cycle by cycle, (iii) the biological event coincides with the pacer signal only when the respective cycling frequencies are equal (Fig. 1a) (*15*), and (iv) phase leads and lags occurring with unequal pacers approximate the respective differences between pacing and free-run periods. Further, the timer-resetting idea, whether in the restricted format which our best firefly data appear to approach as a limit, or modified to cover different oscillator behaviors (*14*), offers the following persuasive advantages over most previously suggested mechanisms for animal synchronization (*16*). First, it accommodates entrainment to both faster and slower pacers in a single mechanism (*17*). Second, it explains not only the fact of consistent phase differences between pacer and animal during entrainment but their magnitudes and directions. Third, it provides for entrainment to a wide variety of foreign rhythms without requiring (*7*) that the animal be able to discriminate the sequence of his act in relation to the acts of neighbors in the synchronizing community. Finally, it allows the animal to duplicate a variety of pacer cycles without actually changing the intrinsic period of its endogenous timer.

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8. Ability to change flash frequency has also been suggested by N. Baldaccini, V. Fiaschi, and F. Papi [*Monit. Zool. Ital.* **3**, 239 (1969)].
9. We thank the University of California, San Diego, for support, travel, and facilities on the expedition. J.B. thanks the American Philosophical Society (Penrose Fund grant 5017) and the National Geographic Society for technical support. J.F.C. had additional support from ONR contract N00014-69-A-0200-8006 and from the University of California Faculty Research Fund. F.E.H. received additional support from a faculty grant, University of Texas.
10. The *Pteroptyx* fireflies studied were from three localities on New Britain, Territory of Papua and New Guinea: Navuneram Village and Keravat, 10 miles apart, and Cape Hoskins, 100 miles to the southwest. By present criteria all belong to the same species. We thank Lesley A. Ballantyne and Miriam R. McLean for identifying the fireflies investigated. Taxonomic details will be found in L. Ballantyne and M. McLean, *Trans. Am. Entomol. Soc.* **96**, 223 (1970).
11. The minimum conduction plus organ excitation delay, measured during strong electrical stimulation of the brain, and presumably bypassing any central delay, varies from 70 to 110 msec in various species [(*1–3*) and present work]. Stimulation by light in the eye usually adds in the order of another 100 msec, hence almost certainly involves more than just visual processing delay.
12. We have no direct evidence of what "resetting the endogenous timer" means in terms of the neuroeffector control mechanisms of flashing. However, in another insect, the roach, the oscillator that controls the circadian rhythm of locomotion has been localized in the optic lobes of the brain, and the eye has been shown to be the organ by which the oscillator is entrained by light [J. Nishiitsutsuji-Uwo and C. S. Pittendrigh, *Z. Vergl. Physiol.* **58**, 1, 14 (1968)].
13. Phase differences between pacer and timer during entrainment have been observed in a variety of other biological systems, for example, crustacean heart [D. M. Maynard, *Biol. Bull.* **109**, 420 (1955)], circadian rhythms [C. S. Pittendrigh and D. H. Minis, *Am. Nat.* **98**, 261 (1964); A. T. Winfree, *J. Theoret. Biol.* **28**, 327 (1970)], and pacemaker neurons [D. H. Perkel, J. H. Schulman, T. H. Bullock, G. P. Moore, J. P. Segundo, *Science* **145**, 61 (1964)]; however, we defer comparisons until the full presentation of our findings.
14. Time relations during resetting depend strongly on the model chosen for the relaxation oscillator. In the minimal or "ideal" situation, in which the timer charges at a linear rate throughout its cycle, triggers always at a fixed level of excitation, and discharges in all-or-none fashion, response time must equal the free-run period exactly, and leads and lags in steady-state pacing must be symmetrical with respect to the free-run period. However, response times shorter or longer than the free-run period could result if the pacer discharged the timer respectively less or more completely than the normal endogenous process or at a slower or faster rate. Similarly, linear phase versus pacer plots (Fig. 2) with slopes different from 1.0, or with inverse or curvilinear relations, can be accommodated readily. Examples of such modified resettings in other firefly species will be given elsewhere.
15. With a pacer period equal to the free-run period it is sometimes not possible, except in the introductory transient, to be absolutely sure that the firefly is actually entrained since equal rhythms could stay in association for a long time by chance. There can be doubt also with pacer cycles shorter than the free-run firefly period (unless there are several different pacers with different apparent lags, as in Fig. 2) because entrainment with constant lag could represent a series of direct sequential responses rather than successive resettings [see figure 17B in (*4*)]. In this connection, the name "paced," applied by Buck and Buck (*7*) to synchrony involving minimum latency triggering by a premature flash, should be renamed "led" synchrony to distinguish it from the present hypothesis of pacer action.
16. Many of the older theories (*6*) can also be questioned for various a priori reasons (*7*) or for lack of precise measurement. For example, a visual observation of exact synchrony in fireflies is of dubious value in relation to possible entraining mechanisms in view of the report that the human eye cannot, under field conditions, distinguish asynchrony between flashes closer together than about 110 msec (*8*).
17. T. J. Walker [*Science* **166**, 894 (1969)], studying entrainment of tree crickets to series of artificial chirps, found the response chirps earlier than the pacer chirps when the pacer rhythm was slower than the cricket's and later when faster. He reported that crickets synchronize "by responding to the preceding chirp of their neighbors" but concluded that the period shortening ("S response") and period lengthening ("L response") are due to qualitatively different mechanisms.

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Hemispheric Asymmetry of Electrocortical Responses to Speech Stimuli

Abstract. *In a group of normal adults, averaged cortical evoked responses to natural speech stimuli were recorded from scalp electrodes placed symmetrically over the two cerebral hemispheres at frontal, Rolandic, and temporoparietal leads. The amplitude of the most prominent component was consistently larger in left hemisphere derivations, with the major hemisphere difference observed in the temporoparietal records. These electrophysiological measures may be sensitive indicators of hemispheric specialization of function.*

It has long been recognized that the neural structures of the left cerebral hemisphere play a dominant role in the mediation of human language. The major source of evidence has come from patients with localized cerebral lesions. Language impairment is much more likely with left hemisphere involvement than with lesions of comparable size, nature, and locus on the right. Over a century ago Paul Broca demonstrated that aphasia was associated with lesions restricted to the third frontal convolution of the left hemisphere. Serious and lasting language defects occur when lesions involve the gyri surrounding the posterior tip of the Sylvian fissure. This region has been considered by some authors as the indispensable speech cortex (*1*). One of the posterior speech regions, namely, the classical area of Wernicke, is significantly larger by gross anatomical measurement in the left hemisphere (*2*).

This report concerns the applica-

tion of electrophysiology to the study of the neural organization of language. The method involves the mapping over the two hemispheres of the averaged cortical potentials evoked by the sounds of human speech. In a group of normal adults, such responses were not symmetrically distributed over the two hemispheres. The electrical responses recorded from the left hemisphere were larger than those recorded from the right hemisphere with the major differences observed between the corresponding temporoparietal regions.

Recordings were obtained from chlorided silver disk electrodes placed at frontal, central, and temporoparietal locations over both hemispheres (*3*). These points were selected because they overlie cortical areas important for speech reception and production. Bilaterally, the central (or Rolandic) leads lie over the sensorimotor field of the muscles of vocalization. On the left, and presumably speech-dominant, side, the recording field of the frontal electrode includes Broca's area while the temporoparietal lead includes activity from Wernicke's area.

A linkage between the earlobes served as a common reference. The brain potentials were amplified and recorded simultaneously with a 14-channel system (Ampex DAS-100) whose band-pass was flat between 1 and 100 hertz.

The subject's task was to monitor an audio tape and report each stimulus heard. The tape consisted of five nonsense words ([pi], [pa], [əpik], [əpak], [a]) which were dubbed 50 times each and recorded in quasi-random order at intervals averaging 8 seconds. The words were spoken by a male adult with clear articulation. Stimuli were presented binaurally through matched insert earphones (Grason-Stadler D58) at a comfortable listening level. The leading edge of the electrical record of each speech stimulus triggered a LINC computer which was used to average the 50 cortical potentials related to each of the five different signals.

Seven right-handed adults participated in the study (ages 21 to 39). Subjects were asked to delay their verbal report until a given stimulus ended and also to keep their eyes fixated upon a small circle 1 m in front of them. Electrodes were placed over lip muscles and about the eyes; activity from neither of these areas contributed to the averaged scalp-recorded responses during the analysis epoch of interest.

The most consistently observed feature of the electrocortical responses to natural speech stimuli were a prominent negative wave (here termed N1) commencing at 40 to 50 msec and reaching a maximum around 90 msec after signal onset and a succeeding positive-going event peaking at a mean latency around 160 msec (P2). Figure 1 presents the data obtained from one subject and labels these components. Other wave components were frequently noted but were more difficult to measure reliably; these were an early positivity preceding the N1 wave (often polyphasic and of low amplitude) and a late negative-positive complex of variable latency which followed P2.

Analysis of variance was conducted separately for the N1, P2, and peak-to-peak (N1-P2) amplitudes. There were no systematic differences between the various speech stimuli for any of these measures in either amplitude or latency (*4*). Figure 2 presents the mean values at all locations for both N1 and P2 for the pooled observations.

The amplitude of the N1 component was significantly larger from left hemisphere recordings than from the right ($P < .01$). Left-right comparisons from homologous scalp locations revealed that the major hemispheric asymmetry occurred at the temporoparietal leads (*5*); left hemisphere responses were on the average 40 percent greater than those recorded from the corresponding right location ($P < .05$). Responses from the Rolandic area were larger than those recorded frontally for both hemispheres ($P < .05$). However, the gradients were different for the two hemispheres. On the left the largest N1 responses were obtained at the temporoparietal region, with progressive attenuation as measurements were made more anteriorly; temporoparietal responses were significantly larger than those from the frontal region ($P < .01$). In contrast, for the right hemisphere the largest responses were recorded at the Rolandic site, with a significant falloff in amplitude in both anterior (frontal) and posterior (temporoparietal) directions ($P < .05$).

The positive-going wave peaking at around 160 msec (P2) was rather uniformly distributed over the various cortical areas. No consistent differences between the hemispheres or among the

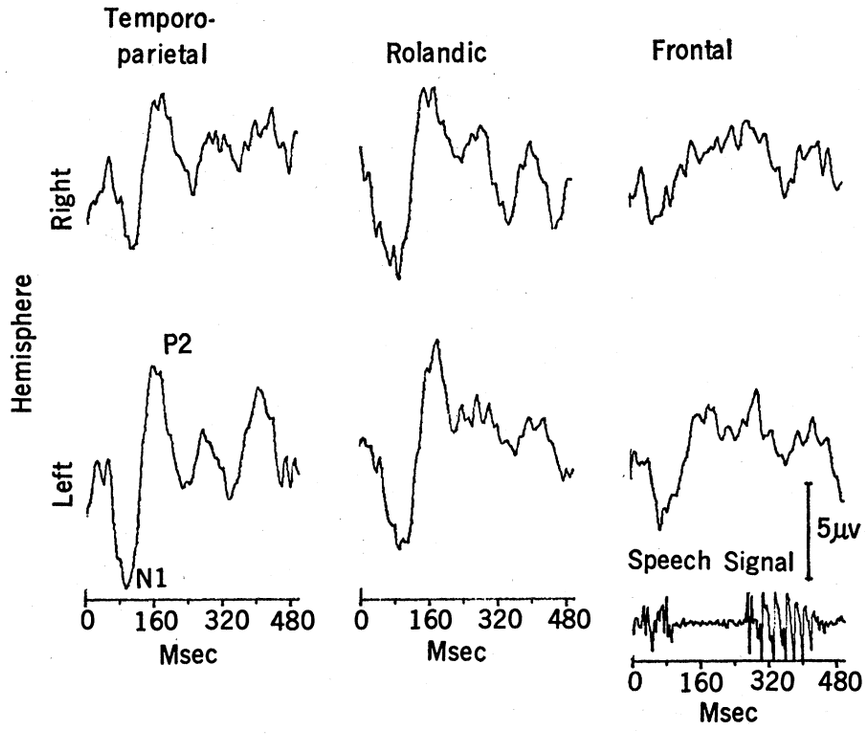

Fig. 1. Averaged evoked responses to the verbal stimulus [əpik] recorded from homologous left and right hemisphere locations in a normal male adult. Lower left response indicates the N1 and P2 components. At bottom right is filtered electrical record of speech signal. Sweep: 500 msec. Calibration: 5 μv.

electrode locations were noted in the present series. Generally, the average amplitude of the P2 wave was smaller than that of the N1 component. The greatest disparity between N1 and P2 was observed at the left temporoparietal location.

As just shown, the information which distinguished between the two hemispheres in electrical response to speech stimuli was basically carried by the amplitude of the N1 rather than the P2 component in this series. Peak-to-peak measurement tended to obscure hemispheric differences (*6*). Peak-to-peak amplitudes were slightly larger from the left temporoparietal region (mean 8.6 μv) when compared with the right (mean 7.5 μv). This represents a difference of 14 percent ($P < .10$) and contrasts with the 40 percent differential between these two areas when the N1 component was measured separately. Electrode location was a significant variable for the peak-to-peak index ($P < .01$). Frontal responses were markedly smaller than those recorded centrally for both hemispheres. However, as with N1, different hemispheric gradients were observed. On the right, temporoparietal responses (mean 7.5 μv) were significantly smaller than the central ones (mean 9.5 μv) but of comparable level to those measured frontally (mean 7.2 μv). This right hemisphere pattern is similar to that reported by Vaughn and Ritter (*7*) for evoked responses to tones. (That investigation by Vaughn and Ritter did not deal with possible hemisphere differences.)

In contrast, at the left hemisphere the central and temporoparietal regions did not differ from each other. Furthermore, the left temporoparietal responses (mean 8.6 μv) were notably greater ($P < .01$) than the left frontal (mean 6.1 μv).

The hemispheric asymmetry of electrocortical responses to speech stimuli found in this study is congruent with clinical, anatomical, and behavioral evidence for a lateralized and localized speech-processing mechanism (*8, 9*). The most reliable and distinctive component of the response is the wave recorded as negative at the scalp surface which by its latency commences after the thalamocortical input to the auditory receiving areas. This neuroelectric event most probably represents a succeeding stage of cortical-cortical transmission from deep to more superficial layers. It is of greatest amplitude when recorded from the posterior speech region, the same area shown by Geschwind and Levitsky to be grossly larger on the left (*2*). The present observations suggest that specialized neural pathways are activated in the left temporoparietal cortex when speech sounds are perceived.

The corresponding posterior right cortex shows another type of specialization, namely, for certain types of visual perception. Here too there is a congruence of clinical and electrophysiological observations. Direct electrical stimulation of the right posterior cortex is much more likely to elicit visual experiences in neurosurgical patients than comparable stimulation of the left hemisphere (*1*). A relationship has been noted between right temporoparietal lesions and disorders of visual space perception (*9*). A number of studies from Beck's laboratory have shown that evoked responses to simple visual stimuli are larger in right (as compared to left) parietal recordings in normal children and adults. This asymmetry may be abolished by ingestion of alcohol. Further, these hemispheric differences are not observed in mongoloid or mentally retarded children (*10*). The findings of Beck *et al.* in conjunction with the present results suggest that hemispheric differences of electrocortical response may be of considerable utility for evaluating brain-behavior relationships in both normal and pathological conditions.

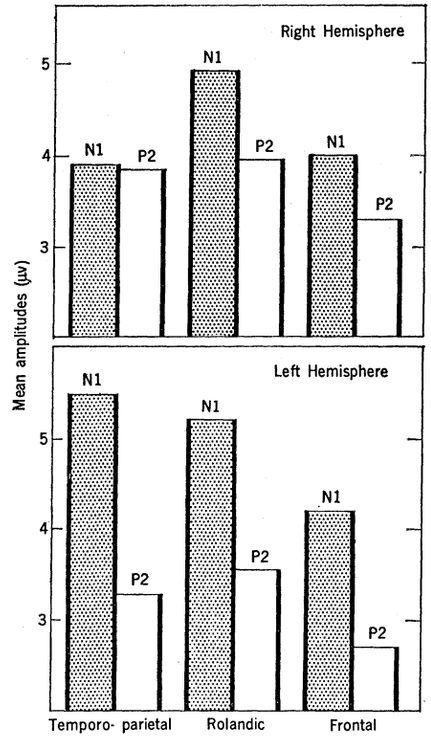

Fig. 2. Mean amplitudes (in microvolts) of N1 (stippled bars) and P2 (open bars) components at temporoparietal, Rolandic, and frontal recordings for left (bottom) and right (top) hemispheres. Each mean based upon 35 observations (five stimuli for each of seven subjects).

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3. Left and right hemisphere frontal, Rolandic, and temporoparietal electrode placements correspond respectively to F7 and F8, C5 and C6, and points midway between T5 and P3, and T6 and P4 of the International 10-20 System.
4. Detailed examination of the actual onset of triggering of the computer showed that speech tokens commencing with an initial vowel were detected within 2 msec; the electrical record of speech signals beginning with a plosive did not reach trigger voltage (set just above tape noise level) until 40 msec (for [pa]) or 56 msec (for [pi]). A LINC program permitted averaging of brain activity for a 1-second period preceding the trigger point as well as the subsequent epoch, and the data were corrected for latency.
5. At these leads, for each subject and each of the five speech stimuli the electroencephalographic baseline was integrated for the one-half second preceding signal onset, with preservation of the net algebraic sign. There was no consistent relationship between this presignal baseline value and the amplitude of the N1 component.
6. In a quite different setting (namely, a study of the effect of attentional variables upon evoked responses to clicks) R. T. Wilkinson and H. C. Morlock also noted that the component comparable to N1 was more closely correlated with experimental variables than was peak-to-peak scoring [*Electroencephalog. Clin. Neurophysiol.* **23**, 50 (1967)].
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