was about 90 percent of the control value. The loss at 21 kHz was somewhat greater for finger spelling. The best subjects (which include four of the five laboratory and university employees and just two or three of the others) were generally the youngest subjects in this sample: their ASL performance at 4.4 kHz was 40 to 50 percent of control. Their performance at 4.4 kHz is about what would be obtained with hearing subjects listening to voice communication over a 1.5-kHz (low-pass) channel (*15*). Thus, while a television picture may use more than 1000 times the bandwidth of a telephone line, visual transmission of ASL requires only a few times more bandwidth than voice communication.

The minimum bandwidth required for the video transmission of ASL by a raster scan is not known; the design of the present study yields only an upper bound. A more judicious choice of raster variables (fewer frames per second, more lines per frame, interlace, and so forth) would almost certainly reduce this upper bound substantially. The low bandwidth—the low information rate—makes possible more advanced picture coding by any of many schemes to further reduce the bandwidth.

Present video transmission systems thus use a much wider bandwidth than is required for transmission of ASL. By a sevenfold further reduction in the bandwidth required for ASL transmission or by a sevenfold increase in the bandwidth that can be carried on telephone facilities, the ASL-signing deaf population could—with appropriate video terminals—use our present telecommunication facilities for ASL communication.

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12. The recordings were made with a camera (Panasonic WV204P) having 4-mm and 8-mm lenses (Apollo) and with a cassette recorder (Sony V0-2800). The playback monitor was a 17-inch cathode-ray tube (Conrac QQA 17/N). For bandwidth calibration, vertical gratings that produced different numbers of cycles per centimeter on the monitor were recorded, and their modulation depth was measured with a microphotometer. Modulation depth diminished sharply to 50 percent of the maximum depth for coarse gratings at 2.0 ± 0.1 MHz. The sharp cutoff at 2 MHz was caused by the recorder; the camera and monitor easily passed 4 MHz. Recordings were made without interlace, producing 60 full pictures per second.
13. The number of horizontal raster lines in the four bandwidth conditions, respectively, was 79, 38, 17, and 9. (The full, noninterlaced television picture contained 262 lines.)
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Electroconvulsive Shock: Progressive Dopamine Autoreceptor Subsensitivity Independent of Repeated Treatment

Abstract. *Repeated electroconvulsive shock, applied to rats, induces a subsensitivity of dopamine autoreceptors located in the substantia nigra as indexed by single-unit electrophysiological techniques. This reduced sensitivity is time-dependent, since effects similar to those seen with repeated treatment were also observed when single electroconvulsive shock was followed by an appropriate treatment-free interval. These data, coupled with identical results after the repeated administration of tricyclic antidepressants, raise the possibility that a reduction of dopamine autoreceptor sensitivity could underlie both electroconvulsive shock and pharmacological treatment of depression.*

Electroconvulsive shock (ECS) is generally thought to be the most effective means of treating endogenous depression (*1*). It affects a variety of putative neurotransmitter or neuromodulator substances including norepinephrine, dopamine, serotonin, γ-aminobutyric acid, and the endorphins (*2*). Considerable attention has recently been directed toward receptor mechanisms possibly underlying the therapeutic efficacy of both ECS and the tricyclic antidepressants (TCA's) (*3*); the focal point of this research has been the norepinephrine system. Several studies have now demonstrated changes indicating decreased β-adrenergic receptor function after repeated treatment with either ECS or TCA's (*4*). Our own recent research has shown that repeated TCA administration induces a progressive subsensitivity of dopamine autoreceptors [dopamine receptors on the soma and dendrites of dopamine-containing neurons located in the zona compacta of the substantia nigra (SNC)] independent of daily drug administration (*5*). In light of these findings we inquired whether ECS could produce similar effects. We now report that ECS also gradually reduces dopamine autoreceptor sensitivity and that this process depends on the passage of time rather than on repeated shock treatment.

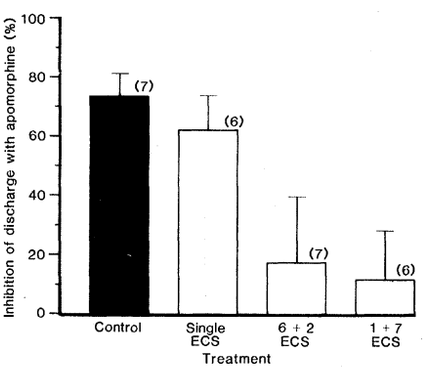

Fig. 1. The effects of various ECS treatments on the apomorphine-induced (0.004 mg/kg, intravenously) inhibition of the spontaneous activity of dopaminergic neurons of the SNC (*16*). For each treatment, *N* is shown in parentheses. Abbreviations: *1 + 7 ECS*, single ECS followed by 7 days without treatment; *6 + 2 ECS*, six daily ECS's followed by 2 days without treatment.

Twenty-seven male albino rats (200 to 250 g; Zivic-Miller) housed two per cage with free access to food and water were maintained on a 12-hour light/dark cycle. After a period of ECS treatment the animals were anesthetized with chloral hydrate (400 mg per kilogram of body weight, injected intraperitoneally), and the electrophysiological activity of dopaminergic neurons located within the SNC [anterior 1300 to 2400 μm, lateral 1300 to 2400 μm (*6*)] was monitored (*7*). Briefly, single unit neuronal activity was recorded through glass micropipettes filled with 2*M* NaCl saturated with Fast Green dye (in vivo impedance, 8 to 15 megohms). Action potentials were fed

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into a differential amplifier with high-input impedance (band-pass filters, 0.1 to 3 kHz) whose output was led into a window discriminator. The digital output of the window discriminator was simultaneously fed into a signal integrator and a digital frequency meter. Pharmacological agents were administered through a femoral catheter. All drug doses are based on the weight of the salt. At the end of each recording, the site of the micropipette tip was marked for later histological examination by passing a −40-μA current for 10 minutes (*8*).

Individual dopaminergic neurons were located on the basis of the electrophysiological criteria of Bunney *et al.* (*9*). That is, dopaminergic cells within the SNC spontaneously fire at 1 to 9 Hz, have biphasic (+/−) action potentials with amplitudes of 0.4 to 1.5 mV, and typically burst upon discharge. These cells are inhibited by the administration of the dopamine agonist apomorphine and subsequently increase their firing rate in response to the injection of the dopamine antagonist, haloperidol, thus also meeting the pharmacological criteria for identifying dopaminergic neurons (*10*).

In experiment 1, animals received ECS either once a day for 6 days or on only a single occasion 60 minutes before recording. Handled but unshocked animals with ear clips were controls. The ECS consisted of a 0.7-second pulse of a 105-V alternating current (60 to 100 mA) administered through ear-clip electrodes. The animals were recorded from 48 hours after the last ECS. Once a dopaminergic neuron was located, its spontaneous activity was monitored for a minimum of 4 minutes to establish a reliable baseline. Apomorphine was then administered. Since a 0.004-mg/kg intravenous dose of apomorphine inhibits the spontaneous activity of dopaminergic neurons by stimulating only dopamine autoreceptors (*11*), we used the inhibition obtained with this drug dose as our index of autoreceptor sensitivity.

We found that long-term ECS reduces the sensitivity of dopamine autoreceptors (Fig. 1). Six daily exposures to ECS significantly attenuated apomorphine's ability to inhibit the spontaneous discharge of dopaminergic neurons relative to both the single ECS group (Mann-Whitney U test, $U = 8$, $P = .037$) and controls ($U = 7$, $P = .013$). Single ECS had no effect on the inhibition produced by this presynaptic dose of apomorphine.

To determine the possible functional significance of the reduced sensitivity of dopamine autoreceptors, we also examined their responsiveness to tail pressure, a mildly activating, nonpainful environmental stimulus. Tail pressure induces dopamine-dependent behaviors and alters the discharge rate of mesencephalic dopaminergic neurons (*7*). The 6-day ECS group displayed a trend (though not significant) to be more responsive to tail pressure than the handled controls [mean percent change in discharge ± standard error (S.E.); controls, 43.2 ± 7.9; 6-day ECS group 70.9 ± 22.4].

We recently found that the progressively diminished dopamine autoreceptor sensitivity that follows repeated TCA treatment depends on the passage of time rather than on daily drug treatment (*5*). The same diminution of autoreceptor sensitivity was seen whether recording was done 2 days after 10 days of drug treatment or 10 days after 2 days of TCA administration. These findings raised the question of whether ECS-reduced sensitivity is also independent of daily shock treatment. We thus administered ECS a single time and 7 days later recorded from dopaminergic neurons. Rats in this group developed the same reduced sensitivity as the 6-day ECS group and differed significantly from both the control group ($U = 0$, $P = .001$) and from the group also given one ECS but tested only 60 minutes later ($U = 4$, $P = .013$) (Fig. 1). All cells reported were located within the SNC (Fig. 2).

Fig. 2. Histological localization of dopaminergic neurons sampled.

Our data indicate that ECS can reduce the sensitivity of dopaminergic autoreceptors. As with TCA's the diminished sensitivity seen with ECS depends on the passage of time. That is, a single ECS reduced sensitivity as much 7 days later as ECS given for 6 days. The only contrast between the present findings and those previously obtained with TCA's is in the responsiveness of dopaminergic neurons to mild tail pressure. Whereas all TCA treatments resulted in a significant augmentation of the change in firing rate produced by tail pressure (*5*), this did not occur with ECS. However, this difference may be more apparent than real since the same trend was seen with both ECS and pharmacological antidepressants.

Repeated (but not single) ECS has recently been reported to result in neuronal changes indicative of a decrease in β-adrenergic receptor functioning (*4*). It is possible that our data can be explained in terms of a similar mechanism. That is, ECS may also reduce the density of dopaminergic autoreceptors within the substantia nigra. Moreover, since repeated ECS is able to block reuptake in noradrenergic neurons (*12*), a similar blockade of dendritic reuptake may reduce autoreceptor sensitivity as a compensation for overstimulation.

An obvious issue raised by our findings is whether the modification of dopamine autoreceptor sensitivity has any relevance to depression and its treatment. While any attempt to answer this question is speculative, several points can be made. Depression may be in part related to a decrease in the functional activity of dopaminergic systems. Indirect support for this hypothesis comes from the finding that depression is often associated with parkinsonism (*13*), a disorder that reflects a severe decrease in nigrostriatal dopaminergic activity. Of greater relevance to endogenous affective illness is that dopaminergic function can also be attenuated in the absence of brain dam-

age by an increase in the sensitivity (supersensitivity) of dopaminergic autoreceptors. The development of such supersensitivity would make the dopaminergic neuron more vulnerable to the inhibitory effects of dendritic dopamine release (*14*). This in turn would result in increased autoregulation (that is, increased self-inhibition) of dopaminergic neuronal activity, thereby causing a decreased responsiveness to the environment and possibly depression. If our speculation is correct, the dopamine autoreceptor subsensitivity, such as we have now demonstrated with both ECS and tricyclics (*5*), might gradually reverse this supersensitivity and, in so doing, ameliorate depression. The passage of time required for the reduction of sensitivity is consistent with the delayed onset of the therapeutic influence of both ECS and tricyclic antidepressants (*15*). These data tentatively suggest, as previously proposed for TCA's (*5*), that it may not be necessary for ECS to be repeatedly administered in order for it to be therapeutically effective. Instead, a single ECS (or short-term treatment) may be sufficient to trigger autoreceptor subsensitivity, which, as it progresses, becomes functionally manifest as an antidepressant effect. Alternatively, autoreceptor sensitivity reduced by ECS may be an intermediate process providing the impetus for other neuronal changes necessary to achieve clinical efficacy. In either case, the hypothesis that repeated antidepressant treatments may not always be required warrants testing in a controlled clinical trial.

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Treatment	Inhibition of discharge (mean % ± S.E.)	*t*	d.f.	*P*
Control	73.6 ± 7.3			
Imipramine				
2 days	34.1 ± 12.4	4.134	12	.001
10 days	13.6 ± 13.9	6.550	11	.001
Amitriptyline				
2 days	40.7 ± 26.1	2.119	10	.05
10 days	2.7 ± 17.4	8.985	9	.001
Iprindole				
10 days	15.7 ± 9.4	4.832	9	.001

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16. One of the six animals in the single ECS + 7-day delay group and three of the six in the six ECS + 2-day delay group showed an increase in spontaneous activity after the administration of a presynaptic dose of apomorphine (0.004 mg/kg, intravenously). However, the dopaminergic nature of these cells was supported by the finding that they all decreased their activity when a postsynaptic dose of apomorphine was administered (50 μg/kg, intravenously) and increased their activity in response to haloperidol (0.2 mg/kg, intravenously) [L. A. Chiodo and S. M. Antelman, *Eur. J. Pharmacol.* **66**, 255 (1980)].
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Monkey Responses to Three Different Alarm Calls: Evidence of Predator Classification and Semantic Communication

Abstract. *Vervet monkeys give different alarm calls to different predators. Recordings of the alarms played back when predators were absent caused the monkeys to run into trees for leopard alarms, look up for eagle alarms, and look down for snake alarms. Adults call primarily to leopards, martial eagles, and pythons, but infants give leopard alarms to various mammals, eagle alarms to many birds, and snake alarms to various snakelike objects. Predator classification improves with age and experience.*

A central but neglected issue in the study of animal communication is that of semantics. Have species other than man evolved the ability to make systematic use of signals to refer to objects in the external world? Captive chimpanzees (*Pan troglodytes*) and gorillas (*Gorilla gorilla*) can be taught to use symbols to represent different objects without support from contextual cues (*1*), but whether nonhuman species exhibit a similar ability under natural conditions has yet to be investigated.

If an organism is to communicate semantically, using different signals to represent distinct objects in its environment, it must either employ many signals or sort objects into groups. Present indications are that repertoires of natural, meaningfully distinct signals are limited in animals. Thus any study of semantic communication under natural conditions must consider how animals "categorize" objects in the external world. The extent to which nonhuman species divide objects into groups for the apparent purpose of communicating about them is not known (*2*). We present field data on responses of free-ranging vervet monkeys (*Cercopithecus aethiops*) to playbacks of recorded predator alarm calls. Additional data illustrate how vervets classify predators and suggest how such categorization develops.

Three groups of free-ranging vervet monkeys were studied in Amboseli National Park, Kenya, for 14 months. Groups contained a mean of 4 adult males (range 2 to 7), 7.6 adult females (range 7 to 8), 6.2 juveniles (range 1 to 11), and 6.5 infants (range 6 to 8). During 1500 hours of systematic sampling of social behavior, tape recordings were made of as many vocalizations as possible (*3*). Analysis was limited to those instances in which observers identified the vocalizer, the stimuli that occasioned vocalization, and apparent responses of nearby individuals.

Field recordings of more than 100 alarm calls supported Struhsaker's finding (*4*) that vervets in Amboseli gave acoustically different alarm calls to at least three different predators: leopards

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