Immune cells appear to be critical for the initiation, progression and manifestation of arterial hypertension. For instance, monocytes can induce vascular inflammation as well as tissue remodelling and (mal)adaptation by secreting chemokines and cytokines, producing ROS, expressing coagulation factors and transforming into macrophages. A multitude of adhesion molecules promote the infiltration and accumulation of inflammatory cells into the kidney, heart, brain and vasculature in hypertension. All these facets offer the possibility to pharmacologically target these cells. This may represent novel therapeutic ways to treat hypertension, attenuate hypertension-associated end organ damage or prevent the development or worsening of high blood pressure.