positive cylindrical lens placed so that its axis is parallel to one set of the illusory crisscrossing lines eliminates those lines while the other lines remain.

A two-dimensional optical Fourier transform of the pincushion grid was produced and photographed (Fig. 1) with coherent, collimated light from a helium neon laser, which passed through a 35-mm slide of the pincushion grid and was then focused with a lens having a focal length of 228.6 cm. There are no diagonal components evident in the two-dimensional transform of the pincushion grid.

The illusion of lines seen in the grid may be related to the demonstration by Hubel and Wiesel that the visual cortices of the cat and the monkey have a predilection for straight lines (*3*). The points of the pincushion grid suggest a line to the visual cortex and the illusion of a line occurs. Consistent with this explanation are that the color of the line is determined by the color of the pincushion and that the illusion does not occur when the pincushion grid is out of focus.

The observations that the human visual system has greater visual resolving power for vertical and horizontal lines than diagonal ones (*4*) is consistent with the intensification of the illusion when the grid is rotated 45°. The human visual cortex, like that of the monkey (*5*), may contain more units maximally sensitive to vertical and horizontal contours.

The lack of diagonal components in the two-dimensional Fourier transform of the pincushion grid demonstrates that the relationship between visual perception and Fourier theory may be fortuitous, and prediction on the basis of Fourier analysis may be unwarranted.

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6. A special thanks to F. H. Beckman, chief scientist of the Franklin McLean Memorial Research Institute, University of Chicago, who produced and photographed the two-dimensional Fourier transform of the pincushion grid; J. Pokorny of the Departments of Ophthalmology and Psychology, University of Chicago, for his guidance, suggestions, and encouragement; and to the mason who tiled my bathroom floor.

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Eyeblink Inhibition by Monaural and Binaural Stimulation: One Ear is Better than Two

Abstract. *Airpuff-elicited eyeblink, like many other reflexes, may be inhibited when an auditory stimulus precedes the reflex-eliciting stimulus by approximately 100 milliseconds. This inhibition is greater when the auditory stimulus is delivered to one ear than when it is presented binaurally.*

A number of reflexes, the human eyeblink among them, may be inhibited if a relatively weak auditory stimulus precedes the reflex-eliciting stimulus by about 100 msec (*1*). We have been evaluating such reflex inhibition as an audiometric technique in which airpuff-elicited eyeblinks would be measured with and without antecedent auditory stimuli of various intensities and frequencies. The presence of inhibition should reflect integrity of the auditory system. This technique would be valuable in testing infants and others from whom voluntary responses cannot be reliably obtained. Since the effect does not depend upon an associative process, its assessment requires no active cooperation on the part of the subject. In our investigations, subjects viewed color slides of works of art throughout each session, and they typically came to ignore the test stimuli completely.

It became necessary to examine the inhibitory effect of monaural stimulation, since the audiologist usually wishes to test each ear separately. Therefore, we compared tone stimuli of equal loudness when presented to one or both ears. We also evaluated the effect of various left-right combinations of tone stimulus, blink-eliciting stimulus, and response transducer.

The 12 subjects in this study were students at Bryn Mawr College who had been screened for normal hearing. Each was fitted with TDH-39 earphones with MX-41/AR cushions. A tube was mounted on each side of the headset and adjusted to permit delivery of a mild (10 msec, 2100 newton/m^2) puff of air to the skin lateral to the eye. A modified d'Arsonval meter on each air-delivery tube was linked to the eyelid so that eyeblinks generated a voltage in the meter coil, which was amplified and measured with a digital voltmeter having storage capability (*2*). The duplication of the air-delivery and response-transducer systems permitted independent stimulation and recording from either side in order to verify the symmetry of the inhibitory effect.

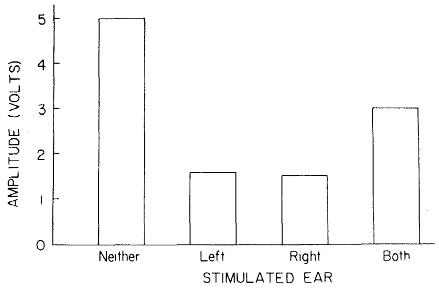

Fig. 1 Mean eyeblink amplitude elicited by an airpuff alone or one preceded by monaural or binaural stimulation.

At the beginning of each test session, the subject's hearing threshold for 1-khz tones was determined for each ear, then an alternating binaural loudness balance test was performed (*3*). On the basis of these measures, the tone stimulus intensities were set at a 70-db sensation level for the more sensitive ear and at an equal loudness for the other ear. The subject then viewed a sequence of color slides during which six trials were presented under each of four conditions: airpuff preceded by a 1-khz tone stimulus (i) to the left ear, (ii) to the right ear, (iii) to both ears in phase, or (iv) airpuff alone. In those stimulus configurations that involved an acoustic signal, the tone duration was 20 msec; its rise and decay times were 2.5 msec, and the interval between onset of the tone and the airpuff was 100 msec.

Trials were approximately 30 seconds apart, and slide changes occurred in the intervals between trials. For half of the session the airpuff was delivered to the left temple and for the other half, to the right temple. Also within each session, half of the responses were measured at each eye. All these manipulations were varied according to an orthogonal design so that all left-right combinations occurred equally often within each stimulus condition.

The eyeblink response was symmetrical; the amplitude was essentially the same whether recorded ipsilaterally or contralaterally to the airpuff stimulus. Likewise, prior monaural auditory stimulation inhibited the response of both eyes equally. Because of this symmetry, the results for the various conditions have been pooled (Fig. 1). Binaural tone stimulation, as expected, reduced the blink response relative to the silent condition, but more inhibition was engendered by monaural tone stimulation of either ear. A repeated-measures analysis of variance (*4*) showed the tone stimulus manipulations to have a significant effect ($F = 36.08$; d.f. = 3, 33; $P < .01$). The Newman-Keuls test for individual comparisons among means showed no significant difference between tone stimulation of the left ear alone or the right ear alone although each of these conditions was significantly different from the binaural and silent conditions ($P < .01$ in each case). The binaural condition, too, differed from the silent one ($P < .01$). The results of these statistical analyses coupled with the observation that every subject showed the trend illustrated (Fig. 1) demonstrates that the effects are reliable.

The finding that the tone stimulus presented monaurally produced almost twice as much inhibition as when presented binaurally is surprising since (i) in numerous loudness matching experiments (*5*), binaural acoustic signals are perceived to be slightly louder than the same signals presented monaurally, and (ii) previous investigations (*6*) have always revealed that the inhibitory effect is a function of the intensity of the inhibiting stimulus.

We do not know why monaural stimulation should produce such a strong inhibitory effect. The fact that the effect is not diminished when the tone stimulation is delivered to the contralateral ear argues that the responsible mechanisms must be central, but more research will be necessary before those mechanisms can be identified and understood. In the meantime, the effect enhances the potential of reflex inhibition as an audiometric technique.

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2. This device measures velocity rather than displacement, which is the customary measure of eyelid reflexes; however, the two indices covary over a wide range [E. Hilgard, *J. Gen. Psychol.* **8**, 85 (1933)].
3. Thresholds were determined by the method of R. Carhart and J. Berger [*J. Speech Hear. Disord.* **24**, 330 (1959)], in which the test tone is presented repeatedly in steps of increasing loudness until it becomes audible. Loudness matching was accomplished by alternating between a 70-db SL tone in the better ear and a tone of variable intensity in the less sensitive ear until the subject indicated that the variable tone was of equal loudness. Tones in these procedures were adjusted in 2-db steps rather than the 5-db increments common in clinical practice. To insure that stimuli were of equal intensity in the monaural and binaural conditions, earphones were calibrated under both conditions with a General Radio ANSI Type 1 coupler, P-7 microphone, and 1561-A sound level meter.
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d-Amphetamine–Induced Inhibition of Central Dopaminergic Neurons: Mediation by a Striato-Nigral Feedback Pathway

Abstract. *Lesions of the striato-nigral pathway (that is, crus cerebri or vicinity of the tail of the caudate nucleus) markedly attenuate depressant effects of intravenous* d-*amphetamine on central dopaminergic cell activity. These results, coupled with previous data showing that microiontophoretic application of* d-amphetamine *directly onto dopaminergic cells does not produce significant slowing, provide direct support for the hypothesis that the depressant effect of* d-*amphetamine on these cells is mediated through a striato-nigral neuronal feedback loop.*

In behaviorally effective doses, the primary action of *d*-amphetamine (*d*-AMPH) in the central nervous system appears to be that of increasing the release and blocking the reuptake of the catecholamines (*1*). In 1967, Corrodi *et al.* (*2*), on the basis of biochemical evidence, suggested that some of the effects of *d*-AMPH on dopamine (DA) metabolism might be secondary to a decrease in the firing rate of dopaminergic neurons. They further suggested that this decrease of DA neuronal activity might be due to the ability of *d*-AMPH to cause, indirectly, an increase in the stimulation of postsynaptic DA receptors, leading to a compensatory decrease in the firing rate of DA neurons mediated by a neuronal feedback pathway. Previously, we demonstrated that low doses of *d*-AMPH (0.25 to 2.0 mg/kg, intravenously) cause a marked depression of DA neuronal activity in the zona compacta of the substantia nigra (A9) (Fig. 1) and the ventral tegmental area (A10) (*3*). Furthermore, we provided evidence that this depressant effect of *d*-AMPH on A9 neurons is primarily an indirect action of *d*-AMPH, which is mediated by a neuronal feedback pathway (*4*, *5*). Thus we showed that, whereas postsynaptic cells in the caudate nucleus are very sensitive to microiontophoretically applied amphetamine as well as DA (*6*), microiontophoresis of *d*-AMPH directly onto A9 DA neurons fails to produce any depression of activity at low ejection currents (*5*) [at high ejection currents

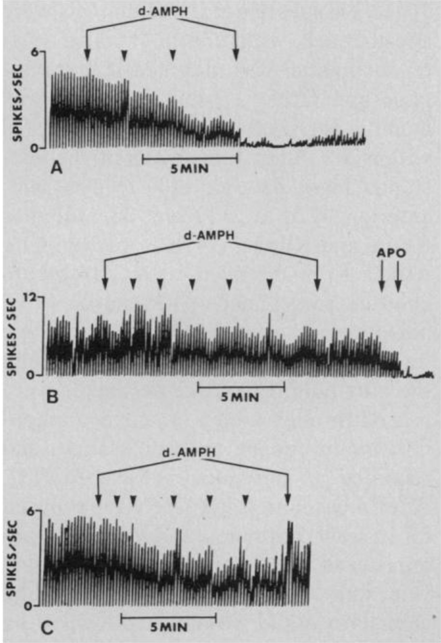

Fig. 1. (A) Typical *d*-AMPH–induced depression of the activity of a zona compacta (A9) dopaminergic neuron in an unlesioned animal. *d*-Amphetamine, in a total dose of 1.6 mg/kg (0.2, 0.2, 0.4, and 0.8 mg/kg, at arrows), temporarily stopped this cell. Recovery was very slow. Control lesioned animals yielded identical results. (B) Effect of *d*-AMPH and apomorphine (*APO*) on dopaminergic cell activity after lesion of the ipsilateral crus cerebri. *d*-Amphetamine, in a total dose of 25.6 mg (0.4, 0.4, 0.8, 1.6, 3.2, 6.4, and 12.8 mg/kg), produced a minimal slowing of this cell. In contrast, apomorphine (0.05 and 0.05 mg/kg) produced its usual depressant effect despite the presence of the lesion. (C) Effect of *d*-AMPH on A9 dopaminergic cell activity after an ipsilateral lesion of the tail of the caudate nucleus. *d*-Amphetamine, in a total dose of 25.6 mg/kg, produced a temporary 30 percent decrease in neuronal activity. All drugs were administered intravenously. Drug dosages are given in terms of the weight of their salts.