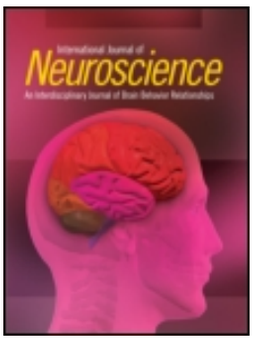

International Journal of Neuroscience

ISSN: 0020-7454 (Print) 1543-5245 (Online) Journal homepage: http://www.tandfonline.com/loi/ines20

Enhancement of heartbeat-related brain potentials through cardiac awareness training

Rainer Schandry & Rolf Weitkunat

To cite this article: Rainer Schandry & Rolf Weitkunat (1990) Enhancement of heartbeat-related brain potentials through cardiac awareness training, International Journal of Neuroscience, 53:2-4, 243-253, DOI: 10.3109/00207459008986611

To link to this article: https://doi.org/10.3109/00207459008986611

Published online: 07 Jul 2009.

Intern. J. Neuroscience, 1990, Vol. 53, pp. 243–253
Reprints available directly from the publisher
Photocopying permitted by license only

ENHANCEMENT OF HEARTBEAT-RELATED BRAIN POTENTIALS THROUGH CARDIAC AWARENESS TRAINING

RAINER SCHANDRY and ROLF WEITKUNAT

Institut für Psychologie, Ludwig-Maximilians-Universität, München, FRG

(Received March 20, 1990)

Heartbeat synchronous potentials were found to be sensitive to differences in perceptual acccuracy. Since heartbeat perception can be improved using appropriate training procedures, increased ability to perceive cardiac performance should also influence heartbeat-related potentials. In the present study, subjects in either of two groups had to press a button immediately after the occurrence of a heartbeat-feedback tone. Whereas the first group was given acoustical heartbeat-feedback throughout the entire training, the second group was provided with tone signals that became fainter during the course of the training phases. The better posttraining performance in heartbeat perception of the group receiving full-intensity feedback was also reflected in the evoked potentials. They differed markedly before and after training, especially between 250 to 400 ms (after the EKG-R-wave), the biggest effect being at Fz and F7. The findings are interpreted as the brain electrical reflection of an increased perception susceptibility to a cadiovascular signal occurring at about 200 ms after the R-wave.

Keywords: event-related potentials, viscероception, heartbeat perception, training, perceptual learning, biofeedback

An important new development during the last five years has been the application of event-related potential (ERP) research and its methodology to potentials evoked by stimuli, occurring *within* the body, especially in the *autonomic* system. Badr, Carlsson, Fall, Lindström, and Ohlsson (1982) were able to demonstrate in human subjects that electrical stimulation of the inner bladder wall results in typical evoked brain potentials primarily at central locations. Peak latencies were between 50 and 100 ms and amplitudes ranged from 10 to 20 μV. In a subsequent study, Badr et al. (1984) demonstrated that stimulation of the bladder evokes a waveform composed of four components in the latency range between 45 and 100 ms. A similar study (Haldeman, Bradley, Bhatia & Johnson, 1982), investigating the urogenital tract (N. pudendus), revealed evoked potentials consisting of two to three components. Once again, activity was mainly observed above the central brain areas, with latencies varying between 50 and 200 ms, and amplitudes ranging between 1 to 3 μV. Sarica, Karacan, Thornby and Hirshkowitz (1986) reported evoked potentials due to electrical stimulation of urogenital tissue (vesico-urethral junction) in the latency range from 50 to 200 ms, with peak amplitudes between 0.5 and 4 μV. Here, electrodes were located at central sites. As a result of these and other studies, a new approach to the investigation of viscero-cortical interaction is becoming established.

Nonetheless, we chose a different approach in investigating brain electrical responses to visceral events. Instead of applying nonbiological stimuli (such as external

This research was supported by the Deutsche Forschungsgemeinschaft. We gratefully acknowledge the assistance of Bernd Sparrer in the data analysis.

Address requests for reprints to Prof. Dr. Rainer Schandry, Institut für Psychologie, Universität München, Leopoldstr. 13, D-8000 München 40.

electrical voltages) to visceral tissue, signals generated *by* an organ (i.e., viscerogenic stimuli) were explored. Schandry, Sparrer and Weitkunat (1986a) were the first to report a brainwave evoked by the *heartbeat*. In sampling EEG epochs time-locked to the EKG-R-wave, a negative waveform in the latency range of 250 to 400 ms post-R-wave was observed that was most prominent at the frontal electrode and exhibited amplitudes ranging from 1 to 2 μV. This heartbeat evoked potential (HEP) proved to be affected by the subject's ability to detect heartbeats and by the subject's focus of attention. When attention was directed toward heartbeats, the wave was more prominent than when attention was directed toward external signals. In a subsequent study (Schandry, Sparrer & Weitkunat, 1986b), the effects of heartbeat discrimination ability and focus of attention were confirmed. The influence of EKG artifacts on the effects could be ruled out in both studies since no comparable effects were observed at EKG leads.

A similar line of investigation was pursued by Jones and coworkers (Jones, Leonberger, Rouse, Caldwell & Jones, 1986 and Jones, Rouse & Jones, 1988). In two preliminary papers, the authors reported the occurrence of cortical activity at central electrodes which is triggered by the heartbeat. On the basis of concomitant recording and analyzing EKG and EOG, the authors were able to rule out the possibility of relevant influences of extracortical sources on the observed scalp potentials. In both experiments, the HEPs of good and poor heartbeat perceivers showed amplitude differences.

The primary aim of the present study was to investigate whether an *improvement* in cardiac perception has an influence on HEP. The heartbeat is a bodily signal that is (under resting conditions) only weakly perceived or undetectable by most individuals (cf. Katkin, 1985; Jones, Jones, Cunningham & Caldwell, 1985). In fact, the heartbeat is somewhat comparable to a subthreshold or near-threshold external signal. With respect to evoked potentials on near-threshold stimuli, it has been amply demonstrated that the response to detected signals is with higher amplitudes (especially the P300 component) than the response to undetected events (Hillyard, Squires, Bauer & Lindsay, 1971; Squires, Squires & Hillyard, 1975; Wilkinson & Seales, 1978; Ruchkin, Sutton, Kietzman & Silver, 1980). Thus, we postulate that an increase in cardiac perception (e.g., by means of an appropriate training procedure) will be reflected in heightened HEP amplitudes.

Training procedures designed to increase cardiac awareness have been employed by several authors (e.g., Katkin, Blascovich & Goldband, 1981; Grigg & Ashton, 1982; Davis, Langer, Sutterer, Gelling & Marlin, 1986), generally resulting in improved accuracy of heartbeat perception: An increase in hit rate from a chance level to as much as 70% or 80% has frequently been observed. Training usually encompasses the repeated performance of a heartbeat perception task, accompanied by performance feedback or knowledge of results (upon completion of each trial). However, we see a drawback in these procedures since the extent to which the results can be interpreted as a sign of increased *sensitivity* for the heartbeat signal as opposed to greater *familiarity* with the testing procedure seems questionable. The latter interpretation is supported by observations related to the detection of *external* auditory signals (above auditory threshold). Here discrimination scores as measured by Katkin's procedure (Katkin et al., 1981) were dependent to a large extent on subjects' familiarity with the testing procedure (Weitkunat, 1987).

We, therefore, employed training procedures that provided immediate feedback on the occurrence of heartbeats. Subjects had to respond to each feedback tone by pressing a button. During the first procedure, heartbeat-feedback was always given, with intensity of feedback stimuli held constant. In the second procedure, however,

the intensity of feedback tone was gradually diminished to zero during training. Thus, the first approach was characterized by maximal external information on the internal process. The second procedure was designed to compel subjects gradually to switch to the use of *interoceptive* information in order to respond correctly to diminishing "feedback" signals. This procedure was adopted from Hefferline's (Hefferline and Perera, 1963) research on conditioning responses to interoceptive stimuli.

The aim of the present study was, thus, twofold: First, the effectiveness of two different training methods (both based on more *direct* heartbeat sensitization procedures, as compared to Hefferline and Perera's method, where subjects were not informed about the operant) in improving heartbeat perception was tested. Second, we investigated whether differential effects of the two training procedures were reflected in HEPs. (Results pertaining to the differential effects of the two training procedures on behavioral measures are described in greater detail in Weitkunat, Schandry, Sparrer & Beck, 1987).

METHODS

Subjects

Forty-two subjects (students and employees of the University of Munich), aged 18–40 years (mean: 25.03), were paid 40 DM for their participation. All subjects (21 male, 21 female) were right-handed and free of any present or former cardiovascular or brain disease.

Experimental Design and Procedure

Subjects were randomly assigned to one of two training groups (22 to "fading-tone" group, 20 to "constant-tone" group, with equal numbers of males and females in both groups). With the exception of the training procedures, the course of the experiment was identical for both groups. Subjects sat in a reclining chair with their eyes closed during physiological recordings. Experimental sessions consisted of a rest period, a series of heartbeat perception tests, and heartbeat perception training.

Rest During the first part of the experiment, subjects were instructed to sit quietly and relax in order to achieve a resting baseline. As in all other recording phases, physiological activity was recorded until 200 heartbeats had occurred. Beginning and ending of the recording phases were signaled by a single or double tone, respectively.

Initial assessment of cardiac awareness A heartbeat perception testing procedure, based on counting heartbeats (Schandry, 1981), was conducted. This condition was introduced in order to collect additional normative data on performance in the cardiac awareness test. It has no direct relation to the topic of the present study, nor was EEG recorded.

Attention-1 To control for the possibility that simply attending to heartbeats over a certain length of time (as is required during the ERP-recording conditions and during training) could exert an influence on performance in subsequent heartbeat perception tasks, a preliminary attention period was conducted before the first heartbeat perception test. Subjects were instructed to attend to their cardiac activity and to count heartbeats silently. EEG and EKG were recorded. Again, recording was done while 200 heartbeats occurred.

Heartbeat perception test 1 The heartbeat perception test as described by Hantas, Katkin and Reed (1984) was employed in a slightly modified version. Subjects were administered 100 tone series, consisting of ten tones (50 ms, 2000 Hz) each. The latency between heartbeat and tone was either fixed (50 series) for all tones within one series or variable (50 series). The two different types of series occurred in a semirandom order. In the case of fixed latency, tones occurred 130 ms after the R-wave; during variable latency, tones occurred with a delay of $N + 30i$ ms after the R-wave. N denotes a random number between 0 and 200, and i is the number of the tone in a series of ten tones. Thus, variable latencies varied between 30 ms ($i = 1$ and $N = 0$) and 500 ms ($i = 10$ and $N = 200$) after the R-wave. At the termination of each series, subjects had to denote whether a fixed latency or a variable latency series was given. A discrimination score $P(\bar{A})$ was computed from subject's responses and normalized by computing $2\arcsin \sqrt{P(\bar{A})}$ (see McNicol, 1972).

Attention-2 This attention period was conducted in the same manner as Attention-1.

Heartbeat perception training The training procedure consisted of nine identical sections, with three phases each. During phase 1 (1 min) of every section, subjects heard heartbeat feedback tones (100 ms, 2000 Hz) 130 ms after each EKG-R-wave. Phase 2 (2 min) was different for the two experimental groups: For the "fading-tone" group, heartbeat feedback tones were faded out logarithmically to the individual hearing threshold by controlling the tone-line via the computer's D/A interface. The "constant-tone" group heard tones at full intensity during these phases. During phase 3 (1 min), no acoustic heartbeat feedback was administered. All subjects were instructed to press a switch immediately after the occurrence of a heartbeat (signaled or not signaled by the feedback tone) in all phases. They were told that "correct" responses would be followed by a different tone. That is, responses which followed the EKG-R-wave within 100 ms to 500 ms ("correct" responses) were "rewarded" with a second acoustic feedback signal (100 ms, 1000 Hz). By applying this response-time window, button presses without any direct relationship to the events of the preceding heartbeat went unrewarded. Between sections, pauses of 30 s each were inserted.

Attention-3 This period was identical to Attention-2.

Heartbeat perception test 2 This test was conducted in the same way as heartbeat perception test 1. Changes in performance due to training were measured by comparison of heartbeat perception tests 1 and 2.

Recording Procedures

EEG was derived from Fz, Cz, F7, and F8 according to the international 10–20 system (Jasper, 1958) and referred to linked mastoids. An additional electrode was placed at the tip of the nose in order to obtain a reference signal for the reduction of EKG artifacts from evoked responses (see below). Since no EOG-contamination of HEPs was to be expected, no EOG-registration was undertaken.[1] Before Ag/AgCl

[1] In an independent study, the assumption of heartbeat synchronous eye movements was tested. In addition to EEG at Fz, Cz, and EKG, horizontal and vertical EOG was recorded using 5 subjects. While a first resting condition was comparable to the rest period of the present study, subjects were instructed in a second condition to perform ballistic eye movements along with occasional eye-blinks in a second "worst case" condition. All heartbeat synchronous averaged EOG-signals of both conditions were well below noise level and no EOG-contamination was observed in the HEPs.

electrodes were positioned and affixed with collodion, the skin was cleaned with 90% isopropyl alcohol. Electrode impedances were kept below 5 kΩ. A common ground electrode for EEG and EKG was placed on the frontal left foot angle joint. EKG was recorded from right midclavicula against the 7th left rib. EEG-signals were preamplified (gain: 1000, low frequency rolloff: 0.03 Hz, high frequency rolloff: 30 Hz) by Princeton Applied Research 113 preamplifiers. The EKG-signal was amplified on a Beckman R-411 Dynograph recorder and filtered in the same manner as the EEG.

Stimulus presentation and data acquisition were governed by a PDP 11/34 computer. Digitizing of physiological signals was done at 200 Hz. "Triggers" were generated by a hardware peak detector at the occurrence of the EKG-R-wave (ascending limb). Sweep length was 800 ms, including a 100 ms pretrigger period.

Data Analysis

Sampling epochs were averaged across subject separately for each phase and location. To remove 50 Hz line-frequency interference, a 4-point moving average filter was used. This filter smoothes the 50 Hz curve effectively, since the length of the filter-period (20 ms) equals exactly one line frequency period. Averages (consisting of 200 sweeps each) were baseline-corrected by subtracting the mean of the first 15 samples (75 ms) of the pretrigger epoch from the entire average.

Reduction of EKG artifact was accomplished in the following manner: The EKG-influence could theoretically be eliminated by subtracting from the EEG sweep a cranial waveform caused solely by cardiac potentials. Estimation of this artifact portion of scalp potential was accomplished by recording EKG from a relatively EEG-free head electrode, located at the tip of the nose. Since EKG-influence can not be expected to be identical at all cranial recording sites (due, for example, to different electrode impedances), the two signals to be subtracted from each other had to be adjusted with regard to their amplitudes. Amplitude fitting was based on the relations of the amplitudes in the latency range of the EKG-R-wave (25 ms before to 50 ms after the QR-slope). Since no evoked EEG-activity is to be expected as early as the latency range of the EKG-R-wave, average waveform components within this time period are considered to be entirely due to EKG-activity. For amplitude fitting, a factor α was computed so that the difference of the amplitude sums around the R-wave would be minimized:

$$\sum_{i=R-25ms}^{R+50ms} (Y_i - \alpha Z_i)^2 = \min.$$

Here, Z represents the voltage at the tip of the nose. Y denotes the voltage derived from Fz, Cz, F7, and F8 (contaminated by EKG-artifact). The corrected EEG-signal Y' for the total epoch is then the difference between the raw evoked potential and the weighted nose-signal:

$$Y' = Y - \alpha Z \qquad \text{(for all sampling points } i\text{)}$$

RESULTS

Effects of Heartbeat Perception Training

A 2 × 2 (group × period, i.e., constant-tone/fading-tone by pretraining/posttrain-

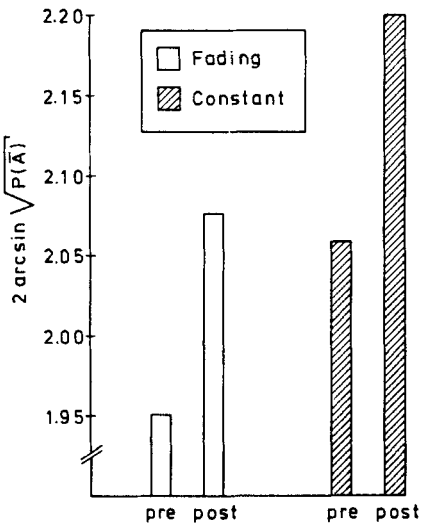

FIGURE 1 Heartbeat discrimination scores in "fading-tone" group and "constant-tone" group before and after training

ing) repeated measures ANOVA was computed on heartbeat discrimination scores. A significant main effect was revealed for pre-post [$F(1/40) = 5.02, p < .05$], but the difference between groups did not reach significance. The changes in group means are shown in Figure 1. The performance level of the "constant-tone" group obviously exceeded that of the "fading-tone" group.

Event-Related Potentials

Figure 2 presents average ERP and EKG waveforms during Attention-2 (pretraining) and Attention-3 (posttraining) periods for the "fading-tone group" and the "constant-tone" group respectively. These uncorrected HEP waveforms clearly show the

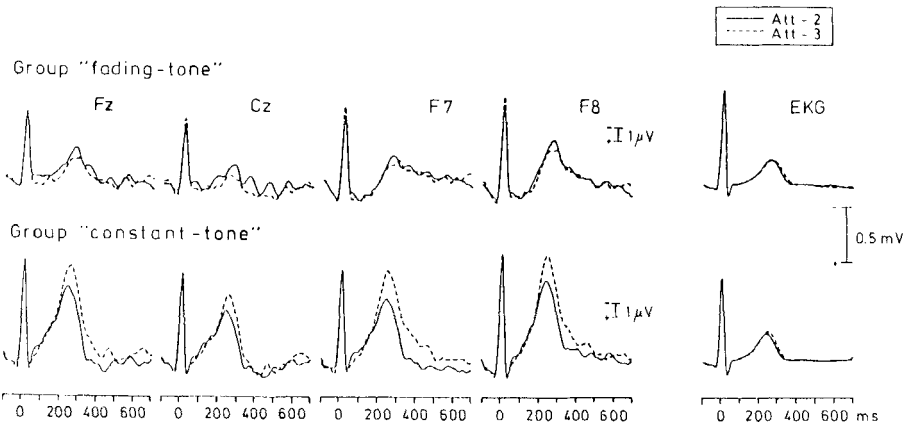

FIGURE 2 Average HEP and EKG waveforms for "fading-tone" group and "constant-tone" group in periods Attention-2 and Attention-3

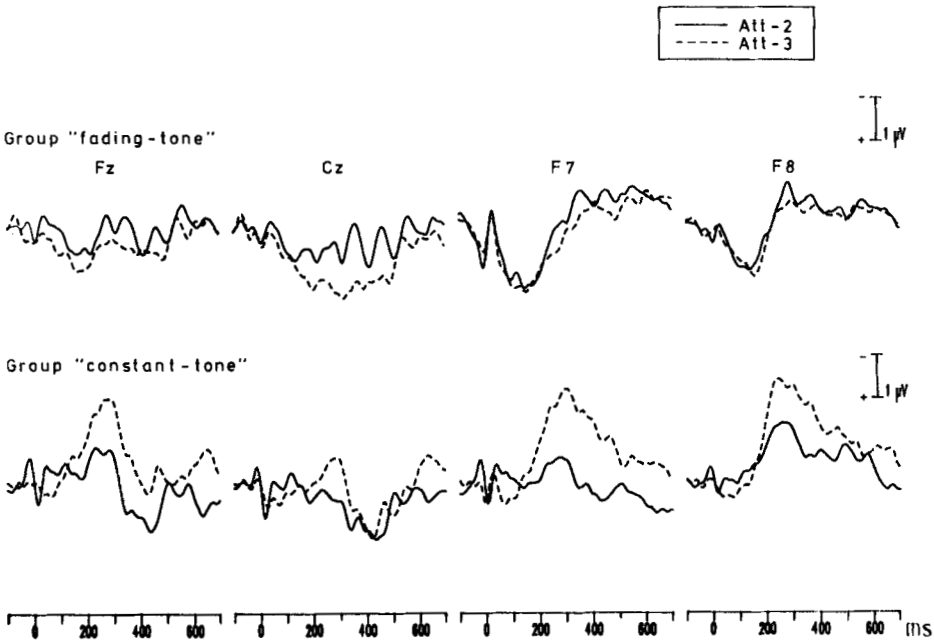

FIGURE 3 Corrected average HEP waveforms for "fading-tone" group and "constant-tone" group in periods Attention-2 and Attention-3

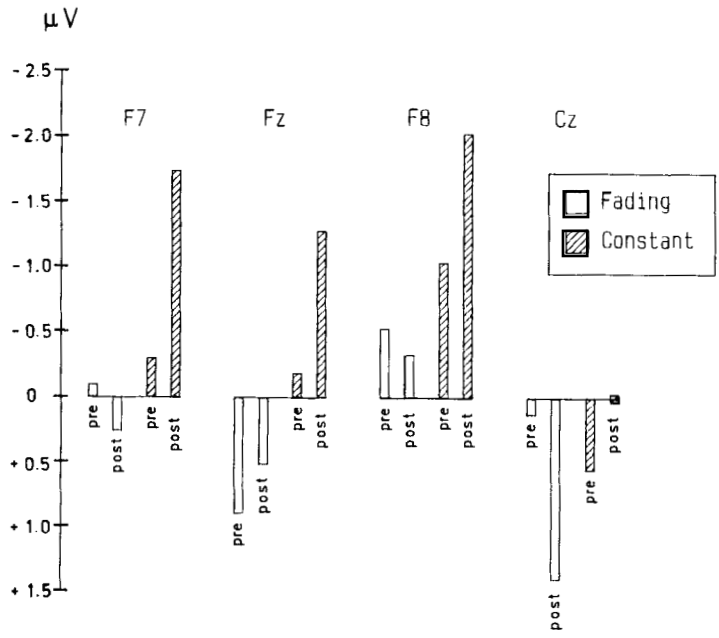

FIGURE 4 Mean HEP amplitudes in the latency range of 200 to 350 ms, for "fading-tone" group and "constant-tone" group at the four locations. Each pair of bars represents the pre- and posttraining means

influence of EKG-artifact for both groups. In both periods and at all locations, a sharp negative maximum occurs in the latency range of the EKG-R-wave. Around the EKG-T-wave latency a broad negative deflection is visible. Here, it is important to note that the standard EKG-T-wave amplitude is usually only about one third of the R-wave amplitude. However, in the EEG in the corresponding latency range, we find electrical activity that is more than half the amplitude of the peak in the R-wave region. (Already in the pretraining phase, a remarkable difference exists between the Grand Averages of the "fading-tone" and the "constant-tone" group potentials in the latency range 250 to 400 ms. Since the T-wave amplitude is also slightly higher in the EKG for the "constant-tone" group and, in addition, this difference in scalp potentials disappears almost completely after EKG-artifact correction [see below], this is attributed to a-priori group differences in electrocardiac potentials.)

Heart synchronous evoked potentials for the "fading-tone" group showed no marked amplitude alterations from pre- to posttraining. For the "constant-tone" group the ERPs showed enhanced activity in the latency range of 250 to 400 ms at all locations. This effect was not present in EKG waveforms.

EKG-artifact corrected average waveforms for both groups from all locations and attention periods (Attention-2 and Attention-3) are presented in Figure 3.

Once again, a strong negative amplitude increment is visible for the "constant-tone" group in Attention-3, as compared to Attention-2. This effect is most pronounced in the latency range from 200 to 450 ms and is not observable for the "fading-tone" group. The peak is most profound at locations Fz and F7, with a maximum located between 280 to 300 ms after the R-wave (which is considerably later than the peak of the EKG-T-wave).

In order to analyze this component further, the mean amplitudes between 200 and 350 ms were computed for each subject, location, and period. The group means are shown in Figure 4. Mean amplitudes were analyzed separately for each electrode location using 2 × 2 ANOVAs with the factor "group" and the repeated measures factor "pre-post training." Whereas no significant main effects were obtained, interactions between "group" and "pre-post" were significant for all locations, with the exception of F8; Fz: $F(1/30) = 8.44$, $p < .01$, Cz: $F(1/30) = 8.08$, $p < .01$, F7; $F(1/30) = 6.79$, $p < .05$. A similar analysis was conducted for EKG waveforms; no significant effects were observed.

These statistical results, in correspondence with visual inspection of Figures 2 and 3, reveal that only "constant-tone" group waveforms showed an increment of ERP-amplitude in the latency range of interest. Further evidence is provided by the results of paired t-test between Attention-2 and Attention-3 mean amplitudes, computed separately for each location and group. The increase in amplitude after training was significant at Fz [$t(14) = -2.71$, $p < .05$] and F7 [$t(14) = -2.37$, $p < .05$] for the "constant-tone" group, whereas no such effect was observed for the "fading-tone" group.

DISCUSSION

Consistent with our hypothesis, the differential effect of cardiac awareness training on (behavioral measures of) cardiac perception is reflected in the heartbeat evoked potential (HEP). A negative waveform within the time range from 250 to 400 ms post-R-wave, with an amplitude between 1 to 2 μV, is enhanced by the effects of increased detection performance for the heartbeat signal. The results of statistical analyses for the two levels of dependent measurement were, however, not parallel.

Whereas, in the cardiac discimination measure, we observed a main effect of the two training procedures with respect to an overall increase in detection performance (differential group effects were reported in Weitkunat et al., 1987), the analysis of the HEP measure resulted only in a significant *interaction* effect between treatments (fading- vs. constant-tone) and period (pre- vs. posttraining). As Figure 1 shows, the discrimination score increased for both groups, but reached a much lower level for the "fading-tone" group (i.e., to about the pre-training level of the "constant-tone" group).[2] In the ERP-amplitudes, the effect reached significance only for the "constant-tone" group. Thus, only partial agreement between behavioral and electrophysiological effects at the level of testing for significance was observed. We attribute this, at least partially, to the different effects, exerted by physical as well as mental intervening variables on cortical and peripheral responses. Moreover, in the case of evoked potentials to near-threshold stimuli, only small and broad waveforms are to be expected (cf. Hillyard, Squires, Bauer & Lindsey, 1971), thus resulting in a generally lower signal-to-noise ratio.

The latency of the observed waveform follows the R-wave within 250 to 400 ms. The R-wave is, however, in our opinion, *not* the eliciting event for this waveform. It has been postulated that *mechanical* events due to the propagation of blood generate the perceivable heartbeat signal (Whitehead et al., 1977; Jones et al., 1987; Schandry & Specht, 1981; Katkin, 1985). Speculatively, we assume that cardiac sensations arise as a consequence of the transfer of mechanical energy during the ejection period. An energy transer to pressosensitive tissue in the heart and in the inner chest wall may partially be explained as a rebound process during rapid acceleration of the blood volume by ventricular ejection. In addition, transfer of mechanical momentum to pressosensitive tissue may occur during deceleration of the blood upon reaching the smaller vessels. (These processes are responsible for the generation of the ballistocardiography-signal, which can be used to estimate stroke volume.) The relevance of such mechanical events in the vicinity of the heart for cardiac awareness is supported by first results of a study utilizing impedance cardiography to measure cardiodynamic parameters (Bestler, Weitkunat, Keller & Schandry, 1988).

If we assume that the heartbeat signal is generated by the rapid ejection of blood volume into the aorta and the larger vessels in the vicinity of the heart, its intensity

[2]The numerical values of the mean heartbeat discrimination scores obtained with the procedure described by Hantas et al. are relatively high, as compared to scores usually observed with the Katkin test. As Katkin, Reed and DeRoo (1983) have pointed out, the procedure employed by Hantas et al. allows misclassifications of poor heartbeat perceivers as good perceivers. This happens when poor perceivers are able to discriminate tone series of fixed latency from those of variable latency solely upon the acoustic information. As the data published by Katkin et al. (1983) show, this misclassification may occur in 10 to 20% of the cases. We see the unexpectedly high mean scores in cardiac perception as a consequence of this effect. Since this experiment investigated brain electrical effects of cardiac perception *training*, one has to consider the possibility that our specific training procedure may artificially increase heartbeat perception scores. During training, subjects were provided with delayed acoustic heartbeat feedback. This may result in an increase of subjects' knowledge about the rhythm of their heartbeat. This in turn may be used as a cue to discriminate fixed latency series from variable latency series during heartbeat perception task 2 without an increased sensitivity to heartbeats. The possibility that the higher posttraining discrimination scores are solely due to such an effect can be ruled out on the basis of the finger tapping data during training phases 3. In each of the nine training sections (during phase 3) subjects had to track their heartbeats by finger taps solely on the basis of interoceptive cues. During these phases no external feedback was given. The intrasubject variability in the latencies between heartbeats (R-wave) and finger taps can be used as a behavioral measure of the accuracy of heartbeat perception. As has been reported in Weitkunat et al. (1987) these latencies decreased significantly for both groups during training, again with a better performance in the "constant-tone" group. This indicates that the training led in fact to a higher interoceptive sensitivity to heartbeats.

should be maximal around the pressure peak in the left ventricle and the aorta, i.e., between 180 and 220 ms post-R-wave. Given also that the heartbeat signal yields a brain electrical response similar to the response stemming from stimulation of somatosensory nerves of another visceral organ, namely the urinary bladder, a negative waveform is to be expected in the latency epoch between 100 and 150 ms (cf. Hillyard, Picton & Regan, 1978; Sarica et al., 1986; Badr et al., 1984; Haldeman et al., 1982). Consequently, if the response follows the peak of mechanical energy transfer during systole, it should be observable around 280 (= 180 + 100) to 370 (= 220 + 150) ms. Similarly, if we consider the heartbeat evoked response as comparable to a somatosensory evoked potential then a latency for the "N1" of 130 to 140 ms has to be assumed (e.g., Hillyard et al., 1978). In this case, a peak latency for the HEP of 310 (= 180 + 130) to 360 (= 220 + 140) ms is to be expected. Roughly speaking, the HEP peak should occur between 250 and 400 ms, which was the actual range for the registered HEP peak.

The amplitude of the HEP-component is rather small (1 to 2 μV). Yet, ERP-peaks elicited by (electrical) stimulation within the urogenital tract are also in the range of 1 to 2 μV, as reported by Sarica et al. (1986) and Haldeman et al. (1982). A comparable peak in the somatosensory evoked potential may be the N140, which also ranges only from 2 to 3 μV (Josiassen et al., 1982). Taking into account the comparatively slow rise-time of the assumed (mechanical) eliciting process, amplitudes exceeding a few microvolts are not to be expected.

In accordance with earlier observations (Schandry et al., 1986a), regarding the influences of cardiac awareness and focus of attention on HEP, the observed effects were most prominent at the frontal electrodes. Neuroanatomical knowledege about the cortical representation of visceroafferent input is still comparatively poor. It is assumed that primary projection areas are found, i.e., in Brodmann's areas 1 and 2 (Newman, 1974), in the posterior central gyrus, and in the vicinity of the projection areas of somesthetic input from the trunk. This latter, comparably small, area is connected to the peripheral receptors by rapidly conducting fibers. Another projection area for visceral input lies more frontally, in the premotor and orbital areas of the frontal lobe, where it is assumed that a more diffuse distribution of visceroafferent projections is located (Netter, 1987; Rohen, 1978). The visceroafferent fibers projecting into this area are presumably relayed in the reticular formation, the tegmentum, and the hypothalamus. Thus, longer conduction times are to be expected for brain electrical responses in this area. We tentatively interpret the more *frontal* distribution of the observed HEP as a reflection of these frontal visceroafferent projection areas.

Certainly, further work is necessary in this new area of ERP research. Not only the nature of the evoking event needs further elucidation, but also other important questions should be addressed, particularly with respect to the topography of visceroafferent brain activity and the relation of visceral perception to brain electrical correlates.

REFERENCES

Badr, G. G., Carlsson, C. A., Fall, M., Lindström, L. and Ohlsson, B. (1982). Cortical evoked potentials following stimulation of the urinary bladder in man. *Electroencephalography and Clinical Neurophysiology, 54*, 494–498.

Badr, G. G., Fall, M., Carlsson, C.-A., Lindström, L., Friberg, S. & Ohlsson, B. (1984). Cortical evoked potentials obtained after stimulation of the lower urinary tract. *The Journal of Urology, 131*, 306–309.

Bestler, M., Weitkunat, R., Keller, W. & Schandry, R. (1988). Heartbeat perception and body position (Abstract). *Journal of Psychophysiology, 2*, 130–131.

Davis, M. R., Langer, A. W., Sutterer, J. R., Gelling, P. D. & Marlin, M. (1986). Relative discriminability of heartbeat-contingent stimuli under three procedures for assessing cardiac perception. *Psychophysiology, 23*, 76–81.

Grigg, L. & Ashton, R. (1982). Heart rate discrimination viewed as a perceptual process: A replication and extension. *Psychophysiology, 19*, 13–20.

Haldeman, S., Bradley, W. E., Bhatia, N. & Johnson, B. K. (1982). Pudendal evoked responses. *Archives of Neurology and Psychiatry, 39*, 280–283.

Hantas, M. N., Katkin, E. S. & Reed, S. D. (1984). Cerebral lateralization and heartbeat discrimination. *Psychophysiology, 21*, 274–278.

Hefferline, R. T. & Perera, T. B. (1963). Proprioceptive discrimination of a covert operant without its observation by the subject. *Science, 139*, 834–835.

Hillyard, S., Picton, T. & Regan, D. (1978). Sensation, perception, and attention. In P. T. E. Callaway & S. Koslow (Eds.), *Event-related brain potentials in man*. New York: Academic Press.

Hillyard, S. A., Squires, K. C., Bauer, J. W. & Lindsay, P. H. (1971). Evoked potential correlates of auditory signal detection. *Science, 172*, 1357–1360.

Jasper, H. H. (1958). Charting the sea of brain waves. *Science, 108*, 343–347.

Jones, G. E., Jones, K. R., Cunningham, R. A. & Caldwell, J. A. (1985). Cardiac awareness in infarct patients and normals. *Psychophysiology, 22*, 4809–487.

Jones, G. E., Leonberger, T. F., Rouse, C. H., Caldwell, J. A. & Jones, K. R. (1986). Preliminary data exploring the presence of an evoked potential associated with cardiac visceral activity (Abstract). *Psychophysiology, 23*, 445.

Jones, G. E., Jones, K. R., Rouse, C. H., Scott, D. M. & Caldwell, J. A. (1987). The effect of body position on the perception of cardiac sensations: an experiment and theoretical implications. *Psychophysiology, 24*, 300–311.

Jones, G. E. Rouse, C. H. & Jones, K. R. (1988). The presence of visceral evoked potentials elicited by cutanous palpitation of heartbeats in high and low awareness subjects. *Psychophysiology, 25*, 459.

Josiassen, R.C., Shagass, C., Roemer, R.A., Ercegovac, D.V. & Straumanis, J.J. (1982). Somatosensory evoked potential changes with a selective attention task. *Psychophysiology, 19*, 146–159.

Katkin, E. S. (1985). Blood, sweat, and tears: Individual differences in autonomic self-perception. *Psychophysiology, 22*, 125–137.

Katkin, E. S., Blascovich, J. & Goldband, S. (1981). Empirical assessment of visceral self-perception: individual and sex differences in the acquisition of heartbeat discrimination. *Journal of Personality and Social Psychology, 40*, 1095–1101.

McNicol, D. (1972). *A primer of signal detection theory*. London: Allen & Unwin.

Netter, F. H. (1987). *The Ciba collection of medical illustrations*. Vol. 5. Stuttgart: Thieme.

Newman, P. P. (1974). *Visceral afferent functions of the nervous system*. London: Edward Arnold (Publishers) Ltd.

Rohen, J. W. (1978). *Funktionelle Anatomie des Nervensystems*. Stuttgart: Schattauer.

Ruchkin, D. S., Sutton, S., Kietzman, M. L. & Silver, K. (1980). Slow wave and P300 in signal detection. *Electroencephalography and Clinical Neurophysiology, 50*, 35–47.

Sarica, Y., Karacan, I., Thornby, J. I. & Hirshkowitz, M. (1986). Cerebral responses evoked by stimulation of vesico-urethral junction in man: Methodological evaluation of monopolar stimulation. *Electroencephalography and Clinical Neurophysiology, 65*, 130–135.

Schandry, R., Sparrer, B. & Weitkunat, R. (1986a). From the heart to the brain: A study of heartbeat contingent scalp potentials. *International Journal of Neuroscience, 30*, 261–275.

Schandry, R., Sparrer, B. & Weitkunat, R. (1986b). Brain-electrical correlates of the heart-beat signal (Abstract). *Psychophysiology, 23*, 459.

Schandry, R. & Specht, G. (1981). The influence of psychological and physical stress on the perception of heartbeats (Abstract). *Psychophysiology, 18*, 154.

Squires, K. C., Squires, N. K. & Hillyard, S. A. (1975). Vertex potentials in a rating scale detection task: Relation to signal probability. *Behavioral Biology, 13*, 21–34.

Weitkunat, R. (1987). *Psychologische und elektrokortikale Korrelate kardiozeptiver Prozesse*. München: Minerva.

Weitkunat, R., Schandry, R., Sparrer, B. & Beck, K. (1987). Zur Trainierbarkeit der Herzwahrenehmung. *Zeitschrift für experimentelle und angewandte Psychologie, 34*, 340–350.

Whitehead, W., Drescher, V. M., Heiman, P. & Blackwell, P. (1977). Relation of heart rate control to heartbeat perception. *Biofeedback and Self-Regulation, 2*, 371–392.

Wilkinson, R. T. & Seales, D. M. (1978). EEG event-related potentials and signal detection. *Biological Psychology, 7*, 13–28.