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Slow brain potentials in a visual-spatial memory task: topographic distribution and inter-laboratory consistency

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Abstract

Slow brain electrical potentials (SPs) were investigated in a visual-spatialmemory task. Two issues were addressed: (1) the nature and topographic distribution of the potentials obtained under such conditions; and (2) the consistency of the SPs when recorded in six identically configured laboratories. Fifteen young male subjects were studied at each laboratory (total $n = 90$). The paradigm entailed presentations of paired visual patterns (S1 and S2), to which subjects responded with a choice reaction time response indicating whether or not the two patterns matched. A biphasic contingent negative variation (CNV) was produced which consisted of an early symmetric component with bilateral foci at posterior temporal sites and a subsequent mid-parietal dominant wave later in the retention interval. Although the CNVs from all laboratories were similar in waveform and in topographic distribution, there were significant inter-laboratory differences in amplitude of the slow potential components. The topographic distributions of the components and the possible role of sampling effects are discussed. © 1997 Elsevier Science B.V.

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1. Introduction

Recent studies have emphasized the diversity of slow potentials (SPs) and have continued to affirm their value as measures of the brain processes underlying cognitive function. The most well known SP is the Contingent Negative Variation (CNV), first described by Walter et al. (1964). The CNV is traditionally observed as a negative SP that develops during the foreperiod in forewarned reaction time situations. It is now generally accepted that what is commonly called the

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CNV comprises multiple waves, each of which depends individually on the specifics of the task used to elicit it. In simple tasks, at least two major components can be distinguished: an early 'O wave' complex apparently related to the characteristics of the warning signal (S1) and a second component apparently related to preparation for motor response at the time of the imperative signal (S2) (Rohrbaugh and Gaillard, 1983). A separate component, the 'stimulus preceding negativity,' appears under conditions in which S2 provides informative feedback (Ruchkin et al., 1986; Brunia, 1988).

More recently, it has been established that a distinctive family of SPs is also obtained within the CNV paradigm under conditions in which a memory load is imposed at the time of S1 (Rugg, 1984; Barrett and Rugg, 1990; Ruchkin et al., 1990). The specific timing, amplitude and topographic representation of these memory-related SPs depend on the character of the material to be remembered. A particularly compelling demonstration of this specificity was described by Ruchkin et al. (1992). In separate conditions, subjects made judgments based on memory for either the phonological or the spatial aspect of visually-presented letter displays. Associated differences in the SPs were taken as evidence for the existence of separate brain systems involved in short-term memory of phonological and visual-spatial material. The SP recorded in the visual-spatial memory condition was characterized by a preponderance over occipito-temporal sites during the encoding phase early in the interval, whereas later in the retention interval it assumed a midline parietal focus. The amplitudes of both early and late phases varied directly with the memory load.

The present report presents additional data describing the character of the CNV in conditions involving visual-spatial memory. Two general aspects are emphasized. First, the basic waveform and topographic distribution of the CNV obtained under such conditions are examined in detail. Although it is not without precedent (e.g. Sanquist et al., 1981), the posterior focus described by Ruchkin et al. (1992) differs markedly from the fronto-central distribution that is traditionally ascribed to the CNV and thus is in need of additional study. Care was taken in the present study to use pattern stimuli that were devoid of any obvious or readily imparted semantic or phonological content.

Second, the consistency of the CNV when recorded under standardized conditions in multiple laboratories was assessed. This is an important issue since it defines the ultimate acceptability of the CNV as a basis for clinical diagnosis, or for the study of individual differences or phenotypic markers. An opportunity to address this issue was presented by the Collaborative Study on the Genetics of Alcoholism (COGA) project, which involves six participating laboratories at US medical schools and research institutions, at which the ascertainment and measurement procedures have been carefully standardized. A variety of clinical assessment and biological measures are taken from the family members of alcoholic and control subjects. Included among these measures are the CNV, as well as other EEG and event-related potential (ERP) measures. The present visual-spatial memory CNV paradigm was selected for study in this context because of prior evidence that the CNV is affected by alcohol acutely (Kopell et al., 1978; Rohrbaugh et al., 1988) and chronically (Skerchock and Cohen, 1984) and indications of visual-spatial processing deficits in alcoholics and their genetic relatives (see review by Parsons, 1993).

2. Methods

2.1. Subjects

Data were obtained from groups of paid normal control subjects who were tested explicitly for the purpose of studying inter-laboratory consistency. Data from $n = 15$ subjects were contributed by each of the six participating coga laboratories, for a total $n = 90$ subjects. These laboratories are located at the University of Connecticut Health Center (CT), State University of New York Health Sciences Center at Brooklyn (NY), University of Iowa (IA), Indiana University (IN), Washington University in St. Louis (MO) and The University of California, San Diego (CA).

Each laboratory is configured identically with a sound attenuated and electrostatically shielded chamber, reclining chair and stimulation and recording apparatus. Subjects were males, between 18 and 29 years of age, who denied any history of neurological or psychiatric illness, uncorrected vision defects, or the use of any psychoactive medication at the time of testing. All but six were right handed, as determined by questionnaire. Subjects were determined by breath analysis not to have measurable levels of blood alcohol at the time of testing. A screening interview was used as a basis for excluding subjects who had a history of drug or alcohol abuse, or who had a family history of drug or alcohol abuse in first or second degree biological relatives.

2.2. Visual-spatial memory task

S1 and S2 stimuli were visual pattern stimuli, consisting of 3–16 connecting or intersecting line segments, which were displayed on the face of a computer monitor. Representative stimuli are illustrated in Fig. 1. At a viewing distance of 1 m, the stimuli subtended 3–6° visual angle. The patterns were displayed for durations of 100 ms with the S1 and S2 onsets separated by 3250 ms and constant inter-trial intervals of 8 s. Trials were presented until 25 artifact-free trials (see below) were obtained, or until a total of 50 trials were presented. On a random half of the trials, the S1 and S2 patterns matched. Subjects were instructed to respond with a button press response (within 1000 ms) with the index finger of the right hand on matching trials and with the left hand on non-matching trials. The memory task was considered easy with these simple stimuli and yielded performance accuracy of essentially 100%.

A square outline frame, defining the field of view, appeared 587 ms before the onset of S1 and remained on continuously until 587 ms after the termination of S2. At the viewing distance of 1 m, this frame subtended 9.6° visual angle. Subjects were instructed to maintain focus on a central fixation mark and were encouraged to avoid eye movements or blinks during the period in which the frame was displayed.

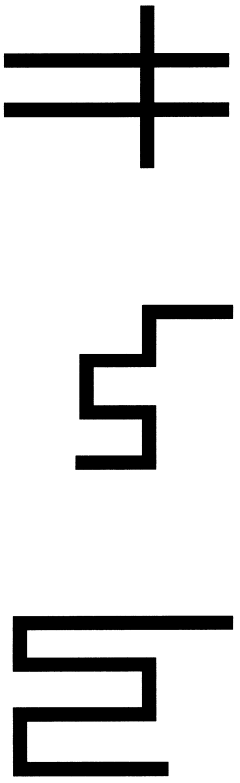

Fig. 1. Samples of visual pattern stimuli.

2.3. CNV recording

CNV data were recorded from the 19 standard 10–20 sites using Electro-caps with tin electrodes, plus additional sites at the left outer canthus and mid-forehead to monitor horizontal and vertical EOG. Electrode attachment impedances were below 5 kΩ and all channels were referred to the tip of the nose. A ground electrode was attached at a location 3 cm anterior to Fz. Signals were amplified with a bandpass of 0.02–50 Hz (3 dB down, 6 dB per octave slope) and digitized at a rate of 256 Hz. Individual trials were rejected automatically if the voltage (with respect to the baseline beginning 100 ms before S1) in any channel exceeded ± 73 μV. A single average was

[1] Laboratories are listed here and elsewhere in random order.

formed for each subject, in which both match and non-match trials were combined. Individual subject averages reported here are all based on at least 15 artifact-free trials and contain EOG activity during the foreperiod of less than 12.5 μV.

3. Results

3.1. Reaction time (RT)

The mean RTs (and associated standard deviations) from the six laboratories are presented in Table 1. Because of technical problems the values were available for only four subjects at the CT laboratory; all other laboratories contributed 15 values. A one-factor ANOVA revealed that there were no significant differences among Laboratories in mean RTs ($F[5,73] = 0.61$).

3.2. CNV component identification

The grand averaged CNV waveforms from each of the 19 electrode sites, plus the vertical EOG, are plotted in Fig. 2. These waveforms begin 100 ms before S1 and are truncated 500 ms after S2 because of excessive EOG activity beyond that time.

The CNV waveforms are distinctly biphasic in appearance. An early peak (approximate latency 1000 ms post-S1) is prominent at posterior temporal (T5, T6) and occipital (O1, O2) sites, whereas a subsequent ramp-shaped negative wave late in the interval is greatest at the midline parietal (Pz) site. These two components were quantified in terms of the average amplitudes (with respect to the 100 ms pre-S1 baseline) within two measurement epochs: 750–1250 ms post-S1 (Early CNV) and 2750–3250 ms post-S1 (Late CNV).

The topographic distributions of the grand averaged Early and Late CNV measures are depicted in Figs. 3a and 4a, respectively. These distributions emphasize the topographic differences between Early and Late CNV measures, with the Early activity appearing largely along a bilaterally symmetric, posterior crescent between foci at T5 and T6 and the Late component distributed more broadly with a focus at Pz.

The distinction between Early and Late CNV components was verified statistically by analyzing the respective distributions along the transverse chain between T5 and T6 (Fig. 5). An ANOVA including the factors of Laboratory ($n = 6$), Electrode Site (T5, P3, Pz, P4, T6) and Component (Early, Late CNV) yielded significant interactions between Electrode Site and Component ($F[4,336] = 104.5$, $P < 0.0001$, based on Greenhouse–Geisser corrected degrees of freedom for this and other tests), as well as a significant effect for Laboratory ($F[5,84] = 3.00$, $P < 0.05$).

Profile analysis, as described by Ruchkin et al. (1988), was used to ensure that possible interactions with Electrode Site reflected distributional differences separate from possible main effects of amplitude. The Early and Late CNV measurements were scaled individually so that the respective root mean square (rms) amplitudes of the average measurements were equal (McCarthy and

Table 1
Means (+SD) of reaction time and Early and Late CNV amplitude at the six laboratories

Laboratory	Reaction time (ms)	Early CNV[a] (μV)	Late CNV[b] (μV)
CT	655 (186.9)[c]	−5.38 (4.62)	−6.26 (6.08)
NY	670 (142.1)	−5.19 (3.70)	−5.21 (3.90)
IA	755 (166.4)	−8.79 (4.23)	−8.54 (4.20)
IN	734 (195.3)	−9.02 (5.37)	−6.85 (6.89)
MO	685 (112.6)	−8.02 (3.83)	−10.54 (3.34)
CA	702 (125.1)	−5.37 (3.07)	−4.23 (5.72)

[a] At T6 electrode.
[b] At Pz electrode.
[c] Because of a technical problem the RT values for CT are based on only four subjects.

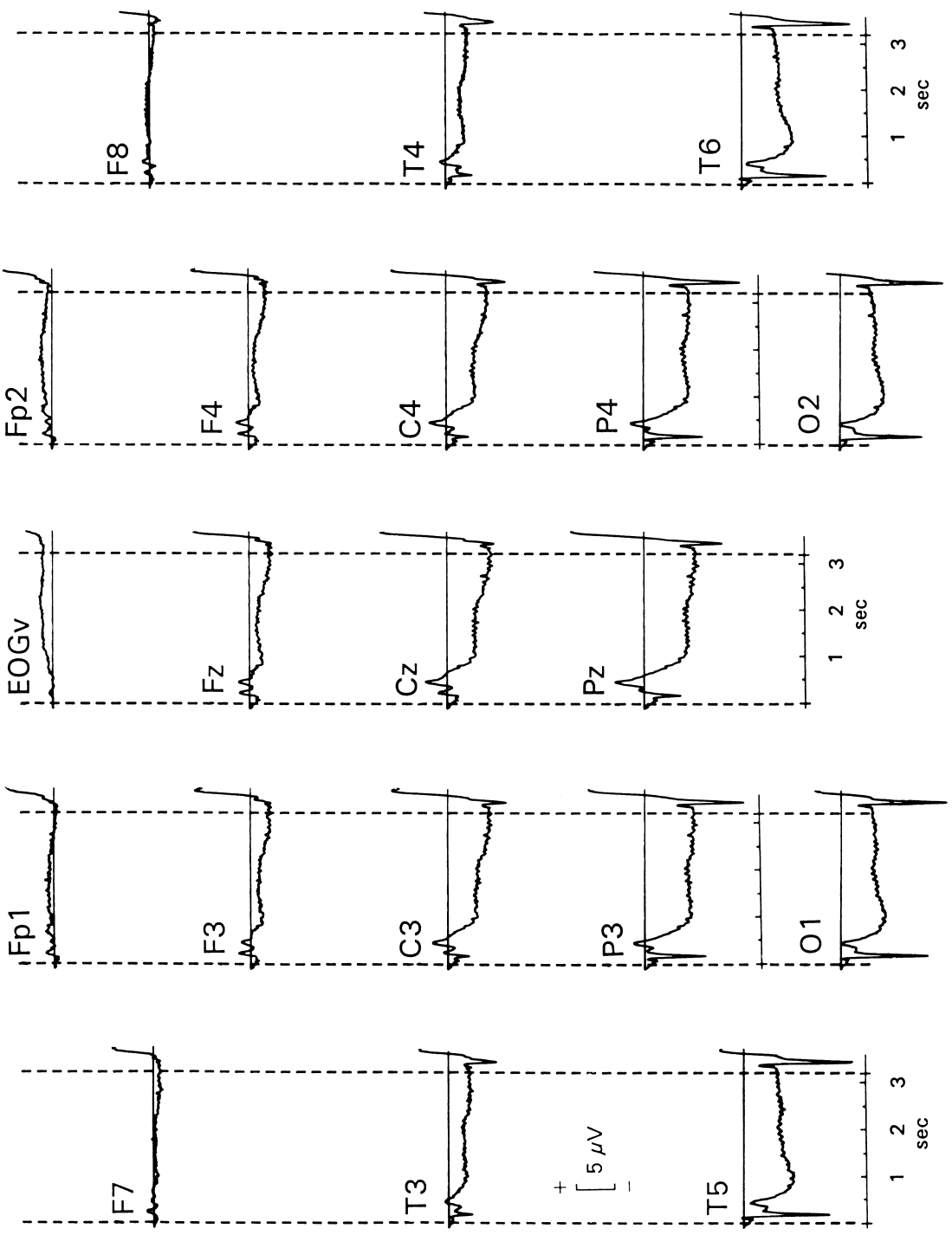

Fig. 2. Grand averaged CNV waveforms for each of the 19 scalp sites, and associated vertical EOG. Waveforms have been digitally low-pass filtered at 32 Hz.

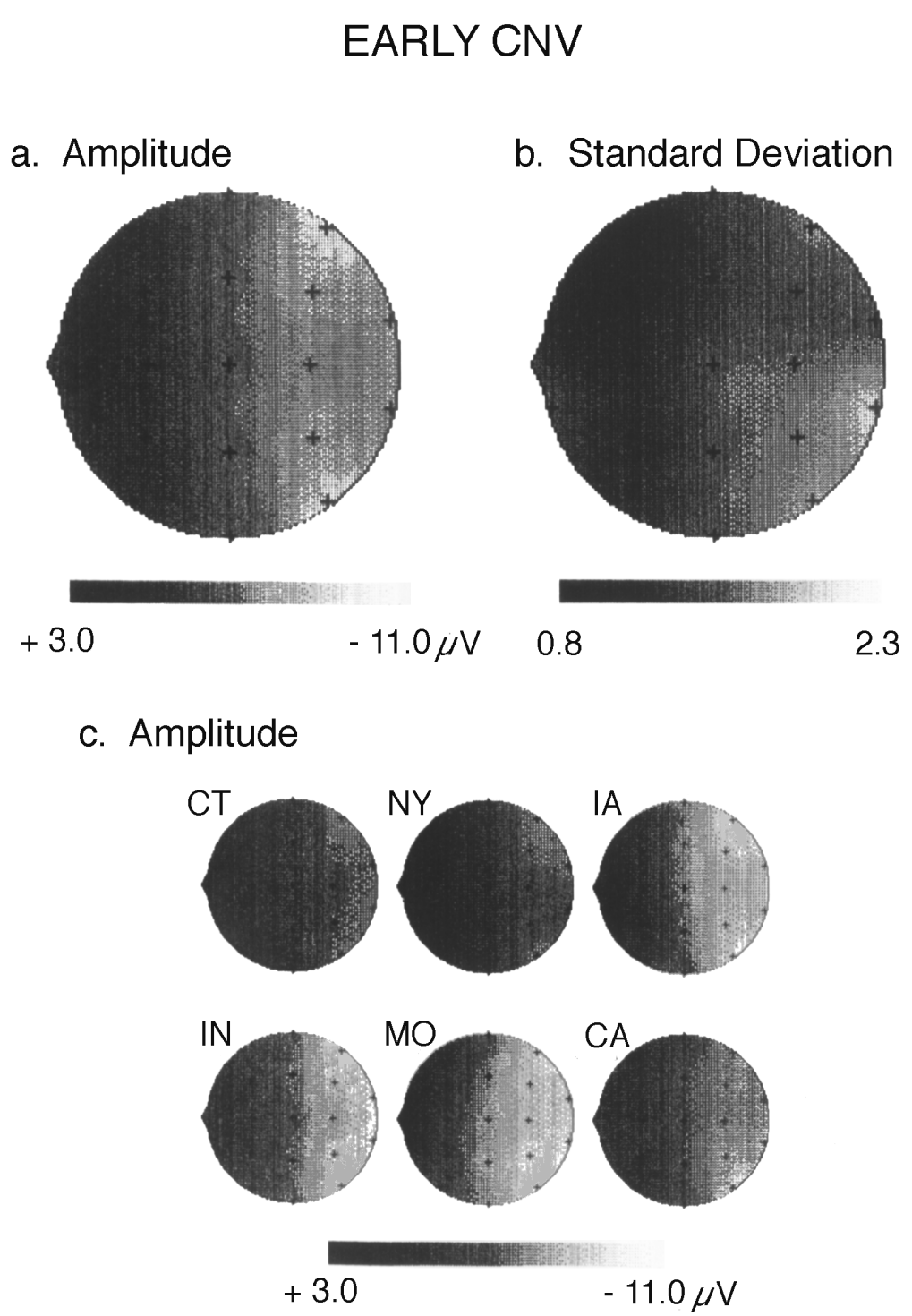

Fig. 3. Maps depicting the topographic distribution of the grand averaged Early CNV measure (a), the corresponding distributions for each of the six contributing laboratories (c), and the associated standard deviations of these six values at the individual scalp loci (b). Maps were constructed using the spherical spline interpolation method incorporated in the EEGSYS program library.

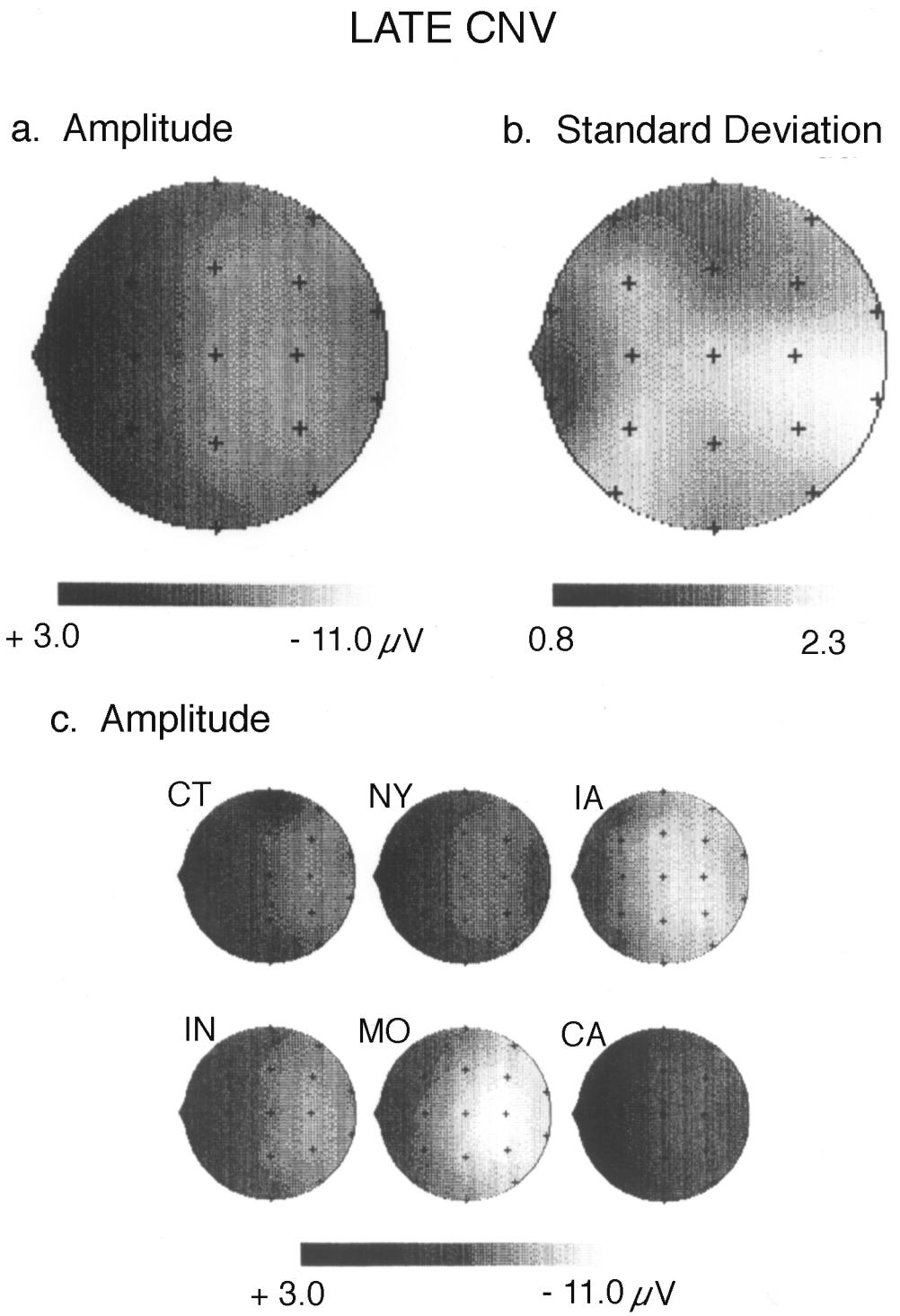

Fig. 4. Maps depicting the topographic distribution of the grand averaged Late CNV measure (a), the corresponding distributions for each of the six contributing laboratories (c), and the associated standard deviations of these six values at the individual scalp loci (b).

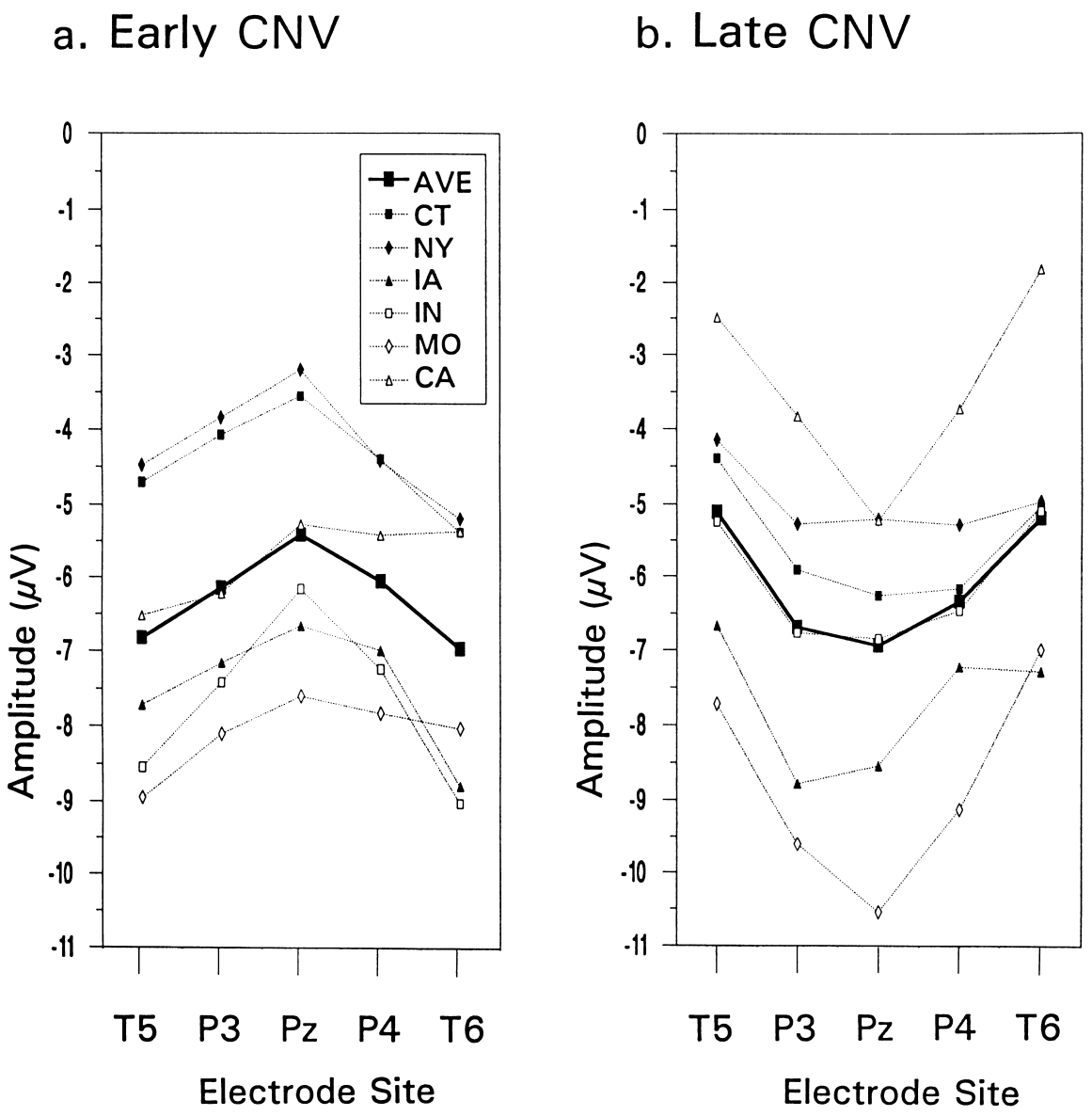

Fig. 5. Amplitudes of the Early (a) and Late (b) CNV measures along the transverse chain of electrode sites between T5 and T6, for the grand average waveform and the average waveforms from each of the contributing six laboratories.

Wood, 1985). An ANOVA of the scaled measures again provided evidence for independence in the form of a significant Component X Electrode Site interaction ($F[4,336] = 95.4$, $P < 0.0001$), but no significant interactions involving Laboratory.

The degree of association between the Early and Late CNV components was assessed further by computing the Pearson correlations, across all subjects, of the amplitudes of the early and late components at the Pz electrode site (Woods and Courchesne, 1987). The correlations were significant but only of modest magnitude ($r = 0.54$ at Pz over $n = 90$ subjects, $P < 0.001$).

A number of additional ERP components also

were identified. These included prominent, occipital-maximum negative components following both S1 and S2 (peak latency ca. 175 ms post-stimulus at O1) and a parietal-maximum P300 positive component following S1 (peak latency ca. 415 ms post-S1 at Pz). The visual appearance of these components suggested that all were affected markedly by overlap with the SPs and thus a formal analysis of amplitude is not presented here. There were no significant differences among laboratories in the latencies of these components ($P > 0.05$).

3.3. Inter-laboratory consistency

The CNV waveforms at T6 and Pz from each of the individual laboratories are plotted in Fig. 6. Although the waveforms from each laboratory showed a biphasic character, the amplitudes varied appreciably. The average Early CNV (at T6) from the different laboratories ranged from −5.19 (NY) to −9.02 (IN) μV and the Late CNV (at Pz) varied from −4.23 (CA) to −10.54 (MO) μV (Table 1).

The inter-laboratory amplitude differences are depicted more completely in the topographic maps of the Early and Late CNV measures from the individual laboratories (Fig. 3c and Fig. 4c), which disclose a considerable amount of variability at all electrode sites (see also Fig. 5). This variability is quantified in Fig. 3b and Fig. 4b, which depict topographic maps of the variability in component mean amplitudes from the six individual laboratories. In general, the variability was greater for the Late CNV than for the Early CNV. The variability within laboratories was also substantial for both components, with standard deviations in excess of 6.0 in some instances (Table 1).

The inter-laboratory differences in mean amplitudes were found to be statistically significant. An ANOVA of the Early CNV component, in which all Electrode Sites ($n = 19$) and Laboratories ($n = 6$) were included, yielded a significant effect of Laboratory ($F[5,84] = 2.62$, $P < 0.05$) and a significant interaction between Laboratory and Electrode Site ($F[90,1512] = 1.74$, $P < 0.05$). Individual one factor ANOVAs of the amplitudes at each Electrode Site yielded a significant main effect of Laboratory ($P < 0.05$) at all sites along the inter-aural line and posterior to it, except C4, P4 and O2. Profile analysis of the rms scaled Early CNV data failed to attain significance for the interaction between Laboratory and Electrode Site ($F[90,1512] = 1.05$), indicating that the apparent interaction was secondary to an amplitude main effect. A corresponding ANOVA of the Late CNV component (involving the factors of Laboratory and Electrode Site) yielded a significant main effect of Laboratory ($F[5,84] = 3.07$, $P < 0.05$) but not a significant interaction between Laboratory and Electrode Site either when based on raw scores or on the rms scaled scores ($F < 1$). Individual one factor ANOVAs of the unscaled amplitudes for each Electrode Site revealed significant effects of Laboratory ($P < 0.05$) at F7, F3, Fz, F4, C3, Cz, Pz and O1.

Separate ANOVAs of the rms scaled data in which hemisphere was introduced as a factor (including all homologous pairs) yielded no significant effects ($P > 0.05$) associated with hemisphere for either the Early CNV or Late CNV measures.

In general these analyses indicated that appreciable differences among laboratories existed for absolute amplitude, but not for the essential biphasic character of the CNV waveforms nor for the respective distributions over the scalp of the Early and Late components.

4. Discussion

The data presented here bear on the character of SPs recorded in a CNV paradigm under conditions requiring visual-spatial memory and on their consistency when recorded in separate, identically configured laboratories. The CNV waveforms were found to consist of distinct Early and Late components, distinguishable on the basis of topographic distribution. Although the waveforms were similar in this respect to those traditionally observed in simple CNV paradigms, the topographic representations of the Early and Late components on the whole were more posterior than those customarily ascribed to the CNV (see Rohrbaugh and Gaillard, 1983).

The present data agree remarkably well with

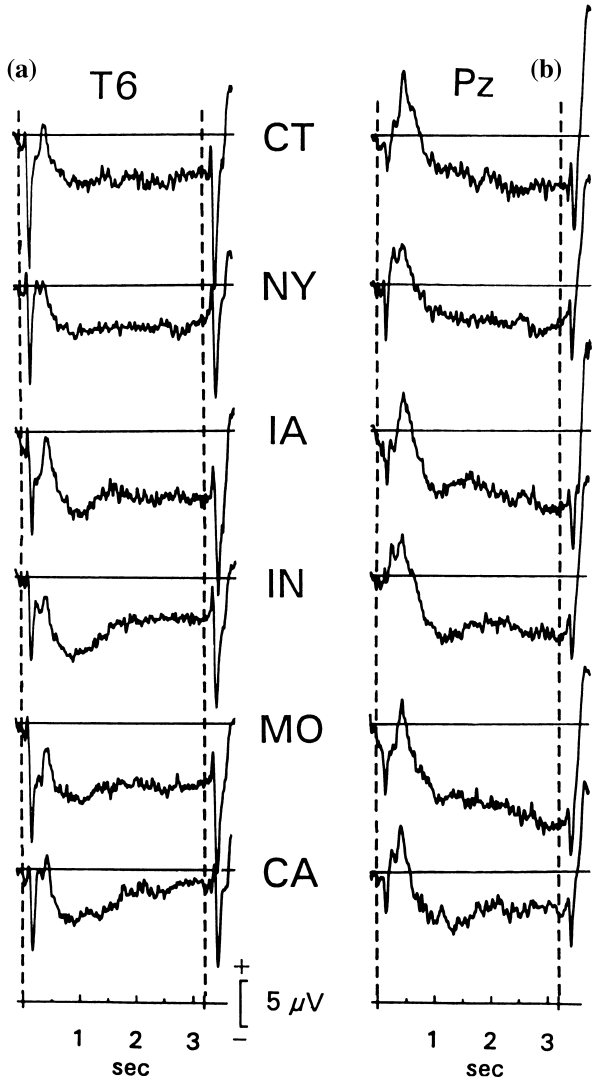

Fig. 6. Plots of the CNVs from the T6 (a) and Pz (b) electrode sites for each of the six contributing laboratories, illustrating the associated degree of waveform variability.

the findings from the visual-spatial condition reported by Ruchkin et al. (1992), who also obtained an early occipito-temporal component followed by a parietal dominant wave. In that study the amplitudes of both were dependent on the memory load, which suggested that this posterior distribution reflected the imposition of a visual-spatial memory demand. The early occipito-temporal component was hypothesized to be related to encoding processes, in accord with evidence for the involvement of the underlying cortex in object identification and recognition processes. The subsequent parietal dominant component was thought to reflect more general maintenance rehearsal processes.

The present data confirm these observations and extend them to abstract geometric forms that were expressly developed to be devoid of any readily discernible semantic or phonological properties. One important difference is that, in

contrast to the findings of Ruchkin et al. (1992) and others (e.g. Barrett et al., 1988; Barrett and Rugg, 1990; Howard et al., 1992), no evidence of hemispheric asymmetries was found in the present study for either the Early or Late CNV components. This lack of asymmetries is noteworthy given the large number of subjects ($n = 90$) upon which the present report is based.

It was also observed that the CNV showed substantial inter-laboratory differences in overall amplitude, although not in general biphasic form or in topographic representation. The amplitude variation among laboratories was close to two-fold for both Early and Late CNV measures and the variability within some laboratories was considerable. This degree of variability has obvious unfavorable implications for studies involving group comparisons. A practical implication relates to the sample sizes needed to detect group effects. Analyses of power (Cohen, 1988) were conducted separately for the Early (at T6) and Late (at Pz) CNV components, based on variability estimates derived from the total sample of $n = 90$ subjects. These analyses indicated that fairly large sample sizes would be needed to detect a modest group difference of 2 μV (with an alpha level of 0.05 and power of 0.80): $n = 40$ subjects per group for the Early component and $n = 60$ for the Late component.

Several sources of variability may conceivably contribute to the observed inter-laboratory differences. One source could be differences in experimental apparatus, procedure or laboratory culture. This is seen as unlikely, given the degree of rigor with which these factors were standardized in the COGA project and the absence of appreciable inter-laboratory differences for performance (RT) measures in this task or for other measures including visual (Cohen et al., 1994) and auditory P300 (Alexander et al., 1994) and N400 (Kuperman et al., 1995).

A more likely explanation lies in differences in subject sampling at the different laboratories, which may in turn derive from pronounced individual differences in the abilities tapped by the spatial memory task. The CNV has been the subject of prior studies of inter-laboratory consistency in both control and patient subjects, which have found much higher degrees of agreement (Abraham et al., 1980; Timsit-Berthier et al., 1984; Verhey et al., 1986). These prior studies, however, used simple, non-discriminative tasks, with short foreperiods under which the CNV measure was based on a homogenized representation of the individual components (Rohrbaugh and Gaillard, 1983). Howard et al. (1992) have emphasized the importance of individual differences in determining the amplitude and asymmetry of the CNV recorded in a visual-spatial memory task, as is attested to by their findings of strong relationships (correlations as high as 0.75) with sex, performance and personality variables. As such, the visual-spatial CNV may provide a rich source of phenotypic variability, which may be useful for genetic studies such as COGA, but which imposes rather severe constraints with respect to the evaluation of group differences. Such findings argue against any casual or uni-dimensional ascertainment strategy and they emphasize the need to obtain comprehensive assessments of subject characteristics until such time as the sources of individual variation are understood more thoroughly.

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