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I. Keynote Presentation

The Significance of Antidromic Potentiation and Induced Activity in the Retina

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Our work on the role of the centrifugal gamma fibres to the muscle spindles in the late forties and early fifties made me consider centrifugal fibres to the mammalian eye. RAMON Y CAJAL (1) has reported the existence of such fibres in the retina of the dog and I did not think it very likely that he could have been mistaken. Others have since shown less faith in the Old Master, but in the laboratory of another highly competent histologist (POWELL) centrifugal fibres have again been found in the optic nerve of the cat. I quote: "These electron microscopic observations of the retina following lesions of the central visual pathway may be accepted as valid evidence for the presence of centrifugal fibres to the retina in the mammal" (BROOKE, DOWNER and POWELL (2)).

I mention this to explain why my stereotaxic attack on this problem made me in the first instance go for the superior colliculus in the cat, rather than for the lateral geniculate body or the optic tract itself (GRANIT (3)).

At the time it was held that merely slow fibres pass to the colliculus and so it came as a surprise that it proved very easy to activate the large ganglion cells by antidromic shocks to that region. When the pick-up electrode was shifted from the point of entry of the optic tract toward the periphery, the latent period increased from about 1.2 ms at the lamina cribrosa to between 4 and 6 ms further out. I concluded that, inasmuch as timing has informative relevance, the retina is admirably organized for translating surface coordinates into time coordinates, provided that the eye moves. These relations were then systematically explored by DODT (8). I was thinking of the familiar Pulfrich effect.

I then tried tetanizing antidromically for some 10 or 20 s and, to my great

surprise, as soon as stimulation was stopped, the isolated ganglion cell started firing at a rate greatly exceeding its previous spontaneous activity. Orthodromic potentiation was well known at the time but nobody had yet succeeded in obtaining an antidromic potentiation. The prevailing notion was that the antidromic spike entering its axonal ganglion probably extended its depolarizing action into the dendrites but not any further. In motoneurons the most striking effect known today is that of DECIMA and GOLDBERG (5,6): if an antidromic ventral-root shock is suitably timed relative to a conditioning, adjacent dorsal root potential, the shock fires a dorsal root spike.

In my experiments all cells did not produce a post-tetanic potentiation but all those that did had to be driven by the antidromic shocks. This suggested that the post-tetanic discharge hardly could be a centrifugal phenomenon, even though a centrifugal contribution could not be excluded. Driving of the ganglion spike was a too obvious conditio sine qua non. Frequency of tetanization and its duration were decisive in determining the duration and firing rate of the post-tetanic discharge. This sometimes reached values as high as 200-300 per s maintained for minutes. As I remember, most of my preparations were BREMER'S encephale isole, some were on pentobarbitone anaesthesia. The facilitation sometimes lasted for a couple of minutes and active cells often began firing already during tetanization, rate of stimulation permitting. The effect could be obtained also from the geniculate body and the optic tract but from these structures it was commonly complicated by inhibitory phenomena.

An idea of the degree of post-tetanic facilitation could be gained by translating firing rates into light intensity. Thus, for instance, a test flash of 5 l.c. was raised in effectiveness in the post-tetanic state so as to correspond to one of 600 l.c. DODT (7), later experimenting with rabbits, found the flicker-fusion frequency of a given test light raised from 16.5 before to 35 flashes per second after antidromic tetanization.

Decisive for my conclusion that the antidromic spikes actually entered the retina were a number of experiments on interference between tetanization and light stimulation. Thus, for instance when the antidromic spike failed to enter the ganglion cell during an inhibitory phase of the light test, it could be made to do so by merely increasing stimulus strength of the shock thereby bringing in other collicular terminals.

Recent experiments by others have since pushed the study of antidromic potentiation one step further and so I shall not review my old work in greater detail. Sixteen years later the problem was taken up by FUKADA (8) who confirmed my findings and connected them with ENROTH-CUGELL and ROBSON'S (9) important subdivision of the retinal ganglions into X- and Y-cells. FUKADA showed that the potentiation only occurred in the transiently responding Y-cells which he called Type I and not in the tonic Type II or X-cells which to a stationary stimulus, focused on the centre of the receptive field, respond with sustained discharge. The significant point here is that the post-tetanic effect was confined to an identified cell, even though at that stage the X-Y identification was tentative.

The next step (FUKADA and SAITO (10)) was to demonstrate that a similar long lasting after-discharge followed a flickering stimulus to the Type I-cell receptive-field organization. FUKADA proposed the term "induced activity" which from now on I, too, intend to make use of. SAITO and FUKADA (11) confirmed the capacity of flickering stimulation to elicit induced activity and studied the responses of Type I and Type II cells to intermittent light. Even when they combined antidromic and flickering stimulation, only the Type I cell proved capable of generating induced activity.

These findings were confirmed by CLELAND and LEVICK (12) who found induced activity only with their transient class of cell, apparently FUKADA'S Type I. From latency studies of the ganglion spike, combining light and optic tract stimulation, they arrived at the conclusion that the induced discharge "is associated with the appearance of an active spike-generating focus located somewhere along the axis of that cell".

The optic tract loses its myelin sheath at the lamina cribrosa and so the axons in their intraretinal course may acquire the complex properties of dorsal root C fibres. These are known from GASSER'S (13,14) studies of their spikes and after-potentials. This analogy may or may not be valid, but if it be, then one would expect the effect of a long-lasting tetanus to emerge as GASSER'S (14) P_2 or second positive after-potential which is of very long duration. During this hyperpolarization negative after-potentials of spikes are increased. It is not easily understood how a positive after-potential could be conducive to facilitation of the ganglion. If on the other hand after loss of its myelin sheath a fibre retains the original properties of A fibres, a tetanus would probably be followed by a brief hyperpolarization, P_1 , rapidly changing into a depolarization, also of relatively brief duration compared with the final, long-lasting, positive P_2 , the only event of a duration long enough to approach that of an induced discharge. However, the polarity of P_2 is of the wrong sign.

In the intraretinal optic tract fibres of the monkey OGDEN and MILLER (15) noted an "intense negative post-tetanic overshoot". There was little in the way of positive after-potential. While this transient effect may aid the ganglion cell in forwarding OGDEN'S P-wave into the internal plexiform layer--I shall come to it below--no correlation is thereby established with the long-lasting induced discharges, so far not at all studied antidromically in the monkey retina. The post-tetanic negativity of OGDEN and MILLER is too brief to explain the long-duration of the induced activity. These problems clearly require more experimentation, an attack with microelectrodes on the internal plexiform layer.

At the moment we had better hold onto the two most significant new observations in this field: (i) that the induced activity can be elicited both by flicker from the orthodromic end as well as by repetitive antidromic stimulation (ii) that both routes of activation presuppose a specific set of large ganglions, apparently those representing the final common path of the synaptic organization that are driving the Y-cells of ENROTH-CUGELL and ROBSON. An explanation based on a purely extraretinal axonic focus, not yet demonstrated, suffers from the weakness of not being able to account for the orthodromic effect of flicker and for the restriction of induced activity to merely one type of the approximately 200,000 fibres counted by HUGHES and WÄSSLE (16) in the cat's optic tract.

Some of the steadily multiplying studies which now are devoted to cat ganglion cells seem to be of particular interest in the present connection. BOYCOTT and WÄSSLE (17) described three main types of ganglions: large alpha cells with dendritic networks spreading laterally up to $1000\mu\text{m}$, smaller beta cells with a field diameter of $25\text{-}300\mu\text{m}$, and still smaller gamma cells with a dendritic field between 180 and $300\mu\text{m}$. The identification proposed was: Y-alpha, X-beta, and W-gamma. The identification was based on the size of the perikaryon in combination with that of the dendritic network. For all cells the latter expands in size towards the periphery. I shall only be concerned with the Y-alpha type. It is generally accepted that the larger the perikaryon, the greater also the axonal diameter and hence the conduction velocity. The ganglion cells responding with induced activity are found among the large ones that are supported by extensive dendritic networks.

This identification was fully supported by HOFFMANN (18) and by CLELAND, LEVICK and WÄSSLE (19) who added the further specification that the Y-cells are the brisk-transient units of CLELAND, DUBIN and LEVICK (20) and CLELAND and LEVICK (21).

According to a suggestion by OGDEN (22) the antidromic spikes in the optic tract may enter the inner plexiform layer by mediation of the tight junctions discovered by DOWLING and BOYCOTT (23), which for good reasons were held to be electrical in nature. SAITO and FUKADA similarly assumed these junctions to give access to the internal plexiform layer. The tight junctions are axosomatic ones between bipolar terminals and somata of ganglion cells. DOWLING and BOYCOTT did not find them in all cone portions of the primate retina and suggested that they were characteristic of rod bipolars.

When OGDEN endowed them with the role of gate openers to the internal plexiform layer, this was done in order to explain the positive P-wave that he and BROWN (24) had found in that layer in response to antidromic shocks. OGDEN did not find any P-waves in the cat retina. In similar work GOURAS (25) recorded a graded potential at the internal surface of the monkey retina. This potential became positive in the internal plexiform layer, had a shorter latency in the periphery, longer in the centre where it also was larger and more drawn-out. The views of these two authors on the nature of the P-wave differ, but a more serious difference from the present point of view is that tight junctions were not found in the centre where the positive wave of GOURAS had its maximum size.

As such it is of course a plausible notion that an antidromic spike--whatever it does afterwards--is gated into the internal plexiform layer by such apposition contacts. Again, however, we must conclude that there is room for more studies of the microphysiology of this region.

Returning to the question of why the Y-cells or, probably, only certain Y-cells generate induced activity, it stands to reason that a large dendritic network, better than a small one, by chance alone is bound to provide more targets for the output of those amacrine cells that are charged with the task of maintaining lateral spread of excitation. Additionally, it is pointed out by DOWLING and BOYCOTT (23) that "no ganglion cell dendritic spread is large enough to account for these effects (meaning the long-distance McIlwain effect); the only direct pathways in the retina for the peripheral effects are via the amacrine-amacrine synapses" (p. 107).

However, since the explanation of specificity in producing induced activity somehow implicates Y-cells, it is not permissible to neglect their curious property of non-linear summation within the receptive field. To ENROTH-CUGELL and ROBSON (9) this was an essential criterion in their definition of Y-cells. I call this property curious, because my own experience with motoneurons, in both intra- and extracellular work, is that, within a large range of firing rates highly complex reflexes add in a strictly linear fashion (summary, GRANIT, 26). Similarly ENROTH-CUGELL and ROBSON found linear summation in the X-cells.

I therefore suggest that the non-linear summation of the Y-cells is a consequence of positive feedback within the amacrine circuits that support their activity. This, at the same time, would explain their proneness to excessive activity, noted also by ENROTH-CUGELL and ROBSON (9) when they stated that "the mean discharge of the Y-cells (unlike that of X-cells) was greatly increased when grating patterns drifted across their receptive fields". The nowadays commonly used Y-cell criteria, high conduction velocity, transient response, and a more peripheral location, need not in every case tally with that of non-linearity. In present day usage of the X-

Y nomenclature the original Y definition is mostly neglected. If we had had experiments correlating induced activity with non-linearity of summation, we would now be better off in discussing the nature of Y-specificity.

Of relevance for this line of thinking are some data by DUBIN (27) dealing with the serial synapses of KIDD (20). DUBIN found them to be characteristic of amacrine cells and published a table showing among other correlations the percentages of amacrine synapses in serial configuration in different animals. These are some of his figures: human parafovea 1.9; monkey fovea 2.5, parafovea 7.1, periphery 5.1; cat (2 animals) 8.2 and 7.5 respectively; rabbit (2 animals) 10.0 and 15.2 respectively. With these challenging figures we again come up against questions of correlation which only can be answered by appropriately designed experiments.

One task of those Y-cells which respond as if they were actuated by positive feedback could be to facilitate the recording of movement in the peripheral visual field and conduct the message at maximum speed to the cortex. This notion presupposes that the X-Y differentiation be maintained up to the central visual stations. For the geniculate body this has been found to be the case, in the cat (HOFFMANN, STONE, and SHERMAN (20); FUKUDA and STONE (30) and in the monkey (DREHER, FUKUDA and RODIECK (31). In this animal the Y-cell projections are found in the magnocellular layer, the X-cells in the parvocellular layer.

In addition to serving as transient fast detectors of movement, the Y-cells also contain information on luminosity. In now forgotten papers and in reviews (GRANIT (32) being the latest) the evidence was summarized that long ago led me to the conclusion that the dominators also in the cat are composite curves carrying a message of luminosity and not one of color. It was shown that the same, large ganglion cell could serve as both scotopic and photopic dominator, this being true also for the retina of the cat. The destination of a message that has this character could hardly be a specifically color-sensitive mechanism in the cortex. I had, in fact, postulated that in all animals the dominator originated a luminance channel.

In now proceeding to discuss some results of primate physiology in terms of Y- and X-cells, I am fully aware of gaps in our knowledge that have to be bridged by hypotheses. I am defending myself with an enlightening quotation referable to PEYTON ROUS: "Yet since what one thinks determines what one does in cancer research, as in all else, it is as well to think something" (from obituary by DULBECCO (33)). And, to begin with, I think that the MCILWAIN effect (34) and its younger descendant, the "shift-effect" of FISCHER and KRÜGER (35), may well be exponents of the particular Y-cells that give induced discharges. It was pointed out by WERBLIN and COPENHAGEN (36) that the MCILWAIN effect is restricted to the Y-cells.

The relation between spectral sensitivity, conduction velocity, and phasic versus tonic properties has been studied in the monkey by GOURAS (37,25), later continued in work with De MONASTERIO and TOLHURST (38, 39, 40). Antidromic stimulation differentiated two main groups of fibres, large ones responding phasically with a conduction velocity of 3.8 m/sec and small tonically responding fibres conducting at 1.8 m/sec. The small ones were found everywhere but had their greatest density in the center. They had opponent color properties and thus the two opposing regions of the receptive field had different color sensitivities, e. g., one red, the other green. The large phasic cells represented the same spectral sensitivity in both center and periphery of the receptive field and so the antagonism between center and surround did not differentiate wavelength. In this lot would be found the Y-cells with dominator properties or, in other works, the fast luminosity instrument of vision. I

shall come to some other papers, psychophysical or based on evoked potentials that separate luminance from color channels, but let me, to begin with, consider the flicker phenomenon.

My first question is so obvious that I do not think it has ever been raised in the present era of sophisticated search for detectors: why is it that heterochromatic photometry is possible by the flicker method? My answer is that this is a fairly selective response of fast Y-cells of the dominator type specializing on transients. There would be more of them in the periphery (ENROTH-CUGELL & ROBSON (9)), hence more facilitation by interaction in the peripheral retina (GRANIT (41)).

In 1929-30, when I was keen on proving that psychophysics could be translated into the kind of neurology that Sherrington's laboratory had pushed into the foreground of research, I used flicker fusion as an index of excitability. Comparisons were made between center and periphery at 10° , area and intensity of the stimulus being varied. For the photopic fusion frequency as a function of intensity, one had the approximation known as the Ferry-Porter rule

$$f = a \text{ Log } I + b.$$

It was known at the time that stimulus area played a role for the fusion frequency but until our work (GRANIT and HARPER (42)) there had been no systematic analysis of it. A similar relationship was found to hold for area,

$$f = c \text{ Log } A + d.$$

By combining these two rules into one, the equation may be formulated as

$$f = \alpha \text{ Log } I \text{ Log } A + \beta \text{ Log } I + \gamma \text{ Log } A + \delta$$

Tabulating the values of these constants for center and periphery, they came out as in

Table 1

Center	0.90	4.76	1.79	15.40
Periphery	1.68	4.87	4.28	14.03

These values show that the constants α and γ which enter the equation in terms containing Log Area are the ones that undergo a significant increase from center towards periphery. The potent peripheral spatial summation could also be demonstrated with stimuli separated by a portion of the illuminated background. For later contributions to this problem, see BROWN (43).

The assumption that in the peripheral retina there are more Y-cells, of the kind that interact by mutual facilitation implicates a cellular substrate whose existence in 1930 merely could be adumbrated. It was not at the time possible to think in terms of a cellular differentiation that today has become the goal of a steadily increasing number of publications dealing with retinal ganglion cells.

For the cat a study by SAITO and FUKADA (11) differentiates between flicker in Type I and Type II ganglion cells. In the Type I cells the number of spikes per flash increased toward a maximum and then fell off, as repetition rate of stimulation was

increased. The TYPE II cells followed rate of stimulation over a wide range with low and constant average spike frequencies. As stated above, only the Type I cells were capable of generating induced activity.

The psychophysical study of flicker and flicker fusion is a highly formalized field, accessible to quantification from several points of view, e.g. waveform, stimulus intensity, adaptive changes etc. But today, when our interest is centered on cellular identifications, other properties of the perception of flicker should in the first instance attract our attention. One of them is the peculiarly unpleasant sensation of violent flicker that at a certain rate of intermittent stimulation below the fusion point is such a striking experience. If the Y-cells of man, like those of the cat, possess an optimum of spike frequency at a certain rate of intermittent stimulation (SAITO and FUKADA (11)), it may well be that self-excitation of their amacrine loops also is at an optimum at those same stimulus rates.

On the assumption that intermittent stimulation at certain rates is particularly prone to stir up self-excitation, it would, of course, be interesting to study the visual system immediately after some 10-20 seconds of flicker. From the work by myself, DODT, FUKADA and others, reviewed above, one would expect characteristic facilitatory after-effects to occur. This work, to be sure, was restricted to the cat but one would like to have psychophysical experiments in man to fill out the picture. A great deal more could also be done from the point of view of flicker with the cat retina and optic nerve.

When I in 1945 gave up experimental work on color reception I tried to collect what psychophysical evidence there was in favor of some measure of separation of color and luminance channels (GRANIT (44)) but today this task would be a great deal easier. It is no exaggeration to state that the electrophysiological evidence in favor of spectral information being carried by broad-band dominators and narrow-band curves of the type I used to call modulators now has become so convincing that, if psychophysicists fail to find either or both of these channels, one would be entitled to put down their failure to inadequate methods.

KING-SMITH and CARDEN (45) set out to test the idea that visual detection can be based on either channel, depending on which one in a given situation has the lower threshold. They had a white background illumination of 1000 td on which was presented a low test flash, colored, or a white of the same intensity relative to threshold. When the test flash durations were 200 ms all stimuli except yellow were mediated by the chromatic system. But when time of exposure was cut down to 10 ms the chromatic peaks disappeared and what remained was a broad-band curve with maximum at 555 nm. Thus the opponent color system needed a longer integration time than the luminance mechanism. Flicker was found to give the same effect as shortening of time of exposure.

It is interesting to note in ZRENNER'S (46) experiments, in which evoked potentials and psychophysical measurements were compared, that against a white background of 30,000 td and a 10 ms exposure of the test stimulus the chromatic effect was strong in the evoked potentials but barely visible in the psychophysical sensory-threshold measurements. A much longer exposure time was needed for demonstration of color specificity by the psychophysical approach. In monkeys PADMOS and NORREN (27) using evoked potentials in otherwise very similar experiments found intermittent stimulation merely to trace the well-known heterochromatic flicker curve while single exposures gave the three spectral peaks studied in several papers by SPERLING and his co-workers (SPERLING et al. (48)). These are at 450, 530-540, and 610 nm with large dips at 480-490 and 570-590 nm. The technique of SPERLING et al., was behavioral but also psychophysical inasmuch as it made use of trained monkeys rewarded for correct responses.

It is not my intention to discuss color mechanisms. The interest here is focused on the broad-band dominance in the spectral flicker curve by comparison with the prominent peaks and dips in the curves based on single stimuli. The peaks are too narrow and too far removed from the maxima of the three retinal photopigments to represent simple projections of the latter. The nature of the interactions involved has been analyzed by SPERLING and HARWERTH (49).

We need not fall back on psychological interpretations in making our comparison between the two curves. The results of PADMOS and NORREN (47) and those of ZRENNER (46) show that the difference between "flicker curves" and "color curves" also holds for the recording of evoked potentials from monkey and from man who on the evidence of SIDLEY and SPERLING (50) has the same receiving apparatus as the monkey. Rather interesting is the fact that the psychophysically determined color peaks and dips do not come out at short exposures while they do so in the records of evoked potentials. This is not the first experiment in which conscious awareness is shown to be time-consuming. LIBET (51), stimulating the somatosensory area in patients, found each repetitive shock to produce an evoked potential but mobilization of conscious awareness required maintained, iterative stimulation for about half a second.

The gist of my argument should now be clear enough; a luminance channel takes its retinal origin in Y-cells with a dominator distribution of spectral sensitivity. Intermittent stimulation, as employed also in heterochromatic photometry with fusion frequency as an index of brightness, favors this channel of information. The chromaticity channel is likely to be based on the more slowly conducting of X-cells but at the moment it is not possible to conclude that the two channels are wholly independent and incapable of interaction. The degree of their segregation and conditions for their interaction must be established by further experimentation. I have referred above to the work on these lines commenced by GOURAS and his colleagues.

The evidence in favor of self-excitation in Y-cells should not be construed to imply that all Y-cells necessarily have this capacity developed to the degree found in those which in the cat are capable of induced activity. As pointed out above, this antidromic potentiation has not yet been studied in primates. But I have drawn attention to the similarity of the induced effects by flicker and by antidromic stimulation (FUKADA) because it suggests means of approaching the related problems of luminance specificity, Y-cells, their self-excitation, distance effects of the McIlwain type and antidromic potentiation. Intermittent light may well be a good substitute for antidromic stimulation.

I understood from the invitation that I was supposed to speak about my own work in vision, a real challenge considering that it lies so far back. I did not see the need for a fresh summary of it, even though I often find it misinterpreted. Thus, inasmuch as my own work appears in this presentation of recent developments, it serves merely as a kind of accompaniment in the background for a set of ideas that have acquired their form in the last decade.

Summary

"The significance of antidromic potentiation and induced activity in the retina", being the title of this lecture, is held to be that the two effects really are identical and that both are the outward sign of the existence of a specific organization in the retina. For this reason they can serve as a valuable criterion for identifying activity in this organization.

This activity is assumed to be in the nature of a self-excitation by positive feedback in the amacrine circuits of certain Y-cells. Relevant literature has been reviewed.

These Y-cells whose spectral response curve is of the dominator type play a prominent role in stimulation by intermittent light and in the perception of luminosity.

A number of properties of intermittent stimulation are mentioned and held to motivate a renewal of the attention of visual experimenters of "flicker" and its after-effects.

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