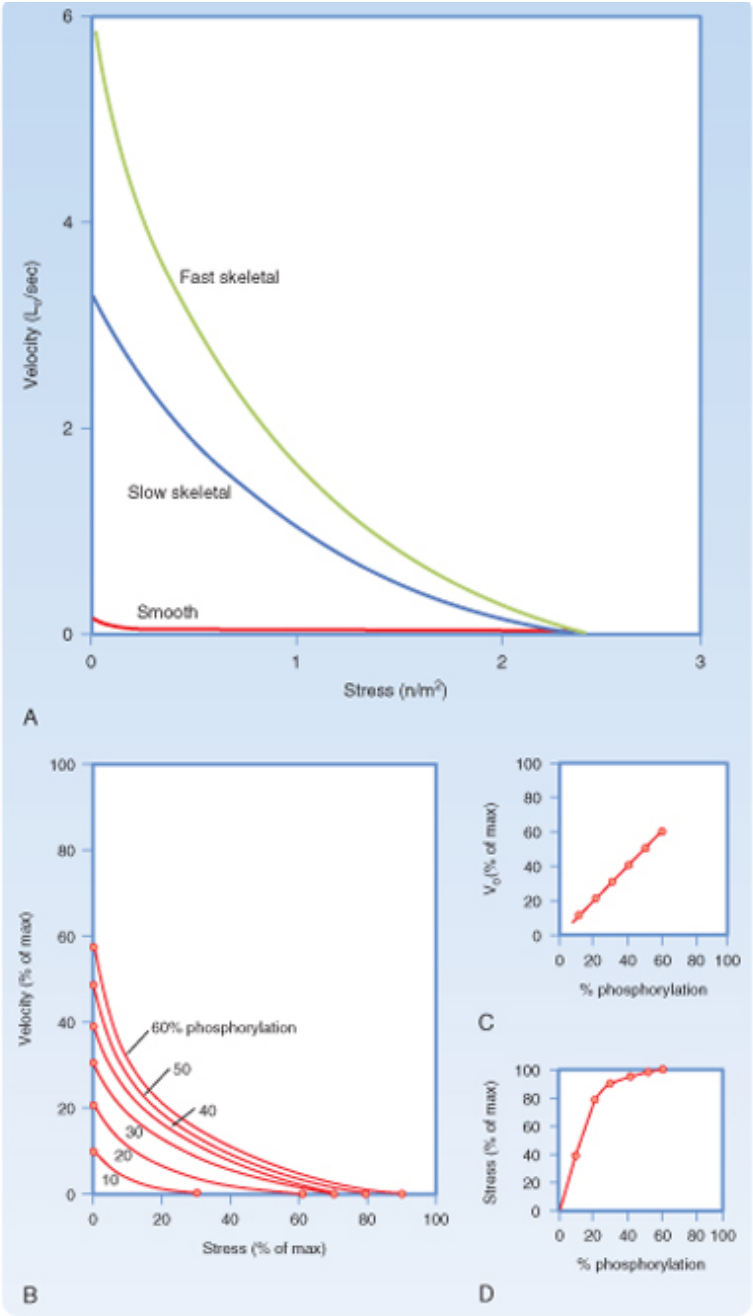


Figure 14-16 A, Force-velocity curves for fast and slow human skeletal muscle cells and smooth muscle. B, Smooth muscles have variable force-velocity relationships that are determined by the level of Ca++-stimulated cross-bridge phosphorylation. C, Maximal shortening velocities with no load (intercepts on the ordinate in B) are directly dependent on cross-bridge phosphorylation by MLCK. D, Active force/stress (abscissa intercepts in B) rises rapidly with phosphorylation and, near maximal stress, may be generated with only 20% to 30% of the cross-bridges in the phosphorylated state.

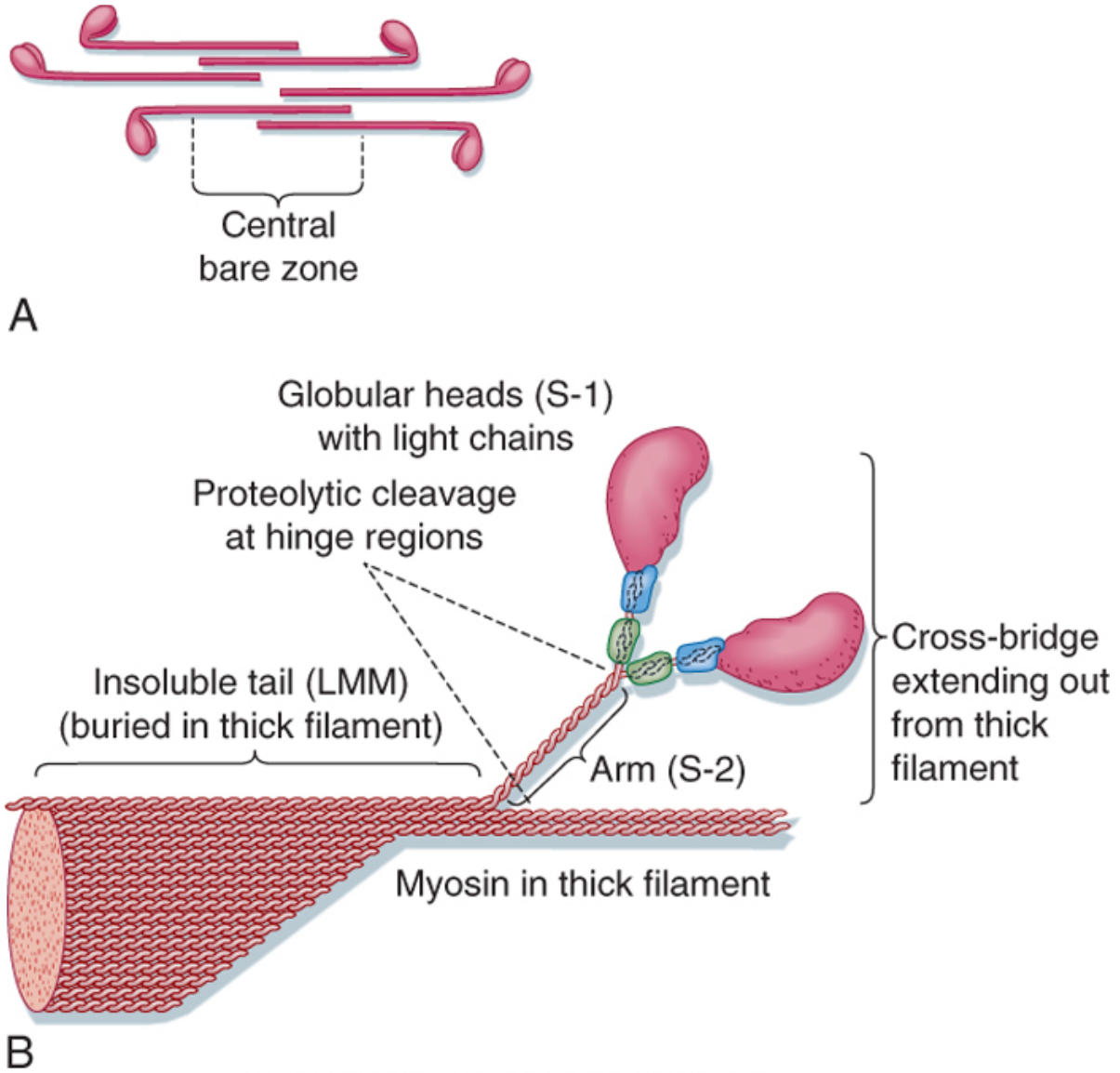


Figure 12-6 Organization of a thick filament. A thick filament is formed by the polymerization of myosin molecules in a tail-to-tail configuration extending from the center of the sarcomere (A). An individual myosin molecule has a tail region and a cross-bridge region. The cross-bridge region is composed of an arm and globular heads (B). The globular heads contain light chains that are important for the function of myosin ATPase activity.

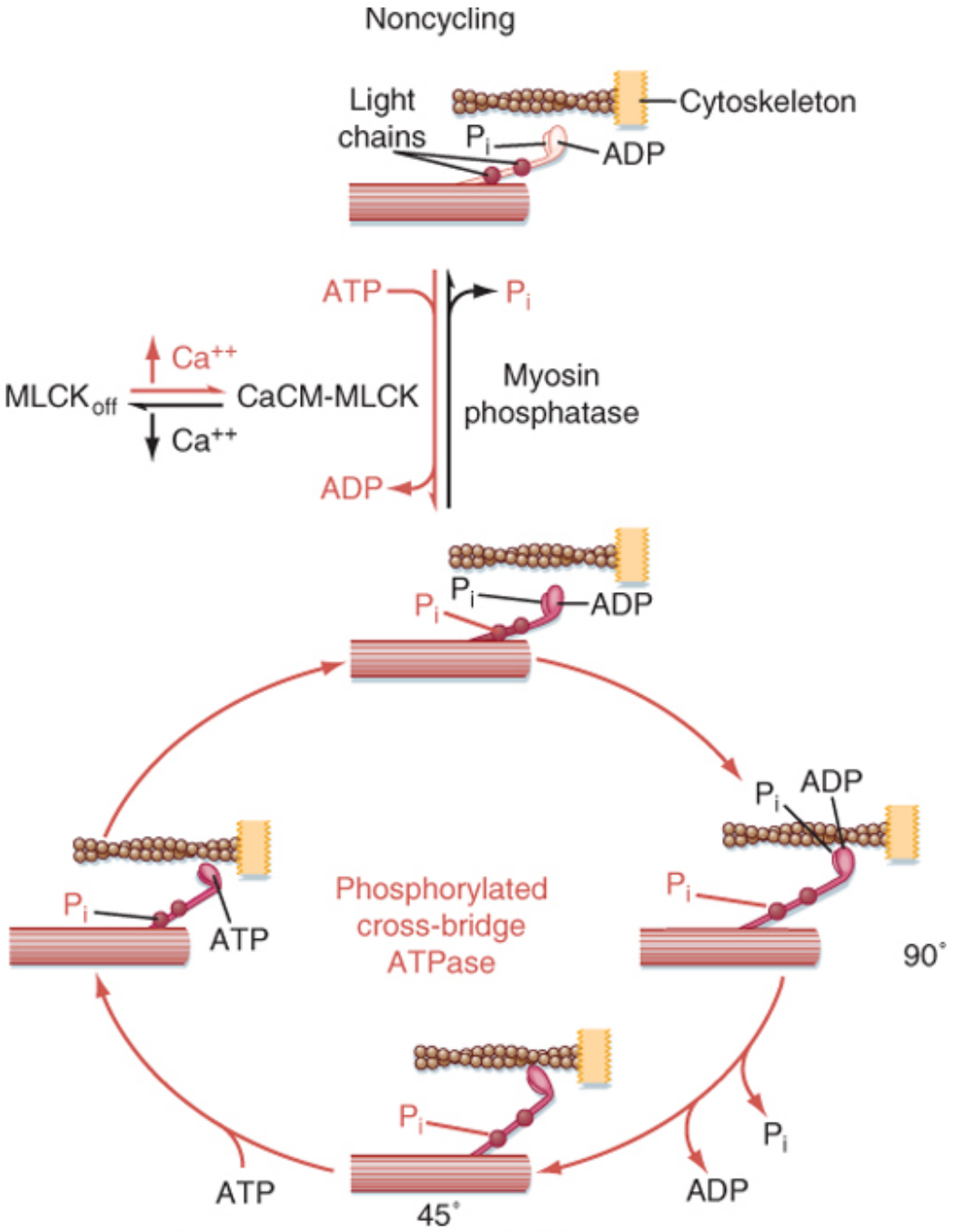


Figure 14-8 Regulation of smooth muscle myosin interactions with actin by Ca^{++}-stimulated phosphorylation. In the relaxed state, cross-bridges are present as a high-energy myosin-ADP-P_i complex in the presence of ATP. Attachment to actin depends on phosphorylation of the cross-bridge by a Ca^{++}-calmodulin-dependent myosin light-chain kinase (MLCK). Phosphorylated cross-bridges cycle until they are dephosphorylated by myosin phosphatase. Note that cross-bridge phosphorylation at a specific site on a myosin regulatory light chain requires ATP in addition to that used in each cyclic interaction with actin.

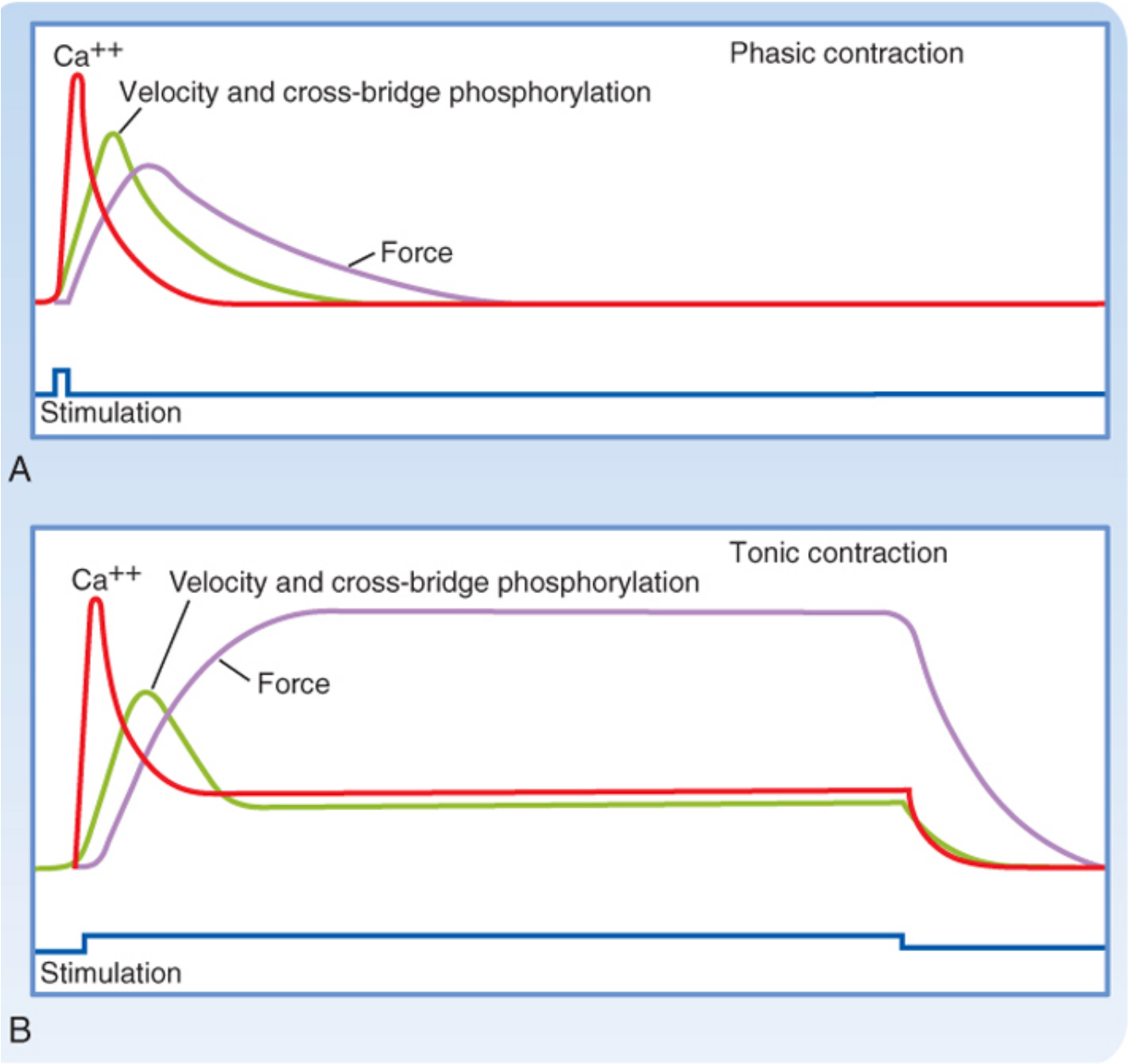


Figure 14-10 Time course of events in cross-bridge activation and contraction in smooth muscle. A, A brief period of stimulation is associated with Ca^{++} mobilization, followed by cross-bridge phosphorylation and cycling to produce a brief phasic, twitch-like contraction. B, In a sustained tonic contraction produced by prolonged stimulation, the Ca^{++} and phosphorylation levels typically fall from an initial peak. Force is maintained during tonic contractions at a reduced [Ca^{++}] (and hence a low level of myosin light-chain phosphorylation), with lower cross-bridge cycling rates manifested by lower shortening velocities and ATP consumption.

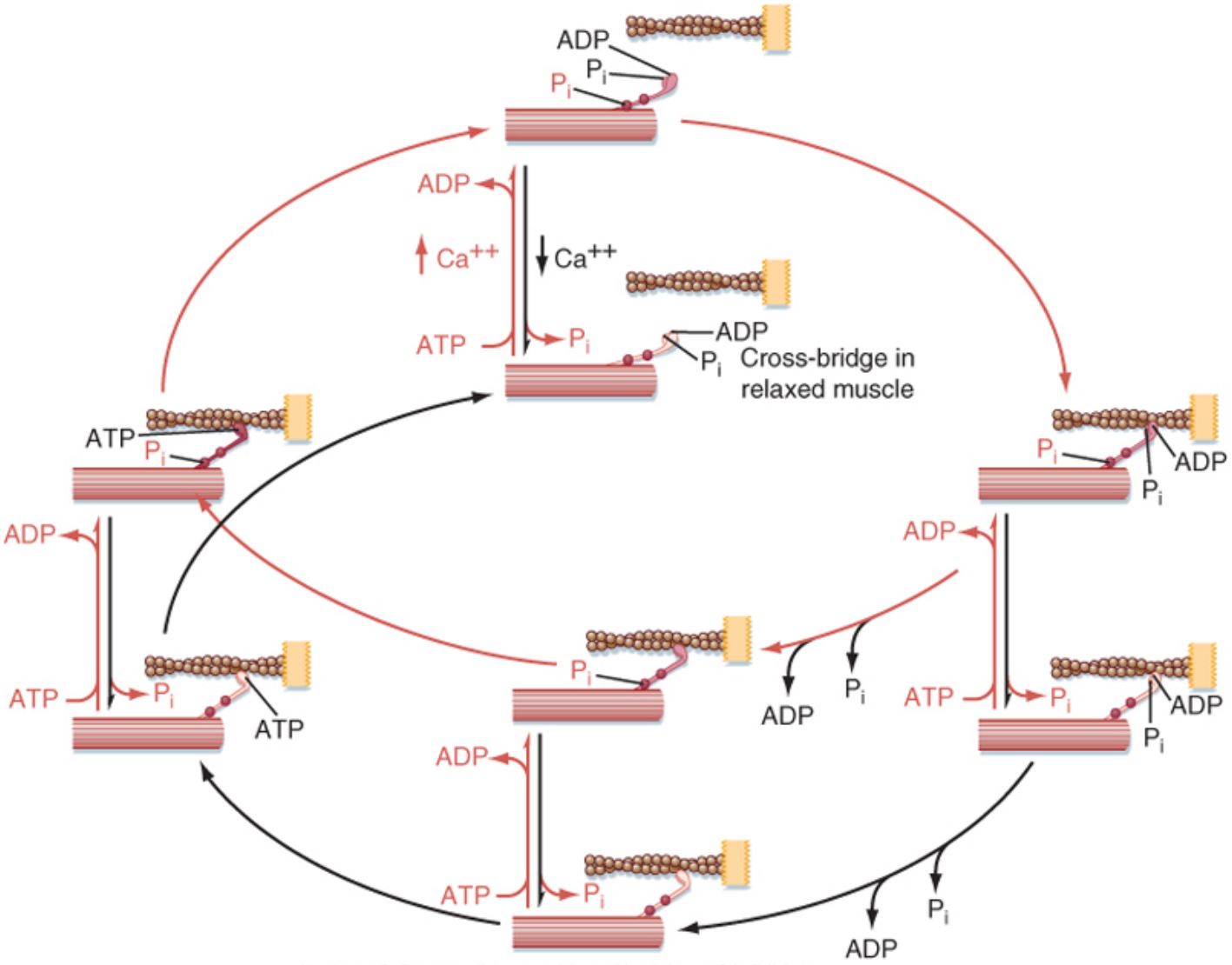
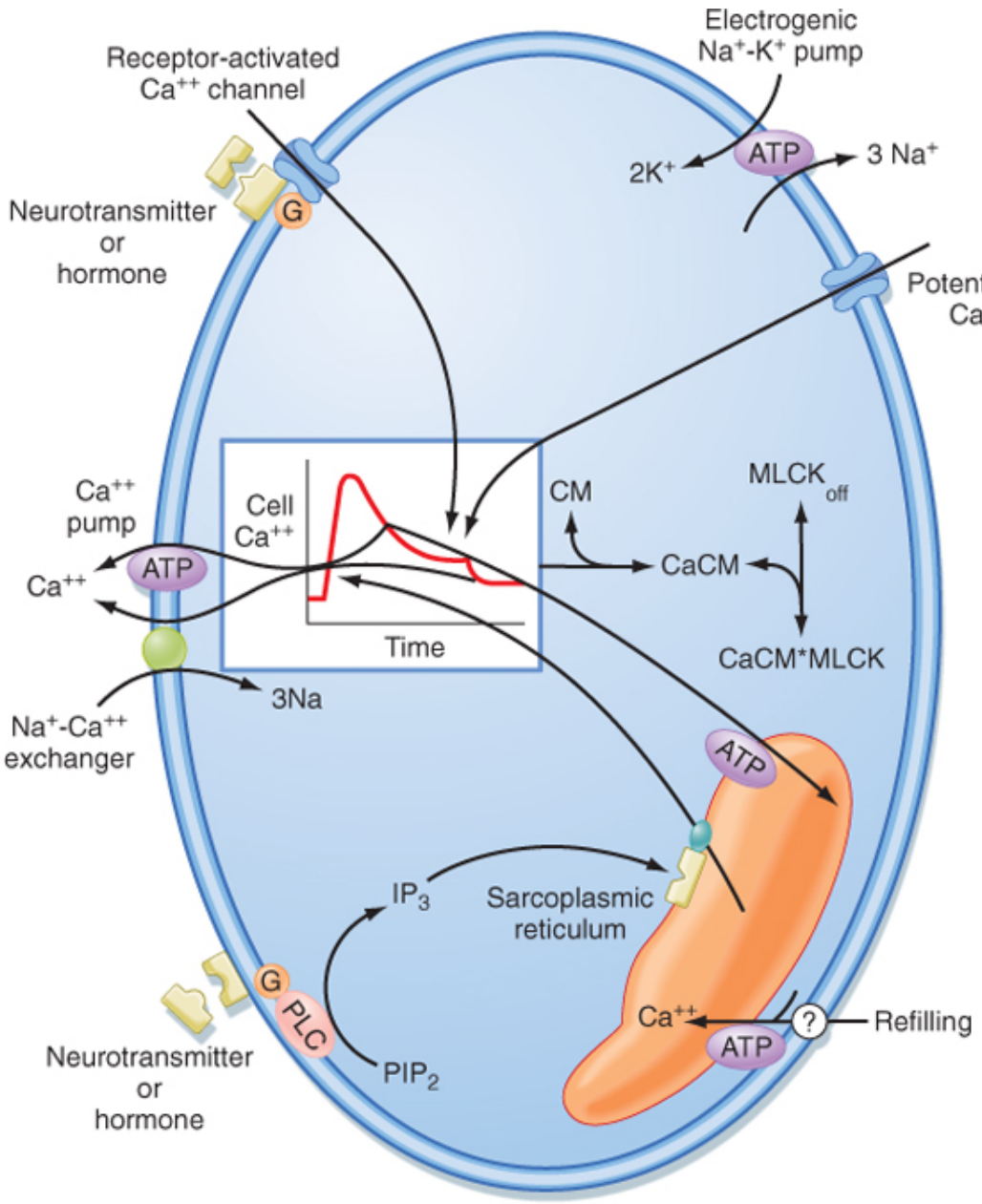


Figure 14-11 Covalent regulation allows eight cross-bridge states in smooth muscle. Phosphorylation by MLCK (vertical red arrows) is obligatory for cross-bridge attachment. Phosphorylated cross-bridges cycle comparatively rapidly. Dephosphorylation of a cross-bridge during a cycle by a constitutively active MP (vertical black arrows) slows cycling rates and produces the latch state. Calcium regulates cross-bridge cycling by determining phosphorylation rates. Note that ATP is required for both regulation (vertical arrows) and cycling (curved arrows).



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

Figure 14-12 Principal mechanisms determining myoplasmic [Ca^{++}] in smooth muscle. Release of calcium from the SR is a rapid initial event in activation, whereas both the SR and the sarcolemma participate in the subsequent stimulus-dependent regulation of myoplasmic [Ca^{++}]. The sarcolemma integrates many simultaneous excitatory and inhibitory inputs to govern the cellular response. Higher-order regulatory mechanisms can alter the activity of various pumps, exchangers, or enzymes (the asterisks designate well-established instances). ATP, process requires ATP hydrolysis; CM, calmodulin; G, guanine nucleotide-binding proteins; IP_3, inositol 1,4,5-trisphosphate; MLCK, myosin light-chain kinase; PIP_2, phosphatidylinositol bisphosphate; PLC, phospholipase C.

END

Video 3, Module 4