
Principles and Practice of Clinical Electrophysiology of Vision

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The b-Wave

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Although the cellular origin of the b-wave and its mechanism of generation remain open to some question, it is generally accepted that this component of the electroretinogram (ERG) is generated in large part by the Müller cells of the retina. The question of b-wave generation has long occupied the efforts of retinal physiologists. Early work in the field demonstrated that the b-wave was generated by the neural retina proximal to the photoreceptors. Application of compounds that block synaptic transmission from the photoreceptors to horizontal and bipolar cells, for example, Mg^{2+} , Co^{2+} , and aspartate,^{18, 22, 63} abolishes the b-wave but leaves photoreceptor responses intact. Occlusion of the central retinal artery, which supplies oxygen to the neural retina but not to the photoreceptors, also abolishes the b-wave but spares photoreceptor responses.¹⁰ These studies clearly indicate that the b-wave is postreceptoral in origin.

Identification of the cell(s) within the retina that generate the b-wave has been a more difficult task. The first evidence concerning the cellular origin of the b-wave was obtained from recordings of the local ERG made with intraretinal microelectrodes positioned at different depths within the retina (Fig 11-1). Such measurements demonstrated that the b-wave had a peak negative amplitude in the distal retina near the outer plexiform layer and that the largest change in amplitude of the intraretinal b-wave occurred across the inner nuclear layer.^{3, 12, 70, 71} These results suggested that the b-wave was generated in the retina by current flowing through a radially oriented cell that acted as a dipole. Bipolar cells, which are the only radially oriented neurons that

span the inner nuclear layer, were thus implicated in b-wave generation.

MÜLLER CELL ORIGIN OF THE b-WAVE

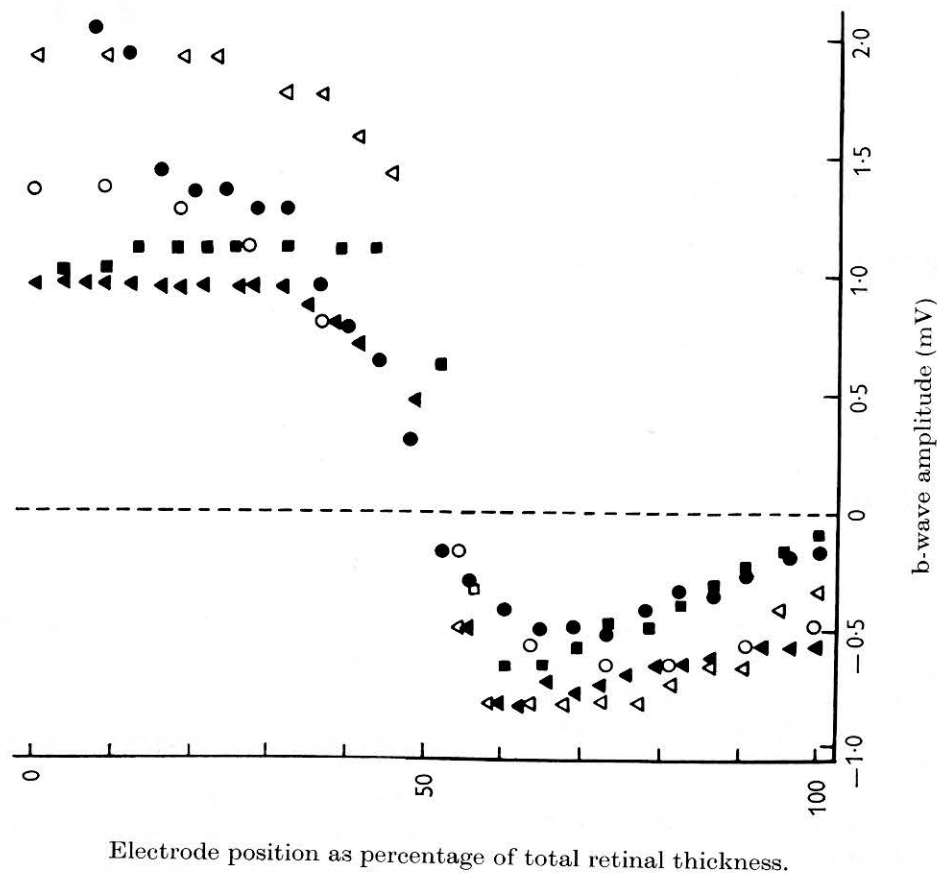
Müller cells, which are the principal glial cells of the retina, are (like bipolar cells) oriented radially within the retina. These cells span the entire width of the neural retina from the inner limiting membrane to past the outer limiting membrane.⁵³ The Müller cell was identified as the likely origin of the b-wave by two important observations: current source density (CSD) analysis and intracellular recording.

Current Source Density Analysis

Faber¹⁹ analyzed b-wave generation by recording the intraretinal b-wave in the rabbit retina and computing CSD profiles from the resulting data. He found that the b-wave in the rabbit was generated principally by a current sink near the outer plexiform layer and by a current source extending from the sink all the way to the vitreal surface of the retina. Faber reasoned that this source sink pattern must arise from current flow through the Müller cell because the Müller cell is the only retinal element that extends from the distal region of the neural retina all the way to the inner limiting membrane.

Intracellular Recording

The conclusion that the b-wave was generated by current flow through Müller cells received consider-

**FIG 11-1.**

Amplitude of the b-wave recorded with an intraretinal electrode in the intact cat eye. Amplitude is plotted as a function of retinal depth, with 0% corresponding to the inner limiting membrane and 100% to the retinal pigment epithelium. The b-wave response reaches its maximal negative amplitude in the outer plexiform layer and undergoes its greatest change in amplitude in the inner nuclear layer. Data are from five separate electrode penetrations (*indicated by different symbols*). The vitreous humor was replaced by a nonconducting oil, and responses were referenced to an electrode in the mouth. (From Arden GB, Brown KT: *J Physiol (Lond)* 1965; 176:429-461. Used by permission.)

able support from the work of Miller and Dowling.⁴² These investigators monitored the intracellular responses of Müller cells of the mudpuppy retina during b-wave generation (Fig 11-2). They found that the b-wave in the mudpuppy closely resembled the light-evoked voltage response of Müller cells in many respects. Müller and b-wave cell responses had similar latencies, time courses (under certain conditions), and responses to flickering stimuli. The two responses also had similar intensity-vs.-response amplitude relations. Both had a dynamic range of approximately five decades, much larger than the one and one half to two decade dynamic range of some retinal neurons.

The Müller cell hypothesis of b-wave generation has received considerable additional support. Newman^{45, 46} performed CSD analyses of b-wave generation that confirmed Faber's findings and contributed

additional details. He found that in the frog the b-wave was generated principally by two current sinks located near the inner and outer plexiform layers and by a large current source at the inner limiting membrane (Fig 11-3). Newman estimated that 65% of the transretinal b-wave response in the frog was generated by the outer plexiform layer current sink, which had a more transient time course than did the inner plexiform layer sink.

A CSD analysis of the b-wave was also conducted in the intact monkey eye by Heynen and van Norren.²⁷ These investigators found a source-sink distribution similar to that of the rabbit: a current sink near the outer plexiform layer and a current source that extended throughout the proximal retina (Fig 11-4). The CSD results from both frogs and monkeys support the hypothesis that the b-wave is generated by a radial current flow through Müller cells.

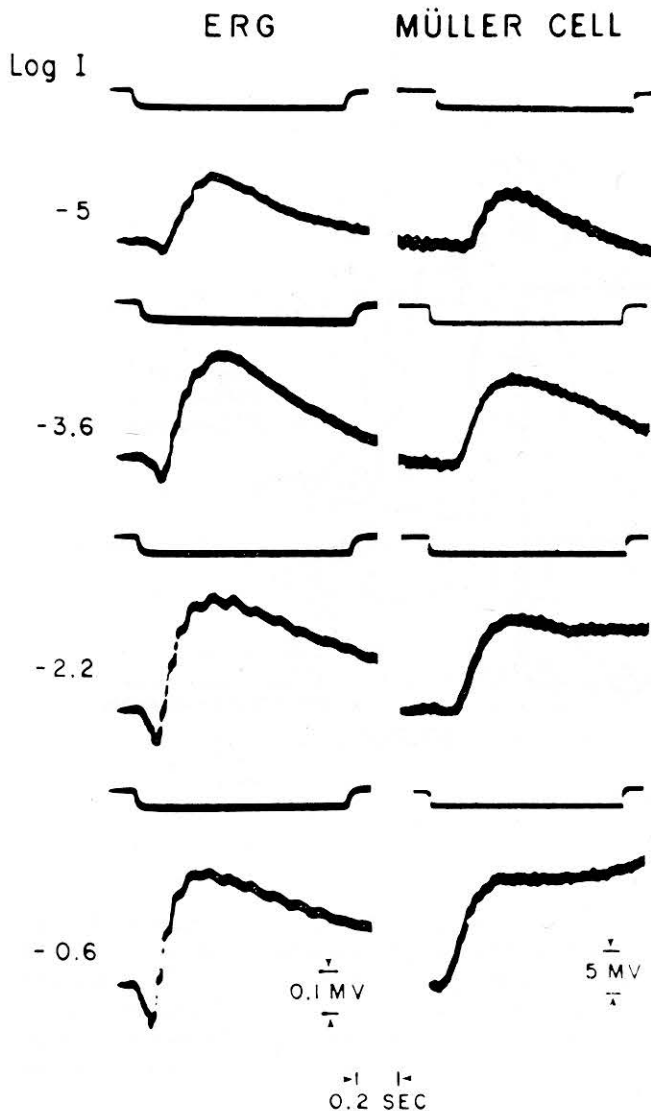


FIG 11-2.

Intracellular Müller cell responses (*right column*) and the ERG (*left column*) recorded from the mud puppy (eyecup preparation). Both the negative a-wave and the larger, positive b-wave are seen in the ERG records. The time course of the Müller cell responses resemble those of the b-wave, particularly at the lower stimulus intensities (-5 and -3.6). Stimulus intensity is given to the *left* of the traces. (From Miller RF, Dowling JE: *J Neurophysiol* 1970; 33:323. Used by permission.)

A developmental study of the chick retina also supports the theory of a Müller cell origin of the b-wave. Rager⁶⁰ found that Müller cell membranes within the outer plexiform layer underwent a marked increase in surface area at the same developmental stage at which the b-wave first appeared.

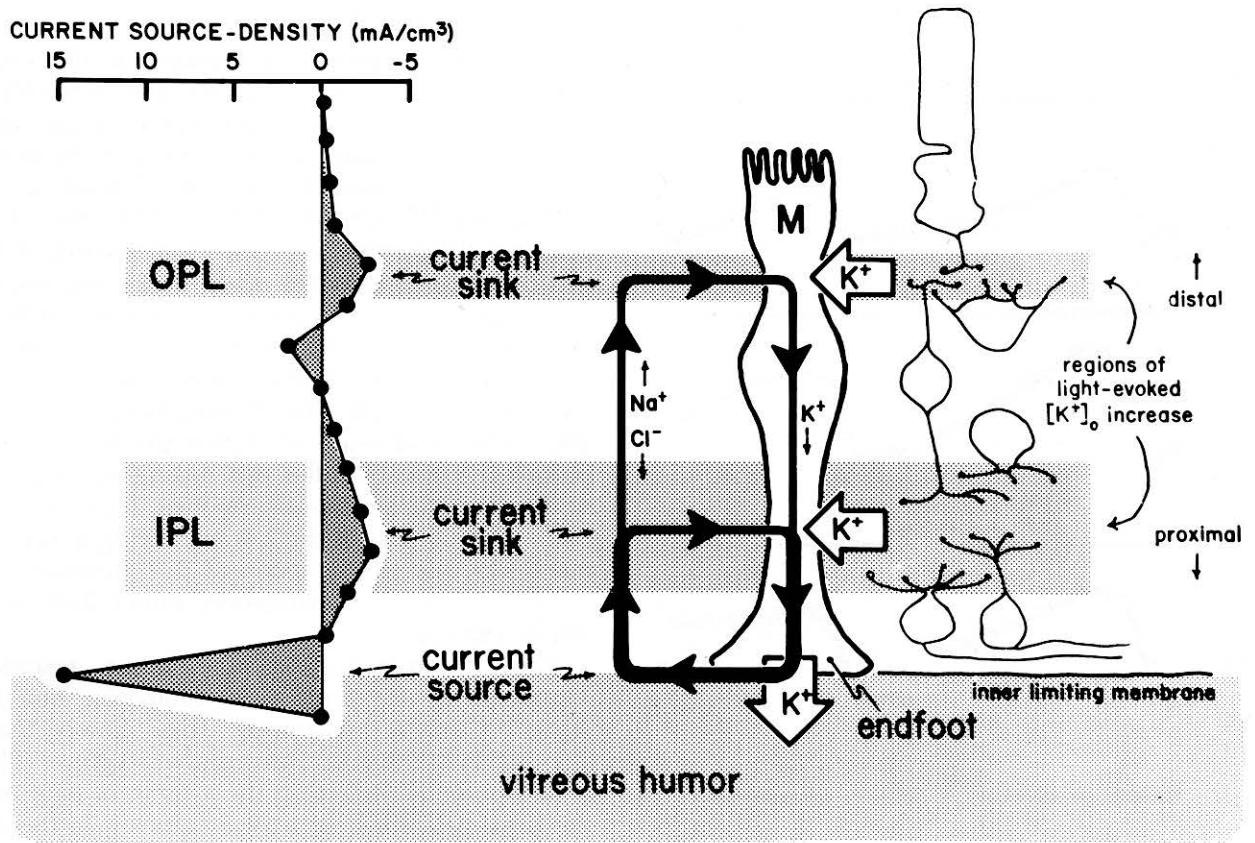
α -Aminoadipic Acid

The gliotoxic action of α -aminoadipic acid (α -aaa) has also been used to test the validity of the Müller cell hypothesis of b-wave generation. α -aaa causes morphological damage to Müller cells in the skate,⁶⁹ rabbit,⁶² and the frog and chicken^{5, 6} while causing little damage to retinal neurons. α -aaa treatment also abolishes the b-wave response while leaving, at least under certain conditions, other visual responses intact. These results suggest that Müller cells are needed to generate the b-wave. However, Zimmerman and Corfman⁷⁴ showed that α -aaa can abolish the b-wave without directly damaging Müller cells. The L-isomer of α -aaa abolished the b-wave while leaving a normal Müller cell resting potential and sparing the Müller cell off-response. They suggested that the initial loss of the b-wave during α -aaa treatment was due to the action of this glutamate analogue on synaptic transmission, rather than on the Müller cell itself.

This interpretation of the α -aaa results is supported by the findings of Shimazaki and Karwoski^{30, 63} in the mudpuppy. They showed that α -aaa abolished the proximal negative response (PNR), a field potential reflecting the activity of neurons in the proximal retina, at least as quickly as the drug affected the b-wave.

MECHANISM OF b-WAVE GENERATION

Both Faber¹⁹ and Miller and Dowling,⁴² in their original studies on the origin of the b-wave, suggested that current flow through Müller cells was generated by light-evoked variations in extracellular K^+ concentration ($[K^+]_o$). This theory derives originally from the work of Kuffler,³⁷ who demonstrated that glial cells are selectively permeable to K^+ and can generate large field potentials in response to changes in $[K^+]_o$. Faber¹⁹ and Miller and Dowling⁴² suggested that a light-evoked increase in $[K^+]_o$ in the distal portion of the neural retina would lead to an influx of K^+ into Müller cells in this retinal region. This K^+ influx would depolarize Müller cells and drive an equal amount of K^+ out from the more proximal regions of the cell. The return current flowing through extracellular space from the proximal retina to the distal retina would generate a vitreal-positive transretinal potential: the b-wave (see Fig 11-3).

**FIG 11-3.**

Left: CSD profile of the b-wave. Data are from a single penetration of the frog retina (eyecup preparation). The b-wave in this species is generated by current sinks in the inner and outer plexiform layers and by a large current source at the inner limiting membrane. *Right:* summary of the Müller cell/ K^+ hypothesis of b-wave generation. Light-evoked K^+ increases in the inner and outer plexiform layers generate an influx of K^+ into Müller cells in these retinal layers (open arrows). This current influx is balanced by a current efflux from the cell endfoot, which has high K^+ conductance. The return current flow through extracellular space (solid lines) generates the b-wave (M = Müller cell; OPL = outer plexiform layer; INL = inner nuclear layer). (From Newman EA: *Trends Neurosci* 1985; 8:156–159. Used by permission.)

$[K^+]_o$ Variations

Measurements of light-evoked $[K^+]_o$ made with K^+ -selective microelectrodes have lent support to this theory of b-wave generation. Oakley and Green⁵⁷ and Karwoski and Proenza³³ showed that large, sustained, light-evoked K^+ increases occurred in the inner plexiform layer of the amphibian retina at the onset and offset of a light stimulus. Additional $[K^+]_o$ measurements in skate,^{34, 35} amphibian,^{15, 16} rabbit,¹⁷ and cat²⁰ retinas demonstrated that, in addition to the sustained K^+ increase in the inner plexiform layer, a transient $[K^+]_o$ increase occurred in the outer plexiform layer at light onset. Karwoski et al.^{31a} showed that these two light-evoked K^+ increases are generated within the two plexiform layers rather than in the nuclear layers (Fig 11-5).

The presence of these two light-evoked $[K^+]_o$ in-

creases lends support to the hypothesis that the b-wave is generated by a K^+ current flow through Müller cells arising from $[K^+]_o$ variations. In the amphibian retina, in particular, there is a good correspondence between the location and time course of the two light-evoked $[K^+]_o$ increases with the location and time course of the two b-wave current sinks measured by Newman.⁴⁶

Pharmacological manipulation of the light-evoked $[K^+]_o$ increases lends further support to the Müller cell/ K^+ hypothesis of b-wave generation. γ -Aminobutyric acid (GABA) or GABA in combination with ethanol enhances the outer plexiform layer $[K^+]_o$ increase while depressing the inner plexiform layer increase.^{15–17} These drugs cause a simultaneous enhancement of the b-wave, which suggests that the outer plexiform layer increase contributes substantially more to the response than does the inner plex-

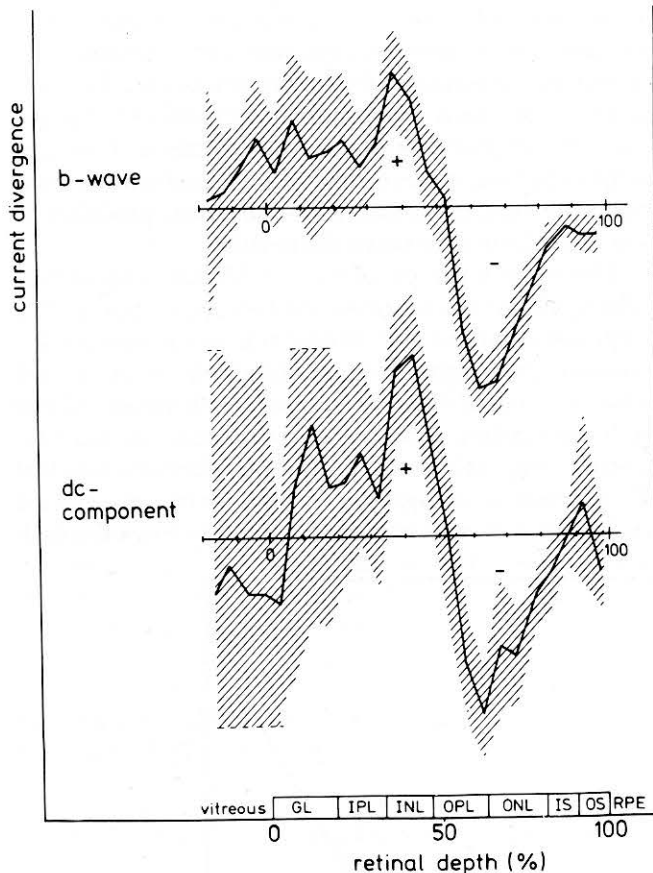


FIG 11-4.

CSD profiles of the b-wave and the dc component of the ERG recorded from the intact monkey (macaque) eye. The two ERG components have similar source/sink distributions, with a large sink near the outer nuclear layer and a distributed source extending to the vitreal surface of the retina (+, current sources; -, current sinks). Plots represent means ± 2 SEM of 26 penetrations. (From Heynen H, van Norren D: *Vision Res* 1985; 25:709-715. Used by permission.)

iform layer increase. Similarly, aspartate (soon after it is applied to the retina) enhances the b-wave while augmenting the distal $[K^+]_o$ increase and reducing the proximal $[K^+]_o$ increase.⁶³ In addition, the b-wave is reduced in magnitude when the amphibian retina is perfused with a high $[K^+]_o$ solution,⁴¹ a result to be expected if b-wave currents are proportional to the relative increase in light-evoked $[K^+]_o$.

Source of $[K^+]_o$ Changes

The source of the distal light-evoked $[K^+]_o$ increase is believed to be the depolarizing bipolar cell.¹⁷ These are the only cells in the distal retina that depolarize and thus release K^+ upon light stimulation. 2-Amino-4-phosphonobutyric acid (APB), an

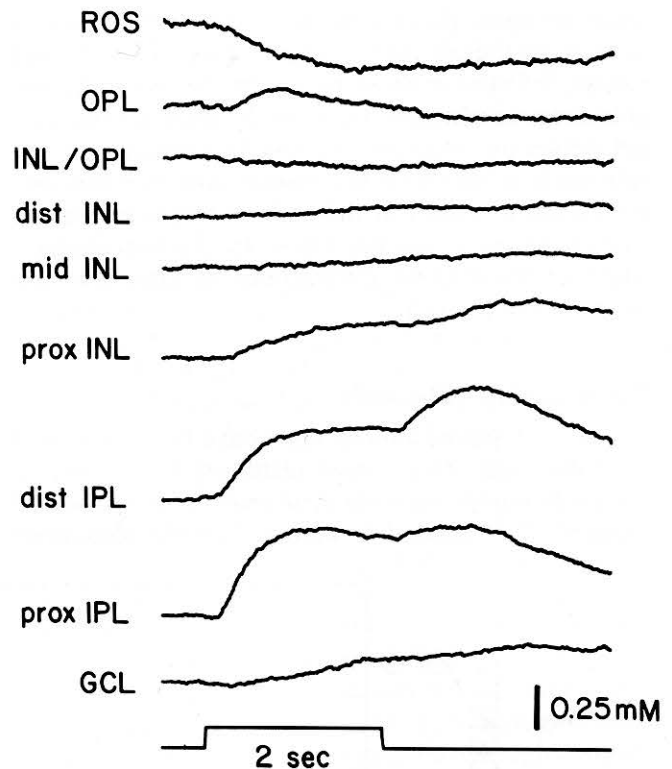


FIG 11-5.

Light-evoked variations in extracellular K^+ concentration recorded at different depths in the frog retina (retinal slice preparation). Increases in $[K^+]_o$ are maximal within the inner plexiform (IPL) and the outer plexiform (OPL) layers. The $[K^+]_o$ increase in the inner plexiform layer has both on and off components. The $[K^+]_o$ increase in the outer plexiform layer occurs only at stimulus on and is more transient. (From Karwoski CJ, Newman EA, Shimazaki H, Proenza LM: *Gen Physiol* 1985; 86:189-213. Used by permission.)

analogue of glutamate, selectively blocks the responses of on-bipolar cells⁶⁶ and abolishes the b-wave^{36, 39, 68} as well as the on-response of Müller cells.⁷⁴ The larger, more prolonged proximal $[K^+]_o$ increase in the inner plexiform layer is believed to be generated by amacrine and ganglion cells,^{17, 32, 63} although bipolar cells may also contribute to the response.⁶⁸

MÜLLER CELL MEMBRANE PROPERTIES

K^+ Conductance

The electrical properties of the membranes of Müller cells lend further support to the Müller cell/ K^+ hypothesis of b-wave generation. Müller cells are selectively permeable to K^+ .^{14, 48} Furthermore, Newman and others have demonstrated that this K^+ conductance is not distributed uniformly over the cell surface but is concentrated in conduc-

tance hot spots (Fig 11–6). In species with avascular retinas, including fish,⁵⁰ amphibians,^{9, 47, 48, 51} and rabbits,^{47, 61} the endfoot of the Müller cell adjacent to the vitreous humor has a much larger K^+ conductance than do other cell regions. In vascularized retinas such as those of the mouse and monkey, K^+ conductance is large at the endfoot but is larger still within the inner nuclear layer. In the vascularized retina of the cat, K^+ conductance is largest at the distal end of the cell.⁴⁷

Müller Cell Ion Channels

Several types of ion channels have been identified in Müller cells. The inward rectifying K^+ channel is the predominate channel type over most of the cell surface.^{9, 54, 56} Inward rectifying channels also occur

at the endfoot,⁹ but nonrectifying K^+ channels may predominate in this cell region.⁵⁶ Ca^{2+} -activated K^+ channels, fast-inactivating K^+ A channels, and Ca^{2+} channels are seen in amphibian cells as well.⁵⁴ However, these channels are normally activated only by large depolarizations (which do not occur under normal physiological conditions) and, thus, probably do not contribute to b-wave currents.

The membrane properties of Müller cells fit well with a Müller cell origin of the b-wave response. The high selectivity of the Müller cell membrane for K^+ ensures that light-evoked variations in $[K^+]_o$ will lead to a radial flow of K^+ current through Müller cells. In species with avascular retinas, the high K^+ conductance of the cell endfoot further ensures that K^+ current entering cells in regions of light-evoked $[K^+]_o$ increase will exit predominately from the end-

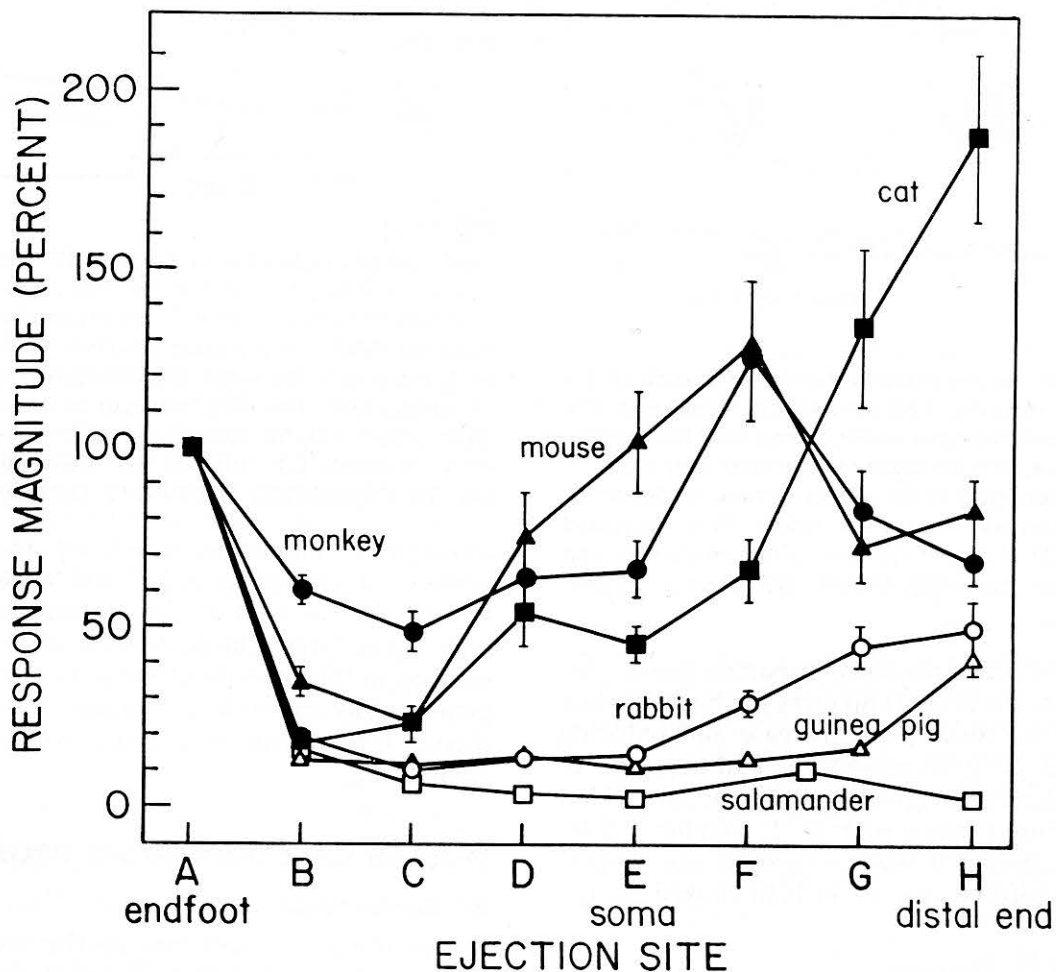


FIG 11–6.

Distribution of K^+ conductance over the surface of enzymatically dissociated Müller cells. The magnitude of cell depolarizations in response to focal K^+ ejections are plotted as a function of ejection location along the cell surface. Responses are normalized to response magnitude at the endfoot. In species with avascular retinas (salamander, rabbit, guinea pig), K^+ conductance is largest at the endfoot. In vascularized species, conductance is largest near the cell soma (mouse, monkey) or at the distal end of the cell (cat). (From Newman EA: *J Neurosci* 1987; 7:2423–2432. Used by permission.)

foot. In these species, a $[K^+]_o$ increase in the outer plexiform layer establishes a current loop that extends all the way to the vitreal surface of the retina (see Fig 11–3). If K^+ conductance were distributed uniformly throughout the Müller cell, the current loop would be shorter, and a smaller transretinal voltage would result. Thus, the distribution of K^+ conductance in Müller cells is critical to the generation of the b-wave.

A quantitative model of b-wave generation in the frog retina was constructed by Newman and Odette⁵⁵ to test the Müller cell/ K^+ hypothesis of b-wave generation. Their computer simulations demonstrated that light-evoked increases in $[K^+]_o$ in the inner and outer plexiform layers could indeed lead to the generation of a transretinal b-wave potential. Their work also showed that the time course of the $[K^+]_o$ increases and the intracellular Müller cell response do not necessarily have to match that of the resulting b-wave response (see Fig 11–2).

SUMMARY OF MÜLLER CELL/ K^+ HYPOTHESIS

The Müller cell/ K^+ hypothesis of b-wave generation holds that light-evoked increases in $[K^+]_o$ in the inner and outer plexiform layers result in an influx of K^+ into Müller cells, which in turn results in cell depolarization and the establishment of a radial current flow through the cells. The return current flow through the extracellular space generates a transretinal voltage that is recorded as the b-wave. The distal (outer plexiform layer) $[K^+]_o$ increase (generated by depolarizing bipolar cells) is thought to predominate over the proximal $[K^+]_o$ increase in generating the response. Thus, the b-wave may be thought of as a glial (Müller cell) response reflecting the activity of depolarizing bipolar cells.

Intracellular Müller cell recordings, CSD analysis, and measurements of light-evoked $[K^+]_o$ within the retina all support this hypothesis. The membrane properties of Müller cells are consistent with such a theory, and the distribution of K^+ conductance across the Müller cell surface determines, in large part, the distribution and magnitude of the currents that are generated.

CHALLENGES TO THE MÜLLER CELL/ K^+ HYPOTHESIS

The Müller cell/ K^+ hypothesis of b-wave generation is not without its problems. Perhaps the most significant challenge to the theory concerns the ef-

fects of Ba^{2+} on the generation of the b-wave. Ba^{2+} effectively blocks K^+ channels in Müller cells⁴⁹ and therefore should abolish ERG components generated by K^+ current flow through Müller cells. Indeed, Ba^{2+} effectively blocks the slow PIII response^{4, 24, 40, 72} and the M-wave,^{30, 31} both thought to be generated by Müller cells in response to changes in $[K^+]_o$. However, Ba^{2+} is less effective in blocking the b-wave^{13, 24, 28, 72} and, in the cat, can sometimes enhance it.²¹

The failure of Ba^{2+} to abolish the b-wave does not rule out the Müller cell hypothesis, however. Although Ba^{2+} does not abolish the response, it can reduce the b-wave amplitude substantially.^{13, 24} In addition, Coleman et al.¹³ have observed that Ba^{2+} reverses the normal $[K^+]_o$ standing gradient within the retina. They have suggested that this reversal of the gradient may explain why Ba^{2+} blocks the slow PIII response more effectively than it does the b-wave. They propose that the inward rectifying K^+ channels of Müller cells would be biased by the $[K^+]_o$ gradient in such a way that they would have a low conductance to a decrease in $[K^+]_o$ (which generates the slow PIII response) but would have a high conductance to an increase in $[K^+]_o$ (which may generate the b-wave). In addition, a Ba^{2+} -induced reduction in the resting $[K^+]_o$ in the distal retina would diminish the light-evoked $[K^+]_o$ decrease (slow PIII) while accentuating the light-evoked distal $[K^+]_o$ increase (b-wave).

Another persistent criticism of the Müller cell/ K^+ hypothesis of b-wave generation concerns the magnitude of the distal $[K^+]_o$ increase. Although depth recordings and CSD analysis indicate that the distal $[K^+]_o$ increase contributes substantially more to the generation of the b-wave than does the proximal $[K^+]_o$ increase, ion-selective microelectrode measurements indicate that the distal increase is much smaller than is the proximal increase. A recent quantitative test of the Müller cell/ K^+ hypothesis⁵⁸ indicated that the experimentally measured distal K^+ increase was only ~30% the magnitude predicted by the hypothesis.

Once again, however, this criticism does not rule out the Müller cell/ K^+ hypothesis. It is possible that ion-selective microelectrode measurements of the distal $[K^+]_o$ increase have underestimated its true magnitude. (The distal increase is more transient and is generated in a much narrower tissue layer than is the proximal increase. Consequently, it is more difficult to detect.) In addition, the currents generated by the distal $[K^+]_o$ increase could be substantially larger than presently believed if Müller cell K^+ conductance in the outer plexiform layer were larger than presently estimated.

In summary, although substantive criticisms of the Müller cell/ K^+ hypothesis of b-wave generation remain, they do not condemn the theory.

ALTERNATE THEORIES OF b-WAVE GENERATION

Recent work has suggested that Müller cells may generate the b-wave, not in response to changes in $[K^+]_o$, but rather in response to light-evoked changes in the extracellular concentrations of other substances. Müller cells possess an electrogenic Na^+ /glutamate transport system^{8, 25} that can generate substantial transmembrane currents in response to changes in extracellular glutamate, a likely transmitter of photoreceptors and bipolar cells. Brew and Attwell⁸ have suggested a specific model whereby variations in light-evoked glutamate generate the b-wave. Their model cannot account for the generation of the greater part of the b-wave, however, because it leads to a vitreal negative rather than vitreal positive potential. It is likely, however, that the Müller cell glutamate transport system contributes, in some part, to b-wave generation.

Müller cells also possess $GABA_A$ receptors and respond to the application of GABA with an increase in Cl^- conductance.³⁸ It is possible that light-evoked variations in GABA, a likely transmitter of horizontal and amacrine cells, generates in Müller cells Cl^- currents that contribute to b-wave generation. Pharmacological studies support this hypothesis. Ethanol, which can potentiate $GABA_A$ receptor responses, GABA itself, or ethanol in combination with GABA, enhanced the b-wave in amphibians,¹⁶ rabbits,¹⁷ and cats.⁴⁴ In addition, picrotoxin, a $GABA_A$ antagonist, reduced the b-wave substantially in cats (Frishman and Steinberg, unpublished observations). These results only suggest a role of Müller cell GABA receptors in b-wave generation, of course, because GABA agonists and antagonists may act on neuronal GABA receptors rather than on those in Müller cells.

Müller cells also possess an electrogenic Na^+ / HCO_3^- cotransport system that generates substantial currents in response to changes in external HCO_3^- concentration (Newman and Astion, unpublished observations). Light-evoked changes in pH within the retina, recently observed by Borgula et al.,⁷ may activate this system and contribute to the generation of the b-wave.

At present, little is known about variations in the extracellular concentrations of glutamate and GABA

with light stimulation. Even less is known about how Müller cells respond in vivo to changes in these substances and to changes in pH. Thus, it is impossible at this time to determine how important Müller cell responses to neurotransmitters and to pH are in b-wave generation. One or more of these processes may turn out to be instrumental in generating the b-wave.

Early studies of the b-wave implicated bipolar cells in the generation of this response. It is possible, indeed quite likely, that currents generated by bipolar cells contribute to b-wave generation to some degree.⁷³ However, it is unlikely that bipolar cells contribute substantially to the response since bipolar cell responses cannot account for the b-wave current source that extends to the inner limiting membrane within the retina. It is likely that the b-wave of the ERG is generated principally by Müller cells.

dc COMPONENT OF THE b-WAVE

The b-wave is only one of the two positive components of the ERG that Granit²³ called PII. The other component, which has been studied only in mammals, is a steady potential termed the dc component.^{11, 12} The dc component continues at reduced amplitude in the ERG after the transient response (the b-wave) and then decays at stimulus offset. This component emerges in the dark-adapted ERG at lower intensities than do b- or c-waves.¹¹ As shown in a series of ERG records from the intact cat eye (Fig 11-7), the dc component of PII has the lowest threshold of the positive components of the ERG. Its threshold is about 1.5 log units higher than the threshold of the negative, scotopic threshold response (STR). At higher intensities, PII dominates over the STR, and above $7.1 \log q \text{ deg}^{-2} \text{ s}^{-1}$ it clearly peaked to form the b-wave. The dc component had a similar depth distribution to the b-wave in cats^{12, 64, 65} and monkeys,²⁷ and in a CSD analysis performed in monkeys it had a similar source-sink distribution to the b-wave (see Fig 11-4).²⁷ However, experiments in cats indicated that the dc component may not have exactly the same neuronal origin as the b-wave since (1) the dc component was unaffected by low doses of intravitreal xylocaine whereas the b-wave was removed,^{12, 67} (2) the dc component summed over a smaller area than the b-wave did,⁶⁷ and (3) the dc component was relatively less suppressed by light-adaptation than was the b-wave.⁶⁷ In fact, in the monkey, the dc component was more easily detected in the light-adapted

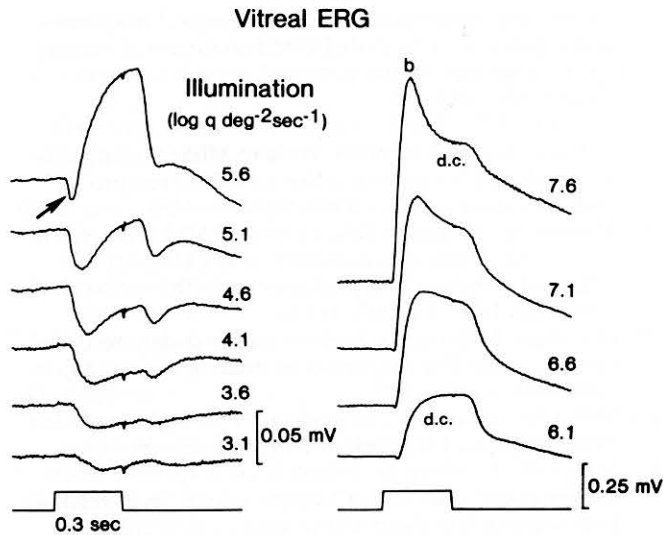


FIG 11-7.

Dark-adapted ERG recorded from an intact cat eye with an electrode in the vitreous humor. Responses are to diffuse stimuli of increasing illumination. The b-wave (*b*) is indicated at 7.6 log q deg⁻²s⁻¹. The dc component (*d.c.*) of the PII-dominated ERG is seen clearly in records 6.1, 6.6, and 7.1. An arrow marks where the STR (truncated by the dc component) resembles an a-wave. (From Sieving PA, Frishman LJ, Steinberg RH: *J Neurophysiol* 1986; 56:1049–1061. Used by permission.)

retina, and it was under light-adapted conditions that depth and CSD analyses were performed.^{26, 27}

ROD VS. CONE b-WAVE

The b-wave can be generated by either the rod or the cone systems. The cone b-wave (sometimes termed b1) is generally a small response of short latency, whereas the rod b-wave (b2), at its maximum, is a relatively large response of longer latency. In cat^{64, 65} and monkey²⁶ retinas, the depth profile of the rod b-wave measured under scotopic conditions and the cone b-wave measured under photopic conditions were very similar, thus suggesting a similar locus of origin for the two responses. Under conditions that stimulate both rods and cones, it is possible to simultaneously record both b-waves. In fact, the photopic b-wave was first identified by Motokawa and Mita⁴³ in the human ERG, where it was observed as a sharp positive peak that occurred prior to the more prolonged major portion of the b-wave in response to intense flashes from darkness. They called this response the x-wave. It could be distinguished from the rod response by its sensitivity to long-wavelength stimuli.^{1, 2, 43} Curiously, in an intraretinal analysis of the ERG in the mangabey

monkey, which has a prominent x-wave,²⁹ Ogden⁵⁹ did not detect a negative potential that matched the time course of the x-wave in the midretina, where the b-wave was maximal.

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