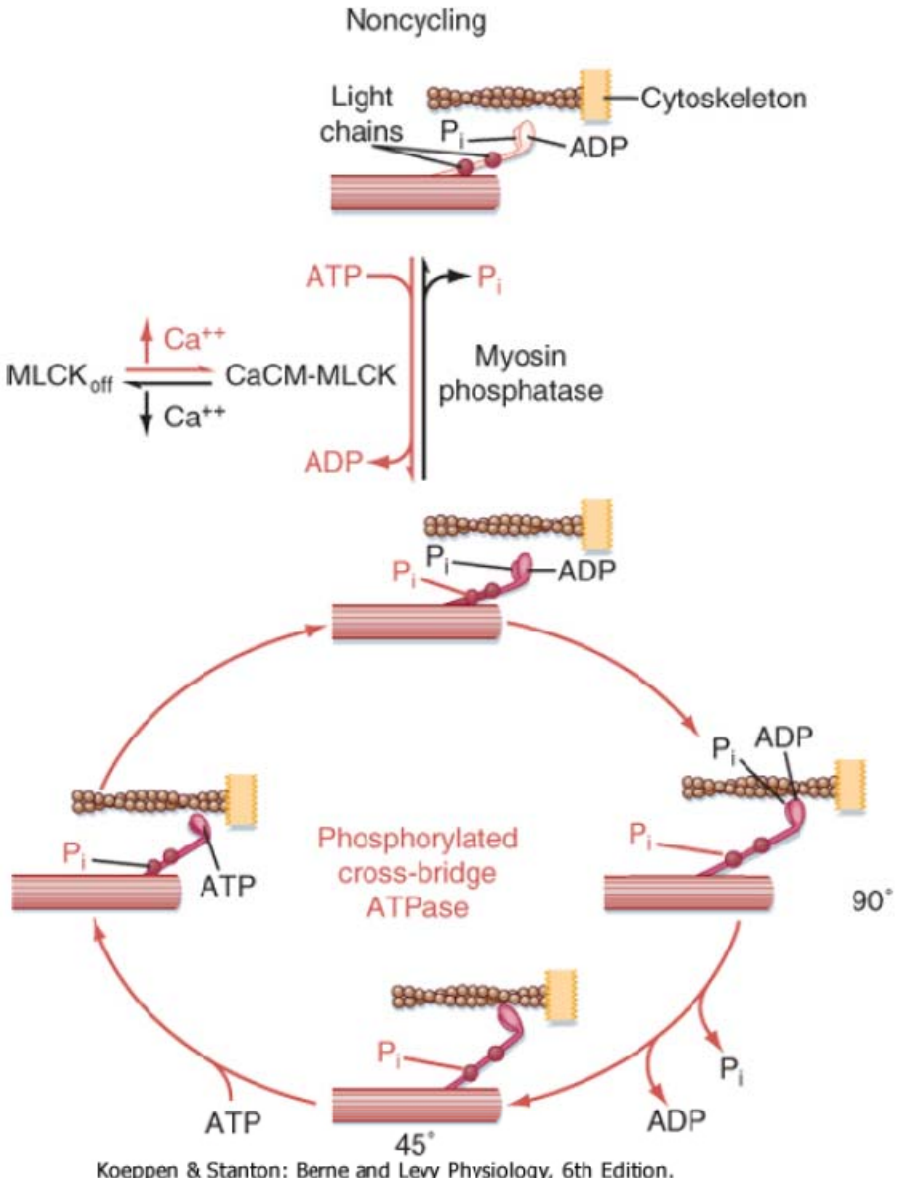
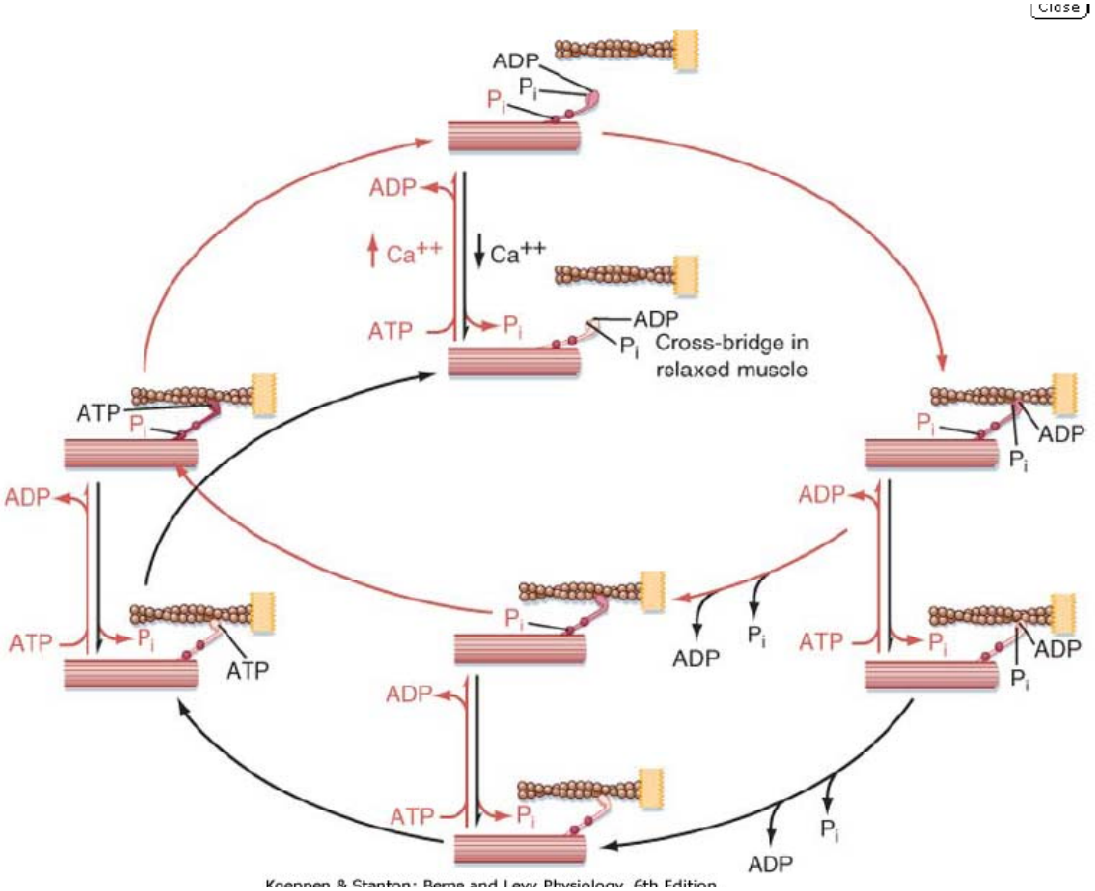
This supplement is meant to answer some reoccurring questions about smooth muscle contraction and control. Below is a figure showing the 2 steps in smooth muscle contraction. Step 1 = Phosphorylation of the regulatory Myosin light chains. Step 2 is like that seen with skeletal muscle cross bridge cycling; however myosin phosphatase can take the Pi off of the regulatory myosin light chains at different points in the cross bridge cycle as seen on the next page on the latch state. In addition the cross bridge cycle is much slower in smooth muscle than it is in skeletal muscle.



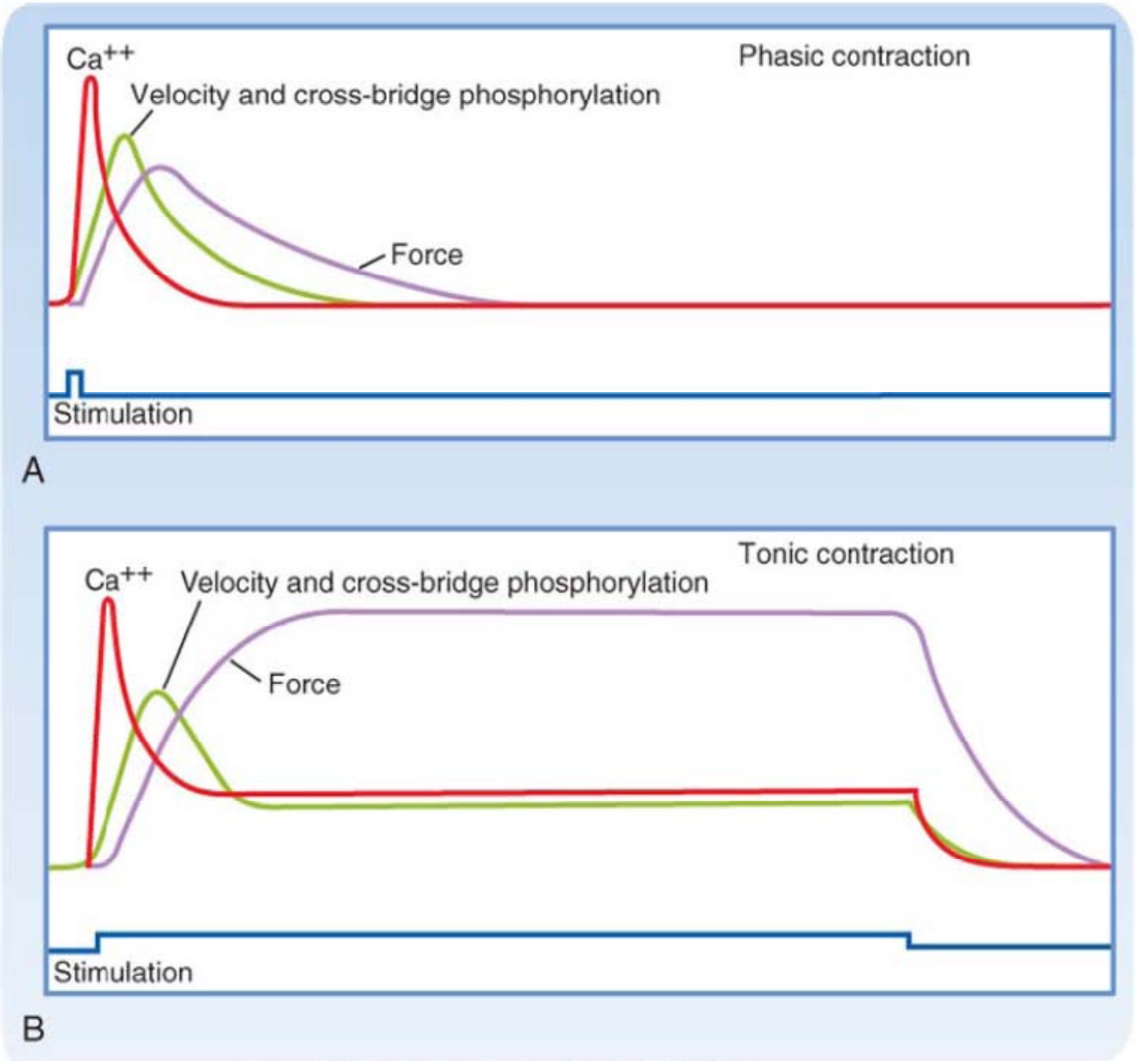
Koeppen & Stanton: Berne and Levy Physiology, 6th Edition.

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.

Below one can see the cross bridge cycling for smooth muscle and the different stages that myosin phosphatase can take of the Pi from the regulatory light chain of myosin. Recall that once the Pi is taken off of the light chain the ATPase activity of myosin slows down even more. This activity is needed to release the myosin from the actin and leaves ADP and a Pi on the myosin, rather than an ATP, see the last two steps in the cycle below. See the last figure on skeletal muscle for more on this partial hydrolysis of ATP to ADP and Pi.



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)*.



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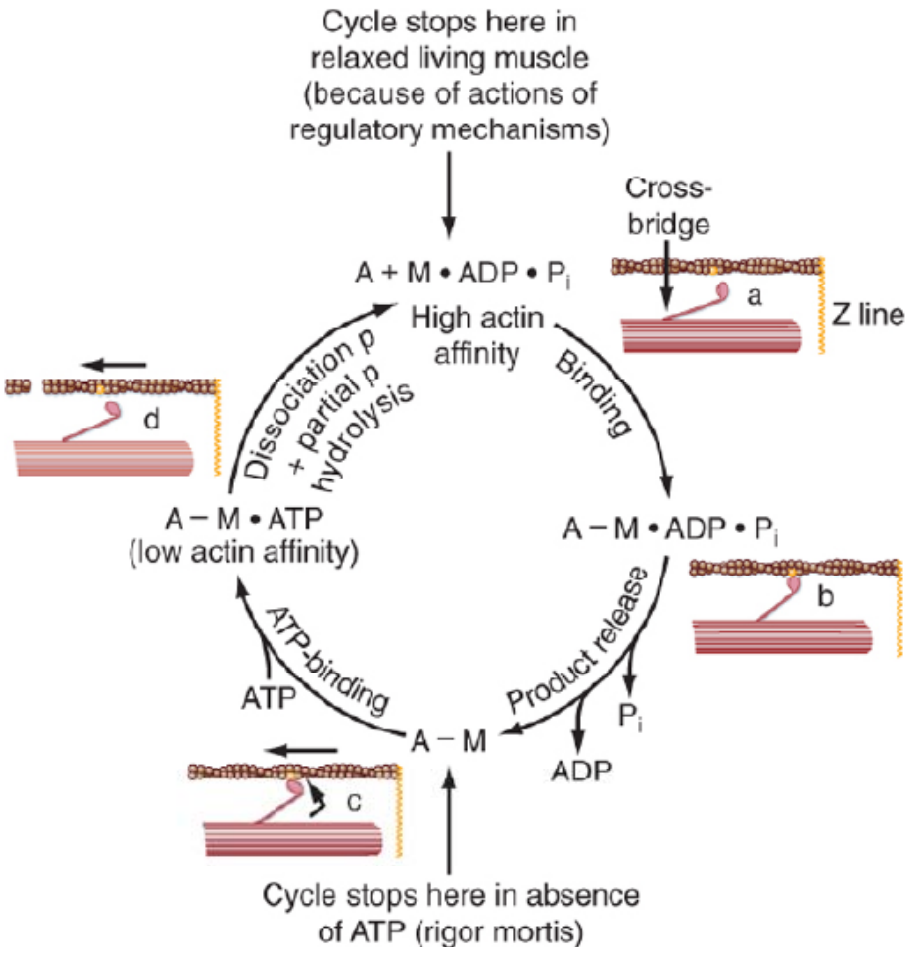

Cross-bridge cycle. **State a,** In the relaxed state, ATP is partially hydrolyzed (M · ADP · P_i). **State b,** In the presence of elevated myoplasmic Ca^{++}, myosin binds to actin. **State c,** Hydrolysis of ATP is completed and causes a conformational change in the myosin molecule that pulls the actin filament toward the center of the sarcomere. **State d,** A new ATP binds to myosin and causes release of the cross-bridge. Partial hydrolysis of the newly bound ATP recocks the myosin head, which is now ready to bind again and again. If myoplasmic [Ca^{++}] is still elevated, the cycle repeats. If myoplasmic [Ca^{++}] is low, relaxation results.

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