

16. In analyses of variance, there were significant main effects of drug groups in terms of total errors over the 12 problems (experiment 1, $F=19.02$; d.f. = 1,11; $P<.005$; experiment 2, $F=8.11$; d.f. = 2,21; $P<.01$) and mean trials to criterion (experiment 1, $F=13.71$; d.f. = 1,11; $P<.005$; experiment 2, $F=8.78$; d.f. = 2,21; $P<.01$). No significant interactions of drug with problems or with sex occurred. Subsequent tests in experiment 2

showed that both the groups fed TCAP and thiouracil made significantly ($P<.01$) more errors than the controls.

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Directional Hearing: Effect of Unilateral Change of the Sound Duration

Abstract. *Monaural change of the duration of a binaural acoustic stimulus within the range of 0.5 to 1.5 milliseconds caused a considerable shift of the sound image. This could be counterbalanced by a unilateral change of the signal intensity. Further unilateral lengthening of the stimulus did not affect the sound localization, but it revealed a masking aftereffect (4 to 10 milliseconds) of the binaural signal. The data define the temporal characteristics of binaural effect formation and the relative importance of duration and intensity parameters at different stages of binaural interaction.*

Intensity difference of the acoustic signal in the right and the left ear is one of the main factors determining sound localization. In this study I have attempted to substitute the relative sound duration for this factor.

Bursts of white noise of equal loudness (5 or 25 db above the auditory threshold) and equal initial duration (from 0.7 to 70 msec) were presented through earphones simultaneously to both ears of human subjects; in this way a single sound image was created, perceived on the midline of the head. After several presentations, the duration of the signal at one ear was increased or reduced by steps (Fig. 1), while at the other ear it remained unchanged. The subjects were instructed to report after each stimulation whether they perceived the sound strictly on the midline, or to the right, or to the left.

In 160 experiments on 12 subjects unilateral changes in stimulus duration within the range of 0.1 to 1.5 msec produced a significant (P from .001 to .02) shift of the sound image in the direction of the longer stimulus. This duration effect could be counterbalanced, that is, the sound could be brought back to the midline, by changing the intensity of the simultaneous stimulation of the other ear. The compensatory change in intensity (in decibels) served as a measure of the lateralization of the sound image (Fig. 1).

The extent of the lateralization did not depend on the initial loudness (5 or 25 db). The relative duration proves to be a very effective factor of the binaural interaction. It is much more effective than is relative intensity. Thus,

a unilateral reduction of duration from 1.0 to 0.6 msec, or by less than half, was counterbalanced by a change in the intensity of the contralateral stimulus by 20 db, or ten times, that is, by

a much greater amount of total energy.

However, the efficiency of the duration factor, that is, of the temporal summation, is confined to a limit of about 1.5 msec. A further unilateral lengthening of the stimulus does not affect the position of the sound image—the lines become parallel to the abscissa (Fig. 1).

For other auditory functions the temporal summation is much longer. Thus, the lowering of the auditory threshold and the augmentation of the loudness, caused by prolongation of the acoustic stimulus, do not cease until the duration of the stimulus exceeds 100 to 150 msec (1). But the binaural interaction is achieved, as it is seen, within 1.5 msec; the binaural effect is formed by this time, and later events cannot influence its formation.

It seems likely that the observed lateralization of sound is a manifestation of unilateral changes in auditory thresholds and, hence, in loudness which are brought about by monaural changes in sound duration. Indeed, the obtained lateralization values are very

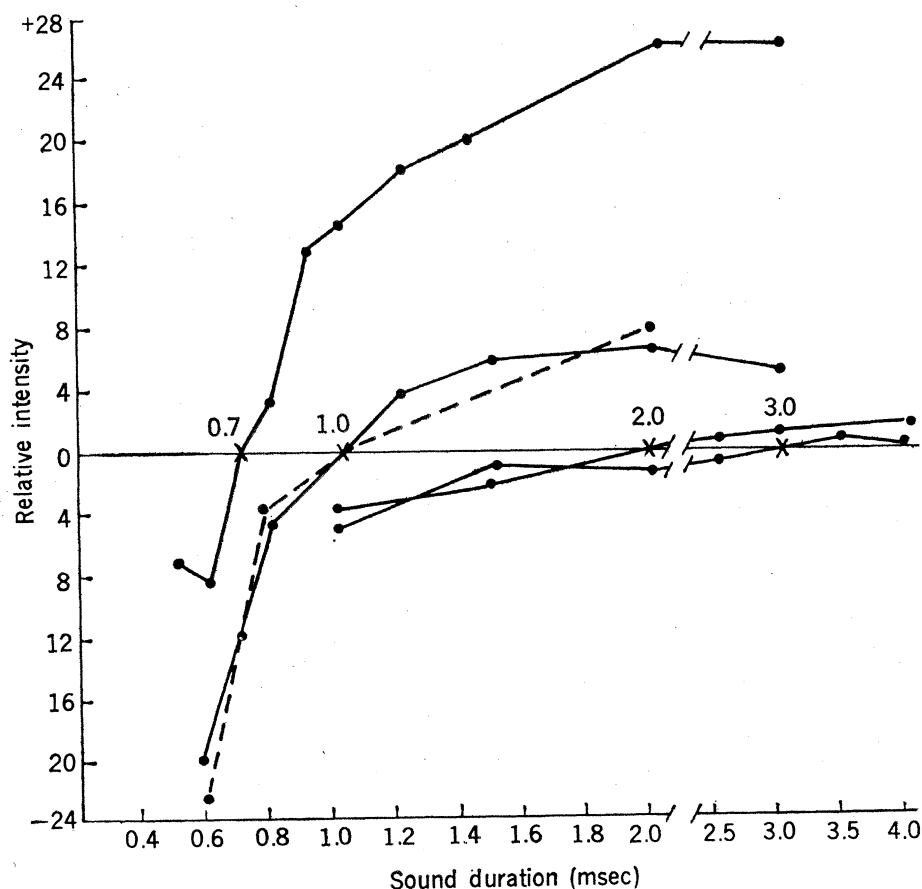


Fig. 1. Sound image shifts caused by unilateral changes of stimulus duration and compensated by an increase (+) or decrease (−) of the contralateral stimulus intensity. Crosses and figures on the base line represent initial sound durations. The dotted line represents auditory thresholds for sounds of different durations; the threshold for 1.0-msec stimulus is taken as a reference. Mean values.

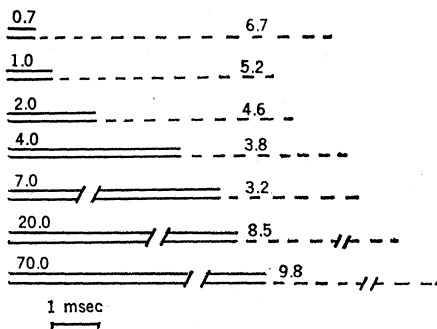


Fig. 2. Duration of the masking aftereffect (.....) exerted by the binaural stimulus (==) on its unilaterally prolonged part. Mean values.

similar to the monaural threshold shifts recorded in the same experiments as a function of stimulus duration and represented by the dotted line in Fig. 1 (mean values). On this curve the threshold for a sound of 1.0-msec duration is taken as a base line to allow a comparison with the lateralization curve for the same initial duration. Both curves coincide almost completely. If the threshold for a sound of some other duration were taken as a base line, then the dotted line would run close to the corresponding lateralization curve. Some discrepancies of the two parameters which arose in several individual experiments were probably due to a redistribution of monaural thresholds for the right and left ears caused by binaural interaction (2).

Changes of the thresholds dependent on sound duration are much more drastic for shorter signals than for longer ones. It has been shown by others (1), as well as in this study, that a shortening of auditory stimuli from 100 to 1.0 msec is accompanied by a rise of the auditory threshold by 24 to 26 db. A similar rise is produced by a very slight reduction of stimulus duration from 1.0 to 0.5 msec only (Fig. 1).

As stated above, a unilateral prolongation of the signal beyond the limit of about 1.5 msec had only a slight effect on the position of the sound image. But it brought to light a masking action of the binaural effect—the subject perceived a single fused sound and did not hear separately the subsequent monaurally prolonged part of the stimulus. The extent of this inhibitory aftereffect was similar to that found for the interaural successive masking (3); it amounted to about 4 to 10 msec (Fig. 2).

When one of the two stimuli was made still longer, its protracted part became free from the masking influ-

ence and was heard separately in addition to the binaural signal. But, despite the prevalence of its duration over that of the contralateral signal, it was only necessary to augment the intensity of the latter by 2 to 4 db to restore the masking of the longer stimulus. In other words, contrary to the relations recorded at the first stage of the experiment, it was not the relative duration of the stimulus, but its relative intensity which became specifically effective.

Thus, during the unilateral prolongation of the noise burst, three successive effects were recorded. At first the position of the sound image was greatly affected. In this phase the relative duration of the stimulus exerted a much stronger influence on the binaural interaction than its relative intensity. At a later stage the position of the sound

image did not change anymore, but the binaural signal had a masking aftereffect on the monaurally prolonged signal. Finally, the latter was heard separately from the preceding binaural stimulus. In this phase it was the relative intensity of the sound that mainly influenced the binaural interaction.

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Unilateral Inhibition of Sound-Induced Convulsions in Mice

Abstract. Loud sound repeatedly presented to one ear after an initial bilateral exposure produces a lateralized inhibition of convulsibility in SJL/J mice. Inhibition of the right side does not affect the convulsible state of the left side. Processes leading to inhibition and convulsibility may develop independently within the same animal.

Mice of many strains convulse to loud sounds. Some seize during the first exposure to noise; others convulse only to subsequent stimulation (1, 2). An assignable genetic defect underlies initial seizure susceptibility in one strain (3). Normally resistant mice may become convulsible within 30 to 36 hours after as little as a 5-second exposure to intense sound (2). The onset of convulsibility may be delayed if mice are stimulated repeatedly at 6- or 12-hour intervals after first exposure to sound (priming). This inhibition is absent or reduced when intervals are longer than 18 hours (2). The induced convulsible

state can be lateralized and may be limited to one half of the brain. Mice primed with one ear open later convulse only when stimulated through the ear open at priming (4). In this study I examined repeated unilateral stimulation following bilateral priming. The results show that inhibition to audiogenic seizures can be localized to the side of acoustic input, that the processes leading to convulsibility and inhibition may develop independently and coincidentally within the same animal, and that unilateral inhibition confers limited protection against convulsing in mice tested bilaterally.

Male mice of the highly inbred SJL/J strain aged 21 ± 3 days were obtained from the production branch of the Jackson Laboratory. Each mouse was primed by placing it in a test chamber and exposing it to 30 seconds of bell ringing at a sound level of 95 db above 0.0002 dyne/cm². In the first experiment 50 mice were bilaterally primed, and at 12-hour intervals thereafter each was reexposed to bell ringing for 30 seconds. In half the mice (group R-), the right ear was open and the left was flooded with glycerin at each exposure. In group L-, the left ear only was open. Immediately after each exposure the ear previously open was

Table 1. Proportions of audiogenic seizures in R and L mice, primed at 0 hr, according to the side of input receiving auditory stimulation. R, right ear open; L, left ear open; 0, both ears flooded with glycerin.

Postpriming stimulation 12, 24, and 36 hr	Test 48 hr	Proportion of clonic convulsions
<i>Experiment 1</i>		
R	R	2 of 11
R	L	12 of 15
L	R	10 of 12
L	L	0 of 12
<i>Experiment 2</i>		
0	R and L	26 of 27
R	R and L	13 of 16
L	R and L	10 of 16