Visual Illusions in Neural Networks:  
Line Neutralization, Tilt After Effect, and Angle Expansion

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Certain visual illusions occur in neural networks that are capable of storing partially contrasted enhanced spatial patterns in short term memory (STM), and whose feature detectors are interconnected by nontrivial generalization gradients. These include neutralization, or adaptation, of nearly vertical or horizontal lines, tilt after-effect of successively viewed lines, and perceived angle expansion. Neutralization can be achieved by networks whose vertical and horizontal representations have higher saturation levels, broader tuning curves, or stronger input pathways. Tilt after-effect and angle expansion can be achieved by shunting lateral inhibition that causes an outward peak shift in the orientationally-coded STM pattern. The amount of outward peak shift is also dependent on the size of the potassium equilibrium point. Differences between the directions of tilt aftereffect (successive contrast) and angle expansion (simultaneous contrast) are ascribed to a normalization of total activity in the STM buffer whereby present stimuli and representations in STM of past stimuli interact to form a consistent action-oriented consensus.

1. Introduction

Why do visual illusions occur? Why is the exquisitely formed visual system susceptible to so many distortions of perception? This paper lists several distortions of neural pattern processing that are consequences of basic requirements on a pattern processor. In particular, certain distortions arise

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in a neural network which is capable of storing partially contrast enhanced patterns in short term memory, and whose feature detectors are interconnected by nontrivial generalization gradients. Analogies of these distortions are found in the neutralization and tilt after-effects discussed by Gibson (1933) and Gibson & Radner (1937) and in the angle expansion described by Blakemore, Carpenter & Georgeson (1970). Hysteresis effects (Fender & Julesz, 1967) also occur, but these will be discussed elsewhere. Section 2 briefly describes the illusions, section 3 summarizes the basic network principles that imply illusion-like properties, sections 4 and 5 analyze the network behavior, section 6 discusses relevant theoretical and experimental facts that are already in the literature, and section 7 discusses aspects of feature detector organization that are suggested by the illusions.

2. Some Visual Illusions

The illusion discovered by Blakemore et al. (1970) is as follows. If two lines forming an acute angle are presented to a subject and he is asked to place a third line parallel to one of the two, he will err in the direction of perceiving the angle as larger than it really is (Fig. 1). The effect is maximal when the angle is about 10° and drops to 0 when the angle approaches 0° or 90°. When the angle is obtuse, it is perceived as smaller than it really is, which may be interpreted as a complementary effect to the original one. Blakemore et al. (1970) conjectured that the shifted angle perception is a result of lateral inhibition between cortical neurons that are maximally tuned to different orientations. They postulated that each perceived line generates a distribution of activity centered at a particular column in the visual cortex, and that this distribution is the net effect of summing lateral excitatory and inhibitory influences (Fig. 2). The two distributions add in such a way that the peaks of the sum are shifted outward from the original peaks. If it is assumed that populations which are further apart on the cortex have orientations that are further apart, then the peak shift represents a perceived angle expansion.

![Diagram](image)

**Fig. 1.** Angle expansion due to an acute angle between A and B. Line B₀ is parallel to B. Line C is perceived as parallel to B.

**Fig. 2.** Excitatory (C) and inhibitory (D) gradients that are centered at 0° and 10° add in (a) and (b), respectively. The two net gradients add to yield a peak shift in (c). After Blakemore et al. (1970).
There is a similarity, noted both by Blakemore et al. (1970) and by Coltheart (1971b), between the angle-expansion effect and the tilt after-effect described by Gibson (1933) and Gibson & Radner (1937). If a tilted line which is nearly vertical is presented to a subject, and then it is removed from the visual field and replaced by an objectively vertical line, the vertical line will look tilted in the opposite direction; a tilted line which is nearly horizontal creates an analogous shift in perceived orientation [Fig. 3(a)]. Thus the tilt after-effect of \( T \) due to prior inspection of \( I \) (a); neutralization of \( I \) to \( I' \) due to sustained inspection of \( I \) (b).

"Angle" between the two lines (though presented successively rather than simultaneously as in the Blakemore experiment) is perceived as larger than it actually is. In effect, the stored representation of the first line interacts with the representation of the second line, and thereby tilts it. Campbell & Maffei (1971) confirm this result using vertical and horizontal gratings, and reference other experiments.

Gibson also showed that the tilted line itself, as it is viewed, will approach (but never reach) being seen as vertical [Fig. 3(b)]. He calls this effect adaptation, but since that term has many other meanings, we prefer the term neutralization, used by Coltheart (1971a). It is obvious, by symmetry arguments, that if there are no biases that favor one orientation over another, then there is no reason for a neutralization shift to occur in one direction rather than the opposite one. Hence some bias in network parameters as a function of orientation is anticipated. Mansfield (1974) has, for example, reported that in the visual cortical projection of the fovea in rhesus monkeys, vertical and horizontal orientations are more abundant than oblique orientations.

3. Basic Network Properties

Below we review how recurrent on-center off-surround networks undergoing mass action (or passive membrane, or shunting) interactions can be derived from first principles. Grossberg (1973) asked how parallel processing of patterns can be accomplished in the presence of noise and saturation. His solution is reviewed in Grossberg & Levine (1975). It is briefly outlined below for completeness. In effect, the solution shows how statistical properties of individual cells, or cell populations, can be married to geometrical properties of cell interactions to overcome noise and saturation.

Let \( n \) states (or cells, or cell populations) \( v_i, i = 1, 2, \ldots, n \), receive inputs \( I(i) \) whose intensity depends on the presence of a prescribed feature, or features, in an external pattern. Let each \( v_i \) have \( B \) excitable sites of which \( x_i \), the average potential of \( v_i \), are excited at time \( t \). Suppose that unexcited sites, which number \( B-x_i \), are excited by \( I_i \) via mass action. Also let excited sites \( x_i \) spontaneously become unexcited at a rate \( A \). Then

\[
\dot{x}_i = -Ax_i + (B-x_i)I_i
\]

so that \( x_i \) approaches the equilibrium point 0 when no input \( I_i \) is present. The relative input intensity \( \theta_i = I_i/I^{-1} \), where

\[
I = \sum_{k=1}^{n} I_k
\]

is the total input intensity, measures the relative importance of the feature coded by \( v_i \) in any given input pattern. How well do the equilibrium values of \( x_i \) code the \( \theta_i \) values? Grossberg (1973) notes that at high \( I \) values, the pattern of \( \theta_i \)'s is lost due to saturation of all \( x_i \) at \( B \), whereas at low \( I \) values, the pattern of \( \theta_i \)'s is confounded by any noise that exists in the system.

How can a better record of the \( \theta_i \)'s in \( x_i \) responses be achieved? Interactions between inputs and/or states must occur, because the \( \theta_i \)'s are defined by an interaction of all the inputs \( I_k, k = 1, 2, \ldots, n \); namely, \( \theta_i = I_i/I^{-1} \). The simplest solution is to let the inputs \( I_i \) be distributed in a nonrecurrent (feed-forward) on-center off-surround anatomy (Fig. 4). Then equation (1)
becomes

\[ \dot{x}_i = -Ax_i + (B - x_i)I_i - x_i \sum_{k \neq i} I_k, \]  
\[ \text{(2)} \]

so that at equilibrium (\( \dot{x}_i = 0 \)),

\[ x_i = \frac{0}{A + J_i}. \]  
\[ \text{(3)} \]

Equation (3) shows that automatic gain control via the off-surround, that is, term

\[ ( -x_i \sum_{k \neq i} I_k) \]

in equation (2), eliminates saturation as \( I \) increases, and maintains \( x_i \)'s sensitivity to the ratio of center-to-surround excitation. Moreover, the total activity

\[ x = \sum_{i=1}^{n} x_i \]

adapts, or is normalized, since \( x \leq B \) no matter how intense \( I \) is.

How can short term memory (STM) of input patterns be achieved? Some mechanism is needed that can maintain activity in the \( v_i \) after inputs cease and that can also let this activity be rapidly inhibited by competing inputs. Reverberatory interactions between populations are therefore assumed to exist. The simplest recurrent network capable of overcoming saturation utilizes feedback signals distributed in an on-center off-surround anatomy, as in system

\[ \dot{x}_i = -Ax_i + (B - x_i)[f(x_i) + I_i] - x_i \sum_{k \neq i} f(x_k) + J_i, \]  
\[ \text{(4)} \]

where \( f(w) \) is the average feedback signal that is produced by an average activity level \( w \); for example, term \( (B - x_i)f(x_i) \) in equation (4) describes self-excitation of \( v_i \) via the recurrent signal \( f(x_i) \) at activity level \( x_i \). Function

\[ J_i = \sum_{k \neq i} I_k, \]

as in equation (2).

The signal function \( f(w) \) must be properly chosen to store important input data in STM and yet to prevent amplification and storage of noise in STM. A complete classification is given in Grossberg (1973) and is reviewed in Grossberg & Levine (1975). A sigmoid (S-shaped) signal function \( f(w) \) [Fig. 6(a)] can suppress noise and other activity that falls below a prescribed quenching threshold (QT), yet contrast enhances and stores in STM all activity that exceeds QT [Figs 6(b) and (c)]. The sigmoid function balances two undesirable tendencies: too little vs. too much noise suppression. Linear signals \( f(w) = aw \) and slower-than-linear signals [e.g., \( f(w) = w(b + w)^{-1} \)] both amplify noise. Faster-than-linear signals [e.g., \( f(w) = w^2 \)] suppress noise so strongly that they also "choose" the population with initial maximal activity for STM storage and suppress all nonmaximal activities [Fig. 6(d)].

The sigmoid signal can store partially contrast-enhanced patterns, because it has a QT. A main question therefore arises: what other mechanisms exist for storing partially contrast enhanced in STM, as in Fig. 6(c)? How can the two extremes of noise amplification and choice-making be avoided so that a continuum of activity levels can be simultaneously stored in STM?

Elias & Grossberg (1975) study STM in a wide variety of related systems; for example, systems of the form

\[ \dot{x}_i = -Ax_i + (B - x_i) \left[ \sum_{k=1}^{n} f(x_k)C_{ik} + I_i \right] - x_i \left[ \sum_{k=1}^{n} g(y_k)D_{ik} + J_i \right], \]  
\[ \text{(5)} \]

\[ \dot{y}_i = -Ey_i + \sum_{k=1}^{n} x_kF_{ik}, \]  
\[ \text{(6)} \]
where $x_i$ is the mean activity in the $i$th excitatory population, $y_i$ is the mean activity in the $i$th inhibitory population, $I_i$ is the $i$th excitatory input, and $J_i$ is the $i$th inhibitory input. They find that a powerful tendency for either choice-making or noise amplification exists, but that partial contrast in STM can be achieved by two main mechanisms: (1) a system such as equation (4) in which the off-surround has uniform strength between all distinct populations and the signal functions for excitatory and inhibitory signals have the same form; or (2) a system in which excitatory signal strength decreases with distance more rapidly than inhibitory signal strength, as in Fig. 7. Case (2) describes situations where there are many feature detectors and some detectors are mutually more closely coupled than others. Anatomies of this type have a plausible psychological interpretation; for example, color detectors sensitive to similar wavelengths, or line detectors sensitive to similar orientations, etc. Such preferred couplings between populations are presumably the anatomical substrate on which innate behavioral generalization gradients are built. There are also some simple physiological reasons why the connection strengths between neurons can decrease as a function of the distance between them; for example, a random growth of axon branches will cause the number of synaptic connections to decrease with distance (Sholl, 1956). In fact, excitatory signals often traverse narrower distance ranges than inhibitory signals (Colonnier, 1965; Szentagothai, 1967; Scheibel & Scheibel, 1970; Bishop et al., 1971). The excitatory coefficients $C_{kl}$ will correspondingly be assumed to decrease with distance faster than the inhibitory coefficients $D_{kl}$. Below it is shown that when systems of this type are constrained to be capable of graded activity in STM, then they also exhibit behavior analogous to certain illusions. Even if other mechanisms contribute to these illusions in vivo, the minimal mechanisms described below must also be implicated whenever recurrent on-center off-surround networks undergoing shunting interactions participate in pattern processing.

Our results will study the equilibrium behavior of such networks after they adjust to prescribed input patterns. We therefore analyze the simpler networks

\[
\dot{x}_i = -Ax_i + (B_i - x_i) \left[ \sum_{k=1}^{n} f(x_k)C_{ki} + I_i \right]
- x_i \left[ \sum_{k=1}^{n} g(x_k)D_{ki} + J_i \right]
\]

(7)

in which the inhibitory potentials have already equilibrated to their excitatory inputs. We also study the related networks

\[
\dot{x}_i = -Ax_i + (B_i - x_i) \left[ \sum_{k=1}^{n} f(x_k)C_{ki} + I_i \right]
- (x_i + E) \left[ \sum_{k=1}^{n} g(x_k)D_{ki} + J_i \right],
\]

(8)

$E > 0$, in which the inhibitory equilibrium level $-E$ and the passive equilibrium level 0 are different; $E$ is related to the Nernst potential for
of \( m \) (one of which is set equal to 0 for our computation) and \( K_m \) is some positive constant for \( m = 0 \) and \( m = l \). \( C_{ik} \) and \( D_{ik} \) are assumed to be functions of the minimum distance between \( i \) and \( k \) on the circle. Thus only the distance between the two populations receiving inputs is important, not their absolute location. We can therefore write \( C_{ik} = C(i-k) \) and \( D_{ik} = D(i-k) \) with this convention in mind. Suppose that \( l \leq n/2 \) and \( i \leq n/2 \) for definiteness. Then at equilibrium \( x_i = 0 \), equation (10) implies

\[
x_i = A^{-1}K[C(l)+C(l-i)-D(l)-D(l-i)].
\]

Since \( x_i \) is symmetric around \( l/2 \) as a function of \( i, 0 \leq i \leq l \), it suffices to consider its behavior as \( i \) approaches \( l \). In particular a peak shift will occur if \( x_{i+1} > x_i \). This is the same as

\[
[D(l) - D(l+1)] - [C(i) - C(l+1)] > [C(0) - C(1)] - [D(0) - D(1)].
\]

Thus, the right hand side of equation (11) is always positive. To make the left hand side even more positive it is sufficient that \( C(i) \ll 0 \), and thus that \( C(i) - C(l+1) \approx 0 \), at an \( l \) value where \( D(l) \) is significantly greater than \( D(l+1) \). The absolute size that \( D(l) - D(l+1) \) must attain to satisfy equation (11) depends on the breadth of the peaks of \( C(l) \) and \( D(l) \); as the breadth increases, the likelihood that \( D(0) \approx D(1) \) also increases. In any case

\[
D(l) - C(l) > D(l+1) - C(l+1),
\]

so that the net inhibition at \( l \) is greater than the net inhibition at \( l+1 \). This will not be necessary in the case of shunting interactions.

In the shunting case, computer studies were first carried out on system (8) with

\[
I_t = \sum_{m=1}^{n} K_m C_{ml},
\]

all \( J_i = 0 \), \( K_0 = K_1 = K_2 \), and \( K_m = 0 \) otherwise. In particular, the excitatory coefficients \( C_{ml} \) were chosen to have a Gaussian distribution; e.g., for \( m-l < n/2 \), let \( C_{ml} = G \exp \{-H^{-2}|m-l|^2\} \). The inhibitory coefficients \( D_{ml} \) were similarly distributed. No inhibitory inputs existed, but inhibition spread through the network via the recurrent off-surface. Even if the maximal strength of inhibition equalled that of excitation, either no peak shift occurred or it occurred inwardly rather than outwardly. Further analysis showed that the inhibitory interactions were still not strong enough to overcome the combined effects of excitatory inputs and excitatory interactions.

To see this, consider what happens if the excitatory inputs are so strong that they dominate all recurrent interactions. Then the recurrent network (8)

4. Angle Expansion

This section explicates Blakemore's heuristic additive model and analyses the behavior of analogous shunting models. In Blakemore's model, each line generates a net gradient due to the summation of excitatory and inhibitory influences, as in Figs 2(a)–(b). When two lines are presented, their gradients combine additively to produce a net gradient whose peaks are outwardly shifted, as in Fig. 2(c). This peak shift is due to the addition of net excitation in one gradient with net inhibition in the other gradient. It will be seen below that in the shunting case, net inhibition is not needed to produce a shift. In other words, even if the net gradient due to one line shows no net inhibition, the interaction between two such gradients gives indirect evidence that inhibition is operative. No net inhibition in response to one input line will occur if \( E = 0 \) in equation (8), yet the network reaction to two input lines ("angle expansion") will give clear evidence of inhibitory interaction. As \( E \) is parametrically increased, net inhibition in response to one line will occur, and angle expansion is achieved for a more robust choice of other network parameters.

Blakemore tacitly introduces a model which we explicate as follows:

\[
\dot{x}_i = -Ax_i + \sum_{m=1}^{n} K_mC_{ml} - \sum_{m=1}^{n} K_mD_{ml}.
\]

Assume that each population \( v_i \) is maximally sensitive to a different line orientation (Hubel & Wiesel, 1962, 1968), and that the \( v_i \) are arranged in a circle going from 0° to 180° (which is the same as 0° here). This is done to avoid boundary effects, such as spurious bumps in the STM pattern, due to the existence of only finitely many cells; cf. Ellias & Grossberg (1975). A stimulus of two lines is represented by letting \( K_m = 0 \) for all but two values of \( m \) (one of which is set equal to 0 for our computation) and \( K_m \) is some positive constant for \( m = 0 \) and \( m = l \). \( C_{ik} \) and \( D_{ik} \) are assumed to be functions of the minimum distance between \( i \) and \( k \) on the circle. Thus only the distance between the two populations receiving inputs is important, not their absolute location. We can therefore write \( C_{ik} = C(i-k) \) and \( D_{ik} = D(i-k) \) with this convention in mind. Suppose that \( l \leq n/2 \) and \( i \leq n/2 \) for definiteness. Then at equilibrium \( x_i = 0 \), equation (10) implies

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x_i = A^{-1}K[C(l)+C(l-i)-D(l)-D(l-i)].
\]

Since \( x_i \) is symmetric around \( l/2 \) as a function of \( i, 0 \leq i \leq l \), it suffices to consider its behavior as \( i \) approaches \( l \). In particular a peak shift will occur if \( x_{i+1} > x_i \). This is the same as

\[
[D(l) - D(l+1)] - [C(i) - C(l+1)] > [C(0) - C(1)] - [D(0) - D(1)].
\]

Consider Fig. 2(a). There, the right hand side of equation (11) is always positive. To make the left hand side even more positive it is sufficient that \( C(l) \ll 0 \), and thus that \( C(l) - C(l+1) \approx 0 \), at an \( l \) value where \( D(l) \) is significantly greater than \( D(l+1) \). The absolute size that \( D(l) - D(l+1) \) must attain to satisfy equation (11) depends on the breadth of the peaks of \( C(l) \) and \( D(l) \); as the breadth increases, the likelihood that \( D(0) \approx D(1) \) also increases. In any case

\[
D(l) - C(l) > D(l+1) - C(l+1),
\]

so that the net inhibition at \( l \) is greater than the net inhibition at \( l+1 \). This will not be necessary in the case of shunting interactions.

In the shunting case, computer studies were first carried out on system (8) with

\[
I_t = \sum_{m=1}^{n} K_mC_{ml},
\]

all \( J_i = 0 \), \( K_0 = K_1 = K_2 \), and \( K_m = 0 \) otherwise. In particular, the excitatory coefficients \( C_{ml} \) were chosen to have a Gaussian distribution; e.g., for \( m-l < n/2 \), let \( C_{ml} = G \exp \{-H^{-2}|m-l|^2\} \). The inhibitory coefficients \( D_{ml} \) were similarly distributed. No inhibitory inputs existed, but inhibition spread through the network via the recurrent off-surface. Even if the maximal strength of inhibition equalled that of excitation, either no peak shift occurred or it occurred inwardly rather than outwardly. Further analysis showed that the inhibitory interactions were still not strong enough to overcome the combined effects of excitatory inputs and excitatory interactions.

To see this, consider what happens if the excitatory inputs are so strong that they dominate all recurrent interactions. Then the recurrent network (8)
can be approximated by the nonrecurrent network
\[ \dot{x}_1 = -Ax_1 + BK[C(i) + C(i-l)]C(i-l) - x_1, \]
with equilibrium value
\[ x_1 = \frac{BK[C(i) + C(i-l)]}{A + K[C(i) + C(i-l)]}. \] (12)

Since \( x_1 \) is a monotone increasing function of the input sum \( C(i, l) = C(i) + C(i-l) \), and since \( C(i, l) \) has its maximal value(s) between 0 and \( l \), the peak(s) of the graph \( x_1 \) must also occur between 0 and \( l \).

The strength of inhibition can be increased either nonrecurrently, using inhibitory inputs to balance the excitatory inputs, or recurrently, via the inhibitory interactions. Both mechanisms work, and they do so in a way that interestingly contrasts with the case of additive interactions.

First consider the nonrecurrent network in which each line generates both an excitatory and inhibitory gradient. Then
\[ \dot{x}_1 = -Ax_1 + (B-x_1) \sum_{m=1}^{n} K_m C_m - (x_1 + E) \sum_{m=1}^{n} K_m D_m. \]
At equilibrium,
\[ x_1 = \frac{\sum_{m=1}^{n} K_m (BC_m - ED_m)}{A + \sum_{m=1}^{n} K_m (C_m + D_m)}. \] (13)

An interesting contrast with the additive case occurs if \( E = 0 \). Suppose that only one line perturbs the network, say at 0. Then, considering values of \( i \) near zero for definiteness,
\[ x_1 = \frac{BK(i)}{A + K[C(i) + D(i)]}. \]
Interpolate a continuous function \( x(s) \) through \( x_1 \) by letting
\[ x(s) = \frac{BK(s)}{A + K[C(s) + D(s)]}, \]
and suppose that \( C(s) \) falls faster than \( D(s) \), as in Fig. 7, in the sense that
\[ C^{-1}(s)C(s) < D^{-1}(s)D(s). \] (14)
Condition (14) holds, for example, if the excitatory and inhibitory coefficient strengths have a Gaussian distribution. Given equation (14), \( x(s) \) has one maximum at 0 because if \( x(s) = 0 \) at some other value of \( s \),
\[ C^{-1}(s)C(s) = [AK^{-1}(s) + D(s)]^{-1}D(s) > D^{-1}(s)D(s), \]
which is a contradiction. Thus if \( E = 0 \), the net gradient of excitation and inhibition is unimodal with no net inhibitory trough, by contrast with the subtractive case of Fig. 2(b). Although there is no inhibitory trough if \( E = 0 \), such a system is nonetheless capable of an outward peak shift if two lines are presented, say at 0 and \( l \). Then at equilibrium,
\[ x_1 = \frac{BK[C(i) + C(i-l)]}{A + K[C(i) + C(i-l) + D(i) + D(i-l)]}. \]
An outward peak shift occurs if \( x_{i+1} > x_n \) which is the same as
\[ C(l+1)D(i) + C(1)D(i) + D(0)C(l+1) + D(0)C(1) + C(l+1)D(l) + C(l)D(l) \]
\[ -C(0)D(l+1) - C(0)D(l) > F[C(l) - C(l+1) + C(0) - C(1)], \] (15)
with \( F = AK^{-1} \). As in the case of additive interactions, such an outcome is facilitated by the conditions \( C(0) \approx C(1) \), \( C(l) \approx C(l+1) \), \( D(0) \approx D(1) \), and \( D(l) - D(l+1) \) relatively large. Then equation (15) becomes approximately
\[ [C(0) + C(l)][D(l) - D(l+1)] > 0, \]
which is trivially satisfied. How can these conditions be guaranteed? In all cases where inhibition falls off more slowly with distance than excitation, condition \( C(0) \approx C(1) \) implies \( D(0) \approx D(1) \), and values of \( l \) exist such that \( C(l) \approx C(l+1) \) whereas \( D(l) - D(l+1) \) is still relatively large. Condition (15) is also easier to achieve for smaller values of \( F = AK^{-1} \), which supports the assumption, used to motivate the nonrecurrent approximation, that inputs are large compared to interaction strengths. The existence of an outward peak shift shows that inhibition can have an important influence even if no net inhibitory trough exists.

If \( E \neq 0 \) in equation (13), then a net inhibitory trough can exist, and when it does a peak shift can be more readily produced. In fact, the conditions that guarantee an inhibitory trough in response to one line when \( E > 0 \) are qualitatively the same as the conditions that guarantee a peak shift in response to two lines given any values of \( E \). To see this, suppose that a line perturbs the network at \( n = 0 \). Then at equilibrium, equation (13) implies
\[ x_1 = \frac{K[BC(i) - ED(i)]}{A + K[C(i) + D(i)]}. \]
For convenience, interpolate the \( x_i \) 's using
\[ x(s) = \frac{K[BC(s) - ED(s)]}{A + K[C(s) + D(s)]}. \]
A critical point of \( x(s) \) is characterized by
\[ F[BC(s) - ED(s)] = (B + E)[C(s)D(s) - C(s)D(s)]. \] (16)
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By equation (14), the right-hand side of equation (16) is positive. On the left-hand side of equation (16), $B$ and $E$ are positive, whereas $C(i)$ and $D(i)$ are negative. Equality in equation (16) can be achieved at values $s$ where $C(s) = 0$ and $D(s)$ is sufficiently negative to match the right-hand side; for example, where $C(a)$ is already near zero and $D(s)$ is falling rapidly. In other words, the conditions that guarantee an inhibitory trough in response to one line when $E > 0$ are qualitatively the same as the conditions that guarantee a peak shift in response to two lines when $E = 0$. We now show that the range of parameters that produce a peak shift for $E > 0$ is wider than the range of parameters that produce a peak shift for $E = 0$. Thus, although a trough is not necessary for a peak shift to exist, it can only make a peak shift easier to obtain. To see this, present lines at $m = 0$ and $l$. Then at equilibrium, equation (13) becomes

$$x_t = \frac{K[B(i) + BC(i-l) - ED(i) - ED(i-l)]}{A + K[C(i) + C(i-l) + D(i) + D(i-l)]}. \quad (17)$$

Suppose $x_{t+1} > x_t$. Defining $U_0 = C(i) + C(0)$, $U_1 = C(i+1) + C(1)$, $V_0 = D(i) + D(0)$, and $V_1 = D(i+1) + D(1)$, this inequality becomes

$$\frac{BU_1 - EV_1}{F + U_1 + V_1} > \frac{BU_0 - EV_0}{F + U_0 + V_0}. \quad (18)$$

The analogous inequality when $E = 0$ is

$$\frac{U_1}{F + U_1 + V_1} > \frac{U_0}{F + U_0 + V_0}. \quad (19)$$

We now show that equation (19) implies (18). Multiply both sides of equation (19) by $B + E$ and subtract $E$. Then

$$\frac{BU_1 - EV_1 - EF}{F + U_1 + V_1} > \frac{BU_0 - EV_0 - EF}{F + U_0 + V_0}. \quad (20)$$

Since $F + U_1 + V_1 < F + U_0 + V_0$, it is also true that

$$\frac{EF}{F + U_1 + V_1} > \frac{EF}{F + U_0 + V_0}. \quad (21)$$

Adding equations (20) and (21) yields equation (18). Thus a positive $E$ makes a peak shift easier to achieve than $E = 0$. In other words, shifting the potassium equilibrium point should influence the size of outward peak shift, other things equal. More generally, Grossberg (1976) shows that increasing $E$ produces a new form of contrast enhancement in on-center off-surround networks. A sufficiently large $E$ can inhibit all but the non-uniform part of the activity pattern across a network.

In Appendix A, the recurrent network with inputs coupled to inhibition as above is studied by means of computer simulation. Parameters are employed which produce angle expansion in the non-recurrent case treated above. The full network is equation (8) with all $B_i = A_i$,

$$I_i = \sum_{m=1}^{n} K_m C_{mi}, \quad J_i = \sum_{m=1}^{n} K_m E_{mi}, \quad C_{mi} = G_1 \exp \{-H_1^{-1} |m-i|^2\},$$

and $D_{mi} = G_2 \exp \{-H_2^{-1} |m-i|^2\}$. Here $G_1 = G_2$ and $H_1 < H_2$. The signal function $f(w) = w^2$ grows faster than linearly, but since our maximum activities are all small, it is a good approximation to the small activity range of a sigmoid signal function. Parameter values and their justification are contained in Appendix A.

The inhibition needed to produce an outward peak shift can also be introduced via the inhibitory connections in the absence of inhibitory inputs. Then equation (8) holds with

$$I_i = \sum_{m=1}^{n} K_m C_{mi}$$

and $J_i = 0$. In order to obtain such a shift, it was necessary to make the maximum inhibitory interaction strength enough greater than the maximum excitatory interaction strength to balance the additional contribution due to sustained excitatory inputs. Computer studies of this phenomenon are found in Appendix A.

5. Gibson Neutralization and After Effect

The Gibson & Radner experiments contain two parts, one on line neutralization, in which a line close to vertical (horizontal) under sustained gaze gradually looks more vertical (horizontal), and the second on tilt after-effect, in which after the first line is removed, a second line that is vertical (horizontal) looks tilted away from the first line. Below we will find a neutralization effect in a network in which there are either of two kinds of biases. Either the interaction gradients are amplified in strength near the vertical and horizontal populations, or the maximal number of excitatory sites in these populations is greater.

The speed with which neutralization sets in will depend on the choice of network parameters. An arbitrarily slow drift can be achieved. Thus one cannot reliably argue that the slow onset of neutralization implies the existence of plastic changes in network parameters. Once the neutralized line is stored in STM by the reverberation, the shift in the second line's apparent orientation can be viewed as an angle expansion phenomenon in which the gradient created by the actually perceived line interacts with the
stored gradient of the first line. Two comments should be made about this interpretation of the after-effect. First, it is not essential that the stored representation be of a neutralized line, but only that it be of a line that has two properties: it is on the same side of the second line as the first line was, and it makes a sufficiently small angle with the second line. Second, this explanation faces the issue that items can be stored in STM without being perceived at every moment that they are stored; for example, a telephone number can be stored without each digit being continuously heard. Yet evidence abounds that when new items related to stored items are presented, the stored items can transform the way in which the new items are perceived. In other words, a feedback exists between the STM buffer and the population which subserves the percept. Our explanation suggests that recurrent on-center off-surround interactions are occurring in either or both of these structures, but does not require a precise synthesis of the feedback mechanism to make its qualitative point. Therefore the remainder of this section will be devoted to the neutralization effect. Grossberg (1977) discusses this feedback.

It is obvious, by symmetry arguments, that if there are no biases that favor one orientation over another, then there is no reason for a neutralization shift to occur in one direction rather than the opposite one. Hence some bias in network parameters as a function of orientation is anticipated. Speaking formally, two main types of parameters can be biased: the maximum activity levels, or number of excitable sites, $B_i$ and the interaction strengths $C_{ij}$ and $D_{ij}$, or tuning, between populations. For the case that we studied, an increase in certain $B_i$ is the same as an increase in $C_{ij}$ and $D_{ij}$. This case is defined by equation (8) with $f(w) = w^2$ and $E = 0$. The choice of a quadratic $f(w)$ was made to simulate the effect of a sigmoid signal function at small activity levels. Defining $B_i = \lambda_i B_i$, equation (8) can be transformed, using the change of variable $q_i = \lambda_i^{-1} x_i$ into system

$$
q_i = -\lambda_i q_i + (B - q_i) \left[ \sum_{m=1}^n f(q_m) C_{mi} + I_i \right] - q_i \left[ \sum_{m=1}^n f(q_m) D_{mi} + J_i \right],
$$

where $\lambda_i = \lambda_i C_{mi}$ and $\beta_{mi} = \lambda_i^2 F_{mi}$. In other words, an amplification of $B_i$ is formally the same as an amplification, or retuning, of the interaction coefficients of the $i$th population. We will therefore only discuss a non-uniform distribution of $B_i$'s; that is, a non-uniform choice of the number of cell sites that code for prescribed orientations. In a similar fashion, biases in the connection strengths of pathways bringing inputs to the network can be transformed into a non-uniform distribution of network parameters. Grossberg (1975b) describes a model of cortical tuning that shows how such an asymmetric parameter distribution can develop using shunting network mechanisms and cross-correlational synapses.

In our computer studies, we let population $i = 0$ code a vertical orientation, and choose a Gaussian distribution $B_i = G^{-1} \exp \{-H_i/2\}$ as well as Gaussian distributions of interaction strength as a function of the distance between populations. A sustained input is delivered to a population $v_i$ which codes a nearly vertical orientation. This input creates a focus of maximal excitation at $v_i$ and we study whether, and how fast, this focus drifts towards the vertical. For small values of $H_i$, there is no drift; for intermediate values of $H_i$, the drift is slow; and for large values of $H_i$, the drift is fast. In other words, the drift rate depends on how non-uniform is the pattern of $B_i$'s of the $v_i$ with which $v_i$ interacts; cf. equation (9). In the Gibson–Radner experiments, neutralization takes place on a time scale of minutes, and is therefore a "slow" phenomenon.

Various other data suggest that asymmetries exist in the orientational coding of certain neural populations. Rose & Blakemore (1974) have done a statistical study of the tuning curves of orientation-sensitive cells in the cat striate cortex. Two types of cells were investigated, the so-called "simple" and "complex" cells. In both types of cells, the average half-width of tuning curves of cells coded vertically or horizontally differs from the half-width of other cells. For complex cells, the vertical and horizontal tuning is broader; for simple cells, it is narrower. The tuning of complex cells is compatible with our model. Our above considerations therefore suggest that if the complex cells are partly responsible for neutralization, then either they are interconnected by a recurrent on-center off-surround network, or they interact with a STM buffer that has this structure.

6. Discussion of the Lateral Inhibition Model and Alternatives

As sections 4 and 5 indicated, shunting on-center off-surround networks undergoing distance-dependent interactions can simulate various visual illusions. There are also heuristic models in the literature for all these phenomena, and it is instructive to compare these models with network mechanisms.

For the Gibson & Blakemore phenomena, the two major theories that have been proposed, other than lateral inhibition, are known as normalization and satiation.

The normalization theory was first stated by Gibson (1937), who conjectured that neutralization results from the horizontal and vertical orientations being in some sense norms, such that a given line adapts toward the norm to which it is closest. He viewed the tilt after-effect as a product of
neutralization, claiming that the shift in perceived orientation of the first
line will shift the perceived orientation of the second vertical line in the same
direction.

Gibson does not propose a specific neural mechanism for the normali-
zation, but he implies that the vertical and horizontal are special directions
in mammalian (or at least human) visual perception. Gibson & Radner
(1937) showed that human subjects, when asked to set a pointer at prescribed
orientations, are most accurate at setting vertical and horizontal orientations,
and often set oblique orientations by estimating how far they deviate from
vertical or horizontal. Campbell, Kulikowski & Levinson (1966) support
this idea by constructing gratings out of parallel lines, and showing that
acuity is best when the lines are vertical or horizontal and worst when the
lines are tilted by 45°. Maffei & Campbell (1970), using an evoked potential
technique, show that the human visual cortex responds more to vertical and
horizontal axes than to oblique axes.

There exists some psychological evidence that these “norms” develop due
to more frequent exposure to the vertical and horizontal orientations in the
early visual environment. Annis & Frost (1973) showed in a cross-cultural
study of white Canadians and Cree Indians that the whites exhibited selective
acuity for horizontal and vertical gratings, but the Indians did not. They
attribute the Indian data to such factors as the lesser degree of horizontal
and vertical orientations in Indian architecture.

The idea that the “norms” are strongly influenced by early visual environ-
ment is also supported by some neurophysiological evidence. Blakemore &
Cooper (1970) showed that if kittens are raised in a visual environment
consisting solely of vertical (horizontal) stripes, then they are virtually blind
to horizontal (vertical) stimuli and develop fewer cells in the visual cortex
tuned maximally to the horizontal (vertical). Hirsch & Spinelli (1970)
raised kittens with one eye seeing only horizontal stripes and the other eye
seeing only vertical stripes; they later found a preponderance of cells in the
visual cortex driven only by one eye (instead of by both eyes as in normal
cats) and with receptive fields oriented in the direction to which that eye had
been exposed. Moreover Pettigrew (personal communication) has raised
kittens in an environment consisting solely of stripes tilted at an oblique
orientation, and has found that animals so raised developed a bias among
cortical cells in favor of that particular tilted orientation. In all these experi-
ments, there had not been degeneration of cortical tissue, so it would seem
that experience had altered the tuning curves of cortical cells.

We can therefore consider a particular orientation a “norm” if visual
experience has made certain classes of cells sensitive to that orientation more
numerous than others, or perhaps more broadly tuned than others. In that
dense, the model proposed in section 5 sharpens Gibson’s idea that line
neutralization results from adaptation to a norm.

Tilt after-effect need not, however, be explained by adaptation to a norm.
In fact, more recent experiments have shown that such an after-effect can
take place even when there has been no neutralization. Köhler & Wallach
(1944), Prentice & Beardslee (1950), and Templeton, Howard & Easting
(1965) did experiments similar to that of Gibson & Radner (1937); an
inspection line was presented to a subject, then removed and followed by a
test line of a slightly different orientation, but not necessarily horizontal or
vertical. As in the Gibson–Radner effect, the perceived orientation of the
test line was not its actual orientation but slightly shifted away from that of
the inspection line. In particular, an after-effect took place even if the
inspection line was vertical or horizontal, and therefore could not have been
neutralized. Coltheart (1971a) reports data in which the after-effect develops
even if the inspection line is viewed only for a few seconds. Typically,
neutralization occurs on a time-scale of minutes. This data, therefore, also
suggests that the after-effect can occur in the absence of neutralization. In
our model, it is not necessary for the inspection line to be neutralized, but
only for it to have a STM representation that can interact with the represen-
tation of the test line to produce angle expansion.

Gibson found support for normalization as the cause of after-effect in the
following “indirect effect”, whose existence was confirmed by Campbell &
Maffei (1971). If the inspection line is tilted slightly counterclockwise from
the vertical, not only will a vertical test line appear tilted in the clockwise
direction but so will a horizontal test line. Gibson explained this phenomenon
by stating that the neutralization of the first line causes not only the nearest
norm to shift but both norms to shift together. One is initially tempted by
this data to suppose that all orientations are tilted in the neutralized direction.
This does not explain, however, why the indirect effect is always smaller
than the direct effect. In the discussion below, we will see that other
models can explain the indirect effect in a manner that also explains this
difference.

The satiation theory was first stated by Köhler & Wallach (1944) to
explain the after-effect alone and not the neutralization. They claimed that
orientation-sensitive cells in the visual cortex that respond to the first line
will be satiated after prolonged exposure to that line; thus for a short period
afterwards, the responses of the visual cortex will be biased away from those
cells. A second line will therefore be shifted in its perceived orientation away
from the orientation of the first line. The theories of after-effect that are
named “neural enhancement” (Over, 1971) and “adaptation” (Coltheart,
1971b) are really variants of the satiation theory.
The indirect effect was explained by Coltheart (1971a) using a satiation theory. He cited Hubel & Wiesel's (1965, 1968) result that there exist hypercomplex cells in the visual cortex having two preferred orientations that are 90° apart. Thus if the first line is tilted slightly counterclockwise from vertical, that orientation will satiate and so will the orientation 90° away, which is slightly tilted counterclockwise from horizontal; a horizontal second line will therefore be perceived as tilted clockwise. This explanation does not explicitly describe the feedback pathways from hypercomplex cells to the other processing stages that contribute to perceiving a line. Nor have the feedback pathways from simple cells or complex cells been described. Nonetheless, Coltheart gives a plausible account of why the indirect effect is smaller than the direct effect. He claims that the network representation of a horizontal test line is influenced only by satiated hypercomplex cells, whereas the representation of a vertical test line is also influenced by satiated complex cells, among others.

The above explanation of the indirect effect can, however, also be recast in a theory of norms: let the neutralized representation of the inspection line excite the corresponding hypercomplex cell population, which in turn excites the representation of perpendicular lines. This normative effect can only be increased if the asymmetric representation of horizontal and vertical lines as a result of development induces a corresponding asymmetry in the coding properties of hypercomplex cells.

The satiation theory seems to be less satisfactory as an explanation of angle expansion. Blakemore et al. (1970, 1971) reject the satiation theory based on experiments that are compatible with a disinhibitory mechanism, but not a satiation mechanism. They place a third line near one of the two lines forming an acute angle (Fig. 8). This third line decreases the angle expansion that would otherwise occur, whether it lies inside or outside the angle. If satiation theory were true, when the third line is inside the angle, there should be an increase in angle expansion. In a theory based on lateral inhibition, the third line inhibits the activity of the adjacent lines, thereby decreasing its inhibitory effect on the nonadjacent line and decreasing the angle expansion.

Recent neurophysiological experiments on the visual cortex of adult cats (Creutzfeldt & Heggelund, 1975) suggest that an adaptation of cortical populations can be induced by prolonged exposures to oriented displays. Whether this is due to a slowly building satiation or to enhancement of inhibitory connection strengths has not been decided. In either case, these results do not compromise the suggestion that lateral inhibition can yield illusions over shorter time scales.

7. Normalization of Activity in STM

Two discrepancies between the Gibson data and the Blakemore data still need to be explained. Figures 9(a) and (b) reproduce Fig. 3 of Gibson & Radner (1937) and Fig. 3 of Blakemore et al. (1970), respectively. First, as the graphs show, the direction of the Gibson after-effect changes when the lines are 45° apart. Morant & Harris (1965) find that the cross-over point is closer to 60°, and argue that neutralization and satiation effects add up to produce this effect. By contrast, the direction of the Blakemore angle expansion does not change until the lines are 90° apart. Second, the contraction of obtuse angles in the Blakemore experiment is opposite to the indirect effect of Gibson, where an obtuse angle between the inspection and test lines is expanded if the angle is less than 135°.

Both discrepancies are compatible with properties of the hypercomplex cells mentioned above, which have two preferred orientations at right angles to each other. We have already discussed Coltheart's explanation of Gibson's indirect effect by means of these hypercomplex cells; the Gibson effect is dealt with by noting that the hypercomplex cells divide up angle space into four equal quadrants. Let axis 1 denote the orientation of the inspection
Analogously, suppose that a behavioral plan is being carried out, with commands for action stored in STM ready to be elicited. How are new commands generated if an incompatible event occurs? How do the new commands inhibit the old commands? Speaking intuitively about such situations, one wants to say that strengthening one command or representation somehow weakens another. Such a concept of total activity normalization also arises in mechanisms of reinforcement (Grossberg, 1972a); of STM for parallel processed patterns (Grossberg, 1973); of perceptual constancies (Grossberg, 1972b); of pattern discrimination (Grossberg, 1970); and of attention and discrimination learning (Grossberg, 1975a). The normalization property is illustrated by the adaptational property of shunting networks that was described by equation (3). Given such a property, the difference between successive contrast (Gibson) and simultaneous contrast (Blakemore) would be explicable if the highest-order discriminative cells, such as hypercomplex cells, have greater activity in STM, and therefore greater potency for transforming the data in perceptual fields, when they are less inhibited by lower-order representations of present events. Thus, when more external cues are on (Blakemore), the activity of higher-order cue representations is partially inhibited by active lower-order cue representations. When the external cues are shut off (Gibson), the tendency for data to be coded in the highest-order, and most selectively tuned, populations is released from inhibition by lower order activities. To test this idea, experiments should be designed wherein increasing the strength of certain cues in STM weakens the strength in STM of related cue combinations. For example, increasing the duration of certain cues (and presumably their strength in STM) might well weaken the strength of related cues presented later. In particular, the Gibson indirect effect might well be greater if the inspection line is presented longer.

A detailed synthesis of how the internal representations of present events interact with the stored STM representations of past events in the hierarchy of discriminative cells falls beyond the scope of this paper. Nonetheless, the above results sharpen the idea that common mechanisms, such as recurrent on-center off-surround networks undergoing mass action interactions, are operating on several, if not all, levels of the hierarchy.

REFERENCES

In the Blakemore study, the orientation-sensitive populations were arranged in a circle, as described in section 4. Here \( n = 90 \), and there are two reasons for using such a large number of populations: (1) the spread of excitatory and inhibitory interactions must be made sufficiently wide to achieve the conditions \( C(0) - C(1) \geq 0 \), \( C(1) - C(1 + 1) \geq 0 \), and \( D(1) - D(1 + 1) \) relatively large; (2) in Blakemore et al. (1970), the maximum angle expansion is \( 2^\circ \), and if the whole circle of orientations is \( 180^\circ \) wide, then \( 2^\circ \) is the difference in presentation between neighboring populations if \( n = 90 \).

In the first simulation (Table 1), both excitatory and inhibitory inputs were present. The parameters were chosen so that the recurrent network was approximately a nonrecurrent network, i.e., inputs were intense relative to the maximum possible \( f(x) \). Parameter \( A \) was chosen small to facilitate equation (15), and the \( E_i \) were all set to 0.

In the second simulation (Table 2), inputs were purely excitatory. The inputs were less intense than above, so as not to dominate recurrent inhibition. The strength of self-inhibition was made three times as great as that of self-excitation to balance the excitatory inputs. Note that the inward peak shift at \( T = 0 \) and 0.2 in Fig. 10 (cf. equation (12)) becomes an outward peak shift at \( T = 0.6 \) when the recurrent inhibition takes effect.

**APPENDIX A**

**Blakemore Simulation**

The computer programs used in both appendices were written to numerically solve system (8) for chosen parameter ranges. Both programs employ the Runge–Kutta method, they were both written in FORTRAN and run on an IBM 360 and PDP-9. Programs are available on request by writing to Dr. Levine.

**Table 1**

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<th>Intense excitatory and inhibitory inputs simulate angle expansion</th>
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**Table 2**

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The populations were again arranged in a circle. One of the populations ($i = 6$) was assumed to code vertical, and $B_i$ was a Gaussian function of the distance modulo 11 between $i$ and 6. A sustained stimulus line was delivered to a population coding a nearby vertical line ($i = 5$) as

$$A = e^{-w - x^2}, \quad a = 4, 4.5,$$

$p = \frac{1}{2(l-1)}, \quad (k-l)$ means distance mod 11

### APPENDIX 2

**Gilson Simulation**

**Parameters for neutralization simulation**

**Table 3**

![Diagram of Fig. 10: Inward peak shift becomes outward as recurrent inhibition builds up.](image)

![Diagram of Fig. 11: Fast and nonexistent peak shifts at small and large standard deviations of maximal excitability.](image)
both an excitatory and inhibitory input. If a drift of maximal \( x_i \) occurred, it always was in the direction of the vertical population.

The excitatory and inhibitory interaction strengths were fixed Gaussians and the sums of the two Gaussians were the same. The sum of the Gaussian defining \( B_i \) was kept constant and its standard deviation was varied. The drift in peak response was either nonexistent, slow, or fast, as the standard deviation was decreased (Table 3). The graphs are shown in Figs 11 and 12.

**Fig. 12.** Slow peak shift at an intermediate standard deviation of maximal excitability.