Asthma & Chronic Hyperventilation

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www.ButeykoClinic.com
www.ButeykoDVD.com
www.AsthmaCare.info
"Noisy and deep" breathing of an asthmatic had always been considered an outcome of the disease. Nobody could even suspect that "deep breathing" was the cause of bronchial asthma, and increased depth of breathing could provoke the appearance of the symptoms of the disease”.

K P Buteyko M D
What is normal breathing?

- Normal breathing minute volume is 4-6 litres (W.H.O.)
How much air do asthmatics breathe?

15 litres minute ventilation


Resting minute ventilation 13.3 +/- 2.0 L/ min &
Exercise minute ventilation, 41.9 +/- 9.0 L/ min)


14.1 litres minute ventilation

Mouth breathing among asthmatics

We speculate that asthmatics may have an increased tendency to switch to oral breathing, a factor that may contribute to the pathogenesis of their asthma.

Enforced oral breathing causes a decrease in lung function in mild asthmatic subjects at rest, initiating asthma symptoms in some. Oral breathing may play a role in the pathogenesis of acute asthma exacerbations.

Enhanced perception of nasal loading may trigger increased oral breathing in asthmatics, potentially enhancing exposure to nonconditioned inhaled gas and contributing to the occurrence and/or severity of bronchoconstrictive exacerbations.
Hypocapnia and asthma
Airway hypopcapnia

Smooth-muscle contraction

Increased mucosal edema

Increased airway resistance

Increased work of breathing

Increased fatigue

Increased sensation of dyspnea

Hyper-ventilation

Progressive respiratory failure

Asthmatic group had a significantly higher resting respiratory frequency and minute ventilation, and had lower mean end-tidal carbon dioxide (ETCO$_2$) [37 mm Hg] than the other two groups (40 mm Hg and 41 mm Hg, respectively).

30 asthmatic and 15 healthy volunteers. Asthmatic group had lower mean resting ETCO$_2$ when compared to the healthy subjects (36 mm Hg v’s 40 mm Hg).

Forty adult patients with acute severe asthma were assessed for arterial blood gases and acid-base changes. Marked degrees of hypoxaemia (mean 60.15mmHg), hypocapnia P(a)CO₂ (34.65mmHg) with apparently normal pH (mean 7.384) were documented in the majority of these patients.


Odhiambo JA, Chwala RD.
“hypocapnia due to voluntary hyperventilation in man causes increased resistance to airflow.” Furthermore, when subjects inhaled an air mixture containing five per cent carbon dioxide “bronchoconstriction was prevented, indicating that it had been due to hypocapnia, not to mechanical factors associated with hyperventilation.”

“As a common aspect of many acute disorders, hypocapnia may have a pathogenic role in the development of systemic diseases.”

“Increasing evidence suggests that hypocapnia appears to induce substantial adverse physiological and medical effects.”

Mild asymptomatic asthma is not associated with clinically significant hyperventilation but is associated with a significant reduction in both arterial and end tidal PCO₂ which relates to airway hyperresponsiveness rather than to the degree of airway obstruction or mucosal inflammation. Anxiety and depression appear not to be implicated.

“In the guinea pig lung, Hypocapnia-induced bronchoconstriction is mediated by tachykinins that are released after the activation of bronchial axonal reflexes.”

Tachykinins mediate hypocapnia-induced bronchoconstriction in guinea pigs  

A. M. Reynolds and R. D. McEvoy  

Journal of Applied Physiology December 1989 vol. 67 no. 6 2454-2460
“Maximum expiratory flow decreased significantly when the alveolar CO2 tension was below 30-35 mmHg, while there was only slight or no influence of CO2 on the maximal flow when the tension was above 35 mmHg. The decrease is taken as evidence of a constrictor effect on peripheral bronchi of hypocapnia”

Hypocapnia caused a consistent increase in flow resistance. Thus, for a minute volume of approximately 30 liters/ min the mean inspiratory flow resistance was 133% greater and the mean respiratory work of ventilating the lungs 68% greater at PaCO\textsubscript{2} 20–25 mm Hg compared to values at 45–50 mm Hg.
Hypocapnia caused a prompt and marked constrictor response in the peripheral lung not associated with cholinergic mechanisms or those involving histamine H1-receptors or prostaglandins.

Changes of PaCO₂ from 20-65 mmHg cause increasing bronchodilation in anesthetized, paralyzed subjects, this effect being attenuated or abolished by drugs. The carbon dioxide bronchodilating effects are probably direct for peripheral structures and are paralleled by a tendency of lung tissue resistance to decrease.

It is possible that hypocapnia creates symptoms that asthma patients cannot control by using their antiasthmatic medication, thus compromising their perceived control over the management of their asthma, and consequently their perceived health. Behavioural interventions should address the problem of hyperventilation in asthma.


Ritz T, Rosenfield D, Meuret AE, Bobb C, Steptoe A.
Ambulant, transcutaneous PCO2 monitoring has been used to show that hyperventilation precedes exacerbation of asthma in a patient. Brief treatment was shown to give him greater control of his breathing and enable him to avoid attacks of asthma.

With the severe load, minute ventilation and respiratory frequency were significantly lower and PETCO₂ was significantly higher during nasal breathing than during oral breathing.

Why does chronic hyperventilation syndrome receive very little attention in the treatment of asthma?

1) It is very difficult to make a diagnosis of hyperventilation in laboratory tests

2) Secondly “no mention is made of any link” between hyperventilation syndrome and asthma

The American Journal of Medicine; December 1986; Volume 81; p989. Hyperventilation Syndrome and Asthma. (Demeter, Cordasco.)
3) “Hyperventilation, leading to airways cooling, will cause bronchoconstriction in vulnerable individuals” but, “because attacks of asthma are accompanied by hyperventilation of physiological origin, the role of hyperventilation in causing asthma attacks may be overlooked”.

British Journal of Psychiatry; 1988; 153, 687-689; Demonstration and treatment of hyperventilation causing asthma.
Resulting in “a large minority of patients may be experiencing avoidable morbidity because of inappropriate diagnoses and ineffective treatment.”

Cooling or dehydration of the airways?

“repeated dry air challenge in dogs in vivo causes persistent airway obstruction and inflammation not unlike that found in human asthma.”

“Winter athletes have an increased incidence of asthma, suggesting that repetitive hyperventilation with cold air may predispose individuals to airways disease. We conclude that repeated DAC causes peripheral airways inflammation, obstruction, hyperactivity, and impaired B-agonist-induced relaxation.”

Davis and Freed American Journal of Respiratory Care Vol: 164, Num5, Sept. 2001
Airway dehydration

- Airway dehydration triggers exercise-induced bronchoconstriction in virtually all patients with active asthma. Dehydration of the expired air is present in asthmatic patients in the emergency department. The bronchoconstriction triggered by dry-air tachypnea challenge in the laboratory can be prevented by humidifying the inspired air.

- Airway Dehydration* A Therapeutic Target in Asthma? Edward Moloney, MB; Siobhan O’Sullivan, PhD; Thomas Hogan, MD; Leonard W. Poulter, DSc; and Conor M. Burke, MD, FCCP
Prolonged overbreathing

“Prolonged hyperventilation (for more than 24 hours) seems to sensitize the brain, leading to a more prolonged hyperventilation.”

Hyperventilation becomes habitual or long term, so even when the primary cause is removed, the behavior is maintained.

Hyperventilation Syndrome and Asthma, Dr Stephen Demeter
Practical examples
Laughter induced asthma- its no joke

- A report published by the American Thoracic Society in May 2005 concluded that laughter causes symptoms among 57% of asthmatics. In the same report Dr. Garay commented, “Nobody knows how laughter brings on asthma, but it might involve hyperventilation.”
Exercise induced asthma

• The greater the breathing volume in comparison with metabolic activity, the greater are the symptoms

• You will recognise how practical situations cause wheezing, cough and breathlessness...
Exercise induced asthma: the protective role of CO2 during swimming-

The Lancet. Peter M Donnelly. Institute of Respiratory medicine, Royal Prince Albert Hospital. Aus.

“In most land based forms of exercise, patterns of breathing are not constrained, VE increases proportionately throughout exercise, and end tidal CO$_2$ tensions are either normal or low. Therefore there is no hypercapnic stimulus for bronchodilation and asthmatics have no protection..... Because end tidal CO$_2$ tensions have not been measured in asthmatics, the potentially protective property of hypercapnia may have been overlooked”.
Buteyko trial results for asthma
Mater Hospital Brisbane 1995

At 12 weeks, BBT;

70% less symptoms
90% less need for reliever medication
50% less need for ICS
Lung function - no change

Control group - taught physiotherapy
No change
A randomised controlled trial of the Buteyko technique as an adjunct to conventional management of asthma. University of Calgary, Canada. Cowie RL, Conley DP, Underwood MF, Reader PG

At six month follow up the Buteyko group had:
Improved asthma control from 40% to 79%
39% of patients decreased inhaled corticosteroids
21% eliminated inhaled corticosteroids
Results at six months;

**Buteyko Group**
Beta agonist decrease 85%
ICS decrease 50%

**Control Group**
Beta agonist decrease 37%
ICS no change
Buteyko Group at six months

Bronchodilators use decrease 66%
Inhaled steroids use decrease 41%
A clinical trial of the Buteyko Breathing Technique in asthma as taught by a video. Opat Aj, Cohen MM, Bailey MJ, Abramson

“Our results demonstrated a significant improvement in quality of life among those assigned to the BBT compared with placebo (p = 0.043), as well as a significant reduction in inhaled bronchodilator intake (p = 0.008).”
Lung function unchanged; Why?

Preventer medication was halved in the BBT. Unrealistic to expect an improvement to lung function while at the same time reducing preventer medication.

Law of diminishing returns. The Buteyko group are under conventional care so it is reasonable to expect that medication has already improved lung function.
The British Thoracic Society recently upgraded the Buteyko Method to "B" classification indicating that there are "high quality systematic reviews of case control or cohort studies" and "High quality case control or cohort studies with a very low risk of confounding or bias and a high probability that the relationship is causal."
Breathing exercises for asthma: panacea or placebo?
Mike Pearson. Thorax 2007;62;1033-1034

The mantra for managing asthma in the 1980s became “asthma is an inflammatory condition- prescribe an inhaled steroid”. While the emphasis on inflammation dominated research and treatment plans, it was easy to overlook other demonstrable means of inducing bronchospasm.

Exercise-induced asthma and voluntary hyperventilation without exercise can both induce bronchospasm in sensitive individuals with relatively little inflammation.
Contd.

Regardless of whether the answers lie in a new physiological explanation or in an understanding of psychological reactions to the presence of a disease, the finding that something has made patients feel better means we cannot ignore this challenge.

Breathing exercises for asthma: panacea or placebo? Mike Pearson. Thorax 2007;62;1033-1034