# Principles and Practice of Clinical Electrophysiology of Vision


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# 10

# Proximal PIII

Chester Karwoski

The corneal-negative component of the electroretinogram (ERG) can be divided into three subcomponents: distal, slow, and proximal. Distal PIII is directly generated by the responses of rods and cones. It is also called the late receptor potential, and it is probably the major contributor to the ERG a-wave (see Chapter 7). Slow PIII is probably generated by Müller cells in response to the light-evoked decrease in $[K^+]_o$ that is produced by the photoreceptors in the distal retina, and it contributes to the ERG c-wave (see Chapter 9). Slow PIII has sometimes been considered a "proximal PIII" since Müller cells are located proximal to the photoreceptors. However, I will reserve the term *proximal PIII* for certain other corneal-negative ERG components that are generated by cells located proximal to the photoreceptors. Proximal PIII has not been widely investigated, but its existence would have potentially great importance to electroretinography.

Proximal PIII was named and described by Murakami and Kaneko[6] in studies on the intraretinal ERG of frogs and turtles where it was observed in the proximal retina as a rapid response preceding the b-wave. Proximal PIII was distinguished from distal PIII in that proximal PIII had a different depth distribution, greater sensitivity to drugs, and longer latency. Proximal PIII is distinguished from slow PIII in that proximal PIII has a faster rise time than the b-wave[6] (1966) whereas the rise time of slow PIII is slower than the b-wave (see Chapter 9). Also, aspartate blocks proximal PIII but not slow PIII (fish,[8] rabbit[5]). Proximal PIII is very small, if present, in cats[4] and primates,[7] and additional study is needed to resolve whether it can be identified in these species as well as in humans.

The cellular origin of proximal PIII is unknown. One reason is that proximal PIII has not been recorded in isolation from other proximal field potentials; thus its waveform cannot be compared with intracellular response types. The $a_2$ component of the vitreal a-wave is found in many vertebrates, and it may be considered a proximal PIII. Fatechand[3] has argued that $a_2$ is generated by the same cells as the proximal negative response (PNR). Hanitzsch[4, 5] described a proximal PIII ("fast peak of PIII") in rabbit retina, and she suggests amacrine or horizontal cells as its origin. In fish, a proximal PIII is particularly well developed, and it may be associated with horizontal cell activity.[2] These authors argue that proximal PIII arises via spatial buffer currents in Müller cells in response to $K^+$ movements across horizontal cell membranes, but the response is quite rapid and a *direct* contribution from horizontal cells should not be excluded. It is unclear whether a response generated by the former mechanism should be considered part of slow PIII or proximal PIII. Evidence for a contribution of horizontal cell $K^+$ to slow PIII has also been reported by Dick.[1] In sum, proximal PIII might be generated by any neuron in the inner nuclear layer, or by Müller cells in response to $K^+$ changes initiated by these cells.

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