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Role of calcium and potassium channels in effects of hydrogen sulfide on frog myocardial contractility

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Abstract

The effects of sodium hydrosulfide NaHS, a donor of hydrogen sulfide H 2S, on the force of muscle contraction were examined on isolated myocardial strips from frog ventricles. NaHS decreased the amplitude of muscle contractions in a dose-dependent manner under normal conditions and during inhibition of Ca channels with nifedipine. In contrast, under conditions of blockade of ATP-dependent potassium channels with glibenclamide, NaHS exerted a positive inotropic effect from the first minute of application. Neither blockade, nor activation of ATP-dependent K-channels with glibenclamide modulated the negative inotropic effect of NaHS. Inhibition of K-channels with tetraethylammonium (TEA) (3, 5, 10 mM) or 4-aminopyridine increased the amplitude of myocardial contractions. Preliminary application of 4-aminopyridine or TEA (3 mM) did not eliminate NaHS-induced negative inotropic effect, although higher TEA concentrations (5 or 10 mM) prevented it. The data indicate that the targets of H2S in frog myocardium are ATP-dependent, Ca-activated, and voltage-dependent K-channels. © 2011 Springer Science+Business Media, Inc.

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Keywords

ATP-dependent K-channels, Ca-activated K-channels, calcium channels, frog myocardium, hydrogen sulfide, voltage-dependent K-channels