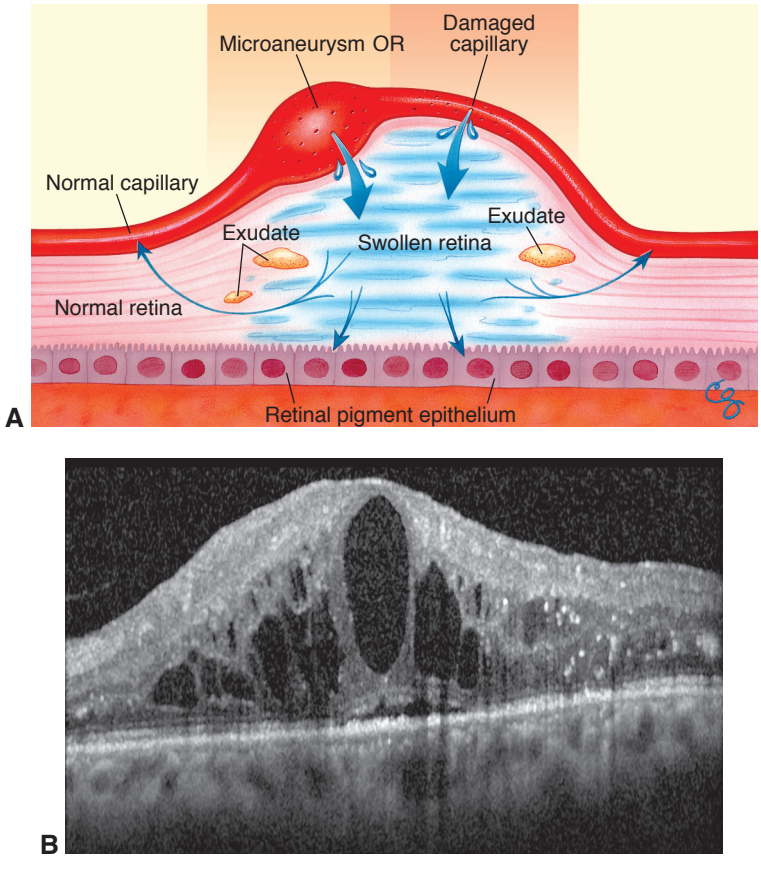

Figure 5-9 Diabetic macular edema (DME). **A,** Artist's rendering of the mechanism of DME, demonstrating development of retinal thickening from a breakdown of the blood–retina barrier. **B,** Spectral-domain OCT (SD-OCT) scan of DME. Whereas there are extensive cystic changes in the outer plexiform and outer nuclear layers, the external limiting membrane line appears intact across the extent of the scan, with the exception of shadowing artifacts from more superficial hyperreflective lesions. Note the foveal detachment. *(Part A from Ginsburg LH, Aiello LM. Diabetic retinopathy: classification, progression, and management.* Focal Points: Clinical Modules for Ophthalmologists. *San Francisco: American Academy of Ophthalmology; 1993, module 7. Illustration by Christine Gralapp. Part B courtesy of Colin A. McCannel, MD.)*

FA is useful in demonstrating the breakdown of the blood–retina barrier by showing local areas of retinal capillary leakage. However, leakage shown on the angiogram may occur in the absence of macular retinal thickening and is thus not considered macular edema. Examination with OCT or slit-lamp biomicroscopy are the most appropriate methods to evaluate eyes for the presence or absence of macular thickening.

Classification of Diabetic Macular Edema

Current algorithms for pharmacologic intervention in DME use a simple, OCT-based definition to classify DME as *center-involved* or *non–center-involved*. In *center-involved DME,* the central retinal subfield appears thickened on OCT scans. DME that does not affect the central subfield is termed *non–center-involved* (Fig 5–10; Activity 5-1).

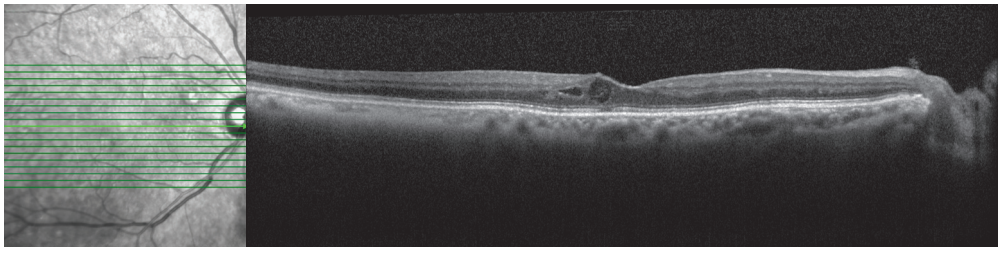

Figure 5-10 OCT volume scan of an eye with DME from a 75-year-old Hispanic male patient with a long history of poorly controlled type 2 diabetes mellitus (A_{1C} levels typically in the 9–9.6 range). The right eye is affected with moderate NPDR. The patient has undergone previous treatment with bevacizumab and aflibercept, but there is persistent center-involved DME. The cystic changes involving the temporal and inferior foveal region are most noticeable in slices 10 through 7 in Activity 5-1. *(Courtesy of Colin A. McCannel, MD.)*

ACTIVITY 5-1 OCT Activity: OCT of diabetic macular edema.
Courtesy of Colin A. McCannel, MD.
Access all Section 12 activities at www.aao.org/bcscactivity_section12.

The ETDRS was the first prospective, randomized clinical trial of photocoagulation in diabetic patients with less than high-risk PDR in order to establish standard treatment paradigms for managing DME (see Clinical Trial 5-3). It defined *clinically significant diabetic macular edema (CSME)* as the indication for focal laser photocoagulation treatment in the following settings:

- retinal thickening located at or within 500 μm of the center of the macula
- hard exudates at or within 500 μm of the center if associated with thickening of adjacent retina
- a zone of thickening larger than 1 disc area, if located within 1 disc diameter of the center of the macula

CSME is an older term that predates diagnoses made with OCT technology. Now that anti-VEGF treatment has supplanted macular laser photocoagulation as the first-line therapy for DME, the CSME diagnosis, which is made clinically, is much less frequently used.

Regardless of whether it is center-involved or non–center-involved, DME may manifest as focal or diffuse retinal thickening. *Focal macular edema* is characterized by areas of local fluorescein leakage from specific capillary lesions, such as microaneurysms (Fig 5-11A). *Diffuse macular edema* is characterized by extensive retinal capillary leakage and widespread breakdown of the blood–retina barrier, often accumulating in a cystoid configuration in the perifoveal macula (cystoid macular edema) (Fig 5-11B). Studies have not demonstrated any difference in treatment response corresponding to the pattern of macular edema, whether focal, diffuse, or a combination of these.

Treatment of Diabetic Macular Edema

In parallel with medical management and optimizing the health habits of the patient, ocular therapies should be considered to maximize visual function and prevent progressive vision loss. These therapies include ocular pharmacologic management and laser photocoagulation treatment. Treatment is typically indicated when the macular edema is center-involved and affects visual acuity. For patients with DME who are asymptomatic