

**Glucocorticoids/salbutamol/  
theophylline interaction**

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**Metabolic acidosis with hyperventilation in  
paediatric patients: 2 case reports**

A 20-month-old boy and an 8-year-old girl developed metabolic acidosis with hyperventilation while receiving salbutamol [albuterol] and salbutamol/ipratropium bromide; treatment with theophylline and/or glucocorticoids may have potentiated the salbutamol toxicity [dosage information incomplete].

The boy was admitted with a mild fever, progressive breathlessness and a 3-day history of cough. His treatment had included salbutamol syrup, followed by intermittent inhalation of nebulised salbutamol that was progressively increased as his condition deteriorated [durations of treatment to reaction onset not clearly stated]. On admission, he was drowsy and had inspiratory wheeze and bilateral ronchi; his respiratory rate was 38 breaths/minute. He received three doses of inhaled salbutamol/ipratropium bromide, as well as frequent nebulised salbutamol and an injection of hydrocortisone. He continued to be tachypnoeic over the next 2 hours, and analysis showed persistent acidosis. The possibility of  $\beta$ -agonist toxicity was considered, and salbutamol was withdrawn. His tachypnoea subsequently resolved and his blood gas findings normalised. He was later discharged in a stable condition.

The girl was admitted with chest discomfort, progressive cough and a 1-week history of dyspnoea. She had been diagnosed with asthma 2 months earlier, and was receiving inhaled salmeterol/fluticasone propionate twice daily and inhaled salbutamol once daily. Two days before admission, she had received several back-to-back doses of nebulised salbutamol without relief. Examination revealed wheezing with chest retractions, and she had difficulty speaking. She had a respiratory rate of 42 breaths/minute and an oxygen saturation of 92% on room air. She received three doses of salbutamol/ipratropium bromide and hydrocortisone [routes not stated]; however, she had persistent tachypnoea. Tests showed lactic acidosis, and due to impending respiratory failure, she received a theophylline infusion. Her acidosis was suspected to be secondary to increased nebulised salbutamol, which was then reduced to inhalation every 6 hours. Theophylline and ipratropium bromide were continued. Her condition gradually improved, with normalisation of blood gases and decreasing dyspnoea. All medications were subsequently stopped, and she was later discharged.

**Author comment:** "[A] number of other agents used to treat asthma, such as glucocorticoids and theophylline, may potentiate the metabolic effects of beta adrenergic agonists by increasing the intracellular levels of [cyclic adenosine monophosphate] . . . Thus development of lactic acid induced metabolic acidosis causes hyperventilation which should be recognized as a compensatory mechanism to maintain body pH and not mistaken as sign of worsening respiratory condition."

Tomar RPS, et al. Metabolic acidosis due to inhaled salbutamol toxicity: A hazardous side effect complicating management of suspected cases of acute severe asthma. Medical Journal Armed Forces India 68: 242-244, No. 3, Jul 2012.  
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