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TEMPORAL FACTORS IN DOMINANCY FOR SOUND IMAGE SPACE – A NEURAL MODEL OF BINAURAL INTERACTION –*

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Abstract

A neural model of binaural interaction in the lower auditory nervous system was presented on the basis of Bergeijk's model in order to analyze the mechanism of binaural information processing for sound localization. The model system could detect interaural phase differences using the interaction of excitatory and inhibitory post synaptic potentials evoked by the respective ipsilateral and contralateral outputs of the cochlear nuclei rather than Bergeijk's or, originally, Békésy's traveling-waves on phase detection cells. The effect of 'phase-intensity trade' as well as 'time-intensity trade' was simulated in the model to compare with the experimental results. Dominancy for binaural information processing was discussed and the possibility of inter- and intra-modal information processing involving temporal operations among post synaptic potentials from verious sensory inputs was suggested.

1. Introduction

The localization of a sound in space depends on the interaural acoustic difference. When a brief stimulus is presented equally both in terms of arrival time and intensity to both ears, the binaural image is located in the midline. If the interaural arrival time or intensity is made unbalanced, the image shifts toward the earlier or louder side. The shifted image can be recentered by adjusting other acoustic cues. This phenomenon is known as the "time-intensity trade." However, for sustained stimuli of low-frequency (lower than 1,500 Hz), the binaural image moves following the interaural phase difference rather than the absolute time delay.[14 15 30] The sensation of binaural beat, evoked by presenting sinusoidal stimuli with interaurally different frequencies, is due to this effect.[16]

In various relay nuclei, etc. of the mammalian auditory system, there have been found neurons sensitive to such interaural cues. Cassaday and Neff[3] observed the cat's inability at localization after lesion in one of the three binaural pathways in the brain-stem. They concluded that the superior olivery complex and trapezoid body are involved in the fundamental processing of binaural information, which is then projected to higher nuclei and the cortex. Anatomically, the two major masses in the SOC, i.e., the lateral superior olive, or S-segment, and the medial superior olive, or accesory olive, are recognized as the first relay nuclei from the bilateral cochlear nuclei.

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Based on these psychological, physiological, and anatomical data, numerous models have been proposed on the binaural system since Békésy first presented a hypothetical model of binaural interaction. In Békésy's model, a pair of excitation waves from both ears proceed through binaural-interaction neurons till the two waves meet. The location of the image follows the balance of the two spaces occupied by the waves. Van Bergeijk refined Békésy's model to explain binaural interaction for bilateral MSOs driven by inhibitory output of the ipsilateral CN and excitatory output of the contralateral CN. The balance of the two MSOs in terms of firing rate determines the location of the image. In this paper, Bergeijk's model[27] will be been examined at the synaptic level to observe the effect of 'phase-intensity trade' as well as 'time-intensity trade.'[22]

2. Model

The present model consists of bilateral groups of four relay nuclei, i.e., the cochlear nucleus (CN); the medial nucleus of the trapezoid body (MNTB); the lateral superior olive (LSO); and the inferior colliculus (IC). The CN, projected by the cochlear nerve, outputs to the ipsilateral LSO and contralateral MNTB, which is connected to the LSO on the same side. The LSO sends output to both ipsilateral and contralateral ICs. The system processes binaural information as follows.

(1) Each CN receives a train of pulses with density analogous to the acoustical wave of the input for low-frequencies (frequency-following) or to the rectified-integrated wave for high-frequencies.[7]
(2) Each neuron in CN responds transitively as the pulse density of the CN input passes over its own threshold.[24]
(3) A burst of pulses as a mass response of all the CN neurons is conducted to the ipsilateral LSO as well as to the contralateral MNTB, which acts as an inverter to discharge a burst of inhibitory pulses in the LSO on the same side.[12]
(4) These two types of pulses from the ipsilateral CN and from the contralateral CN (via the MNTB) cause excitatory post-synaptic potentials (epsp) and inhibitory post-synaptic potentials (ipsp), respectively.[10 26]
(5) The LSO generates a burst of pulses corresponding to the peak amplitude of the combined epsp and ipsp.
(6) The IC counts and compares the two bursts from the bilateral LSOs in reciprocal action like Bergeijk's model.

Cochlear Nucleus

The CN in the model is divided into separate groups with their proper characteristic frequencies. The number of neurons activated in each group depends on the input intensity of its frequency component.[21] Each neuron generates a pulse at the point where the component crosses over its threshold (= 0 in the model).

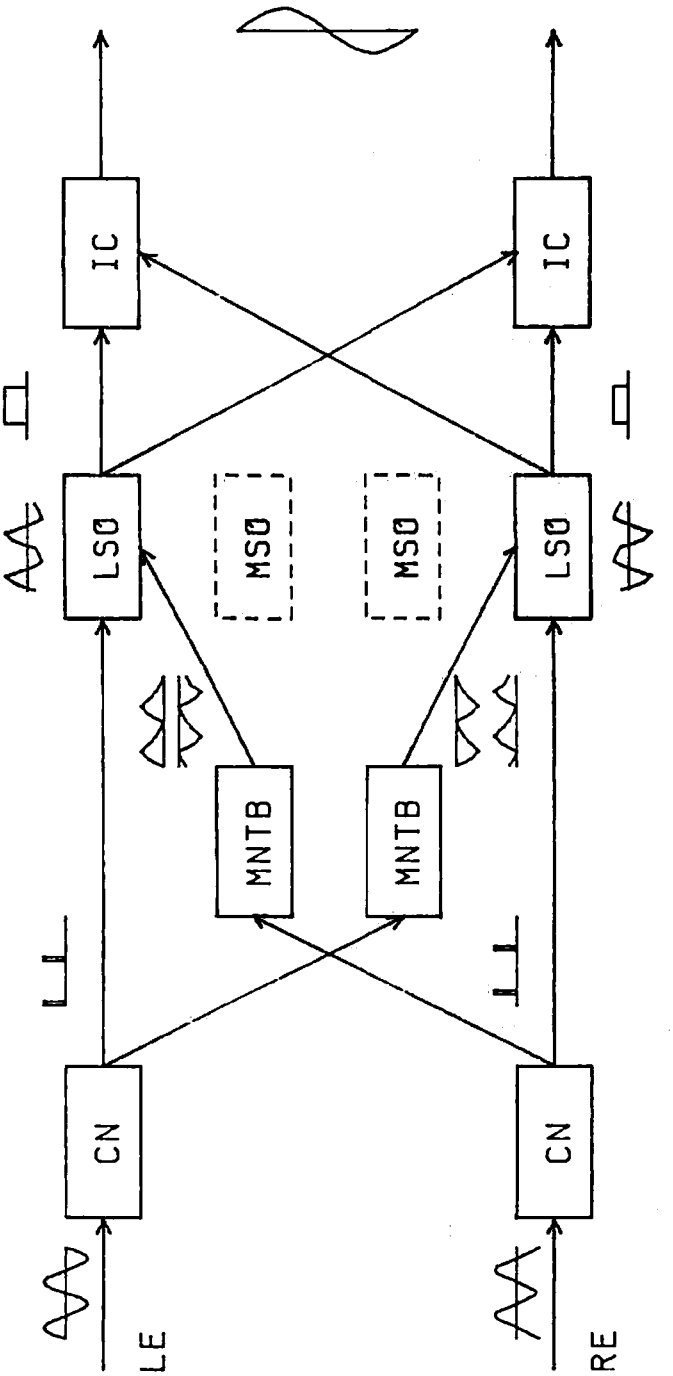

Fig. 1 *Block diagram of the binaural system. CN: coclear nucleus; MNTB: medial nucleus of trapezoid body; LSO: lateral superior olive; MSO: medial superior olive; IC: inferior colliculus.*

Let,

$$S_L(t) = \text{(input of left CN)} = \sum_{i=1}^{n} s_{Li}(t) = \sum_{i=1}^{n} A_L(t) * a_{Li} * \sin(2\pi f_i t)$$

$$S_R(t) = \text{(input of right CN)} = \sum_{i=1}^{n} s_{Ri}(t) = \sum_{i=1}^{n} A_R(t) * a_{Ri} * \sin(2\pi f_i (t-d))$$

$T_L(t)$ = (the set of outputs from the left CN groups)

$T_R(t)$ = (the set of outputs from the right CN groups)

$p(x)$ = (threshold function)

$= 1 \quad (0 < x)$

$\quad 0 \quad (x \leq 0)$

where n is the number of the frequency components ($f_1, \ldots, f_n$); a_{Li} and a_{Ri} are amplitudes of the i-th frequency component in the left and right CN inputs, respectively; d is the time delay of the right CN input compared to the left CN input; and A_L (t) and A_R (t) are the gate functions of input events, i.e.,

$$A_L(t) = \begin{cases} 0 & (t < 0) \\ 1 & (0 \leq t < t_E) \\ 0 & (t_E \leq t) \end{cases}$$

$$A_R(t) = \begin{cases} 0 & (t-d < 0) \\ 1 & (0 \leq t-d < t_E) \\ 0 & (t_E \leq t-d) \end{cases}$$

Then,

$$T_L(t) = (N(a_{Li})*p(s_{Li}(t))*(1-p(s_{Li}(t-dt)))_{i=1,n}$$

$$T_R(t) = (N(a_{Ri})*p(s_{Ri}(t))*(1-p(s_{Ri}(t-dt)))_{i=1,n}$$

where N is the number of activated neurons or the number of pulses in a output burst from a CN group at each crossing-point. If δ is a delta function

$$\delta(t) = \begin{cases} 1 & (t = 0) \\ 0 & (t \neq 0) \end{cases}$$

T_L (t) and T_R (t) are represented as follows

$$T_L(t) = \left(\sum_{j=0}^{m_i-1} N(a_{Li})*\delta(t-jT_i)\right)_{i=1,n}$$

$$T_R(t) = \left(\sum_{j=0}^{m_i-1} N(a_{Ri})*\delta(t-d-jT_i)\right)_{i=1,n}$$

where T_i (= 1/2 f_i) is the period of the i-th frequency component; and m_i is the number of zero-crossings of the i-th frequency component for the stimulus duration T_E.

Lateral Superior Olive

Two burst trains from the ipsilateral and contralateral (via the ipsilateral MNTB) CNs produce excitatory postsynaptic potentials (epsp) and inhibitory postsynaptic potentials (ipsp), respectively, on the membranes of LSO neurons.

Synaptic potential can be modelled by an equivalent circuit composed of resting membrane potential (E), resistance (1/G), and capacitance (C). A pulse of conducted action potential activates synapses to release quanta of transmitters which generate shunt conductance mg (g: a unit conductance of one quantum; m: the number of quanta transmitted by one pulse of action potential).[17] If a pulse causes a rectangular change in the shunt conductance,[17] the time course of the post synaptic potential (psp) elicited by a periodic train of pulses can be represented as the output of a relaxation oscillator.[13]

In the present model, the time course unit for epsp or ipsp is simplified as follows[28]

$$v_E(t) = \begin{cases} \sin 2\pi(T'/4)t & (0 \leq t < T') \\ 1 - \sin 2\pi((T-T')/4)t & (T' \leq t < T) \\ 0 & (t < 0,\ T < t) \end{cases}$$

$$v_I(t) = -v_E(t)$$

where $v_E(t)$ and $V_I(t)$ are units of epsp and ipsp, respectively; and T and T′ are total durations of the respective conductance changes.

If the synapses in each LSO group projected by a CN group with the proper characteristic frequency generate psp with the same parameters (t′ and T), and neurons in each LSO group have different thresholds within a rectangular distribution, the output of each LSO group can represent the peak amplitude of the combined epsp and ipsp.

Let,

$U_L(t)$ = (output of left LSO)

$U_R(t)$ = (output of right LSO)

$V_{Li}(t)$ = (membrane potential on neurons in the left LSO group)

$V_{Ri}(t)$ = (membrane potential on neurons in the right LSO group)

Then,

$$V_{Li}(t) = \sum_{j=0}^{m_i-1} (N(a_{Li})*v_E(t-jT_i) + N(a_{Ri})*v_I(t-d-jT_i))$$

$$V_{Ri}(t) = \sum_{j=0}^{m_i-1} (N(a_{Ri})*v_E(t-d-jT_i) + N(a_{Li})*v_I(t-jTi))$$

$$U_L(t) = (\sum_{k=1}^{l_{Li}} p(V_{Li}(t) - c_{Lik})*(1-p(V_{Li}(t-dt) - c_{Lik})))_{i=1,n}$$

$$U_R(t) = (\sum_{k=1}^{l_{Ri}} p(V_{Ri}(t) - C_{Rik})*(1-p(V_{Ri}(t-dt) - c_{Rik})))_{i=1,n}$$

where, l_{Li} and l_{Ri} are the numbers of neurons in the left and right i-th LSO groups, and c_{Lik} and c_{Rik} are the thresholds of k-th neurons in the left and right i-th LSO groups, respectively.

Inferior Colliculus

The bilateral ICs count and compare independently the two outputs from the left and right LSOs.

Let,

$I_L(t)$ = (the set of the number of pulses from the left LSO groups during a unit epoch e_i from t)

$= (E_{Li}(U_L(t))_{i=1,n}$

$I_R(t)$ = (the set of the number of pulses from the right LSO groups during a unit epoch e_i from t)

$= (E_{Ri}(U_R(t))_{i=1,n}$

$Y(t)$ = (laterality index of $I_L(t)$ and $I_R(t)$)

$$= \begin{cases} (I_L(t) - I_R(t))/(I_L(t) + I_R(t)) & (I_L(t)+I_R(t)\neq 0) \\ 0 & (I_L(t)+I_R(t)=0) \end{cases}$$

3. Simulation

Phase Detection in Steady State

If each pair of parameters in the bilateral system is the same, the left and the right systems work reciprocally. Figs. 2 (A), (B), and (C) show the change in the steady-state detectability of interaural phase differences in binaural stimuli of the sinusoid f(= 1/T) balanced bilaterally in amplitude when the psp parameter T′ is shifted from T/4 to T/2. The system failed in phase detection in the case of T′ = T/2 (Fig. 2 (C)). As can been seen in Fig. 2 (B), the result for T′ = T/3 agrees with Colburn and Durlach's experimental data,[6] which indicate that left- and right-most shifts in the binaural image occur at about 90° and 270° of the interaural phase difference, respectively.

Transient Response

Fig. 3 shows the transient (initial) response of the system with T′ = T/3 immediately after ths stimulus presentation. For more than 180° of the interaural phase difference, the sound image located at left initially even though the steady-state image located at right, as in Fig. 2 (B).

Single Response

Fig. 4 shows an example of the system response for a single event (one cycle of the sinusiod, isolated pulse, etc.). In this case, the interaural phase difference should be replaced by the intraural time difference as the binaural information.

Phase-Intensity Trade

Fig. 5 shows an example of the steady state response for binaural stimuli with an interaural amplitude difference ($a_{Ri} = 0.9 * a_{Li}$). The phase angle at the zero-crossing

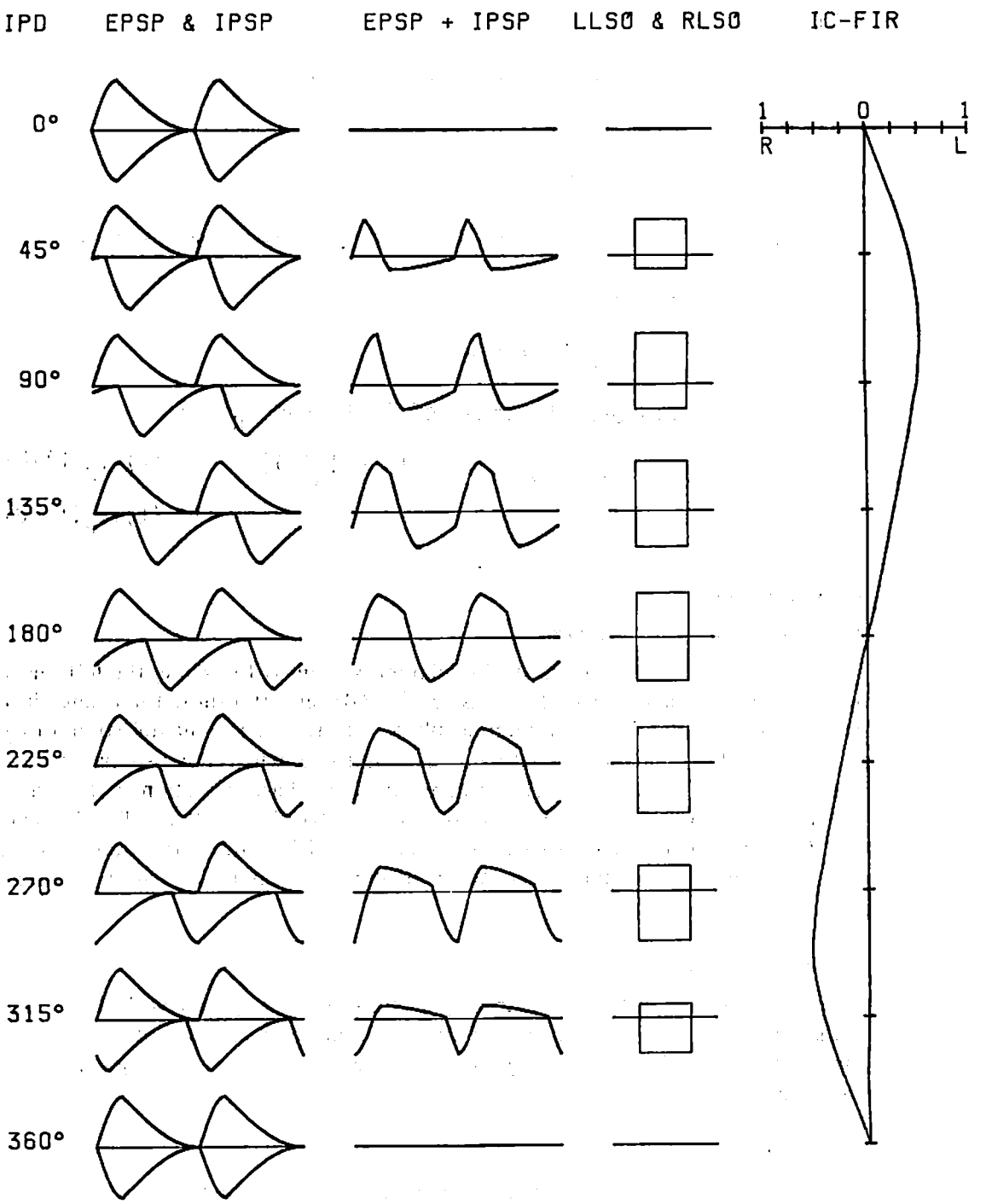

Fig. 2 (A) *Steady -state response of the system for binaural stimuli of the sinusoid of frequency f(= 1/T) bilaterally balanced in amplitude in the case of psp charging duration T' = T/4. IPD: interaural phase duration (360*d/T); EPSP: excitatory post synaptic potential (v_E (t)); IPSP: inhibitory post synaptic potential; EPSP + IPSP: combined potential of epsp and ipsp on LSO neurons (V_{Li} (t) = $-V_{Ri}$ (t)); LLSO: pulses from the left LSO (I_L (t)) (upward); RLSO: pulses from the right LSO (I_R (t)) (downward); IC-FIR: output of the inferior colliculus representing the laterality index (Y (t)). One division corresponds to 1/4 of the image position of a monaural stimulus in left/right ear.*

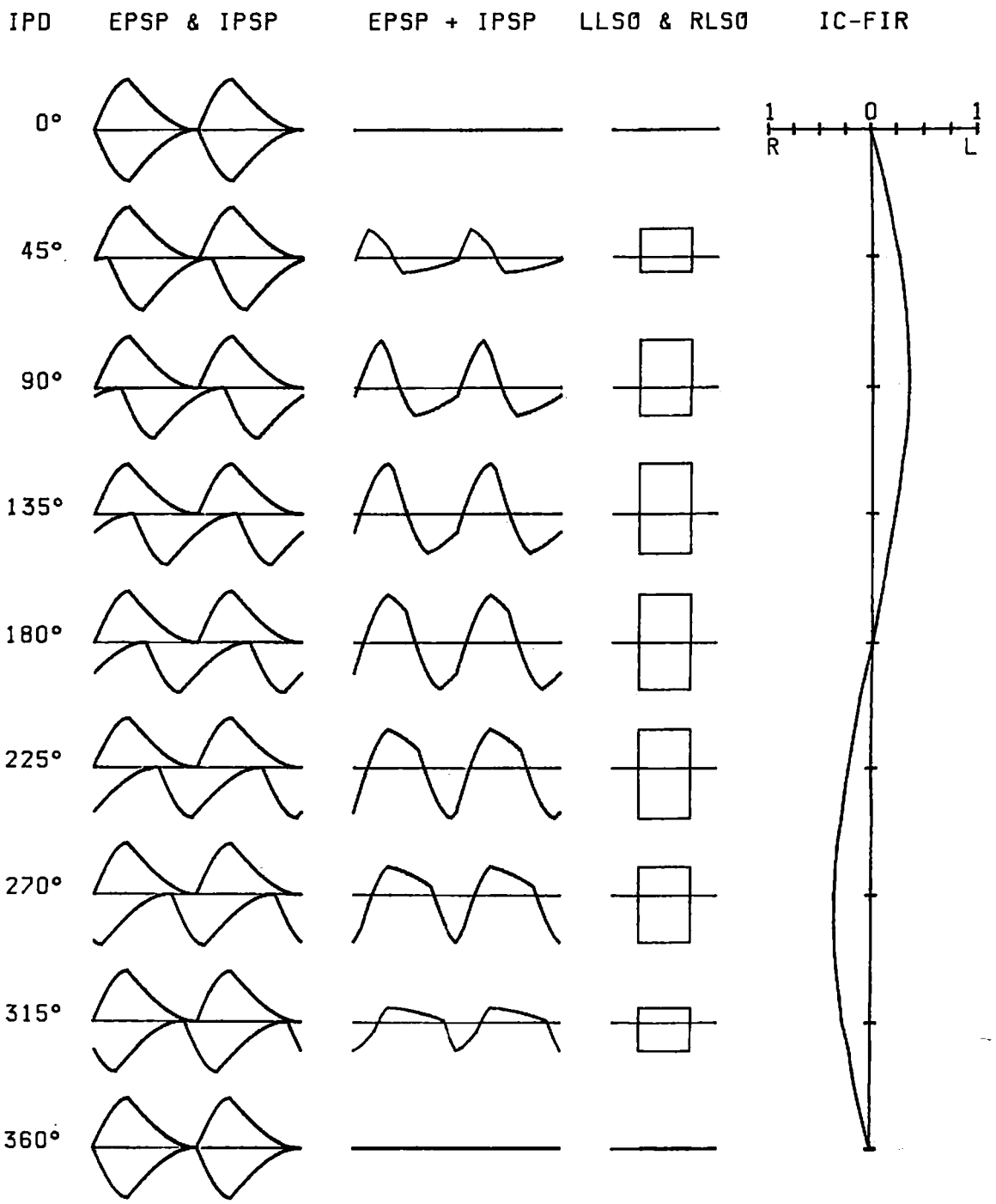
IPD
EPSP & IPSP
EPSP + IPSP
LLS0 & RLS0
IC-FIR
0°
45°
90°
135°
180°
225°
270°
315°
360°
1
0
1
R
L

Fig. 2 (B) $T' = T/3$.

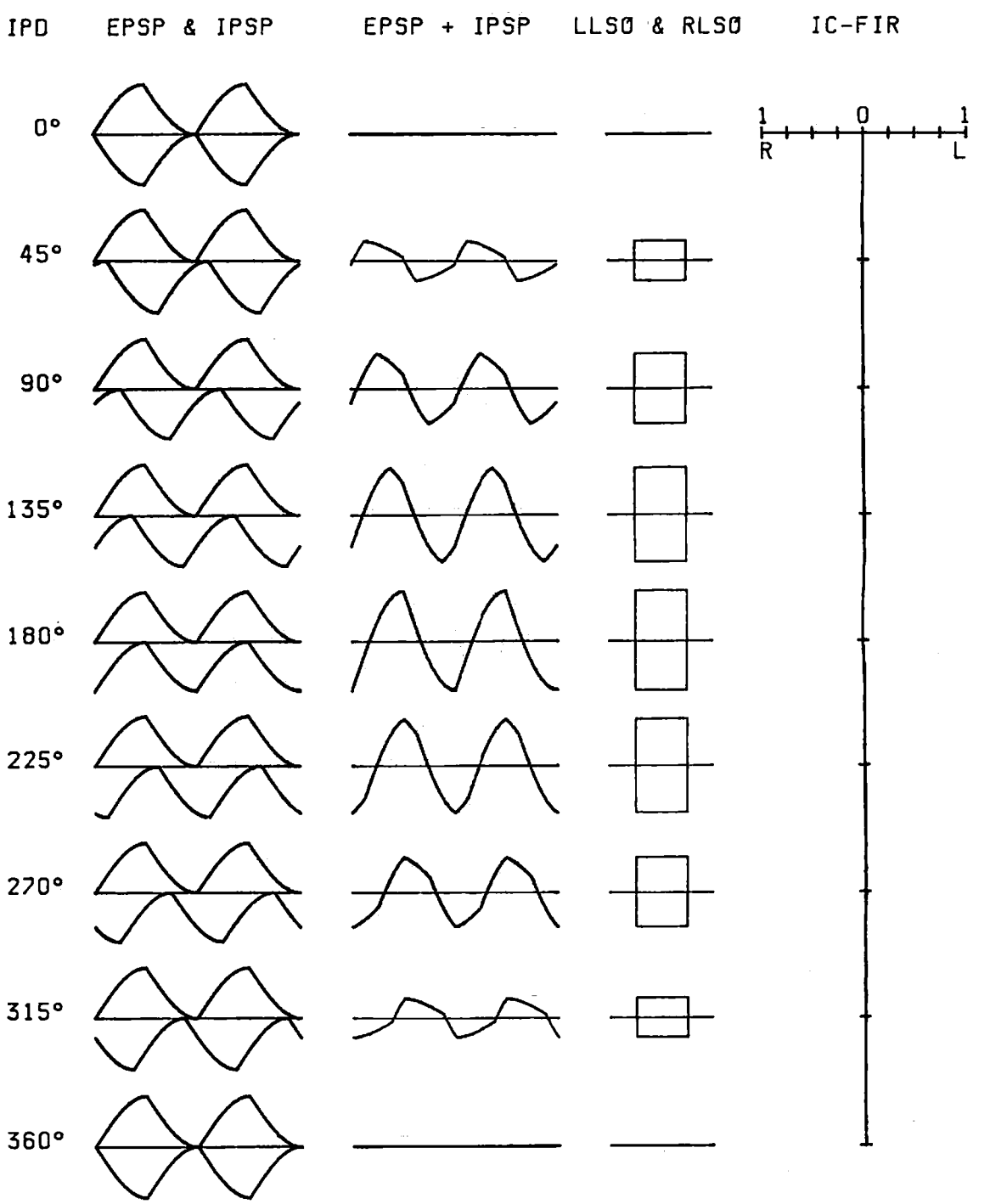
IPD
EPSP & IPSP
EPSP + IPSP
LLSO & RLSO
IC-FIR
0°
45°
90°
135°
180°
225°
270°
315°
360°
1
0
1
R
L

Fig. 2 (C) $T' = T/2$.

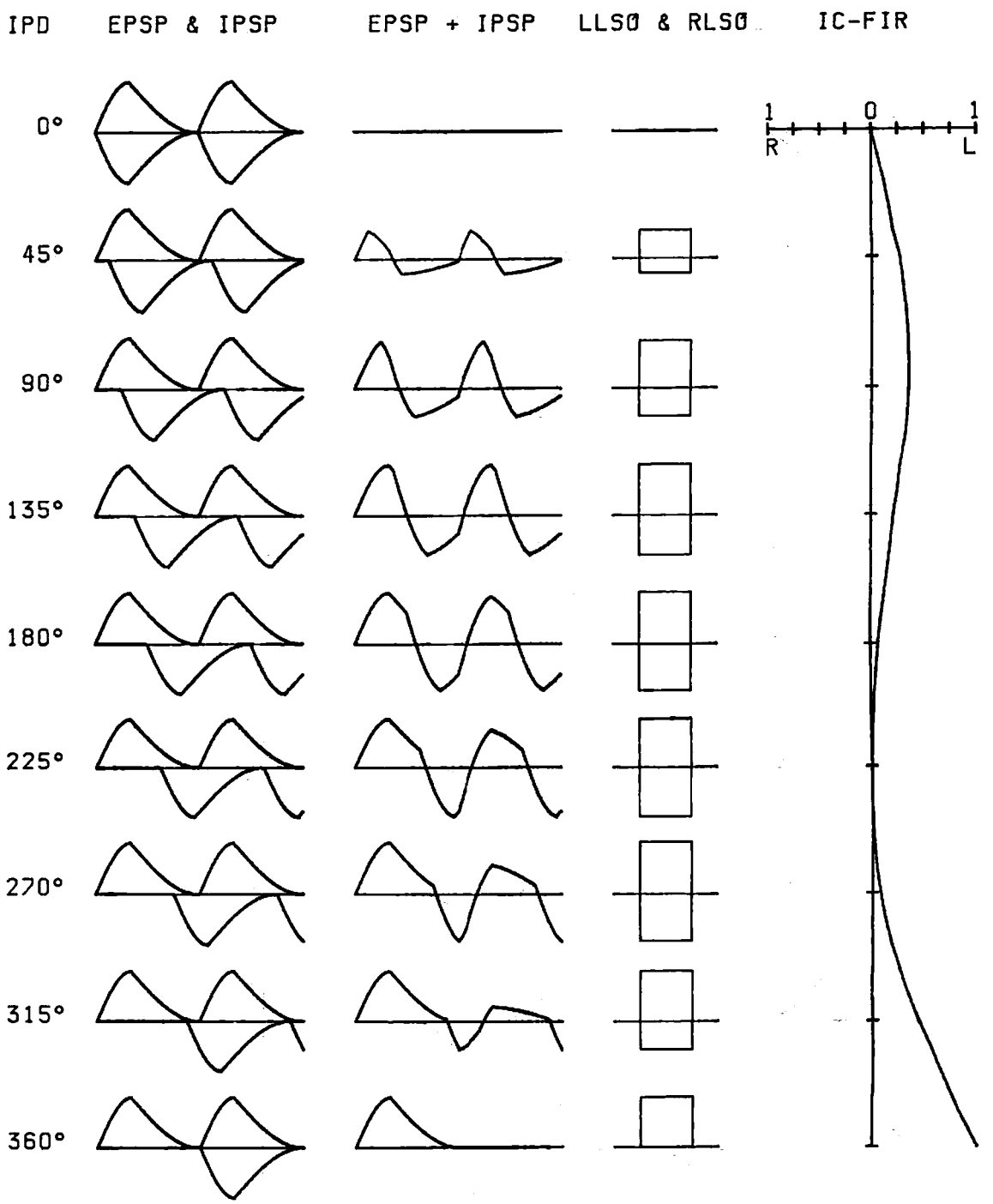

Fig. 3 *Transient response of the system with T' = T/3 immediately after stimulus presentation of a sustained sinusoid f(= 1/T).*

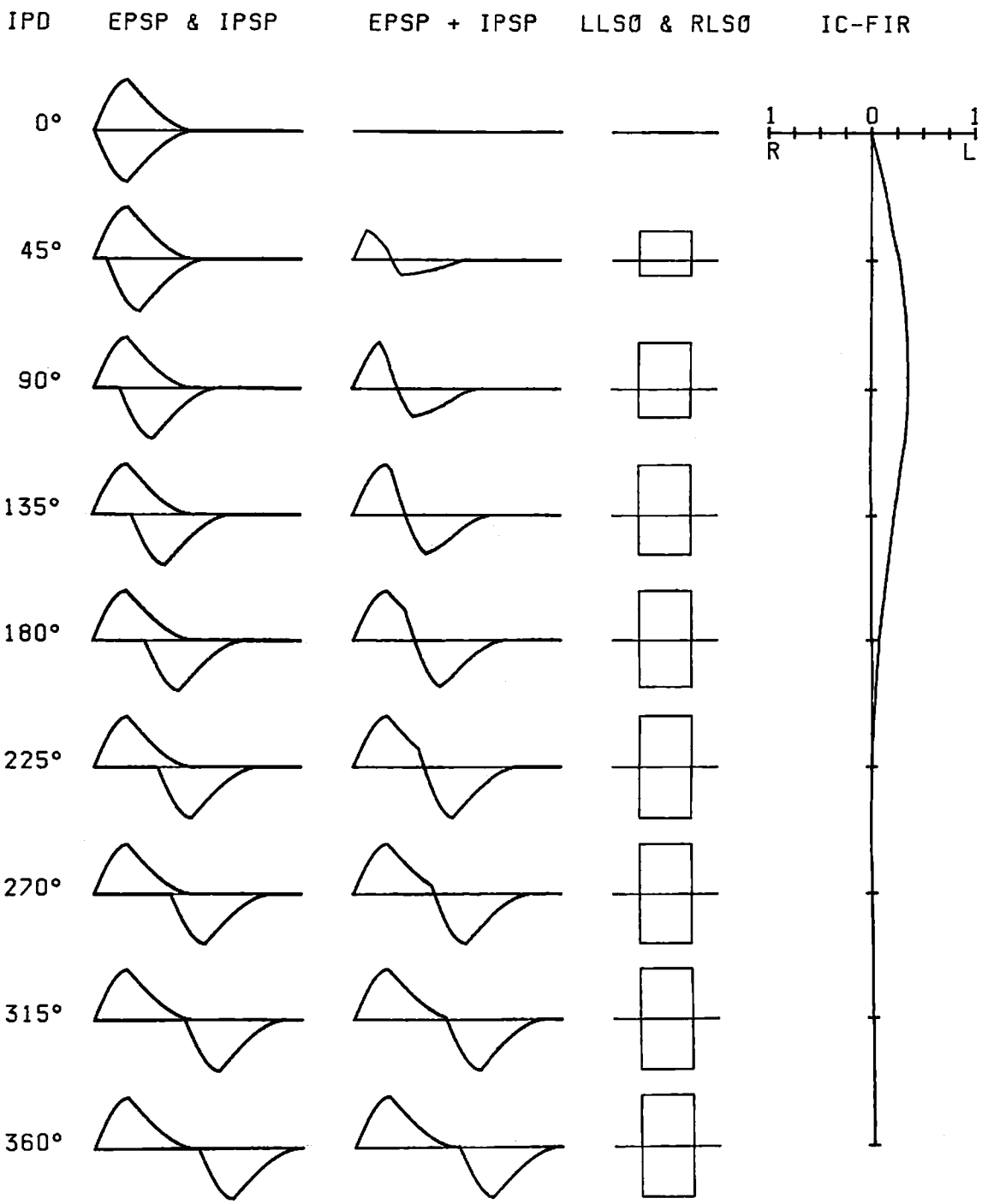

Fig. 4 *Response of the system with T' = T/3 for a single event (one cycle of the sinusoid, isolated pulse, etc.). IPD is the interaural time difference (ITD = IPD*T/360).*

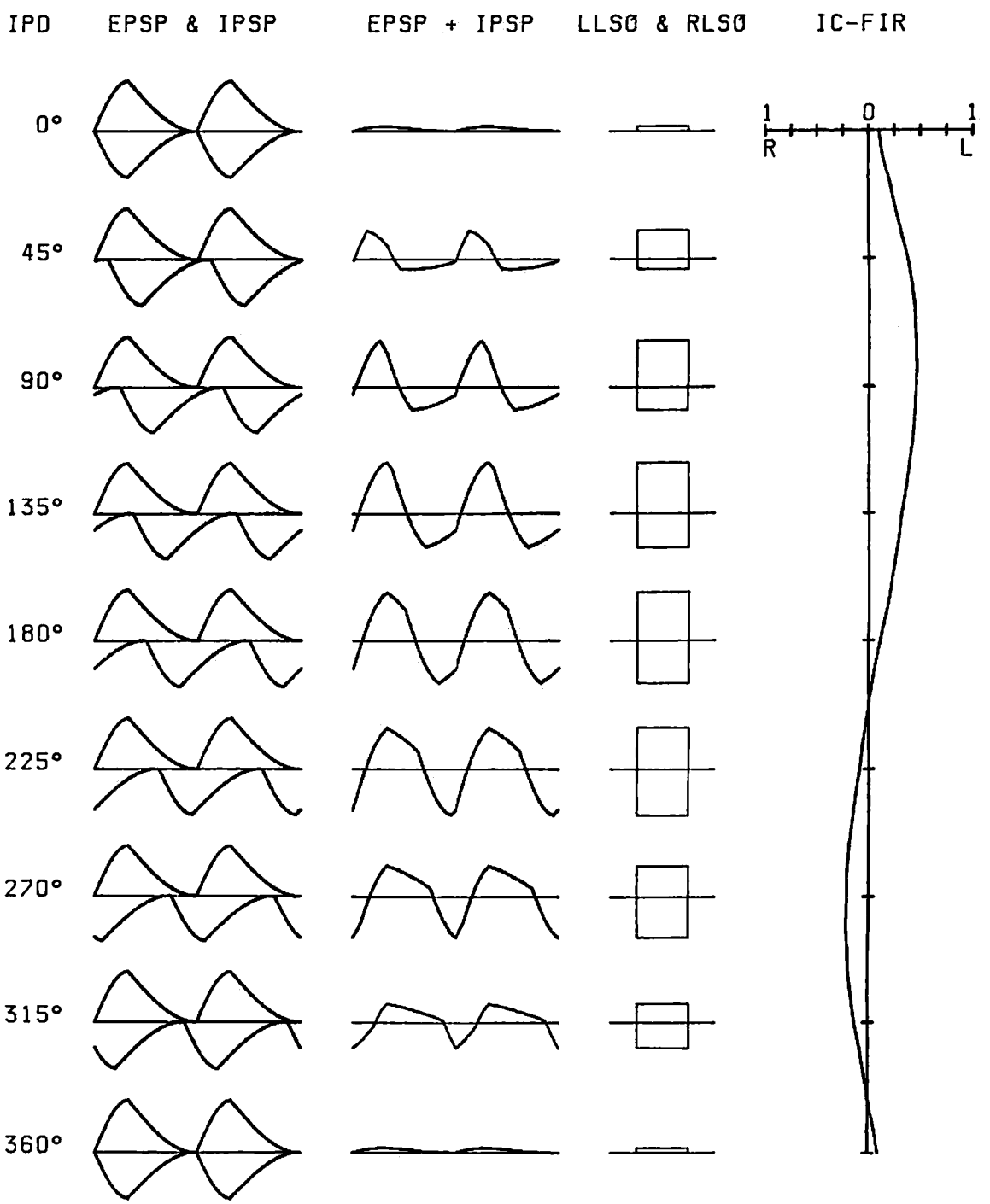

Fig. 5 *Steady-state response of the system with T' = T/3 for unbalanced binaural stimuli ($a_{Ri} = 0.9 * A_{Li}$).*

point of the IC output corresponds to the phase-advantage of the right ear necessary for centering the image of the binaural input with unbalanced amplitude, i.e.. the 'phase-amplitude trade' when the number of the CN output pulses increases lineary as the input amplitude is strengthened. The phase-intensity trade ratios (deg/dB) are plotted for the various input intensities in Fig. 6.

Dominancy for Sound Image Space

Fig. 7 shows an example of the simulated results for binaural stimuli balanced in amplitude from the model with a smaller (80%) right LSO output. The sound image space of the lateralization indicates a left space dominancy, though the phase advance is not necessary for centering the image at a zero interaural phase difference, unlike the case of an unbalanced input amplitude.

4. Discussion

The present model is based on the interaction of epsp and ipsp with unequal charging and discharging time constants. The simulated and experimental results for interaural phase detection almost agree for $T' = T/3$, which is identical with the ratio in Walsh and Tuckwell's psp model. However, it is necessary to keep the ratio for periodic stimuli with different frequencies. Two possible mechanisms for keeping the psp ratio constant can be considered: 1) The duration of the shunted capacitance decreases in the equivalent circuit of the psp as the repetitive frequency of the stimulus increases. 2) The output of a CN group with a characteristic frequency f, maps onto a corresponding LSO group, where a psp with the proper ratio is exclusively generated. Some evidence on LSO neurons in the location of arrays from high to low characteristic frequencies and in rather strict response patterns support the second mechanism, which gives the system an ability as a parallel analyzer of complex sounds.[19]

Bergeijk modelled the MSO as a system of binaural interaction with ipsilateral inhibitory and contralateral excitatory inputs. However, the MSO receives excitatory inputs both from the ipsilateral and contralateral CNs. The LSO is the nucleus accepting excitatory and inhibitory inputs. The present model consists of bilateral multistage processor of relay nuclei in the lower auditory system. If the MSO is also an analyzer of binaural information, the neural circuit for such a multistage processing system should be realized in the MSO itself with bilateral excitatory inputs.[29] The MSO is well organized into a two-dimensional array of fine columns which include marginal cells, peripheral cells, multipolar cells, and bipolar cells. The medial and lateral parts of each MSO column are projected from the contralateral and ipsilateral AVCNs (anteroventral cochlear nucleus), respectively. If some inhibitory cells in the columns can act as inverters of inputs, the MSO would be modelled as a neural circuit by the present system.

Recent anatomical and physiological data[25] suggest that the MSO plays a role in reflex movements of the eyes and head in response to the direction of sounds rather than binaural sound localization. There have been found synapse endings with small vesicles which do not come from the auditory system but possibly from the visual system.[20] If the MSO is the lowest system of audiovisual interaction,[18]

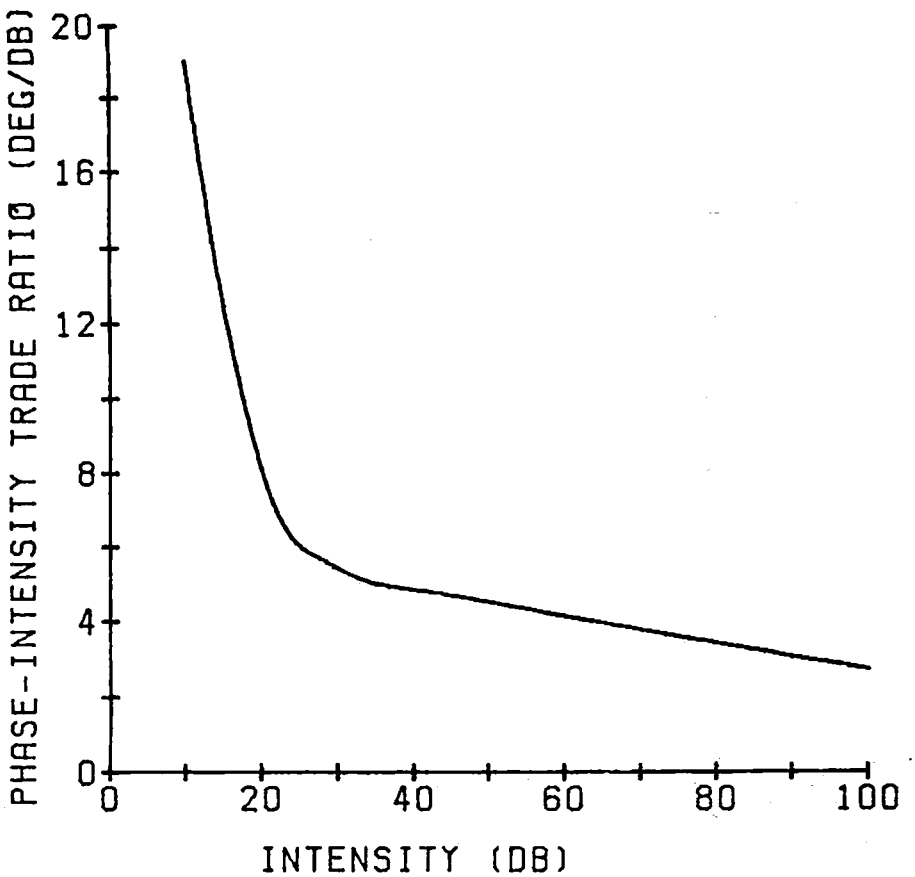

Fig. 6 *Phase intensity trade ratio estimated by this model at various stimulus intensities when CN output is assumed to increase linearly as the input intensity is strengthened.*

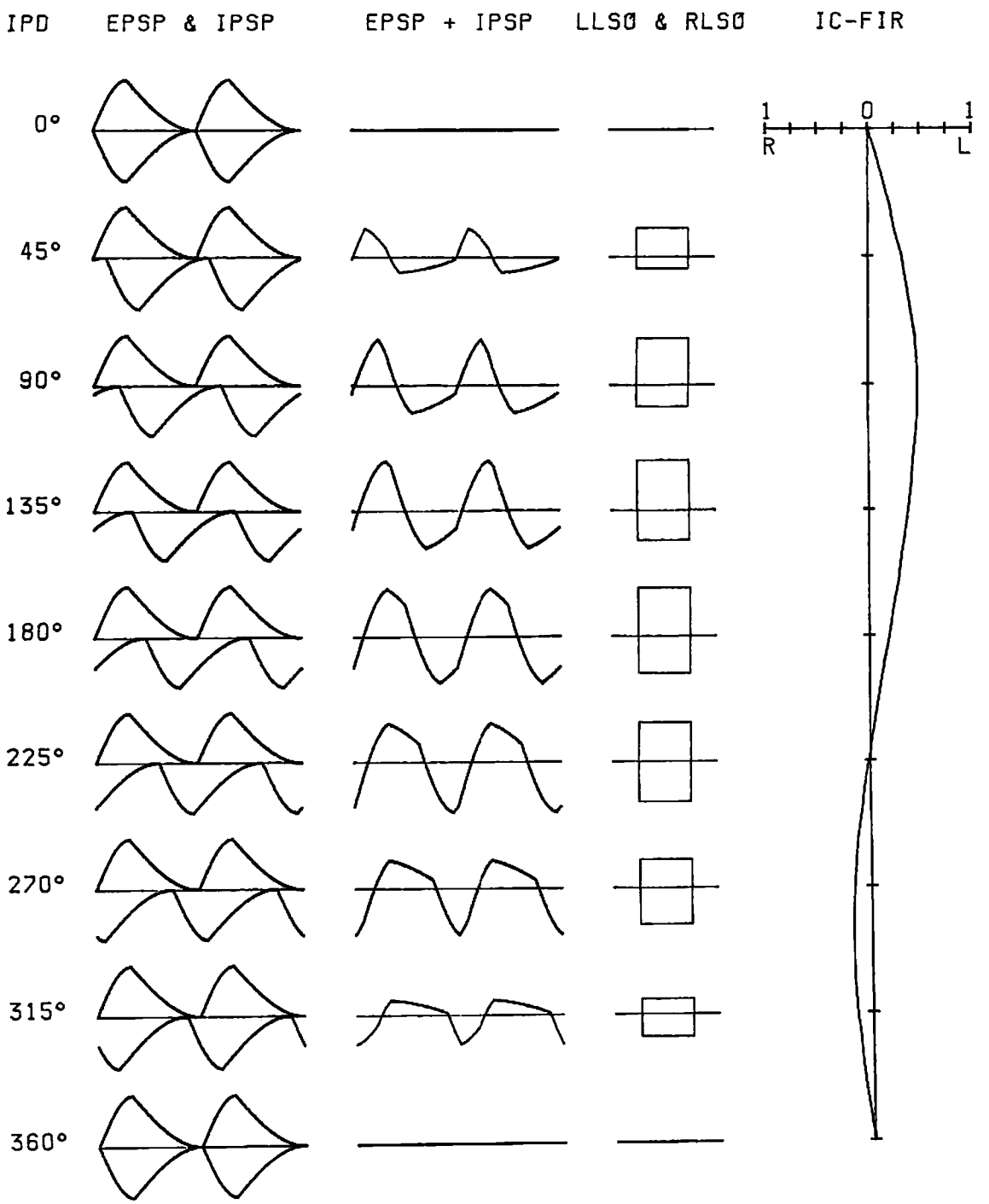

Fig. 7 *Steady-state response of the system with T' = T/3, but with unbalanced LSO outputs ($E_{Ri} = 0.8*E_{Li}$), showing left space dominance for sound image.*

further modelling of the MSO innervated by the visual system as well as the LSO innervated by the higher auditory system[8] is interesting for analyzing the mechanism of the interaction between sound localization and visual orientation[11] in the egocentric perceptual field[5 31] to explain the dominancy for binaural information processing.[2 9 23]

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References

1. Aitkin, L.M. & C.E. Kenyon (1981); The auditory brain stem of a marsupial. Brain Behav. Evol., 19, 126-143.
2. Bloom, F.E. (1981); Chemical signaling and cortical circuitry: integrative aspects. In Schmitt F.O., F.G. Worden, and S.G. Dennis (eds): The organization of the cerebral cortex. MIT Pr, Cambridge, 359-370.
3. Casseday, J.H. and W.D. Neff (1975): Auditory localization: role of auditory pathways in brain stem of the cat. J. Neurophysiol., 38, 842-858.
4. Colburn, H.S. (1973); Theory of binaural interaction based on auditory nerve data. I. General strategy and preliminary results on interaural discrimination. J. Acoust. Soc. Amer., 54, 1458-1470.
5. Cook, M. (1977); Gaze and mutual gaze in social encounters. Amer. Scientist, 65, 328-333.
6. Durlach, N.I. and H.S. Colburn (1978); Binaural phenomena. In Carterette, E.C. and M.P. Friedman (eds): Handbook of perception, IV, hearing. Academic Pr, New York, 365-466.
7. Evans, E.F. (1975); Cochlear nerve and cochlear nucleus. In Keidel, W.D. and W.D. Neff (eds): Handbook of sensory physiology, vol. V/2. Auditory system – physiology (CNS), behavioral studies, psychoacoustics. Springer, Berlin, 1-108.
8. Gibson, J.M. and H.R. Hirsch (1975); Psychoneural models of the auditory masking process. J. Theor. Biol., 51, 135-147.
9. Glick, S.D., D.A. Ross, and L.B. Hough (1982); Lateral asymmetry of neurotransmitters in human brain. Brain Res., 234, 53-63.
10. Goldberg, J.M. and P.B. Brown (1969); Response of binaural neurons of dog superior olivary complex to dichotic tonal stimuli: some physiological mechanisms of sound localization. J. Neurophysiol., 32, 613-636.
11. Hassler, R. (1978); Interaction of reticular activating system for visilance and the trancothalamic and pallidal systems for directing awareness and attention under striatal control. In Buser, P.A. and A. Rougeur-Buser (eds): Cerebral correlates of conscious experience. Elsevier, Amsterdam, 111-129.
12. Hirrison, J.M. (1978); The auditory system of the brain stem. In Naunton, R.F. and C. Fernandez (eds): Evoked electrical activity in the auditory nervous system. Academic Pr, 353-371.

13. Huang, C.-M. (1981); Time constants acoustic adaptation. Electroenceph. Clin. Neurophysiol., 52, 394-399.

14. Itoh, K. and S. Kikkawa (1981); Sound image space and dominance. Ann. Bull. RILP. 15. 55-61.

15. Jeffress, L.A. (1975); Localization of sound. In Keidel, W.D. and W.D. Neff (eds): Handbook of sensory physiology, vol. V/2. Auditory system – physiology (CNS), behavioral studies, psychoacoustics. Springer, Berlin, 449-453.

16. Kuwada, S., T.C. Yin, and R.E. Wickesberg (1979); Response of cat inferior colliculus neurons to binaural beat stimuli: possible mechanisms for sound localization. Science, 206, 586-588.

17. Martin, A.R. (1976); The effect of membrane capacitance on non-linear summation of synaptic potentials. J. Theor. Biol., 59, 179-187.

18. McCarley, R.W. and J.A. Hobson (1975); Neuronal excitability modulation over the sleep cycle: a structural and mathematical model. Science, 189, 58-60.

19. Mizuno, C., T. Ifukube, and C. Yoshimoto (1975); Analysis of tone perception system using sound image lateralization. I.E.C.E. Tech. Rep., MBE-75, 46.

20. Perkins, R.E. (1973); An electron microscopic study of synaptic organization. J. Com. Neurol., 148, 387-416.

21. Rose, J.E., L.M. Kitzes, M.M. Gibson, and J.E. Hind (1979); Observations on phase-sensitive neurons of anteroventral cochlear nucleus of the cat: nonlinearity of cochlear output. J. Neurophysiol., 37, 218-252.

22. Sayers, B.M.A. and P.A. Linn (1968); Interaural amplitude effects in binaural hearing. J. Acoust. Soc. Amer., 44, 974-978.

23. Scheibel, A.B. (1981); The problem of selective attention: a possible structural substrate. In Pompeiano, O. and C.A. Marsan (eds): Brain mechanisms and perceptual awareness. Ravan Pr, New York, 319-326.

24. Schroeder, M.R. (1975); Models of hearing. Proc. IEEE, 63, 1332-1350.

25. Schwartz, I.R. (1977); Dendritic arrangements in the cat medial superior olive. Neuroscience, 2, 81-101.

26. Tsuchitani, C. (1977); Functional organization of lateral cell groups of cat superior olivary complex. J. Neurophysiol., 40, 296-318.

27. van Bergeijk, W.A. (1962); Variation on a theme of Békésy: a model of binaural interaction. J. Acoust. Soc. Amer., 34, 1431-1437.

28. Walsh, J.B. and H.C. Tuckwell (1978); Repetitive subthreshold sysnaptic excitation and transmitter depletion. J. Theor. Biol., 70, 467-469.

29. Worden, F.G., J.T. Marsh, and F.J. Bremer (1966); Electrophysiological analog of the interaural time-intensity trade. J. Acoust. Soc. Amer., 39, 1086-1089.

30. Yost, W.A. (1981); Lateral position of sinusoids presented with interaural intensitive and temporal difference. J. Acoust. Soc. Amer., 70, 397-409.

31. Zihl, J. and D. von Cramon (1979); The contribution of the 'second' visual system to directed visual attention in man. Brain, 102, 835-856.