Obstructive sleep apnoea (OSA) is a sleep disorder where repeated upper airway obstruction during sleep leads to a decrease in blood oxygen saturation and disrupted sleep. Current treatment options include oral appliances, surgery, and/or the use of a continuous positive airway pressure (CPAP) machine. However, breathing retraining with the Buteyko Institute Method (BIM) is a safe, effective and convenient approach to OSA that could help eliminate the need for surgery or CPAP. This paper examines the role of breathing retraining in OSA and presents a case study to illustrate its effectiveness.

By Mary Birch

Obstructive sleep apnoea and breathing retraining

About the author
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Introduction

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Hyperventilation, OSA and breathing retraining

During his medical studies in 1953, Russian doctor, Professor Konstantin Buteyko, was asked to do a project on breathing. Through his research, Professor Buteyko concluded that hyperventilation or over-breathing caused many conditions, including asthma and OSA.

Until his death in 2003, Professor Buteyko dedicated his life to researching and developing breathing retraining techniques. The method Professor Buteyko developed spread internationally in 1991, and is now used throughout the world for a number of conditions.1,2

OSA profile

In obstructive sleep apnoea, the muscles of the soft palate relax during sleep, leading to oro-pharyngeal and uvular collapse, causing obstruction of the airway and a drop in oxygen saturation. OSA is characterised by the periodic cessation (apnoea) or reduction (hypopnoea) of airflow during sleep.3 Apnoea is defined as the total cessation of airflow at the nose and mouth, lasting at least 10 seconds, associated with a decrease in oxygen saturation of 4% or more. Hypopnoea is generally taken to mean a greater than 50% reduction in airflow lasting 10 seconds or more, associated with a 4% or greater fall in oxygen saturation.4 However, the difference between the two is of little clinical importance since both events have similar pathophysiology.

There is no clear consensus in medical literature as to why OSA occurs, although it is thought to be associated with ageing, obesity or airway anatomy. Both children and adults, however, can suffer from OSA, and many people who do are of normal weight.

Although the cluster of symptoms which occur with sleep apnoea has been described in medical literature for thousands of years, it is only in the last three decades the significance of this sleep disorder has begun to be recognised, according to a National Health and Medical Research Council report.5

Constant reductions in oxygen saturation and wakening or semi-wakening from sleep lead to daytime symptoms associated with lack of quality sleep. People with OSA find they are constantly tired, irritable, sleepy during the day, lack concentration and suffer from headaches.

Dysfunctional breathing and OSA

Hyperventilation is considered to be the fundamental cause of OSA, according to Professor Buteyko’s theory. He argues the apnoeas or pauses in breathing which occur in OSA are the body’s defence mechanism against the excessive loss of carbon dioxide due to hyperventilation and consequent hypoxia (see illustration).
Hyperventilation

When a person hyperventilates, they exhale excessive amounts of carbon dioxide (CO₂). Lowered CO₂ levels in the body are critical in many conditions.

The optimal level of CO₂ in the air sacs is around 6.5%. If the CO₂ level falls below this due to hyperventilation, there is a gradual alkaline reaction in the lungs.

When alveolar CO₂ is lowered, this gradual alkaline reaction in the lungs is carried through to the blood and total blood CO₂ will also decrease.⁶

Oxygen (O₂) in the blood is carried by means of a haemoglobin (Hb) molecule. When CO₂ is low due to over-breathing, O₂ is bound tighter than normal to the Hb molecule due to a chemical bond. For this bond to loosen, CO₂ levels need to increase and the blood pH needs to become less alkaline.

As blood pH decreases, the Hb/O₂ bond decreases in strength, hence more O₂ is available to the tissues. Therefore, when CO₂ levels rise to normal, O₂ is more readily released from haemoglobin and it can then oxygenate the body and maintain homeostasis.

This phenomenon, called the Bohr effect, the Verigo-Bohr effect, or oxygen-haemoglobin disassociation curve, is described in standard physiology textbooks.

A case study

When John, (not his real name) a 44 year-old man, attended the Buteyko Breathing Centre in Melbourne, his medical history included asthma from childhood and OSA of 18 months duration. His symptoms included shortness of breath, frequent deep breaths, snoring, frequent waking at night sweating, irritability, lack of concentration, and abdominal bloating.

John’s medication was Seretide 250/50, one puff at night (an asthma medication consisting of fluticasone and salmeterol). Sleep studies taken 18 months previously indicated John suffered from severe OSA. He was prescribed a CPAP machine, but found it was not effective in controlling the OSA symptoms.

John’s doctor had also mentioned the possibility of surgery to alleviate his OSA. Despite using the CPAP machine, John found his condition was getting worse so he only used the CPAP intermittently.

Assessment

When John first attended the breathing centre, he displayed signs of sleep deprivation. His speech was very rapid and incoherent at times, he was obviously tired and distressed, unable to concentrate or listen effectively, and at times appeared agitated.

He was of lean build and had never been overweight. John’s initial pulse was high (at 96) and his respiration rate was 20 per minute.

There was also evidence of over-breathing, ie. mouth-breathing constantly, taking large breaths while talking, breathing loudly at times, sighing, yawning, shoulders raised, and breathing from the upper chest (not from the diaphragm).

BIM breathing retraining

Following an explanation of what was involved, John enrolled to attend a Buteyko breathing retraining course of five consecutive daily sessions of 90 minutes per day, plus follow-up.

John’s course included breathing techniques to unblock the nose, promote nasal breathing and control symptoms (eg. when waking at night, when feeling short of breath or when having asthma symptoms during the day), and guidelines and techniques to enhance sleep quality and to improve breathing while performing daily living activities and exercising.

John was also asked to perform a series of breathing exercises three times per day. These exercises, totalling around 15 minutes per session, included breathing pauses and other techniques to reduce breathing and thereby retain CO₂.

John was asked to record his results on a daily diary sheet, and to list any symptoms, plus medication used, including use of the CPAP.

John’s progress

John’s sleep and asthma gradually improved from day two of the course. On day three, he was still having sleep problems but felt a little better.

By day four, his breathing had improved considerably. He was sleeping much better and the night sweats had stopped. He was using the CPAP for part of the night only and stated that he was feeling much better.

His concentration was also much improved as were his asthma symptoms. By day four he had seen his physician and the Seretide had been withdrawn.

He was now taking Flixotide (fluticasone) 250mcg once daily and not requiring
any bronchodilators. This reduction in asthma medication is consistent with results obtained in the first clinical trial of the Buteyko method in the western world, and recently confirmed by a further study.1,9

By day five, John was looking well and had used the CPAP for only two hours the previous night.

One month later, John attended the centre for review. He looked very much improved. His pulse rate was now down to 64 and he was sleeping well throughout the night without the CPAP.

His breathing had improved significantly, his tiredness had gone, and his speech patterns and concentration appeared normal. He was advised to continue the breathing exercises three times per day.

After five months, John phoned, rather elated, to say: ‘I’ve had repeat sleep studies done and I don’t have apnoeas at all. Based on the results, I don’t need surgery. The snoring has gone from loud to mild. I used to wake myself up with snoring… that’s all gone. My oxygen saturation is up to 97%; before it was around 87%.’

Reviewed by telephone two years after the course, John says his current health status is good. He sleeps well and does not use a CPAP machine. He does not do the breathing exercises except when his nose blocks occasionally, generally when the weather is cold and dry.

He still continues to use one puff of Flixotide (very low dose) daily for ‘security and insurance’.

Incidence, morbidity and mortality

In Australia, a 1995 clinical study found 24% of men and 6% of women over 55 had clinically significant OSA.9

Studies have also shown that OSA has been implicated in a number of clinical conditions, including pulmonary and systemic hypertension and cor pulmonale.

There have also been links between daytime sleepiness associated with OSA and road accidents. In addition, a number of studies have suggested that OSA is associated with premature mortality, largely as a result of vascular diseases.10

OSA Diagnosis

Diagnosis of OSA can be made by monitoring an individual’s sleep patterns overnight at a sleep clinic. The number and duration of apnoeas and hypopnoeas, stage of sleep (REM/non-REM), heart rate, oxygen saturation, position and limb movements are monitored electronically during sleep, and a statistical report is provided.

From these details, it can be determined whether the person suffers from OSA, and the severity of their condition. People with severe OSA can have over 20 episodes of apnoea or hypopnoea per hour.

Treatment options

Current treatment options for OSA generally involve surgery or nightly use of oral appliances (such as mandibular advancement splints), or CPAP machines.11

A Cochrane Review of trials of drug treatments for OSA concluded the data available do not support the use of drugs as a therapy for OSA.12

Further, since the advent of nasal CPAP, surgical options for OSA are now less common.13

Most commonly, moderate to severe OSA is treated with the use of a nasal CPAP machine, which forces air through a nasal mask, thus maintaining the person’s airway while sleeping.

However, there are problems with compliance in using nasal CPAPs, as there are numerous side-effects.14 These include dry nose, mouth or throat, rhinitis, noise, nasal congestion, sore eyes, headache, mask discomfort and chest discomfort.15

The machines require cleaning and maintenance, users and partners may find them noisy and intrusive, and people have the inconvenience of having to take the machine with them when away from home.

A Cochrane review of clinical trials for surgery for OSA found that no completed trials were identified to compare surgical interventions for OSA with other surgical or non-surgical interventions or no intervention.16

This review concluded there was an urgent need for high quality randomised controlled trials to be carried out in the field of surgery for OSA, and that more research should be undertaken to identify and standardise techniques to determine the site of airway obstructions.

Conclusion

Breathing retraining with the Buteyko Institute Method offers a safe, effective, convenient, and more appealing option for people with OSA, which can eliminate the need for surgery, oral appliances, or CPAP.

The benefits of breathing retraining have become apparent from many years of clinical practice in teaching BIM courses to people with OSA. Unlike other alternatives, there are no side effects with breathing retraining.

Trials of breathing retraining have demonstrated benefits in asthma, however, there are currently no trials available for OSA.

Further information:

www.butey kobayside.com
www.bibhs.org

References

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9 NHMRC, op. cit. p.2.
10 NHMRC, op. cit. p.3.
11 Ibid.
13 NHMRC, op. cit. p.36.
14 NHMRC, op. cit. p.4.
15 Ibid.

Correction

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Associate Professor Paul Desmond, MBBS FRAC and co-author of Hepatitis C. A medical and social diagnosis (Clinical Update 73, ANU July 2004, pp.23-25) is director of gastroenterology, St Vincent’s Hospital, Melbourne, Victoria. The ANU apologises for its error.