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Does conscious intention to perform a motor act depend on slow cardiovascular rhythms?

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ABSTRACT

Slow oscillations around 0.1 Hz are characteristic features of both the cardiovascular and central nervous systems. Such oscillation have been reported, e.g. in blood pressure, heart rate, EEG and brain oxygenation. Hence, conscious intention of a motor act may occur only as a result of brain activity changes in frontal and related brain areas, or might be entrained by slow oscillations in the blood pressure. Twenty-six subjects were asked to perform voluntary, self-paced (at free will) brisk finger movements. Some subjects performed self-paced movements in relatively periodic intervals of around 10 s at the decreasing slope of the slow 0.1-Hz blood pressure oscillation. Our study reveals the first time that self-paced movements, at least in some subjects, do not stem from "free will" based on brain activity alone, but are influenced by slow blood pressure oscillations.

Voluntary, self-paced finger movement is preceded by an increased level of brain activity in form of a negative potential shift, the so called "Bereitschaftspotential", starting 1–2 s prior to movement onset [6]. Functional magnetic resonance imaging (fMRI) studies have recently demonstrated that free decisions to perform right or left finger movements showed activation in medial and lateral frontopolar corticies 7 s before the subject's motor decision [20]. This raises an interesting question: does conscious intention of a motor act occur in any phase-relationship to slow oscillations in the blood pressure? In other words, does conscious free will stem from brain activity alone [10], or can it also depend on the interoceptive system, including internal bodily events such as slow blood pressure waves [4]?

Functional MRI studies during rest, sometimes combined with EEG [14] or heart rate [7], have shown that there are fluctuations between 0.01 and 0.1 Hz in the resting-state fMRI BOLD signal. This work shows that the 0.1 Hz blood pressure oscillations and/or heart rate oscillations are among many variables responsible for fluctuations of brain activity.

People have various physiological rhythms, such as cardiovascular, respiratory and blood pressure rhythms [13]. Of special interest are slow blood pressure waves, with a frequency of around 0.1 Hz and respiratory rhythms between 0.2 and 0.5 Hz. The 0.1 Hz component may be related to the "eigenfrequency" of the baroreflex, which implies that this frequency can be seen as a type of resonance phenomenon within the baroreflex loop and/or the cardiovascular system [21,23]. Other slow oscillations also exist, such as the 0.15-Hz rhythm in the heart rate and arterial blood pressure, which exhibit varying phase and frequency coupling with the respiration induced through neurons in the reticular formation of the brain stem [16].

Twenty-six naive subjects (12 males, 14 females), aged 19–31 years ($M = 23 \pm 2.8$), participated in the present study. All subjects were right-handed and had normal or corrected to normal vision. 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 experiment. First, a 5-min recording was made while subjects rested to assess the spontaneous low-frequency oscillations around 0.1 Hz in the blood pressure during rest conditions. Next, subjects were instructed to perform voluntary, self-paced (at free will) brisk finger movements within a period of 10 min. No other instruction was given. Each subject's right index finger rested on a micro switch with a defined pressure point. Hence, a sloping edge was detected and stored that was synchronous to each movement onset. Similarly, a rising edge was synchronous to the offset.

Data from four subjects were rejected from further analysis due to high blood pressure, irregular respiration, extra ventricular contractions or recording artefacts. We measured the movement-to-movement intervals (MMIs) and calculated the mean ± SD and median for each of the remaining 22 subjects. Analysing the MMIs revealed a great intersubject variability. We formed three groups

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Table 1
Characteristic data from group A.

Subj./sex	Age (years)	Mean HR during rest (bpm) ± SD	Mean BP during rest (mmHg) ± SD		MMI		Number of MMIs	Rest EF (Hz) BPdia
			BPsys	BPdia	Mdn (s)	SDn		
AS2/m	26	70 ± 4	96 ± 6	64 ± 3	7.3	0.25	80	0.10
AS4/f	23	76 ± 3	107 ± 2	72 ± 1	9.9	0.24	59	0.11
AS6/m	23	92 ± 5	114 ± 4	77 ± 3	9.2	0.13	63	**0.09**
AS7/m	20	70 ± 5	108 ± 5	62 ± 2	7.3	0.36	79	**0.11**
AS8/f	20	75 ± 4	100 ± 5	54 ± 2	12.4	0.30	47	**0.09**
AU2/m	27	72 ± 6	125 ± 7	75 ± 3	12.2	0.28	48	0.10
AU3/m	23	65 ± 11	126 ± 4	74 ± 3	8.9	0.39	66	0.12
AT7/m	23	62 ± 5	113 ± 8	60 ± 2	10.4	0.35	43	**0.11**
AQ9/m	26	70 ± 3	117 ± 3	86 ± 1	12.0	0.25	58	0.08
Mean	23.4	72	112	69	10.0	0,28	60	0.10
STD	2.5	8	10	10	2.0	0,08	13	0.01

The table presents age, heart rate (HR), systolic (BPsys) and diastolic (BPdia) blood pressure (BP), movement to movement intervals (MMIs) with median and normalized SD (SDn), number of MMIs and the dominant spectral peaks around 0.1 Hz during rest (EF). This dominant peak is termed "eigenfrequency".

from the self-paced condition according to the median and the normalized SDn (mean/SD):

Group A: median ≥7 s and <13 s and SDn <0.4
Group B: median ≥13 s.
Group C: median <7 s and SDn <0.4

Group A: 9 subjects showed relatively constant intervals between consecutive movements of 10.0 s ± 2.0 (mean ± SD). Group B: 8 subjects performed self-paced movements in random intervals of 20.8 s ± 6.3. Group C: 5 subjects performed relatively fast and periodic self-paced movements in intervals of 4.55 s ± 1.32. Subjects in group C were excluded from further analysis because of their short inter-movement intervals related frequently to respiratory cycles. The remaining subjects executed the self-paced movements either relatively periodically, with a frequency close to 0.1 Hz (group A), or randomly (group B). Tables 1 and 2 present additional analyses of data from these two groups.

We continuously recorded the ECG bipolarly at electrodes placed on the thorax (filter setup: 0.5-100 Hz), blood pressure (BP), respiration and prefrontal changes of (de)oxyhemoglobin (Hb/HbO_2). For BP recording, a continuous non-invasive monitoring system (CNAP™ Monitor 500, CNSystems) 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). Hb/HbO_2 were recorded with a custom made one-channel near-infrared spectroscopy (NIRS) system [1]. The system uses the continuous wave (CW) method and measures hemodynamic concentration changes in HbO_2 and Hb. 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. A digital 3 Hz low pass Butterworth filter of order 5 with an attenuation of 30 dB in the stop band was used to allow down sampling to 10 Hz. Afterwards, a 0.01 Hz high pass filter was used to remove baseline drifts. For more details about the system see [1].

From the ECG, the QRS complexes were automatically detected based on an algorithm using a filter bank to decompose the ECG signal into various subbands. Afterwards, an interval series was formed, linearly interpolated, resampled at 4 Hz and displayed as HR intervals (RRI) series. From the BP recording, the beat-to-beat systolic (BPsys) and diastolic (BPdia) pulse pressures were extracted, linearly interpolated, resampled by 4 Hz and also time series generated. A transfer function model [9] was used to remove respiratory-related variability from instantaneous HR-, RRI-, BPdia- and BPsys-time series and Hb/HbO_2 signals. Auto spectra were calculated from the RRI, BP beat-to-beat time series and HbO_2 by applying Welch's method with a Hanning window. The single segments had a length of 100 s, with 50 s overlap. Welch's method was also used to estimate cross power spectral density and magnitude squared coherence (COH^2). For the phase-relationships, we chose the frequency with the highest squared coherence between RRI and BPdia series. With this frequency, it is possible to identify the phase- and time-delay from the cross power spectra. (time-delay = phase-delay/(2 × pi × frequency)). Fig. 1 presents the examples of different time series and their spectra. Each time series displays slow oscillations as documented by the 0.1-Hz peaks in the power spectra. In the spectra of BP and HbO_2, additional peaks at ~0.24 Hz are visible due to the respiration.

The power spectrum was calculated from the 5-min BPdia time series during rest. When one dominant peak was found in the

Table 2
Characteristic data from group B (For further explanation see Table 1).

Subj./sex	Age (years)	Mean HR during rest (bpm) ± SD	Mean BP during rest (mmHg) ± SD		MMI		Number of MMIs	Rest EF (Hz) BPdia
			BPsys	BPdia	Mdn (s)	SDn		
AS3/f	22	62 ± 3	113 ± 3	72 ± 2	16.5	0.47	24	0.11
AS5/m	31	68 ± 4	94 ± 4	56 ± 2	14.2	0.65	36	**0.08**
AT8/f	24	63 ± 4	102 ± 3	72 ± 2	15.4	0.1	39	0.08
AU1/m	23	89 ± 3	146 ± 2	87 ± 2	31.1	0.32	19	**0.11**
AU4/f	20	64 ± 3	107 ± 4	67 ± 2	28.0	0.35	20	**0.09**
AU5/f	24	70 ± 7	141 ± 3	108 ± 5	16.4	0.14	36	**0.11**
AU7/f	20	65 ± 3	103 ± 4	69 ± 2	24.4	0.31	24	0.1
AT6/m	27	68 ± 5	109 ± 4	71 ± 2	20.5	0.17	30	**0.1**
Mean	23.9	69	114	75	20.8	0.31	29	0.1
STD	3.7	9	19	16	6.3	0.18	8	0.01

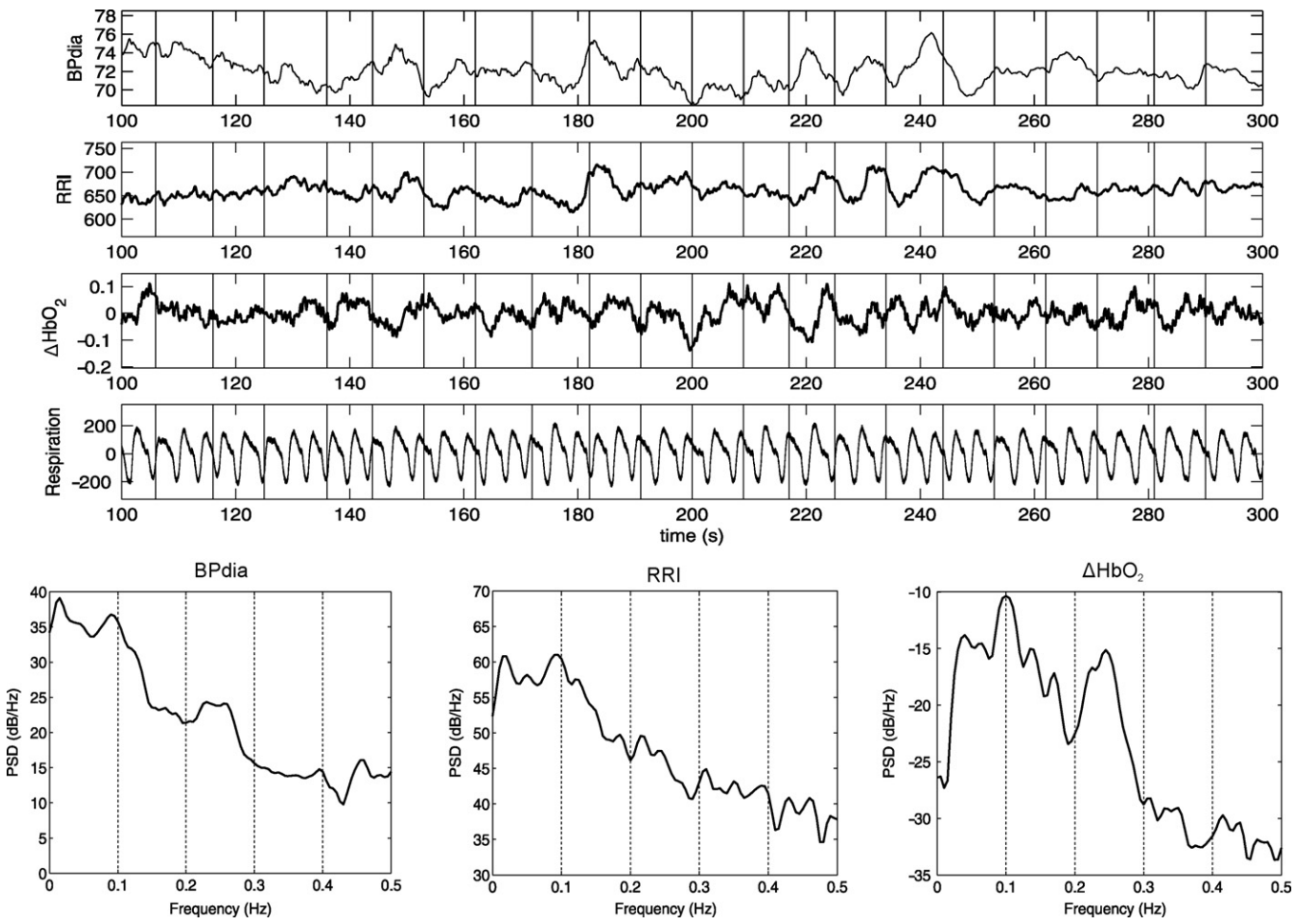

Fig. 1. Example of blood pressure (mmHg), RR intervals (ms), ΔHbO_2 and respiration time courses during self-paced movements (indicated by vertical lines) in subject AS6 with the corresponding power spectra for BP, RRI and HbO_2. Dominant peaks were found in only 4 subjects in group A and 5 subjects of group B (indicated in Tables 1 and 2 by bold numbers in the far right column). All other subjects showed either unclear peaks or two peaks within the investigated interval, whereby always the largest peak was selected automatically.

power spectrum between 0.07 and 0.13 Hz, it was interpreted as the "eigenfrequency" of the cardiovascular system (see Tables 1 and 2).

In a first step, both the respiration corrected beat-to-beat blood pressure and heart rate interval time series were averaged across all subjects of each group starting 6 s prior and ending 6 s after each movement onset. Fig. 2 presents the examples of the individual averaged diastolic blood pressure time courses and beat-to-beat heart rate responses, together with their grand average time courses. Prior to movement onset, the beat-to-beat heart rate intervals increased in all 9 group A subjects, who also clearly displayed a decreasing slope in their 0.1-Hz blood pressure oscillations. Both the blood pressure decrease and the beat-to-beat increase were highly significant (see indicated time windows in Fig. 2; paired sample t-test $p < 0.01$ and $p < 0.005$, respectively). No such movement-related changes were found in group B.

Second, the phase-relationship between RRI and BPdia were analysed for both groups by calculating cross spectra. The results are summarized in Table 3. We found that the mean time delay varied between 2.6 and 2.9 s without any significant differences between both groups, or between rest and movement conditions. Therefore, in all subjects from groups A and B, across both conditions, the 0.1-Hz BPdia oscillation led the 0.1-Hz HR oscillation by ~2.6 s.

Third, we investigated whether any relationship exists in group A between the "eigenfrequency" derived from the resting state and the movement-to-movement intervals. The subjects who exhibited relatively constant movement-to-movement intervals around 10 s displayed a weak negative correlation ($r = -0.524$, $p = 0.182$), with its "eigenfrequency". This weakness of the relationship is not surprising, since only 4 subjects displayed clear spectral peaks in the BP time series during rest (marked in Fig. 3 by circles). The weak correlation might also result from the small number of subjects, and because (in some subjects), one pressure wave sometimes occurred between consecutive movements, and other times, two blood pressure waves occurred instead. Summarizing, the weak correlation seen here may result from the small sample size and some confounds.

One of the goals of the complex, non-linear cardio-baroreceptor control system [18,21] may be to guarantee the blood and oxygen supplies, respectively, of all body parts and especially the central nervous system (CNS). An interesting feature of this system is the slow oscillation with a strong preference for frequencies around 0.1 Hz [23]. Noteworthily, such 0.1-Hz oscillations have been reported at different brain locations and in different physiological signals. For example, Cooper et al. [3] reported irregularly rhythmical changes at a frequency of about 0.1 Hz in the oxygen availability of cortical tissue, Schroeter et al. [17] reported on spontaneous low-frequency oscillations of oxyhemoglobin at optodes places over the occipital cortex, and Witte et al. [24] described the occurrence of EEG bursts in full-term newborns ("tracé alternant" EEG pattern) in intervals of approximately 10 s. Recently, slow frequency fluctuations in the range between 0.01 and 0.1 Hz have been

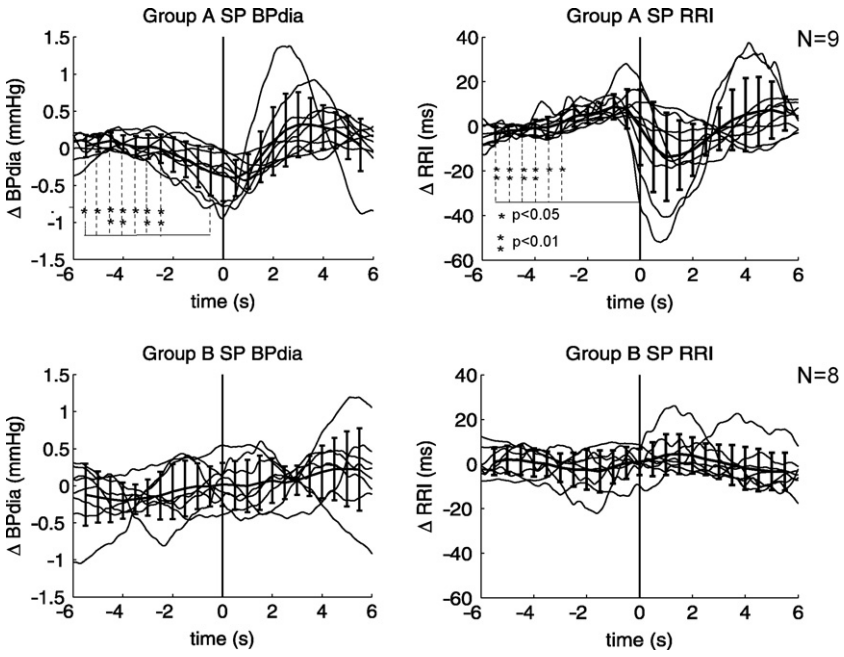

Fig. 2. Diastolic blood pressure and HR interval time series changes obtained in groups A (upper panels) and B (lower panels). (Left): Diastolic blood pressure changes ((BPdia); (Right): HR interval time series changes ((RRI). Individual and grand average time courses and the mean ± SDs calculated in consecutive 500-ms windows are displayed. The horizontal line at second 0 marks movement onset. Note also the 500-ms time windows displaying significant differences (*$p < 0.05$; **$p < 0.01$) either to 0 s (RRI) or 0.5 s prior to movement onset (BPdia).

Table 3
Phase-delays between BPdia and RRI oscillations around 0.1 Hz for groups A (left) and B (right) for rest and self-paced (SP) movement conditions with $COH^2 > 0.5$.

Subj.	BPdia vs. HR group A						Subj.	BPdia vs. HR group B					
	SP			Rest				SP			Rest		
	Freq (Hz)	Angle (°)	Time (s)	Freq (Hz)	Angle (°)	Time (s)		Freq. (Hz)	Angle (°)	Time (s)	Freq. (Hz)	Angle (°)	Time (s)
AS2	0.12	−102	−2.37	0.12	−111	−2.57	AS3	0.10	−72	−2.01			
AS4	0.12	−131	−3.04	0.10	−100	−2.77	AS5	0.10	−98	−2.72	0.08	−90	−3.14
AS6	0.10	−129	−3.60	0.10	−134	−3.72	AT8	0.10	−88	−2.45	0.08	−114	−3.96
AS7	0.12	−113	−2.60	0.12	−108	−2.49	AU1	0.12	−132	−3.05	0.11	−130	−3.29
AS8	0.12	−85	−1.97	0.08	−50	−1.72	AU4	0.10	−85	−2.36	0.10	−91	−2.53
AU2	0.12	−116	−2.70	0.13	−104	−2.23	AU5	0.10	−85	−2.35	0.09	−68	−2.24
AU3	0.13	−77	−1.65	0.13	−73	−1.56	AU7	0.08	−89	−3.08	0.08	−95	−3.28
AT7	0.12	−99	−2.59	0.12	−93	−2.15	AT6	0.10	−85	−2.37	0.10	−73	−2.02
AQ9	0.12	−112	−2.60	0.10	−94	−2.61	Mean	0.10	−92	−2.55	0.09	−95	−2.92
Mean	0.12	−107	−2.57	0.11	−96	−2.58	STD	0.01	18	0.37	0.01	22	0.69
STD	0.01	18	0.56	0.02	24	0.71							

reported in the resting-state fMRI BOLD signal related to HR [7,19] or EEG power fluctuations in various frequency bands [14]. All these observations suggest a close relationship between brain activity fluctuations at frequencies around 0.1 Hz and the timing of mental processes associated with (at least) the preparation and initiation of self-paced movements. Here, we report for the first time that a slow decrease of the 10-s blood pressure slope may act as a type of trigger for cortical activity changes associated with the initiation of voluntary movements.

In group A, we have to consider two types of mutually interacting 0.1 Hz oscillations. The first are the motor actions that result from central changes in motor excitability induced through 0.1 Hz oscillations (motor responses being entrained to 0.1 Hz pressure oscillations). The second oscillations are the induced blood pressure changes ("exercise pressor response" [12]) through muscle activation with a frequency of 0.1 Hz (physiological oscillations being entrained to behavioral responses). During a "steady-state" condition with two mutually interacting 0.1-Hz oscillations, the important condition of the baroreflex is fulfilled: the blood pressure change leads clearly the heart rate change (see Table 3). The heart rate changes are induced by "central commands" [22] which impinge upon brainstem cardiovascular nuclei and induce the pre-movement deceleration ([15], see Fig. 2 upper right diagram) closely related to the pre-movement blood pressure decrease (Fig. 2 upper left diagram). The preparatory HR deceleration is followed by a fast return to the baseline and a post-movement cardiac acceleration [9,11].

Why did only some subjects execute self-paced movements in ~10-s intervals, and how did the cardiovascular features differ between groups A and B? No difference between both groups was found in the "eigenfrequency" during rest (see Tables 1 and 2). No significant difference was found in the phase-coupling of 0.1-Hz oscillations in BPdia and RRI time series (see Table 3). Remarkably, 7 males were in group A, but only 2 males were in group B. This is interesting because there is strong evidence that males have a higher baroreflex gain than females [2]. This could indicate that subjects with higher baroreflex gain are more likely to exhibit self-paced movements in close phase-relationship to the 10-s blood pressure oscillations. Further research should explore whether "central commands" and/or stronger CNS inhibition through baroreceptor activation [8] could also differentiate between groups.

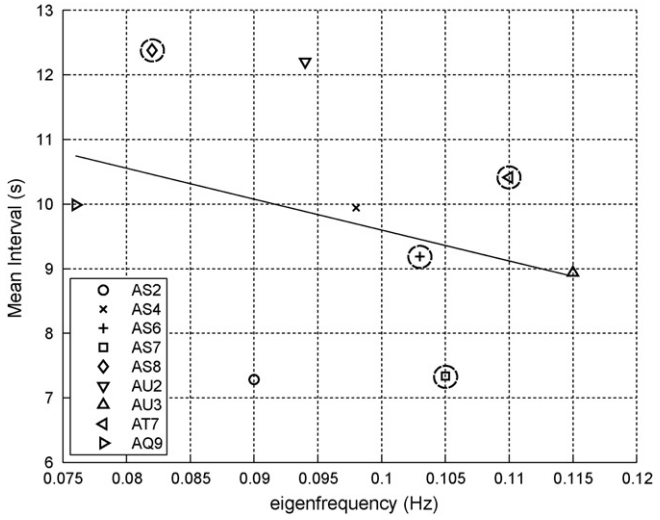

Fig. 3. Relationship between mean movement-to-movement intervals and "eigenfrequency" defined in the resting state without movement while performing voluntary, self-paced finger movements. Each subject is illustrated by a distinct symbol. The line fits the relationship in a least squares calculation (coefficient of determination $R^2 = 0.28$). The encircled subjects displayed clear spectral peaks in the BP time series.

Summarizing, some subjects (group A) performed voluntary, self-paced movements in relatively periodic intervals of around 10 s at the decreasing slope of the slow 0.1-Hz blood pressure oscillation. This means somatic markers [5] might be responsible for initiation of self-paced movements in some subjects, where the "eigenfrequency" of the blood pressure waves play a special role. Our study indicates that self-paced movements, at least in some subjects, do not stem from "free will" from brain activity alone, but are influenced by slow blood pressure oscillations.

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References

[1] G. Bauernfeind, R. Leeb, S. Wriessnegger, G. Pfurtscheller, Development, set-up and first results of a one-channel near-infrared spectroscopy system, Biomed. Tech. 53 (2008) 36–43.
[2] S.D. Beske, G.E. Alvarez, T.P. Ballard, K.P. Davy, Gender difference in cardiovagal baroreflex gain in humans, J. Appl. Physiol. 91 (2001) 2088–2092.
[3] R. Cooper, H.J. Crow, W.G. Walter, A.L. Winter, Regional control of cerebral vascular reactivity and oxygen supply in man, Brain Res. 3 (1966) 174–191.
[4] H.D. Critchley, S. Wiens, P. Rotshtein, A. Öhman, R.J. Dolan, Neural systems supporting interoreceptive awareness, Nat. Neurosci. 7 (2004) 189–195.
[5] A.R. Damasio, B.J. Everitt, D. Bishop, The somatic marker hypothesis and the possible functions of the prefrontal cortex [and discussion], Phil. Trans. R. Soc. Lond. Ser. B Biol. Sci. 351 (1996) 1413–1420.
[6] L. Deecke, B. Grözinger, H.H. Kornhuber, Voluntary finger movement in man: cerebral potentials and theory, Biol. Cybernetic 23 (1976) 99–139.
[7] J.C. de Munck, P. van Gelderen, J.A. de Zwart, S.G. Horovitz, M. Fukunaga, J.M. Jansma, J.H. Duyn, Low-frequency fluctuations in the cardiac rate as a source of variance in the resting-state fMRI BOLD signal, NeuroImage 38 (2007) 306–320.
[8] B.R. Dworkin, T. Elbert, H. Rau, N. Birbaumer, P. Pauli, C. Droste, C.H.M. Brunia, Central effects of baroreceptor activation in humans: attenuation of skeletal reflexes and pain perception, Proc. Natl. Acad. Sci. U.S.A. 91 (1994) 6329–6333.
[9] G. Florian, A. Stancak, G. Pfurtscheller, Cardiac response induced by voluntary self-paced finger movement, Int. J. Psychophysiol. 28 (1998) 273–283.
[10] P. Haggard, Conscious intention and motor cognition, Trends Cogn. Sci. 9 (2005) 290–295.
[11] J.R. Jennings, M.W. Van der Molen, R.J. Somsen, C. Terezis, On the shift from anticipatory heart rate deceleration to acceleratory recovery: revisiting the role of response factors, Psychophysiology 27 (1990) 385–395.
[12] M.P. Kaufman, H.V. Forster, Reflexes controlling circulatory, ventilatory, and airway responses to exercise, in: Handbook of Physiology. Exercise: Regulation and Integration of Multiple Systems, Am. Physiol. Soc., Bethesda, MD, 1996, pp. 381–447.
[13] H.P. Koepchen, Physiology of rhythms and control systems: an integrative approach, in: H. Haken, H.P. Koepchen (Eds.), Rhythms in Physiological Systems, Springer, Berlin, 1991, pp. 3–20.
[14] D. Mantini, M. Perucci, C.D. Gratta, G.L. Romani, M. Corbetta, Electrophysiological signatures of resting state networks in the human brain, Proc. Natl. Acad. Sci. U.S.A. 104 (2007) 13170–13175.
[15] D. Papakostopoulos, N.K. Banerji, P.V. Pocock, Performance, EMG, brain electrical potentials and heart rate change during a self-paced skilled motor task in Parkinson's disease, J. Psychophysiol. 4 (1990) 163–183.
[16] V. Perlitz, M. Lambertz, B. Cotuk, R. Grebe, R. Vandenhouten, G. Flatten, E.R. Petzold, H. Schmid-Schönbein, P. Langhorst, Cardiovascular rhythms in the 0.15-Hz band: common origin of identical phenomena in man and dog in the reticular formation of the brain stem? Pflügers Archiv: Eur. J. Physiol. 448 (2004) 579–591.
[17] M.L. Schroeter, M.M. Bücheler, C. Preul, R. Scheid, O. Schmiedel, T. Guthke, D. Yves von Cramonet, Spontaneous slow hemodynamic oscillations are impaired in cerebral microangiopathy, J. Cereb. Blood Flow Metab. 25 (2005) 1675–1684.
[18] H. Seidel, H. Herzel, Bifurcations in a nonlinear model of the baroreceptor-cardiac reflex, Physica D 115 (1998) 145–160.
[19] K. Shmueli, P. van Gelderen, J.A. de Zwart, S.G. Horovitz, M. Fukunaga, J.M. Jansma, J.H. Duyn, Low-frequency fluctuations in the cardiac rate as a source of variance in the resting-state fMRI BOLD signal, NeuroImage 38 (2007) 306–320.
[20] C.S. Soon, M. Brass, H.J. Heinze, J.D. Haynes, Unconscious determinants of free decisions in the human brain, Nat. Neurosci. 11 (2008) 543–545.
[21] A.M. Van Roon, L.J.M. Mulder, M. Althaus, Introducing a baroreflex model for studying cardiovascular effects of mental workload, Psychophysiology 41 (2004) 961–981.
[22] A.J.M. Verberne, N.C. Owens, Cortical modulation of the cardiovascular system, Prog. Neurobiol. 54 (1998) 149–168.
[23] K.H. Wesseling, J.J. Settels, Baromodulation explains shortterm blood pressure variability, in: J.F. Orlebeke, G. Mulder, L.P.J. Van Doornen (Eds.), The Psychophysiology of Cardiovascular Control, Plenum Press, New York, 1985, pp. 69–97.
[24] H. Witte, P. Putsche, K. Schwab, M. Eiselt, M. Helbig, T. Suesse, On the spatio-temporal organisation of quadratic phase-couplings in 'tracé alternant' EEG pattern in full-term newborns, Clin. Neurophysiol. 115 (2004) 2308–2315.