Prenatal hypoxia restricts fetal growth and increases risk of adult cardiovascular disease. Programming of β-adrenergic receptor mediated inotropic effects are likely involved since they are desensitized in fast-growing chickens. To understand the mechanisms underlying receptor desensitization, we incubated eggs in normoxia or hypoxia and determined cardiac contractility before and after β-adrenergic stimulation when the chickens were 5 weeks old. Prenatal hypoxia caused contractile dysfunction. cAMP levels were lower and so was the β1AR/β2AR ratio in hypoxic birds, Gi expression was unchanged, while Gs was increased. These changes resemble those reported in early human heart failure. We conclude that prenatal hypoxia has a programming effect on adult β-adrenergic function and speculate that prenatal hypoxia leads to heart failure.