

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. Analogs 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 analyse 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

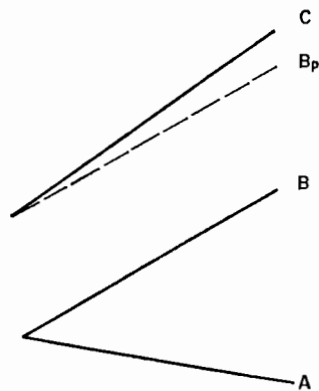


FIG. 1. Angle expansion due to an acute angle between A and B. Line B_p is parallel to B. Line C is perceived as parallel to B.

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 code orientations that are further apart, then the peak shift represents a perceived angle expansion.

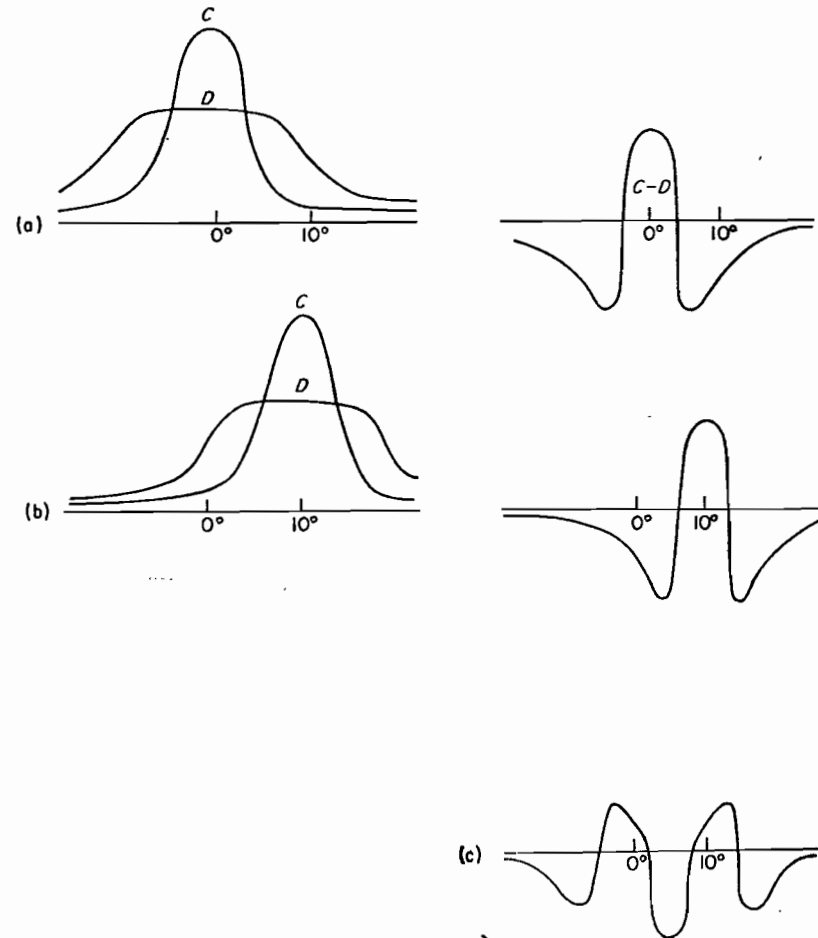


FIG. 2. Excitatory (C) and inhibitory (D) gradients that are centered at 0° and 10° add in (a) and (b), respectively. The two net gradients add to yield a peak shift in (c). After Blakemore *et al.* (1970).

There is a similarity, noted both by Blakemore *et al.* (1970) and by Coltheart (1971*b*), 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

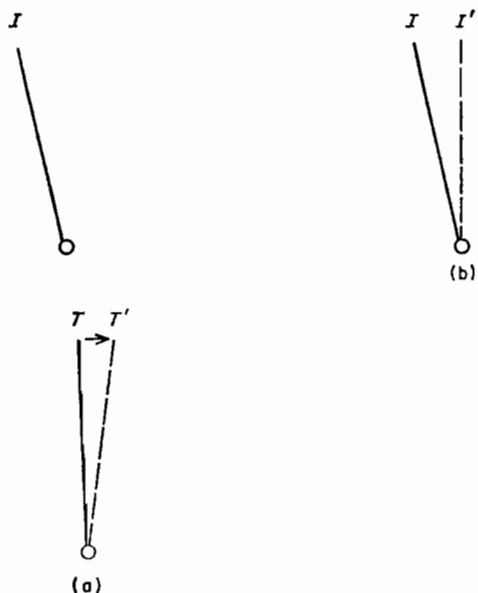


FIG. 3. Tilt after-effect of T due to prior inspection of I (a); neutralization of I to I' due to sustained inspection of I (b).

“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 (1971*a*). 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 , $i = 1, 2, \dots, n$, receive inputs $I_i(t)$ 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 $x_i(t)$, 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

$$\dot{x}_i = -Ax_i + (B - x_i)I_i, \quad (1)$$

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 θ_i values? Grossberg (1973) notes that at high I values, the pattern of θ_i 's is lost due to saturation of all x_i at B , whereas at low I values, the pattern of θ_i 's is confounded by any noise that exists in the system.

How can a better record of the θ_i 's in x_i responses be achieved? Interactions between inputs and/or states must occur, because the θ_i 's are defined by an interaction of all the inputs I_k , $k = 1, 2, \dots, n$; 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)

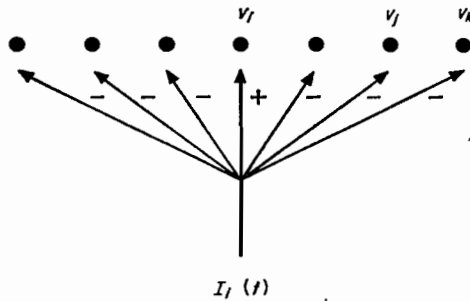


FIG. 4. Nonrecurrent on-center off-surround network.

becomes

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

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

$$x_i = \theta_i \frac{BI}{A + I}. \quad (3)$$

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

$$\left(-x_i \sum_{k \neq i} I_k\right)$$

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

$$x = \sum_{k=1}^n x_k$$

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

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

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

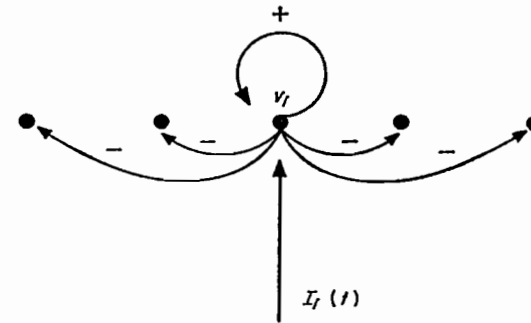


FIG. 5. Recurrent on-center off-surround network.

I_i in equation (4) is the excitatory input to v_i , and J_i is the inhibitory input to v_i ; e.g.,

$$J_i = \sum_{k \neq i} I_k,$$

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?

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

$$\dot{x}_i = -Ax_i + (B_i - x_i) \left[\sum_{k=1}^n f(x_k)C_{ki} + I_i \right] - x_i \left[\sum_{k=1}^n g(y_k)D_{ki} + J_i \right] \quad (5)$$

$$\dot{y}_i = -Ey_i + \sum_{k=1}^n x_k F_{ki}, \quad (6)$$

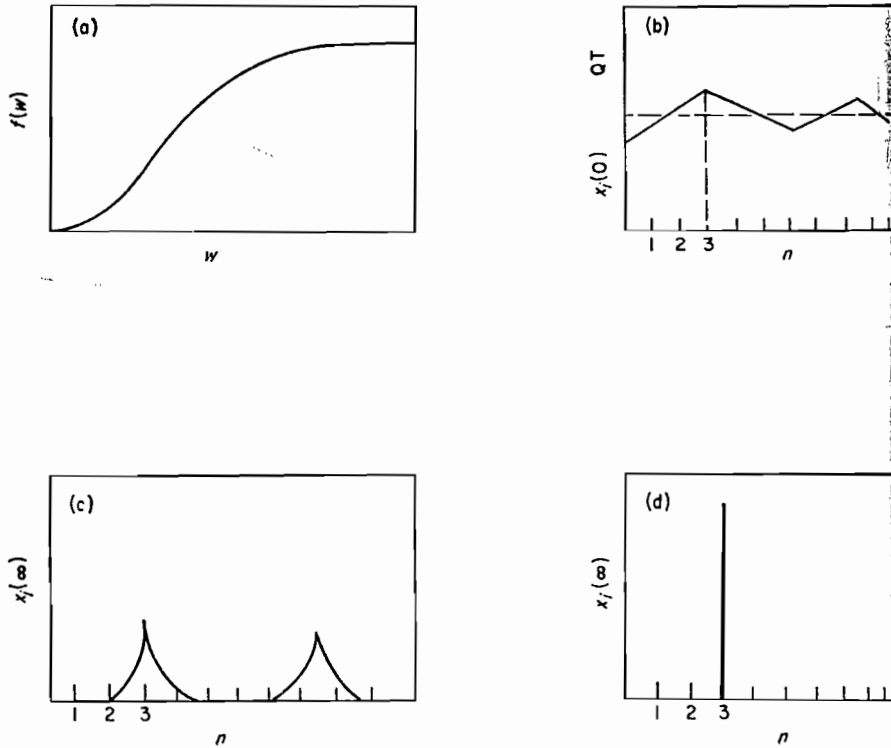


FIG. 6. Dependence of contrast enhancement in STM on choice of signal function.

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

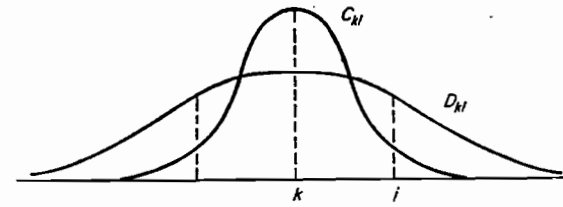


FIG. 7. Fall-off of excitatory (C) and inhibitory (D) connection strengths as a function of distance.

behavioral generalization gradients are built. There are also 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_{ki} will correspondingly be assumed to decrease with distance faster than the inhibitory coefficients D_{ki} . 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 analyse the simpler networks

$$\begin{aligned} \dot{x}_i = & -Ax_i + (B_i - x_i) \left[\sum_{k=1}^n f(x_k)C_{ki} + I_i \right] \\ & - x_i \left[\sum_{k=1}^n g(x_k)D_{ki} + J_i \right] \end{aligned} \quad (7)$$

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

$$\begin{aligned} \dot{x}_i = & -Ax_i + (B_i - x_i) \left[\sum_{k=1}^n f(x_k)C_{ki} + I_i \right] \\ & - (x_i + E) \left[\sum_{k=1}^n g(x_k)D_{ki} + J_i \right], \end{aligned} \quad (8)$$

$E > 0$, in which the inhibitory equilibrium level $-E$ and the passive equilibrium level 0 are different; E is related to the Nernst potential for

potassium (Hodgkin, 1964; Katz, 1966). Indeed, each term in equation (8) can be interpreted as a voltage times a conductance.

Grossberg & Levine (1975) study a special case of equation (7) that is important below; namely

$$\dot{x}_i = -Ax_i + (B_i - x_i)[f(x_i) + I_i] - x_i \left[\sum_{k \neq i} f(x_k) + J_i \right], \quad (9)$$

where the number of excitable sites B_i in each v_i can be arbitrary positive numbers. They prove that the v_i with the largest B_i tend to totally quench the STM of v_j with smaller B_j , other things equal. Total quenching is due to the fact that each v_i can inhibit all v_k , $k \neq i$, with equal strength $f(x_i)$. Section 5 shows that when populations interact as in Fig. 7, then the total quenching becomes partial and is interpreted as line neutralization.

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:

$$\dot{x}_i = -Ax_i + \sum_{m=1}^n K_m C_{mi} - \sum_{m=1}^n K_m D_{mi}. \quad (10)$$

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 $K_m =$ some positive constant K 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 ($\dot{x}_i = 0$), equation (10) implies

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

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 $x_{l+1} > x_l$. This is the same as

$$[D(l) - D(l+1)] - [C(l) - C(l+1)] > [C(0) - C(1)] - [D(0) - D(1)]. \quad (11)$$

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 $C(l) \cong 0$, and thus that $C(l) - C(l+1) \cong 0$, 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 $C(i)$ and $D(i)$; as the breadth increases, the likelihood that $D(0) \cong D(1)$ 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

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

all $J_i = 0$, $K_0 = K_l = K$, and $K_m = 0$ otherwise. In particular, the excitatory coefficients C_{mi} were chosen to have a Gaussian distribution; e.g., for $|m-i| < n/2$, let $C_{mi} = G \exp\{-H^{-2}|m-i|^2\}$. The inhibitory coefficients D_{mi} were similarly distributed. No inhibitory inputs existed, but inhibition spread through the network via the recurrent off-surround. 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

$$\dot{x}_i = -Ax_i + K(B - x_i)[C(i) + C(i-l)],$$

with equilibrium value

$$x_i = \frac{BK[C(i) + C(i-l)]}{A + K[C(i) + C(i-l)]}. \quad (12)$$

Since x_i 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_i 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

$$\dot{x}_i = -Ax_i + (B - x_i) \sum_{m=1}^n K_m C_{mi} - (x_i + E) \sum_{m=1}^n K_m D_{mi}.$$

At equilibrium,

$$x_i = \frac{\sum_{m=1}^n K_m (BC_{mi} - ED_{mi})}{A + \sum_{m=1}^n K_m (C_{mi} + D_{mi})}. \quad (13)$$

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,

$$x_i = \frac{BKC(i)}{A + K[C(i) + D(i)]}.$$

Interpolate a continuous function $x(s)$ through x_i by letting

$$x(s) = \frac{BKC(s)}{A + K[C(s) + D(s)]},$$

and suppose that $C(s)$ falls faster than $D(s)$, as in Fig. 7, in the sense that

$$C^{-1}(s)\dot{C}(s) < D^{-1}(s)\dot{D}(s). \quad (14)$$

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 $\dot{x}(s) = 0$ at some other value of s ,

$$C^{-1}(s)\dot{C}(s) = [AK^{-1} + D(s)]^{-1}\dot{D}(s) > D^{-1}(s)\dot{D}(s),$$

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,

$$x_i = \frac{BK[C(i) + C(i-l)]}{A + K[C(i) + C(i-l) + D(i) + D(i-l)]}.$$

An outward peak shift occurs if $x_{l+1} > x_l$, which is the same as

$$C(l+1)D(l) + C(l)D(l) + D(0)C(l+1) + D(0)C(l) - C(l)D(l+1) - C(l)D(l) - C(0)D(l+1) - C(0)D(l) > F[C(l) - C(l+1) + C(0) - C(1)], \quad (15)$$

with $F = AK^{-1}$. As in the case of additive interactions, such an outcome is facilitated by the conditions $C(0) \cong C(1)$, $C(l) \cong C(l+1)$, $D(0) \cong D(1)$, 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 $C(0) \cong C(1)$ implies $D(0) \cong D(1)$, and values of l exist such that $C(l) \cong C(l+1)$ 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 $m = 0$. Then at equilibrium, equation (13) implies

$$x_i = \frac{K[BC(i) - ED(i)]}{A + K[C(i) + D(i)]}.$$

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

$$F[BC\dot{s}(s) - E\dot{D}(s)] = (B + E)[C(s)\dot{D}(s) - \dot{C}(s)D(s)]. \quad (16)$$

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 $\dot{C}(s)$ and $\dot{D}(s)$ are negative. Equality in equation (16) can be achieved at values s where $\dot{C}(s) \cong 0$ and $\dot{D}(s)$ is sufficiently negative to match the right-hand side; for example, where $C(s)$ 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

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

Suppose $x_{l+1} > x_l$. Defining $U_0 = C(l) + C(0)$, $U_1 = C(l+1) + C(1)$, $V_0 = D(l) + D(0)$, and $V_1 = D(l+1) + D(1)$, 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 $B_l = A$,

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

and $D_{mi} = G_2 \exp\{-H_2^{-2}|m-i|^2\}$. 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 excitable 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 populations which subserve 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 $B_i = \lambda_i B$, equation (8) can be transformed, using the change of variable $q_i = \lambda_i^{-1} x_i$, into system

$$\begin{aligned} \dot{q}_i = & -Aq_i + (B - q_i) \left[\sum_{m=1}^n f(q_m) \hat{C}_{mi} + I_i \right] \\ & - q_i \left[\sum_{m=1}^n f(q_m) \hat{D}_{mi} + J_i \right], \end{aligned} \quad (22)$$

where $\hat{C}_{mi} = \lambda_m^2 C_{mi}$ and $\hat{D}_{mi} = \lambda_m^2 D_{mi}$. 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_j which codes a nearly vertical orientation. This input creates a locus of maximal excitation at v_j and we study whether, and how fast, this locus drifts towards the vertical. For small values of H , there is no drift; for intermediate values of H , the drift is slow; and for large values of H , 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_j 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 normalization, 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 environment 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 experiments, 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

sense, 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 (1971*a*) 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 representation 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, 1971*b*) 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

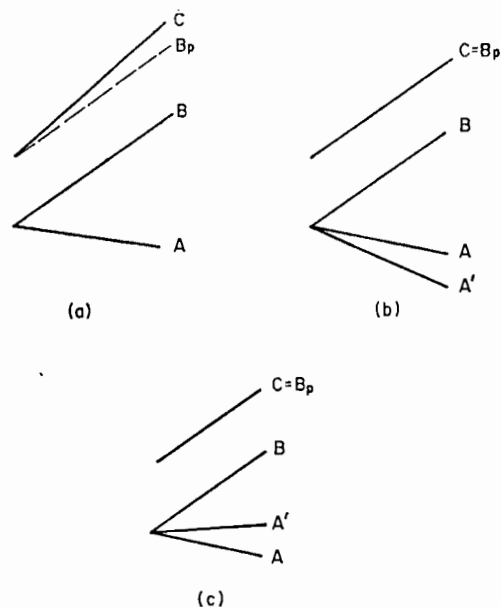


FIG. 8. Disinhibition by a third line (A') of the angle expansion that would otherwise show at C due to A and B .

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 disinhibition 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 (especially) of the adjacent line, thereby reducing 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

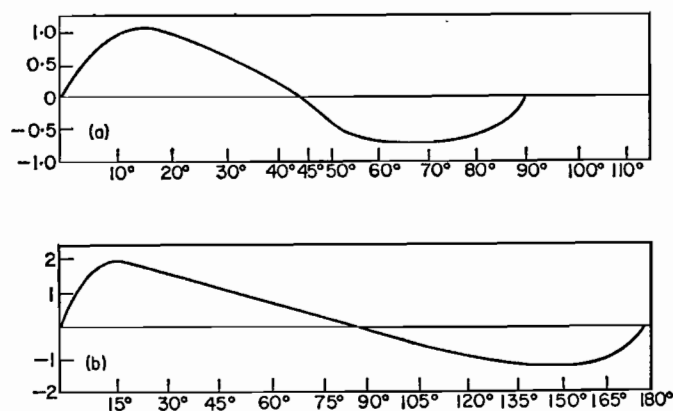


FIG. 9. Dependence of Gibson & Blakemore illusions on angle.

line. By the above arguments, test lines within at least $\pm 45^\circ$ of this orientation will be displaced away from the inspection orientation. Imagine that the inspection line excites the appropriate hypercomplex cells, which feed back to cells coded at $\pm 90^\circ$ from the inspection orientation. These indirectly excited cells will also cause an outward shift in the perceived orientation of test lines within $\pm 45^\circ$ of them. The directly and indirectly excited cells hereby break orientation space into four quadrants.

If this argument is accepted, then it remains to ask why the hypercomplex cells do not break up orientation space into four quadrants in the Blakemore experiments, where instead orientation space is broken into two halves? The two situations differ in terms of two kinds of parameters: simultaneous versus successive presentations of lines, and time scales for the effects to develop. The second of these differences is probably less important, considering Coltheart's claim that after-effects can occur even if the inspection line is only briefly viewed. The main difference, therefore, is that Gibson-type experiments, because of the successive presentation, depend on a memory effect, presumably a STM effect, whereby the stored representation of a past line interacts with the perceived representation of a present line. We are therefore led to consider the following conceptual framework to unify this discussion.

Suppose that a tug-of-war exists between the data already stored in STM and the demands of present events. For example, suppose that a list is stored in STM ready for retrieval and an incompatible event occurs. How is a consensus between the old and new data generated? In particular, how does the new event weaken the STM activities of the old incompatible events?

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.

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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.

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 $C(0) - C(1) \cong 0$, $C(l) - C(l+1) \cong 0$, and $D(l) - D(l+1)$ relatively large; (2) in Blakemore *et al.* (1970), the maximum angle expansion is 2° , and if the whole circle of orientations is 180° wide, then 2° 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

TABLE 1

Intense excitatory and inhibitory inputs simulate angle expansion

A	0.05
B	1
E	0
C_{ki}	$e^{-(k-i)^2/7^2}$
D_{ki}	$e^{-(k-i)^2/8^2}$
I_i	$3(C_{39, i} + C_{52, i})$
J_i	$3(D_{39, i} + D_{52, i})$

$k-i$ means distance mod 90

approximately a nonrecurrent network, i.e., inputs were intense relative to the maximum possible $f(x_i)$. 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 $T = 0.1$ and 0.2 in Fig. 10 [cf. equation (12)] becomes an outward peak shift at $T = 0.6$ when the recurrent inhibition takes effect.

TABLE 2

Excitatory inputs balanced against recurrent interactions simulate angle expansion

A	0.05
B	1
E	0.1
C_{ki}	$e^{-(k-i)^2/7^2}$
D_{ki}	$4e^{-(k-i)^2/8^2}$
I_i	$0.5(C_{39, i} + C_{52, i})$
J_i	0

$k-i$ means distance mod 90

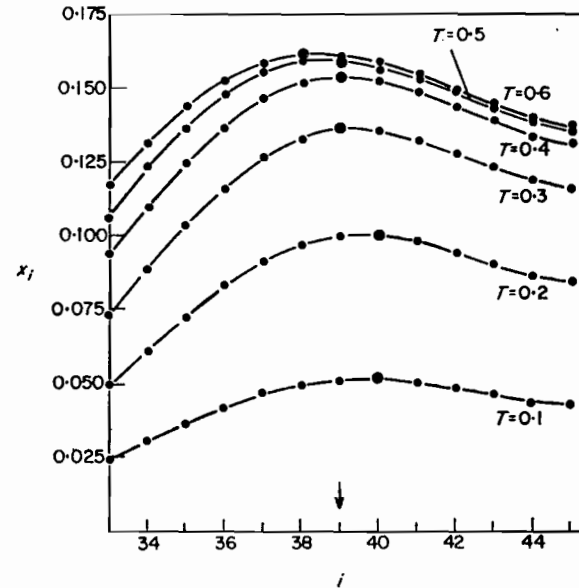


FIG. 10. Inward peak shift becomes outward as recurrent inhibition builds up.

APPENDIX B

Gibson Simulation

Here $n = 11$, and 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 nearly vertical line ($i = 5$) as

TABLE 3

Parameters for neutralization simulation

A	1
B_i	$2e^{-(i-6)^2/\sigma^2}$, $\sigma = 4, 4.4, 5$
E_i	0
C_{ki}	$e^{-(k-i)^2/4^2}$
D_{ki}	$e^{-(k-i)^2/5^2}$
I_i	C_{5i}
J_i	D_{5i}

} $k-i$ means distance mod 11

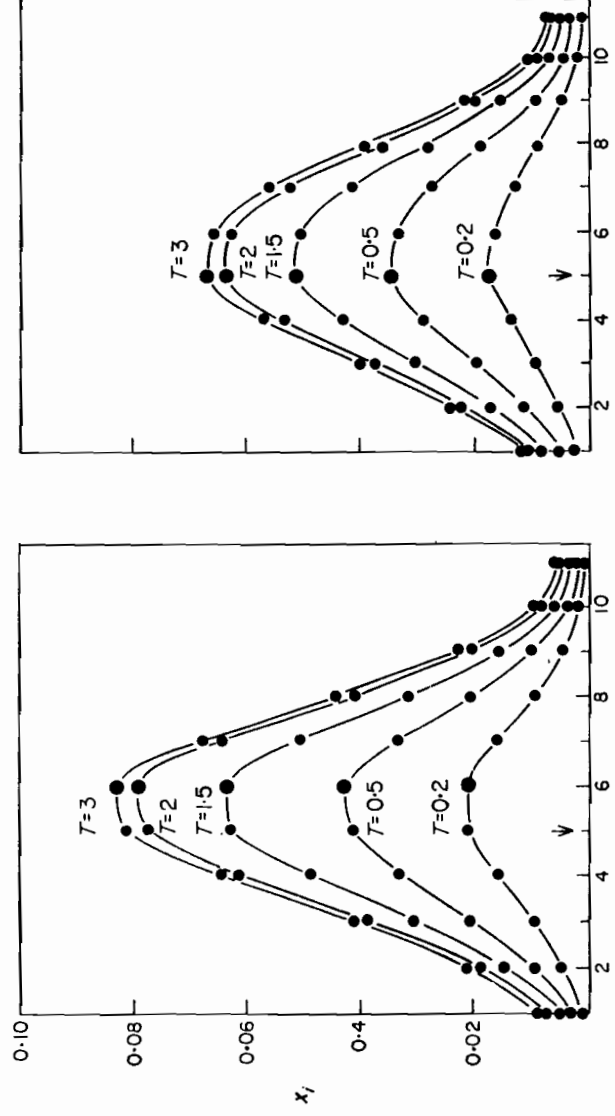


FIG. 11. Fast and non-existent peak shifts at small and large standard deviations of maximal excitability.

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.

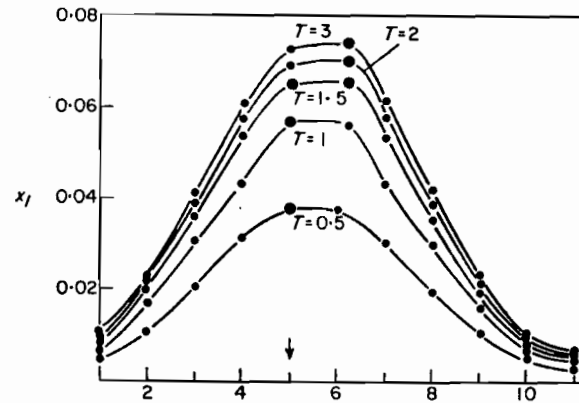


FIG. 12. Slow peak shift at an intermediate standard deviation of maximal excitability.