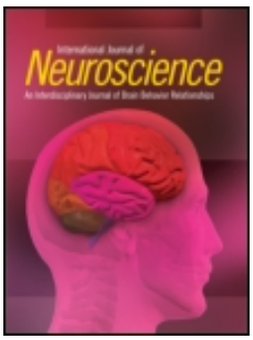

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FROM THE HEART TO THE BRAIN: A STUDY OF HEARTBEAT CONTINGENT SCALP POTENTIALS

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Heartbeat perception has become the most widely studied example of visceral perception. In the present study scalp potentials contingent to the visceral event "heartbeat" were investigated. Scalp potentials, averaged time-locked to the EKG-R-wave, were studied at *Fz*, *Cz*, and *Pz* under four conditions: resting (baseline), heartbeat discrimination task, signal detection task, and heartbeat discrimination task after physical exercise. 22 subjects were assigned to the two groups "good" and "poor" perceivers, according to their performance in an initial heartbeat perception test. Event related potentials (ERPs) of "good" perceivers were more stable across conditions than those of "poor" perceivers. Peak latency within the range of 200 to 300 ms differed significantly between conditions. A principal component analysis performed on the ERP averages extracted five components. Subsequent ANOVAs across factor scores yielded significant main effects for the "groups" factor, experimental conditions and electrode sites. The strongest effects occurred over the frontal region in the latency range of 250–400 ms (following the EKG-R-wave). These were found to be not due to artifactual EKG influences.

Detection of heartbeats is possible for a certain percentage of healthy subjects under resting conditions (cf. Jones & Hollandsworth, 1981; Katkin, Morell, Goldband, & Bernstein, 1982; Schandry, 1981). Thus, cortical processing of cardiac activity takes place during normal functioning of the heart, and certain events within the cardiac cycle must serve as perceivable stimuli. The nature of this stimulus is not yet known. Probably different physiological events serve as stimuli for different subjects.

The processing of *external* stimulation is manifested in event related potentials (ERPs). The analogue for visceral events has been shown in man only for *electrical stimulation* of the urinary bladder (Badr, Carlsson, Friberg, Lindström, & Ohlsson, 1982) and the pudendal nerve (Haldeman, Bradley, Bhatia, & Johnson, 1982). An evoked potential as a response to the regular activity of a visceral organ has not yet been reported.

Cardiac perception is an area of psychophysiological research that has been investigated increasingly for about 15 years. Previously it was regarded as mainly within the context of psychology of emotions (e.g., Mandler, Mandler, & Uviller, 1958; Valins, 1970) and was not treated as a psychophysiological variable that could be measured and manipulated in a quantitative manner.

With the advent of biofeedback research the methods of psychology of perception and models from information processing theory were applied to cardiac perception. Brener's theoretical concept of visceral awareness as a prerequisite of autonomic control (e.g., Brener, 1974) influenced experimental work on this question, and different methods to quantify cardiac awareness were developed (e.g., by Brener &

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Jones, 1974; Whitehead, Drescher, Heimann, & Blackwell, 1977). Cardiac awareness was improved by training procedures (e.g., Ashton, White, & Hodgson, 1979), and the accuracy of such awareness was affected by body position (Rouse, Jones, & Jones, 1984). Sexual differences in the effectiveness of cardiac discrimination training have been established (Katkin, Blascovich, & Goldband, 1981), and a relation has been demonstrated between cardiac awareness and emotional experience (Hantas et al., 1982; Katkin, 1985; Schandry, 1981). Many studies report interindividual differences in cardiac awareness. Although the majority of untrained subjects (about 80–90%) seem to have no sensations of heartbeats at normal resting conditions, a small proportion of subjects show good or even exact heartbeat perception.

There is no doubt that *cortical processing* of cardiovascular activity can be demonstrated in all humans. At the behavioral level a dependence of reaction time and signal detection performance on heart rate and cardiac phase has been reported (e.g., Lacey & Lacey, 1974; Sandman, McCanne, Kaiser, & Diamond, 1977). Following the Laceys' theory these findings can be explained as a result of the modulating effect of baroreceptor discharge on cortical activity and processing capacity (e.g., Lacey & Lacey, 1970). However, direct electrophysiological measurement in humans of cortical responses to changes in baroreceptor activity, or other cardiovascular processes, has, to our knowledge, not been reported.

Some EEG studies on cardiovascular activity have been published. Callaway and Layne (1964) and Walker and Sandman (1982) demonstrated that ERP waveforms change when sensory stimuli are given at different temporal positions of the cardiac cycle. Walker and Sandman interpreted this effect in terms of baroreceptor influences on the cortex.

The abovementioned findings indirectly support the hypothesis that cardiovascular afferent information interacts with higher central nervous functions. The present study employs a more direct psychophysiological approach. Under the premise that cardiovascular events like heartbeats exert an influence on cortical processes and are detectable as a sensory signal (at least within certain subjects and/or under conditions of stress and physical exertion), it is assumed that they evoke cortical responses analogous to "classical" sensory event-related potentials. The waveform of this "heart synchronous cortical potential" should be-in analogy to sensory ERPs-dependent, for example, on physical signal characteristics, attention and motivation.

The heart synchronous cortical potential may be studied simply by averaging EEG-samples time-locked to the EKG-R-wave or to any other event of the cardiac cycle. However, artifacts of noncortical origin have to be expected. Due to the averaging procedure a "scalp-EKG" may become prominent in any averaged scalp potential (cf., Callaway & Layne, 1964). Further, other artifacts may be generated by pulsing of cerebral fluids, and also become enhanced due to the time-locked averaging.

In order to separate cortical from noncortical electric activity, two different approaches may be applied. Firstly, experimental conditions can be manipulated in a way that presumably influences only one of the sources of potentials and leaves the other unchanged. For example, variation of cognitive set should yield electrocortical changes, (Donchin, 1981) but leave the EKG unchanged. Secondly, separation of different signals can be attempted by employing signal processing and filtering methods. In the present study both methods are reported.

The aim of the present study was the investigation of ERPs that accompany the cortical processing of the heartbeat signal. In detail the following questions were investigated: (a) Is there electrical activity at the scalp that is contingent upon the

heartbeat? (b) Is this activity influenced by attentional processes? (c) Is this activity dependent upon individual performance in heartbeat discrimination?

METHODS

Subjects

22 university students (11 male, 11 female) served as unpaid volunteer subjects. None of them suffered from heart or brain diseases, including prior injuries, or were taking prescribed medicine. Age ranged from 22 to 41 years (mean age 26.9 years).

Procedure

During the whole session subjects were seated in a comfortable reclining-chair. Eyes were closed during experimental conditions and only opened during resting intervals. Instructions were given via an intercom which was in operation during the whole session.

The session consisted of nine phases (see Table 1). The start and end of each phase was signaled by a 1000 Hz (55 dB) single or double tone, respectively. All acoustic stimuli were presented via loudspeaker located 50 cm behind the subject's head. In detail the session consisted of the following elements.

Rest. During these phases subjects were allowed to open their eyes. Also they received instructions concerning the following experimental phases. No biosignals were recorded.

Baseline (BASE). This phase was introduced to establish a resting level of physiological activity. Subjects were instructed to sit quietly and relax (duration: 400 heartbeats).

Heartbeat perception (HBP). In this phase the ability of subjects to perceive their heart activity was quantified by a heartbeat discrimination task so as to distinguish "good" from "poor" perceivers (Schandry, 1981). During three signaled time intervals (35, 25, 45 s) subjects counted their heartbeats silently. The start and the end of each time interval was signaled by a single or double tone, respectively. Subjects were instructed to detect their heartbeats by concentrating on bodily feelings associated with the action of the heart. Subjects were instructed *not* to take their own pulse or try any other physical manipulations which might facilitate the detection of heart-

TABLE 1
Order and duration of experimental phases

Phase	1 BASE	2 REST	3 HBP	4 REST	5 ATT	6 REST	7 SDT	8 REST	9 EX/ATT
Time (s)		30	180	30		30		30	
No. of Heartbeats	400				200		200		200

beats. At the end of each interval they reported the number of heartbeats counted. No information was given on to the duration of the three time intervals. No EEG signals were recorded.

Attention (ATT). In order to direct attention towards the "signal" (heart activity), subjects had to count silently their heartbeats. At the end of the phase, i.e., after 200 beats, subjects reported the number they had counted.

Signal Detection Task (SDT). In order to direct the subject's attention away from heart activity a signal detection task was introduced: Fifteen 800 Hz tones buried in white noise (white noise: 64 dB SPL, tone: 65 dB SPL) had to be detected. The number of detected tones had to be reported.

Exercise (EX). A condition invoking physical exercise was introduced to increase heartbeat and so change the characteristics of the assumed cardiac "stimulus." Subjects had to move their extended legs alternately upwards and downwards quickly until heart rate increased to 120 bpm. Subsequently, the ATT procedure was repeated.

White noise and tones in phase SDT were generated by a Roland JX-3P synthesizer.

Data Acquisition

EEG was recorded from *Fz*, *Cz* and *Pz* with Grass silver disc electrodes (E4S, fixed with self-adhesive Grass EC2 electrode-paste) referenced to linked mastoids. EKG was recorded from right midclavicula against the 7th left rib, using Beckman Ag/AgCl electrodes. The ground electrode was located at the forehead.

EEG-signals were preamplified by Princeton Applied Research 113 preamplifiers (gain: 1000, low frequency rolloff: .03 Hz, high frequency rolloff: 100 Hz). EEG and EKG were amplified by a 6-channel Beckman Type R-411 Dynograph recorder, EEG and EKG with a low-pass cutoff of 100 Hz and 30 Hz, respectively. Time constant for EKG was 1 s, for EEG 5.6 s.

Data acquisition was controlled by a PDP 11/34 computer digitizing the physiological data at a sampling rate of 200 per second and storing them on-line on 9-track digital tape. "Triggers" were generated by a hardware peak detector at the occurrence of the EKG-R-wave (ascending limb). Triggers also were stored on tape. A calibration signal of 13 μV was sent through the total recording system before each session.

Data Analyses

Averaging and filtering. Each raw data sweep (140 sampling points) started 100 ms pretrigger and terminated 600 ms posttrigger. The number of sweeps averaged per phase varied between 170 and 185, because sweeps contaminated by body- and eye-movement artifacts were excluded (exclusion was based on visual inspection at the oscilloscope, whenever voltage overflow (100 μV) occurred or a DC shift of more than 100 μV was visible). In order to remove residual 50 Hz line-frequency interference from the averages a 4-point moving average filter was used. This filter causes a (tolerable) phase shift of 2.5 ms. Averages were baseline-corrected by subtracting the mean of the first 15 samples (75 ms) of the pretrigger epoch[1] from the entire 700-ms sample.

[1]Because the Q-wave peaks within the last 25 ms of the pre-trigger epoch, the amplitude of the Q-wave would have largely determined the overall baseline value. For this reason, only the first 75 ms of the 100 ms pre-R-wave interval were used to compute each trial's baseline.

Grand averages across the averaged waveforms of the 22 subjects were computed separately for the 3 electrode sites in each of the 4 experimental conditions. Grand averages of the EKG were computed also for the 4 conditions.

Principal component analysis (PCA). A strong EKG-artifact has to be expected when averaging EEG in synchrony to an event in the cardiac cycle. This artifact could obscure components of a heart synchronous cortical potential. Because electrocardiac and electrocortical potentials at the skull are held to originate from independent generators, a PCA is expected to separate these two sources of variation.

The covariance matrix was factored using 264 ERP-waveforms (22 Subjects $\times$ 4 Conditions $\times$ 3 Locations) which were reduced from 140 to 45 points by averaging 3 adjacent data points and omitting the last 5 points, so that each data point represented 15 ms of EEG-activity. The criterion for factor extraction was set to Eigenvalue greater than 1 (cf., Wood & McCarthy, 1984). The five components obtained were Varimax rotated. In order to retain the original voltages, nonstandardized factor scores were calculated.

RESULTS

Heartbeat Discrimination

The accuracy of perception of heartbeats was quantified as an error score. The difference between reported and actual number of heartbeats was divided by the actual number of heartbeats. The sum of the absolute values of these three quotients as obtained in the three perception phases was computed. Thus, a high error score reflected a poor accuracy of overall perception. The sample was divided around the median into groups of "good" and "poor" perceivers.

Latency Effects

Figure 1 shows the grand mean EEG waveforms across all subjects for the 4 conditions, 3 locations, and the corresponding EKGs.

Visual inspection of the EEG grand averages reveals a similarity with the EKG grand average. In the latency range of the R-wave a sharp peak is visible for all locations and phases. In the time epoch of the T-wave a relatively high and broad peak appears at *Fz* and less distinct at *Cz*. At the parietal lead this peak was not seen.

In the attention condition, at locations *Fz* and *Cz*, a shift of this peak to longer latencies, as compared to the baseline-condition, was observed. Although only a minimal shift in the EKG-T-wave was obtained (BASE: 241 ms, SD: 5.53; ATT: 238 ms, SD: 5.61), the possibility that this latency shift was produced by a T-wave shift was investigated. The latency differences were measured between the average EKG-T-wave for each subject and the highest peak in the averaged EEG-epochs at *Fz* and *Cz* in the baseline and attention conditions between 200 and 300 ms. An ANOVA was calculated (Groups $\times$ Conditions $\times$ Locations, the latter two treated as repeated measures) on the mean values obtained (Table 2). A significant effect was obtained only for experimental conditions ($df = 1$, $F = 6.98$, $p < .05$). No interaction occurred with accuracy of heartbeat perception.

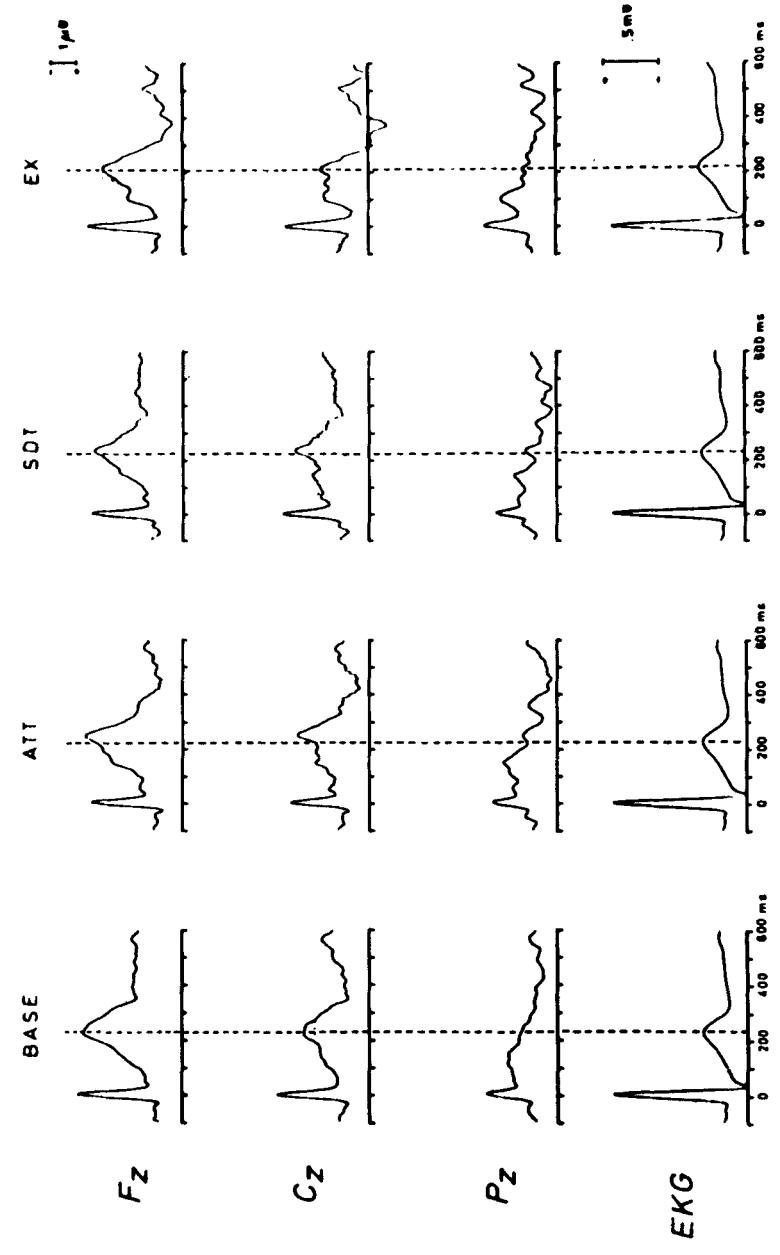

FIGURE 1 Grand averages across 22 subjects for conditions baseline, attention, signal-detection-task and exercise at *Fz*, *Cz* and *Pz*. Below are the corresponding EKG averages. (Dashed vertical lines indicate positions of maximum of EKG-T-wave. Since each individuals' averaged waveform consists of approximately 180 sweeps, each grand average represents about 3960 single sweeps.)

TABLE 2

Mean latency differences (ms) between the EKG-T-wave and the EEG-peak in the latency range 200–300 ms

Condition	Total Group Location		Good Location		Poor Location	
	Fz	*Cz*	*Fz*	*Cz*	*Fz*	*Cz*
BASE	−6.6	−12.1	−5.0	−12.7	−8.2	−11.4
	(8.2)	(8.8)	(13.9)	(14.5)	(9.2)	(10.8)
ATT	10.5	16.4	21.4	25.9	−.5	6.8
	(7.8)	(8.8)	(11.0)	(12.8)	(10.5)	(11.9)
BASE−ATT	17.1	28.5	26.4	38.6	7.7	18.2

ANOVA Results

Source	*df*	*F*	*p*
Groups	1	.67	.758
Exp. Conditions	1	6.98	.016*
Locations	1	.00	.962
Conditions × Groups	1	1.29	.270
Locations × Groups	1	.15	.703
Conditions × Locations	1	1.75	.200
Groups × Conditions × Locations	1	.01	.918

*$p<.05$

ERP Stability and Heartbeat Perception

The hypothesis that the detection of heartbeats is reflected in the ERP waveform, as a certain part of the total registered scalp potential (but not necessarily visible as a classical "peak"), suggests that interindividual differences in the stability of the waveform may be seen: ERPs of subjects which are able to discriminate heartbeats may include a certain segment, where processing of cardiac signals is reflected. Within this time domain, the ERPs of subjects *un*aware of heartbeats may show only noise. Thus, the ERPs of the latter are expected to be less stable than ERPs of good heartbeat perceivers.

Intraindividual stability of the ERPs across experimental conditions was evaluated in each subject at each location by calculating the cross-correlation coefficient between averaged ERP waveforms for pairs of experimental conditions (phase 1 and 2, 1 and 3, 1 and 4, 2 and 3, and so on). This yielded six correlation coefficients per subject and location. After Fisher Z-transformation of the correlation coefficients an average (stability score) from these Z-scores was computed for each individual.

The relation between ERP-stability and heartbeat discrimination was tested for the three locations by computing the Spearman rank-correlation between the Z-transformed average cross-correlations and the results of the heartbeat perception task. The three rank-correlation coefficients were in the expected direction, i.e., the better the heartbeat discrimination (i.e., lower scores), the higher was the stability score (overall $\rho = -.431$, $p<.05$).

Wave Form Analysis

The principal component analysis yielded five components, together explaining 85% of total variance. Figure 2 shows the factor loading functions of the Varimax-rotated components so that they correspond to the original waveform in polarity.

Table 3 shows the results of ANOVAs for the factor scores corresponding to the 5 components ($2\times4\times3$ ANOVA for factors groups, experimental conditions and locations; the latter two factors were treated as repeated measures). Component 4

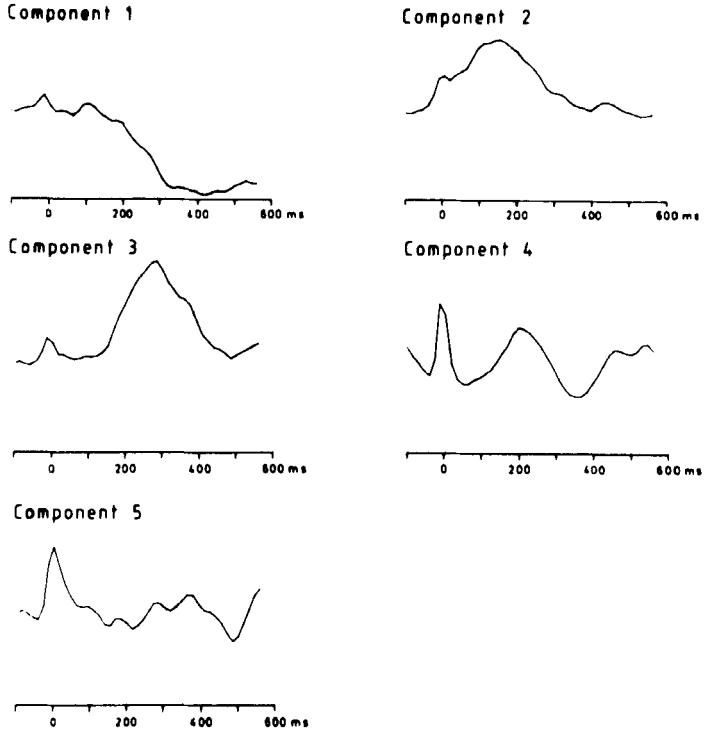

FIGURE 2 Varimax-rotated components from PCA across EEG averages. (Variance explained by components 1 to 5: 33%, 16%, 26%, 6%, 5%, respectively).

TABLE 3
Results of ANOVAs of factor scores

	Main effects					
	HB-Discrimination		Conditions		Locations	
Source	*F*-ratio	*df*	*F*-ratio	*df*	*F*-ratio	*df*
Component 1	.008	1	1.285	3	1.903	2
Component 2	1.874	1	.030	3	1.403	2
Component 3	1.628	1	4.015*	3	12.684**	2
Component 4	4.942*	1	3.234*	3	32.808**	2
Component 5	.790	1	.224	3	17.171**	2

*$p<.05$ **$p<.01$

showed significant main effects for the factors groups, conditions and locations, component 3 for the factors conditions and locations, and component 5 yielded a significant main effect for locations. Figure 3 shows the variations of the mean scores for the significant main effects in the ANOVAs. No significant interactions were observed.

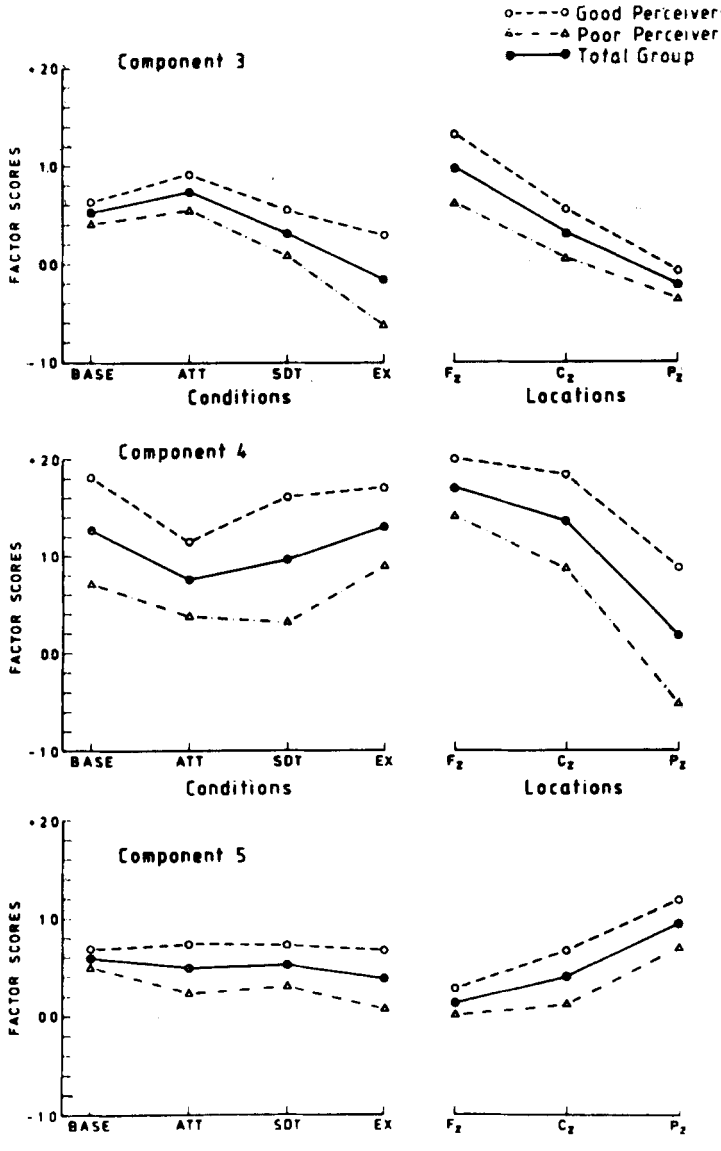

FIGURE 3 Mean factor scores of components 3, 4, and 5 reflecting the main effects of ANOVAs.

Reliability of PCA Results

Stability of component structure. The stability of component structures obtained from PCA after Varimax-rotation was investigated by splitting the raw data into two samples: single sweeps with even numbers ("even sweeps"), and single sweeps with odd numbers ("odd sweeps"). The two data sets were factored in the same way as the original data. Split-half data PCAs extracted six components (probably due to decreased S/N-ratio). Figure 4 presents the factor loading functions for the two subsamples.

The intercorrelations between the five similar factor loading functions obtained from the three separate PCAs (total data set, split half even, and split half odd) yielded high Pearson correlation coefficients between .53 and .99 ($p<.001$).

Estimation of EKG-influence. In order to examine whether the ANOVA results of the scalp potentials were determined by electrocardiac activity, a separate PCA for only the EKG was computed. This resulted in five components, each showing high factor loadings in the latency-range of the R-wave (Figure 5). Thus, all these components contributed to the explanation of variance in the R-wave range. ANOVAs

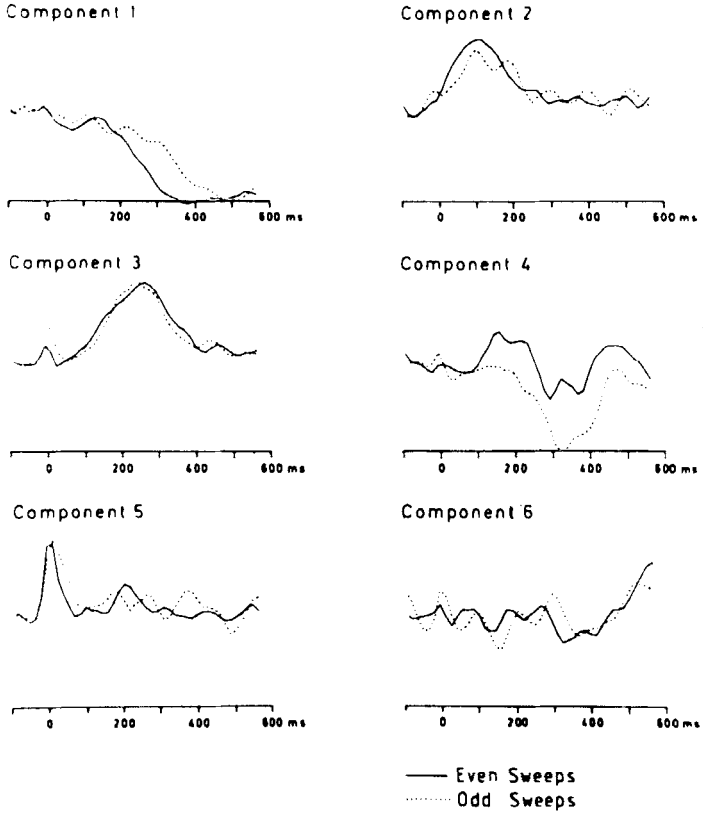

FIGURE 4 Varimax-rotated components from the separate PCAs across the split-half data. (Variance explained by components 1 to 6 for even sweeps 42%, 14%, 19%, 4%, 5%, 4%, for odd sweeps 28%, 10%, 18%, 18%, 7%, 4%.)

across factor scores (2×4 ANOVA for the groups and conditions factors) yielded significant effects *only for experimental conditions* (not for groups), for component 1, which was mostly representative of the R-wave, and for component 3, which represented parts of the R-wave and the descending limb of the T-wave (Table 4). The differences in factor scores between condition EX and all other conditions were responsible for the significant effects (cf., Table 4).

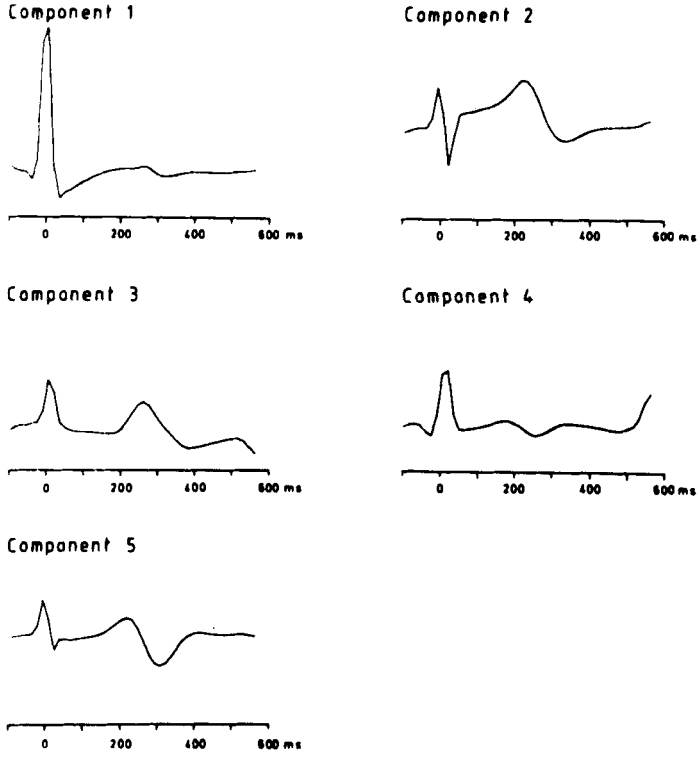

FIGURE 5 Varimax-rotated components from PCA across EKG averages. (Variance explained by components 1 to 5 43%, 22%, 12%, 9%, and 7%.)

TABLE 4
Mean factor scores obtained from PCA across EKGs and results of ANOVAs

	Experimental conditions				ANOVA results	
	BASE	ATT	SDT	EX	*F*-ratio	*df*
Component 1	30.03	30.63	31.13	33.04	7.49**	3
Component 2	20.47	19.91	20.04	20.00	.49	3
Component 3	−2.37	−.27	.23	5.73	10.91**	3
Component 4	4.24	5.26	5.13	4.78	.42	3
Component 5	1.03	.16	.69	.34	.26	3

**$p<.01$

Evaluation of PCA components

Usually in ERP studies PCA is used to quantify waveform characteristics of the averages by comparing "classical" peaks with certain component loading functions. In the present case clear peaks were not identifiable and PCA served primarily to evaluate the effectiveness of experimental manipulations and to give some information about latency ranges where relevant activity may occur.

The possibility that the PCA in the present case had generated a spurious "component structure," as could be obtained from randomly distributed data, was evaluated in that the component structures of the separate PCAs across the split-half data were well reproduced.

Component 1. This factor loading function has to be interpreted as a "baseline-component": Whenever the single waveforms submitted to a PCA are set to a common baseline level, a DC-shift over the sampling epoch, which is present to some extent in every averaged waveform, causes the extraction of a component that explains variance of this shift. A second reason for the appearance of a component of this shape is the averaging procedure, time-locked to an event at the beginning of the sampling epoch. With increasing distance (in time) from the "trigger," time jitter of the signal waveform also increases, thus leading to a higher variance at later time points.

Component 2. Neither loading function nor factor scores provide information about the significance of this component. It is present also in the PCA across the split-half data, but not in the EKG–PCA. Thus, it seems to be of cortical origin, but independent of experimental manipulations.

Component 3. A superficial inspection of component 3 may give the impression (Figure 2) that it reflects mainly EKG-interference in the latency of the T-wave. If this were true, the PCA across the EKG should generate a corresponding component, which additionally should show similar differences in factor scores. Such a component was not found, so it can be assumed that signals from *cortical* sources respond sensitively to experimental conditions and electrode placement (ANOVA, Table 3). The strongest expression of this component is observed at *Fz* under the attention condition (Figure 3), during the conditions signal detection task and exercise, parietally as well as frontally, this component appears significantly weaker. Probably, it is a component common to all subjects that is enhanced primarily during relaxed, but attentive states. We also assume that this component reflects the same kind of activity that generates the peak in the 200–300 ms range, which was observable in the grand averages.

Component 4. The shape of this component resembles that of the components 2 and 5 from the EKG–PCA (Figure 5). Between -50 and 50 ms a sharp peak can be observed, probably generated by electrocardiac interference (R-wave). The subsequent biphasic complex (150 to 450 ms) can be found in the abovementioned EKG-components also (Figure 5): The first peak (150 to 280 ms) in EKG-component 2, the second (280 to 450 ms) in EKG-component 5. In spite of this similarity it cannot be assumed that these two peaks are mainly a reflection of electrocardiac activity. The factor scores of this EEG-component behave differently as compared to the two EKG-components: While the EKG-components are only minimally sensitive to a manipu-

lation of experimental conditions (Table 4), for EEG-component 4 three significant main effects were yielded by the ANOVA (Table 3). It was sensitive to performance in heartbeat discrimination (generally higher scores for "good" perceivers), electrode placement (decreasing from frontal to parietal sites), and experimental conditions (lowest scores under condition attention, especially for "good" perceivers).

Component 5. For this component the ANOVA yielded a significant main effect for electrode placement (highest factor scores at *Pz*). In the latency range of the R-wave a prominent peak was observed (see also component 4 of the EKG–PCA). Probably, the ANOVA main effect is due to different EKG-interferences at the three electrode positions (see also grand averages, Figure 2). The factor scores maximum at *Pz* may be attributable also to a quite different reason. EEG-components responding to independent variables appear at *Pz* generally quite weak (cf., Figure 3). Thus, if there is no relevant cortical activity present at *Pz*, variance of noise will lead to another (artificial) component (due to Varimax-rotation).

DISCUSSION

The present study demonstrated a stable average waveform at the scalp (using the R-wave as a "trigger"). EKG influence contributed to a certain degree to this waveform which was dependent on electrode location. In the grand averages a peak, in the latency range from 200 to 300 ms, was observed which was relatively broad during baseline. When attention was directed towards the heartbeat this peak became sharper and appeared later. The intraindividual stability of the scalp potential correlated with performance in a cardiac perception task. A PCA yielded five reliable components. ANOVAs on factor scores proved that experimental manipulations exert effects on the components. EKG-influences cannot account for these effects.

The *demonstration* of a reliable ERP waveform as a consequence of heartbeat synchronous EEG recording was the main aim of this study. Influences of electro*cardiac* origin have to be considered seriously when averaging synchronously to any cardiac event. In our data EKG interference became obvious as a dominant peak in the time range of the R-wave at all locations under the four conditions. However, EKG artifact is not considered relevant in determining between conditions effects. Firstly, a PCA computed with the *EKG-data* resulted in components that were different from the EEG components (differences in shape of factor loading functions and different results in the ANOVAs performed on factor scores). Secondly, the ERP peak in the range of 200 to 300 ms varied independently from the EKG between the conditions attention and baseline. Thirdly, the correlation between stability of scalp potentials and performance in the cardiac discrimination task is not explicable as an effect of EKG interference.

The effects of the independent variables upon the waveforms have to be assessed in light of other possible sources of potentials other than electrocortical and electrocardial. Cerebral blood volume changes have to be taken into account. Recently, Sandman, O'Halloran, and Isenhart (1984) showed that brain blood volume changes occur after auditory stimulation (synchronized with diastolic cerebral pulse pressure wave). We cannot exclude the possibility that systematic blood volume changes exert some influence on the registered waveforms. However, there are no indications in the literature that cerebral blood volume may change with the kind of experimental manipulations we introduced in our study.

The latency range of 200 to 300 ms, where major effects due to experimental manipulations were observed, is important from different points of view. The "stimulus," serving as the trigger for the detection of a heartbeat, most likely occurs in the time interval of 50–200 ms following the R-wave. Within this epoch the aortic valve opens (R + 50 ms), the blood is propelled into the aorta by the maximum contraction of the left ventricle (R + 100 ms), and a pressure wave travels along proximal areas of the aorta producing a rapid distension of the aortic walls. It seems reasonable to accept these dramatic dynamic processes as the "stimulus" in heartbeat perception. The high density of mechanoreceptors in this area of the cardiovascular system as well as in the surrounding thoracaic tissue guarantees the afferent transport of cardioceptive information.

Behavioral data on heartbeat perception also lend support to locating the heartbeat "stimulus" within this time interval. Whitehead et al. (1977) found that it is possible for "good heartbeat perceivers" to discriminate between "immediate" heartbeat feedback, presented 128 ms after the R-wave, and "delayed" feedback (384 ms after the R-wave). Whitehead et al. chose the delay of 128 ms for the "immediate" condition, because "mechanical contraction of the heart requires about 100 ms following the R-wave, and the blood pulse wave may not reach the neck or the wrist until approximately 150 ms after the R-wave." Clemens (1984), who manipulated the delay of heartbeat feedback systematically in the range of 0 to 400 ms after the R-wave, reported that subjects found external stimuli delayed by 0 or 100 ms from the R-wave as more "synchronous" to the heartbeat than signals delayed by 400 ms, and that signals occurring in the first 250 ms of the cardiac cycle were perceived as more representative of heart action than later signals.

If we hypothesize that the relevant (perceivable) cardiac event occurs between 100 to 200 ms after the ascending limb of the R-wave in the cardiac cycle, then electrocortical evidence may be expected 150 to 600 ms following it. The N100, known from the ERP literature on exteroceptive signals as the first prominent peak, may be elicited in the time range of 200 to 300 ms.

At *Fz* and *Cz* a dominant peak between 200 and 300 ms was obtained. When attention was directed to heartbeats, this peak appeared as a rather distinct waveform at about 250 ms after the R-wave. This effect was mainly due to the "good" perceivers within the total group (Table 2). We assume tentatively that this peak reflects central processing of cardiac information. Under resting conditions, this component is probably smaller and is obscured by overlapping T-wave activity. Under condition of selective attention to heartbeats it may become sufficiently enhanced to override T-wave interference.

The PCA factor loading functions being most marked in this time range are components 3 and 4 (cf., Figure 2). Both components proved to be sensitive to a conditions effect. Factor scores of component 4 also were different for the groups of good and poor heartbeat perceivers (cf., Figure 3). Thus, the PCA results also support the assumption of a cardiac "stimulus" event occurring in the time range 100 to 200 ms after the R-wave and a brain-electrical representation of this event after another 100 to 200 ms.

In conclusion, this study lends strong support to the assumption that: (a) the cortical processing of cardiovascular events is accompanied by scalp potentials of electrocortical origin, and (b) these potentials are influenced by psychological variables.

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