(*Ovis dalli*) hunters heard wolf howls on the nights of 9 to 10 and 11 to 12 August, and I heard two howls on 8 September (Fig. 1).

During 20 to 22 March 1978 I conducted 5 hours of aerial survey in a Turbo-Beaver aircraft along the Jago, Okpilak (located between the Jago and Hulahula rivers), Hulahula, and Canning rivers. A complete survey would have required at least 15 hours. Two black wolves and one gray wolf were 11 km north of the den location shown in Fig. 1. A sheep hunter had seen a black wolf in this area in early September 1977. These wolves may represent a pack that existed north of the Hulahula pack prior to the outbreak of rabies; there were no black wolves in the Hulahula pack. Two gray wolves were on the Canning River 48 km west of the den. Tracks of at least two wolves were on the Okpilak River 42 km northeast of the den. Wolf tracks were also seen on the Jago River and in most of the Canning drainage. These findings suggest that only the Hulahula pack was affected by rabies.

Indeed, several factors would make it unlikely in northeastern Alaska for a rabid wolf to infect a wolf in another pack. Wolf densities there are usually lower than one wolf per 180 km^2 (*22*). A rabid wolf, infective for only a few days, would have to leave its home range, then contact and infect another wolf before it died. [There is one report of a rabid wolf traveling at least 24 km after attacking a man (*12*).] Evidence described herein suggests that most rabid wolves seek familiar areas. Supporting data are sparse, but no one has reported significantly lowered wolf densities following local outbreaks of rabies in wolves (*12, 21, 23*), which suggests that pack-to-pack transmission of rabies is uncommon. Predator control, dispersing wolves filling vacant territories, and subsequent reproduction would mask the devastating effects of the disease.

Since there was a rabies outbreak among arctic foxes along the Arctic Coast (*7*), the most probable source of rabies was a rabid arctic fox. Because of a lack of prey on the coastal plain in northeastern Alaska, wolves are rarely seen near the coast. It is not uncommon, however, for arctic foxes to travel south from the coast, particularly when at high densities as they were in 1976 to 1977. A few were even seen south of the Brooks Range during this period (*7*). Moving south brings them in contact with wolves. Wolves will chase and attempt to kill foxes which they encounter (*14, 24*), and in so doing, may be bitten or may eat such a fox. If the fox is rabid, the wolf may become infected. In August at RS-2, I found the skull of an arctic fox showing evidence of chewing by a wolf, and the fox's chipped canines indicated that it may have bitten rocks, not done by normal foxes.

In an earlier incident, three wolves (one confirmed rabid) had attacked men or dogs within a 13-day period in a small area near Aklavik, Northwest Territories, Canada (*8*), which suggests that the three were packmates and were infected about the same time. Because members of wolf packs are socially close, most if not all pack members will be exposed to rabies if one member becomes rabid, decimating the pack. If this is usually true, wolves, which occur in discrete social units (packs) and low densities, would be sporadic hosts of rabies.

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Retrograde Amnesia Produced by Several Treatments: Evidence for a Common Neurobiological Mechanism

Abstract. *This experiment examined the effects on memory of various amnestic treatments in animals earlier treated with the α-adrenergic antagonist phenoxybenzamine (PBZ). Thirty minutes before being trained in a one-trial inhibitory (passive) avoidance task, animals received an injection of PBZ or saline. Immediately after training, each animal received one of the following amnestic treatments: stimulation of the frontal cortex or amygdala, pentylenetetrazol, diethyldithiocarbamate, or cycloheximide. In control animals, each treatment produced retrograde amnesia. However, PBZ-treated animals did not develop amnesia. These findings suggest that there may be a common neurobiological mechanism underlying the amnesias produced by many treatments.*

During the past 30 years, various treatments have been used to produce retrograde amnesia in animals (*1*). In many studies, a specific amnestic treatment was chosen because of a particular known neurobiological response to it. These studies represent indirect attempts to learn about the nature of memory storage processing by relating posttraining interference with the activity of certain neurobiological systems to deficits in later retention (*2*). Thus, the amnesias produced by electroconvulsive shock, some forms of direct electrical

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Table 1. Retention performance of rats and mice in a one-trial inhibitory avoidance task. Abbreviations: PBZ, phenoxybenzamine; PTZ, pentylenetetrazol; DDC, diethyldithiocarbamate; and CXM, cycloheximide.

Pretraining injection	Posttraining treatment	*N*	Median latency (sec)	Interquartile range (sec)	Animals at maximum latency (%)
		Control groups			
Saline	None	20	180	180 to 180	80
PBZ	None	10	180	180 to 180	90
Saline	Saline	15	180	180 to 180	80
PBZ	Saline	10	180	180 to 180	90
Saline	Sham*	10	180	134 to 180	70
PBZ	Sham*	10	180	180 to 180	80
Saline†	Saline	10	300	300 to 300	90
PBZ†	PBZ	10	300	300 to 300	90
		Experimental groups			
Saline	Cortex‡	11	56§	12 to 105	27
PBZ	Cortex‡	14	180‖	180 to 180	79
Saline	PTZ	15	44§	23 to 170	27
PBZ	PTZ	10	180‖	180 to 180	90
Saline	DDC	10	52§	15 to 150	30
PBZ	DDC	10	180	142 to 180	70
Saline	Amygdala‡	10	15§	11 to 36	20
PBZ	Amygdala‡	10	180‖	180 to 180	90
Saline†	CXM	10	150§	102 to 210	10
PBZ†	CXM	10	300‖	300 to 300	90

*Electrodes were implanted in the amygdala, but the stimulation was not delivered. †Subjects in the CXM study were mice rather than rats. ‡Electrical stimulation of the designated area. §Latencies significantly lower than those of pooled rat or mouse control groups ($P < .02$, two-tailed, Mann-Whitney U test). ‖Latencies significantly higher than those of saline-treated animals that received the same amnestic treatment ($P < .05$, two-tailed, Mann-Whitney U test).

stimulation of the brain, and convulsant drugs might be a consequence of behavioral or brain seizures. However, the evidence does not entirely support a causal relationship between seizures and amnesia (*3*). For example, direct subseizure electrical stimulation of specific brain regions also produces amnesia in some cases, which indicates that seizures are not necessary to impair memory (*3, 4*). Such findings might indicate that the regions stimulated (or areas receiving major anatomical projections from the stimulation sites) are involved in memory storage processing. In other studies, inhibitors of neurotransmitter synthesis (*5*) or protein synthesis (*6*) have been used to produce amnesia; these studies were generally attempts to determine the possible roles of these systems in memory storage. These studies, then, provide evidence supporting the view that several different neurophysiological, neuroanatomical, and neurochemical systems are involved in memory processing.

Recent findings indicate that peripheral injections of some hormones, such as adrenocorticotrophic hormone and epinephrine, can produce retrograde amnesia (*7*). In addition, pretraining treatment with phenoxybenzamine (PBZ), an α-adrenergic blocking agent, attenuates the amnesia produced by epinephrine (*8*). These results suggest that many amnestic treatments impair memory not because of their putative "major" action but because of an effect on neuroendocrine components of a stress response. Most amnestic treatments are in fact physiological stressors having the potential of causing excessive release of stress-related hormones and neurotransmitters (*9*). It is therefore possible that many amnestic treatments may act on memory either by producing an extensive neuroendocrine response to the combined stress of training and treatment or perhaps in some cases (as with direct subseizure electrical stimulation) by more directly influencing neuroendocrine activity after training.

Because PBZ attenuated the amnesia produced by epinephrine injections, we examined the possibility that it might attenuate amnesia produced by other treatments. Such findings would be consistent with the hypothesis that retrograde amnesia produced by many treatments can be mediated by a single mechanism. The results indicate that amnesia produced by (i) supraseizure electrical stimulation of the frontal cortex or injections of the convulsant agent pentylenetetrazol, (ii) subseizure electrical stimulation of the amygdala, (iii) injections of diethyldithiocarbamate (a dopamine-β-hydroxylase inhibitor that blocks norepinephrine synthesis), or (iv) injections of cycloheximide (a protein synthesis inhibitor) is blocked by injections of PBZ given before training.

Male Swiss-Webster mice (90 to 120 days old) were used in the cycloheximide experiment because the drug is toxic to rats at relatively low doses; male Sprague-Dawley rats (70 to 90 days old) were used in all other experiments. Mice and rats were trained in one-trial inhibitory (passive) avoidance tasks (*10*). The apparatus used for mice was a trough-shaped alley divided by a hurdle (2.5 cm high) into a well-lit start compartment and a dimly lit shock compartment. The walls and floors of the apparatus consisted of stainless steel plates through which the footshock (0.6 mA, 0.4 second) could be delivered. The training apparatus for rats was similar. In this case, rats were placed in a well-lit white start compartment separated from a black shock compartment by a sliding door. After the door was opened, rats crossed into the black compartment where they received a footshock (3 mA, 2 seconds) through a grid floor. Animals received intraperitoneal injections of either saline or PBZ (2 mg per kilogram of body weight, 0.2 ml per 100 g of body weight) 30 minutes before training (*11*). Immediately after training, each animal in a drug-treatment group received an intraperitoneal injection of saline, diethyldithiocarbamate (680 mg/kg, 0.1 ml per 100 g), cycloheximide (160 mg/kg, 0.5 ml per 100 g), or pentylenetetrazol (45 mg/kg, 0.2 ml per 100 g). In the brain-stimulation experiments, animals were removed from the training apparatus and placed in a stimulation-recording chamber during a 5-second interval between footshock and stimulation. The animals received frontal cortex stimulation (5-mA, 1-second, 60-Hz sine wave), bilateral amygdala stimulation (30 μA per side, 10-second, 100-Hz, 0.1-msec monophasic pulses), or sham stimulation (*12*). Retention tests were administered 24 hours after training. The latency to enter the shock compartment was used as the measure of retention (180-second cutoff latency for rats, 300 seconds for mice). Short latencies were interpreted as reflecting amnesia under these conditions.

The results of these experiments are shown in Table 1. All control groups had median retention latencies equal to the maximum allowed. Groups that received pretraining saline injections followed by a posttraining amnestic treatment had latencies in each case lower than those of the control groups. All groups that received PBZ injections before training and amnestic treatment had long la-

tencies comparable to those of the control groups. In addition, except in the diethyldithiocarbamate experiment, the PBZ-treated animals had latencies significantly longer than those of the corresponding saline-treated groups that received an amnestic treatment.

These results are consistent with our previous findings that epinephrine-produced amnesia is blocked by prior treatment with PBZ. In addition, the results indicate that PBZ attenuation of amnesia has considerable generality; it seems clear that PBZ can block the amnesia produced by most classes of amnestic agents. The treatments chosen differ substantially in the mechanisms generally presumed to mediate the effects on memory. However, because a single drug can block the amnestic effectiveness of these various treatments, the results suggest that many, if not all, amnestic treatments may act through a common neurobiological mechanism. We therefore caution against interpreting the effect of a particular treatment on memory in terms of one biological effect of that treatment.

The mechanism by which PBZ blocks the development of amnesia is not yet clear. Because PBZ readily penetrates the blood-brain barrier and is distributed throughout the central nervous system (*13*), our findings do not address the question of whether or not the adrenergic antagonist acts centrally or peripherally to block amnesia. This question has some relevance here because our previous examinations of PBZ attenuation of epinephrine-produced amnesia indicate that a rapid but transient decrease in brain norepinephrine and adrenal epinephrine after training may be related to amnesia. When we examined the effects on biogenic amines of PBZ injected before training and epinephrine injection, we found that the absence of amnesia was correlated with an attenuated decrease in both brain norepinephrine and adrenal epinephrine concentrations (*8*). Therefore, the release of one or both of these amines may be responsible for the development of amnesia. Our present findings indicate that it may be useful to examine posttraining changes in amine concentrations (as well as other physiological responses to stress) after a variety of amnestic treatments to determine the generality of the relationship between changes in these systems and impaired memory formation. Whatever the specific mechanism underlying PBZ attenuation of amnesia, it may be possible to view many amnestic treatments in terms of a common nervous system action. Findings such as these may significantly reduce the number of neurobiological systems that must be considered in order to understand the mechanisms responsible for retrograde amnesia.

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11. The dose of PBZ was chosen on the basis of preliminary findings that this dose has no effect itself on retention performance in animals trained with either low or high footshock (*8*). At higher doses (for example, 8 mg/kg), the drug impairs later retention performance. Of particular relevance to the findings reported here, we have not observed enhancing effects on retention of PBZ-treated animals under low footshock training conditions. This finding is important because it indicates that the drug's attenuation of amnesia is not simply the result of better acquisition during the training trial, an effect that would not be observed in the present study because the control groups' retention latencies are at or near the maximum allowed.
12. The electrode leads were isolated during the footshock. Electrographic recordings were made after brain stimulation. No differences in brain seizure patterns in the groups receiving stimulation of the frontal cortex were observed in PBZ-treated animals compared with saline-injected rats. Stimulating the amygdala did not produce seizures or other electrographic abnormalities in animals that received either saline or PBZ.
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The Compleat Angler: Aggressive Mimicry in an Antennariid Anglerfish

Abstract. *A case of aggressive mimicry is described in which an anglerfish of the genus* Antennarius *(order Lophiiformes) utilizes a lure that mimics a small fish. The lure provides not only a highly attractive visual cue but presumably also a low-frequency pressure stimulus for potential prey with a minimum of energy expenditure.*

I have an artificial minnow, . . .' [the body made of] cloth, . . . the belly, shadowed as perfectly as you can imagine, . . . the tail and fins . . . of a quill . . ., the eyes of two little black beads, and the head so shadowed, and all of it so curiously wrought, and so exactly dissembled that it would beguile any sharp-sighted trout in a swift stream.—The Compleat Angler, IZAAK WALTON, 1654

Luring as a mode of energy capture appears to be widespread in both the plant and animal kingdoms. From spiders to whales (*1-3*), numerous organisms conserve energy by using a feeding strategy of remaining motionless and offering enticement to would-be predators. Some animals lure with specialized structures of attraction that mimic food items or provide false sexual cues, while others attract prey in more passive ways by offering what is mistaken by smaller organisms as suitable shelter or feeding substrate (*4*). Fishes of the teleost order Lophiiformes are the best known anglers, and have evolved one of the most complex and efficient luring mechanisms (*5, 6*). Nearly all of the more than 200 species of the order (*7*) are equipped with a modified first-dorsal fin spine placed on the tip of the snout, consisting of a sup-

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