tients with manic-depressive and schizoaffective depressions was examined separately (Fig. 1a), although neither MHPG nor D time appeared to be related to age in this subgroup.

In conclusion, MHPG excretion was lower in patients with manic-depressive depressions than in patients with chronic characterological depressions. We also observed a significant inverse correlation between MHPG excretion and D time which was most pronounced in the manic-depressive disorders. Taken in conjunction with pharmacological studies that have suggested an inverse relationship between D time and central catecholaminergic activity, our results support the view that MHPG excretion reflects central noradrenergic activity, particularly in patients with manic-depressive disorders. Further studies in a larger series of patients are needed to confirm and extend these observations.

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References and Notes

1. J. J. Schildkraut, *Neuropsychopharmacology and the Affective Disorders* (Little, Brown, Boston, 1970).
2. J. W. Maas and D. H. Landis, *J. Pharmacol. Exp. Ther.* **163**, 147 (1968).
3. S. M. Schanberg, J. J. Schildkraut, G. R. Breese, I. J. Kopin, *Biochem. Pharmacol.* **17**, 247 (1968).
4. S. M. Schanberg, G. R. Breese, J. J. Schildkraut, E. K. Gordon, I. J. Kopin, *ibid.*, p. 2006.
5. T. N. Chase, G. R. Breese, E. K. Gordon, I. J. Kopin, *J. Neurochem.* **18**, 135 (1971).
6. J. W. Maas, H. Dekirmenjian, D. Garver, D. E. Redmond, Jr., H. D. Landis, *Brain Res.* **41**, 507 (1972); G. R. Breese, A. J. Prange, Jr., J. L. Howard, M. A. Lipton, W. T. McKinney, R. E. Bowman, P. Bushnell *Nature New Biol.* **240**, 286 (1972).
7. K. Greenspan, J. J. Schildkraut, E. K. Gordon, L. Baer, M. S. Aronoff, J. Durell, *J. Psychiat. Res.* **7**, 171 (1970).
8. P. A. Bond, F. A. Jenner, G. A. Sampson, *Psychol. Med.* **2**, 81 (1972); F. D. Jones, J. W. Maas, H. Dekirmenjian, J. A. Fawcett, *Science* **179**, 300 (1973).
9. J. J. Schildkraut, R. Watson, P. R. Draskoczy, E. Hartmann, *Lancet* **1971-II**, 485 (1971).
10. W. E. Bunney, Jr., F. K. Goodwin, D. L. Murphy, K. M. House, E. K. Gordon, *Arch. Gen. Psychiat.* **27**, 304 (1972).
11. J. W. Maas, J. A. Fawcett, H. Dekirmenjian, *ibid.* **19**, 129 (1968); *ibid.* **26**, 252 (1972); J. J. Schildkraut, P. R. Draskoczy, E. S. Gershon, P. Reich, E. L. Grab, *J. Psychiat. Res.* **9**, 173 (1972); J. J. Schildkraut, *Amer. J. Psychiat.* **130**, 695 (1973).
12. M. H. Ebert, R. M. Post, F. K. Goodwin, *Lancet* **1972-II**, 766 (1972); R. T. Rubin, R. G. Miller, B. R. Clark, R. E. Poland, R. J. Arthur, *Psychosom. Med.* **32**, 589 (1972); J. W. Maas, H. Dekirmenjian, J. Fawcett, *Nature* **230**, 330 (1971).
13. B. Angrist, B. Shopsin, S. Gershon, *Psychopharmacologia* **26**, 1 (1972).
14. Preliminary findings were presented at the annual meeting of the American College of Neuropsychopharmacology, San Juan, Puerto Rico, December 1972; and at the annual meeting of the Federation of American Societies for Experimental Biology, Atlantic City, New Jersey, April 1973 [J. J. Schildkraut, E. Hartmann, B. A. Keeler, *Fed. Proc.* **32**, 340 (1973)].
15. The diagnosis of manic-depressive depression, made on the basis of a history of at least one manic or hypomanic episode, corresponds to the "bipolar depressive disorders" of certain other systems of classification and to the "manic-depressive illnesses, circular type, depressed" of the American Psychiatric Association DSM-II. According to our diagnostic criteria, three patients with chronic characterological depressions and one patient with a manic-depressive depression had signs and symptoms (such as agitation, somatic preoccupations, feelings of guilt, and paranoid ideation) suggesting possible involutional depressive syndromes.
16. H. Dekirmenjian and J. W. Maas, *Anal. Biochem.* **35**, 113 (1970).
17. M. Reiner, Ed., *Standard Methods of Clinical Chemistry* (Academic Press, New York, 1953), vol. 1, p. 55.
18. We cannot meaningfully compare MHPG excretion in these two subtypes of depressive disorders to "control" values, since data on MHPG excretion are not available on "control" subjects studied under comparable conditions of hospitalization.
19. J. J. Schildkraut, *Amer. J. Psychiat.* **122**, 509 (1965); in *Psychopathology and Psychopharmacology*, J. O. Cole, A. Freedman, A. Friedhoff, Eds. (Johns Hopkins Press, Baltimore, 1973), 231–249.
20. D. L. Murphy and R. J. Wyatt, *Nature* **238**, 225 (1972); D. L. Murphy and R. Weiss, *Amer. J. Psychiat.* **128**, 1351 (1972); A. Nies, D. S. Robinson, C. L. Ravaris, J. M. Davis, *Psychosom. Med.* **33**, 470 (1971).
21. S. Spector, R. Gordon, A. Sjoerdsma, S. Udenfriend, *Mol. Pharmacol.* **3**, 549 (1967).
22. A similar difference in age has been noted in other studies and may be explained by the fact that the age of onset is lower for manic-depressive disorders (bipolar) than for other types of depressive disorders (unipolar) [G. Winokur, P. J. Clayton, T. Reich, *Manic-Depressive Illness* (Mosby, St. Louis, 1969)].
23. H. Dekirmenjian and J. W. Maas, personal communication.
24. M. Hamilton, *J. Neurol. Neurosurg. Psychiat.* **23**, 56 (1960). The correlations between MHPG excretion and agitation or somatic anxiety were negative for manic-depressive depressions, but positive for chronic characterological depressions; this further suggests that different pathophysiological processes may be operative in these two types of depressive disorders.
25. Patients with chronic characterological depressions tended to score higher on hypochondriasis (and related items) than did manic-depressive patients; however, this difference was not statistically significant ($.20 > P > .10$).
26. However, we cannot exclude the possibility that the items on the Hamilton depression rating scale were not specific or sensitive enough to detect differences in retardation, agitation, or anxiety related to MHPG excretion.
27. R. M. Post, E. K. Gordon, F. K. Goodwin, W. E. Bunney, Jr., *Science* **179**, 1002 (1973).
28. A. Rechtschaffen and A. Kales, Eds. *A Manual of Standardized Terminology, Techniques and Scoring System for Sleep Stages of Human Subjects* (NIH Publication No. 204, Government Printing Office, Washington, D.C., 1968).
29. E. Hartmann, T. J. Bridwell, J. J. Schildkraut, *Psychopharmacologia* **21**, 157 (1971); E. Hartmann, R. Chung, P. R. Draskoczy, J. J. Schildkraut, *Nature* **233**, 425 (1971); R. J. Wyatt, *Biol. Psychiat.* **5**, 33 (1972); D. J. Kupfer and M. B. Bowers, Jr., *Psychopharmacologia* **27**, 183 (1972); E. Hartmann, in *Sleep and Dreaming*, E. Hartmann, Ed. (Little, Brown, Boston, 1970), pp. 308–328.
30. M. Jouvet, *Science* **163**, 32 (1968); C. D. King, *Advan. Pharmacol. Chemother.* **9**, 1 (1971).
31. An alternative was to include data for the patient with a recurrent endogenous depression with data for the manic-depressive depressions rather than with that for chronic characterological depressions, since recurrent endogenous depressions are clinically indistinguishable from manic-depressive depressions except for the absence of the history of mania or hypomania (*1*). When this was done, the inverse relation between MHPG excretion and D time in the remaining five patients with chronic characterological depressions appeared more meaningful.
32. E. Hartmann, *Arch. Gen. Psychiat.* **19**, 312 (1968).
33. Supported in part by PHS grant MH 15,413; M.P. was the recipient of fellowship 70.495, from the Foundations' Fund for Research in Psychiatry. We thank E. L. Grab, J. Kantrowich, and G. Rege for their assistance.

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Equipotentiality Quantified: The Anatomical Distribution of the Engram

Abstract. *Sensory events and the representation of past experience cause distinctive changes in the electrical activity of widespread regions of the brain. These regions have similar roles in the engram in the sense that they all seem to participate in responses to external events and in subsequent representations of these events. However, the relative contribution of these processes to the activity of different brain regions is quantitatively different, in that some regions are much more strongly affected than others. These results may constitute the basis for reconciliation of localizationist and antilocalizationist views of brain function.*

Almost half a century ago, Lashley formulated his well-known law of mass action (*1*), a quantitative assessment of the effects on learned behaviors observed after brain lesions in rats, asserting that the amount of functional loss was roughly proportional to the volume of tissue excised, independent of locus. Implicit in these findings was the law of equipotentiality, that is, a variety of brain regions had the potentiality to perform the functions normally mediated by some specific region in the intact brain. More recent studies of brain damage in man have provided some support for these ideas, in that

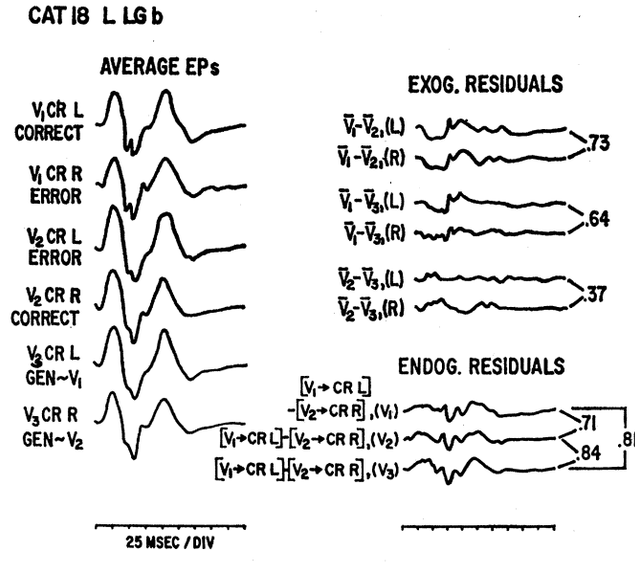

Fig. 1. (Left) Average evoked potentials obtained from the left lateral geniculate body (bipolar derivation) of a cat subjected to differential behavioral generalization during trials in which six different stimulus-response contingencies were represented: V_1CR L, the cat pressed the left lever in correct response to stimulus V_1; V_1CR R, the cat pressed the right lever in erroneous response to V_1; V_2CR L, the cat pressed the left lever erroneous response to V_2; V_2CR R, the cat pressed the right lever in correct response to V_2; V_3CR L, the cat pressed the left lever in response to the novel stimulus V_3 during test of differential generalization; V_3CR R, the cat pressed the right lever in response to V_3. (Top right) Waveshapes representing pairs of exogenous residuals obtained by manipulating the appropriate averages as described in the text. (Bottom right) Waveshapes representing independent estimates of endogenous residuals, obtained by subtraction of the appropriate average response waveshapes. Numbers at the right refer to Pearson product moment correlation coefficients between the bracketed waves.

many different signs of deficit can be observed after damage to a particular brain region (*2, 3*) and deficit in a specific function can be demonstrated after damage to a wide variety of brain regions (*3, 4*). Such findings, vaguely suggestive of the capacity of the brain as a whole to process information, are difficult to reconcile with the specificity of anatomical connections and the dramatic functional deficits sometimes caused by localized brain lesions. The studies reported here may provide the basis for reconciliation of what might be called the "localizationist" and "anti-localizationist" views of brain function.

In a series of publications (*5–8*) we have provided evidence that the evoked potential contains components which reflect the release of memories activated by afferent input as well as the direct impact of that input upon the electrical activity of a brain region. These two kinds of processes have been respectively identified as "endogenous" and "exogenous." In this report we describe a set of procedures that has enabled us to quantify the contribution of endogenous processes to the average evoked potentials (AEP's) recorded from a brain region. Because the effects we wanted to detect might well constitute weak contributions to the AEP, easily obscured by the effects of noise and variability in the processes, our procedures were designed to evaluate the statistical features of a very large amount of data so that we would be able to detect even weak effects.

Eighteen adult cats were trained to differentiate between light stimuli flickering at two different frequencies, V_1 and V_2. The conditioned responses, CR_1 and CR_2, were approach-approach for 4 cats, avoidance-avoidance for 3 cats, and approach-avoidance for 11 cats. Lever-pressing responses were used in some cats, and hurdle-jumping in others. Instrumental shaping and observational training (*9*) were used. Cue frequencies ranged from one to eight flashes per second. Flicker was delivered from a silent fluorescent tube mounted in the top of a dimly illuminated cage (60 by 60 by 60 cm) with a Lucite work panel in one wall and a grid floor. Evoked potentials were recorded from 34 electrodes permanently implanted in each cat (*10*). After periods of overtraining ranging from 6 months to 7 years, evoked potentials were recorded during several long sessions in which the cats were subjected to differential behavioral generalization: a novel flicker stimulus, V_3, whose frequency was intermediate between V_1 and V_2, was occasionally interspersed within a random sequence of the conditioned stimuli, V_1 and V_2 (*8*).

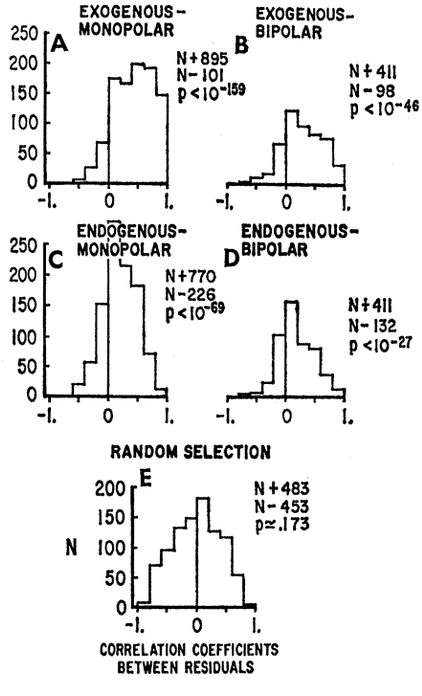

Fig. 2. Distribution of correlation coefficients between exogenous (A and B) and endogenous (C and D) residual waveshapes, computed according to the equations shown in Table 2. (A and C) Distributions obtained from data derived from monopolar leads; (B and D) from data derived from bipolar leads. (E) Distribution obtained by performing the same algebraic manipulations on waveshapes randomly selected from the records obtained from each derivation. Beside each histogram, $N+$ denotes the number of correlation coefficients with positive sign and $N-$ those with negative sign. P values represent the exact probability of obtaining the observed distributions from a symmetrical population with zero median.

Because evidence exists that some interactions among evoked potentials are approximately linear (*5, 11*), we assumed that the constituent exogenous and endogenous processes were separable by algebraic manipulation. The exogenous and endogenous processes postulated to be present in the AEP's

Table 1. Equations representing the exogenous and endogenous processes corresponding to six stimulus-response contingencies. The symbols $\bar{V}_1$, $\bar{V}_2$, $\bar{V}_3$ denote exogenous processes corresponding to the three different flicker stimuli. The expressions in parentheses denote endogenous processes. The arrows, as in $(V_1 \rightarrow CR_1)$, for example, indicate that the stimulus V_1 means CR_1, that particular stimulus-response contingency having been learned.

Trials resulting in CR_1	Trials resulting in CR_2
$V_1CR_1 = \bar{V}_1 + (V_1 \rightarrow CR_1)$	$V_1CR_2 = \bar{V}_1 + (V_2 \rightarrow CR_2)$
$V_2CR_1 = \bar{V}_2 + (V_1 \rightarrow CR_1)$	$V_2CR_2 = \bar{V}_2 + (V_2 \rightarrow CR_2)$
$V_3CR_1 = \bar{V}_3 + (V_1 \rightarrow CR_1)$	$V_3CR_2 = \bar{V}_3 + (V_2 \rightarrow CR_2)$

corresponding to the six stimulus-response contingencies are represented by the equations shown in Table 1. If we assume that the AEP's are composed of the sums of exogenous and endogenous processes indicated in these six equations, then six representations of exogenous processes and three representations of endogenous processes can be independently constructed by carrying out the algebraic manipulations shown in Table 2. These nine equations show that algebraic manipulation of independently obtained AEP's yields three pairs of identical residuals of exogenous processes, that is, Eq. 1 = Eq. 2 = $\bar{V}_1$–$\bar{V}_2$; Eq. 3 = Eq. 4 = $\bar{V}_1$–$\bar{V}_3$; Eq. 5 = Eq. 6 = $\bar{V}_2$–$\bar{V}_3$, and three identical residues of endogenous processes, that is, Eq. 7 = Eq. 8 = Eq. 9 = $(V_1 \rightarrow CR_1)$–$(V_2 \rightarrow CR_2)$. If, in fact, the stimuli V_1, V_2, and V_3 and the decision-making processes resulting in performance of CR_1 and CR_2 were represented by separable time-locked neural activity in the AEP, then the putatively identical residual waves that would result from our performing the indicated operations on AEP's from trials with the appropriate stimulus-response contingencies should have similar shape. Thus, the cross-correlation coefficients between the various appropriate residual waveshapes provide a quantitative scale which represents the variance in the AEP's recorded from a given brain region contributed by stimulus-related exogenous and memory-released endogenous processes.

We have applied these operations to all the data obtained from the 18 trained cats during a total of 79 experimental sessions in which the cats were subjected to differential generalization. Figure 1 illustrates typical AEP's recorded from trials representing the six possible stimulus-response contingencies and shows the three pairs of exogenous residuals and the three endogenous residuals computed according to the equations shown in Table 2. The numbers to the right of the residuals show the Pearson product moment correlation coefficients computed between the bracketed residual waves. There is

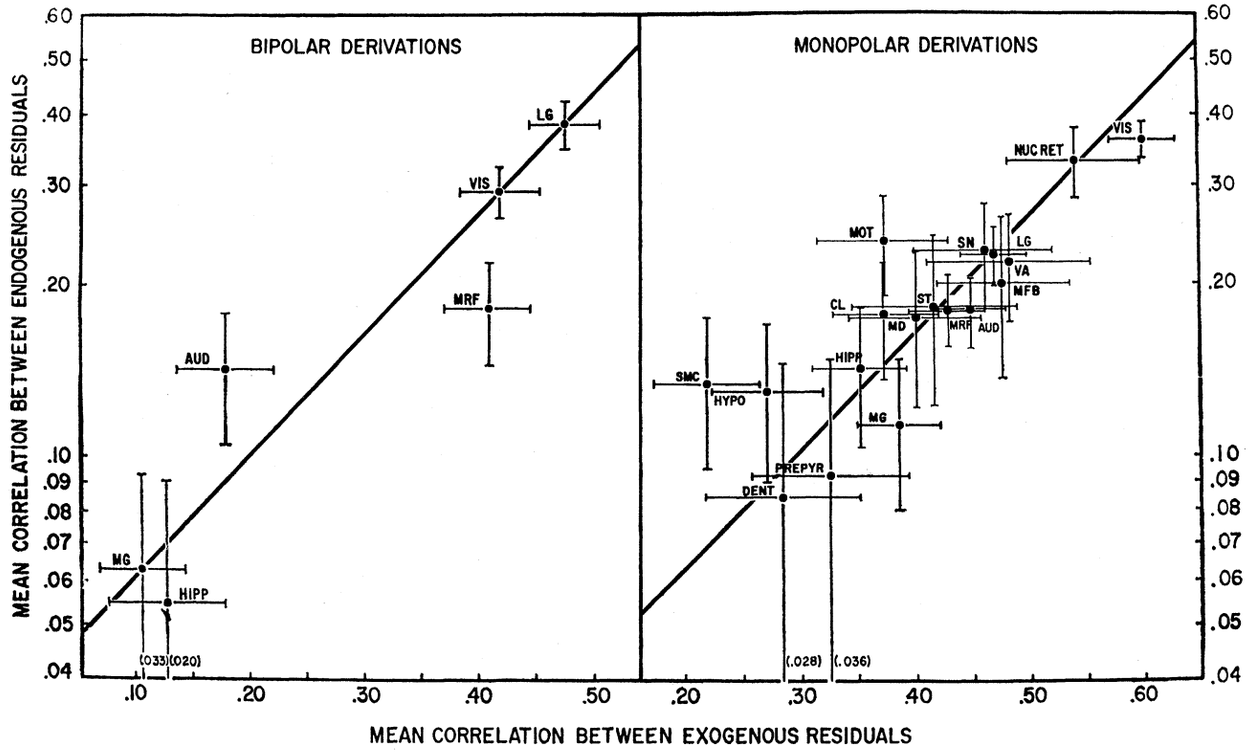

Fig. 3. Mean correlation coefficients between exogenous residuals plotted against those between endogenous residuals for different brain regions. Brackets indicate one standard error. (Bipolar derivations) *AUD*, auditory cortex ($N = 38$, 8 cats); *HIPP*, hippocampus ($N = 49$, 14 cats); *LG*, lateral geniculate ($N = 81$, 16 cats); *MG*, medial geniculate ($N = 65$, 14 cats); *MRF*, mesencephalic reticular formation ($N = 54$, 16 cats); *VIS*, visual cortex ($N = 77$, 16 cats). (Monopolar derivations) *AUD*, auditory cortex ($N = 103$, 16 cats); *CL*, nucleus centralis lateralis ($N = 47$, 10 cats); *DENT*, dentate ($N = 25$, 5 cats); *HIPP*, hippocampus ($N = 62$, 16 cats); *HYPO*, hypothalamus ($N = 27$, 6 cats); *LG*, lateral geniculate ($N = 124$, 18 cats); *MD*, nucleus medialis dorsalis ($N = 32$, 5 cats); *MFB*, medial forebrain bundle ($N = 24$, 6 cats); *MG*, medial geniculate ($N = 89$, 16 cats); *MOT*, motor cortex ($N = 22$, 4 cats); *MRF*, mesencephalic reticular formation ($N = 91$, 18 cats); *NUCRET*, nucleus reticularis ($N = 26$, 5 cats); *PREPYR*, prepyriform cortex ($N = 24$, 4 cats); *SMC*, sensorimotor cortex ($N = 29$, 4 cats); *SN*, substantia nigra ($N = 25$, 9 cats); *ST*, nucleus subthalamus, $N = 24$, 5 cats); *VA*, nucleus ventralis anterior ($N = 22$, 9 cats); *VIS*, visual cortex ($N = 105$, 18 cats). *N* denotes the number of independent measurements; data were included only from regions from which more than 20 independent measurements were available.

Table 2. Equations representing independently obtained AEP's manipulated to yield three pairs of identical residuals of exogenous processes and three identical residuals of endogenous processes.

Eq. No.	Trial outcomes	Processes represented in AEP's	Residuals
		Exogenous processes	
1	$V_1CR_1 - V_2CR_1$	$= [\bar{V}_1 + (V_1 \rightarrow CR_1)] - [\bar{V}_2 + (V_1 \rightarrow CR_1)]$	$= \bar{V}_1 - \bar{V}_2$
2	$V_1CR_2 - V_2CR_2$	$= [\bar{V}_1 + (V_2 \rightarrow CR_2)] - [\bar{V}_2 + (V_2 \rightarrow CR_2)]$	$= \bar{V}_1 - \bar{V}_2$
3	$V_1CR_1 - V_3CR_1$	$= [\bar{V}_1 + (V_1 \rightarrow CR_1)] - [\bar{V}_3 + (V_1 \rightarrow CR_1)]$	$= \bar{V}_1 - \bar{V}_3$
4	$V_1CR_2 - V_3CR_2$	$= [\bar{V}_1 + (V_2 \rightarrow CR_2)] - [\bar{V}_3 + (V_2 \rightarrow CR_2)]$	$= \bar{V}_1 - \bar{V}_3$
5	$V_2CR_1 - V_3CR_1$	$= [\bar{V}_2 + (V_1 \rightarrow CR_1)] - [\bar{V}_3 + (V_1 \rightarrow CR_1)]$	$= \bar{V}_2 - \bar{V}_3$
6	$V_2CR_2 - V_3CR_2$	$= [\bar{V}_2 + (V_2 \rightarrow CR_2)] - [\bar{V}_3 + (V_2 \rightarrow CR_2)]$	$= \bar{V}_2 - \bar{V}_3$
		Endogenous processes	
7	$V_1CR_1 - V_1CR_2$	$= [\bar{V}_1 + (V_1 \rightarrow CR_1)] - [\bar{V}_1 + (V_2 \rightarrow CR_2)]$	$= (\bar{V}_1 \rightarrow CR_1) - (\bar{V}_2 \rightarrow CR_2)$
8	$V_2CR_1 - V_2CR_2$	$= [\bar{V}_2 + (V_1 \rightarrow CR_1)] - [\bar{V}_2 + (V_2 \rightarrow CR_2)]$	$= (\bar{V}_1 \rightarrow CR_1) - (\bar{V}_2 \rightarrow CR_2)$
9	$V_3CR_1 - V_3CR_2$	$= [\bar{V}_3 + (V_1 \rightarrow CR_1)] - [\bar{V}_3 + (V_2 \rightarrow CR_2)]$	$= (\bar{V}_1 \rightarrow CR_1) - (\bar{V}_2 \rightarrow CR_2)$

a high correspondence between residuals representing hypothetically similar processes.

Figure 2 shows that the histograms of correlation coefficients representing hypothetically similar processes are markedly skewed to the right, while the distribution of correlation coefficients between randomly selected AEP's is symmetrical around zero. These results indicate that the hypothetically similar residuals predicted by the equations did in fact exist, supporting the contention that the postulated separable exogenous and endogenous processes were present in the AEP's recorded under these circumstances.

Figure 3 shows, for numerous anatomical regions, the results obtained by plotting the mean correlations between the residuals due to exogenous influences against the mean correlations between residuals due to endogenous influences. These data reveal a hierarchical organization in the representation of exogenous and endogenous processes in different structures and suggest that a logarithmic relationship exists between these two different processes in any given brain region.

Evidence that endogenous influences represent the readout of specific memories and are not to be attributed to nonspecific factors has been presented elsewhere (*7*). This evidence was obtained from several experiments in each of which a group of animals provided a control for a particular nonspecific factor. Results from all these experiments were subsequently combined to provide the largest possible amount of data on which to base the statistical computations described herein. Although examination of the data from each control group revealed no clearly apparent contradiction to the relationship illustrated in Fig. 3, the possibility of heterogeneous variance among these groups has not been excluded.

These findings suggest that the representation of an experience, the engram, is widely distributed throughout the neuraxis. This diffuse representation of the engram might explain the resistance of memories to lesions and might underlie the phenomena that led Lashley to the formulation of the laws of mass action and equipotentiality. On the other hand, the data show that the participation of an anatomical region in the representation of an experience is logarithmically proportional to the impact of those sensory events upon that region. The great quantitative range spanned by these data may explain why severe specific functional deficits are sometimes caused by localized lesions in some structures but not in others.

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References and Notes

1. K. S. Lashley, *Science* **73**, 245 (1931).
2. H. L. Teuber, in *Evolution of Nervous Control* (AAAS, Washington, D.C., 1959), p. 157.
3. A. R. Luria, *Higher Cortical Functions in Man* (Basic Books, New York, 1966).
4. W. Penfield and L. Roberts, *Speech and Brain Mechanisms* (Princeton Univ. Press, Princeton, N.J., 1959).
5. E. R. John, *Mechanisms of Memory* (Academic Press, New York, 1967).
6. ———, in *Psychobiology*, J. McGaugh, Ed. (Academic Press, New York, 1971), p. 199.
7. ———, *Science* **177**, 850 (1972); ———, F. Bartlett, M. Shimokochi, D. Kleinman, *J. Neurophysiol.*, in press; E. R. John and D. S. Ruchkin, *Science* **153**, 209 (1966).
8. E. R. John, M. Shimokochi, F. Bartlett, *Science* **164**, 1534 (1969).
9. E. R. John, P. Chesler, F. Bartlett, I. Victor, *ibid.* **159**, 1489 (1968).
10. E. R. John, *Methods Med. Res.* **2**, 251 (1964).
11. D. Arnol and P. Gerin, *Electroencephalogr. Clin. Neurophysiol.* **26**, 325 (1969); M. Clynes, *Proceedings of the 6th International Conference on Medical Electronics and Biological Engineering*, Tokyo (1965); E. Donchin and D. B. Lindsley, *Electroencephalogr. Clin. Neurophysiol.* **19**, 325 (1965); D. A. Jeffreys, *ibid.* **24**, 596 (1968); S. Sutton, M. Braren, J. Zubin, E. R. John, *Science* **150**, 1187 (1965); M. W. Van Hof, *Acta Physiol. Pharmacol. Neer.* **9**, 210 (1960).
12. This work was supported by NIH grant MH20059, NSF grant GB-27559X, and the Health Research Council of New York grant I-752.

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Operant-Controlled Evoked Responses: Discrimination of Conditioned and Normally Occurring Components

Abstract. *Rats were rewarded for signaling large and small sensory evoked components with appropriate bar presses. Most rats operantly generated large components and correctly signaled only these. Two rats correctly signaled successful and unsuccessful attempts to generate large waves. One rat discriminated component amplitudes without operantly attempting to generate specific wave types.*

Operant conditioning of sensory evoked components has been frequently reported (*1*, *2*). The phenomenon in humans and subhumans is not trivially mediated by changes in receptor orientation or by execution of discrete skeletal responses (*1*, *2*). It has been assumed that organisms control their neural activity by learning to discriminate and generate familiar psychological states whose neural correlates are the reinforcement-specified changes (*1*). Such a view suggests that organisms should be able to discriminate differences in a conditionable neural parameter, since operant conditioning of the neural event hypothetically proceeds by the organism's first learning to discriminate the reinforced event from other events. We explored this possibility by reinforcing rats for correctly signaling the size of a flash-evoked cortical component with an appropriate bar press. It was found that such discrimination behavior can be acquired, although rarely in the absence of attempts by the animals to generate particular kinds of evoked potentials.

Discrimination of neural events has been previously reported (*3*). In this