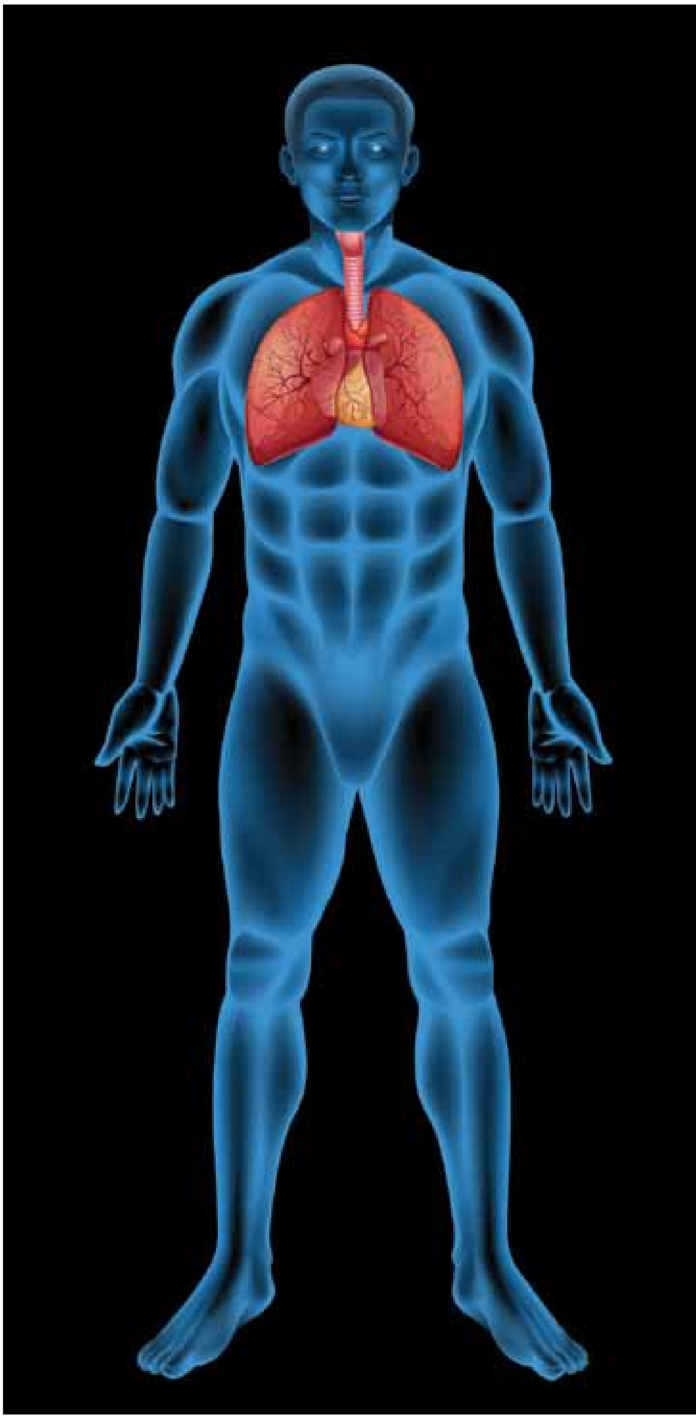
Breath, buffers and performance



IT'S EASY TO UNDERSTAND THE IMPORTANCE OF BREATHING FOR PHYSICAL PERFORMANCE, BUT WHAT ARE THE EFFECTS OF OVER-BREATHING? **DR DAVID BEALES** EXPLAINS THE PHYSIOLOGY OF BREATHING, CARBON DIOXIDE CONTROLS, ACID-ALKALINE BUFFERING AND ELECTROLYTES.

As I sit here, preparing to exercise my brain to write this article, I ask myself if the way in which I breathe is important for my clarity of thought and my ability to be energetic with good health and performance. This article aims to show just that and to explain some of the science, showing that how we breathe profoundly affects both health and performance.

Perhaps you will, as you read this introduction, pause quietly for several breaths and consciously ask yourself; 'how am I breathing?' Notice the in-breath, where air is entering – through the nose, or mouth, or both? If you are breathing through the nose, the air is warmed by contact with the lining of the nose. Under the lining, close to the surface, circulating cells are carrying oxygen to the tissues through small blood vessels called arterioles. These arterioles have smooth muscle in their walls, which responds to chemical triggers to tighten or relax.

How are you placing the breath?

As you breathe in, are the outer ribs moving out and the soft belly rising? Are the shoulders rising and most of the movement taking place in the upper chest? If it is the former scenario, the diaphragm (the largest involuntary muscle in the body) is working and moving. As you can see in Diagram 1, it is wrapped around the ribs and attached to the back. It acts like bellows, drawing air into the lungs, where oxygen is exchanged for carbon dioxide.

In days gone by, carbon dioxide was written off by physiologists as a waste product and of no importance. As a doctor specialising in behavioural medicine, I now realise how mistaken this commonly-held view was. Managing normal carbon dioxide levels is the key to maintaining buffer reserves. My aim is to explain why, when everyday stress or overtraining results in over-breathing, buffer reserves, which are controlled by the kidneys, reduce in the body. The individual is then unable to buffer the acidic products of muscular exertion; mainly lactic acid. This causes the athlete to 'hit the buffers' much earlier than they would want and as a consequence, suffer from a drop in performance.

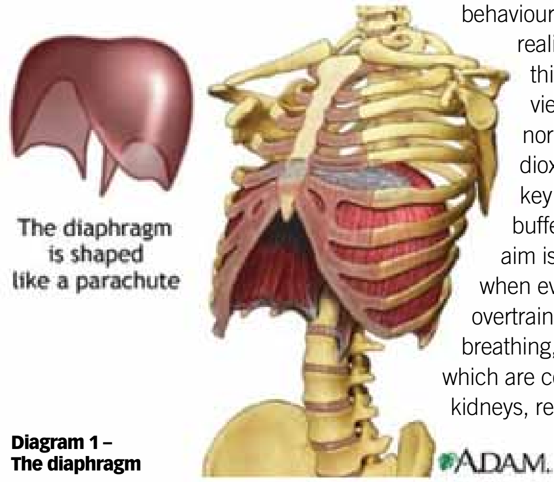


Diagram 1 – The diaphragm

Joe's story

While in Florida, attending the funeral of her father, already anxious and worried, Joe was bitten by a

"For each 1mm drop in carbon dioxide levels from a normal baseline of 40mmHg, there is a 2% reduction in transfer of oxygen to the tissues."

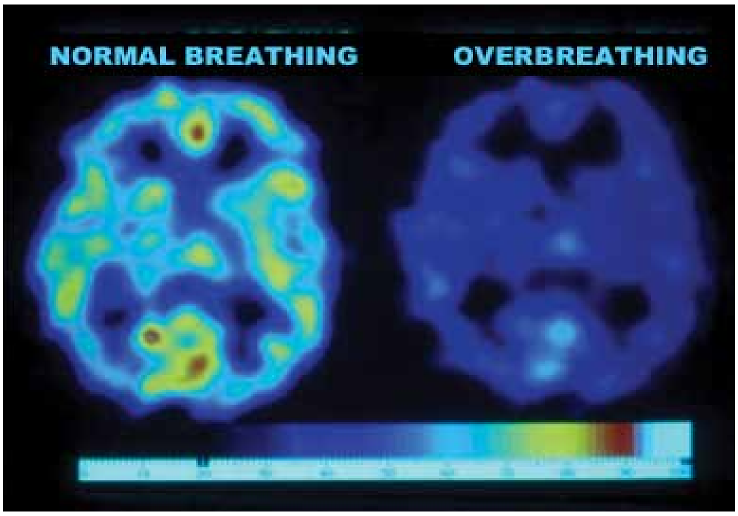


Diagram 2 – reduced carbon dioxide causes hypoxia

poisonous insect on her lower leg. The bite became infected. She was on her own and felt increasingly isolated and fearful. Although she was treated well, she experienced increasing pain and anxiety, with difficulty in concentrating, and decided to return home earlier than planned. On the flight back to the UK, she developed chest pain and breathlessness. As a result, the plane was diverted to Iceland, where she was treated as an emergency. Pulmonary embolus and other diseases were excluded. She had normal oxygen levels. She came back to England and the chest pain, breathlessness and marked dizziness continued.

She was admitted to hospital, and although investigated for ten days, no diagnosis was made. When I saw her, however, she had a very low resting carbon dioxide level at 25mm of mercury partial pressure (mmHg): the norm is 40mmHg in the out-breath. I also asked her to complete a validated questionnaire for chronic hyperventilation and her Nijmegen Score was 49: to put this into context, a normal upper level score is 23 (1). I also obtained the hospital discharge notes and saw that her bicarbonate level had been reduced at 20meq/l and her potassium level was reduced at 3meq/l. I made a diagnosis of chronic over-breathing, resulting in reduced carbon dioxide levels (hypocapnia). When I asked her to walk gently, while monitoring the carbon dioxide levels in her out-breath with capnography (a method of measuring carbon dioxide in the out-breath), I found that she did not raise her carbon dioxide levels and she quickly became breathless.

With the aid of a breathing re-training programme, using biofeedback of her carbon dioxide levels, combined with cognitive behavioural coaching, Joe recovered completely within eight weeks.

So, what was happening in Joe with her hypocapnia (over-breathing/hyperventilation)? Over-breathing means that:

- Too much carbon dioxide is being moved out of the lungs, resulting in reduced levels in tissues for our metabolic needs.
- The breathing style is linked with upper chest breathing without the full engagement of the diaphragm.
- This can be measured as reduced carbon dioxide in the blood and out-breath.

How does a low carbon dioxide level in Joe's findings lead to:

- An inability to exercise without breathlessness?
- The inability to raise her carbon dioxide levels with exercise?
- Reduced resting carbon dioxide levels?
- Reduced bicarbonate (HCO_3) levels?
- Reduced electrolyte concentration of potassium – and if measured, the likely reduction of other minerals with a positive electronic charge such as calcium, magnesium, chromium, zinc and other trace elements?
- Brain fog and difficulty in concentrating, as well as many other symptoms in the 16 questions of the Nijmegen Questionnaire?

Low carbon dioxide levels result in oxygen starvation in tissues – i.e. there is a reduction in the release of oxygen to the cells. The binding affinity of haemoglobin to oxygen is directly correlated with carbon dioxide levels. For each 1mm drop in carbon dioxide levels from a normal baseline of 40mmHg, there is a 2% reduction in transfer of oxygen to the tissues. A Pulse Oximeter, measuring oxygen saturation in the blood, shows how much oxygen is attached to haemoglobin, and will be normal at around 98% saturation. The effect of reducing carbon dioxide levels from 40mm to 20mmHg is a 40% fall in oxygen release, and is shown in the adjacent functional magnetic scan (see Diagram 2).

As carbon dioxide levels drop from 40 to 20 mmHg, it results in a 40% reduction in oxygen uptake.

Is it a surprise that the whole mind/body system is profoundly disturbed by over-breathing? Joe's brain fog and difficulty in concentration are explained by a reduced transfer of oxygen to her brain, as well as the cerebral tightening of smooth muscle in the walls of her millions of arterioles, resulting in reduced flow.

What about Joe's breathlessness, low bicarbonate levels and reduced electrolytes and minerals?

Reduced carbon dioxide levels precipitate diverse changes:

- **Blood vessels constrict in the brain and the heart.**
- **Blood flow to the brain is reduced by as much as 50%.**
- **Oxygen and glucose supplies to the brain are radically reduced.**
- **Brain cells become more excitable and are more likely to be anaerobic.**
- **Smooth muscles constrict around the bronchioles, as well as the placenta and gut.**
- **Sodium and potassium deficiencies (electrolyte imbalance) may develop.**
- **Calcium-magnesium ratios in muscles become imbalanced.**
- **Buffers (bicarbonates) required for regulating acids (e.g. lactic acid are depleted.**
- **The release of oxygen and nitric oxide by haemoglobin is inhibited.**

- **Increased airway resistance occurs and mouth breathing further reduces airway resistance, as nitric oxide is not released through the nasal lining of the nose.**

Mechanism of buffer depletion

Henderson-Hasselbalch's Equation

$$pH = \frac{HCO_3 \text{ (bicarbonate)}}{CO_2 \text{ (carbon dioxide)}}$$

If carbon dioxide levels are reduced as a result of over-breathing, in order to maintain a neutral pH, the compensatory body mechanism is to release bicarbonate from the kidneys. For this process to occur there is a delay, depending on the individual; from eight to 48 hours. As you look at the Henderson-Hasselbalch's equation, you will see that if the CO_2 levels are reduced and there is a delay in bicarbonate excretion, the pH will rise, resulting in a state called respiratory alkalosis. The body's pH level needs to be maintained within the narrow range of 7.35 to 7.45. So, if the pH rises, the body/mind goes into a catabolic state, with physiological hyper-arousal.

If the breathing style remains one of over-breathing, resulting in too much carbon dioxide being moved out of the body for our metabolic requirements, then bicarbonate continues to be moved out of the kidneys and levels become depleted – as in Joe's reduced bicarbonate level. Since bicarbonate ions have negative charges, as it is removed from the body, it also drags minerals with a positive charge along with it. These minerals include magnesium, calcium, zinc, sodium and trace elements like chromium. Several of these minerals make up electrolyte salts, which are commonly supplemented by athletes.

So with reduced bicarbonate levels within the body, the acidic products of exercise, such as lactic acid, cannot be buffered effectively.

Because of Joe's chronic over-breathing, I found very low levels of carbon dioxide in her out-breath during easy exercise. As a result, her buffering capabilities were very low, including depleted levels of bicarbonate and certain mineral salts.

In an average athlete, incremental exercise using a step protocol, while measuring End Tidal carbon dioxide through a nasal cannula with the mouth closed, gives a good measure of buffer reserve. As the athlete reaches their limit of tolerance and moves into buffer reserve depletion, the measurement of carbon dioxide shows a 'point of acidaemia', where the level of carbon dioxide starts to fall. The baseline is normally around 40mmHg if there is no history of chronic over-breathing. The mechanisms are explained further in the online article (3) and textbook (4).

My advice to athletes in training is to stop pushing exercise beyond the limit of tolerance of their muscles: this will usually be signalled by more than normal tiredness and fatigue. Excessive fatigue is the signal that you are reaching your point of acidaemia, which signals buffer depletion. There is undue strain on the buffering system and de-conditioning of muscle strength, which over time is likely to result in 'overtraining'.

This is not to say that incremental exercise will not steadily improve muscle strength. However, overtraining and reduced performance is likely to occur if, in training, the athlete consistently pushes their body into acidaemia.

If, on the other hand, the training is steady within these constraints, when faced with a final race effort when anaerobic and buffer-depleted muscles are pushed beyond a limit of tolerance, there is likely to be potential for optimal performance. After the race, there is a need to recover with good sleep, reduced stress and a releasing of physiological over-arousal. Good breathing practices can also help restore a return to physical well-being and psychological self-esteem when performance has been disappointing.

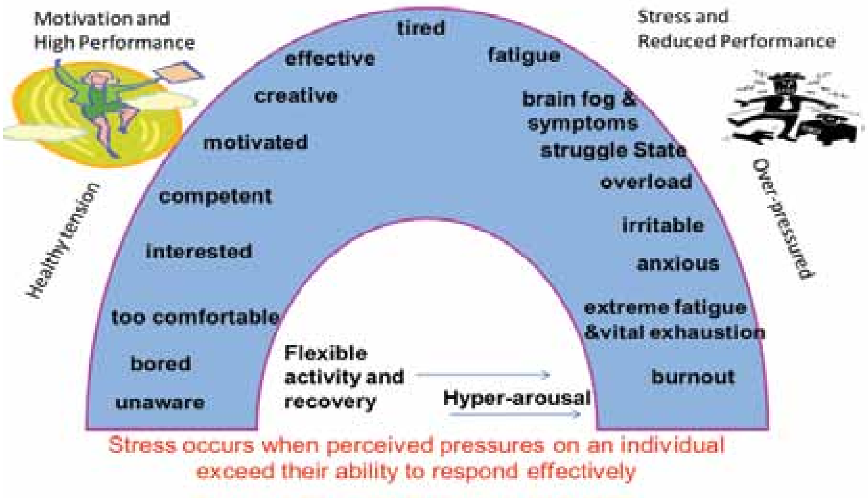


Diagram 3 – Maintaining performance without hyper-arousal/stress

Stress and performance

In Joe's story, we can see how anxiety and fear contributed to her over-breathing and buffer depletion. The link between stress and over-breathing is confirmed by science (4) and suggests that 'breathing is exquisitely sensitive to stress'. A fuller explanation of the connection between over-breathing and anxiety can be found in the online article that I wrote with osteopath Jonathan Nunn for Positive Health Online (3).

To perform well in sport, we need to lead balanced lives, without sustained excessive pressures. We need to keep away from the down slope of the human function curve, as shown in the above diagram. It's easy to do too much in today's world. Mindfulness training is therefore extremely helpful when our body-mind signals that we are moving into overload; mindfulness enables us to step back, thereby retaining our resilience and health. FSN

REFERENCES

1. Dixhoorn J and van Duivenvoorden H (1985). Efficacy of Nijmegen Questionnaire in recognition of the hyperventilation syndrome. *Journal of Psychosomatic Research.* 29:(2):199-206.
2. Northbridge D et al (1990). Novel exercise protocol suitable for use on a treadmill or a bicycle ergometer. *British Heart Journal.* 54:313-316.
3. David Beales and Jonathan Nunn (2010). www.positivehealth.com/article/breathing/mindfulness-and-the-breath.
4. Chaitow L et al (2001). Breathing Pattern Disorders. Churchill Livingstone.
5. Wilhelm F, Gervitz R and Roth W (2001). Respiratory Dysregulation in Anxiety. *Functional Cardiac and Pain. Behav Modif.* 25(4):513-545.

About the author

David Beales, MD, is a specialist in behavioural medicine. He is a Fellow of the College of Physicians, trained in rheumatology at Guy's Hospital, and then worked within general medicine and general practice. He is medical advisor to CNELM, where he teaches, as well as at the British School of Osteopathy. He works with individuals and small groups and also lectures and offers workshops to help maintain health and vitality, including a workshop to help therapists maintain resilience for Confer on March 22nd; www.confer.uk.com/well-being.html. He also runs workshops on 'Mindfulness and the Breath', and is giving a two day retreat on May 11th and 12th at Hawkwood College, Stroud. Details at; www.mindfulphysiology.co.uk, www.emotionalhealinguk.co.uk and dbeales@heartsandminds.fsnet.co.uk.