Gestational age and levels of lysozyme in human amniotic fluids

Groups	Gestational age (weeks)	Number tested	Levels of lysozyme in individual samples (mg/l)	Mean values
A	9	1	4.8	
	10	4	1; 4.2; 6; 7.9	
	11	3	1.2; 2; 5	4.6
	12	5	1.2; 4.8; 5.9; 7; 8.8	
B	13	3	5; 6.8; 8	
	14	8	3.1; 4; 6; 6; 6; 7.2; 11.2; 13.2	
	15	1	4.4	7.4
	16	4	5.2; 8; 9.2; 15	
C	17	3	4.8; 7.2; 12.1	
	18	4	5.6; 8.8; 8.8; 15	
	19	2	9; 15.2	9.7
	20	3	5.8; 11; 13.9	
D	21	–		
	22	3	5.5; 8.5; 18	
	23	–		11.8
	24	2	8; 11.8	

The levels of LZM varied in 46 amniotic fluids from 1 to 18 mg/l, and considerable variations were observed in samples collected at about the same stage of gestation. LZM activity was detected in the amniotic fluid of the youngest fetus tested, 9 weeks old. When the samples were divided into 4 groups according to the age of the fetuses (Table) the mean levels of LZM in each group were found to increase as pregnancy progressed. The detection of LZM in amniotic fluids at an early stage of gestation is in agreement with the observed early synthesis of the enzyme during fetal development[15,16].

Normal levels of LZM were observed in the amniotic fluids from 7 fetuses with neural-tube defects; the individual values were found to range between 3 and 16 mg/l.

The high counts of macrophages observed in amniotic fluids of fetuses with CNS defects[8,9] do not seem to affect the levels of LZM. The clinical significance of estimating the levels of LZM in amniotic fluids to monitor bacterial infections of the fetus remains to be established.

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Deep Temporal Lobe Projections to the Nucleus of the Diagonal Band of Broca

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Summary. Electrophysiological studies with acutely prepared cats found that stimulation of deep temporal lobe structures (e.g., amygdala, prepyriform cortex) evoked responses in the nucleus of the diagonal band of Broca. An analysis of field, extra- and intracellular unitary responses points to the existence of a monosynaptic excitatory connection.

Early electrophysiological studies by Stoll et al.[2] and Gloor[3] suggested the presence of a pathway to the septal area from deep regions of the anterior temporal pole. Specifically, a pathway from the amygdala to the septal component of the diagonal band of Broca (nDBB)[4] has been suggested by anatomical techniques[5–8]. The cortex subjacent to the amygdala (e.g., the prepyriform cortex) might also project to the nDBB[9]. The present report investigates some of the physiological properties of these deep temporal lobe pathways to the nDBB.

Cats were anesthetized with thiamylal sodium (25 mg/kg) and chloralose (40 mg/kg). The septum and hippocampus were exposed by removing the overlying cerebral cortex and corpus callosum. Bipolar stimulating electrodes with an inter-pole distance of 0.5 mm were stereotaxically placed in the amygdala and prepyriform cortex. In most of the experiments, the amygdala stimulating electrodes were arranged so that the basolateral group or the anterior amygdaloid area from AP levels +12 to +15 was stimu-

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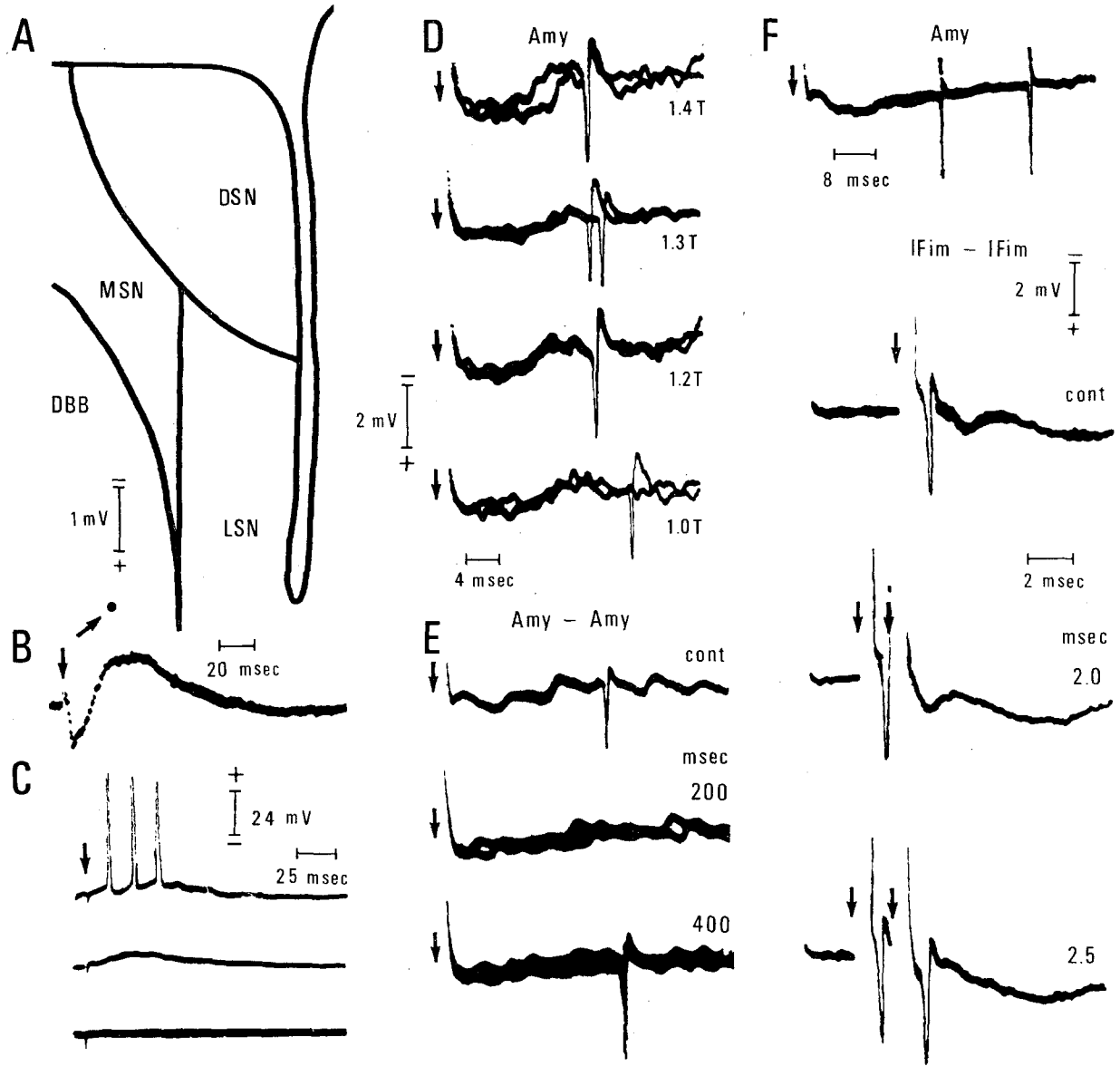
lated[10]. Stimulation sites were verified histologically. An additional stimulating electrode was visually placed upon the ipsilateral fimbria (IFim) to antidromically identify cells in the nDBB. In most experiments, the stria terminalis was sectioned in the caudate-thalamic sulcus at a level anterior to the placement of the IFim electrode.

Microelectrodes were used to record field potentials, extra- and intracellular unitary responses. A detailed account of the preparatory procedures is given by DEFRANCE et al.[11].

Field potentials recorded in the nDBB following supramaximal amydala or prepyriform cortex stimulation resulted in a response which presented an early positivity followed by a dominate, prolonged negativity. An example, recorded at a location indicated in Figure A, is shown in B. Occasionally, the negativity, was followed by a shallow positivity. The prominent positive-negative response was unaltered by stria terminalis section, indicating the involvement of an alternate pathway to the nDBB.

An example of an intracellular recording following amygdala or prepyriform cortex stimulation is shown in C. To supramaximal stimulation, the cell issued a series of spikes (upper trace). After deterioration of the spike mechanism, an initial depolarizing potential remains (middle trace). These depolarizing intracellular events



A) Tracing of histological section showing recording site (arrow) for field potential of B) at a depth of 4.4 mm from the dorsal septal surface. B) Field response following stimulation of anterior amygdaloid area; averaged 16 times. C) Intracellular recording from a cell in nDBB following stimulation of the anterior amygdaloid area (upper and middle trace). The steady transmembrane potential was 56 mV. Lower trace is the extracellular control. D) Testing of an extracellular unit in the nDBB to changes in the intensity of stimulation of the amygdala. Stimulus intensities are shown in times threshold (T). E) Paired-stimulus testing of a unit using an Amy-Amy combination. Responses shown are the test control (cont) and test response for 200 msec and 400 msec interstimulus intervals. F) Extracellular unit responding to Amy stimulation and paired-stimulus testing of the unit utilizing an IFim-IFim combination.

In all cases, downward arrow indicates the onset of the stimulus artifact.

DSN, dorsal septal nucleus; MSN, medial septal nucleus; LSN, lateral septal nucleus; nDBB, nucleus of the diagonal band of Broca; Amy, amygdala; IFim, ipsilateral fimbria.

correspond, temporally, to the extracellular negativity. Furthermore, when recording with an electrode with a fortunate tip size, extracellular unitary discharges could be seen riding upon the slow negative wave. Such negativity, then, seems to represent an envelope of spike activity and associated EPSPs.

An analysis of extracellular unitary responses was undertaken; 165 units were examined. The conduction velocity of fibres from the deep temporal lobe was determined from the analysis of the difference in latencies of extracellular units, with stimulating electrodes in both the anterior amygdaloid area and the diagonal band of Broca. The modal conduction velocity from the amygdala to the nDBB, of 11 estimates, was 0.6 M/sec; with a range of 0.5 M/sec to 0.9 M/sec. GLOOR[3] has reported a conduction velocity of fibres from basolateral amygdala stimulation to the anterior hypothalamus of 1 M/sec.

The relative stability of latency for unitary discharges with changes in stimulus intensity is shown in D. With submaximal stimulation, beginning at 1.4 times threshold (1.4 T), the latency was 18 msec. The latency remained stable until threshold (1.0 T) is reached where the latency increased to 22 msec. This relative constancy in discharge latency is taken to represent the monosynaptic excitation of cells. The latency shift at threshold is understandable in terms of slow rise times and temporal dispersion of the EPSPs. In addition to those units which showed stable latencies, units were encounted which showed marked instability, sometimes even at a constant stimulus intensity. These, undoubtably, represent polysynaptic activation.

With the stria terminalis severed, stable, as well as unstable, extracellular units could still be recorded to amygdala and prepyriform cortex stimulation. Consideration of the conduction velocity and conduction distance made the observed latencies of the stable units reasonable for a monosynaptic connection. These results suggest a combination mono- and polysynaptic input from the amygdala and/or prepyriform cortex to the nDBB.

Extracellular units which displayed stability in latency showed durations of test response suppression with paired-stimulus testing from 100 msec to 1000 msec. The modal time to recovery was 400 msec. An example is shown in E. The upper trace is the test control. The conditioning responses are not shown. As seen in this instance, the unitary discharge is absent in a 200 msec interval, but re-appears in a 400 msec interval. This characteristic recovery time was also seen for the test field response. No differences were found in the behavior of the test responses between amygdala and prepyriform cortex stimulation.

Extracellular units in the nDBB responding to deep temporal lobe stimulation could be antidromically activated by IFim stimulation; 45 of 133 units tested were antidromically by IFim stimulation. F shows an example where amygdala stimulation evokes two unitary discharges and where IFim stimulation produces a short latency discharge. The latency in this instance was 1.5 msec. Paired-stimulus testing of the unit with an IFim-IFim combination found it to recover in a 2.5 msec interval, suggesting its antidromic nature. The modal conduction velocity of the antidromically activated units was 4.0 M/sec, with value ranging from 2.8 M/sec to 4.8 M/sec.

There is a problem concerning the origin of the projections to the nDBB. RAISMAN[12] and DEOLMOS[13] indicate that amygdala projections are restricted to the tract of the diagonal band. Moreover, the prepyriform cortex has been shown to project through the amygdala[5,9]. Hence, in this study, amygdala stimulation should also activate fibres of passage from the prepyriform cortex. What contribution the amygdala makes to the projection to the nDBB could not be determined with the present techniques. This difficulty led us to examine inputs from both areas and include these results in the term 'deep temporal lobe projections'.

These results suggest that the deep temporal lobe projections to the nDBB are monosynaptically excitatory and that some of the target neurons project to the hippocampal formation. The nDBB, therefore, provides a synaptic station for prepyriform cortex and possible amygdala input for the influence of hippocampal activity[14].

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Adrenaline and the Electrogenic Sodium Pump in *Rana catesbeiana* Sympathetic Ganglion Cells

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Summary. The effect of adrenaline on the Na^+-pump in bullfrog (*Rana catesbeiana*) sympathetic ganglion cells was studied by use of electrophysiological methods. The rate of removal of excess Na^+ injected into a ganglion cell was increased by adrenaline. The K^+-activated hyperpolarization of cell membrane, which might be produced by an electrogenic Na^+-pump, was also increased by adrenaline. These results suggested that adrenaline was able to accelerate the Na^+-pump, possibly the electrogenic Na^+-pump.

When adrenaline (Ad) is directly applied to bullfrog's sympathetic ganglia, the ganglion cells produce depolarizing (Ad-depolarization) and also hyperpolarizing (Ad-hyperpolarization) responses[1]. The nature of Ad-hyperpolarization is essentially similar to that of the slow inhibitory postsynaptic potential (slow IPSP)[1], which seems to be produced by an electrogenic sodium pump[2]. It was, therefore, suggested that Ad might be able to accelerate the electrogenic sodium pump and thus to produce the Ad-hyperpolarization[1]. The present experiment was designed to demonstrate the experimental

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