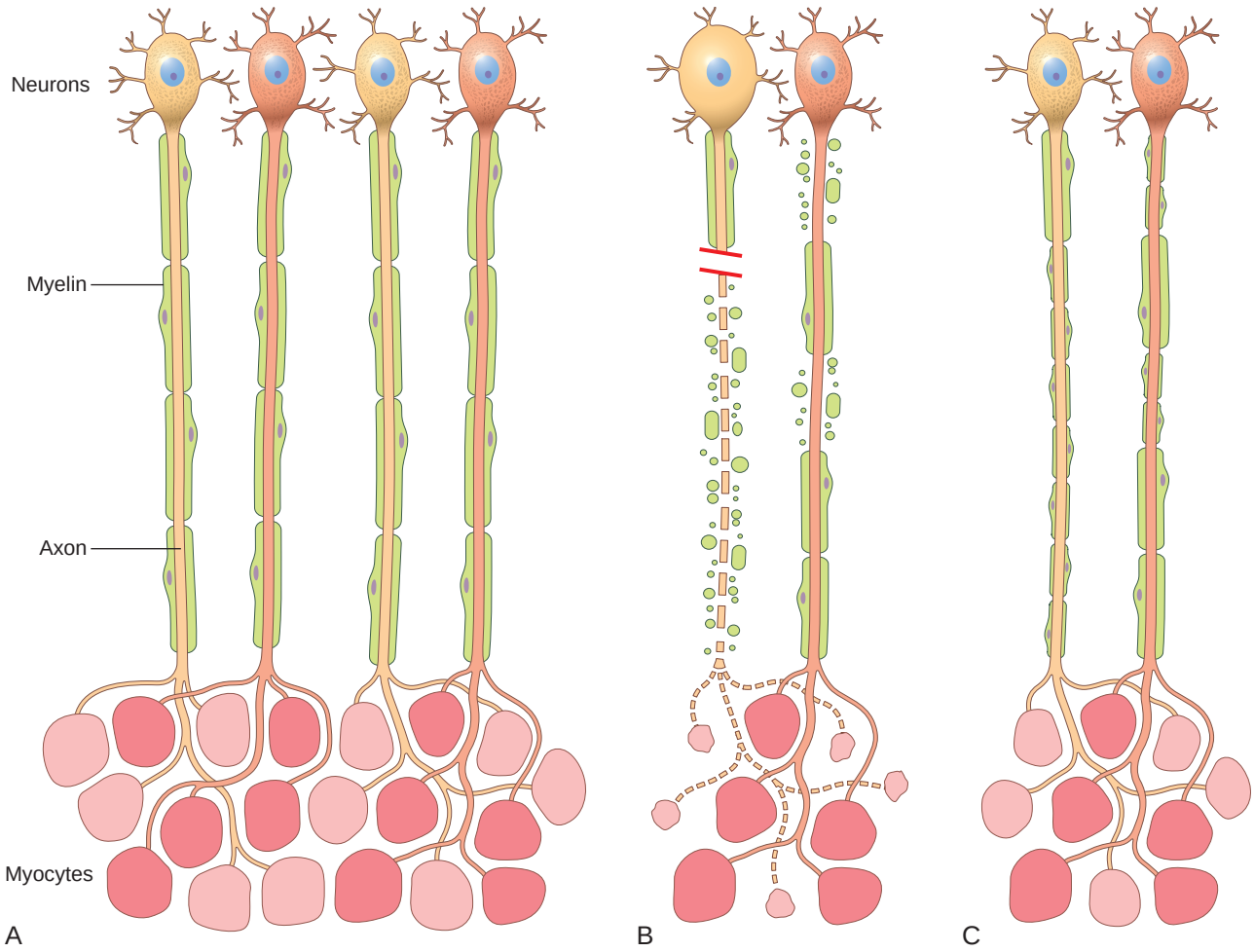

Figure 27-2 Patterns of peripheral nerve damage. **A,** In normal motor units, type I and type II myofibers are arranged in a "checkerboard" distribution, and the internodes along the motor axons are uniform in thickness and length. **B,** Acute axonal injury (*left axon*) results in degeneration of the distal axon and its associated myelin sheath, with atrophy of denervated myofibers. In contrast, acute demyelinating disease (*right axon*) produces random segmental degeneration of individual myelin internodes, while sparing the axons. **C,** Regeneration of axons after injury (*left axon*) allows reinnervation of myofibers. The regenerated axon is myelinated by proliferating Schwann cells, but the new internodes are shorter and the myelin sheaths are thinner than the original ones. Remission of demyelinating disease (*right axon*) allows remyelination to take place, but the new internodes also are shorter and have thinner myelin sheaths than flanking normal undamaged internodes. See Table 27-1 and Fig. 27-7 for comparison.

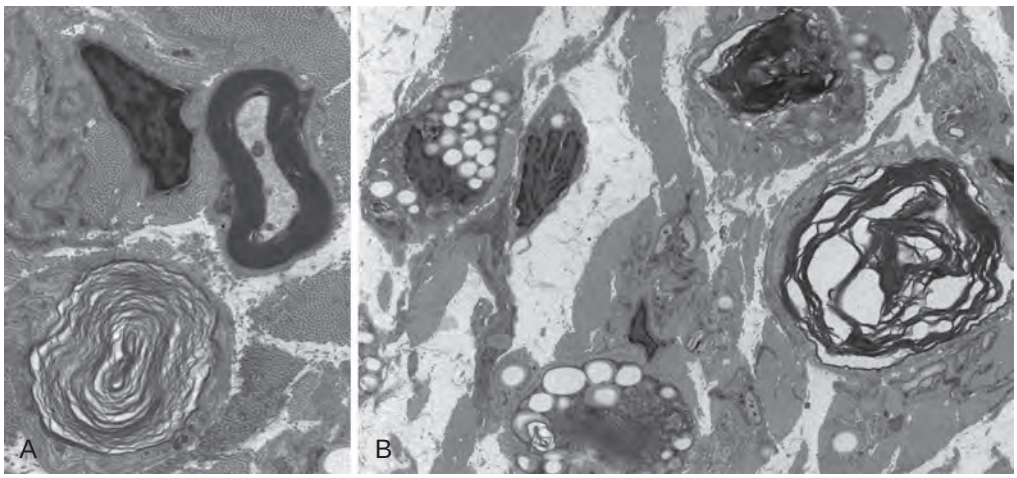

Figure 27-3 Electron micrographs illustrating features of axonal degeneration. **A,** Degenerating myelin with loosened myelin layers is seen in the degenerating axon in the lower left corner, to be contrasted with a normal myelin sheath with tightly packed myelin and intact axon in the upper right corner. **B,** In addition to an unraveling myelin sheath, several cells contain lipid droplets (seen as vacuoles) derived from degenerating myelin.