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A synaptic mechanism possibly underlying directional selectivity to motion

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A specific synaptic interaction is proposed as the mechanism underlying the directional selectivity to motion of several nervous cells. It is shown that the hypothesis is consistent with previous behavioural and physiological studies of the motion detection process.

Detection of movement is one of the most basic and elementary computations performed by visual systems. Hence it is not surprising that the mechanisms and principles underlying movement detection have been approached in various species with a variety of techniques from behavioural analysis and psychophysics to physiology. Although several investigators have provided a wealth of information in the last years, the early analyses of Hassenstein & Reichardt (1956), Reichardt (1957, 1961), Barlow & Hill (1963), and Barlow & Levick (1965) still represent the extent of our understanding of this function. These studies are in many respects complementary. Those of Reichardt & Hassenstein are centred on the functional principles of movement detection as inferred from the average optomotor behaviour of a whole insect, whereas Barlow & Levick attack the problem of the neural circuitry underlying directional selectivity in the ganglion cells of a vertebrate retina.

Figure 1*a* and *b* summarize the main conclusions of the two approaches. Both models postulate the existence of two types of channels (1 and 2, from two adjacent receptor regions) with different conduction properties. In figure 1*a*, channel 1 and channel 2 are low pass filters with a short and a long time constant, respectively, while in figure 1*b*, channel 2 simply contains a delay.

Perhaps the most significant contribution of Barlow & Levick consists of the experimental recognition that movement detection, at the level of direction selectivity of the ganglion cells, results primarily from an inhibitory mechanism that 'vetoes' the response to simultaneous signals from the receptors (after appropriate asymmetric delay) rather than from the detection of the conjunction of excitation from two regions (see figure 1). On the other hand, the main thrust of Hassenstein & Reichardt's analysis is the demonstration that the interaction underlying movement detection must be nonlinear and, in particular, of a multiplicative type. Many experimental data suggest that this is indeed the functional scheme underlying movement detection in insects (Poggio & Reichardt 1976).

Barlow & Levick did not explicitly specify the inhibitory interaction they proposed but from their notation (see figure 1*b*) it can be clearly understood to be nonlinear. They stress, for instance, that the response to movement cannot be predicted from the map of the receptive field showing the regions yielding 'on' or 'off'-responses to stationary spots.

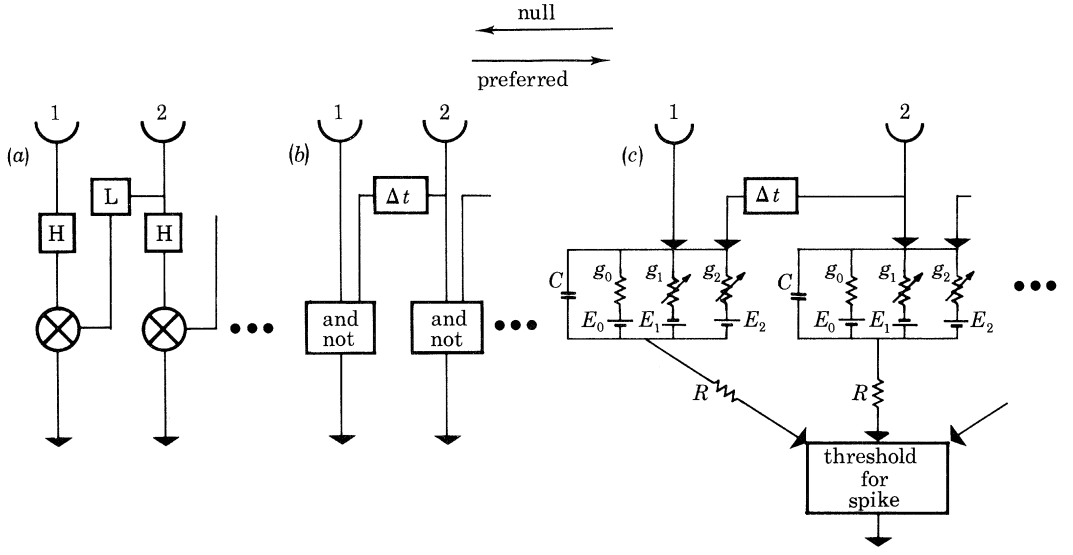

FIGURE 1. (*a*) A part of the model of movement detection of Hassenstein & Reichardt (1956). The two inputs are multiplied after low pass filtering with different time constants. If an average operation is made on the output the overall operation is equivalent to cross-correlation of the two inputs. The output of this and its complementary scheme represents the behavioural optomotor response of the whole insect. (*b*) The scheme proposed by Barlow & Levick to account for direction selectivity of the ganglion cells in the rabbit retina. The output of the model is spike rate of the ganglion cells. A pure delay Δt is not really necessary: a low pass filtering operation is sufficient (inhibition needs only to last longer than excitation). There is a complementary scheme for the opposite direction. (*c*) The equivalent electrical circuit of the synaptic interaction assumed to underly direction selectivity. The interaction implemented by the circuit is of the type $g_1 - \alpha g_1 g_2$ (see text). Addition of the outputs of this and the complementary circuit cancels at the motor level the linear contribution g_1, effectively leaving the multiplicative term $\alpha g_1 g_2$. The delayed channel could, in principle, also carry the excitatory input g_1, instead of g_2.

The simplest neural implementation of the 'and-not' (or 'veto') gate of Barlow would consist of linear inhibitory interaction of two inputs followed by a threshold operation. For such a scheme to be feasible it is necessary that there exist direction selective subunits where the threshold operation is performed. Their outputs could be then added by a ganglion cell. The threshold operation cannot take place at the level of the large field ganglion cells themselves since no selectivity could be present when a large number of input channels are stimulated (i.e. during movement of a periodic grating) with many different phases (Poggio & Reichardt 1976). The subunits that discriminate the sequence of receptor excitation in individual

pairs of regions were tentatively identified by Barlow & Levick to be bipolar cells. However, bipolar cells do not have a spike generating mechanism. Moreover, there is now some evidence indicating that directional selectivity in the vertebrate retina is restricted to the ganglion cells; bipolar cells and even amacrine cells, although important for determining directional sensitivity of ganglion cells, are not, apparently, direction selective themselves (Werblin 1970; Marchiafava 1978). Thus, the simple idea of a threshold operation on linear combinations of inputs as the physiological equivalent for the 'and-not' gate or multiplication of figure 1 cannot apparently hold, at least if threshold elements are identified with cells.

In conclusion, the two studies of Hassenstein & Reichardt and Barlow & Levick have outlined the basic principles underlying movement detection: (*a*) a multiplication-like, inhibitory interaction between pairs of inputs or input regions, after (*b*) asymmetric lowpass or delay operations. The nature of the underlying mechanism is still, however, an open question. The object of this paper is to propose a physiologically plausible synaptic mechanism for the actual neural implementation of the and-not gates (or multiplications) of figure 1*a*, *b*.

Our hypothesis is sketched in figure 1*c*. Each *R–C* circuit is the electrical equivalent of a patch of dendritic membrane (of a direction-selective cell) receiving two distinct inputs corresponding to channels 1 and 2 of figure 1*a* and *b*, through two distinct, *closely adjacent* synapses. The two inputs control conductances g_1 and g_2, respectively. The depolarizations induced in each patch of dendritic membrane propagate passively according to the cable properties of the cell (symbolized in figure 1*c* by the resistances R) and summate in the soma where they eventually exceed the voltage threshold for initiation of a spike. The conductances g_1, g_2, g_0 are postulated to be associated with different ionic channels: g_1 could, for instance, control a Na channel and g_2 a Cl channel. The resting conductances and batteries of the ionic channels are lumped together in the resting conductance g_0 and in the resting potential E_0, respectively. In this representation g_1 and g_2 are therefore identically zero in the resting state. The circuit equation can be written as

$$C\,\mathrm{d}V/\mathrm{d}t+(g_1+g_2+g_0)V = g_1\,E_1+g_2\,E_2+g_0\,E_0. \tag{1}$$

Equation (1) defines a nonlinear relation between the inputs $g_1(t)$, $g_2(t)$ and the membrane potential 'output', $V(t)$.

We show now (*a*) that the circuit of equation (1) with adequate numerical values of its parameters can subserve direction selectivity, in a way consistent with Barlow & Levick's description, and (*b*) that the operation performed by the circuit of figure 1*c* on the two inputs g_1 and g_2 can be characterized as a multiplication-like operation.

The first argument (*a*) is made explicit by the numerical solutions of equation (1) shown in figure 2. Under the assumptions $E_1 \gg E_0$ and $E_2 \approx E_0$ (the reversal potential for inhibitory channels is usually close to the resting potential) the circuit behaviour can underly direction selectivity of a cell. The potential change for movements (for instance of a light spot) in the preferred direction occurs when

the changes of g_1 and g_2 are separated by an appropriate delay: in this case the increase of g_2 does not offset the depolarization induced by the increase of g_1, and may even result in a small depolarization, if E_2 is actually slightly more positive than E_0 (as in the example of figure 2). Movements in the null direction result in almost simultaneous changes of g_1 and g_2, leading to a reduction of the membrane depolarization.

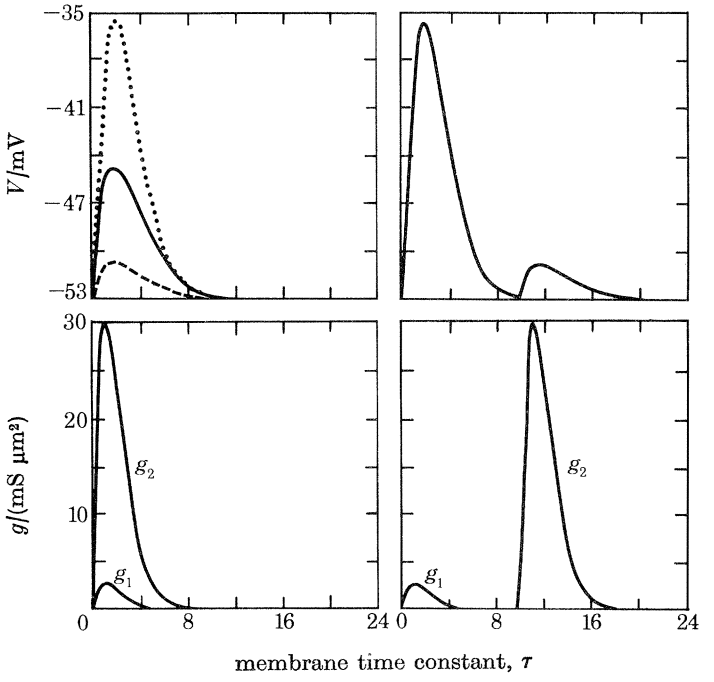

FIGURE 2. Time course of changes of g_1, g_2 and the resulting membrane potential V. Equation (1) has been solved with parameter values $E_1 = 40$ mV, $E_2 = -50$ mV, $E_0 = -53$ mV, $g_0 = 3.5$ mS μm². The time course of the inputs $g_1(t)$ and $g_2(t)$ is given by the standard 'alpha' function (Jack *et al.* 1975). The time scale is in units of the membrane time constant $\tau = C/g_0$. The broken lines represent the synaptic voltage elicited by g_1 and g_2 independently. The diagrams at the left represent the situation expected for stimuli moving in the null direction, while the case of the preferred direction is shown at the right. A useful parameter for characterizing the various cases is the total input charge at the synapses, since the presynaptic potential at the soma remains roughly proportional to it (Jack *et al.* 1975): the total charge is, in arbitrary units, 66 for g_1 alone, 10 for g_2 alone, 38 for the 'null' direction case, 76 for the 'preferred' direction case.

Inhibition clearly takes place in the null direction case. Facilitation (for the preferred direction) in the cell's firing rate may easily follow from temporal integration at the soma and the presence of the threshold mechanism there, since the total charge in this case (76) is larger than either for g_1 alone (66) or for g_2 alone (10).

With the parameter values used here (but $C = 0$) the representation equation (4) does not converge for all t, and must be continued analytically. The Volterra representation that has to be used instead of equation (4) (since $C \neq 0$) converges for all t ($0 \leqslant t \leqslant T$) and all bounded inputs g_1, g_2 (Poggio & Torre 1978). For a good *quantitative* approximation, terms of order higher than the second are needed with these parameter values.

The two inputs g_1 and g_2 clearly interact in a nonlinear way in their effect upon the membrane potential. The conductance change g_2 essentially shunts the membrane potential towards E_2. Notice that the effect of a change in g_2 is small (actually disappearing if $E_2 = E_0$), unless there is a simultaneous change in g_1. This has to be expected if g_1 and g_2 interact in a multiplicative manner. In order to gain some insight (*b*) into the nature of this transduction, the transmembrane potential V, solution of equation (1), can be expanded in a functional power series in $g_1(t)$ and $g_2(t)$. This representation shows explicitly the dependence of the output V on the inputs g_1 and g_2; it has here a deeper justification since the membrane potential V is (in the general cable-like case) an analytic functional of bounded conductance inputs (Poggio & Torre 1978). In order to simplify the calculations that follow, the membrane capacitance C will be neglected (time changes of the conductances are assumed to be slower than the membrane time constant). Equation (1) leads, under this hypothesis, to

$$V = \frac{g_1 E_1 + g_2 E_2 + g_0 E_0}{g_1 + g_2 + g_0}, \tag{2}$$

which can be rewritten as

$$V = \left(E_0 + \frac{g_1}{g_0} E_1 + \frac{g_2}{g_0} E_0\right) \left(1 + \frac{g_1}{g_0} + \frac{g_2}{g_0}\right)^{-1}. \tag{3}$$

The term $(1 + g_1/g_0 + g_2/g_0)^{-1}$ can be expanded in a Taylor series, for small $g_1(t)/g_0$ and $g_2(t)/g_0$, yielding (up to the second order in g_1, g_2):

$$V = E_0 + (E_1 - E_0)\frac{g_1}{g_0} + (E_2 - E_0)\frac{g_2}{g_0} - (E_1 - E_0)\left(\frac{g_1}{g_0}\right)^2 - (E_2 - E_0)\left(\frac{g_2}{g_0}\right)^2 + (2E_0 - E_1 - E_2)\frac{g_1 g_2}{g_0^2} + \ldots. \tag{4}$$

Higher order terms can often be neglected (it can be shown (Poggio & Torre, 1978) that the qualitative properties of equation (2) are displayed by the second order approximation, equation (4)). Assume now that $E_1 \gg E_0$, as is usually the case for excitatory channels, but $E_2 \approx E_0$. This allows us to neglect terms containing $(E_2 - E_0)$. The first term (E_0) in equation (3) is the resting potential: for $g_1 = g_2 \equiv 0$, $V = E_0$. The term

$$(E_1 - E_0)\, g_1/g_0 \tag{5}$$

is linear in the input conductance g_1 and its effect is to depolarize the membrane. The term

$$-(E_1 - E_0)\ (g_1/g_0)^2 \tag{6}$$

is a second order term which provides a correction for nonlinear summation of input g_1. Finally, the term

$$(2\ E_0 - E_1 - E_2)\ g_1\ g_2/g_0^2 \tag{7}$$

is the first term that shows a nonlinear interaction between the two changes of conductances. The interaction is of the multiplicative type and the sign depends on the sign of $2E_0 - E_1 - E_2$, which is negative according to our hypotheses ($E_2 \approx E_0$, $E_1 > E_0$). Thus, for relatively small inputs, the interaction implemented by the circuit of figure 1*c* is of the type $(g_1 - \alpha g_1 g_2)$. Note that the crossterm is different from zero only when the two conductance changes are simultaneous at the level of the postsynaptic membrane. Otherwise only the selfterms remain. Therefore, if the two conductance changes derive from movement of a spot in front of the receptors 1 and 2 of figure 1*c* the crossterm will be zero in the 'preferred' direction and will give an inhibitory contribution in the 'null' direction, eventually offsetting the excitatory input. Thus, the specific 'shunting' mechanism of figure 1*c* could show the same behaviour as was observed by Barlow & Levick, while implementing a non-linear interaction which is almost identical to the Reichardt's multiplication. The idea that this operation may be realized by some form of shunting inhibition was proposed by Thorson (1966).

There are three points here that are worth mentioning. First, this type of multiplicative inhibition is critically dependent on the spatial arrangement of the synapses. If the two synapses are not spatially adjacent (their distance being larger than about one-tenth of the membrane length constant) the circuit of figure 2 is not a faithful model: in this case the effects of the conductance changes would essentially summate linearly (Rall 1964; Jack *et al.* 1975). Linear summation of the effects of the conductance changes would also occur if the conductance changes were very brief (much briefer than the membrane time constant). The second point is that the mechanism of figure 1*c*, localized at the various dendritic branches of a cell, can subserve direction selectivity of this cell over a large receptive field. The objection to the simple threshold scheme does not apply here, because the multiplicative interaction would take place at the level of the dendritic membrane before spike generation. A third remark concerns the facilitatory effect that has been reported under some conditions in the preferred direction (Barlow & Levick 1965; Mimura 1972). If the two conductance changes do not overlap in time (the crossterm then disappears) and if E_2 is slightly more positive than E_0, an increase of g_2 leads to a flow of depolarizing current (through the g_2 terms of equation (4)), which adds at the soma to the current induced by g_1.

The nonlinearity of the interaction between conductance changes clearly plays a very important rôle in our proposal. Instead of being an undesirable side-effect of the mechanism of synaptic transduction, nonlinearity is in our postulate the key property. Thus, it is to be expected that synapses implementing this type of interaction will satisfy the following conditions that are known to maximize the degree of nonlinearity: (*a*) adjacent locations (within one-tenth of the membrane length constant); (*b*) suitable (roughly simultaneous) timing and relatively long duration of conductance changes; (*c*) small input conductance of the dendritic cable relative to the synaptic conductances. Condition (*c*) is, for instance, increasingly satisfied with decreasing diameter of the dendritic branch and with

increasing distance of the synapses from the soma. Synapses on dendritic spines would also show, for the same reason, a high degree of nonlinear interaction. Thus condition (*c*), together with the requirement that inhibition must be selective in its action, suggests that the best location for these synapses is on distal, thin dendrites. This also ensures the slowest passive decay of postsynaptic potential at the soma. Reasonable approximations of these properties are required by our model and may characterize synapses that implement this or similar nonlinear interactions. Equations (1) to (6) can be shown to be a special case of a general theory that yields the exact dependence of the somatic potential on conductance changes at arbitrary locations in a dendritic tree (Poggio & Torre 1978).

It must be stressed that our proposal leaves open the problem of the detailed circuitry underlying direction selectivity in either the fly, the rabbit retina, the geniculate or the cortex (Wyatt & Daw, 1975; Schiller *et al.* 1976; Emerson & Gernstein 1977; Sillito 1977). The existence of presynaptic pairs (two 'adjacent' presynaptic elements, one postsynaptic), that could implement the circuit of figure 1*c*, is not ruled out by known structural studies (Boschek 1971; Stell 1972; Wong-Riley 1974; Boycott 1974). The origin of the delay or lowpass operation remains speculative. The duration of the required delay depends on the time courses of g_1 and g_2 in a patch of the dendritic membrane. A delay (or lowpass operation) could arise from slower conduction through channel 2 and/or from the different kinetics of the conductances g_1 and g_2 (Gardner & Kandel 1972; Kehoe 1972; Levitan & Tauc 1972; Berry & Cottrell 1975; Kandel 1976).

Of course, simple alternatives to the scheme proposed here are, for instance, presynaptic inhibition and action potential mechanisms in dendrites. Although we have some arguments against these possibilities, only experiments can decide between the various hypotheses. Recent data of Marchiafava (1978) on directionally selective ganglion cells in the turtle's retina suggest that postsynaptic inhibition of the type proposed here underlies, at least in part, the property of directional selectivity.

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