The forces that arise from the actin cortex play a crucial role in determining the membrane deformation. These include protrusive forces due to actin polymerization, pulling forces due to transient attachment of actin filaments to the membrane, retrograde flow powered by contraction of actomyosin network, and adhesion to the extracellular matrix. Here we present a theoretical model for membrane deformation resulting from the feedback between the membrane shape and the forces acting on the membrane. We model the membrane as a series of beads connected by springs and determine the final steady-state shape of the membrane arising from the interplay between pushing/pulling forces of the actin network and the resisting membrane tension. We specifically investigate the effect of the gel dynamics on the spatiotemporal deformation of the membrane until a stable lamellipodium is formed. We show that the retrograde flow and the cross-linking velocity play an essential role in the final elongation of the membrane. Interestingly, in the simulations where motor-induced contractility is switched off, reduced retrograde flow results in an increase in the rate and amplitude of membrane protrusion. These simulations are consistent with experimental observations that report an enhancement in protrusion efficiency as myosin II molecular motors are inhibited.