Figure 1.1
Lamination of the retina and formation of the foveal depression.
(A) Cross section of retinal layers corresponding to (B), which is a representation of the neuronal components in the retina. Vertical processing of information from photoreceptors to bipolar cells and ganglion cells provides the most direct route for sending visual information to the brain. Horizontal cells and amacrine cells mediate lateral interactions between adjacent groups of photoreceptors, bipolar cells and ganglion cells, enabling neighbouring parts of the retina to compare light intensity from different regions of the visual field. (C) Bipolar and ganglion cells are laterally displaced from the inner retinal layers to form the fovea centralis, allowing light to interact directly with cone photoreceptors in the outer retinal layers. (Adapted from Pritchard and Alloway, 1999)
Figure 1.2
Electron micrograph of retinal pigmented epithelial (RPE) cells with electron dense melanin granules. Bruch’s membrane (BM) is seen between the RPE and the choriocapillaris. Endothelial cell fenestrations (arrows) are seen facing the basal aspect of RPE. An intravascular leucocyte (*) is visible. (Courtesy of Diana van Driel)
Figure 1.3
Fundus photograph. Severe hemorrhages (asterisk) and cotton wool spots (arrows) that may accompany macular oedema. (Courtesy of Mark Gillies)

Figure 1.4
Fundus photograph. Hard exudates (arrow) representing lipid exuding from damaged macular vessels.

Figure 1.5
Fundus photograph. Persistent cystoid macular oedema (CMO) (asterisk) despite heavy laser treatment. CMO often has a "petaloid" pattern like the petals of a flower; this is consistent with swelling occurring in the parafoveal region rather than in the foveola.
Figure 1.6
The location of the perifoveal blood vessels and the plexiform layers (intraretinal diffusion barriers) determines the sites where cystic spaces are formed in cystoid macular oedema (CMO). (A) The inner retina of the macula contains three vascular plexuses: within the GCL, and the superficial and deep vascular plexuses of the INL (left side of A). The right side of A displays the typical ‘Z-shaped’ morphology of Müller cells in the macula. (B) When the RPE becomes leaky, serum proteins (which bind water) accumulate in the outer retina up to the level of the OPL, causing a thickening of tissue and the development of cystic splitting of the Fibres of Henle. Leakage of the inner retinal blood vessels results in cyst formation in the INL, as the two plexiform layers (IPL and OPL) act as diffusion barriers for extruded serum proteins and for the movement of water. NFL, nerve fibre layer; GCL, ganglion cell layer; IPL, inner plexiform layer; INL, inner nuclear layer; OPL, outer plexiform layer; ONL, outer nuclear layer; PRS, photoreceptor segments. (Adapted from Bringmann et al, 2004)
Figure 1.7

Proposed mechanism of K⁺-induced Müller cell swelling due to redistribution of Kir4.1-K⁺ channel protein (refer to text) during transient ischemia-reperfusion of the rat retina. (A) In control retinal slices, Kir4.1 immunoreactivity is predominantly expressed by Müller cell end-foot membranes which abut on retinal blood vessels (arrow) and the vitreous body (arrowhead). A similar expression pattern was observed for the aquaporin-4 (AQP-4) water channel protein. (B) In a retina two days after transient ischemia, the Kir4.1 protein is redistributed from normal expression sites around vessels and at the inner limiting membrane, while the expression of AQP-4 is largely unaltered. This closes the pathway which normally extrudes K⁺ ions out of the retina into the blood; K⁺ accumulates in the Müller cell bodies and causes an osmotic gradient at the gliovascular interface which drives water into Müller cells, via the aquaporins. (Adapted from Bringmann et al, 2004)
Figure 1.8
Optical coherence tomography (OCT) showing (A) cystoid macular oedema, and (B) resolution of macular oedema after laser therapy. Fluid has distorted the macula in A compared with B in which the foveal depression is obvious in the centre of the image. (Courtesy of Mark Gillies)
Figure 1.9

Representation of the molecular complexes localised to interendothelial junctions. The general organisation of these complexes includes transmembrane proteins acting as receptors and cytoplasmic proteins transducing signals inside the cells, and often modulating activity of the transmembrane component. Some cytoskeletal elements are shown. **Tight junctions** are comprised of the transmembrane protein occludin, claudins and a group of cytoplasmic proteins (ZO-1, ZO-2, cingulin and 7H6) as well as actin microfilaments. **Adherens junctions** are formed by VE-cadherin dimers which are connected inside the cell to a cluster of catenins (α and β catenins, plakoglobin and p120) and actin microfilaments. PECAM occurs at endothelial cell-cell contacts where it expresses homophilic binding properties. The **complexus adherens** comprises VE-cadherin as the transmembrane component and possibly plakoglobin, desmoplakin and vimentin inside the cell. (Adapted from Lampugnani and Dejana, 1997)
Figure 1.10
Diagram indicating barrier and fence functions of tight junctions. In its barrier function, tight junctions inhibit the passage of ions and small solutes between cells, whereas in its fence function, it prevents intermixing of mobile molecules in apical and basolateral membrane domains. Both occludin and claudins have been shown to be directly involved in barrier and fence functions of tight junctions. In the paracellular pathway, tight junctions form the sole resistor ($R_p$) compared with the transcellular route, in which the current is impeded by apical ($R_{1T}$) and basolateral ($R_{2T}$) plasma membranes that form two resistors in parallel. (Adapted from Schneeberger and Lynch, 1992)