sponse during the CS+ within a given trial. Heart rate showed a significant deceleration during the first 2 minutes of the CS+ and an acceleration toward the original pre-CS+ level during the 3rd minute. Bar pressing, on the other hand, showed a significant deceleration during all 3 minutes of the CS+ period.

Also, there were more individual differences in the direction of the heart-rate change to the CS+ than in the direction of the bar-pressing change. On acquisition day 6, for example, only five rats showed a heart-rate deceleration during both of the first 2 minutes of the CS+. Bar-pressing rate during the CS+ decelerated for all rats.

Our data on direction of the heart-rate change during the CS+ do not agree with those of Stebbins and Smith (*4*). These authors reported that the heart rate accelerated during the CS+; we found the predominant change to be a deceleration. While such differences in direction of change have been reported before, our knowledge of the variables controlling direction is still too meager to identify the factors which could account for this difference (*7*).

The response to the shock (UCR) was invariably an increase in heart rate; the response to the CS+ was predominantly a decrease. Thus, the direction of the heart-rate CR does not seem to be determined simply by the direction of the UCR. Almost from the first publication of Pavlov's work in English there have been doubts about his conclusion that the CR is simply a copy (a partial copy in some cases) of the UCR (*8*). These doubts are further substantiated by the data of the present experiment.

A significant difference between CS+ and CS— was found on the 1st day of discrimination for both heart rate and suppression. Furthermore, there was no significant generalization to the CS— on its first presentation. Thus, the discrimination occurred too rapidly to produce data that would have permitted a comparison of the development of the discrimination for heart rate and suppression. These data do, however, provide a control for the effects of unpaired presentations of shock and CS; if our results were produced by pseudoconditioning or sensitization, discrimination should not have taken place.

Our results support the view that conditioned responses can be relatively independent in the way they are affected by the same classical conditioning procedure. In fact, it would seem that the effects of a classical conditioning procedure are more easily seen in the CS's modulation of ongoing operant behavior than in its modulation of autonomic responses such as heart rate. This conclusion can, of course, be criticized. One might be tempted to suggest that heart-rate changes were simply artifacts of changes in bar-pressing rate. That is, that the heart decelerated when bar pressing decreased during the CS+ and accelerated, or decelerated less, if bar pressing continued. This interpretation does not seem likely since the conditioned heart-rate response appeared only toward the end of the experiment—long after suppression had reached asymptote. If this heart-rate response were a simple function of bar-pressing rate, the relationship should have become apparent as soon as the bar-pressing rate during the CS had stabilized.

Another possibility is that the rapid conditioning of suppression was a confounding effect of operant reinforcement. One could argue that heart rate was controlled only by classical conditioning, whereas suppression of bar pressing was controlled not only by classical conditioning but also by the operant punishment of bar pressing. This argument, of course, holds only if one assumes that bar pressing can be punished by shock onset while heart rate cannot. While punishment of bar pressing may have affected the course of suppression, it does not seem likely that it can account for all of the present results since suppression is known to occur rapidly even when conditioning trials are given in a different apparatus with no bar present (*9*).

It would seem then that the conditioning of the two responses did proceed relatively independently and, further, that the conditioning of suppression was more rapid and stable than the conditioning of heart rate. If this conclusion is correct, then the results of this experiment pose a problem for research in which a single CR is employed as an index of some internal state (conditioned fear, anticipatory goal response, association, or expectation). For example, suppression of bar pressing alone (*10*) or heart rate alone (*11*) have been employed as indexes of fear during avoidance conditioning. But, since the conditioning procedure affects these CR's in different ways, one's description of the development of fear during avoidance conditioning would differ considerably depending on which CR was employed.

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31 March 1966

Control of Sensory Fields by Stimulation of Hypothalamus

Abstract. *Stimulation of the cat's hypothalamus, which elicits attack, also establishes sensory fields for two reflexes related to biting. Touching a perioral region leads to head movement, bringing the stimulus to the mouth. Touching the lip-line leads to jaw opening. The size of the fields depends on the intensity of stimulation.*

This report deals with the control of jaw opening, one of the final events in a sequence of responses comprising attack behavior in the cat. Evidence obtained earlier in this laboratory clearly indicated that section of the infraorbital maxillary and infraalveolar branches (both sensory) of the trigeminal nerve precludes jaw opening in attack evoked by electric stimulation of the hypothalamus. The failure of biting to develop is not due to motor impediment (*1*).

We can now attribute this effect to an unusual sort of sensory loss. In brief, in the normal cat, stimulation of areas of the hypothalamus associated with attack causes the appearance of sensory fields for head-orienting and jaw-opening responses. The extent of both of these sensory fields is a function of the intensity of stimulation. The sensory denervation mentioned above involved those areas of the face in which these fields would normally appear.

Observations were made on 21 cats. Each was fitted with several electrode guides mounted stereotaxically on the skull over the hypothalamus. After the cats had recovered from surgery, sterile calibrated electrodes were advanced in small steps through the guides into the brain tisse of each waking animal. Stimulation occurred at each step, and the animal's behavior was noted. Most of the tissue along the electrode track could be explored in a short time. When relevant responses appeared, the exploring electrodes were cemented in place. One-millisecond biphasic pulses repeated at a frequency of 62.5 per second were used as stimuli.

Stimulation of the regions of the brain under study ordinarily produces savage attack. This required restraint of the animals in a loose-fitting canvas sack which was of no apparent discomfort to them. Their heads were free to move when we studied head-orienting responses. Measurement of the sensory fields for jaw-opening required complete immobilization of the head, a condition achieved with a head holder whose suspending elements had been attached to the skull during surgery. The cats were quite tolerant of this device: they purred, ate, and even slept while immobilized. Many objects were used for tactile stimulation of the face, but when we measured changes in the extent of the jaw-opening sensory field we used a narrow, pointed stick, and measurements were read from a millimeter rule moulded to the contours of the cat's lip-line.

In the normal adult cat, light tactile stimulation of the muzzle (Fig. 1, top) produces no observable activity beyond a slight tendency, in some cats, to close the lips. A striking change occurs in the waking cat during stimulation of the attack areas of the hypothalamus. Stimulation of the tissue above these areas in the electrode track was never observed to produce the effects now described.

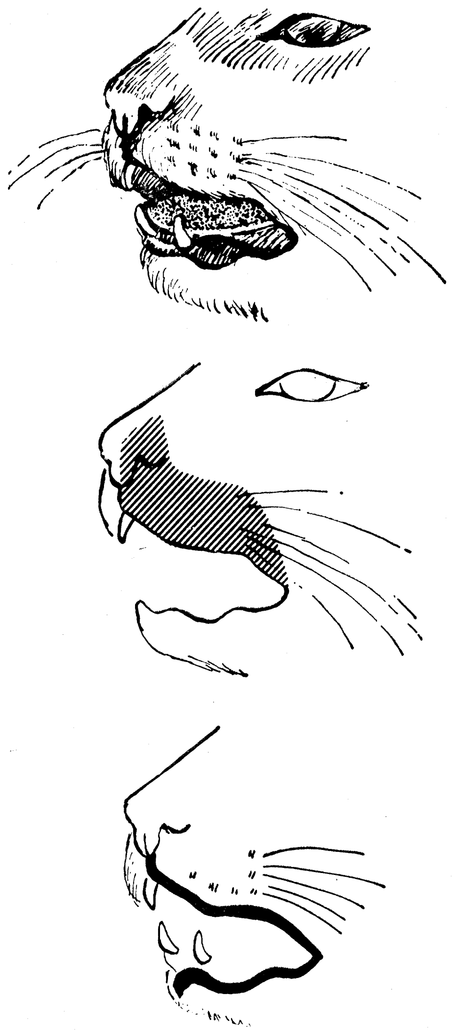

Fig. 1. (Top) The cat's muzzle. (Middle) Maximum extent of the maxillary sensory field for head-orienting responses during relatively intense stimulation. A similar mandibular field has not yet been mapped in detail. (Bottom) Maximum extent of the sensory field for the jaw-opening response during relatively intense stimulation.

With hypothalamic activation a sensory field for a rapid jaw-opening response appears in an area of the muzzle strictly limited to the lip-line (Fig. 1, bottom). Light tactile stimulation in this region of the face, and in no other, produces spring-like depression of the mandible. Not only is the appearance of this "trigger zone" dependent on hypothalamic activity, but the extent of the sensory field from which jaw-opening may be triggered is a function of the intensity of central stimulation (Fig. 2).

Just suprathreshold stimulation provides for jaw-opening to light touch only in a region of the lip close to the midline. Increasing hypothalamic stimulation is accompanied by lateral extension of the trigger zone along the lip-line until, at high levels of stimulation, the corner of the mouth is included in the field. The manner of extension is similar in both upper and lower lip-lines.

Light touch in the zone depicted in Fig. 1 (middle) does not trigger jaw-opening during hypothalamic stimulation but does prompt immediate head-orienting responses. A touch at any point in the effective sensory field initiates rapid movement of the head in such a way as to bring the midline of the mouth to the stimulating object. Midline stimulation of this zone produces a powerful forward thrust of the head. Movement continues only so long as the probe maintains contact.

As in the case of jaw-opening, the effective field size for the head-orienting response is a function of the intensity of central stimulation. The mapped zone illustrated in Fig. 1 (middle) represents the maximum extent of the sensory field for this response in the maxillary region. It is clear that the ophthalmic branch of the trigeminal nerve does not contribute to the field.

Although the maxillary field coincides roughly with the area occupied by the vibrissae, these structures are not particularly effective in triggering the response. The hairless portion of the external nares is more sensitive in this respect than the vibrissae.

One notable feature of the sensory fields is their distribution largely to the side of the muzzle contralateral to the side of central stimulation. High levels of stimulation activate sensory fields ipsilaterally whose extent lags behind the contralateral fields by a wide margin.

The results of this study could be viewed as an example of behavioral control consonant with the general concept of selective perception, a set of ideas important in explanations of instinctive and attentive behavior. Discussions relating to selective perception have often centered on exclusion of sensory events from effective entry to the central nervous system. Sensory filtering, operating through passive receptor or central nervous system characteris-

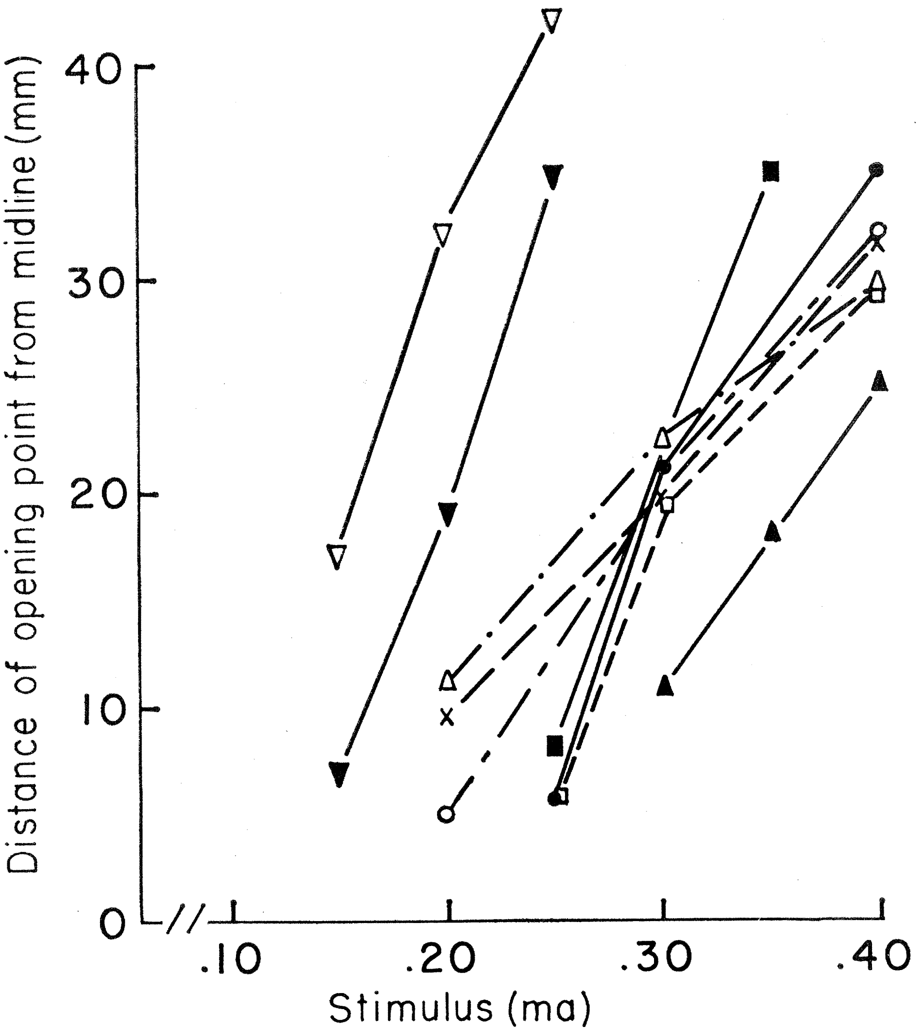

Fig. 2. Extent of sensory fields for jaw-opening, as determined by intensity of stimulation. The data are from eight different cats, and all but one (the solid circles) of the curves represent the maxillary lip-line field. Each point is the mean of ten trials.

tics, is thought to limit the availability of a particular response to narrow bands of stimulus spectra (*2*). Entire sensory modes have been interpreted as being damped by active mechanisms in achieving a focus of perception (*3*).

The data presented here suggest a rather different manner of achieving what is, in effect, selective perception. During central stimulation a certain response (for example, jaw-opening) becomes available through peripheral stimulation of a limited sensory field which, prior to central stimulation, had been inoperative. This is an active mechanism and does not proceed through sensory exclusion. The field is unspecific with respect to the relevant sensory mode: any object making contact with the effective sensory field triggers the response. The sensory field itself is limited; but, within the bounds illustrated in Fig. 1, the greater the hypothalamic activity, the more inclusive the effective sensory field. The specific events described in this paper constitute a highly adaptive mechanism for an aroused, attacking animal.

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Pesticide Residues in Total Diet Samples: Bromine Content

Duggan, Barry, and Johnson provide interesting data on pesticide residues in total diet samples in food ready for consumption [*Science* **151**, 101 (1966)]. Most of the analytical procedures are specific for the pesticide or group of pesticides; but in the case of bromine and arsenic the analytical procedures determine the total amount of the element present, and the figure so obtained is not necessarily related to a pesticide containing one of these elements. It is true that the high concentrations of bromide ion in grain can often be associated with use of methyl bromide or ethylene bromide fumigants, but this association is probably not true of all the other diet components named by the authors.

Bromides occur naturally in sea water and soils; some soils contain appreciable amounts—for example, the tidewater area of Virginia contains 10 to 20 parts per million. It is not surprising that some of this bromine is taken up by plants or that natural bromine, usually in the range 0.5 to 10 ppm, is found in the edible portion of the plants. Part of the bromine is conveyed in the food chain to animals, and up to 8 ppm can occur in milk [G. E. Lynn, S. A. Schrader, O. H. Hammer, C. A. Lassiter, *J. Agr. Food Chem.* **11**, 87 (1963)].

Interest in the herbicide bromoxynil, 3,5-dibromo-4-hydroxybenzonitrile, containing over 50 percent bromine, and in its octanoyl ester, has led me to study the bromine levels in grain from treated plants. Control samples, which have received no treatment with a pesticide containing bromine, have a natural bromine content that varies with the location of the site but has ranged from 0.5 to 25 ppm. Clearly it would be incorrect to relate this natural bromine content to bromoxynil, or, for that matter, to any other pesticide containing bromine. I therefore suggest that, when Duggan *et al.* list bromide-ion levels as derived from pesticides, this classification could have unwarranted implications.

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