XVIII. COMMUNICATION BIOPHYSICS*

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A. SUMMARY OF RESEARCH – PUBLICATIONS

The following list of publications and theses presented by members of the Communications Biophysics Group during 1964 was omitted from Quarterly Progress Report No. 76, January 15, 1965.

Publications

1. J. S. Barlow, Evoked Responses in Relation to Visual Perception and Oculomotor Reaction Times in Man, Ann. N.Y. Acad. Sci. 112, 432-467 (1964).
2. J. S. Barlow, Some Statistical Characteristics of Electrocortical Activity in Relation to Visual-Oculomotor Tracking in Man, First Conference on Neurobiology: Feedback Systems Controlling Nervous Activities, Alfonso Escobar (ed.), Asociedad Mexicana de Ciencias Fisiologicas: Mexico City, D.F., 1964, pp. 385-408; Boletín del Instituto de Estudios Médicos y Biológicos, 21, 497-517 (1963).
3. J. S. Barlow, R. L. Rovit, and P. Gloor, Correlation Analysis of EEG Changes Induced by Unilateral Intracarotid Injection of Amobarbital, Electroenceph. clin. Neurophysiol. 16, 213-220 (1964).

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Publications (continued)

4. M. A. B. Brazier, Evoked Responses Recorded from the Depths of the Human Brain, Ann. N. Y. Acad. Sci. 112, 33-59 (1964).

5. M. A. B. Brazier, Information Carrying Characteristics of Brain Response, The Physiological Basis of Mental Activity, R. Hernandez-Peon (ed.), Suppl. 24, Electroenceph. clin. Neurophysiol., 1963, pp. 55-67.

6. A. Cavaggioni and M. H. Goldstein, Jr., Facilitation and Inhibition in the Visual System after Photic Stimulation, Archives Italiennes de Biologie (in press).

7. N. I. Durlach, Note on Binaural Masking-Level Differences at High Frequencies, J. Acoust. Soc. Am. 36, 576-581 (1964).

8. N. I. Durlach, Note on Binaural Masking-Level Differences as a Function of the Interaural Correlation of the Masking Noise, J. Acoust. Soc. Am. 36, 1613-1617 (1964).

9. M. Eden, A Note on Short-Term Storage of Information in Vision, Perceptual and Motor Skills 19, 93-94 (1964).

10. M. Eden, Taxonomies of Disease, The Diagnostic Process, John A. Jacques (ed.), Proceedings of a conference sponsored by the Biomedical Data Processing Training Program, University of Michigan, Ann Arbor, Michigan, April 1964, pp. 47-49.

11. Denise Albe-Fessard, J. Masson, Robert Hall, et Walter Rosenblith, Modifications au cours de la veille et du sommeil des valeurs moyennes de réponses nerveuses centrales induites par des stimulations somatiques chez le Chat libre, Compt. Rend. Acad. Sci. (Paris) 258, 353-356 (6 janvier 1964).

12. G. L. Gerstein and W. A. Clark, Simultaneous Studies on Firing Patterns in Several Neurons, Science 143, 1325-1327 (1964) (Abstract).

13. G. L. Gerstein and N. Y-s. Kiang, Responses of Single Units in the Auditory Cortex, Exptl. Neurol. 10, 1-18 (1964).

14. G. L. Gerstein and B. Mandelbrot, Random Walk Models for the Spike Activity of a Single Neuron, Biophys. J. 4, 41-68 (1964).

15. J. L. Hall II, Minimum Detectable Change in Interaural Time or Intensity Difference for Brief Impulsive Stimuli, J. Acout. Soc. Am. 36, 2411-2413 (1964).

16. N. Y-s. Kiang, Stimulus Coding in the Auditory Nerve and Cochlear Nucleus, Acta Oto-Laryngologica (in press).

17. N. Y-s. Kiang and R. R. Pfeiffer, Nerve and Nucleus: A Study of Stimulus Coding in the Initial Stages of the Auditory Nervous System, Science 146, 432 (1964) (Abstract).

18. R. R. Pfeiffer, Response Characteristics of Some Single Units in the Cochlear Nucleus to Tone-Burst Stimulation, J. Acoust. Soc. Am. 36, 1017 (1964) (Abstract).

19. R. R. Pfeiffer and N. Y-s. Kiang, Patterns of Spontaneous and Continuously Stimulated Spike Discharges in the Cochlear Nucleus of Anesthetized Cats, Biophys. J. (in press).

20. M. B. Sachs, Responses to Acoustic Stimuli from Single Units in the Eight Nerve of the Green Frog, J. Acoust. Soc. Am. 36, 1956-1958 (1964).

21. W. M. Siebert, Some Implications of the Stochastic Behavior of Primary Auditory Neurons, Kybernetik (in press).

22. D. C. Teas and N. Y-s. Kiang, Evoked Responses from the Auditory Cortex, Exptl. Neurol. 10, 91-119 (1964).

Theses

1. Richard S. Bair, Pitch of Short Tones, S. B. Thesis, Department of Physics, M. I. T.
2. Gerald J. Burnett, Masking Effects of Pure Tones in the Human Ear, S. B. Thesis, Department of Electrical Engineering, M. I. T.
3. Stephen K. Burns, Neuroelectric correlates of Behavioral Abnormalities Induced by Early Sensory Restriction, S. M. Thesis, Department of Electrical Engineering, M. I. T.
4. Robert R. Capranica, The Evoked Vocal Response of the Bullfrog – A Study of Communication by Sound, Sc. D. Thesis, Department of Electrical Engineering, M. I. T. (to be published as Special Technical Report Number 12 of the Research Laboratory of Electronics and as Research Monograph No. 33 by The M. I. T. Press, Cambridge, Mass.).
5. Ambrose W. Clay, The Effects of Clutter on the Echolocation System of Bats, S. B. Thesis, Department of Electrical Engineering, M. I. T.
6. Richard J. Clayton, Rhythmic Forebrain Potential Correlated with Cessation of Movement in the Rat, S. M. Thesis, Department of Electrical Engineering, M. I. T.
7. Harry S. Colburn, Time-Intensity Relations for Binarual Masking, S. M. Thesis, Department of Electrical Engineering, M. I. T.
8. Edwin G. Duffin, Electrical Properties of Indium-Filled Microelectrodes, S. B. Thesis, Department of Electrical Engineering, M. I. T.
9. Daniel S. Frischmuth, Effects of Control of Transmitted Signal Reception on Human Echolocation, S. B. Thesis, Department of Electrical Engineering, M. I. T.
10. John J. Guinan, Jr., The Transfer Characteristic of the Cat's Middle Ear, S. M. Thesis, Department of Electrical Engineering, M. I. T.
11. Harold W. Ingels, Noise Masked Threshold of Periodic Pulses, S. M. Thesis, Department of Electrical Engineering, M. I. T.
12. Kenneth C. Koerber, Spontaneous Activity in the Cochlear Nuclei of De-cochleated Cats, S. M. Thesis, Department of Electrical Engineering, M. I. T.
13. Clifford L. Laurence, Studies of Masking Noise Phase Delays in Binaural Masking Level Differences, S. B. Thesis, Department of Physics, M. I. T.
14. James P. McGaughy, Auditory Localization of Signals in the Presence of Masking Noise, S. B. Thesis, Department of Electrical Engineering, M. I. T.
15. Donald J. Mided, The Role of Psychophysics in the Law, S. B. Thesis, Department of Electrical Engineering, M. I. T.
16. Michael J. Murray, Analysis and Synthesis of the Mating Croak of the Bullfrog, S. B. Thesis, Department of Electrical Engineering, M. I. T.
17. John T. Motor, Binaural Detection of Signal with Angular Dispersion of Masking Noise, S. B. Thesis, Department of Electrical Engineering, M. I. T.
18. Richard F. Otte, A Miniature Microscope Extension Using Fiber Optics, S. M. Thesis, Department of Electrical Engineering, M. I. T.
19. Lawrence R. Rabiner, Binaural Masking – The Effects of Interaural Delay of the Noise on the Detection of Tones, S. M. Thesis, Department of Electrical Engineering, M. I. T.
20. Murray B. Sachs, Characteristics of Primary Auditory Neurons in the Green Frog. S. M. Thesis, Department of Electrical Engineering, M. I. T.

Theses (continued)

21. Abba Weinstein, Measurement of the JND in Intensity, S. B. Thesis, Department of Electrical Engineering, M. I. T.

22. George E. Wien, A Preliminary Investigation of the Effect of Head Width on Binaural Hearing, S. M. Thesis, Department of Electrical Engineering, M. I. T.

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B. MOTION OF MIDDLE-EAR JOINTS

To determine whether there is any significant articulation of the middle-ear joints, we measured the phase and amplitude of three points, (i) on the body of the stapes, (ii) on the long process of the incus, and (iii) near the end of the handle of the malleus. The measurements were made on anesthetized cats by a method described earlier.[1,2] The phase delays and amplitudes of the stapes and incus relative to the malleus are plotted in Fig. XVIII-1.

Most investigators (see, for example, Møller[3] and Zwislocki[4]) have stated that the malleus and incus move as a unit, whereas the stapes moves separately with slippage occurring in the incudo-stapedial joint. The data indicate, however, that there is movement in the malleo-incudal joint, and little, if any, in the incudo-stapedial joint. Figure XVIII-1 shows a slight increase in the incus

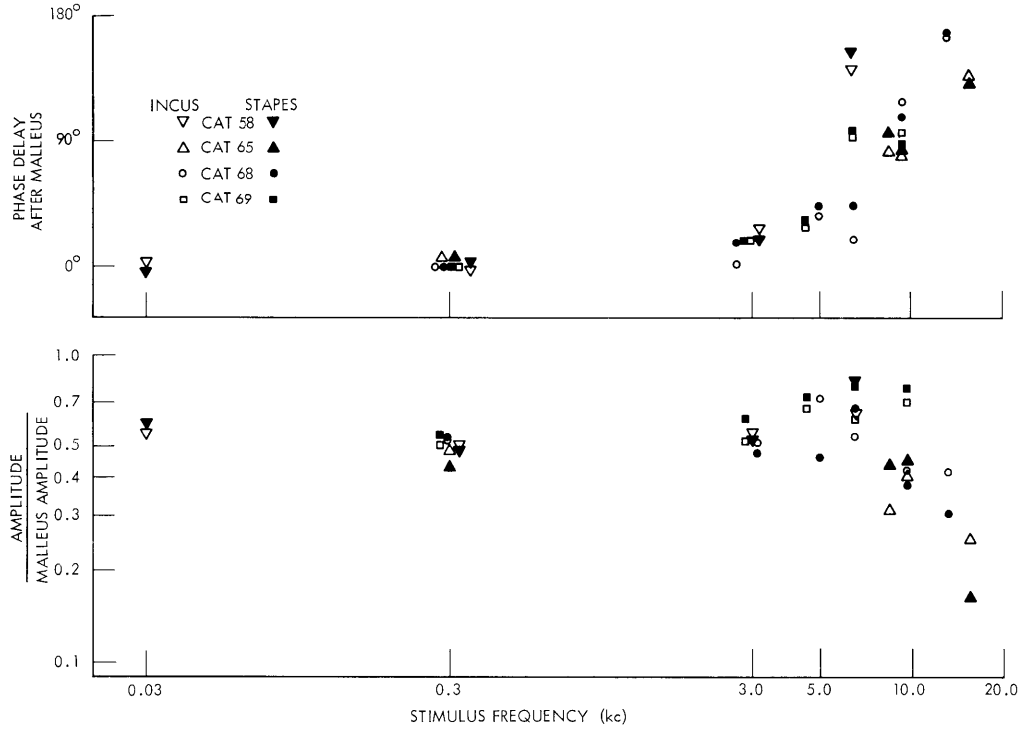

Fig. XVIII-1. Phase and amplitude of the incus and stapes relative to the malleus.

and stapes movement (relative to the malleus), in the 4-9 kc region, a decrease in the relative movement at higher frequencies, and an increase of the relative phase delay above 4 kc. All of these observations agree with a model of the middle ear in which the malleus and incus are coupled elastically.

J. J. Guinan, Jr., W. T. Peake

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C. DEPENDENCE OF EFFERENT INHIBITION OF AUDITORY NERVE RESPONSES ON INTENSITY OF ACOUSTIC STIMULI

Continuation of earlier studies[1] on the efferent olivocochlear bundle (OCB) has confirmed the impression that the effect of electrical stimulation of the crossed OCB is dependent on the strength of the acoustic stimuli. In particular, the data presented here indicate that stimulation of the OCB, which produces considerable reduction in the amplitude of auditory nerve responses to low-intensity clicks, has little, if any, effect on the neural responses to high-intensity clicks. This finding differs from results published by Desmedt.[2]

The data presented here were obtained from anesthetized cats with both middle-ear muscles cut. Responses were recorded from an electrode near the round window (reference on headholder) and from bipolar concentric electrodes in the auditory nerve. In all of the results presented here electrical stimulation of the crossed OCB consisted of a burst of 32 shocks at a rate of 400 per second. The pulse that produced the click was delayed 2 msec from the last of the shocks.

Figure XVIII-2 shows averaged responses recorded from the round-window electrode over a range of 80 db. Figure XVIII-3 is a similar presentation of responses recorded from the concentric electrode in the auditory nerve.

Figure XVIII-4 shows a plot of the amplitude of the first neural component, N_1, of the round-window click response as a function of click intensity. Figure XVIII-5 is a similar intensity series for the responses recorded from the concentric electrodes. It can be seen from Figs. XVIII-2 through XVIII-5 that throughout the upper 40 db of the

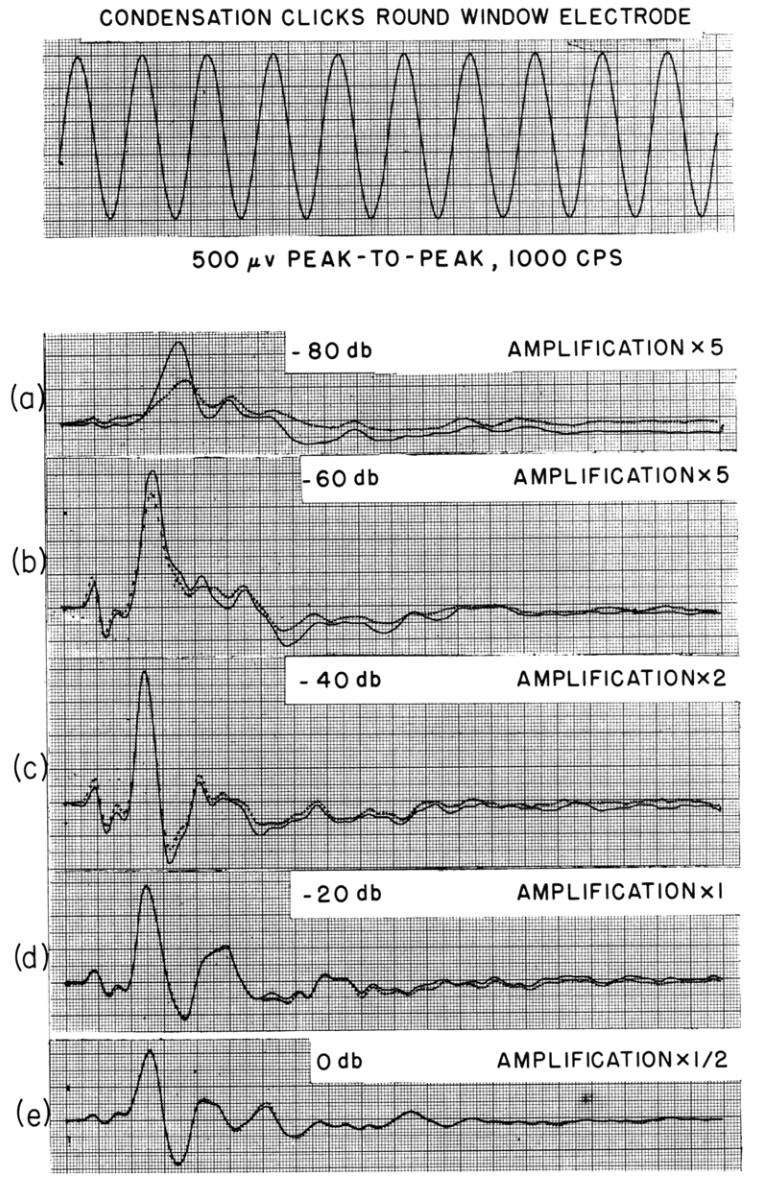

Fig. XVIII-2. Averaged responses to condensation clicks recorded at the round window. Solid-line traces are click responses with no OCB stimulation; dotted-line traces are responses to clicks presented 2 msec after the last of 32 shocks, 1.8 volts peak-to-peak, delivered at a rate of 400/sec to the crossed OCB at its decussation. Click intensity (re 4 volts to PDR-10 earphone, 0.1 msec square pulses) is indicated on each trace. The vertical scale amplification, relative to the calibration signal, accounts for number of responses averaged and changes in amplifier gain. Visual detection level for single responses, -90 db. Number of responses averaged (a) 64, (b), (c), (d), (e), 32.

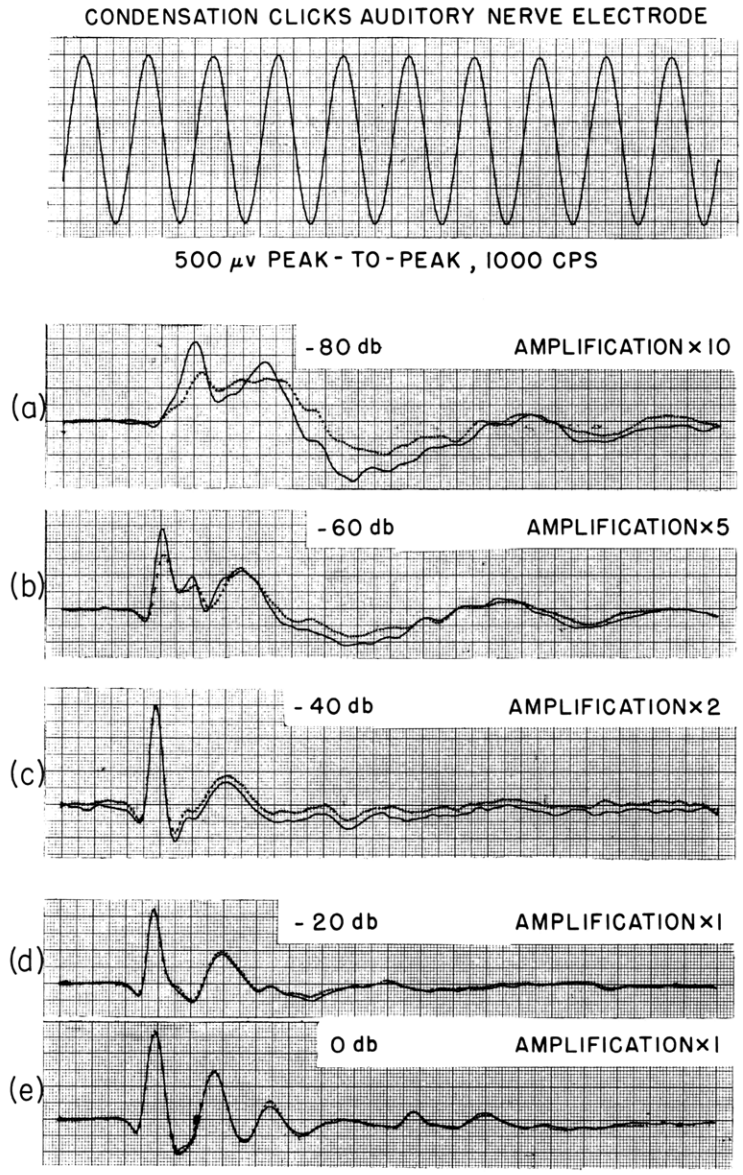

Fig. XVIII-3. Same as Fig. XVIII-2 except that responses (to same stimuli) were recorded from concentric bipolar electrode in the auditory nerve. Number of responses averaged (a) 64, (b), (c), (d), (e), 32.

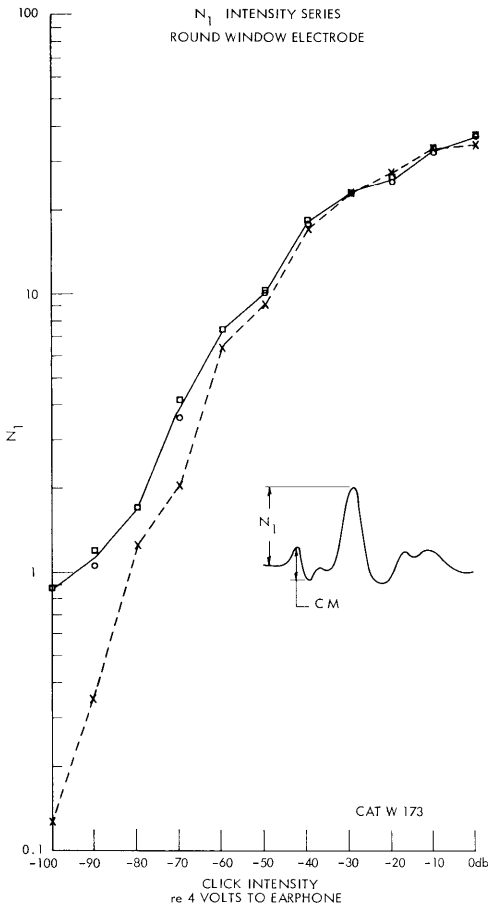

Fig. XVIII-4.

Amplitude of N_1 component of round-window response to condensation clicks. Insert shows how N_1 was measured. Data from same series shown in Fig. XVIII-2. Open circles represent response amplitude with no OCB stimulation; crosses, response amplitude with click preceded by OCB stimulation; open squares, N_1 response amplitude in a control series without OCB stimulation run immediately after the shock series at each intensity. Solid line represents the intensity series without efferent stimulation; dashed line, intensity series with OCB stimulation.

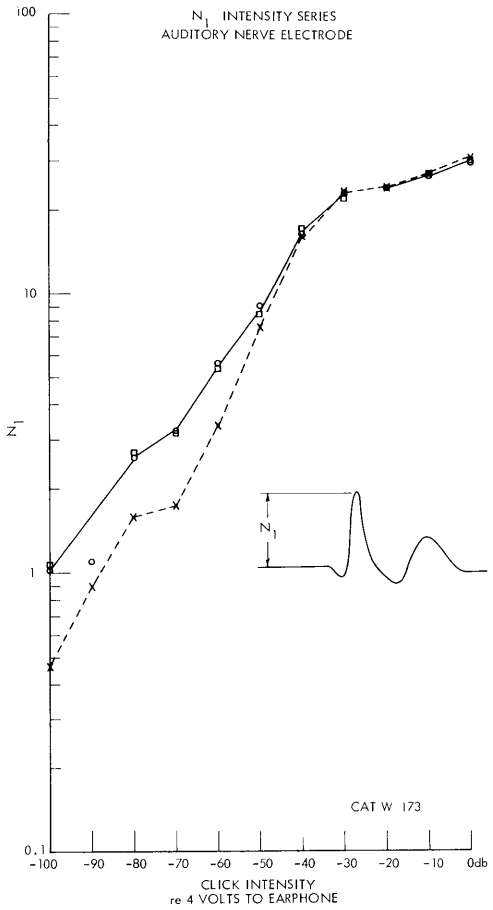

Fig. XVIII-5.

Same as Fig. XVIII-4 except that responses were recorded from concentric bipolar electrode in auditory nerve. Data from same series as shown in Fig. XVIII-3.

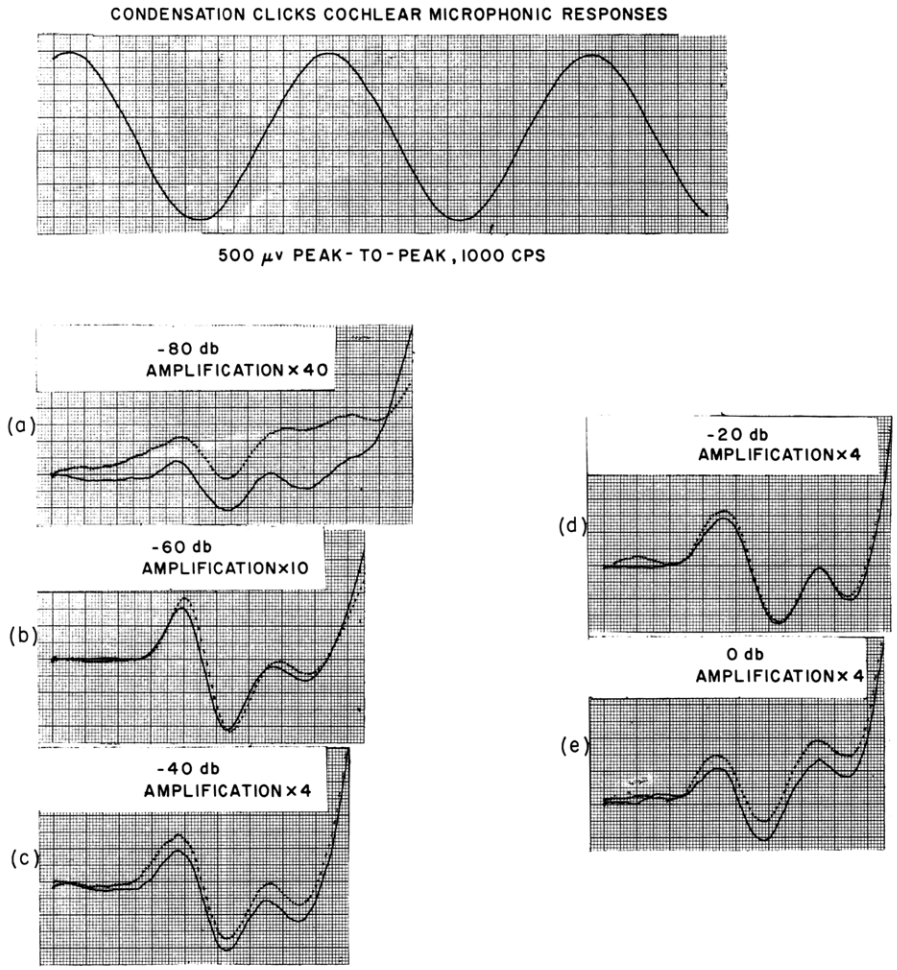

Fig. XVIII-6. Averages of same responses as shown in Fig. XVIII-2 with time scale expanded by a factor of four and vertical scale expanded as shown in each trace relative to calibration signal. Solid line traces represent round-window response with no OCB stimulation; dotted line traces, response to click preceded by OCB stimulation. Number of responses averaged, (a) 64, (b), (c), (d), (e), 32.

intensity range the OCB stimulation seems to have little effect on the neural part of the click response.

Figure XVIII-6 shows averaged responses from the round-window electrode with the scales expanded to better display the cochlear microphonic (CM) response. As has been reported[2,3] by other workers the CM component increases after OCB stimulation. Figure XVIII-7 is a plot of peak-to-peak amplitude of CM as a function of click intensity.

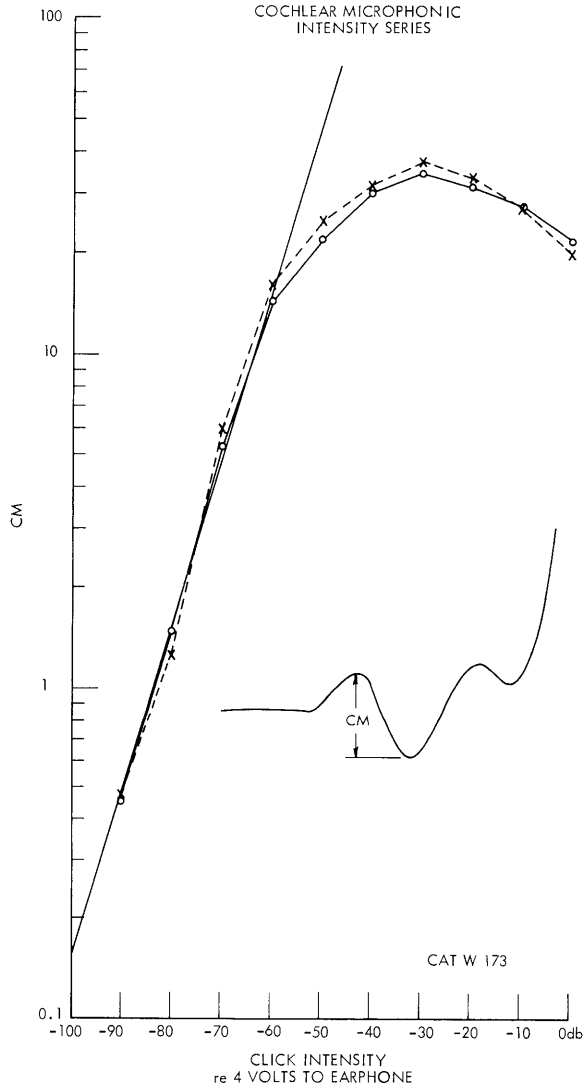

Fig. XVIII-7. Cochlear microphonic intensity series. Open circles, crosses, solid and dashed lines same as in Figs. XVIII-4 and XVIII-5. CM amplitude measured peak-to-peak as indicated in insert. (Fine line indicates a linear relationship between CM and sound-pressure level.)

The increase of CM from OCB stimulation can be seen to extend over a larger intensity range than does N_1 reduction. In other preparations this increase has existed throughout the intensity range.

Desmedt[2], in similar studies confined to low acoustic intensities, concluded that the effect of OCB stimulation on neural responses to clicks can be thought of as reducing the intensity of the acoustic stimulus. The amount of equivalent reduction, he concluded, is largely independent of the acoustic intensity for a given configuration of OCB stimulation. If this were true over a large intensity range, the dashed curves in Figs. XVIII-4 and XVIII-5 would be shifted to the right by a constant amount relative to the solid curves. The dashed and solid curves of Figs. XVIII-4 and XVIII-5 are clearly not parallel; there appears, rather, to be a continuous decrease of the OCB effect on N_1 with increasing acoustic intensity. At intensities 70 db or more above threshold for detection of N_1, the OCB seems to be incapable of reducing the neural response.

The effects demonstrated here can be enhanced somewhat by increasing the voltage of the electrical stimulus to the OCB. (In all experiments discussed here the shocks were at a level that caused no observable muscular response; no paralyzing drug was given.) It was observed, however, that even with shocks strong enough to cause gross movements of the preparation there was no evidence of OCB reduction of the N_1 response to 0 db clicks.

We have not yet been able to interpret these data in terms of possible characteristics of the mechanisms of action of the OCB. It appears from preliminary data that the frequency spectrum of the acoustic stimulus is also an important parameter in determining the effectiveness of efferent inhibition. The combination of frequency and intensity dependence is under investigation at the present time.

M. L. Wiederhold, W. T. Peake

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2. J. E. Desmedt, Auditory-evoked potentials from cochlea to cortex as influenced by activation of the efferent olivocochlear bundle, J. Acoust. Soc. Am. 34, 1478 (1962).

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D. CORRELATION ANALYSIS OF EEG AND TREMOR ACTIVITY

In the account of studies on normal or "physiological" tremors, which was included in the report[1] on investigation of tremors in Quarterly Progress Report No. 76, it was suggested that it would be of interest to compare by correlation techniques simultaneously recorded EEG and tremograms, in view of the appearance in the latter of components of the same frequencies as that of alpha activity (8-13/sec in the parieto-occipital EEG). In the present report findings are presented from analyses of such recordings from two normal controls (the same subjects as were studied in the previous report[1]) and in two patients, the first having a pronounced unilateral rhythmic tremor of the outstretched upper extremity, and the second having a rather rhythmic physiological tremor. The techniques of recording analysis of the data were the same as those described in the earlier report.[1] All recordings were made with eyes closed and were of 2 minutes duration, except for those carried out during intermittent photic stimulation, which recordings were of 40 sec duration.

In Fig. XVIII-8 are shown a portion of the ink trace and correlograms for the first normal control. The location of the first peak in the autocorrelograms of the tremograms at a delay of 100 msec is indicative of tremor activity of an average frequency of 10/sec. The last is very close to the average EEG frequency of 9.5/sec which is evidenced from the autocorrelogram for the latter. From the crosscorrelograms in Fig. XVIII-8, it is apparent that there is only minimal crosscorrelation between the tremors on the two sides. It is also apparent that the crosscorrelograms for the midline parieto-occipital EEG with the tremors on the two sides, are essentially flat.

Results for similar recordings from the second normal control subject, from whom bilateral EEG recordings in the parieto-occipital region were made, are shown in Fig. XVIII-9. From the autocorrelograms, it is apparent that the average tremor frequency, 8/sec, is much lower than that of the average EEG frequency, which is 11.1/sec. The crosscorrelation coefficient between the tremors from the two sides is seen to be 0.25. The crosscorrelation coefficient for the EEG on the two sides is approximately 0.6 at zero delay. The crosscorrelograms for the tremors with the EEG on the same and on opposite sides are essentially flat.

In Fig. XVIII-10, results are shown from recordings, carried out at the suggestion of Dr. G. F. Rossi of Genoa, Italy, during a visit to the Neurophysiology Laboratory of the Massachusetts General Hospital, to investigate the possibility of altering the frequency characteristics of the tremor by repetitive photic stimulation of the subject. It is apparent from the autocorrelograms in Fig. XVIII-10 that the stroboscopic stimulation was without effect upon tremor activity, in contrast to its effect upon the EEG. In all instances, crosscorrelograms of EEG with tremor activity were essentially flat, and hence none is shown in Fig. XVIII-10.

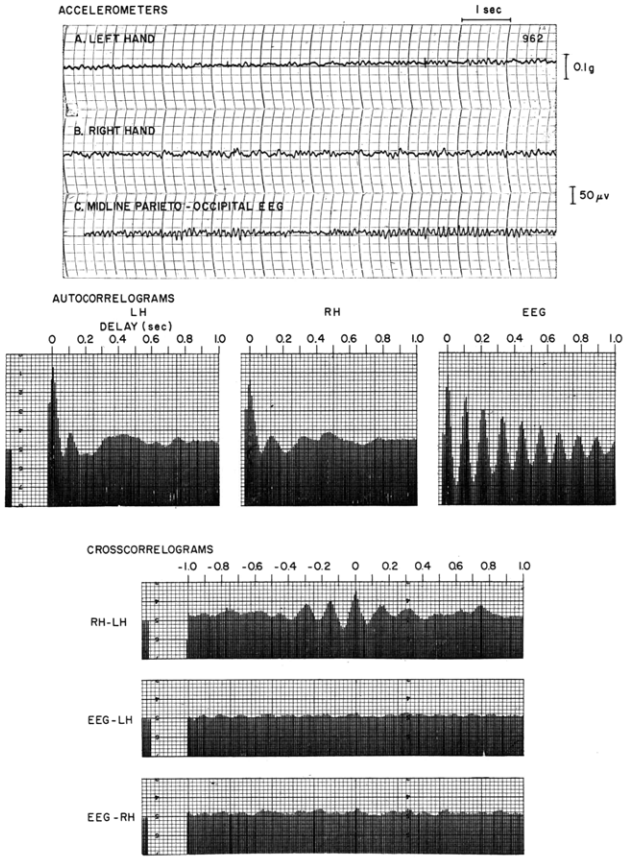

Fig. XVIII-8. Simultaneously recorded midline parieto-occipital EEG and accelerometrically monitored physiological tremors of the outstretched upper extremities for a normal subject (B. J.). Accelerometers mounted on the upper surface of the hands. Eyes closed in this and subsequent figures. Duration of this and subsequent recordings, 2 minutes, except for recordings during intermittent photic stimulation, for which 40-sec recordings were made. Calibration is in terms of g, the acceleration caused by gravity.

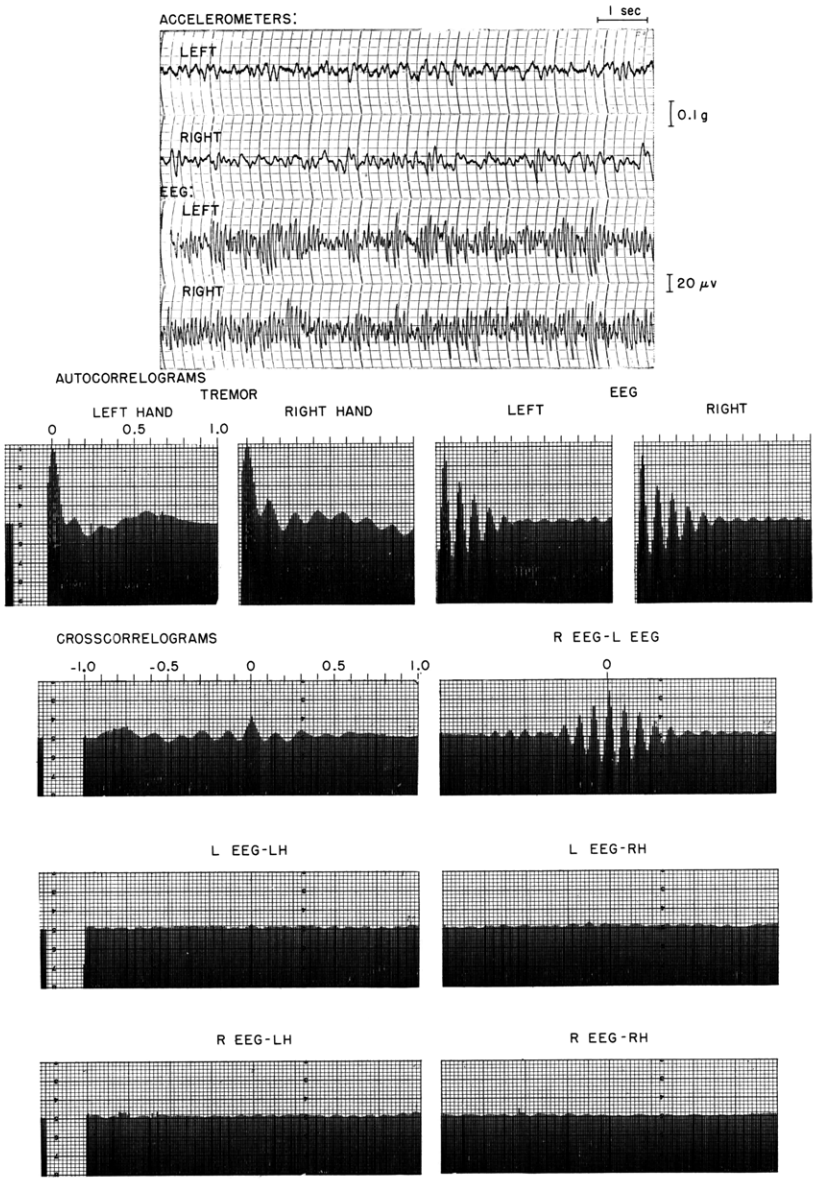

Fig. XVIII-9. Bilateral tremograms and parieto-occipital EEG recordings for a normal subject (B. K.).

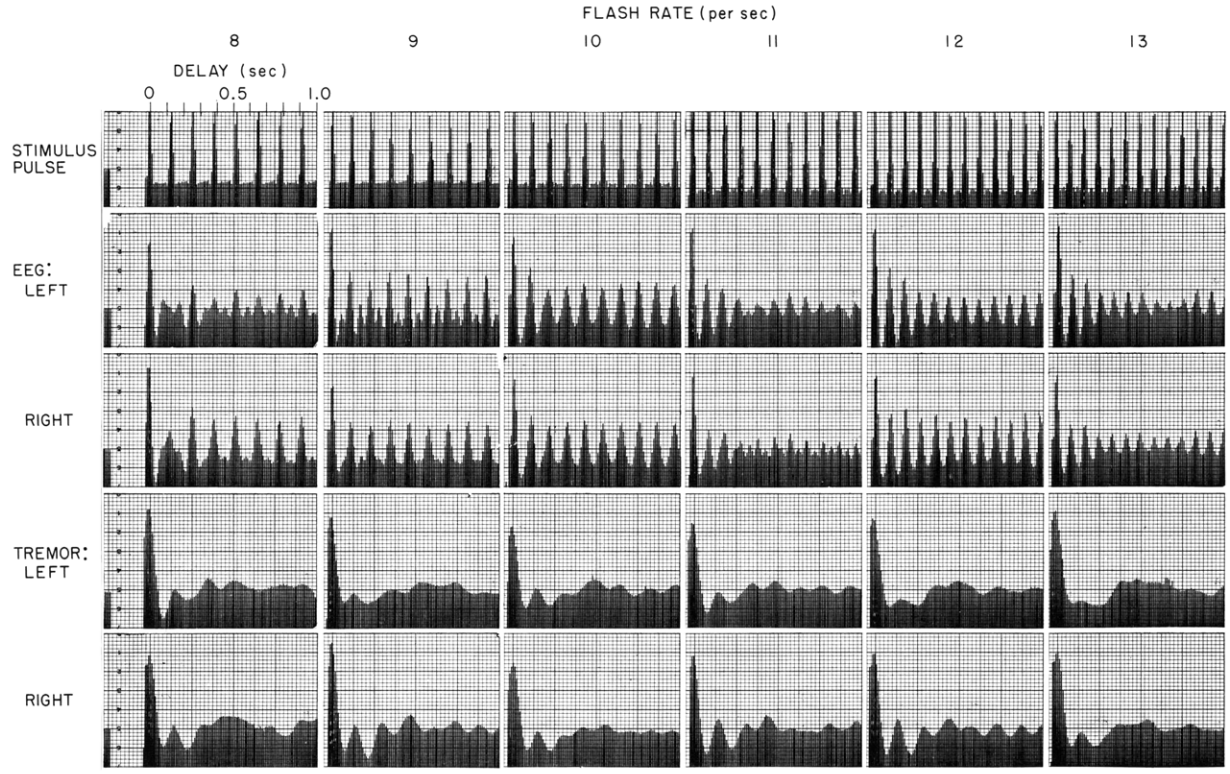

Fig. XVIII-10. Autocorrelograms for EEG and tremograms for intermittent photic stimulation at various flash rates. (Normal Subject B. K.)

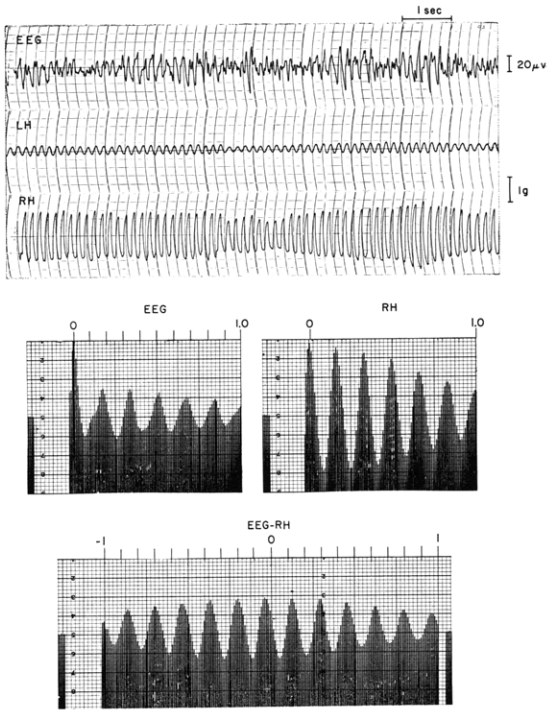

Fig. XVIII-11.

Midline parieto-occipital EEG and tremograms for a patient (E. C.) with a pronounced unilateral rhythmic tremor of the outstretched right upper extremity. Apparent tremor recorded from the accelerometer on the left side represents purely transmitted motion from the right side. For this recording, the upper frequency response of the system was limited to 15 cps (1/2 amplitude) in order to eliminate a prominent EMG content in the EEG leads.

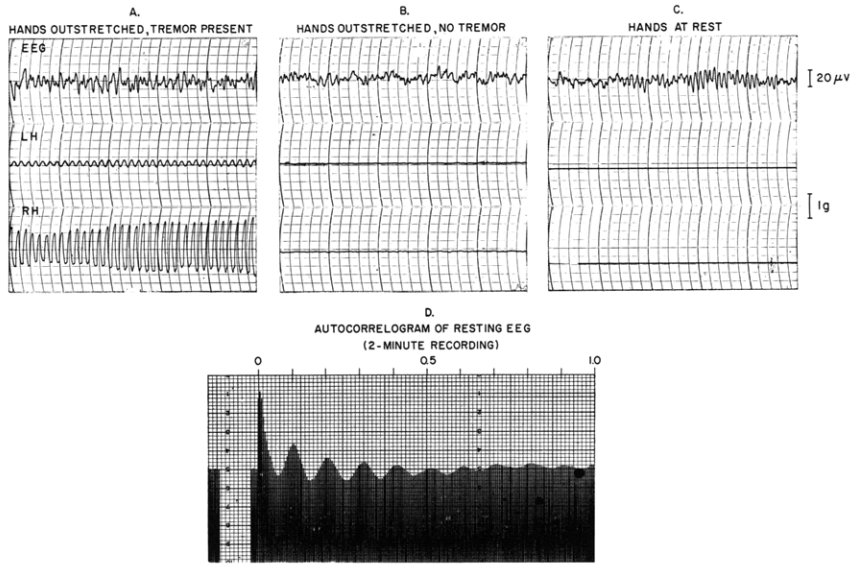

Fig. XVIII-12. Portions of EEG and tremogram for Patient E. C. for various conditions. (A). Both upper extremities in outstretched position. (B). Tracing obtained just after extremities are outstretched but before the rhythmical tremor had appeared. (C). Upper extremities at rest on the knees. (D). Autocorrelogram of resting EEG.

Recordings are shown in Fig. XVIII-11 from a patient with a pronounced rhythmical tremor that appeared upon sustained posturing of the right upper extremity in the outstretched position. No tremor was present at rest on either side, and since the tremor appeared only the right side upon extending the limbs individually, the output of the accelerometer on the left side in Fig. XVIII-11 must be considered to have resulted from mechanical crosscoupling, through the trunk of the body, from the tremor on the right side. The average tremor frequency is seen from the autocorrelogram to be 6/sec. A very prominent 6/sec component is also present in the autocorrelogram of the EEG, and a crosscorrelation coefficient of 0.35 is apparent from the crosscorrelogram for this frequency component in the EEG and in the right-sided tremor. Identical findings resulted from a second recording. The question of the possibility of an artefact of movement of the EEG electrodes or leads, as a source of the 6/sec component in the EEG trace must, of course, be immediately raised. A third accelerometer was not available to permit direct monitoring of motion of the head, but inspection of the ink traces shown in Fig. XVIII-12 suggests that the 6/sec component in the EEG ink trace did not derive from movement artefact. Thus, upon close inspection of the EEG recorded when the upper extremities are in the outstretched position (A, in Fig. XVIII-12) a 6/sec component is evident, but its amplitude does not appear to parallel that of the tremor, as would be expected if the former were purely a movement artefact. Moreover, a 6/sec component is evident in the EEG in the few seconds just after the upper limbs are outstretched, but before the tremor has developed, as is apparent in B, in Fig. XVIII-12. The EEG in the latter instance contrasts with that recorded when the upper extremities are completely at rest (C, in Fig. XVIII-12), which is characterized by rather prominent alpha activity of an average frequency of 10/sec, as is evident from the autocorrelogram shown in D in Fig. XVIII-12. Hence it appears unlikely that movement artefact is a source of the 6/sec component in the EEG in Fig. XVIII-11, but this possibility cannot be excluded entirely.

Recordings from a patient with rather rhythmic tremors of the physiological type bilaterally are shown in Figs. XVIII-13 through XVIII-19. In Fig. XVIII-13, the tremograms and the parieto-occipital EEG tracings are very similar, but no such similarity is apparent between the tremograms and the central EEG leads. The central EEG leads were included in order to explore possible relationships between the tremor and the EEG overlying the pre-central motor area. The similarity of the autocorrelograms of the tremor recordings and those of the parieto-occipital EEG recordings is also evident from inspection of Fig. XVIII-14. The average frequency of the tremor on the left side is approximately 8.5/sec, on the right it is slightly lower, approximately 8/sec. The average frequency in the parieto-occipital EEG is approximately 9.5/sec; no prominent rhythmic activity is present from the central EEG leads, as evidenced by the autocorrelograms for the latter.

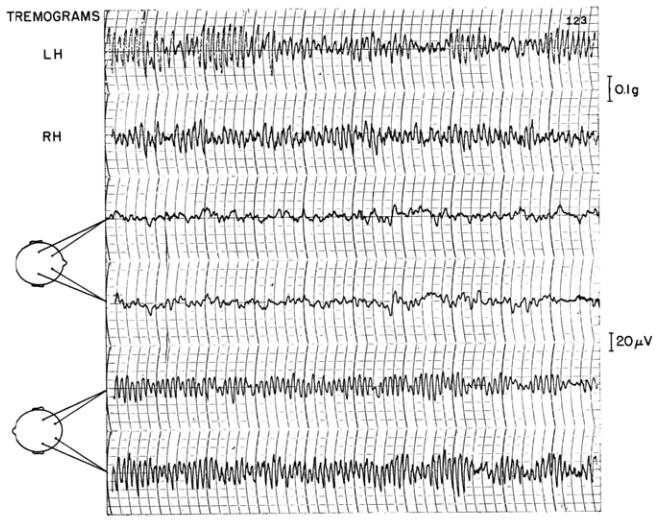

Fig. XVIII-13. Bilateral tremograms and EEG recordings from bilateral fronto-central and parieto-occipital electrodes for a patient (P. D.) with rather rhythmic physiological tremors bilaterally.

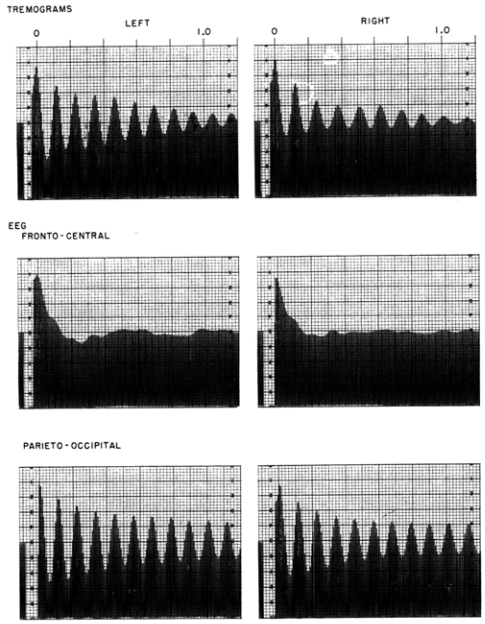

Fig. XVIII-14.

Autocorrelograms of tremograms and EEG for Patient P. D.

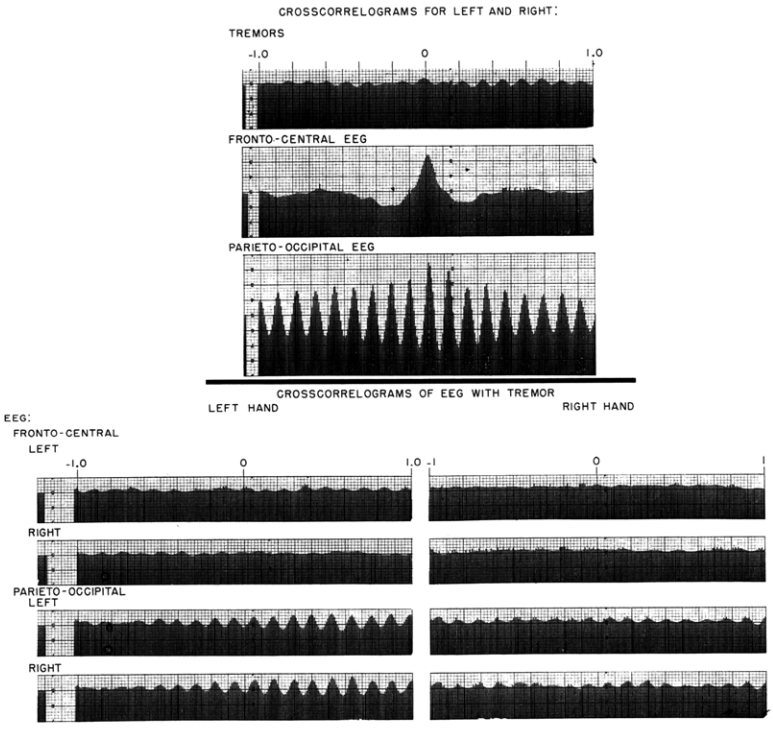

Fig. XVIII-15.

Crosscorrelograms of EEG with tremograms for Patient P. D.

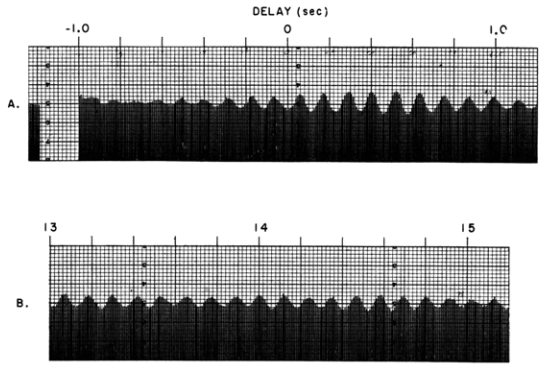

Fig. XVIII-16.

Crosscorrelogram of left parieto-occipital EEG with tremogram of left upper extremity: (A) for delays in the range −1 - +1 sec; (B) for delays in the range +13 - +15 sec. Duration of recordings analyzed, 2 minutes. (Patient P. D.)

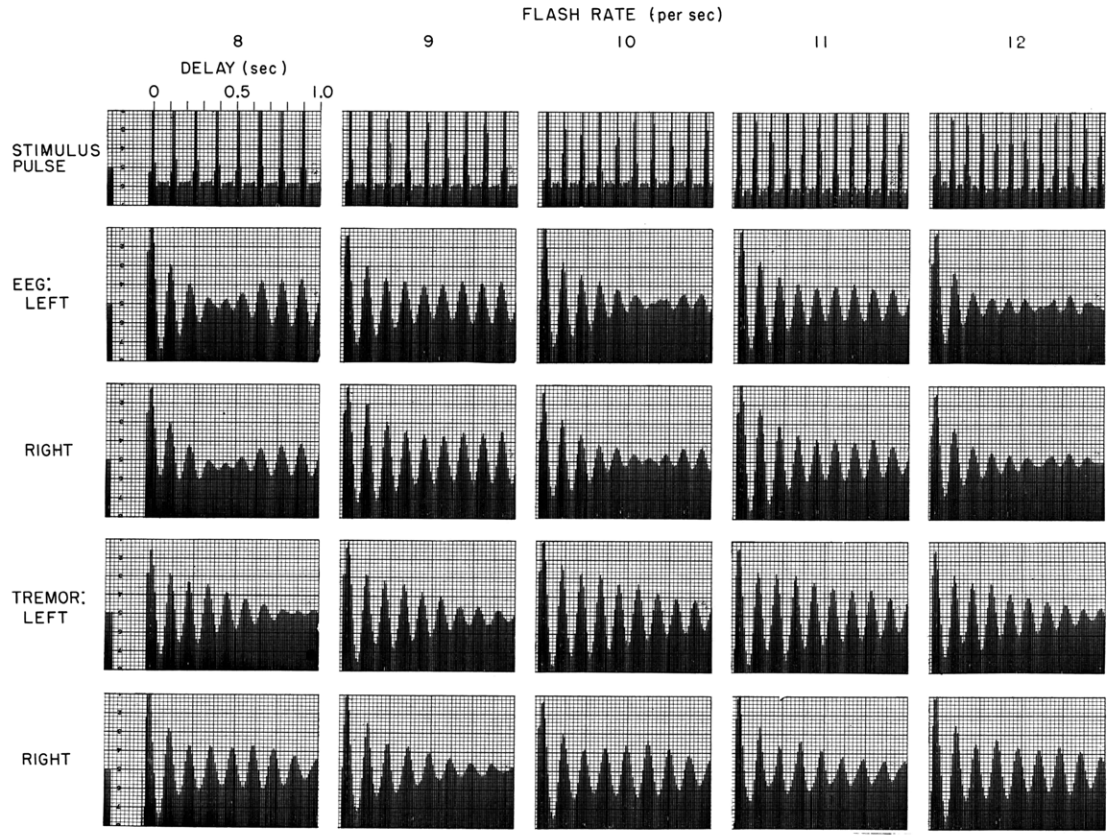

Fig. XVIII-17. Autocorrelograms of parieto-occipital EEG and tremograms for Patient P. D. for various flash rates of photic stimulation. Duration of recordings, 40 sec.

Crosscorrelograms of the tremor recordings on the two sides with the several EEG leads are shown in Fig. XVIII-15, and it is apparent that the amplitudes of none of the crosscorrelograms exceed the level of sampling artefact, with the possible exception of the correlograms for the tremor on the left with the parieto-occipital EEG, a finding that may be of interest in view of the fact that this patient was left-handed. Since the parieto-occipital EEG on the two sides are strongly correlated, as shown in the lower correlogram in Fig. XVIII-15, it is not surprising that the crosscorrelograms of the tremor on the left side with the parieto-occipital EEG from both sides are quite similar.

In view of the very similar autocorrelograms for the tremor on the left with the parieto-occipital EEG (Fig. XVIII-14), the question arose as to whether the apparent crosscorrelation of these two resulted merely as an artefact of sampling in this 2-minute recording, particularly in view of the fact that the peak in the crosscorrelograms is at a delay of ~0.5 sec, rather than nearer zero delay. To examine this possibility, the crosscorrelogram was recomputed for the full range of delays extending from -1 to +15 sec (the upper limit available with the correlator), under the assumption that any apparent correlation remaining after such large delays would be due solely to sampling artefact. The initial and final segments of this continuously computed curve are shown in Fig. XVIII-16. Following the peak in the crosscorrelogram at 0.62 sec at no intermediate delay did the amplitude exceed the value apparent in Fig. XVIII-16 at 14.6 sec delay, that is, for no delays following the former did the amplitude of the crosscorrelogram exceed more than 0.6 its value at 0.62 sec, although, at several intermediate delays, the amplitude was approximately the same as that at 14.6 sec. The last may then be taken as an indication of the peak level of spurious correlation caused by sampling artefact, and it is clearly exceeded for delays in the vicinity of 0.6 sec. (As a check on the performance of the over-all system for correlation at such large delays, a 10/sec sine wave was recorded on the magnetic tape and processed in exactly the same manner as for the crosscorrelogram in Fig. XVIII-16, the crosscorrelogram being computed out to a delay of 15 sec (150 wavelengths). The amplitude of the envelope of the correlogram at a maximum delay of 15 sec had decreased by only 14 per cent of amplitude at zero delay, the decrease being due to slight irregularities in the speed of the magnetic drum and of the magnetic tape recorders.) It is apparent, then, that sampling artefact does not appear to account for the crosscorrelation in the range of ~0.6 sec delay between the left-sided tremor and the parieto-occipital EEG. The possibility that movement artefact in the EEG could be the basis for the apparent correlation between the left-sided tremor and the EEG would appear to be excluded by the absence of correlation of the left-sided tremor with the EEG from the central leads, and of the right-sided tremor (whose amplitude was approximately the same as that on the left) with any of the EEG leads.

Correlograms for this patient from recordings with repetitive photic stimulation are

shown in Figs. XVIII-17 through XVIII-19. From the autocorrelograms of the tremor on the left and right sides, as shown in Fig. XVIII-17, it is apparent that the average frequencies of the tremor on the two sides remain virtually unchanged for different flash rates, the frequencies being only slightly different from those with no flash (Fig. XVIII-14). At the same time, from Fig. XVIII-17, it is evident that the dominant rhythmic activity in the parieto-occipital EEG is little altered by the flashing. As was the case with no photic stimulation (Fig. XVIII-14) the average frequency of the tremor on the left side is very similar to that of the parieto-occipital EEG activity, of 9/sec. The tremor rate on the right side remained at 8/sec.

For a closer examination of the question of relationships between tremor and EEG, the crosscorrelograms shown in Figs. XVIII-18 and XVIII-19 were computed. Upon first sight, the crosscorrelograms of the tremor on the left side with the parieto-occipital EEG activity (Fig. XVIII-18) for these very brief (40-sec) recordings appear to suggest that these two phenomena are in fact correlated. Upon closer inspection of the crosscorrelograms, however, it becomes apparent that the location of the peaks nearest zero delay are not consistently located on the delay scale; they appear in fact to be randomly distributed about zero delay. Hence we must conclude that the apparent crosscorrelation of tremor with EEG activity represents entirely an artefact of sampling in these short recordings. The same is true for the crosscorrelograms of tremor on the right side with EEG activity, which are shown in Fig. XVIII-19, the amplitudes of the crosscorrelograms being generally smaller than those in Fig. XVIII-18. From these results, it is clear that the tremor in this patient was not entrained by the photic stimulation, nor was the tremor synchronized with the parieto-occipital EEG during the stroboscopic stimulation.

To summarize, correlation analysis of recordings from two normal controls has not indicated that the accelerometrically monitored physiological tremors from the outstretched upper extremities are correlated with the parieto-occipital EEG. There is evidence that such a relationship was present in recordings from a patient with a pronounced unilateral rhythmic tremor, but the possibility that this apparent correlation may have arisen from a component of movement artefact in the EEG has not been completely excluded. Recordings from a left-handed patient with rather rhythmic tremors of the physiological type bilaterally were suggestive of a crosscorrelation between the tremor on the left side with the parieto-occipital EEG. Such a correlation was not distinguishable from that caused by sampling artefact in shorter recordings (40 sec instead of 120 sec) with photic stimulation at flash rate in the alpha-frequency range. The latter stimulation at several flash rates was without effect upon the frequency of the tremor. Similarly, no effect of intermittent photic stimulation upon tremor was found for one of the normal control subjects.

The fact that the frequency of physiological tremors, the significance of which is

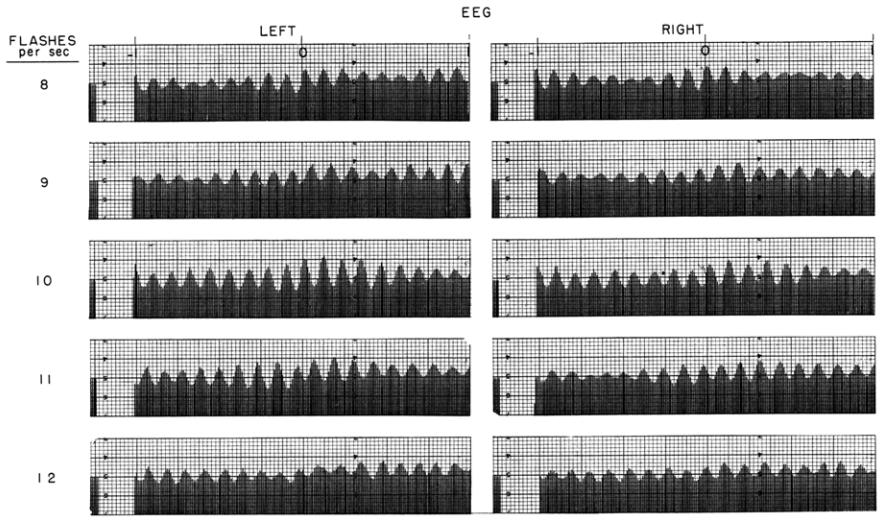

Fig. XVIII-18. Crosscorrelograms of parieto-occipital EEG with tremograms of left side for various flash rates for Patient P. D. Duration of recordings, 40 sec.

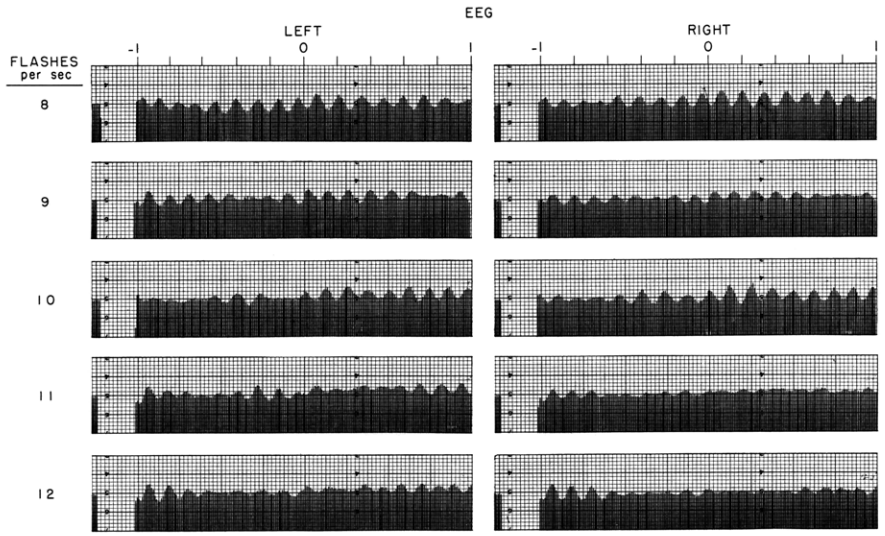

Fig. XVIII-19. Crosscorrelograms of parieto-occipital EEG with tremograms of right side for various flash rates for Patient P. D. Duration of recordings, 40 sec.

still under discussion,[2,3] may lie within the alpha-frequency range of the EEG has long been of interest.[4-13] (Despite the fact that in some instances the characteristics of the monitoring system for the tremors may influence the frequency of the recorded tremor,[14,15] the fact that tremors have been recorded with several different techniques, some of which (for example, photoelectric) cannot alter the frequency characteristics of the tremor itself, indicates that the basic phenomenon cannot be an artefact of the recording system.) There has been no agreement, however, concerning the question of whether physiological tremor activity is consistently correlated with EEG activity. Thus, in some instances, short-time wave-to-wave correspondence,[4-7] as well as statistical agreement between the mean frequencies of the two phenomena[9] (as determined by visual analysis), have been reported, but in other series[13] no such agreement has been found.

It seems clear that physiological tremor is not solely dependent upon rhythmic activity of the cerebral cortex, for the former has been observed when the latter is not present, or when the connections between brain and limb, at the level of the spinal cord, are interrupted.[7,8] The possibility that components in both, under particular circumstances, could be entrained by a common pacemaker at such a subtle level that no wave-to-wave correspondence would be evident upon inspection of the simultaneously recorded inked traces, might be considered. Such records might or might not exhibit the same average frequency, as determined by visual or automatic frequency analysis. By cross-correlation analysis, however, the presence of such a common (entrained) component could be detected, even if it were not at all evident in the original traces.[19]

Alternatively, for two quasi-rhythmic systems possessing similar or even identical frequency characteristics which in reality are quite independent of one another, a wave-to-wave correspondence, of an entirely fortuituous nature, could appear for brief intervals of time. Such true independence could be demonstrated with the aid of crosscorrelation analysis, appropriate attention being paid to the possibility of correlation errors arising from sampling artefact.[20]

The present study, which demonstrates the particular suitability of correlation analysis for study of tremor-EEG relationships, has not unequivocally indicated the existence of a consistent relationship between physiological tremor and the EEG, within the limitations of the present experimental conditions, for the subjects who were examined. Further studies of the problem, including simulation ones, are planned.

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J. S. Barlow

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15. J. G. L. Williams, A resonance theory of "microvibrations," Psychol. Rev. 70, 547-558 (1963).

16. J. S. Barlow, Autocorrelation and crosscorrelation analysis in electroencephalography, IRE Trans. Vol. ME-6, pp. 179-183, 1959.

17. W. A. Rosenblith (ed.), Processing Neuroelectric Data, The M.I.T. Press, Cambridge, Mass., 2d printing, 1962, 127 pp.

18. W. B. Davenport, Correlator errors due to finite observation intervals, Technical Report 191, Research Laboratory of Electronics, M.I.T., March 8, 1951.

19. Compare Fig. 4 in J. S. Barlow[16] and Fig. 3.17 of W. A. Rosenblith (ed.)[17], p. 54.

20. Compare W. B. Davenport,[18] and W. A. Rosenblith (ed.)[17], pp. 56-60.

E. ACTIVE AND COMPENSATORY VISUAL-OCULOMOTOR TRACKING IN RELATION TO THE VESTIBULAR SYSTEM

In earlier experiments[1-3] designed to study quantitatively the nature of the disorder of stabilization of the visual image which follows complete loss of vestibular function in man, it was found, for one such patient, that the compensatory oculomotor tracking (fixation point stationary, head rotated passively while the subject was seated in the motionless chair) was indistinguishable from compensatory oculomotor tracking by a normal subject. The possibility that neck proprioceptive mechanisms might provide the sensory basis for the remarkably accurate passive oculomotor tracking by the patient was considered, but this possibility could not be directly tested experimentally in those experiments.

To further explore the nature of visual-oculomotor tracking in relation to vestibular and neck proprioceptive mechanisms, the present series of experiments, employing a Bárány chair, were carried out. For this purpose, a Bárány chair was modified so that its angular position in the horizontal plane could be monitored electrically by means of a potentiometer whose wiper is driven by the pivot of the chair. The electro-oculographic (EOG) recording technique and the use of correlation techniques for analyses of the data have been described previously.[1] For the present recordings, which were limited to study of eye movements in the horizontal, one electrode was placed adjacent to the outer canthus of each eye.

Experiments were carried out with two patients who had suffered loss of vestibular function following streptomycin toxicity to the labyrinthine apparatus, and with one neurological patient with normal vestibular function and two normal adults who served as controls. The recordings from one of the patients with vestibular loss were technically unsatisfactory for analysis (because of poor cooperation by the patient). Results from the other patient with this disorder and from the control subjects are described below.

Recordings were carried out as follows:

1. Compensatory visual oculomotor tracking, eyes open.

2. Compensatory oculomotor tracking, eyes open, with proprioceptive input: same as (1), except that the subject maintains fixation on his thumb, which overlies the spot on the oscilloscope, the hand grasping the front of the oscilloscope.

3. Compensatory tracking, eyes closed: the subject is instructed to attempt to visualize and track the stationary object of (1) above, as he is moved passively in the Bárány chair.

4. Compensatory tracking, eyes closed, with proprioceptive input: same as (3), but with proprioceptive input as in (2).

5. Active tracking (eyes open): the subject attempts to track the spot of light on

the oscilloscope as it moves randomly along the x-axis of the oscilloscope screen. (Input signal to the x-amplifier of the oscilloscope: filtered noise in the band with DC to 0.8 cps with an attenuation of 36 db per octave for higher frequencies.)

Results for the two young normal controls are shown in Figs. XVIII-20 through XVIII-28. In Fig. XVIII-20, ink tracings and correlograms for compensatory visual tracking are shown for subject T.D. The electrical positions of eye and chair are remarkably similar, as are the two autocorrelograms. In the crosscorrelogram, the appearance of the peak at zero delay indicates that there is no time lag between eye position and chair position, the former compensating exactly for the latter, as the gaze is maintained at the stationary object. No change is apparent upon the addition of proprioceptive input which is afforded as the subject fixates on his stationary thumb, as is apparent from comparison of Fig. XVIII-21 with Fig. XVIII-20.

In Fig. XVIII-22 results are shown for the recording in which tracking of the stationary object was attempted (by imagining the fixation point) while the eyes were closed. Although it is evident from inspection of the ink tracings that there is some tendency for eye movements to parallel those of the chair, the EOG is much more irregular in comparison with the tracing of the chair position than was the case in Fig. XVIII-20 with the eyes open. Moreover, it is apparent from the crosscorrelogram that eye position lagged the chair position by approximately 50 msec. The addition of proprioceptive information while the eyes were closed (Fig. XVIII-23) was ineffective in improving the quality of the attempted tracking. The time lag in the latter instance of eye position with respect to chair position was approximately 60 msec.

For comparison with the results of compensatory oculomotor tracking shown in Fig. XVIII-20, the findings for active tracking of the horizontally moving object (the spot of light on the oscilloscope screen, the subject remaining stationary in the Bárány chair) are shown in Fig. XVIII-24. The irregular EOG tracing which results from successive saccadic jumps is in striking contrast to the completely smooth EOG for compensatory tracking shown in Fig. XVIII-20. The much greater irregularity of the eye movements in Fig. XVIII-24 cannot be accounted for solely by the fact that the average frequency of the tracking signal in the latter instance was somewhat higher than that used in Fig. XVIII-20, inasmuch as saccadic movements are apparent even for the lowest frequency components in Fig. XVIII-24. Despite this irregularity, however, the average lag of eye movement with respect to spot movement is only 8 msec, as is indicated by the location of the peak in the crosscorrelogram in Fig. XVIII-24.

Results of similar recordings from another normal subject are shown in Figs. XVIII-25 through XVIII-28. In Fig. XVIII-25 are shown the findings for compensatory tracking with eyes open, and it is evident that the EOG tracing is an almost exact replica of the electrical recording of the chair position. The addition of proprioceptive input resulted in no change in the precision of oculomotor tracking.

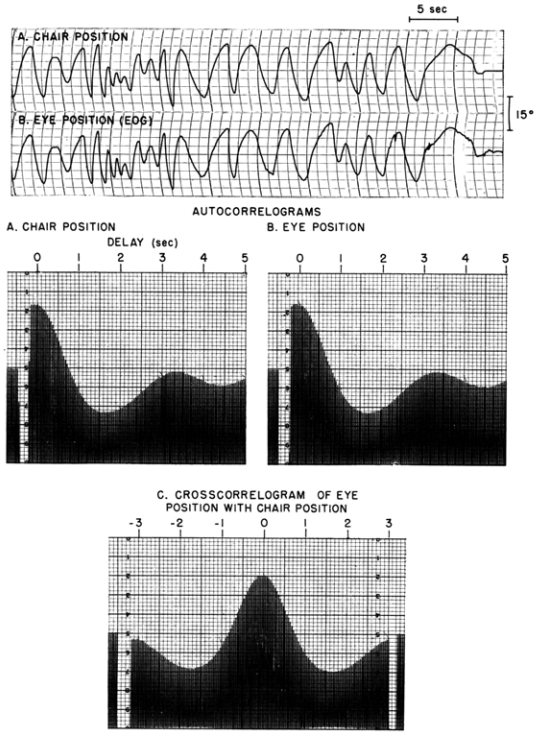

Fig. XVIII-20.

Compensatory visual-oculomotor tracking of a stationary object (spot of light on an oscilloscope screen). Subject seated in Bárány chair whose angular position is monitored electrically. Eye position monitored electro-oculographically by means of electrodes at the outer canthus of each eye. The angular calibration is the same for Figs. XVIII-20 through XVIII-33. (Note that a different angular calibration is employed for Figs. XVIII-34 through XVIII-38.) Duration of recording analyzed, 4 minutes. Delay scale in seconds. (Subject T. D.)

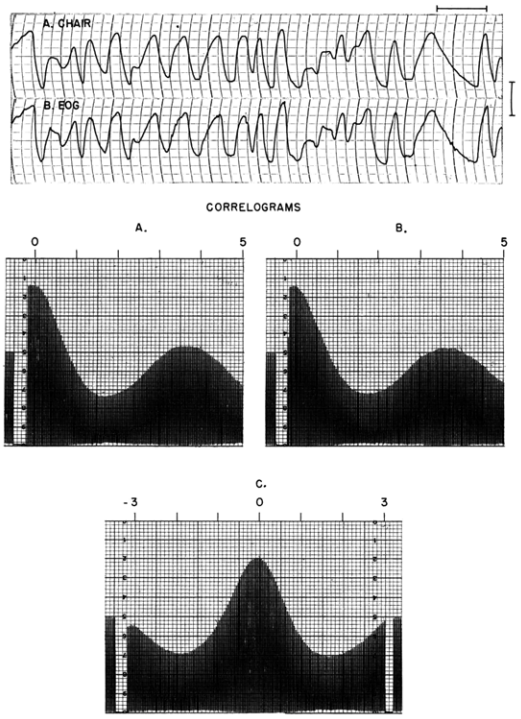

Fig. XVIII-21. Compensatory tracking, eyes open, with proprioceptive input. (Subject T. D.)

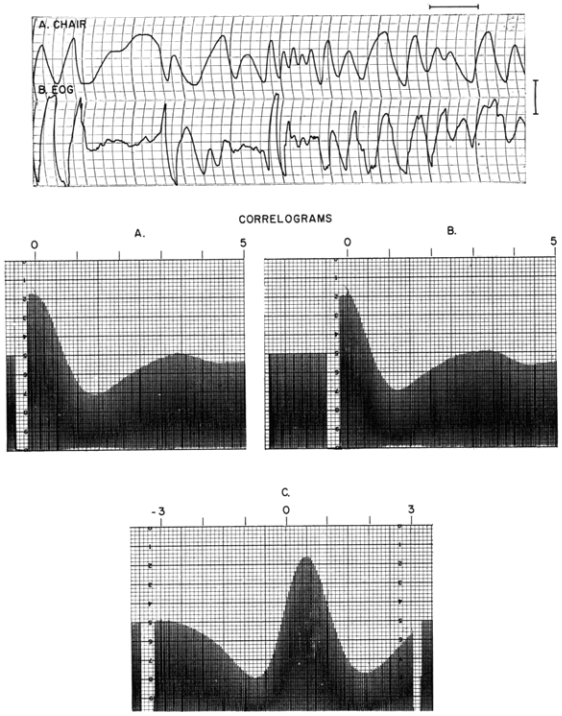

Fig. XVIII-22.

Attempted compensatory tracking with eyes closed. (Subject T. D.)

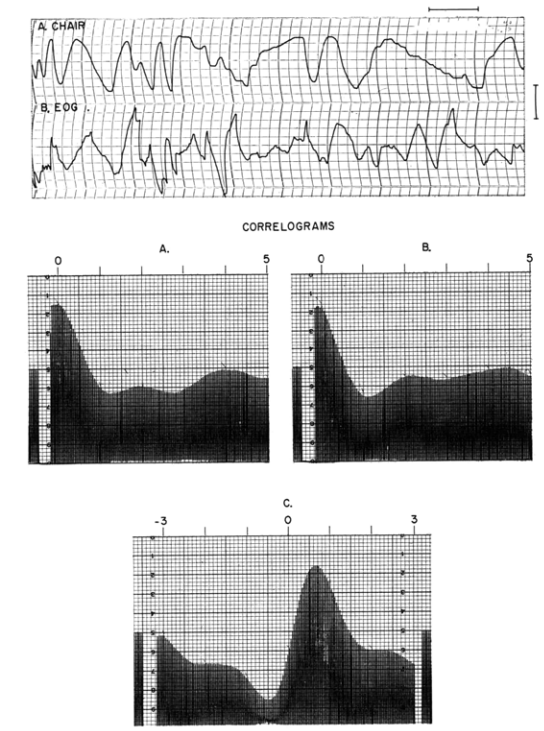

Fig. XVIII-23.

Attempted compensatory tracking, eyes closed, with proprioceptive input. (Subject T. D.)

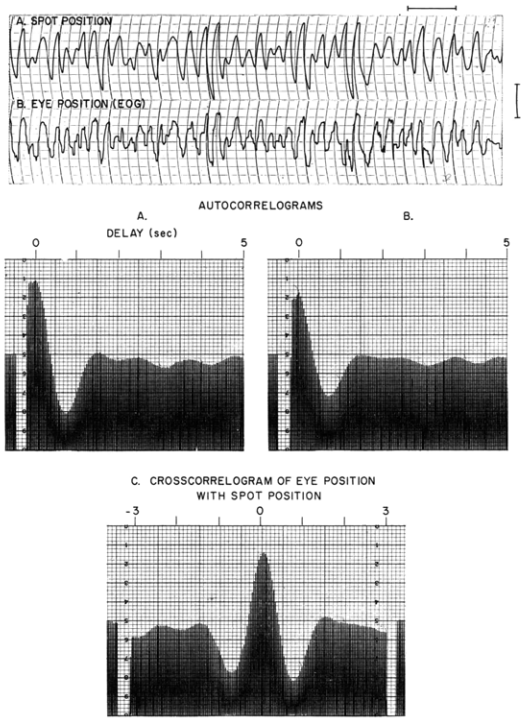

Fig. XVIII-24. Active tracking of a horizontally moving object (spot of light on an oscilloscope screen, X-input to oscilloscope, filtered noise, DC-0.8 cps). Subject seated motionless in Bárány chair. (Subject T. D.)

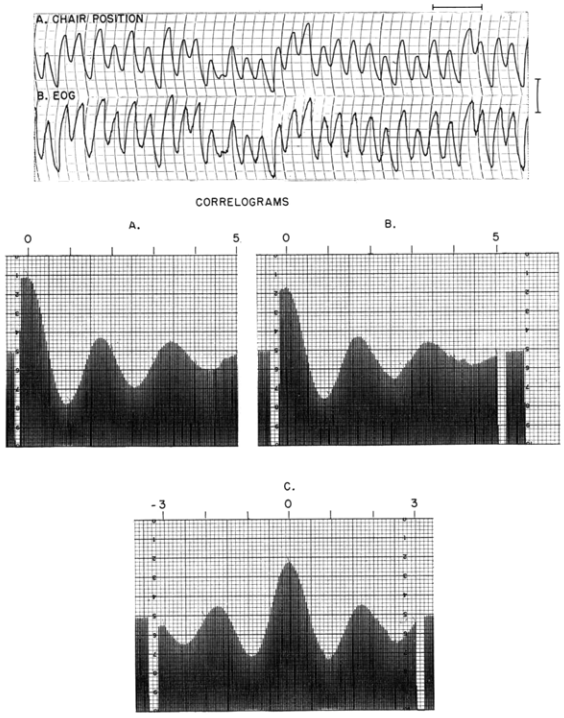

Fig. XVIII-25. Compensatory tracking, eyes open. (Subject C. M.)

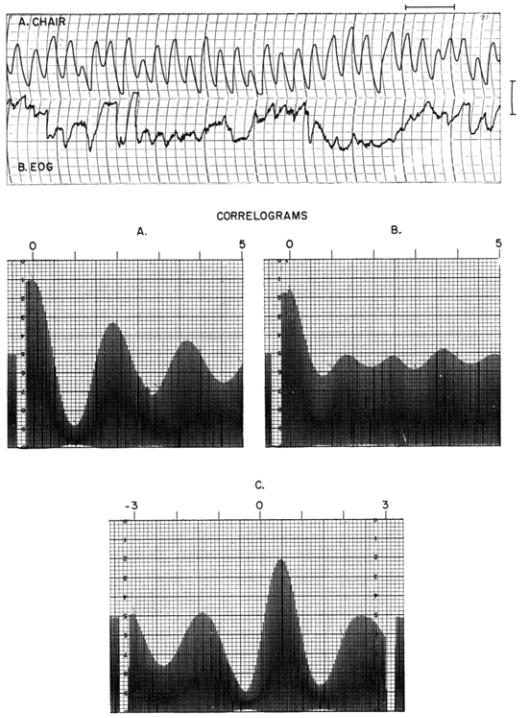

Fig. XVIII-26. Attempted compensatory tracking with eyes closed. (Subject C. M.)

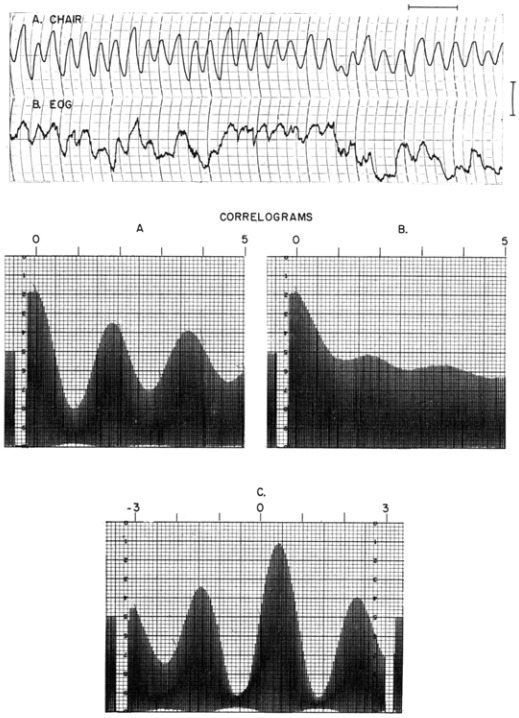

Fig. XVIII-27.

Attempted compensatory tracking, eyes closed, with proprioceptive input. (Subject C. M.)

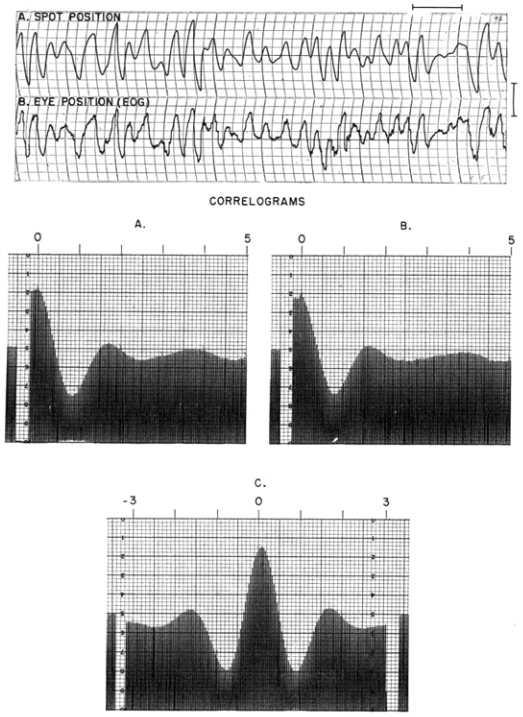

Fig. XVIII-28.

Active tracking of horizontally moving target, eyes open, subject stationary. (See Fig. XVIII-24.) (Subject C. M.)

Attempted tracking of the imagined fixation point, while the eyes were closed, results in an EOG tracing that bears little resemblance to the tracing of chair position (Fig. XVIII-26). There does, however, remain some correlation between the two, as is indicated by the crosscorrelogram, which shows that for the component common to the two, the eye position lagged chair position by ~ 500 msec on the average. As is evident from Fig. XVIII-27, the addition of proprioceptive input results in little change.

Findings for active tracking of the moving spot by the stationary subject (Fig. XVIII-28) indicate the presence of prominent saccadic components in the EOG, and the crosscorrelogram indicates an average lag time of approximately 100 msec.

Results of recordings from a patient with motor neuropathy, but with a normally functioning vestibular system, are shown in Figs. XVIII-29 through XVIII-33. Compensatory tracking with eyes open by this patient (Fig. XVIII-29) is much the same as that for the normal subjects shown in Figs. XVIII-20 and XVIII-25, and no change resulted when proprioceptive input was added (Fig. XVIII-30). Upon closing of the eyes, however, there was almost complete "decoupling" between the EOG and the electrical signal of chair position, as is evident from Fig. XVIII-31, a finding that remained the same when proprioceptive input was added while the eyes were closed (Fig. XVIII-32). It is of interest that this patient was unable to carry out active tracking of the tracking signal that was employed in these recordings (Fig. XVIII-33), a finding that contrasts strikingly with the excellent compensatory tracking (Fig. XVIII-29).

Results of recordings from a patient who had lost vestibular function from streptomycin toxicity to the vestibular apparatus approximately one year previously are shown in Figs. XVIII-34 through XVIII-38. The absence of vestibular function for this patient was evident from the fact that there was no response with electro-oculographic monitoring to air calorics, iced alcohol calorics, nor any nystagmus on positional testing. The extent to which the EOG tracing for this patient resembles that of the electrical indication of chair position, in passive oculomotor tracking, as shown in Fig. XVIII-34, is truly remarkable. Provision of proprioceptive input results in no change (Fig. XVIII-35); the time lag between the EOG and the chair-position signal in both instances is zero, as is indicated by the location of the peak in the crosscorrelograms in Figs. XVIII-34 and XVIII-35. Compensatory tracking attempted when the eyes are closed (Fig. XVIII-36) indicates that the degree of "coupling" between the eye position and chair position is considerably less than when the eyes are open, although it is not completely absent in the former instance. It is of interest that the crosscorrelogram of Fig. XVIII-36 indicates that the component of the EOG which was correlated with the chair position leads the latter by approximately 120 msec. Provision of proprioceptive input with eyes closed resulted in no essential change (Fig. XVIII-37).

Active visual oculomotor tracking by this patient (Fig. XVIII-38) is remarkably smooth; in fact, it is better than that for any of the control subjects (Figs. XVIII-24,

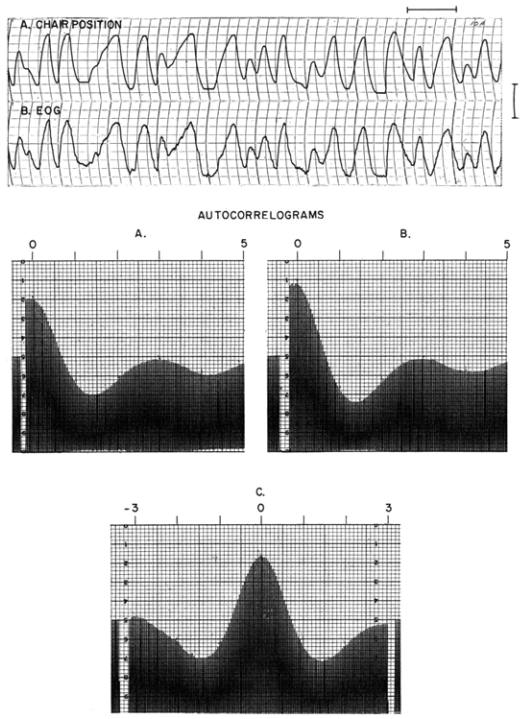

Fig. XVIII-29. Compensatory tracking, eyes open. (Patient C. E.)

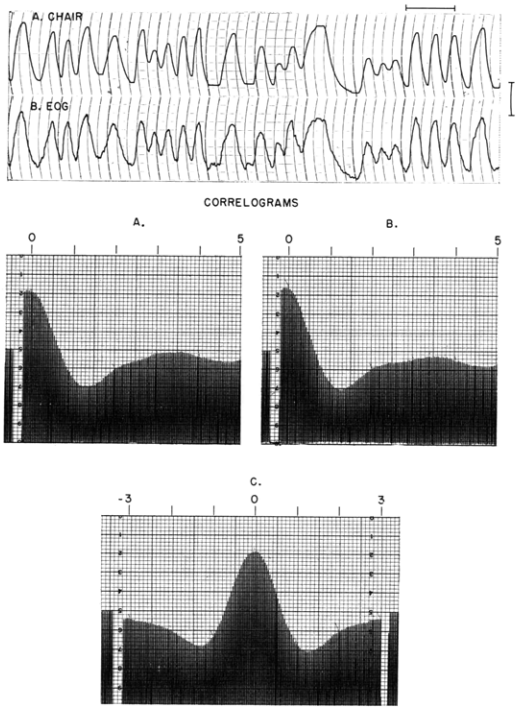

Fig. XVIII-30.

Compensatory tracking, eyes open, with proprioceptive input. (Patient C. E.)

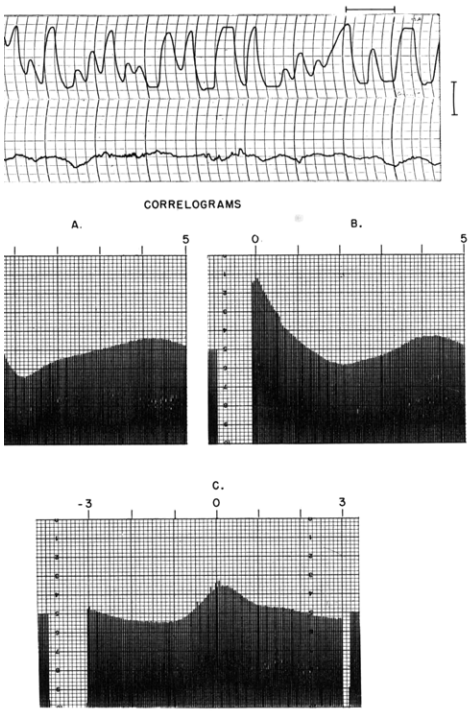

Fig. XVIII-31.

empted compensatory tracking,
s closed. (Patient C. E.)

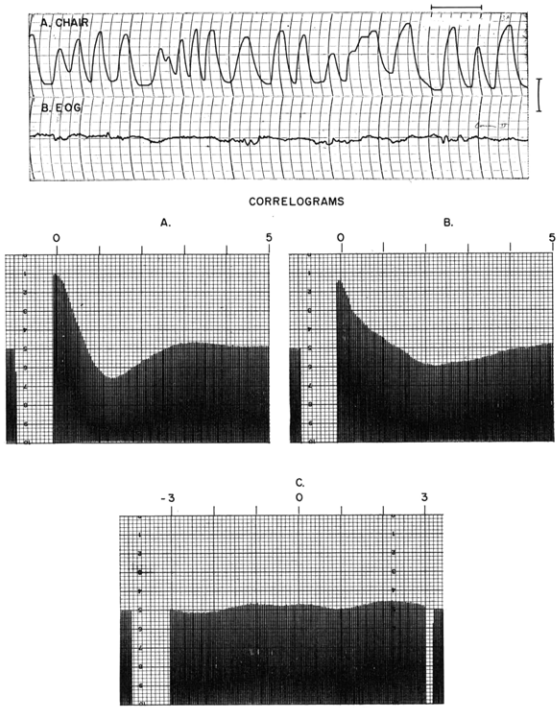

Fig. XVIII-32.

Attempted compensatory tracking, eyes closed, with proprioceptive input. (Patient C. E.)

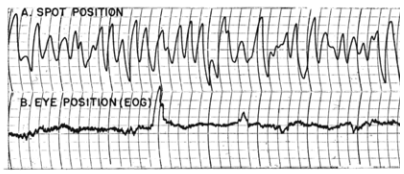

Fig. XVIII-33.

Attempted active tracking, of a horizontally moving Patient C. E. The patient to execute this maneuver; correlograms were comput

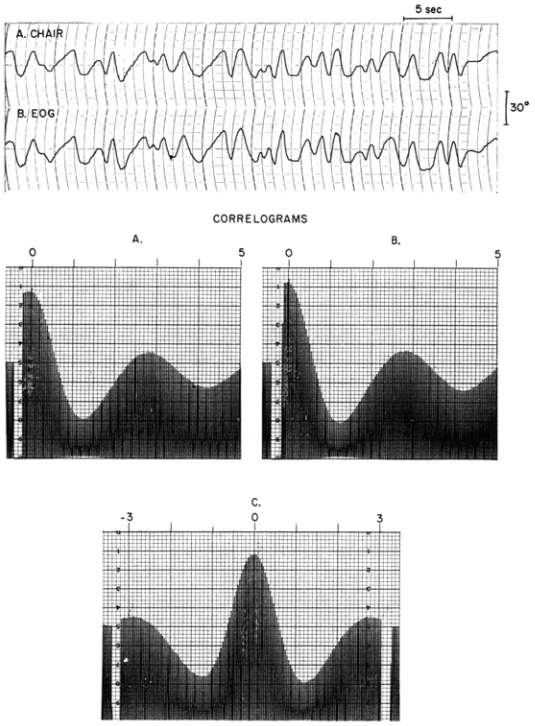

Fig. XVIII-34. Compensatory tracking, eyes open. (Patient K. H.)

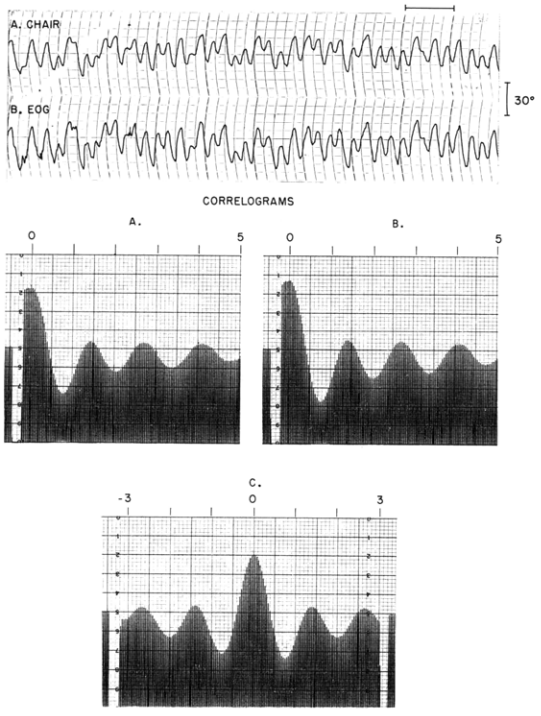

Fig. XVIII-35. Compensatory tracking, with proprioceptive input, eyes open. (Patient K. H.)

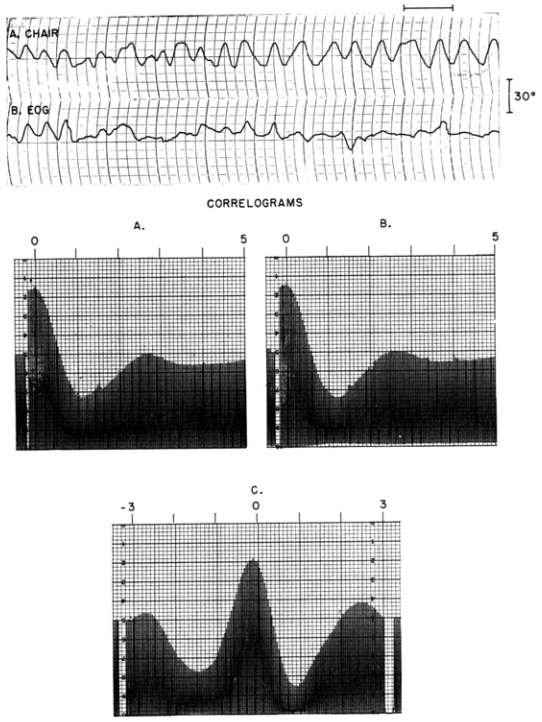

Fig. XVIII-36. Attempted compensatory tracking, eyes closed. (Patient K. H.)

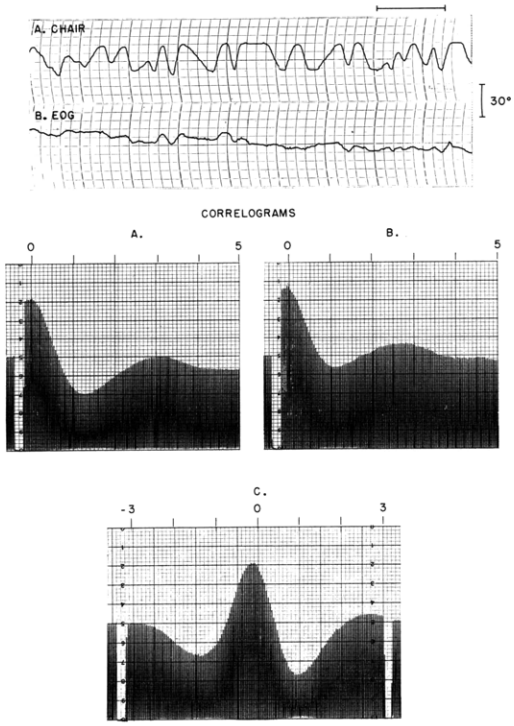

Fig. XVIII-37. Attempted compensatory tracking, eyes closed, with proprioceptive input. (Patient K. H.)

XVIII-28 and XVIII-33). Moreover, there is no time lag, on the average, between EOG and the position of the spot (the tracking signal) as evidenced by the location of the peak in the crosscorrelogram in Fig. XVIII-38 at zero delay.

The findings from the present series of experiments, together with those previously reported,[1] provide further quantitative information concerning the nature of active and compensatory visual-oculomotor tracking, and suggest the following points in relation to individuals with a normally functioning vestibular system:

1. Compensatory oculomotor tracking (stationary fixation point, passively moving subject) is executed quite as well if the whole body is moved as a unit in the Bárány chair as it is when only the head is moved and the rest of the body remains stationary. It is therefore evident that neck proprioceptive mechanisms have no essential role in compensatory oculomotor tracking of this type.[4]

2. The "decoupling" of ocular movements which appears when the eyes are closed is quite pronounced both in movements of the head with respect to the neck,[5] and in movements of the entire body as a unit, while subjects are seated in a Bárány chair (Figs. XVIII-22, XVIII-26, and XVIII-31). Therefore, neck proprioceptive mechanisms appear to have no essential role in eye movements when the eyes are closed.

3. Compensatory oculomotor tracking in response to passive motion of the subject in a Bárány chair when the eyes are open, is equally good with or without the proprioceptive input that is provided by the stationary thumb as an object for fixation of gaze. (Compare Figs. XVIII-20 and XVIII-29 with Figs. XVIII-21 and XVIII-30.)

4. The extent of "decoupling" of ocular movements when the eyes are closed is quite pronounced with or without the above-mentioned type of proprioceptive input. (Compare Figs. XVIII-22, XVIII-26, and XVIII-31 with Figs. XVIII-23, XVIII-27, and XVIII-32.) It is therefore apparent that proprioceptive information from the upper limbs is not effective for the control of eye position when the eyes are closed.

5. Compensatory oculomotor tracking, carried out either during passive movement of the head alone, or during passive movement of the body as a unit, is much more smoothly accomplished than is active oculomotor tracking, the former being completely free of the saccadic jumps that characterize the latter. It is therefore evident that the fundamental neurophysiological bases of these two different kinds of tracking are different.

The following additional points are apparent from the results from the present and the previously reported findings in patients with vestibular loss:

6. Compensatory tracking either with motion of the head alone, or during motion of the body as a unit, is carried out quite normally in the absence of vestibular function.[6] Vestibular mechanisms, therefore, appear to have no essential role in the compensatory tracking observed in these experiments.

7. Active tracking was accomplished more smoothly by the present patient with

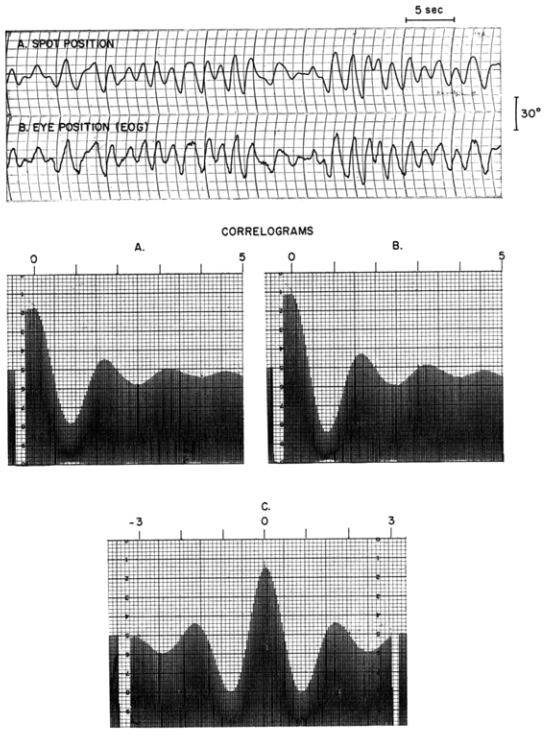

Fig. XVIII-38. Active tracking, eyes open, of a horizontally moving target. (Patient K. H.)

vestibular loss than by the controls, a finding that suggests a tendency to a compensatory effect for loss of vestibular function. The EOG tracings for active tracking by this patient (Fig. XVIII-38) are, however, still much less smooth than those for compensatory tracking (Fig. XVIII-34). It is apparent, then, that the physiological mechanisms for active tracking do not provide a basis for the smooth compensatory tracking that remains after vestibular loss in this patient.

These findings thus appear to indicate that the smooth compensatory visual-oculomotor tracking observed in these experiments is not based upon proprioceptive, nor vestibular mechanisms, nor upon the physiological mechanisms that are responsible for active or pursuit tracking. Accordingly, the physiological basis for compensatory tracking remains unclear. In view of the fact that the present patient had lost vestibular function approximately a year before these recordings, the possibility may exist that, in the intervening time, some as yet unelucidated mechanism may have compensated for the loss of vestibular function. It would therefore be of interest to make serial recordings from patients beginning soon after vestibular loss, in order to explore possible changes with time in the nature of compensatory tracking. Furthermore, in view of the fact that active tracking was carried out by this patient more smoothly than by the control subjects, it would be of interest to determine, by crosscorrelation analysis, oculomotor reaction times to step-function tracking signals.[7]

In future experiments, we plan to explore further the nature of the "decoupling" of ocular movements which occurs when the eyes are closed. As one step in the investigation of this phenomenon, recordings of compensatory tracking while the subject is in the dark and attempting to maintain gaze directed at an imagined stationary fixation point are planned, for comparison with recordings of compensatory tracking of a visible fixation point while the eyes are open, and for comparison with recordings of attempted compensatory tracking with the eyes closed.

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2. J. S. Barlow, Study of control of eye movements in man by means of autocorrelation and crosscorrelation, Biometrics 18, 421, (1962).

3. J. S. Barlow, Analysis of Control of Eye Movements in Man by Means of Autocorrelation and Crosscorrelation, DIGEST of the 1962 15th Annual Conference of Engineering in Medicine and Biology, p. 4.

4. Compare Figs. XVIII-20, XVIII-25, and XVIII-29 with Fig. XVIII-8 of J. S. Barlow, Quarterly Progress Report No. 65, op. cit.

5. J. S. Barlow, Quarterly Progress Report No. 65, op. cit.; see Fig. XVIII-11.

6. Compare Fig. XVIII-34 with Fig. XVIII-15, J. S. Barlow, Quarterly Progress Report No. 65, op. cit.

7. J. S. Barlow, Quarterly Progress Report No. 65, op. cit., see Figs. XVIII-6 and XVIII-12.