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CHAPTER 4

Very Slow Brain Potentials Relating to Expectancy: the CNV

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INTRODUCTION

SINCE THE publication by W. G. Walter and his colleagues (1964), in which the contingent negative variation (CNV) was initially reported, there has been a steady growth of interest in this phenomenon. Grey Walter's findings were confirmed and extended in several laboratories. The clinical applications of such techniques are now being explored.

Dr. Walter's group reported that the effect consists of a slow shift in the average baseline potential that is correlated with conditional expectancy and thus represents a cerebral response in the Pavlovian sense. They hypothesized that the CNV is a shift in the apical cortical dendritic potentials in the direction of depolarization that "primes" the cortex for action and that reducing the excitability threshold facilitates cortical responsivity, with the result that the efficiency of overt activity is increased (Walter, 1964b).

In this paper, I shall review studies of "steady" cortical potentials, sometimes referred to as dc shifts or very slow potential changes. This includes phenomena with a latency of 200 to 300 milliseconds and a duration of 0.5 second or more. There is no implication of a dc generator as a source of a steady potential between the surface of the brain and a neutral reference. I am dealing with activity slower than delta waves (1 to 4 Hz). Sensory evoked responses (ER) have rapid primary components with a latency of about 50 milliseconds, later secondary components with a latency of 100 to 200 milliseconds, and often "slow" components from 200 to 500 milliseconds with variable durations.

There has been interest in the study of baseline changes since the development of stable, high-input-impedance dc amplifiers. Caspers

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(1961) concluded from his investigations and those of others that the steady-potential gradient between the surface of the brain and an extracerebral reference electrode is built up in the upper cortical layers in the apical dendritic network. He hypothesized that the slow changes in potential are synchronized in large numbers of neurones. Clare and Bishop (1955) demonstrated that dendritic excitation does not conform to the "all or nothing" principle of axonal discharge. Accordingly, either direct electrical stimulation of the cortex or neuronal stimulation from ascending fibres would cause a negative dc shift at the cortex. Goldring and O'Leary (1951, 1958) have demonstrated actual shifts in the dc level of the cortex using both electrical and physiological stimuli. They have recorded long-lasting slow potential shifts during spontaneous spike discharges of epileptogenic foci (O'Leary and Goldring, 1960). Kohler et al. (1952, 1955a,b, 1957) demonstrated a slow potential shift that accompanied prolonged visual and auditory stimuli in cats, humans, and monkeys. They found the maximal negative shift at the vertex rather than near the primary sensory projection areas as they had expected. Contrary to results with animals in which they found a steady negative potential shift over the visual cortex, they found in humans a positive potential because they used an electrode at the vertex as the reference lead and the occipital position as the active lead. Since we have found the vertex to be the area of greatest negativity of the CNV and it is often the position for recording the maximum amplitude of the secondary components of ER, it is the least neutral area that could be chosen for the electrical reference.

Caspers (1961) demonstrated a slow shift in rats in the negative direction in connection with locomotion, exploratory behavior, alerting, and orienting behavior. Grooming behavior, on the other hand, was accompanied by a positive shift. He found the shift to be nonspecific to the type of stimulation and maximal in the central and frontal areas of the rat's brain. It related to increased firing of cell units in the reticular system. The steady potential shifted to positive polarity when the animal's alertness decreased in the transitional stage between waking and sleep. Arousal from sleep was accompanied by a shift in cortical activity in the negative direction.

Rowland (1961) reported slow-potential shifts in cats after a conditioning signal. It was initially positive and then shifted to negative during a 10-second application of clicks that signalled that electric shock was to follow. He measured negative shifts of 300 to 500 μV, lasting up to 30 to 70 seconds, and demonstrated the extinction of the response during nonreward trials and also a swing from negativity to positivity after several such trials. The maximal negative shift occurred in the early acquisition trials, and the positive shift in nonrein-

forced trials appeared after only a few extinction trials. He also showed that the degree of negative shift was related to drive-induced states caused by food deprivation and feeding reinforcement (Rowland and Goldstone, 1963). Somewhat similar slow potential shifts in rats caused by electrical reinforcement following conditional signals were reported by Wurtz (1966).

A potential shift in humans preceding a voluntary motor response by a half second was reported by Kornhuber and Deecke (1965). They termed that slow negative potential the "readiness potential" and found it to be bilateral, but maximal on the contralateral side to the responding limb. This was also observed by Gilden et al. (1966) who described its distribution and amplitude, attributing it to a generator in the Rolandic area corresponding to the neural area involved with the initiation and control of a voluntary movement. The readiness potential will be related to the CNV in the discussion.

DESCRIPTION OF THE CNV IN HUMANS

The original experimental paradigm that was utilized in the early work of Grey-Walter et al. and subsequently adopted in many other laboratories, involved a first, or conditional, signal (S_1) such as a click, a constant delay of 1 second or more, and then a second or imperative stimulus (S_2) such as a series of repetitive flashes to which the subject responded by pressing a button. The development of the CNV in such a paradigm is shown in figure 4–1. The baseline measure of potential is established to each of the signals when presented alone, and then when paired, no change in slow potential is seen in the interstimulus interval. With instructions to press the button, a slow wave shift of about 20 microvolts at the vertex is seen arising in the interval just after the ER to S_1 and ending with the ER after S_2. Averages of ten to twelve 4-second intervals are commonly taken to enhance the signal-to-noise ratio in order to make the electrical response clearly visible. Figure 4–2 shows the CNV in a series of single trials in a subject with a high-amplitude CNV that is seen clearly without averaging.

METHODS OF RECORDING

We have recorded the CNV in this basic paradigm in more than 100 normal individuals of college age and adults and also in about 90 children. The CNV response is present in practically all normal and cooperative adults. In the few cases of failure, we have observed that the recording equipment or the experimental procedures were faulty.

Presently we record the EEG on an Offner type TC EEG recorder with the time constant altered to either dc or 8 seconds. In working with children, however, we find it sometimes necessary to record

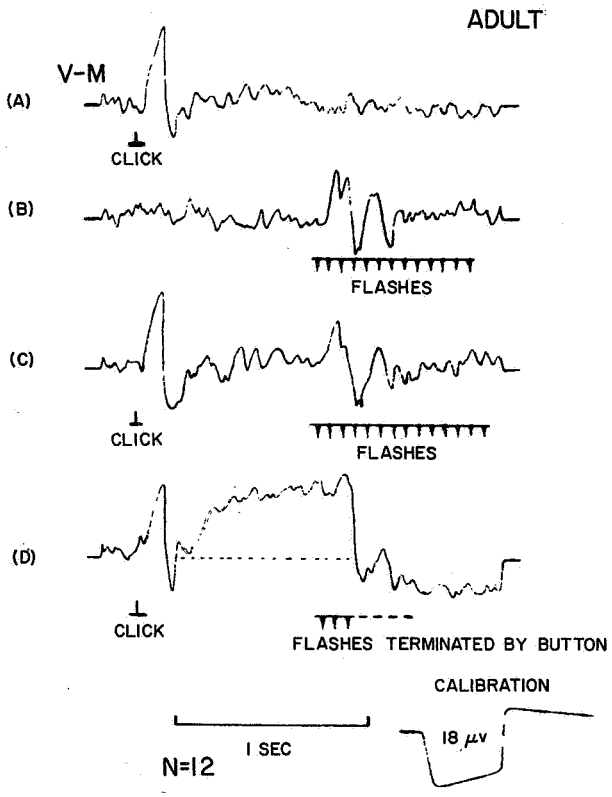


FIGURE 4–1.—Development of the CNV as a conditional response. (A) Vertex response to click alone, (B) response to series of flashes, (C) response to click followed by flashes, and (D) CNV appears as slow negative wave following the click when subject presses button to stop flashes.

with a 1-second time constant to obtain sufficient stability. Silver chloride disc electrodes are attached to the skin with collodion after the cleaning of the skin with ether and application of conducting jelly. The resistance is reduced to 3 to 4 kilohms, and the offset potential between electrodes is minimized by keeping them shorted together in saline solution when not in use.

The physiological stimulus and response data are recorded on an 8-channel P.I. Co. magnetic tape recorder; two channels of data are monitored on the CAT averager on line. After the experiment, all data channels are analyzed by the CAT and written out on a plotter from which data are measured by hand.

REVIEW OF RESEARCH ON THE CNV

After the initial discovery by Walter's group of the CNV as the electrical correlate of expectancy, related work has been done in other laboratories. Cohen and Walter (1966) found that the CNV is seen in anticipation of a pictorial presentation with no overt response on the part of the subject as well as when an operant response is required. There have been many attempts to define the psychological process as associated with the CNV. Cohen and Walter take the view that it relates to the psychological state of "expectancy" and have sometimes used the term "E Wave" as an interchangeable term for the CNV.

Irwin et al. (1966b) studied the CNV as a function of motivation and reported that the amplitude of the response is subject to attitudinal effects. Chiorini (1966) demonstrated the CNV in cats during the acquisition of a conditioned avoidance response; however, in humans, Irwin et al. believed that the motivational aspects of the response are more significant than the conditional expectancy.

Low et al. (1966a) demonstrated the CNV in humans as a conditioned response. They considered conation as the important part of the mental state relating to the response and suggested the term "conative negative variation" as more appropriate. They also reported the scalp distribution of the CNV and assessed the possible role of eye movements as its origin. Low et al. (1966b) were the first workers to record the CNV in monkeys as a conditional response in a paradigm similar to the one used with human subjects. The animals pressed a lever to terminate a shock as the second stimulus, following an auditory stimulus. Discrimination training showed a negative shift follow-

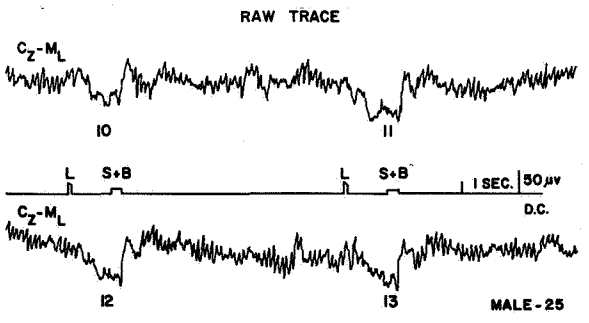


FIGURE 4–2.—CNV in original record. The vertex-to-mastoid record is negative down, showing a high-amplitude CNV and alpha blocking in response to a light flash L, followed by a sound and terminated by a button S + B.

ing punitive reinforcement trials and almost no slow response to a stimulus that was not reinforced by shock, so that no motor response was made after sufficient training. Cant and Bickford also reported the presence of CNV in a monkey in an avoidance conditioning paradigm (personal communication). Even in animals the CNV appears to be stable only during operant conditioning, when the subject is prepared to respond to the stimuli and not in cases where it must passively endure reinforcement. Cant and Bickford (1967) also observed the CNV in humans and found that its amplitude is related to changes in motivational level. Hillyard and Galambos (1967) produced similar results to the work of Walter in demonstrating the CNV as a brain conditional response. They also demonstrated a relationship between the average amplitude of the CNV and the rapidity of response in a reaction time experiment with a constant foreperiod.

The current work in this area represents elaborations of the original paradigms to include a variety of stimulus response sequences of greater complexity. Walter et al. (1967) telemetered EEG from free-ranging human subjects in order to study brain responses in a naturalistic, unrestrained setting. The CNVs recorded from four subjects by telemetry were identical to those recorded under restraint. A rubber ball thrown to the subject on the first signal produced a CNV that bore close resemblance to the trajectory of the ball. Records were made while subjects were sitting, walking, talking, riding a bicycle, and affected by distracting activities.

Walter (1967) found the CNV to be similar whether the stimuli were presented to the subject at random time intervals or whether the subject initiated the presentation of stimuli by pressing a button to start the procedures himself. When he pressed a "start" button, the action was preceded by the "readiness potential" already described, or as Walter calls it, the "intention wave." This wave merges with, and becomes the CNV before the flashes, which the subject stops either by pressing a button or producing a negative shift above a trigger threshold to stop the stimuli. In this case, the physiological response controls the stimuli rather than an overt action, termed by Walter, "autostart" and "autostop." The temporal relationships of the CNV to different delay intervals have been reported both by Walter et al. and by Irwin et al.

The temporal course of the CNV is found to have a similar pattern by all of the workers who used similar experimental paradigms. The time course of the response relates specifically to the temporal relationships of the S–R intervals. The CNV is maintained until the S_2 and its reaction. McAdam et al. (1969) found that maximal average amplitudes are significantly greater for 0.8 and 1.6 seconds than for 1.8-second intervals between stimuli. The point in time at which

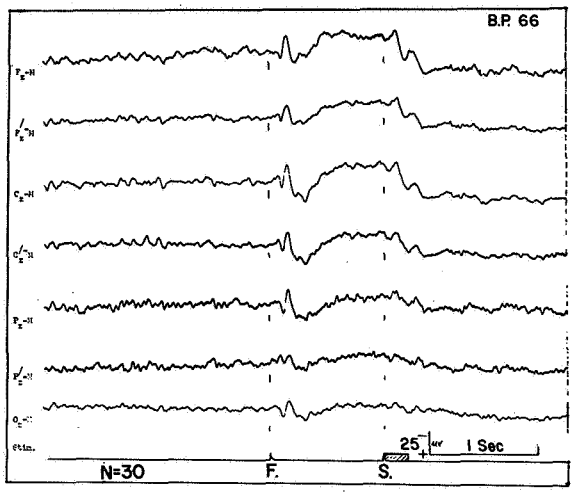


FIGURE 4–3.—A–P distribution of the CNV in a normal subject. Average of 30 trials of dc recording from six monopolar leads referred to mastoid. Positions are standard and intermediate locations from the midfrontal to the midoccipital.

maximal amplitude is reached depends on the time at which the response is to be made; if the interval is broken up into quarters, the growth of the CNV by quarters of the total interval between S_1 and S_2 is similar for the different intervals. The rise time for the shortest interval is the fastest. McAdam (1966) showed that the CNV developed during trials of time estimation and that it was maximal in amplitude during acquisition trials, decreasing during later practice trials. (See figs. 6–23 and 6–24.)

Irwin et al. (1966b) varied the amount of effort needed to press a bar for operant response. They found the CNV to be significantly larger when it was necessary to exert 14 pounds of force than 2 pounds.

DISTRIBUTION OF THE CNV

The combination of multichannel tape recorders and averagers or other specialized computers allows responses from several scalp positions to be compared simultaneously. We have recorded from all of the standard electrode positions of the 10–20 system in order to compare the CNV response from various locations. Figure 4–3 shows the anterior-posterior distribution of the CNV in an adult. Large—but consistent—individual differences can be observed.

In agreement with Walter (1964b), we found that the maximal amplitude is usually seen at the vertex lead, with a mastoid reference.

Walter thought the response to be mainly frontal and, in some subjects, to sweep back in time from the frontal pole, reaching its peak at the vertex. We were able to establish normative spatial distributions of the CNV which provide a baseline for the study of development in children and also pathological changes by recording from all of the standard positions during several sessions.

About 20 subjects were tested using bipolar combinations of leads from both standard and nonstandard electrode placements in the A–P line, as well as across the head in the standard coronal positions, M1–T3, T3–C3, C3–Cz, etc. Bipolar recording permits use of higher gain, and some of the movement and skin artifacts common to wide areas of the scalp are reduced. Measurements from bipolar leads are comparable to the unipolar data cited in the last figure. Figure 4–4 shows the distribution of the CNV in seven channels of bipolar combinations in a normal subject, with the maximal amplitude of the CNV at the vertex. The amplitude peaks a bit earlier in the frontal leads, indicating that it is not always a standing wave but that it may move from the front to the center of the head confirming Walter's observation. The maximal posterior gradient usually is seen between the vertex and the Pz position, and the maximal anterior gradient lies between the frontal and frontal pole position.

We have tried many reference positions and found none that was sufficiently neutral to be more stable for slow components than the mastoid leads. Most of our left and right comparisons were made to the linked mastoids to provide a common lead for both sides since one mastoid may be more active than the other. On many runs we have used a mastoid lead linked to a lead just over the middle of the brow of one eye through a potentiometer, so that we could compensate for eye movements in the vertical direction as was suggested by Walter. The resistance between the two reference leads is adjusted so that the contribution of voltage induced by an eye blink is sufficient to cancel the blink artifact at the vertex electrode. This also partially reduces the blink artifact to the frontal leads. One argument for continuing to use the mastoid reference is that the data from many laboratories will continue to be comparable.

We have found the average maximal amplitude of the CNV at the vertex in 60 young adults to be 21.4 microvolts with a standard deviation of 4 microvolts. This is in close agreement with Walter (1964b) and with Low and his colleagues (1966a). CNV below 5 microvolts is very difficult to detect with our averaging methods against the background activity; a CNV of 35 microvolts is the largest value measured in my laboratory. The amplitude seems to be fairly stable if the same situation is repeated. Subjects who were retested during two sessions separated by 2 to 8 days had a product moment correlation of 0.8

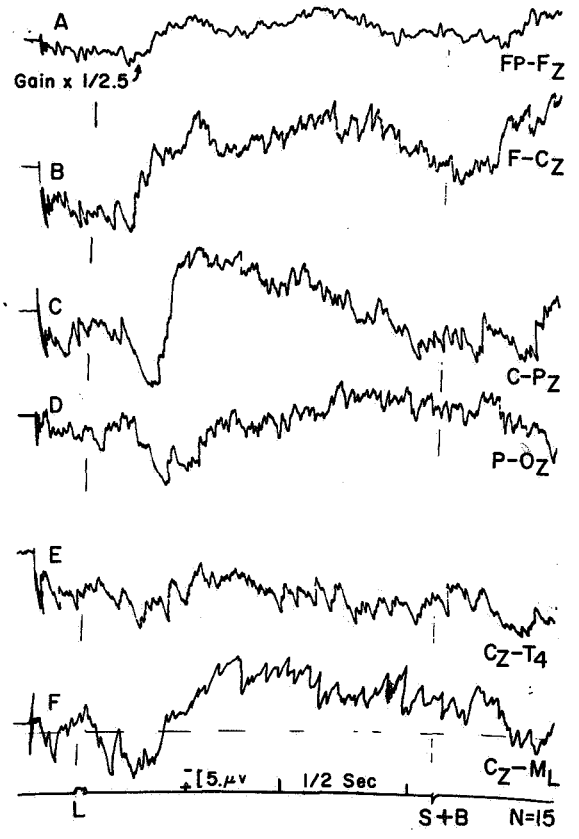


FIGURE 4-4.—Bipolar recording of CNV. The first four traces are a standard midline bipolar run. The trace E is vertex to a midtemporal lead, and F is a vertex-to-mastoid lead, indicating the shape of the CNV.

between average maximum vertex CNV on the test and retest trials (N=34).

The amplitude of the CNV is reduced in the frontal positions, more so in the parietal positions, and is minimal in the occipital and posterior temporal positions. It is quite small also in the frontal pole positions, which is evidence against its generation by the electrical field of the eyes, or even its being primarily a frontal lobe phenomenon. The CNV is reduced fairly symmetrically as the transverse distance from the vertex is increased, reaching a maximal gradient between the mid-Rolandic and midtemporal positions. There is still a considerable CNV in the midtemporal positions, but using the mastoid references probably minimizes its amplitude because of the small separation of

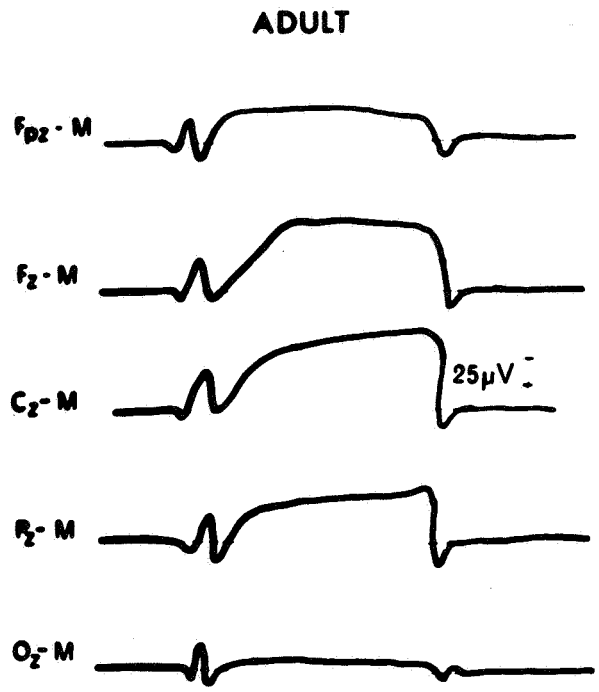


FIGURE 4–5.—Typical A–P distributions of the CNV in average adults.

the active and reference leads. The amplitude in the anterior temporal positions is quite minimal but greater than in the posterior temporal leads. Figure 4–5 shows the amplitude at the various standard lead positions (see Table I).

Low et al. (1966a) presented data comparable to ours in 30 subjects in a similar S–R paradigm. They observed an earlier maximal amplitude in frontal regions and a later maximal amplitude in posterior regions. They also confirmed Walter's and Cohen's observations that the modality or intensity of the stimulus has no significant effect on the CNV. The decrease of CNV amplitude with distance from the midline is comparable to our findings. The distribution in the A–P longitudinal plane is not in complete agreement although the average amplitude at the vertex is the same. Their study shows an almost linear reduction in amplitude from about 23 microvolts in the frontal pole position to about 19 microvolts in the occipital-parietal position, while we obtained an average maximum of about 12 microvolts in the frontal pole position and 9 microvolts in the midoccipital position.

TABLE I.—*Spatial Distribution of Maximal Amplitudes of the CNV**

	FP_1	FP_z	FP_2	
	11. 3	12. 1	11. 9	
F_7	F_3	F_z	F_4	F_8
12. 3	16. 3	18. 7	15. 5	12. 9
T_3	C_3	C_z	C_4	T_4
14. 2	17. 7	21. 4	18. 3	14. 8
T_5	P_3	P_z	P_4	T_6
7. 4	12. 2	16. 6	13. 7	8. 3
	O_1	O_z	O_2	
	8. 1	9. 3	7. 6	

*Entries are the means of average maximal amplitudes of the CNV in microvolts in 60 adult subjects. Standard deviations ranged from 2.4 to 3.8 at different positions.

FORMATION AND MORPHOLOGY OF THE CNV

The form of the CNV is characteristic of both the individual subject and the experimental parameters as shown in figure 4–6. About 40 percent of the adults tested produced a ramp-shaped CNV as in (A), and about 33 percent produced a rectangular CNV as in (B). The other 27 percent were divided among the mixed or atypical shapes remaining. In some individuals, the CNV remained for a short time after the imperative signal, or it dropped for about 0.1 second and then resumed a marked negativity, returning only gradually to a baseline within 1 or 2 seconds. This is an identifiable effect that has been called "rebound" by Dongier's group in Belgium (Bostem et al., 1967).

When a novel stimulus is first presented, there is in addition to the sensory evoked response an indication of the alerting or orienting response. The alpha rhythm is blocked; there may be a decrease in skin resistance or change in skin potential, and a slow negative potential shift lasting 300 to 500 milliseconds and about 10 to 15 microvolts at the vertex often occurs. This is best seen with stable electrodes in a small number of trials in the average. This response becomes habituated rapidly unless the stimulus is given significance by its association with a response; in this case, it blends with the "intention" wave, which then remains as long as the person voluntarily responds.

If the first stimulus is a click and the click is followed by an imperative stimulus such as flashes, the slow negative potential blends into the early part of the CNV by rapidly lengthening in duration and increasing in amplitude. At that stage, it is impossible to separate the brain response of orienting and conditional expectancy since psychological expectancy is a consequence of novelty. The CNV is acquired fairly rapidly as a conditional response. Figure 4–7 shows the percentage of subjects reaching their maximal CNV amplitude as a function of

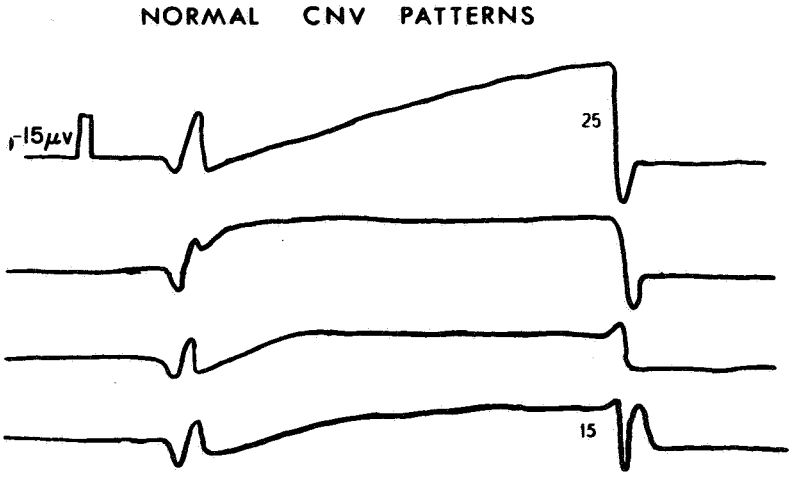
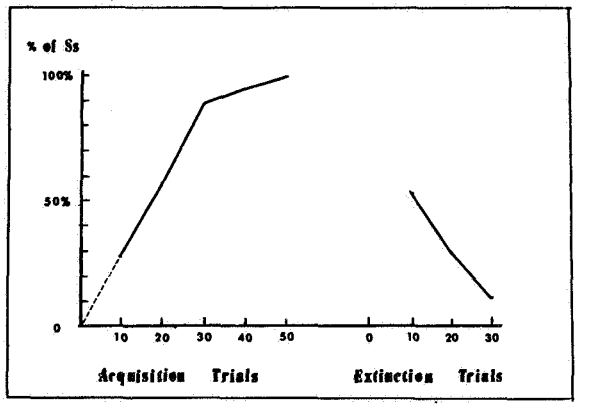


FIGURE 4–6.—Typical patterns of the CNV in normal subjects. A calibration pulse is shown on the top trace, and the CNV is shown between the evoked responses to the first and second stimuli. The numbers represent the usual range of amplitudes.



No. of trials to reach maximum CNV and % of Ss whose CNV is above 10 μvolts during Extinction.

FIGURE 4–7.—Mean CNV during acquisition and extinction trials.

number of trials when the subject pushed a button to terminate a tone (S_2) as quickly as possible after the onset of the tone. First, 25 trials of tone alone—without a conditional signal—were presented in order to establish the baseline of reaction time, and then the conditional signal of a single light flash was introduced 1 second before the tone. With anticipation of the response, the average reaction time

shortened from 360 milliseconds to 190 milliseconds in the last set of trials. Over one-half of the subjects reached their maximal CNV within the second set of ten conditional trials. The CNV stayed near its maximal level indefinitely as long as the experimental conditions remained constant. Several hundred trials were presented to two subjects at single sessions lasting over 3 hours with no appreciable change in CNV amplitude as their motivation and responsivity continued at a satisfactory level.

RELATION OF THE CNV TO OTHER BRAIN POTENTIALS

The maximal amplitude of the ER to light flashes and sounds was measured for each subject from repeated presentations of the stimuli alone at 4-second intervals and from presentations during CNV trials. There is a tendency for subjects with large ER also to have large CNVs; however, many subjects with low-amplitude ER also have high-amplitude CNV. Several examples of the various types of relationships are shown in figure 4–8. The maximal average amplitudes of the ER derived from the vertex to mastoid are compared with CNV amplitudes from the same derivation. The peak-to-peak deflection of the

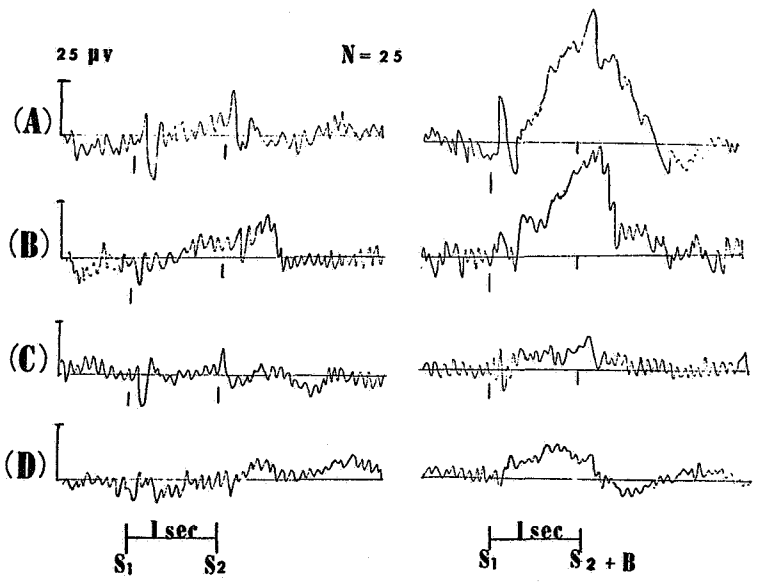


FIGURE 4–8.—Comparison of evoked response and CNV amplitudes. The left-hand traces show averaged records when the subject makes no response to S_2, and the right-hand traces show the CNV when the subject pushes a button to end the S_2. (A) shows a subject with high-amplitude ER and CNV, (B) a subject with small ER, but a large CNV, (C) a subject with large ER and a small CNV, and (D) a subject with both small ER and CNV.

highest negative to the highest positive potential of the ER components within about 300 milliseconds of the stimulus is taken as the maximal amplitude.

The ER amplitude to light flash varied from 6 to 19 microvolts with an average of 12.4 microvolts and a standard deviation of 2.6 during trials when the flash had conditional signal value. During flash presentations alone, the mean amplitude of the AEP was 10.7 microvolts with a standard deviation of 2.4 microvolts. The hypothesis that the mean amplitudes under the two conditions are not different was rejected, using a t test at the .05 level of significance. The increase of the ER with significance of the signal agrees with the finding of Sutton et al. (1965a) in which the amplitude of an ER is increased as stimulus uncertainty is increased and the subject guesses what stimulus is going to occur.

The product-moment correlation of the mean amplitudes of CNV and ER to flash as a conditional signal for 60 subjects is 0.43, significant at the 1-percent level. The greatest response to flash did not always occur at the vertex lead, and the correlation might have been higher if more posterior leads had been considered; however, a uniform lead position seems advantageous.

The ER to S_2 is usually obscured by the CNV, and no accurate measurements of amplitude may be made. Often the negative peak extends beyond the CNV, but it is not quantifiable since the neutral baseline is not known. We did not present a sound stimulus as a first stimulus to a sufficient number of subjects to make a meaningful correlation of its amplitude. Observation so far, however, indicates that results are similar to the flash response. The amplitude seems a little larger when the signal is given conditional significance, and it bears some relationship to the amplitude of the CNV.

The latency of the CNV is difficult to measure since it develops out of the complex secondary ER to the S_1. It seems on the average to begin within 200 to 400 milliseconds after the onset of the S_1 and to reach its peak within 400 to 900 milliseconds after the S_1 when the interstimulus interval is 1 second. The latency and delay time to peak value are of course related to the characteristic shape of an individual's CNV with the "rectangular" shape leading to shorter latencies and a rapid rise to the peak amplitude.

Visual inspection suggests that the average latency for a 1-second interval is 260 milliseconds and 295 milliseconds when the subject is conditioned to expect a 2-second delay. The difference in time to reach peak amplitude is more marked. It took an average of 820 milliseconds to reach the peak with a 1-second delay and an average of 1530 milliseconds to reach peak amplitude with a 2-second delay, a significant difference at much below the 1-percent confidence level.

The motor potential—or the intention wave as called by Walter (1967)—mentioned earlier is thought by some to bear a relationship to the CNV. We have not yet looked systematically at that variable; however, in a few subjects tested, there seems to be no strong relationship. It has a more restricted distribution, is much smaller in amplitude, and is found by Vaughan et al. (1968) to be bilaterally asymmetrical. The strongest evidence against it as a possible basis for the CNV is that no overt motor response is necessary to elicit a CNV; CNV to a variety of S–R acts are quite similar indeed, even when the response is subjective or ideational. At first, Walter thought the CNV to be a cortical priming response preparatory to making a voluntary action, but he now conceives the CNV as related to the psychological state of expectancy (Walter, 1965b). It can be elicited by the expectancy of almost any discrete event in time that bears significance for the subject. It is convenient for research purposes to endow significance by asking the subject to press a key when a stimulus occurs. If the key press becomes a passive act, the CNV amplitude is reduced. If the act is given operant significance by controlling something in the situation, then the CNV is maintained at a high level.

Figure 4–9 shows what can happen when a motor response loses its

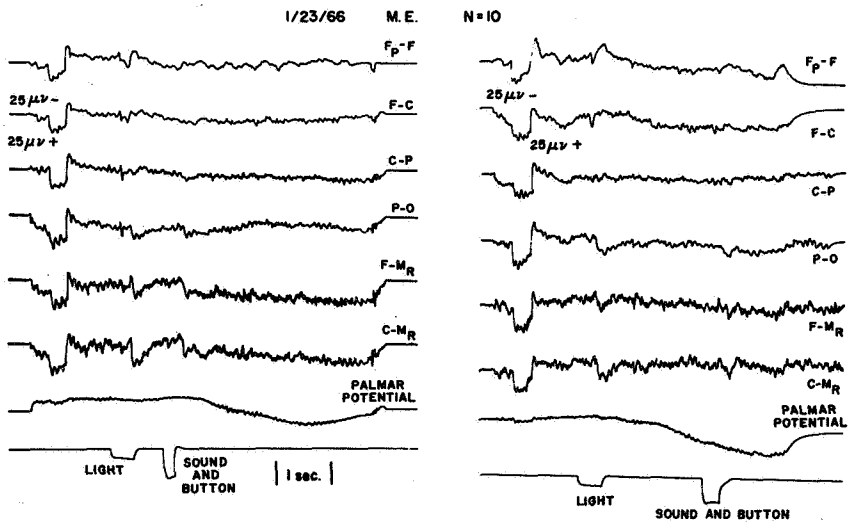


FIGURE 4–9.—Drop in CNV with change in stimulus-response conditions. Top five traces are a midline bipolar run and a frontal monopolar run. The sixth trace is vertex-to-mastoid, the next is average palm potential, and the last line is the stimulus program. The left-hand traces show a fully developed CNV, and the right-hand traces show the flattening of the CNV with increasing the interval and removing the function of pressing the button.

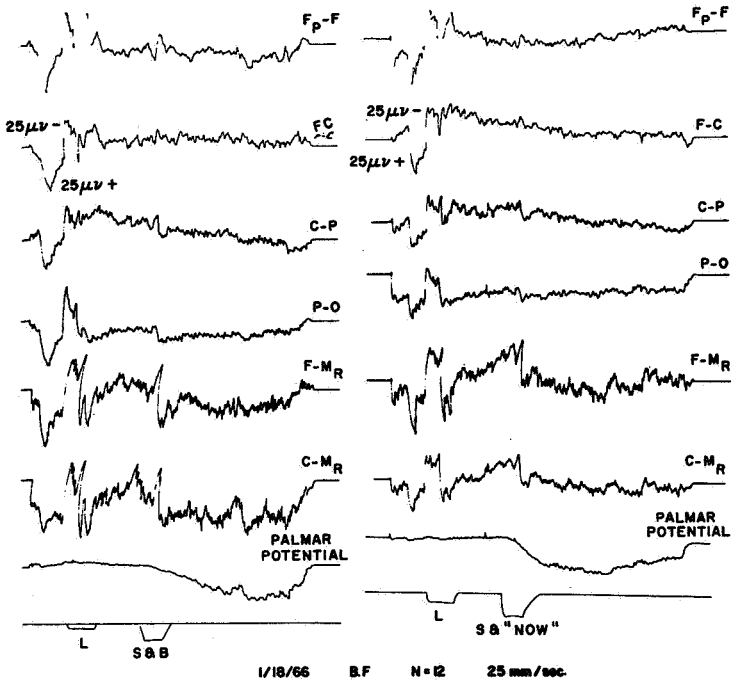


FIGURE 4–10.—The CNV terminated by a thought. The left side traces show the CNV and palm potential response in the conventional situation of flash, sound, and button. The right side traces show the CNV response in the same subject to the instruction, "Think 'Now' at the time that you would press the button, but make no movement." There is very little difference either in the CNV or the skin potential response between the two situations.

operant value. We routinely ran an experimental extinction series by presenting S_1 alone after the subject was well trained. The CNV extinguishes as the subject perceives that he no longer has any task. When the S_2 is restored, the CNV returns to its former value as the subject again expects to press the button and stop the sound. In this case, by accident, the push-button jack was loose, and the subject could not terminate the sound with the push button; although he continued to press the button to the sound, it accomplished nothing. He was no longer motivated, and the CNV remained at a minimal level.

An ideational response alone is sufficient to produce a CNV in a well-trained subject, so that instead of pressing the button to S_2, the subject is instructed to just think "Now" at the time that he normally would press the button. An electromyograph revealed no movement of his hand during the CNV response seen in figure 4–10.

The CNV also is present in a situation in which subjects expect a projected picture as the S_2; the CNV is similar whether or not he is to make an overt response (Cohen and Walter, 1966). Actually, although the CNV is a fairly generalized response, the distribution over the head is often slightly more posterior when a picture is presented than when the subject makes a motor response, as shown in figure 4–11.

The CNV is elicited when the subject responds with a word to S_2 which he freely associates to a word presented as S_1. It does not matter whether he says the word aloud or merely "thinks" the word to himself as an ideational response (fig. 4–12). In this case, the subject has a higher amplitude CNV to a verbal series than the motor reaction series of trials. This probably reflects his value system since the subject is a professional writer and is more interested in words than fast hand reactions.

SUMMARY CONSIDERATIONS

The CNV develops in human subjects as the electrical response of the brain to a conditional signal that an operant response is to be made after a delay. As illustrated, a wide variety of S–R paradigms result in the CNV in human beings even when verbal or ideational responses are made instead of overt motor acts.

The CNV varies in amplitude, shape, latency, consistency, and distribution over the head in different subjects. The experimental parameters of the stimuli may vary in relation to temporal sequences, sensory modality, prior number of trials, instructions to the subject, and in countless other ways. The electrophysiological response is related to psychological events or states of mind identified as expectancy, decision (Walter, 1964b), motivation (Irwin et al., 1966b; Cant and Bickford, 1967), volition (McAdam et al., 1966), preparatory set or conation (Low et al., 1966a), and arousal or the physiological state of excitability (McAdam, 1969).

The evidence that eye movements do not account for the CNV is conclusive. The CNV is similar when recorded from surface or intracranial electrodes (Walter et al., 1964), it has a different spatial distribution than the eye field, and it has been reported in a subject with glass eyes when no electroocular field (Low et al., 1966a) was present. Data from a subject who moved his eyes in opposite directions as an overt response and careful recording of eye position during the production of the CNV also confirm this opinion, as seen in figure 4–13. However, the fact that eye movements can simulate the CNV makes it imperative to monitor them for both experimental and clinical work.

We should consider another possible internal brain source for the CNV, that is, the generator for the motor potential (MP) described by Gilden et al. (1966), who report the maximal negativity of up to

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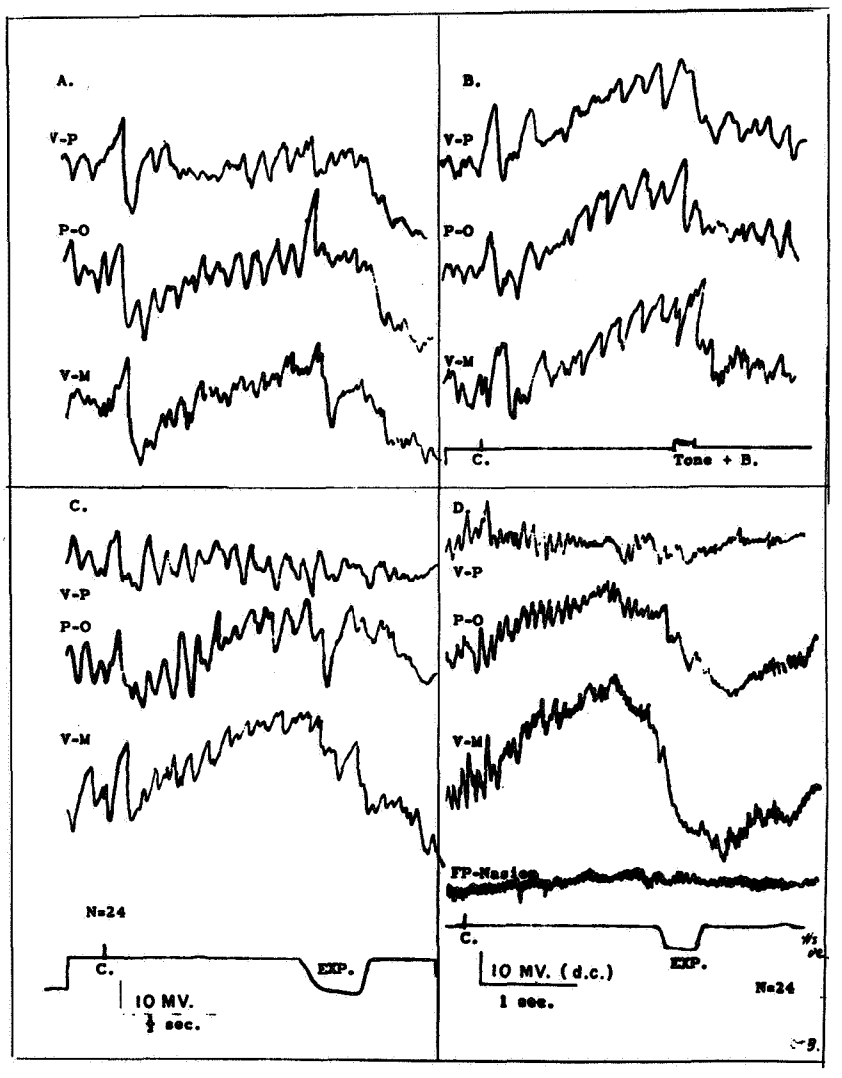

FIGURE 4–11.—Comparison of the CNV in anticipation of making a motor response and of seeing a picture; (A) and (C) are two sets of averages showing that maximal gradient of the CNV is posterior between the P and O leads when a picture is expected; (B) shows that the gradient is more anterior when the subject expects to make a motor response (same subject); (D) shows the result when the subject must press a button to make the picture appear, indicating a larger CNV than either alone. The last trace shows the stability of the leads around the eye.

25 microvolts beginning from 0.5 to as much as 2 seconds before a voluntary movement. The most compelling argument against the CNV being nothing but "motor potential" is the finding that CNVs are recorded in situations that do not involve movement such as

anticipation of meaningful visual or auditory presentations as shown in the previous figures. It is conceivable that the MP is a special instance of a CNV, with the S_1 and S_2 both being internalized, the hand movement being the response that operates to satisfy the induced set of the subject. The internalized realization that it is "now time to press the button" or make another instructed response is S_1; the initiation of the voluntary action that has been delayed until the "proper" time is analogous to S_2 in the S–R paradigm of the CNV. The electrophysiological event mediating the time between the origin of the wish and the consummation of the act is a slow negative shift, and the

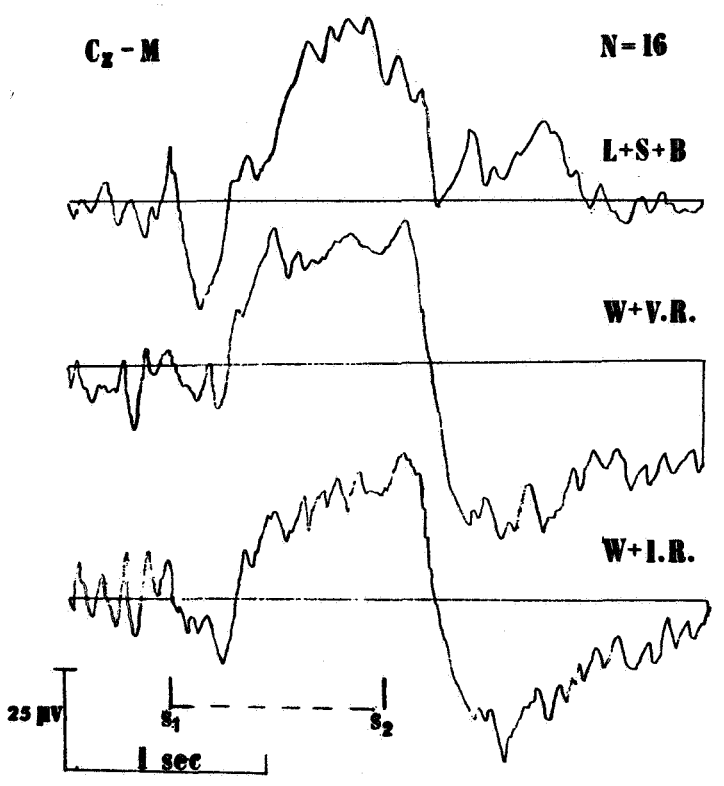

FIGURE 4–12.—The CNV to verbal stimuli.

A. The average of 16 trials recorded at the mastoid to the usual light, sound, and button trials.

B. Same subject when a word is called to the subject as S_1 and he responds aloud with an associated word as S_2.

C. Same situation, but subject merely thinks a word to the sound of S_2, making a subjective or ideational response.

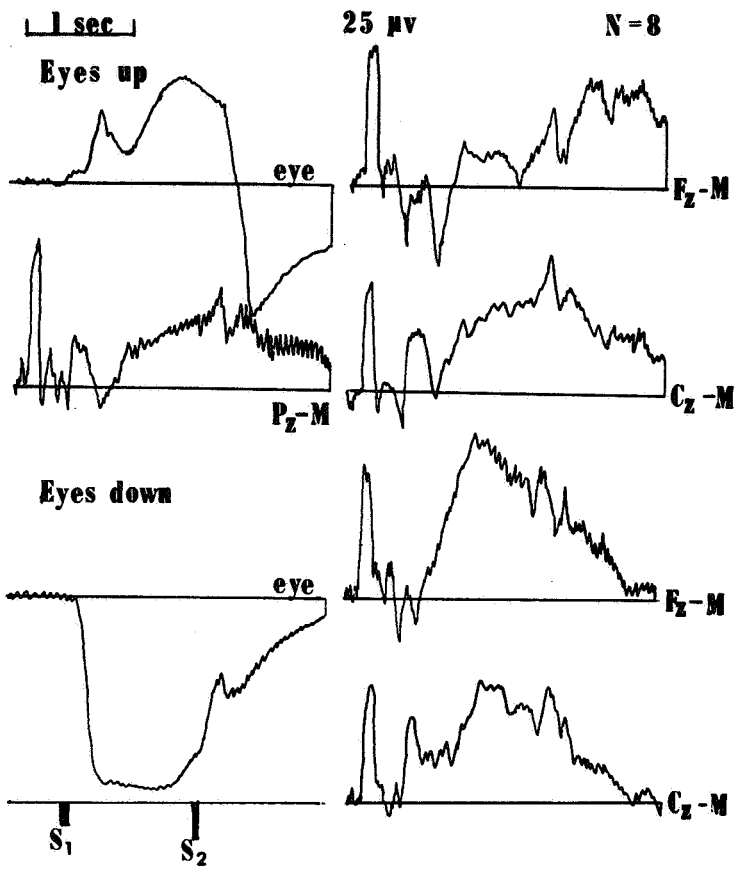

FIGURE 4–13.—Independence of the CNV from eye movement artifacts. The subject makes a voluntary eye movement response to a visual stimulus as S_1 and returns the eyes to the original position at S_2 (recorded at the Burden Inst., Bristol, England).

psychological correlate may well be expectancy or, in other terms, preparatory set.

Walter's original hypothesis that the CNV relates to efficiency of action such as shortening of reaction time because of "cortical priming" is confirmed by the other investigations. McAdam (1969) found that late components of somatosensory AEP between 200 to 400 milliseconds are shorter when the stimulus is presented during the CNV trials compared to presentation during the resting state. Other measures of levels of arousal are consistent with the hypothesis that the CNV is present, representing heightened arousal or alertness, but no change is seen in the early components of the ER.

The work on one very slow potential wave—the CNV—has been reviewed here. We are continuing our work toward understanding the physiological origins and the psychological significance of the CNV as well as exploring its clinical utility (Walter, 1966). We are now exploring a variety of psychiatric and neurological disorders and developmental problems in children. We still conceive of the CNV as the electrical correlate of psychological expectancy and prefer the generality of the term "contingent" since there is such a variety of contingencies which it can represent.

DISCUSSION

Dr. Low: The CNV certainly is contingent upon something. The question is, upon what? Is it always contingent upon the same thing? If and when these questions are answered, we may find some reliable clinical or diagnostic application for CNV studies.

Since this is supposed to be a workshop session to consider problems involved in conducting and interpreting experiments related to the study of AEP, I would like to begin this discussion of Dr. Cohen's review by emphasizing the most serious methodological problem in CNV experiments. It is obvious that eye movements can introduce a very significant contaminant into recordings of slow activity at the scalp or from the brain surface. These movements must be accounted for in any CNV experiment. In humans, this may be done in several ways. One method, described by McCallum and Walter (1968), consists of "balancing out" eye movement between the active electrode and the reference. Another simple method—and the method we prefer with cooperative subjects—is to make all recordings with the subject visually fixating a target.

In animal work, accounting for and eliminating eye movement is not so simple. We have solved the problem in two ways. The obvious method is to enucleate the eyes of the animal, as first suggested by Chiorini (1966); however, this is not always practical or desirable. Another method uses a subcortical reference electrode. This works well if both the surface and the reference electrodes are away from the anterior frontal regions, and this point will be discussed in more detail later.

The most extensive work quantifying the relationship between eye movement and the CNV in man was done by Hillyard (1968). He partitioned the CNV into two components, the Eye Artifact Potential (EAP) and the true CNV (tCNV). In experiments performed with the subject's eyes closed, the mean contribution of eye movement artifact to the total negative shift at the vertex was 23 percent over all subjects. Significantly, the EAP introduced as much variability into the vertex potential as did the tCNV. He concluded that changes re-

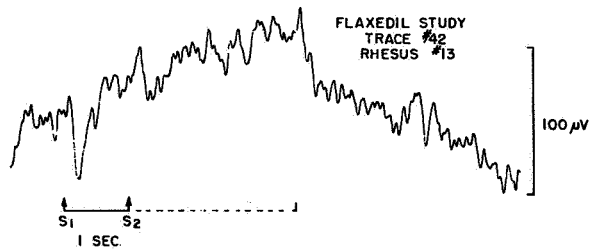

FIGURE 4.–14.—CNV recorded from a chemically paralyzed monkey. Trace is an average of 48 trials, recorded with the active electrode on frontal cortex referred to a reference in occipital bone (negative up).

ported in the CNV by other workers without controls for eye movement effects may have been determined by ocular rather than brain potentials. His data are in general agreement with those of Low (1966).

If ocular movements are excluded as the source of the CNV by proper experimental procedure, then what is the source of this potential? Our major research effort in Houston was directed towards attempting to answer this question, and I will show some of the results of this work.

A CNV-like potential may be recorded from Rhesus monkeys using a variety of stimulus-response conditioning paradigms. The simplest paradigm—and the one most closely resembling the S_1–S_2–R situation in human studies—is escape-conditioning with a warning cue. Using such a paradigm, we have recorded CNV from monkeys with and without eyes; with and without chemically induced paralysis; with cortical surface electrodes; with bone, cortical, and subcortical reference electrodes; and with intracortical, extracellular microelectrodes.

Figure 4–14 is a sample CNV recorded from a monkey that was completely paralyzed by Flaxedil. The animal had been trained using a variation of our usual S_1–S_2–R paradigm. The S_1 was a loud click, and S_2 was a 1000-Hz tone lasting 2.5 seconds, with a shock, across the feet occurring at the end of S_2. The tone could be terminated, and the shock avoided if a lever was pressed during S_2. Lever presses in the S_1–S_2 interval were punished by shock. After training to criterion, i.e., 90 percent correct trials, this animal was paralyzed and intubated, maintained with a respirator, and given a series of S_1–S_2 trials. The illustration is an "average" of 48 trials recorded from frontal cortex and referred to an occipital bone reference. The form of this potential is very similar to CNV recorded in humans without the sharp cutoff at S_2, possibly because the animal was not able to make the required response.

Figure 4–15 illustrates similar shifts recorded with an identical paradigm from a different animal. This monkey's eyes were surgically removed 8 days before this experiment. Each of the three traces is an "average" of 40 trials and is a result of cortical-cortical simultaneous multichannel recording. Frontal cortex to parietal cortex leads show a marked anterior-dominant negative shift in the S_1–S_2 interval and beyond. When the recording is from frontal cortex to sensory-motor cortex, the more anterior electrode is still recording the greater negativity. Sensory-motor cortex to parietal cortex leads show relatively little potential difference between the two, with only a small, late rise of the baseline in the S_1–S_2 interval.

Figure 4–16 illustrates the positions of the electrodes in this animal.

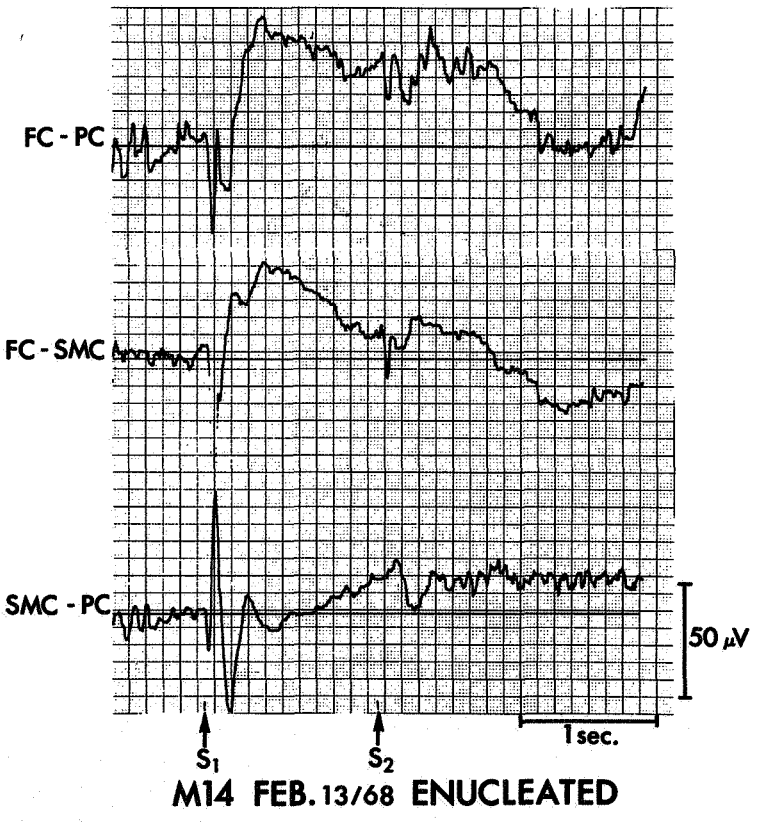

FIGURE 4–15.—CNV recorded from an enucleated animal. Cortical-cortical recording. Each trace is an average of 40 trials, and the epochs are simultaneous (negative up). FC=frontal cortical, SMC=sensory-motor cortical, and PC=parietal cortical.

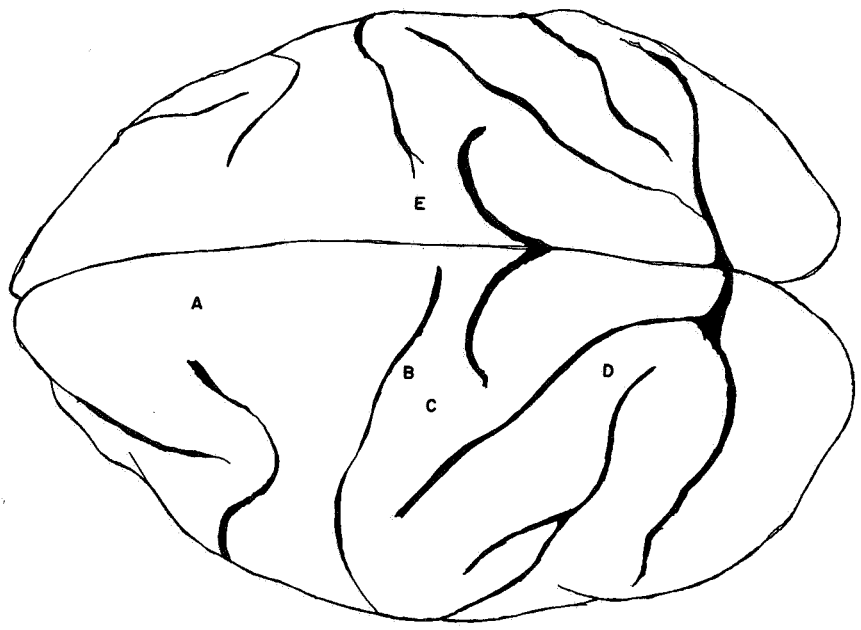

FIGURE 4–16.—Electrode positions for Figure 4–15 and Figure 4–17. A=frontal cortex, B=sensory-motor cortex, C=subcortical reference, D=parietal cortex, and E=subcortical reference.

The frontal surface electrode (A) is well anterior to the motor region. The sensory-motor electrode (B) is near the motor-arm area, not as close as we intended, but certainly closer to it than electrode (A). Electrode (D) is on posterior parietal cortex. Marks (C) and (E) indicate the insertion points of subcortical reference electrodes. For technical reasons, electrode (C) could not be used, and the subcortical reference for the following illustration was in the opposite hemisphere to the surface electrodes, a circumstance that introduced no significant variation as compared to similar recordings using an ipsilateral subcortical reference in another animal.

Figure 4–17 shows three traces, each an average of 40 trials using the same "enucleated" animal. The first trace is from the frontal cortex, the second from sensory-motor cortex, and the third is from parietal cortex, each referred to the same subcortical reference. There is an obvious early rise of the negative shift in the anterior region, with a later, slightly delayed peaking in the sensory-motor and parietal regions.

Using this same technique in the same animal before enucleation produced very similar findings except that the negative shift in the anterior lead was greater before than after the eyes were removed.

There were no significant differences noted in amplitude measurements of the CNV at the sensory-motor area referred to the subcortical reference when comparing pre-enucleation and postenucleation records.

Other recordings from three other monkeys produced similar results, i.e., a potential shift between S_1 and S_2, which was negative at the surface of the cortex with respect to subcortical reference. While this particular observation may be modified with more careful measurement, there were no apparent differences noted whether the reference

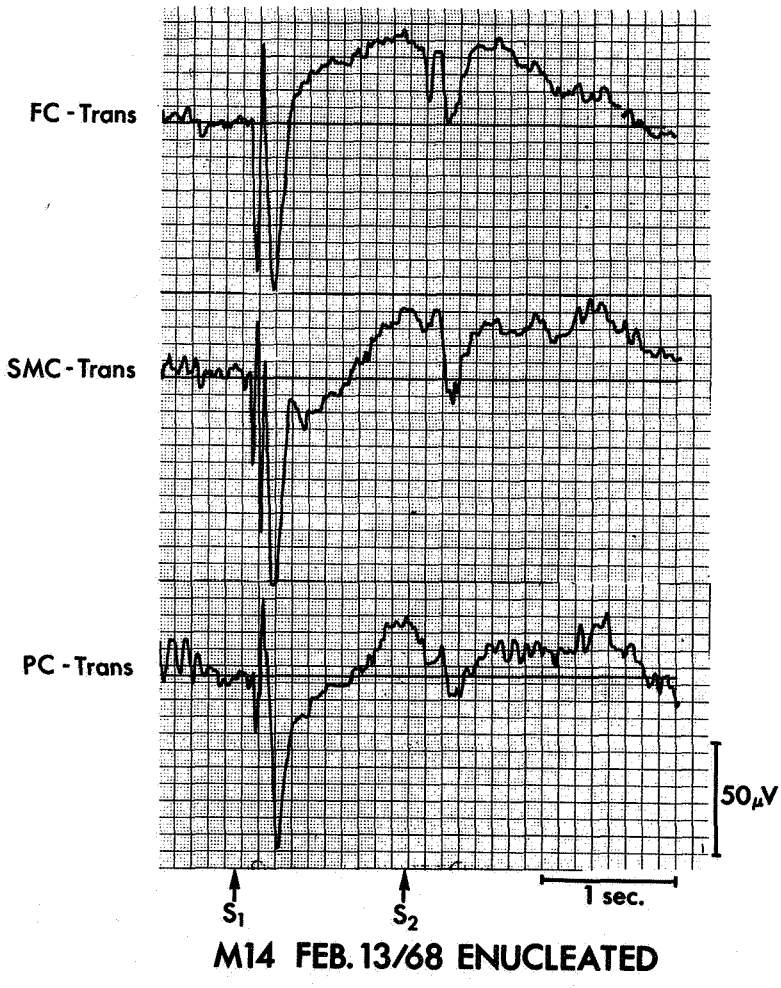

FIGURE 4–17.—CNV recorded from an enucleated animal. Cortical-subcortical recording. Each trace is an average of 40 trials, and the epochs are simultaneous (negative up). FC=frontal cortical, SMC=sensory-motor cortical, PC=parietal cortical, and Trans=subcortical.

was 3, 7, 10, or 15 mm deep, as long as the contact was in white matter. In every animal, the anterior cortex became negative first, with reference to more posterior cortex, with a later increase in negativity of posterior areas.

Whether this potential in Rhesus monkeys is the same thing as the CNV in man is a moot point. It looks very similar to the human CNV; it appears in apparently analogous situations; its distribution is similar, and we have considered it to be that which serves as the CNV for monkeys.

Regarding another point, i.e., the relationship of the CNV to arousal and/or alertness in man, we have acquired some clarifying data. By varying the intensity of S_2 around threshold, it was demonstrated (Low et al., 1967) that CNV magnitude is correlated positively and CNV variability is correlated negatively with level of attentiveness in man. The question then arose concerning whether this increased CNV magnitude is simply a reflection of generalized arousal.

Another experiment was done with nine volunteer subjects, using essentially the same procedure; i.e., the subject's threshold for clicks was determined using the Bekesy trace method. Then a series of flash-click pairs was given with the intensity of S_2 systematically varied around threshold. Twenty flash-click pairs were given at each arbitrarily chosen intensity level of S_2, and the trials were averaged in blocks of ten, giving two CNV measurements for each level. A figure called percent variance of CNV magnitude was calculated as σ/μ where σ was the difference between the two CNV measurements at any given level multiplied by $1\sqrt{2}$ (analogous to the standard deviation), and μ was the mean value of the two measurements at the same level. The CNV measurements included peak amplitude and area.

Correlations were then made between these measurements and several variables, including attenuation of S_2 in decibels. Figure 4–18 shows the relationship for all nine subjects of percent variance of CNV area to S_2 intensity. The variance is markedly lower when S_2 is at threshold than at other intensities, and the differences between the variances at T and at T+3 dB, and T and T−10 dB are significant at the 0.05 level. Pearson product-moment correlation coefficients were then calculated for S_2 intensity against CNV area, CNV amplitude (peak), reaction time, heart rate, respiration rate, and GSR reactivity. Table II shows these coefficients of correlation. There are three significant correlations, i.e., a strong negative correlation between S_2 intensity and CNV area, and positive correlations between S_2 intensity and respiration rate and S_2 intensity and GSR reactivity.

These data were interpreted as indicating that the mechanisms responsible for the increased magnitude of the CNV with increased

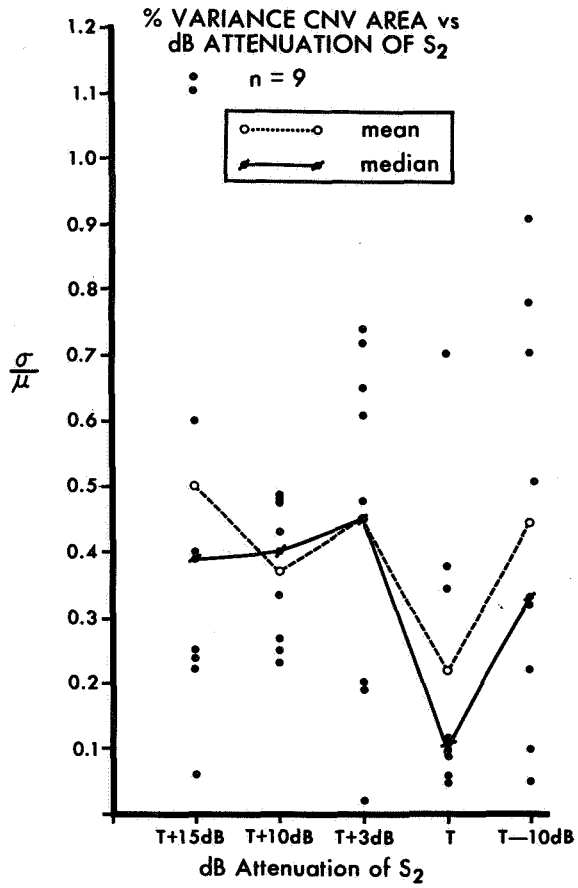

FIGURE 4–18.—Percentage value σ/μ of CNV area vs dB attenuation of S_2. See text for explanation of σ/μ. Nine subjects, 20 trials at each intensity level for each subject.

attentiveness are not necessarily part of a global, physiological arousal response.

The method of measuring the CNV is a matter of concern. It is evident that a single-point amplitude measurement, whether it is peak amplitude or amplitude at a given time after the warning signal, or before the command signal, is not alone a sufficient descriptor of the CNV. The shape of the CNV varies markedly, depending in part upon the length of the interstimulus interval (McAdam et al., 1969), and yet it is difficult to accommodate multiple measurements such as rise time, amplitudes at different points along the CNV, duration, etc.

TABLE II.—*Correlation Between S_2 Intensity and Other Variables*

Variable	r	p
Heart rate	−0. 210	0. 100
GSR reactivity	+0. 405	*0. 005
Respiration rate	+0. 323	*0. 025
Reaction time	−0. 222	0. 100
Peak CNV amplitude	+0. 176	0. 150
CNV area	−0. 612	*0. 005

*Significant correlation.

Also, it is often difficult to measure peak amplitude with accuracy because of the fast activity superimposed on the curve of the CNV.

For these reasons, we have adopted the method of measuring CNV area as well as peak amplitude. Figure 4–19 illustrates the area of a CNV as we measure it. The area is obtained as an integration of CNV amplitude as a function of time between point A and point B. Point A is the point of origin of the CNV, and B is its point of termination; both points are obtained by inspection.

Using the data obtained in the last described experiment, correlations were determined between CNV areas and S_2 intensity, peak CNV amplitude, CNV duration, reaction time, and the other measured physiological variables. Table III shows the correlation coefficients in table form. The only significant correlation was between area and S_2 intensity. There was little or no correlation between area and peak amplitude or duration of the CNV, or between area and reaction time.

It was concluded that the area measurement is a useful parameter, without which valuable information may be lost and that the area of a CNV is not necessarily a simple function of peak amplitude, duration, or reaction time.

One other point should be stressed. The exact relationship of the CNV to background rhythmic activity or to the so-called resting dc level has not yet been satisfactorily resolved. For example, we have obtained traces such as those in figure 4–20, indicating that, at least in some cases, the resting dc level of the brain moves positively as the CNV increases in magnitude, as though the CNV were momentarily returning the cortex to the zero state. Knott and Irwin (1967) have shown that low-anxiety subjects will develop higher amplitude CNV than high-anxiety subjects in a stressful experimental situation. They postulate that the cortex may have a fixed capacity for shifting negatively and that the CNVs of the high anxiety subjects "run into" this ceiling from a variable baseline, with anxiety or arousal factors af-

fecting the level of this baseline. The specific CNV generators presumably provide a transient negative rise toward the postulated "ceiling."

Low and McSherry (1968) have shown that in the usual low-stress single S_1–S_2–R paradigm, the physiological system for generation of the CNV is not saturated since the CNV magnitude may be increased by superimposition of tasks in time.

Finally, I feel less brave about making assertions regarding the psychological-physiological significance of the CNV than I once was. It may well be that what we call the CNV is not a single entity but is several different potentials with similar appearances, occurring alone or recorded together in a variety of circumstances. All negative shifts recorded at the surface of the brain do not necessarily signify the same physiological-neuronal process. Since there is no general agreement about the question of whether a cortical surface-negative potential always indicates either excitation or inhibition or some mixture of both, it would seem quite adventurous to speculate about the physiological function of a phenomenon that may not even be a discrete potential.

Dr. Lombroso: I would like to make some remarks on this subject, and I apologize if they will further add to the complexity—already alluded to—of this phenomenon. It has been stated earlier that cortical

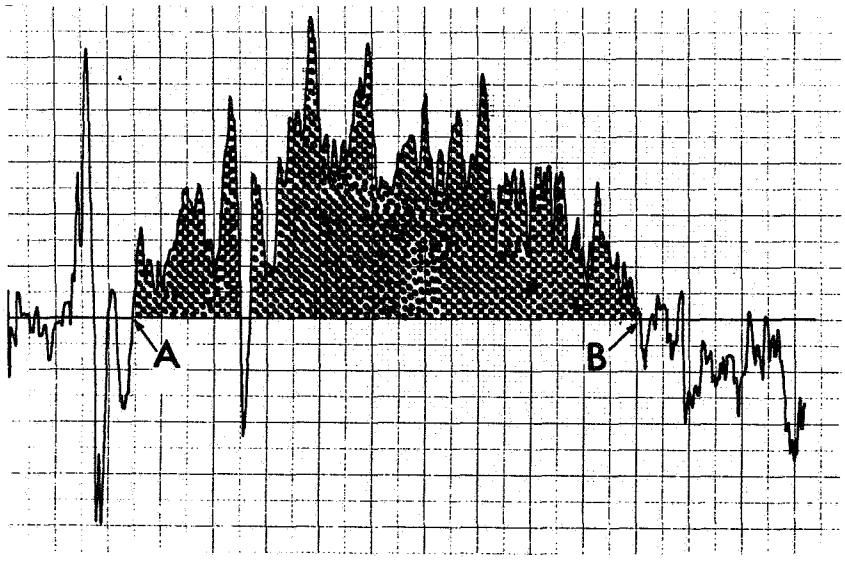

Figure 4–19.—Sample CNV (monkey, sensory-motor cortex-to-subcortical reference) illustrating area of the potential (stippled); (negative up).

TABLE III.—*Correlation Between CNV Area and Other Variables*

Variable	r	p
S_2 intensity	−0. 612	*0. 005
Heart rate	+0. 138	0. 200
GSR reactivity	+0. 002	>0. 500
Respiration rate	−0. 004	>0. 500
Reaction time	−0. 040	0. 450
Peak CNV amplitude	−0. 002	>0. 500
CNV duration	+0. 090	0. 300

*Significant correlation.

or scalp-derived dc shifts (1) are synchronized with behavioral processes; (2) may form an integral part of the bioelectric activity of the cortex; (3) may provide information on the functional state of the cortex.

We have made some observations that may be interpreted as raising some questions on these claims (Lombroso, in press). These were obtained on adult subjects, and the usual technique for the study of the CNV was used. The only difference in our setup was that 1 second following a flash (2 logarithmic units above threshold) the subject received via earphones a 10-msec tone delivered to either his right or left ear according to a program provided by a random pattern generator. There was background white noise. After baseline trials with no instructions, the subject was asked to respond to each tone regardless of the ear it reached. Thus, we could observe the development of the CNVs obtainable when the tone reached either ear and "averaged" separately on a CAT–1000 from an FM tape deck. There was no difference between these CNV, as could be expected. We also were recording and averaging both vertical and horizontal EOG, as well as the EMG derived from the "acting" arm, and from two pairs of electrodes placed orthogonally presumably over the sensory projection area of the contralateral hemisphere. The response requested was the activation of a microswitch that in some cases gated an electronic counter that measured reaction time. For some experiments, the response was "mental"; i.e., a serial subtraction. A continuous strip-chart monitored all parameters and permitted discarding of trials with mistakes or blinking and other artifacts.

After the CNV became established, the subject was asked to respond only when the tone reached one of his ears, and not to respond when it reached the other. Two to four trials were run consecutively in the same manner. Then the subject was asked to reverse his response to the time when the tone now reached the opposite ear. This again was

repeated for two to four trials. Finally, runs were made with the subject instructed to ignore the tone to either ear.

Figure 4–21 shows what happened consistently in the CNV obtained separately when the tone reached one ear or the other at random. Each graph is the average of 12 flash-tone sets. The broken lines indicate when the flash and the tone were given—1-second apart. In the left column are displayed the CNV developing when the tone reached the left ear, for which the subject was asked not to respond (NR) during the first three runs, and to respond (R) during the subsequent two. Conversely, in the right column are the CNV developing when the tone arrived at the right ear, for which a response was required in the first three runs, and none for the two consecutive ones.

Note the similarity of the CNV developing when the tone had reached the left or the right ear during the first run when a choice had to be made. Remember that the subject was performing (correctly as monitored all the time) a different task for each stimulated ear—press a switch or subtract a number in one case, or do nothing in the other. As the program continued, however, note the difference developing at the termination of the CNV. Naturally, none would occur during either the first evoked response nor during the development of the negative dc shift since no difference in the program became known to the subject until the imperative signal arrived.

As you can see, at the second such trial (run 4 of fig. 4–21) the CNV tends to terminate earlier when the tone reaches the ear for which

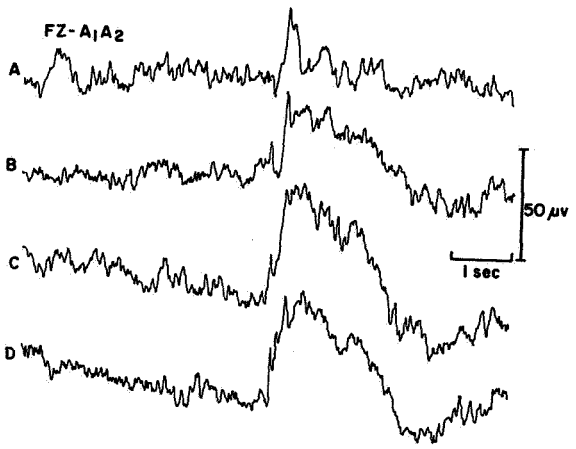

FIGURE 4–20.—Sample CNV (man, vertex-to-paired ear reference) obtained while recording for 4 sec before and 4 sec after the occurrence of S_1. Each trace is an average of 12 trials (negative up).

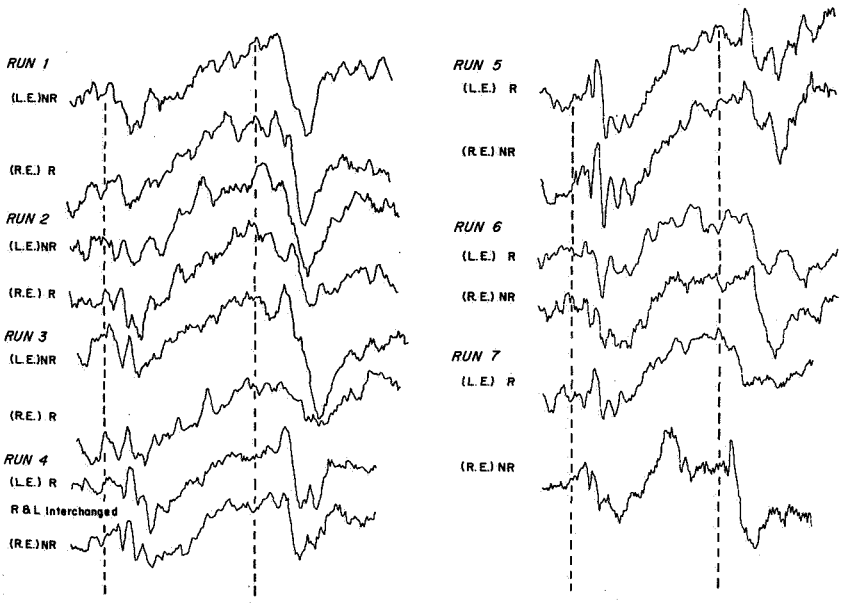

FIGURE 4–21.—Broken lines indicate times of flash-tone stimuli, 1 second apart. The CNV, obtained when the tone reached at random the left or the right ear, are displayed in the left and right columns, respectively. R and NR denote "response" and "no response," respectively. During runs 3, 4, and 5, the subject was responding only when the tone reached his right ear, while during runs 6 and 7, the instructions were reversed. Note the difference in the termination of the CNV between R and NR that appears at run 4 and its rapid reversal at run 6. During the last run, the subject was told not to respond to tones reaching either his right or left ear. All runs were performed consecutively and consisted of 24 flash-tone pairs, with tones distributed randomly to right or left ear.

the subject was told to respond. More strikingly, the positive deflection of the CNV when the tone reaches the ear for which no response is requested now has "grown"—so to speak—becoming notably greater than it was for the previous trial and falling below "baseline". Conversely, note how much smaller the positive deflection of the ending CNV has become when the stimulus reaches the ear selected for response. These differences become even more marked at the third consecutive trial (run 5 of fig. 4–21). Note what happens when the response parameters are reversed, that is, when at run 6 the subject is told to respond only when the tone reaches his left ear. The CNV termination sweep has changed also quite markedly, becoming less than one-half of its value for the previous one. Conversely, the end sweep of the CNV developing when the tone reaches the right ear for which now no response is requested, is about double what it was for the

previous run. Similar differences are seen for the next consecutive trial (run 7) and for the last (run 8). No differences are seen between the two sides when the subject is told not to respond to any tone, and no CNV develops.

These differences in the termination of the CNV were not caused by muscle or eye contaminants although I argue—like many others—that strict monitoring of both is necessary. Also "averaging," especially of eye movements, should be done in conjunction with CNV averaging since only by "averaging" one may discover their contribution. Likewise, there was no significant contamination in our CNV from the SER because of the contralateral fingering of the microswitch. These differences in the termination of the CNV occurred equally over both hemispheres and were unrelated to the subject's errors.

Now, I would like to illustrate briefly a second observation. This shift in the morphology of the CNV termination, with the reversing of instructions, can occur right away following the change in instructions and in response. But in other instances, we found a remarkable lag in the shift of the CNV morphology as it related to the behavioral response. Figure 4–22 illustrates this point. Here the two CNV (each an average of 12) are displayed in pairs. Note again the little difference between the NR and R CNV on the first trial, and the developing of a marked difference as trials progress (runs 2 and 3). But now, when the instructions are reversed at run 4, there is no immediate and parallel shift in the CNV termination. If anything, the end of the R CNV is still greater than the NR CNV. Only at run 6 and especially at run 7 do we see a well-established reversal of the CNV positive deflection. In other words, while the subject performed the requested shifts in his response immediately and correctly, there was a considerable time lag for a parallel shift to appear in his CNV.

It is possible that the described differences in the termination of the CNV obtainable when the subject responds or does not respond, may be related to a surge of negativity and to a further positive dc shift, respectively, and that these relate to aspects of discriminatory behavior such as "attending to" or suppressing" and so forth. Interesting as these differences I have described might be, I find it strange that the electrical signals accompanying these high-level neuronal processes should lag, at least in some subjects, so far behind their performance. It would seem reasonable to question, for example, the concept that the CNV represents a "priming" process of the frontal cortex preparatory for the discharging of its motor neurons, when it may take so long for an aspect of its morphology to "catch up" with a change in motor performance. For the same reasons, how could we relate these changes to either "excitation" or "inhibition" in neuronal assemblies, as has been claimed?

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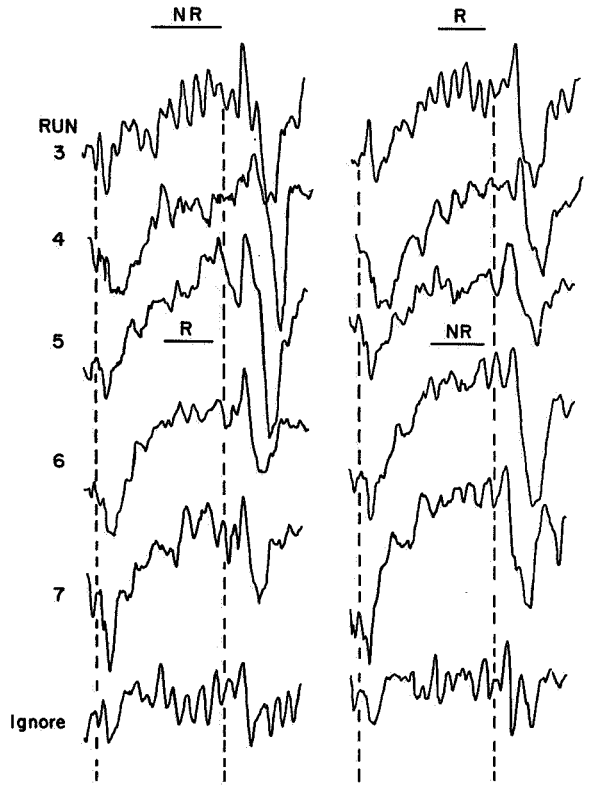

FIGURE 4–22.—The experimental situation is the same here as for the preceding figure and the CNV are displayed in pairs, those obtained when the tone reached the left ear being the first for each run. R and NR denote "response" and "no response," respectively. A notable difference develops in the termination of the CNV as the trials progress, being maximal at run 3, when the instructions were reversed. At runs 6 and 7 the reversal in the CNV termination takes several trials to become clear.

While it is conceivable that some components of the CNV are "neuronal" in origin—and we would agree that the AEP to flash and tone were indeed "neuronal" events—it is at least plausible that others may be non-neuronal. Referring in particular to the dc shifts, we should remember that parallel to neuronal events of excitation or inhibition, many metabolic and physiochemical processes are occuring in glial cells, capillary endothelium, and the like, all capable of inducing slow current shifts. Adey (1963), for instance, has shown that rapid changes in impedance of small volumes of cortex closely relate

to relatively rapid shifts of blood flow and gas exchange, and that the impedance of dendritic structures has been noted to change in several of those behavioral states during which negative dc shifts have been measured on the cortex. It is conceivable that a phenomenon such as the one we have described before, namely, the time lag between reversing changes in CNV when behavioral parameters are reversed, might be explained easily on the basis of such extraneuronal sources whose time basis is much less rigid than one would expect from neuronal populations.

Dr. Chapman: I would like to make two points—the first about the various ways that eye movements might affect electrical responses and the second about the relation between CNV and AEP. We all know that eye movements can cause much difficulty, and this holds equally true for the slow potentials, as well as the faster ones which we categorize as evoked potentials. I would like to point out that there are several ways in which such eye movements might affect our responses. The one that has been discussed primarily is the direct electrical effect resulting from movement of the eye. The voltages recorded from the eye as the corneoretinal potential or electrooculogram (EOG) may be carried by volume conduction to electrodes at other sites on the head.

There are also indirect ways in which eye movements might affect the evoked potentials that we ought to keep in mind because the effects may be larger although the mechanisms are more subtle. I call them indirect because they involve the visual pathways themselves, rather than simple spread of potential. There are at least two ways in which the indirect effects might occur.

One kind of indirect effect is caused by displaced retinal images producing neural activity in the visual system. A static light pattern moved across the retina by eye movements is a very effective stimulus for retinal activity because both on and off responses are produced as the light moves onto fresh retina and off of previously stimulated retina. This effect was demonstrated in experiments by Gaarder et al. (1964), who had their subjects fixate a static target and obtained an AEP by synchronizing the computer with the subject's eye movements. The AEP they obtained depended upon stimulus characteristics showing that it was mediated by the visual system. This indirect effect may be large enough to be seen as lambda waves in the EEG. This indirect effect can be eliminated in many experiments by keeping the subject in the dark and keeping the presentation of visual stimuli so brief that the retina does not have time to sweep across the light. It is to be noted that experiments purporting to use nonvisual stimuli are not immune from this eye movement effect if a visual field is present, since the nonvisual stimulus may synchronize eye movements, which in turn

result in visual evoked responses. If we are interested in differential effects, we might worry about differential eye movements producing the differences between the evoked responses or CNV.

The second indirect effect of eye movements concerns direction of gaze and is not so easy to cope with. A light stimulus may not reach the same retinal locations from one stimulus presentation to the next, even within an averaging run. Even having an experienced subject fixate is only an approximation to reproducible conditions since eye movements are so ubiquitous (Riggs, Armington, and Ratliff, 1954). The eye need move only little for the light stimulus to reach a fresh piece of retina, and it is well known that adaptation effects in the visual system are very large. Also a shift in gaze may shift the visual stimulus onto a part of the retina that contributes more or less to the AEP (Rietveld et al., 1965; Tepas and Armington, 1962); for example, consider the difference between peripheral and foveal representation. We need to consider the problem that differences in electrical responses associated with independent variables of interest may be caused by differences in gaze direction, and the problem is more complicated than knowing the average gaze during an averaging run. Reviewing briefly, eye movements may have direct effects from the EOG and indirect effects via the visual system—i.e., retinal image displacement during the individual stimulus presentation and from one presentation to the next.

The second issue concerns the relation between slow wave potentials and the AEP. The data in figure 4–23 were obtained in a study of AEP (Chapman, 1965); however, slow wave effects show up. Two classes of stimuli, numbers and letters, were presented in a sequence that was fixed for a given run of trials. For example, the data in the top row were from runs when each trial had the following sequence of light flashes: number, letter, number, letter, blank. The particular numbers and letters in each position were randomly selected. The subject was given a task that involved one set of stimuli. In the top row, the letters were relevant to the task, and the numbers irrelevant. In the second row, the same physical stimuli were used in the same sequence, but the numbers were relevant to solving the problem, and the letters irrelevant. Vertical comparisons showed the tendency for larger AEP when the stimuli were task-relevant.

These AEP appear to be superimposed on a slow wave change running across the trials. This experimental design has features in common with the one used to obtain the CNV, namely, trials in which there are fixed temporal relations among the stimuli; also, the subject must respond to certain imperative or relevant stimuli.

The question is whether the enhanced positive response (positive is up in fig. 4–23) to the relevant stimulus might be caused by the

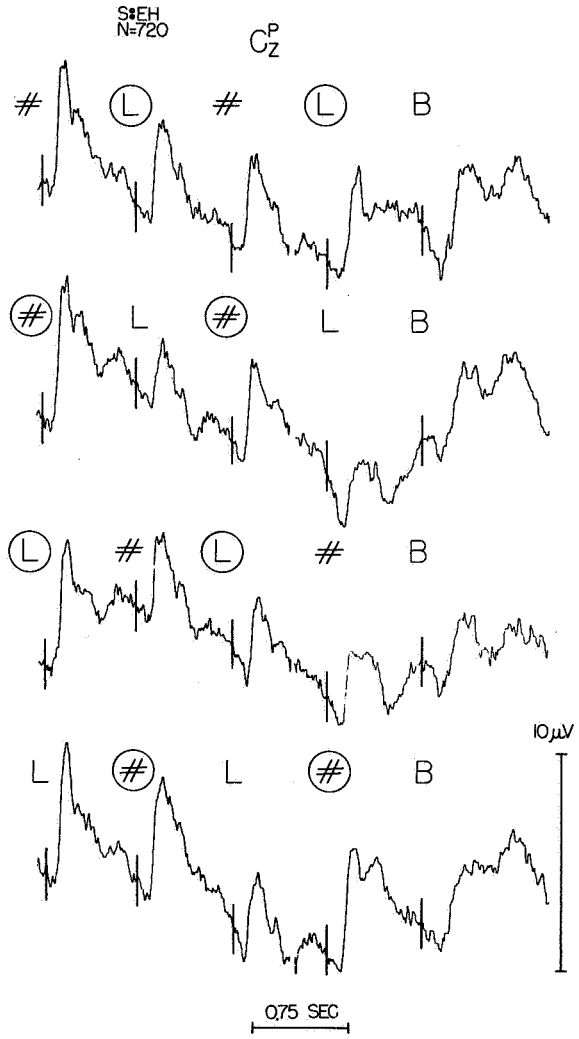

FIGURE 4–23.—Averaged evoked potentials to numbers (#) and letters (L) during runs when one stimulus class was relevant (circled), and the other irrelevant. Monopolar recording from electrode between C_z and P_z with reference to linked earlobes (negative down). Time constant was 0.4 sec. Onset time of brief visual stimuli shown by vertical lines on traces.

termination of an anticipatory negative wave. If so, AEP and CNV investigations may be studying the same process.

The data from this subject suggest that they are independent to some extent. For the sake of this discussion, the AEP is defined as

the amplitude of the major positive component with reference to the voltage level at the time of the stimulus (vertical lines in fig. 4–23). The slow wave is defined as the voltage levels "across" the trial at the times of the stimuli. Although there is a statistical tendency for higher AEP peaks to occur when the trace at the time of the stimulus is more negative, this does not appear to be a tight, causal connection. For example, the largest AEP tended to occur at the first stimulus on all types of trials (first AEP in each row) although for this subject the most negative part of the slow wave occurred later in the trial when the fourth stimulus was flashed. Moreover, the pattern of slow wave change across the trials was different in other subjects although there was a tendency for the relevant stimuli to evoke higher-amplitude AEP.

Aside from the question of the relation to CNV, these data illustrate a problem with regard to AEP measurement. Most of us take our AEP measurements in relation to the particular response we are looking at, and not in relation to the entire sequence of responses or an absolute reference level. For example, many of us establish a baseline using the very first part of the response or the potential level found a short time before the stimulus was delivered and measure amplitudes from there. The data in figure 4–23 show that the AEP amplitudes would be profoundly different if they were measured from a common baseline for all the AEP in the sequences.

Dr. Walter: Dr. Low's espousal of the area under the CNV curve raises a question. Why not use a low-pass filter on the data? It would seem that this would be an entirely equivalent operation. Is there some positive reason for not treating the data that way, rather than waiting until the Linc can process it?

Dr. Cohen: To answer Dr. Walter, I guess my reason for not filtering that way is that I am interested in the faster responses as well at the same time.

Dr. Walter: It still might be easier if you separated them onto two channels.

Dr. Cohen: In regard to Dr. Lombroso's finding, we have made similar observations. When stimuli are to be discriminated, the negative stimulus is followed by a positive wave. I wonder whether he thinks that positivity, in that case, represents inhibition. If the negativity is priming excitation, do we have an opposite process in the positivity? I realize we must have some balance.

There is a lag in the CNV compared to the behavioral response. I think that the reason for the lag is that we are looking at synchronous activity. We don't know what in the cortex is mediating a particular behavior and how that is reflected electrically at a given point. However, unless there is synchrony, we won't see a wave at the surface.

Young children can perform the tasks used in CNV experiments before the CNV can be recorded. This is also found in children with learning problems and behavioral disorders. There, behaviorally they may be able to perform the task, but the CNV is very much retarded for the age level and may be absent. We obtain behavioral changes before we see a synchronized electrical activity at the scalp. But that should not be too surprising since I think maximal cortical response is not required in order to mediate a simple task.

DR. ALLISON: I have a question for Dr. Vaughan which was triggered by one of Dr. Cohen's figures (fig. 4–5). He showed the topography of the CNV, and it was largest in the vertex region. It is curious to me that there is a whole variety of evoked responses, all of which are largest in the vertex region. Of course, there are the vertex potentials themselves, which may or may not be largest at the vertex. There are certainly differences between modalities that are focused in the region of the vertex.

Some of the early auditory responses that Goff described this morning are largest at the vertex. Goff and I have recently been recording odorant evoked responses (Allison and Goff, 1967). This response is a long latency response that is also largest at the vertex. It seems unlikely to me that the area of the brain under that electrode is really responsible for all this activity. I gather that you are rather optimistic about your volume conduction model, allowing you to infer the generators of scalp-recorded responses. I am wondering how your model would accommodate this conglomerate, all of which seems to be large at the vertex.

To be specific, would the model allow us to specify superficial generators, as the CNV might be, for example, if it were generated transcortically, as opposed to a deep generator, as I would expect the odorant response, for example, to be?

DR. VAUGHAN. As I have noted in Chapter 2, there are several steady-potential shifts (SPS) that differ both spatially and in their behavioral correlates from the CNV. My present evaluation of this situation is tentative at best, both because of the artifact problem and the somewhat ephemeral nature of these phenomena. Because of these uncertainties, I have not published more data obtained over the past few years in my laboratory. I have serious reservations concerning the reliability and interpretation of some of the published observations made elsewhere. For a time, we were convinced that the CNV was, in fact, merely the early slow component of the motor potential (MP) (the "readiness potential" of Kornhuber and Deecke, 1965). Our evidence was the somatotopic distribution over motor cortex and the fact that it seemed to be more closely time-locked to the motor response than to the stimulus in both the reaction time paradigm and the time esti-

mation paradigm (Vaughan and Cost, 1968). We have never been able to confirm conclusively the existence of a frontal SPS independent of the MP or the EOG. The issue has been complicated further by our confirmation of the early observations by Köhler, et al., (1955a, b) of SPS associated with novel stimulation and the discovery of SPS over visual cortex in discrimination tasks. Since these seem to be specific potentials comparable to those recorded by Gumnit and Grossman (1961), the SPS to auditory stimulation is maximal at the vertex. Only for visual stimuli has it been possible for us to make a clear spatial differentiation of the SPS associated with sensory set or orientation and those which seem to be related to preparation for a motor response.

My own bias on this problem is that the physiological origin and functional significance of the SPS need to be elucidated in experimental animals. At the present time, there is virtually no reason even to suppose that they are either wholly or partly of neural origin; nor do I feel that the phenomena are sufficiently reliable within or across subjects, even under the quite close behavioral controls we employ, to feel very comfortable about suggestions that these phenomena might have diagnostic value in clinical populations. Although much of the interest in steady potentials in man has derived from the presumption that "complex" psychological variables exist which may be defined by such vague terms as "expectancy," I suspect that observations made in animals under carefully controlled behavioral conditions will provide the insight into the functional significance of these potentials.

Dr. Donchin: I want to support Dr. Chapman's comment on the relationship between the CNV and the AEP. Dr. Smith and I have obtained very similar results (Donchin and Smith, 1968). I would like to stress that whatever we decide about the CNV, its nature, its physiological source, and its functional significance, we must consider the relationship between the CNV and the evoked response. While an investigator might have no interest in the CNV in a particular study, the subject determines to a large extent the nature of the experiment. If the instructions produce constraints that make the contingencies between the stimuli in a series important to the subject, as for example when the stimuli follow each other at a fixed interval and each becomes an S_1 to the following S_2, a CNV might—and usually does—develop between the successive stimuli. If there is a relationship between the CNV and fast responses—and we have no information on that yet—then the results of such an experiment would be difficult to interpret. In the study I refer to, we were basically interested in the task-relevance of stimuli and its effect on the AEP. The stimuli were presented randomly, and averaged evoked potential differences were indeed found that depended on task-relevance. However, if the

stimuli are presented at a fixed rate and the proper amplifiers are used, a CNV develops between the stimuli. Any relationship between the CNV and evoked response thus would greatly affect our results.

DR. KNOTT: I want to give Dr. Dale McAdam an opportunity to comment on the matter of evoked response and the CNV. He may answer your question for you. However, I would like to return to the data presented by Jasper at the Cold Spring Harbor conference of 1936. Using rather long-time-constant amplifiers, he recorded very slow potentials in the electroencephalogram. At that time, Jasper made some analogies between shifts to a negative polarity and increases of excitability, and shifts to a positive polarity related to a decrease in excitability. In this, he led the field by many years.

Our data on the anxiety problem did show that there was a lesser rise in the CNV in moderately anxiety-prone individuals placed under stress than in less anxious subjects under stress; this led us to believe that there is a finite dc level that can be achieved. This has been followed up by my collaborator, Dr. Don Irwin, who has been able to show that while the dc level of the cortex can be increased by constant stimulation, there consequently is a limit to the remaining shift under the expectancy paradigm. This is important, because I think it may explain the Chapman data, and I think it clearly is related to the comments just made by Donchin.

DR. MCADAM: I would like to summarize briefly some recent work (McAdam, 1969) showing changes in somatosensory evoked potentials to noncue stimuli presented during a CNV. Figure 4–24 shows sample CNV and the procedure we used. We generated CNV by asking subjects to respond as quickly as possible to the offset of an 1800-msec tone. On a random one-half of the trials, shock to the median nerve was given 1 second after tone onset. The median nerve shocks were of sufficient intensity to cause a small but reliable thumb twitch. Subjects were instructed to pay no attention to the shocks, but to attend only to the tone and respond as quickly as possible to its offset. Shocks were also delivered during the intertrial intervals, i.e., when no CNV was present.

In addition to recording the CNV from a vertex-mastoid derivation, evoked potentials to median nerve shock were recorded from contralateral frontoparietal scalp using RC-couplied amplifiers. Examples of these potentials are presented in figure 4–25. These are tracings of responses from two subjects, A and B, under conditions when the shock was presented during the CNV (A′ and B′), and when it was presented in the intertrial interval (A and B). The lower set shows the most complex potential, while the components in the upper set were seen in all 24 subjects. All scoring was based upon the seven compo-

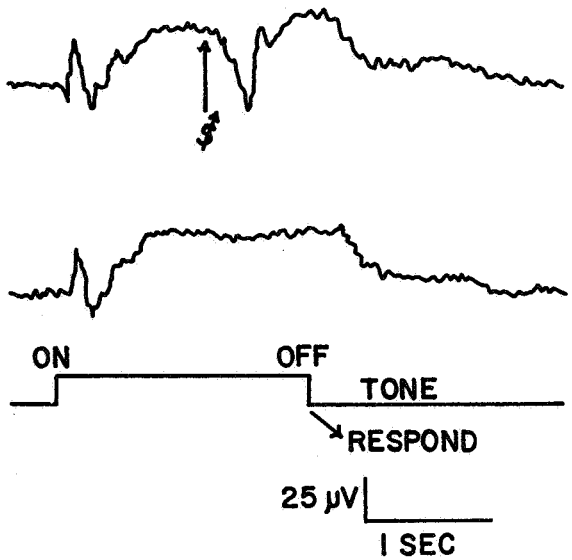

FIGURE 4–24.—CNV from one subject. Averages of 50 responses each. Vertex-to-mastoid reference. Upper record: trials when median nerve shock was presented. Lower record: trials with tone alone (negative up).

nents that were common to all subjects; the component numbers used in tables IV and V are those shown in this figure.

In addition to the 16 subjects in the experimental group, 8 subjects were run under a "tone-control" condition. These subjects were given the same stimuli as were those in the CNV group, but made no responses. No CNVs were seen in this group.

Analysis of amplitude changes between potentials recorded when the shock was given during the tone as compared to those recorded when shock was given during the intertrial interval showed an across-the-board decrease in amplitude of nearly all components when the shock was presented in combination with another stimulus (see table IV). These amplitude decreases during tone were present whether a CNV was generated or not.

Latency changes, on the other hand, were found to be unique to the CNV group; the latencies of components 9, 10, and 11 were significantly shorter when the shock was given during the CNV than when it was given during the intertrial interval. No corresponding difference was found in the tone-control group data (see table V). This result lends support to the hypothesis advanced by Walter, et al. (1964) that the CNV represents "the electric sign of cortical prim-

ing." There is, however, nothing in these data that would limit the mechanism for the observed responsiveness changes to a cortical site. In fact, since the changes in the evoked potentials that were unique to the CNV group were seen only with the later components (beyond 200 msec), subcortical structures such as the reticular formation are very probably playing a role in producing these changes.

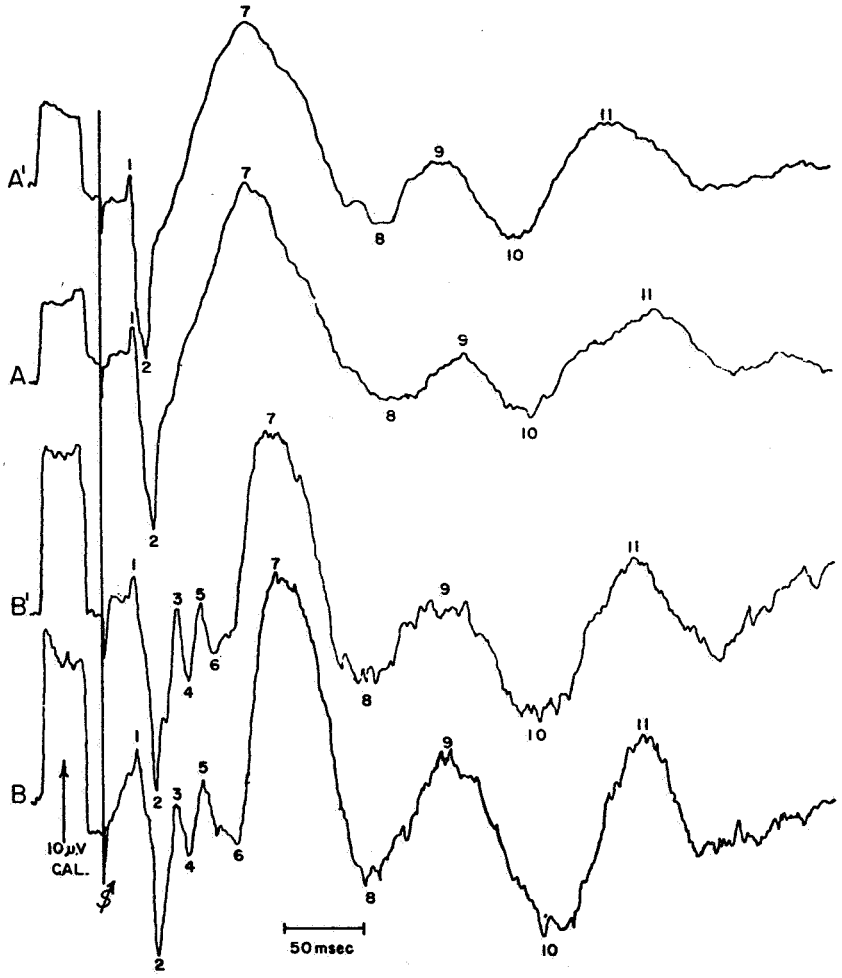

FIGURE 4–25.—Somatosensory evoked potentials from two subjects, A and B, in the CNV group. Averages of 50 responses each. Parietal electrode relative to a reference placed 6 cm anterior to it. Square wave at left of each trace is 10–μV calibration signal; A and B responses obtained during the intertrial interval and A′ and B′ are responses obtained during presentation of the tone (negative up).

TABLE IV.—*Mean Differences in Peak-to-Peak Amplitudes of Evoked Potential Components*

Components[a] (and mean peak-to-peak amplitudes, in μV, response during intertrial)	Tone control group		CNV group	
	Difference (μV) (intertrial-tone)	t	Difference (μV) (intertrial-CNV)	t
1–2 (14.5)	1. 3	[b] 3. 33	0. 9	1. 02
2–7 (19.0)	1. 6	[b] 2. 71	3. 7	[b] 2. 75
7–8 (11.4)	1. 2	[b] 2. 67	4. 1	[b] 4. 17
8–9 (8.9)	2. 0	[b] 5. 00	2. 7	[b] 2. 48
9–10 (9.5)	1. 6	[b] 2. 71	1. 9	3. 92
10–11 (9.4)	−0. 4	0. 585	1. 1	1. 43

[a] Identified by numbers as in figure 4–2.
[b] $P<0.05$; all others not significant.

TABLE V.—*Mean Differences in Peak Latencies of Evoked Potential Components*

Component[a] (and mean latency, in msec, response during intertrial)	Tone control group		CNV group	
	Difference (msec) (intertrial-tone)	t	Difference (msec) (intertrial-CNV)	t
1 (18.7)	0. 4	. 800	−0. 2	. 610
2 (33.4)	0. 1	. 164	0. 1	. 151
7 (104.9)	1. 4	. 318	−2. 2	1. 93
8 (158.1)	−4. 1	. 579	3. 6	1. 26
9 (210.6)	2. 0	. 769	6. 5	[b] 2. 20
10 (273.7)	2. 6	. 565	7. 3	[b] 4. 68
11 (343.9)	−0. 8	. 333	10. 3	[b] 4. 11

[a] Identified by numbers as in Figure 4–2.
[b] $p<0.05$, all others not significant.

I would like now to discuss another point in Dr. Vaughan's support by helping bridge the gap between what he calls the "motor potential" (Vaughan et al., 1968) and the CNV. The motor potential consists in part of a slow, surface negative shift recordable over motor cortex, which occurs before the performance of a voluntary motor act. It is topographically distributed over the motor area, the site of maximum amplitude being determined by the muscle groups involved in making the response.

The motor potential was first described by Kornhuber and Deecke (1965), and they gave it the name *Bereitschaftspotential* or "readiness potential" (RP). They noted that the RP is enhanced by "intentional engagement" on the part of the subject in the performance of the response, but they do not report quantitative data on this point. "Intentional engagement" sounded a lot like motivation to us, and since a number of papers on the CNV had expressly implicated motivation as one of its most potent psychological determiners (Irwin et al., 1966a, b; Rebert et al., 1967; McAdam et al., 1968), David Seales and I decided to look at the RP under conditions of varying motivation (McAdam and Seales, 1969).

Figure 4–26 shows examples of RP that we recorded from electrodes located at C_3 and C_4 (contralateral and ipsilateral motor areas, respectively) while the subject was making a simple button-press response with his right thumb. Two conditions were run; in one, labeled "baseline" subjects were instructed simply to make a response every 3 or 4 seconds. There was no consequence whatsoever. Under the "reward" condition, the subjects were given purposely vague instructions that if they responded in the "right way" or at the "right time" they would receive a monetary reward for the response. In fact, they were rewarded on a random 50 percent of the trials in this situation.

Figure 4–27 summarizes the data from the 11 subjects tested under these conditions. Analysis of variance on these data showed no interaction effect between conditions and electrode placement. However, RP amplitudes were significantly larger under the "reward" condition than under the "baseline" condition for both ipsilateral and contralateral placements, and RP amplitudes were larger for contralateral placements than for ipsilateral placements under both conditions. It appears, therefore, that the RP changes with increased motivation in much the same way as does the CNV; i.e., larger amplitude responses are found. Since the changes in RP amplitudes were the same for both ipsilateral and contralateral electrode locations, it implicates a general activating system (possibly the reticular formation) as a neural substrate for these changes.

It is interesting that a laterality effect has never been reported for the CNV. This is certainly caused in most cases by the fact that experimenters did not look for it; the CNV is "traditionally" recorded as a midline phenomenon. Nonetheless, both Low et al. (1966a) and Cohen (in this volume) report that the CNV is distributed symmetrically in the coronal plane with a peak amplitude at the vertex. It may be that the increased complexity of the CNV situation over that used to evoke an RP and the fact that stimuli have been presented bilaterally, served to wash out any laterality effect. Mr. Seales and I are currently exploring this problem, but we have no data which we can report as yet.

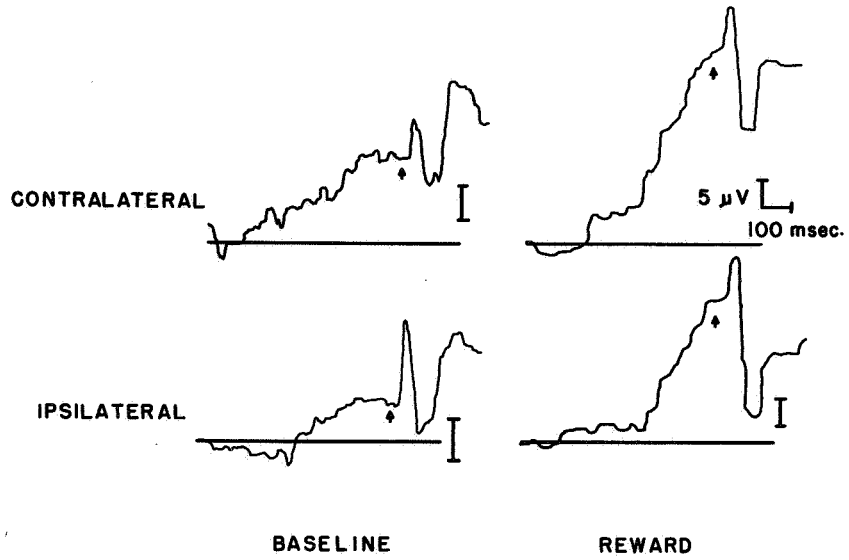

FIGURE 4–26.—Sample RPs obtained from one subject. The active electrode is C_3 or C_4. Arrow indicates occurrence of response (negative up).

DR. LEHMANN: I would like to ask Dr. McAdam if he measured the amount of pressure exerted by the subject in the reward and non-reward situation.

DR. MCADAM: I would be very happy to say that I did, but I didn't. We were not equipped to do it.

DR. HILLYARD: Both Low and Cohen pointed out that the corneoretinal potential can be a source of artifact in the CNV, and I would like to present some data that will document the seriousness of this problem.

Figure 4–28 (Hillyard and Galambos, in press) shows computer-averaged tracings of the CNV from the vertex-mastoid derivation and the simultaneous transorbital EOG, recorded with dc electrodes above and below one eye. These recordings were taken in the standard S_1–S_2-lever-pressing situation, wherein S_1 was a click and S_2 was a tone that signalled the motor response. These are typical records from nine different subjects, each of whom displayed a different, characteristic pattern of eye movements during the S_1–S_2 interval.

In the top row, for example, the waveform of the CNV is paralleled closely by the waveform of the EOG deflection (lower tracing). An upward deflection in the EOG indicates a negative shifting of the supraorbital electrode, caused by elevation of the negative, posterior end of the corneoretinal dipole. Most commonly, there was a downward eye rotation in the S_1–S_2 interval if the eyes were closed; however,

the mechanisms and significance of such involuntary eye movements preceding lever presses remain a mystery. Some subjects, however, did not display any sizable eye movements under identical circumstances.

My next task was to relate the amplitudes of these transorbital potential shifts to the artifacts produced concurrently in the vertex-mastoid montage during different-sized eye rotations. Accordingly, the recorded CNV was subdivided into one component caused by the corneoretinal fields called the Eye Artifact Potential (EAP), and a second component which I called the "true" or tCNV, which probably comes from the brain. These two potentials are summed and confounded in most recordings of the CNV unless special precautions are taken.

A separate "calibration" procedure was designed to relate the amplitude of the EOG deflection to that of the EAP induced at the

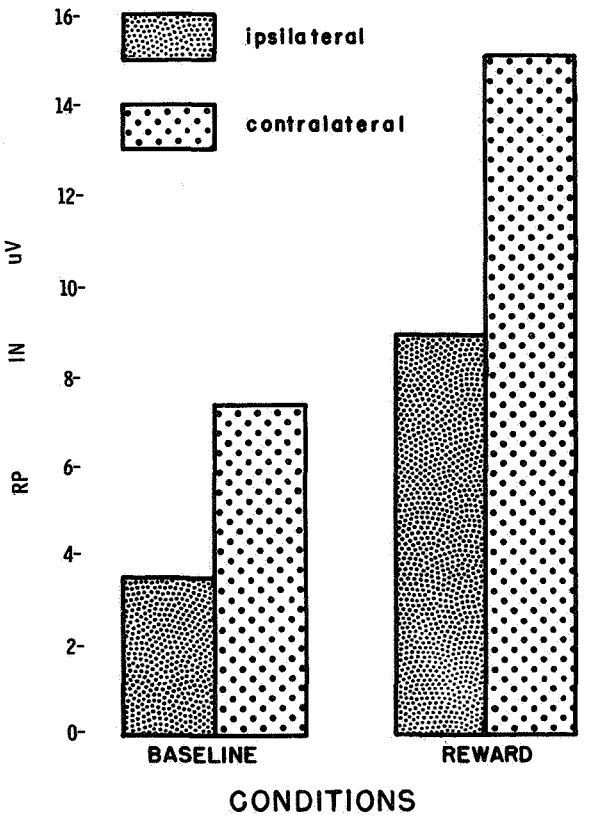

FIGURE 4-27.—Group means of RP amplitudes for ipsilateral and contralateral locations under baseline and reward conditions.

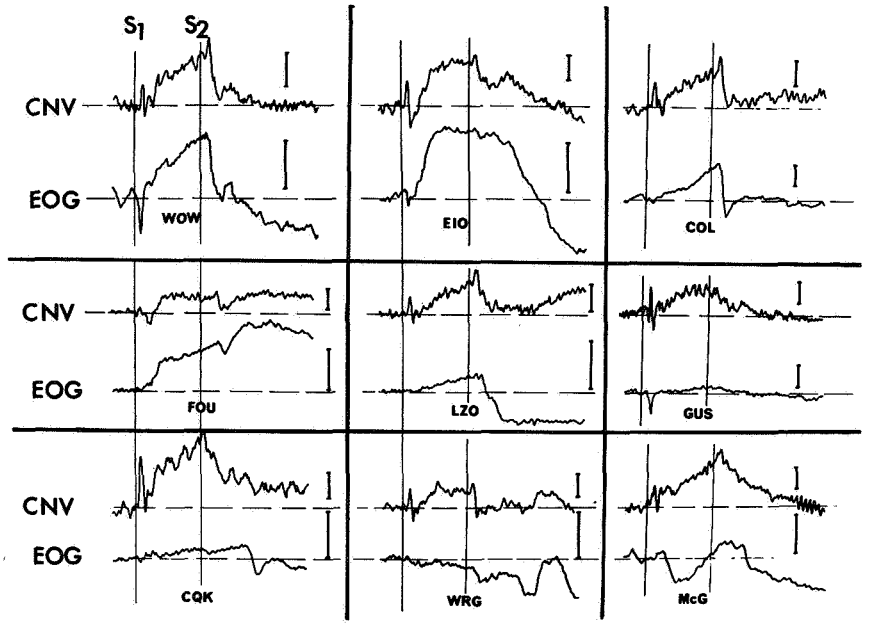

FIGURE 4–28.—Simultaneous recordings of the CNV and the deflection in the vertical EOG, caused by synchronous involuntary eye movements. Calibrations are 20 μV for the CNV and 100 μV for the EOG (negative up).

vertex. Each subject made upward and downward eye rotations of specified angles, and the EOG deflections were measured along with the EAP, which could be algebraically separated from the concurrent tCNV by a subtraction method, described fully in Hillyard and Galambos (in press).

The amplitude of the EAP induced at the vertex (in microvolts) is plotted in figure 4–29 as a function of the potential shift recorded across the eyes during eye rotations of different sizes. This relationship was linear in all subjects, and the parameters of the lines of best-fit are given for the vertex-mastoid channel. The solid circles represent EAPs that were induced simultaneously in a frontal electrode, placed 4 cm anterior to the vertex. In the frontal electrode, which was closer to the eyes, a greater proportion of artifact was induced per unit of eyeball rotation. By taking such calibration curves and applying them to CNVs that were recorded in the S_1–S_2-lever pressing situation, the appropriate amount of EAP at the vertex could be calculated. By such procedures, it was found that 23 percent of the CNV was composed of negative EAP in the average subject, because of a net tendency to move the eyes downward in synchrony with the CNV.

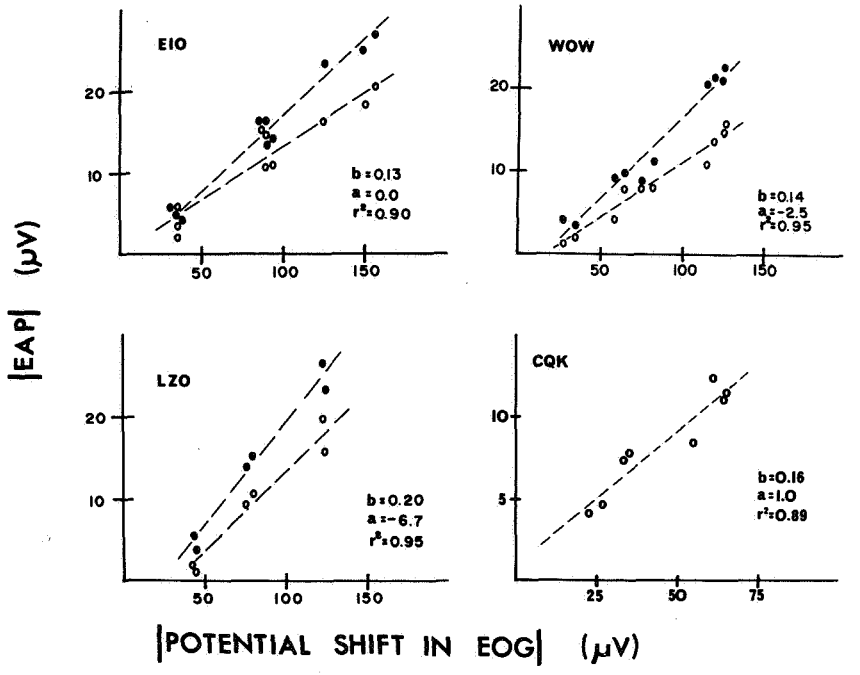

FIGURE 4–29.—Linear increase in eye-movement artifact (EAP) with increasing potential shift in the EOG, both induced by voluntary eye rotations. Parameters of best-fit lines: b=slope, a=intercept, r^2=Pearson correlation coefficient, squared. Solid circles: frontal-mastoid. Open circles: vertex-mastoid.

Within a given subject, however, there was considerable variability from trial to trial in the magnitude of the eye rotation (fig. 4–30). At times, the involuntary eye movements were downward, thus incrementing the tCNV; however, on other sets of trials, the EAPs were absent or even positive, thus partially cancelling out the tCNV. Eye movements introduced a tremendous amount of variability into the CNV, even under constant conditions. It is therefore possible that experimental manipulations, such as a change in the stimuli or task conditions, could produce change in the CNV, either by affecting oculomotor mechanisms (and the EAP) or by altering the tCNV.

Figure 4–31 gives a graphic illustration of how the eye artifact can affect what is recorded from the vertex. For each subject, three blocks of 12 trials are shown, selected and summed on the basis of the size of the concurrent EOG deflection. In the set of trials labelled A, the eye was rotated downward in subject McG; in B the eyes didn't rotate much, and in C the eyes rotated upwards, producing negative, zero, and positive EAPs, respectively. Notice the reduction in the CNV caused by positive EAP and its enhancement by negative EAP.

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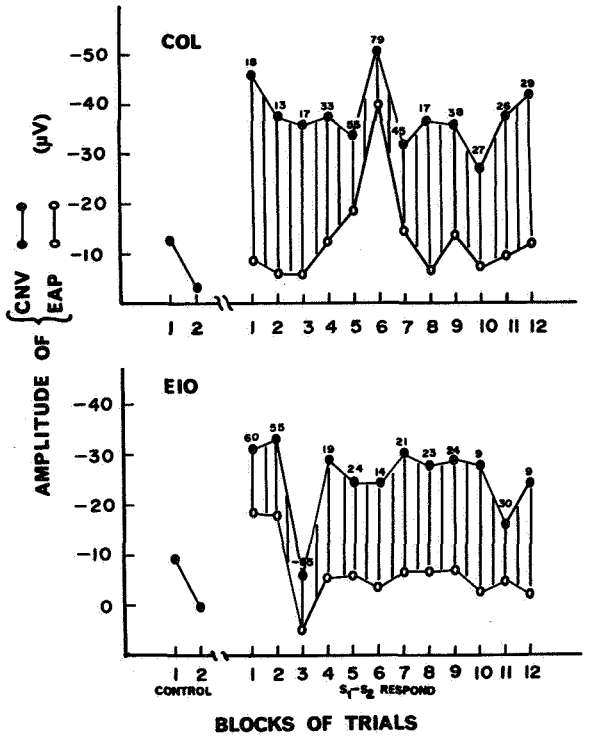

FIGURE 4–30.—Spontaneous variability to the CNV, broken down into EAP and tCNV (hatched area) components. Number above each point is the percentage of CNV comprised of EAP, or simply "percentage artifact".

The story is the same for subject EIO; CNV and EOG from three blocks of 12 trials are shown, and the CNV was progressively diminished as EAPs became more positive. In the frontal electrode, the EAP was larger than at the vertex for a given amount of eye rotation, while the tCNVs were smaller by about 25 percent.

These relationships are shown graphically in figure 4–32. In each subject, 96 trials were subdivided into blocks of 12 on the basis of the EOG deflections. The CNV amplitudes recorded from the vertex were a linear function of the eyeball rotation, which was indexed by the transorbital EOG.

One way to eliminate ocular artifacts was to perform the CNV experiments with the eyes fixated on a point. Typical CNVs recorded from four subjects with the eyes fixed are shown in figure 4–33; since the eyes did not move, these potential shifts represent only the tCNV component. In WOW and EIO, there was a small "twitch" in the EOG

after the click (S_1), which could have contaminated the click-evoked potential, but not the tCNV. Eye blinks frequently occurred after the lever press, causing large deflections in the EOG because of upward rotation of the ocular dipole. The resultant EAP caused the CNV to "cut off" more sharply than it would have if no blinks occurred.

The amplitudes of the tCNV that were recorded directly with the eyes fixated were equal to those of tCNV produced with the eyes closed and free to rotate, calculated by subtraction of the appropriate amounts of EAP. This equality substantiates the validity and accuracy with which the CNVs were partitioned into additive tCNV and EAP components.

Further studies were made of the relationship of the CNV and tCNV to the reaction time (RT) of the lever press (Hillyard, in press). Previously, it had been shown that an inverse relation between large CNV and short RT occurred in the context of acquisition of the CNV (Hillyard and Galambos, 1967; Walter, 1965a); that is, on the first few trials, before the subject had learned the S_1–S_2 association, the CNVs were small, and RTs were long. With practice, the CNV grew larger while RT decreased, thus producing a significant negative correlation over acquisition trials.

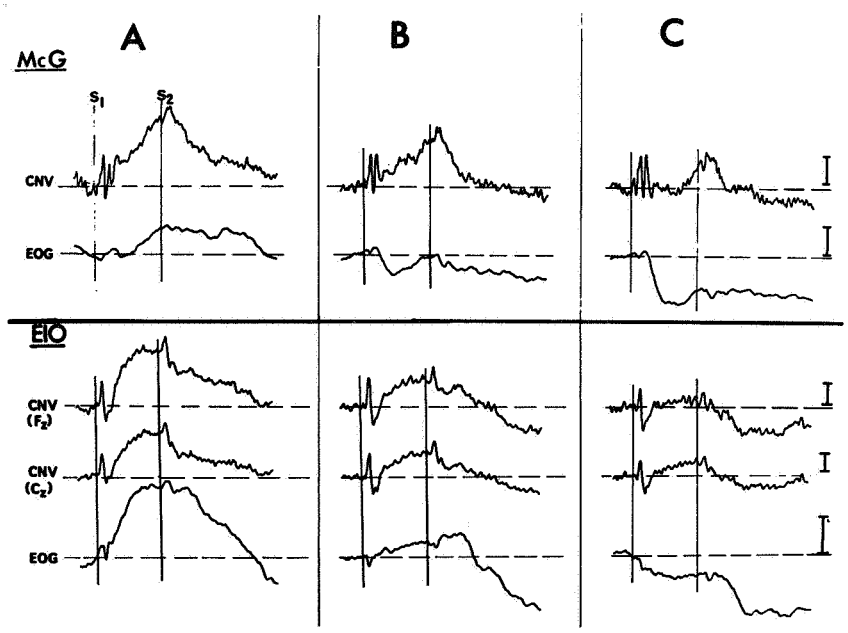

FIGURE 4–31.—Correlation of CNV amplitude with ocular potential shifts. Calibrations: CNV=20 μV, EOG=100 μV (negative up).

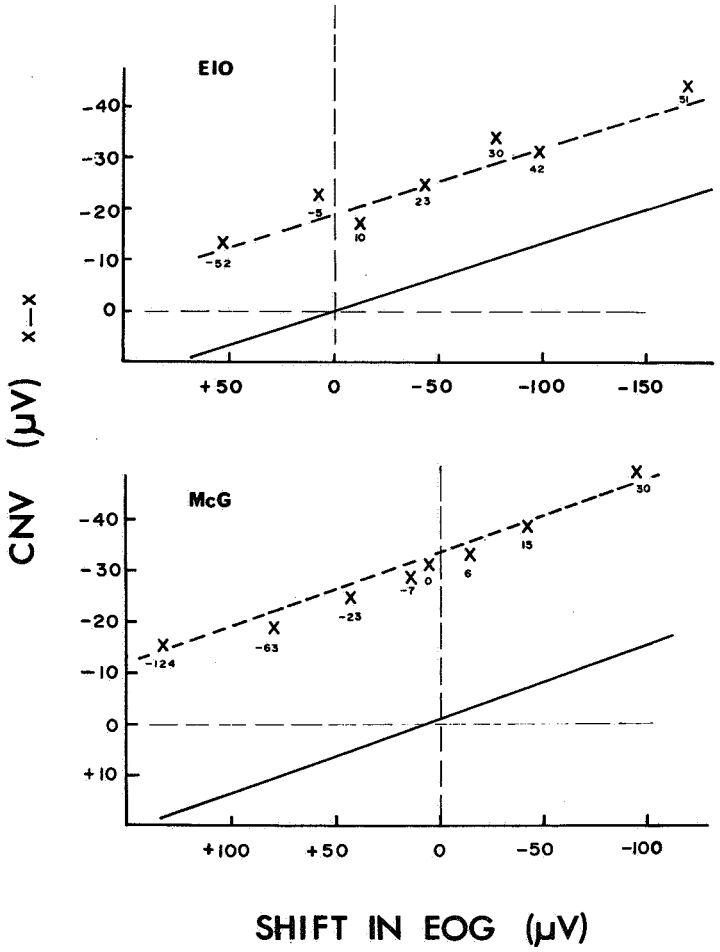

FIGURE 4–32.—Linear increase in CNV as a function of EOG deflections spontaneously produced. Solid line gives predicted value of EAP for given amount of EOG deflection.

I was interested in the trial-to-trial relationships between CNV and RT, within a long series of trials in which there was no net trend of increasing CNV or decreasing RT. A total of 96 trials was subdivided into blocks of 12 each on the basis of RT. The tape-recorded CNVs were summed together on each block as shown in figure 4–34.

The uppermost tracing is the averaged CNV from the 12 trials with the fastest RT, ranging from 104 to 148 msec. The second tracing is from the 12 trials with the next fastest RT, and so on. For this particular subject, CNVs were significantly smaller when RTs were longer, with RT fluctuating spontaneously on a trial-to-trial basis.

Eye movements were very small in this subject, and the tracings contain only tCNV. A second procedure was to consider 15 pairs of immediately adjacent trials, one of which had a fast RT while the other had a much slower RT, and sum the two sets of CNV separately. The tCNV was significantly larger on the trials with the faster RT (−26.9 μV versus −13.2 μV), even though the two kinds of trials occurred within seconds of each other. There seems to be a moment-to-moment fluctuation of a response-governing process, resembling concentration or attention, which is reflected in the amplitude of the tCNV. In many subjects, the tCNV amplitude could serve as a predictor of the RT of the ensuing motor response.

This inverse relationship between tCNV and RT is plotted in five subjects in figure 4–35. The CNV were averaged in blocks of 12 trials, and the mean tCNV is plotted against the median RT (msec) of the 12 trials. In each case, there was a statistically significant negative correlation. This analysis was made on ten subjects, but only in these five did a significant correlation emerge between tCNV and RT. I have no good explanation why some subjects did not display the correlation, but they did tend to have RTs that were somewhat faster and more narrowly distributed, and/or tCNVs that were smaller and less variable.

Dr. Walter: Could I ask a question on this figure? The line con-

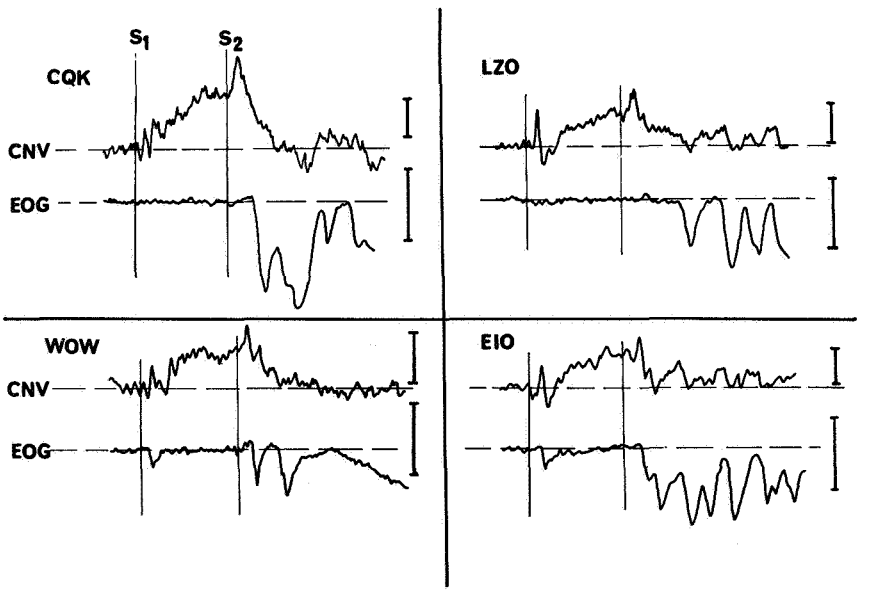

Figure 4–33.—CNVs recorded with eyes fixated. EOG deflections and EAP are negligible. Calibrations: CNV=20μV, EOG=100 μV.

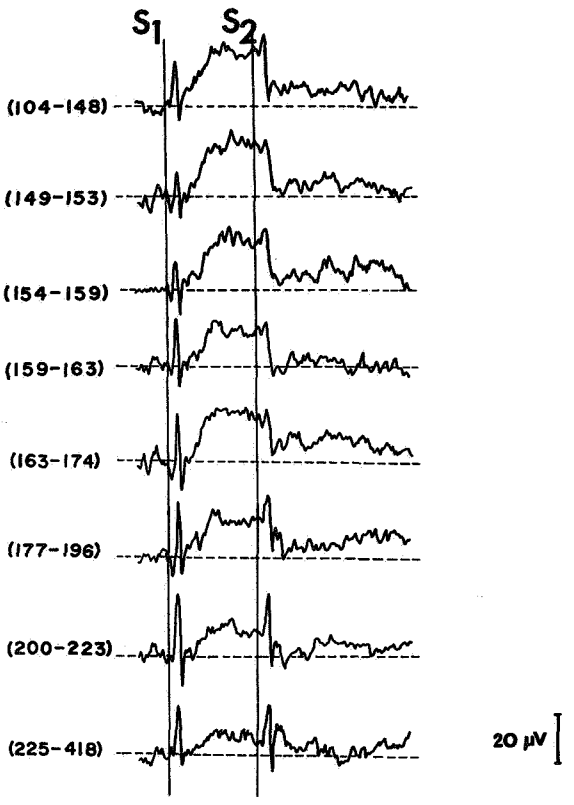

FIGURE 4–34.—Correlation between spontaneous variability in RT and the amplitude of CNV preceding the motor response. Each tracing is the average of CNV from 12 trials, having the range of RT shown at left in msec (negative up).

necting the x does not really imply anything about sequence. Is that just so you can find all the x?

DR. HILLYARD: Yes; RT and CNV of different magnitudes were distributed evenly throughout the series of trials. Therefore, the speed of RT was independent of sequential position.

DR. CALLAWAY: I want to ask Dr. Vaughan a question concerning that figure. Do you think that if you had averaged backwards from the response that this would have disappeared?

DR. VAUGHAN: I would like to ask Dr. Hillyard if he did that.

DR. HILLYARD: As I understand your argument, it is that there is greater variability in RTs that are longer, and hence the peak latencies of CNV associated with longer RTs would be more dispersed in time relative to the triggered epoch of computer averaging. Thus, CNV

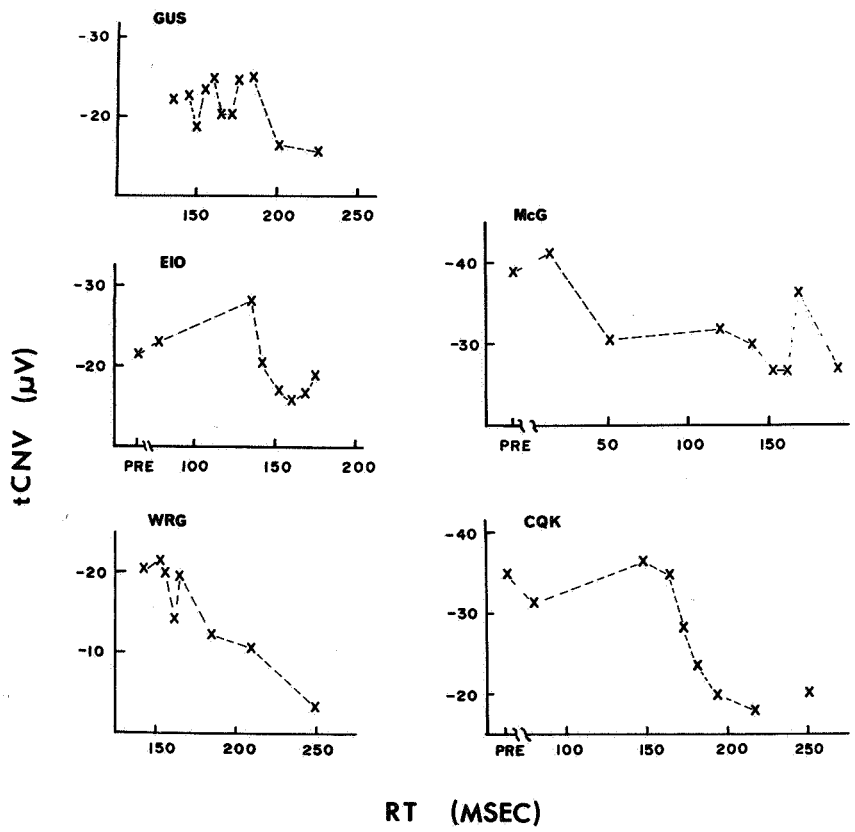

FIGURE 4–35.—Various forms of the relationship between increasing RT and decreasing tCNV in five different subjects. "Pre" indicates trials on which erroneous, premature lever presses were made before the onset of S.

of variable latency would not sum to their full amplitude because of reduced time-locking of the response with the averaging epoch.

DR. VAUGHAN: This is true. It may not be the whole story. It may be that there is in addition, of course, a true relationship between speed and the CNV.

DR. HILLYARD: I don't believe that that criticism applies to these data, because the CNV were summed in blocks having relatively constant response latencies (RT); this is equivalent to summing with the lever press used as the time-locked reference point. Furthermore, the CNV waveforms shown in figure 4–34 had reached plateau amplitudes well before S_2 arrived, and a plateau is not reduced in amplitude upon averaging by small desynchronizations of time-locking. Also, the difference in magnitude between "fast" and "slow" CNV was so great that a small failure of time-locking could not have accounted for it.

DR. LINDSLEY: In the first records that you showed us, what was the subject instructed to do with his eyes? Were his eyes closed, was he in the dark, or what? You had him fixating, and there was no oculogram during the first part, during the S_1–S_2 interval. What was he doing when there was correspondence between the oculogram and the CNV?

DR. HILLYARD: The only instructions given were to press the lever as fast as he could.

DR. LINDSLEY: What were the eyes doing? Were the eyes closed, or were they fixating something?

DR. HILLYARD: The eyes were closed. Systematic involuntary eye movements only occur when the eyes are closed, and the large correlation between the CNV and the EOG deflection is seen only then. If the eyes are open but not fixated, there will be irregular eye movements that are not closely synchronized with the CNV, but nonetheless can contribute artifact to it.

DR. COHEN: When you determined eye movement effects on the vertex, what were the instructions to the subject to determine the eye artifact on the vertex?

DR. HILLYARD: I had them make small square-waves of eyeball rotation; the eyes were rotated downward at S_1 and upward at S_2. This produced a square-wave deflection in the EOG, with its amplitude and polarity dependent upon the direction and extent of the eye movement. A square-wave of EAP was concurrently induced in the vertex-mastoid montage, at a reduced level of amplitude, of course.

DR. COHEN: That sounded very similar to one of my records (fig. 4–13) where the instruction was to move the eyes, and this produced a true CNV.

DR. HILLYARD: That is right.

DR. COHEN: So it may be very possible that some of your eye movement effect at the vertex is true CNV, in addition to possible ocular movements.

DR. HILLYARD: That is correct; the total potential shift induced at the vertex by voluntary eye movements is a composite of EAP and tCNV. The magnitude of the EAP depends upon the size of eye movement and is completely independent from the tCNV, which is increased when eye movements are made with greater speed and vigor. The details of the separation of the potential shifts during eye movements into the EAP and the tCNV are somewhat complicated (Hillyard and Galambos, in press), and I didn't want to go into that here.