Dynamics of SCR, EEG, and ERP activity in an oddball paradigm with short interstimulus intervals


Abstract

Studies of concurrent central, and autonomic activity using a conventional event-related potential (ERP) oddball paradigms, are considered useful in elucidating the relationship between central and autonomic responses, but the autonomic response tends to overlap. A new method was used to decompose and score overlapping skin conductance responses. This method enabled examination of dynamic relationships of phasic SCR, prestimulus electroencephalogram (EEG), and ERP to auditory target stimuli in 50 normal adults. SCR amplitude was negatively correlated to EEG and N200 amplitude. The SCR amplitude changes over time exhibited an exponential decline opposite to those of N200, alpha, and beta. All the fitted exponential functions had a time constant of 1–2 min. The findings suggest that a N200 component, active in the auditory sensory discrimination, is concomitant with the SCR. The narrow range of the time constant may provide a clue to the concomitant processes underlying central and autonomic adaptive functions.

Descriptors: EEG, N2b, SCR, Electrodermal activity, Orienting reflex, Time constant

Research studies on electrodermal activity (EDA), electroencephalography (EEG), and event-related evoked potential (ERP), have become essentially separate disciplines since their respective inception in 1888 (Bloch, 1993), 1929 (Brazier, 1961; Gloor, 1969), and 1964 (Halliday, 1980). This paper serves to: (a) briefly review possible common adaptive functions of EEG, ERP, and SCR; (b) present the difficulties of concurrent central nervous system (CNS) and autonomic nervous system (ANS) studies; (c) describe a solution to these difficulties; and, (d) report for the first time the interrelationships between concomitant CNS–ANS variables in a paradigm with short interstimulus intervals (ISI).

EEG

The possible genesis of various rhythmic EEG activities have been reviewed extensively (Lopes da Silva, 1991; Steriade, Gloor, Llinàs, Lopes da Silva, & Mesulam, 1990). Variation of brainstem tonic excitation to the cortical neurons leads to arousal, EEG desynchronization, and shifts in EEG frequency (Cobb, 1963; Kellaway, 1990; Montplaisir, 1975; Moruzzi & Magoun, 1949; Niedermeyer, 1987). Event-related desynchronization (ERD) following auditory or visual stimulation has also been demonstrated (Pfurtscheller & Arambar, 1977), and poststimulus alpha blockage (desynchronization) has been linked to the orienting reflex (Voronin, Bonfitto, & Vasilieva, 1975). A functional link between EEG and electrodermal activity, as indices of arousal and orienting is expected, but poststimulus EEG contains both ERD and ERP. Prestimulus EEG measures, on the other hand, can be used as an index of the brain state modulating ERP responses (Basar, Basar-Eroglu, Rosen, & Schütz, 1984; Intriligator & Polich, 1994). Prestimulus alpha and beta activity have been associated with prestimulus electrodermal level modulated by arousal (Lim et al., 1996).

ERP

Multiple components have been found to be active around the time of N100 (Näätänen & Picton, 1987). N100 is generally regarded as an exogenous component of ERP modulated by stimulus parameters and attention. Enhancement of ERP late components (N200 and P300) to stimuli with temporal uncertainty (Sutton, Baren, Zubin, & John, 1965), with an unpredicted sudden change (Ritter, Vaughan, & Costa, 1968), and to selective attention (Ford, Roth, Dirks, & Kopell, 1973; Hillyard, Hink, Schwent, & Picton, 1973) have been demonstrated. N200 has been suggested to reflect a central orienting response (Ford, Roth, & Kopell, 1976; Squires, Squires, & Hillyard, 1975) and is thought to reflect a decision process related to sensory discrimination of attended stimuli (Ritter, Simon, Vaughan, & Friedman, 1979). In the time zone of N200, there are two recognized components, N2a and N2b (Perrault & Picton, 1984). N2a is regarded as an automatic response to deviant stimuli in a series of standard stimuli with its maximum amplitude over the anterior head region (Näätänen & Gaillard, 1983). A subset of N2a, the mismatch negativity (MMN), has been
proposed as the cerebral initiation of the orienting reflex (OR; Näätänen, 1992).

N2b is a broad, centrally distributed wave between 150 and 215 ms in response to infrequent attended auditory stimuli. N2b has been interpreted as a brain event associated with transient arousal and the OR (Loveless, 1983; Näätänen & Gaillard, 1983). In attend oddball conditions, N2b is often followed by P3a (frontocentrally distributed, peaking between 220 and 280 ms), also associated with the OR (Squires et al., 1975). The N2b–P3a wave complex has been widely linked to the orienting response (Halgren & Marinkovic, 1994; Loveless, 1983; Lytyinen & Näätänen, 1987; Näätänen & Gaillard, 1983).

P3b (P3 or P300) has a longer latency and is more prominent in the parietal region. It is thought to reflect “context updating” (Donchin, 1979), or “context closure” (Verleger, 1988). P3b has not been shown to habituate and it is, therefore, not considered a likely correlate of autonomic OR (Donchin, 1981; Donchin et al., 1984). These findings indicate that any central adaptive changes that relate to electrodermal activity are most likely to be seen in the time zone of N200.

SCR
Skin conductance (SC) reflects the activity of the eccrine sweat glands innervated by the sympathetic sudomotor nerves of the autonomic nervous system (Fowles, 1986; Wallin, 1981). SC is composed of two components—a phasic, stimulus-related SCR, and a tonic arousal-related skin conductance level (SCL) (Boucsein, 1992). SCR waveform is considered to reflect sweat activity in the sweat ducts in the skin and the opening and the collapse of the sweat duct near the pores (Edelberg, 1993). SCR is regarded as the most useful laboratory test for an autonomic response (Damasio, 1994). SCR is a widely investigated autonomic response (Graham, 1973) and is usually regarded as an index of OR (Fowles, 1986; Näätänen, 1992, p. 63). Sokolov (1960) regarded what Pavlov (1927) described as a generalized aroused state following an unexpected external stimulus as a manifestation of a generalized OR, and later extended it to include focal OR (Sokolov, 1975), of which SCR is a principal component (Boucsein, 1992, p. 218). The SCR habituation associated with repetitive, regular, unattended stimuli is well known (Boucsein, 1992; Graham, 1973; Roy, Sequeira, & Delerm, 1993; Siddle, Remington, Kuiack, & Haines, 1983; Siddle, Smith, & Marcer, 1974). Most electrodermal studies have used paradigms of long ISIs. Recently, SCR habituation in the nonattend condition has been shown using a very short ISI of 1.1 s (Barry, Feldmann, Gordon, Cocker, & Rennie, 1993). The demonstration suggests that the adaptive character of SCR is not compromised with the use of short ISI stimulation.

CNS–ANS Integration
Concurrent measures of central and autonomic function have proved difficult because ERPs have a time course in the order of a fraction of a second, whereas the SCR has a time course in the order of several seconds (and SCL varies over an even longer time scale). A paradigm with a short ISI, such as conventionally used in ERP research, results in overlapping SCRs (Figure 1), making quantitation of responses difficult (Boucsein, 1992; Dawson, Schell, & Filion, 1990). This difficulty has contributed to the paucity of research into concurrent ANS and CNS activities.

Some ANS and CNS studies have employed intermediate ISIs (Roth, Goodale, & Pfefferbaum, 1991), whereas others used ANS data qualitatively to study EEG (Davies & Krkovic, 1964; Voronin et al., 1975) or ERP activity (Lytyinen, Blomberg, & Näätänen, 1992). Short ISI auditory stimulation is known to be more useful for study of focused attention (Schwent, Hillyard, & Galambos, 1976), and alpha and galvanic skin responses (GSR) to light stimuli of ISI of 2–3 s show closer correspondence than those responding to stimuli of longer ISI (Voronin et al., 1975). Therefore, a conventional paradigm with short ISI was used in this study.

Subaveraged ERPs have long been used in ERP studies to uncover ERP features that would otherwise be obscured by the conventional averaging (Ritter, Simon, Vaughan, & Friedman, 1979; Starr, Sandroni, & Michalewski, 1995). Few studies have assessed the dynamic of central and autonomic changes across a trial. This was the focus of the current study. We predicted that:

![Figure 1](https://example.com/figure1.png)

**Figure 1.** Skin conductance traces (upper) from an individual attending and responding to 40 targets presented at random but fixed sequential times (upward markers on the time axis) during a trial. The responses to the targets were complex, consisting of numerous overlapping skin conductance responses, each with a fast rise and a slow decline.
The electrodes were excited by a constant voltage of 0.5 V (Fowles et al., 1981; Lykken & Venables, 1971) so that the recorded signal, proportional to the resultant current, was also proportional to the conductance.

**Skin Conductance Response Evaluation System (SCORES)**

SCORES scans the entire SC data of a session and for each response complex associated with a stimulus, iteratively optimizes the model parameters so as to minimize the mean square difference between the actual and the fitted curves. The fitted curves have been shown to be almost indistinguishable from the actual raw response complex (the top two superimposed SCR curves in Figure 2). At this point (for an example see Figure 2), in the vast majority of cases, the mathematically generated pattern fitted the actual raw response complex well. None had residuals exceeding 5% of the response amplitude. The decomposed parts are the tonic level, the decaying residual from the last response, and the two “pure” SCRs (SCR 1 and SCR 2) displaced by time interval of two stimuli (the four parts below the superimposed raw and fitted curves in Figure 2). A range of variables including peak amplitude and latency of the first “pure” response associated with a stimulus (SCR 1) are measured automatically. More details regarding the mathematics are available elsewhere (appendix in Lim et al., 1997).

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**Method**

**Subjects**

Fifty normal subjects (25 men and 25 women aged 18–70 years, mean = 45.4 years, SD = 15.0 years) participated in the study. Subjects were recruited from the surrounding community. Subjects with a history of a major medical, neurological, or psychiatric disorder or with history of alcohol or other drug abuse were excluded. Subjects were asked to avoid tobacco or caffeinated drinks for 3 hr and alcoholic beverages for 1 day before the recording sessions.

**Stimulus Paradigm and Data Acquisition**

The subjects were seated comfortably in a quiet, dimly lit laboratory with air-conditioned ambient temperature set at 24 ± 1°C. Subjects sat facing a monitor screen and wearing a pair of headphones. The operator was in an adjacent control room with audiovisual links. A conventional auditory oddball paradigm was used, with a constant ISI of 1.32 s. The hearing threshold was determined. Auditory stimulation consisted of tones at 60 dB above threshold with duration 50 ms and with a rise and a fall time of 10 ms. Tones of 1000 Hz were designated as background and tones of 1500 Hz as target. A total of 40 target and 247 background tones were presented in a random but fixed sequence, with intertarget intervals (ITI) varying between 2.64 and 15.84 s. Subjects were instructed to attend to the target tone and respond with a button press “as quickly as possible” when they were “sure” they had heard a target stimulus. They were also asked to look at a colored dot on the center of the video screen. SC, EEG, single trial ERP, and electrooculogram (EOG) were recorded simultaneously using a 32-channel Syn Amps DC amplifier system (Neuro Scan Inc., Sterling, Virginia, USA). The horizontal EOG was recorded via a pair of electrodes placed 1 cm lateral to the outer canthi and the vertical EOG was monitored via a pair of electrodes placed above and below the right eye in line with the center of the pupil. The EOG signals were used for correction of their contribution to the scalp recorded EEG by a technique based on Gratton, Coles, and Donchin (1983). The signals were band-limited to 50 Hz, digitized at 250 Hz, and stored continuously for more than 6.5 min for subsequent offline processing.

**SC Recording**

SC was recorded by a pair of silver-silver chloride electrodes, approximately 0.8 cm² in contact area, filled with electrode paste of 0.05 M sodium chloride in an inert ointment and placed on the volar surface of the distal phalanges of digits II and III of the nondominant hand. The skin area was wiped with an alcohol swab.
All 10-s epochs containing an SCR within 1–3 s following each of the 40 targets in each subject were evaluated. The segments containing composite SC signals and often overlapping responses were decomposed into phasic SCR and tonic SCL components by curve-fitting using our software package SCORES. The SCL and the “pure” SCR amplitude and latency associated with each target stimulus were obtained, sorted by target and averaged across subjects. The four parameter values of all the SCRs were grouped into 40 target bins and subaveraged (missing values did not contribute to the average). An SCR waveform was constructed from the four parameter values for each target for the group.

**EEG**

EEG was recorded from 19 head sites placed according to the 10-20 system (Jasper, 1958) in reference to linked-ear electrodes. The EEG was amplified by 200, digitized, and stored continuously during the session. After EOG correction, any prestimulus EEG epoch containing signal in excess of ±500 μV was excluded from analysis. Fast Fourier transforms (FFT) of 1-s EEG segments prior to each target were then performed after applying a windowing function to minimize leakage and end effects. The EEG spectral amplitudes from 0 to 48 Hz were grouped into sub-bands as follows: delta band (1–3 Hz), theta band (4–7 Hz), alpha band 1 (8–9 Hz), alpha band 2 (10–13 Hz), alpha (8–13 Hz), beta band 1 (14–18 Hz), beta band 2 (19–24 Hz), beta band 3 (25–30 Hz), and beta band 4 (31–48 Hz). EEG data from only Cz site was analyzed. As the delta band, theta band, and beta band 4 data did not show a systematic changes over target presentation times, no further analysis was performed on them. The two alpha bands 1 and 2 exhibiting similar trends were lumped together and averaged as a single alpha band. Similarly, beta bands 1, 2, and 3 were grouped into a single beta band. The root mean squares (rms) amplitudes were used for trend analysis. The alpha and beta rms amplitudes associated with each target stimulus were averaged across subjects.

**ERP**

Target ERPs at Cz were extracted from the continuous recordings, with each epoch extending from 200 ms prestimulus to 800 ms poststimulus. The average of the 200-ms prestimulus EEG served as the baseline for amplitude measurement. Target ERPs were subjected to the same artifact rejection method as employed for EEGs. The single trial epochs associated with each of the 40 targets were averaged across subjects, resulting in an ensemble of 40 ERPs corresponding to targets 1 to 40. N100, P200, N200, and P300 ERP peak latencies and amplitudes falling within the following windows (80–140 ms for N100; 150–200 ms for P200; 180–280 ms for N200; 280–550 ms for P300) were scored automatically. The peak within each window was determined by the signs of the gradients before and after a specific data point. The specific data point was defined as the peak if one sign of its preceding gradients sustained for more than three data points, and an opposite sign of its ensuing gradients also sustained for more than three data points. The peak amplitude was measured from a baseline defined as the mean of the data over a 200-ms prestimulus period. The peak latency was determined from the stimulus onset to the peak time.

The automatic scores were checked by two reviewers and were re-scored when required in a small number of cases.

**Statistics and Analysis**

Preliminary exploratory analysis of the trends of all measured variables for each targets revealed that only SCR, alpha amplitude, beta amplitude, and N200 amplitude varied systematically across the trial. Temporal trends of these measures were curve-fitted using a standard nonlinear least-squares routine known as the Marquardt-Levenberg method (Press, Flannery, Teukolsky, & Vetterling, 1992). Before statistical analysis, SCR amplitudes were transformed logarithmically to reduce skewness in the distribution and to improve normality. To avoid undue influence of individual spurious measurements and to satisfy the normality assumption of the analysis of variance (ANOVA), outliers were removed. For each of the three variables, cases in each target with values greater (or less) than 1.5 times the interquartile range from the upper (or lower) quartile were considered outliers (0.82% of alpha data, 0.92% of beta data, 1.08% of SCR data). Separate correlation studies of SCR amplitudes versus total alpha, beta, and N200 amplitude data over trial were performed.

**Results**

The fact that the decomposed parts of the SCR summated to the raw trace with little error suggests that the overlapping SCRs (Figure 1) had been scored successfully (Figure 2). A total of 1,421 SCRs (or 71% of targets) were obtained from all 50 subjects. SCR, N200, and EEG frequency spectra showed systematic dynamic patterns from the 1st to the 40th target stimulus (Figure 3). The first SCR was by far the largest, followed by the next six target responses, which were larger than the rest (Figure 3A). Alpha band amplitude showed the most prominent changes among the four EEG bands. Alpha amplitude was initially low (first three targets) and became increasingly larger up to the 25th target and varied in between these levels thereafter (Figure 3B). Beta changes followed a similar pattern, but were less obvious because of their low amplitude relative to the alpha band. The N200 amplitude was the only ERP component that showed a systematic change over the trial (Figure 3C). N200 amplitude increased markedly after the first five targets.

Three key features of the CNS and ANS activity were identified. (1) The dynamic amplitude changes of SCR, alpha, beta, and N200 were best modeled as exponential functions of the time of target presentation. The parameters of the exponential time functions are listed in Table 1. SCR amplitude exhibited an exponential decline with time with a high goodness of fit (Figure 4A, Table 1). There was an exponential rise from an initial low amplitude, both in EEG alpha and beta amplitudes over the trial (Figure 4B and N200 ERP (Figure 4C, Table 1). (2) SCR, alpha, beta, and N200 dynamic profile over the trial reached a plateau within 3 min and had time constants between 1 and 2 min (Figure 3, Table 1). (3) Log (SCR) was significantly correlated individually with alpha, beta and N200 amplitude (Table 2).

**Table 1. The Variables and Parameters of the Fitted Exponential Time Functions**

<table>
<thead>
<tr>
<th>Dependent variable</th>
<th>Time constant (TC) (s)</th>
<th>Initial level (a0) (μS)</th>
<th>Constant (c)</th>
<th>goodness-of-fit (r²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>SCR (μS)</td>
<td>59.6</td>
<td>0.25</td>
<td>0.21</td>
<td>0.82</td>
</tr>
<tr>
<td>EEG alpha (μV)</td>
<td>70.1</td>
<td>1.06</td>
<td>2.26</td>
<td>0.59</td>
</tr>
<tr>
<td>EEG beta (μV)</td>
<td>101.6</td>
<td>0.82</td>
<td>2.76</td>
<td>0.54</td>
</tr>
<tr>
<td>ERP N200 (μV)</td>
<td>55.8</td>
<td>7.86</td>
<td>0.00</td>
<td>0.49</td>
</tr>
</tbody>
</table>

Note: SCR = skin conductance response; EEG = electroencephalogram; ERP = event-related potential.
Discussion

The pattern and directions of change of SCR amplitude, alpha and beta amplitudes, and N200 amplitude across the trial are evident in Figure 3. SCR amplitude correlated negatively with EEG alpha, EEG beta, and N200 amplitude (Table 2). In addition, the SCR amplitude exhibited an exponential decline opposite to the incline of EEG alpha, EEG beta, and N200 ERP over time of target presentations (Figure 4). Moreover, their fitted exponential functions had a time constant between 1 and 2 min (Table 1). It is notable that the dynamics of concurrent CNS and ANS activity with ISIs of 20–30 s in nonattend condition, in which responses usually extinguish after 5–20 min of stimulation and reappear when there is a change in any stimulus feature (Näätänen, 1992, p. 64). To date, little data have been published on the characteristics of SCRs obtained in an oddball paradigm with short ISIs of 1–2 s. One of the reasons is that scoring overlapping SCRs has been a difficult task (Barry et al., 1993; Lyytinen et al., 1992) until recently (Lim et al., 1997). The other is the desire to avoid overlapping responses all together (Roth et al., 1991).

A number of EDA habituation results obtained using ISIs between 15 and 120 s indicate that longer ISIs produce larger responses and slower response habituation than shorter ISIs (Siddle, Stephenson, et al., 1983). Responses to attended stimuli of short ISIs can be obtained and scored, and the differences between responses to attended stimuli of short ISI from those obtained using a classical habituation paradigm with long ISI have been described elsewhere (Lim et al., 1999). Briefly, the former exhibit higher

Figure 3. The grand-average waveforms of skin conductance responses (SCRs) (A), electroencephalogram power spectra (B), and event-related potentials (ERPs) (C) evoked by targets 1 to 10 and targets 15, 20, 25, 30, 35, and 40. The divisions on the vertical axis in panels (A), (B), and (C) are 0.25 µS, 2 µV/Hz, and 10 µV, respectively. Each target label marks the position of the zero baseline for the target trace. In (C), the dotted line is the zero baseline based on 200 ms prestimulus data for each target ERP trace. Upward deflection indicates negativity. The SCR peak amplitude is decreasing (A) and the alpha and beta band powers are increasing (B) and the N200 amplitude at about 200 ms is also increasing (C) with the advancing sequence of targets. Note in C, the relative sizes of N100 and N200 and that the N200 amplitude above the zero was small for targets 1 to 5, with an increase in N200 amplitude thereafter.
response rate, greater response prevalence, and faster rate of decline than the latter. Despite these differences, a key common feature is the response habituation in both paradigms. The rate of SCR decrement in this current study appears to be consistent with the general trend of faster habituation with shorter ISI, and it perhaps defines a new boundary of response habituation rate. Because habituation is widely regarded as an elementary form of learning (Stephenson & Siddle, 1983), this boundary may contribute to the understanding of learning process especially in its early stages of memory consolidation.

Could the close correspondence of ERP and SCR response characteristics be due to the sweat gland activity on the scalp being embedded in the ERP? This explanation is unlikely, because the N200 onset occurred at about 150 ms and lasted less than 200 ms, whereas SCR onset in the fingers was about 1,720 ms. The net difference in onset time on the scalp was more than 900 ms after allowing for the time difference of 800 ms for the head-finger conduction time difference over a head-hand length difference of 0.8 m for a c fibre conduction velocity of about 1 m/s (Shahani, Halperin, Boulu, & Cohen, 1984). Moreover, the SCR rise time (peak time to onset time) of about 2,500 ms is an order of magnitude greater than that of N200.

The increment of N200 over trial, in an attend condition, has not been reported previously. N200 in nonattend conditions has been found to increase progressively in size from wakefulness through stage I to stage II sleep, and has been considered to reflect an arousal effect (Picton et al., 1974). This finding can possibly explain N200 increment with advancing targets across the trial, perhaps associated with decreasing level of arousal with lapsed time. However, our paradigm involved active auditory discrimination and reaction time response in a relatively short test, during which the tonic skin conductance level was reasonably stable. The SCL did not correlate with any of the ERP wave peak or with SCR. This argues against the influence of global tonic variations in arousal. An ERP peak (O-wave) increment with trial repetition has been reported in an unattend condition (Simons et al., 1987). We believe that the N200 increment seen in our study reflects a central adaptive response to stimulus repetition not directly associated with general tonic arousal.

Because button press to target detection has been found to cause substantial slow negative shift in ERPs prior to stimulus (Starr et al., 1995), our observed N200 amplitude increment could potentially be due to differences in negative shifts over trial, namely reflecting changes in the readiness potential. This possibility is unlikely, because the ERP peaks in this study were measured relative to the averaged level in the prestimulus 200-ms period, and according to these authors when the slow shifts were removed, the ERPs associated with count and button press paradigms did not differ (Starr et al., 1995). The lack of ERP waveform differences between count and button press paradigms in their study also argues...
against the influence of motor-related potentials on endogenous ERP. Reaction time correlates more with N200 latency than P300 latency, and increasing reaction times is associated with increasing N200 amplitude (Ritter et al., 1979). Furthermore, N200 peaks 50 ms before EMG response onset, and P300 peaks 100 ms after EMG responses onset (Starr et al., 1995). These findings indicate that sensory discrimination decisions have been made around the time of N200, and suggest ERP components contributing to N200 play a role in sensory discrimination.

The close temporal relationship between SCR and N200 amplitude in this study suggests a link between the central and autonomic OR. The dynamic nature of the N200 and its smaller amplitude than P300 might have contributed to the paucity of description of N200 in the literature. The N200 in this study may contain the bulk of N2b, and perhaps part of the positive-going slope of P3a in the N2b–P3a wave complex discussed in the introduction. An alternative view is that N2b does not change in response to repetitive targets and what is varying has been P3a, being large in the initial few targets and habituated later. The summated N2b and P3a waves could have resulted in an apparent reduction of N200 initially, increasing later with habituation. The summated N2b and P3a waves could have resulted in an apparent reduction of N200 initially, increasing later with habituation of P3a. Conceptually, the two scenarios are plausible and the literature seems to favor the notion of an N2b–P3a wave complex. Because the positive P3a peak is small, has always been embedded in the positive going slope of P3b (P300 or P3), and is difficult to measure, the N200 wave peak provides a measurable quantity reflecting this complex. The temporal patterns over trial of both SCR and N200 shared an almost identical time constant (Table 1). This close dynamic relationship between N200 and SCR, sharing identical time constant provides further evidence for the N2b–P3a wave complex as a central concomitant of an autonomic electrodermal OR. Further studies are needed to differentiate the two scenarios.

Interpretation of N2b, P3a, or N2b–P3a wave complex, being a central OR, leads to the general expectation of a positive relationship between N200 and SCR. But N200 was found to increase with SCR decrement in this study. This apparent directional discrepancy between the empirical data and the general expectation has also been observed in a nonattend condition (Simons et al., 1987). This discrepancy may be explained as follows: The progressive increment of N200 responses to the initial six or seven targets as seen in our study, would have been lost in a conventional average because the grand-averaged ERPs would have been dominated by the later, larger responses, leading to a final averaged tracing with a sizeable N200. The initial increment is lost in the process. A corollary to the explanation is that a fully developed N200 or a diminished P3a represents an habituated central OR and the initial incremental change of N200 reflects central OR habituation in progress.

If this explanation is accepted, the network substrate underlying incremental dynamic changes of N200 may also be linked to progressive consolidation of the neuronal model according to Sokolov’s comparator theory (1960, 1963, 1975, 1990). The substrate for the neuronal model has been associated with cortical regions and the hippocampus (O’Donnell et al., 1993; Salisbury, O’Donnell, MCarley, Shenton, & Benavage, 1994; Sokolov, 1975; Vinogradowa, 1975). Sokolov (1990) argues that there are two forms of OR, an involuntary (novelty related, unattended) and a voluntary process (significance-related, attended). Näätänen (1992, p. 64) proposes two components for OR, namely arousal (nonspecific generalized) and attentional (informational). MMN falls into the arousal category, and apart from the first stimulus during the session, the bulk of our data may be classified into their attentional category. Näätänen (1992, pp. 241–243) argues that in sensory discrimination tasks, N2b is an index of detecting MMN exceeding a threshold. Association of N2b with an integrative detector, accumulating evidence of mismatches to surpass a threshold, has been made by others (Loveless, 1986). An adaptive process might unfold, with iterative threshold changes over time, from the initial fuzzy boundary to a critical boundary as described by the exponential rise time constant for N200 (and SCR) over trial. The iterative process may include the strengthening of the neuronal representation of the stimulus, which occurs during conscious deviance detection as part of the ongoing sensory discrimination task (Schroger, 1997). This dynamic aspect of N2b, evidenced in our data, has not been demonstrated before. Its temporal variation with SCR response has been too difficult to demonstrate without the SC decomposing tool.

Finally, the EEG alpha and beta trends across the trial needs to be differentiated from those of SCR and N200, in that the EEG was a prestimulus measure and hence not a direct consequence of the target stimulation. The pseudorandomized target presentation in the paradigm would have minimized the influence of an anticipatory process. The initial lower EEG amplitude or EEG attenuation (Figures 3B and 4B), may be attributed to increased arousal. As the experiment progressed, the level of arousal decreased, EEG synchrony was enhanced and EEG amplitude increased—findings all largely consistent with previous research (Davis, 1939; Lindsley, 1952). Accordingly, one may argue that the background cortical activity reflects the general level of arousal, which might influence (either separately or collectively) N200 and SCR. However, in this study, the SCL, which is an index of arousal (Bohlin, 1971; Boucsein, 1992), did not show any trend over the trial. Therefore changes in EEG cannot be solely due to lowering arousal. On the other hand, EEG desynchronization has been positively linked to activation and attentional demands (Boiten, Sergeant & Geuze; 1992; Dujardin et al., 1993; van Winsum, Sergeant, & Geuze, 1984), and it is thought to be due to cortical excitation through disinhibition of thalamic interneurons (Pfurtscheller & Aranibar, 1977). Furthermore, coexistence of both ERD in discrete cortical regions engaged in a task, and of event-related synchronization in regions not engaged in the task (Pfurtscheller, 1992), argues more for changes associated with information processing than for global changes in arousal.

In summary, it is not surprising that there are modulations underlying central and autonomic functions. What was surprising in this study was the similarity in temporal patterns displayed by different measures of brain function, which had time constants of 1–2 min, and the selective covariation of the N200 component with SCR. This is the first demonstration of the selective and dynamic relationships between SCR and N200. The 1–2 min time constant provides a clue to the conjoint processes that underlie these central and autonomic adaptive functions.

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