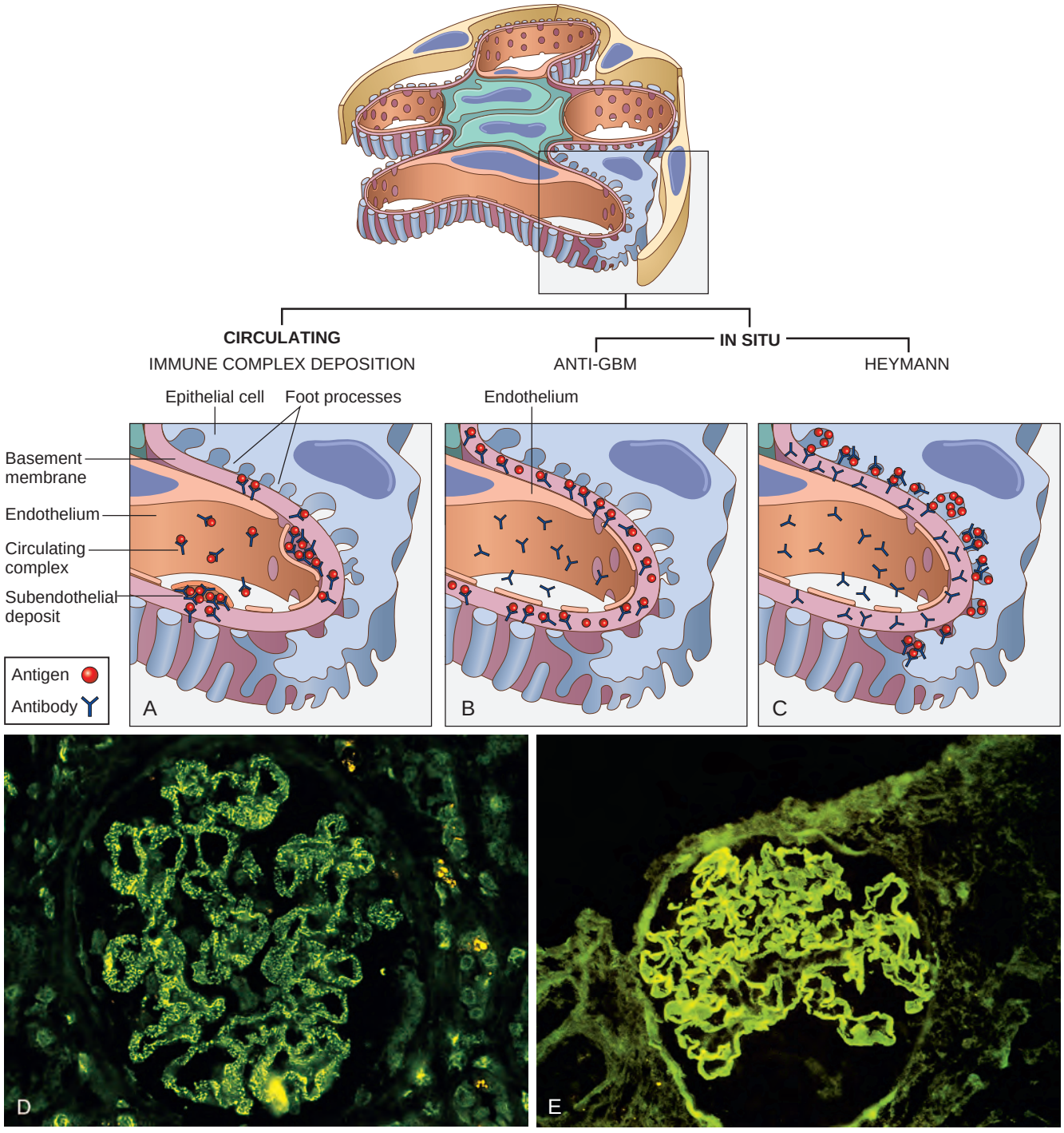


**Figure 20-4** Antibody-mediated glomerular injury can result either from the deposition of circulating immune complexes **(A)** or, more commonly, from in situ formation of complexes exemplified by anti-GBM disease **(B)** or Heymann nephritis **(C). D** and **E,** Two patterns of deposition of immune complexes as seen by immunofluorescence microscopy: granular, characteristic of circulating and in situ immune complex nephritis **(D),** and linear, characteristic of classic anti-GBM disease **(E).**

tissue components. What triggers these autoantibodies is unclear. Secondary forms of membranous nephropathy can be experimentally induced by drugs (e.g., mercuric chloride), and graft-versus-host disease (Chapter 6). In some of these situations there may be uncontrolled B-cell activation, leading to the production of autoantibodies that react with renal antigens.

### Antibodies Against Planted Antigens

**Antibodies can react in situ with antigens that are not normally present in the glomerulus but are "planted" there**. Such antigens may localize in the kidney by interacting with various intrinsic components of the glomerulus. Planted antigens include cationic molecules that bind to anionic components of the glomerulus; DNA,

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