Effects of removal of external Na^+ ions on vascular smooth muscles

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Summary

In single cells dispersed from the rabbit portal vein, Na-Ca exchange currents could be recorded with the whole-cell clamp method, under the condition in which all other possible ionic pathways had been blocked. Although Ca-influx mediated by the Na-Ca exchange may be responsible for contraction produced by Na removal (0 Na-contraction) observed in several vascular muscles in the guinea-pig, there are some discrepancies between properties of Na-Ca exchange current and 0 Na-contraction. Na-Ca exchange current was transient, lasting for about 1 min, whereas 0 Na-contraction was slow and long-lasting. The exchange current may be inactivated relatively quickly, but intracellular Ca concentration increased by this process may not be reduced without Na. 0 Na-contraction was only slowly reduced in Ca-free solution, containing 0.1 mM DGTA, suggesting a partial contribution of intracellular Ca release, in addition to a slow decrease in Ca concentration in the extracellular space in muscle strips. Excess Mg was less effective in inhibiting 0 Na-contraction compared with Na-Ca exchange current.

Removal of external Na decreased intracellular pH from about 7.2 to 6.7 when Na was replaced with choline or N-methyl-D-glucamine (NMDG), keeping Cl concentration contant. When NaCl was replaced with sucrose, however, intracellular acidification was much less, but 0 Na-contraction was similar to, or even larger than, those produced by choline or NMDG substitution, suggesting that intracellular acidification is not an important factor in 0 Na-contraction.

When Ca was readmitted after more than 10 min exposure to Ca- and Na-free solution, a quick large contraction could be elicited in the presence of verapamil. This suggests that there is some Ca-influx pathway which is Na-independent and is unlikely voltage-gated Ca channel. This pathway seems resistant to excess Mg and the contribution of this Na-independent pathway to 0 Na-contraction may explain the discrepancies described above. 0 Na contraction was greatly potentiated with ouabain (50 μM) in all vascular muscles studied. On the other hand, ouabain increased resting trension in the pulmonary artery and the portal vein, while it had a marginal effect on the vena cava. Activity of Na-Ca exchange mechanism relative to that of Ca-pump in the plasma membrane seems to vary in different vascular muscles.