

primary studies - published RCT

Efficacy of salbutamol and ipratropium bromide in decreasing bronchial hyperreactivity in children with cystic fibrosis.

Code: PM1534166

Year: 1992 **Date:** 1992

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Study design (if review, criteria of inclusion for studies)

Placebo controlled cross-over trial over 3 days

Participants

11 participants (5 males) with history of RAD, mean (SD) age 10.5 (2.3) years SK score 60 - 95

Interventions

Baseline spirometry Treatment with single dose of either nebulised placebo, 2.5 mg salbutamol or 0.25 mg ipratropium 45 minutes later FEV1 Methacholine challenge doubling concentrations of inhaled methacholine 5 minutes apart with FEV1 taken after each increase

Outcome measures

Drop of greater than 20% of FEV1 from baseline to give the concentration of methacholine or maximum concentration of methacholine

Main results

FEV1 (mean +/- S.E.) did not change following pretreatment with saline, salbutamol, or ipratropium (1.64 +/- 0.22, 1.63 +/- 0.16 and 1.67 +/- 0.19, respectively). All patients demonstrated airway hyperreactivity with a PC20 below 8 mg/mL (geometric mean, 0.41 mg/mL) after saline pretreatment. Salbutamol inhalation significantly increased the PC20 to 1.24 mg/mL (P less than 0.01), but ipratropium bromide was found to be even more effective than salbutamol (PC20 = 7.37 mg/mL) (P less than 0.0001). We conclude that the variable response to bronchodilator is not secondary to impaired aerosol distribution since ipratropium bromide effectively blocked the response to methacholine.

Authors' conclusions

The improvement in PC20 without a change in baseline FEV1 following salbutamol suggests that the adrenergic agent altered the contractile mechanism of smooth muscle.

<http://dx.doi.org/10.1002/ppul.1950130109>

See also

Pediatr Pulmonol. 1992 May;13(1):34-7.

Keywords

Adolescent; Albuterol; Biomarker; Bronchodilator Agents; Child; Ipratropium; Methacholine; non pharmacological intervention - diagn; pharmacological_intervention; Salbutamol; Inhalation OR nebulised; Adrenergic beta-Agonists; Respiratory System Agents; Anticholinergic Agents; Respiratory Tract Diseases;