the nest and group quietly near their nest entrance, with the head and thorax lowered to the ground, the gaster raised and arched, and the intersegmental membrane between the last two segments dorsally extended (Fig. 1). Males flying out from other nests are attracted by these "calling" females. On drawing close to a female, the male first touches her with his antennae, then grasps the female's thorax with his mandibles. While riding on her back, he extends his copulatory apparatus in search of the female's genitalia. If she is ready to mate, she turns her abdomen slightly to the side, so that the male is able to couple. Then the male releases his mandibular grip on the female's thorax. With the pair in this position the copulation can last several minutes (Fig. 1).

A similar calling behavior has been described in *Harpagoxenus subleavis*, whose calling females release a sex pheromone from the poison gland (*5*). Although calling *Rhytidoponera* frequently have their sting slightly exposed, we were not able to demonstrate that the poison gland or the Dufour's gland produce a sex attractant. Instead, we discovered a large gland which opens dorsally between the last two abdominal tergites (Fig. 2). This organ, which we propose to call the tergal gland, consists of many single glandular cells. Each cell sends a channel into a voluminous reservoir consisting of two paired sacs. The glandular secretion is readily discharged to the outside when the intersegmental membrane between the last two tergites is expanded.

When exposed to the secretions of the tergal gland, *Rhytidoponera* males, which have previously emerged from their nests, respond with agitated locomotion and attraction. When we placed males in an arena (diameter, 60 cm) into which a weak air current (0.5 m/sec) was blown carrying the scent of the secretions of the tergal gland, the males were strongly attracted to the scented air current. They did not respond to a control air current simultaneously blown in through a second opening (Table 1). Several males exposed to tergal gland secretions attempted to mount one another. When a worker was made available, some males tried to mate with it, even though it was not "calling." These results suggest that some *Rhytidoponera metallica* workers produce a sex attractant in the tergal gland, which they discharge during sexual calling. This is the first evidence of sexual chemical communication in the primitive ant subfamily Ponerinae. A first histological survey of 11 ponerine species (*6*) indicates that the new gland is a common structure in Ponerinae. We have also found it in *Myrmecia vindex*, representing a second primitive subfamily, the Myrmeciinae. This fact suggests that the gland is a very primitive phylogenetic trait in ants generally. We suppose, however, that the primary function of the gland is not the secretion of sex pheromones, because we have found it to be present in workers of species that have no ergatoid reproductives.

Table 1. After males of *Rhytidoponera metallica* had left their nests, they were placed in an arena, which was then covered by a red glass plate. Weak air currents (0.5 m/sec) were next blown into the arena, one being first led over moist (H_2O) filter paper (control), the other over the secretion of a single tergal gland (test). A count was made of the number of males being attracted to either one of the air currents within a 5-minute interval. The openings through which the test current and the control current were led were alternated in position in successive replication. For each replication a new population of males was used.

Replication	Number of males tested	Number of males responding to	
		Tergal gland scent	Control
1	18	6	0
2	27	13	2
3	12	8	1
4	21	16	3
5	9	7	0
6	11	6	0
7	26	12	3

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6. Ponerine ants found to possess the tergal gland include species of the genera *Amblyopone, Paraponera, Ectatomma, Odontomachus, Pachycondyla,* and *Platythyrea*. More detailed accounts of the sexual behavior of ponerine ants and the functioning of the tergal gland are being prepared.
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Amygdaloid Projections to Prefrontal and Motor Cortex

Abstract. *Direct projections from the amygdala to the entire frontal cortex were demonstrated in the cat using the retrograde transport of horseradish peroxidase. Injections throughout the prefrontal cortex labeled neurons in the ipsilateral basal magnocellular amygdaloid nucleus; injections in the premotor and motor cortices labeled neurons in the same nucleus plus a few cells in the anterior amygdaloid area.*

The frontal lobe of the brain has, in spite of a clear regional differentiation (*1*), a relevant unitary function in the neurobiological correlates of the highest forms of behavior (*2*). Stimulation of the amygdaloid complex, on the other hand, elicits certain motor and behavioral phenomena closely related to responses obtained by stimulation of the frontal lobe cortex (*3*). Such a relation instigated this search for a direct monosynaptic connection between the amygdala and the frontal cortex.

Studies of neocortical monosynaptic afferent connections are facilitated by using horseradish peroxidase (HRP) as a retrograde tracer [for example, see (*4* and *5*)]. In the present study we used HRP to trace systematically the afferent connections to the whole frontal cortex, including prefrontal area [gyrus proreus, comparable to the frontal granular cortex of primates (*6*)] and motor and premotor areas (gyrus sigmoideus and both banks of the sulcus cruciatus).

The entire frontal lobe was explored in a group of 34 adult cats, each of which received an intracortical injection of 0.3 to 1.5 μl of a 25 to 50 percent aqueous solution of HRP (Sigma VI). All injections were unilateral and most remained limited (Fig. 1). The animals were anesthetized 30 to 60 hours later and were perfused with a solution of paraformaldehyde and glutaraldehyde. In most cases perfusion commenced with 10 percent dextran. The brains were removed and processed according to a modified LaVail and LaVail method for revealing HRP (*7*).

Animals that received frontal lobe injections of HRP exhibited HRP-labeled cells in the amygdala; direct connections from this nucleus to the motor, pre-

motor, and prefrontal cortices were revealed. No such connections were seen in control animals that received an injection of either seroalbumin in the frontal cortex, or HRP in other neocortical areas.

Earlier anatomical work in various mammals has shown amygdaloid projections to nonfrontal neocortical regions (*8, 9*). Amygdalo-prefrontal connections have also been described but only in primates (*8, 10*); results depended, however, on the procedure. Methods in which silver was used (*8*) showed amygdaloid fibres projecting to the orbital region of the prefrontal cortex, a phylogenetically older area that receives fibers from the magnocellular portion of the mediodorsal nucleus of the thalamus, in turn receiving direct connections from the amygdala. Methods in which HRP was used (*10*) showed amygdaloid fibers from the lateral division of the basal nucleus [equivalent to our basal magnocellular nucleus (*11*)] projecting only to the prefrontal convexity. Our present findings show that all injections in the gyrus proreus in 15 cats, either in the portion corresponding in the primate to the orbital region or in that corresponding to the prefrontal convexity, produced labeled neurons in the basal magnocellular nucleus of the ipsilateral amygdala (Fig. 1, A and B, and Fig. 2C). The number of HRP-positive cells depended far more on the local extension of the injection than on its location in the gyrus.

Direct projections from the amygdala to the motor and premotor cortices have not, to our knowledge, been reported previously. In the present series, however, HRP injected in the gyrus sigmoideus anterior (areas 4 and 6) (*12*), in the lower bank of the sulcus cruciatus (areas 4 and 6), and medially in the upper bank of the same sulcus (area 4, extending in some cases to 3a) gave HRP-positive cells in the ipsilateral basal magnocellular nucleus of the amygdala (Fig. 1, C and D, and Fig. 2, A and B). Their number was consistently lower than that produced by injections in the gyrus proreus. A few cells appeared also in the anterior amygdaloid area.

Areas 4 or 6, or both, were injected in 14 cats. Ten of them showed the results described above. The remaining four did not: in each of them the injected area turned out either to be minuscule or to involve only the lateral portion of area 4.

Although no direct amygdalo-motor cortical connections had been demonstrated, it has long been known that amygdaloid stimulation produces certain

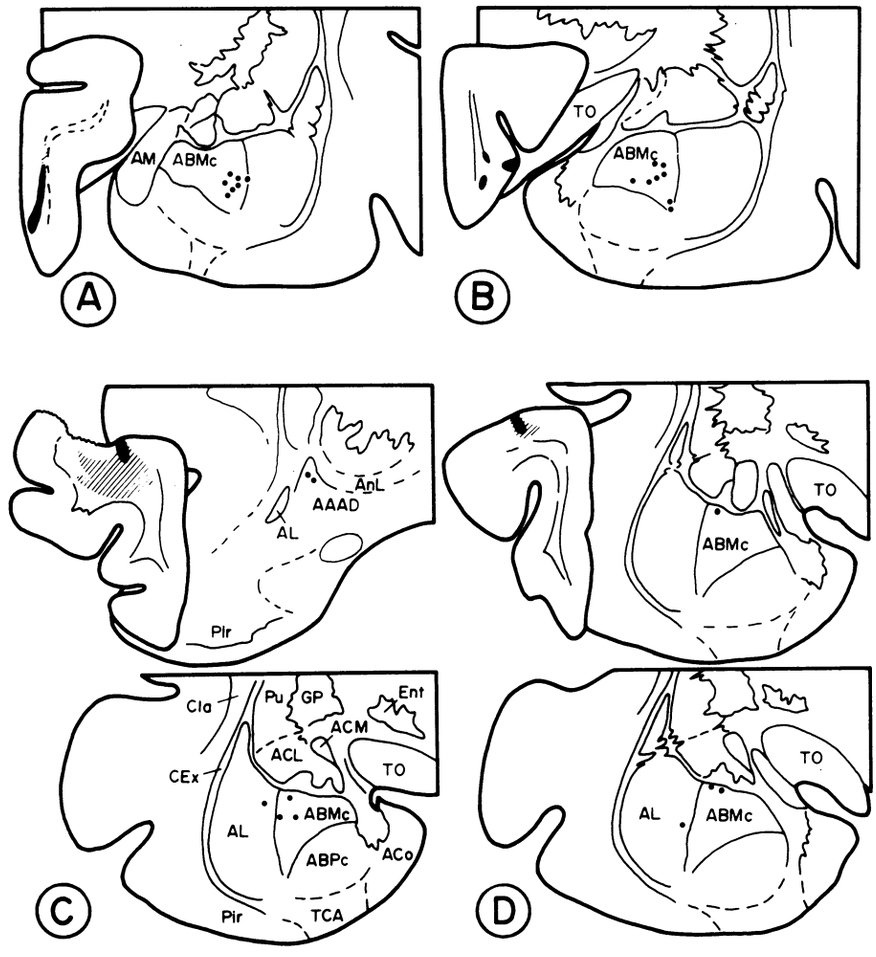

Fig. 1. Distribution of labeled neurons (dots) after small injections of horseradish peroxidase (solid black) into (A) the anteromedial and (B) basolateral parts of gyrus proreus, (C) the lower bank of sulcus cruciatus, and (D) gyrus sigmoideus anterior in four different cats. The cortical projections arise from the nucleus basalis magnocellularis amygdalae (*ABMc*) in every case; some labeled neurons were found also in the area amygdalina anterior (*AAAD*) and in the nucleus lateralis (*AL*). Other abbreviations: *ABPc*, nucleus basalis parvocellularis amygdalae; *ACL*, nucleus centralis lateralis amygdalae; *ACM*, nucleus centralis medialis amygdalae; *ACo*, nucleus corticalis amygdalae; *AM*, nucleus medialis amygdalae; *AnL*, ansa lenticularis; *CEx*, capsula externa; *Cla*, claustrum; *Ent*, nucleus entopeduncularis; *GP*, globus pallidus; *Pir*, cortex piriformis; *Pu*, putamen; *TCA*, area transitionis cortico-amygdalina; and *TO*, tractus opticus. The peculiar morphology of the injected area in the first two drawings (A) is due to the rostro-caudal penetration of the needle, which entered three times in the second case and which, in both cases, delivered a very small amount of HRP. For this reason, the HRP injection site is almost restricted to the needle track, producing an almost inappreciable light-brown halo of diffusion.

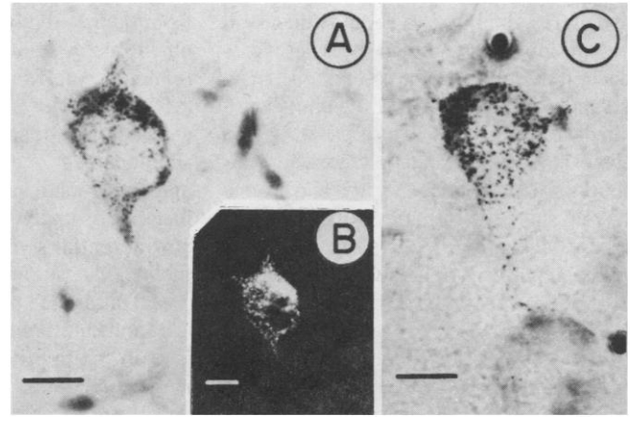

Fig. 2. Neurons labeled by horseradish peroxidase in the nucleus basalis magnocellularis amygdalae ipsilateral to the injections into the motor (A and B) or the prefrontal (C) cortices. (B) Dark-field photomicrograph of the same neuron as in (A). Motor cortex injections gave rise to a significantly lower number of labeled cells than the prefrontal cortex ones. Scale, 20 μm.

stereotyped motor phenomena (*3*) which cannot be interpreted as behavioral responses (*13*). It does not seem, however, that our present findings can explain these phenomena, since the most immediate and constant motor responses are ipsilateral to the stimulated amygdala (not crossed, as they would be if the ipsilateral connection were involved), and bilateral cortical ablation does not suppress them (*14*).

The main source of amygdalo-frontal projections, that is, the basal magnocellular nucleus, is very rich in acetylcholinesterase (AChE) (*15*); hence the influence of the amygdala on the neocortex may well involve cholinergic projections. This fact, together with a recent demonstration of basal prosencephalic AChE-rich cells projecting to the motor cortex (*16*), increases the importance of making a comprehensive study of a cholinergic input to the motor cortex that interacts with noradrenergic and dopaminergic afferences.

The amygdala has played, throughout phylogeny, an important role in elaborating behavioral patterns. In lower mammals its input is chiefly olfactory; in higher mammals the amygdala becomes complicated by the increasing importance of other sensory inputs (*17*). Simultaneously there has been an elaborate development of the cerebral cortex as the ultimate associative level. Probably, the amygdala had to widen its field of action, its basolateral complex undergoing a "vertiginous" development together with the associative neocortex and the mediodorsal nucleus of the thalamus (*18*). It is likely, therefore, that two-way monosynaptic relationships between the amygdala and the neocortex and thalamus appear only above a certain evolutionary stage (*17*).

In conclusion, the amygdala has been considered as the site of evaluation of the motivational significance of stimuli from the environment and the internal milieu (*1, 19*), exerting its influence by modulating hypothalamic drive mechanisms (*19*). To this one should add that it has a double influence on the frontal cortices: indirectly through a thalamic relay (*8*) and directly by way of the paths identified here. In this way, possibly the higher the evolutionary stage, the connections between neocortex and limbic system increase in complexity, allowing progressively more complex and finely adjusted patterns of behavior.

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Differential Sensitivity for Smell: "Noise" at the Nose

Abstract. *The ability of subjects to resolve differences in concentration of chemicals in the vapor phase by smell rivaled the optimum performance of chromatographs. In some instances, subjects resolved a difference in concentration of only 5 percent. The reported inability of olfaction to register fine differences in intensity seems to be largely a result of fluctuations in the stimulus.*

The absolute sensitivity of olfaction is recognized as remarkable, exceeding that of the keenest physical instruments. However, its differential sensitivity is considered poor. The first thorough investigation of the differential sensitivity of olfaction implied that one concentration would smell just noticeably different from another if the two differed by about 25 to 33 percent (*1*). Neither the magnitude nor the generality of this estimate has since been challenged. Somewhat higher and lower values were sometimes obtained, both in the original and in subsequent studies; but the constancy of the value always seemed more impressive than the differences, both within any particular study and, to a large extent, between studies (*2*).

The apparatus used to obtain difference thresholds for smell has ranged from the relatively crude Zwaardemaker olfactometer of 19th-century origin to a relatively sophisticated flow-dilution olfactometer. Use of these devices, whether crude or sophisticated, has not been accompanied by direct assessment of the magnitude and variability of the stimulus. Accordingly, the various estimates of differential sensitivity have rested on the risky assumptions that (i) the supposed difference between one concentration and another is the true difference, and (ii) moment-to-moment fluctuations in the concentration of a "constant" stimulus make only a trivial contribution to the size of the difference threshold. The notion that noise in a sensory stimulus limits discrimination arose in the 19th century and survives in the modern theory of signal detection (*3*). It is ironic that the limiting influence of such noise has not been acknowledged explicitly for olfaction, where the stimulus is particularly difficult to control.

This study explored differential sensitivity to three odorants: *n*-butyl alcohol, ethyl *n*-butyrate, and *n*-amyl alcohol. Two untrained subjects attempted to decide, in a two-alternative forced-choice task, which of two slightly different concentrations smelled stronger (*4*). In any particular 1-hour session, a subject performed the task 100 times with the same two concentrations. After each trial, he was told whether or not he had chosen correctly. He served twice for each pair of concentrations; there were six pairs per odorant.

Concentrations were prepared by diluting the reagent-grade odorants with deionized water. A small volume (1 ml for *n*-butyl alcohol and *n*-amyl alcohol; 2 ml for ethyl *n*-butyrate) of the appropriate concentration was placed onto an absorbent cotton ball that rested on a perforated platform in a glass vessel (60 ml) designed for olfactory testing (*5*). The vessel contained two small ports, one below and one above the platform. The subject inhaled through a monorhinic nosepiece placed at the upper port.