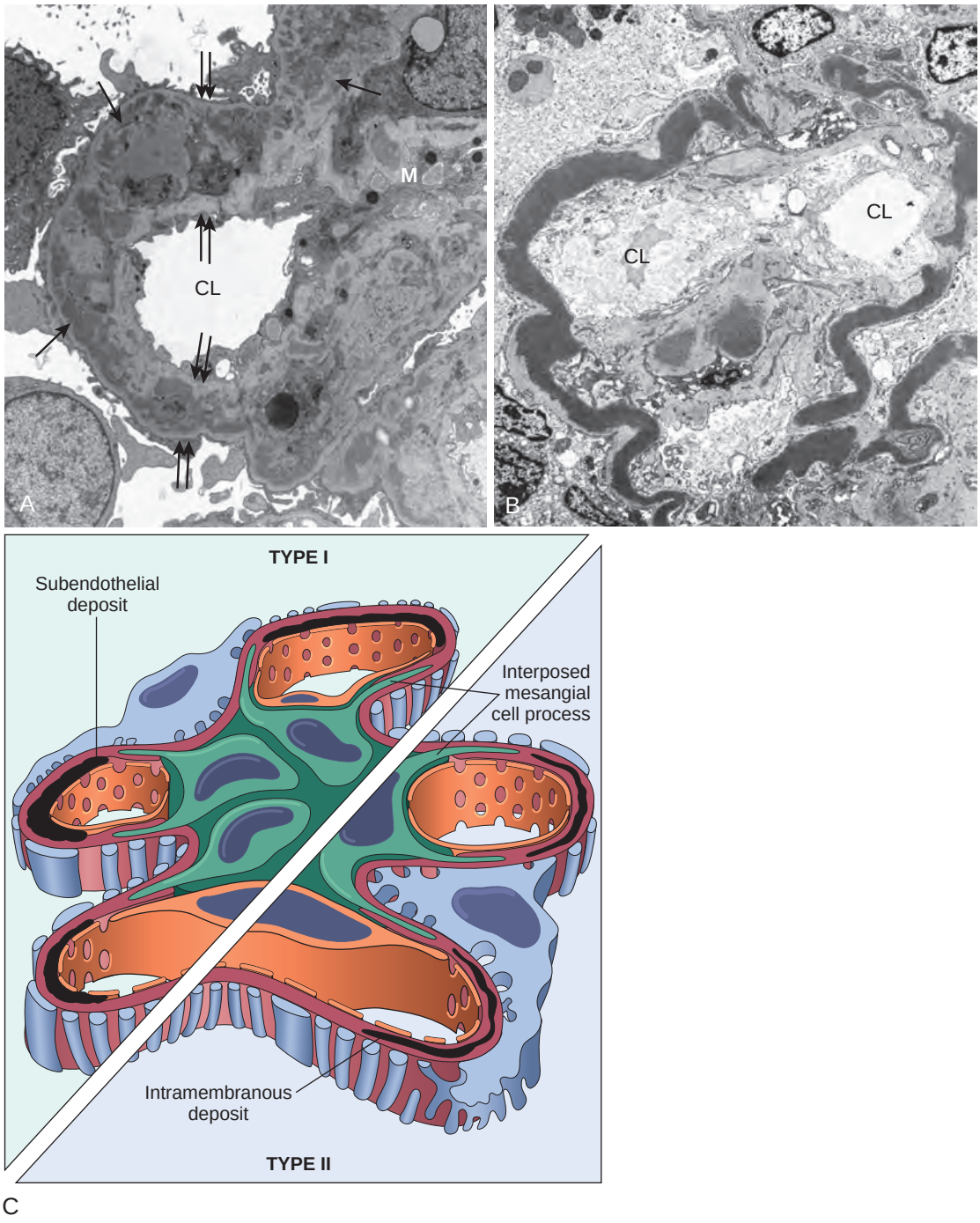

Figure 20-17 **A,** Membranoproliferative glomerulonephritis, type I. Note discrete electron-dense deposits *(arrows)* incorporated into the glomerular capillary wall between duplicated (split) basement membranes *(double arrows)*, and in mesangial regions (M); CL, Capillary lumen. **B,** Dense-deposit disease (type II membranoproliferative glomerulonephritis). There are dense homogeneous deposits within the basement membrane. CL, Capillary lumen. In both, mesangial interposition gives the appearance of split basement membranes when viewed in the light microscope. **C,** Schematic representation of patterns in the two types of membranoproliferative glomerulonephritis. In type I there are subendothelial deposits; type II is characterized by intramembranous dense deposits (dense-deposit disease). In both, the basement membranes appear split when viewed in the light microscope. (**A,** Courtesy Dr. Jolanta Kowalewska, Cedars-Sinai Medical Center, Los Angeles, Calif.)

Clinical Features. Most patients with primary MPGN present in adolescence or as young adults with nephrotic syndrome and a nephritic component manifested by hematuria or, more insidiously, as mild proteinuria. Few remissions occur spontaneously in either type, and the disease follows a slowly progressive but unremitting course. Some patients develop numerous crescents and a clinical picture of RPGN. About 50% develop chronic renal failure within 10 years. Treatments with steroids, immunosuppressive agents, and antiplatelet drugs have not proven to be of any benefit.