taken (with updates) from Maynard and Dando's review of stomach anatomy. Their CP (cardiopyloric), GP (gastro-pyloric), LC (lateral cardiac), and LG (lateral gastric) neurons are equivalent to the DG, MG, LG, and LPG neurons, respectively of (2). The names of other STG neurons are AM (anterior median), GM (gastric mill), Int 1 (interneuron 1), PD (pyloric dilator), AB (anterior burster), LP (lateral pyloric), VD (ventricular dilator), IC (inferior cardiac), and PY (pyloric) of which there are two subgroups, PE (early PY) and PL (late PY); D. K. Hartline and D. V. Gassie, Jr., in preparation.
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15. Although plateaus were most clearly demonstrated while a cell's firing was suppressed by steady hyperpolarization, plateau properties are present in nonpolarized cells, as we have shown for most cell types by prematurely triggering and terminating ongoing bursts with current pulses (as in Fig. 1, G and H).
16. Some of the rare spontaneous gastric rhythms in the isolated STG (2) may have been due to the occurrence of plateau potentials in gastric neurons even though central inputs were severed. We have observed the CP to burst endogenously in one such isolated STG to date.
17. Plateau behavior in the isolated STG, either spontaneous or following stimulation of the input nerve, rules out the possibility of its being due to network interactions involving neurons in central ganglia.
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20. We also tried replacement of Ca^{2+} with 1 to 5 m*M* Ba^{2+} in isolated STG's, which resulted in the occurrence of prominent plateaus in the same gastric neurons as exhibited plateaus under central input. We hesitate to use Ba^{2+} plateaus as a criterion for identifying "bursty" neurons since neurons which do not normally generate plateaus will do so with Ba^{2+} [for example, the crayfish and lobster stretch receptors; Y. Washizu, *Comp. Biochem. Physiol.* **15**, 535 (1965)].
21. We thank D. Gassie and C. Sirchia for technical assistance, and I. Cooke for critical comments. Supported by NIH grant NS13138.

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Norepinephrine in Chronic Paranoid Schizophrenia: Above-Normal Levels in Limbic Forebrain

Abstract. *In postmortem examination of brains of four patients with chronic paranoid schizophrenia, above-normal norepinephrine levels were measured in the ventral septum, the bed nucleus of the stria terminalis, the nucleus accumbens, and the mammillary bodies. No changes were detected in other limbic forebrain regions, including the hypothalamus and the medial olfactory (preoptic) area. The results point to the possibility of a malfunction of limbic noradrenergic mechanisms in schizophrenia, especially the paranoid variety.*

Brain catecholamines (CA), both dopamine (DA) and norepinephrine (NE), have been implicated in the pathophysiology of schizophrenia (*1*). In this respect, the evidence includes (i) the psychotogenic activity of drugs (such as *d*-amphetamine and *l*-dopa) that increase the synaptic availability of brain CA (*2*); (ii) the ability of neuroleptics with antipsychotic activity to block peripheral (*3*) and central (*4*) CA receptors; and (iii) the reduced activity of DA β-hydroxylase in the brain of schizophrenic subjects (*5*); this latter finding, however, has been disputed (*6*). [Studies on the CA-related enzymes monoamine oxidase (*7*) and DA-stimulated adenylate cyclase (*8*), as well as a preliminary study on DA and homovanillic acid (*9*), have failed to provide a uniform picture.] The limbic forebrain has been suggested as a possible seat of the behavioral abnormalities seen in schizophrenic subjects (*10*). The limbic forebrain in the rat contains not only a rich dopaminergic innervation and a DA-sensitive adenylate cyclase (*11*), but also an NE-sensitive adenosine 3′,5′-monophosphate generating system which is blocked in a dose-dependent manner by neuroleptics with antipsychotic activity (*12*). Thus, although the apparent correlation between the antipsychotic potency of neuroleptics and their action on dopaminergic systems (*13*) in general favors the view that brain DA is implicated in schizophrenia (*14*), there also is evidence suggesting a malfunction of noradrenergic mechanisms (*5, 15, 16*).

Our studies on the distribution of NE (*17*), DA (*9*), and serotonin (*18*) in the human brain have shown that NE has the strongest limbic representation of the three major brain amines in this species. Thus, the highest levels of NE in the forebrain (means of 1 to 2 μg per gram of wet tissue) occur in the hypothalamus, nucleus accumbens, medial olfactory (preoptic) area, bed nucleus of the stria terminalis, and the central amygdaloid nucleus; the nuclei of the ventral septum as well as the mammillary body and the paramedian thalamic region also contain appreciable concentrations of NE (means of about 0.5 μg/g).

We present data here on the distribution of NE in limbic forebrain regions of four patients with chronic paranoid schizophrenia. In this study postmortem brain material was used; it included the following groupings (*19*): (i) four patients diagnosed by Bleuler's criteria as chronic paranoid schizophrenics, (ii) three individuals with no diagnosed disease who committed suicide, and (iii) 12 controls with no evidence of psychiatric or neurologic disease. In an earlier study of neu-

Table 1. Norepinephrine in limbic brain areas of four schizophrenic subjects compared with controls specifically matched as to age and postmortem interval. Of a given control brain, not all of the listed regions were available for analyses. The statistical significance of differences was determined with a two-tailed *t*-test; *N*, number of controls; S.E.M., standard error of mean.

Brain region	Norepinephrine (micrograms per gram of wet tissue)				
	Controls			Schizophrenics	
	N	Mean ± S.E.M.	Range	Mean ± S.E.M.	Percentage of control
Hypothalamus, total	12	1.83 ± 0.18	0.91–2.71	1.86 ± 0.22	102
Hypothalamus, anterior	4	2.29 ± 0.31	1.48–2.29	2.07 ± 0.30	90
Hypothalamus, posterior	4	1.64 ± 0.36	0.75–2.41	1.88 ± 0.18	115
Hypothalamus, lateral	12	1.49 ± 0.12	0.89–2.20	1.63 ± 0.17	109
Nucleus accumbens	8	1.58 ± 0.16	1.21–2.36	2.40 ± 0.27*	152
Medial olfactory (preoptic) area	8	1.49 ± 0.27	0.56–2.51	1.69 ± 0.19	113
Bed nucleus of stria terminalis	4	1.23 ± 0.17	0.84–1.69	2.72 ± 0.26†	221
Ventral septum	4	0.53 ± 0.11	0.33–0.82	1.59 ± 0.24‡	300
Mammillary body	12	0.45 ± 0.02	0.33–0.58	0.69 ± 0.16§	153
Paramedian thalamic nuclei	4	0.48 ± 0.02	0.44–0.51	0.53 ± 0.06	110

*$P < .02$. †$P < .005$. ‡$P < .01$. §$P < .05$.

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Table 2. Influence of neuroleptic drug treatment and suicide on norepinephrine concentrations in limbic forebrain areas of schizophrenic cases. Values are expressed as percentage of those of controls (Table 1); *N*, number of cases.

Group	Norepinephrine concentration (percentage of control)				
	Hypothalamus	Medial olfactory	Nucleus accumbens	Nucleus striae terminalis	Ventral septum
		Neuroleptic drug treatment			
Schizophrenic					
No neuroleptics (case 1)	75	101	180*	217*	166*
Acute chlorpromazine (cases 2 and 3)	134, 105	117, 88	108, 142	265,* 165*	342,* 347*
Chronic piperacetazine (case 4)	95	146	178*	236*	343*
		Suicide			
Nonschizophrenic ($N = 3$)	89	95	102	ND	108†
Schizophrenic (cases 2, 3, and 4)	111	117	142	222	343

*Value outside the upper range of control (Table 1). †Only two cases examined.

rologically normal brains NE levels did not vary with either patient age (19 to 77 years) or postmortem interval (6 to 33 hours) (*17*). However, the controls in group (iii) were matched to the schizophrenic cases with regard to both age and postmortem interval. All procedures in handling and freezing of the brains following autopsy and dissection of discrete brain regions were performed as described (*17, 20*). Norepinephrine was assayed either spectrofluorimetrically after separation on Dowex columns (*21*) or by a radioenzymatic procedure (*22*). All values were corrected for dilution factors and losses during the extraction procedure; they are expressed as micrograms of NE per gram of wet tissue. The chemical analyses, but not the dissections, were performed without knowledge of the subject's clinical history.

In the schizophrenic cases the most conspicuous finding was a significantly above-normal concentration of NE in several of the NE-rich areas of the limbic forebrain (Table 1). The nucleus accumbens and the mammillary body of the schizophrenics had approximately 50 percent higher NE levels than did those of controls ($P < .02$ and $< .05$, respectively). For the bed nucleus of the stria terminalis the difference was more than 100 percent ($P < .005$). However, the most conspicuous change was in the ventral septum, where the mean NE level in schizophrenics was threefold higher than the corresponding control mean ($P < .01$).

Sharply contrasting with this increase of NE levels in these regions of the limbic forebrain (the change in the mammillary body possibly being of borderline significance only), none of the other limbic areas examined, including the NE-rich hypothalamus and medial olfactory (preoptic) area, displayed any clear changes. Thus the observed above-normal limbic NE values in these cases of chronic paranoid schizophrenia appear to reflect a rather localized change, possibly confined to a functionally distinct subsystem within the limbic forebrain, rather than a derangement of the whole system.

Two major sources of error not related to the disease process could have influenced the neurochemical parameters in our schizophrenic subjects. These are drug treatment (neuroleptics) and mode of death (suicide). Three of four schizophrenics received some neuroleptic treatment and died from traumatic suicide (*19*). However, neither of these factors can account for the specific pattern of NE changes found in our cases (Table 2). While in respect to neuroleptic drug treatment the schizophrenic sample was quite inhomogeneous (one untreated, one chronically treated, and two acutely treated patients), the pattern of limbic NE changes in each case was basically the same. [Neither acute nor subacute chlorpromazine administration influenced brain NE levels in laboratory animals (*23*)]. Also, the NE levels in three suicides with no known disease were comparable with the control values (Table 2). Thus, it seems reasonable to conclude that the above-normal NE levels in certain limbic forebrain areas in these cases of paranoid schizophrenia were related to the disease process.

Depending on the synaptic neurotransmitter dynamics underlying the steady-state condition, our measurement of above-normal limbic NE in the examined cases of paranoid schizophrenia could, in principle, be due to either increased or decreased activity of the NE neurons involved. A deficit in noradrenergic brain activity has been hypothesized as a possible factor in schizophrenia (*5, 16*). However, a denser-than-normal NE innervation of the limbic areas in question also could produce the observed effect of above-normal NE levels (*24*). More definitive conclusions may be drawn when data on the behavior of NE metabolites become available.

Although our results suggest a malfunction of the limbic NE system, much evidence implicates brain DA in schizophrenia. It may be that the NE and DA systems converge on a common "crucial" receptor structure in the limbic forebrain. If the functions of these CA systems are dynamically interrelated (*25*), a malfunction of the NE system could result in a relative predominance of the DA system. Thus, the therapeutic efficacy of the antidopaminergically active neuroleptics in schizophrenia may be an example of an indirect therapeutic effect rather than chemotherapy aimed at correcting the primary neurochemical disturbance.

In conclusion, we suggest that the significantly above-normal NE levels within some limbic forebrain regions, especially the ventral septum and the bed nucleus of the stria terminalis, may be a specific neurochemical feature, or concomitant, of chronic paranoid schizophrenia. If these results are confirmed in a larger series of cases, the theoretical and therapeutic implications would be manifold.

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19. Group (i): schizophrenic patients (chronic paranoid). There were three females and one male; mean age, 42 ± 12.1 years; postmortem interval, 13.1 ± 4.7 hours. *Case No. 1*: female, age 75; duration of illness, 43 years (institutionalized); treatment, no neuroleptic drugs at any time, Trimeton (antihistaminic), Noludar (hypnotic), Lasix, Feosol, vitamin C; cause of death, coronary thrombosis. *Case No. 2*: female, age 45; duration of illness, 10 years; treatment, no indication of chronic neuroleptic treatment, chlorpromazine, 650 and 750 mg daily (orally) 2 days before death, 250 mg 4 hours before death; cause of death, suicide (jumped from high window). *Case No. 3*: male, age 24; duration of illness, not known but chronic; treatment, no indication of chronic neuroleptic treatment, 1100 mg of chlorpromazine (orally) within 24 hours before death; cause of death, suicide (hanging). *Case No. 4*: female, age 24; duration of illness, 9 years; treatment, piperacetazine, 400 mg daily until death; cause of death, suicide (jumped in front of train). Group (ii): mode-of-death controls. There were three males, all suicides; mean age, 39.7 ± 10.7 years, postmortem interval, 17.8 ± 3.9 hours. They received no antidepressant or other known drug therapy. Death was caused by CO poisoning, jumping in front of a bus, and jumping from a high window. Group (iii): normal controls. There were ten males and two females; mean age, 44.7 ± 6.1 years. The ratio of young to middle aged to old was 2:1:1, same as for group (i); this ratio was maintained for brain regions for which fewer than 12 controls (subgroups of eight or four in Table 1) were analyzed. The postmortem interval was 16.5 ± 2.5 hours for the group, 16.3 ± 2.5 hours for subgroup of eight, and 13.9 ± 2.7 hours for subgroup of four. Causes of death were myocardial infarction (eight cases), accidental chest trauma (three cases), and stabbing (one case).
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24. A relative increase in the density of NE terminals could result from a degeneration of the cellular elements in the affected areas with the NE terminals remaining intact. However, this possibility appears somewhat unlikely because we have evidence that other biochemical parameters, such as DA and serotonin levels, do not change in an analogous manner in the areas in question (unpublished observations). An absolute increase in the density of NE terminals could be due to several possible processes, such as faulty overdevelopment of the corresponding NE system or systems; failure of some NE terminals to regress, as part of a normal process, during postnatal development (analogous to the apparently normal decrease of the nigrostriatal DA during adolescence) [A. Carlsson and B. Winblad, *J. Neural Transm.* **38**, 271 (1976)]; or sprouting of NE nerve endings in response to damage of some other neuronal system or systems impinging on the same perikarya [G. Raisman, *Brain Res.* **14**, 25 (1969); R. Y. Moore, A. Björklund, U. Stenevi, *ibid.* **33**, 13 (1971)]. In this latter respect, the possibility of a primary damage of the neuron system containing γ-aminobutyric acid is especially provoking [E. Roberts, *Neurosci. Res. Program Bull.* **10**, 468 (1972)]. Any of these factors may confer on the afflicted brain regions the kind of special biological vulnerability that has been hypothesized as one of several possible factors predisposing the affected individual to schizophrenia [S. S. Kety, *Semin. Psychiatry* **4**, 233 (1972); W. Pollin, *Arch. Gen. Psychiatry* **27**, 29 (1972)].
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Zooplankton Niches and the Community Structure Controversy

In a recent report Makarewicz and Likens (*1*) interpret the results of their study of the zooplankton community in Mirror Lake, New Hampshire, as supporting the individualistic concept of natural communities (*2*). They state that "the finding parallels Ramensky's . . . and Gleason's . . . concepts of species individuality and community continuity." Ecologists have long argued about whether natural communities constitute highly structured entities. Pielou (*3*) has defined community structure as "the amount of interdependence among the species." Thus, the more biological interactions that occur in a given community, the stronger the statistical associations among species and the more definite the structure. Obviously, the degree of structure will vary with the taxonomic unit that is considered. For example, much of the support for the individualistic concept has come from studies of terrestrial plant communities (*4*). Zooplankton communities in lakes, however, exhibit a definite structure which invalidates the application of the individualistic concept as proposed by Makarewicz and Likens (*1*). My argument consists of two parts: why they did not find community structure, and how it can be found. Because the community structure controversy has important ecological implications, a careful evaluation of their interpretation is warranted.

Makarewicz and Likens (*1*) report on 48 productivity estimates of 15 zooplankton species. They collected nonreplicated samples at four depths each month for a year. They plotted isopleths (isolines) of these productivity values using two to four species per graph, with depth on the *Y* axis and time of year on the *X* axis. The isopleth representations are termed population response surfaces. Because mean monthly production values for the species are continuous and because the population response surfaces do not overlap greatly, Makarewicz and Likens conclude that "this intracommunity population structure is analogous to the extensive or intercommunity population continuum formed by communities in relation to habitat gradients." They do suggest, however, that the observed division of the niche space has resulted from species interactions.

The productivity estimates themselves are a valuable contribution to zooplankton ecology. My criticism concerns only the representation of the results and subsequent conclusions concerning community structure. Three central questions are (i) Do 48 environments (samples) per year adequately characterize the overlap patterns of a zooplankton community? (ii) Have the sets of species used to represent niche separation been appropriately chosen? (iii) Does the use of productivity values give a good estimate of species importance? The following remarks apply mostly to zooplankton communities: sweeping generalizations for all communities should be avoided until biological insights are better developed.

First, it is crucial in delineating community structure to select an appropriate spatial-temporal framework for the orga-

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