predator. He shows that the greater the number and novelty of stimuli, and the less expected and more variable their sequence, the greater is the conflict induced. He also demonstrates that the amount of conflict is directly related to increase in reaction time.

Protean displays involve all the above factors, so, besides the possibility that a predator may be confused into making an incorrectly directed attack, it will in any case suffer a delay in its reaction time. Any such delay is bound to be of survival value to the prey. Therefore, unlike cryptic, mimetic, or warning displays, the protean display does not systematically deny or falsify information; instead it creates confusion by simultaneously arousing conflicting responses. Furthermore, to the extent to which it is unsystematic, it is resistant to defeat by learned modification of responses in the predator.

There is thus both theoretical (*15*) and observational (*8*) evidence that erratic movements during antipredator displays have survival value. In terms of evolutionary theory, therefore, their unpredictability is not accidental but has appeared as a result of natural selection during phylogeny. Protean display is an entirely distinct principle in antipredator behavior—a principle that awaits more detailed and exact analysis.

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Brain Catecholamines: Relation to Defense Reaction Evoked by Acute Brainstem Transection in Cat

Abstract. *The concentration of noradrenaline, but not serotonin, in the brainstem of cats is reduced 3 hours after the production of a defense reaction by supracollicular decerebration, even when movements and changes in blood pressure are abolished by transection of the spinal cord. After midcollicular decerebration, which does not elicit a defense reaction, noradrenaline concentrations do not change. The decrease in its concentration accompanying the defense reaction produced by brain lesions probably reflects activity, in this behavior, of neurons containing noradrenaline.*

When the defense reaction (or sham rage) is elicited by electrical stimulation of the amygdala or hypothalamus in the cat, it is accompanied by a decrease in the concentration of noradrenaline (NA) in the brain and of NA and adrenaline (A) in the adrenals, without change in the amounts of dopamine and serotonin (5HT) in the brain (*1*). Furthermore, with fluorescence histochemistry it has been demonstrated that the decrease in the NA in the brain is the result of depletion of NA located within axon terminals of neurons containing NA (NA neurons) (*2*). Since concentrations of NA do not change when electrical stimulation fails to elicit the defense reaction, we previously suggested a relationship between activity in these NA neurons and this behavior (*1*).

To determine if the defense reaction elicited without electrical stimulation is accompanied by a decreased concentration of NA in brain, we produced recurrent spontaneous outbursts of rage in cats by decerebrating them above the colliculi and preserving the posterior hypothalamus (high decerebration). Concentrations of NA and 5HT in the medulla, pons, and the lower part of the mesencephalon and of NA and A in adrenals have been compared with those in identical regions of the lower brainstem and in the adrenals of cats decerebrated by transection between superior and inferior colliculi (low decerebration). These cats do not show spontaneous rage.

Mature cats of both sexes were anesthetized with ether, cannulas were placed in one femoral artery and in the trachea, the common carotid arteries were bilaterally ligated, and the cat was then placed in a stereotaxic frame. Decerebration was performed with a spatula and completed by suction. Blood pressure measured by a Statham pressure transducer, heart rate measured by a cardiotachometer triggered by the arterial pulse, expired CO_2 measured by an infrared CO_2 meter, and respiration and chest movements measured by a pneumograph were amplified and displayed on channels of an Offner-Beckman polygraph. Body temperature was maintained at 37°C by an infrared lamp thermostatically controlled by the rectal temperature. After the transection of the brainstem was completed, administration of the anesthetic was discontinued. The animals were decapitated 3 hours later, the brains were rapidly removed, the levels of transection were noted, and a piece of lower brainstem extending from 1 cm be-

Table 1. Changes in mean concentration (± standard error) of catecholamines in cat brainstem and adrenal glands and of serotonin in brainstem 3 hours after decerebration at midcollicular (low decerebrate) or supracollicular (high decerebrate) levels. Figures in parentheses are the numbers of observations. Abbreviations: NA, noradrenaline; 5HT, serotonin; A, adrenaline. The differences between the nonoperated and ether controls and between the nonoperated and low decerebrate cats are not significant.

Group	Brainstem (ng/g)		Adrenal gland (ng/g)	
	NA	5HT	NA	A
	Control animals			
Nonoperated	227 ± 13 (10)	516 ± 47 (4)	512 ± 32 (10)	484 ± 30 (9)
Ether	216 ± 6.8 (5)	512 ± 28 (2)	505 ± 38 (5)	473 ± 65 (5)
	Operated animals			
Low decerebrate (no defense reaction)	200 ± 9.5 (5)	648 ± 45 (6)	542 ± 45 (7)	459 ± 55 (7)
High decerebrate (defense reaction)	149 ± 10† (7)	531 ± 39 (4)	325 ± 46* (8)	258 ± 50* (8)

* Difference from nonoperated controls significant, $P < .01$. † Difference from nonoperated controls significant, $P < .001$.

low the obex to a plane between superior and inferior colliculi dorsally and 1 mm rostral to the pons ventrally was dissected out. The cerebellum was discarded, and the section of the lower brainstem was weighed and then frozen on dry ice. Preparation of this block required removal of the hypothalamus in high decerebrate animals, but the block usually corresponded in its rostral extent to the plane of section in low decerebrate animals. Both adrenal glands were removed for assay. The concentrations of NA and 5HT in brain and of NA and A in adrenals were determined by methods previously described (*3*). For controls, the concentrations of NA and 5HT in the lower brainstem and of A and NA in the adrenals were measured in animals decapitated after being anesthetized with chloroform (Table 1, nonoperated controls) and in animals anesthetized with ether for 45 minutes (the usual time required for preparation of experimental animals) and then decapitated 3 hours later under chloroform anesthesia (Table 1, ether controls).

Within 15 minutes after the administration of the anesthetic was stopped, rudiments of the defense reaction appeared spontaneously in high decerebrate cats, and by 90 minutes recurrent spontaneous outbursts of rage were well established; these continued until the animals were killed. Typical attacks consisted of tail lashing, alternating limb movements, extension of claws, snarling, pilo-erection, elevation of blood pressure and heart rate, tachypnea, pupillary dilatation, retraction of nictitating membranes, and centering of eyes. The relative intensity of each component, however, varied from animal to animal. Each attack lasted from 5 to 20 seconds, but occasionally several attacks would blend. In some animals, recurrent attacks occurred with a frequency of six per minute maintained over several hours (Fig. 1). Attacks were easily provoked by any cutaneous contact, movement of the animal, or, even, sudden noise. In contrast, low decerebrate animals remained immobile during the 3-hour test period without notable alteration in blood pressure or pulse or the appearance of spontaneous movements. In these animals, tail lashing, alternating limb movements, elevation of blood pressure and heart rate, and tachypnea could be induced by deep pressure on limb muscles or by other noxious stimuli. The intensity and duration of attacks so provoked, however, were less than those seen in the high decerebrate preparations.

Table 2. Changes in mean concentration (± standard error) of catecholamines in cat brainstem and adrenal gland and of serotonin in brainstem 3 hours after spinal cord transection and mid-collicular (low) or supracollicular (high) decerebration. Figures in parentheses are the numbers of observations. Abbreviations are the same as in Table 1. The difference between the nonoperated and low decerebrate group is not significant. The differences in 5HT and adrenal NA of high decerebrate and nonoperated controls are not significant.

Group	Brainstem (ng/g)		Adrenal gland (ng/g)	
	NA	5HT	NA	A
Low decerebrate spinal	206 ± 11 (6)	532 ± 32 (3)	616 ± 50 (8)	418 ± 47 (9)
High decerebrate spinal	159 ± 13* (6)	544 ± 29 (4)	514 ± 33 (8)	363 ± 21* (8)

* Difference from nonoperated controls significant, $P < .001$.

The ether controls were not significantly different from the nonoperated controls, and no changes occurred in animals without appearance of the defense reaction (low decerebrate) (Table 1). There was a significant decrease in the concentration of NA in the brainstem along with a decrease in the concentrations of NA and A in adrenals of animals with high brainstem transection, all of which demonstrated recurrent attacks of the defense reaction. In these animals concentrations of 5HT in the brainstem did not change.

To evaluate whether the struggling movements or recurrent changes in blood pressure and respiration contributed to the decline in the concentration of brain NA in the defense reaction, in another group of cats we first transected the spinal cord at the first cervical segment and then proceeded to decerebrate them as described. The cats were maintained with artificial ventilation with expired CO_2 maintained at 2 to 3 percent, decerebration was immediately performed, and the animals were killed 3 hours later.

In high decerebrate spinal cats, all the movements of limbs and trunk and periodic changes of blood pressure and respiration characteristic of the defense reaction were absent. Within an hour after supracollicular decerebration, however, periodic attacks of blinking, swallowing, conjugate eye movements, baring of the teeth, and occasionally slowing of the heart for one or two beats would occur every 1 to 5 minutes. We could provoke these responses by pulling the cat's whiskers, pressing on its snout or eyeballs, or opening its mouth, maneuvers which will elicit an outburst of rage in the nonspinal high decerebrate cat, and thus they were interpreted as abbreviated cranial manifestations of a defense response. In low decerebrate spinal cats such move-

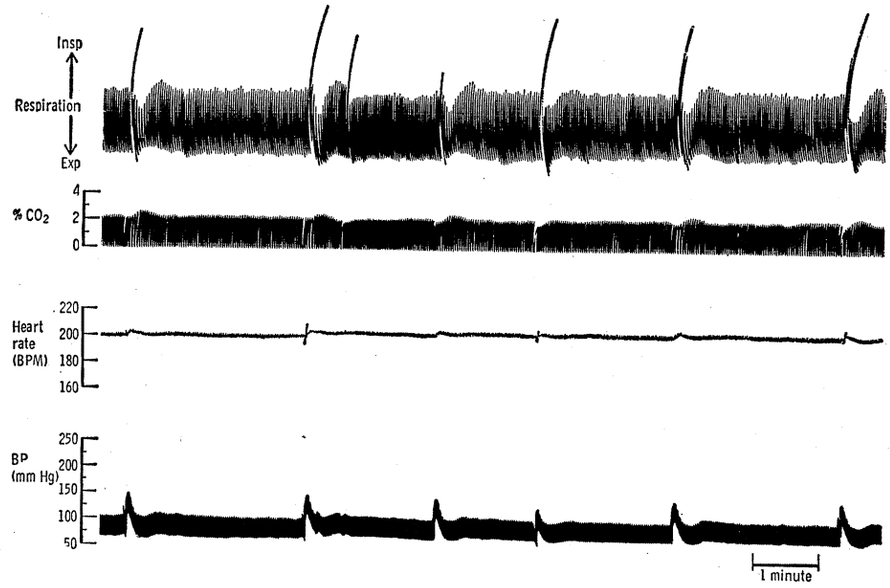

Fig. 1. Recurrent spontaneous outbursts of the defense reaction 90 minutes after acute brainstem transection in cat. Top channel, pneumograph; second channel, expired CO_2; third channel, heart rate; bottom channel, blood pressure. Outbursts of the defense reaction occur with each elevation of blood pressure.

ments did not occur spontaneously nor could they be evoked by manipulation.

Abolition of movement or changes in blood pressure resulting from section of the spinal cord does not avert the decrease in the concentration of brain NA occurring with high decerebration (Table 2). On the other hand, the absence of any change in its concentration in low decerebrate spinal cats or in that of brainstem 5HT in either group indicates that spinal section does not by itself produce a change in the amounts of these amines in the lower brainstem within 3 hours. Spinal section prevents the depletion of adrenal NA but, surprisingly, not that of A occurring with high decerebration. The decrease in the concentration of adrenal A in high decerebrate spinal cats is unexplained but may only be a consequence of the small sample size, since the difference between the amounts in low and high decerebrate spinal cats is not significant ($P > .05$).

The defense reaction, when elicited by a lesion of the brainstem, results in a reduction in the amount of brainstem NA unassociated with any change in the amounts of 5HT; it also causes a decrease in the amount of adrenal NA and A. Such a pattern is similar to that produced by electrical stimulation of the amygdala and hypoththalamus, which evokes the defense reaction (*1*). That the decline in brainstem NA is not the result of depletion of NA reserves secondary to transection of tracts is supported by the fact that the depletion of NA in animals with permanent sections of central tracts is not seen for at least 2 days (*4*), and also by the finding that the amount of NA in the brainstems of animals with low decerebration does not decline. Nor is it the result of the principal somatic and autonomic concomitants of the defense reaction since the concentration of NA in the brainstem falls to the same degree in high decerebrate cats as it does when expression of the defense response is abolished by transection of the spinal cord.

When peripheral sympathetic neurons are electrically stimulated, depletion of NA in sympathetic terminals is the result of a disproportion between the release and synthesis of NA (*5*). That depletion of NA within neurons in the brain is to some extent proportional to the degree of neural activity has also been demonstrated (*6*). Thus, it seems likely that the decline in brainstem NA seen when the defense reaction is elicited by high decerebration or by electrical stimulation of the hypothalamus or amygdala (*1*) is the result of augmented activity in NA neurons. The absence of changes in the amounts of 5HT during rage suggests that the activity of neurons containing 5HT is of less importance in this behavior than is that of the neurons containing NA. On the other hand, neurons containing 5HT may be able to maintain a synthesis great enough to compensate for increased activity. It is also likely that the depletion of adrenal NA and A in the defense reaction results from the release being in excess of synthesis, during the increased sympathetic discharge to the adrenal.

Our study supports the premise that in the brain neurons containing NA are active in the expression of the defense reaction in the cat. That depletion of NA is not unique to this behavior should be emphasized, for depletion of brain NA associated with preservation or even elevation of 5HT concentrations may occur in drug-induced excitement or physical and emotional stress (*7*). It is likely that neurons containing NA are a component of the loosely defined arousal mechanisms of the brainstem which are maximally engaged in any form of excited behavior, including the defense reaction.

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Hampea and the Boll Weevil: A Correction

In reporting the occurrence of the cotton boll weevil (*Anthonomus grandis* Boh.) on plants of the genus *Hampea* in Veracruz, Mexico, I stated that the host plants were "tentatively determined to be *H. integerrima* Schlecht." and added that "the plants differ clearly in floral characters from *H. rovirosae* Standl." (*1*). These statements require correction.

I recently examined a wide range of material of *Hampea*, including the relevant types. It became clear that the specimens (*2*) cited in the earlier paper represent *Hampea rovirosae* Standl. This observation constitutes a significant northward extension of the range of this species.

The basis for the earlier misconception lies in the incorrect measurement of the calyx that Standley reported in his description of *H. rovirosae*, and in the emphasis that he gave to this feature in his key to the genus (*3*). Standley stated that the calyx of *H. rovirosae* is 10 mm, whereas I measured calyces of the several buds on the holotype (*4*) and obtained a mode of 7 mm. In only one bud, in which the calyx is torn, was a measurement of 10 mm obtained. In the specimens cited (*2*), calyces range from 4 mm to 7 mm with a mode of 5 mm. Flowers of staminate and pistillate plants do not differ in this respect. Comparisons with the type leave no doubt that the plants observed to be the host of *Anthonomus grandis* Boh. are *Hampea rovirosae* Standl.

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Hexagonal Diamonds in Meteorites

The recent synthesis of hexagonal diamond at high pressure and its subsequent discovery in the Canyon Diablo and Goalpara meteorites [R. E. Hanneman, H. M. Strong, F. P. Bundy, *Science*, **155**, 995 (1967)] constitute strik-