the switch in stages were 12.3 and 60.1, respectively. This is a statistically significant difference ($P < .025$).

To be certain that the reaction was in fact due to the auditory-visual discrepancy and not to the shift in voice locus per se, we observed four additional infants. These infants, whose age range was from 28 to 56 days, received the experimental procedure as described above, except that the mother was not visible to the infant. She stood behind the curtain backdrop, completely hidden from the infant as she spoke to him. Voice locus and intensity were the same as in our original procedure. Two infants served at each of the 2-minute and 5-minute periods of stage 1. The sessions were filmed and scored for tonguing.

These infants remained calm throughout the procedure. There was almost no visible reaction to the lateral displacement of the mother's voice. The mean tonguing scores for the 45-second periods before and after the switch in stages were 15.5 and 20.8, respectively.

We observed three additional infants in order to determine whether the effect is dependent upon a speaker who is familiar to the infant. We thus repeated the experimental procedure but substituted a female laboratory assistant for the mother. Two infants served in the 2-minute condition of stage 1, and one infant in the 5-minute condition.

All three infants reacted with distress after the shift in voice locus. The mean tonguing scores for the 45-second periods before and after the switch in stages were 8.1 and 50.6, respectively.

We interpret our results as indicating that infants as young as 30 days are perceiving auditory and visual information within a common space. Perceived discrepancies within this space produce agitation and upset. The spatial dislocation thus is apparently a violation of the young infant's perceptual world, in which speaker and voice share the same spatial position. Further experiments will be required to determine which stimulus factors control the perception of spatial discrepancies. Communicative movements of the speaker, notably movements of the mouth and lips, are one obvious potential source of stimulus control. Michotte's (*4*) psychophysical investigations of causality perception in adults suggest that the perceptual unit is temporally defined; that is, the infants register the synchrony between lip movements and vocalizations, at least to the extent of differentiating the gaps between speech segments.

The lower age limit of our sample leaves open the possibility that the infants could have developed this capacity for spatial coordination during the first few weeks of postnatal life. The consistency with which our procedure produced visible distress in the infants does not, however, lend support to this interpretation. The expectation that voice and speaker are a spatial unit is presumably learned, but the learning would require the prior existence of a perceptual system that has access to and reliably coordinates information from separate modes. If the infant does not initially perceive the spatial integrity of such information, he must at least register the temporal correspondence between modes and, somehow, must begin to spatially coordinate the intermodal temporal unit. He must do so at a time in his life when his processing capacities are decidedly underdeveloped (*5*). If learning is to account for the auditory-visual spatial coordination, the learning process must necessarily be an extremely rapid and efficient one.

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## Attention-Related Increases in Cortical Responsivity Dissociated from the Contingent Negative Variation

Abstract. *Certain tasks which increase attention to stimuli also elicit the contingent negative variation and increase the amplitude of the P300 component of the sensory evoked response. Therefore it appeared possible that the contingent negative variation and attention-related increases in P300 are either confounded by artifact or generated by common neural mechanisms. The fact that we have recorded attention-related increases in P300 amplitude independent of corresponding systematic changes in contingent negative variation indicates that neither of these possibilities is correct. The two phenomena are independently variable modulations of cortical activity.*

When a sensory stimulus is made sufficiently interesting or relevant to the performance of a task, the cortical response (but not the peripheral nerve response) evoked by that stimulus is larger than it is when the stimulus has no significance for the subject (*1*). This increase in amplitude is particularly large and reliable in the so-called P300 wave, a late positive evoked response component best recorded slightly anterior to the vertex in man and having a maximum between 230 and 360 msec after the stimulus (*1–3*). We shall refer to attention-related increases in the amplitude of this component as the P300 effect. Recently several authors (*4–6*) have pointed out the similarity between conditions used to demonstrate the P300 effect and those conditions which produce a surface-negative baseline shift in the human electroencephalograph (EEG), commonly called the contingent negative variation (CNV) (*7–8*). The P300 effect appears to occur when there is either a preparatory increase in concentration immediately before the task stimulus, a condition which also elicits CNV, or a reactive decrease in concentration after a response to the stimulus, which occasions a positive-going shift in the CNV back to the EEG baseline before the trial.

The probable covariance of CNV with the P300 effect has suggested two hypotheses about the possible relationship of these phenomena. One hypothesis stated by Karlin (*4*) proposes that the P300 effect is an artifact of averaging the evoked potential at the same time that the CNV is returning to pretrial baseline, which thereby causes summation of the resulting positive-going baseline shift into the sensory response. This type of artifact was demonstrated by Donchin and Smith

(*5*). In their experiment the CNV developed during the 2-second intervals between trials of a vigilance task only when a stimulus was predictable and relevant to the task; the CNV baseline return summed with the P300 wave, increasing P300 amplitude selectively on task-relevant trials. Any design allowing the subject differential prediction of a significant or relevant stimulus would be similarly confounded unless the CNV baseline return was delayed beyond the occurrence of the P300 wave. Furthermore, since the latency of CNV baseline return varies with reaction time in tasks requiring a decision (*9*), it is possible that differences in the latency of CNV baseline return could confound the P300 effect when differential preparation is precluded but a differential reaction to the relevant stimulus is permitted (*3*). If the CNV baseline return is the sole cause of the apparent P300 increase, then prolonging the CNV beyond the occurrence of P300 should abolish the P300 effect. The second hypothesis, stated by Nataanen (*6*) holds that the P300 effect reflects a genuine increase in the P300 wave, but that this increase and the CNV are both produced by the activity of a common electrocortical activation system. This theory predicts that the magnitude of the P300 effect should be directly proportional to the difference in CNV amplitude between experimental and control groups, even after controls for artifact due to the baseline return latency of CNV. McAdam (*10*) reported a rough correlation between CNV and a late evoked response component latency. He interpreted this as evidence in support of the activation theory. However, his placements of bipolar electrodes did not permit direct measurement of the P300 wave. Moreover, he compared "CNV" trials with "non-CNV" trials which differed on the same covarying attentional factors under study here. Thus his results could have been due either to an interaction with CNV or to attentional variables possibly unrelated to CNV. A preliminary study by Donald (*11*) suggesting that CNV and attention-related changes in P300 might be independently variable led us to a more comprehensive and explicit test of these theories.

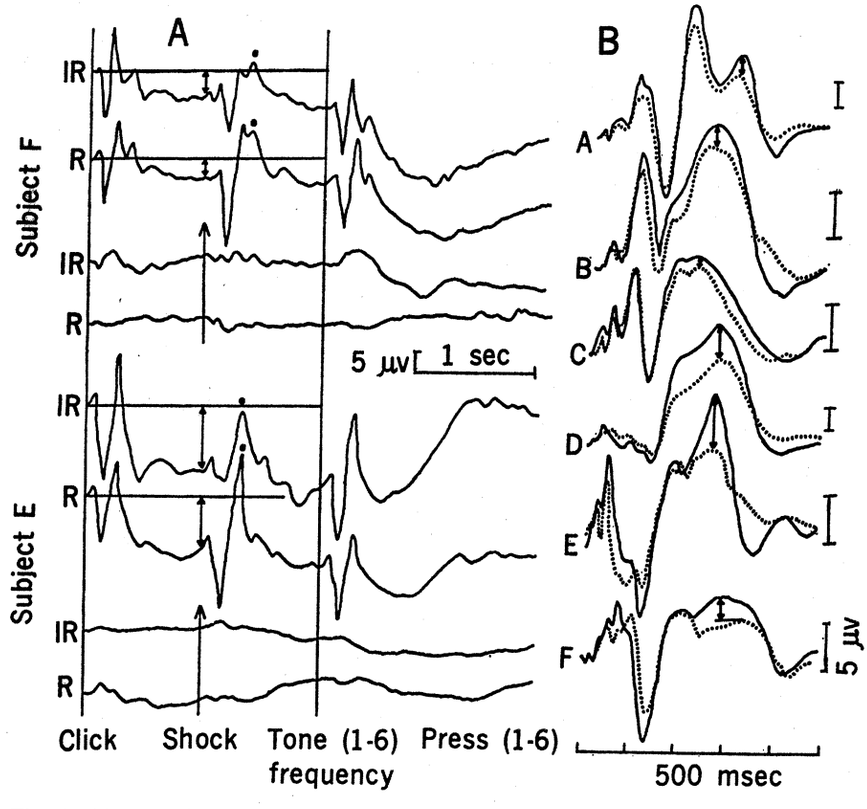


Fig. 1. (A) Contingent negative variations and eye-movement potentials from single sessions (48 summations) for two subjects. IR, shock-irrelevant trials; R, shock-relevant trials. The upper and lower pairs of R and IR have been matched on the basis of equal CNV amplitude. The bottom two traces of each pair are averages of eye-movement. Relative negativity at vertex is downward in the CNV averages. Reference electrode is at left earlobe. Amplifier gain is similar in all traces. The amplitude of CNV was measured just before shock (arrows). The P300 wave following shock is indicated by dots. (B) Averaged somatic evoked potentials (432 summations per trace) averaged over six sessions for each subject. Relative negativity at contralateral hand area is downward. Reference electrode is at right earlobe. Arrows indicate where latency and amplitude measurements of P300 were made. Dotted line, IR; solid line, R.

In this study we elicited the P300 effect while both the CNV amplitude and the latency of its baseline return were controlled. Because of the similarity of the conditions used to produce the CNV and the P300 effect, we did not use a "non-CNV" condition. Instead, using a paradigm known to elicit CNV's of similar amplitudes under both stimulus-relevant and stimulus-irrelevant conditions (*9*), we tested each subject repeatedly under both conditions, and measured the magnitude of the P300 effect with CNV amplitude held constant and the latency of CNV baseline return delayed well beyond the occurrence of P300. Subjects were required to perform a difficult task of tone identification with a 2-second foreperiod signaled by a warning click (*12*). During the foreperiod, an unpredictable stimulus (shock of the left median nerve at an intensity 3 ma above the threshold for the thumb twitch) was delivered 500, 1000, or 1500 msec after the warning click on 75 percent of the trials. On the remaining trials, distributed randomly throughout the session, no shock was delivered. Since the subject could not predict when or if he would be shocked, the shock could not serve as a timing cue. The shock was made irrelevant (IR) to the task on certain sets of trials, and relevant (R) on others. On R trials, occurrence of the shock signaled the subject to identify the tone; but if no shock was delivered he had to press a "no-shock" button rather than identify the tone. Thus on those trials when the shock-evoked potentials were measured, subjects were performing the same tone-discrimination task under both R and IR conditions with exactly the same stimulus parameters, the only difference being the relevance of the shock to the task. Since the button press occurred at least 2000 msec after the shock, CNV baseline return was delayed well beyond the shock-evoked response-averaging epoch. One female and five male adults were each tested in a min-

imum of three IR and three R sessions in a counterbalanced order. Each session included 192 discrimination trials, 48 for each of the three intervals between click and shock and 48 trials with no shock in a constrained random order. Intertrial intervals were random and ranged from 8 to 14 seconds, averaging 11 seconds, long enough to minimize the use of discrimination stimuli as timing cues for succeeding trials. The presentation of stimuli, sampling, and storage of data were achieved by a LINC computer program described in detail elsewhere (*13*). The CNV, eye movements, and shock-evoked potentials were simultaneously averaged on-line and stored separately for each click-shock interval. The CNV was recorded from a nonpolarizable Ag-AgCl (*14*) vertex electrode referred to the left earlobe (*15*). Similar electrodes were placed above the left supraorbital ridge and on the left external canthus to record eye movement potentials. The subject's eyes were fixated throughout the sampling epochs to minimize eye movements. The electrodes were connected to low-level d-c amplifiers (Grass model 7PI), and recording was delayed at least 20 minutes after attachment of the electrode to stabilize baseline drifts (*16*). Shock-evoked responses were recorded from a silver disk electrode placed over the contralateral hand area (4 cm posterior, 7 cm lateral to the vertex) referred to the right earlobe. The disk electrodes were connected to an a-c amplifier (Grass model 7P5A) set at a gain of $10^4$ with upper and lower one-half amplitude frequency settings at 500 hz and 0.15 hz, respectively. Averaging epochs were 3840 msec for CNV and eye movements and 500 msec for shock-evoked responses.

In all subjects CNV developed shortly after the warning click and did not terminate until 1 to 3 seconds after the stimulus for tone discrimination. So that we might compare P300 amplitudes of R and IR trials without the confounding effect of uncontrolled variation in CNV amplitude, each subject was tested until he had produced three R and three IR sessions with approximately equal ranges of CNV amplitudes. Each subject's R and IR trials for each click-shock interval were then matched in pairs by ranking the CNV amplitude separately for R and IR sessions and then pairing trials of corresponding rank CNV amplitudes. Paired comparison *t*-tests (*17*) showed no significant differences in CNV amplitude between matched R and IR trials. Two matched pairs are shown in Fig. 1A for the 1-second click-shock interval. Note that the records of eye movement show none of the abrupt d-c shifts visible at the vertex, that CNV amplitude and waveform are similar under both R and IR conditions, and that no blocking of the CNV followed the shock (*18*). Differences between matched R and IR trials in P300 latency and base-to-peak amplitude (measured by a LINC quantification program) were evaluated with paired comparison *t*-tests (*17*). Although there were individual differences in latency and amplitude of P300, the reliability for each subject was high, and comparisons were always made with each subject serving as his own control. Although the two groups did not differ significantly in CNV amplitude, and although CNV termination occurred long after the response to the shock in both groups, the P300 wave was higher in amplitude on R trials at both the vertex ($P < .005$) and the contralateral hand area ($P < .01$). This increased amplitude is visible both in comparisons of single sessions (Fig. 1A) and in the shock-evoked responses of each subject averaged across all sessions (Fig. 1B). The P300 and CNV amplitudes of R and IR trials averaged across all subjects are shown graphically in Fig. 2A. The magnitude of P300 enhancement on R trials ranged from 10 to 35 percent. There were no significant differences in P300 latency between R and IR groups. In order to compare the magnitude of the P300 effect with the differences in CNV amplitude, Spearman rank correlations were calculated between the ratios of R to IR for CNV and P300 for each subject (*19*). Four subjects showed no significant correlation (.37, .39, .14, and .19), one showed a positive (.77, $P < .05$), and one showed a negative (− .60, $P < .05$) correlation. The lack of correlation indicates independence of P300 effect from differences in CNV amplitudes between R and IR trials. As a further test of this independence, the R and IR trials were each subdivided for each subject into high (above median) and low (below median) CNV groups and two comparisons were made (Fig. 2, B and C). In the first comparison CNV was higher in the R group; in the second comparison CNV was higher in the IR group; in both comparisons P300 amplitude was significantly larger on R trials ($P < .05$). In neither case was the P300 effect reversed or eliminated, even though in both comparisons the CNV amplitude of the high group was 150 to 200 percent the amplitude of the low group (*20*). A final statistical test involved comparison of the relative magnitudes of the P300 effect at click-shock intervals of the 500, 1000, and 1500 msec. The P300 amplitude was significantly ($P < .001$) higher at intervals of 1000 and 1500 msec than it was at 500 msec, which probably indicates more recovery of P300 from the cortical response to the warning click at the longer delays. The recovery of P300, expressed as the percentage increase in amplitude of P300 from the 500 to the 1500 msec delay, was 17 percent on IR trials, and 26 percent on R trials; this indicates a significantly ($P < .05$) faster recovery on shock-relevant trials.

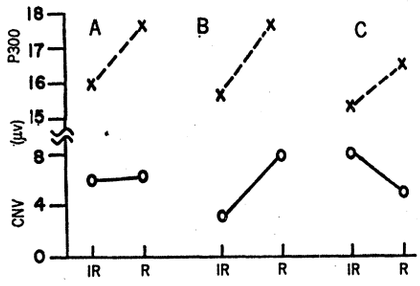


Fig. 2. Mean amplitudes of contingent negative variation and P300 wave on shock-relevant (R) and shock-irrelevant (IR) trials. (A) The R and IR groups have equal CNV amplitudes. (B) The R group has 200 percent the CNV amplitude of the IR group. (C) The IR group has 160 percent the CNV amplitude of the R group. In (A–C) the P300 wave is always larger in the R group.

In summary, the P300 component was significantly higher in amplitude on stimulus-relevant trials, regardless of whether CNV amplitude on those trials was made to be higher than, lower than, or similar to CNV amplitude on stimulus-irrelevant trials. We conclude that there is no absolute proportionality between CNV amplitude and magnitude of the P300 effect, and that some process not represented by CNV waveform or amplitude produced the P300 increase on shock-relevant trials. It remains to be established whether this residual evoked potential increase is present in all of the paradigms thought to elicit the P300 effect and to what extent previous studies were confounded by the baseline return factor. The suggestion that the P300 effect is nothing but an artifact of the CNV baseline re-

turn is clearly disproven. Similarly the theory that CNV and the P300 effect reflect the action of a common "activation" mechanism must be rejected. It is likely that all EEG activity is correlated with the gross arousal level of the subject and that this common element may introduce covariances between different phenomena derived from the EEG. But another process is required to explain the dissociability of CNV from the P300 effect. Apparently there are at least two independently variable modulators of cortical electrical activity correlated with moment-to-moment efficiency in human performance. Although these data are not addressed directly to Nataanen's (*6*) or Karlin's (*4*) speculations about the psychological processes underlying CNV and the P300 effect, such speculation must now account for the independent variability of these two phenomena.

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12. For the identification task a 0.1-msec warning click was followed 2 seconds later by a 32-msec tone pip of either 1000, 1100, 1200, 1300, 1400, or 1500 hz, which the subject identified by pressing one of six corresponding keys. A click train (15 cycle/sec) immediately followed the offset of the tone pip; this train could be terminated immediately by a correct key press; a response delayed more than 3 seconds was recorded as a failure, and the click train was automatically terminated. The click train made the task more difficult by partially masking the tone, and its immediate termination indicated a correct response. Responses and failures were recorded on a teletype printout.
13. W. R. Goff and G. D. Goff, *Behav. Res. Method Instrum.* **2**, 77 (1970).
14. Beckman Instruments, Inc., Fullerton, Calif. Polarization voltage stabilized at approximately 0.25 $\mu$v/hour within 20 minutes after application. For d-c recording, electrode impedances were kept below 2500 ohms.
15. Topography data on five of the subjects indicated that in this paradigm the foreperiod CNV is largest at the vertex, falling off sharply in amplitude at $P_z$, $T_3$, $T_4$, and FP electrodes (International 10-20 System).
16. On about 20 percent of the recording sessions a steady baseline drift persisted throughout the experiment. On these occasions the baseline slope was calculated and subtracted from the averaged records in the quantification procedure. This permitted a more accurate measurement of CNV amplitude than would have been possible if amplifier time constants had been used. Large irregular baseline changes were occasionally observed; these were usually due to movement artifact which could be eliminated by keeping electrode impedances below 3 kilohms.
17. W. J. Dixon and F. J. Massey, Jr., *Introduction to Statistical Analysis* (McGraw-Hill, New York, ed. 2, 1957), p. 126.
18. Compare J. Tecce and N. Scheff, *Science* **164**, 331 (1969) and C. McCallum, in *Attention in Neurophysiology*, C. R. Evans and T. B. Mulholland, Eds. (Butterworths, London, 1969), p. 40. Both references report CNV blocking by distracting stimuli. Such blocking was not observed here, presumably because no task-irrelevant operations were linked to the shock stimulus.
19. The ratios of R to IR for P300 and CNV were calculated from measurements of amplitudes from base to peak.
20. Figures 2B and 2C suggest a positive within-condition correlation between CNV and P300 amplitude on R trials. In fact, although no subjects yielded significant Spearman rank correlations between CNV and P300 on IR trials, three of the six subjects did yield significant ($P < .05$) correlations on R trials. Since performance of the task requires attention to the shock on R trials, but not on IR trials, these correlations probably reflect the established covariance of both P300 on IR trials, three of the six subjects within conditions (*4*, *8*). This covariance would not be predicted in the IR condition since the P300 wave to the shock is unrelated, whereas CNV is related to performance efficiency. In any case, the within-condition correlation is borderline and gross variations in stimulus significance across the R and IR conditions, although not altering CNV amplitude, introduce P300 changes so large as to destroy any absolute proportionality between CNV and P300.
21. Supported by the Veterans Administration, PHS grant M-05286 and NSF grants GB-3919 and GB-5782. We thank Thomas C. Fisher for technical assistance.

6 January 1971

## Potassium-Uranium Systematics of Apollo 11 and Apollo 12 Lunar Samples and of Some Deep Earth Rocks

Fanale and Nash have discussed the K-U systematics of lunar, chondritic, and terrestrial samples (*1*). They discerned distinct trend lines for Apollo 11 and Apollo 12 samples and for terrestrial rocks and showed that chondrites are not members of these trend lines. They pointed out that, if the earth and the moon were formed from the same material, then the lunar rocks

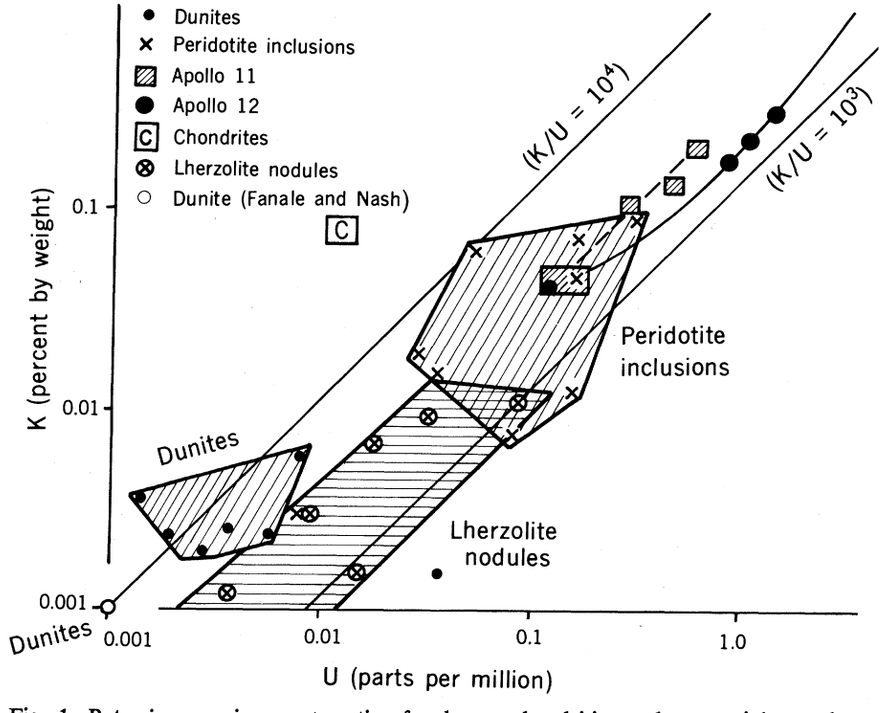


Fig. 1. Potassium-uranium systematics for lunar, chondritic, and terrestrial samples. Lunar data from figure 1 of Fanale and Nash (*1*) (see their references for original data); chondritic data from Fisher (*5*); dunite and peridotite data from Fisher (*2*); lherzolite data from Green *et al.* (*3*).