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Short communication

Early contingent negative variation of the EEG and attentional flexibility are reduced in hypotension

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Abstract

This study explored the question as to whether hypotension is related to decreased attentional performance and reduced cortical activation. A total of 50 females aged 19–44 years participated in the study. Attentional performance was assessed using three subtests of the Attentional and Cognitive Efficiency (ACE) battery. Contingent negative variation (CNV) as a measure of cortical activation was registered during a constant fore-period reaction time paradigm: two conditions were defined using tones as S1 (80 or 60 dB) and S2 (70 dB). The following results were obtained. Hypotensive patients performed significantly more poorly on one subtest of the ACE, which indicates a reduced speed for switching from a routine to a controlled response (quantifying attentional flexibility). They also had longer reaction times and revealed a significantly smaller amplitude of the early CNV component. In addition, a significant correlation was observed between systolic blood pressure and the amplitude of the early CNV component. The data support previous findings that hypotension can be related to lowered cortical activation and indicate that specific aspects of attentional performance might be negatively affected by hypotension.

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Essential hypotension can be described as the chronic condition of low blood pressure that is independent from another illness or pathology. There is no general agreement as to when exactly the diagnosis ‘hypotension’ applies. Most authors

suggest a systolic blood pressure (SBP) below 100–110 mmHg as a cut-off value. (Weiss and Donat, 1982; Pschyrembel, 1993). We refer to the ‘essential’ form when speaking of hypotension in this work, which should not be confused with secondary (i.e. symptomatic) or orthostatic forms. Even though essential hypotension is by far the most common type of hypotension (90%; Phillip, 1993), its aetiology is still unknown.

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Most medical textbooks emphasise that hypotension cannot be regarded as an illness, mainly because hypotension might have cardioprotective effects, and thus results in a higher life expectancy (Phillipp, 1993). In the face of hypertension as a major risk factor for coronary heart and cerebrovascular diseases, hypotension might therefore seem to be a desirable state at first glance. However, physicians are frequently faced with patients displaying subjective symptoms that seem to be very common, but not specific, among hypotensive patients such as: drop in performance, reduced drive and concentration, tiredness, problems getting started in the morning, enhanced urge for sleep, dizziness, buzzing in the ears, palpitations, heartache, headache, lack of appetite or cold hands and feet (Weiss and Donat, 1982; Pilgrim, 1994).

Considering the relatively high prevalence of hypotension, especially among younger women (10–20% of the female population aged 20–40 years according to Baenkler et al., 1999), psychophysiological research on hypotension has been very sparse and largely focused on personality psychological aspects (e.g. Schalling and Svensson, 1984; Berry and Lester, 1997; see also Pilgrim, 1994). In contrast to the large number of studies investigating cognitive performance in hypertension (for a review see Waldstein and Elias, 2000), such studies on hypotension were lacking until recently.

In a pioneering study, Stegagno et al. (1996) observed reduced performance in a hypotensive group in comparison to normotensive subjects in a free recall test of 16 words and a mental arithmetic task. In a second study, Costa et al. (1998) compared hypotensive and normotensive female participants on various variables of performance and on Contingent Negative Variation of the EEG (CNV; interstimulus interval, ISI: 2 s), which was recorded from the vertex (Cz). Their interpretation of reduced cognitive—especially attentional—performance relied on the following observations:

- On two standard German paper–pencil tests of sustained attention (a cancellation test, Brickenkamp, 1994; and a number-connecting test, Oswald and Roth, 1987), hypotensive subjects

demonstrated significantly lower performance than normotensive subjects.

- Hypotensive subjects remembered significantly fewer words than normotensive subjects in a free recall test (Baddeley, 1982) of a list of 16 words.
- With regard to CNV, hypotensive subjects exhibited a significantly less negative amplitude (calculated over seven time points: 500, 750, 1000, 1250, 1500, 1750 and 2000 ms) as compared with normotensive subjects.

The present study aimed at following questions:

- a. Which component of the CNV is affected in hypotensive individuals? Generally, the CNV is not a unitary concept: the waveform is made up of (at least) two different components—an early and a late wave—that are difficult to separate by choosing an ISI below approximately 3 s. These components seem to reflect functionally and neurobiologically different underlying processes: Tecce (1972) and Tecce and Cattanaach (1993), for example, proposed that the early component is invertedly U-related to the arousal level and that the late component is monotonically related to attention towards S2. The late wave also reflects processes related to motor preparation, thus constituting a ‘hybrid wave’ (Tecce, 1972). In a more neurobiological approach, Birbaumer et al. (1990) interpreted the early CNV as an indicator of prefrontal activation that—via the striatum and thalamus—selectively regulates activation thresholds of more posterior regions, as reflected by the late component. The study by Costa et al. (1998) left out this question, partly because of the short ISI of 2 s, and also partly because of the rather global concept of CNV (i.e. CNV as reflecting attention) by which the authors were guided. By extending the ISI to 4 s in this study, differential effects may be observed on the CNV components, thus giving more information as to which attentional and neurophysiological processes may be affected by hypotension.
- b. Do differences in CNV and reaction times (RT) depend on arousal level, as suggested by many subjective symptoms (and not a genuine prob-

lem of attention as suggested by Costa et al., 1998)? For this purpose we varied the intensity of the warning signal (S1) of the CNV paradigm. Although most work on CNV and RT has been carried out focusing on the late wave (Sanquist et al., 1981; Brunia, 1988; Van Boxtel and Brunia, 1994), potential modulations of the early wave was of special interest, due to its suggested association with arousal (Tecce, 1972; Tecce and Cattanaach, 1993).

A total of 50 female right-handed participants aged 19–44 years (mean 24.26, S.D. ± 5.07) took part in the experiment. They were requested to refrain from drinking coffee or alcohol, smoking and eating salty food for at least 3 h prior to the beginning of the experiment. A blood pressure screening was taken at least 1 week before the actual experiment to exclude hypertensive individuals with a blood pressure (BP) above 140/95 mmHg and to check the reproducibility of the measurements, which were accomplished by a sphygmomanometer. During the main experiment, the participants were subjected to four blood pressure measurements: (1) baseline, (2) between the two CNV recordings, (3) after the end of the second CNV recording and (4) after completing the ACE battery. Based on the averaged SBP of the four measurements, the sample was divided via median-split into a hypotensive (mean 101.77 mmHg, S.D. ± 5.63) and a normotensive group (mean 119.78 mmHg, S.D. ± 6.12) of 25 participants each. Retest-reliability between the systolic BP at screening and the first measurement of the actual experiment was $r=0.88$.

EEG was recorded in a sound-attenuated room using Syn-Amps DC amplifiers controlled by NEUROSCAN software (version 3.0; 1993). A notch filter was used to eliminate 50-Hz interference. Corresponding to Costa et al. (1998), the vertex (Cz) was selected as the recording site for CNV activity. Vertical and horizontal eye movements and eye blinks were checked by placing an electrode above the left orbit and beside the left outer canthus. Electrodes (Ag/AgCl) were filled with Elefix paste. Impedance values were below 5 k Ω . EEG and eye movement electrodes were refer-

enced to linked mastoids. Sampling epochs started 200 ms before S1 and ended 5500 ms after the onset of S1. Data were recorded with a sampling rate of 1 kHz and digitally filtered online with a 0–70-Hz band-pass. All data were saved on hard disk for offline analysis and data reduction.

As a CNV paradigm, we chose a constant fore-period reaction-time task, using pure tones as S1 (1000 Hz) and S2 (800 Hz, 70 dB). Two conditions consisting of 40 trials each were defined: they differed by the intensity of S1, which was either 60 or 80 dB. The duration of all tones was 100 ms. All participants underwent both conditions, which were presented in a counterbalanced order. The interstimulus interval was 4 s, which permits the differentiation of an early (250–1000 ms) and a late (3250–4000 ms) component. We also calculated an average CNV (250–4000 ms). Reaction times following S2 were recorded using a Hewlett Packard 5300A time-counter (accuracy in ms).

The Attentional and Cognitive Efficiency (ACE) battery developed by Mialet and colleagues (Mialet et al., 1995, 1996; Pope et al., 1997) is a set of five computerised tests of attentional and cognitive performance, from which three subtests were chosen: a test of attentional flexibility (AZE test), of visual sustained attention (Letter-Grid test) and of visuospatial memory (Checker-Board test). Since the AZE test alone leads to significant differences, only this task is described here. One of the three letters 'A', 'Z' or 'E' is presented on the computer screen. The participant has to respond to the presentation of one of these letters by pressing the corresponding key, which was one of the three arrow-keys at the bottom right of the keyboard. The rationale of the test is to induce a sensomotor routine in the participant by frequently presenting 'A' (left side of the screen; response: ' \leftarrow ' key) and 'Z' (right side of the screen; response: ' \downarrow ' key) in an alternating manner. In only 10% of the presentations, and at random, the letter 'E' (response: ' \rightarrow ' key) appears at either side of the screen. In these cases, an interruption of the automatic response is required in favour of a controlled response. Dependent variables are: (1) speed, measured as total number of correct responses; (2) accuracy, measured as total number

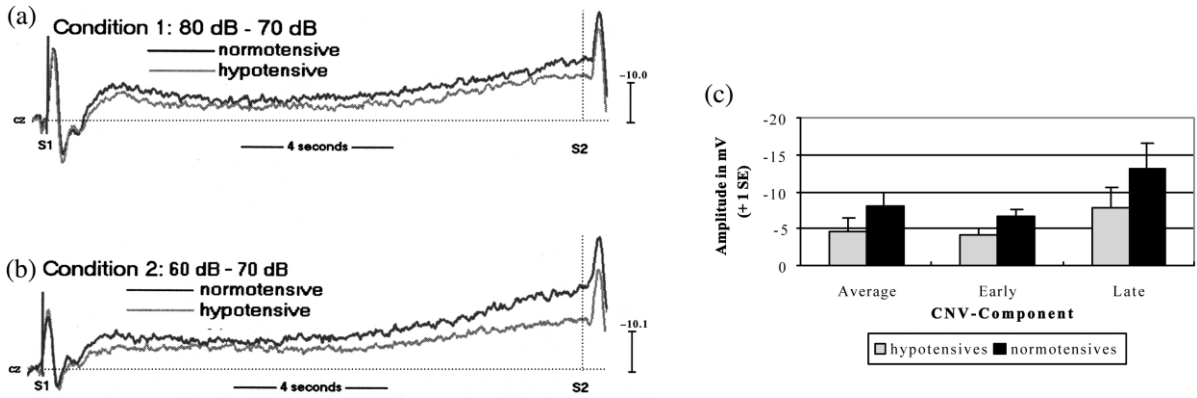


Fig. 1. Grand averages for both groups on the two conditions (a) S1=80 and (b) S1=60, S2=70 dB. (c) Mean CNV amplitudes (μV ; \pm S.E.) for both groups averaged across both conditions. Note that negativity is depicted upwards.

of incorrect responses; and (3) attentional flexibility (E/A), measured as RT('E') divided by RT('A'). This E/A fraction normally lies above 1, reflecting the effort in switching from automatic to higher-level controlled processing.

Concerning electrophysiological data analysis, 40 epochs were averaged for each condition. The algorithm of Semlitsch et al. (1986) was used for the correction of trials containing eye movements. Trials contaminated by eye blinks or excessive eye movements ($>100 \mu\text{V}$) were excluded from data analysis. Participants with less than six (Tecece, 1972; Tecece and Cattanaach, 1993) usable trials in one or both conditions were not included in the mixed ANOVA. This was the case for eight participants. One participant was identified as an outlier (values exceeding the 99% confidence interval of the linear regression) and two had to be excluded

due to technical problems during the EEG recording. As dependent variables, the total average (250–4000 ms after onset of S1), the early (250–1000 ms) and the late CNV (3250–4000 ms) were computed for the remaining 22 hypotensive and 17 normotensive subjects and entered into a 2×2 mixed ANOVA group with between-subject factors (hypotensive vs. normotensive) and repeated measurements on condition (60 vs. 80 dB).

Concerning the RT, one participant was excluded as an outlier, thereby leaving 25 hypotensive and 24 normotensive subjects for a same 2×2 mixed ANOVA as for CNV. Data obtained with the ACE battery were analysed using ANOVA with the between-subject factor group for the AZE test. Because the factor group contains only two levels and a priori hypotheses about the direction of differences existed, the given probability (P) values for the factor group are one-tailed.

No main effect was obtained for the condition or condition \times group interaction (all $F < 1$) for the CNV. In accordance with our hypothesis, hypotensive subjects expressed less negativity on all three CNV parameters. The group difference was significant for the early component ($F_{1,37} = 4.07, P < 0.03$), and insignificant for the average ($F_{1,37} = 1.92, P < 0.09$) and the late CNV ($F_{1,37} = 1.51, P < 0.11$). Fig. 1 depicts the waveform of the CNV for both conditions and a histogram for the components averaged over both conditions. A signifi-

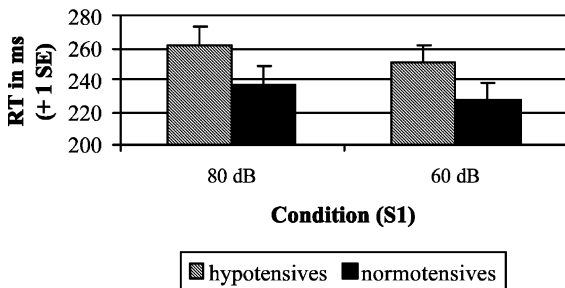


Fig. 2. Mean reaction time (RT) in ms (\pm S.E.) for both groups for the two conditions.

cant correlation was observed between the systolic BP and the early CNV ($r=0.37$, $P<0.01$).

Fig. 2 illustrates the mean reaction times for the two groups for both conditions. The interaction between group and condition did not reach significance. On average, hypotensive subjects were significantly slower than normotensive subjects (257 vs. 232 ms, $F_{1,47}=2.83$; $P<0.05$). With regard to condition, the mean reaction time was approximately 10 ms faster for the 60 dB condition (239 vs. 249 ms), although this difference was not significant ($F_{1,47}=2.85$, $P<0.10$).

Hypotensive subjects required significantly longer to switch from an automatic to a controlled response, as indexed by the mean scores for attentional flexibility (E/A=1.54 vs. 1.40; $F_{1,47}=7.18$; $P<0.005$) in the AZE test.

Overall, similar to Costa et al. (1998) and Stegagno et al. (1996), our results suggest that hypotension is associated with a reduction in some aspects of cognitive and psychomotor performance. Since the differences on CNV and RT between the two groups were similar, independent of stimulus intensity, we conclude that arousal is not an adequate explanation for our results.

The finding of lowered CNV in chronic hypotensive subjects in the study by Costa et al. (1998) and the present one is especially interesting for the following reason. It has to be assumed that *phasically* induced lowering of blood pressure leads to baroreceptor inhibition, which is, in turn, related to enhanced cortical activation that can be observed *in more negative* CNV amplitudes (Elbert et al., 1992; Rau et al., 1993). This suggests that results on phasically lowered blood pressure cannot be generalised to chronic low blood pressure, or vice versa. Furthermore, the results of phasically induced hypotension do not offer a straightforward answer to the question concerning the underlying physiological mechanisms relating chronic hypotension with cortical activation. Similar to the theory of Dworkin et al. (1979), who postulated that chronic hypertension can be instrumentally learned by reducing cortical sensitivity to stressful stimuli, a physiological explanatory approach for hypotension may also include the baroreceptors. A more detailed account of this idea is given below.

The cardiovascular activity is capable of influencing central nervous activity via afferent projections (for reviews see Vaitl and Gruppe, 1991; Lacey, 1992; Vaitl and Schandry, 1995). Considering the findings of Lacey and Lacey (1980) and Lacey (1992), cardiac deceleration facilitates sensorimotor and attentional performance by increasing cortical activation, and vice versa for cardiac acceleration. A close relationship between cardiac deceleration, on the one hand, and early and late negativity in the CNV, on the other, was demonstrated in several studies (for a review see Rockstroh and Elbert, 1990). It is assumed that the baroreceptors, which are mainly located in the carotid sinus and aortic arch and are normally engaged in the regulation of arterial blood pressure, play a major role in mediating cardiovascular influences on brain activity (e.g. Rau et al., 1988, 1993). Thus, by an increase and decrease in cardiovascular activity (cardiac activity and blood pressure), baroreceptors are excited or inhibited, and in this way facilitate performance in different tasks by altering cortical activity. It could be hypothesised that hypotensive subjects have a reduced 'ability' in adopting cardiovascular activity to situational demands, therefore being less able to benefit from its modulating cortical effect.

This notion can only be integrated into the framework of the work of Lacey and Lacey—whose work mostly concerned phasic cardiovascular changes—by postulating altered response characteristics of baroreceptors in hypotensive subjects. Baroreceptor activity is mainly influenced (a) by its threshold, which determines at which value blood pressure is stabilised, and (b) the sensitivity or slope, indicating the amount of activity induced by a certain change in blood pressure (stimulus–response curve) (Dembowsky and Seller, 1995). In these two aspects, hypertensive differ from normotensive subjects by having elevated firing thresholds (stabilising blood pressure at higher values) and hyposensitivity or reduced slope (Chapleau et al., 1989; Koushanpour, 1991). The hyposensitivity of baroreceptors is—according to Sleight (1991)—a risk factor for myocardial infarction due to reduced regulative processes, which are reflected in a greater variability of blood pressure in hypertensive subjects. Concerning

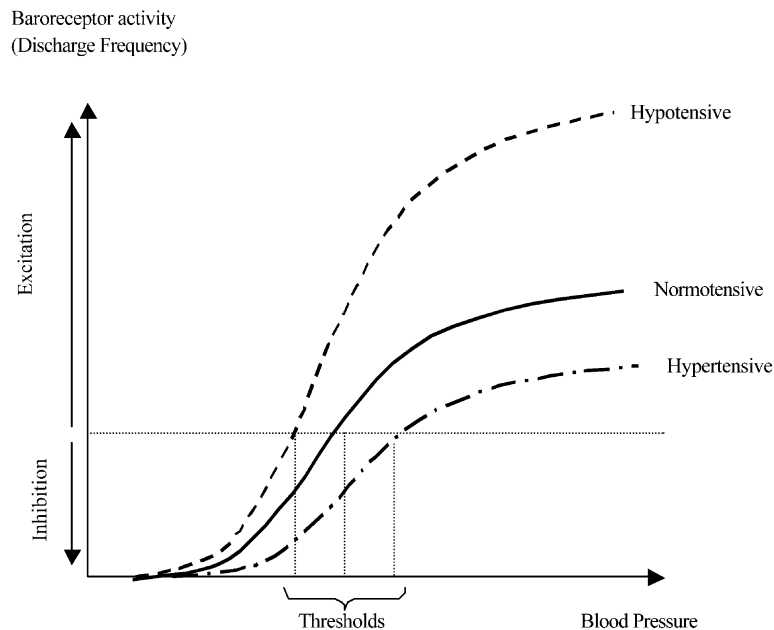


Fig. 3. *Hypothetical* blood pressure–baroreceptor activation curve for hypotensive subjects. Baroreceptor activity is excited above firing thresholds and inhibited below firing thresholds. Note the different slopes proposed for the three chronic blood-pressure levels.

hypotension, it is known that the threshold for baroreceptor activity is reduced, thus stabilising blood pressure at a lower value (Dembowsky and Seller, 1995), but to the best of our knowledge, sensitivity has not yet been a matter of investigation. We could therefore hypothesise that baroreceptors might be hypersensitive to changes in blood pressure in hypotensive individuals (steeper stimulus–response curve). Fig. 3 illustrates the hypothesised stimulus–response curve for hypotensive subjects. This hypersensitivity would keep blood pressure within stricter limits, thus making it more difficult for hypotensive individuals to modulate cardiovascular activity. Changes in blood pressure via cardiac deceleration or acceleration would be detected and regulated to normal values in a faster and more effective manner than for normo- and hypertensive individuals. At the performance level, this notion could be combined with the hypothesis proposed by Lacey and Lacey, which implies that a reduced cardiovascular variability is to be negatively judged due to decreased capability of modifying cortical excitability. At this point it is interesting to consider the findings

on attentional flexibility and early CNV in one context. In both cases, the prefrontal cortex is implicated to play an eminently important role (for early CNV see Birbaumer et al., 1990; Tecce and Cattanach, 1993; Rosahl and Knight, 1995; for a review on attention see Cohen and O'Donnell, 1993). It is well established that the cardiovascular system possesses afferents to the prefrontal cortex (see e.g. Montoya et al., 1993). Therefore, it seems reasonable to speculate that cardiovascular input may modulate higher-order prefrontal functions. Hypersensitivity of baroreceptors could be responsible for a diminishment of these modulatory—and normally supportive—cardiovascular effects. This hypothesis would predict that hypotensive individuals should perform worse in situations in which the modulation of cardiovascular activity is of an advantage, and that differences should be mostly pronounced in situations requiring attentional flexibility.

Overall, the question of causality remains a problem in this field of research and, of course, there are other theoretical approaches to this issue (e.g. reduced cerebral blood flow in hypotension).

However, regardless of the mechanisms involved in hypotension, this study demonstrated that there is empirical evidence of reduced cognitive performance in hypotension, along with a decreased early wave of the CNV. It is conceivable that at least parts of the hypotensive population remain below their optimum cognitive possibilities.

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