Supplemental Figure 1 (movie). A 3-dimensional reconstruction of a vibratome section (65 µm) of the retina. Horizontal cells and one type each of amacrine and ON cone bipolar cells are stained with anti-calbindin (red). Ribbons are stained with anti-RIBEYE (green). Examination of RIBEYE puncta during rotation reveals which puncta are associated with the calbindin-stained ON cone bipolar axons.

Supplemental Figure 2. Synaptic markers are colocalized at the sites in sublamina a where axons of calbindin-positive bipolar cells contact ipRGCs and DACs. (a) A spine from an ipRGC (blue) stained with the ipRGC antibody contacts a calbindin-positive bipolar cell axon (green). (b) Magnification of the image in (a) shows that a GluR4 puncta (red; arrow) is apposed to the axon of the bipolar cell (green). This axon makes a short horizontal turn within stratum 1 before it continues its descent to sublamina b. (c) The ipRGC specialization (blue) contacts the axon of the bipolar cell and contains a GluR4 puncta (red; arrow) at that site. (d) GluR4 puncta (red; arrowheads) are also contained at the conjunctions of calbindin-positive bipolar cell axons (green) and DAC processes (blue). Scale bars (a) 2 µm; (b-d) 0.5 µm. All panels are single 0.3 µm optical sections.

Supplemental Figure 3. The extensive arbor of the bistratified diving ganglion cells in sublamina a apparently contributes no excitatory drive to this cell, as all spiking is abolished in the presence of 100 µM L-APB, an agonist of the mGluR6 glutamate receptor exclusively located on ON cone bipolar cells. The stimulus was a spot of light covering the entire dendritic field of the cell.