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A NEURO-SYNAPTIC MODEL OF THE MASKING AND UNMASKING PROCESS IN THE BILATERAL AUDITORY SYSTEM

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Summary

A model of bilateral information processing in the auditory system was presented on the basis of the interaction of excitatory and inhibitory synaptic potentials intra- and internuclei in order to analyze the mechanism of binaural unmasking as well as monaural and central masking. The system was composed of a bilateral pair of auditory relay nuclei, i.e., the cochlear nuclei, sperior olives, trapezoid bodies, inferior colliculei, and medial geniculate bodies. These nuclei, except the medial geniculate were organized in pairs of bodies of afferent systems from the cochlear nucleus to the inferior colliculus, as well as an efferent system from the inferior colliculus to the cochlear nucleus. The medial geniculate body, which was regarded as a set of macro columns composed of projection columns, interacts the bilateral spectral information from the inferior colliculei. The model system could detect interaural differences using the interaction of excitatory and inhibitory postsynaptic potentials evoked on the lateral superior olive neurons (interaural difference detector). The equalization process for binaural information was explained as a mechanism of the threshold controls of analog-to-digital conversion neurons in the cochlear nucleus by the interaural difference detector. The binaural equalized inputs were converted to combined potentials of excitatory and inhibitory postsynaptic potentials for cancellation or summation in the medial geniculate body. The monaural and central masking emerged from the overall inhibition process within the macro column receiving the monaural and binaural spectral inputs. The binaural unmasking could be explained as a result of disinhibition in the macro column by the cancellation of binaural masker inputs. The forward and backward effects in monaural as well as central masking were simulated in the model to discuss the mechanism of transformations of nervous phasic discharges to tonic activity for the recognition of auditory signals in the medial geniculate body connected with the reticulo-thalamic system.

1. Introduction

In a previous paper[1], I presented a model of bilateral interaction in the auditory nervous system with the intention of analyzing the mechanism of binaural unmasking as well as image lateralization. The model could simulate both the processes of detecting the bunaural difference for image lateralization and of equalizing and cancelling binaural inputs. However, I could not explain the process of binaural unmasking, because the model had no ability to interact with auditory signals in the spectral domain for masking.

When a noise is added to a signal on one channel the threshold for signal detection increases (monaural simultaneous masking). The masking can be observed to occur even when applying a forwarded or following noise to the signal ('forward masking' and 'backward masking'). If the noise is added to the channel opposite the signal, the threshold shift decreases substantially ('central' masking'). When the signal and masker are presented to both ears, the masking effect decreases in some conditions ('binaural unmasking'). The greatest recovery of the threshold shift can be obtained in the case of the maximum discrepancy between the interaural phase differences of signal and noise.[2,3]

In the present paper, I would like to to improve on my previous model by adding a new stage for the transformation of phasic neural discharges to tonic activity in the medial geniculate body in order to explain the processes of binaural unmasking as well as of monaural and central maskings in the bilateral auditory nervous system connected with the reticulo-thalamic system.

2. The Model

The present model consists of bilateral groups of six relay nuclei, i.e., the anteroventral cochlear nucleus (AVN); the dorso lateral cochlear nucleus (DCN); the medial nucleus of the trapezoid body (NTB); the lateral superior olive (LSO); the inferior colliculus (ICC); and the medial geniculate body (MGB). The AVN, projected by the cochlear nerve, outputs to the ipsilateral LSO and contralateral NTB, which is connected to the LSO on the same side. The LSO sends output to both the ipsilatelal and contralateral ICCs.[5] The ICC receives direct input from the DCN,[6] which receives auditory input through the AVN on the same side. The ICCs, which project to the bilateral MGBs,[7] innervate the LSOs on both sides. The LSOs, then, innervate the DCNs on the respective sides.[8]

Post Synaptic potentials

The post synaptic potentials (PSPs) which are generated over a certain time course on the cell bodies of neurons by the synapses are are classified into two types of monopolar potentials, i.e., positive (excitatory) PSP (EPSP) and negative (inhibitory) PSP (IPSP).

In the present model, the time course for the EPSP ($V_E(t)$) or IPSP ($V_I(t)$) was simplified as follows,

$$V_E(t) = \begin{cases} \frac{1}{2}\left(1-\cos 2\,\frac{t}{2Tc}\right) & (0 \le t < Tc) \\ e^{-(t-Tc)/\tau} & (Tc \le t) \end{cases} \qquad (1)$$

$$V_I(t) = -V_E(t) \qquad (2)$$

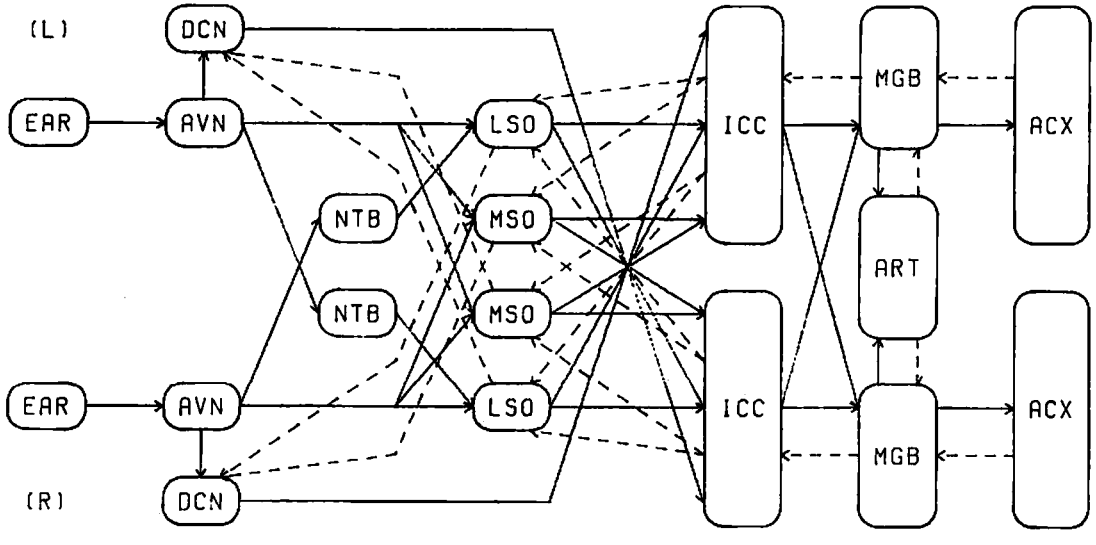

Fig.1 Blockdiagram of the bilateral auditory nervous system with afferent (solid lines) as well as efferent (broken lines) pathways. ACX:auditory cortex; ART:auditory reticularis thalami; AVN: anteroventral cochlear nucleus; DCN:dorsal cochlear nucleus; EAR: auditory input (via cochlear nerve); ICC:inferior colliculus; LSO:lateral superior olive; MGB:medial geniculate body; MSO:medial superior olive; NTB:medial nucleus of trapezoid body.

where Tc is the duration of the conductance change and is the discharging time constant after the completion of the conductance chage.

When the PSP are generated repetitively, the EPSP and IPSP are

$$V_E(t)=\begin{cases}\frac{1}{2}(1-\cos 2\frac{t}{2Tc}) & (0\leq t<Tc)\\ e^{-(t-Tc)/\tau}/(e^{-Tc/\tau}-e^{-T/\tau}) & (Tc\leq t<T)\end{cases} \quad (3)$$

$$V_E(t+T)=V_E(t) \qquad (T\leq t) \quad (4)$$

$$V_I(t)=-V_E(t) \quad (5)$$

where T is the time period of the repetitive stimuli.

The combined EPSP and IPSP potential is described by

where n_E and n_I are the respective excitatory and inhibitory syn-gains, and d_n is the delay in the IPSP from the EPSP.

In the model, the parameters n_E/n_I and d_n are, $n_I/n_E = 2$, $d_n = Tc/3$.[9)]

Performance of the Model System

The model system deals with binaural information in four processes, i.e., the detection of the binaural difference (D-process), the equalization of the binaural inputs (E-process), the cancellation of the nonsignal components in the equalized binaural inputs (C-process), and masking and unmasking (M-process).

These four processes are performed as follows.
1) Each AVN receives a train of pulses with a density corresponding to the acoustic wave of the input.
2) The AVN conducts a burst of pulses with a density proportional to the amplitude for the ipsilateral LSO as well as for the contralateral NTB, which act as an inverter to discharge a burst of inhibitory pulses in the LSO on the same side.
3) The two bursts from the ipsilateral and contralateral AVN (via the NTB) cause the EPSP and IPSP on the LSO neuron, respectively.
4) The LSO generates a train of pulses corresponding to the positive peak amplitude of the combined potential Of the EPSP and IPSP.
5) The ICC counts and compares the two pulse trains from the bilateral LSOs in reciprocal action to determine the laterality of the binaural image (D-process).
6) The ICC on the side of the image lateralized, or the side of the larger LSO output, sends a train of excitatory pulses to the ipsilateral LSO as well as a train of inhibitory pulses to the contralateral LSO.

7) The LSO conducts a train of inhibitory/excitatory efferent pulses in proportion to the LSO output with some compensation to the contralateral DCN, according to the excitatory/inhibitory LSO output.
8) The LSO inhibitory/excitatory efferent pulses cause a upward/downward shift in the thresholds of DCN neurons.
9) The DCN neurons, which receive a train of pulses corresponding to the acoustic wave of the input from the ipsilateral VCN, respond transitively to generate a sequence of bursts containing input intensity information as pulse density and phase information as the timing of the bursts in the sequence.
10) The sequences from the DCNs on the side of the lateralized image and on the opposite side can be, then, delayed and advanced in phase to equalize the neural timing of the binaural inputs (E-process).
11) The equalized sequence of pulse bursts from the DCN are sent to the contralateral ICC where the combined potentials of the EPSP and IPSP are respectively generated by each burst in the sequence.
12) Each MGB receives the two ICC combined potentials to superimpose them into one complex potential.
13) The MGB binaural neurons respond to the positive peak of the complex potential to send the inhibitory output laterally to relay cells in projection columns within a macrocolumn.
14) Since the combined potential of the EPSP and IPSP is bipolar, the complex potential decreases in terms of its positive peak as the phase difference between the combined potentials increases (C-process).
15) Then, the lateral inhibition by the binaural neurons decreases (disinhibition) when the C-process is active (binaural unmasking).
16) There are similar inhibitory interneurons within a macrocolumn but showing no binaural interaction because of the monaural innervation (M-process).

Laterality Index and Probability Fusion

The outputs of the bilateral LSOs as a pair of reciprocal peak detectors for the combined potentials of the EPSP and IPSP can be represented as follows,

$$L=\max(a_E.V_E(t)+a_I.V_I(t-d)) \quad (7)$$

$$R=\max(a_E.V_E(t-d)+a_I.V_I(t)) \quad (8)$$

where L and R are the left and right outputs; a_E and a_I are the intensity coefficients of the ipsilateral and contralateral inputs, respectively; and d is the interaural time delay.

The bilateral ICCs compare independently the two LSO outputs (L and R) to determine the laterality index, which is represented in terms of the normalized output of the ICC on the left or right side,

$$l=\begin{cases}\dfrac{L-R}{L_O+R_O} & (L\geq R)\\ 0 & (L<R)\end{cases} \tag{9}$$

$$r=\begin{cases}\dfrac{R-L}{R_O+L_O} & (R\geq L)\\ 0 & (R<L)\end{cases} \tag{10}$$

where L_O and R_O are the left and right LSO outputs for monaural presentation, respectively.

The probability fusion u is calculated as follows.

$$u=1-\frac{L+R}{L_O+R_O} \tag{11}$$

The Equalization Process

If the binaural inputs to the left ($S_L(t)$) and right ($S_R(t)$) ears are sinusoidal waves with the interaural phase difference d, the optimal threshold shifts for the left (H_L) and right (H_R) inputs are

$$d=\min|\sin^{-1}(H_L)|+\min|\sin^{-1}(H_R)| \tag{12}$$

where H_L is positive and H_R is negative.

In the model, H_L and H_R are determined as follows.

$$H_L=-R \quad (l\geq 0) \tag{13}$$

$$H_R=L \tag{14}$$

$$H_L=-H_L \quad (l<0) \tag{15}$$

$$H_R=-H_R \tag{16}$$

where u_f is the threshold of probability fusion which determines the region in the IPD domain for compensation. In the region where u becomes less than u_f, the downward threshold-crossing points is adopted for the side with larger threshold.

Masking

The MGB consists of a set of projection columns like functional columns in cortex,[10] groups of which form macrocolumns. These projection columns receive binaurally, their respective component of the corresponding characteristic frequency.

Each macrocolumn includes some intra-macrocolumnar (Golgi type II) cells which receive neighbouring components binaurally,[11] and inhibit laterally all of the relay cells in projection columns within macrocolumn.[12] In the model, each column is simplified to receive a sum of inhibitory inputs from all of the components except itself within the macrocolumn.

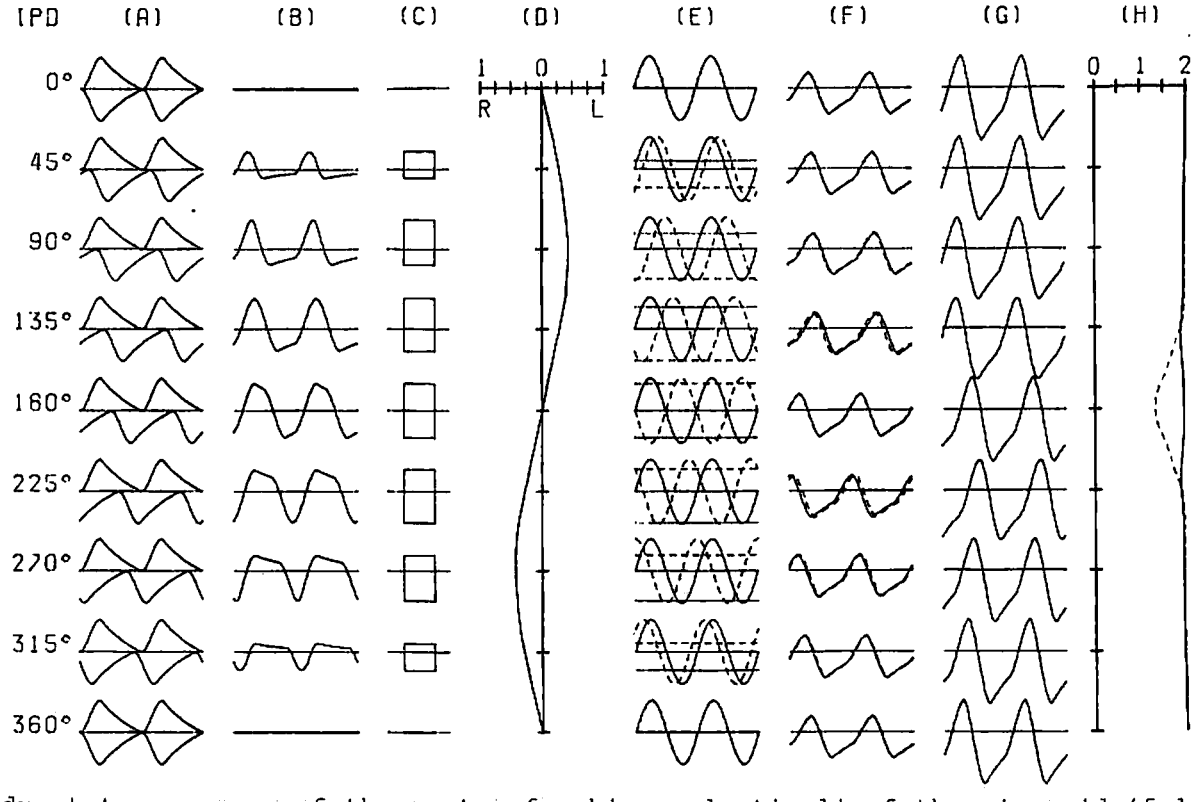

Fig.2 Steady-state response of the system for binaural stimuli of the sinusoid (f=1/T), bilaterally balanced in amplitude, in the case of the PSP charging duration Tc=T/3, the discharging time constant Γ=2Tc, the synaptic gain ratio n_I/n_E=2, and the neuron delay d_n=2Tc. IPD:interaural phase difference; (A):EPSP and IPSP evoked by the ipsilateral (left) and contralateral (right) (via NTB) AVNs; (B):superimposed potential of the EPSP and IPSP on LSO; (C):the left and right outputs corresponding to the positive (upward) and the negative (downward) peaks of the superimposed potential; (D):laterality index (ldev=1/4 of the image position of a monaural stimulus); (E):input sinusoids and the shifted threshold of the left (solid line) and right (broken line) ICCs, respectively; (F):two combined potentials on the left (solid line) and right (broken line) ICCs, respectively; (G):superimposed potential of two combined potentials; (H):positive peak of the superimposed potential with (solid line) and without (broken line) compensation of threshold-crossing.

Let the bilateral MGB inputs F_1 and F_2 be as follows.

$$F_i(t) = \sum_j a_{ij} . f(t) \qquad (i=1,2;\ j=1,N) \qquad (17)$$

Then, the sum of inhibitory inputs to one projection column are

$$I_i = \sum^{i} \sum^{j} a_{ij} . f(t) + \sum^{k} \sum^{i} a_{ij} . f(t) \quad (i=1,2; j=m_L, m_H; k=s_L, s_H) \quad (18)$$

where m_L/m_H and S_L/S_H are the lower/upper boundaries of binaural and monaural macrocolumns, respectively.

The excitatory and inhibitory nerve impulses cause EPSPs and IPSPs with longer periods than the neurons in the lower nuclei. However, the amplitudes of longer PSPs increase in proportion to the density of nerve impulses. Then, the phasic nerve discharges from the ICCs ca be transformed to a tonic activity in each column. In this model, the output of the column is assumed to correspond to the peak amplitude of the combined potential of such longer EPSPs and IPSPs.[13]

3. Results

Phase-Difference Detection and Equalization

Figure 2 shows the detectability of the interaural phase differences in binaural stimuli of the sinusoid (f=1/T) balanced bilaterally in terms of amplitude as well as in terms of ability of equalization-summation. In the figure, responses corresponding to two cycles of the sinusoid are illustrated for nine examples of the interaural phase difference (IPD). The positive and negative waves in (A) represent the EPSPs and IPSPs evoked by the output bursts of the ipsilateral (left) and contralateral (right)(via the NTB) VCNs. The upward and downward pointing bars in (C) denote the left and right LSO outputs corresponding to the positive and negative peaks of the superimposed potential of the EPSPs and IPSPs in (B), respectively. The curve in (D) indicates the change in the laterality index, l, as the IPD shifts from 0° to 360°. In (E) are shown the threshold shifts of the DCNs in proportion to the LSO outputs in (C) with compensation. The two waves represented by solid and broken lines in (F) indicate the combined potentials of EPSP and IPSP with delay in the ICC caused by the equalized sequences of bursts from the left and right DCNs, respectively. The two potentials were superimposed as in (G) and the positive peak is plotted in (H). The data represented by a broken line in (H) represents the data without compensation.

Phase-Difference Filter

Figure 3 shows the system responses when the threshold control on the DCNs was fixed at one of various IPD levels from 0° to 360°. Each curve indicates the characteristics of band-pass filters in the phase-difference domain.

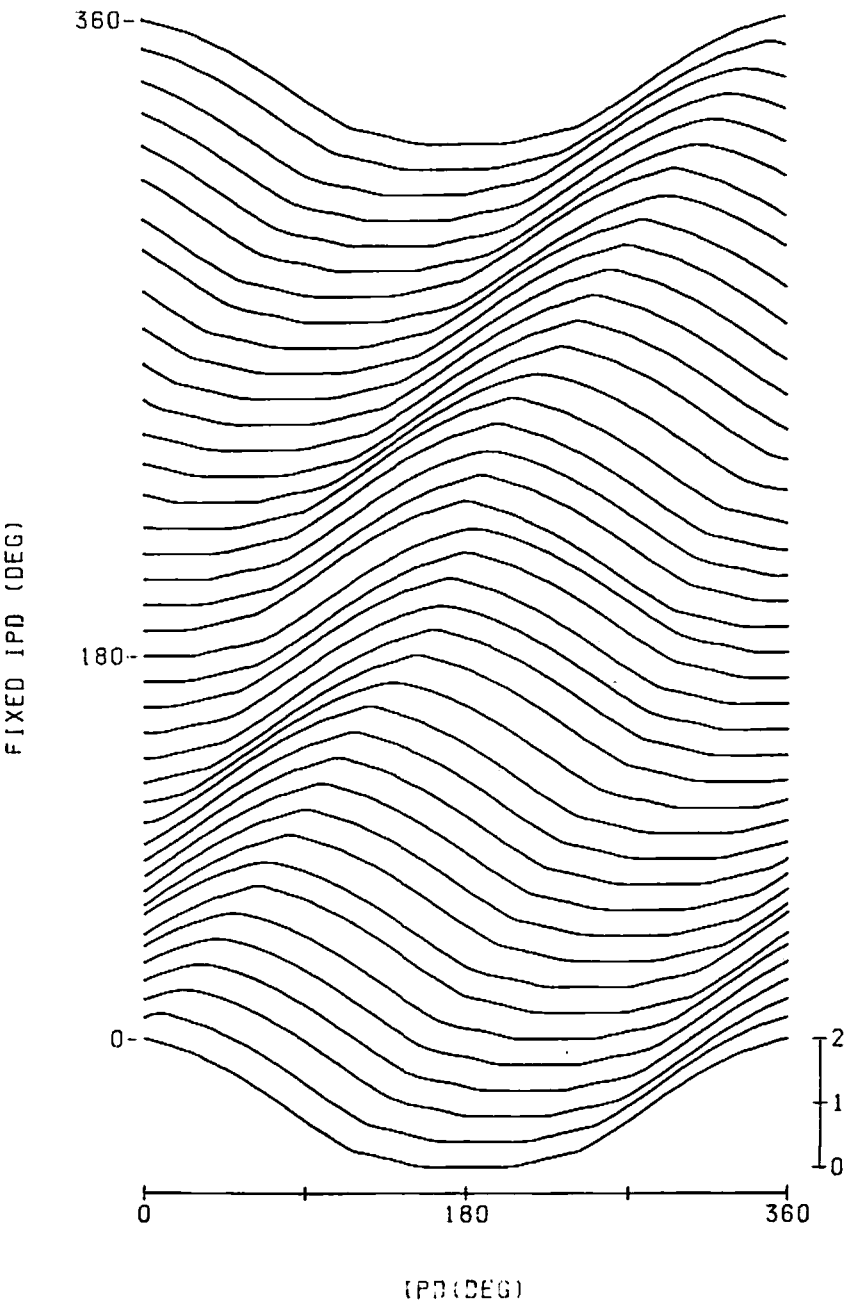

Fig.3 System responses as band-pass filters in the phase-difference domain when the threshold control on the DCNs was fixed at one of IPD levels from 0° (bottom) to 360° (top).

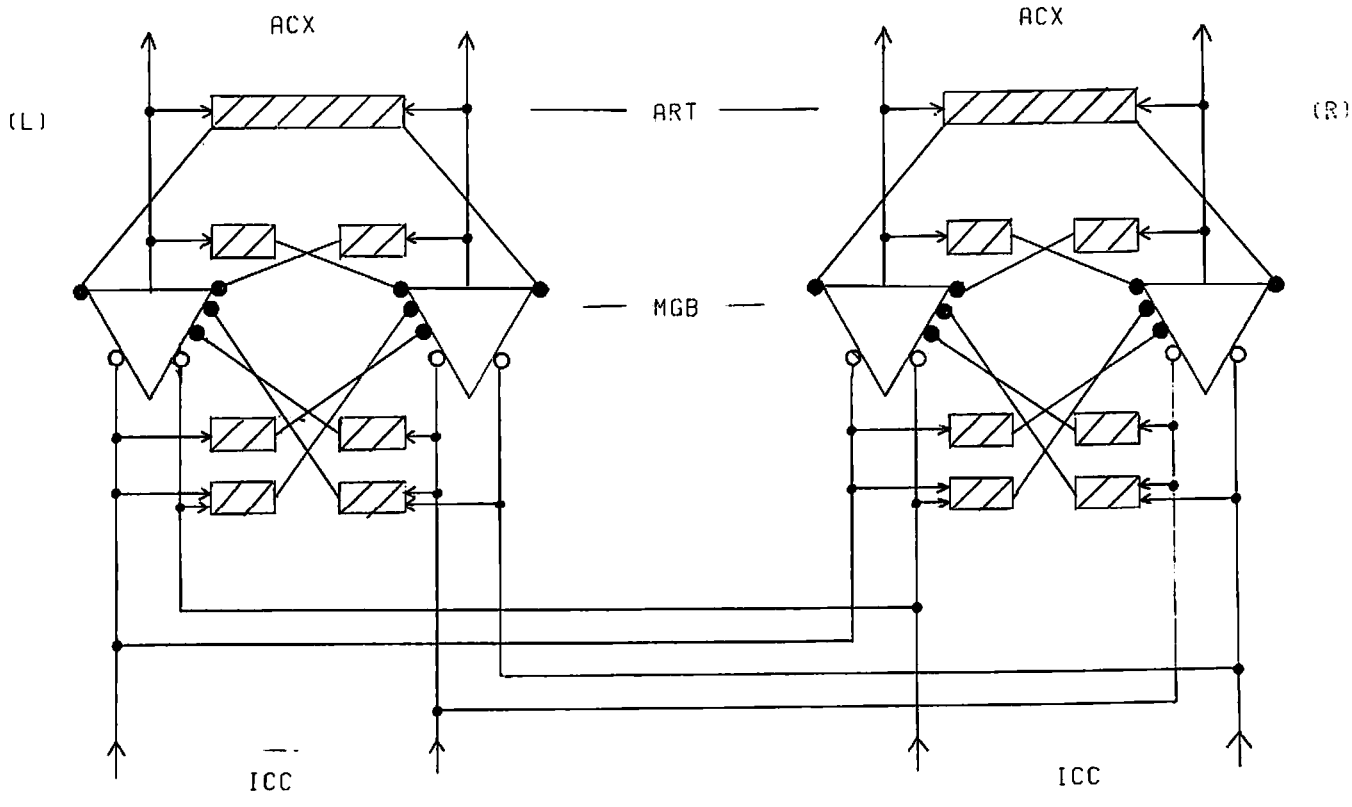

Fig.4 Schema of neural circuits in MGBs. ACX, ART, ICC, and MGB :see Fig.1; Triangle: excitatory relay cell; hatched rectangle: inhibitory interneuron; open circle:excitatory synapse; filled circle:inhibitory synapse; small filled circle:branch of nerve fiber. Two projection columns were illustrated in each MGB. The respective upper and lower interneurons inhibit the relay cells recurrently and feedforwardly.

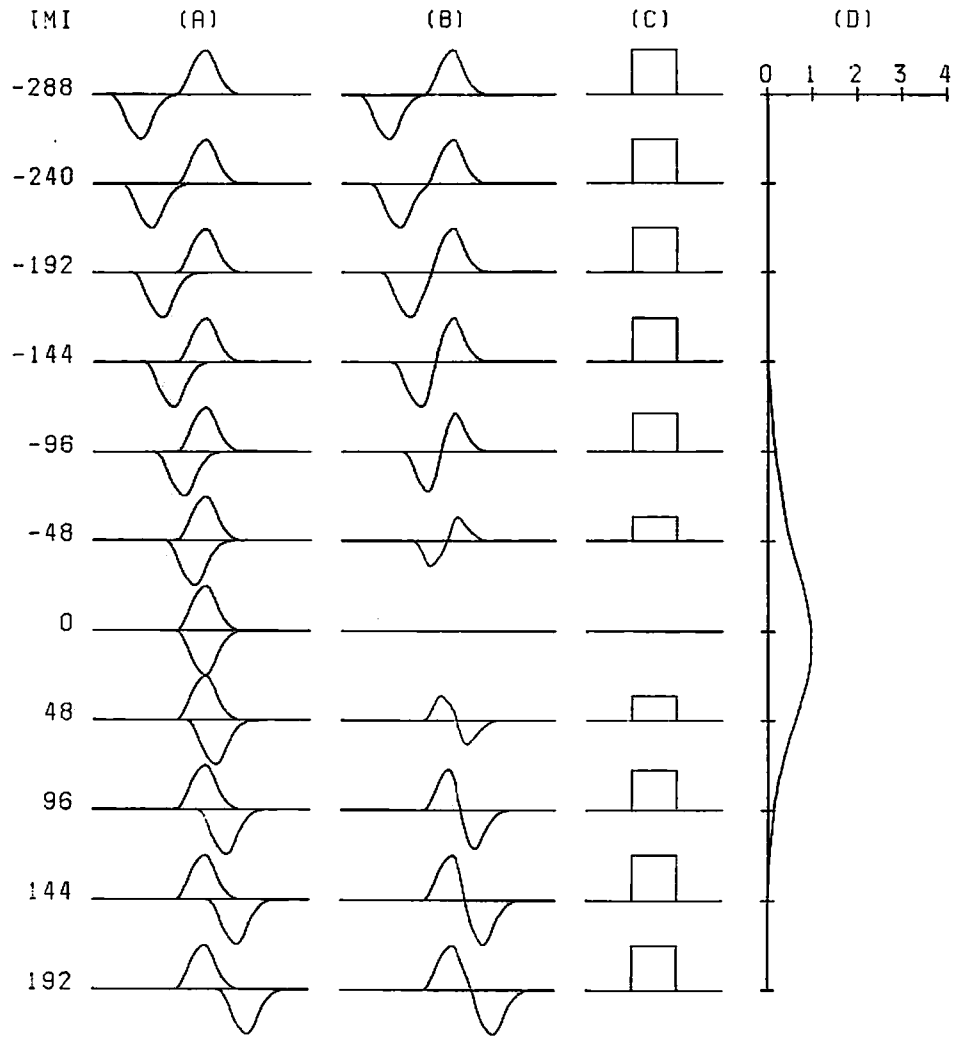

Fig.5(a) System response of the neuro-synaptic model of phasic to tonic transformation process in the MGB for stimulus of the signal in masker with various masker-to-signal intervals in the case of the PSP charging duration Tc=60ms, the discharging time constant τ=2Tc, the masker duration d_m=120ms, and the signal duration d_s=120ms. IMI:masker onset interval from the signal onset (positive IMI and negative IMI less than τ=-120ms correspond to backward masking and forward masking, respectively); (A):integrated signal EPSP (positive) and masker IPSP (negative) balanced in amplitude; (B):superimposed potential of the signal EPSP and the masker IPSP on the relay cells in the projection column receiving the signal spectral component; (C): positive peak value of the superimposed potential (the tonic output of the projection column of signal component in the MGB to the ACX); (D): signal increment necessary for recovering the output of signal component to the level with no masker interaction (ldev=the amplitude of signal with no increment).

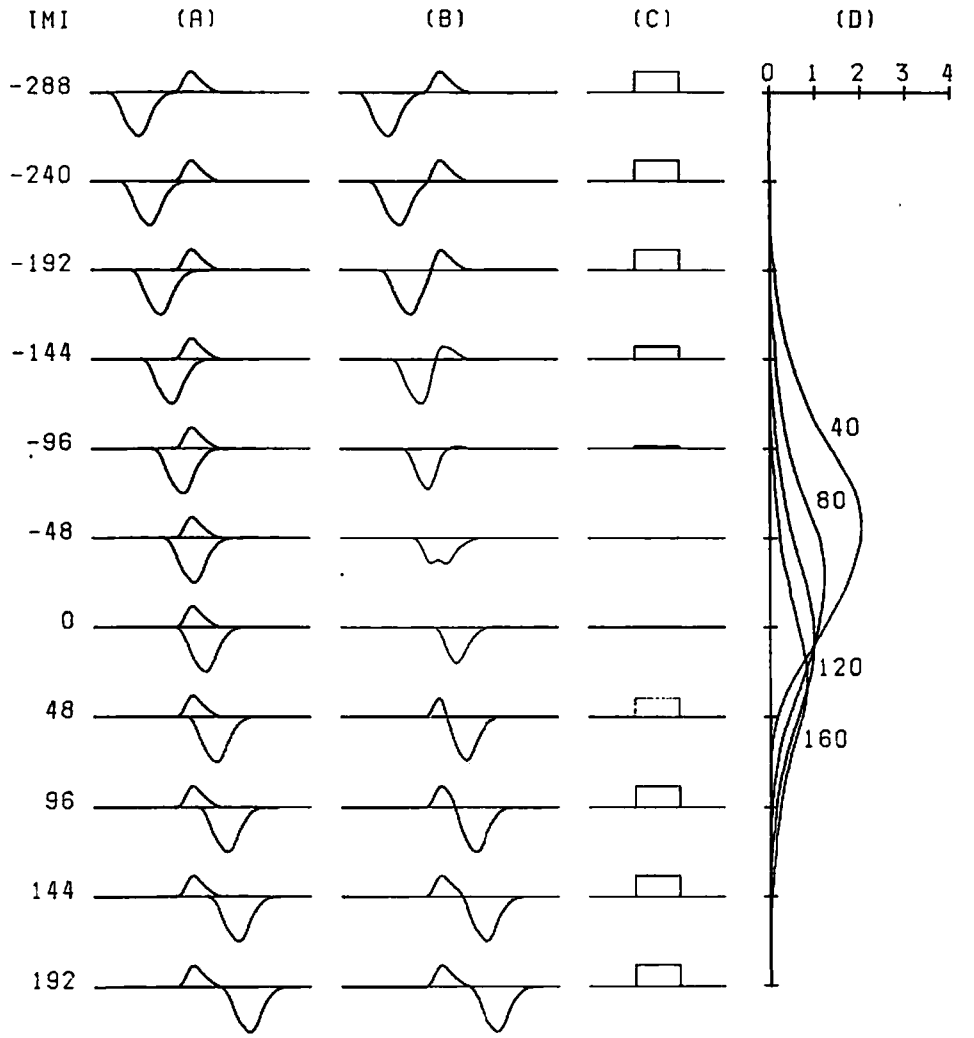

Fig.5(b) System response under the same parameters as in Fig.5(a) when the duration of signal was varied from 40 to 160ms, but the masker duration was fixed at 120ms. (A)-(C): response to the signal with the duration of 40ms; (D):four examples of signal increments for the signal duration d_S=40, 80,120,160ms (peak level of each integrated EPSP was not normalized).

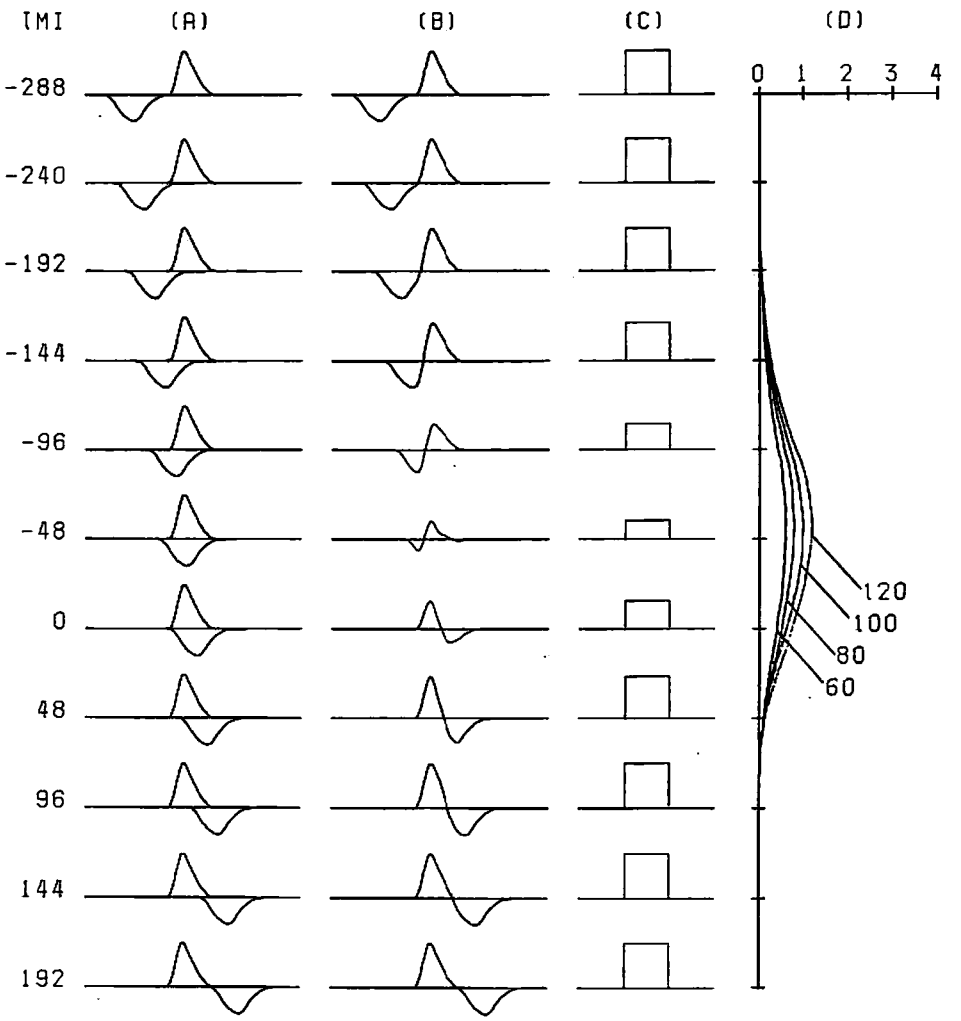

Fig.5(c) System response under the same parameters as in Fig.5(a) when the masker intensity was varied from 120 to 60% of the signal, but the signal and masker durations were fixed at 40ms and 120ms, respectively. (A)-(C): response to the signal with the 60% masker; (D): four examples of signal increments for the signal with 60, 80, 100, and 120% maskers. The results for the decreased maskers explain the MLDs in binaural unmasking process (disinhibition of binaural interneurons by the cancellation of masker component).

Masking and Unmasking Process

Figure 4 shows a schema of bilateral MGBs and the fundamental masking process. Figure 5 indicates an example of the simulated results for forward and backward masking.

4. Discussion

A model of bilateral information processing in the auditory nervous system was presented on the basis of the interaction of excitatory and inhibitory synaptic potentials intra- and internuclei in order to analyze the mechanism of binaural unmasking, as well as monaural and central masking. The model could simulate the process of masking, including forward and backward masking, using the interaction of the EPSP and IPSP in MGB.

In the present model, masking was explained as the lateral inhibition within a macrocolumn of feed-forward type. Gibson and Hirsh have presented two types psychoneural models of masking process, i.e., forward and recurrent inhibition.[14] Recently, Singer has proposed a neural circuit model of the lateral geniculate body which is reciprocally connected with the inhibitory nucleus reticularis thalami.[15] Such recurrent inhibition should be considered for higher masking processes[16-20] if the MGB is really a transformer of phasic input to tonic activity. The nonspecific thalamic nuclei such as the reticularis thalami are controlled by both the ascending reticular system as well as the centrifugal pathways.[21] It is necessary to analyze the mechanism of cortico-thalamo-reticular interactions for the processing of signals in three domains (Triplex theory), i.e., time, frequency, and lateralization.[22]

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