Preeclampsia (PE) is a novel risk factor for cardiovascular disease. PE not only results in pregnancy-induced hypertension but also a multi-organ syndrome characterised by widespread endothelial damage. Anti-angiogenic factors, soluble vascular endothelial growth factor (VEGF) receptor-1 (commonly known as sFlt-1) and soluble endoglin (sEng) are increased dramatically in pregnancy prior to the clinical onset of PE. Several studies, including ours have demonstrated that sFlt-1 is quite possibly the “final common pathway” responsible for the signs of PE. Recently, we showed that introduction of sFlt-1 into pregnant mice (which increased plasma levels of sFlt-1) was accompanied by hypertension and proteinuria and this could be reversed by VEGF. Collectively, these studies demonstrate that high levels of circulating sFlt-1 mimic PE-like maternal signs in several animal models. In a landmark publication, we showed that the heme oxygenase-1 (HO-1) pathway inhibits sFlt-1 and sEng in cultured endothelial cells and human placental tissue explants [Circulation, 115:1789-97, 2007]. HO-1 is an anti-inflammatory enzyme that generates three molecules (Biliverdin, Fe$^{2+}$ and CO). They are unique in that they all have biological activity. Carbon monoxide (CO), like nitric oxide (NO) promotes vasodilatation, however, the molecular mechanisms responsible for inhibiting sFlt-1 production or PE-like signs are unknown. These will be discussed in line with the above findings to address the question “Can the Biology of VEGF and Heme Oxygenases help solve Preeclampsia?” and we will identify new cheap effective therapies.