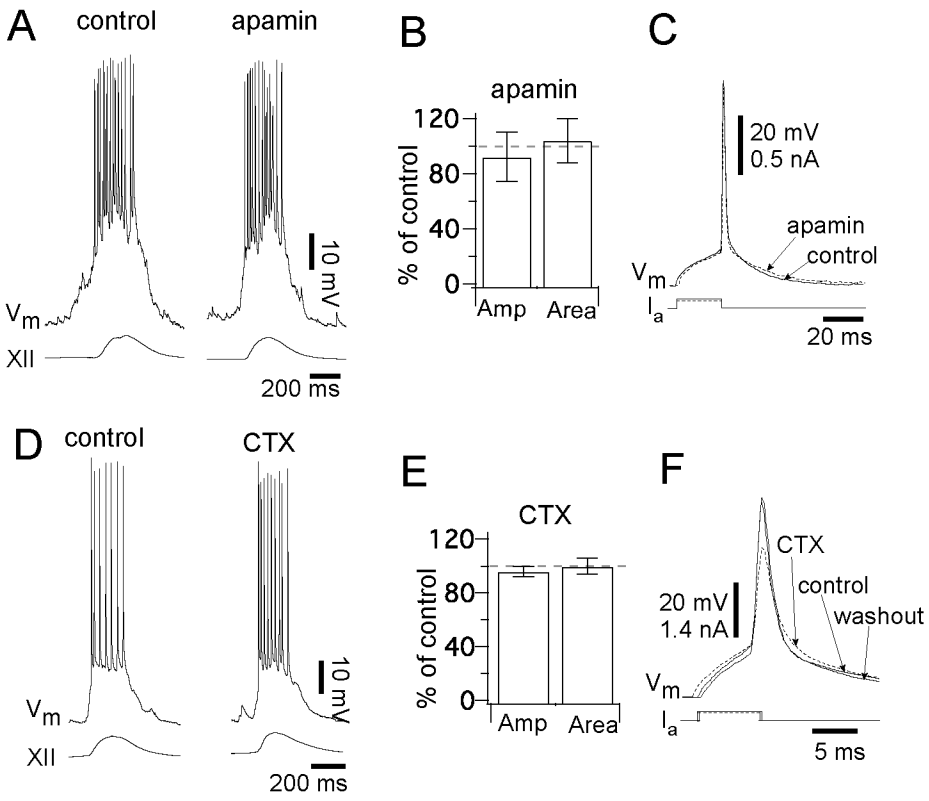

Supplementary Figure 1. Ca2+-activated K+ currents (IK-Ca) in neonatal mouse preBötC neurons do not contribute significantly to inspiratory burst generation. **A-B**, Bath applied (SK-type) IK-Ca blocker apamin (1 μM) had no significant effect on drive potential amplitude and area, which were 96±18% and 108±16% of control, respectively ($p>>0.05$, n=8). Sequential traces show membrane potential (Vm) and hypoglossal nerve (XII) response of a representative inspiratory neuron in the preBötC exposed to apamin. Both control and apamin examples represent steady-state behavior. **C**, SK-type IK-Ca causes after-hyperpolarizations (AHPs). However inspiratory neurons do not exhibit notable AHPs, thus apamin caused little change in evoked spikes. In particular, apamin caused no significant change in the width, amplitude, or rheobase of evoked spikes; these measures were 104±4%, 99±7%, and 100±3% of control, respectively ($p>>0.05$, n=6). **D-E**, Bath applied (BK) IK-Ca blocker charybdotoxin (CTX; 100 nM) had no significant effect on drive potential amplitude or area, which were 100±4% and 104±6% of control, respectively ($p>>0.05$, n=7). **F**, However, CTX reversibly and significantly increased spike width and decreased spike amplitude to 135±10% and 86±3% of control, respectively (width $p<0.05$ and amplitude $p<0.01$, n=6).