila, and M. Kähönen, "Fine structure of the low-frequency spectra of heart rate and blood pressure." *BMC Physiol.*, vol. 13, pp. 3–11, Oct. 2003.
[57] G. Pfurtscheller, D. S. Klobassa, G. Bauernfeind, and C. Neuper, "Cardiovascular responses after brisk finger movement and their dependency on the "eigenfrequency" of the baroreflex loop," *Neurosci Lett.*, vol. 490, pp. 31–35, 2011.
[58] J. Jennings, M. V. der Molen, R. Somsen, and C. Terezis, "On the shift from anticipatory heart rate deceleration to acceleratory recovery: revisiting the role of response factors," *Psychophysiol.*, vol. 27, pp. 385–395, 1990.
[59] R. Diehl, D. Linden, D. Lücke, and P. Berlit, "Phase relationship between cerebral blood flow velocity and blood pressure. a clinical test of autoregulation." *Stroke*, vol. 3, pp. 1801–1804, Oct. 1995.
[60] M. Wolf, U. Wolf, V. Toronov, A. Michalos, A. L. Paunescu, J. Choi, and E. Gratton, "Different time evaluation of oxyhämoglobin and deoxyhämoglobin concentration changes in visual and motor cortices during functional stimulation: A near-infrared spectroscopy study." *Neuroimage*, vol. 16, pp. 704–712, 2002.
[61] G. Florian, A. Stancak, and G. Pfurtscheller, "Cardiac responses induced by voluntary selfpaced finger movements," *Int J Psychophys.*, vol. 28, pp. 273–283, 1998.
[62] P. Haggard, "Conscious intention and motor cognition," *Trends Cogn Sci.*, vol. 9, pp. 290–295, Jun. 2005.
[63] M. Naito, Y. Michioka, K. Ozawa, Y. Ito, M. Kiguchi, and T. Kanazawa, "Hemodynamics for brain-computer interfaces: optical correlates of control signals." *IEEE Signal Process Mag.*, vol. 25(1), pp. 87–94, 2008.
[64] G. Pfurtscheller, B. Allison, C. Brunner, G. Bauernfeind, T. Solis-Escalante, R. Scherer, T. Zander, G. Müller-Putz, C. Neuper, and N. Birbaumer., "The hybrid BCI," *Front Neurosci.*, vol. 4, p. doi:10.3389/fnpro.2010.00003, 2010.

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