STRYCHNINE BLOCKS BINAURAL INHIBITION IN LATERAL SUPERIOR OLIVARY NEURONS[1]

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Abstract

The present study seeks to identify neurotransmitters mediating binaural inhibition in lateral superior olivary nucleus neurons. Neurons in this auditory structure receive inputs from both ears and are thought to code for localization of sound in space. Iontophoretic application of glycine during monaural stimulation was found to mimic the inhibition observed with binaural stimulation. Binaural inhibition was blocked by application of the glycine receptor antagonist, strychnine, as were the effects of iontophoretic application of glycine. The post-strychnine recovery time course for return of synaptically mediated binaural inhibition and recovery of the effects of iontophoretic glycine application were identical. Although the superior olivary complex (SOC) neurons displaying binaural inhibition could in some cases be inhibited by GABA, the binaural inhibition rarely was blocked by iontophoretic application of the GABA receptor antagonist, bicuculline. These findings suggest that glycine may be a neurotransmitter mediating binaural inhibition in certain SOC neurons and that the projection to the lateral superior olivary nucleus from the medial nucleus of the trapezoid body may be glycinergic.

The superior olivary complex (SOC) is the first structure in the ascending auditory pathway to receive bilateral input (for review see Boudreau and Tsuchitani, 1970; Brugge and Geisler, 1978; Tsuchitani, 1978). The auditory nerve on each side projects from the cochlea to the cochlear nucleus on that side. Binaural neurons in the lateral superior olive (LSO) receive their predominant contralateral input from the opposite ventral cochlear nucleus (VCN) via a secure synapse in the ipsilateral medial nucleus of the trapezoid body (MNTB; see Fig. 1). Ipsilateral input comes directly from the homolateral VCN to the same neuron. Ultrastructural studies indicate the existence of at least three types of synaptic endings upon the neurons of the medial superior olive (MSO). The morphological characteristics of these endings suggest that some may be inhibitory but others may be excitatory (Clark, 1969; Perkins, 1973; Schwartz, 1980). Although comparable morphological evidence has not been reported for the lateral superior olive (LSO), elec-

trophysiological evidence indicates the existence of neurons in both the MSO and LSO which are excited by inputs from one ear and inhibited by simultaneous stimulation of the opposite ear (Galambos et al., 1959; Goldberg and Brown, 1968, 1969; Guinan et al., 1972a, b; Tsuchitani, 1977, 1978).

The nature of the neurotransmitter mechanisms mediating this inhibition is not known, but the existence of binaural neurons within the SOC provides an opportunity to study the interaction between excitatory and inhibitory synaptic inputs by varying the acoustic stimulus to each ear. Recently, Zarbin et al. (1981), using autoradiographic evidence, demonstrated that strychnine, the specific glycine antagonist, displays significant receptor binding in the rat LSO and minimal binding in the rat MSO.

The present study examines binaural inhibition and attempts to antagonize the synaptically mediated binaural inhibition by iontophoretic application of the inhibitory amino acid antagonists, strychnine or bicuculline. The actions of exogenously applied glycine and GABA then are compared to the inhibition produced by the endogenous synaptically released inhibitory neurotransmitter.

Materials and Methods

Chinchilla, *Chinchilla laniger*, weighing 300 to 600 gm were anesthetized initially with sodium pentobarbital (40 mg/kg), followed 30 min later by ketamine hydrochloride

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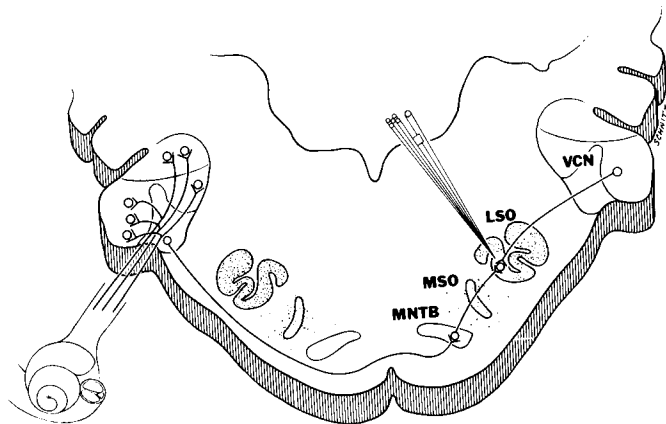
Figure 1. A schematic representation of the innervation of
binaural neurons in the lateral superior olive (*LSO*). Ipsilateral
input to these neurons derives from the anterior regions of the
ventral cochlear nucleus (*VCN*). Contralateral input from the
interstitial nucleus of the opposite VCN traverses the midline
to synapse in the medial nucleus of the trapezoid body
(*MNTB*), which in turn projects to the LSO. The present study
finds that strychnine blocks binaural inhibition in neurons of
the LSO which receive a prominent input from the MNTB.

(40 mg/kg). Supplementary injections of sodium pento-
barbital were given as needed to maintain anesthesia.
Because of the effect of pentobarbital on neuronal
response patterns (Young and Brownell, 1976) and on
amino acid neurotransmitter release (Potashner and
Lake, 1981), some animals were anesthetized with and
maintained on ketamine alone. The animal was placed in
a sound-attenuating chamber (Industrial Acoustics Co.,
Inc.) and body temperature was monitored and main-
tained by a thermostatically controlled DC heating pad.

A posterior fossa approach was used to allow alignment
of the electrode parallel to the major axis of the MSO.
This permitted independent punctures of the three main
nuclei comprising the SOC in chinchillas. Details of the
surgical procedures have been described previously (Cas-
pary, 1972; Caspary et al., 1979) with the following addi-
tions. Extracellular recordings were obtained with a glass
micropipette (tip diameter, 1.0 to 1.5 μm) glued alongside
a five-barrel micropipette (Glass Co. of America) (outer
tip diameter, 8 to 10 μm) so that the single barrel tip
protruded 3 to 7 μm beyond the tip of the five-barrel
electrode. A balancing or summating channel was em-
ployed to alleviate current effects, and this barrel and the
recording micropipette were filled with 2.0 M potassium
acetate. In some experiments the recording barrel was
filled with Alcian blue (8% in 0.5 M sodium acetate (Lee
et al., 1969)) to allow for marking of individual recording
locations. The remaining four barrels contained 0.5 M
glycine, pH 3.5 to 4.0 (Sigma Chemical Co.); 0.002 M
strychnine (Sigma); 0.5 M GABA, pH 3.5 to 4.0 (Sigma);
and 0.001 M bicuculline (Sigma). In some experiments 0.5
M picrotoxin (Sigma) was substituted for bicuculline.
Ejection, retention, and balancing currents were pro-
duced by a constant current source (Medical Systems,
Inc. model BH-2).

Upon termination of the experiment, the animal was
sacrificed with sodium pentobarbital and underwent car-
diac perfusion with saline followed by 10% formalin. The
relevant portion of the brain was serially sectioned in the

plane of the electrode and tracings were made of all
sections containing electrode tracks to enable reconstruc-
tion of penetrations through the SOC.

Fifty coherent tone bursts (50 or 100 msec duration, 5
or 10 msec rise/fall) were presented at the rate of 2/sec.
Auditory stimuli were delivered through TDH-49 ear-
phones (Telephonics) and calibrated at the level of the
external auditory meatus using a Brüel and Kjaer 0.25-
inch condenser microphone. The system was calibrated
from 0.05 to 30.0 kHz and attenuation data were cor-
rected to dB SPL re 0.0002 dyne/cm^2. No binaural inten-
sity differences of greater than 20 dB sound pressure
level (SPL) were used in the present study, and all decibel
values are in SPL.

Neuronal responses were processed on-line with a
PDP-8E computer (Digital Equipment Corp.) and were
stored simultaneously on analog magnetic tape (Tand-
berg 100) for subsequent off-line processing. Rapid onset
noise bursts (3 msec duration, 20 msec interaural delay)
were used as search stimuli. Both excitatory and inhibi-
tory response areas and best frequency were obtained for
all neurons encountered. Poststimulus time histograms
(PSTHs), interspike interval histograms (ISIHs), and
spike counts were obtained for all neurons before, during,
and subsequent to the iontophoretic administration of
the putative amino acid transmitters and their antago-
nists.

The experimental paradigm used varied somewhat de-
pending upon the response characteristics of the neuron
being studied. To classify neurons, the following PSTHs
were obtained: (*a*) monaural contralateral, (*b*) binaural,
and (*c*) monaural ipsilateral. When binaural stimulation
revealed inhibition, intensities and phase were varied to
maximize the inhibitory effect. The following iontophor-
etic paradigm then was implemented: (*a*) GABA or gly-
cine was applied during monaural stimulation in an effort
to mimic the endogenous binaural inhibition; (*b*) strych-
nine, bicuculline, or, in some cases, picrotoxin was applied
continuously during binaural stimulation in order to
block the synaptically mediated inhibition; and (*c*)
GABA or glycine was introduced during or immediately
following application of its respective antagonist.

Results

One hundred and nineteen neurons were tested with
iontophoretic application of GABA, glycine, strychnine,
and bicuculline or picrotoxin and were located in or
immediately surrounding the LSO. Only neurons from
punctures that penetrated a major portion of the LSO
are included, while data from punctures which pene-
trated the MNTB or MSO are not included in the present
study. Thirteen neurons responded to monaural stimu-
lation alone, either ipsilaterally or contralaterally, while
106 neurons responded to binaural stimulation. Seventy-
nine of the binaural neurons were inhibited during
binaural stimulation, while 27 were not. Of neurons show-
ing binaural inhibition, 81% could be inhibited by glycine
during monaural stimulation, which mimicked the
binaural condition (synaptic inhibition) (Table I). Ion-
tophoretic application of strychnine resulted in blocking
of endogenous inhibition in 82% of these neurons while
blocking the effects of subsequent simultaneous ionto-
phoretic application of glycine in 97% of the neurons

TABLE I
Effects of iontophoresis on binaurally inhibited LSO neurons

Substance	Stimulus Condition	Number of Neurons Tested	Percentage of Neurons Affected
Glycine	Monaural	68	81
Strychnine	Binaural	54	82
Strychnine and glycine	Monaural	33	97
GABA	Monaural	67	72
Bicuculline	Binaural	15	7
Bicuculline and GABA	Monaural	50	50

examined (Table I). GABA was also effective in mimicking binaural inhibition (72%), but only 7% of the neurons tested showed blocking of the synaptically mediated binaural inhibition with the application of the GABA antagonist, bicuculline (Table I). A χ^2 test of the difference between the effects of bicuculline and strychnine on binaural inhibition was highly significant ($\chi^2 = 25.76$; $df = 1$; $p < 0.001$). Bicuculline was effective only 50% of the time in blocking the effects of iontophoretically applied GABA (Table I).

An example of a neuron that exhibited a phasic response and was typical of the largest population of LSO neurons is seen in Figure 2. Ipsilateral stimulation (right ear) (Fig. 2A) results in an "on-type" response, while binaural stimulation results in complete inhibition of the ipsilateral response (Fig. 2B). Glycine application during ipsilateral stimulation (Fig. 2C) greatly reduces the response, thus mimicking the binaural condition. Figure 2E displays the blocking of the endogenous synaptic inhibition by strychnine which is seen to return the response to a pattern similar to the ipsilateral (monaural right) stimulation. Glycine inhibition is blocked by strychnine (Fig. 2F) and GABA application has little effect (Fig. 2L). The neuron displayed in Figure 2 and another extensively studied neuron (R47-3, Figs. 3 and 4) are representative of the consistent blocking of binaural inhibition produced by iontophoretic application of strychnine. The neuron in Figure 3 also is driven by ipsilateral stimulation and is inhibited binaurally (Fig. 3, *A* and *B*). Glycine application (Fig. 3C) mimics the binaural condition and the effect is blocked by strychnine (Fig. 3F). Figure 3E shows that strychnine blocks much of the binaural inhibition (compare with Fig. 3B).

Recovery from strychnine was monitored by a series of binaural histograms and monaural histograms accompanied by glycine application. The return of glycine effectiveness coincided with the reappearance of a control level of binaural inhibition. The recovery time from strychnine application varied between 5 and 25 min. Rate intensity curves for the neuron seen in Figure 3 are displayed in Figure 4. Binaural inhibition develops at intensities greater than 14 dB (Fig. 4B) and is mimicked in the monaural plus glycine condition (Fig. 4C). Iontophoretic application of strychnine at each intensity (Fig. 4D) results in a curve similar to that of the monaural control (Fig. 4A) except for a slight increase in overall response. Differences seen between curves *B* and *D* in Figure 4 are representative of findings for other neurons for which data at multiple intensities were obtained.

An example of a neuron displaying excitation with monaural stimulation, ipsilateral or contralateral, and

inhibition with binaural stimulation is displayed in Figure 5. The ipsilateral stimulation plus glycine (Fig. 5D) mimics the binaural condition (Fig. 5B). Strychnine reverses the synaptically mediated binaural inhibition (Fig. 5E) and blocks the effects of simultaneously applied glycine (Fig. 5F) during binaural stimulation.

Among purely excitatory binaural neurons, GABA appeared to be somewhat more effective (80%; 16 of 20 neurons) than glycine (69.6%; 16 of 23 neurons) in producing inhibition, but the difference was not statistically significant.

Certain low frequency neurons exhibit binaural inhibition only when there is a fixed temporal relationship between the two ears. This temporal difference corresponds to a fixed phase relationship at any given frequency when a given frequency is delivered binaurally. Fourteen of these neurons were tested iontophoretically. Seven of 10 neurons showed blocking of the phase-dependent, synaptically mediated binaural inhibition with
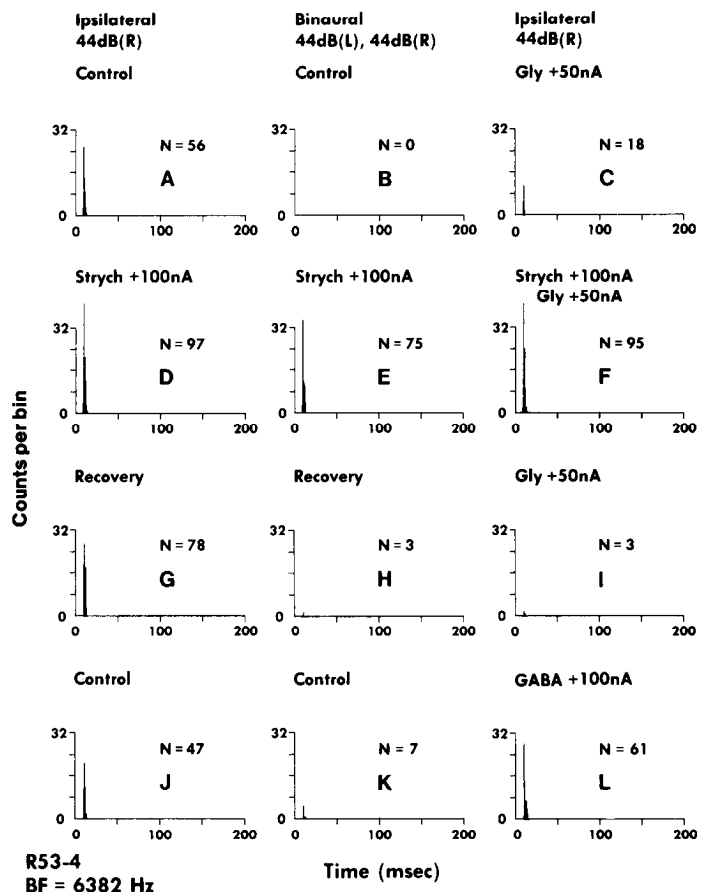

Figure 2. PSTHs of neuronal activity to 100-msec tone burst stimuli showing the effects of iontophoretically applied glycine (*Gly*), strychnine (*Strych*), and GABA on a phasic responder from the lateral superior olive (*LSO*). The *left* and *right* columns display the response to ipsilateral stimuli at 44 dB SPL, while the response to binaural stimuli at 44 dB SPL is seen in the *center column*. This neuron was excited by an ipsilateral stimulus (*A*) and was inhibited by binaural stimuli (*B*). Glycine application reduces the ipsilateral response (*C*) or mimicry of the binaural condition. Strychnine application blocks binaural inhibition (*E*) and glycine inhibition (*F*). Note that GABA application has little effect (*L*). *BF*, best frequency.
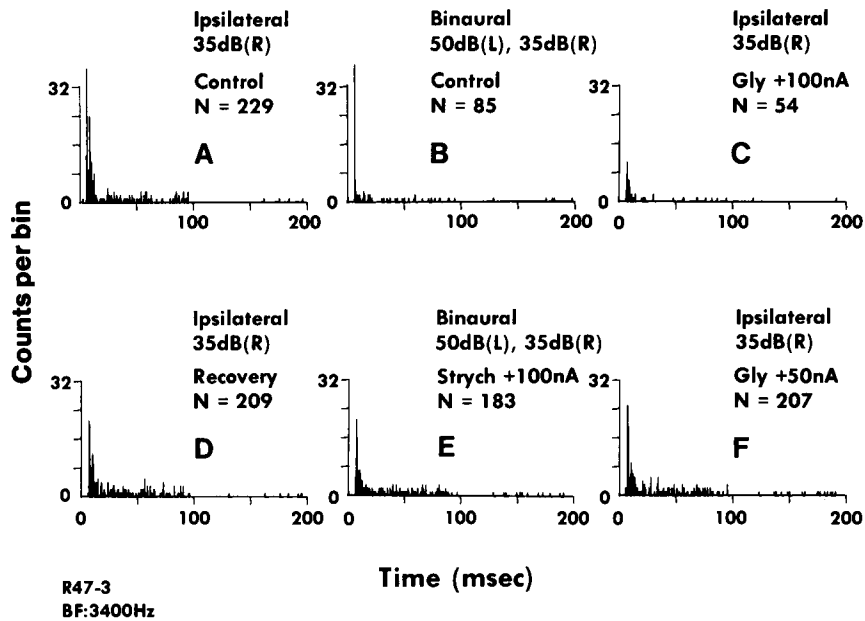
Figure 3. PSTHs of activity of an LSO neuron for 100-msec tone presentations. This neuron was driven by ipsilateral stimulation (*A*) and was inhibited binaurally (*B*). Glycine application mimics the binaural response (*C*). Strychnine antagonizes binaural inhibition. The residual level of strychnine is sufficient to block the effect of iontophoretic application of glycine (*F*). *BF*, best frequency.
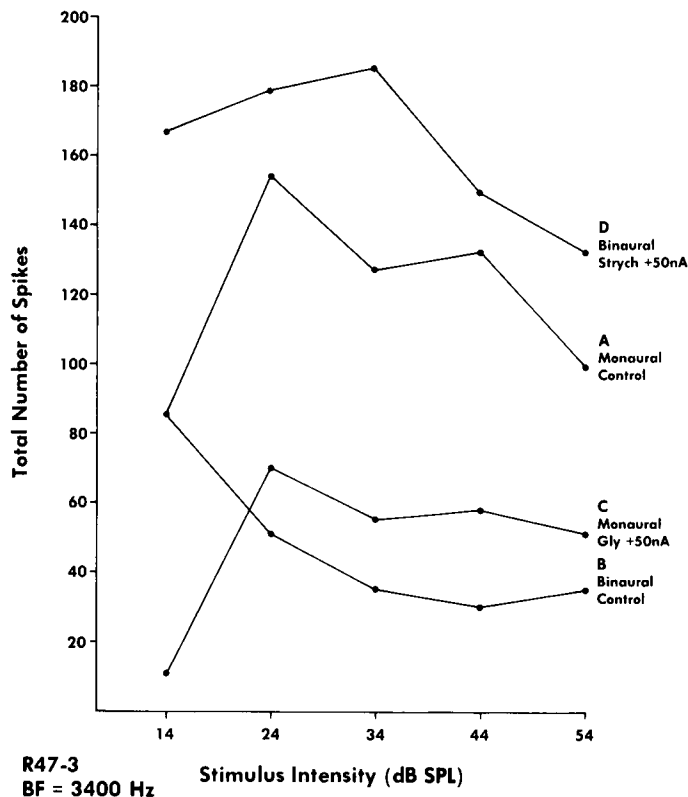
Figure 4. Response rates for each of the experimental conditions as a function of stimulus intensity. Intensities at both ears were set to the same level for the binaural condition. Strychnine (*D*) blocks the inhibition produced by binaural stimulation (*B*). Strychnine also raises the overall activity beyond the monaural control level (*A*). Glycine reduces the level of response (*C*) without alteration of the profile across stimulus intensity.

iontophoretic strychnine. A 180° out-of-phase binaural stimulus inhibits the monaural response (Fig. 6, *A* and *B*) of this neuron located in the lateral lobe of the LSO. The in-phase condition (not shown) has no effect, while the application of glycine during monaural stimulation (Fig. 6*C*) mimics the out-of-phase condition. Strychnine blocks both the effect of iontophoretic application of glycine and the synaptically released transmitter evoked by binaural out-of-phase stimuli (Fig. 6, *D* and *E*). The binaural inhibition returns (Fig. 6*F*) simultaneously with the return of the effect of iontophoretic glycine application. None of the phase-sensitive neurons tested displayed reversal of inhibition with bicuculline. Ninety-five percent of these neurons (12 of 14) were inhibited by glycine, while only 48% (6 of 13) were inhibited by GABA.

Discussion

Neurons located in the LSO receive inputs from both ears and are thought to act as comparators to produce an output coded for spatial localization of sound (for review see Erulkar, 1972; Brugge and Geisler, 1978; Tsuchitani, 1978). The inputs to the LSO nucleus are from the ipsilateral spherical cell area of the anteroventral cochlear nucleus with the contralateral input originating from ipsilateral MNTB neurons which receive input from the globular cell in the contralateral interstitial nucleus and the anterior portion of the posterior ventral cochlear nucleus (Rasmussen, 1967; Harrison and Feldman, 1970; Browner and Webster, 1975). The ultrastructural studies of the afferent terminals in the SOC of the cat (Clark, 1969; Lindsey, 1975; Schwartz, 1980) and chinchilla (Perkins, 1973) indicate the presence of type I, II, and III endings, suggesting that both excitatory and inhibitory endings are present (Uchizono, 1975). Although this is best documented in the MSO, a similar
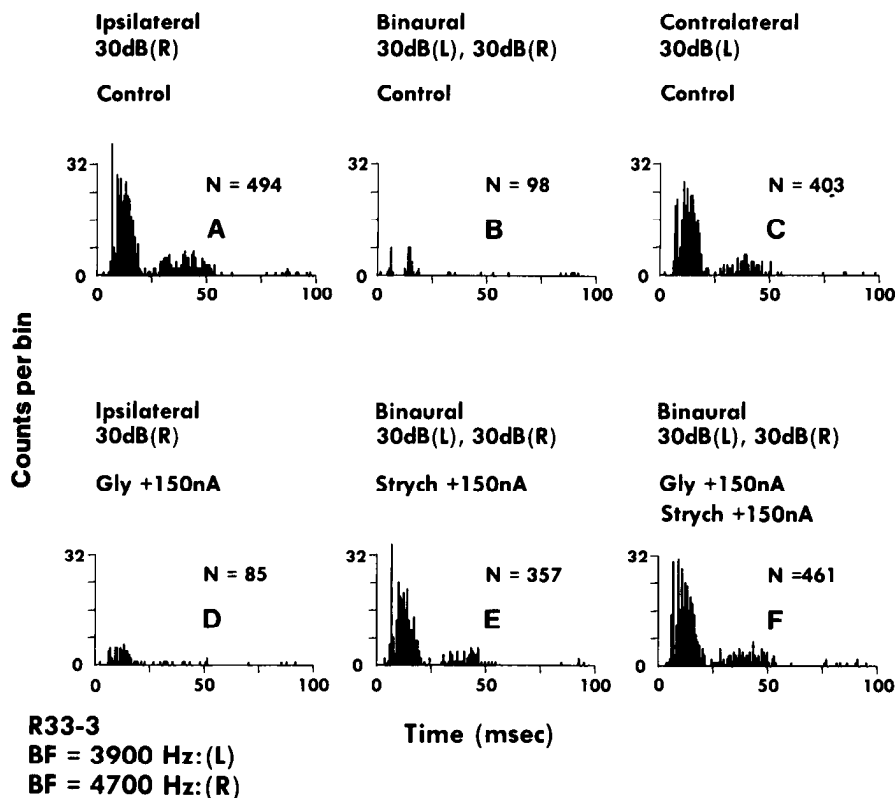
Ipsilateral 30dB(R) · Binaural 30dB(L), 30dB(R) · Contralateral 30dB(L)

Control · Control · Control

N = 494 A · N = 98 B · N = 403 C

Ipsilateral 30dB(R) · Binaural 30dB(L), 30dB(R) · Binaural 30dB(L), 30dB(R)

Gly +150nA · Strych +150nA · Gly +150nA / Strych +150nA

N = 85 D · N = 357 E · N =461 F

R33-3
BF = 3900 Hz:(L)
BF = 4700 Hz:(R)

Time (msec)

Counts per bin

Figure 5. PSTHs (50-msec stimulus duration) of a neuron displaying excitation with monaural stimulation, ipsilateral (*A*) or contralateral (*C*), and inhibition with binaural stimulation (*B*) at 30 dB SPL. Glycine application during ipsilateral stimulation (*D*) mimics the binaural response. Subsequent application of strychnine blocked both binaural inhibition (*E*) and the combined effect of glycine application and binaural stimulation (*F*).

Ipsilateral Control A · Binaural Out of Phase Control B · Ipsilateral Gly + 125nA C

N=579 · N=175 · N=191

Ipsilateral Post Strych Gly + 125nA D · Binaural Out of Phase Strych + 100nA E · Binaural Out of Phase Recovery F

N=424 · N=465 · N=175

Time (msec)

R52-12
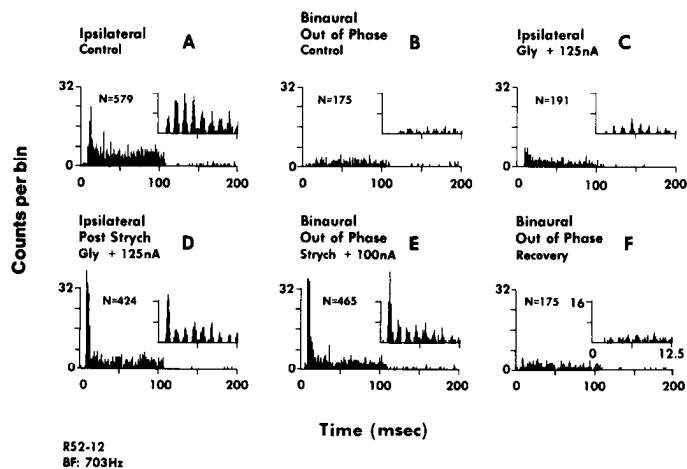BF: 703Hz

Counts per bin

Figure 6. PSTHs showing the responses of a neuron located in the lateral lobe of the LSO (100-msec stimulus duration). Interspike interval histograms with the *ordinate* as 32 intervals are seen in the *upper right corner* of the PSTHs. The monaural response (*A*) is inhibited by the binaural 180° out-of-phase stimulus (*B*). Glycine inhibits the response before strychnine application (*C*) which subsequently blocks the glycine inhibition (*F*) and the synaptic inhibition seen in the binaural condition (*E*).

arrangement of synaptic inputs is thought to be present in the LSO (I. R. Schwartz, personal communication). Evidence supporting the presence of excitatory and inhibitory endings impinging on LSO neurons is provided

by electrophysiological studies (Galambos et al., 1959; Boudreau and Tsuchitani, 1970; Guinan et al., 1972a, b; Tsuchitani, 1977) which indicate that many of these neurons show inhibition with binaural stimulation. Two previous studies describe 86% (222 of 259) of binaurally inhibited-ipsilaterally excited (IE) neurons were located in the LSO (Guinan et al., 1972a, b; Tsuchitani, 1977). The present study also found the IE response type to be the prevalent pattern located within the LSO. The localization of tracks to the LSO in the present study was simplified by the long passage through the medulla and the orientation parallel to the axis of the MSO in para-horizontal section. Only neurons from punctures that passed through a major portion of the LSO were included in the present study. The two detailed structure-function studies of the cat SOC (Guinan et al., 1972a, b; Tsuchitani, 1977) indicate that the two most proximate structures, the lateral trapezoid nucleus and the dorsolateral periolivary nucleus, show binaural inhibition in only 18% of the neurons studied.

The precision with which excitation and inhibition may be controlled by manipulating the acoustic input to SOC neurons provides an excellent substrate for the study of excitatory and inhibitory interactions. The present study finds that contralateral inhibition can be blocked with strychnine and mimicked by iontophoretic application of glycine. Light microscopic autoradiographic studies (Zarbin et al., 1981) show binding of [³H]strychnine in the LSO but not in the MSO. In addition, there appears to be a lateral to medial increase

in the number of binding sites found in the LSO (M. A. Zarbin, personal communication). Although the locus of action has not been defined, alterations of auditory sensation have also been reported by humans suffering from strychnine toxicity (Gosselin et al., 1976) or following the administration of subconvulsant doses (Clark, 1938). The inability of bicuculline to block GABA inhibition could be due to the presence of bicuculline-insensitive GABA receptors (Bowery et al., 1981). It is also possible that after several hours within the brain, bicuculline at the electrode tip could be degraded by mixing with cerebrospinal fluid, causing an increased pH and loss of stability (Olsen et al., 1975).

The presence of strychnine binding in the LSO and our findings that strychnine can block synaptically mediated binaural inhibition and that glycine can mimic synaptically mediated inhibition suggest that glycine may be a neurotransmitter mediating contralateral inhibition in the LSO (Fig. 1). Since the source of this inhibition is thought to be the fibers projecting from the medial nucleus of the trapezoid body onto neurons of the LSO of the same side, this suggests that the projection pathway utilizes glycine as a neurotransmitter.

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