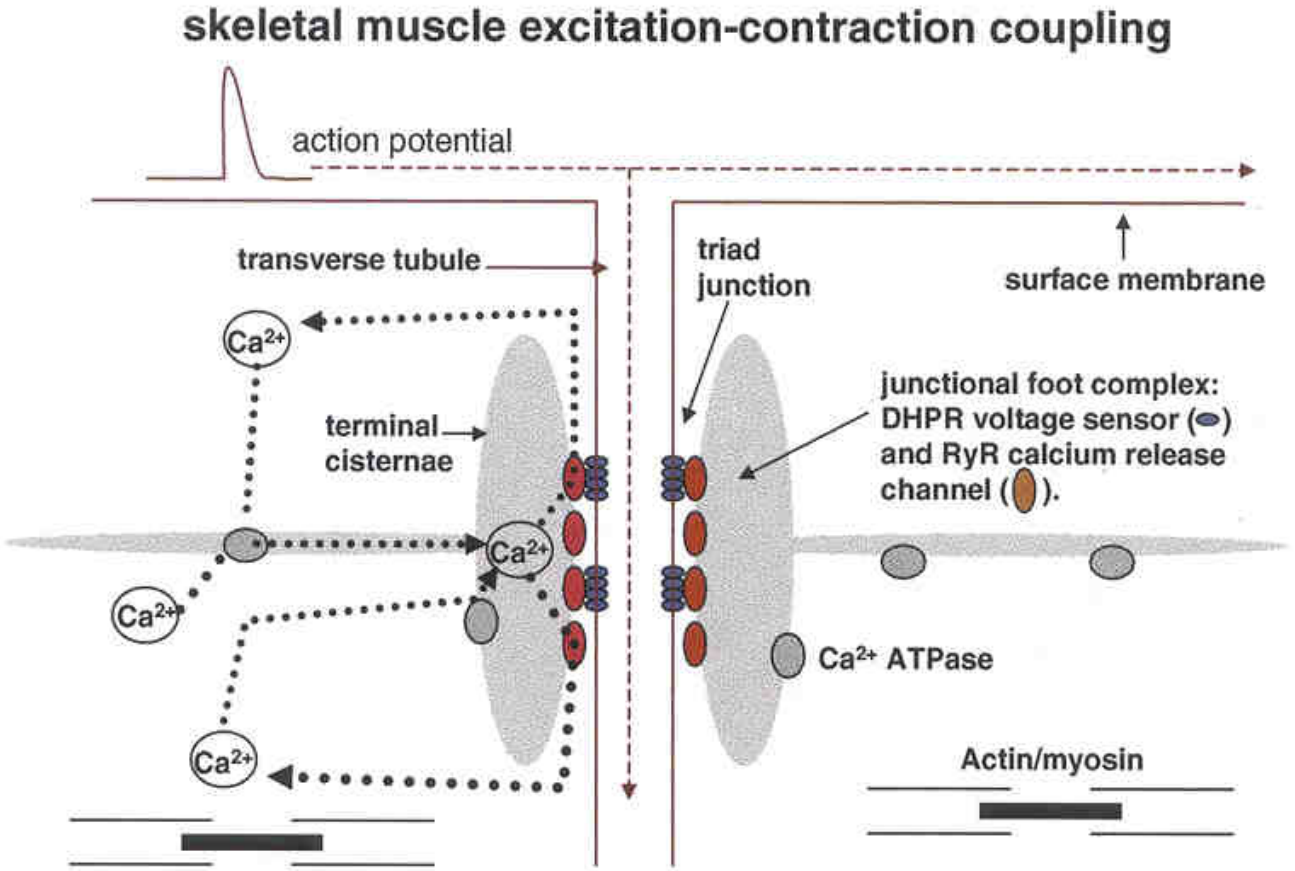


Fig. 1. Membrane systems and membrane proteins involved in excitation-contraction coupling. The action potential propagates along the surface and transverse tubule membranes. The signal resulting from depolarization is transmitted across the triad junction that is formed between the transverse tubule membrane and the terminal cisternae membrane of the sarcoplasmic reticulum Ca^{2+} store. A triad is the usual junction between the t-tubule and sarcoplasmic reticulum in skeletal muscle and is named because it consists of a central t-tubule with a terminal cisternae of sarcoplasmic reticulum on either side. The voltage sensor for EC coupling is the dihydropyridine receptor (DHPR) in the transverse tubule membrane. The Ca^{2+} release channel in the sarcoplasmic reticulum is the ryanodine receptor (RyR). A tetrad of four DHPRs oppose every second RyR. The contraction/relaxation cycle is terminated when Ca^{2+} is taken back into the sarcoplasmic reticulum by the Ca^{2+} pump (Ca^{2+}-ATPase).

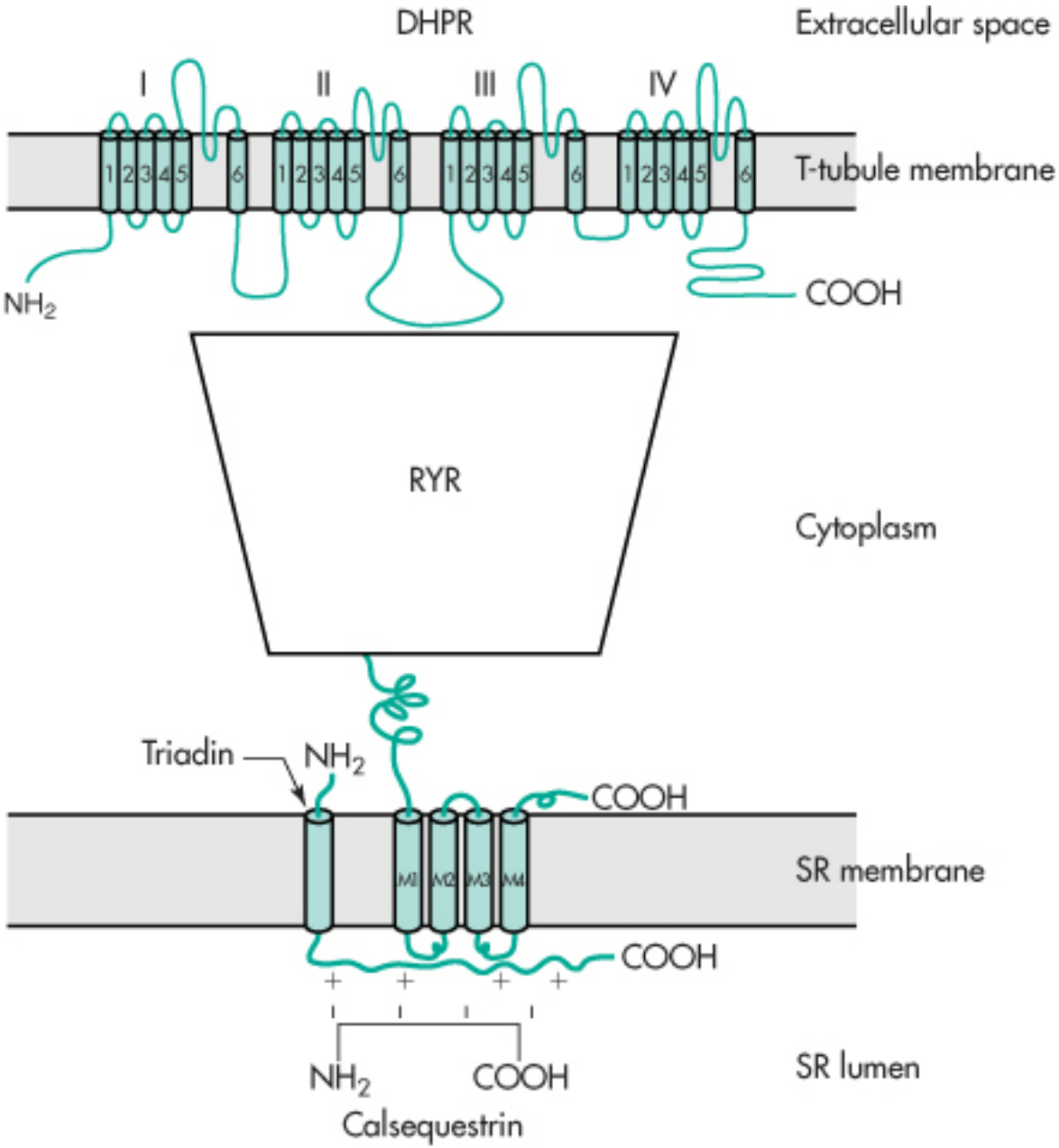


Figure 12-8 Molecular structure and relationships between the dihydropyridine receptor (DHPR) in the T-tubule membrane and the ryanodine receptor (RYR) in the SR membrane. Triadin is an associated SR protein that may participate in the interaction of the RYR and DHPR. Calsequestrin is a low-affinity Ca^{2+}- binding protein that helps to accumulate Ca^{2+} in the terminal cisternae. See text for details. (From Franzini-Armstrong C, Protasi F: Physiol Rev 77(3):699, 1997.)

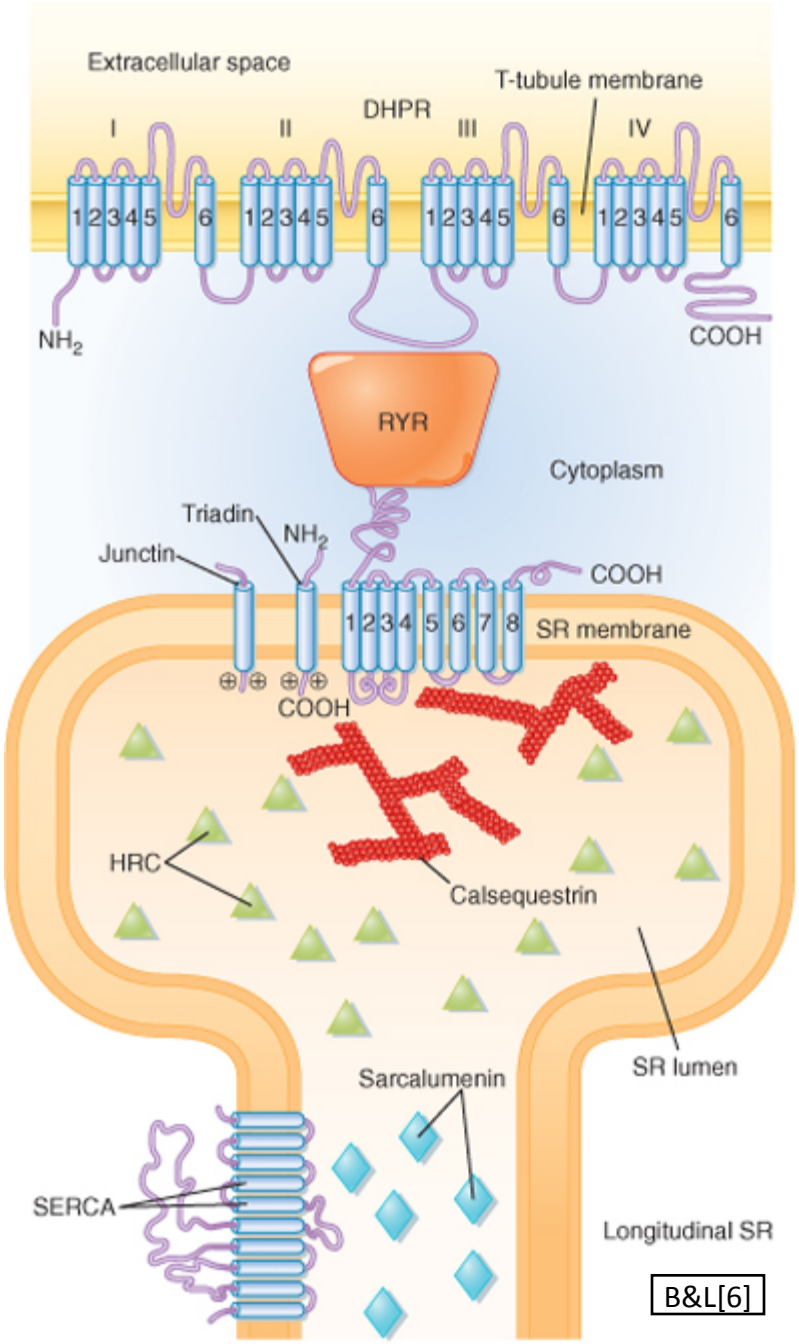


Figure 12-10 Molecular structure and relationships between the dihydropyridine receptor (DHPR) in the T-tubule membrane and the RYR in the SR membrane. Triadin is an associated SR protein that may participate in the interaction of RYR and DHPR. Calsequestrin is a low-affinity Ca^{++}-binding protein that helps accumulate Ca^{++} in the terminal cisternae. See text for details. (From Rossi AE, Dirksen RT: Muscle Nerve 33:715, 2006.)

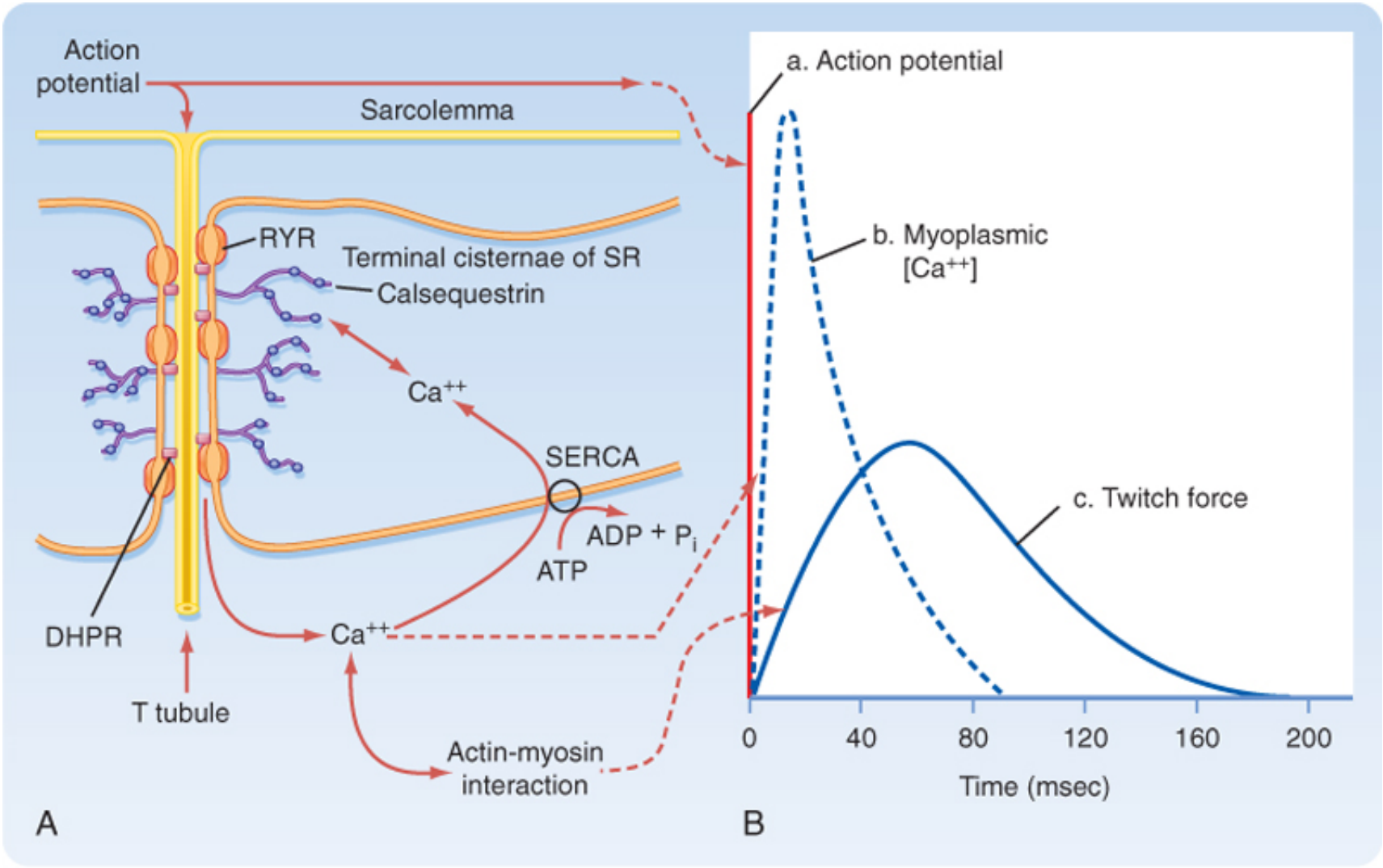
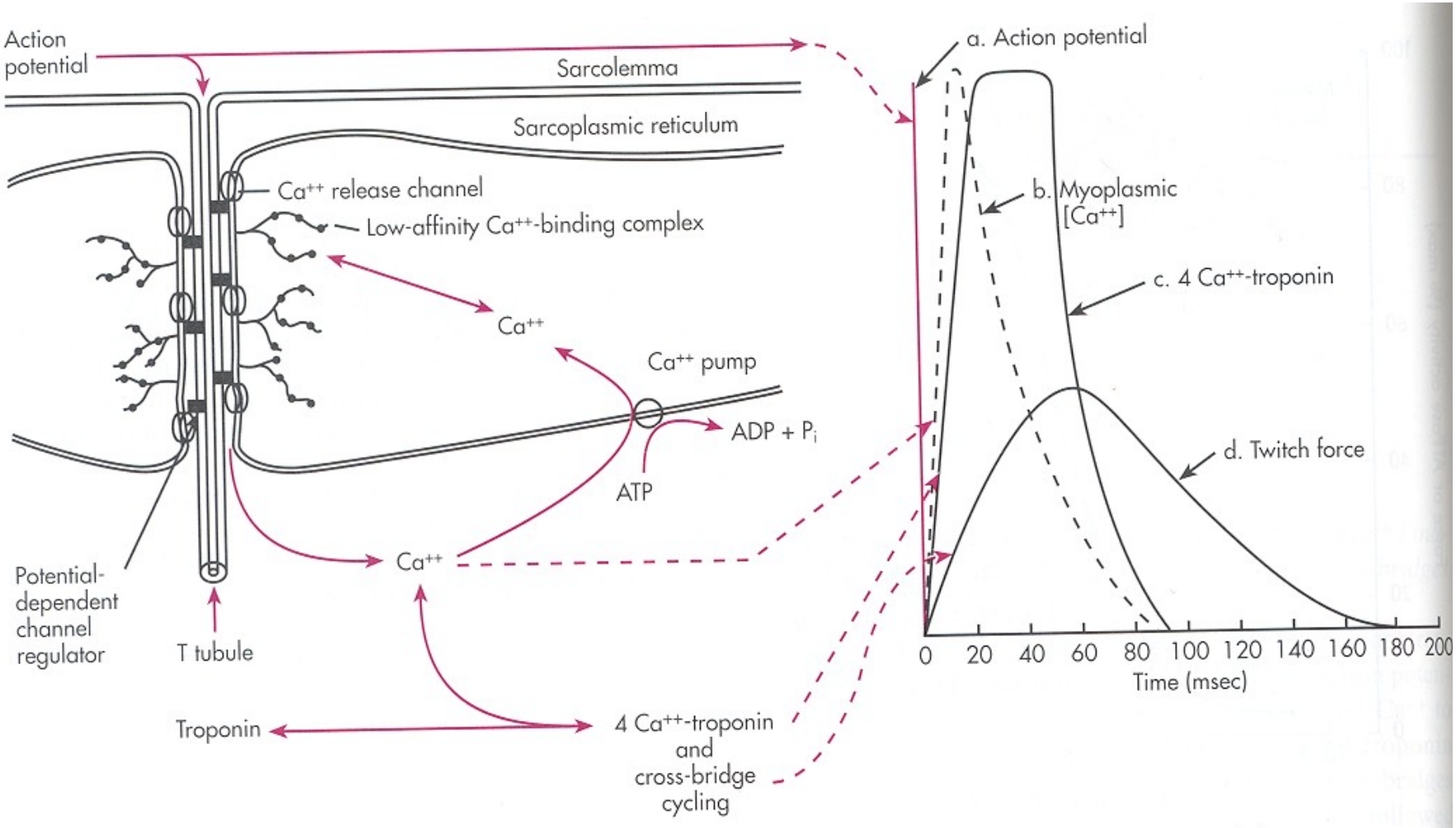


Figure 12-8 Stimulation of a skeletal muscle fiber initiates an action potential in the muscle that travels down the T tubule and induces release of Ca^{++} from the terminal cisternae of the SR (A). The rise in intracellular [Ca^{++}] causes a contraction. As Ca^{++} is pumped back into the SR by Ca^{++}-ATPase (SERCA), relaxation occurs. B, Time courses of the action potential, myoplasmic Ca^{++} transient, and force of the twitch contraction.



■ **Fig. 18-3** **A**, Membranes and proteins involved in the regulation of myoplasmic Ca^{++} in skeletal muscle. Action potentials propagating along the sarcolemma (**B**, *a*) depolarize T-tubular membranes containing voltage-sensitive elements that regulate the opening of Ca^{++} channels in the adjacent membranes of the sarcoplasmic reticulum. A pulse of Ca^{++} ions (**B**, *b*) diffuses out of the sarcoplasmic reticulum into the myoplasm while the channel is open. In the myoplasm, the Ca^{++} can bind to troponin (**B**, *c*) and initiate cross-bridge cycling (**B**, *d*) or to Ca^{++} pumps that return it to the sarcoplasmic reticulum where most Ca^{++} ions reversibly associate with low-affinity Ca^{++}-binding proteins.

B&L[4]

END

Video 5, Module 3