Hypertrophic cardiomyopathy (HCM) is an alarmingly common and often deadly genetic heart disease (1 in 200 people) that is both poorly understood and difficult to treat with currently available therapies. One well established characteristic of anesthetics is their depressant effect on the heart. Recent data suggests this action occurs via a mechanism that may be desirable in the setting of HCM to counteract the cardiac hypertrophy that defines this disease process. As sedative-hypnotic medications, anesthetic themselves could not readily be applied as HCM treatment. However, we have studied a fluorinated propofol derivative (fropofol) that possesses the same cardiac depression without any activity as an anesthetic. Preliminary data supports direct interaction with proteins in the cardiac muscle fibers, which suggests novel therapeutic potential for fropofol. This work seeks to further understand the molecular mechanism of fropofol-induced cardiac depression and its possible application to HCM.