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Physiology, pseudoscience, and Buteyko

Comments by Holt and Beasley (N Z Med J. 2004;117(1188). URL: http://www.nzma.org.nz/journal/117-1188/754) concerning the Buteyko technique ignore an increasing body of evidence suggesting that the breathing re-education might have a therapeutic role in the management of lower respiratory tract disease.[1–3]

Few studies are perfect. Instead of pouring vitriol on the study[4] perhaps it might be appropriate to ask why people are able to decrease their asthma medication, not deteriorate significantly and maintain objective measures of lung function. As medical scientists, we should not become stuck in the comfort of the familiar but welcome observations that cannot be explained by current theories. They may hold the keys to new solutions.

While I personally disagree with some aspects of the Buteyko technique, perhaps it might be appropriate to briefly examine some of the Buteyko ideas.

The three basic tenets of the Buyteko technique appear to be:

- Encouraging nasal breathing.
- Slowing the respiratory rate.
- Possibly resetting arterial pCO_2 levels at a higher level through breath-holding.

Firstly, could the nose have a role in improving lower respiratory tract function? The nose is well known to have subtle influences on the lower respiratory tract thought to be due to the warming, filtering, and humidification of the incoming air. Patients, who have their jaws wired forcing them to breathe largely through their noses, not only have increases in residual volume (23%), functional residual capacity (13%), and total lung capacity (5%) but their PaO_2 also increases by 8%.[5] The reasons for such a change have been subject to debate; however, in asthmatics, the increased resistance provided by the nose may affect closing volumes and thus reduce the extent of physiological shunt.

Secondly, does changing the respiratory rate influence asthma? It might change the pattern of breathing. In my opinion, a deficiency of the Buteyko technique as opposed to yogic breathing techniques[6] is that it does not emphasise the importance of diaphragmatic breathing.

West documents that the upper 7% slice of lung brings in 4 ml of oxygen per minute as opposed to a lower 13% volume slice bringing in 60 ml of oxygen per minute. The oxygen cost of quiet breathing using the diaphragm is less that 5% of total oxygen consumption—whereas, in voluntary hyperventilation, it can increase to up to 30% of total oxygen consumption.[7] It makes physiological sense to breathe slowly using your diaphragm.

Most respiratory physiology books teach that expiration relies totally on the elasticity of the lung and chest wall. Careful observation proves them wrong. If you lie flat and breathe out the diaphragm lengthens eccentrically. If you lie flat, and breathe in and

out slowly and then relax after a normal inhalation, you will feel the air whoosh out faster, proving that some tension is held in the diaphragm during supine exhalation.[6]

There is also a braking contribution from the laryngeal musculature. Weightlifters build up muscle strength more quickly through slow eccentric contraction. A slow breathing pattern might have a role in the maintenance of diaphragmatic strength.

Changing your breathing pattern to a slow abdominodiaphragmatic breathing pattern may also influence the sensation of dyspnoea[8] and the need to use bronchodilators. There is a very poor correlation between measures of pulmonary function and dyspnoea. The sensation of dysnoea correlates best to changes in respiratory rate, an increase in the inspiratory component of the respiratory cycle and the use of accessory respiratory musculature particularly the sternomastoid muscle. Bearing in mind the high prevalence of hyperventilation syndrome amongst asthmatic patients some asthmatic patients might be better off focusing on their breathing pattern before they resort to using bronchodilators.

The third puzzling question is whether it is possible to reset central pCO_2 receptors so that your arterial pCO_2 runs at a higher level. West teaches that the limbic system is able to alter the pattern of breathing.[7] The limbic system would also appear to have the ability to independently override the pontomedullary respiratory centre, which maintains normal pCO_2 homeostasis. The ventilatory response to CO_2 is significantly lower in yoga practitioners and their end tidal pCO_2 (as a measure of arterial pCO_2) is significantly higher (3.7 mmHg).[9]

In contrast, people who are prone to anxiety attacks would appear to have lower arterial pCO_2 levels (5 mmHg on average) as compared to controls and their pCO_2 receptors are more responsive to changes in arterial pCO_2.[10] The physiological significance of changing the settings of central pCO_2 receptors remains to be determined.

Like all good studies, the Gisborne studies raises more question than it answers. The challenge is not to dismiss it out of hand but to extract from the Buteyko technique answers to our patients' problems. As an ear nose and throat specialist, my knowledge of the lower respiratory tract is limited, but hopefully this letter offers some potential avenues that could be explored.

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