The Journal of Neuroscience, April 1990, 10(4): 1055–1061

Unit Responses Evoked in the Amygdala and Striatum by Electrical Stimulation of the Medial Geniculate Body

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Unit activity was recorded from cells and cell clusters in the amygdala and striatum in response to electrical stimulation of the medial geniculate body (MGB) in rats anesthetized with chloral hydrate. Responses were mostly excitatory and were evoked against a relatively silent background (i.e., the units seldom fired between stimuli). The shortest latency responses were recorded in the caudate putamen (CPU), lateral amygdaloid nucleus (AL), and amygdalostriatal transition area (AST). Longer latency responses were obtained from neurons in the basolateral (ABL), basomedial (ABM), and central (ACE) nuclei of the amygdala. Moreover, while responses were evoked in AL, AST, and CPU with 300–500 μA stimuli delivered once every 10 sec, more intense and higher-frequency stimuli were required to obtain responses in ABL, ABM, and ACE. These findings are consistent with anatomical tracing studies showing that AL, AST, and CPU receive direct projections from the MGB and related acoustic processing areas of the thalamus but that ACE, ABL, and ABM do not.

The mammalian medial geniculate body (MGB) is the auditory thalamocortical relay nucleus: it receives afferent fibers from the inferior colliculus and sends efferent fibers to the auditory cortex (Morest, 1964; Harrison and Howe, 1974; Erulkar, 1975; Diamond, 1983; Morest and Winer, 1986). In addition to projecting to the cortical areas, the MGB also projects subcortically to the amygdala and striatum (Ebner, 1969; Ryugo and Killackey, 1974; Veening, 1978; Ottersen and Ben-Ari, 1979; Turner and Herkenham, 1981; LeDoux et al., 1984, 1985, 1990a; Russchen, 1982; Kudo et al., 1986, 1989). Behavioral studies suggest that thalamo-amygdala pathways play an essential role in the establishment and maintenance of auditory emotional memories (LeDoux et al., 1984, 1986, 1990b; Iwata et al., 1986; LeDoux, 1986, 1987). Little is known, however, about the functional properties of these projections. In the present experiments, we have, therefore, recorded unit activity in the amygdala and striatum while stimulating the MGB electrically. The aims were to determine (1) whether units in these subcortical forebrain areas could be entrained by MGB stimulation and, if so, (2) whether properties of the evoked discharges are consistent with the existence of direct synaptic projections.

Materials and Methods

Rats ($N = 35$) were anesthetized with chloral hydrate (7% in H_2O i.p., 420 mg/kg). Some rats ($N = 7$) were also immobilized with *d*-tubocurarine chloride (1.8 mg/kg, i.v.). Additional doses of both drugs were applied as needed to maintain anesthesia and/or paralysis. Anesthetic supplements (115 mg/kg, i.p.) were usually required 2 hr after the initial dose and then every 30–45 min thereafter. In studies involving paralysis, chloral hydrate (115 mg/kg) and *d*-tubocurarine chloride (0.9 mg/kg, i.v.) supplements were given alternatively every 30 min. Body temperature was maintained at 37°C throughout the experiment. The cranium above the MGB and amygdala was exposed and the dura retracted. The MGB was stimulated with twin pulses (500 μA, 0.1 Hz, 60 μsec each, 200 μsec apart) or single pulses (500 μA to 1.3 mA, 0.2–1 Hz, 500 μsec) produced from a Grass S88 constant-current stimulator and delivered through a bipolar concentric stimulating electrode ($R = 10$ kΩ) positioned at a 20° angle to the coronal plane, leaving as much space as possible for manipulation of the recording electrode. Coordinates, measured from the interaural line, for the MGB at a 20° angle (anterior 4.6, medial 3.0, dorsal 3.7) were calculated on the basis of Paxinos and Watson (1986). Single- or multiple-unit activity was recorded with steel microelectrodes ($R = 2$–5 MΩ) or with conventional glass microelectrodes filled with Pontamine sky blue in 1.5 M NaCl ($R = 10$–15 MΩ), amplified (Grass 511), filtered (AP Circuit), and discriminated (Frederick Haer Company). Discriminated output was viewed on a Tektronix storage oscilloscope and digitized for the construction of rate or poststimulus histograms using a Cambridge Electronic Design 1401. After initial positioning 4 mm below the surface of the brain in the striatum above the amygdala, electrodes were lowered in steps of 25–100 μm with a hydraulic micropositioner (Kopf). Recordings were made in parallel tracks (250–500 μm apart) along the mediolateral axis (from 4.2 to 5.1 mm, relative to the mid-sagittal suture) and the anteroposterior axis (from 7.2 to 4.8 mm, relative to the interaural line).

When steel microelectrodes were used, the locations of the recording tracks were determined from the position of 2 small lesions (100 μA DC, 7 sec) 2 mm apart in the DV plane. The position of the stimulating electrode was also marked by introducing a small lesion (150 μA DC, 10 sec). Animals were perfused with 10% buffered formalin with potassium ferricyanide (5%) and potassium ferrocyanide (5%). The brains were removed, frozen, and postfixed overnight. Sections (50 μm) were cut on a sliding microtome. Every other section was counterstained in thionin (0.25%). Tracks were reconstructed by means of an overhead projector.

When glass microelectrodes were used, Pontamine sky blue was iontophoretically injected (10 μA for 10–20 min) in order to mark the location of the tip of the microelectrode. Animals were perfused with 10% buffered formalin. To prevent the diffusion of the dye, sections were dehydrated and coverslipped without staining. The cytoarchitectonic location of the dye spot was determined under dark-field illumination using fiber bundles for landmarks. Camera lucida drawings were made and tracks were reconstructed.

Received June 9, 1989; revised Aug. 24, 1989; accepted Sept. 19, 1989.

Supported by MH38774 and a Grant in Aid from the New York Heart Association. J.E.L. is an Established Investigator of the American Heart Association. The studies described in this paper were performed in the Laboratory of Neurobiology at Cornell University Medical College.

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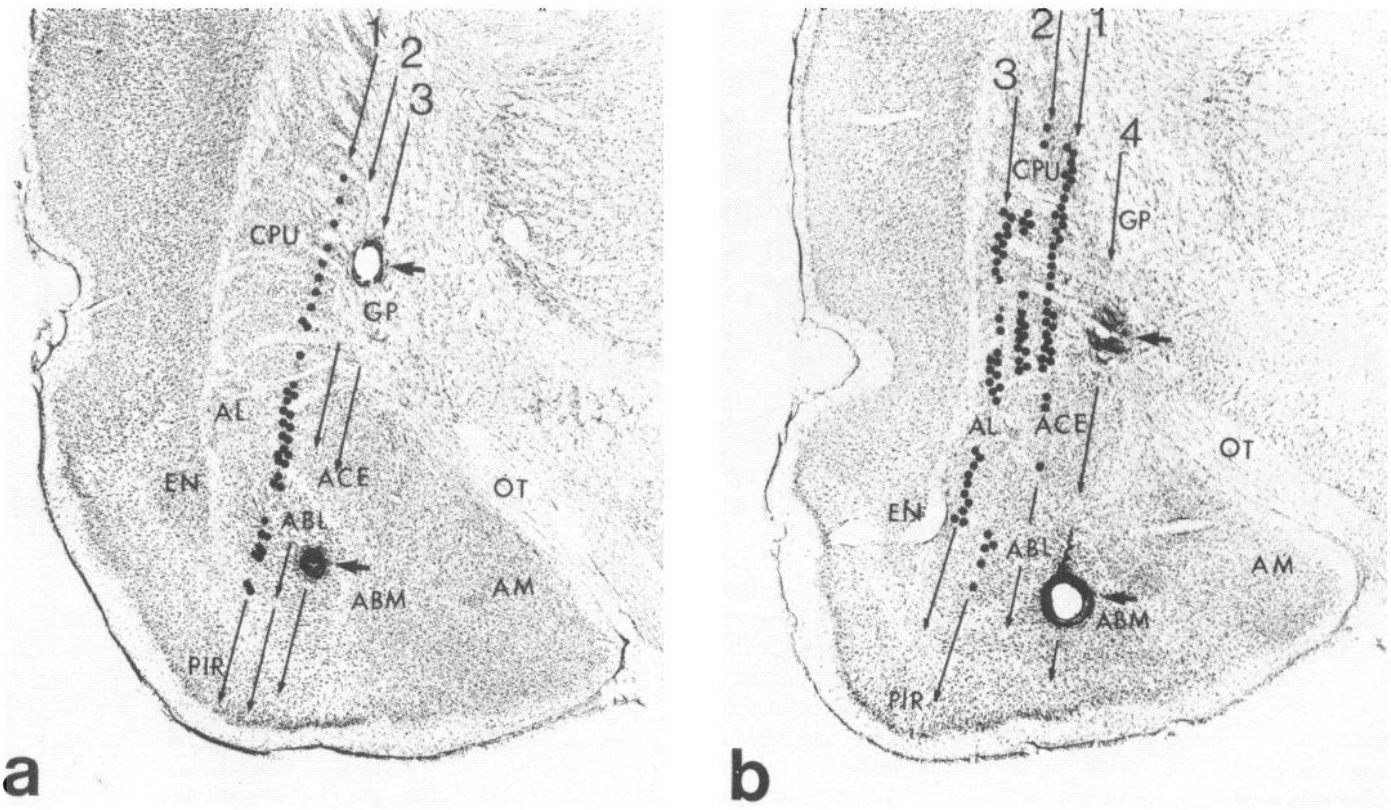

Figure 1. Reconstruction of tracks through the amygdala in 2 rats. Small marker lesions are indicated by *horizontal arrows.* Note in both cases the absence of responses in the medial tracks. In *a,* which is rostral, the ACE does not exhibit any responses, while *b* illustrates a small number of responses encountered in the lateral but not medial part of the ACE.

Results

Stimulus evoked response latency

Recordings were made in the amygdala and overlying areas of the striatum during electrical stimulation of the MGB in 28 rats anesthetized with chloral hydrate and in 4 rats anesthetized with chloral hydrate and immobilized. Responses to MGB stimulation were obtained from 552 cells or cell clusters in the amygdala and striatum. In most instances, responses were evoked against a relatively silent background (i.e., spontaneous discharge rates were either low or absent between stimulations). The onset latency to the initiation of the responses, as determined from poststimulus histograms, was used as the primary measure. Photomicrographs illustrating typical recording tracts are shown in Figure 1. Representative responses of units in striatum and amygdala are shown in Figure 2, and poststimulus histograms are presented in Figure 3.

As the electrode descended through tracks in the striatum en route to the amygdala (Fig. 1), short pulses (twin shocks, 60 μsec each, 200 μsec apart, 500 μA, 0.1 Hz) applied to the MGB readily evoked responses (Figs. 2, 3). The mean onset latency of neuronal responses in the striatum was 4.3 ± 0.09 msec (N = 311), with units in the lateral striatum responding with somewhat shorter latencies (4.0 ± 0.1 msec; range, 3–5 msec, n = 177) than units in the medial striatum (4.7 ± 0.1 msec; range, 3–10 msec, n = 134) (Fig. 4). In most instances, the onset latencies varied slightly from stimulation to stimulation, but sometimes single units were entrained with fixed, invariant latencies.

Responses were also evoked from units in the amygdalostriatal transition zone (AST) lying between the ventral border of the caudate putamen and the dorsal nuclei of the amygdala. The mean onset latency of the responses in AST was 4.5 ± 0.2 msec (n = 53).

As laterally placed electrodes entered the lateral nucleus of the lateral amygdala (AL; Fig. 1*b*), short pulses applied to the MGB continued to evoke unit responses (Figs. 2, 3). The mean onset latency of responses (N = 90) in AL was 6.7 ± 0.2 msec (Fig. 4), with a range of 4–12 msec. Responses were similar in the dorsal, ventral, medial, and lateral parts of AL.

Once the electrode passed through the AL and entered the basolateral nucleus (ABL; Fig. 1), short pulses were generally no longer effective in evoking responses. However, long (500 μsec) and intense (500 μA–1.3 mA) stimuli did evoke responses from some (n = 57) neurons in the basolateral amygdala (Figs. 2, 3). The onset latencies ranged between 6 and 20 msec in this area, with a mean onset latency of 9.6 ± 0.3 msec. In a narrow strip of the ABL bordering the external capsule, short-duration stimuli sometimes evoked responses.

In more medial tracks, unit responses were evoked from cells (n = 35) in the lateral part of the central nucleus of the amygdala (ACE; Fig. 1). Units in this region did not respond to the short pulses that were effective in the CPU, AST, and AL and were entrained only by long and often intense pulses similar to those usually needed to excite cells in the ABL. Typically only one unit responded. The onset latency of the responses was 9.6 ± 0.3 msec (Fig. 4), with a range of 7–15 msec. Units were more easily entrained caudally in the lateral ACE. Cells in the medial ACE never responded to MGB stimulation (Figs. 1, 4).

Responses to long, high-intensity pulses were also recorded

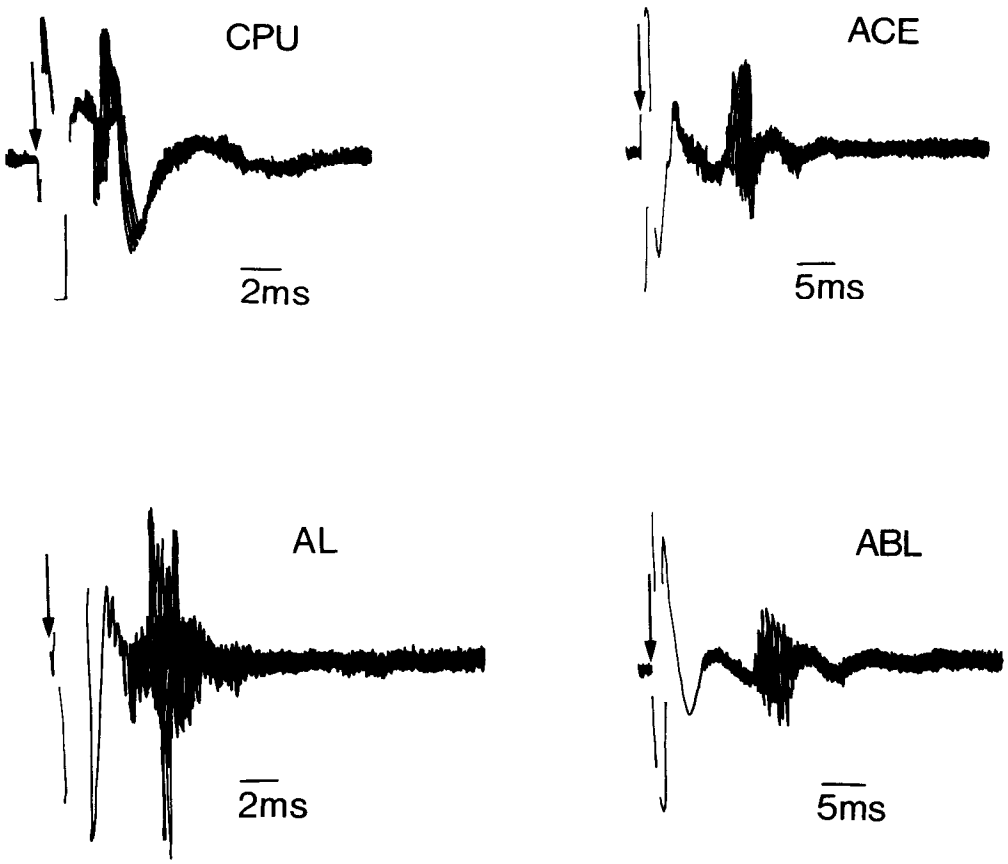

Figure 2. Typical responses to MGB stimulation in the CPU, AL, ACE, ABL. Note that latencies vary between areas, with shorter latencies in the CPU and AL and longer latencies in the ACE and ABL. Track trace represent 5 sweeps. Traces reconstructed from oscilloscope photographs.

from units in the basomedial amygdala (N = 59). As in the central amygdala, the higher the intensity, the fewer the units that were entrained. The mean onset latency was 13.2 ± 0.5 msec, with a wide range of onset between 8 and 30 msec (Fig. 4).

As mentioned, cells in the ACE, ABL, and ABM generally only responded to long, high-intensity pulses. The minimal intensity required to trigger responses in both the striatum and the lateral amygdala was 250–300 μA (twin pulses, 60 μsec each, 200 μsec apart), whereas responses in the central, basolateral, and basomedial amygdala were never evoked with intensities lower than 500 μA. Moreover, cells in the ACE, ABL, and ABM required higher-frequency stimuli (0.2–1 Hz) than cells in the AL, AST, and CPU.

Mean onset latencies of samples obtained were compared by means of Student's t test. Within the amygdala, latencies were shorter in AL than in ACE [t (df = 34) = 4.33, $p < 0.001$], ABL [t (df = 56) = 4.76, $p < 0.001$], and ABM [t (df = 58) = 10.14, $p < 0.001$]. Latencies were shorter in the striatum than in AL [t (df = 89) = 10.50, $p < 0.001$] and shorter in the lateral striatum than in the medial striatum [t (df = 133) = 5.08, $p < 0.001$].

Stimulus evoked inhibitions

Unit activations produced by MGB stimulation were followed by long periods of silence. However, since spontaneous firing rates were very low, it was difficult to determine whether the effect was due to stimulus induced inhibition. Therefore, in 4 preparations anesthetized with chloral hydrate and immobilized, 42 cells or cell clusters were subjected to double shocks. The delay between the 2 shocks was reduced until cells did not respond to the second one. Cells in AL (N = 18), AST (N = 6), and CPU (N = 18) generally failed to respond to a second shock applied 20 msec after the first one. Recovery of the response to the second shock usually began within a 30 msec interval but was complete only with 50–100 msec intervals between the 2 pulses. Because of the unusual stimulation requirement of ACE, ABL, and ABM (see above), these areas were not tested.

Firing frequencies

The cells studied above were identified by their reaction to MGB stimulation. These cells mainly fired in response to the stimulus and seldom discharged spontaneously. This low level of spontaneous activity was confirmed in control studies where the rate of spontaneous discharge of cells in the amygdala and striatum was characterized in the absence of MGB stimulation.

Firing frequencies of 23 cells were analyzed in 7 rats anesthetized with chloral hydrate (420 mg/kg, i.p.) and paralyzed with d-tubocurarine chloride (1.8 mg/kg, i.v.). These cells were found by lowering the recording electrode very slowly until spontaneous unit activity was detected. The low rate of spontaneous activity made it difficult to locate cells. Twelve cells in AL were studied. Three had firing frequencies above 1 Hz (1.5, 2.8, and 5.8 Hz). The firing frequencies of the others ranged from erratic discharges (for example, 4 spikes in 300 sec) to 0.85 Hz. Three cells located in ABL also had firing frequencies between 0.05 and 0.40 Hz, and a fourth had a firing frequency of 2 Hz. Seven cells in the striatum also had very low firing frequencies, ranging from 0.2 to 0.8 Hz.

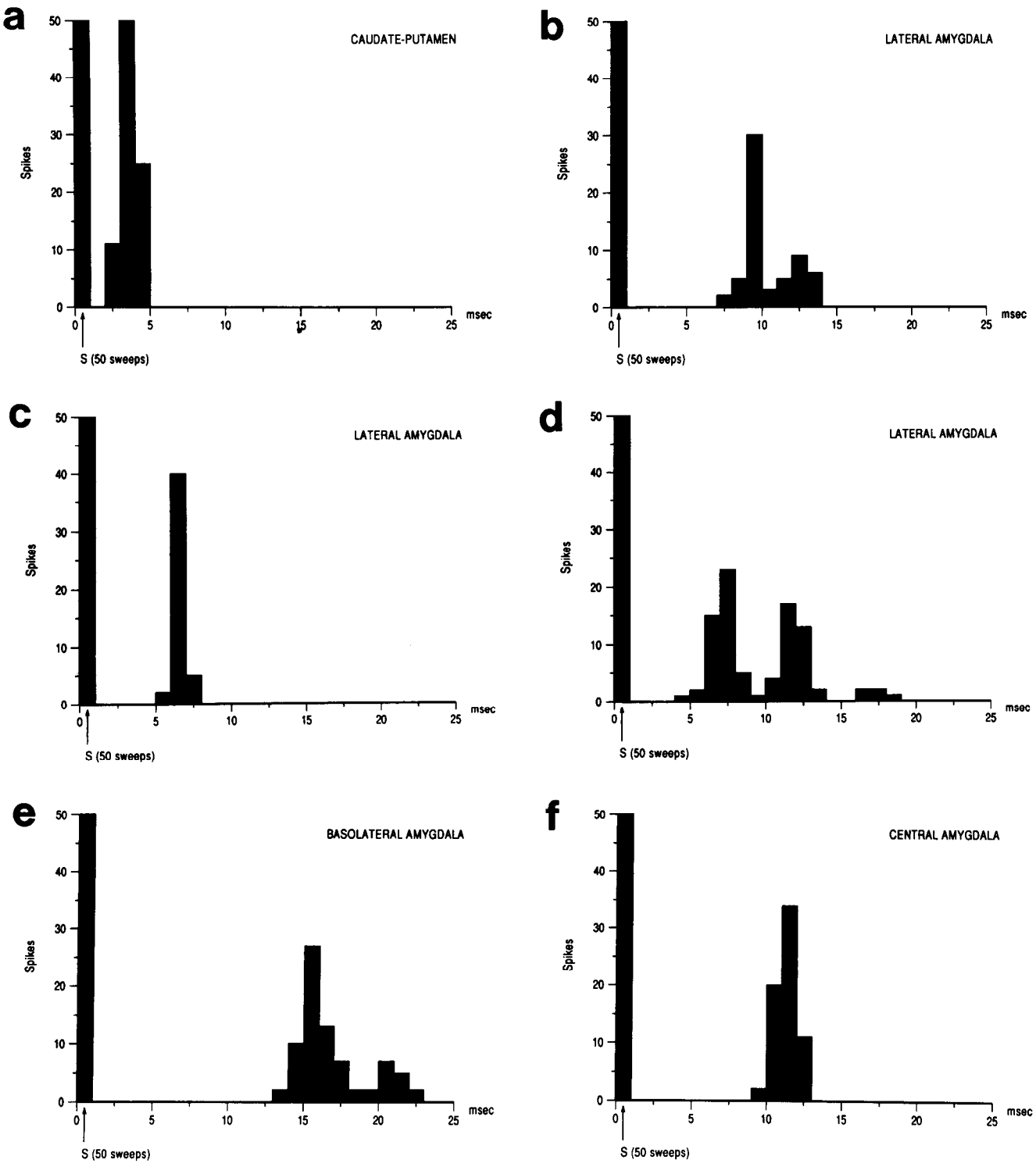

Figure 3. Typical poststimulus time histograms: Note that units were entrained by MGB stimulation against a completely silent background (e.g., cells entrained by the stimulation seldom discharged until the next stimulation. *a*, A very short latency response in the CPU; *b–d* illustrate both short and somewhat longer latencies in the AL; *e* and *f*, long-latency responses in the ABL and ACE. Responses in ABL and ACE required long, intense, high-frequency stimuli.

Discussion

In the present study we have demonstrated that electrical stimuli delivered to the MGB elicit action potentials in neurons within the amygdala and striatum. The responses with the shortest latencies were recorded from cells in the posterior CPU, AST, and AL. Longer latencies were observed in the ACE, ABL, and ABM. Further, the response thresholds were lower in AL, AST, and CPU than in the other areas. Responses were easily evoked by stimuli in the range of 250–500 μA in CPU, AST, and AL,

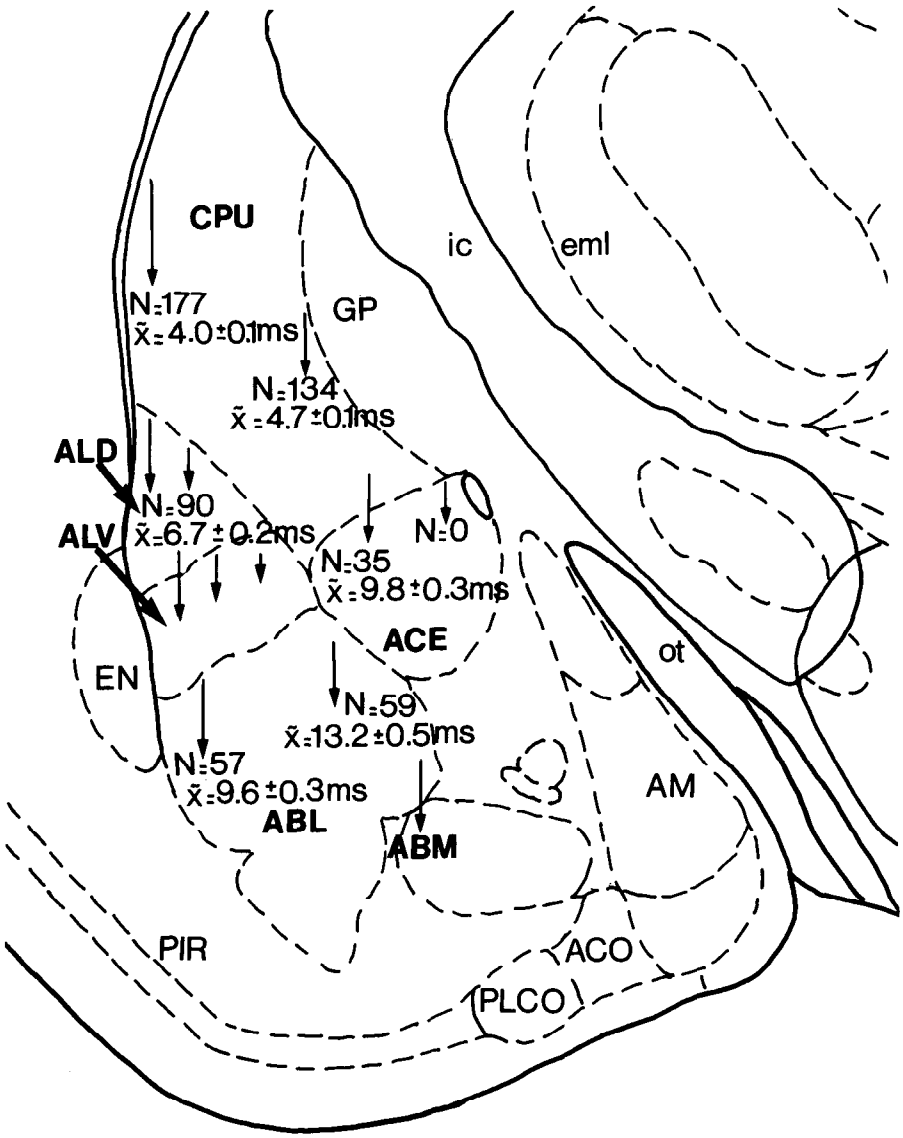

Figure 4. Summary of the mean latencies in response to MGB stimulation in each area studied. Latencies were shorter in CPU, AST, and AL than in ACE, ABL, and ABM. Also, longer, more intense and higher-frequency stimulation was required to elicit the responses in ACE, ABL, and ABM than in AL, AST, or CPU.

whereas in ACE, ABL, and ABM, relatively intense (500 to >1000 μA) and high-frequency (0.2–1 Hz) stimuli were required. These observations, like the anatomical findings presented in the preceding paper, suggest that CPU, AST, and AL receive direct anatomical projections from MGB but that connections between the MGB and ACE, ABL, and ABM may be indirect and multisynaptic.

In general, excitatory responses were evoked from cells with low spontaneous firing rates. The cells fired to the stimulus pulses and then were usually silent until the next occurrence of the stimulus. There is a common agreement throughout studies of firing frequencies in the amygdaloid complex that cells are usually either silent or exhibit very low firing frequencies (Ben-Ari et al., 1974; Ben-Ari and Kelly, 1976; Wang et al., 1979; Le Gal La Salle and Ben-Ari, 1981; Tagaki and Yamamoto, 1981; Zhang et al., 1986). The low spontaneous activity may be related to the presence of anesthesia. However, previous studies have suggested that AL and ABL contain a robust inhibitory system due to the presence of GABA interneurons (Le Gal La Salle, 1978; Ottersen and Storm-Mathisen, 1984; McDonald, 1985; Nitecka and Frotscher, 1989). There is also evidence for the existence of a GABA-mediated feedback loop via axonal collaterals of stellate cells contacting an interneuron which, in turn, synapses on dendrites of the former (Le Gal La Salle, 1978; Tagaki and Yamamoto, 1981). This observation is in agreement with our results showing failure to respond to double shocks separated by 20–100 msec and may explain why the excitatory activity of the cells was so short-lived in response to MGB stimulation. However, inhibition in the amygdala is not limited to GABA interneurons. Dopaminergic fibers issue from the substantia nigra and project to all parts of the amygdala (Fallon, 1981) and iontophoretic applications of dopamine suppress unit activity in the ABL (Ben-Ari and Kelly, 1976; Ben-Ari, 1981). Dopaminergic fibers also innervate the striatum (Dahlström and Fuxe, 1965), where pharmacological studies show a depressant effect of dopamine in this area (see Ben-Ari and Kelly, 1976, for review). Serotonergic fibers issuing from the raphe nucleus project to the AL, ABL, and ACE and iontophoretic applications of serotonin depress unit activity in the ABL (Wang et al., 1979).

The results reported here are largely consistent with anatomical tracing studies (LeDoux et al., 1984, 1985, 1990a). Injections of anterograde axonal markers into the MGB produce terminal-like labeling in the AL, AST, CPU, ABM, and the ventromedial part of ACE (but not the lateral ACE). Injections of retrograde tracers into AL, AST, or CPU label neurons in the medial division of the MGB and the underlying and associated PIN. Injections in the ventromedial ACE and ABM result in retrograde transport to POM, which lies medial to the MGB. Injections in ABL result in no labeled cells in the posterior thalamus. Thus, as observed in the present study, cells in AL, AST, and CPU might be expected to respond to stimulation of the medial MGB, whereas cells in ABL and lateral ACE should respond poorly. Why cells in ABM and the ventromedial ACE responded poorly is more difficult to explain. Since the POM, which projects to these areas, is adjacent to the medial MGB, why didn't the current spread to POM during stimulation and excite these areas? While MGM/PIN is present from the caudalmost to the rostral-most levels of MGB, POM is absent caudally. This is significant since the stimulating electrode was located in the caudal areas of MGB in most experiments. However, even when stimulation was applied more rostrally, strong excitatory responses did not appear in these areas. Alternatively, the MGM/PIN projection may be excitatory and the POM projection inhibitory. Since background neural activity was so low, the effects of inhibitory stimulation would have gone undetected.

It is also possible that the high-threshold, long-latency responses in ACE, ABL, and ABM are mediated by multisynaptic pathways. Anatomical tracing and immunocytochemical studies suggest that AL projects to ACE (especially lateral ACE) and ABM (Krettek and Price, 1978a; Roberts et al., 1982; Amaral, 1987; Nitecka and Frotscher, 1989). In unpublished studies we have injected PHA-L into AL and found a strong projection to ABL and ABM and a weaker but clear projection to ACE. It is thus possible that activation of ABL, ABM, and ACE depended upon an intra-amygdala relay from AL. AL also projects to the prefrontal cortex, which projects to the ACE (Krettek and Price, 1977).

The distinction between areas that receive projections from MGM/PIN and from POM is important. While MGM and PIN receive afferents from the inferior colliculus and thus can be considered part of the acoustic thalamus, POM does not (LeDoux et al., 1987a). The activation of cells in AL, AST, and CPU by thalamic stimulation may, therefore, mimic the effects of peripheral acoustic stimuli.

One possible complication in explaining our results in terms of stimulation of the acoustic thalamus is the fact that the substantia nigra (SN) lies immediately ventral to the MGB and is known to project to the striatum and amygdala (Dalström and Fuxe, 1965; Fallon, 1981). However, it seems unlikely that the spread of current to SN can account for our results. The current would have had to spread at least 2 mm in order to reach pars compacta and reticulata, which project to the amygdala and striatum. The closer lateral nucleus of SN receives afferents from the amygdala (ACE) (Krettek and Price, 1987b) but does not project to amygdala or striatum. If anything, we should have found antidromically activated cells with constant-onset latencies that consistently followed high-frequency stimulation in ACE. Further, we should have had many antidromically activated cells in the striatum as a result of stimulation of striatonigral pathways. While cells with constant-onset latencies were occasionally seen, this was not a common occurrence. Furthermore, these cells never followed very high-frequency stimulation, which indicates that such responses were likely to be orthodromic.

In conclusion, the correspondence between physiological results reported here and axonal transport findings in the preceding paper provides strong support for the existence of pathways that transmit information to the amygdala directly from acoustic processing areas of the thalamus. Since the thalamo-amygdala projections arise in areas where cells tend to be broadly tuned (Winer and Morest, 1983; Weinberger and Diamond, 1987), these circuits may provide the amygdala with only a crude stimulus representation. However, since the relay is monosynaptic, it may offer temporal processing advantages over the multisynaptic circuits that transmit sensory inputs to the amygdala by way of the neocortex (Whitlock and Nauta, 1956; Jones and Powell, 1970; Herzog and van Hoesen, 1975; Turner et al., 1980; Aggleton and Mishkin, 1986; Amaral, 1987). Moreover, thalamic and cortical auditory inputs converge in the amygdala (LeDoux et al., 1987b; Kudo et al., 1989), thus suggesting that the early arrival of crude stimulus information from the thalamus may prepare the amygdala to receive more detailed information from the slower cortical pathways. Further understanding of the mechanisms by which sensory stimuli arouse emotions through the amygdala will depend upon the elucidation of the functions of these parallel sensory inputs to the amygdala.

Appendix

Abbreviations

ABL, amygdala, basolateral nucleus;
ABM, amygdala, basomedial nucleus;
ACE, amygdala, central nucleus;
ACO, anterior cortical amygdaloid nucleus;
ALD, amygdala, lateral dorsal;
ALV, amygdala, lateral ventral;
AM, amygdala, medial nucleus;
AST, amygdalo-striatal transition area;
CPU, caudate putamen;
eml, external medullary lamina;
EN, endopiriform nucleus;
GP, globus pallidus;
ic, internal capsule;
MGB, medial geniculate body;
PLCO, posterolateral cortical amygdaloid nucleus;
PIR, piriform cortex;
POM, posterior thalamic complex; and
OT, optic tract.

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