Abdominal aortic aneurysm (AAA) rupture is a common cause of mortality in the United States. Current treatments are only employed once the risk of rupture outweighs the risks associated with surgery. Murine models have been developed to characterize AAA pathogenesis in the hope that new treatments will be developed. For this study, angiotensin II (AngII) was infused subcutaneously into apolipoprotein E-deficient (ApoE\(^{-/-}\)) mice using an osmotic mini-pump over 28 days. ApoE\(^{-/-}\) mice (16-week-old, 3 females, 2 males) were imaged using a VisualSonics Vevo 2100 high frequency ultrasound before pump implantation and 3, 7, 14, 21, and 27 days following implantation. Images were acquired in the transverse and longitudinal planes from the suprarenal region of the aorta. Blood pressure measurements were taken using a tail-cuff system (CODA, Kent Scientific). Three mice (1 female, 2 male) developed aneurysms within the first 14 days of infusion. Pre-study abdominal aortas had a diastolic diameter of 0.84±0.09 mm and a systolic diameter of 0.96±0.08 mm. By day 21, AAAs had a diastolic diameter of 1.51±0.59 mm and a systolic diameter of 1.56±0.59 mm. Initially, mice had a systolic blood pressure of 111.94±6.53 mmHg and a diastolic pressure of 82.38±5.13 mmHg. These pressures steadily elevated but eventually began to plateau. By day 27, systolic pressure had risen to 154.92±11.43 mmHg and diastolic pressure to 115.77±10.25 mmHg. Color Doppler images revealed complex, recirculating flow within the aneurysms, a phenomenon which could affect vessel remodeling. In conclusion, this study utilized \textit{in vivo} sonographic methods to characterize AAA development.