Supplemental Digital Content 1

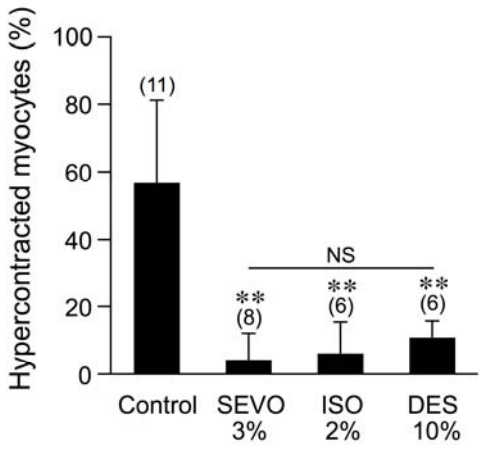

Fig. 1. Protective effects of volatile anesthetics against H_2O_2-induced cellular Ca^{2+} overload and hypercontracture. Percentage of hypercontracted myocytes measured at 15 min after exposure to H_2O_2 (100 μM) in the absence (Control) and presence of 3% sevoflurane (SEVO), 2% isoflurane (ISO) or 10% desflurane (DES). The data shown were obtained from multiple experiments of confocal imaging of fluo-3 fluorescence in ventricular myocytes (*N* = 3 for each volatile anesthetic) at room temperature (23-25°C). **, *P*<0.01 compared with Control. Note that there is no statistical significance (NS) among the three volatile anesthetic groups.

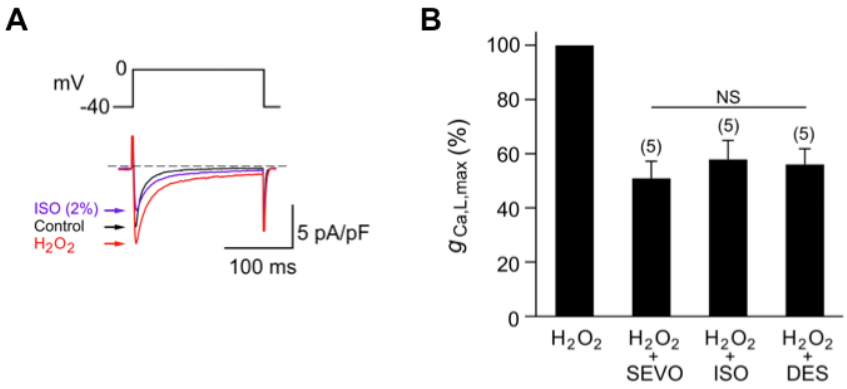

Fig. 2. The inhibitory effects of volatile anesthetics on H_2O_2-stimulated L-type Ca^{2+} current ($I_{Ca,L}$) in mouse ventricular myocytes. (*A*) Superimposed current traces of $I_{Ca,L}$ in response to 200-ms depolarizing steps to 0 mV applied from a holding potential of -40 mV before (Control; black) and during exposure to H_2O_2 (100 μM; red) initially without and then with 2% isoflurane (ISO; purple). (*B*) Summarized data for the reduction of H_2O_2-stimulated $I_{Ca,L}$ by 3% sevoflurane (SEVO), 2% isoflurane (ISO) or 10% desflurane (DES). Maximal conductance for $I_{Ca,L}$ ($g_{Ca,L,max}$) was calculated from $I_{Ca,L}$ recorded during exposure to H_2O_2 (100 μM) initially without and then with each volatile anesthetic, and $g_{Ca,L\ max}$ in the presence of each volatile anesthetic was normalized with reference to the value measured in its absence. Note that there is no statistical significance (NS) among the three volatile anesthetic groups (N = 3 for each volatile anesthetic). The experiments were conducted at 35-37°C.

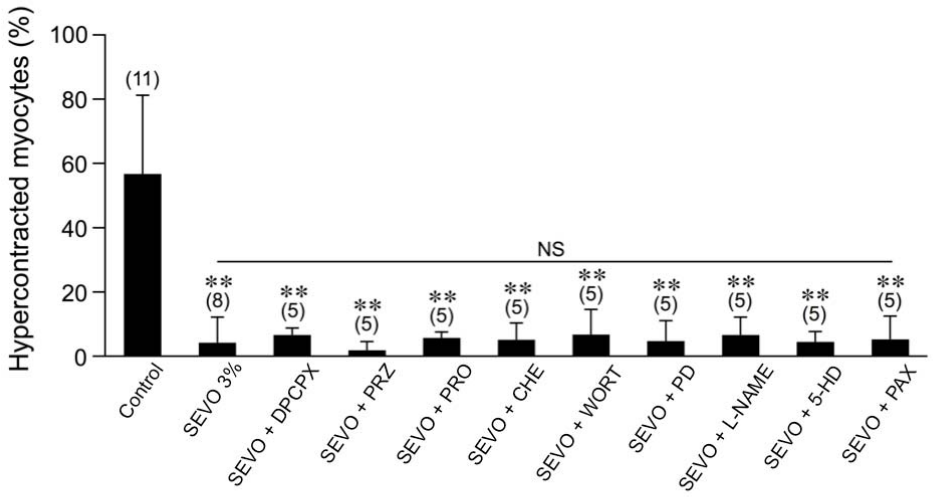

Fig. 3. Protective effects of sevoflurane against H_2O_2-induced Ca^{2+} overload and hypercontracture in the presence of blockers for G-protein coupled cell surface receptors, downstream signalling molecules and targets. Percentage of hypercontracted myocytes measured at 15 min after exposure to H_2O_2 (100 μM) in the absence (Control) and presence of 3% sevoflurane (SEVO) without or with various blockers. The data shown were obtained from multiple experiments of confocal imaging of fluo-3 fluorescence in ventricular myocytes (N = 3-4) at room temperature (23-25°C). **, P<0.01 compared with Control. Note that the protective effect of sevoflurane was not significantly influenced by the presence of any blockers. DPCPX, 8-cyclopentyl-1,3-dipropylxanthine (1 μM); PRZ, prazosin (1 μM); PRO, propranolol (1 μM); CHE, chelerythrine (1 μM); WORT, wortmannin (100 nM); PD, PD098059 (50 μM); L-NAME, *N*-nitro-L-arginine methyl ester (1 mM); 5-HD, 5-hydroxydecanoic acid (100 μM); PAX, paxilline (10 μM).