buteyko
breathing clinics

20 Arthur Street
Freemans Bay
Auckland 1011
New Zealand
+64 9 360 6291
info@buteykobreathing.nz
www.buteykobreathing.nz

HYPERVENTILATION AND CARDIOVASCULAR HEALTH

The Buteyko Clinic breathing retraining programme

The Buteyko Clinic offers a health education programme based on the Buteyko Institute Method. [1] Clients attend a minimum of five 60 to 90-minute breathing training sessions where they learn breathing awareness and are taught the appropriate breathing exercises for their condition.

The programme starts with an initial assessment and breathing exercises are tailored to the individual to assist them establish a more normal breathing pattern. Dysfunctional breathing patterns include mouth breathing, upper chest breathing, absence or restriction of diaphragm breathing, hyperventilation (chronic over-breathing), erratic or irregular breathing, and poor posture.

The programme is designed to assist people to improve the way they breathe. It consists of a series of lectures and practical training sessions including breathing exercises. Clients are taught specific breathing exercises that need to be practised daily for a minimum of six weeks. The breathing exercises have two purposes:

1. To help relieve symptoms associated with breathing-related disorders including but not limited to: asthma, bronchitis, COPD, chronic cough, hay fever, sinusitis, nasal congestion, sleep apnoea, snoring, primary hypertension and anxiety/panic attacks.

2. To normalise each aspect of the breathing pattern (rate, rhythm, volume, mechanics, use of the nose), for all situations (awake, asleep, at rest and, during eating, speech and exercise). [2]

The specific goal of breathing retraining is to achieve physiologically normal breathing. As a client's breathing improves, and in order to achieve this goal, it may be helpful to review prescribed medications with the client's referring GP or specialist. The following sets out these suggestions including the rationale and supporting evidence.

Hyperventilation, hypocapnia and cardiovascular health

Breathing in excess of normal metabolic requirements causes a loss of carbon dioxide (CO_2) from the blood, leading to hypocapnia. Hypocapnia alters oxygenation of the heart and cardiac rhythm.[3]

Reduced arterial CO_2 affects cardiac functioning in three ways. Firstly, it reduces blood flow to the heart. [4] [5] [6] Secondly, the bond between red blood cells and oxygen is strengthened, leading to reduced delivery of oxygen to the heart muscle (Bohr Effect).[5] Thirdly, there is increased oxygen demand on the heart attributable to myocardial contractibility [7] and blood vessel constriction. [8] [9]

An experimental study on 9 human subjects showed that voluntary over-breathing that reduced arterial partial pressure of CO_2 from normal levels of 40 mmHg to 20 mmHg, increased coronary blood vessel resistance by 17% and decreased coronary blood flow by 30.3%. [10]

Rutherford et al. (2005) noted that, "During voluntary hyperventilation in un-anaesthetised humans, hypocapnia causes coronary vasoconstriction and decreased oxygen supply and availability to the heart. This can induce local epicardial coronary artery spasm in susceptible patients." [11]

In a 1990 study of 100 patients and 25 control subjects it was noted that, "Hyperventilation causes hypocapnia and respiratory alkalosis and thereby predisposes to coronary vasoconstriction and cardiac arrhythmia". [12]

Hashimoto et al. (1990) presented the effects of CO_2 on oxygenation of the heart during haemorrhagic shock under conditions of normocapnia (normal CO_2), hypocapnia (low CO_2) and hypercapnia (high CO_2). Hypocapnia was found to decrease blood flow and oxygenation of the heart, whereas hypercapnia increased it. [13]

Similar results were found by Tateyama et al. (1995) who investigated the effects of increased arterial CO_2 ($PaCO_2$ not equal to 60 mmHg) on heart tissue oxygen tension and metabolism in anaesthetised dogs. The authors found that hypercapnia increases blood flow and oxygenation of the heart. [14]

Myocardial infarction (heart attack)

Myocardial infarction, otherwise known as a heart attack, occurs when blood flow that brings oxygen to the heart is severely reduced or cut off altogether. The stoppage of blood resulting in oxygen starvation and damage to part of the heart muscle is a heart attack.

Heart attacks often occur during or following physical exercise or emotional stress. Both increase breathing volume, and when breathing volume is greater than metabolic needs, carbon dioxide is removed from the lungs and, hence, the blood, resulting in reduced blood flow and oxygenation of the heart.

In a paper entitled "Hyperventilation and Myocardial Infarction," Chelmowski et al. (1988) wrote, "In addition to causing peripheral and cerebral vasoconstriction, hyperventilation has also been shown to cause diminished coronary blood flow. Oxygen delivery to the myocardium and other tissues is further decreased in alkalosis because of increased haemoglobin oxygen affinity according to the Bohr effect". [15]

In the ensuing sections, we investigate whether patients with chronic heart disease breathe more heavily than normal, whether hyperventilation during resuscitation can adversely affect outcomes, and whether breathing exercises aimed at correcting breathing volume may reduce the risk of further cardiac problems.

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Do patients with chronic cardiac failure hyperventilate?

In a study of 20 patients at the Royal Victoria Hospital, Belfast with moderate to severe chronic cardiac failure, ventilation was noted at 15.3 to 18.5 litres per minute. In addition, researchers observed that patients with chronic cardiac failure have higher breathing volume per minute during rest and physical exercise compared with normal subjects. (Normal breathing volume at rest is 4 to 6 litres per minute.) Higher breathing volume was observed to contribute to breathlessness during exercise, with a reduction to a minute volume level corresponding to an improvement in the degree of breathlessness. [16]

A study of 30 patients with stable chronic heart failure at the National Heart Hospital London, Buller et al. (1990) found increased ventilatory response (heavier breathing than normal) to exercise and that the size of this abnormality was related to the severity of chronic heart failure. Furthermore, there was a close linear correlation between breathing volume and the rate of CO_2 production during exercise, which in turn was closely related to the severity of chronic heart failure. In other words, the heavier the breathing volume relative to exercise, the greater the severity of chronic heart failure. [17]

This finding is supported by a number of researchers who found an excessive ventilatory response to exercise as characterised by an increase in the slope of the relationship between ventilation and CO_2 production. [18 19 20]

In a paper entitled "The Development of Hyperventilation in Patients With Chronic Heart Failure and Cheyne-Stokes Respiration," which was published in the journal Chest, 132 patients were studied to analyse the relationship between daytime respiratory and cardiac function in patients with compensated chronic heart failure with and without periodic breathing or Cheyne-Stokes respiration. [21]

The authors observed that patients with breathing problems had increased breathing volume per minute and reduced arterial CO_2. Furthermore, "patients with problem breathing or Cheyne-Stokes respiration have greater impaired cardiac function". They concluded, "Patients with daytime breathing disorders have chronic hypocapnia." [21]

These studies clarify the relationship between over-breathing and resultant cardiac problems in susceptible individuals.

Hyperventilation during cardiopulmonary resuscitation (CPR)

While numerous studies show that hyperventilation causes reduced blood flow and oxygenation of the heart, other studies show that hyperventilation during cardiopulmonary resuscitation is detrimental to survival.

In a study of 12 patients, the minute volume was found to be 13 litres. The researchers noted that "hyperventilation was common, and that the persistently high airway pressures are likely to have a detrimental effect on blood flow during CPR." [22]

In another study by the Department of Emergency Medicine, Medical College of Wisconsin, USA published in the journal Critical Care Medicine, researchers tested the hypothesis that excessive breathing during CPR causes a significant decrease in coronary perfusion pressure and an increased likelihood of death.

In 13 adults receiving CPR, the average ventilation rate was 30 breaths per minute (the normal rate is 12). None of the 13 adults survived. A study was conducted to investigate the number of breaths per minute and survival rate during cardiac arrest in pigs. Three groups of seven pigs were treated with 12 breaths per minute, 30 breaths per minute and 30 breaths per minute plus addition of supplemental CO_2. Survival rates in the groups were as follows: six out of seven pigs treated with 12 breaths per minute, one out of seven treated with 30 breaths per minute and one out of seven pigs treated with 30 breaths per minute plus CO_2.

The authors commented "despite seemingly adequate training, professional rescuers consistently hyperventilated patients during out-of-hospital CPR," and that "additional education of CPR providers is urgently needed to reduce these newly identified and deadly consequences of hyperventilation during CPR." [23]

Hyperventilation as a test for diagnosis of coronary artery spasm

The hyperventilation provocation test involves patients breathing excessively for a certain period to lower CO_2 tension to produce symptoms. A weight of evidence exists to support the role of hyperventilation as a diagnostic tool for cardiac disease. [24] [25] [26]

Researchers from the Division of Cardiology, Kumamoto University School of Medicine, Japan investigated the hyperventilation test as a clinical tool to induce coronary artery spasm (narrowing of blood vessels to the heart). The study involved 206 patients with coronary spasm and 183 patients without angina at rest (non-spasm). Each patient performed hyperventilation for six minutes. Of the spasm group, 127 showed positive responses to the test, including electrocardiographic changes attributable to reduced blood flow. No one in the non-spasm group showed any ischemia (restriction of blood flow). When clinical characteristics were compared; high disease activity, severe arrhythmias and multi-vessel spasm were significantly higher in the hyperventilation test positive patients than in the negative patients (69% vs. 20%). The authors concluded that "hyperventilation is a highly specific test for the diagnosis of coronary artery spasm, and that hyperventilation test-positive patients are likely to have life-threatening arrhythmias during attacks and multi-vessel spasm." [26]

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Breathing exercises for cardiac rehabilitation

Breathing exercises aimed at normalising breathing volume provide therapeutic benefits to recovering cardiac patients.

A 2004 study examined 55 men two months following a heart attack. Assessment of acid base balance and capillary blood gases were performed at rest and repeated after ten days and six months. Exercises to correct breathing were taught over five days, with monthly meetings for six months.

Twenty patients were randomly selected for breathing correction while 10 patients made up the control group. Following the practice of breathing exercises, ventilation per minute significantly decreased from 18.5 to 9.8 litres. (Note that normal breathing volume per minute is 4 to 6 litres.) CO_2 increased from 33.2 mmHg to 44.2 mmHg (normal CO_2 is 40 mmHg.)

Based on improvements to minute volume and respiratory function, the authors recommended that breathing retraining could be an additional rehabilitation measure after heart attacks. [27]

A 1992 study enlisted 41 subjects to practise breathing exercises for reversing chronic hyperventilation. 16 of the subjects were diagnosed with mitral valve prolapse. Results demonstrated the effectiveness of breathing retraining on modifying respiratory physiology and reducing functional cardiac symptoms in subjects with signs associated with hyperventilation syndrome. Results demonstrated significantly higher end-tidal CO_2 levels and lower respiratory rates when compared with pre-treatment levels measured three years earlier. The authors concluded, "Breathing retraining has lasting effects on respiratory physiology and is highly correlated with a reduction in reported functional cardiac symptoms." [28]

A call to action

Research and clinical evidence indicate that over-breathing is a significant factor in cardiovascular disorders. There is a clear relationship between over-breathing and cardiac arrest and heart attack. Cardiac symptoms are commonly triggered in clinical settings with voluntary hyperventilation tests. Detrimental outcomes involving CPR with hyperventilation have been identified. Higher breathing volumes are recognised among some cardiac patients and the positive effect from breathing exercises designed to reduce minute volume post heart attack are well documented.

In fifteen years of clinical practice I have found that a need exists to improve awareness of physiologically normal breathing in the management of respiratory, cardiovascular, sleep and anxiety/stress-related disorders. Awareness of what constitutes physiological normal breathing also has important implications for elite athletes and for all sports.

Adapted and updated from an original article by Patrick McKeown, Buteyko Ireland.
Glenn White
Buteyko Breathing Clinics
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