human processing is not essential for these particular tasks, and they suggest that alternative explanations merit further consideration. The fact that the chinchillas respond to the synthetic speech as though an abrupt qualitative change occurs in the short voicing-lag region of the VOT continuum at precisely the place where many languages separate two phonemic categories lends support to the idea that speech-sound oppositions were selected to be highly distinctive to the auditory system. By this reasoning, one might infer that there is at least one other natural psychophysical boundary, located in the voicing-lead region of the VOT continuum, which serves as a basis for the phonemic distinction between prevoiced and voiceless-unaspirated plosives of languages such as Spanish, Thai, and Kikuyu. In any case, further experiments with animals should help to pinpoint which speech-perception tasks require "special processing."

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14. Beginning with training on individual vowels, all positive and negative stimuli for a new condition were added simultaneously.
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Ventricular Obstruction: Effect on Drinking Induced by Intracranial Injection of Angiotensin

Abstract. *Lesions of the subfornical organ (SFO) severely attenuated drinking induced by injections of angiotensin II into the lateral ventricles, but a few days (4 to 14) later a recovery of the drinking response is observed. A possible explanation for this is that other dipsogenic sites are involved which are beyond the interventricular foramen and that SFO lesions produce an obstruction by edema or debris at the foramen which blocks access of cerebrospinal fluid–borne angiotensin to those sites. This hypothesis is supported by tracer studies and by direct injection into the third ventricle of SFO-lesioned animals. Other studies reported implicate the anteroventral third ventricle as a likely site for angiotensin receptors.*

Angiotensin has been shown to induce drinking when administered systemically or intracranially (*1*), but its site, or sites, and the mechanism of action remain in question. Early studies of intracranial mapping implicated a diffuse number of tissue sites (*2*). However, a more recent study indicates such data need reevaluation, since mapping with low doses of angiotensin revealed that the critical aspect of the intracranial injection was that the peptide must gain access to the brain ventricular system (*3*). Other evidence supporting ventricular involvement includes the finding that the ventricles and periventricular tissue, but not other brain regions, show a rapid accumulation of radioactivity after systemic injection of labeled angiotensin (*4*). These results suggest that peripheral angiotensin gains access to periventricular sites which are important central nervous structures for the dipsogenic response to angiotensin.

Recently Simpson and Routtenberg (*5*) have suggested that, in the rat, the subfornical organ (SFO) contains the exclusive receptors for angiotensin-induced drinking. The SFO is one of several highly vascularized structures which lies outside the blood-brain barrier, and it protrudes into the anterior dorsal third ventricle adjacent to the interventricular foramen (*6*). Simpson and Routtenberg observed a cessation of drinking in response to angiotensin injections in the basal telencephalon after lesions of the SFO. In two different laboratories, one at the University of Iowa and one at the University of Pittsburgh, we have independently investigated the role of the SFO in the mediation of drinking behavior (*7*); our data do not support the conclusion (*5*) that SFO is the exclusive receptor site for the dipsogenic action of angiotensin.

Both of our groups found independently that SFO lesions blocked drinking to lateral ventricle (LV) injections of angiotensin II on the first test after the lesion, but with subsequent tests (4 to 14 days later), a recovery of elicited drinking behavior was regularly observed. Adult male rats were implanted with 23-gauge guide cannulae to ensure delivery of angiotensin to the LV (*3*) and were screened to establish the drinking response to the hormone after injection through a 30-gauge inner cannula. Then lesions aimed at SFO were made and animals were again tested for response to angiotensin in two tests after the lesion (*8*). In the Pittsburgh experiments both water and a 1.8 percent solution of NaCl were available (*9*), and tests were carried out during the dark phase of the 24-hour cycle. In the Iowa experiments, only a bottle of water was available and testing was done during the light phase.

Of 55 rats that passed the screening test and were lesioned, 22 showed severe deficits in drinking in response to angiotensin on the first test after the lesion (see Table 1; Pittsburgh study, $N = 12$; Iowa study, $N = 10$). However, on the second test after the lesion animals showed a significant recovery of drinking behavior in response to angiotensin (Pittsburgh study, $P < .05$; Iowa study, $P < .01$). Most of the animals with drinking deficits on the first test after the lesion sustained extensive SFO damage (19 of 22 had 70 to 100 percent destruction). It is not likely that recovery or enhancement of function of residual SFO tissue could explain recovery of the drinking response to angiotensin since recovery was noted in animals with complete as well as partial lesions. Furthermore, each laboratory identified one or more animals (a total of four) that failed to show any deficit in angiotensin-induced drinking after 70 to 100 percent destruction of SFO (*10*).

The Pittsburgh group initiated research for brain sites from which the dipsogenic response to angiotensin did not vary as a function of SFO lesions. Pilot work in-

dicated that the optic recess of the third ventricle (OR) was particularly sensitive to the dipsogenic action of angiotensin. Therefore, a series of animals were prepared with two intracranial cannulae, one aimed at LV and the other at the OR. Twenty-five animals that showed reliable drinking responses to antiotensin injections in both cannula sites were then subjected to the previously outlined procedure for lesioning (*8*). Three days later they were tested for the drinking response to angiotensin (100 or 200 ng in 1 μl of isotonic saline) at each cannula site. Eight animals evidenced severe deficits in drinking after LV injections of angiotensin. However, despite massive damage to SFO (70 to 100 percent) in these eight rats, no drinking deficit was observed when angiotensin was injected into the OR (see Table 2).

With these results, the hypothesis that SFO contained the exclusive dipsogenic receptors for angiotensin was untenable. The SFO lies at the confluence of the lateral and third ventricles. Considering this unique location of the structure and the recovery data, each group hypothesized that lesions of SFO could block the interventricular foramen, either by edema or lesion-produced debris, thereby retarding passage of sufficient cerebrospinal fluid–borne angiotensin from LV to the rest of the ventricular system (*11*). With this hypothesis, the initial deficit in drinking after LV injections would occur as a result of foramen blockage, while the observed recovery would be correlated with recovery of ventricular flow. The unattenuated response to angiotensin injections into the OR after SFO lesions is also consistent with this hypothesis, since the hormone was introduced into the third ventricle "downstream" from the ventricular obstruction.

To assess this hypothesis, the Iowa group used tracer analysis of cerebrospinal fluid collected from the cisterna magna to determine the effects of SFO lesions on movement of angiotensin through the ventricles. Nine animals were initially screened for the drinking response to angiotensin delivered to the LV. Four days after lesions aimed for the SFO, animals were retested for drinking; they were then anesthetized and 10 ng of angiotensin II (5-L-isoleucine) uniformly labeled [^{14}C]isoleucine (specific activity, 250 mc/mmole; New England Nuclear) were administered to the LV. Four minutes later cerebrospinal fluid was collected from the cisterna magna and 10-μl samples were analyzed for radioactivity with a liquid scintillation counter (*12*).

A complete elimination of drinking was observed in two lesioned rats; recovered radioactivity was only 4.0 count/min ± 3.5 (standard error) compared to 85.8 count/min ± 33.7 for nonlesioned controls (N = 4). In seven lesioned rats with no deficit in drinking, radioactivity averaged 37.7 count/min ± 15.1. Thus an initial deficit in drinking after lesions aimed at the SFO was accompanied by a virtual absence of recovered radioactivity, supporting the hypothesis of the blocked cerebrospinal fluid flow from LV injection sites. The two rats with drinking deficits were retested 14 days after the lesion and recovery of drinking to the prelesion levels was observed; interestingly, this recovery of drinking paralleled recovery of high counts of radioactivity (73.0 count/min ± 3.53), suggesting a reestablishment of the flow of cerebrospinal fluid from LV. Histological analysis of the two rats with drinking deficits revealed one complete SFO lesion and one lesion with minimal SFO damage (< 20 percent).

To reproduce the effect of ventricular obstruction, the Pittsburgh group placed cold cream barriers (*13*) in the third ventricle at the level of the interventricular foramen in animals with implanted cannula in the lateral preoptic area or the LV and OR. These plugs prevented the LV from communicating with the third ventricle and also prevented injections into the OR from reaching the SFO. The integrity of the plugs was confirmed in each case by radioactive or colored dye tracings. After plugging, the drinking response to angiotensin (40 or 100 ng) injected into the preoptic area or the LV was reduced from 8.3 ± 1.2 ml to 1.2 ± 0.4 ml (P < .001) total fluid intake, while drinking following injections into the OR was unchanged (12.3 ± 2.5 ml to 11.7 ± 2.1 ml) in the same 14 animals. The order of the angiotensin drinking tests was counterbalanced and tests were conducted on the same day that the plugs were inserted. Thus the application of an obstructive plug within the ventricles reproduces the results that we believe are due to edema or debris obstruction following SFO lesions.

In summary, our findings support the hypothesis that disruption of ventricular flow by lesion-produced debris or edema can be responsible for deficits, after SFO lesion, in the drinking response to intracranial injections of angiotensin. The reported attenuation of the drinking response to angiotensin after SFO lesions (*5*) can be accounted for on this basis. Furthermore, SFO cannot be the exclusive dipsogenic receptor site for angiotensin since drinking after injections of angiotensin remains even when access to the SFO via cerebrospinal fluid is eliminated or when SFO is totally ablated. Given this conclusion we should consider the location of other sensitive tissue. Data from other studies in our laboratory (*14*) indicate that more posterior injection sites in the fourth ventricle are negative, even at doses of 500 ng, and posterior third ventricle sites are

Table 1. Recovery of drinking following angiotensin injections into the lateral ventricles after subfornical organ lesions. Values are expressed as the mean fluid intakes (in milliliters) ± the standard error of the mean; N is the number of animals.

Fluid	Pittsburgh study (N = 12)		
	Before lesion	First test after lesion (~day 3)	Second test after lesion (~day 8)
Water	9.8 ± 1.4	0.7 ± 0.4	4.1 ± 1.1
NaCl (1.8%)	4.3 ± 1.2	0.4 ± 0.2	0.8 ± 0.6
Total	14.1 ± 1.6	1.1 ± 0.5	4.9 ± 1.5
	Iowa study (N = 10)		
	Before lesion	First test after lesion (day 4)	Second test after lesion (day 14)
Water	8.1 ± 2.2	0.4 ± 0.8	6.2 ± 2.2

Table 2. Effect of SFO lesions on fluid intake following angiotensin injections into the cerebral ventricles of eight rats. Values are expressed as mean fluid intakes (in milliliters) ± the standard error of the mean; NS, not significant.

Fluid	Injections into lateral ventricle			Injections into third ventricle		
	Before lesion	After lesion	P*	Before lesion	After lesion	P*
Water	10.83 ± 1.91	0.93 ± 0.94	.01	9.08 ± 0.94	10.22 ± 2.04	NS
NaCl (1.8%)	5.33 ± 1.72	0.60 ± 0.27	.05	6.53 ± 1.62	2.98 ± 0.96	NS
Total	16.17 ± 1.90	1.53 ± 0.71	.001	14.62 ± 1.61	13.23 ± 1.73	NS

*Probability values based on t-tests comparing intakes before and after lesions.

equivocal with respect to the dipsogenic effect of angiotensin. The lateral ventricles are also without active sites, as shown by the rats with interventricular blockage by lesion debris or cold cream plugs. Since the evidence points to a periventricular site, these data limit the area to the anterior-ventral part of the third ventricle at the preoptic and hypothalamic level. It is in this region that we suggest that further experimentation would be fruitful.

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10. It should be noted Simpson and Routtenberg (5) also reported that in one of seven animals with SFO lesions there was no attenuation of drinking after intracranial injection of angiotensin.
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Light for All Reasons: Versatility in the Behavioral Repertoire of the Flashlight Fish

Abstract. *The flashlight fish,* Photoblepharon, *possesses headlight-like luminous organs situated in the orbit just below the eyes. On the basis of direct field and laboratory studies, it is postulated that the bioluminescence is used by the fish for many different functions: to assist in obtaining prey, to deter or escape predators, and for intraspecific communication. The fish also uses its light to see by.*

A biologically generated light may be used by an organism in different specific ways for several distinct functions (*1*–*3*). In most of the luminous organisms that have been analyzed, the proposed functions of light emission fall in three major categories. The first is assisting predation (offense). For example, certain of the mesopelagic ceratioid angler fish, such as *Melanocetus murrayi* (*4*) and *Oneirodes acanthias* (*5*), have a luminous organ (esca) which presumably functions as a lure (*1*, *2*). Similarly, the firefly "femme fatale" (*Photuris versicolor*), in addition to using her light organ to signal the male of her own species, mimics the courtship signal of other species, attracting males whom she then eats (*6*). The second major function is

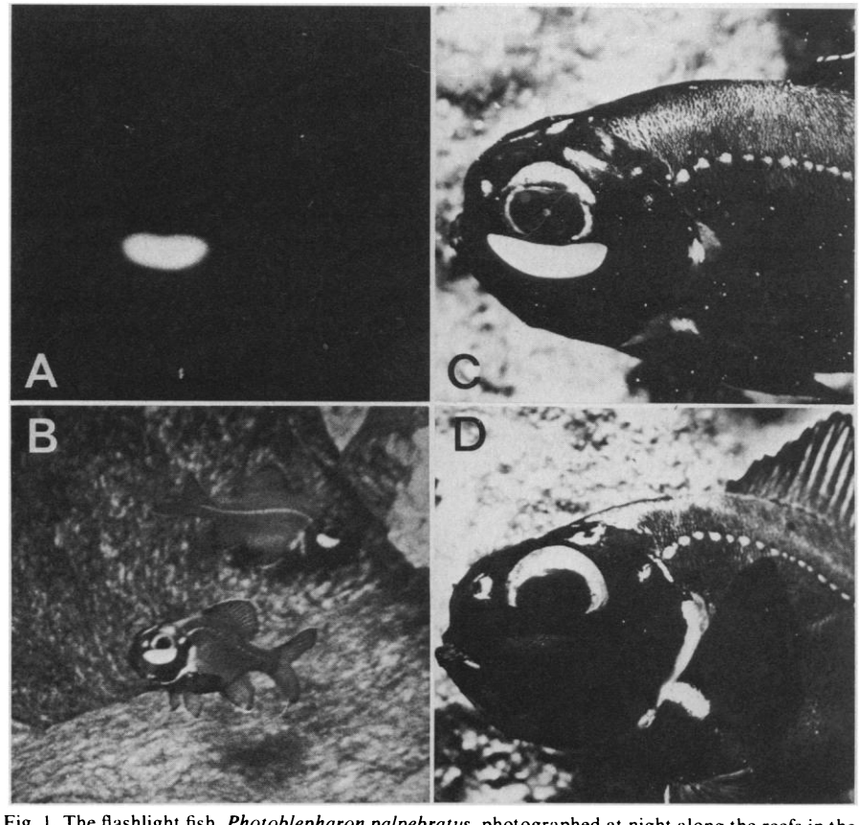

Fig. 1. The flashlight fish, *Photoblepharon palpebratus*, photographed at night along the reefs in the Gulf of Elat, Israel, by the light emitted from its own luminescent organ (A) and with an underwater strobe light (B, C, and D). The reflective areas of the lateral line, the edges of the fin rays, and the operculum are not luminescent. (B) A pair of *Photoblepharon* in their intertidal territory. (C) Closeup of *Photoblepharon* with the lid of the luminescent organ open. (D) Closeup with the lid closed. The fish are about 6.5 cm long.