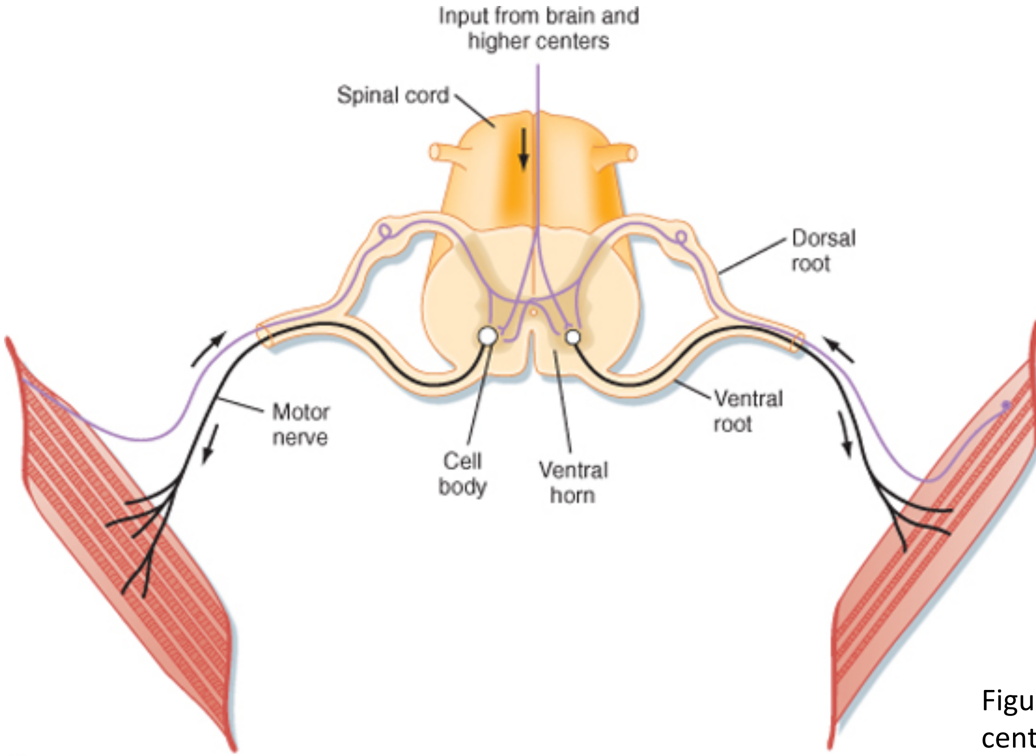
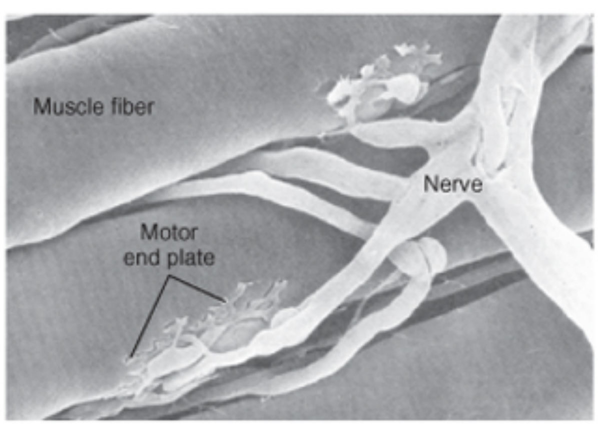
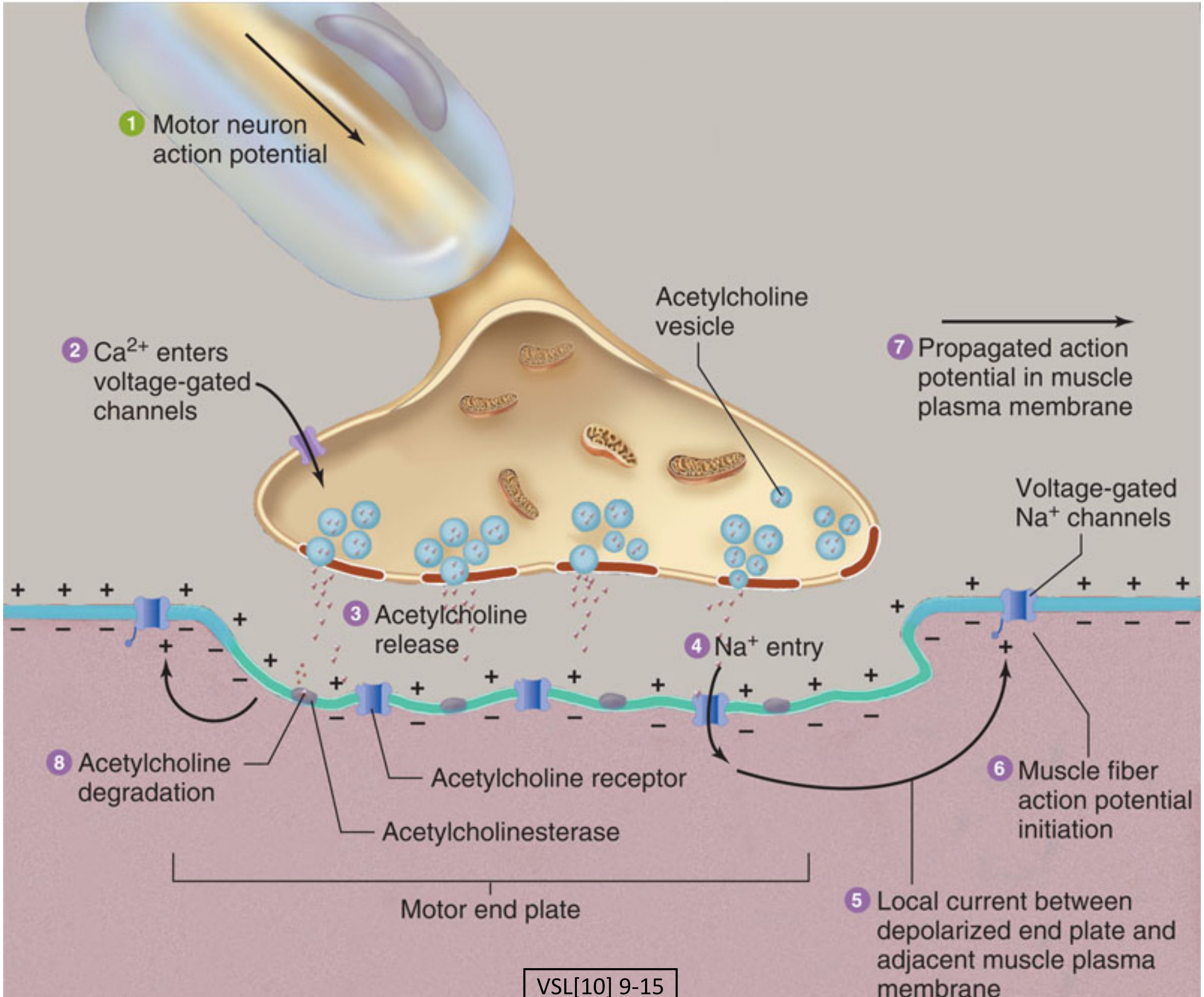




Koeppen & Stanton: Berne and Levy Physiology, 6th Edition.

Figure 12-7 Skeletal muscle is a voluntary muscle controlled by the central nervous system, with efferent signals (i.e., action potentials) passing through an α motor neuron to muscle fibers. Each motor neuron may innervate many muscle fibers within a muscle, although each muscle fiber is innervated by only one motor neuron (A). B, Scanning electron micrograph showing innervation of several muscle fibers by a single motor neuron. (B, From Bloom W, Fawcett DW: A Textbook of Physiology, 12th ed. New York, Chapman & Hall, 1994.)



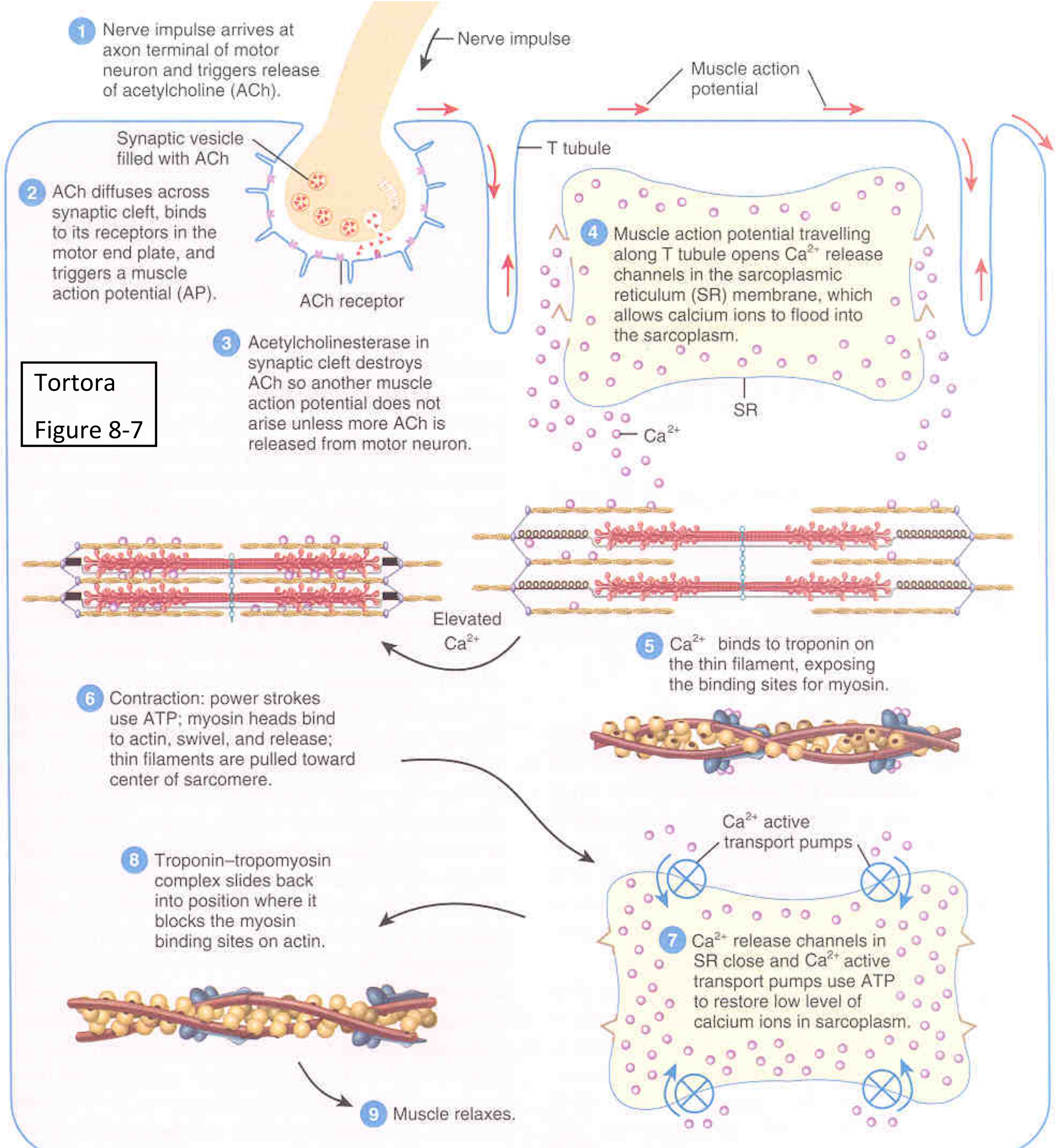
1 Motor neuron action potential
2 Ca^{2+} enters voltage-gated channels
Acetylcholine vesicle
7 Propagated action potential in muscle plasma membrane
Voltage-gated Na^{+} channels
3 Acetylcholine release
4 Na^{+} entry
8 Acetylcholine degradation
Acetylcholine receptor
Acetylcholinesterase
6 Muscle fiber action potential initiation
Motor end plate
5 Local current between depolarized end plate and adjacent muscle plasma membrane

VSL[10] 9-15

TABLE 9–2 *Sequence of Events Between a Motor Neuron Action Potential and Skeletal Muscle Fiber Contraction*

VSL[10]

1. Action potential is initiated and propagates to motor neuron axon terminals.
2. Calcium enters axon terminals through voltage-gated calcium channels.
3. Calcium entry triggers release of ACh from axon terminals.
4. ACh diffuses from axon terminals to motor end plate in muscle fiber.
5. ACh binds to nicotinic receptors on motor end plate, increasing their permeability to Na^+ and K^+.
6. More Na^+ moves into the fiber at the motor end plate than K^+ moves out, depolarizing the membrane and producing the end plate potential (EPP).
7. Local currents depolarize the adjacent muscle cell plasma membrane to its threshold potential, generating an action potential that propagates over the muscle fiber surface and into the fiber along the T-tubules.
8. Action potential in T-tubules induces DHP receptors to pull open ryanodine receptor channels, allowing release of Ca^{2+} from lateral sacs of sarcoplasmic reticulum.
9. Ca^{2+} binds to troponin on the thin filaments, causing tropomyosin to move away from its blocking position, thereby uncovering cross-bridge binding sites on actin.
10. Energized myosin cross-bridges on the thick filaments bind to actin:
$$A + M \cdot ADP \cdot P_i \rightarrow A \cdot M \cdot ADP \cdot P_i$$
11. Cross-bridge binding triggers release of ATP hydrolysis products from myosin, producing an angular movement of each cross-bridge:
$$A \cdot M \cdot ADP \cdot P_i \rightarrow A \cdot M + ADP + P_i$$
12. ATP binds to myosin, breaking linkage between actin and myosin and thereby allowing cross-bridges to dissociate from actin:
$$A \cdot M + ATP \rightarrow A + M \cdot ATP$$
13. ATP bound to myosin is split, energizing the myosin cross-bridge:
$$M \cdot ATP \rightarrow M \cdot ADP \cdot P_i$$
14. Cross-bridges repeat steps 10 to 13, producing movement (sliding) of thin filaments past thick filaments. Cycles of cross-bridge movement continue as long as Ca^{2+} remains bound to troponin.
15. Cytosolic Ca^{2+} concentration decreases as Ca^{2+}-ATPase actively transports Ca^{2+} into sarcoplasmic reticulum.
16. Removal of Ca^{2+} from troponin restores blocking action of tropomyosin, the cross-bridge cycle ceases, and the muscle fiber relaxes.



Tortora

Figure 8-7

END

Video 6, Module 3