THE SUPERIOR COLLICULUS AS A DECISION SYSTEM CONTROLLING EYE MOVEMENT[†]

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COINS Technical Report 74C-3

(June 1974)

Abstract

The decision making process necessary to select one location in the visual scene as a new target for orientation is investigated. The analysis shows how a network, receiving spatially patterned input, corresponding to the spatial positions of objects in the visual field, can generate a single locus of activity corresponding to the location of the desired target. It is shown that decision-making processes of this type require interaction between systems that are unstable over some range of operation. The neural system is described by a set of analytic equations, and the necessary conditions for decision-making are derived.

[†]This work was supported in part by NIH Grant No. 5 R01 NS09755-03 COM awarded to M. A. Arbib, Computer and Information Sciences, University of Massachusetts, Amherst, Mass., and by the Neurosciences Research Program, Jamaica Plain, Mass.

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1. Introduction

Active search for visual input, as expressed by scanning of the world by eyes and head, is one of the important aspects of visual perception. These scanning movements occur in almost all situations and are modified by the visual input (Yarbus, 1967). In the simplest situation, a flash of light causes orientation of the eyes in the direction of the flash. In the more complex, real-world situation, the eyes move from object to object and from feature to feature, with scanpaths that differ for different scenes but may show some similarities for repetitions of the same scene.

In this paper a model of an eye-movement controller is developed. The controller is hypothesized to be a two-layered neuronal network, representing the superior colliculus (one of the centers concerned with eye movement control, as will be discussed later). This controller receives spatial patterns of intensities from various visual areas, such as the primary visual cortex, and supplies a spatial pattern of intensities as output to motor centers.

The formalisation of the neural network, and of its input and output, is based on available neuroanatomical and neurophysiological data (see Sec. 2.2). An analysis of the dynamics of such a network is carried out, and the conditions that will produce the desired spatio-temporal output

sequence are delineated. The effects of external visual input and internally generated hypotheses are examined. The concept of spatial bias is introduced, and this, together with adaptation, is hypothesized to be the basis of the cyclic scanpaths observed by Noton and Stark (1971). On the basis of this model, a possible mechanism for the occurence of saccades during REM (rapid eye movement) sleep is discussed.

2. Biological Basis of the Model

2.1. Eye-Movement Patterns

What features of the visual input attract an eye fixation? A fixation may be in response to a bright flash, that is, a simple intensity stimulus, and animals without visual cortex (the pattern-analysing region, according to traditional belief) retain their ability to fixate a bright patch or a moving object (Humphrey, 1970; Sprague and Meikle, 1965).

Feature clusters may also attract fixations. Yarbus (1967) showed that, when scanning paintings, a major fraction of the fixations were on the representations of objects, leaving bright but relatively featureless areas unfixated. Individual features, such as lines or angles, are not usually the elements that are fixated. Rather, the fixation may fall in between a number of features (Noton and Stark, 1971; Dev, unpublished experiments) but so as to permit a number of features to fall within the high-resolution foveal area.

However, a region of high feature concentration is not always sufficient to attract a fixation. Whole areas of high feature density, for example, trees in a forest, may be ignored in favor of a region of lower feature density, such as the figure of a man (Yarbus, 1967). In problem-solving situations (Newell and Simon, 1967; Yarbus, 1967), regions of

high fixation density can be drastically altered depending on the problem under consideration. In this case, eye movements may be driven either by the expectation of a piece of data in that location, that is, by a hypothesis, or because the region being fixated is the one with the most new information. Thus, information may also drive eye movements.

Saccadic eye movements also occur in the dark or during sleep. Eye movements in the dark show a large, side-to-side, horizontal component. During sleep, eye movements are greatly reduced, corresponding to a lower level of arousal. However, during the REM phase of sleep, the number of eye movements is greatly increased. This suggests that, besides visual sensory input to the eye-movement controller, there may be some component of random or noise input. This could be a non-specific input, such as an arousal input from the reticular formation, and is perhaps a random spatial pattern of neuronal activity.

Thus, the eye-movement controller receives input of varying degrees of complexity -- a single flash, a feature cluster, a hypothesis -- as well as a noise input. It is hypothesized that the sensory input to the controller overrides the noise input as long as no adaptation to the sensory input occurs, but that when the sensory input has been adapted to or is absent, the noise input provides the drive for the exploratory eye movements that are always seen in the awake animal. Figure 1 schematizes the basic structure of the eye-movement controller.

2. Neurological Data Relevant to Visual Search

Aside from the loss of eye movement produced by lesions of the motor nuclei (III, IV and VI) or of the surrounding areas, gross defects in eye movements are observed for lesions of the superior colliculus (Sprague and

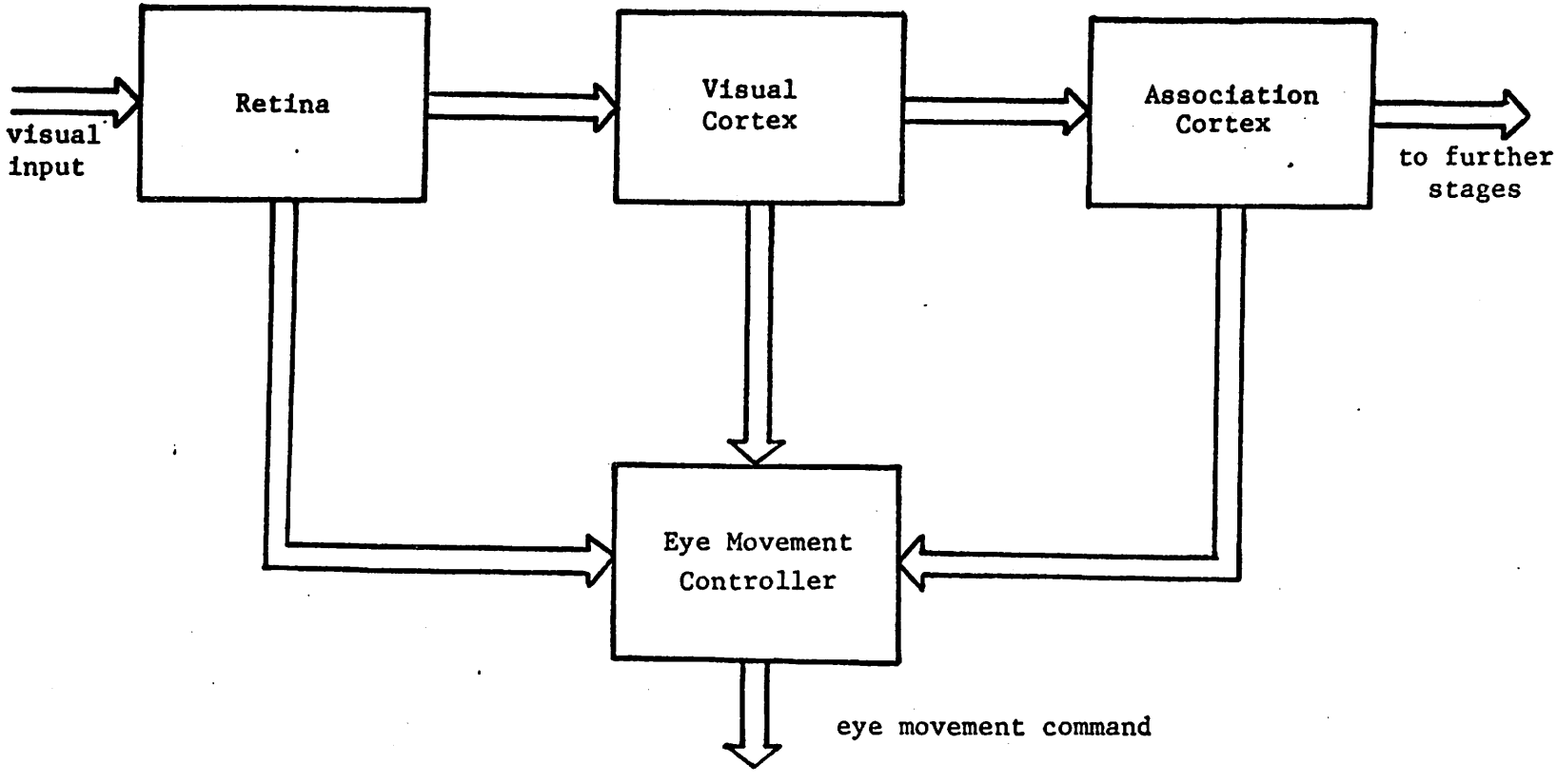


Fig. 1. Block Diagram Illustrating the Inputs to the Eye Movement Controller

Meikle, 1965; Humphrey, 1970), pretectum (Sprague et al., 1970), occipital cortex (Crosby et al., 1962), the frontal eye fields (Robinson, 1968), and the internal medullary lamina (Schlag et al, 1970). Eye movements are elicited by stimulation of the superior colliculus (Apter, 1945; Schaeffer, 1970), frontal eye fields (Robinson, 1968), and the internal medullary lamina (Schlag and Schlag-Rey, 1971).

The question arises whether any one of these areas is the main controller for the oculomotor system, with the others supplying a modifying influence either to the controller or to the oculomotor areas, or whether they all have independent control and the net result is the interaction of all these influences.

Figure 2 outlines the anatomical connections relevant to eye movement control in the monkey. It is possible to include other pathways, for example, connections to the ventral lateral geniculate or to the limbic system, but initially we assume that any effect they have on the oculomotor system is generally a modifying one, and we ignore possible situations in which these centers may become prepotent.

As can be seen from the diagram there are many inputs to the oculomotor area. However, there is no known connection directly from the retina to the oculomotor region, though Oyster et al. (1972) suggest that at least a functional direct path exists for the rabbit.

The most direct route appears to be via the superior colliculus and the pretectum. The neuroanatomical data confirm the behavioural data cited earlier that the controller, that is, the superior colliculus receives inputs from many regions, corresponding to information at different stages of processing.

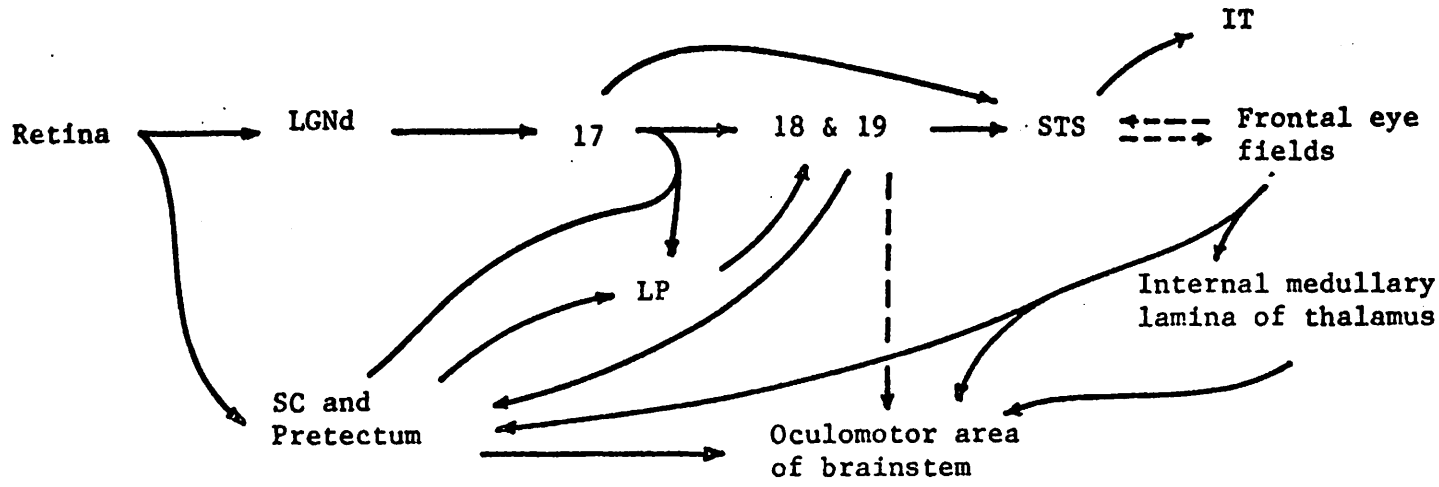


Fig. 2. Outline of Neuroanatomical Connections relevant to Eye Movement Control in the Monkey (IT: inferotemporal cortex; LGNd: dorsal lateral geniculate nucleus; LP: latero-posterior nucleus; SC: superior colliculus; STS: superior temporal sulcus; 17,18 & 19: areas of visual cortex)

2.3. The Superior Colliculus

While stimulation and recording experiments both show the involvement of the superior colliculus in eye movements (Schaeffer, 1970; Robinson, 1972; Schiller and Koerner, 1971; Goldberg and Wurtz, 1972), ablation of the superior colliculus does not eliminate eye movements (Sprague and Meikle, 1965; Wurtz and Goldberg, 1972). In the monkey, focal lesions in the lower layers of the superior colliculus cause an increased latency in the generation of eye movements, but the movement when made is brisk and accurate. Larger lesions cause a slight decrease in speed but no loss in accuracy. In the cat, however, total unilateral lesion of the superior colliculus results in neglect of the contralateral visual field except for violently moving objects. Horizontal following upto 30 degrees beyond the midline (in cat) is possible for slow moving objects, but fast moving objects are lost earlier. A species difference appears to exist -- Denny-Brown (1962) found no deficit in pursuit eye movements for unilateral colliculectomy in the monkey.

Thus the data indicate that the superior colliculus is not the only structure mediating eye movements. However, it appears to be necessary for rapid response to a visual stimulus, whether stationary or moving. Wurtz and Goldberg (1972) suggest that the superior colliculus acts to limit the area of the visual field to which attention should be directed, leaving other systems to carry out more accurate fixation, instead of directing the fixation itself. In the model that we develop, we will show that besides serving to limit the visual area of interest, the superior colliculus, by means of unstable loops, may also supply the tone necessary for rapid eye movement responses.

Further, random changes in noise input to the superior colliculus (in contrast to the more structured changes of visual input) cause these unstable loops to generate the exploratory drive which is missing in animals without the superior colliculus.

The superior colliculus receives a retinotopic projection from retinal ganglion cells, from striate cortex (area 17) and from peristriate cortex (areas 18 and 19) (Apter, 1945; Abplanalp, 1970; Cynader and Berman, 1972; etc.). The importance of the cortical projection increases with phylogeny (Crosby et al., 1962) until in the monkey, the foveal part of the retina does not project to the superior colliculus at all (Wilson and Toyne, 1970). This retinotopic organisation, rather than a random one, greatly simplifies the mechanism of any control system directing attention to locations in space.

The superior colliculus shows a dissociation of function between its superficial and deeper layers. In the cat, lesion of the superficial layers or lesion of the retinal or cortical input tracts to the superior colliculus causes visual neglect and apparent inability to see an object in the visual field (Sprague and Meikle, 1965). However, species differences may occur -- Schneider (1969) shows that, in the hamster, pattern recognition can still take place. Lesions of the deeper layers, of the efferent pathways of the superior colliculus, or of the tegmentum result in motor deficits such as circling and misreaching. Anatomically, the superficial layers are the ones which receive retinal and cortical input, while the deeper layers supply output to the tegmentum and reticular formation. A similar structural and functional dissociation occurs in the tree shrew (Casagrande, 1972), and possibly in other mammals. (Note that

the phrase 'deeper layers' is used to indicate both intermediate and deep collicular layers.)

Single-cell recording reveals a similar difference between superficial and deeper layers (Goldberg and Wurtz, 1972; Wurtz and Goldberg, 1972). Neurons in the superficial layers possess visual receptive fields and show little or connection with motor activity. Some are inhibited during an eye movement but show no specificity for direction or length of eye movement. A little deeper, but still within the superficial layer, some neurons show enhancement of response if the stimulus within the receptive field is the one fixated next. The response does not occur without a visual stimulus. In deeper layers, however, the response of a neuron is correlated with an eye movement of specific length and direction -- a movement that would result in foveation of a stimulus in the receptive field of that neuron. This response occurs both for a visual stimulus and in darkness. The response precedes the eye movement by 30-50 msec. for some neurons and upto 200-300 msec. for others.

The size of movement fields in the deeper layers is large, at least 10-20 degrees in diameter at 20 degrees from the fixation point. Receptive fields for neurons in the superficial layers are large too -- less than a degree in diameter at the fovea to 10-30 degrees at the periphery, and often even larger. They show insensitivity to size or shape of stimulus.

The superior colliculus can therefore be modelled as a two-stage system. The first stage, consisting of the superficial layers, is sensory in nature, receiving mostly visual input at various stages of processing,

and is hypothesized to be an evaluation stage. The second stage consists of the deeper layers and is hypothesized to be a decision stage, closely coupled to subsequent motor stages. Inputs from other modalities, such as auditory, to these deeper layers is a superposition onto this stage of decisions taken elsewhere.

3. Formulation of the Model

3.1. The Basic Model

The superior colliculus can be considered as one of several controllers influencing the plant, or the oculomotor system. We are interested in modelling the structure of this controller using data on the neural structure of the superior colliculus, and in observing whether the output of the model controller is able to generate eye-movement commands characteristic of visual search (Fig. 3).

The plant, that is, the oculomotor system, is multi-input single-output. The inputs are from the superior colliculus as well as the frontal eye fields, the internal medullary lamina of the thalamus, and striate, peristriate, and temporal cortex. The output is a command to the eye muscles. (At present we have ignored the commands to the rest of the body, transmitted through the reticulospinal and other tracts. These mediate whole body orientation in correlation with eye movements.)

The superior colliculus controller is modelled as a two-stage system, with its properties being determined by the neural connectivity within each stage. The first stage, or evaluation stage, consists of an array of neurons which receives inputs from retina, striate cortex and peristriate cortex (all of which are highly correlated), integrates them at each point on the array (the possible interactions between these inputs are developed

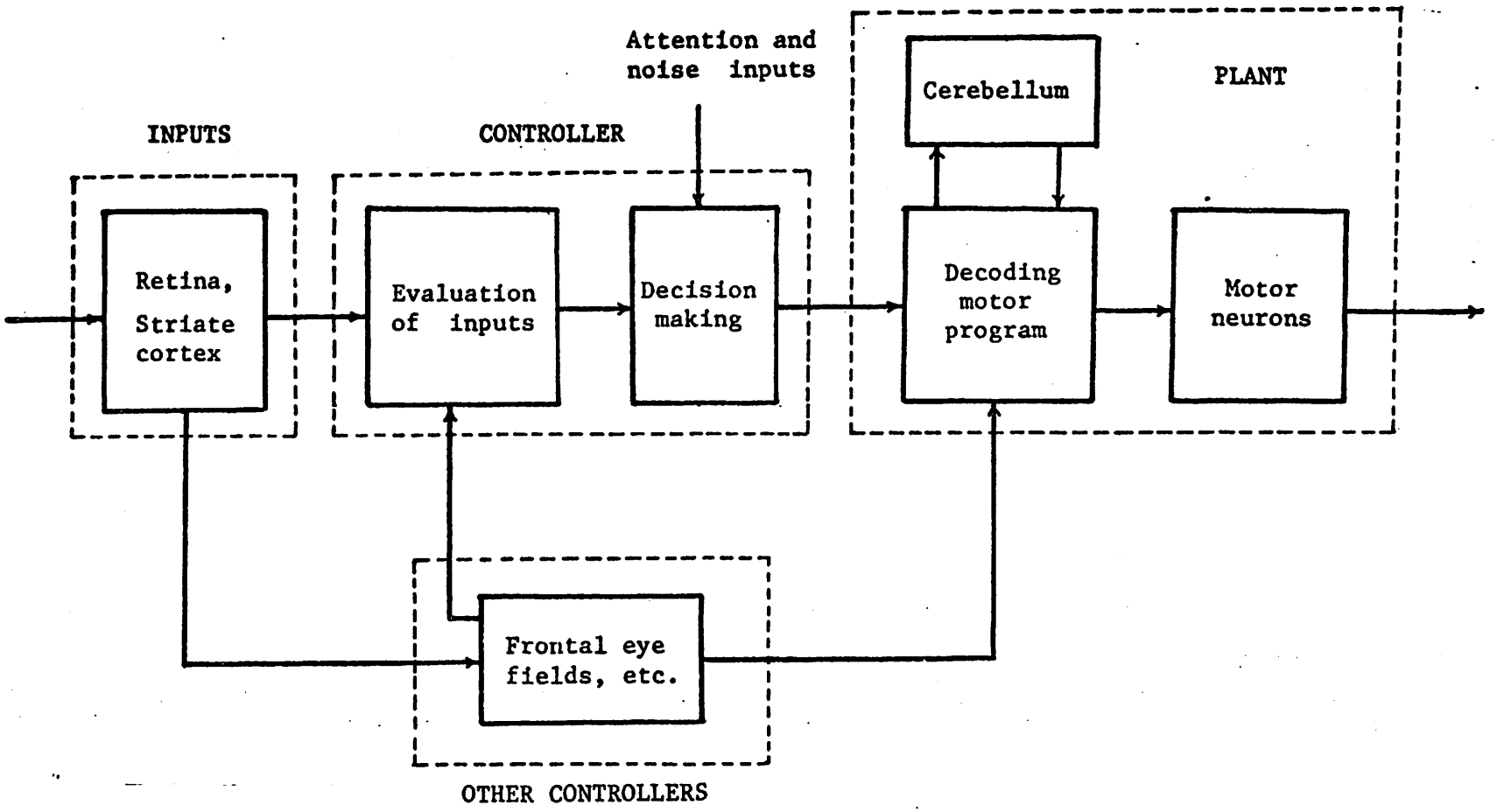


Fig. 3. The Basic Model

in a later section), and allows them to interact over the array. The output of the evaluation stage is a spatial pattern of activity over this array with peaks of activity whose intensities serve to rank-order the various inputs to this stage. The decision about which of the peaks is to control eye movements is taken by the decision stage, and its output is a spatial array with a single intense peak of activity.

The assumption that the output of the superior colliculus controller is a single peak in the spatial pattern of activity is based on the data of Schiller and Koerner (1971) and Wurtz and Goldberg (1972) which indicate that the spatial location of an activity peak is the relevant variable in the control vector for saccadic eye movements. The temporal sequence of the locations of the peaks is assumed to provide the motor command for visual scanning. This model of the eye-movement controller ties in with the concept of distributed motor control (Arbib, 1972).

3.2. Neural Representation of Model

Data on neural interconnections within the superior colliculus is sparse. Also, the number of layers in the superior colliculus and the connections within and between layers vary from species to species (Karten, 1972). Electron-microscopic studies of ultrastructure in the superior colliculus have shown the presence of rich dendro-dendritic connections in the superficial layers (Szekely, 1971), but their function is not yet understood.

Because of the scarcity of data, the simplest neural representations consistent with the data available are chosen for both the evaluation and the decision stage.

The Evaluation Stage

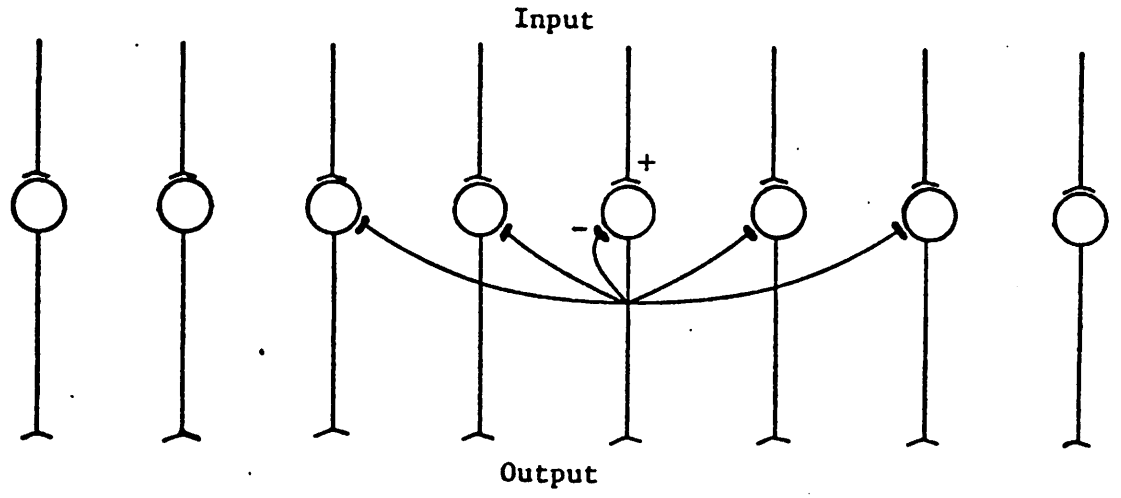
This stage is assumed to be a layer of neurons with inhibitory interactions between these neurons (Fig. 4a). Input to the array is retinotopic, that is, each location on the array corresponds to a location on the retina. Because the representation at each stage of visual processing remains retinotopic, the inputs from all these stages to the superior colliculus are in register with each other.

The processing capabilities of each stage depend on the connectivity between its neurons. The parameters to be considered are the form of the inhibitory weighting function and the region over which a neuron can inhibit others. In the next section, a mathematical analysis is carried out to determine the properties of the evaluation stage.

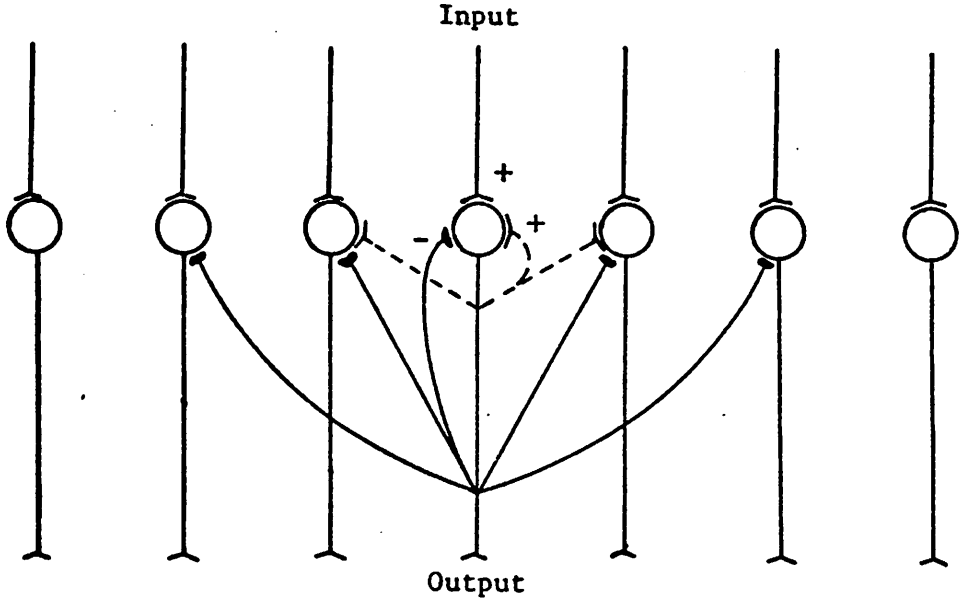
The Decision Stage

This stage is assumed to be a layer of neurons receiving a retinotopic projection from the previous stage, that is, the evaluation stage. (Neuroanatomically, it has been shown that each region in the superficial layers projects to the immediately underlying region in the deep layers.) Each neuron provides excitatory feedback to itself, thus setting up a reverberatory loop (Fig. 4b). Each neuron also inhibits all other neurons in the array in a diffuse manner. (Because neurons cannot be both excitatory and inhibitory, in mammals inhibitory feedback is probably mediated by a group of inhibitory neurons whose inhibitory output is proportional to the activity in the excitatory neurons.)

Output from the decision stage is to the oculomotor area which eventually recodes the 'move' command into a motorneuronal pattern. Modifications and extensions of this basic model are considered in sec. 5.



a) The Inhibitory Connectivity of a Neuron in the Evaluation Stage



b) The Localized Excitatory and Widespread Inhibitory Connectivity of a Neuron in the Decision Stage

Fig. 4. Diagram of the Neural Connectivities in the Evaluation and the Decision Stages of the Eye-Movement Controller

4. Mathematical Analysis of the Controller

4.1. The Evaluation Stage

The evaluation stage is one where inputs at different locations on the superior colliculus are compared with each other. It is shown that the comparison process causes increase of activity at locations which correspond to localised regions of high contrast in the input. The input to the superior colliculus is a superposition of outputs from a number of different regions of the brain. Thus, for retinal output, 'contrast' implies correspondence with the actual intensity contrast in the visual input pattern. But, for cortical output, 'contrast' applies to the spatial pattern of neural activity. For the particular cortical output corresponding to feature detection 'contrast' implies contrast in feature density.

Consider a two-dimensional array of neurons, with (x,y) being the coordinates of the location of a neuron in the array. Each neuron receives input from neighbours lying within a region S surrounding it (edge effects are ignored here). A spatial weighting function b(x,y), defined over S, weights this input.

Therefore the activity of the neuron at (x,y) is given by

$$E(x,y;t+1) = \sum_{(\xi,\eta)\in S} \sum b(\xi,\eta).E(x-\xi,y-\eta;t) + c.P(x,y;t) \qquad (1)$$

where P(x,y;t) is the external input to the neuron at (x,y), and c is a constant. (In this equation, ξ and η are dummy variables of summation.)

For simplicity of analysis, the one-dimensional case is considered, and the variables are assumed to be continuous. We then have

$$E(x;t+1) = b(x)*E(x;t) + c.P(x;t) \qquad (2)$$

where * indicates the operation of spatial convolution.

The spatial weighting factor, or spatial impulse response b(x), causes a spatial transformation of any input pattern. The transfer function corresponding to this transformation is uniquely determined by the spatial weighting factor.

From neurophysiological and neuroanatomical data, it is possible to assume two different basic forms of the spatial weighting factor. In one form, b(x) is a negative unimodal function, with its trough at x = 0 and with a gradual return to zero with distance from the center. This implies that each neuron inhibits all other neurons in the region S surrounding it, including itself. In the other form, b(x) is very much the same as before, except that, in the immediate vicinity of x = 0, over a region small compared to S, b(x) is positive. For each neuron, this implies that it excites those neurons in the immediate vicinity, but inhibits all other neurons in the surrounding region S.

a) An Inhibitory Spatial Weighting Factor

Consider a simple form of the spatial weighting factor, such as the rectangle function

$$b(x/a) = -K, \quad \text{for } -a/2 \leq x \leq a/2, \quad \text{and}$$
$$= 0 \quad \text{elsewhere} \qquad (3)$$

where K is a constant. The corresponding spatial transfer function of such a system is

$$B(as) = -K.a \operatorname{sinc}(as) \qquad (4)$$

where

$$\text{sinc}(s) \triangleq (\sin \pi s)/\pi s, \qquad (5)$$

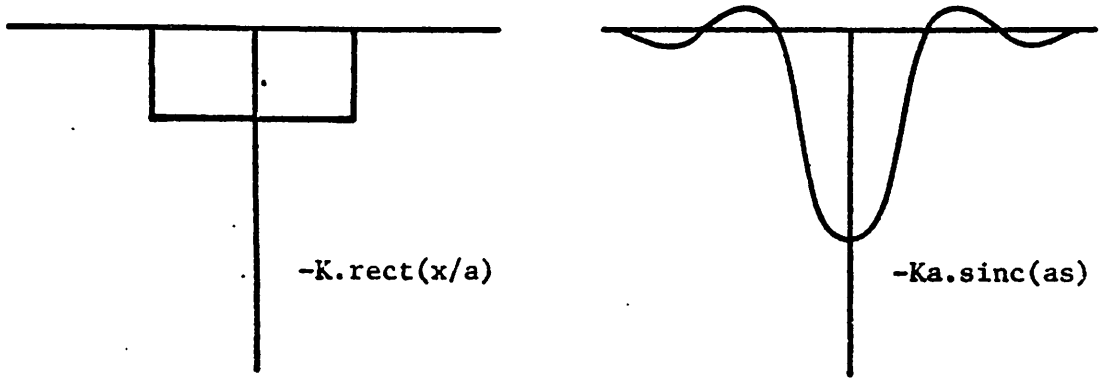
and s is the spatial frequency of the input. The spatial weighting factor and the spatial transfer function are shown in Fig. 5a.

Interpreting the spatial transfer function in terms of excitatory and inhibitory inputs to a neuron, it can be said that negative amplitudes of a spatial frequency correspond to inhibition, while positive amplitudes correspond to excitation. In Fig. 5a, it is seen that the low frequency and zero frequency components are negative, implying that the influence of neighbouring neurons is to inhibit the response of a neuron to these low frequencies. The positive sidelobes in the spatial frequency transfer function, on the other hand, indicate that there is a range of spatial frequencies for which the effective interaction between neurons is excitatory, in spite of the fact that each neuron only inhibits its neighbours. This paradoxical effect of excitation is actually a disinhibition, that is, the inhibition of an inhibitory interaction.

The effect of such suppresion of low frequencies and enhancement of higher frequencies is an output whose contrast is enhanced with respect to that of the input. It is noteworthy that the same principle of generalized lateral inhibition as described here, has long been used in photographic techniques to increase visual contrast, and is known by the term 'unsharp masking' (Yule, 1967).

A slightly different form of the spatial weighting factor, the normal or Gaussian curve, may be more common in the nervous system than the rectangular function described above. The spatial transfer function of a

a)



b)

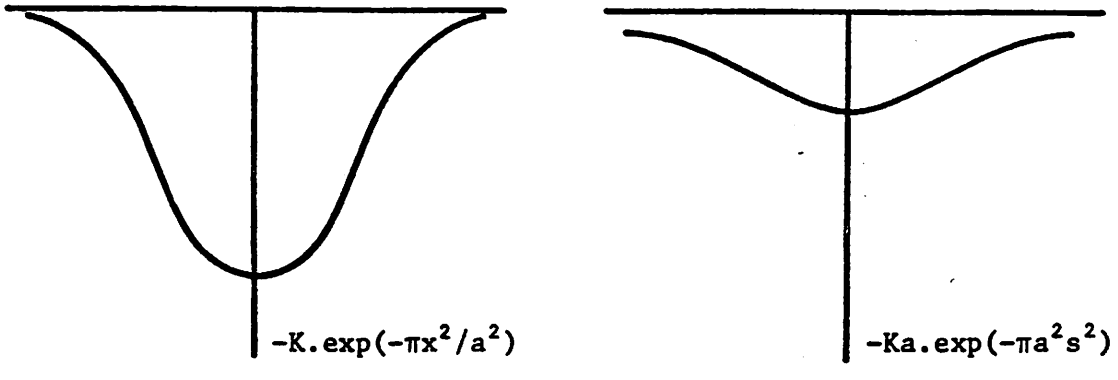


Fig. 5. Spatial Weighting Functions and their Spatial Transforms
a) a Rectangular Spatial Weighting Function
b) a Gaussian Spatial Weighting Function

Gaussian weighting factor is shown in Fig. 5b. For a spatial weighting factor of the form

$$b(x/a) = -K.e^{-\pi(x/a)^2}, \qquad (6)$$

the corresponding spatial transfer function of the system is

$$B(as) = -K.ae^{-\pi(as)^2}, \qquad (7)$$

as shown in Fig. 5b.

The spatial transfer function of the Gaussian weighting factor has no positive sidelobes and hence does not enhance any spatial frequencies. However, the relatively greater inhibition of the lower frequencies itself generates an increase in contrast.

b) A Spatial Weighting Factor with Excitation and Inhibition

Consider the spatial weighting factor in Fig. 6, consisting of the sum of a broad rectangular inhibitory factor and a sharp rectangular excitatory factor. In such a case, the spatial weighting factor is

$$b(x) = b_1(x/a_1) + b_2(x/a_2) \qquad (8)$$

where

$$b_1(x/a_1) = K_1, \quad \text{for } -a_1/2 \leq x \leq a_1/2,$$
$$= 0 \quad \text{elsewhere}, \qquad (9)$$

and $b_2(x/a_2) = -K_2$, for $-a_2/2 \leq x \leq a_2/2$,

$$= 0 \quad \text{elsewhere}. \qquad (10)$$

The corresponding transfer function is the sum of the transfer functions of these two factors, because of the assumed linearity of the system. Therefore, the spatial transfer function is

$$B(s) = K_1.a_1 \operatorname{sinc}(a_1 s) - K_2.a_2 \operatorname{sinc}(a_2 s) . \qquad (11)$$

As can be seen from Fig. 6, the effect of the added excitation is to reduce inhibition at all significant spatial frequencies, thus reducing contrast. Precisely the same conclusion holds in the general case, that is, for other forms of the excitatory and inhibitory components of the spatial weighting factor as well as for the rectangular and the Gaussian forms.

When the excitatory factor is sufficiently large, that is, when $K_1.a_1 > K_2.a_2$, in eqn. 11, the system may become unstable. The reason that such a situation is of interest is that under certain constraints unstable systems form the basis for decision-making systems. This is discussed in the following section.

4.2. The Decision Stage

In our model, a decision-taking system is one which responds to a spatial input pattern of intensities, by generating activity at only one location in the decision array. The position of this activity focus codes the location to which the eyes will turn. In this section, unstable systems are investigated as a possible basis for decision-making systems.

Consider a two-dimensional array of neurons, with (x,y) being the coordinates of the location of the neuron in the array. Each neuron receives excitatory input from near neighbours lying within a spatial region S_1 surrounding it (edge effects are ignored here). A spatial weighting function a(x,y), defined over S_1, weights this input. Each neuron also receives inhibitory input from neighbours lying within a larger region S_2 surrounding it. Another spatial weighting function b(x,y), defined over S_2, weights this inhibitory input.

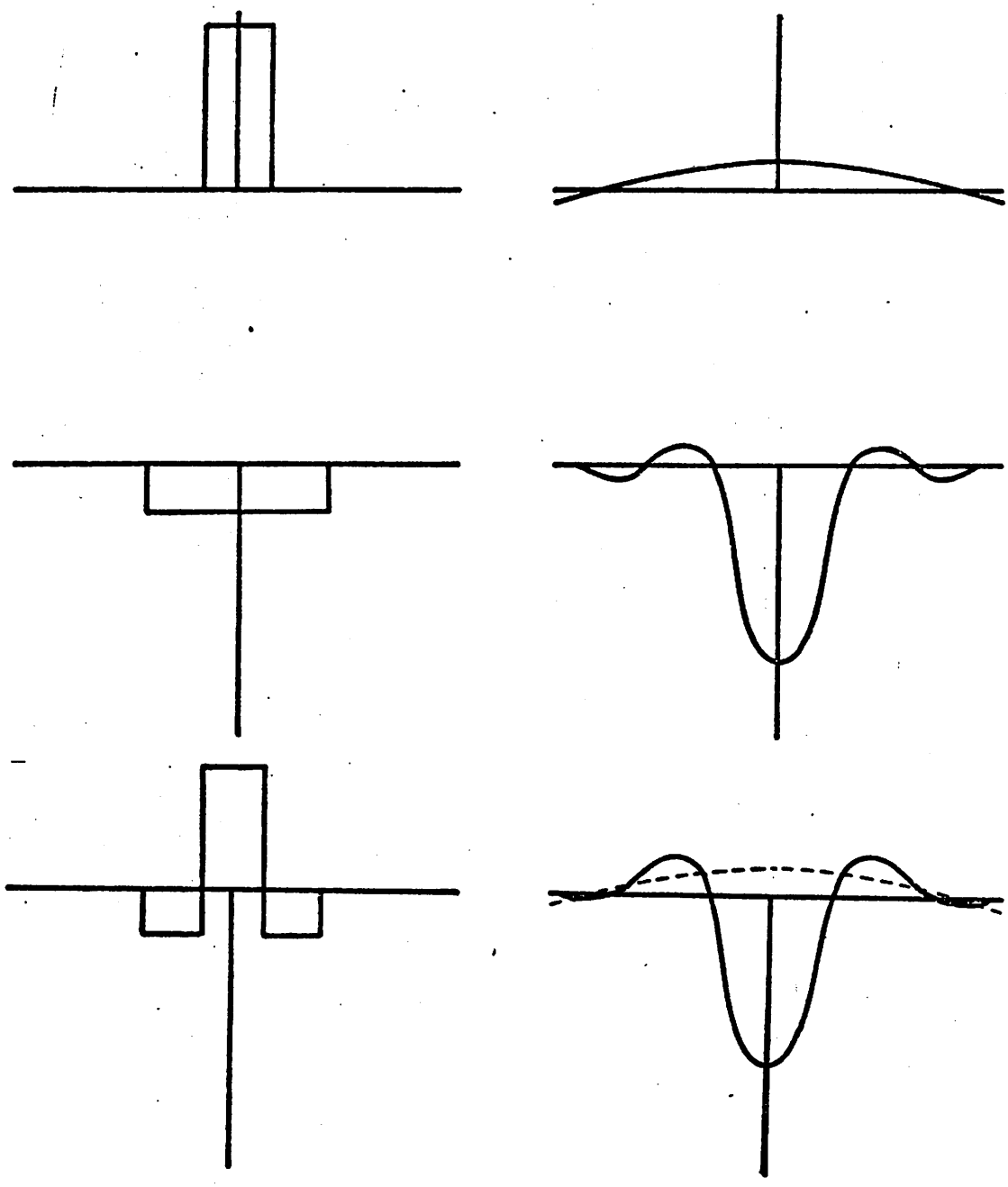


Fig. 6. Illustrating the Effect of Combining an Excitatory and an Inhibitory Spatial Weighting Function on the Spatial Transfer Function. The effect of the added excitatory spatial weighting function is to reduce inhibition at all significant spatial frequencies.

Therefore, the activity of the neuron at (x,y) is given by

$$E(x,y,t+1) = \sum_{(\xi,\eta)\in S_1} \sum a(\xi,\eta).E(x-\xi,y-\eta,t) - \sum_{(\xi,\eta)\in S_2} \sum b(\xi,\eta).E(x-\xi,y-\eta,t) + P(x,y,t) \quad (12)$$

where $P(x,y,t)$ is the input from the evaluation stage to the neuron (x,y). (In this equation, ξ and η are variables of summation.)

For the purposes of further analysis, the one-dimensional case is considered, and the variables are assumed to be continuous. We then have

$$E(x,t+1) = \int_{S_1} a(\xi).E(x-\xi,t)d\xi - \int_{S_2} b(\xi).E(x-\xi,t)d\xi + P(x,t) \quad (13)$$

or $$E(x,t+1) = a(x)*E(x,t) - b(x)*E(x,t) + P(x,t) \quad (14)$$

where * indicates the operation of spatial convolution.

Based on neurophysiological and neuroanatomical data, the assumption is made that both $a(x)$ and $b(x)$ are unimodal, with their peaks at $x = 0$. This implies a decrease of interaction with increasing distance between the neurons.

In the following subsections, instead of investigating general forms of the functions $a(x)$ and $b(x)$, three special cases are considered. In the first subsection, the excitatory interaction is assumed to be sharply localised while the inhibitory effect of each neuron is spread uniformly over the entire array. The length of the array is defined as 2L. The spatial weighting factors are described by

$$a(x) = A.\delta(x), \text{ for all } x, \quad (15)$$

where $\delta(x)$ is defined by

$$\delta(x) = 1 \text{ , for } x = 0$$

$$= 0 \text{ , elsewhere,} \qquad (16)$$

and $$b(x) = \frac{B}{2L} \text{ , for all } x, \qquad (17)$$

where $$B = \int_{-L}^{L} b(x).dx \qquad (18)$$

is the area under the spatial weighting function $b(x)$.

In the second subsection, the excitatory effect is still assumed to be sharply localised, but the inhibitory effect is limited to a region of length 2X surrounding each neuron. Therefore, the spatial weighting functions are described by

$$a(x) = A.\delta(x) \qquad (19)$$

$$b(x) = \frac{B}{2X} \text{ , for } -X \leq x \leq X$$

$$= 0 \text{ , elsewhere} \qquad (20)$$

and $$B = \int_{-X}^{X} b(x)dx. \qquad (21)$$

In the third subsection, the excitatory effect is no longer sharply localised. Each neuron receives excitatory input from neurons in a region of length 2X surrounding it. The inhibitory effect of each neuron is assumed to be spread over the entire array. The spatial weighting functions are now described by

$$a(x) = \frac{A}{2X} \text{ , for } -X \leq x \leq X$$

$$= 0 \text{ , elsewhere} \qquad (22)$$

and $$b(x) = \frac{B}{2L} \text{ , for all } x \qquad (23)$$

where $$A = \int_{-X}^{X} a(x).dx \qquad (24)$$

is the area under the spatial weighting function a(x).

These three special cases, with rectangular spatial weighting functions of differing extent, permit analysis of input conditions and neural connectivity under which decision-making can occur. In general, it is shown that the sharper the localisation of the excitatory interaction, the more effective is the decision-making process.

a) Localized Excitation and Uniform Inhibition

Let the spatial weighting functions be described by

$$a(x) = A.\delta(x) \qquad (15)$$

and $$b(x) = \frac{B}{2L}, \text{ for all } x. \qquad (17)$$

The activity of a neuron at location x and time (t+1), obtained by substituting eqns. 15 and 17 in eqn. 13, is

$$E(x,t+1) = A.E(x,t) - B.\bar{E}(t) + P(x,t) \qquad (25)$$

where $$\bar{E}(t) = \int_{-L}^{L} E(x-\xi,t)\,d\xi \qquad (26)$$

is the average neural activity in the entire array.

To analyse the dynamics of the array response, use is made of the autonomous system, that is, a system with initial conditions E(x,0) and without any input. Therefore, the activity at location x and time (t+1) is

$$E(x,t+1) = A.E(x,t) - B.\bar{E}(t) \; . \qquad (27)$$

Since decision-making is defined as the generation of a single locus of high activity, with suppression of activity at all other locations, it is required that the system be globally stable. The integration of eqn. gives

$$\bar{E}(t+1) = (A-B).\bar{E}(t) \; . \qquad (28)$$

Hence, for global stability, the condition is that

$$(A-B) < 1 \ . \qquad (29)$$

In Sec. 3.4.1, it was shown that when

$$\int_{-L}^{L} a(x)dx < \int_{-L}^{L} b(x)dx \ , \qquad (30)$$

or $$A < B \ , \qquad (31)$$

the system response is the increasing of the contrast present in the input. Such a system, besides being globally stable, is also stable at every location. Hence, to obtain localised instability, it is at least necessary that

$$(A-B) > 0 \ . \qquad (32)$$

Returning to the investigation of instability at specific locations in the array, a new variable $\theta(x,t)$, the activity, at x, relative to the average activity, is defined as

$$\theta(x,t) = \frac{E(x,t)}{\bar{E}(t)} \ . \qquad (33)$$

Therefore, dividing eqn. 27 by eqn. 28, we obtain

$$\theta(x,t+1) = \alpha.\theta(x,t) - \beta \ , \qquad (34)$$

where $$\alpha = \frac{A}{A-B} \ , \qquad (35)$$

$$\beta = \frac{B}{A-B} \ . \qquad (36)$$

The relative change in activity at a location x and time (t+1) is defined as

$$\Delta(x,t+1) = \theta(x,t+1) - \theta(x,t) \ . \qquad (37)$$

Therefore, from eqn. 34, we obtain

$$\Delta(x,t+1) = \beta\{\theta(x,t) - 1\} . \qquad (38)$$

For increase of activity at a location, it is required that

$$\Delta(x,t+1) > 0 \qquad (39)$$

and instability of that increase occurs if

$$\Delta(x,t+1) > \Delta(x,t) . \qquad (40)$$

Applying condition 39 to eqn. 38 the condition for increase of activity at x is obtained as

$$\theta(x,t) > 1 . \qquad (41)$$

Condition 41 implies that only those locations where the activity is above average will show an increase in activity. Similarly, it can be shown that those locations at which activity is already below the average will show a further decrease. Therefore, condition 41 is a necessary condition for unstable increase in neural activity at any location.

Applying condition 40 to eqn. 38 the condition for unstable increase of activity is obtained as

$$\theta(x,t) > \theta(x,t-1) , \qquad (42)$$

or $$\Delta(x,t) > 0 . \qquad (39)$$

But this condition is known to be true as long as

$$\theta(x,t) > 0 . \qquad (41)$$

Therefore, condition 41 is both a necessary and sufficient condition for the occurrence of unstable activity at location x in the decision array.

Hence it is found that sharply localised excitatory interaction and uniform inhibition have the effect of defining a threshold, $\theta = 1$. All neurons with above threshold activity increase further in activity,

while activity in neurons with below threshold levels decreases even further. Both types of change are unstable.

b) Localized Excitation and Limited Spread of Inhibition

Let the spatial weighting functions be defined by

$$a(x) = A.\delta(x) \text{ , for all } x, \qquad (19)$$

and $$b(x) = \frac{B}{2X} \text{ , for } -X \leq x \leq X$$

$$= 0 \text{ , elsewhere.} \qquad (20)$$

The activity, in the autonomous system, of a neuron at location x and time (t+1), obtained by substituting eqns. 19 and 20 in eqn. 13, is

$$E(x,t+1) = A.E(x,t) - \frac{B}{2X}.\int_{-X}^{X} E(x-\xi,t)d\xi \qquad (43)$$

The average neural activity of the array is again

$$\bar{E}(t+1) = (A-B).\bar{E}(t) \qquad (28)$$

where, for global stability,

$$(A-B) < 1 \text{ .} \qquad (29)$$

The relative activity of the autonomous system is

$$\theta(x,t+1) = \alpha.\theta(x,t) - \frac{B}{2X}.\int_{-X}^{X} \theta(x-\xi,t)d\xi \text{ ,} \qquad (44)$$

and the incremental activity is

$$\Delta(x,t+1) = \beta\{\theta(x,t) - \frac{1}{2X}.\int_{-X}^{X} \theta(x-\xi,t)d\xi\} \text{ .} \qquad (45)$$

For increase of activity at location x and time (t+1), it is required that

$$\Delta(x,t+1) > 0 \text{ ,} \qquad (39)$$

or, from eqn. 45 that

$$\theta(x,t) > \frac{1}{2X}.\int_{-X}^{X}\theta(x-\xi,t)d\xi \ . \qquad (46)$$

Physically, condition 46 implies that the activity at a location x will increase if it is already above the average activity within a region of length 2X surrounding it. Note that this local average may be above or below the global average for the entire array. For X = L, the system reduces to case a). Condition 46 is most easily satisfied at the maxima of $\theta(x,t)$. At a sharp, isolated peak, $\Delta(x,t+1)$ is large, while at a broad, flat region, $\Delta(x,t+1)$ is small or zero.

For an unstable increase in $\Delta(x,t+1)$, it is required that

$$\Delta(x,t+1) > \Delta(x,t) \ , \qquad (40)$$

or, from eqn. 45 that

$$\theta(x,t) - \frac{1}{2X}.\int_{-X}^{X}\theta(x-\xi,t)d\xi > \theta(x,t-1) - \frac{1}{2X}.\int_{-X}^{X}\theta(x-\xi,t-1)d\xi \ , \qquad (47)$$

or $$\Delta(x,t) > \frac{1}{2X}.\int_{-X}^{X}\Delta(x-\xi,t)d\xi \ . \qquad (48)$$

Condition 48 implies that if the relative increment at any location is greater than the average increment in a region 2X surrounding it, then the increase in activity at that location is unstable.

Substituting eqns. 45 and 37 in condition 48 gives

$$\beta\{\theta(x,t-1) - \frac{1}{2X}.\int_{-X}^{X}\theta(x-\xi,t-1)d\xi\} > \frac{1}{2X}.\int_{-X}^{X}\theta(x-\xi,t)d\xi - \frac{1}{2X}.\int_{-X}^{X}\theta(x-\xi,t-1)d\xi \ . \qquad (49)$$

If $\beta = 1$, (that is, A = 2B), eqn. 49 reduces to

$$\theta(x,t-1) > \frac{1}{2X}.\int\theta(x-\xi,t)d\xi \ . \qquad (50)$$

Note the difference between conditions 50 and 46.

Condition 50 implies that instability occurs only if the activity at x is greter than the average activity in the surrounding region will be one time interval later. Suppose x is the location of a local maximum, but this local peak is situated in the middle of a large trough. The activity at x will increase because of condition 46, but the average activity of the surrounding region also increases because of the flanking regions of high activity. The increase in average activity may be sufficiently great so that condition 50 is not satisfied, and no unstable increase of activity occurs at x. At a global maximum, however, both conditions 46 and 50 are satisfied.

Therefore, spatial weighting functions of the form defined in eqns. 19 and 20, generating inhibition with limited spread and localised excitation, favor rapid development of unstable increase in activity at locations of isolated activity peaks.

c) Limited Spread of Excitation and Uniform Inhibition

Let the spatial weighting functions be described by

$$a(x) = \frac{A}{2X} \text{ , for } -X \leq x \leq X$$

$$= 0 \text{ , elsewhere} \qquad (22)$$

and $$b(x) = \frac{B}{2L} \text{ , for all } x . \qquad (23)$$

The activity, in the autonomous system, of a neuron at location x and time (t+1), obtained by substituting eqns. 22 and 23 in eqn. 13, is

$$E(x,t+1) = \frac{A}{2X} \cdot \int_{-X}^{X} E(x-\xi,t)d\xi - B.\bar{E}(t) \ . \qquad (51)$$

The average neural activity is

$$\bar{E}(t+1) = (A-B).\bar{E}(t) \ . \qquad (28)$$

The relative activity at location x is

$$\theta(x,t+1) = \frac{\alpha}{2X}.\int_{-X}^{X}\theta(x-\xi,t)d\xi - \beta \ . \qquad (52)$$

The relative increment in activity at x is

$$\Delta(x,t+1) = \frac{\alpha}{2X}.\int_{-X}^{X}\theta(x-\xi,t)d\xi - \theta(x,t) - \beta \ . \qquad (53)$$

For an increase in activity at location x, it is required that

$$\Delta(x,t+1) > 0 \ , \qquad (39)$$

or $$\frac{\alpha}{2X}.\int_{-X}^{X}\theta(x-\xi,t)d\xi - \theta(x,t) - \beta > 0 \ . \qquad (54)$$

Since $\alpha = \frac{A}{A-B}$ and $\beta = \frac{B}{A-B}$, condition 54 can be rewritten as

$$\frac{1}{2X}.\int_{-X}^{X}\theta(x-\xi,t)d\xi > 1 + \frac{A-B}{A}\{\theta(x,t) - 1\} \ . \qquad (55)$$

From conditions 31 and 32, it can be seen that

$$0 \leq (A-B) < 1 \ . \qquad (56)$$

Therefore, at any location x, the average of the surrounding input must be greater than the global average, by a quantity dependent on the parameters A and B and on the value of $\theta(x,t)$, if activity at x is to increase. Therefore, broad regions of high input are more likely to increase in activity than isolated peaks of activity.

For an unstable increase in activity at location x, the condition is that

$$\Delta(x,t+1) > \Delta(x,t) \ , \qquad (40)$$

or $$\frac{\alpha}{2X}.\int_{-X}^{X}\theta(x-\xi,t)d\xi - \theta(x,t) > \frac{\alpha}{2X}.\int_{-X}^{X}\theta(x-\xi,t-1)d\xi - \theta(x,t-1) \ . \qquad (57)$$

$$\text{or} \quad \Delta(x;t) < \frac{\alpha}{2X} \cdot \int_{-X}^{X} \Delta(x-\xi,t)\,d\xi \ . \qquad (58)$$

Note that condition 58 is the opposite of condition 48 in case b), if $\alpha \approx 1$. For instability at location x, the relative increase of activity at that location must be less than the average relative increase in the surrounding region. This ensures smoothing-out of any peaks within a broad region of high input.

Therefore, in case c), where there is some spread of excitation, and the inhibition is uniformly distributed, unstable increase of activity occurs preferentially at broad regions of high activity. Isolated peaks of activity do not show instability and tend to be smoothed out.

d) Evaluation of Different Spatial Weighting Functions

The input to the decision stage from the evaluation stage is a spatial pattern of intensities where the contrast has been enhanced because of the characteristics of the evaluation stage (Sec. 4.1). At the decision stage, a single locus of high activity must develop, corresponding to the direction in which the eyes are to be turned.

Of the three cases examined above, none develops just one locus of activity. In case a), where the excitation is sharply localised and the inhibition is uniformly spread over the array, any location at which the activity is above the average level of array activity will show an unstable increase. In case b), where the excitation remains sharply localised but the inhibition has limited spread, isolated activity peaks are emphasized while regions of uniform activity show little or no increase. In case c), where the excitation

has limited spread, but the inhibition is uniformly distributed over the array, it is broad regions of high activity that show instability. Isolated peaks are smoothed out.

Case b), where isolated activity peaks are emphasized, is the one most appropriate for handling the high contrast input received from the evaluation stage. Neuroanatomical and neurophysiological investigations are required to determine whether excitatory loops in the deep layer of the superior colliculus are indeed sharply localised.

Though isolated locations show increase in activity, in case b), more than one such location does exist. However, a decision requires the existence of just one such locus. Since activity at these different loci increases at different rates, depending on the activity in the surrounding regions, one can postulate a threshold, such that when activity at some locus increases beyond the threshold, it triggers the corresponding motor program and suppresses activity elsewhere in the array. The threshold may correspond to the maximum firing rate possible in these regions.

The consequences of postulating a threshold, above which activity in the decision stage triggers a motor program, are the following:
i) If there are two locations at which the activity reaches threshold simultaneously, simultaneous triggering of two motor programs occurs. While the result of this simultaneous triggering of motor programs depends on the mechanism of decoding of the movement command, one possible result is the generation of an 'average motor program.' Such an effect is observed when a frog, presented with two identical target flies, snaps between the two, at an 'average fly' (Ingle, 1968).

ii) There is a latency between the time of arrival of the input at the decision stage and the time at which the activity at some location reaches threshold. Low intensity inputs will show greater latency. Experimentally, this is an observed fact.

iii) Triggering of a motor program by activity at one location in the decision stage should simultaneously suppress or 'erase' activity at all locations. This erasure prevents triggering of a second motor program by the continued increase of activity at some other location. It also clears the decision stage in preparation for the new input pattern resulting from the change in the direction of fixation of the eyes.

Though unstable increase of activity at specific locations has been emphasized, these systems also show unstable decrease of activity at other locations. Since neural firing rates cannot fall below zero, the unstable decrease of activity cannot decrease below this bound. Neurons also have a maximum firing rate and hence an upper bound exists for unstable increase of activity. The average level of neural activity in the array may be situated asymmetrically between these two bounds. The nonlinearity introduced by the upper and lower bounds is difficult to analyse and requires computer simulation. However, the assumption of a threshold for the triggering of motor programs removes the problem of the existence of an upper bound to neural firing. The existence of a lower bound reduces the rate of increase in activity at the locations of instability because, if a neuron's activity is prevented from falling, its inhibitory effect is greater than that predicted by the linear analysis above.

5. Discussion

As has been discussed earlier, the superior colliculus is not the only controller of saccadic eye movements. However, it appears to have a special role in certain kinds of saccades--fast saccades orienting the animal to objects in the visual field. Lesion of the superior colliculus does not eliminate eye movements (Wurtz and Goldberg, 1972). There is a greater latency preceding the movement, but the movement is as brisk as before. Other deficits, but not solely an increase in saccade latency, have been described for lesions of other visual areas, such as frontal eye fields or visual cortex (Crosby et al., 1962). The ability to naviagate past walls and barriers, that is, to perceive a moving visual field, is not lost with a collicular lesion (Casagrande, 1972) nor is optokinetic nystagmus, which functions to maintain a stationary image of a moving visual field. What is lost is the ability to orient to small objects introduced into the visual field (Casagrande, 1972; Schneider, 1969).

The model proposed is an effective mechanism for generating fast saccades that orient the animal to small objects in the visual field. An unstable excitatory loop, as in the decision layer, rapidly generates a standard firing rate, determined by the maximal firing rate of the loop. The evaluation layer increases the contrast present in the input, preferentially enhancing isolated peaks of input intensity (corresponding to inputs localised in space, such as small objects) rather than extended regions of high intensity (corresponding to large regions of the visual field).

5.1. Extensions of the Model

The superficial layers of the superior colliculus probably have richer information-processing capability than has been suggested in the model.

McIlwain and Buser (1968) hypothesize an override system, with retinal input having priority over cortical input. They suggest that the neural basis may be a more proximal location of retinal input on the dendrites of the collicular neurons, with cortical input arriving at the distal ends of the dendrites.

Szekely (1971) points out the extensive dendro-dendritic connections in this layer, and discusses possible functional implications. Dendro-dendritic interaction may be analogous to neural lateral inhibition but on a more localised basis. Repeated interaction would cause sharp contrast enhancement and suppression of regions of similar input.

The evaluation layer operation is based on the criterion of enhancing activity in that region which receives maximum input, with the enhancement being reduced if neighboring regions also receive high input. This would make the animal repeatedly look at the most intense stimulus in the visual field, contrary to the exploratory behavior which is observed. Thus some process of adaptation must exist. This adaptation cannot occur in the colliculus because the collicular representation of the visual field is retinotopic. Any collicular adaptation would be to a region in the visual field and not to a stimulus whose retinal image shifted with every movement of the eye. (Ingle [1972] shows that, in the frog, collicular adaptation does occur, but only in response to repeated presentation of a stimulus in a fixed region of the visual

field.) The adaptation must be present in the input to the colliculus, and may be considered the opposite of the hypothesis input, which sensitizes a locality in the evaluation layer.

Thus, the evaluation layer, rather than being in a state of constant activity because it is bombarded with input, probably exists at a low level of activity. Only hypotheses, or sudden changes in visual input, create regions of high activity, which then drive the decision layer.

The necessity of an 'erase' mechanism for the decision stage was pointed out in Sec. 4.2.* Does the activity peak in the output command also trigger the erase mechanism? Or is the circuitry of the decision layer such that the activity peak is self-erasing? Can the erase mechanism prevent simultaneous activation of two motor programs? Is an eye movement necessary in order to erase activity in the decision layer?

A noise, or arousal, input to the decision stage of the controller has been hypothesized in Sec. 2.1. The reticular formation may be one of the sources of this non-specific input. It is known to project diffusely to the superior colliculus and is part of the arousal system, located in the brain stem (Magoun, 1963). A noise input to the unstable loops of the decision array would generate intensity peaks and a temporal sequence of motor commands, similar to that generated by visual input, except that the saccades are to randomly chosen points.

Such an input may provide the exploratory drive that causes the eyes to continue scanning even when the sensory input is adapted to or

*While the form of the 'erase' mechanism is not known, it may be related to the observed transient inhibition of collicular activity after each eye movement.

is absent. This input may also be the neural basis for the rapid eye movements observed during REM sleep, when output from the locus coeruleus, in the reticular formation, is high (Jouvet, 1967).

5.2. Experiments and Model-Testing

Essential elements of model-making are testing the validity of the model's hypothesis, supplying numerical parameters and quantifying the model, and testing the operation of the model against the operation of the real system. Numerical estimation of some model parameters is possible through experimentation.

Wheeles et al. (1960) show that it takes 100 msec to make an irreversible decision to move the eyes towards a stimulus. A second stimulus, presented within the first 100 msec, may modify the decision, causing orientation towards the new stimulus. Therefore the time scale developed for the model should be such that from the time input begins to arrive at the decision layer, it takes 100 msec for an activity peak to build to threshold, and tigger a motor program.

Young and Stark (1963) find that there is at least a 200 msec interval between saccades. In terms of our model, it implies either that the 'erase' mechanism prevents activation of the decision stage for 200 msec or that the oculomotor areas after the decision stage are not available for 200 msec after a motor program has been triggered. Neurons in the deep layer of the superior colliculus show a reduction in steady state firing around the time that the eye movement is initiated and this reduction lasts for about 70 to 150 msec (Goldberg and Wurtz, 1972a).

Testing the validity of the hypothesis on which the model is based, and testing the operation of the model against that of the real

system, are often difficult to distinguish.

The main hypotheses are (1) that localised unstable loops in the decision layer generate activity peaks, (2) that a threshold mechanism coupled with an erase mechanism allow only one of these peaks to activate a motor program and suppress all the others, (3) that noise input to the decision layer provides the main exploratory drive, with sensory input interrupting exploration, and (4) that lateral inhibition and override mechanisms in the evaluation layer produce sharp contrast and peaks in the input to the decision layer.

The simplest means of checking for an unstable loop is by opening the feedback pathway and observing the change in activity. However, this is difficult because these loops are assumed to lie wholly within the deep layers of the superior colliculus. Undercutting and stimulation experiments, analogous to those of Burns (1968), may test for the existence of loops.

A histogram of the number of spikes per second, preceding and during a movement, shows the time course of development of activity in the deep layers. An analysis of this curve would indicate whether the best fit is provided by an unstable loop.

An unstable loop will continue firing if there is no inhibition, that is, no erase mechanism. Ingle (1972) finds tht tectal neurons in frog fire for much longer periods after a thalamic lesion which removes thalamo-tectal inhibitory input. Behaviorally, the frog turns repeatedly by a certain angle. This implies the existence of unstable, excitatory loops in the frog tectum, with thalamic inhibition holding the activity of the tectal loops in check. The thalamic inhibition is not necessarily

identical with the 'erase' mechanism postulated earlier. Chemical blocking of inhibition, together with stimulation of a location in the deep layers, should activate an unstable loop and cause a set of 'staircase' saccades.

Wheeles et al. (1960) show that when two consecutive flashes are presented at different locations, and the time interval between them is varied, for intervals between 0 and 100 msec, the eye may turn to either of the two targets. The probability of looking at the second flash increases as the interval decreases. In terms of our model, this indicates a variability in rate of build-up of activity in the loops, such that an activity peak may develop more rapidly at the location corresponding to the second flash than at that corresponding to the first.

The importance of the noise input to the deep layers of the colliculus, the decision stage, may be tested by correlating neural activity in this layer with the level of arousal of the animal as measured for example by activity in the reticular formation. Sensory input, such as visual and auditory input, should be blocked, so that the decision stage is not activated by these systems. The period of REM (rapid eye movement) sleep is well suited for such an experiment because the threshold to sensory input is greatly increased and yet reticular formation activity is high, and is accompanied by a great deal of eye movement.

The existence of an 'erase' mechanism for the decision layer was postulated in Sec. 4.2. However, if actual movement of the retinal image is the signal to erase activity peaks in the decision array, then

holding the eyes stationary should prevent erasure. The motor program generated can be observed, using the method of Koerner and Schiller (1972). The optical stimulus is presented to the paralyzed eye, and movements of the non-stimulated, normal eye are observed.

Electrical stimulation of two locations in the deep layers generates a motor program causing orientation to some location midway between the two appropriate targets (Schaeffer, 1970). However, optical stimulation almost never creates such a situation (Wheeles et al., 1960), though Ingle (1972) shows that a frog will snap in between two flies if thalamic inhibition of the tectum is removed. Therefore, any erase mechanism involved is very direct, perhaps triggered simultaneously with the motor program.

5.3. Existing Models for Visual Search

Existing models for eye movements in visual search are very varied. Pitts and McCulloch (1947) proposed a servo-type error-reducing eye movement mechanism. However, the results do not fit with known ballistic type of eye movements exhibited by mammals.

Didday (1970) has presented a model for the orienting and snapping behavior of the frog, based on the analysis by Lettvin et al. (1959) of the frog's optic tectum. (Note that the eye movements of destriated monkeys are believed to resemble the frog's orienting behavior [Humphrey, 1970]). Our model, by separating the evaluation and decision processes, permits greater complexity to be built into the evaluation procedure and also allows decisions arrived at by other modalities to drive this decision stage, for example, visual orientation towards an auditory stimulus. However, the basic mechanism of decision-making by means of

unstable elements is explored by Didday by means of computer simulation of case a) (Sec. 4.2).

Noton and Stark (1971) propose that the memory of an object includes a record of the eye movements used to scan the object, such that, when the object is viewed again, the scan path is reproduced. In this model eye movements are completely under central control and the role of eye movement controllers, such as the superior colliculus, is not discussed. It ignores the possibility that cyclic scanpaths may be a consequence of rather low-level, mechanistic criteria of eye movement control.

Prokoscki (1971) has advanced a model based on the cross-correlation between foveal input with the entire visual array. While the model reproduces many of the features of normal scanpaths, it is difficult to relate the mechanism to known neural structure.

The model for eye movement control presented in this chapter constitutes an extension of a model of control of orientation by the frog tectum (Didday, 1970). Our model is based on data from mammals such as the cat and the monkey, which handle a visual world much more complex than that of the frog. The evaluation and decision processes are separated, introducing a greater possibility of interaction of this controller with other systems. The effects of different connectivities are explored mathematically permitting the prediction of neuroanatomical connectivity in the deep layers of the colliculus.

6. Conclusion

This chapter presents a model of the superior colliculus as an eye movement controller. Neuroanatomical and neurophysiological data are used to develop a two-stage model of the colliculus. Evaluation of

the input is modelled as occurring in the superficial layers of the colliculus. Lateral inhibition and override mechanisms enhance the contrast present in the input and generate peaks of activity. The deeper layers of the superior colliculus are modelled as the decision-making layers. By means of unstable loops, they develop localised regions of high activity. The assumption of unstable loops results in a system where the rapid build-up of activity reduces the latency with which an eye movement is triggered in response to a visual stimulus. This accounts very well for the observation (Wurtz and Goldberg, 1972) that removal of the superior colliculus does not eliminate the occurrence of saccades but does increase the latency of the saccades.

The strength of this model lies in its close adherence to neural structure, so that properties of the model system can be translated into neural terms, thus permitting prediction of neuroanatomical and neurophysiological properties of the superior colliculus. The usefulness of the model is enhanced by considering it as a subsystem within a larger system for the control of eye movement.

REFERENCES

Abplanalp, P. (1970): Some subcortical connections of the visual system in treeshrews and squirrels.
Brain, Behav. Evol. 3, 155-168.

Apter, J.T. (1945): Projection of the retina on superior colliculus of cats.
J. Neurophysiol. 8, 123-134.

Arbib, M.A. (1972): The metaphorical brain: An introduction to cybernetics as artificial intelligence and brain theory.
New York: Wiley-Interscience.

Burns, B. De L. (1968): The uncertain nervous system.
London: Edward Arnold.

Casagrande, V. (1972): Superior colliculus of the treeshrew: a structural and functional subdivision into superficial and deep layers.
Science 177, 444-447.

Crosby, E.C., Humphrey, T., and Lauer, E.W. (1962): Correlative anatomy of the nervous system.
New York: Macmillan.

Cynader, M., and Berman, N. (1972): Receptive field organisation of monkey superior colliculus.
J. Neurophys. 35, 187-201.

Denny-Brown, D. (1966): The cerebral control of movement.
Liverpool: Liverpool University Press.

Didday, R.L. (1970): The simulation and modelling of distributed information processing in the frog visual system.
Ph.D. Thesis, Information Systems Laboratory, Stanford Univ.

Goldberg, M.E., and Wurtz, R.H. (1972a): Activity of superior colliculus in behaving monkey. I. Visual receptive fields of single neurons.
J. Neurophysiol. 35, 542-559.

Goldberg, M.E., and Wurtz, R.H. (1972b): Activity of superior colliculus in behaving monkey. II. Effect of attention on neuronal responses.
J. Neurophysiol. 35, 560-574.

Humphrey, N.K. (1970): What the frog's eye tells the monkey's brain.
Brain Behav. Evol. 3, 324-337.

Ingle, D. (1968): Visual releasers of prey-catching behavior in frogs and toads.
Brain Behav. Evol. 1, 500-518.

Ingle, D. (1972): Disinhibition of frog tectal units after pretectal lesions.
Abstract in the Program of the Second Annual Meeting of the Society for Neuroscience.

Jouvet, M. (1967): Neurophysiology of the states of sleep.
In "The Neurosciences: A Study Program," G.C. Quarton, T. Melnechuk and F.O. Schmitt (eds.).
Rockefeller University Press.

Karten, H.J. (1974): In the Neurosciences Research Program Bulletin "Sensory-Motor Function of the Midbrain-Tectum," D. Ingle and J.M. Sprague (eds.). To be published.

Lettvin, J.Y., Maturana, H., McCulloch, W.S., and Pitts, W.H. (1959): What the frog's eye tells the frog's brain.
Proc. I.R.E. 47, 1940-1951.

McIlwain, J.T., and Fields, H.L. (1971): Interactions of cortical and retinal projections on single neurons of the cat's superior colliculus.
J. Neurophysiol. 34, 763-772.

Newell, A. and Simon, H. (1971): Human Problem Solving.
Englewood Cliffs, New Jersey: Prentice-Hall.

Noton, D. and Stark, L. (1971): Scanpaths in eye movements during pattern perception.
Science 171, 308-311.

Oyster, C.W., Takahashi, E., and Collewijn, H. (1972): Direction-selective retinal ganglion cells and control of optokinetic nystagmus in the rabbit.
Vision Res. 12, 183-193.

Pitts, W.H., and McCulloch, W.S. (1947): How we know universals: the perception of auditory and visual forms.
Bull. Math. Biophys. 9, 127-147.

Prokoscki, F.J. (1971): A model for human eye movements during viewing of a general two-dimensional, dynamic display.
Ph.D. Thesis for the School of Engineering at the University of Connecticut, Storrs.

Robinson, D. (1968): Eye movement control in primates. The oculomotor system contains specialized subsystems for acquiring and tracking visual targets.
Science 161, 1219-24.

Robinson, D. (1972): Eye movements evoked by collicular stimulation in the alert monkey.
Vision Res. 12, 1795-1808.

Schaeffer, K.P. (1970): Unit analysis and electrical stimulation in the optic tectum of rabbits and cats.
Brain Behav. Evol. 3, 222-240.

Schiller, P.H., and Koerner, F. (1971): Discharge characteristics of single units in the superior colliculus of the alert rhesus monkey.
J. Neurophysiol. 34, 920-936.

Schlag, J., and Schlag-Rey, M. (1970): Induction of oculomotor responses by electrical stimulation of prefrontal cortex in the cat.
Brain Res. 22, 1-13.

Schlag, J., and Schlag-Rey, M. (1971): Induction of oculomotor responses from thalamic internal medullary lamina in the cat.
Exp. Neurol. 33, 498-508.

Schneider, G.E. (1969): Two visual systems: Brain mechanisms for localisation and discrimination are dissociated by tectal and cortical lesions.
Science 163, 895-902.

Sprague, J.M., Berlucchi, G., and DiBerardino, A. (1970): The superior colliculus and pretectum in visually guided behavior and visual discrimination in the cat.
Brain Behav. Evol. 3, 285-294.

Sprague, J.M., and Meikle, T.H. (1965): The role of superior colliculus in visually guided behavior.
Exp. Neurol. 11, 115-146.

Szekely, G. (1971): The mesencephalic and diencephalic optic centers of the frog.
Vision Res. 3, 269-280.

Wheeles, L.L., Boynton, R.H., and Cohen, G.H. (1960): Eye movement responses to step and pulse-step stimuli.
J. Opt. Soc. Amer. 56, 956-960.

Wilson, M.E., and Toyne, M.J. (1970): Retinotectal and corticotectal projections in Macaca mulatta.
Brain Res. 24, 395-406.

Wurtz, R.H., and Goldberg, M.E. (1972a): Activity of superior colliculus in behaving monkey: III Cells discharging before eye movements.
J. Neurophysiol. 35, 575-586.

Wurtz, R.H., and Goldberg, M.E. (1972b): Activity of superior colliculus in behaving monkey: IV effects of lesions on eye movements.
J. Neurophysiol. 35, 587-596.

Yarbus, A.L. (1967): Eye movements and vision.
New York: Plenum Press.

Young, L.R., and Stark, L. (1963): Variable feedback experiments testing a sampled data model for eye tracking movements. I.E.E.E. Trans. Human Factors in Electronics HFE-4, 38-51.

Yule, J.A.C. (1967): Principles of color reproduction. New York: John Wiley and Sons.