16. Brody, S. *Bioenergetics and Growth* (Hafner, New York, 1964).
17. Oddy, V. H., Goosen, J. M. & Annison, E. F. *Aust. J. biol. Sci.* **37**, 375-388 (1984).
18. Shutz, Y., Lechtig, A. & Bradfield, R. B. *Am. J. clin. Nutr.* **33**, 892-902 (1980).
19. Martin, R. D. & MacLarnon, A. M. *Nature* **313**, 220-223 (1985).
20. Martin, R. D. *Symp. Zool. Soc. Lond.* **51**, 87-117 (1984).
21. McNab, B. K. *Am. Nat.* **116**, 106-124 (1980).
22. Hennemann, W. *Oecologia* **56**, 104-108 (1983).
23. Hennemann, W. *Oikos* **43**, 363-368 (1984).
24. McNab, B. K. *J. zool. Res.* **199**, 1-29 (1983).
25. Fleming, M., Harder, J. D. & Wukie, J. J. *Comp. Biochem. Physiol.* **70B**, 645-648 (1981).
26. Dryden, G., Gebczynski, M. & Douglas, E. L. *Acta theriol.* **19**, 453-461 (1974).
27. Trojan, P. & Wojciechowska, B. *Ekol. Pol.* **15**, 811-817 (1967).
28. McClure, P. A. & Randolph, J. C. *Ecol. Monogr.* **50**, 199-217 (1980).

Enhanced detection in the aperture of focal attention during simple discrimination tasks

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There is increasing evidence that it is possible to shift an aperture of focal attention to a position in visual space independent of fixation and that this can be done much faster than the eyes are able to move[1-4]. Recently, we showed[4] that such serial scrutiny by the aperture of focal attention is required before an observer is able to tell what a target is (for example, to know whether the orientation of a line segment is horizontal or vertical). Here we considered whether attention directed towards a specific position in the visual field for an orientation discrimination task improves performance on a simple detection task in the area to which attention is directed. We found that a small test flash could be detected when it was positioned near a peripheral line target presented briefly, if the orientation of the target had to be identified. The test flash could not be detected when presented at some distance from the same target or when another target had to be identified. This enhancement implies that even simple identification tasks such as orientation discrimination are not performed passively by the visual system.

Against a dark field, we presented two bright targets each extending 0.7 degrees of arc in length (horizontal or vertical line segments; or the letter L or T), one in the position where the observer was fixating, the other at 4° eccentricity from the fixation marker, but in any random direction from this marker. Observers were asked to identify only one of the targets (either peripheral or central) in a given block of trials, and were told that the peripheral target would always be at the same eccentricity but in any random direction. Presentation time was short (10 ms) to avoid a second fixation on the target by eye movement, and a mask was applied at a specific time, termed stimulus onset asynchrony (SOA), after target onset, in order to limit processing time (see Fig. 1*a*). Simultaneously with this target identification task, we also presented a small test flash with a 4 arc min diameter lasting 10 ms, at various positions across the visual field, and asked observers to report the presence or absence of the test flash, by pressing one of four keys: two for presence or absence of the test flash when target A was identified and another when target B was identified. Detection errors were signalled by ringing of a bell on the computer that ran the experiments. The test flash and the targets were masked at different SOAs so that performance on the two tasks could be controlled independently. The test flash appeared on 66% of the trials, and was presented at positions along two cardinal directions from the peripheral target: on the perimeter of an imaginary circle of the same eccentricity as the peripheral target or on a line running from the central target to the peripheral target and beyond (see Fig. 1*b*). In each block of 50 trials the test flash could appear in any of the four directions and at any of three distances from the discrimination target. As observers had to identify only one of the targets in a given block of trials, we could compare detection rates for the test flash when the peripheral or the central target was identified, thus isolating changes in detection rate caused by the identification processes.

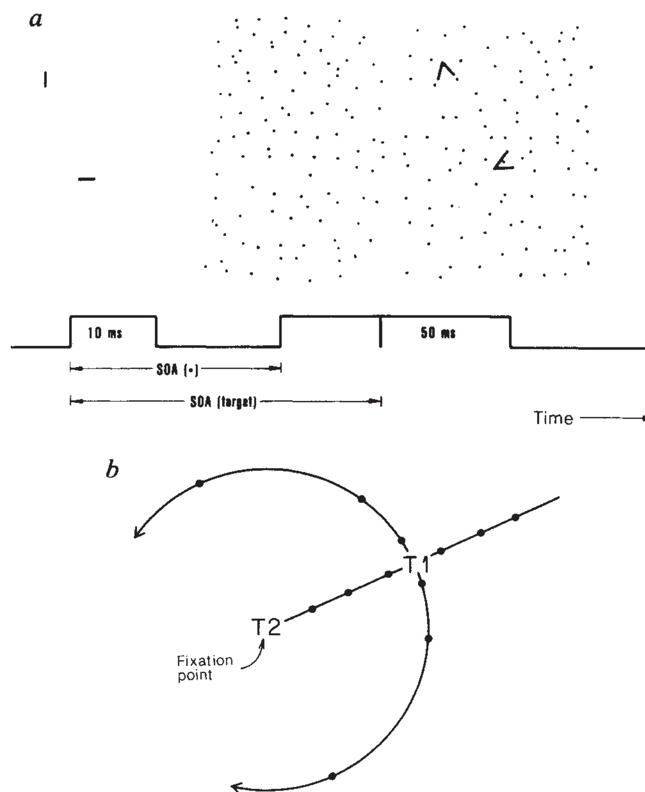

Fig. 1 *a*, The stimulus used in our experiments. The observers were presented first with a pair of targets, one at the fixation point (here a horizontal line) and another at 4° eccentricity. A small dot, the test flash, shown here near the vertical peripheral target, was presented on 66% of the trials. This pattern was presented for 10 ms then masked by random dots after a certain time, SOA(·), and after a longer time, SOA(target), a pair of randomly oriented 'V' symbols were presented at the locations of the targets for 50 ms. A fixation cross was presented at the position of the central target before each trial began, but was removed 50-100 ms (randomized between trials) before the stimulus onset. *b*, Possible locations of the test flash relative to the discrimination target T1. Fixation point is on T2, where the central target was presented. T1 represents the peripheral target; solid lines represent the two cardinal directions from T1 that were used for presenting the test flash. The test flash could be placed in one of 12 positions (indicated by the solid circles). These positions are relative to T1; when T1 appeared in another direction all the possible positions would rotate in this direction. We used the same configuration for experiments involving central and peripheral target discrimination.

The results of experiments in which observers had to identify the peripheral target are shown in Fig. 2*a*. The results are presented as the difference between the frequency of correct detection when the test flash was presented, P(detect), and the false alarm rate, P(FA), representing the frequency of observers falsely reporting the presence of the test flash. As the distances between the test flash and the discrimination target were varied in each block of trials, the same false alarm rate was measured for all distances, so an increase in $[P(\text{detect}) - P(\text{FA})]$ reflects

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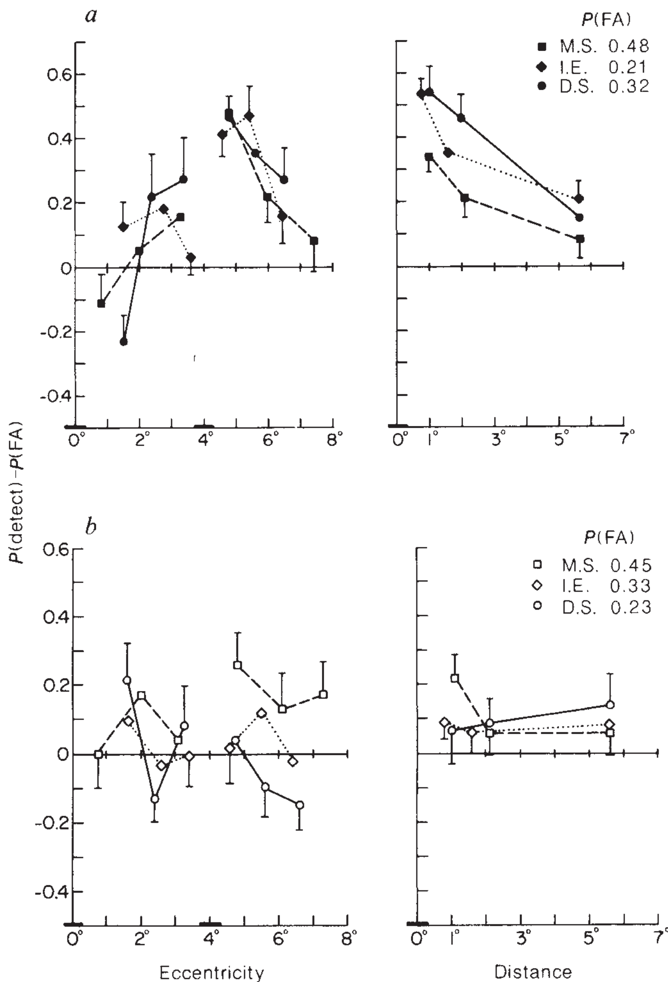

Fig. 2 *a*, Data from experiments in which observers had to identify the peripheral target. Plot on the left shows the dependence of $P(\text{detect}) - P(\text{FA})$ on the radial distance of the test flash from the fixation point, the peripheral target here being at 4° eccentricity. Plot on the right shows the dependence of $P(\text{detect}) - P(\text{FA})$ on the distance from the peripheral target along an equi-eccentricity circle; data for the clockwise and anticlockwise directions were combined. Data shown are for three observers: M.S. and D.S., who performed an orientation discrimination task with SOA(target) = 60 ms for M.S. and 70 ms for D.S. Observer I.E. discriminated between T and L with SOA(target) = 120 ms. For all observers SOA(·) was 60 ms. *b*, Same as *a*, but discrimination was performed on the central target. Target discrimination rates were 0.90 ± 0.01, 0.90 ± 0.02 and 0.89 ± 0.02 in *a* and 0.86 ± 0.02, 0.84 ± 0.02 and 0.83 ± 0.02 in *b* for observers I.E., D.S. and M.S., respectively.

an increase in correct detection for each observer. When examining detection rates for the test flash at different positions in the radial direction, at different eccentricities, a clear enhancement was seen at about 4° eccentricity, where the discrimination target was presented (Fig. 2*a*, left). The same increase in detection rate was seen along the constant eccentricity direction in the vicinity of the discrimination target (Fig. 2*a*, right). To prevent contamination, the test flash was never presented closer than 0.6° from the target centre. To ensure that the increase in detection rate did not originate from trials in which incorrect discriminations were made, we kept the error rate on the discrimination task low by adjusting the masking delay (SOA) for each observer, so that the performance on the discrimination task was ~90% correct (see Fig. 2 legend) for all observers (although one of us served as observer, the other two observers were unaware of the purpose of this study). Surprisingly, the detection rate was lower at positions between the central target (fixation point) and the

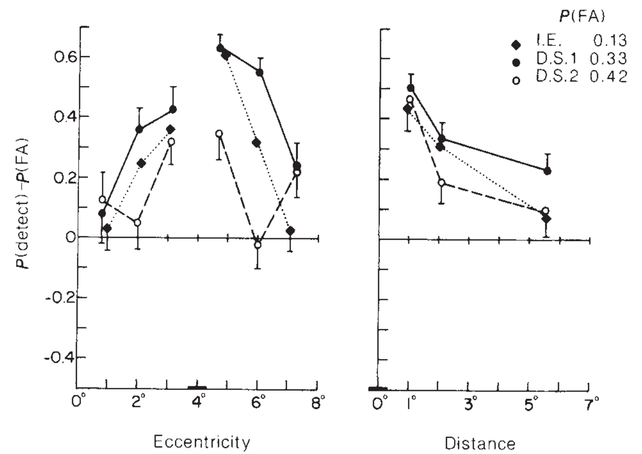

Fig. 3 The same as Fig. 2*a*, but without presentation of the central target. Note that detection at locations between the fixation point and the peripheral target is improved. Data shown are for observers I.E. (T/L discrimination) and D.S. (expt 1, T/L discrimination; expt 2, vertical/horizontal discrimination).

peripheral target than at more peripheral positions. Experiments in which the central target was absent yielded a more symmetrical enhancement around the peripheral target (see Fig. 3). Thus the area of highest detection rate is not around the fixation point, but is shifted to the location of the target being identified; this is demonstrated in a plot of $[P(\text{detect}) - P(\text{FA})]$ isocontours (Fig. 4). The two regions of enhancement were reconstructed from the data of two experiments, a new one with the peripheral target at 2° eccentricity and the one with the peripheral target at 4°, as in Figs 2, 3. The size of the enhancement area is larger at the larger eccentricity, having a diameter about twice that of the enhancement area at 2° eccentricity.

Next we attempted to confirm that this enhancement originated from the discrimination task that our observers were performing, rather than from the presence of the target itself. The presence of the target might cause luminance interaction between itself and the test flash or might bias the observers to detect the test flash when it is located near the target. We performed additional experiments using the same stimuli, but

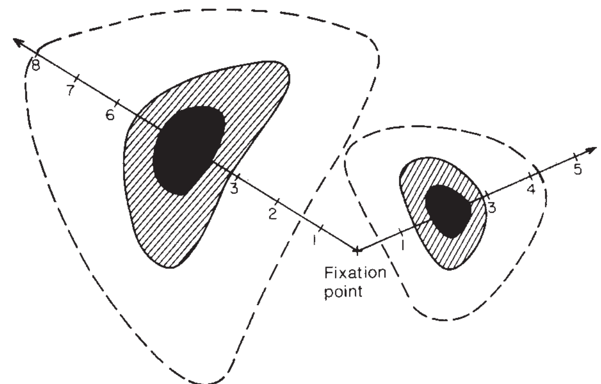

Fig. 4 Test flash detection isocontours plotted at two different eccentricities for observer D.S., in experiments such as those of Fig. 2*a*. The broken lines represent the borders of the enhancement areas where correct detection of the test flash is about the same as without enhancement (Fig. 2*b*), approaching the 'false alarm' rate; within the shaded area the detection rate is more than twice the false alarm rate, and in the solid area about three times the false alarm rate. Note that the linear size of the enhancement area scales linearly with eccentricity, being about twice the size at 4° than at 2°.

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asked the observers to perform discrimination on the central target; the results of these experiments are shown in Fig. 2*b*. The enhancement around the peripheral target disappeared, and performance everywhere was close to chance level [P(detect) ~ P(FA)], with about the same false alarm rates as in the previous experiments). There was also no clear improvement at positions close to the central target. This lack of enhancement might be a result of the coarse sampling used, as the size of the enhancement area around the centre of gaze might be smaller than that at the peripheral positions. This decrease in size would be consistent with that seen with decreasing eccentricity (see Fig. 4).

When attention is focused on the discrimination task, does it cause a change of sensitivity or a change of criterion for detection of the test flash? The psychophysical method we used to investigate this question does not allow a direct answer as it was designed to maximize test-flash position uncertainty and to force attention shifts to the discrimination target. To obtain an answer, we would have to present the test flash at known locations on each block of trials, but that might have allowed the observers to shift their attention to the expected location of the test flash, a strategy we wanted to avoid. As a result, the false alarm responses from all locations were combined, thus it is unknown whether there was an increase in false alarm rate in the vicinity of the discrimination target resulting from the discrimination process. However, the false alarm rates measured using the peripheral target were not significantly larger than those for the central target (FA differences of 0.03 ± 0.06, -0.12 ± 0.06, 0.09 ± 0.08 for the three observers; $\pm$s.e.m.).

We conclude, therefore, that visual discrimination involves a process that increases the probability of detecting a nearby test flash. This increase is not due to the presence of the target to be identified nor is it due to any expectation that the test flash will be found at a particular position, cued by the discrimination target, but results from discriminating the target. At present we cannot decide whether the observed visual enhancement is caused by some sensitivity or criterion change, but we find that this enhancement is involved in a task as simple as discriminating between vertical and horizontal lines. This result indicates that even a simple discrimination task cannot be performed passively by the visual discrimination system. The enhancement might be a property of the serial attentive process that is required for discrimination of orientation[4]. According to our finding, the size of the area processed by the attentive system is scaled with eccentricity, having an average diameter of ~3° at 4° eccentricity, and ~1.5° at 2° eccentricity. Interestingly, this area is the same for the two tasks that we used—orientation discrimination (vertical versus horizontal) and positional relationship discrimination (T versus L). It is possible that the area of visual enhancement that we have plotted here is the 'searchlight' of attention.

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1. Posner, M. I. *Q. J. exp. Psychol.* **32**, 3-25 (1980).
2. Treisman, A. & Gelade, G. *Cognitive Psychol.* **12**, 97-136 (1980).
3. Bergen, J. R. & Julesz, B. *Nature* **303**, 696-698 (1983).
4. Sagi, D. & Julesz, B. *Science* **228**, 1217-1219 (1985).

Phorbol esters block a voltage-sensitive chloride current in hippocampal pyramidal cells

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The importance of second-messenger systems in controlling the excitability of neurones and other cells, through modulation of voltage- and calcium-dependent ionic conductances, has become increasingly clear. Cyclic AMP[1–3], acting via protein kinase A[4–6], has been identified as the second messenger for several neurotransmitters, and recent studies have suggested that activation of protein kinase C may have similar modulatory actions on neurones[7–11]. Calcium and potassium currents have so far been shown to be the major ionic conductances modified by kinase activation[4–11]. We now report that hippocampal pyramidal cells contain a previously undescribed voltage-dependent chloride current which is active at resting potential and is turned off either by membrane depolarization or by activation of protein kinase C by phorbol esters. We propose that this current may reside predominantly in the cell's dendritic membrane and thereby may regulate dendritic excitability.

In 53 out of 55 rat hippocampal pyramidal cells in which K^+ currents were blocked (see Fig. 1 legend), voltage clamp recording with Cl^--containing microelectrodes revealed slow inward current relaxations, in response to hyperpolarizing steps from depolarized holding potentials. These current relaxations appeared to be caused by the turning on of an inward current, as the amplitude of the instantaneous current jump at the beginning of a hyperpolarizing step was smaller than that at the end, suggesting an increase in membrane conductance during the step (Fig. 1*a*). In addition, the amplitude of the instantaneous currents produced by short-duration hyperpolarizing voltage steps increased during the inward current relaxation produced by a long hyperpolarizing voltage step, and returned slowly to baseline amplitude when the membrane potential was stepped back to the holding potential (V_H) (Fig. 1*b*). This is in contrast to the M-current (I_M) recorded in conditions where K^+ currents are present, which turns off with similar voltage steps[12]. Also in contrast to I_M, this current was insensitive to muscarinic receptor activation. This slow inward current was voltage-sensitive over a wide range of membrane potentials (Fig. 1*c*). As determined by steady-state conductance measurements, the current was completely inactive at membrane potentials postive to approximately −10 mV, was largely active at the resting potential (−55 to −70 mV in these cells), and was fully activated at potentials negative to −90 mV (Fig. 1*c*). These voltage values could be overestimates of the actual voltage-sensitive range of the current if it was generated at a site electrotonically remote from the recording electrode in the soma (see below). Relaxations caused by this current could also be evoked by holding the cell at relatively hyperpolarized levels and turning it off with depolarizing commands (see Figs 1*e*, 2*b*).

We suspected that the ionic carrier of this current was chloride because currents carried by K^+, Ca^{2+} and Na^+ should be effectively blocked in the medium used in our experiments (see Fig. 1 legend). Indeed, when cells were impaled with electrodes containing the anion methyl sulphate ($MeSO_4^-$), hyperpolarizing steps produced an outward current (Fig. 1*d*(ii), $n = 11$), rather than an inward current as invariably seen in cells recorded with Cl^--filled electrodes (Fig. 1*d*(i), $n = 53$). The reversal potential of this current was determined with both Cl^-- and $MeSO_4^-$-containing microelectrodes. With the former, when cells were held at hyperpolarized levels and depolarizing steps of increasing size were applied, a turning off of the current was observed, resulting in outward current relaxations which reversed to inward relaxations at a membrane potential of about +15 mV in the cell illustrated (Fig. 1*e*(i)). The average reversal potential of this current with Cl^- in the recording electrode was

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