Recent results in vitro and in vivo demonstrate that application of low-frequency ultrasound to neurons leads to activation of voltage-gated sodium channels (Na\textsubscript{v} channels), thereby increasing the rate of action potential firing. These results suggest that modulation of brain activity by transcranial ultrasound may facilitate radical new approaches to behavioral neuroscience and treatment of neurological disorders. However, it is unclear what the mechanistic basis of this effect is, or even whether it represents a direct action of ultrasound on Na\textsubscript{v} channels. To help answer these questions, and to better understand the interactions of ultrasound with membrane proteins in general, we have studied the electrical response of planar lipid bilayers under voltage-clamp to ultrasound (1 MHz). The response to sufficiently long ultrasound pulses consists of distinct dynamic “on” and “off” components with no apparent steady-state effects; shorter pulses result in interference between the two components. The on and off responses are exponentially decaying sinusoidal current oscillations about the baseline level, and are identical except for the polarity of the current. For bilayers made of POPE and POPG (3:1 by weight) and approximately 200 \( \mu \)m in diameter, the mean frequency and decay constant of the off responses are 1033 Hz and 832 s\(^{-1}\). Similar results were obtained with a focused ultrasound transducer with a 43 MHz center frequency, indicating that the response is independent of ultrasound frequency. The electrical response probably results from distortion of the bilayer by ultrasonic radiation force. The electrical response may be either a capacitive current due to rapid changes in bilayer capacitance (under voltage-clamp, \( I_C = V \frac{dC}{dt} \)), or a flexoelectric current due to changes in bilayer curvature with resulting differences in electrical polarization between the inner and outer lipid monolayers. We hypothesized that ultrasound-induced changes in bilayer structure may alter the activity of membrane proteins by changing the contribution of protein/bilayer hydrophobic mismatch to the free energy difference between protein conformations. To test this hypothesis we examined the effects of ultrasound on gramicidin single channel currents. In preliminary experiments supporting this hypothesis we found that the lifetime of single gramicidin channels was reduced when ultrasound pulses were triggered by crossing of a current threshold corresponding to formation of a single gramicidin dimer. If our hypothesis is correct, ultrasound could affect many types of ion channels and membrane proteins through hydrophobic mismatch, and these effects would be dependent on the protein’s membrane environment.