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ON THE ANALYSIS OF NERVE SIGNALS DEDUCED FROM METACONTRAST EXPERIMENTS WITH HUMAN OBSERVERS

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SUMMARY

1. This paper reviews Alpern, Rushton & Torii's (1970*a–d*) derivation of the size of the inhibitory nerve signal arising from after flashes in the metacontrast experiment.

2. Their geometric argument is recast in terms of simple functional equations. This form of argument clearly displays the role of their assumptions in obtaining their main conclusion: nerve signal is linear in intensity over a range of 3–4 log units.

3. Two disadvantages of their approach are discussed. First, it is noted that in the presence of the data the assumption they employ in their analysis is logically equivalent to their conclusion.

4. Secondly, accepting their claim that the nerve signal generated by the after flash is linear over a broad range of intensities, and that this inhibitory signal simply cancels the excitatory signal of the test flash, leads to the conclusion that over this same intensity range the excitatory nerve signal is a power function with an exponent of close to two. This is incompatible with the suggestion that photoreceptor signals have been measured.

INTRODUCTION

The phenomenon of metacontrast consists of a suppression of one flash (the target flash) by a second flash (the after flash, sometimes called the contrast flash or masking flash) which occurs later in time and falls on a different region of the retina. General reviews of the literature may be found in Lefton (1973), Kahneman (1968), Raab (1963) and Weisstein (1972).

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In an important series of experiments, Alpern, Rushton & Torii (1970*a–d*, henceforth referred to as A.R.T. following their convention) use the phenomenon of metacontrast to determine various properties of the photoreceptors. One of the properties which they investigate is how the size of the inhibitory nerve signal generated by the after flash varies with the light intensity of the after flash. They conclude that the inhibitory nerve signal grows linearly with the intensity of the after flash, over a range of nearly four log units, until saturation begins. The argument which they use to deduce the inhibitory nerve signal size from their data is geometric in character. A review of this argument is provided by Rodieck (1974).

The following section of this paper provides a brief description of their experiments. Then, a convenient mathematical notation will be introduced which will permit us to express simply the empirical results of the metacontrast experiments (eqn. (2)) and the assumption of proportionality between inhibitory nerve signal size and the area of the after flash (eqn. (3)).

This paper makes two points about their analysis. First, it is shown that the assumption that nerve signal is proportional to area is necessary to arrive at the conclusion that nerve signal is proportional to intensity. Once the data have been analysed, it is seen that they demand that the function relating nerve signal to area and the function relating nerve signal to intensity must be the same. The data do not specify what the function is. The choice of a particular function to describe the relationship of inhibitory nerve signal with area is equivalent to choosing the function for light intensity.

Secondly, the data provide us with a quantitative relationship between intensities of the target flash and after flash at threshold (eqn. (1)). If the inhibitory nerve signal just cancels the excitatory signal at threshold, any deductions concerning the inhibitory signal will lead to deductions about the excitatory signal as well. If we are deducing properties of photoreceptors, these two functions should coincide. This follows because the relationship between photoreceptor signal and intensity should be the same independent of the role the signal subsequently subserves. In fact, this turns out not to be the case, which therefore suggests that it is not photoreceptor signals which are being deduced.

BRIEF DESCRIPTION OF THE EXPERIMENTS

In the experiments where the rod response to light intensity was determined by A.R.T., the target flash was a bluish-green dot of 2° in diameter and 10 msec duration. The after flash was an annulus with outer diameter of 8° and inner diameter of 2°. The centre of the target flash fell

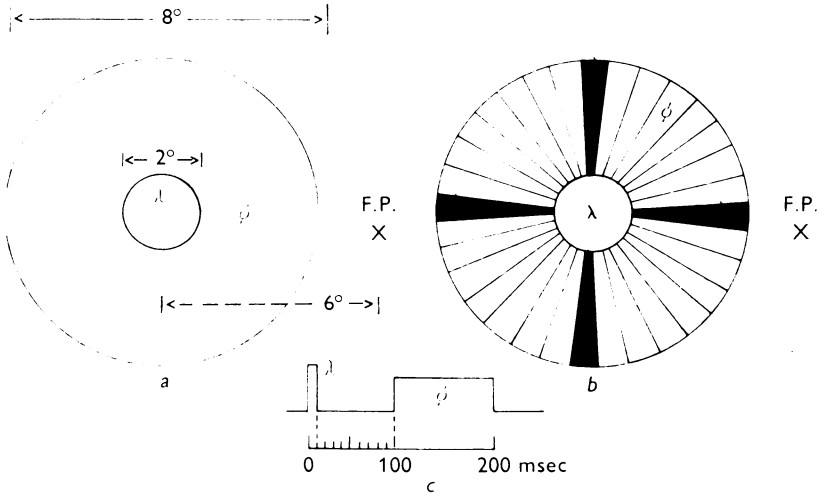

Fig. 1. Spatial and temporal relation of metacontrast stimuli used in rod experiments (redrawn from A.R.T., 1970*a*).

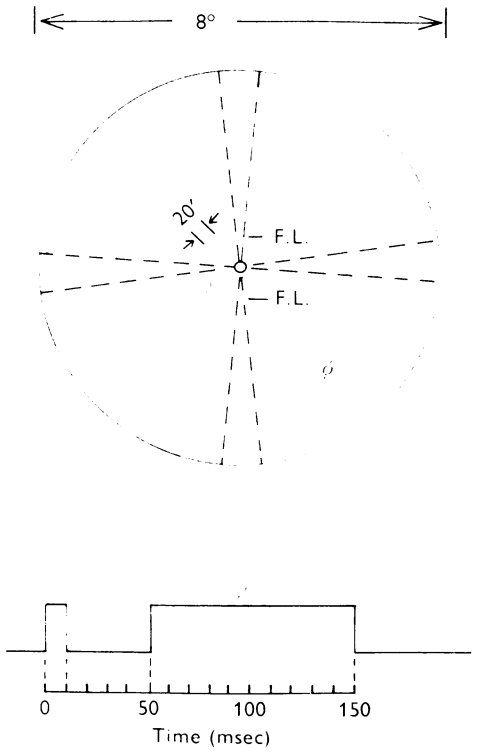

Fig. 2. Spatial and temporal relation of metacontrast stimuli used in cone experiments (re-drawn from A.R.T., 1970*d*).

6° temporal from the fixation point and coincided with the centre of the annulus. The time between target offset and after flash onset (ISI) was 90 msec. In the cone experiments various colour dots were used, depending on the cone system under study. The target flash was reduced to 20′ of diameter and fell on the fixation point. The outer diameter of the annulus was again 8°, but the inner diameter was reduced to 20′. The ISI was also decreased to 40 msec. These facts are summarized in Figs. 1 and 2 which are redrawn from A.R.T. (1970*a*, *d*). The duration of the after flash was always 100 msec.

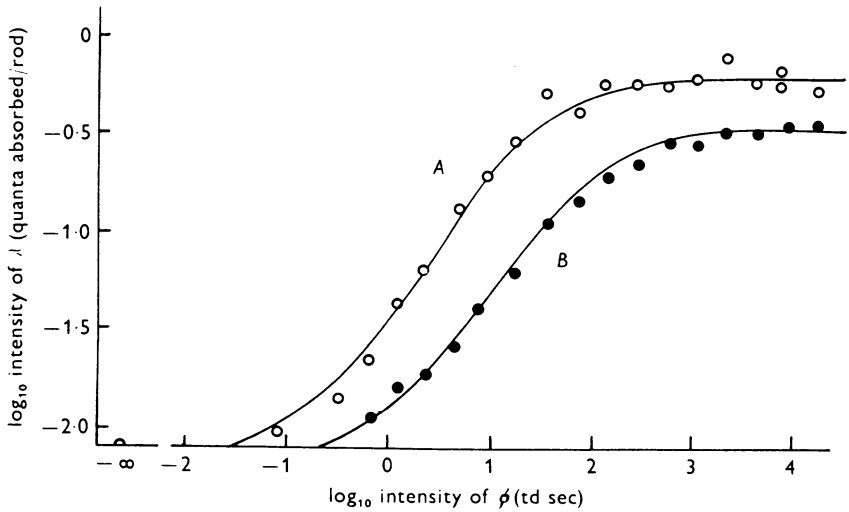

Fig. 3. A plot of log threshold of the test flash as a function of log intensity of after flash for the rod experiment. Curve *A* from experiment with full background and *B* when one-eighth windmill stop was interposed in after flash field (re-drawn from A.R.T., 1970*a*).

Using these stimuli, A.R.T. then performed the following experiments. In one experiment the after flash consisted of the full annulus. The threshold value of the target flash was determined for a wide range of after flash intensities. In the second experiment a 'windmill stop' (see Fig. 1) was interposed in the after flash field. This reduced the area of the after flash by a factor of 8. Then the threshold value for the target was again determined for a wide range of after flash intensities.

Data from the rod experiment are presented in Fig. 3 (from A.R.T., 1970*a*). These are plots of target threshold *vs.* after-flash intensity. The parameter of the curves is the area of the after flash. Similar functions were found for the cone systems (cf. Figs. 2 and 3 in A.R.T., 1970*d*). Since the relevant experiments all employed windmill stops that were symmetric

about the centre of the annulus, when I refer to the area of the after flash it will be understood that this area is a radially symmetric area. The conclusions stated may not hold for other definitions of area.

NOTATION

Throughout the paper I will follow the convention of denoting functions as Latin capital letters and numbers by lower case Greek letters. Following A.R.T. I will reserve the letter λ to mean the intensity of the target flash, and ϕ to mean the intensity of the after flash. The letter ω will be reserved to denote radially symmetric area.

Our task, then, is to find the size of the inhibitory nerve signal as a function of the area and intensity of the after flash. Let us denote this function as $N^*(\omega, \phi)$. We will suppose that the target flash also produces a nerve signal, and we will denote this signal size as $N(\lambda)$. Both of these functions depend on many other experimental parameters and any useful theory will provide a means of ultimately incorporating them. However, since these parameters were fixed throughout the experiments to be discussed, we will not explicitly represent them in our notation.

Finally, it will be convenient to write down an expression which relates the experimentally measured values λ, ϕ, and ω for the portion of the data where intensity trades off multiplicatively with area (A.R.T. refer to this as the linear portion of Fig. 3). The derivation of this expression is in the Appendix, and can be written as

$$\lambda = \beta(\omega\phi)^{\alpha}, \tag{1}$$

where α and β are fixed, real numbers whose values depend on the particular experiment.

DISCUSSION

A.R.T. deduce the form of the inhibitory nerve signal function $N^*(\omega, \phi)$ from the following considerations. First, they describe the main empirical finding: a reduction in the area of the after flash by a factor of 8 must be compensated by an increase in luminance by a factor of 8 to produce an equal test threshold. This fact does not rest on the value 8, but they go on to say (A.R.T., 1970*a*, p. 206), 'It was always found when the surrounding area was reduced to a fraction $1/n$ that the flash had to be increased n times for λ to be maintained at threshold.'

In our notation we can represent this finding as

$$N^*(\omega, \phi) = N^*(\gamma\omega, 1/\gamma \times \phi). \tag{2}$$

Eqn. (2) portrays the fact that if we change the radially symmetric area by a factor of γ, we must change the after flash intensity by a factor of $1/\gamma$ to produce the same inhibitory nerve signal size.

Eqn. (2) is an empirical finding. It is not sufficiently strong to determine the form of N^* uniquely. To accomplish this A.R.T. (1970*a*, p. 197) introduce an assumption, 'The full surround ... may be thought of as consisting of thirty-two sectors each of the size of one windmill sail. Their total inhibitory effect on λ at the centre is thirty-two times the effect of one sector.'

Presumably the number thirty-two is arbitrary. We can represent this assumption by the following equation:

$$N^*(\gamma\omega, \phi) = \gamma N^*(\omega, \phi). \tag{3}$$

By combining eqns. (2) and (3) it follows that

$$N^*(\omega, \gamma\phi) = \gamma N^*(\omega, \phi)$$

or

$$N^*(\omega, \phi) = \phi N^*(\omega, 1). \tag{4}$$

Eqn. (4) states that the inhibitory nerve signal is linear in light intensity. This is the main conclusion of A.R.T.'s analysis of N^* and applies to values of intensity somewhat less than saturation.

Two points need to be made. First, the data alone do not suffice to prove eqn. (4). They do, however, place a strong constraint on N^*. If we set $\gamma = \phi$ in eqn. (2) we may write

$$N^*(\omega, \phi) = N^*(\phi\omega, 1) = G(\phi\omega). \tag{5}$$

where G is an arbitrary, monotonic function. Thus, even though linearity does not follow from eqn. (2), we have reduced N^* from a function which depends on two real numbers to a function which depends only on a single real number. To make further claims about the shape of G further assumptions are needed. The assumption that A.R.T. chose, represented in eqn. (3), yields

$$G(\gamma\omega\phi) = \gamma G(\omega\phi),$$
$$G(\omega\phi) = G(1)\omega\phi.$$

Suppose, however, we were to make a slightly different assumption, such as the more general one.

$$N^*(\gamma\omega, \phi) = F(\gamma)N^*(\omega, \phi)$$

or

$$G(\gamma\omega\phi) = F(\gamma)G(\omega\phi), \tag{6}$$

where we assume only that F is a monotone, increasing function. We then write

$$G(\phi\omega) = F(\phi)G(\omega) \tag{7a}$$

and for $\omega = 1$

$$G(\phi) = G(1)F(\phi). \tag{7b}$$

It is clear, then, that any assumption we choose to make about the area function, F, determines the solution we obtain. Put another way, eqn. (5) says that the function relating inhibitory nerve signal to area and the

function relating it to intensity are the same, namely G. Assuming that nerve signal is related to area linearly is therefore formally equivalent to assuming it is linearly related to intensity.

There are, however, strong consequences of the assumption in eqn. (6). This assumption constrains G even if we make no assumptions about F. Substituting eqn. (7*b*) into eqn. (7*a*) yields

$$G(\phi\omega) = \frac{G(b)G(\omega)}{G(1)}.$$

It is well known (cf. Aczel, 1966) that the only continuous solution to this equation is a power function. So then, eqns. (2) and (6) yield

$$N^*(\omega, \phi) = G(\omega\phi) = G(1)(\omega\phi)^{\rho}.$$

We can summarize the situation as follows. The data alone do not specify the nerve signal function G. They show only that some G exists. If we wish to make the further assumption that G has the property given in eqn. (6) we may conclude that G is a power function. The deduced exponent will be one, i.e. G will be linear, if and only if we assume that it is.

The second point which needs to be made is that eqn. (3) leads to the following asymmetry. Suppose that at threshold the excitatory nerve signal from the target flash is equal in magnitude to the inhibitory signal so that the two precisely cancel. At threshold, then, we write

$$N^*(\omega, \phi) = N(\lambda).$$

If we replace λ by its analytic expression for the linear range in eqn. (1) and N^* by its deduced form under eqn. (3), that is A.R.T.'s assumption, we have

$$N(\beta(\omega\phi)^{\alpha}) = G(1)\omega\phi.$$

For this equation to hold, N must be

$$N(\lambda) = G(1)(\lambda/\beta)^{1/\alpha}.$$

Thus, whereas eqn. (3) leads to a linearity result for the relationship between the inhibitory nerve signal and after flash intensity, it leads to a power law relationship between excitatory nerve signal and target flash intensity.

This discrepancy in nerve signal functions would not arise if the empirically measured exponent, α, were one. As can be easily seen in Fig. 3, α is much closer to one half. Thus, the nerve signal function deduced for the small excitatory flash and the large inhibitory flash differ, when we assume eqn. (3). This makes it unlikely that photoreceptor signals have been deduced.

Moreover, this objection remains valid even if the demand that the signals precisely cancel is weakened to either a proportionality assumption

$$N(\lambda) = \delta N^*(\omega, \phi)$$

or a constant difference assumption

$$N(\lambda) = N^*(\omega, \phi) + \delta.$$

The asymmetry can be resolved, however, if we deny both eqns. (3) and (6) and assume instead

$$G(\phi\omega) = \log \phi\omega$$

and at threshold

$$N(\lambda) = \alpha N^*(\omega, \phi) + \log \beta.$$

Then it follows that

$$N(\lambda) = \log \lambda = G(\lambda)$$

and the asymmetry is resolved.

CONCLUSIONS

Two points have been made in this note. The first is to deny the claim of A.R.T. (1970*a*, p. 193) that they 'have established . . . the [inhibitory nerve] signal *must* be proportional to quantum catch' over the portion of the metacontrast data where area trades off perfectly with intensity. This conclusion is directly traceable to an assumption they make which is never tested directly. Their data suggest that over a large range inhibitory nerve signal follows the same function for area and intensity. The data do not specify what the function might be.

Secondly, their suggestion that the nerve signal function from the inhibitory flash is linear over a broad intensity range, combined with the idea that the signal from the inhibitory flash cancels the signal from the test flash, forces the conclusion that the signals generated by these two flashes are quite different. This is not easily compatible with the claim that their analysis has yielded the function relating photoreceptor response to light intensity.

APPENDIX

To prove eqn. (1) we need to discover the function, say D, which will compute the threshold value, λ, for any symmetric area and light intensity within the linear portion of the data (Fig. 3). On the linear portion of the graph we have

$$\log D(\omega, \phi) = \log \lambda = S(\omega) \log \phi + A(\omega),$$

where S and A area real-valued functions of the symmetric area. Equivalently

$$\lambda = D(\omega, \phi) = 10^{K(\omega)} \phi^{S(\omega)}.$$

Since $D(\omega, \phi)$ also obeys eqn. (2), that is

$$D(\omega, \phi) = D(\gamma\omega, 1/\gamma \times \phi),$$

we may write

$$D(\omega, \phi) = H(\omega\phi).$$

For $\omega = 1$ we have

$$D(\omega, \phi) = H(\omega\phi) = H(\phi) = 10^{A(1)}\,\phi^{S(1)}.$$

Thus H is a power function. Letting $\beta = 10^{A(1)}$ and $\alpha = S(1)$ we conclude

$$\lambda = D(\omega, \phi) = H(\omega\phi) = \beta(\omega\phi)^{\alpha}.$$

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