NORMAL BREATHING "THE KEY TO VITAL HEALTH"

NORMAL BREATHING

For Buteyko Breathing Teachers

www.normalbreathing.com

By Artour Rakhimov

Normal breathing: the key to vital health

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Normal breathing: the key to vital health

Introduction

What do we need in order to be healthy? Most people, including many medical doctors and health professionals, would probably say that a good diet, exercise, healthy environment, and proper rest are all important. Other people may add relaxation exercises, supplements, herbal remedies and other factors. Meanwhile, there is one factor, which is usually missing in typical answers. That is **normal breathing** (or breathing in accordance with existing medical and physiological norms).

Humans can live for days without water and for weeks without food. However, we can survive without breathing for not more than a few minutes. Can it be so, then, that breathing is as important as water and food? In my view, breathing is the missing link in the modern philosophy of health. We know too little about its importance and its effects on various processes, systems, and organs of the human body.

This book is written for an inquisitive reader who has keen interest in respiration, its basic theory, hyperventilation and its effects, the regulation of respiration in health and disease and breathing retraining therapies. The book will also separately describe the discoveries and practical work of Doctor Buteyko, a detailed description of his breathing method, and relevant known medical investigations and physiological experiments. Such attention to the Buteyko method seems justified to me due to its remarkable success in the treatment of various chronic health problems, which are often considered incurable.

While this book does contain scientific terms and methods, it is not required that the reader be medically trained in order to understand the information presented here.

Each chapter of this book starts with an introduction formulating the questions to be discussed. After the main text of theoretical chapters, general conclusions are suggested. Each chapter has a Q&A (Question & Answer) section and chapter-related references.

Finally, let me express my gratitude to all people whose help made the existence of this book possible. In particular, sincere thanks to Stuart B. Wiley (Canada), Paul Ryner, Carol Baglia, Roger Young, Suzanne Nicole (USA), Patrick McKeown, Anne Burns (Ireland), Carolina Gane (Holland), Elizabeth MacDomnic and Duncan Robertson (UK) for proofreading and/or valuable remarks which improved the quality of the manuscript.

Chapter 1. Scientific studies about breathing-health connection

Introduction

In this chapter we will examine what medical science has been studying during last hundred years. Our goal is to establish a relationship between breathing and both health and disease.

How should we breathe? How do sick people breathe? Is there any connection to the severity of the health problems? What has been found out about breathing of severely sick and critically ill patients? How do most people breathe when they die and before that? Are there any simple practical tests, which indicate if personal breathing is normal or not?

1.1 Minute ventilation in health and disease

What is the norm of breathing? How many litres of air per minute should we breathe while sitting at rest? The physiological norm of minute ventilation can be found in many physiological and medical textbooks. It is about 6 litres per minute (Guyton, 1984; Ganong, 1995). So, let us keep in mind this important number: **6 litres of air per minute**.

Table 1.1 summarizes information about minute ventilation at rest in different diseased states.

***One row corresponds to one medical study/publication**

Condition	Minute ventilation	N. of patients	Prevalence of CHV	Reference
Normal breathing	**6 l/min**	-	0 %	Medical textbooks
Heart disease	**15 (±4) l/min**	22	100%	Dimopoulou et al, 2001
Heart disease	**16 (±2) l/min**	11	100%	Johnson et al, 2000
Heart disease	**12 (±3) l/min**	132	100%	Fanfulla et al, 1998
Heart disease	**15 (±4) l/min**	55	100%	Clark et al, 1997
Heart disease	**13 (±4) l/min**	15	100%	Banning et al, 1995
Heart disease	**15 (±4) l/min**	88	100%	Clark et al, 1995
Heart disease	**14 (±2) l/min**	30	100%	Buller et al, 1990
Heart disease	**16 (±6) l/min**	20	100%	Elborn et al, 1990
Pulm hypertension	**12 (±2) l/min**	11	100%	D'Alonzo et al, 1987
Cancer	**12 (±2) l/min**	40	100%	Travers et al, 2008
Diabetes	**12-17 l/min**	26	100%	Bottini et al, 2003
Diabetes	**15 (±2) l/min**	45	100%	Tantucci et al, 2001
Diabetes	**12 (±2) l/min**	8	100%	Mancini et al, 1999
Diabetes	**10-20 l/min**	28	100%	Tantucci et al, 1997
Diabetes	**13 (±2) l/min**	20	100%	Tantucci et al, 1996
Asthma	**13 (±2) l/min**	16	100%	Chalupa et al, 2004
Asthma	**15 l/min**	8	100%	Johnson et al, 1995
Asthma	**14 (±6) l/min**	39	100%	Bowler et al, 1998
Asthma	**13 (±4) l/min**	17	100%	Kassabian et al, 1982
Asthma	**12 l/min**	101	100%	McFadden et al, 1968
COPD	**14 (±2) l/min**	12	100%	Palange et al, 2001
COPD	**12 (±2) l/min**	10	100%	Sinderby et al, 2001
COPD	**14 l/min**	3	100%	Stulbarg et al, 2001
Sleep apnoea	**15 (±3) l/min**	20	100%	Radwan et al, 2001
Liver cirrhosis	**11-18 l/min**	24	100%	Epstein et al, 1998
Hyperthyroidism	**15 (±1) l/min**	42	100%	Kahaly, 1998
Cystic fibrosis*	**13 (±2) l/min**	10	100%	Bell et al, 1996

Cystic fibrosis	**11-14 l/min**	6	100%	Tepper et al, 1983
Epilepsy	**13 l/min**	12	100%	Esquivel et al, 1991
CHV	**13 (±2) l/min**	134	100%	Han et al, 1997
Panic disorder	**12 (±5) l/min**	12	100%	Pain et al, 1991
Bipolar disorder	**11 (±2) l/min**	16	100%	MacKinnon et al, 2007
Dystrophia myotonica	**16 (±4) l/min**	12	100%	Clague et al, 1994

Table 1.1 Minute ventilation of patients with different health problems.

Table 1.1 comments:

- *1. There are many dozens of other medical investigations into minute ventilation of patients with chronic heart failure, which show similar results to those listed above. (The reason for this is the commonness of this health problem among people and the popularity of the stress-exercise test for heart patients among respiration researchers). Here I have quoted only the results of some typical recent studies.*
- *2. "COPD" means chronic obstructive pulmonary disease.*
- *3. Sometimes, measurement of ventilation produces results, which are smaller than in real conditions. This can happen when the test interferes with the normal breathing pattern, such as when the experiment involves the use of facial masks. Since it is harder to breathe through them, these masks reduce minute ventilation. Breathing through a mouth-piece also leads to breathing less air than in reality. Another effect is connected with body weight: people of a lighter weight need less air, as they normally have lower metabolic rates. All these effects should be taken into account when analysing experimental results. For example, in the quoted study (Bell et al, 1996) patients with cystic fibrosis had minute ventilation of 10.4+-1.4 l/min. Not only were they wearing masks during measurement, but also the average weight of these people was 56.5 kg. Hence, the quoted minute ventilation would probably be equivalent to about 15 l/min for typical adults.*
- *4. Similarly, weight should be taken into account when analysing minute ventilation of children. For example, it was reported that 12 children with epilepsy had an average minute ventilation of almost 8 l/min (Esquivel et al, 1991). Their average weight was 43 kg, which corresponds to about 12-15 l/min for adults with normal weights, therefore indicating hyperventilation. Numerous other studies also found evidence of hyperventilation in patients with this health condition.*

Note, that virtually all tested patients with chronic heart failure over-breathe.

The same was true for these limited studies in relation to diabetes, asthma, and other disorders. However, more experiments are required for these and other health problems in order to be certain about the existing links between breathing and diseases.

It is normal, that such studies can find prevalence of over-breathing in investigated subjects. A few health conditions where patients breathe less than the norm will be considered below.

Now we can conclude that many sick people breathe too much.

What about breathing rates in modern healthy subjects? This table comprises 14 published medical studies. We see that healthy subjects breathe about 6-7 l/min at rest.

Table. Minute ventilation (or minute breathing rates)
at rest in healthy subjects (14 studies)

Condition	**Minute ventilation**	**N. of subjects**	**Reference**
Normal breathing	**6 l/min**	-	Medical textbooks
Healthy subjects	**7.7 ± 0.3 l/min**	19	Douglas et al, 1982
Healthy males	**8.4 ± 1.3 l/min**	10	Burki, 1984
Healthy males	**6.3 l/min**	10	Smits et al, 1987
Healthy males	**6.1±1.4 l/min**	6	Fuller et al, 1987
Healthy subjects	**6.1± 0.9 l/min**	9	Tanaka et al, 1988
Healthy students	**7.0 ± 1.0 l/min**	10	Turley et al, 1993
Healthy subjects	**6.6 ± 0.6 l/min**	10	Bengtsson et al, 1994

Healthy subjects	**7.0±1.2 l/min**	12	Sherman et al, 1996
Healthy subjects	**7.0±1.2 l/min**	10	Bell et al, 1996
Healthy subjects	**6 ± 1 l/min**	7	Parreira et al, 1997
Healthy subjects	**7.0 ± 1.1 l/min**	14	Mancini et al, 1999
Healthy subjects	**6.6 ± 1.1 l/min**	40	Pinna et al, 2006
Healthy subjects	**6.7 ± 0.5 l/min**	17	Pathak et al, 2006
Healthy subjects	**6.7 ± 0.3 l/min**	14	Gujic et al, 2007

We can also consider historical changes in breathing rates for normal subjects.

Table 3. Historical changes in minute ventilation
(or minute breathing rates) at rest for normal subjects

Condition	Minute ventilation	Age	N. of subjects	Reference
Normal breathing	**6 l/min**	16	-	Medical textbooks
Normal subjects	**4.9**	-	5	Griffith et al, 1929
Normal males	**5.3±0.1**	27-43	46	Shock et al, 1939
Normal females	**4.6±0.1**	27-43	40	Shock et al, 1939
Normal subjects	**6.9±0.9**	-	100	Matheson et al, 1950
Normal subjects	**9.1±4.5**	31±7	11	Kassabian et al, 1982
Normal subjects	**8.1±2.1**	42±14	11	D'Alonzo et al, 1987
Normal subjects	**6.3±2.2**	-	12	Pain et al, 1988
Normal males	**13±3**	40 (av.)	12	Clague et al, 1994
Normal subjects	**9.2±2.5**	34±7	13	Radwan et al, 1995
Normal subjects	**15±4**	28-34	12	Dahan et al, 1995
Normal subjects	**12±4**	55±10	43	Clark et al, 1995
Normal subjects	**12±2**	41±2	10	Tantucci et al, 1996
Normal subjects*	**11±3**	53±11	24	Clark et al, 1997
Normal subjects	**8.1±0.4**	34±2	63	Meessen et al. 1997
Normal females	**9.9**	20-28	23	Han et al, 1997
Normal males	**15**	20-28	47	Han et al, 1997
Normal females	**10**	29-60	42	Han et al, 1997
Normal males	**11**	29-62	42	Han et al, 1997
Normal subjects	**13±3**	36±6	10	Tantucci et al, 1997
Normal subjects	**12±1**	65±2	10	Epstein et al, 1996
Normal subjects	**12±1**	12-69	20	Bowler et al, 1998
Normal subjects	**10±6**	39±4	20	DeLorey et al, 1999
Normal seniors	**12±4**	70±3	14	DeLorey et al, 1999
Normal elderly*	**14±3**	88±2	11	DeLorey et al, 1999
Normal subjects	**17±1**	41±2	15	Tantucci et al, 2001
Normal subjects	**10±0.5**	-	10	Bell et al, 2005
Normal subjects	**8.5±1.2**	30±8	69	Narkiewicz, 2006
Normal females	**10±0.4**	-	11	Ahuja et al, 2007
Normal subjects	**12±2**	62±2	20	Travers et al, 2008

1.2 Do people notice their over-breathing (hyperventilation)?

They very rarely do. Usually, people agree that their breathing is heavy when they breathe more than about 20 l/min at rest (or over 3 times the norm!).

Why is this? Air is weightless, and breathing muscles are powerful. During rigorous physical exercise or during maximum voluntary ventilation, an average person can breathe about 160 l/min, with about 40 breaths per minute and 4 liters of air for tidal volume for each breath (p.545, Straub, 1998). Some athletes can breathe up to 200 l/min during strenuous exercise. So it is easy to breathe only a small portion of our maximum abilities: for example, "only" 16 l/min (or “only” 10% of the maximum capacity) at rest, throughout the day and night and overlook that it is, in fact, a high rate of breathing. It is nevertheless normal during rigorous exercise to breathe 100 l/min or more since CO_2 and O_2 concentrations in the arterial blood can remain nearly the same as at rest.

Usually it is possible to estimate the breathing rate (or minute ventilation) of a person visually. If the chest is moving at rest, the person is breathing at least twice more air than the physiological norm (over 12 l/min for a 70 kg man). If his shoulders are moving, he is breathing four times the norm. Normal breathing is invisible, inaudible, and regular. Moreover, when healthy people are asked about their breathing, they usually say that they feel nothing, while sick people have various sensations about movements of the air through the nostrils, at the back of the throat, and in the area of the abdomen.

Unfortunately, modern medical doctors are not trained to pay attention to the breathing rate of their patients. A patient can come to the doctor's office while heavily panting, even through the mouth, and the GP or MD will not even mention or suggest to breathe through the nose or to reduce his or her breathing rate.

1.3 The main effect of hyperventilation

The previous section demonstrated that many sick people chronically over-breathe. It is possible to assume that, maybe these people got sick in the first place, and then started to breathe heavier. Alternatively, it is also possible that they got sick because of over-breathing. In order to find out what causes what let us look at the main physiological effects of such over-breathing for healthy people. What would happen with a healthy person, who starts to breathe too much?

Respiration is the process of regulated exchange of two gases, CO_2 (carbon dioxide) and O_2 (oxygen). The human body, as a form of life, produces energy by oxidizing different substances, mainly fats and carbohydrates. Both these substances are mainly composed of carbon with some hydrogen and oxygen. Hence, the main end products of this energy production are CO_2 and water. Normally, one of the functions of breathing, apart from bringing new O_2 for cells to use, is to remove excessive (but not all) CO_2.

When healthy people breathe near the norm, their CO_2 level in the organism is also near the physiological norm. However, breathing too much delivers more O_2 to the lungs and removes more CO_2 from the body.

Let us look at the basic course of events in a case of acute over-breathing. When the person starts to breathe deeply and frequently, the total concentration of CO_2 in the lungs gets smaller since the person intensively blows off carbon dioxide from the lungs. It takes about one to two minutes to reduce the concentration of CO_2 in blood. About 1-10 minutes later, CO_2 concentrations in the nervous tissues, muscles, and most other organs and cells are also reduced due to CO_2 diffusion from these parts to the blood.

Thus, the first effect of hyperventilation is lowered CO_2 concentrations in all body cells. If hyperventilation is chronic, CO_2 deficiency is also chronic.

1.4 Do we need this “poisonous” CO_2?

Carbon dioxide gas is used for killing animals. In small mammals (e.g., rats and mice) the loss of consciousness is quick (seconds). In larger mammals (e.g., guinea pigs) the animals first become very distressed and disturbed. They are restless, breathe deeply, and salivate profusely. For discussion of animal euthanasia with the use of carbon dioxide one may see (Coenen et al, 1995) and (Paton, 1983). Very large relative concentrations or pure carbon dioxide gas are normally used.

Also, there are many books, newspaper articles, and even some medical publications, which claim that one of the main functions of human respiration is “*to remove the poisonous carbon dioxide from the human organism*”. In addition, there are many popular health articles, which state that carbon dioxide is a “*waste*” gas. It follows from this approach that it is better to breathe deeper and faster in order to expel the “poison” at higher rates.

However, there are thousands of medical and physiological publications, studies, trials, and experiments that

state the opposite. Namely, they strongly discourage over-breathing (hyperventilation), both in its acute and chronic forms.

Why? Over-breathing removes too much carbon dioxide from the organism, while carbon dioxide is absolutely necessary to sustain life. When its level becomes about 3-4 times less than the physiological and medical norms, death is an immediate outcome.

Probably, these ideas about CO_2 appeared after French scientist Antoine-Laurent Lavoisier discovered in 1788 the role of CO_2 and O_2 in breathing. He explained why mice and candle both died in CO_2, but could live longer in O_2.

What about killing of animals with high levels of carbon dioxide in air? Could we die in similar conditions? Let us look at another related phenomenon, drowning. Thousands of people die every year because of too much water being taken in through the mouth. About 4-5 litres of water in taken at once would be enough to fill the whole stomach and parts of the lungs with water causing death in a few minutes. However, nobody claims that water is a "*poison*" because water is equally important and vital (in sensible quantities, not 4-5 litres at once) for human and any cellular life. The situation with carbon dioxide is exactly the same.

Every living thing needs normal levels (not too much and not too little) of carbon dioxide for healthy functioning. Moreover, numerous medical studies cited in this chapter clearly show that carbon dioxide is the substance that is most needed by patients with various modern chronic degenerative disorders and ailments.

1.5 CO_2 deficiency: the main physiological effect of hyperventilation

Let us look at some of the known carbon dioxide effects, which are confirmed by professional Western studies. Note that these effects can be found, in varying degrees, in any normal human organism.

Stabilizer of transmission of signals between nervous cells

The normal work of our senses, conscious thinking, decision making, and all other mental activities require stable transmission of electrical signals between nervous cells. Such transmission is possible when CO_2 content in nerve tissues is normal. Logic, sense, reason, wisdom, focus, memory, concentration and many other qualities are based on this stability of signal transmission.

The signal is passed from one nervous cell to another only when the strength or voltage of the signal is higher than a certain threshold value so that accidental signals will not be amplified causing disruption in the work of the CNS. This threshold value is very sensitive to the local CO_2 content.

When we hyperventilate and CO_2 content is suboptimal, accidental weak signals can be amplified and transmitted further interfering with the real signals based on senses, memory, logic and other objective factors.

Hence, CO_2 has a calming effect on excessive excitability of brain areas responsible for conscious thinking (e.g., Krnjevic, 1965). Other researchers (Balestrino & Somjen, 1988; Huttunen et al, 1999) also concluded that increased CO_2 pressure generally reduces cortical excitability, while hyperventilation "*leads to spontaneous and asynchronous firing of cortical neurons*" (Huttunen et. al., 1999).

Hence, breathing too much makes the human brain abnormally excited due to reduced CO_2 concentrations. As a result, the brain gets literally out of control due to appearance of spontaneous and asynchronous ("self-generated") thoughts. Balestrino and Somjen (1988) in their summary directly claimed that, "*The brain, by regulating breathing, controls its own excitability*".

These effects of CO_2 on brain cells are of special importance in understanding anxiety, insomnia, panic attacks, epilepsy and other psychological and neurological problems and disorders to be discussed later. Besides, this effect is important in order to understand the mechanism of the mind-body connection.

Bohr effect (or supply of oxygen to all body cells)

CO_2 is a catalyst for the chemical release of O_2 from haemoglobin cells. This phenomenon is called the Bohr effect and it can be found in many medical textbooks (e.g., Ganong, 1995, Starling & Evans, 1968). Bohr and his colleagues (1904) first described this effect. How does it work?

In normal conditions (when we breathe about 6 l/min), arterial blood is 96-98% saturated with O_2 due to a fresh air supply to the lungs. When the arterial blood arrives at the tissues, some O_2 is released by its carriers, the haemoglobin cells (red blood cells). What is the reason for this chemical release? The cells of the organism also breathe, and the more they breathe the more CO_2 is produced. These elevated values of CO_2 in tissues increase the CO_2 level in the blood due to CO_2 diffusion from the tissues. As a result, the greater the amount of CO_2 in the blood, the

more O_2 is going to be released from the haemoglobin cells for the tissues to use, since CO_2 is a catalyst causing this chemical reaction.

This mechanism is especially effective during physical exercise. Indeed, depending on the type of exercise, some of our muscles work harder than others. Those muscles that produce more CO_2, are going to get more O_2 in exchange (due to the Bohr effect), so they can continue to work at high rates. Were this mechanism to be absent, a human organism would quickly tire at the slightest physical exertion due to lack of oxygen.

Therefore, carbon dioxide is a necessary factor for oxygenation of tissues. No carbon dioxide means no oxygen in the tissues, while no oxygen means no energy for various processes and no life.

Let us look at the events when people over-breathe. On the one hand, breathing more can raise blood saturation from normal 96-98% to 97-99% (by about 1%). However, it follows from the Bohr effect that those who chronically breathe too much (in comparison with physiological norms) suffer from hypoxia (low oxygen concentrations) in tissues due to the low carbon dioxide level in the blood and tissues. (Low tissue oxygenation is normally found in malignant cells, diseased nervous cells, and inflamed tissues of various organs). Meanwhile, normal breathing (about 6 l/min) provides more O_2 for the tissues of the organism.

Hence, the paradox of breathing is in the fact that acute over-breathing, while bringing more oxygen during first seconds, creates the opposite effect: in a few minutes (or even earlier). The cells start to suffer from the lack of oxygen. Therefore, chronic deep breathing causes chronic tissue hypoxia.

Prolonged forceful over-breathing can have disastrous consequences, as Yale Professor Yandell Henderson and his colleagues demonstrated in their work with dogs almost a century ago (Henderson et al, 1908). In these experiments, forceful respiration was created using a suction and exhaust pump. The dogs after many minutes were disconnected from the machine and died without attempting to draw a single breath due to failure of the cardiovascular system. This result was completely unexpected by the researchers. Later, it became clear that hypoxia was one of the factors contributing to these deaths. However, there was also another factor: constriction of small blood vessels due to low carbon dioxide level.

Local vasodilation

CO_2 locally dilates arteries and arterioles making the work of the heart easier, creating conditions for delivering more oxygen to tissues, and removing more waste products.

Vice versa: low carbon dioxide stores have a local vasoconstrictive effect leading to spasms, hypoxia (this time due to poor blood supply) and accumulation of metabolic wastes in different vital organs and tissues.

One may argue that there are many other blood vessels which also contribute to total resistance to blood flow. Why should we concentrate on CO_2 effects on arteries and small blood vessels? Basic physiology of the human organism explains, that the total relative resistance to blood flow in arteries and arterioles is about 3-8 times greater than in any other type of blood vessels (Ganong, 1995). The effect of vasoconstriction due to hyperventilation is so powerful, that Soley and Shock (1938) reported their difficulty in obtaining blood samples from fingers of their patients following voluntary hyperventilation. It is more difficult for the heart to pump the blood through the body when small blood vessels, due to low carbon dioxide, are constricted. Moreover, the heart muscle itself receives less blood if the person is over-breathing.

Therefore, low CO_2 level in the organism produces profound adverse impact on the cardiovascular system and blood supply to the heart and other organs.

What about the human brain? Does it suffer from heavy breathing? The following results were obtained by measuring blood flow through the main artery (the carotid artery) leading to the brain. Voluntary hyperventilation led to 35% reduction in the blood flow to the brain in comparison with the conditions at rest. This result is quoted in the medical textbook written by Starling & Evans (1968), while the effect is well documented and has been confirmed by dozens of professional experiments.

By the way, do you notice that when people passionately argue with each other, or are angry, or violent, they usually breathe heavily? Would it be reasonable, in the light of these physiological studies, to conclude that it is useless to argue or try to reason with the person whose brain is not normally oxygenated due to excessive breathing?

There are numerous studies which indeed do reveal the negative effects of over-breathing on different skills (motor, memory, logic) and general performance, which require combinations of various human abilities.

Hence, a low carbon dioxide level not only reduces oxygenation of tissues, but also impairs blood supply to vital organs of physiological functioning.

Relaxation of smooth muscles

CO_2, when applied locally, is a relaxant of smooth muscles (e.g., Hudlicka, 1973). Dr. Brown in his article "*Physiological effects of hyperventilation*" analysed almost 300 professional studies and stated, "*Studies designed to determine the effects produced by hyperventilation on nerve and muscle have been consistent in their finding on increased irritability*" (Brown, 1953).

This fact, together with the properties of CO_2 mentioned previously, will help us to understand the mechanism by which normal carbon dioxide concentrations can restore the harmonious work of different muscular groups (such as the heart, respiratory muscles, muscles of the digestive tract, etc.) in order to eliminate muscular spasms (e.g., heart attacks, asthma attacks, constipation, etc.). Moreover, since muscles get irritated it is normal to expect that when people breathe too much, they are more likely to be tense, anxious, stressed, aggressive, and violent. Vice versa, normal carbon dioxide concentrations would result in muscular relaxation, composure, and sensible actions.

Bronchodilation

Normal level aCO_2 eliminates possible constriction of bronchi and bronchioles which can appear due to low aCO_2. The article "*The mechanism of bronchoconstriction due to hypocapnia* [low CO_2 concentrations] *in man*" (Sterling, 1968) described the following effect of CO_2 on air passages. Bronchoconstriction (narrowing of air passages), which is the main problem of asthmatics, is mainly mediated by special nerve cells. Low aCO_2 makes them, among many other nervous tissues, more excited, causing narrowing of bronchi and bronchioles.

Therefore, over-breathing can cause bronchoconstriction (as it is observed in asthma) leading to the feeling of suffocation.

Blood pH balance

CO_2 is the most important factor in controlling blood pH, balance of electrolytes and pH of other body fluids (urine, saliva, stomach secretions, etc). Indeed, bicarbonate is the largest CO_2 component of the blood, as well as intra-cellular and extra-cellular fluids, while a typical medical or physiological textbook will indicate its leading role in the control of pH of blood and other body fluids (e.g., medical textbooks by Starling & Evans, 1968; Guyton, 1984; and Ganong, 1995). Hence, changes in bicarbonate concentration must influence the ionic composition of every human cell.

As one of the numerous effects in this area, Carryer (1947) found that "*While no significant change in total calcium of the blood takes place, the readily available, or ionized portion is affected markedly* [due to hyperventilation]… *The decrease in available calcium increases excitability of the neuromuscular mechanism, inducing tetany*".

These medical conclusions point out the cause of problems (low carbon dioxide due to hyperventilation) with calcium metabolism, which is found in osteoporosis, arthritis, and other health conditions.

Participation and catalisation of chemical reactions

CO_2 is a participant of numerous other biochemical reactions involving virtually all vitamins, minerals, amino acids, hormones, carbohydrates and other vital substances. Some of the chemical reactions, all requiring CO_2 as a catalyst or as one of the reagents, were described by Kazarinov (1990).

Apart from these known effects, there are probably many other processes of the human organism that require normal CO_2 levels and normal breathing for optimum physiological functioning.

The first respiratory physiologists were called "cardio-respiratory physiologists" since the link between the cardiovascular and respiratory systems, as they found it, was very intimate. Professor Yandell Henderson was one of the most prominent scientists in this area. His article "*Carbon dioxide*" was published in 1940 in *Cyclopedia of Medicine*. In the section with the title "*Relations of Carbon Dioxide and Oxygen in the Body*" he wrote,

"*Carbon dioxide is, in fact, a more fundamental component of living matter than is oxygen. Life probably existed on earth for millions of years prior to the carboniferous era, in an atmosphere containing a much larger amount of carbon dioxide than at present. There may even have been a time when there was no free oxygen available in the air…*

Another natural, but very obstructive misconception is that oxygen and carbon dioxide are so far antagonistic that in blood a gain of one necessarily involves a corresponding loss of the other. On the contrary, although each

tends to raise the pressure and thus promote the diffusion of the other, the 2 gases are held and transported in the blood by different means…

A sample of blood may be high in both gases, or low in both gases. Moreover, under clinical conditions low oxygen and low carbon dioxide—anoxemia and acapnia—generally occur together. Each of these abnormal states tends to induce and intensify the other. Therapeutic increase of carbon dioxide, by inhalation of this gas diluted in air, is often the effective means of improving the oxygenation of the blood and tissues."

In the section "*As a factor in the Acid-base Balance of the Blood*", he continued,

"*Modern physiology has shown that, in addition to the control and regulation exerted by the nervous system, there are many chemical substances produced in the body that influence function and form. To these active principles Starling gave the name of "hormones." Among the hormones are epinephrine (often called adrenaline), pituitrin, thyroxin, insulin and many other products of the glands of internal secretion and other organs. Carbon dioxide is the chief hormone of the entire body; it is the only one that is produced by every tissue and that probably acts on every organ. In the regulation of the functions of the body, carbon dioxide exerts at least 3 well defined influences: (1) It is one of the prime factors in the acid-base balance of the blood. (2) It is the principal control of respiration. (3) It exerts an essential tonic influence upon the heart and peripheral circulation.*"

Finally, he stated in the section "*In the Control of Respiration and the Circulation*",
"Carbon dioxide is the chief immediate respiratory hormone."

1.6 Medical studies of hyperventilation

The previous section described some effects (there are many more as we are going to see later) of hyperventilation on healthy people. While these effects are normal for any human organism, the degree of particular negative changes and the location of the most affected organs are various in different individuals. Therefore, the individual problems created by over-breathing are going to be different. In order to investigate this issue, let us turn our attention to medical studies.

Medical doctors and professors have written extensive reviews of professional literature and described their own case histories of hyperventilation (Bass, 1990; Brasher, 1983; Lum, 1975; Magarian, 1982, 1983; Morgan, 1983; Tavel, 1990). These studies showed the symptoms of over-breathing and the profound negative influence of both acute and chronic hyperventilation on the whole biochemistry of the human organism.

The first medical article containing a description of the symptoms of hyperventilation, but without understanding their cause, was published by DaCosta (DaCosta, 1871). One group of researchers described the biochemical mechanism by which hyperventilation can gradually cause problems with high cholesterol (hypertension) and high blood sugar levels (diabetes) (Lavrent'ev, 1993).

Acute and, especially, chronic hyperventilation, according to these and many other references, affects every system and organ of the human body causing a wide variety of symptoms. Many medical doctors have mentioned that the physiological response to hyperventilation is individual. Thus, individual genetic predisposition and certain other factors define which system or organ is the most affected by hyperventilation. It can be the heart, brain, kidneys, liver, intestines, stomach, lungs or one of many others. Additional references on the negative effects of hyperventilation can be found in the previously cited works. Magarian (1982), for example, quoted over 180 other scientific articles in order to back up his conclusions about the physiological consequences of hyperventilation.

Since hyperventilation is an important part of our fight-or-flight response, the blood is generally diverted from vital organs to large skeletal muscles. Studies found decreased perfusion of the heart (Okazaki et al, 1991), brain (discussed above), liver (Hughes et al, 1979; Okazaki, 1989), kidneys (Okazaki, 1989), and colon (Gilmour et al, 1980). Moreover, investigations of blood flow in muscles were not consistent in relation to changes in perfusion, while oxygenation of muscles in some studies is even reduced (Thorborg et al, 1988). That could happen probably due to vasoconstriction and the suppressed Bohr effect. Normally, hyperventilation also compromises oxygenation of vital organs (e.g., Hughes et al, 1979; Hashimoto et al, 1989; Okazaki et al, 1991).

Typically, the blood flow to vital organs is directly proportional to aCO_2. Such a linear relationship (between brain blood flow and carbon dioxide concentration) can be found, for example, in *Handbook of Physiology* (Santiago & Edelman, 1986).

Chronic hyperventilation interferes with normal digestion. Decreased perfusion and oxygenation of GI organs can lead to lack of digestive enzymes, accumulation of metabolic waste products, slow digestion, putrefaction of certain nutrients and mal-absorption. That should cause problems with protein metabolism (which usually appear before problems with fat and carbohydrate metabolism), thus adversely affecting normal repair of the body (especially

the GI tract, the largest consumer of amino acids) and the immune system.

In order to experience the effects of breathing on digestion, one may voluntarily hyperventilate after a meal. While normal digestion can take 2 hours, hyperventilation may extend this time up to 5-8 hours or more, depending on the degree of hyperventilation.

Warning. *By mild voluntary hyperventilation, you may almost halt digestion. For many people that can cause GI distress and aggravation of existing gastrointestinal problems. Breathing less (or voluntary hypoventilation) can also make some digestive problems worse.*

It would be normal to expect that the degree of all these negative effects may vary from individual to individual.

Similarly, according to the article entitled "*The effects of hyperventilation; individual variability and its relation to personality*" (Clark, 1982), not only negative cardiovascular changes, but also psychological effects of hyperventilation had individual variability.

Thus, when we over-breathe, there are certain factors (both, genetic and environmental) which create our specific physiological responses to hyperventilation. While the above-mentioned negative consequences of deep breathing are typically found in a normal human organism, genetic predisposition and some other factors (previous events which influenced the organism) probably define the organs, their parts and the systems which are going to suffer most from low carbon dioxide stores and other effects of chronic hyperventilation. More research is required in order to find the effects of hyperventilation and individual differences.

1.7 Studies about the hyperventilation provocation test

One may realize the dangers of over-breathing by performing a HVPT (hyperventilation provocation test), during which the person should breathe very quickly and deeply, usually for about 2-3 minutes. (It would be impossible to do it much longer due to losing consciousness, while forceful involuntary over-breathing, when a pump is used, would cause death in dozens of minutes). This short over-breathing test has a well-recorded history of clinical use and was employed by many medical doctors to provoke the symptoms of the main health problem in order to diagnose it, as well as to demonstrate to patients that hyperventilation was the main cause of their symptoms. Thus, using deep and fast breathing, you can reproduce your specific health symptoms.

Over-breathing is an excellent tool used by medical doctors around the world to find out the most sick organs and systems in any particulat patient.

For example, voluntary over-breathing in asthmatics causes the asthma attack, in people with hypertension – the heart attack, in epileptics – epilepsy attack, etc. Here is a short summary of medical studies regarding different health conditions, number of patients investigated, and the percentage of patients who reproduced their specific health problem.

- coronary artery spasms (Nakao et al, 1997) 206 patients, 100% specific;
- bronchial asthma (Mojsoski N & Pavicic F, 1990) 90 patients, 100% specific;
- panic attacks (Bonn & Readhead, 1984; Holt PE, Andrews, 1989; Nardi et al, 2000), 95% specific;
- epileptic absence seizures (Esquivel, 1991; Wirrel, 1996).

Important notice. *The hyperventilation provocation test should not be performed by people who have certain severe health problems, without professional supervision, due to possible complications.*

The symptoms experienced can be reversed by reducing ventilation and raising carbon dioxide stores to previous values. Another important finding of these and other authors is that most people, including numerous above-mentioned patients, were unaware of their abnormal breathing pattern and believed that they breathed normally.

1.8 Hypoventilation as a health problem

As shown above, many disease states are characterized by hyperventilation. Are there any health problems in which sick people breathe less than the norm?

Low minute volume can be found, for example, in cases of hypothyroidism. Such people usually have abnormally low levels of thyroid hormones. As a result, their cells cannot generate enough energy. Typical symptoms

of hypothyroidism are low energy, hypoxia, apathy, sleepiness, and weight gain. Indeed, since little CO_2 is produced by cells, less O_2 is released to them by haemoglobin cells, due to the suppressed Bohr effect.

Chronic mountain sickness patients can have lower than normal ventilation, but this rare health condition is observed only in those who live about 3,000 m or more above sea level.

Abnormal breathing may be observed in sleep apnoea, in which sleeping patients stop breathing for 10-20 or even up to 40-50 seconds. Such apnoeic spells interrupt the normal functioning of the nervous system. As a result, these spells can awaken the patients many times during the night, interfering with physiological and psychological recovery. These people can breathe too little during spells. The inter-event ventilation (i.e. between spells) has been observed to be usually more than 20 l/min.

Patients with hypothyroidism and sleep apnoea can normalize their breathing patterns using the method described in later Chapters. Moreover, practical work of breathing practitioners revealed that normalization of breathing of these patients dramatically improves their health state. Even more surprising is the fact that these patients can use virtually the same methods and breathing exercises (to be discussed) in order to restore their health.

1.9 End-tidal CO_2 and different health problems

Minute ventilation, although a very important respiratory parameter, needs special equipment and does not always indicate hyperventilation and small aCO_2. Inaccuracies occur in cases of small body weight (found, for example, in children), irregular or very shallow breathing found in some obese patients, and obstruction of airways leading to partial or total closure of some lung areas. This last phenomenon will be discussed in the next section.

As a result, many professional researchers, when investigating respiration, often measure $etCO_2$ (end-tidal CO_2) as a more accurate characteristic reflecting CO_2 content of the lungs. A device called a "capnometer" can continually measure CO_2 level in the expired air. The level of CO_2 gradually rises during exhalation showing an approximate equalization with the CO_2 value in alveoli in the lungs (hence, the phrase "end-tidal"). The normal alveolar CO_2 pressure is about 40 mm Hg pressure (Guyton, 1984; Ganong, 1995) or partial pressure of 5.3% of normal air at sea level. According to "Handbook on physiology" (Severinghaus JW, 1965), "A PCO_2 below 35 mm Hg is indicative of alveolar hyperventilation" (p.1476). 35 mm Hg corresponds to 4.6% CO_2 at sea level (see Appendix 2 in order to find the relationships between aCO_2% and absolute aCO_2 pressure at different altitudes).

All previously quoted studies (section 1.1) indicating hyperventilation should find abnormally low $etCO_2$ for tested patients. Indeed, people who breathe more should generally show smaller CO_2 concentrations in expired air.

What also follows from many studies is that with the deterioration of health $etCO_2$ tension gets even lower.

The investigation of over 100 patients (Tanabe et al, 2001) with different degrees of chronic heart failure revealed that class I patients (light degree) had about 34.5 mm Hg $etCO_2$ pressure, class II patients: 32.5 mm Hg, and class III patients: 30.8 mm Hg. Thus, the heart patients with the more serious heart problems had lower CO_2 levels and, therefore, heavier breathing in terms of minute ventilation.

American scientists from the Brown Medical School in Providence recently published a study *End-tidal carbon dioxide predicts the presence and severity of acidosis in children with diabetes* (Fearon & Steele, 2002). They start this publication with, *"Patients with diabetic ketoacidosis (DKA) hyperventilate, lowering their alveolar (PACO(2)) and arterial carbon dioxide (PaCO(2))"*. Their conclusion was, *"End-tidal CO(2) is linearly related to HCO(3) and is significantly lower in children with DKA"* (Fearon & Steele, 2002).

Expired end-tidal CO_2 values are considered by many emergency professionals as an accurate predictor (life/death) of cardiac arrest. For example, authors of the article "*End-tidal carbon dioxide during cardiopulmonary resuscitation in humans presenting mostly with asystole: a predictor of outcome*" investigated 120 French patients during non-traumatic cardiac arrest. The researchers found that "*end-tidal CO_2 could provide a highly sensitive predictor of return of spontaneous circulation during cardiopulmonary resuscitation (MPR)*" (p.791, Cantineau et al, 1996). More recently a large group of medical doctors from several American hospitals tested over 100 patients and wrote an article "*End-tidal carbon dioxide measurements as a prognostic indicator of outcome in cardiac arrest*" with the same conclusion (Ahrens et al, 2001). There are several other studies written by emergency professionals, with the same conclusions.

Therefore, emergency patients (with cardiac arrest) with the most deep and frequent breathing have the least chances of survival.

Rosen and his colleagues (1990) in the abstract of the article *"Is chronic fatigue syndrome synonymous with effort syndrome?"* wrote: "*Chronic fatigue syndrome (CFS), including myalgic encephalomyelitis (ME) and postviral syndrome (PVS), is a term used today to describe a condition of incapacity for making and sustaining effort,*

associated with a wide range of symptoms. None of the reviews of CFS has provided a proper consideration of the effort syndrome caused by chronic habitual hyperventilation. In 100 consecutive patients, whose CFS had been attributed to ME or PVS, the time course of their illness and the respiratory psychophysiological studies were characteristic of chronic habitual hyperventilation in 93. It is suggested that the labels 'CFS', 'ME' or 'PVS' should be withheld until chronic habitual hyperventilation - for which conventional rehabilitation is available - has been definitively excluded."

Paulley started his article "*Hyperventilation*" (Paulley, 1990), with "*Physicians' and specialists' continued failure to recognize, diagnose and treat adequately the majority of hyperventilators is a disgrace. Hyperventilation Syndrome (H.V.S.), incorrectly labelled myalgic encephalomyelitis (M.E.), is the latest example of the profession's incompetence*."

These doctors claim that chronic fatigue syndrome, myalgic encephalomyelitis, and postviral syndrome can be directly caused by over-breathing since normalization of breathing results in recovery of the patients with these health concerns.

Capnometers (devices to measure carbon dioxide levels in the expired air) have become especially popular among psychologists. For example, Fried and colleagues (1990) studied several groups of subjects with anxiety, panic phobia, depression, migraine, and idiopathic seizures. The abstract claims that "*virtually all the noncontrol subjects were found to show moderate to severe hyperventilation and the accompanying EEG dysrhythmia*" (p.67).

Abnormally low carbon dioxide values ($etCO_2$) were found in all (over 60) patients with neurotic depression and non-retarded endogenous depression (Damas Mora et al, 1976).

Asmundson and Stein (1994) measured carbon dioxide concentrations in over 20 patients with panic disorder. Their average CO_2 was also below the medical norm.

Therefore, various psychological problems are connected and can be the consequences of chronic over-breathing.

1.10 Hypoxia and blood shunting

McFadden & Lyons (1968) showed that in mild and severe asthmatic patients, some parts of the lungs could not carry out adequate air exchange due to airway obstruction. This causes an ineffective exchange of CO_2 for O_2 in venous blood in the obstructed alveoli of the lung. Therefore, this venous blood, after leaving the lungs almost unchanged, is mixed with oxygenated arterial blood. This effect is called "blood shunting". As a result, hypoxia of such patients becomes worse since less O_2 is present in the blood. Meanwhile, aCO_2 rises to or even exceeds, in severe cases, the physiological norm (40 mm Hg). Thus, with further deterioration of health, large aCO_2 (hypercapnia) is observed. In spite of increased aCO_2 pressure and greatly improved Bohr effect, tissue hypoxia is greater than before due to very low arterial oxygenation (it would not be correct to expect that higher aCO_2 concentrations can compensate for lack of oxygenation in the damaged lung areas. The balance between these two gases is indeed delicate.) This problem of ventilation/perfusion mismatch (inadequate air supply to some lung parts) and corresponding blood shunting is especially severe in patients with emphysema.

Normally, in healthy lungs each lung area requires air ventilation, which is approximately proportional to its volume. Meanwhile, in severe cases of ventilation/perfusion mismatch, the working lung part can hyperventilate, but the total ventilation can be less than the norm. Indeed, if, for example, only one-third of the lungs is functional, as in emphysema, this third may use, say, about 3-4 l of air per minute, indicating general hypoventilation (3-4 l/min is less than the physiological norm). Meanwhile, under normal conditions, this working lung part would need only about 2 l/min.

Ventilation/perfusion mismatch is common in patients with mild or severe asthma, emphysema, and cystic fibrosis, and for some patients with obesity, hypertension, and diabetes. However, for most people very low aCO_2 does not cause severe airway obstruction and corresponding blood shunting. Taking into account individual variability of the effects of CO_2 depletion (discussed in section 1.3), it is possible that asthmatics and other groups of people as above have air passages, which are more sensitive to hyperventilation due to their genetically inherited characteristics.

Hypoxic hypoxia can also be the result of the ventilatory failure due to fatigue of respiratory muscles or depression of the respiratory neurons in the brain by morphine and other drugs. (Hypoxic hypoxia is hypoxia resulting from a defective mechanism of oxygenation in the lungs.)

1.11 Critical care patients and arterial CO_2

It was shown above that, in cases of cardiac arrest, carbon dioxide concentration is a reliable predictor of human survival. Meanwhile, critical care professionals often use the most sophisticated and advanced devices to measure different physiological parameters. Analysis of arterial blood usually includes investigation of blood gases (blood concentrations of bicarbonates, total CO_2, oxygenation, etc.) of critically ill patients.

All 29 patients with severe liver damage (in most cases due to metastatic cancer or cirrhosis of liver) had low CO_2, while for 25 patients "*it was also clinically evident that respiratory exchange was increased markedly*" (p.762, Wanamee et al, 1956). Thus, hyperventilation was visually observed by the authors of this publication, "*Respiratory alkalosis in hepatic coma*". They also found that heavy over-breathing led to severe electrolyte abnormalities. These abnormalities included decreased sodium ions and increased chloride ions in the blood. Abnormally high lactic and pyruvic acid concentrations were other frequent effects.

Blood gases and respiratory patterns provided accurate information for survival prognosis in acute cerebrovascular accidents. When these parameters were normal, patients survived. Out of 11 hyperventilating patients with less than 35 mm Hg aCO_2, only one survived (Rout et al, 1971).

The same conclusion (regarding aCO_2 and survival prognosis) was made for head injuries (Huang et al, 1963; Vapalanti & Trouph, 1971).

Summarizing the results of these works and their connection with brain dysfunction, Dr. Plum wrote, "*The combination of hyperpnoea* [increased breathing] *with an elevated pH, and a subnormal or moderately low oxygen tension occurs in many serious illnesses that entirely spare the brain. These include the alveolar-capillary block of diffuse pulmonary carcinomatosis; heart failure; advanced cirrhosis, with or without hepatic coma; acute pulmonary infarction; and many others, including the cryptic pulmonary congestion that accompanies most serious disease in the obtunded and elderly*" (Plum, 1972). Interestingly, all above-mentioned effects (low carbon dioxide concentration, elevated pH, and hypoxia) quoted by Dr. Plum are caused by heavy breathing.

Hence, one can conclude that over-breathing is a normal feature of these severe diseases.

When suffering various serious health problems (heart disease, diabetes, cancer, AIDS, etc.) the patient's life is usually threatened, not by the main health problem, but by complications and infections, such as in the case of bacteremic shock. Analysing a group of patients initially diagnosed with arteriosclerotic heart disease, cerebrovascular insufficiency, diabetes, arthritis, several forms of cancer, fatty liver, and alcoholism, one study showed that complications due to pathogenic microorganisms in the blood caused 46 deaths in 50 patients (Winslow et al., 1973). Pneumonia and urinary tract infections were the foci of pathogenic microorganisms. Now we may ask the following: what was observed with their breathing, when not only a part of the organism, but even the blood was polluted with pathogens? All 50 patients, according to a table accompanying this article, had very disturbed blood gases corresponding to severe over-breathing.

Dr. Simmons and his colleagues wrote an article "*Hyperventilation and respiratory alkalosis as signs of gram-negative bacteremia*" (bacteremia being the presence of bacteria in the blood). This extract is from the beginning of their abstract:

"*Visible hyperventilation was observed clinically in patients with Gram-negative bacteremia. Eleven patients with Gram-negative infections and either proved or probable bacteremias were therefore studied to see if hyperventilation might be a common response to such bacteremia. In every case there was laboratory evidence of hyperventilation, and in 8 cases the hyperventilation was visible to the observer. Since only patients were studied who had no other cause for increased ventilation, this appears to be a primary response to the bacteremia...*" (abstract, Simmons et al, 1960).

Another group of US medical professionals found that the degree of over-breathing has a strong correlation with over-all mortality (Mazarra et al, 1974). Heavier breathing indicated smaller chances of survival. Here is what they wrote in their scientific abstract:

"*Respiratory alkalosis* [blood alkalisation is the normal physiological result of over-breathing] *was the most common acid-base disturbance observed in a computer analysis of 8,607 consecutive arterial blood gas studies collected over an 18 month period in a large intensive care unit.*

Through a retrospective review of the randomly selected hospital records of 114 patients, we defined four groups based upon arterial carbon dioxide tension ($PaCO_2$) and mode of ventilation. Group I, with a $PaCO_2$ of 15 mm Hg or less, consisted of 25 patients with an over-all mortality of 88 per cent. Group II, with a $PaCO_2$ of 20 to 25 mm Hg, consisted of 35 patients with a mortality of 77 per cent. Group III, with a $PaCO_2$ of 25 to 30 mm Hg, consisted of 33 patients with a mortality of 73 per cent, and Group IV, with a $PaCO_2$ of 35 to 45 mm Hg, consisted of 21 patients

with a mortality of 29 per cent (p<0.001). Shock and sepsis were most common in group I patients.

These findings suggest that extreme hypocapnia [low level of carbon dioxide] *in the critically ill patient has serious prognostic implications and is indicative of the severity of the underlying disease*" (Mazarra et al, 1974).

This article indicated that the names of the most common diseases to occur in all 4 groups of people were cerebrovascular disease, hepatic coma, bronchopneumonia, and arteriosclerotic heart disease.

Finally, let us look at the conclusion drawn by a group of US researchers who recently wrote an article with the title "*Can cardiac sonography and capnography be used independently and in combination to predict resuscitation outcomes?*" (Salen et al, 2001).

"CONCLUSIONS: Both the sonographic detection of cardiac activity and ETCO(2) levels higher than 16 torr were significantly associated with survival from ED resuscitation; however, logistic regression analysis demonstrated that prediction of survival using capnography was not enhanced by the addition of cardiac sonography" (Salen et al, 2001).

In other words, they found, probably to their surprise, that monitoring of the heart, as an addition to the monitoring of breathing, does not provide any further information about chances of survival.

A review of these professional studies indicates that critically ill patients usually have very low carbon dioxide level due to visible hyperventilation. Laboured breathing of such patients probably corresponds to minute ventilation of 20-25 l/min or more.

The analysis of Western medical literature suggests that many critically ill patients die in conditions of heavy and deep breathing.

Deep breathing, as we showed above, reduces oxygenation of the body. Are there any simple tests that reflect our ventilation and oxygenation? *"Oxygen content in the organism can be found using a simple method: after exhalation, observe, how long time the person can have no breathing without stress"* (Buteyko, 1977).

1.12 Breath-holding time and its clinical significance

All breathing parameters described above need to be measured using special equipment. Meanwhile, there is a simple test, which can be done at almost any moment by everyone, since only a watch or a clock is required. This is BHT (breath-holding time), or how long one can be without breathing. What are the results of medical studies regarding this test?

According to textbook "*Essentials of exercise physiology*" (McArdle et al, 2000), "*If a person breath-holds after a normal exhalation, it takes about 40 seconds before breathing commences*" (p.252).

Breath holding can be started at different phases of breathing (e.g., after normal inhalation, or exhalation, or taking a very deep inhalation, or a complete exhalation). These different conditions can produce large variations in results (by more than 200%). Moreover, sometimes patients are asked to take 2 or 3 deep breaths before the test. Since researchers use different methods for BHT measurements, the standardization of results is necessary in order for them to be compared.

"*Handbook of physiology*", after analysing numerous relevant publications, suggested the following proportions for BHT measurements (Mithoefer, 1965). If BHT after full inhalation is 100%; then BHT after normal inhalation is 55%; BHT after normal exhalation is 40%; BHT after full exhalation is 24%. Taking an additional full exhalation or inhalation before starting the test increases BHT by about 5 or 15% respectively for each full manoeuvre. This information allows us to compare different BHT tests done during almost a century of clinical investigations, if we use some standard conditions for the test. In order to do that, let me introduce the BHT: **BHT is BHT after quiet or usual expiration.** The under-line can remind the reader about BHT measured at the base level, as when we are totally relaxed (as after usual exhalation).

Different studies and their results can be now compared by changing their BHTs to the standard of measurements, the BHT. These results are given in Table 1.2.

Warning. Usually BHT in physiological or medical studies is measured for as long as possible. This procedure is dangerous if you have certain serious health conditions with inflammation, irritation, ulcers or any other damage to internal organs (this will be fully explained later). The conditions requiring caution include: certain heart conditions, diabetes, hypoglycaemia, severe kidney disease, gastric or intestinal ulcers, acute gastritis, IBS, panic attacks, migraine headaches, etc.

Types of people investigated	N. of subjects	**BHT, S**	BHT, s	Test conditions (order of actions just before BHT test)	%BHT for BHT[1]	Reference
Fit instructors	22	**46 s**	67 s	Full exhalation, normal inspiration	70%	Flack, 1920
Home defence pilots	24	**49 s**	72 s			
British candidates	23	**47 s**	69 s			
US candidates	7	**45 s**	66 s			
Delivery and	27	**39 s**	57 s			
Pilots trained for scouts	15	**42 s**	62 s			
Pilots taken off flying (stress)		**34 s**	49 s			
Normal subjects	30	**23 s**	58 s	Full inspiration	40%	Friedman, 1945
Neurocirculatory asthenia	54	**16 s**	40 s			
Normal subjects	22	**33 s**	45 s	Normal inspiration	73%	Mirsky et al, 1946 norm. inspir., alap,
Anxiety states	62	**20 s**	28 s			
Normal subjects, class 1 heart	16	**16 s**	48 s	Full inspiration, full exhalation, full inspiration	33.3%	Kohn & Cutcher, 1970
Class 2 and 3 heart patients	53	**13 s**	39 s			
Pulmonary emphysema	3	**8 s**	23 s			
Functional heart disease	13	**5 s**	15 s			
Normal subjects	6	**28 s**	76 s	Full exhalation, full inspiration	38 %	Davidson et al, 1974
Asymptomatic asthmatics	7	**20 s**	55 s			
Asthmatics with symptoms	13	**11 s**	27 s	Full inspiration	40 %	Perez-Padilla et al, 1989
Normal subjects	14	**25 s**	74 s	Deep breath of 50% O_2, 50% N_2[2]	33.3%	Zandbergen et al, 1992
Panic attack	14	**11 s**	34 s			
Anxiety disorders	14	**16 s**	49 s			
Outpatients	25	**17 s**	43 s	Full inspiration	40 %	Gay et al, 1994
Inpatients	25	**10 s**	25 s			
COPD, CHF (cong. heart	7	**8 s**	21 s			
12 heavy smokers	12	**8 s**	21 s			
Normal subjects	26	**21 s**	21 s	Normal exhalation	100%	Asmudson & Stein, 1994
Panic disorder	23	**16 s**	16 s			
Normal subjects	30	**36 s**	36 s	Normal exhalation	100%	Taskar et al, 1995
Obstructive sleep apnoea	30	**20 s**	20 s			
Normal subjects	76	**25 s**	67 s	Full exhalation, full inspiration	38%	McNally & Eke, 1996
Normal subjects	10	**38 s**	38 s	Normal exhalation	100%	Flume et al, 1996
Successful lung transplantation	9	**23 s**	23 s			
Successful heart transplantation	8	**28 s**	28 s			
Normal subjects	31	**29 s**	32 s	Normal exhalation in supine position	90%	Marks et al, 1997
Outpatients with COPD	87	**8 s**	9.2 s			

Table 1.2 Breath holding time according to various medical references

Table 1.2 comments.

- *1. "% of BHT for BHT" means the percentage of BHT used to calculate BHT.*
- *2. Zandbergen et al, 1992 conducted their experiments with the mixture of 50% O_2 and 50% N_2. According to Ferris with his colleagues (1945), such mixture increases normal BHT by about 50%.*

Analysing the results of Table 1.2, the following conclusions can be made.

- Normal subjects have the longer BHT (maximum pause) in comparison with sick people who suffer from various health problems.
- The stronger the severity of the health problem, the shorter the BHT.

- Recovering and asymptomatic people have intermediate BHT values.

Let us now turn our attention to the comments expressed by medical professionals about the breath holding time of healthy and sick people.

In 1919 The Lancet published one of the first articles describing the medical application of BHT investigated by military medical doctor and Lieutenant-Colonel Martin Flack (Flack, 1920). As Dr. Flack indicated, less than 35 s BHT was considered to be sufficient to take pilots "*off flying through stress*" (Flack, 1920). The possible reason for such a drastic measure was described by him on the next page. On one occasion a medical doctor wanted to suspend from flying one experienced pilot due to his unusually low BHT (23 s BHT). The pilot was allowed to fly, lost control, crashed the plane and was killed. The commanding officers decided that this test was, indeed, an indicator of the personal health state, especially stress. At the end of his publication Flack suggested, "*...that these tests would also be of value for measuring trench fatigue, industrial fatigue, and fatigue in women workers*..." (Flack, 1920).

According to Dr. Wood, who investigated patients with a variety of symptoms diagnosed as DaCosta's syndrome (one of the previous names for the chronic fatigue syndrome), low BHT was the most common symptom found in his 200 patients (Wood, 1941).

A few years later Dr. Friedman, Director of the Harold Brum Institute for Cardiovascular Research, San Francisco, after analysing his patients with neurocirculatory problems wrote, "*... the breathholding time was found to be directly related* [inversely proportional] *to the severity of the dyspnea suffered*" (Friedman, 1945).

Dr. Mirsky and his colleagues (1946) concluded that "*the difference* [in breath holding time] *between the normal and abnormal patients* [with variety of anxiety states] *is of clinical significance*".

Two American medical doctors, Robert Kohn and Bertha Cutcher, in their article "*Breath holding time in the screening for rehabilitation potential of cardiac patients*" (Kohn & Cutcher, 1970) described the testing of more than 100 cardiac patients. It was found that "*...an individual unable to hold his breath for at least 20 sec* [7 s BHT] *is a poor candidate for vocational rehabilitation*". Furthermore, "*It is now suggested that the determination of the breath-holding time is an effective screening test for rehabilitation potential*" (Kohn & Cutcher, 1970).

Apparently, healthy obese patients were "*unable to hold their breath much beyond 15 s*", whilst all normal non-obese subjects could breath hold for more than 30 s (Hurewitz et al, 1987).

Similarly, African researchers noticed that, *"Significant differences were observed in the mean of the Quetelet index, percent predicted vital capacity and the breath holding time between the normal female and the obese female subjects. A high but inverse relationship was found between estimated body fat and each percent predicted vital capacity and breath holding time in subjects whose Quetelet index was above 30 kg/m2"* (Sanya &Adesina, 1998)

A review of publications on leprosy (Katoch, 1996) revealed that "*respiratory function test studies have shown impaired breath holding time*" (abstract).

Authors of the article "*Rating of breathlessness at rest during acute asthma: correlation with spirometry and usefulness of breath-holding time*" (Perez-Padilla et al, 1989) wrote,

"*These results suggest that: 1) magnitude of dyspnea and breath-holding time correlate with severity of airflow obstruction in acute asthma attacks associated with dyspnea at rest; and 2) breath-holding time varies inversely with dyspnea magnitude when it is present at rest*" (abstract). Thus, BHT has correlation with the most important parameters officially accepted for the diagnosis of asthma.

Later Mexican scientists published the same result in their article, *Estimating forced expiratory volume in one second based on breath holding in healthy subjects*. Their conclusion was *"FEV1 [forced expiratory volume] can be reliably estimated using BHT"* (Nevarez-Najera et al, 2000).

Japanese doctors compared breath holding times for normal subjects and patients with COPD (chronic obstructive pulmonary disease). *"The period of no respiratory sensation [a certain period of no particular respiratory sensation which is terminated by the onset of an unpleasant sensation and followed by progressive discomfort during breath-holding] was also measured in eight patients with chronic obstructive pulmonary disease. The values of the period of no respiratory sensation in patients with chronic obstructive pulmonary disease were apparently lower than those obtained in normal subjects. These findings suggest that measurement of the period of no respiratory sensation can be a useful clinical test for the study of genesis of dyspnoea"* (Nishino et al, 1996).

Kendrick and colleagues used breath holding for more accurate measurements of pulmonary blood flow (Kendrick et al, 1989). It was important for testing that the subjects hold their breath as long as possible for better measurements. The researchers had 33 patients with cardiac problems (but without overt cardiac failure) and noticed that, "*for very dyspnoeic patients a breath-hold time of less than 10 s would be desirable...e.g., 6 s is acceptable.*

However, in very ill patients, even a 6 s breath hold time may be too long" (Kendrick et al, 1989). The authors were clearly disappointed by the short BHTs of their patients.

Magnetic resonance imaging (MRI) is a test in which X-ray films are taken of patients, who must remain motionless during the procedure. Patients must therefore be able to hold their breath for the duration of the test. Sick patients with numerous health problems have been a challenge for MRI professionals since these patients could not hold their breath a sufficiently long enough time in comparison with normal subjects.

For example, one abstract claimed that patients with coronary artery disease "*found it significantly more difficult to perform a steady breath-hold … or attain the same diaphragm position over multiple breath-holds than normal subjects*" (Taylor et al, 1999).

In order to solve this problem, new magnetic imaging techniques requiring shorter BHTs (even as short as a few seconds only) were developed. However, some, most seriously ill patients could not achieve even multiple 1 s breath holds, as reported by Posniak and colleagues (1994):

"*OBJECTIVE. Chest and abdominal CT scans using 1.0-sec scan times are often limited by motion in patients who are unable to hold their breath. With our scanner we can obtain images in 0.6 sec (partial scan)*" (abstract). The breathing pattern of some patients was so strong that they could not stop for even a single second.

Russian medical Doctor K.P. Buteyko and his colleagues tested thousands of patients with a variety of cardiac and bronchial problems and found that sick people usually have about 10-20 s BHT, and the very sick as low as 3-5 s. With approaching death, the breath holding time gradually, day after day, goes down: 5 s, 4, 3, 2, 1 (last frantic gasps for more air), death… (Buteyko, 1977).

Are there any health conditions in which BHT is long in spite of poor, but stable health? It is possible, according to my research, in such rare cases as obesity hypoventilation syndrome, chronic mountain sickness, after carotid body resection (these nervous cells monitor carbon dioxide concentration in the blood and brain and, as a result, control respiration), and curarisation of respiratory muscles (a procedure during which respiratory muscles are cut and cannot obey the central nervous system). Obviously, more research is required before final conclusions can be made.

We can see that the BHT (breath holding time after normal expiration) is an excellent indicator of our health. The sicker the person, the lower the BHT.

Finally it can be noted that these low BHTs were found for sick and severely sick patients. Apparently, there are certain people who have low BHTs (due to chronically heavy breathing), but are not diagnosed (yet?) with any serious organic disease.

1.13 Role of nitric oxide

There are numerous studies published over the past 80 years regarding the negative effects of hypocapnia (low level of CO_2). Hence, CO_2 is the most known and investigated factor that relates to overbreathing. Which other parameters of the body become abnormal during and because of hyperventilation?

Normal nasal breathing helps the body to use its own nitric oxide. This substance is produced, among other places, in nasal passages. During normal breathing, we have quiet prolonged exhalations (that do not prevent accumulation of nitric oxide in some areas of nasal passages) and relatively quick inhalations (that allow inhalation of the accumulated nitric oxide). Duirng hyperventilation exhalations are forceful and quick (as one can observe in many sick people) and inhalations are slow. This reversal of the main stages of breathing decreases the utilization of nitric oxide.

The roles and some important effects of this hormone on the body have been discovered very recently and there are still many questions in relation to this substance. Nitric oxide is found and synthesized in endothelial cells that line the lumen of blood vessels, neurons, and macrophages. As a gas, it is routinely found in nasal passages and measured in exhaled air. The known functions of the NO include:

1. **Vasodilation of arteries and arterioles** (and hence regulation of blood flow to tissues). In this respect, NO is similar to CO_2 acting on the smooth muscles of blood vessels.

2**. Regulation of binding and release of O_2 to haemoglobin**. This NO function is again similar to the CO_2 function known as the Bohr effect.

3. **Destruction of parasitic organisms, viruses, and malignant cells** by inactivating their respiratory chain enzymes in mitochondria.

4**. Inhibition of inflammation in blood vessels**.

5. **Neurotransmission**. Learning, memory, sleeping, feeling pain, and some other processes require NO for

transmission of neuronal signals. On the other hand, brain cells can probably be killed during a stroke due to excessive production of nitric oxide.

6. **Hormonal effects.** NO influences secretion from several endocrine glands. It stimulates the release of adrenaline from the adrenal medulla, pancreatic enzymes from the exocrine portion of pancreas, and Gonadotropin-releasing hormone from the hypothalamus.

Abnormal NO production and its availability are now associated with hypertension, heart failure, stroke, obesity, diabetes (both type I and II), atherosclerosis, rheumatism, aging, and dyslipidemias (particularly hypercholesterolemia and hypertriglyceridemia).

Currently there are numerous studies world-wide related to the role of NO in human health and diseased states. It is beyond the scope of this book to provide these studies.

Practice shows that possibly for some people some health improvements can be achieved mainly through the correction of one's breathing pattern, which can normalize production and utilization of nitric oxide, while CO_2 changes could be small. Hence, in these people nitric oxide can play, during some stages of breathing normalization, the leading role in health restoration.

1.14 Changes in the ANS (autonomous nervous system)

Most of the time, breathing is regulated by the ANS (autonomous nervous system). In healthy people movement of the diaphragm provides at least 75% of the changes in air volume in the lungs during inhalation at rest, as one may see in many medical textbooks (e.g., p. 312, Castro, 2000; p. 595, Ganong, 1995). Inhalation involves activation of the diaphragm (the initially dome-shaped diaphragm is stretched sideways and becomes more flat), and, hence, inhalation, as a process of muscular activation, is normally controlled by the sympathetic part of the ANS. Exhalation, in health, involves relaxation or passive recoil of the diaphragm (p. 314, Castro, 2000) indicating parasympathetic control of this part of the process. In normal conditions (12 breaths per minute) one breathing cycle (inhalation-exhalation) takes 5 seconds. Inhalation lasts about 2 s and exhalation about 3 s (p. 313, Castro, 2000; p.541, Straub, 1998).

Since an average person takes many thousands breaths every day, the parameters of his or her breathing can be considered as a window, through which certain disturbances in the ANS can be detected. Let us consider the typical breathing parameters of sick people.

The above studies in minute ventilation show that people with asthma and heart disease breathe about 2.5 times more air every minute (about 15 l/min instead of 6). How is it possible that they breathe so much? Such breathing rates are possible by breathing faster (not 12 times per minute, but 15-20 or even more times per minute) and deeper (up to 700-1,000 ml of air per breath instead of 500 ml as it should be in health). If a healthy person needs about 3 s to exhale 500 ml through the relaxation of the diaphragm, there is no way for a sick person to exhale more air (700-1,000 ml) in less time using only relaxation. Hence, sick people unconsciously apply muscular efforts to exhale air from the lungs at resting conditions. These muscular efforts need sympathetic control indicating that hyperventilation means abnormal control of this vital function (breathing) by the ANS. Moreover, this fast and deep breathing is usually, but not always, accompanied by chest breathing, when the rib cage, not the diaphragm, does the main job of air movement. Hence, hyperventilation also means abnormal innervations or dis-regulation of control of the breathing muscles by the ANS. Furthermore, practice shows that hyperventilation is usually accompanied by the reversal of the two phases of breathing: inhalations often become longer than exhalations. One may notice how sick people take a prolonged inhale and then the rib cage collapses to expel air with force and an audible noise. Finally, the breathing of sick people is often uneven and irregular with sighing, coughing, snorting, sneezing, etc.

All these abnormal processes take place 24/7 and they indicate pathologies in the functions of the ANS. The ANS, in its turn, regulates contractions of the heart, digestion, production of hormones and many other vital processes. It is logical to expect then that chronic overbreathing can lead to various health abnormalities through negative effects on the ANS, but too little research about these negative effects is currently available.

1.15 Focus on diseases

The modern Western approach to respiration is often based on the following understandings about breathing. "*Respiration is the total process of delivering oxygen to the cells and carrying away the by-product of metabolism, carbon dioxide*", or "*Respiration is the process of taking in oxygen from inhaled air and releasing carbon dioxide by exhalation*", or "*Respiration is the process by which animals take in oxygen necessary for cellular metabolism and*

release the carbon dioxide that accumulates in their bodies as a result of the expenditure of energy".

About a century ago leading world's physiologists had a different understanding about the role of breathing and CO_2 in human health (see Professor Yandell Henderson's quote above). First of all, it is the primary role of breathing to regulate CO_2 (not just to release this by-product). Secondly, while regulation of CO_2 is an important factor, there are many other functions of normal breathing. These other functions can be disturbed or disrupted. Possible abnormalities of breathing are: dominance of chest breathing at rest; fast shallow breathing and diaphragmatic flutter; slow inhalations and quick exhalations; periodic breathing; coughing; sighing; and sneezing. All these and many other irregularities and infringements are connected with pathological processes or abnormalities in the respiratory system, autonomous nervous system, endocrine system, musculoskeletal system, cardiovascular system, gastrointestinal and other systems of the human organism. Let us now review some diseases and their relations to breathing.

Asthma

In 1968 The New England Journal of Medicine published the results of a large study (McFadden, 1968) in which breathing and blood gases of a group of asthmatics were investigated. The researchers found that all 101 tested patients had chronic alveolar hyperventilation. Those asthmatics who had a light or moderate degree of the disease breathed about 15 l of air per min or 2.5 times more than the official medical norm (6 l/min).

More recently, in 1995, American researchers from the Mayo Clinic and Foundation (Rochester) confirmed the same average value (about 15 l/min) for another group of patients diagnosed with asthma (Johnson et al, 1995). This study was published in the *Journal of Applied Physiology*.

Finally, medical professionals from Mater Hospital in Brisbane (Australia) tested 39 asthmatics and found 14 l/min (Bowler et al, 1998). These figures were reported in the *Medical Journal of Australia*.

Clinical Science published in 1968 an article, *The mechanism of bronchoconstriction due to hypocapnia in man* (hypocapnia means abnormally low CO_2 concentrations). In this paper, Sterling explained that CO_2 deficiency causes an excited state of the cholinergic nerve. Since this nerve is responsible for the state of the smooth muscles in bronchi, its excited state leads to the constriction of air passages.

What about modern textbooks on physiology? One states, "*Agents that tend to dilate airways include increased $PaCO_2$ (hypoventilation or inspired CO_2)...*" (p.545, Straub, 1998). This textbook directly claims that slowing down breathing (hypoventilation) or increased CO_2 level dilates airways. Moreover, CO_2 is suggested as the chief chemical substance that promotes this effect.

Did anybody ever suggest before recent years the connection between asthma and ventilation? Doctor Buteyko proposed this link in the 1950s (his first official publications appeared in the 1960s), when he discovered the central role of overbreathing in the development and degree of asthma. (He and his colleagues also found that asthma patients got immediate relief from their asthma attack symptoms, if they practiced reduced breathing). Dr. Herxheimer independently suggested that low CO_2 was the cause of bronchial asthma in 1946 and 1952 (Herxheimer, 1946; 1952).

Let us consider the possible mechanism suggested by Doctor Buteyko. Low CO_2 values in the bronchi cause chronic constriction of airways (that happens in all people). In addition to this direct effect, chronic hyperventilation makes immune reactions abnormal. The immune system becomes too sensitive in relation to intruders from outside (coming with air or food), but weakens the responses to various pathogens, like viruses and bacteria. (Why? Hyperventilation is a defensive reaction and a part of the fight-or-flight response. Hence, hyperventilation indicates a state of stress, increased alertness and emergency for the whole organism, the immune system included. Hence, various intruders are to be attacked.)

The immune system becomes hypersensitive and seemingly innocent events (like breathing cold air or inhaling dust particles, dust mite proteins, cat proteins, tree pollen, etc.) can trigger an inflammatory response in the airways of asthmatics, enlargement of mast cells, excessive production of mucus, a sense of anxiety or panic, more hyperventilation, and further constriction of airways.

As a result, mucus makes air passages narrower (or even blocks some of them) creating a feeling of suffocation and causing asthma attacks. During an attack, an asthmatic may try to clear the mucus by coughing it out, but that further reduces CO_2 concentrations in the lungs and makes air passages narrower.

Heart disease

In 1995 the *British Heart Journal* published a study (Clark et al, 1995) done by researchers from the National Heart and Lung Institute in London. The breathing rate of all 88 heart patients at rest ranged from 10 to 18 l/min (or

about 2-3 times more than the norm).

In 2000, in a study from the *Chest* magazine, a group of American cardiac professionals revealed that patients with chronic heart failure had breathing rates in the range of 14 to 18 l/min (Johnson et al, 2000).

More recently, Greek doctors from the Onassis Cardiac Surgery Center in Athens recorded ventilation values ranging from 11 to 19 l/min for heart patients from their hospital (Dimopoulou et al, 2001).

These and many other similar results raise many questions. Are there any heart patients (with primary hypertension, angina pectoris, and other problems) who have normal breathing parameters? Does the normalization of breathing mean no symptoms and no disease for all heart patients? What are the details of interactions between breathing and heart disease? These questions will be discussed later.

Above-mentioned physiological effects, resulting from a CO_2 deficiency, influence the cardiovascular system.

- Low blood CO_2 values lead to the narrowing of small blood vessels (vasoconstriction of arteries and arterioles) in the whole body. That causes two problems. First, as a group of Japanese medical professionals found, in conditions of CO_2 deficiency, blood flow to the heart muscle decreases (Okazaki et al, 1991). Hence, heart tissue gets less oxygen, glucose and other nutrients. Second, since small blood vessels are the main contributors to the total resistance in relation to blood flow, CO_2 deficiency increases resistance to blood flow and makes the work of the heart harder.
- The suppressed Bohr effect, due to low CO_2 values in the blood, also reduces oxygenation of the heart muscle. That increases anaerobic metabolism and produces excessive amounts of lactic acid. Note that lactic acid is often implicated as a source of pain in any tissue. In the case of the heart, a person can suffer from angina or chest pain.
- The excited nerve cells in the heart (the cells that are called pacemakers) interfere with the normal synchronization and harmony in the working of the heart muscle. (The valves should open and close in proper time, much like a well-tuned engine.) Desynchronization can make the whole process of blood pumping less efficient or more energy- and oxygen-demanding possibly causing pathological adaptive changes in the heart tissue.
- Abnormal metabolism of fats leads, as Russian medical studies revealed, to increased blood cholesterol level in genetically-predisposed people. That condition gradually, over periods of weeks or months, produces cholesterol deposits on the walls of blood vessels. Such deposits can induce primary hypertension. As their published work suggests, the BHT has a linear correlation with the blood cholesterol level. These results are discussed later in more details.
- Chronic hyperventilation affects the normal utilization and conversion of essential fatty acids into prostaglandins causing changes in inflammatory responses and the malfunctioning of the immune system.
- Mouth breathing (at rest, during sleep, exercise, etc.) is an additional adverse stimulus. It further reduces aCO_2 and prevents normal absorption of nitric oxide (a hormone and powerful dilator of blood vessels) synthesized in the nasal passages while the main effect of taking nitroglycerine medication, in case of heart problems, is to provide the organism with additional nitric oxide.
- Since heart patients breathe 2-3 times more than the official norm, they usually have a more frequent and deeper breathing pattern. That must result in other breathing abnormalities, for example, chest breathing, as well as slow inhalations and quick exhalations. These irregularities indicate abnormal states of the autonomous nervous and musculoskeletal systems.

The father of cardiorespiratory physiology, Yale University Professor Yandell Henderson (1873-1944), investigated some of these effects about a century ago. Among his numerous physiological studies, he performed experiments with anaesthetized dogs on mechanical ventilation. The results were described in his publication *Acapnia and shock. - I. Carbon dioxide as a factor in the regulation of the heart rate*. In this article, published in 1908 in the *American Journal of Physiology*, he wrote, "*... we were enabled to regulate the heart to any desired rate from 40 or fewer up to 200 or more beats per minute. The method was very simple. It depended on the manipulation of the hand bellows with which artificial respiration was administered... As the pulmonary ventilation increased or diminished the heart rate was correspondingly accelerated or retarded*" (p.127, Henderson, 1908).

Which parts of the cardiovascular system are going to be most affected? That depends on genetic predisposition, life style and environmental factors. There are so many parameters that can adversely affect the normal work of the cardiovascular system. People are different. Some may get chronic heart failure, others high blood pressure, or stroke, or various abnormalities in the heart muscle.

Western experimental studies suggest that the following cardio-vascular problems can appear as a result of hyperventilation (courtesy of Peter Kolb, Biochemical Engineer, Australia):

- palpitations (Bass C, 1990; Cluff, 1984; Demeter & Cordasco, 1986; Lum, 1975; Magarian et al., 1983; Nixon, 1989; Sher, 1991)

- cardiac neurosis (Bass C, 1990; Cluff, 1984; Nixon, 1989)
- angina pain (Nixon, 1989)
- myocardial infarction (Nixon, 1989)
- Wolfe-Parkinson-White syndrome (Nixon, 1989)
- arrhythmias (Cluff, 1984; Demeter & Cordasco, 1986; Nixon, 1989)
- stenosis of coronary artery (Demeter & Cordasco, 1986; Nixon, 1989; Sher, 1991; Waites, 1978)
- tachycardia (Cluff, 1984; Lum, 1975; Nixon, 1989; Tavel, 1990)
- failure of coronary bypass grafts (Nixon, 1989)
- right ventricular ectopy (Nixon, 1989)
- silent ischemia (Nixon, 1989)
- elevated blood pressure (Nixon, 1989)
- flat or inverted ECG T-wave (Demeter & Cordasco, 1986; Nixon, 1989; Sher, 1991; Tavel, 1990)
- vasoconstriction (Cluff, 1984; Demeter & Cordasco, 1986; Lum, 1975; Nixon, 1989; Sher, 1991)
- reduced cerebral blood flow (Cluff, 1984; Lum, 1975; Magarian et al., 1983; Sher, 1991; Waites, 1978)
- mitral prolapse (Bass C, 1990; Cluff, 1984; Nixon, 1989; Tavel, 1990)
- low cardiac output/stroke volume (Waites, 1978).

Do you know that it is possible to get abnormal ECG tracing from a healthy heart just by voluntary heavy breathing? Later, many cardiac professionals, while analyzing such ECGs, can claim pathological changes in the heart. These changes are different in different people. Vice versa, normal breathing naturally eliminates, either immediately or in due course of time, various, already detected, ECG abnormalities.

Modern medicine and physiology have a very limited understanding of what is going on with the cardiovascular system when breathing gradually change in one or the opposite direction. There are many questions related to individual variability, mechanisms of developing pathologies, and the interaction of hereditary and environmental factors.

Cancer

Let us consider some facts about the appearance, growth and development of malignant tumours; their spread to distant tissues and resistance to standard methods of treatment. What is the abnormal background, which is rarely discussed in popular books and articles about cancer, but which is known to professional oncologists?

It has been known for decades that malignant cells normally and constantly appear and exist in any human organism due to billions of cell divisions and mutations. These abnormal cells, in normal conditions, are quickly detected by the immune system and destroyed. However, the work of macrophages, enzymes and other agents of the immune system is severely hampered under the conditions of hypoxia. That was the conclusion of various studies. For example, Dr. Rockwell from Yale University School of Medicine studied malignant changes at the cellular level and wrote, *"The physiologic effects of hypoxia and the associated micro environmental inadequacies increase mutation rates, select for cells deficient in normal pathways of programmed cell death, and contribute to the development of an increasingly invasive, metastatic phenotype"* (Rockwell, 1997). The title of this publication is *"Oxygen delivery: implications for the biology and therapy of solid tumors"*.

Summarizing the results of numerous studies, Ryan with colleagues chose the following title of their article, *"The hypoxia inducible factor-1 gene is required for embryogenesis and solid tumor formation"* (Ryan et al, 1998).

In normal conditions, even a group of hypoxic cells dies (or is easily destroyed by the immune cells). What about cells in malignant tumours? Researchers from Gray Laboratory Cancer Research Trust (Mount Vernon Hospital, Northwood, Middlesex, UK) concluded,

"Cells undergo a variety of biological responses when placed in hypoxic conditions, including activation of signalling pathways that regulate proliferation, angiogenesis and death. Cancer cells have adapted these pathways, allowing tumours to survive and even grow under hypoxic conditions..." (Chaplin et al, 1986).

Moreover, American scientists from Harvard Medical School noted that *"... Hypoxia may thus produce both treatment resistance and a growth advantage"* (Schmaltz et al, 1998).

There is so much professional evidence about fast growth of tumours in the condition of severe hypoxia, that a large group of Californian researchers recently wrote a paper *"Hypoxia - inducible factor-1 is a positive factor in solid tumor growth"* (Ryan, 2000). As an echo, a British oncologist from the Weatherhill Institute of Molecular Medicine (Oxford) went further with a manuscript *"Hypoxia - a key regulatory factor in tumour growth"* (Harris, 2002).

When the solid tumour is large enough and the disease progresses, cancer starts to invade other tissues. This

process is called metastasis. Does poor oxygenation influence it? *"...Therefore, tissue hypoxia has been regarded as a central factor for tumor aggressiveness and metastasis"* (Kunz & Ibrahim, 2003) was the conclusion of German researchers from University of Rostock and University of Leipzig.

Since dozens of medical and physiological studies yielded the same result, what about just a title again? *"Tumor oxygenation predicts for the likelihood of distant metastases in human soft tissue sarcoma"* (Brizel et al, 1996). The harder one breathes, the faster cancer invades.

Probably, the reader now can guess about the effect of cancer treatment and the chances of survival for those who suffer from severe chronic hyperventilation. Indeed, *"... tumour hypoxia is associated with poor prognosis and resistance to radiation therapy"* (Chaplin et al, 1986).

"Low tissue oxygen concentration has been shown to be important in the response of human tumors to radiation therapy, chemotherapy and other treatment modalities. Hypoxia is also known to be a prognostic indicator, as hypoxic human tumors are more biologically aggressive and are more likely to recur locally and metastasize" (Evans & Koch, 2003).

"Clinical evidence shows that tumor hypoxia is an independent prognostic indicator of poor patient outcome. Hypoxic tumors have altered physiologic processes, including increased regions of angiogenesis, increased local invasion, increased distant metastasis and altered apoptotic programs" (Denko et al, 2003).

Could breathing influence the tumors and if so, how? The authors of one of the studies cited above mused about the origins of all these problems, "*Surprisingly little is known, however, about the natural history of such hypoxic cells*" (Chaplin et al, 1986). Why could they appear? What is the source of tissue hypoxia? We can again suggest that our breathing does influence the breathing process of all body tissues, tumours included.

Is there any experimental evidence indicating the usefulness of CO_2 for malignant tumours?

During the last decade, there has been a steady progress in the investigation and application of CO_2-O_2 gas mixtures called "carbogen" in clinical practice. Carbogen breathing is usually applied for several hours during administration of certain anti-cancer medications. Let us review some results in this area and the reasons for carbogen application.

Several studies from England and the USA found that breathing various carbogen mixtures significantly improves oxygenation of tumours. The general opinion of these researchers is that *"Perfusion insufficiency and the resultant hypoxia are recognized as important mechanisms of resistance to anticancer therapy. Modification of the tumour microenvironment to increase perfusion and oxygenation of tumours may improve on the efficacy of these treatments..."* (Powell et al, 1997).

A large group of British scientists from the Paul Strickland Scanner Centre revealed that when 14 cancer patients breathed various carbogen mixtures (with 2%, 3.5% and 5% CO_2 content, the rest was O_2) *"arterial oxygen tension increased at least three-fold from basal values"* (Baddeley et al, 2000). They also found that *"There were no significant changes in the respiratory rate, heart rate and blood pH. The results suggest that 2% CO_2 in O_2 enhances arterial oxygen levels to a similar extent as 3.5% and 5% CO_2 and that it is well tolerated"* (Baddeley et al, 2000).

Another group of British researchers directly measured oxygen pressure in cancer cells and concluded, *"This study confirms that breathing 2% CO_2 and 98% O_2 is well tolerated and effective in increasing tumour oxygenation"* (Powell et al, 1999).

These results generate the following question. Which gas, CO_2 or O_2 is the main contributor to increased oxygenation and by how much? The amounts of both gases in mixtures were much higher than the amounts of O_2 and CO_2 in normal air.

Let us, first, consider the influence of O_2. There are two O_2 states namely O_2 that is combined with haemoglobin and dissolved O_2, which can increase oxygenation of the arterial blood. As we considered in Chapter 1, the saturation of haemoglobin with O_2 under normal conditions (or when breathing normal air) is about 98%. Increased O_2 pressure can raise this value to almost 100%. This would cause about a 2% increase in arterial blood in comparison with the initial value. In addition, when patients breathe carbogen mixtures more O_2 can be dissolved in the arterial blood (this O_2 is not bound to red blood cells). In normal conditions the contribution of dissolved O_2 is about 1.5% of the total blood O_2 as the remaining 98.5% O_2 is combined with haemoglobin. Increasing O_2 almost five times increases total arterial O_2 content by about 6% in relation to the initial normal value.

Hence, increasing the O_2 component in breathing air (up to almost 100%) causes about 8% increase in total O_2 content in arterial blood.

Similarly, British researchers, as mentioned above reported *"arterial oxygen tension increased at least three-fold from basal values"* (Baddeley et al, 2000). How was it possible to get such a large increase in tissue oxygenation

(about 200%) if arterial blood could carry only about 8% more O_2 during carbogen breathing in comparison with initial conditions?

The remaining increase in tissue oxygenation could be due to larger CO_2 values, which shift the Bohr curves down and enhance O_2 release from haemoglobin cells, and due to the dilation of blood vessels. Therefore, the increase in oxygenation of tissues is mainly due to the larger CO_2 content.

Indeed, the British professionals decided "*to assess the relative contributions of carbon dioxide and oxygen to this response and the tumour oxygenation state, the response of GH3 prolactinomas to 5% CO_2/95% air, carbogen and 100% O_2*" (Baddeley et al, 2000). That was done using magnetic resonance imaging and PO2 histography. They found that,

"A 10-30% image intensity increase was observed during 5% CO_2/95% air breathing, consistent with an increase in tumour blood flow, as a result of CO_2-induced vasodilation, reducing the concentration of deoxyhaemoglobin in the blood. Carbogen caused a further 40-50% signal enhancement, suggesting an additional improvement due to increase blood oxygenation. A small 5-10% increase was observed in response to 100% O_2, highlighting the dominance of CO_2-induced vasodilation in the carbogen response" (Baddeley et al, 2000).

It is not oxygen, but carbon dioxide that is the substance responsible for the main improvement in oxygenation of tissues.

Diseases of the brain and the central nervous system

Physiology and medicine teach us that a CO_2 deficiency produces the following abnormalities in the nerve cells:

- Increased excitability of all nerve cells. We are too excited when we hyperventilate since overbreathing "*leads to spontaneous and asynchronous firing of cortical neurons*" (Huttunen et. al., 1999).
- Reduced blood flow to the brain. Our brains get less blood supply. This physiological fact can be found in many textbooks. As Professor Newton from the University of Southern California Medical Center recently reported, "*cerebral blood flow decreases 2% for every mm Hg decrease in CO_2*" (Newton, 2004). That means that with each second decrease in the BHT, blood flow to the brain is less by almost 1%. Less blood means a decreased supply of glucose (the main fuel for the brain in normal conditions), oxygen, and other nutrients. In addition, it causes gradual accumulation of waste products. All these effects affect all aspects of human performance, including concentration, coordination, memory, logic, etc.
- The suppressed Bohr effect. Not only is the inflow of oxygen less, but also oxygen release from red blood cells is hampered by low CO_2 concentrations in tissues. That further reduces brain oxygenation.

In addition, there are similar negative effects due to likely abnormalities with production and delivery of nitric oxide causing hypoxia, lowered blood perfusion, faulty transmission of signals, and inflammation. Imbalances in the ANS and hormonal system are other destabilizing factors. It is likely that there are other effects of abnormal breathing on the nervous system. Hyperventilation is virtually always manifested in abnormal breathing patterns, including a higher frequency of breathing, shorter exhalations and inhalations, absence of periods of no-breathing, abnormalities in the work of respiratory muscles (e.g., chest breathing), etc.

Do clinical studies show that patients with mental or psychological problems have heavy breathing?

In 1976 the *British Journal of Psychiatry* published a study of CO_2 measurements in 60 patients with neurotic depression and non-retarded endogenous depression (Mora et al, 1976). All patients had abnormally low carbon dioxide values.

Later, in 1990, American psychiatrists from Hunter College (City University of New York) reported results from several groups of subjects with anxiety, panic phobia, depression, migraine, and idiopathic seizures (Fried, 1990). The abstract states "*virtually all the non-control subjects were found to show moderate to severe hyperventilation and accompanying EEG dysrhythmia*". In addition, it notes that hyperventilation and abnormal electrical signals in the brain took place simultaneously.

Canadian scientists from the Department of Psychiatry (University of Manitoba, Winnipeg) measured carbon dioxide concentrations in over 20 patients with panic disorder. Their average CO_2 was also below the medical norm (Asmundson and Stein, 1994). There are many other studies that report abnormally low CO_2 values for people with various psychological and neurological problems.

It has been known in neurology for over 50 years that poor oxygenation and reduced blood supply of the brain are the foundations of virtually all "mysterious" and known neurological and psychological pathologies, ranging from insomnia, depression, addictions and phobias to Parkinson, Alzheimer, and senile dementia. Indeed, if one considers

the places and people who tried breathing retraining (Chapter 4), almost all of them relate to psychology and neurology.

GI (gastrointestinal) problems

How hyperventilation affects the GI system?

- Small blood vessels in the digestive organs get constricted. That reduces their blood supply. Physiological measurements confirm this effect on the stomach, liver, spleen, and the colon. Hence, GI organs get less oxygen, glucose, and other nutrients for their normal work and repair.
- The suppressed Bohr effect, due to low CO_2 values in the blood, reduces the oxygenation of the digestive organs.
- The excited state of the nerve cells in the GI system (the enteric nervous system that orchestrates the normal work of the whole digestive conveyor) interferes with the normal work of the GI organs. That can influence the contraction of the muscular layers, the production and secretion of digestive enzymes and other functions. Indeed, a group of American gastroenterologists from the Mayo Clinic in Rochester recently published a study *Hyperventilation, central autonomic control, and colonic tone in humans* (Ford et al, 1995). They tested the effects of voluntary over-breathing with normal and CO_2-rich air. A drop in the CO_2 level of the blood (hyperventilation) caused abnormalities in the contractility and peristalsis of the colon.
- Chronic hyperventilation can cause autoimmune GI reactions since it is not normal to breathe two-three times the norm 24/7. The immune system, as in case of asthma, can start searching for enemies coming from outside (i.e., with food). This can contribute to the pathology of inflammatory bowel disease, irritable bowel syndrome, Crohn's disease and other problems and complaints.

Hyperventilation can create numerous abnormalities in the GI system. There are no studies that compare these effects or define the individual differences. Similarly, the impact of permanent changes in breathing is also not investigated.

Hormonal problems

How do people with hormonal (endocrine) problems breathe? A group of Italian medical researchers from the University of Ancona reported that 28 patients with diabetes breathed from 10 to 20 l/min (Tantucci et al, 1997). A year later German endocrinologists from Gutenberg University Hospital (Mainz) tested 42 people with hyperthyroidism and found 15 l/min (Kahaly et al, 1998).

How can deep breathing cause hormonal or endocrine problems? Since hyperventilation is a state of emergency for the whole body, it can interfere with the normal production and secretion of various hormones. For example, the immediate effects of stress include surges of adrenalin and cortisol. Chronic hyperventilation often leads to gradual development of deficiencies in these hormones. In addition, all tissues, hormonal glands included, suffer from reduced oxygenation and blood supply. Above-mentioned abnormalities with nitric oxide production and its bioavailability directly cause problems with several hormones (see above). The function of the ANS is compromised in conditions of overbreathing creating another cascade of imbalances in the circardian cycles regulated by these hormones. However, there were no systematic studies that identified the long-range hormonal changes due to heavy breathing. Individual differences, together with life style and environmental parameters, should play their role when it comes to expected effects.

Other health concerns and summary

What about Western research concerning the breathing of people with various other problems? There were few studies relating the quality of breathing to other health problems. However, medical science knows little or nothing about the breathing/disease interaction for many common health problems like cancer, arthritis, diabetes, etc. That especially relates to the situations when breathing parameters gradually change.

What other diseases are related to abnormal breathing? Breathing regulates blood supply and oxygenation of all cells, tissues and organs. Breathing also reflects the state of the autonomous nervous system that regulates the work of all body organs. In conditions of chronic hyperventilation vital organs suffer from reduced blood supply and hypoxia. In addition, chronic hyperventilation interferes with the normal work of the nervous and immune systems. Hence, a wide variety of negative effects is present when we breathe too much.

Furthermore, if reduced blood and oxygen supply, together with abnormalities of the immune and nervous systems, are part of the main problem, then breathing can play a role in the further development of this health problem.

There is some limited but encouraging practical evidence about the healing influence of normalization of breathing on a variety of health conditions.

What diseases are not related to chronic hyperventilation? People with, for example, color-blindness lack certain structures in the retina of their eyes. Whatever their breathing patterns, there are no known cases of the appearance or disappearance of this medical condition. Likely, breathing has nothing to do with this problem. Hemophilia is usually manifested in the absence of one blood clotting substance. Again, this problem is purely genetic and unrelated to breathing.

There are over 30,000 various health problems and abnormalities known to modern medicine. Doctor Buteyko and his Soviet (Russian) medical colleagues, based on their clinical experience with over 200,000 patients, hypothesized that about 150-200 health conditions are connected with abnormal breathing. Hence, less than 1% of all health problems might be affected by our breathing. However, many of these health problems are fairly common for modern people. This, for example, relates to our main killers, like heart disease, cancer, and many others.

1.16 Why breathing?

Patients with modern degenerative diseases usually have many dozens of physiological and biochemical parameters which seen to be abnormal. For critically ill patients this number is much larger and is often estimated to be many hundreds. That means that the concentrations of numerous minerals, vitamins, hormones and many other substances are out of their norms. Why, then, are breathing in general and CO_2 related parameters in particular chosen for our consideration?

Based on still limited studies, when breathing is normal, many diseases are absent. When people are sick, they over-breathe. Even partial normalization of their breathing results in better health, as we saw above and are going to consider later. What exact role breathing plays in the pathology of various diseases is a very big and hard question that needs further studies and trials.

The unique position of breathing among many other health parameters is due to its semi-automatic nature. Professor Ronald Ley, State University of New York, recently wrote a large review "*The modification of breathing behavior*" starting with the statement, "*Breathing is the only vital function under direct voluntary control as well as involuntary control*" (Ley, 1999).

Most of the time the "breathing centre" governs human respiration by keeping minute ventilation, carbon dioxide and oxygen concentrations, and breath holding time or the BHT (breath holding time after normal expiration) within relatively narrow ranges at rest. Meanwhile, people can voluntarily change many breathing parameters. For example: minute ventilation, breathing frequency, and tidal volume (the amount of air taken in per breath) can be decreased or increased by at least a factor of two. The duration of such wilful breathing can be sustained for many minutes or even hours every day. Thus, breathing, to some degree, can be controlled in the short run by human willpower and, over long periods of time, can be normalised through self-discipline and persistence. Moreover, there are numerous life style and environmental factors (to be discussed) that directly influence breathing and that can be adjusted to meet the needs of the human organism.

A person cannot directly order the heart to slow down, the air passages to open, the kidneys and liver to intensify their work and cleanse the blood of pathogens, or any spasm to disappear. However, these and many other problems can be solved by breathing exercises, as will be discussed later.

1.17 Evolution of air on Earth

How is it possible that a human being, one of the smartest species on Earth, can kill itself, and over 90% people die this way, by over-breathing? Is it nature so silly to create this way? In order to answer these questions we need to consider changes in air composition on Earth.

When there were no life on Earth, air has no oxygen (since oxygen is a very reactive substance), while CO_2 was a part of the volcanic gases that formed air during those times. Geological studies suggest that CO_2 concentration was up to 10-12% or even more. Thus, when the first organic substances and life forms appeared on Earth (from about 5 billion to 1 billion years ago), our atmosphere did not have any measurable amounts of O_2, according to Professor Maina (Maina, 1998), who wrote the book *The gas exchangers: structure, function, and evolution of the respiratory processes* about development of respiration and breathing in various creatures living on Earth in the past and now. He is one of the leading modern authorities on respiration of different life forms.

Appearance of the first vertebrates (about 550 millions years ago) and the development of prototypes of human

lungs took place when air was made up of only about 1% O_2, while having much higher percentage of CO_2 (Maina, 1998), likely over 7%. Normal air today has many times more O_2 (about 20%) and only a fraction of the CO_2 (0.03%). However, our cells now still live in the air that existed hundred millions years ago: *"But the cells of animals and humans need about 7 % CO_2 and only 2% O_2 in the surrounding environment. This is the way how our cells live: cells of the heart, brain, and kidneys"* (Buteyko, 1977).

Hence, most of the time our lungs were developing and evolving in conditions when the CO_2 content was high (up to 7-12% during the first stages of development), with gradual decline, and low O_2 values (about 1% or less during the first stages). During these stages the process of control of breathing by the nervous system was also developed. Since this primitive air had very little O_2, our evolutionary predecessors could get more oxygen in tissues by breathing more. Since any stressful situation, digestion, search for food, mating, playing, and any other activity required more oxygen, hyperventilation became the fundamental reflex or instinct. Only totally peaceful stress-free rest had low metabolic rate where heavy breathing would not give any advantage for survival.

On the other hand, however heavy was the breathing of these primitive creatures in the past, they would still get the main nutrient, CO_2, from air. The CO_2 content in tissues had to be even higher than in air and these creatures would never develop spasms of coronary vessels, bronchi, other smooth muscles, or abnormal excitability of the nerve cells, or muscular tension or any other above-mentioned negative effects. Hence, nature did provided primitive creatures with ability to function without all above-discussed physiological flaws.

However, the main parameter of our environment, our air, had dramatic change during later stages of our evolution due to advance of green life that transforms CO_2 into O_2 during photosynthesis. These events could be reflected on the following picture.

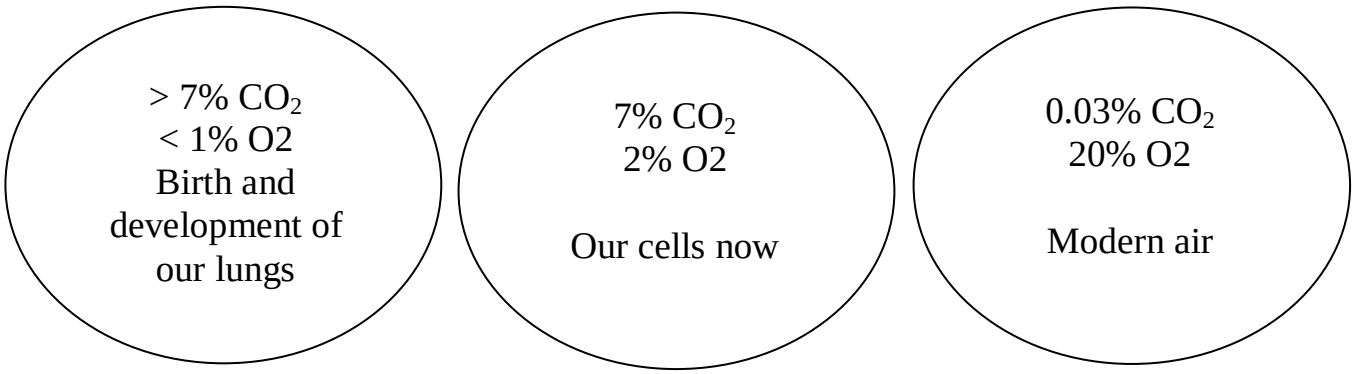


Fig 1.1 CO_2 and O_2 values in air during early stages of development of our lungs, in our cells now and in modern air.

We can see that air had dramatic change during evolution. It now has too much oxygen and almost no CO_2. Hence, the chief parameter of our environment (we can survive for days or weeks with no water or food, but only for minutes with no air) became abnormal in its composition. It is only existence of our lungs that protected us from extinction. Nature could not anticipate this cardinal change in air, but it did provide us with the means for survival.

Conclusions

- Many sick people with modern degenerative health conditions chronically breathe 10-25 l/min, or 2-5 times more than the physiological and medical norms (about 5-6 l/min).
- Such chronic over-breathing usually reduces CO_2 stores in the organism causing, according to physiological laws and studies, the following consequences:

- hypoxia of all cells and organs (especially of the brain and heart);
- local constriction of arteries, arterioles, and capillaries, leading to poor blood perfusion in the heart, brain and other vital organs;
- tension and irritability in smooth muscles;
- excessive excitability of many brain areas and other parts of the nervous system;

- constriction of air passages;
- abnormal changes in blood pH and electrolytic composition of each human cell;
- abnormal biochemical reactions involving vitamins, minerals, amino acids, lipids, hormones, carbohydrates and other vital substances.

• Medical studies have revealed personal variability of physiological and psychological symptoms due to chronic hyperventilation.

• Acute over-breathing, which is produced by the hyperventilation provocation test, often reproduces the symptoms of the main health problem.

• There are only a few, rare health conditions, which are characterised by breathing less than the norm.

• Since over-breathing causes low CO_2 concentrations in the exhaled air, results of such measurements revealed that the carbon dioxide level is low for different groups of sick people, and dangerously low in the severely sick.

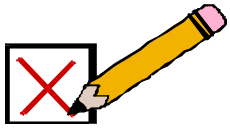
• Breathing of critically ill patients is usually visible, audible, and corresponds to very low carbon dioxide stores and heavy hyperventilation.

• BHT (breath holding time) or the BHT (breath holding time after normal expiration) is a simple and clinically significant indicator reflecting the individual health state of people with asthma and heart disease.

• Breathing has a unique position among other vital physiological functions due to the human ability to control it.

• During early millennia of evolution of the lungs, hyperventilation, as a reaction to psychological, physiological, chemical, bacteriological, viral and any other stress, became the most fundamental reflex of the human organism.

• Despite of dramatic change in air composition during evolution of life on Earth and despite of our ability to kill ourselves just by heavy breathing, Nature provided us with the means of survival: our lungs.



Q&A section for Chapter 1

Q: What were the historical origins of concerns about the dangers of CO_2?

A: In the 1780s, French scientist Antoine-Laurent Lavoisier determined the composition of air. He also discovered the mechanism of gas exchange during respiration and burning. Oxygen is consumed for the production of energy and carbon dioxide is expelled as an end product. In his classical experiments, mice died in a closed glass jar in an atmosphere containing large quantities of carbon dioxide and almost no oxygen. A candle also quickly expired in such air.

That was probably the time when a superficial understanding of respiration produced the idea that carbon dioxide was a "*toxic, waste and poisonous*" gas while oxygen brought life and vigor. "*Take a deep breath*", "*Breathe more air, it is good for your health*", "*Breathe deeper, get more air into your lungs, we need oxygen*", etc. became popular phrases for which there is no scientific basis. Even now, some scientific publications contain such misleading sentences, as "*Respiration is the process of oxygen delivery.*"

Professor Yandell Henderson gave the following explanation of this ignorance, "*Likeness of Life to Fire. - Lavoisier's supreme contribution to science, and particularly to physiology was the demonstration that, in their broad outlines, combustion in a fire and respiratory metabolism in an animal are identical. Both consist in the union of oxygen from the air with carbonaceous material: and both result in the liberation of heat and the production of carbon dioxide…*

The human mind is inherently inclined to take moralistic view of nature. Prior to the modern scientific era, which only goes back a generation or two, if indeed it can be said as yet even to have begun in popular thought, nearly every problem was viewed as an alternative between good and evil, righteousness and sin, God and the Devil. This superstitious slant still distorts the conceptions of health and disease; indeed, it is mainly derived from the experience of physical suffering. Lavoisier contributed unintentionally to this conception when he defined the life supporting character of oxygen and the suffocating power of carbon dioxide. Accordingly, for more than a century after his death, and even now in the field of respiration and related functions, oxygen typifies the Good and carbon dioxide is still regarded as a spirit of Evil. There could scarcely be a greater misconception of the true biological relations of these gases…" (Henderson, 1940).

Q: How did the parameter "40 mm Hg CO_2" appear in textbooks?
A: This number is important because it is present in all main physiological textbooks used nowadays by western students. This is the current medical norm for CO_2 content in alveoli and the arterial blood. The number was established about a century ago by the famous British physiologists Charles G. Douglas and John S. Haldane from Oxford University. Their results were published in the article *The regulation of normal breathing* by the *Journal of Physiology* (Douglas & Haldane, 1909). The investigators analysed arterial blood gases of staff members at Oxford University, including scientists and support personnel, and found the average for the group. It is possible to argue that even during those times many University workers had a sedentary life style with little physical activity. Hence, their CO_2 concentrations could be lower than those for most healthy people a hundred years ago. Indeed, another old study by the also famous Karl Albert Hasselbalch had about 46 mm Hg aCO_2 as the average value for volunteers at rest (Hasselbalch, 1912). Doctor Buteyko suggested about the same value to be the norm for people in good health.

Q: How many people have normal breathing?
A: If we accept medical standards (6 l/min for ventilation, as in most medical and physiological textbooks, and 40 s for the BHT), only a small percentage (less than 10%) of the population satisfies this criterion. Experience shows that on average, only a few, if any, per 1,000 people have breathing with Doctor Buteyko's norm (60 s BHT or more).

Q: How much oxygen is retained in the human organism? In other words, are we efficient in oxygen extraction from air?
A: Typical patients with asthma and heart disease breathe about 15 l/min at rest and have about 15 s BHT. They utilize or absorb only about 10% of inhaled oxygen, the remaining 90% is exhaled back in air. People, who are considered normal by medical standards (6 l/min and 40 s BHT) retain only about a quarter (25%) of the oxygen that they inhale. Their lungs are more efficient at extracting oxygen. Those healthy people, who breathe in accordance with Buteyko's norm (4 l/min; 60 s BHT), can extract up to 30-35% of the oxygen they inhale. People with over 3 min BHT (hatha yoga masters. Dr. Buteyko and many of his colleagues, etc.) would have about 2 l/min for minute ventilation and retain up to 60% of inhaled oxygen.

Q: Which body parts or tissues are particularly sensitive to tissue hypoxia? In other words, how long can various organs and tissues survive without oxygen?
A: The time of survival will relate to initial oxygenation (reflected in the breath holding time) and existing pollution of tissues. This table from the British Medical Journal (Leach & Treacher, 1998) reflects tolerance to hypoxia of various tissues for an ordinary person.

Tissue	Survival time
Brain	< 3 min
Kidney and liver	15-20 min
Skeletal muscle	60-90 min
Vascular smooth muscle	24-72 hour
Hair and nails	Several days

Q: Some people claim that over-breathing can help the organism to "expel toxins". Is this opinion correct?
A: Although some medical and physiological textbooks on respiration state that unwanted substances can be removed from the organism through the air passages, their quantities are small. In addition, over-breathing or hyperventilation is unlikely to be useful due to greatly decreased blood supply to other organs of elimination, which will then function less efficiently. Moreover, poor blood supply to the tissues is going to diminish the rate at which these substances are collected by body fluids and eliminated.

Meanwhile, normal breathing (about 6 l/min), in addition to the described normalisation of body physiology, means that smaller amounts of polluted air, smoke, dust, etc. are taken in to the organism through the lungs.

Q: Does deep breathing help to deliver more fresh air to poorly ventilated parts of the lungs filled with old stale air?
A: Often people also ask, "Is it true that, if I breathe little, I do not exercise my lungs and can develop some lungs

problems?" Vice versa. All people with asthma, emphysema, bronchitis, and many other problems are heavy breathers. They need CO_2 to heal their lungs. In addition, people with heavy or deep breathing are often chest-breathers since the smooth muscle of the diaphragm is in the state of spasm. Hence, their lower layers of the lungs get much less, if any, fresh air. Normal breathing is diaphragmatic allowing homogeneous inflation of the whole lungs with fresh air, similar to what happens in the cylinder of a car due to the movement of the piston.

Q: Can a few deep breaths or sighing relieve tension in the chest?
A: During the first of several deep breaths, not only are all alveoli in the lungs greatly expanded providing more oxygen for all tissues, but also any tightness in the chest muscles can be temporarily relieved, due to their stretching and subsequent relaxation. Periodic sighing (a typical symptom of diabetics, CFS sufferers, cardiac patients, asthmatics, etc.) is an example of chest tension relief, but such deep breaths also remove more CO_2, first, from the lungs, and finally, from all cells.

As a result, any beneficial effects of deep breathing are very short-lived. Moreover, lowered CO_2 levels lead to worsening of the problems which deep breathing was intended to solve causing: 1) further bronchoconstriction, up to partial or total closure of some lung areas and less effective gas exchange; 2) more muscular tightness due to increased hypoxia, excessive excitability and tension in the chest and other muscles, constriction of arteries and capillaries, and certain other physiological disorders discussed above.

Thus, the temporary relief provided by periodic deep breaths or sighing can become a part of the vicious circle. It is no surprise that various medical professionals, authors of the already cited publications, viewed sighing as a clear symptom of the chronic hyperventilation syndrome.

Q: How does breathing affect the quality of sleep?
A: A normal person needs about 5-6 hours of sleep. He falls asleep within a few minutes, sleeps the whole night in the same position without awakening, does not remember his dreams and wakes up fully refreshed. That corresponds to normal breathing and normal breath holding time (about 40 s).

A typical asthmatic with 15 l/min ventilation and about 15 s BHT tends to have 8-10 hours of sleep. He is likely to need some 5-20 minutes to fall asleep. During the night he can awaken, get anxious, change positions, have dreams, etc. In spite of the long period of sleep, he may still feel tired in the morning. How and why are these abnormalities possible?

As mentioned above, hyperventilation "*leads to spontaneous and asynchronous firing of cortical neurons*" (Huttunen et. al., 1999). This phrase, from the professional magazine *Experimental Brain Research*, has very serious personal and even social ramifications (as we are going to see in Chapter 9).

For example, when this asthmatic goes to sleep he has thoughts, which are self-generated by his brain in spite of his conscious attempts to calm down, relax, put everything aside, etc. These *"spontaneous and asynchronous"* thoughts often cause problems with falling asleep.

Let us consider the duration of sleep. Two main known physiological purposes of sleep are to give rest to the brain (especially to cortical areas) and the muscles. The normal person, due to normal aCO_2 concentrations, has had a relaxed, easy attitude, with normal perception during the whole day. He has experienced less stress (since stress in modern people is mainly due to distorted attitudes to outer events and stimuli, not due to life-threatening situations). His muscles have been relaxed (again due to carbon dioxide). Hence, he needs only 5-6 hours of sleep.

The asthmatic, due to chronic hyperventilation, has had tense muscles and over-excited brain during the whole day. Normally, he needs more time for sleep in order to relax and rest his muscles and brain.

Moreover, severely sick and hospitalised people with 5-8 s BHT may need up to 12-14 hours of sleep, usually of miserable quality: with frequent awakenings, changed body positions, dreams, nightmares, etc. The causes are the same: tense muscles and "*spontaneous and asynchronous firing of cortical* [and other] *neurons*".

Certain practical evidence and hatha yoga studies also have found that, when breath holding time is about 1 minute, people need on average only about 4 hours of sleep, while for 2-3 minutes BHT, 2 hours of sleep is sufficient. In my view, that corresponds to the way Nature designed the human organism.

The relationships between sleep and breathing will be considered in more detail later.

Q: Are concentration and other mental skills (like logic, analytical abilities, memory, etc.) similarly affected and why?
A: We know from above, that brain blood flow is proportional to aCO_2. In addition, brain oxygenation is impaired in

such conditions due to the Bohr effect. Both factors produce predictable effects on all our senses and communication within the nervous system. At some moments of time, these "spontaneous and asynchronous firings of cortical" and other neurons may coincide with the normal image of the world. However, considering long periods of time, it is unreasonable to expect that a chronically hyperventilating brain can function normally.

Q: I have heard that in some places pure O_2 can be bought for breathing. Is it good for health?
A: While breathing pure O_2, "*Free radicals (and other toxic metabolites of oxygen) are generated in most cells as a consequence of normal metabolic processes, but cells are protected from injury by antioxidant mechanisms. Several forms of lung injury appear to result from generation of toxic metabolites of oxygen in quantities which exceed the antioxidant capacity of lung cells…*", as stated at the very beginning of the abstract by Brigham (1986).

Moreover, detailed investigation of lung tissues revealed that, "*Exposure of animals to oxidant gases produces a mild emphysema, and O_2-derived free radicals are capable of degrading connective tissues in vitro. It is postulated that degradation of connective tissue by O_2-derived free radicals leads to emphysema in these models" (abstract, Kerr et al, 1987).*

A review, "*Data on oxidants and antioxidants*", conducted by Junod (1986), also found that "*Since O_2 intermediates can affect the general cellular metabolism and inhibit cell replication or reduce protein synthesis, all the biological effects of O_2 and its metabolites should therefore be considered in the pathogenesis of emphysematous lesions in the lung*" (Junod, 1986).

Another related question is why anti-oxidants are important supplements. They are used in order to diminish the possible damage done by oxidants generated by, among other sources, excessive freely-dissolved O_2 concentrations.

Finally, a textbook on medical physiology (Ganong, 1995) contains a section entitled "*Oxygen toxicity*". It starts with: "*It is interesting that while O_2 is necessary for life in aerobic organisms, it is also toxic. Indeed, 100% O_2 has been demonstrated to exert toxic effects not only in animals, but also in bacteria, fungi, cultured animal cells, and plants. The toxicity seems to be due to the production of the superoxide anion (O_2-), which is a free radical, and H_2O_2. When 80-100% O_2 is administered to humans for periods of 8 hours or more, the respiratory passages become irritated, causing substantial distress, nasal congestion, sore throat, and coughing. Exposure for 24-48 hours causes lung damage as well. In animals, more prolonged administration without irritation is possible if treatment is briefly interrupted from time to time, but it is not certain that periodic interruptions are of benefit to humans*" (Ganong, 1995).

In subsequent paragraphs, Professor Ganong describes development of lung cysts and serious visual defects due to retinal damage in infants treated with O_2 for respiratory distress syndrome. Increased O_2 pressure (in some places pure O_2 is administered at increased pressure) accelerates the harmful effects of O_2.

Meanwhile, breathing O_2 for a few minutes would probably not be very harmful. Generally, breathing pure oxygen can be useful as a short-term emergency measure in cases of life-threatening hypoxia.

Q: What is the long-term influence of different air compositions on human health? Has anybody investigated the optimum composition of air?
A: The first experiments in this area were done about a century ago by Yale researchers. Professor John Haldane was, probably, the most prominent scientist of those times. He wrote a classic textbook "R*espiration*" (Yale University Press, New Haven, UK, 1922) which is mostly devoted to the interaction between breathing and arterial blood CO_2 concentrations. During the later years of his career he served in the British Navy, working on air supply for submarines (where people can spend several months). The results of his research are still classified by British government agencies.

Available information about air composition on spaceships indicates that during the first three US space missions astronauts used pure O_2. Pure oxygen would be expected to cause impairment of mental performance and physical health, due to decreased blood flow to the brain, as discussed in section 1.2 and other negative effects mentioned above. On later missions US astronauts were provided with much less O_2 in the air of their spaceships.

In 1960s Doctor Buteyko was the manager of the laboratory of functional diagnostic and studied various breathing –related effects on cardiovascular and other systems of the human organism. His research was supported and funded by the Soviet Ministry of Aviation and Space Exploration for first Soviet space missions. According to Doctor Buteyko, the optimum air for long-term health benefits should be about 10-12% O_2 (as found on high mountains) and 2% CO_2 (Buteyko, 1977). Probably, this extra 2% CO_2 increases aCO_2, improving tissue oxygenation and producing

other positive changes, while 10-12% O_2 (twice less than normal air) is small enough to minimize oxidative lung damage.

Surprisingly little information is published about research on optimum air for submarines. Also, very little is published about the growth processes of plants and animals in CO_2 rich air, while known results are very encouraging.

Q: Plant respiration is the opposite process: consumption of CO_2 and production of O_2. Thus, plants fix CO_2 for synthesis of other chemical substances. Can animals do the same?

A: A review of numerous publications devoted to this subject was given by Waelsch and colleagues (1964) in an article entitled "*Quantitative aspects of CO_2 fixation in mammalian brain in vivo*". They found that aspartic and glutamic amino acids and glutamine were the substances chemically synthesised in mammalian brains.

Glutamine is the most abundant amino acid in the human organism (hence, its popularity among some bodybuilders). It is also the amino acid most required for tissue repair, but "*since the supply of glutamic acid from the circulating blood is insufficient for the formation of additional amount of glutamine, the dicarboxylic acid has to be synthesized in the brain.*" (Berl et al, 1962) This last substance is a CO_2 derivative.

Thus, CO_2 can be fixed by the human organism in order to rebuild nervous tissues in the brain. The rate of CO_2-derived glutamine production is proportional to CO_2 concentration in the brain. It follows that low CO_2 in the brain not only makes the brain unreasonably excited (possibly causing anxiety, fears, panic attacks, aggression, hostility, violence, or other strong emotions), but also has adverse effects on its cellular repair.

There are several other reactions, in which CO_2 is one of the necessary components. These reactions relate to biosynthesis of amino acids, carbohydrates, lipids and several other vital substances. The formulas of these reactions are provided in the article "*The Role of Carbon Dioxide in the Vital Processes of the organism*" (Kazarinov, 1991) [Available at: http://members.westnet.com.au/pkolb/biochem.htm].

"Q: Could you [doctor KP Buteyko] please explain us shortly your principle of breathing?

A: Here it is: we know that deep breathing decreases the concentration of carbon dioxide in the blood, lungs and cells. A Russian scientist from Perm, Verigo discovered this law at the end of 19-th century, which is, as it seems, strange: a fall of carbon dioxide increases the chemical link between oxygen and haemoglobin. As a result, it is more difficult for oxygen to get from the blood to the brain, heart, kidneys, and other organs. In other words, the deeper the breathing, the less the oxygenation of the cells in the brain, heart, and kidneys. This law is in the foundation of our discovery. CO_2 deficiency causes constant spasms in all organs. Hence, it is necessary to learn right breathing" (Buteyko, 1997).

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Chapter 2. The chemical and physiological mechanisms of immediate regulation of breathing

Introduction

The next topic (Chapter 2) is to consider the basic mechanism of immediate regulation of breathing. First, we shall consider the chemical foundations of breathing regulation. How does the body know when to breathe? Which body tissues and structures participate in regulation of respiration?

Second, we are going to consider some practical situations. How and why is our breathing changed due to some simple physiological activities? How do variations in the composition of the inspired air influence human breathing?

Within this portion of the topic, as a first step, we can investigate the control of breathing in healthy people when the human organism is subjected to different tests (voluntary changes in breathing frequency; changes in parameters of inspired air, including barometric pressure and air composition; and physical activity). Later, due to the importance of the BHT (breath holding time) test, we can consider how breathing is controlled during breath holding. Then, we will review the impact of the same tests on the breathing for sick people.

The main ideas about breathing regulation in the healthy have been known for about 100 years, and can be found in medical and physiological textbooks. However, there are still debates regarding chemical and neurological control of breathing.

2.1 Biochemical control of respiration

The central nervous system control our breathing, using special tissues and cells that sense the concentration of carbon dioxide, oxygen, and hydrogen ions, and then send the information to the brain. There are 2 types of respiratory chemoreceptors: central chemoreceptors, which are located in the medulla oblongata of the brain, and peripheral chemoreceptors.

The central chemoreceptors detect changes in the pH of the cerebro-spinal fluid and they are responsible for long-term or chronic changes in breathing. Since CO_2 dissolves in the blood and can penetrate through the blood-brain barrier, the main reason for pH variations in the brain are changes in CO_2 concentrations. Peripheral chemoreceptors monitor immediate changes in CO_2, O_2, and pH concentrations of the blood and control our breathing in the short run. It is agreed that peripheral chemoreceptors include;

1) the carotid body, which is situated at the division of the common carotid artery into the external and internal carotid arteries in the neck, becoming the external and internal carotid artries;
2) the aortic body which is located near the aortic arch.

Both bodies are small pieces of tissues, which are very well perfused. The carotid and aortic bodies detect acute changes in CO_2, pH and O_2 concentrations of the arterial blood. The output from both bodies is relayed to the respiratory centre in the brain for immediate regulation of breathing.

It is possible that there are additional chemoreceptors, which are often called “paraganglia”, located in the thorax and abdomen (Deane et al, 1975; Easton et al, 1983).

In addition, there are other receptors that are connected to the breathing centre, and are located in the lungs. These receptors, too, can influence our breathing by producing, for example, coughing, sneezing, deep inhalations, hyperventilation, breath holding and other effects. These receptors can detect the presence of foreign objects or irritants in the airways, the degree of inflation of the lungs and other parameters. The functions and actions of some of these receptors in the lungs and their relationships to breathing are still poorly understood.

More recent evidence suggests the existence of the respiratory rhythm due to dischange of medullary respiratory neurones. The respiratory rhythm,according to Oxford Medical Dictionary, is *the rhythm of alternating inspiratory and expiratory movements that take place during breathing. Four respiratory centres are thought to control the respiratory rhythm; the inspiratory centre and expiratory centre in the medulla, and an apneustic centre and pneumotaxic centre in the pons.* This rhythm probably plays a central role in situations when we loose consciousness. The respiratory rhythm influences, together with other factors, the duration of breath holding (Parkes, 2005). This study of Parkes *Breath-holding and its breakpoint* indicates that even such a simple event as breath holding has very complicated mechanism of interactions of various parameters and little is known about exact details of this process.

Doctor Buteyko proposed that CO_2 deficiency makes the neurons of the respiratory centre excited fuelling

further hyperventilation and causing reduction in the BHT (Buteyko, 1977).

2.2 The main physiological parameter, which controls the breathing of healthy people

Let us consider healthy volunteers, who were asked the following:

- 1) to breathe several times slower or faster while allowing the depth of breathing to regulate itself in a natural manner (their respiration rate could change from normal 12-15 to 3-4 per minute for slow breathing and up to 24-60 per minutes for fast breathing);
- 2) to breathe normal air at different barometric pressures (from about .5 to 4 atmospheres);
- 3) to breathe different air mixtures (consisting of from 0.04% to 4-5 % CO_2 and from 12% to 80 % O_2);
- 4) to walk 3, 5 or 8 km/h.

Was there any common parameter, which regulated breathing for all these situations? Practical experiments revealed that most parameters varied within wide ranges during these tests. For example, minute ventilation, the respiration rate, the volume of a single breath (tidal volume), arterial and venous O_2 contents changed two or more times in comparison with corresponding values at rest. However, the carbon dioxide concentration in the arterial blood (aCO_2) varied as little as 2-5 % in relation to resting numbers.

These tests were conducted by Yale Professor John Haldane about a century ago when basic ideas about respiration were established. Analyzing these results, Professor Haldane, in his textbook on respiration (probably the oldest one), stated that under normal conditions, exclusive of heavy work, the breathing was regulated by the carbon dioxide level in the lungs or arterial blood (Haldane, 1922). These classic experiments have been repeated many times by other researchers.

Therefore, during normal daily activities, the nervous system preserves a certain level of carbon dioxide concentration in the lungs and the arterial blood.

In order to maintain this nearly constant daily CO_2 level, the human organism employs, using a feedback mechanism, special groups of nervous cells located in two different places, in the medulla of the brain and near the main arteries close to the heart. This system of cells will be called in this book "the breathing centre". How does this feedback mechanism work?

In normal conditions, the immediate aCO_2 value of the person corresponds to aCO_2 norm established by the breathing centre. However, certain activities can change the aCO_2 value in both directions.

For example, during acute voluntary over-breathing the current aCO_2 value becomes smaller than the aCO_2 norm. This change is detected by the breathing centre. The breathing centre instructs (or orders) the respiratory muscles to stop their activity (stop breathing). The organism starts to accumulate carbon dioxide up to the level preset by the breathing centre (aCO_2). That causes a gradually increasing desire to breathe. Normal breathing is resumed when this preset level is achieved.

In certain other situations, the organism can accumulate excessive (in respect to the preset aCO_2 norm) amounts of carbon dioxide. That can happen, for example, during breath holding started after normal breathing. In both cases the breathing centre senses this gradual aCO_2 increase and sends impulses to respiratory muscles to intensify breathing until additional carbon dioxide is removed and aCO_2 again stabilizes near the initial norm.

The same situation takes place during speaking while vigorously exercising since ventilation during heavy exercise can be as large as 150-170 litres per minute. Even during yelling or roaring ventilation numbers are significantly less. Hence, speaking while vigorously exercising causes increases in current aCO_2 values.

Generally, the drive to increase breathing is proportional to the difference between current aCO_2 values and the preset aCO_2 norm. When the difference is negative (the current aCO_2 values are lower than the aCO_2 norm), there is a drive to decrease ventilation, up to its full cessation, as in cases of prolonged voluntary hyperventilation.

The breathing centre also monitors oxygenation of the arterial blood using a similar feedback mechanism, but its importance for the above-mentioned tests is almost negligible. Since CO_2 forms carbonic acid in the blood, the control of oxygenation is achieved by these cells through sensing hydrogen ions or pH of the blood which is kept, in health, within a narrow range (7.35-7.45). Hence, the breathing centre also monitors blood pH, one of the most important and carefully guarded physiological parameters.

Let us look in more details at normal physiological responses for these four tests investigated by Professor Haldane.

- 1. Changing breathing frequency from a normal 12-15 times per minute to 3-4 times per minute caused prolonged inhalations and exhalations in normal people. This naturally produced a slow and deep breathing pattern. The amount of air exhaled during such breathing was almost unchanged. Hence, CO_2 elimination from the organism

during this slow breathing was also nearly the same (as before the test). Hence, the aCO_2 value remains unaffected. Voluntary breathing with high frequencies (24-60 times per minute) resulted in fast shallow breathing also without significant aCO_2 changes. Dogs in hot weather cool their bodies using such breathing. Large amounts of water evaporate from their tongues cooling the blood, while the carbon dioxide level remains almost unchanged.

- 2. Breathing normal air at different barometric pressures (from twice less to four times more, than the normal barometric pressure at sea level) did not have an immediate effect on breathing. Indeed, CO_2 content of the inspired air changed very little in these situations since normal air had only 0.04 % CO_2. Hypoxia at the lowest pressures was too small to produce noticeable changes in breathing. Thus, breathing was almost unaffected.
- 3. When the CO_2 content of the inspired air suddenly increased, the CO_2 pressure in the lungs and, hence, the arterial blood also increased. The breathing centre sensed this aCO_2 rise due to blood acidification and that caused increased ventilation. Small increases in inspired air CO_2 content (up to about 1%) were almost unnoticeable. Air with around 2% CO_2 resulted in marked increase in ventilation. 4-5% CO_2 in inspired air produced laboured breathing with minute ventilation over 20 l/min (e.g., Straud, 1959) in order to remove extra CO_2 and preserve the preset aCO_2.

Variations in O_2 concentration of the inspired air produced no sensations in wide ranges (from 15% to 100%). Thus, breathing pure oxygen usually does not result in immediate specific feelings or symptoms. Hyperoxia (excessive oxygen in air) has never been experienced by an animal species during evolution. Indeed, air O_2 content on the Earth has always been increasing, except for short historical periods when it has had small drops. Thus, breathing pure O_2 is usually not felt, although prolonged breathing is harmful to the lungs and the brain. However, for hypoxia, a slight increase in ventilation and a corresponding aCO_2 decrease were detected when air O_2 concentration became about 12-15%.

- 4. Physical exercise required additional energy provided by muscles due to oxidation of carbohydrates and fats and the generation of additional CO_2. This extra CO_2 was transported by the venous blood to the lungs and detected by the breathing centre. The results were increased breathing frequency and minute ventilation. It is possible for ventilation to reach 100 l/min for moderately heavy physical work, but aCO_2 would be virtually the same as at rest (2-5% relative variations in respect to the initial value).

2.3 Hypoxia and its contribution to regulation of breathing

Let us look at the influence of O_2 content on respiration when CO_2 concentrations in the inspired air are negligible. Healthy people usually notice increased ventilation only when O_2 content is below 10%. Smaller changes (e.g., when breathing 15-19% O_2) are probably also sensed by the breathing centre, but increased ventilation would also result in reduced aCO_2 values. The breathing centre mainly controls aCO_2. As a result, these two drives cancel each other out for O_2 concentrations between 15 and 19%.

What happens during heavy work? When intensity of exercise gets higher, the amount of oxygen in tissues and in arterial blood becomes lower. As a result, increasing energy is generated without direct O_2 participation (anaerobic metabolism). Such metabolism causes the appearance of lactic acid in the blood. A small amount of lactic acid is present in the blood at rest and its level gets larger, but still remains constant, for exercise with moderate intensities. At higher intensities the lactic acid level is constantly increasing causing blood acidosis, which is sensed by the breathing centre.

Fatigue and pain are the normal sensations experienced during and after strenuous physical efforts. According to a textbook on medical physiology, "*Fatigue is a poorly understood phenomenon that is a normal consequence of intense exercise or mental effort. In addition, it is a symptom of many different diseases. During exercise, acidosis and other factors contribute to its production*" (p. 627, Ganong, 1995).

Using these conclusions, it is possible to suggest that the breathing centre can play an important or, probably, the central role in the perception of sensations of fatigue and pain due to physical and mental exercise. Possibly, because it is a part of the nervous system and is sensing blood acidosis caused by increasing blood lactate, the breathing centre sends intensive signals perceived as fatigue or pain. That makes heavy work, at some moment in time, physiologically impossible (blood acidosis to pH=7.0 can be fatal for humans).

In order to keep pH closer to the normal 7.4, the organism starts to remove carbonic acid as a compensation for increasing lactic acid. Carbonates are the major CO_2 forms in the blood and their level can be kept low due to removal of bicarbonates by kidneys and additional ventilation.

In practical terms, only at sub-maximum intensities (80-90% of the maximum possible oxygen consumption), does aCO_2 start to decline from the initial resting level (40 mm Hg) down to about 30 mm Hg for maximum possible intensities. This conclusion can be found in textbooks on exercise physiology (e.g., p.201, Brooks et al, 1996).

2.4 Control of breathing during breath holding

When the lungs are not ventilated, as during breath holding under normal conditions, their carbon dioxide concentration builds up. Indeed, incoming venous blood continuously brings more CO_2 to the lungs. The breathing centre monitors these changes. During initial stages of breath holding, the discomfort is small or unnoticeable. Gradually, the chemical influence on the breathing centre gets stronger over time, creating, at some moment, the first distinctive urge to breathe. This urge can be suppressed by one's will power until another aCO_2 threshold is achieved. At that moment the continuation of breath holding becomes impossible. This is the main process, which regulates the duration of breath holding time in healthy subjects.

Therefore, BHT is mainly defined by 3 parameters:

1) the initial aCO_2;
2) aCO_2 threshold established by the breathing centre;
3) the rate of aCO_2 accumulation.

Let us consider different practical situations and how they influence these parameters and BHT.

- **Initial volume of air in the lungs**. Larger initial amounts of air in the lungs allow more carbon dioxide to be left there by the venous blood. Hence, lungs CO_2 concentrations are going to increase with lower rates during breath holding. Similarly, the rate of aCO_2 increase in the arterial blood leaving the lungs is also smaller. That allows longer BHT.

Indeed, as mentioned in chapter 1, an analysis of several experimental studies found that BHT is linearly proportional to the initial lung volume (Mithoefer, 1965). For example, it was found that BHT after complete exhalation is about 4 times less than BHT after full inhalation. At normal exhalation breath can be held for about 40% and at normal inhalation about 55% as long as BHT at maximum inhalation.

- **Body position**. Depending on the body position, the metabolic rate and, hence, the rate of carbon dioxide production are different. For example, standing requires from about 40 to 70% more energy, than lying on one's back. As a result, the rate of aCO_2 accumulation in the lungs is higher when standing. Normally, BHT when lying is larger, while BHT while sitting is going to be in between the other two positions.
- **Current physical activity**. From the previous paragraph it follows that physical activity generally reduces BHT due to faster accumulation of carbon dioxide. That is especially true for higher intensities, when BHT can be several times smaller and is equal to a few seconds at sub-maximum intensities. However, slight physical activity at the end of breath holding can prolong BHT (explained in this section below).
- **Previous physical activity**. Let us compare two situations. The tests, in both considered situations, can be done while sitting, but, in one case, immediately after fast walking and, in the other case, after 5-10 minutes of being at rest. The results are going to be different, since prior muscular activity generates additional carbon dioxide (in muscles which produced this work). This additional carbon dioxide arrives at the lungs, is detected by the peripheral chemoreceptors of the breathing centre and, as a result, causes decrease in BHT.
- **Previous breathing**. Acute hyperventilation reduces CO_2 content, first, in the lungs, then in the arterial blood, and finally in all tissues. Therefore, depending on the duration and intensity of hyperventilation, the starting CO_2 level is going to be smaller. Hence, additional time will be required in order to achieve, first, the normal aCO_2 level preset by the breathing centre and, later, the upper limits of tolerance which define BHT. For example, 2 minutes of forced hyperventilation produced 2 minutes of natural breath holding at maximum inhalation in healthy people (Ganong, 1995). With increasing intensity and duration of over-breathing (up to a few minutes) this time can be even greater. Meanwhile, long and especially chronic hyperventilation resets the breathing centre to lower aCO_2 normal values. Indeed, results in Chapter 1 revealed that those people who chronically breathe deeply have a short BHT. However, at the moment we are discussing only healthy people with normal breathing. In later chapters, the effects of hypoventilation (breathing less than desired) will be considered.
- **Initial air composition**. Breathing special mixtures of air before the test or as a last breath before breath holding also influences BHT. For example, the higher the starting CO_2 pressure in the lungs, the shorter the BHT (Godfrey & Campbell, 1968). Larger starting O_2 concentrations increase BHT (Mithoefer, 1965).
- **Special chemicals affecting the breathing centre, respiratory muscles or other related systems**. Many drugs, medications, steroids, other synthetic and natural substances can influence the breathing centre and BHT (up to several times in both directions). For example, alcohol is known to increase BHT soon after intoxication (larger BHTs may contribute to the feelings of euphoria and powerfulness), while later, during hangover, BHT is significantly reduced in comparison with the initial value.

• **Physical activity at the end of breath holding**. Different types of light physical activity at the end of breath holding (for example, stomping, clenching fists, swinging movements of arms, swallowing movements, imitation of inhalation by chest or diaphragm muscles without taking any air in) prolong BHT (Bartlett, 1977). Probably, these activities distract the central nervous system from paying exclusive attention to the breathing centre, which generates distress and pain signals. As it was suggested by Bartlett, "*These findings show that a manoeuvre that does not involve the respiratory system can consistently prolong breath-holding time, possibly by providing distraction from the discomfort of the breath hold*" (Bartlett, 1968). He found that, practically, 10 to 20% increases in BHT could be observed. In my view, a similar effect of pain reduction due to distraction takes place in cases of light physical activity, while having, for example, toothache.

• **"Training effect"**. Several successive breath holds essentially improve breath-holding performance, as it was documented by Heath and Irwin (1968) and Bartlett (1977). Professionals investigating diving believe that this increase in breath holding time is due to restoration of what is called the "diving reflex" which is observed in many (even land-dwelling) animals, humans included. What they found is that repetitive breath holds "teach" the human organism to divert blood from the skin to the heart, brain and other vital organs (as during cold water immersion). Practically, the degree of this reflex can be defined by the amount the pulse slows during breath holding.

Does the "traing effect" relates to the maximum possible breath holding time or the breath holding time until the first stress (the first desire to take breath)? Medical doctors from the School of Medicine in Chiba University (Japan) studied, according to the title of their paper, *Changes in the period of no respiratory sensation and total breath-holding time in successive breath-holding trials.* They wrote,

"Immediately after breath-holding at end-expiratory level, there is a certain period of no particular respiratory sensation which is terminated by the onset of an unpleasant sensation and followed by progressive discomfort during breath-holding. This period, defined as the time from the start of voluntary breath-holding to the point where the onset of an unpleasant sensation occurs, is designated "the period of no respiratory sensation'. Although it has been shown that the maximum breath-holding performance is improved with successive trials, it is not clear whether this training effect exerts a similar influence on the period of no respiratory sensation during breath-holding. 2. Since the training effect seems to be associated with the stresses of breath-holding, we hypothesized that the initial period of no respiratory sensation during breath-holding might be less influenced by the training effect. 3. We studied 13 normal subjects who performed repeated breath holds while continuously rating their respiratory discomfort using a visual analogue scale. In addition, we measured the hypercapnic ventilatory response of each individual and obtained the relationship between the slope of the hypercapnic response curve and breath-holding periods. 4. Our results showed that there was little training effect on the period of no respiratory sensation and that the period of no sensation during breath-holding is inversely related to the slope of the hypercapnic ventilatory response curve" (Nishino et al, 1996).

Hence, those people, who frequently perform breath holds (divers, patients with sleep apnoea, etc.), can improve their total or maximum breath holding time without any real improvements in their minute ventilation and actual carbon dioxide stores. However, their breath holding time until the first discomfort (or "*the period of no respiratory sensation*" as the Japanese doctors called it) has only minor improvement.

The "training effect" has important practical implications in relation to BHT measurements, as we will see in later chapters.

• **An individual attitude or current emotional set**. As for any other personal test concerning human performance, the general state of the central nervous system, feelings, attitudes and emotions, all influence BHT. Many studies from Chapter 1 do not specify the exact details for BHT measurements. Obviously, this introduces some level of uncertainty in the measurement process.

Note that in all these situations I use the term BHT (breath holding time), not the different term <u>BHT</u> (breath holding time after normal expiration), since <u>BHT</u> is measured under normal conditions (sufficient rest, no breathing control, no breathing manipulations, normal air composition and pressure, etc.) after normal or usual exhalation.

2.5 Control of breathing in people with chronic hyperventilation

Chronic over-breathing causes numerous negative physiological changes (Chapter 1) due to low CO_2 content in the organism. Tissue hypoxia (due to the suppressed Bohr effect), excessive excitability of the nervous system, and reduced blood supply to the brain, heart muscles and other organs and tissues are the typical consequences of chronic over-breathing.

Let us compare the regulation of breathing of sick people during physical exercise with our previous findings for healthy individuals. From practical experience it is known that the respiration of most sick people usually gets

heavier for moderate and, sometimes even minor physical exertion, like stair climbing. A professional review of medical studies in this area concluded that physical exercise usually caused metabolic acidosis (excessive acidification of the blood) for patients with various health problems (Wasserman & Casebury, 1976).

What were the sources and consequences of increased acidity in sick people? Deep breathing causes chronic tissue hypoxia. Hence, these people experience low tissue oxygenation before exercise. Since all cells need oxygen in order to function, anaerobic metabolism (burning fuels without oxygen participation) becomes more pronounced. Therefore, the resting lactic acid level in the blood of sick people is often elevated.

Exercise needs even more energy and O_2. That results in even a greater incidence of anaerobic metabolism, as compared to healthy subjects, and in further accumulation of lactic acid in the case where physical work is being performed, leading to increased acidity of the blood. That further increases acidity of the blood. If for healthy people high lactic acid levels are normal only for very heavy work, most sick patients have high lactate almost from the onset of light or moderate physical exercise. Such acidosis causes greatly increased breathing and further reduction in body CO_2 stores. For example, an analysis of respiratory parameters of different classes of heart patients revealed, "*The end-tidal CO_2 pressure during exercise can be used to evaluate the functional capacity of patients with chronic heart failure*" (abstract, Tanabe et al, 2001).

Therefore, physical exertion requires more physical and psychological efforts on the part of sick people, while for critically ill patients even light exercise can be fatal, since hypoxia is already near the physiological limits of the human organism.

It follows that certain, biochemical parameters (aCO_2, tissue oxygenation, lactate values, etc.) for severely ill patients during slight physical exertion or even at rest are close to the same parameters of healthy athletes who are exercising strenuously and are near the full physical exhaustion. The difference is that the athletes can rest and then recover quickly, while the severely ill patients often do not know how to recover to physiological norms.

Other previousy mentioned tests also would produce different results in people with impaired health and respiration.

For example, breathing 3 times per minute can be difficult or impossible for sick people to perform. Indeed, in order to preserve their usual resting ventilation (breathing over 15 l/min), sick people need to inspire more than 5 l of air during a single breath. That is unattainable even for most healthy individuals. Breathing less air per minute (relative hypoventilation) during long periods of time may cause other problems. Among them are additional tension in the chest muscles due to maximum inhalations and exhalations (causing more stress) and increased anxiety due to unexpected and often feared feelings of air shortage.

The reaction to breathing different gas mixtures in sick people is usually abnormal too. For example, hyperventilating sick people are over-sensitive to the presence of small amounts of carbon dioxide in the inspired air. Breathing some gas mixtures (e.g., with 2 or 4 % carbon dioxide content) produces excessive over-breathing in people who hyperventilate as compared to the rection of healthy subjects. A similar reaction (over-breathing) is observed, when breathing air with reduced O_2 content. Accordingly, high altitudes are poorly tolerated due to already existing hypoxia.

In the abstract of the review "*Studying the control of breathing in man*", Dr. Foldering wrote,

"*Disruptions of the control of breathing may have serious consequences for several categories of patients, e.g., those with chronic obstructive pulmonary disease (COPD), asthma, sleep apnoea, sudden infant death syndromes, and the hyperventilation syndrome. Adequate investigation of the control of breathing in these patients is of great importance for their treatment*" (Foldering, 1988).

2.6 Breath holding control in diseased states

As it was shown in section 1.8, BHTs and BHTs for people with various health problems are significantly reduced as compared to healthy individuals. What are the possible reasons?

- People who hyperventilate has a lower initial aCO_2 as well as lower tissue carbon dioxide concentrations. That causes excessive excitability of nervous cells (see chapter 1, section 1.2), including, the nerve cells of the breathing centre. The breathing centre becomes increasingly sensitive to aCO_2 increases, as numerous chemical sensitivity tests and published studies confirm.

Thus, the state of excessive excitement of the nervous cells of the breathing centre can make BHTs and BHTs shorter in people who hyperventilate in comparison with healthy people.

- Healthy people have larger amounts of O_2 in their tissues due to a normal aCO_2. Thus, during breath holding they do not suffer, to a large extent, from hypoxia. In many diseased states, the amount of O_2 in tissues is smaller. This

causes a worsening of the already existing hypoxia during breath holding.

Indeed, testing the BHT of patients with neurocirculatory asthenia (another name for chronic fatigue syndrome), Dr. Friedman, Director of the Harold Brum Institute for Cardiovascular Research, San Francisco, found the average BHT of healthy persons after voluntary hyperventilation increased by about 40%, whereas the BHT of his sick patients decreased (!) by almost twice (Friedman, 1945). Why? As we know preliminary voluntary hyperventilation normally increases BHT due to a temporary reduction in carbon dioxide stores. So, what was wrong with the sick patients who breathed deeply?

People who hyperventilate normally suffer from low tissue oxygenation due to initially low carbon dioxide values. Additional forced over-breathing removes even more carbon dioxide making the amount of available oxygen even smaller due to the suppressed Bohr effect (no carbon dioxide – no oxygen to release). There are large amounts of O_2 in the blood, but this O_2 cannot move from red blood cells due to the absence of the chemical catalyser, CO_2.

Since sick, hyperventilating people normally suffer from chronic hypoxia already at rest, their breathing centre is also hypersensitive to further oxygen reductions. That can also contribute to shorter breath holding pauses in people with heavy breathing.

• Human and animal experiments performed by Brown with colleagues (1948, 1949, 1950a, 1950b) revealed that prolonged forced hyperventilation (24 hours) caused the following changes: increased sensitivity to CO_2 inhalation, reduced blood buffer capacities due to increased excretion of urinary sodium and potassium, and marked fall in the brain plasma level of inorganic phosphates. In summary it was suggested "*this reduction in the buffering ability of the brain for CO_2 following prolonged hyperventilation may be the mechanism by which the increased sensitivity of the respiratory centre to CO_2 following prolonged hypocapnia* [low aCO_2] *is brought about*" (Brown et al, 1950b).

In turn, that causes smaller differences between the aCO_2 tolerance threshold and the aCO_2 usual level leading to shorter BHTs.

Thus, there are three factors which can make BHT and the BHT short when people chronically breathe too much:

1) excessive excitability (or irritability) of the breathing centre due to low carbon dioxide level;
2) low tissue oxygenation due to low carbon dioxide concentrations;
3) abnormal brain electrolyte changes (reduced buffering ability).

These suggestions are based on known physiological processes but have not been supported by any practical studies. It is possible that there are some other factors that can play a more important role as to why BHTs are shorter for people with chronic hyperventilation.

2.7 Connection between BHT (breath holding time after normal expiration) and arterial CO_2

Experimental results from Chapters 1 and 2 suggest that for most people it is possible to establish a relationship between aCO_2 and the BHT (breath holding time at normal exhalation). Normal aCO_2 values (about 40 mm Hg) correspond to BHTs of about 35 s. When aCO_2, due to chronic over-breathing, gets smaller, so does the BHT. Critically low aCO_2 numbers correspond to very small BHTs.

Russian medical Doctor Buteyko devoted his life to studying breathing. During the 1960s he created a diagnostic complex, which could measure about 40 different respiratory and cardiovascular characteristics in real time including minute volume, breathing frequency, tidal volume, expired gas parameters, blood gases, pulse, blood pressure, ECG, etc.(Inventor and Efficiency Expert, 1961; Buteyko 1961; Buteyko, 1962). Using this unique machine he investigated thousands of sick and healthy people and found that the relationship between aCO_2 and the BHT is almost linear. Years later he patented (Buteyko, 1986) the formula:

$$\mathbf{aCO_2\% = 3.5\% + .05*\underline{BHT}}$$

(here, aCO_2% is the alveolar CO_2 concentration in %, 3.5% is the minimum CO_2 content, .05 is a coefficient of proportionality, BHT is the breath holding time after quiet expiration, while sitting at rest for 5-10 minutes). Appendix 2 provides the relationships between aCO_2% and aCO_2 values measured in mm Hg at different altitudes. The patent claimed the same accuracy of aCO_2% defining as can be done using professional diagnostic devices. While many Western studies indirectly indicate that BHT is lower when CO_2 values are lower, to my knowledge, there are no Western studies that directly focused on this problem.

Let us calculate the normal BHT value while applying this formula. Normal arterial and alveolar CO_2 pressure is about 40 mm Hg, whereas a normal pressure at sea level is 760 mm Hg. Hence, the normal aCO_2 is 5.26% (this is

the normal carbon dioxide concentration in human lungs and arterial blood at sea level). That corresponds to 35 s BHT (in order to check that just substitute 5.26% into the formula and calculate the BHT), which is close to the average found in numerous observations quoted in chapter 1 (Table 1.2).

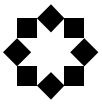
It is likely that this formula works very well in the most important ranges of aCO_2 and BHT values, when the BHT is between about 10 and 60 s. It cannot be applied with severely sick people, when aCO_2 is about 20-30 mm Hg or lower. Indeed, minimum possible aCO_2, according to this formula is 3.5% or about 27 mm Hg, while medical studies often quote 20 mm Hg and lower values for critically ill patients. At the other end, for high aCO_2 and BHT numbers, there were exceptional people (hatha yoga masters), who could hold their breath for many minutes, but their aCO_2 was only slightly above the medical norm.

Those people, who routinely perform breath holding, should measure their BHT until the first distinctive desire to breathe due to the "training effect" discussed in section 2.4.

This formula also practically helps to evaluate the minute ventilation (how much air we breathe in 1 minute). Normal ventilation (about 6 l/min) corresponds to normal BHT (about 38-40 s). Those who have about 17 s BHT (almost twice less than the norm) breathe about two times more air (12 l/min for normal adults). 12 s BHT corresponds to thrice over-breathing.

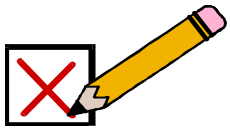
Therefore, the BHT reflects the intensity of breathing. Shorter BHTs mean chronically heavier and deeper breathing. Many western medical doctors came to the same conclsion as described in Chapter 1.

Note about exceptions. The formula cannot be applied for some physiological abnormalities of the respiratory, circulatory, and nervous systems. For example, BHT can be smaller than predicted by the formula, as in cases of blood shunting found in some people with emphysema and severe asthma (McFadden & Lyons, 1968). In certain cases, BHT can be greater than expected, as after surgery of carotid bodies of the breathing centre (Patterson, 1974; Gross et al, 1976) or curarisation of the respiratory muscles (Campbell et al, 1966).



Conclusions

- Under normal conditions, except with very heavy work, the breathing of healthy individuals is regulated by carbon dioxide in the lungs or arterial blood.
- Only in special cases, like very low O_2 content in the inspired air (less than 12-15%), hypoxia affects ventilation of healthy people causing their over-breathing.
- Normally, breath holding in health is limited mainly by the aCO_2 threshold with little contribution due to hypoxia. Other parameters, which define BHT, are initial aCO_2 value and the rate of aCO_2 accumulation.
- Control of breathing in those who hyperventilate is largely defined by chronically existing hypoxia due to initially low carbon dioxide stores. The contribution of hypoxia is proportional to the degree of deep breathing.
- As a result of hypoxia, a more excited state of the respiratory centre, electrolyte changes in the human brain and other factors, the breath holding time of hyperventilators (majority of sick people) is reduced, depending on the degree of over-breathing.
- It is possible that the connection between the BHT (breath holding time after normal expiration) and aCO_2 can be expressed through a linear relationship, which can be applied for most practical situations. The BHT can also reflect the intensity of breathing (or minute ventilation).



Q&A section for Chapter 2

Q: Are there any special reasons to measure the BHT after a quiet expiration?

A: Doctor Buteyko introduced BHTs in order to standardise BHT measurements. BHT is proportional to the volume of air in the lungs. The amount of air after normal or deep inhalation can vary in different individuals. However, after a quiet relaxed expiration there is only anatomical volume of air left in the lungs. Therefore, in order to have better accuracy, it is reasonable to compare the BHT of different people after their quiet expiration (Buteyko, 1977).

Q**: What are the physiological causes of the "training effect"?**
A: In other words, why does breath holding time get larger with test repetitions, while measured CO_2 values can remain unchanged? Many physiologists who have studied the free diving of humans believe that with humans can develop a good diving reflex, similar to that observed in many diving animals. The diving reflex includes numerous adaptive physiological changes as a response to breath holding: slowing of the heart rate, reduced blood circulation to muscles while maintaining blood circulation to the heart and the brain, and many others. Facial contact with cold water can trigger some of these changes in humans as well.

Q: There are various activities mentioned in section 2.3 which extend breath holding time. Are these activities useful for health or not?
A: These activities can have a different impact on breathing and were discussed in section 2.3 only as examples which help to understand the physiology of breath holding. This chapter investigated only the immediate physiological built-in mechanism of breathing regulation (how breathing is controlled in healthy and sick people). Finding the real-life factors which make breathing chronically deep (why breathing gets excessive in certain groups of people or why people get sick) will be the subject of the next chapter.

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Chapter 3. Lifestyle factors that matter

Introduction

Let us look at the main causes of chronic hyperventilation.

Why do modern people chronically breathe too much? Which environmental, life-style, and other factors intensify breathing, causing the breathing centre to permanently readjust to lower carbon dioxide values? What are the mechanisms and degree of influence of these hyperventilation-producing factors?

Most causes described in this chapter were systematically investigated and summarized by Doctor Buteyko (Buteyko, 1969; Buteyko 1970; p.158, Buteyko, 1991; p.177, Khoroscho, 1982), when he used his unique diagnostic complex in different practical situations. Different Russian publications and Buteyko's lectures described hyperventilation-producing factors often without providing detailed information about the mechanisms of their influence. In addition, these papers were in Russian. However, there are relevant Western physiological studies showing such mechanisms and often providing measurements of their influence. Many of these factors are well-known. They are frequently mentioned by medical authorities and various health professionals as factors, which increase the chances of new health problems while aggravating existing ones.

3.1 Stress, anxiety and strong emotions

The concept of "stress" can have different meanings. For example, the presence of bacteria and their toxins in the blood is an example of stress to a medical doctor. Such physiological stress, as it was described in section 1.8, produces visible chronic hyperventilation. Similarly, tiny amounts of other toxins in the blood, as a result of, for example, teeth cavities, also cause mild over-breathing, as we will see later.

However, let us consider here only influences due to individual perception of threat, challenge, or other psychological stress. There are numerous physiological changes generated by anxiety states, fear, fight-or-flight situations, or other situations accompanied by strong emotions. How do these states and factors influence breathing?

An early paper "*Some physical phenomena associated with the anxiety states and their relation to hyperventilation*" (Kerr et al, 1937) included a chart showing physiological changes caused by these states. Hyperventilation is the main factor causing many of the other consequences described.

According to Professor Lum, "*Most authors, with the exception of Rice (1950), have described the clinical presentation of hyperventilation as a manifestation of, and secondary to, an underlying anxiety state*" (p.197, Lum 1976).

Dr. Magarian wrote one of the large hyperventilation reviews, published in "*Medicine*" (Magarian, 1982), choosing the following title for his paper "*Hyperventilation syndromes: infrequently recognized common expression of anxiety and stress*".

A more recent study "*Fear-relevant images as conditioned stimuli for somatic complaints, respiratory behaviour, and reduced end-tidal pCO_2*" (Stegen et al., 1999) discusses psychological and physiological effects providing numerous references in this area.

Dozens, if not hundreds, of professional physiological and medical studies suggest that hyperventilation is a normal outcome of stress and anxiety.

Authors of the article "*Emotions and respiratory patterns: review and critical analysis*" (Boiten et al, 1994) suggested, that "*...normo-ventilatory responses (which are identified by stable end-tidal CO_2 levels that remain within the normal range) seem to be characteristic for behavioural conditions that may either involve withdrawal from the environment, relaxation or active coping...Thus, hyperventilation appears to signify an unsuccessful outcome of the coping process*" (p.121).

Researchers from the University of Oxford found that during voluntary over-breathing "*subjects in the positive interpretation condition experienced hyperventilation as pleasant, and subjects in the negative interpretation condition experienced hyperventilation as unpleasant …"* (Salkovskis & Clark, 1990).

Therefore, not only negative emotions and states, but also many positive strong emotions (e.g., delight, excitement, exhilaration, thrill, etc.) produce hyperventilation.

Stress, according to TV, radio, newspapers and magazine news about medical studies, contributes to the development of most modern health problems.

On the other hand, stress-triggering physiological changes had obvious usefulness in terms of evolutionary survival of our species.

For example, during the fight or flight response, the diversion of the blood from internal organs and the brain to large skeletal muscles prepares the body for possible vigorous physical activity. Increased nervous excitability helped our ancestors to give different psychological interpretations to the surrounding stimuli and events ("*it is me who is in danger now*"). Higher blood sugar values and increased concentrations of numerous hormones (e.g., well-known adrenalin rush) were useful for active physical response.

Indeed, during peaceful periods, an objective perception of the world, coupled with quiet and thoughtful relationships with nature and with other tribal members, proved to be advantageous. At other times (e.g., during hunting, fight-or-flight responses, group conflicts, or mating), subjective perception, in terms of seeing the world in the light of personal needs, was more beneficial to the specie's survival.

Do we have more stress now? From an objective viewpoint, our ancestors living 1, 5 or more thousands of years ago, often had daily life-threatening situations and challenges. Hence, the level of stress and the likelihood of mortality were much higher in the past. Why then, do modern people chronically hyperventilate and get sick because of stress, while our ancestors mostly died from other, more natural causes (like, infectious diseases, accidents, and conflicts)?

A part of the answer lies in breathing. The objective perception of the world for people who suffer from chronic hyperventilation can be difficult and fearful. That predisposes them to strong emotions, excitement, and tendencies to exaggerate, justify, negate, distort, and misinterpret events.

Another part of the answer is what happens after one experiences stress in modern society. Our ancestors hunted, struggled, or ran away under stressful conditions. All these are active coping strategies involving physical exercise. These are also responses of healthy wild animals. Therefore, our attention should be directed to the following question.

What are the effects of exercise on the breathing of healthy people?

3.2 Physical inactivity

The natural lives of our ancestors, or modern primitive people, or modern human-related species and other mammals include many hours of daily exercise of light, moderate, and, sometimes, high intensity. Typical moderate intensities (40-65% from maximum O_2 consumption) produce the following effects in healthy people.

- While aCO_2 remains close to the initial level (about 40 mm Hg), venous CO_2 content rises from resting 46 mm Hg to about 60-65 mm Hg due to the greatly increased metabolism of carbohydrates and fats (p.201, Brooks et al, 1996).

Hence, during moderate exercise, the human organism accumulates large additional amounts of CO_2. Generally, physical activity is characterised by the largest amounts of carbon dioxide stores in the human body, which are significantly higher than during any other activity.

- Blood pH in arteries slightly decreases (acidification) from 7.42 to 7.38 (p.201, Brooks et al, 1996), indicating some adaptation of the breathing centre to a more acidic environment. (Lactic acid is the main cause of additional acidity) and higher aCO_2 values. This adaptation, if the exercise lasts long enough, can positively influence after-exercise breathing.
- Arterial oxygenation is reduced by about 2-5 % and venous O_2 partial pressure is decreased from resting 40 mm Hg to 20 mm Hg.

Thus, moderate exercise takes place in the condition of mild tissue hypoxia for the receptors of the breathing centre. As a result, after the exercise the breathing centre can tolerate lower oxygen levels in the arterial blood. Note that these lower O_2 arterial blood concentrations normally increase tissue oxygenation due to the enhanced Bohr effect.

- Exercise is normally accompanied by movements of the limbs causing neuronal discharges from working muscles to the central nervous system. Such muscular movements, as discussed in Chapter 2 (how light physical activity can prolong BHT), suppress the drive of the breathing centre to increase ventilation.

Thus, any prolonged physical activity with light, moderate, or moderately high intensity usually has positive effects on the subsequent respiration of healthy individuals.

These changes result in increased tolerance to higher aCO_2 and lower vO_2 values. Indeed, several physiological studies revealed "*the increased 'tolerance' for high CO_2 and low O_2 in breath holding, during and immediately after exercise*" (e.g., p.220, Astrad, 1960).

All these factors, if exercise is long enough, should cause a favourable adaptation of the breathing centre and subsequent easy and natural breathing when exercise is finished and important physiological parameters (blood sugar, lactate, concentrations of certain hormones) return back to their normal values. That may take from a few minutes to

many hours depending on the fitness of the person and the intensity and duration of the exercise.

Therefore, exercise for a healthy person trains the breathing centre to reset its aCO_2 norm (the crucial parameter which, according to chapter 2, controls human respiration) to higher values. As a result, exercise reduces ventilation.

"Physical work, sport, and exertion increase CO_2 production. Its level increases in the blood, while oxygen decreases. The higher the intensity, the stronger the excitement of the breathing centre and the deeper the breathing, but it is only deeper formally. Breathing becomes not deeper, but shallower: it is less in relation to metabolism. This is the reasoning behind the usefulness of exercise and sport! During prolonged intensive exercise the receptors, which control breathing, adapt to increased CO_2. If the person regularly works and toils, then he practically follows our method: he is decreasing his breathing using exercise" (Buteyko, 1977).

Vice versa, if we do not exercise after experiencing stress, or anxiety, or strong emotions, we can continue over-breathing that leads to habitual or chronic hyperventilation.

Russian medical professionals from the Buteyko Clinic in Moscow currently believe that a lack of physical activity is the main cause of carbon dioxide deficiency in modern people (for more details one may visit their website www.buteykoclinic.ru).

"Q: Why does hyperventilation form, since nobody specially practices deep breathing…?
A: The main cause is the lack of physical activity, low level of metabolism in general. We must work out at least 4 hours per day, with heavy perspiration. Then you would get high level of metabolism. The method should be practiced until the level of carbon dioxide is stabilized and reached the value when the attacks are impossible. It is practically impossible to maintain this state without physical exercise. If a person, due to some reasons, cannot do physical exercises, he should do the breathing ones. There is nothing wrong with that, since the [breathing] exercises are not hard and can be performed practically in any situation" (Novozshilov, 2003a).

An important notice. *The positive effect of exercise on breathing is the experience of healthy people. As we saw in Chapter 2, physical exercise does not have the same effect on sick people. The aCO_2 of sick people gets lower during exercise with moderate or even light intensities. Thus, in poor health the possible benefits from exercise are limited and, in severe cases, even impossible to achieve. In fact, for critically ill patients any physical exercise can have disastrous health consequences.*

3.3 Overeating

According to Russian studies, when our breathing gets slightly heavier and aCO_2 concentrations decrease, glucose is driven from the blood into fat cells since CO_2 influences permeability of membranes of fat cells in relation to blood glucose. Hence, most people, in condition of chronic hyperventilation, gradually accumulate extra-weight. Eating more, on the other hand, is the stress for organs of digestion. Therefore, breathing becomes heavier at the end of the digestive process.

The amplitude of these changes is proportional to the caloric value and type of meal eaten. Therefore, with larger meals, especially ones with fats and proteins, these effects are more significant. As Doctor Buteyko suggested, when digested substances are in the blood, they are to be used or metabolised by body cells. This cellular consumption means "inner breathing". Thus, the respiration of cells (this term is normally used by certain microbiologists), especially in case of overeating, is intensified. That causes increased ventilation in the human organism (Buteyko, 1977). Overeating, according to Doctor Buteyko, has the worst possible consequences for respiration.

Doctor Buteyko also found that meals rich in proteins (especially when they are quick absorbing animal proteins) and, to a lesser degree, fats considerably intensify breathing, while fresh fruits and vegetables produce the least impact on ventilation. Why? One reason is due to varying availability of digestive enzymes. Fresh fruits, for example, often have their own enzymes for self-digestion making their digestion easy. Cooked meats and fats are hard to digest. Second, the amino acids cause blood acidification. Therefore, the CO_2 removal (or over-breathing) is required to restore the blood's normal pH. Third, some essential amino acids can directly affect the breathing centre and intensify respiration (chapter 8).

An old study by Haselbalch (1912) revealed that after following a vegetarian meal, aCO_2 decreased to 43.3 mm Hg (the initial value was about 45 mm Hg); while a meal with meat resulted in 38.9 mm Hg. Such a difference means that a BHT after a meat meal can be about 12 s less, than after a vegetarian one. Explaining this finding in his textbook on respiration, Professor Haldane suggested, that "*a meat diet, which causes an increase of sulphuric and phosphoric acids in blood, is acid-forming as compared to a vegetable diet, which contains less protein and relative*

abundance of salts yielding carbonates"(p.183, Haldane, 1922). Thus, the breathing centre compensates for the additional acids (amino acids) in the blood and the resulting blood acidification by reducing carbonic acid and CO_2 stores. While with the vegetarian meal, the presence of additional alkaline salts in the blood requires extra acids for blood pH preservation. Among all acids in the blood, carbonic acid is the main component and its concentration can be changed by respiration.

These ideas provide some explanation why alkaline diets are considered to be healthy in the management of various health problems (fruits and vegetables yield alkaline residues in the blood, when they are consumed), while acidic diets (that include meats, fish, eggs, dairy products, most grains, legumes and nuts) less so.

In addition to the immediate effects on respiration, a lack of normally occurring food substances in the diet such as vitamins and minerals can gradually cause chronic hyperventilation. For example, carbohydrates require for their digestion adequate amounts of B vitamins. These vitamins are naturally present in cereals, whole grains and root vegetables and almost absent in sugar, white bread and white rice. Thus, eating these refined products diminishes the B vitamin content in nervous cells gradually leading to chronic hyperventilation (Buteyko, 1977). Doctor Buteyko and his colleagues particularly emphasized the dangers of sugar and refined products. The lack of some minerals (especially Mg, Zn, and Ca) or their biochemical unavailability is another cause of chronic over-breathing.

Therefore, the typical western diets, which are often full of refined products and lack fresh fruits and vegetables, has negative effects on breathing. Most of all, overeating, so prevalent nowadays, is one of the major causes of chronic hyperventilation.

3.4 Deep breathing exercises

Deep breathing exercises, according to Doctor Buteyko, were the main cause of hyperventilation in the general population of the USSR (Buteyko, 1977). Indeed, until the 1980s, physical gymnastics combined with deep breathing were encouraged and promoted by central radio and TV programs, magazine and newspaper articles. As a result of such propaganda ("*breathe deeper in order to bring more oxygen and remove more "poisons" from the organism*"), schools, colleges, universities, boy-scout and military camps, health retreats, sanatoriums, hospitals, many state factories, and other establishments included voluntary deep breathing (hyperventilation), which was synchronised with dynamic movements of body limbs during daily gymnastics. Prolonged and frequent repetitions of such exercises gradually reset the breathing centre to a lower aCO_2 value, causing chronic hyperventilation years later (Buteyko, 1977).

However, this practice was mostly stopped after the Directive of the Soviet Health Minister "*Practical actions for application of the method of voluntary regulation of depth of breathing for treatment of bronchial asthma*" (Directive N 591, 30 April 1985, signed by USSR Health Minister S. Burenkov). Thus, Soviet medical authorities officially accepted the Buteyko method of relative hypoventilation (reduced breathing), as a safe and natural alternative to drugs in relation to asthma and bronchitis.

Some time ago, when I started to research medical literature on respiration, I found only one study, which suggested the usefulness of hyperventilation (over-breathing), in contrast to the several hundred studies about the dangers of hyperventilation. Having said that, there are those rare conditions (discussed in chapter 1 and 6) when medical professionals may recommend hyperventilation, but they are the exceptions and not the rule.

Nevertheless, many people still believe that breathing more is healthy. When I polled people with the question: What is better for human health: to breathe more or less, I found that well over 95% of my respondents believed that over-breathing, deep breathing, and hyperventilation were useful and could be practised. Only less than 5% disagreed. Most of them professionally studied physiology and/or medicine.

It follows from these facts that the ideas about the usefulness of hyperventilation and over-breathing are still among the greatest modern health-related misconceptions.

The actual physiological effects of these misconceptions are probably limited, since few people routinely practice hyperventilation exercises. However, there are many people, who couple deep breathing with physical exercise, dancing, singing, playing musical instruments, praying and other activities. In addition, periodic and deliberate sighing, excessive coughing, and other over-breathing actions are still considered normal by many people.

How is it possible then that people do not notice that acute voluntary over-breathing produces a negative impact on their health? The answer can be found in the results of experiments conducted by researchers at the University of Oxford, England. In their abstract, which was partly quoted above, they wrote,

"A cognitive explanation of the association between acute hyperventilation and panic attacks has been proposed: the extent to which sensations produced by hyperventilation are interpreted in a negative and catastrophic

way is said to be a major determinant of panic. Non-clinical subjects were provided with a negative or a positive interpretation of the sensations produced by equivalent amounts of voluntary hyperventilation. As predicted, there was a significant difference between positive and negative interpretation conditions on ratings of positive and negative affect. Subjects in the positive interpretation condition experienced hyperventilation as pleasant, and subjects in the negative interpretation condition experienced hyperventilation as unpleasant, even though both groups experienced similar bodily sensations and did not differ in their prior expectations of the affective consequences of hyperventilation. When the subjects were given a positive interpretation, the number of their sensations correlated with positive affect; when a negative interpretation was given, the number of bodily sensations correlated with negative affect…" (Salkovskis & Clark, 1990).

It follows from these results that, when a person has positive expectations about his activity, which is accomplished with deliberate over-breathing, his brain, due to the resulting excitement and hypoxia, is inclined to select positive interpretations and final effects, while ignoring any certain rational and/or critical thoughts. For example, a deliberately hyperventilating person can have a tendency to exaggerate his abilities and achievements without noticing his wakneses and the possibilities for own improvement.

Deep breathing for relatively healthy people usually means increased ventilation. It is rather rare for people to voluntarily practice deep breathing that results in lower minute ventilation (so that they breathe less). Even if it were possible, such deep breathing would produce increased tension. You can check this. First, you must relax completely. You may note that such relaxation causes a naturally quiet exhalation and subsequent automatic breath holds or suspended breathing. Vice versa, large inhalations and exhalations will often result in muscular tension in the respiratory muscles. That increased muscular tension will mean more stress, and more stress is the main cause of hyperventilation. Therefore, deep breathing causes over-breathing to follow, due to the stress generated.

Deliberate deep and slow breathing can create another effect due to the large amplitude of chest and/or diaphragm movements. Indeed, such amplitude can be still present many hours later after the practice, but with more normal individual breathing frequency, indicating the initial development of chronic hyperventilation. The person, due to possible nervous excitement and impaired rationality (both caused by low aCO_2), may even periodically remind himself to continue deep breathing in spite of possible chest tightness, dizziness and nausea resulting from excessive ventilation.

Therefore, deep breathing in most people encourages a more stressful state and subsequent hyperventilation.

About exceptions. In my view, only very healthy people (with the usual CP above 60 s) can practice slow deep breathing exercises, like pranayama, while learning hatha yoga. Such people are sufficiently relaxed and their breathing is well mastered. By the way, old hatha yoga manuscripts clearly stated that pranayama could be started by the yoga students only when their bodies were purified and their breath holding time was over 1 minute.

3.5 Overheating

Overheating, according to Doctor Buteyko, is another factor that intensifies breathing. That especially relates to many children, who suffer from the belief by loving parents and care-takers, that excessively warm dressing is healthy. A child's metabolism is 2-3 times higher than that of adults. Hence, their bodies normally generate much more energy. Accordingly, healthy children need much less clothing (Buteyko, 1977). Even when children wear minimum clothing, one may see that their hands and feet are usually warm. Only when their hands and feet are cold, adding more clothes or other actions are necessary.

Similar considerations can be applied to adults. During summer, most people wear shirts, t-shirts, dresses and other very light clothes, while going anywhere, outdoors and indoors. Here it would be useful to spend less time in hot and warm places. When it gets colder, people start to wear pullovers, sweaters, jumpers, jackets, coats, suits, etc. However, they wear these heavy clothes even when they are indoors where the temperatures are high. You can see that phenomenon in libraries and offices, shops and waiting rooms, cars and public transport.

Investigations on the influence of heat on breathing found that changes in air wet-bulb temperature, from 17 to 40 degrees, caused a fall in carbon dioxide pressure from 44 to 33 mmHg for healthy male subjects (Gaudio & Neil, 1968). After some math, it follows, that their BHTs fell from 46 to 8 s due to the effects of the strong heat. Thus, if the assumption of linear influence of temperature on ventilation is accepted (although that may not be true for the whole range of values), one may conclude that each two degrees of increased surrounding temperature produce over 1 mmHg of carbon dioxide down (or minus about 2s BHT).

Rapid changes in temperature can increase breathing as well. Sudden cold immersion usually produces severe hyperventilation during the first few breaths. This topic will be discussed in more detail later.

3.6 Talking with deep inhalations, a loud voice, or a high pitch

During lectures and public speeches, or when just talking, it is important not to take deep in-breaths between phrases (Buteyko, 1977). Often, people start their sentences and phrases after deep inhalations quickly blowing out the air, together with precious carbon dioxide, from their lungs. That is a current feature of modern talking style and it can be routinely observed with many TV reporters and commentators. Such a speaking style makes the speech more appealing or even dramatic for viewers. However, it also increases ventilation causing reduced carbon dioxide stores.

A study entitled "*Influence of continuous speaking on ventilation*" (Hoit & Lohmeier, 2000) revealed the following (abstract).

"This study was conducted to explore the influence of speaking on ventilation. Twenty healthy young men were studied during periods of quiet breathing and prolonged speaking using noninvasive methods to measure chest wall surface motions and expired gas composition. Results indicated that all subjects ventilated more during speaking than during quiet breathing, usually by augmenting both tidal volume and breathing frequency. Ventilation did not change across repeated speaking trials. Quiet breathing was altered from its usual behaviour following speaking, often for several minutes. Speaking-related increases in ventilation were found to be strongly correlated with lung volume expenditures per syllable. These findings have clinical implications for the respiratory care practitioner and the speech-language pathologist."

The authors found that average ventilation increased from resting 7 l/min to almost 14 l/min during speeches. Average initial end-tidal CO_2 pressure of these healthy (by year 2000 standards) young American men was almost 38 mm Hg. After 10 minutes of speaking it dropped to about 31 mm Hg. A quick calculation shows that their average initial BHT was about 29 s, after 10 min speaking their average BHT was correspondent to 14s, if the formula connecting aCO_2 and BHT is applied. For most subjects even many minutes of recovery were not enough to completely restore initial CO_2 levels.

Actual BHT values after such speeches are going to be much larger due to effects created by acute over-breathing. When current carbon dioxide value is less than the usual set-point established by the breathing centre due to acute hyperventilation, BHT gets larger in healthy people (chapter 2), while the BHT (or the CP) measurements require 5-10 min of rest with normal breathing.

Talking with deep inhalations, raising one's voice in terms of loudness, elongation of vowels, and high pitch phrases will lead to worse results, even when such talking is emotionally neutral. Should this talking be infused with emotions, especially strong ones, higher increases in ventilation can expected.

Since nowadays, there are many people, who have to speak for hours daily, the negative effects of poor speaking skills on one's health can be substantial. More detailed suggestions about correct ways of speaking will be given later.

3.7 Mouth breathing

When seeing people on Western city streets and in other public places, one may notice that up to 20-40% of them breathe through their mouths while walking or even while quietly standing or sitting. This phenomenon seems more common among children. What are the negative effects of mouth breathing?

Reduced oxygenation and carbon dioxide stores

Scientific literature on respiration often mentions a physiological parameter known as dead space volume. It is about 150-200 ml in an average adult: inside the throat, nose and bronchi. This space preserves additional carbon dioxide for the organism, since inhalations take CO_2 enriched air from dead space back into the lungs. When the mouth is open, the dead space volume becomes smaller due to a continuous exchange of air. That does not happen with nasal breathing. In addition, nasal breathing provides more resistance to respiratory muscles during breathing as compared to mouth breathing (the mouth-breathing route is shorter and has a larger cross sectional area). During nasal breathing we can either breathe more resulting in more mechanical work for the respiratory muscles or to breathe slightly less while generating less mechanical work. What is the practical result? Due to an in-built tendency to optimise physiological processes, the human organism is ready, as during nose-breathing, to breathe less and tolerate higher aCO_2, than to exert more demands on constantly working respiratory muscles.

In the abstract of a physiological study "*An assessment of nasal functions in control of breathing*" (Tanaka et al, 1988) the researchers wrote:

"Breathing pattern and steady-state CO_2 ventilatory response during mouth breathing were compared with

those during nose breathing in nine healthy adults...We found the following. 1) Dead space and airway resistance were significantly greater during nose than during mouth breathing. ...These results fit our observation that end-tidal PCO_2 was significantly higher during nose than during mouth breathing. It is suggested that a loss of nasal functions, such as during nasal obstruction, may result in lowering of CO_2, fostering apneic spells during sleep."

This Japanese article, which studied a group of healthy volunteers, gives average end-tidal CO_2 43.7 mm Hg for nasal breathing and only 40.6 mm Hg for mouth breathing. Practically, in terms of BHT that means 45 s and 37 s at sea level. Hence, mouth breathing reduces oxygenation of the whole body.

Cleaning, warming and humidification of air

Our nasal passages are created to clean, humidify and warm the incoming flow of air due to the thin layer of protective mucus. This layer of mucus can trap almost all (95-99%?) dust particles, bacteria, viruses and other airborne objects. This is what long, narrow, and intricate nasal airways are for.

When the mouth is used for inhalation, this air route is shorter, wider and almost straight. Now the same airborne objects can easily get into the alveoli and the blood, creating stress, first of all, for the immune system (detection, marking, isolation, and deactivation of intruders) and then for organs of elimination (kidneys, liver, skin, and GI patches). Some pathogens, can even multiply in the lungs causing more severe problems.

If you are an asthmatic and endurance athlete, you should train mostly, or better only using your nose, and for really important competitions mouth-breathing can be used, if you have no problems with your asthma. What is important for training is to have an aerobic training effect. That is possible while breathing through the nose, as these western results indicate.

"The major cause of exercise-induced asthma (EIA) is thought to be the drying and cooling of the airways during the 'conditioning' of the inspired air. Nasal breathing increases the respiratory system's ability to warm and humidify the inspired air compared to oral breathing and reduces the drying and cooling effects of the increased ventilation during exercise. This will reduce the severity of EIA provoked by a given intensity and duration of exercise. The purpose of the study was to determine the exercise intensity (%VO_2 max) at which healthy subjects, free from respiratory disease, could perform while breathing through the nose-only and to compare this with mouth-only and mouth plus nose breathing. Twenty subjects (11 males and 9 females) ranging from 18-55 years acted as subjects in this study. They were all non-smokers and non-asthmatic. At the time of the study, all subjects were involved in regular physical activity and were classified, by a physician, as free from nasal polyps or other nasal obstruction. The percentage decrease in maximal ventilation with nose-only breathing compared to mouth and mouth plus nose breathing was three times the percentage decrease in maximal oxygen consumption. The pattern of nose-only breathing at maximal work showed a small reduction in tidal volume and large reduction in breathing frequency. Nasal breathing resulted in a reduction in FEO_2 and an increase in $FECO_2$. While breathing through the nose-only, all subjects could attain a work intensity great enough to produce an aerobic training effect (based on heart rate and percentage of VO_2 max)" (abstract, Morton et al, 1995).

Autoimmunization effect

The layer of mucus moves as a carpet from sinuses, bronchi and other surfaces towards the stomach. Hence, these trapped by the mucus objects are drained (or swallowed) into the stomach where digestive enzymes and HCl (low pH) make bacteria and viruses either dead or weak. Further along the digestive conveyor, some of these substances can penetrate into the blood due to intestinal permeability effect. But now these pathogens are either dead or weakened and would not do much harm while providing a good lesson for the immune system. This is exactly how immunization, done by medical personnel, often works with success: medical doctors inject a vaccine with either dead or weakened bacteria or virus to teach and strengthen the immune response to these pathogens. Hence, nasal breathing is a natural mechanism of autoimmunization.

Practically, when some household members are sick with for example, flue or cold, the still healthy people can breathe either through the nose teaching the own immune system how to defeat the pathogenic bacteria or virus, or through the mouth allowing the same pathogens to access, settle and multiply in various parts of the human body causing the infection.

Nitric oxide

Production and utilization of own nitric oxide that is synthesized in the sinuses depends on the breathing route. Some important functions of this hormone were discussed in Chapter 1.

Compare old and modern group photos. They also provide a part of the answer regarding causes of poor health in contemporary society.

The mouth, according to Doctor Buteyko, is designed by Nature for drinking, eating and speaking. At all other

times it should be shut. (Teeth flossing and brushing can be other sensible exceptions.)

3.8 Morning hyperventilation

For most people, even healthy ones, breathing is heaviest during the early morning hours. A group of Italian scientists from Centro di Medicina Subacquea e Iperbarica (CEMSI) in Salerno investigated, according to the title of their article *Voluntary breath-holding in the morning and in the evening*. They revealed that, *"During the evening sessions, most of the BHT/Delta P ACO_2 ratios… were higher than the corresponding morning values…"* (Bosco et al, 2004).

In sick people the effect is even stronger. Let us first consider asthma.

American pediatricians from the Washington University School of Medicine in St. Louis in their publication note, *"BACKGROUND: Symptoms from asthma are often prominent at night. In adults significant circadian variation has been shown with reduced peak expiratory flow rates and increased bronchial reactivity to methacholine in the early morning hours"* (Porter et al, 1999).

A group of Brasilian medical scientists investigated, according to their title *Morning-to-evening variation in exercise-induced bronchospasm* (Vianna et al, 2002). Their objective was *"to compare morning and evening EIB [exercise-induced bronchospasm] and minute ventilation during exercise (VE)"* (Vianna et al, 2002). Indeed, baseline FEV1 was significantly lower during early morning hours, while minute ventilation higher.

Over 30 years ago the *Thorax* published a study *Physiological patterns in early morning asthma* (Hetzel, et al, 1977). The goal of the study was also to explain *"the sudden nature of some asthma deaths as these often occur in the early morning"* (Hetzel, et al, 1977).

Several other publications were devoted to effects of sleep on patients with COPD (chronic obstructive pulmonary disease). American scientists from the Yale Center for Sleep Medicine (Yale University School of Medicine, New Haven) wrote, *"Symptoms related to sleep disturbances are common in individuals with moderate to severe COPD, particularly in the elderly, which is commonly manifested as morning fatigue and early awakenings. One major cause of morbidity in this population is abnormalities in gas exchange and resultant hypoxemia as they can lead to elevated pulmonary pressures, dyspnea and in severe cases right ventricular overload and failure. Sleep has profound adverse effects on respiration and gas exchange in patients with COPD…"* (Urbano & Mohsenin, 2006).

Moreover, Sheppard and colleagues in the publication from the *Chest* magazine noted, *"Epidemiologic investigation has revealed that patients with pulmonary disease are at increased risk of dying during the early morning hours"* (Sheppard et al, 1984). The load on the heart muscle during the episodes of hypoxemia during sleep can be, according to their conclusion, *"can be transiently as great as during maximal exercise"*.

If patients with pulmonary conditions can die due to heart problems at night, what about heart patients themselves? *"Coronary spasm occurs most often from midnight to early morning when the patient is at rest"*, (Yasue & Kugiyama, 1997) says the Japanese study *Coronary spasm: clinical features and pathogenesis* published in the *Internal Medicine* magazine.

Intensive care professionals from Department of Anaesthesia and Intensive Care Medicine of the Hadassah Medical Centre in Jerusalem, Israel also decided to investigate, according to their title, *In-hospital cardiac arrest: is outcome related to the time of arrest?* They wrote,

"BACKGROUND: Whether outcome from in-hospital cardiopulmonary resuscitation (CPR) is poorer when it occurs during the night remains controversial. This study examined the relationship between CPR during the various hospital shifts and survival to discharge…CONCLUSIONS: Although unwitnessed arrest is more prevalent during night shift, resuscitation during this shift is associated with poorer outcomes independently of witnessed status" (Matot et al, 2006)

Trying to explain the cause of deaths, Turkish cardiologists from Ankara published a study *Circadian variations of QTc dispersion: is it a clue to morning increase of sudden cardiac death?* They explained, *"BACKGROUND: Several studies related to cardiac events including sudden death have shown a peak incidence in the early morning hours. Our data suggest that QTcD has a circadian variation with an increase in the morning hours, especially in patients with coronary artery disease. This finding was thought to be an explanation for the role played by sympathetic nervous system in the occurrence of acute cardiac events and sudden death during these hours"* (Batur et al, 1999).

A group of American cardiologists from the Georgetown University Medical Center in Washington, D.C. was also interested in the most likely time of death, *"The time of death was available in… 96 of the 139 patients who died suddenly. There was a circadian variation of all SCDs [sudden cardiac deaths] compared with other deaths with a*

distinct peak during the morning (p = 0.04)" (Behrens et al, 1997).

Swiss medical doctors explained in their abstract, *"Prinzmetal's angina is a variant of the classic exertion dependent angina pectoris. Typical is the appearance of the symptoms at rest during early morning hours. It is due to spasms in the coronary arteries. Various provocation tests may be used to trigger spasms, among others hyperventilation which leads to vasoconstriction of coronary arteries"* (Jacob et al, 1994).

Even healthy people have heavier breathing and lowered oxygenation of the brain during early morning hours, as Australian scientists from Latrobe University in Melbourne revealed. After testing health subjects, these scientists concluded, *"These data indicate that normal diurnal changes in the cerebrovascular response to CO(2) influence the hypercapnic ventilatory response as well as the level of cerebral oxygenation during changes in arterial Pco(2); this may be a contributing factor for diurnal changes in breathing stability and the high incidence of stroke in the morning"* (Cummings et al, 2007).

It is not a surprise then that British researchers from the National Heart and Lung Institute (Imperial College, London) also noted, *"The reduction in hypercapnic cerebral vascular reactivity that occurs in the morning after sleep is associated with an increased risk of cerebral ischemia and stroke"* (Meadows et al, 2005).

Similarly, Californian neurologists wrote, "*This reduced morning response to hypercapnia suggests diminished vasodilator reserve during this period, and may be related to the increased stroke risk during the morning hours"* (Ameriso et al, 1994).

Patients with diabetes also suffer from lower oxygenation in tissues during nights: *"Circadian rhythms of tissue oxygen balance and blood rheological properties were investigated in 40 patients with insulin dependent diabetes mellitus... Preserved blood hyperviscosity and increasing tissue hypoxia at night indicated stable disturbance of hemorheological properties and tissue oxygen balance"* (Galenok et al, 1988)

Japanese doctors from the Department of Pathology for the Handicapped in Ehime University warn that those who care about people with epilepsy should know about higher chances of seizures during nights, *"…S-w paroxysms combined with clinical symptoms and continuing for more than four seconds were fewer during the afternoon than the morning and, moreover, during sleep. …Therefore, the observation of typical absence seizures during the morning should be regarded as important"* (Nagao et al, 1990)

A marker of inflammation, C-reactive protein, was measured during different parts of the day in obese patients (Punjabi & Beamer, 2007). The conclusion of these medical scientists from the Johns Hopkins University in Baltimore was in the title of the study, C-reactive protein is associated with sleep disordered breathing independent of adiposity. Hence, it is not just obesity, but disordered breathing at night that can lead to inflammation: *"…the results of this study suggest that mechanisms other than adiposity per se could contribute to the inflammatory state seen in adults with SDB [sleep disordered breathing]"* (Punjabi & Beamer, 2007).

Since inflammation can get worse during nights in many patients, is it possible that cortisol production (cortisol is one of the key hormones to fight inflammation) also decreases at nights? German researchers suggested, *"Some studies found patterns of enhanced or blunted waking cortisol responses observed under chronic stress, burnout, or post traumatic stress disorder… The morning cortisol increase typically observed in healthy subjects and also observed in the control group was absent in the amnesic patients… Further studies are needed to understand the neurological or psychological mechanisms leading to a missing morning cortisol response in amnesic patients"*(Wolf et al, 2005) .

"Approximately two-thirds of women experience nausea or vomiting during the first trimester of pregnancy. These symptoms are commonly known as morning sickness" (Flaxman & Sherman, 2000). It is known that pregnancy of modern women means chronic hyperventilation (see below). Many of these women have even heavier breathing in comparison with their usual daily chronic hyperventilation.

What are the causes of morning hyperventilation? They are numerous.

Presence of disease and existing damage in the body

Dr. Buteyko, during his public lecture at the Moscow State University, said, *"The horizontal position, lying intensifies breathing. Patients with asthma, heart disease, hypertension, and stenocardia often have acute states at night. If they lie down during the day time, or lie for 2-3 hours – the breathing gets heavier, the attacks come. Many severely sick patients sit, afraid to lie down. This is sensible. We should lie down only for sleeping. Our patients cannot control their breathing at night, and hence, sleep is poison for them"* (Buteyko , 1977).

Sleeping too long

Sleeping too long, according to Buteyko, intensifies breathing causing prolonged periods of gradually increasing hyperventilation (p.177, Khoroscho, 1982)

Sleeping on the back

Among body positions, sleeping on the back (supine position) is worst (some people start snoring in this position). Sleeping on the right side also causes increased ventilation in comparison with sleeping on the left side or on the chest. Children's ventilation is minimum when they are sleeping on their tummies (Buteyko, 1977).

There are no physiological studies known to me in which such parameters of breathing, as minute ventilation and aCO_2, are monitored in different sleeping body positions. Meanwhile, there are at least 8 studies devoted to the physiological effects of different body night positions on the stomach. All these investigations, trials included, advocate the left position due to the least night-time recumbent reflux (e.g., Khoury et al, 1999). That means that the liquid content of the stomach, for the left position cannot escape or leak due to the absence of physical contact with any of the stomach valves. This position is also the most favourable for general peristaltic waves in the large intestine due to the gravitational effect in the transverse colon. There are, possibly, some other reasons to explain the advantages of the left position (e.g., the influence of the heart position). Some people, due to congenital reasons, have their hearts on the right side. In such cases the effects of sleeping positions are also in reverse.

A simple "sleeping positions" self-test. By measuring breath holding time in different sleeping positions, one can find out what is the optimum position for breathing and health. Note that one needs to spend about 10-15 minutes in a certain position in order to achieve a stable metabolism correspondent to this position. A ticking mechanical clock can be used for that purpose. Generally, sleeping on the left side will produce the highest BHT, followed by sleeping on the tummy. Sleeping on the back is worst and causes lowest BHT.

Other causes

Among other causes are poor air quality, soft beds, abnormal thermoregulation, etc.

3.9 Embryonic and foetal development in a woman hyperventilating during her pregnancy

Thorp & Rushing (1999) devoted their study to the review of over 50 references regarding umbilical cord blood analysis. They found that "*because of the progesterone-induced hyperventilation of pregnancy, maternal PCO_2 decreases to 31 to 34 mm Hg from the nonpregnant state value of 39 to 40 mm Hg*". Their article also presented a table of studies reporting umbilical cord arterial blood gas values. Average carbon dioxide numbers ranged from 49 to 56 mm Hg, while oxygen content was only between 15 to 24 mm Hg, indicating extreme hypoxia in the embryo.

A textbook "*Fetal Physiology and Medicine*" (Hugh & Hugh, 1984) also revealed that "*In comparison to maternal blood, the pCO_2 is higher, pO_2 and sO_2* [oxygen saturation] *values are strikingly lower* [for fetal blood]..."(Hugh & Hugh, 1984). The oxygenation of the blood of the human foetus is usually about 5-7 times less than that of the normal adults. At the same time, carbon dioxide content of the growing foetus is significantly higher than normal adult values. For example, in the middle of pregnancy Soothill and colleagues (1986) measured the umbilical arterial carbon dioxide as 10 mm Hg greater than maternal arterial CO_2 pressure.

Hence, the human embryo grows in an environment containing much less oxygen and more CO_2 in comparison with normal adult values. But since many women hyperventilate before pregnancies, their aCO_2 values during pregnancies are much lower, than their physiological norms. Her embryo receives all its nutrients, carbon dioxide included, through the umbilical cord via the blood. Therefore, the embryo gets exactly the same CO_2 concentrations, as his mother. Due to Nature’s design, carbon dioxide concentrations of the embryo are much greater than normal human values, but the lowered aCO_2 of the mother tends to reduce the CO_2 stores of the embryo.

Hence, when the mother hyperventilates, her embryo also hyperventilates in relation to its physiological norms. Such a baby is already developing and will be born in the state of over-breathing.

Moreover, studies of pregnant women done by Doctor Buteyko revealed that numerous complications during pregnancies and deliveries are possible only when carbon dioxide stores are low. That relates, for example, to spasms of ovaries, excessive hypoxia of the embryo, and spontaneous abortions. Babies born with over-breathing often suffer from various infections, skin rashes, pneumonia, asthma, eczema, and other problems. These ailments are absent in babies with normal aCO_2 (Buteyko, 1977).

During the birth there are dramatic changes in CO_2 and O_2 tensions. The summary of these studies indicates that, on average, carbon dioxide concentration drops from over 8% to about 5% during the first hours of life (Respiration and Circulation, 1971). Scientists often describe the birth as a powerful stress. Indeed, such large changes in blood gases must profoundly affect the biochemistry of the whole organism.

3.10 Special factors for infants

Before the baby is born, the fetus gets all its blood supply from the mother through the umbilical cord. This includes CO_2 and O_2. Because of this, the breathing of the fetus depends solely on the mother's breathing and when she hyperventilates, her unborn baby also hyperventilates.

Birth itself is a severe shock for the baby. Probably, the central part of this shock is a drastic drop (about 30%) in blood CO_2 concentrations. The process of delivery and new environmental conditions cause stress and make their breathing heavy. In order to make the transition more gradual, all primitive and recent cultures used swaddling (or tight wrapping) of babies. When meeting people born and raised on different continents, I was reassured that swaddling was the norm in Africa, America, Europe and Asia. In Asia, due to hot climate, they used, instead of clothes, wooden sticks, which were applied along the trunk and tightly tied using ropes. In Scotland, swaddling blankets were passed from generation to generation. Swaddling prevents unduly deep chest breathing and restricts their total ventilation. In other words, they breathe less. Modern western civilization and our health care systems have gradually lost this wise cultural tradition.

In his public lectures, Buteyko said, *"But the cells of animals and humans need about 7 % CO_2 and only 2% O_2 in the surrounding environment. This is the way our cells live: cells of the heart, brain, and kidneys. But now air has 10 times more oxygen and 250 less carbon dioxide, i.e., it is not suitable for our cells and is poisonous in its composition. This is confirmed by embryology. During recent years detailed studies of gas blood exchange in embryos of humans and animals were done. It was found that during 9 months we live in an environment, which has 3-4 times less oxygen and 1.5 times more CO_2 (both as partial pressures) in comparison with adults. Obviously, the organism of the mother creates such conditions for the embryo, as they were billions of years ago. This supports the Law of Gekkel-Severtsev: the embryon, in its development, repeats the filogeneses. After birth, during the first breaths, there is a sudden increase in blood oxygenation and a sudden drop in CO_2. It is known that the child is virtually disease-free in the womb of the mother. Only after the birth, do diateses and all other abnormalities of metabolism appear. Why? There is a sudden change in air. The wisdom of East surprises us: the just-born infant is tightly swaddled, and in some places even tightened to a wooden plate. The chest is covered by layers of heavy material [voilok]. Our grandmothers covered the cradle with the infant using material covering [leaving a small hole for air exchange], and used swaddling too… Folk wisdom understood, that this air, so poisonous for the newborn, requires gradual adaptation"* (Buteyko, 1977).

Another special factor for children relates to their high metabolic rate. Metabolism and heat generation of children is much higher than that for adults. Having simlar clothes, as adults, can damage their health. What is the criterion of feeling cold for the babies? It is too cold for them when their feet or arms are cold. A quick touch by hand and feet can check if the baby is warm or cold. Overheating is one of the leading causes of poor health in modern babies and children.

"The metabolism of the child is about 2-3 times higher. When it is cold for an adult, children feel comfortable. But they are provided with 5 layers of clothing and then a hat on top … Overheating intensifies breathing and the child gets a cold, not from a draught, but from own hyperventilation. Then he is even more insulated, more overfed… It is well known: in a large and poor family, where bread and potatoes, there is only one shirt for all [children], [they] run on the snow barefoot and all are healthy" (Buteyko, 1977).

3.11 Nutritional deficiencies

Due to intensive farming and food refining methods, our food is less nutritious than 100 or more years ago. We get less essential fatty acids (omega oils), calcium/magnesium, fiber, zinc, trace-metals and other essential nutrients. Nutritional deficiencies, depending on one's personal make-up, create stress for certain organs, body parts, and systems of the human organism. This stress intensifies chronic hyperventilation.

3.12 Exposure to toxic chemicals

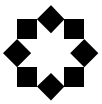
Toxic chemicals, once in the human organism, can generate different waste products, interfere with hormonal balance, and influence the nervous, digestive, cardiovascular and other systems. These negative changes sooner or later cause over-breathing. The mere appearance of bacteria or large amounts of waste products from bacteria in the blood would be sufficient to cause heavy over-breathing (chapter 1).

Therefore, environmental, professional, dietary and any other exposure to heavy metals, pesticides, herbicides, and chemicals due to pollution are also causes of chronic hyperventilation. Many medical drugs intensify respiration.

"Antibiotics (penicillin, streptomycin etc.) intensify breathing. After 2-3 weeks of such treatment, the state [of health] unavoidably gets worse. What is the mechanism? Antibiotics fight microbes, suppressing the breathing of micro organisms. All of the living world has one common foundation: metabolism. Therefore, antibiotics suppress the breathing of microbe cells and our cells. This causes excitement of the breathing centre, its disruption, in the direction of intensification. Moreover, antibiotics create conditions for new allergies. Senseless, widespread use of antibiotics causes tremendous damage. Camfora, codein, cordiamin, adrenalin, theoephedrine, ephedrine – also intensify breathing. People use them senselessly, trying to cure themselves, and cause tremendous harm to themselves" (Buteyko, 1977).

Alcohol, soon after intoxication, suppresses the breathing centre and can increase the breath holding time about 2 times. Simialrly, marijuana and cocain can increase the BHT about 3-4 times. This change in breathing, according to considered physiogical laws, profoundly influences perception and feelings of the intoxicated person leading to increased confidence, logic, feeling of energy, coordination, sharper sensations in relations to smells, colours, etc. Later, the abnormal substances are to be removed by the immune system and the liver and kidneys causing heavy breathing and hangover. The breath holding time plunges below the initial values. Low oxygenation, poor blood perfusion generates many negative effects described above.

As a short summary that connects this chapter with diseases and individual symptoms discussed in Chapter 1, Doctor Buteyko noticed, "If the cause of hyperventilation is in the wrong lifestyle, that means you are bound to change your lifestyle or to fight with the symptoms all the time" (from Novozshilov, 2003b).



Conclusions

The following factors can cause chronic hyperventilation:

- Stress, anxiety and strong emotions
- Physical inactivity
- Overeating
- Deep breathing exercises
- Overheating
- Talking with deep inhalations, a loud voice, or a high pitches
- Mouth breathing
- Sleeping too much, sleeping on the back or on the right side
- Embryonic and foetal development in a woman hyperventilating during her pregnancy
- Lack of swaddling
- Nutritional deficiencies
- Exposure to toxic chemicals.

The references of this book provide experimental results in respect to various respiratory parameters of people over a period of about 100 years. By analysing this data and some other articles (all published in respectable medical and physiological magazines), the following average changes for people, who were considered healthy, took place during the 20-th century:

- Normal minute ventilation increased from 5-6 to 9-12 l/min. Healthy people breathe more.
- Normal breathing frequency increased from 9-12 to 12-15 times per minute. Healthy people breathe faster.
- Normal tidal volume (the amount of air for one breath) increased from 500-600 to 600-700 ml per breath. Healthy people breathe deeper.

As normal outcomes:

- Carbon dioxide concentrations (in the lungs and blood) decreased from 40-43 mm Hg to 36-39 mm Hg.
- The BHT (BHT after quiet exhalation) decreased from 40-50 s to 25-35 s.

The crucial parameter here is carbon dioxide concentrations, while other characteristics only support the idea that modern people, who are considered to be healthy, hyperventilate in relation to previous healthy generations.

In addition to that, there are sharp increases (ten times or more) in chronic degenerative health conditions.

As a result, average breathing of the typical modern Earth citizen indicates some degree of hyperventilation.

Hence, due to readjustment of the breathing centre to abnormal, from the evolutionary viewpoint,

environmental conditions and individual choices (discussed in this chapter), people now breathe much heavier than, for example, 100 years ago.

Q&A section for Chapter 3

Q: Do all emotions cause hyperventilation?
A: According to Doctor Buteyko, all strong emotions result in over-breathing. However, some emotional attitudes, in my view, do not have a negative impact on breathing. For example, when people experience reasonable shame, their breathing is normal or can be even reduced. Similarly, admiration in respect to art creations, Nature's phenomena, and human deeds causes naturally reduced breathing. These experiences are often described as "breath-holding" or "breath- taking (away)". By the way, healthy people, when opening their mouths, cease to breathe.

Q: What are the effects of music on respiration?
A: They are complex and individual. Studies found that one's breathing rhythm can follow the musical rhythm. Also, heavy or loud music and music with uneven rhythm cause more muscular tension increasing ventilation. Quiet, soft, and peaceful musical pieces induce relaxation, slower heart rate, and establish easy breathing. Music is another cultural factor, which often adversely influences the breathing of modern people, especially our younger generations.

Q: What is the dynamic of changes in ventilation and aCO_2 during sleep?
A: Healthy people breathe slower, ventilate less, and have higher aCO_2 during night sleep. Their sleep is short and refreshing. Many sick people usually also breathe less due to decreased metabolic rate, but their carbon dioxide concentrations get smaller. Therefore, they over-breathe. This process is especially pronounced during early morning hours (4-6 a.m.), as mentioned above.

As for the effects of different sleep stages, during deep sleep stages (or slow wave sleep), ventilation is reduced and that helps to deeply relax skeletal muscles. Carbon dioxide, a powerful muscle relaxant (chapter 1), can play a central role in this effect. REM (rapid eye movement) sleep can result in relative hyperventilation and low aCO_2 (Krieger, 1986).

Q: What is known about breathing and natural processes, like defecation and urination? Can they produce hyperventilation?
A: If a person has a desire to defecate but does not have a chance to do that, the breathing can become heavier. Indeed, from chapter 1 it is known, that raised aCO_2 increases blood flow to the brain and internal organs, together with the group of muscles, which is responsible for effective elimination (this group includes muscles of the descending colon, sigmoid colon, rectum and sphincter). Vice versa, lowered aCO_2 reduces blood flow to these muscles up to the possible spasmodic state. The relief, relaxation, and easy breathing after the defecation indicate reduced ventilation and increasing CO_2 stores.

The act of urination seems to be less connected with hyperventilation. Meanwhile, distension of the bladder causes more slow and shallow breathing (Schodorf & Polosa, 1980) leading to higher carbon dioxide values. Thus, deliberately keeping one's bladder full, somehow, reduces ventilation and, possibly, is not detrimental to one's health.

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Chapter 4. Western methods of breathing retraining

Introduction

Whenever the breathing of sick people is shown to be abnormal, it is then reasonable to inquire about the available methods of breathing retraining developed by medical professionals. This chapter provides a review of various Western methods, including the types of breathing exercises used, their duration and frequency, length of the therapy, and other auxiliary activities, as well as the immediate and long-term effects of each treatment.

The goal of almost all these methods was to reduce breathing and increase carbon dioxide values. The location names given before the titles of the publications listed below, identify the different research centres where a particular breathing retraining method has been employed.

It is possible that the researchers, as well as ordinary people, often have a misconception that breath means spirit (something immaterial); hence, breathing retrainig can address only emotional or psychological problems, not organic or metabolic diseases, which are accompanied by damaged organs or tissues or hormonal abnormalities. That was probably the reason why the patients for these therapies were usually diagnosed with “hyperventilation syndrome” or "panic attacks" as their main health symptom and concern.

References to the publications, which describe these therapies, are given at the beginning of the appropriate sections.

The last section of this chapter discusses the common features and differences of these Western methods of breathing retraining, as well as their advantages and disadvantages.

It must be noted that there is one method of breath retraining (the Buteyko method) which has not been considered in this chapter. This method shall be discussed later, as it deserves special attention.

4.1 University of California Medical School, San Francisco, USA

Kerr WJ, Dalton JW, Gliebe. *Some physical phenomena associated with the anxiety states and their relation to hyperventilation*, Ann Int Med 1937, 11: 962-992.

In the 1930s, it became obvious to a group of doctors that a large number of their patients (from one-fourth to one-third) had "*a variety of symptoms referable to many structures of the body; and in whom hyperventilation precipitates and maintains a state of hyperirritability approaching clinical tetany. The symptoms may be so well localized in some cases that local disease is suspected without discovery of universal functional disturbance*" (p. 961, Kerr et al, 1937).

As a treatment, the inhalation of O_2-CO_2 gas (70% O_2 and 30% CO_2) was found to be most effective both in speed and adequacy. In other situations, “*a paper sack, inverted over patient’s head and sealed with adhesive tape, is successful*” (p.989, Kerr et al, 1937).

Additionally, a prescription with ammonium chloride, in order to reduce ventilation, was given, although the authors noted that chloride ions were probably not as effective as carbon dioxide.

This demonstrated to the patient “*the mechanism of the physiological difficulties which he himself caused* [due to hyperventilation], *and the results are so dramatic, that he is able to follow the procedure and to appreciate what the results mean to him*” (p.989, Kerr et al, 1937).

However, the authors decided that the use of several chemical drugs, which could suppress the heart action and the respiratory rate, should be tried as the way to success.

4.2 Papworth Hospital, Cambridge, UK

Lum LC, *Hyperventilation: The tip and the iceberg*, J Psychosom Res 1975; 19: 375-383.
Lum LC, *The syndrome of habitual chronic hyperventilation*, in *Modern Trends in Psychosomatic Medicine*, edited by OW Hill, Butterworth, London, 1976, vol 3: 196-230.

Professor Lum insisted that the therapy must start with the HVPT (hyperventilation provocation test), which demonstrated to the patients that reappearance of their symptoms was directly related to their over-breathing. That brought psychological relief to the patients (normally causing easier breathing), since they were now convinced that one of the causes of their symptoms was proven by the test and could soon be treated by their learning about breathing retraining. Lum found that there are two factors in how soon the patients got well: the duration of symptoms and the

age of the individual. Indeed, it is normal to expect that the recovery rates should depend on the previous damage accumulated in the organism.

Usually, patients were referred to a physiotherapist in order to learn 3 key components of the therapy: awareness about normal and abnormal breathing patterns, relaxation, and diaphragmatic breathing.

Thus, patients were informed about the importance of achieving constant control of their breathing, as well as learning how to prevent such harmful acts as sighing, sniffing, coughing, deep breathing, chest inflation, and other unhealthy habits and factors, which encourage excessive breathing.

Relaxation physiotherapy included teaching the patients how to loosen up their habitually tense posture, and especially how to recognise and prevent muscular tension in the chest, shoulders, head and neck.

It was found that the majority of his patients had little, if any, control and use of their diaphragm. Hence, the physiotherapist taught them how to control it and to use it almost exclusively at rest, and how to suppress unnecessary thoracic movements or relax the breathing muscles of the chest.

In order to achieve normal breathing, "*an extremely slow respiratory rhythm is encouraged, in order to gradually persuade the respiratory centre to readjust to a higher level of arterial carbon dioxide: a slow process if the disorder has persisted for many years*" (p.226, Lum, 1976). Then "*the patient is initially instructed to devote two periods of 20 minutes each day to breathing exercises, and to constantly check his breathing throughout the day*" (p.226, Lum, 1976).

He treated 320 patients (up to 1974) using this therapy. 70% were rendered completely asymptomatic. 25% had improved and 5% failed to respond. 18 patients were diagnosed by a cardiologist as having organic cardiac disease. Out of these patients, only 3 failed to improve.

4.3 Portland Veterans Administration Medical Centre, USA

Magarian GJ, *Hyperventilation syndrome: infrequently recognized common expressions of anxiety and stress*, Medicine 1982; 61: 219-236.

Magarian GJ, Middaugh DA, Linz DH, *Hyperventilation syndrome: a diagnosis begging for recognition*, West J Med 1983; 38: 733-736.

As a first practical step with this approach, it was "*important that the patients be confronted with the cause-and-effect relationship and their symptoms. A hyperventilation trial is crucial for therapeutic success*" (p. 736, Magarian et al, 1983). During the HVPT the patient breathed deeply at a rate 30 to 40 times per minute. Most patients experienced their symptoms within minutes or seconds. Such recognition was a major factor for successful overall outcome of the therapy.

Given its nature, the test should be conducted cautiously for patients with ischemic coronary disease, sickle cell disease, cerebrovascular insufficiency and baseline hypoxemia. Meanwhile, "*it is of far greater detriment for the patient not to recognise the relationship between over-breathing and their symptoms than the potential risks of performing the test*" (p.231, Magarian, 1982). The suggested duration of the test was 4-5 minutes.

When the HVPT was done, breathing into a bag resulted in quick alleviation of the symptoms. Finally, the patients were encouraged to use relaxation therapy and also taught how to retrain their own breathing pattern, from thoracic to diaphragmatic. The authors reported about the low efficiency of different medications for the normalisation of breathing.

4.4 St. Bartholomew's Hospital, London, UK

Bonn JA, Readhead CP, Timmons BH, *Enhanced adaptive behavioural response in agoraphobic patients pretreated with breathing retraining*, Lancet 1984 Sep 22; 2(8404): 665-669.

21 patients with agoraphobia (sensations of fear, panic, and terror), first, were administered the HVPT (with breathing frequency 60 times per minute, for 3 min maximum, breathing through both mouth and nose as vigorously as possible). Over 95% of patients recognised their symptoms, but which were not as severe as usual. Two thirds of the patients were unable to complete the 3-min HVPT due to dizziness and distress. In contrast, only 4% of the normal subjects were unable to hyperventilate for 3 minutes. The authors emphasised the importance of the HVPT and the subsequent helpful "*shock of recognition*".

After positive recognition of their symptoms, 12 patients were given a 10-week course of the therapy (1-2

laboratory sessions per week, 2h for each session). The laboratory sessions included instructions about diaphragmatic respiration. Patients, while lying down, were told to maintain a breathing pattern of 8-10 breaths per minute.

Techniques for coping with panic attacks were suggested - "*the use of a paper bag for rebreathing or, in public places, breathing into hands tightly cupped over nose and mouth...The establishment of a normal breathing pattern requires regular and persistent practice over several months...Any relaxation technique that entails the use of deep breathing is contraindicated, since it will probably exacerbate hypocapnia* [low aCO_2]" (p.668, Bonn et al, 1984).

The results were recorded at the end of the therapy, and for 1-month and 6-month follow-ups. The improvements were similar for all periods. Breathing rate dropped from 28 to 15-19 breaths per minute. The weekly number of panic attacks was reduced from 4-5 to 0.2. Other somatic characteristics were also significantly better.

4.5 Institute of Stress Research, Netherlands

Grossman P, de Swart JCG, Defares PB, *A controlled study of a breathing therapy for the treatment of hyperventilation syndrome*, J Psychosom Res 1985; 29 (1): 49-58.

The researchers employed the HVPT for 3 minutes with the goal to be reached of 2.5% $etCO_2$ (end-tidal CO_2). Then the physical and psychological symptoms, experienced by the patients, were discussed.

The treatment was 10-weeks long and included 7 laboratory sessions (each for about 30 min) and daily home assignments. A ventilatory training device was individually adjusted to generate a periodic pattern for breathing with slightly less frequency than the initial one. That was achieved by the use of auditory stimuli for inspiration, expiration, and the pause. Verbal emphasis on abdominal breathing was provided. Laboratory sessions also included the CO_2 analysis of the expired air.

As a result of the therapy, the average breathing rate of the experimental group (25 subjects) decreased from 17 to about 11 breaths per minute, and resting $etCO_2$ increased from 4.2 to 4.7%, while scores in all somatic complaints significantly improved.

Although the subjects of this study were free from serious physical ailments, the researchers wrote, "*In a broader sense, the findings indicate that by means of direct voluntary training of respiration, it may be possible to effect long-term alterations in ventilatory control mechanisms. This may have implications for the behavioural treatment of other respiratory disorders (e.g., asthma, sleep apnea and emphysema). Since alterations in ventilatory parameters are known to induce substantial changes in a range of other physiological systems (e.g., cardiovascular and CNS; see [16]), long-term modification of ventilatory control, via breathing therapy may also be useful in treating specific disorders of these other systems*" (p. 58, Grossman et al, 1985).

4.6 Department of Psychiatry, University of Oxford, Warneford Hospital, UK

Clark DM, Salkovskis PM, Chalkley AJ, *Respiratory control as a treatment for panic attack*, J Behav Ther Exp Psychiatry 1985 Mar; 16(1): 23-30.

Salkovskis PM, Jones DR, Clark DM, *Respiratory control in the treatment of panic attacks: replication and extension with concurrent measurement of behaviour and pCO_2*, Br J Psychiatry 1986 May; 148: 526-32.

"Eighteen patients who experienced frequent panic attacks were given a treatment derived from the literature on hyperventilation and anxiety. The treatment consisted of (i) brief, voluntary hyperventilation. This was intended to induce a mild panic attack; (ii) explanation of the effects of over-breathing and reattribution of the cause of a patient's attacks to hyperventilation; (iii) training in a respiratory control technique. Substantial reductions in panic attack frequency and in self-reported fear during a behaviour test were obtained after 2 weeks' treatment and these reductions occurred in the absence of exposure to feared situations. Further reductions in panic attack frequency were evident at 6-month and 2-year follow-up though interpretation of these results is complicated by the addition of exposure and other psychological treatments" (abstract, Clark et al, 1985).

In this study all patients were successfully treated *"...Large and rapid reductions in panic attack frequency and questionnaire report of fear were observed. Patients' resting pCO_2 was significantly lower than controls and rose to normal levels during treatment"* (abstract, Salkovskis, 1986).

The high success rate of this therapy could be partly explained by the absence of patients with an organic or metabolic illness and absence of the elderly among the subjects (the average mean age was about 30 years, from 26 to

44). The patients used a recorded tape, set at 12 breaths per minute. This tape was to be practiced at home daily. Laboratory sessions were organised about once per week involving ongoing respiratory training and homework instructions.

Statistical analysis revealed that patients with particularly low resting $etCO_2$ were more likely to recognize the symptoms of over-breathing as being similar to their panic attacks.

4.7 Department of Psychiatry, University of Utrecht, Netherlands

Ruiter de C, Ryken H, Garssen B, Kraaimaat F, *Breathing retraining, exposure and a combination of both, in the treatment of panic disorder with agoraphobia*, Behav Res Ther 1989; 27(6): 647-655.

The study was completed with 40 patients diagnosed with agoraphobia. All participants were selected on the basis of their ability to recognize their symptoms during the HVPT. The patients were informed as to how hyperventilation and catastrophic thinking and worrying could cause panic attacks.

The treatment consisted of 8 individual sessions lasting about 60 min each, as well as instructions how to practice relaxation and slow breathing in daily situations. The main goal of the therapy was to gradually reduce the respiration rate. For that purpose a pacing tape was used. Each time it was used, the breathing frequency proved to be slightly lower than the previous one.

Slow diaphragmatic breathing was *"encouraged by suggesting patients put one hand on their abdomen, and breathing 'against the hand'"* (p. 649, Ruiter et al, 1989). *"Treatments that included breathing retraining techniques seemed to result in a decrease in respiratory rate, but not in an increase in alveolar pCO_2"* (p. 652, Ruiter et al, 1989), since $etCO_2$ slightly decreased during the treatment. Meanwhile, *"the present study found breathing retraining plus cognitive restructuring ineffective in reducing panic"*(p. 654, Ruiter et al, 1989).

Professor Ronald Ley, State University of New York, wrote comments on this study, concluding

"Efforts to reduce ventilation through exclusive attention to a reduction in respiratory frequency may not only be unsuccessful in reducing ventilation, but may, as in the study in question, produce a paradoxical increase in ventilation, an effect opposite to the express purpose of the breathing retraining. The results here indicate that pCO_2 monitoring should be an integral part of breathing retraining process" (p. 304, Ley, 1991).

4.8 Cornell University Medical College, New York, USA

Fensterheim H, Wiegand B, *Group treatment of the hyperventilation syndrome*, Int J Group Psychother 1991 Jul; 41(3): 399-403.

"Hyperventilation (hv) is increasingly recognized as being significant in a number of psychological and medical conditions. The core of treatment for hv is breathing retraining, usually on an individual basis. This article describes a group therapy for breathing retraining for patients with hv-induced panic reactions. An analysis of group process suggests that such treatment is helpful in ways impossible for individual retraining and that further exploration of this modality is warranted" (abstract, Fensterheim & Wiegand, 1991)

The novelty of this study was the use of group therapy during the breathing retraining sessions. The authors found that their patients had a warm supportive acceptance of each other and of the group itself. That "*facilitated cooperative performance of their assignments and taking the risks in life situations that are so necessary for progress in this area*" (p. 401, Fenshterheim & Wiegand, 1991).

Moreover, it was found, that group processes helped to alleviate the disturbed reaction to symptoms and to accelerate the learning of correct breathing patterns. In particular, the authors emphasized that group treatment sessions, as an addition to individual sessions, should be especially helpful to those patients who had the most difficulty in learning a normal breathing pattern.

4.9 California School of Professional Psychology, San Diego, USA

DeGuire S, Gevirtz R, Kawahara Y, Maguire W, *Hyperventilation syndrome and the assessment of treatment for functional cardiac symptoms*, Am J Cardiol 1992 Sep 1; 70(6): 673-677.

DeGuire S, Gevirtz R, Hawkinson D, Dixon K, *Breathing retraining: a three-year follow-up study of treatment for hyperventilation syndrome and associated functional cardiac symptoms*, Biofeedback Self Regul 1996 Jun; 21(2): 191-198.

"*Three methods of breathing retraining (guided breathing retraining, guided breathing retraining with physiologic monitoring of thoracic and abdominal movement plus peripheral temperature, and guided breathing retraining with physiologic monitoring of thoracic and abdominal movement, peripheral temperature and end-tidal carbon dioxide) were compared with a no-treatment control group to determine the effectiveness of breathing retraining on modifying respiratory physiology and reducing functional cardiac symptoms in subjects with signs associated with hyperventilation syndrome. Of 41 subjects studied, 16 were diagnosed with mitral valve prolapse. Results demonstrated that all 3 methods of breathing retraining were equally effective in modifying respiratory physiology and reducing the frequency of functional cardiac symptoms. Results determined that respiratory rate and subject's perception that training had generalized were the best predictors of treatment success. Furthermore, it was found that subjects with mitral valve prolapse responded as well to treatment as did those without prolapse*" (abstract, DeGuire et al, 1992).

Each person had 6 individual breathing retraining sessions over a 3-week period. During the first laboratory session, all of them were informed about respiratory physiology and the relation between hyperventilation and functional cardiac symptoms. The patients were shown diaphragmatic breathing and their practice was corrected by the experimenter. Although many patients reported discomfort with slow diaphragmatic breathing, they were encouraged to tolerate the discomfort and were reassured that their symptoms would disappear with time. The patients were carefully observed to ensure that they did not increase their tidal volumes. Later sessions were focused on correcting errors in diaphragmatic breathing and setting a slow-paced respiratory rate of less than 14 breaths per min.

Average etCO_2 in all 3 groups increased from about 35 to 40 mm Hg. It was of special interest to the authors, that some functional cardiac problems could be treated with breathing retraining by the normalisation of etCO_2.

Four years later, the researchers found that the positive effects of breathing retraining were still present in the tested subjects:

"*This study was designed to evaluate the long-term effects of paced diaphragmatic breathing on subjects who reported functional cardiac symptoms and who also demonstrated associated signs of hyperventilation syndrome. Subjects were a representative sample composed of 10 out of the original 41 subjects who had participated three years previously in a study designed to evaluate the short-term effects of breathing retraining on functional cardiac symptoms and respiratory parameters (respiratory rate and end-tidal carbon dioxide). The results of this follow-up study indicate that breathing retraining had lasting effects on both respiratory parameters measured. Subjects evidenced significantly higher end-tidal carbon dioxide levels and lower respiratory rates when compared to pretreatment levels measured three years earlier. Subjects also continued to report a decrease in the frequency of functional cardiac symptoms when compared to pretreatment levels. We conclude that breathing retraining has lasting effects on respiratory physiology and is highly correlated with a reduction in reported functional cardiac symptoms*" (abstract, DeGuire et al, 1996).

Medical professionals often interpret abnormal EKGs to be the result of certain organic conditions or damage to the heart. However, this and other experiments clearly pointed out that EKG results depend on the breathing pattern. When breathing is normal, so are the results. Over-breathing often produces certain abnormal EKG changes (see Chapter 1), in accordance with an individual's predisposition.

4.10 Lothian Area Respiratory Function Service, City Hospital, Edinburgh, UK

Tweeddale PM, Rowbottom I, McHardy GJ, *Breathing retraining: effect on anxiety and depression scores in behavioural breathlessness*, J Psychosom Res 1994 Jan; 38(1): 11-21.

22 patients with behavioural breathlessness were selected for breathing retraining. The HVPT involved 3-minute periods of rest, voluntary hyperventilation, and recovery. During an initial assessment special attention was paid to observing the existing breathing patterns of the patients (upper chest or abdominal breathing; nasal or oral breathing; presence of sighing, gulping, or yawning; and inappropriate patterns during speech), identification of hyperventilation-triggering situations and clinical details.

During their 7 weekly visits to the laboratory, the patients were taught to develop awareness of their breathing patterns, to practice individual breathing exercises, to implement breathing control during speech, to develop control of hyperventilation-triggering situations, and finally, to achieve an effortless breathing pattern.

As a result of such intervention, "*both groups* [with behavioural breathlessness and chronic fatigue] *showed improvements in breathing patterns, end tidal CO_2 levels and scores for HV-related symptoms which were sustained*"

(abstract, Tweeddale, 1994). Moderate increase in BHT was noticed for the patients with lowest initial BHT values.

The authors also made an important practical conclusion. "*In any patient, if there is either resistance to the idea of breathing being related to symptoms, or lack of commitment to regular performance of breathing exercises or unwillingness to check out breathing patterns and exercise control during daily activities, breathing retraining is unlikely to be of benefit*" (p. 20, Tweeddale, 1994).

4.11 Service de Psychosomatique, Hopital du Sacre-Caeur de Montreal, Quebec, Canada

Monday J, Gautrin D, Cartier A, *Chronic hyperventilation syndrome. The role of respiratory re-training* [in French], Rev Mal Respir 1995; 12(3): 291-298.

"This study compares three non-pharmacological approaches to the chronic hyperventilation syndrome (CHS). Eighteen subjects were evaluated at the start of the study then one and 6 months after having received in a random fashion one of the following treatments: group I (teaching approach of one hour on the respiratory physiology of the CHS and on breathing techniques; n = 5); group II (same approach as in group I with breathing retraining of 8 sessions; n = 8); group III (same as group II with the addition of a modified Jacobson's progressive relaxation; n = 5). Whereas all three groups had a similar symptomatic score at the beginning of the study (although subjects of group III had in general higher scores and were symptomatic for a longer period), our results show that all subjects improved after 4 weeks, those in group II showing the greatest improvement (p < 0.05). This confirms the relevance of applied and repeated pedagogy in approaching subjects with the CHS" (abstract, Monday et al, 1995).

4.12 Laboratory of Pneumology, U. Z. Gasthuisberg, Katholieke Universiteit Leuven, Belgium

Han JN, Stegen K, De Valck C, Clement J, Van de Woestijne KP, *Influence of breathing therapy on complaints, anxiety and breathing pattern in patients with hyperventilation syndrome and anxiety disorders*, J Psychosom Res 1996 Nov; 41(5): 481-493.

"The effect of breathing therapy was evaluated in patients with hyperventilation syndrome (HVS). The diagnosis of HVS was based on the presence of several suggestive complaints occurring in the context of stress, and reproduced by voluntary hyperventilation. Organic diseases as a cause of the symptoms were excluded. Most of these patients met the criteria for an anxiety disorder. The therapy was conducted in the following sequence: (1) brief, voluntary hyperventilation to reproduce the complaints in daily life: (2) reattribution of the cause of the symptoms to hyperventilation: (3) explaining the rationale of therapy-reduction of hyperventilation by acquiring an abdominal breathing pattern, with slowing down of expiration: and (4) breathing retraining for 2 to 3 months by a physiotherapist. After breathing therapy, the sum scores of the Nijmegen Questionnaire were markedly reduced. Improvements were registered in 10 of the 16 complaints of the questionnaire. The level of anxiety evaluated by means of the State-Trait Anxiety Inventory (STAI) decreased slightly. The breathing pattern was modified significantly after breathing retraining. Mean values of inspiration and expiration time and tidal volume increased, but end-tidal CO_2 concentration ($FETCO_2$) was not significantly modified except in the group of younger women (< or = 28 years). A canonical correlation analysis relating the changes of the various complaints to the modifications of breathing variables showed that the improvement of the complaints was correlated mainly with the slowing down of breathing frequency. The favorable influence of breathing retraining on complaints thus appeared to be a consequence of its influence primarily on breathing frequency, rather than on $FETCO_2$" (abstract, Han et al, 1996).

This study involved 92 patients diagnosed with hyperventilation syndrome. However, the work of the physiotherapists was not targeted to decrease ventilation of the subjects and to raise their CO_2 values. The article states, that "*the therapy consisted of: (1) a reattribution of the threatening symptoms to faulty breathing habits; and (2) training of slow breathing and learning to use the diaphragm more (abdominal breathing) and less the upper part of the thorax*" (p. 482, Han et al, 1996).

Results of the therapy revealed that the patients acquired a slow deep breathing pattern. Their average tidal volume rose from abnormal 740 ml/breath (the norm is about 500-600 ml/breath) to 880 ml/breath, while $etCO_2$ was almost unchanged (4.76% before and 4.84% after the treatment). Normal $etCO_2$, according to most medical sources, is about 40 mm Hg or 5.25% CO_2 at sea level.

32 patients improved markedly, according to their questionnaires, and were feeling well. Meanwhile, several

symptoms related to hyperventilation did not improve at all after the treatment. These included "*confusion or feeling of losing contact with surroundings, feeling of faster and deeper breathing, bloated abdomen, stiff fingers or arms, tightness around the mouth, and cold hands or feet*" (p. 485, Han et al, 1996). That should not be a surprise, since the main problem (hyperventilation or low aCO_2) was not identified and resolved in this study.

4.13 New Zealand Guidelines Group

The New Zealand Guidelines Group is a large group of medical professionals, including numerous professors and physicians (for more details visit their website at http://www.nzgg.org.nz). The Chairman of the group is Norman Sharpe, Head of the School of Medicine in the University of Auckland.

In their "*Guidelines for Assessing and Treating Anxiety Disorders, Appendix 6: Slow Breathing Exercise*", the Group states,

"You will remember that when you get anxious your rate of breathing increases. This over-breathing is often referred to as 'hyperventilation'. When you over-breathe you breathe out too much carbon dioxide which leads to a decrease in the level of carbon dioxide in the blood. The decreased level of carbon dioxide causes or worsens a number of symptoms such as breathlessness or light- headedness. You may experience these symptoms if you have panic attacks.

To get rid of these symptoms, the level of carbon dioxide in the blood must be increased and steadied. One way of achieving increased levels of carbon dioxide is to breathe into a paper bag. A large portion of the air you breathe out is carbon dioxide, therefore, by rebreathing your old air you are taking higher amounts of carbon dioxide into your lungs.

Although breathing into a paper bag is simple and effective, it may not always be convenient or socially appropriate to pull out a paper bag in public! Additionally, although breathing into a paper bag is effective during a panic attack, this method cannot prevent hyperventilation in the future. An alternative method which is less obvious to other people and more effective in the long run is the slow breathing exercise. This method will help you to control your hyperventilation. Also, by learning slow and regular breathing habits you will help to prevent future episodes of hyperventilation and other symptoms of panic.

The following exercise is to be practised four times every day for at least five minutes each time, AND at the first signs of panic or anxiety.

Combining slow breathing with relaxation is particularly helpful.

SLOW BREATHING EXERCISE (TO BE PRACTISED REGULARLY AND AT THE FIRST SIGNS OF ANXIETY OR PANIC).

If you recognise the first symptoms of over-breathing, STOP what you are doing and sit down or lean against something. If you are driving, pull over and park in a safe place.

1. Hold your breath and count to 5 (do not take a deep breath).

2. When you get to 5, breathe out and say the word 'relax' to yourself in a calm, soothing manner.

3. Breathe in and out slowly through your nose in a six second cycle. Breathe in for three seconds and out for three seconds. This will produce a breathing rate of 10 breaths per minute. Say the word 'relax' to yourself every time you breathe out.

4. At the end of each minute (after 10 breaths) hold your breath again for 5 seconds and then continue breathing using the six second cycle.

5. Continue breathing in this way until all the symptoms of over-breathing have gone. It is important for you to practise this exercise so that it becomes easy to use any time you feel anxious. (Treatment Protocol Project 1997)

Among considered methods of breathing retraining, this is the only Western study that involves breath holding as a method of CO_2 accumulation.

4.14 Stanford University, Palo Alto, USA

Meuret AE, Wilhelm FH, Roth WT, *Respiratory biofeedback-assisted therapy in panic disorder*, Behav Modif 2001 Sep; 25(4): 584-605.

The value of this small study (only 4 patients) is that after the treatment all patients were "*below the clinical threshold for the diagnosis of panic disorder*" (p. 596, Meuret et al, 2001).

The patients performed 5 mild HVPT, each 3 min long. These tests were followed by 8 min quiet sitting

period. The therapy had 5 individual sessions, each about 80 min long, over the course of 4 weeks. The major components of the therapy were:
- education about hyperventilation and its central role in the mechanism of the panic attack;
- teaching techniques to control respiration;
- directing attention to dangerous breathing patterns;
- instruction in home breathing exercises.

Home assignments were performed twice per day, each about 20 min long.

The abstract of the publication states,

"The authors describe a new methodologically improved behavioral treatment for panic patients using respiratory biofeedback from a handheld capnometry device. The treatment rationale is based on the assumption that sustained hypocapnia resulting from hyperventilation is a key mechanism in the production and maintenance of panic. The brief 4-week biofeedback therapy is aimed at voluntarily increasing self-monitored end-tidal partial pressure of carbon dioxide (PCO_2) and reducing respiratory rate and instability through breathing exercises in patients' environment. Preliminary results from 4 patients indicate that the therapy was successful in reducing panic symptoms and other psychological characteristics associated with panic disorder. Physiological data obtained from home training, 24-hour ambulatory monitoring pretherapy and posttherapy, and laboratory assessment at follow-up indicate that patients started out with low resting PCO_2 levels, increased those levels during therapy, and maintained those levels at posttherapy and/or follow-up. Partial dissociation between PCO_2 and respiratory rate questions whether respiratory rate should be the main focus of breathing training in panic disorder" (abstract, Meuret et al, 2001).

4.15 Common features of Western methods of breathing retraining

• **Usefulness or necessity of the HVPT (hyperventilation provocation test)**

Most authors used the over-breathing test and indicated its necessity for the successful outcome of their therapy. Indeed, since many patients (usually over 90%) could recognize their symptoms during the test, these patients realised the cause of their problems (excessive breathing). It is normal to expect that this important practical discovery should immediately bring relief to many patients since anxiety (fear of the unknown) was greatly reduced. In addition, this insight created a better relationship with their doctors that was based on trust, as well as it also sparked a strong interest and enthusiasm of the patients that they would soon be able to play an important role in solving their own problems.

When this test was not done, many patients would continue to have reasonable doubts about the value of the whole therapy and the doctors' ability to help. The crucial difference between these two situations (with and without the test) is the positive change in attitude of the patients towards the suggested approach.

• **Awareness about normal and abnormal breathing patterns**

The patient must learn the main characteristics of normal and abnormal breathing patterns in order to recognise them in every day life.

First, it was explained to the patient that behaviours leading to hyperventilation cause a reduction in carbon dioxide stores. These behaviours included coughing, sneezing, sighing, sniffing, and chest inflation. In order to become healthy, all of those abnormal behaviours must be stopped or prevented.

Second, it was important for the patient to know the features of thoracic and diaphragmatic breathing. Diaphragmatic breathing could be characterised by the following adjectives: smooth, quiet, slow, and regular. Thoracic (chest) breathing, on the other hand, is often uneven, noisy, fast, and irregular.

• **Relaxation**

Since tension is a normal response to stress and hyperventilation, one can conclude that relaxation, due to a feedback mechanism, reduces minute ventilation, decreases pulse rate, and raises carbon dioxide concentrations. Therefore, relaxation is a valuable therapeutic tool in reducing hyperventilation and in improving one's health as numerous studies of various medical conditions have validated.

Moreover, relaxation can be useful in eliminating the negative consequences of stress, hyperventilation, and their subsequent abnormal physiological changes. In particular, the reduction of tension in the large skeletal muscles, especially of the chest-shoulders-neck-jaws region, results in more calmness and easier breathing.

• **Diaphragmatic breathing**

Many authors suggested to their patients that they use the instructions of physiotherapists in order to learn diaphragmatic breathing. Two previously discussed features, which are the awareness of normal and abnormal

breathing patterns, as well as relaxation, could be considered both the theoretical preparation and the first practical steps in acquiring a normal diaphragmatic breathing pattern.

• **Durations of daily sessions and the therapy**

The typical duration of recommended daily sessions was from 20 min to 1-2 hours, in addition to a daily regime of breathing control. Are there any physiological grounds for such programs?

The breathing centre has two groups of specialized cells (chemoreceptors) which monitor carbon dioxide and oxygen concentrations (Chapter 2). One group is located near the main arteries close to the heart, and the other in the medulla of the brain.

Those cells, which are located near the heart (the peripheral chemoreceptors), can detect changes in carbon dioxide levels in arterial blood in less than a second. Indeed, these cells are in contact with the arterial blood leaving the heart. Therefore, their adaptation and training starts and finishes almost immediately with a voluntary reduction in ventilation. Thus, they are being retrained while simultaneously the person breathes less. (The adaptation of these cells to acute carbon dioxide changes during breath holding causes the “training effect” discussed in chapter 2).

The second group of cells located in the brain (the central chemoreceptors) is bathed in the CSF (cerebro-spinal fluid). Their trainability depends on carbon dioxide pressure and the pH of CSF. Hence, it is important to know how CO_2 in the arterial blood influences CO_2 and pH concentrations in the CSF of the brain, since these fluids are separated by a blood-brain barrier. It is a well-established fact that this barrier allows only certain substances to cross it and only at particular rates. What would be the time required for an increase in arterial CO_2 to penetrate this barrier in order to influence the cells located in the main part of the breathing centre? There were several studies by physiologists and neurologists devoted to this subject.

Measurements of carbon dioxide tension on the cerebral cortex in anaesthetized cats revealed that this parameter reaches a plateau in 5-10 min after aCO_2 in the blood experience a 5-10% absolute increase (Seisjo, 1961). In another series of experiments with anaesthetized rats, brain tissue CO_2 had an exponential increase, with half-time 6-7 min, as a response to 1 mm Hg CO_2 pressure increase in inspired air (Seisjo, 1963). Other physiologists recorded 12 min in maximum specific activity of CO_2 isotopes in the brain after their appearance in the blood of rabbits (Coxon & Swanson, 1965). Finally, a more recent investigation found that "*changes in pH and pCO_2 with hyperventilation and hypoventilation occurred rapidly in both arterial blood and CSF. Steady state values were reached within 15 min for hypoventilation and 30 min for hyperventilation... These results are consistent with previous research*" (abstract, Andrew et al, 1994).

Therefore, it would be reasonable to assume that about 10-15 min is required in order to achieve maximum influence on the central chemoreceptors of the breathing centre.

The optimum duration of this influence (how long it will continue) would probably depend on the amplitude of the change and current trainability of the breathing centre. Too long time of the session would lead to fatigue and other unpleasant symptoms.

The recommended length of supervised therapies was from 1 to 3 months, while unsupervised sessions at home would take more time. Meanwhile, most patients experienced decisive improvement in their health during the supervised part.

• **Respiratory rate**

Increased initial breathing frequencies of patients participating in these therapies were commonly noticed. Most authors suggested to their patients to practice slower breathing patterns in order to derive the assumed benefits of reduced ventilation and increased aCO_2. However, from section 2.2 we know that, when people are asked to voluntarily decrease their respiratory rate, while having naturally regulated breathing, a slow deep breathing pattern is the likely outcome. However, we also know that most hyperventilators already have deep breathing.

Is there any likelihood that some of them may start to breathe even deeper after these exercises as a result of such breathing practice? Probably, for just such a possibility prompted Bonn along with his colleagues to suggest that "*any relaxation technique that entails the use of deep breathing is contraindicated, since it will probably exacerbate hypocapnia* [low aCO_2]" (p.668, Bonn et al, 1984).

Furthermore, researchers from Stanford University (Meurel et al, 2001) directly questioned (see above) whether the respiratory rate should be the main focus of breathing training. Professor Ley expressed similar ideas (quoted above).

Indeed, the main goal of all these therapies was to normalise (increase) carbon dioxide values by diminishing ventilation, or in other words, by breathing less while at the same time experiencing relaxation. That, by itself indeed, is not an easy task, as it requires the persistent use of patient's concentration and will power. In addition, monitoring

one's breathing frequency may only result in deeper breathing, causing further tension and more over-breathing. All of this calls for learning to create a delicate balance between both consistent and relaxed effort.

There also is one technical aspect involving the use of capnometers. As the reader may remember these devices measure end-tidal CO_2 in the expired air. In normal conditions this value is close to the carbon dioxide pressure in the lungs and arterial blood.

However, many studies revealed that, in cases of deep breathing or prolonged exhalations, the difference between end-tidal and arterial CO_2 gets larger when compaed to normal conditions (Jones et al, 1979; Robbins et al, 1990). Jones and colleagues, for example, found that in different exercise conditions "*$PETCO_2$-$PaCO_2$ varied between -2.5 and +9.1 Torr* [1 Torr = 1 mm Hg], *was inversely related to the frequency of breathing (r = 0.475), and directly related to tidal volume*" (abstract, Jones et al, 1979). Hence, the slower the breathing, the larger the difference; and the deeper the breathing, the larger the difference.

As a result, in cases of slow deep breathing, the reading of capnometers significantly over-estimates arterial CO_2 tension. That creates the illusion that carbon dioxide in the blood and in the whole organism is increasing, whereas real values may in fact remain unchanged or even decrease.

• **Drug-free nature of therapies**

Only the earliest study (Kerr et al, 1937), conducted before World War II, relied on the use of chemical substances in order to normalize ventilation and aCO_2. Other therapies were drug-free and some authors claimed that drugs were useless when working towards breathing normalization. This commitiment to the use drug-free procedures is what makes these breathing methods different from the traditional approaches practiced and advocated by most medical authorities and doctors.

There are many known problems with drugs. In the next section, one of the crucial differences between a traditional drug-based approach and any drug-free therapy is discussed.

• **Acquisition of control over own health by the patients**

Let us consider two possible scenarios in which patients seek help for their disease: in one situation, the doctor administers a drug, while in the other, a natural approach is recommended, which requires a change in personal habits, priorities, and routines.

Modern medicine usually cannot give a clear explanation as to what causes patient's problems, and therefore, most suggested solutions are useless. The well-meaning doctor often will rely on drugs as a solution. The problems with drugs are numerous and well-documented. A few of those drugs have survived several decades and are still prescribed. A typical scenario is as follows:

The patient arrives at the doctor's office with his complaints. The doctor listens, asks question, thinks about the problem, conducts tests, make a diagnosis, and prescribes a chemical substance, which is foreign to the human body. The patient innocently accepts the doctor's approach because he has little understanding of the nature of the problem. What he does know is that his disease is said to result from a combination of stress, genetics, a deteriorating environment and other factors, all of which are typically outside of his control. The doctor rarely suggests a natural approach (diet, exercise, relaxation, etc.). Instead, the latest miracle drug, recently approved by medical authorities, becomes the key to the treatment.

After taking the drug for months or years (if he survives), the patient may realise that it does not work or that it even causes his health to deteriorate further. By that time, this "new" wonder drug may be withdrawn (as many are), or banned due to dangerous side-effects. Nevertheless, the well-meaning doctor is still dispensing the 'miracle' pill. The role of the pill is to fuel the imagination and hopes of the excited, hyperventilating patient.

Retraining one's breathing is, in contrast, a natural therapy based on ideas, some of which are expressed in Chapter 1. If this natural approach is accepted by the patient, he then realizes that his breathing was and is the cause of his problems. It is possible that some patients may still dream of a miracle cure.

Consequently, breathing retraining, as compared to other methods of treatment, provides the student with the ability to impact the development of his disease with practical coping strategies, which when combined with the patient's self-discipline, persistence, and determination, can indeed provide a way to recover.

• **High success rates**

Most cited studies report a high rate of success, indicating that their patients were soon enjoying easier breathing. Is it always a normal outcome? How long does it take practically to restore normal breathing?

As mentioned above, the patients chosen for these experiments did not have any organic damage according to preliminary screening investigations. Therefore, their progress often took only a few weeks or months. However, longer time was required for people who had certain damaged or abnormal organs and tissues.

Q&A section for Chapter 4

Q: Why are these therapies used mainly for patients diagnosed with panic attacks and hyperventilation syndrome when most other sick people also over-breathe?

A: Historically, capnometers (devices to measure CO_2 in exhaled air) became popular among psychologists. In addition, there is an almost direct relationship between hyperventilation and panic attacks. It takes longer time for the development of an asthma attack, cholesterol deposits, tumours and certain other pathological tissues and phenomena.

With regards to other patients, it is indeed reasonable to ask medical authorities, "*Why not treat modern patients with different chronic degenerative conditions from their hyperventilation and then see what happens with their symptoms and complaints, when their breathing is normalized in accordance with medical standards?*"

Q: Is pursed-lip breathing helpful?

A: Pursed-lip breathing involves exhalation against partially closed (pursed) lips, as if one is getting ready to whistle. This technique is supposed to prevent bronchiolar collapse and includes small inhaled breaths and a long pursed-lip exhalation. Its use is advised during exercise and panic attacks. One study of patients with cystic fibrosis managed to demonstrate improved FEV (forced expiratory flow) and FVC (forced vital capacity) (Delk, 1994) without any improvements in their quality of life. Patients with COPD (chronic obstructive pulmonary disease) after pursed-lip breathing sessions increased their exercise performance, while making their blood gases at maximum speed worse (Casciari, 1981).

In my view, pursed-lip breathing, pranayma and some other slow deep breathing exercises can be very beneficial for certain groups of people (e.g., with heart disease and sleep apnea) during certain stages of breahing retraining. However, it should be kept in mind, that restoration of normal breathing reuires a program of actions that addresses all the essential factors causing chronic hyperventilation, including strictly nasal breathing, prevention of sleeping on the back, etc.

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Chapter 5. History and advance of the Buteyko breathing method

Introduction

This chapter, first, gives a short history and information about the theoretical findings and practical work of Doctor Buteyko and his pupils in Russia and abroad. Later, some basic features and trials of the Buteyko breathing method are considered.

5.1 Some historical facts about the origins of the method

Doctor Buteyko received his medical degree from the First Medical Institute in Moscow, where he studied from 1946 to 1952. During this period he had a medical practice attending and dealing with severely sick and critically ill patients. A series of events helped him to realize the connection between the respiration and health of patients with hypertension, angina pectoris, asthma, and some other serious diseases. He noticed that with approaching death, patients' respiration got heavier. By visual observation of patients' breathing in the hospital, he could predict how many days or hours of life were left. Later he discovered that deliberate acute hyperventilation (which we explored as the HVPT or hyperventilation provocation test in previous chapters) quickly worsened the health of patients, while breathing less caused elimination of their symptoms. Buteyko also confirmed these findings in his own problem, hypertension. He then decided to devote his life to studying respiration, in general; and CO_2 properties, in particular.

After graduation with Honours, in 1952, he joined the Department of Clinical Therapy of the same institute, working as the manager of the Laboratory of Functional Diagnostics in Moscow. Among his concerns were lack of qualified personnel, inadequate equipment, and financial problems. He then had more time to study western publication about breathing. During these years the Soviet state was developing the unique program of outer space exploration for the first space missions. It was of exceptional importance to know and study effects of air parameters (air pressure and its composition) on human health. Hence, Soviet officials were looking for bright young scientists who could lead such projects in physiology and medicine. At the end of 1950's he was chosen to head such a project in Novosibirsk. Due to importance attached to the project, his laboratory was provided with the best available equipment and best qualified support.

Obviously, in this research, as, for example, in any similar NASA research, its people and facilities were heavily guarded by Soviet state officials, including KGB agents. The aims of this research included:
- finding optimum air parameters for human functioning during space missions depending on the stage of the flight and initial parameters of astronauts;
- breathing of healthy and sick people and interactions between various diseases and respiration;
- effects of various environmental factors (sleep, sleeping postures, exercise, posture, meals, diets, daily activities, temperature, thermoregulation, emotions, etc.) on breathing and health.

Thus, in 1960 Buteyko became the manager of another Laboratory of Functional Diagnostics organised at the Institute of Experimental Biology and Medicine in Novosibirsk. Buteyko created in his laboratory a unique diagnostic complex, which included several physiological devices to measure 40 important health parameters in real time (or with each breath). According to Buteyko and Dyomin, "*One such investigation, lasting about 1 hour, produces about 2,000 recordings of 40 main parameters of respiratory and cardiovascular processes, resulting in about 100,000 numbers…*" (Buteyko & Dyomin, 1963). These parameters included pulse, EKG, blood pressure, tidal volume, respiratory rate, minute ventilation, arterial and venous blood gases and chemical analysis of the expired air. The complex produced many thousands of measurements per hour, analysed by a computer. The unique features of this complex were described in the Soviet magazine "*Inventor and Efficiency Expert*" (Inventor and Efficiency Expert, 1961; Buteyko 1961; Buteyko, 1962). Some characteristics and abilities of this machine were also reported in more than 20 scientific articles written by Buteyko with his colleagues and published in medical, physiological and diagnostic magazines and conference proceedings.

Research with the use of this complex was done from 1960 to 1968. That allowed Buteyko to receive information about physiology and respiration of the human organism in health and disease and relationships between respiration and different factors, including those described in chapters 2 and 3.

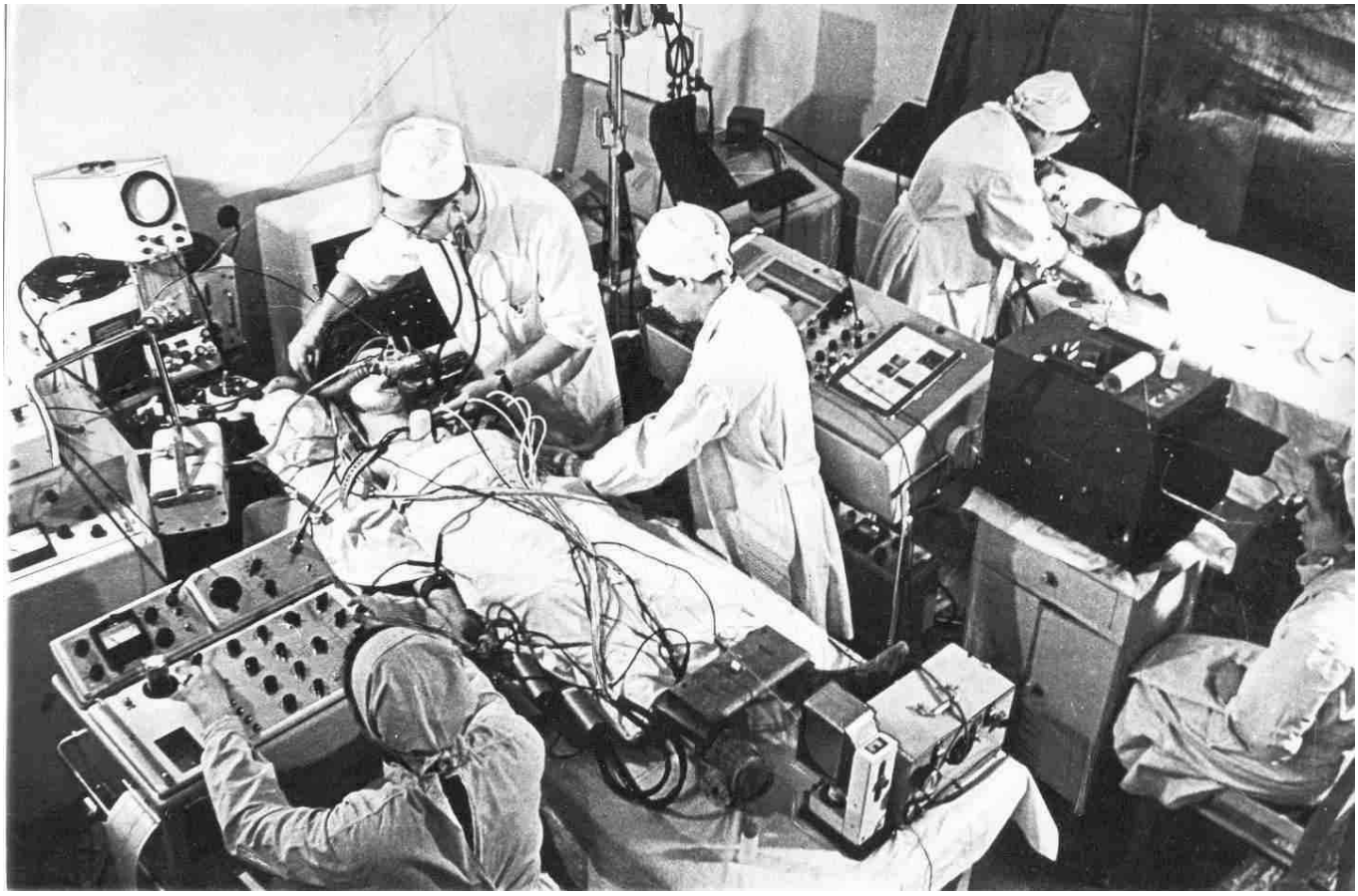


Photo 1. This is the unique diagnostic complex created in 1960s for USSR's first space missions in the Laboratory of Functional Diagnostic in Novosibirsk headed by Doctor Buteyko. The volunteers were to breathe air with varying composition. About 40 physiological parameters of the body were measured by the complex in real time 24/7, thousands of numbers every hour, literally with each breath of the person. The information was recorded and analyzed by a computer. Simultaneously Doctor Buteyko studied the interactions between breathing and diseases, breathing and life style factors and other breathing-related and CO_2-related challenges.

Not only did he brilliantly conduct and complete these studies funded and initiated by the USSR's Ministry of Aviation and Space Exploration, he also discovered and practically confirmed the fundamental role of breathing in development and treatment of various health conditions, including asthma, bronchitis and heart disease. Given the confidential nature of the project, it was normal that many results remained classified for years. (Compare Buteyko's situation with the situation of the famous Oxford Professor John Haldane, who was hired in 1920-1930's by British Navy to study air in submarines and whose name then disappeared from the world scientific community.) This, however, was not the case with Buteyko and his colleagues. They had dozens of publications in open Soviet literature (in Russian) about their research in 1960's. Moreover, in 1990's Buteyko and his numerous pupils were able to travel in Western countries and share the word about the Buteyko method and his discoveries.

5.2 Breathing and modern diseases

Let us now consider and analyse some ideas and hypothesis discovered by Doctor Buteyko. They can be found in his scientific publications and articles, and described during his interviews and public lectures.

Most patients (over 90 %) with a variety of modern chronic health problems ("diseases of civilization") hyperventilate and have chronically low aCO_2 (Buteyko & Shurgaya, 1965; Buteyko, 1969, 1972; Buteyko et al, 2005a, 2005b).

Acute overbreathing worsens their health state, while reduced breathing improves it (Buteyko & Shurgaya, 1965; Buteyko, 1969, 1972; Buteyko et al, 2005a, 2005b).

Chronic forms of the "diseases of civilizations" are impossible without dis-regulation of external respiration (Buteyko, 1969, 1972; Buteyko et al, 2005a, 2005b)

Development of these diseases is proportional to the degree of hyperventilation. The lower the aCO_2 and BHT, the worse the health of the patient (Buteyko, 1969, 1972; Buteyko et al, 2005a, 2005b).

The health state of a person can be defined by own BHT and alveolar CO_2 in accordance with the following table (Buteyko 1969, 1972, 1991; Buteyko et al, 2005a, 2005b)

	Super-health	Normal health	1-st stage of the disease	2-nd stage of the disease	3-rd stage of the disease	DEATH
BHT	> 60 s	40-60 s	20-40 s	10-20 s	<10 s	
$\%CO_2$	> 6.5 %	5.5-6.5 %	4.5-5.5 %	4.0-4.5 %	< 4.0 %	

Restoration of normal breathing is equal to elimination of these diseases and their symptoms (Buteyko, 1969, 1972; Buteyko et al, 2005a, 2005b)

Studies of Russian doctors practicing the Buteyko method, as well as Western publications, reveal that over 150 health conditions relate to heavy breathing, or are possible only in conditions of hyperventilation. These are less than 1% of all health pathologies (about 30,000) currently known to the medical science.

"I hear a whisper of indignation and joy: "So, are all diseases from deep breathing?" No, not all. Deep breathing causes one single disease: the disease of deep breathing. It causes a wide spectrum of pathologies. Why? It interrupts the metabolism of the substances – the foundation of life in all cells. These or other symptoms are manifested, and their strong expression is qualified as disease… "(Buteyko, 1977).

The theory suggested by Buteyko has some restrictions and cannot completely describe all physiological processes in the following cases: infectious diseases; severe psychological abnormalities; traumas; extreme states due to intoxication, poisoning, radiation, and reanimation; aging; unconscious states; childbirth; sex; and cancer (Buteyko et al, 2005a; Buteyko et al, 2005b). However, application of the Buteyko method allows natural regulation and normalization of these and many other physiological processes.

According to the book "Buteyko's cure" ("*Istcelenie po Buteyko*") compiled by the Moscow Buteyko clinic, "*1. Essence of the discovery.*

The essence of the Buteyko's discovery consists of the idea that during deep breathing too much CO_2 is removed from the body. This causes changes in the homeostasis, which are partially neutrloised by various compensatory mechanisms. The developing disbalance of homeostasis, accumulating during long periods of time (for a fetus due to breathing of the mother) leads to disruption of previous biosyntheses and other processes. The symptoms of the patients with carbon dioxide deficiency is worsened due to defensive reactions of the organism, which are expressed in disruptions of the respiratory and cardiovascular systems. However, it is necessary to say that should these defensive reactions, worsening the symptoms, be absent, the death of the person would take place much sooner.

2. The first proposition of the theory.

…Respiratory alcolosis is accompanied, as a ruler, by the state of oxygen deficiency,…which leads to appearance of incompletely oxidised products of metabolism, and, hence, to development of metabolis acidosis, which partially compensate pH fluctuations, caused by by the respiratory alcolosis, although it should be noted, that this sompensation is not complete. As a result of these processes, there are following changes in the organism:

1) the electrolytic composition of the extracellular fluid is changes;

2) pH of the extracellular fluid is changes;

3) since the cells are participants of the pocess of pH normalization, their pH changes;

4) since the kidneys try to normalise the PH and remove HCO3, the buffer abilities of blood plasma is reduced;

5) due to changes in the ratio HCO3 of the cell wo HCO3 of the plasma, the rate of diffusion of HCO3 from the cell to

plasma is changed;
6) [Haemoglobin cells can not release oxygen with the same efficiency.]
7) Synthesis of aminoacids, nitrous bases, lipids, and carbohydrates is disrupted.
The results of these changes are:

1. *disruption of synthesis of proteins, peptides, nucleoacids, lipids, and carbohydrates;*
2. *disruption in the work of enzymes, due to changes in pH, changes in the rate of carboxolozation;*
3. *changes in the energy production of the organism (decreased synthesis of ATP)…"* (p. 247, Buteyko's cure, 2000).

5.3 Development of specific health problems

Let us look at the suggested mechanisms of developments of particular health conditions.

• **Asthma**

Doctor Buteyko claimed that he knew since 1952 that chronic hyperventilation was the cause of asthma. This was his clinical observations. Normally, during later years it was merely a technical matter for him to establish the causes of asthma. Low aCO_2, due to chronic hyperventilation, resulted in chronic moderate bronchoconstriction and abnormal sensitivity of the immune system in relation to various substances or processes (Buteyko, 1964; Buteyko & Odintsova, 1968; (Buteyko, 1977). The final triggers, in order for asthma attack to take place, can be overeating, exercise, allergens, stress, overheating, and other factors which cause autoimmune response in airways, further intensification of respiration, more bronchoconctriction, production of extra mucus, chest tightness, wheezing and other negative effects.

Peter Kolb's web site (http://members.westnet.com.au/pkolb/buteyko.htm) contains large amounts of practical and theoretical information about asthma, hyperventilation, carbon dioxide, the Buteyko method and its development.

Practical evidence and Western clinical trials (see below) suggest that the Buteyko method is successful in dealing with asthma. Normalization of breathing and maintenance of normal CO_2 values for certain time leads in elimination of abnormal immune and allergic reactions in former asthmatics. As a result, they have clinical remission and usually can lead normal (in relation to work, diet, exercise, and exposure to allergens) lifestyle. For more details about asthma one may visit the website of the Buteyko Clinic in Moscow (www.buteykoclinic.ru).

While Russian practice and Western trials demonstrate the power of the Buteyko method for asthma, it is still unclear what the immunological mechanisms and biochemical causes of its success are. There are certain known CO_2 effects discussed above. These CO_2-related effects provide the foundation for appearance of asthma. Apart from that, there are other possible causes known to those who are teaching or learned the Buteyko method. What are these?

- Prevention of asthma attacks by reduced breathing and relaxation, instead of using reliever medication, provides patients with the sense of control. Since **psychological component** and accompanying feeling of helplessness were essential parts of asthma attacks in the past, what are the changes on psychological, neurological, biochemical and other levels? How strong are they?
- Since many asthmatics are mouth breathers and since nasal breathing provides them with dilator of small blood vessels **nitric oxide**. What is the influence of nitric oxide when asthmatics patients start breathing through the nose? Does it play a role for those patients who is able to stop their morning asthma attacks (4-6 a.m.) by taping their mouth for continuous nasal breathing? Does nasal breathing reduce irritation of air passages in comparison with mouth breathing?
- The effects of **transition to diaphragmatic breathing** are also poorly investigated. The role of this factor should not be underestimated since abnormal breathing pattern is predominant feature for people with asthma.
- Duration of inhalations in many asthmatics are longer than their exhalations. These phases, at the same time, are controlled by **parasympathetic and sympathetic parts of the autonomous nervous system**. If the therapy reverses the lengths of these phases (exhalations becomes longer), does it affect autonomous nervous system and how?
- Similarly, what are the effects of correct posture and relaxation on various components and parameters of the disease?

Apart from these general questions, the biochemical and immunological changes due to changes in breathing are poorly understood areas. There are just few limited Russian unpublished studies indicating normalisation of IgA, IgE, IgG, and IgM concentrations as a result of breathing retraining in a small group of children. What are the effects of chronic hyperventilation on the mechanisms of inflammation and allergy? Are the effects of leukotrines on airways

are different in conditions of chronic hypocapnia for asthmatics? What is the role of mast cells, basophils, eosinophils, and T lymphocytes? Can chronic hyperventilation be a triggering factor in imbalance of helper T cell Type 1 and 2 immune responses?

More general question can relate to interaction between respiration and immune responses. Hyperventilation is a defensive reaction and a part of the "flight-or-flight" response, or a state of increased alertness and emergency for the whole organism, the immune system included. It is possible to hypothesise that the immune system should be in the state of increased sensitivity, when certain agents can provoke strong immune reactions. Which immune reactions do become abnormal? How do they appear? Why only asthmatics get certain abnormal processes and cascades of events?

There are many other theoretical and practical questions that can help us to better understand the underlying mechanism of asthma pathology and create the optimum program for management of this disease.

• **Hypertension and angina pectoris**

Due to his own health concerns, Buteyko had keen interest in cardiovascular problems. According to his public lecture, "*In 1952 I had blood pressure of 220-120, headaches, insomnia, and heartaches. Since my professional occupation was the general form of malignant hypertension, I predicted my remaining life span to be about 1.5 years*" (Buteyko, 1970).

During the 1960's, while testing patients at his diagnostic complex, Doctor Buteyko, together with his colleagues, found relationships between aCO_2 and parameters of the cardiovascular system. In particular, they found that low aCO_2 causes low tone and spasmodic conditions in small arterial blood vessels and capillaries (Buteyko et al, 1964a; Buteyko et al, 1964b; Buteyko et al, 1965; Buteyko, 1968) and the relationship between aCO_2 and blood cholesterol level for people with hypertension (Buteyko et al, 1965). Practically, for each 0.1% decrease in CO_2 (due to chronic over-breathing) there is a resulting increase of 10 mg/dL of cholesterol, for those people who are genetically predisposed to cholesterol deposits.

These results are consistent with our findings from chapter 1 and other Western studies. For example, we already know that chronic over-breathing normally causes tissue hypoxia and decreased blood supply to various vital organs. In particular, blood flow to the heart itself is found to be smaller in numerous animal experiments (for example, see Gelman et al, 1985; Karlsson et al, 1994). Such adverse conditions were observed by many Western doctors (see Chapter 1) and cause various cardio-vascular problems listed in chapter 1, including hypertension and angina pectoris.

While these heart conditions were investigated in Russia, no studies or trials were attempted in Western countries. As in case of asthma, there are many questions. What are the practical factors of the Buteyko method and changes in breathing that cause these favourable changes? What are the psychological, biochemical and neurological changes due to changes in blood CO_2 values? What are the effects of continuous nasal breathing? Since sympathetic dominance is a typical abnormality for many heart conditions, what are the effects of the breathing retraining on the autonomous nervous system?

• **Other defensive reactions**

All these problems (asthma, hypertension, and angina pectoris), according to Buteyko, are not abnormal pathological states, but defensive mechanisms of the organism against excessive CO_2 losses ((Buteyko, 1977). Indeed, narrowing of air passages (as in asthma), or narrowing of arterial blood vessels (as in hypertension and angina pectoris), or pooling of blood (as in varicose veins) causes delays in CO_2 removal. Similar defensive mechanisms are observed in many other situations: blocked nose, polyps, excessive mucus production, varicose veins, spasmodic states of different organs leading to migraine, gastritis, ulcers, Raynoud's disease etc. One needs just to imagine the mechanism how these created obstacles delay excessive carbon dioxide elimination.

As a result, numerous health conditions can be considered as the desperate attempts of the organism to preserve higher carbon dioxide concentrations.

• **Gastrointestinal diseases**

GI (gastrointestinal) diseases are typically accompanied by spasmodic states of small blood vessels leading to hypoxia and problems with an inadequate blood supply to GI organs (Guzman et al, 1999), while hyperventilation leads to reduced perfusion and oxygenation of the liver, colon and other vital organs (for references see Chapter 1). Elimination of hyperventilation was found to be an effective method to deal with gastritis, gastric and intestinal ulcers, IBS, IBD, Crohn disease, and other GI problems. How is it possible that so different health problems can have the

same cause?

From chapter 1, it is known that the GI tract has its own brain (the second brain), which is located and functions relatively independently from the central nervous system. We also learned that chronic hyperventilation often causes various mental and cognitive problems (panic attacks, anxiety, depression, etc.) depending on individual predisposition of people. Hence, it is possible to suggest that low CO_2 stores can affect, in a similar way, this second brain causing variety of GI problems depending on individual differences that include genetic make-up and currently existing abnormalities in the GI tract.

Chronic hyperventilation leads to abnormal intestinal permeability and abnormal protein metabolism since GI tract is the main consumer of amino acids. That suggests that the mechanism of development of these conditions is based on overbreathing. Normal breathing, on the other hand, is incompatible with these pathological processes.

• **Diseases connected with bone metabolism**

Since low aCO_2 causes abnormal changes in ionic composition of the blood, as well as abnormal redistribution of ions in extra-cellular and intra-cellular fluids, that affects regeneration and destruction of bone tissues. In particular, changes in bio-available calcium concentrations were discussed in chapter 1. Therefore, it is sensible to assume that osteoporosis, osteochondrosis, abnormal growth of bone tissues, brittle teeth, and other related conditions are directly influenced by over-breathing.

• Allergies and abnormal immune reactions

Low aCO_2, by interfering with activities of vitamins, catalysts, hormones, and other body chemicals, alters normal human immune reactions. Moreover, physiological studies indicate that low carbon dioxide stores directly affect blood perfusion and oxygenation of the spleen, and other tissues and organs involved in the production of the immune cells (Guzman et al, 1999). As a result of over-breathing, in some cases, pathogenic objects do not cause a normal response of the immune system leading to colds, infections, tonsillitis, while in other cases the reaction of the immune system is excessive, as in hypersensitivity, allergies, and autoimmune disorders. Practical work on breathing normalization revealed efficiency of the Buteyko breathing therapy for colds, respiratory viral diseases, allergies, allergic rhinitis, multiple sclerosis, rheumatism, arthritis, lupus, nephrosis and other related disorders.

• Endocrine diseases

By affecting production and concentrations of different hormones, chronic hyperventilation can cause problems with thyroxin, insulin, estrogen, epinephrine, norepinephrine, calcitonin, pancreatic endocrine hormone and other hormones. That leads to hypo- and hyperthyroidism, diabetes, hyperparathyroidism, diseases of the adrenal cortex, and other health concerns.

For example, Doctor Buteyko suggested the following mechanism for diabetes development (Buteyko, 1962): During times of stress, it is physiologically normal for the organism to have certain hormonal changes in order to prepare the organism for rigorous physical activity ("fight or flight" response). While increased production of adrenalin and nor-adrenalin (the main stress hormones) are well-known effects, decreased insulin level and higher blood glucagon concentrations increase blood sugar level. Such adaptations are obviously useful from the evolutionary viewpoint in order for this physical activity to be efficient. However, chronic stress without physical activity results in chronically high glucagon and low insulin values. These and some other biochemical changes (due to the changed direction of the Krebs cycle) cause hyperglycaemia.

Practical studies done by Doctor Buteyko revealed that diabetics usually have 32-34 mm Hg aCO_2 and 5-10 s BHT (breath holding time after quiet expiration). Therefore, diabetes can be also viewed as a normal effect of hyperventilation in people with a certain genetic predisposition.

More information on diabetes can be found on Christopher Drake's website at www.buteyko.com.au/Chris.html. Numerous cases of complete remission of insulin-dependent diabetes are described in the next chapter.

The practice of Russian doctors revealed that bleeding between menstruations, if present, usually disappears when the CP is above 20 s. Furthermore, females, who have problems with regularity of their monthly cycles, experience normalization in periodicity when their CPs are over 40 s. There are also various infertility problems, which are eliminated when the CP is greater than 40 seconds.

• **Cancer**

Let me describe here the possible chain of events. Chronic hyperventilation washes out CO_2 from each cell of the human organism. Since CO_2 is a powerful dilator of small blood vessels, hypocapnia (low CO_2 concentrations) lead to constrictions of arterioles causing problems with oxygen delivery. As a response, the heart work harder, but less blood arrives at the tissues. Moreover, low cellular CO_2 values cause inability of red blood cells to release whatever little oxygen they bring (the Bohr effect). Abnormalities with the use of the own nitric oxide roduced in the nasal passages intensify vasoconstriction and hypoxia. The final outcome is no CO_2 and no O_2 in the tissues. When oxygen supply is near the critical level, due to chronic hyperventilation, the hypoxic cells are in a dangerous state. Moreover, certain cells, tissues and organs can be predisposed to higher, than average, level of hypoxia due to hereditary characteristics. In addition, some cells, tissues and organs can be especially vulnerable, for example, due to direct exposure to harmful substances (like smoking for lungs, certain chemicals for kidneys, liver, brain, stomach, colon, kidneys, etc.,).

Such local abnormal environmental parameters can cause death in some clusters of cells. All these processes affect the existence of neighbouring cells and the whole organ where these events take place. As a result, the affected tissue or the organ has two choices: to die or to modify parameters of existence in order to survive. Death of cells can be particularly quick and the whole process can be more invasive if certain gangrene-causing pathogens are present. For example, Clostridium perfringens, Cl. welchii, Cl. septicum, Cl. novyi, Cl. histolyticum, Cl. sporogenes, and various other bacterial strains, including Streptococcus and Staphylococcus cause gas and moist types of gangrene. Hypoxia is a known powerful factor in the spread of gangrene, while dozens of professional studies revealed the usefulness of hyperbaric oxygen therapy in the treatment of gangrene. That is a normal current practice in hospitals of various countries.

However, gangrene means death of the tissue. As an alternative to death, the hypoxic cells can change their phenotypes in order to survive in conditions of extreme hypoxia (Charles Maguire, 2003, private communication). The cells can be reprogrammed to the new environment and become similar, in some respect, to the cells of human embryo, which successfully and quickly grows in conditions of severe hypoxia. That causes the formation of a tumour.

Let me again repeat a quote from above,

"Cells undergo a variety of biological responses when placed in hypoxic conditions, including activation of signalling pathways that regulate proliferation, angiogenesis and death. Cancer cells have adapted these pathways, allowing tumours to survive and even grow under hypoxic conditions..." (Chaplin et al, 1986).

Hence, the tumour represents a life form, which adapted to the conditions of the environment. Since severe hypoxia was the norm in the early years of life on Earth, when O_2 content in air was less than a few percent, it is likely that primitive animal life forms were similar, in many respects, to tumours (Charles Maguire, 2003, private communication), while development of tumours is a normal defensive reaction of the organism due to changed environmental conditions.

Hence, cancer and malignant tumours are due to the interaction of the following factors:

1) level of tissue hypoxia;
2) hereditary predisposition (weakness of certain tissues and organs);
3) life-style factors and environmental influences.

Practically, as medical evidence indicates, hypoxia remains the central factor in appearance, survival, growth, and metastasis of tumours.

We may conclude that, apart from other positive effects, normalization of breathing (or steps in this direction) is an efficient method to improve tissue oxygenation and fight cancer. Since the Buteyko method is directly intended to restore normal CO_2 stores, normal breathing and normal cellular oxygenation, it is no surprise that disappearance of tumours is a normal and frequently reported effect of this therapy. However, it usually takes months or years to get this result. At the same time, optimum anti-cancer treatment also needs special dietary changes, which, fortunately, have been investigated and described by nutritionists and medical and other heath professionals in popular and professional literature. According to published observations of Russian doctors, the Buteyko method is very effective during earlier stages of cancer (stages 1 and 2), while there is a definite improvement in quality of life for patients who have cancer in stages 3 and 4.

• **Other health concerns**

Appendix 3 (from Novosibirsk Buteyko web site) provides a list of health conditions, which are characterised by hyperventilation and low aCO_2 and which favourably respond to correct application of the Buteyko method. Appendix 4 (from Moscow Buteyko web site) gives symptoms of hyperventilation syndrome, which are treated in the

Moscow Buteyko Clinic.

• **Formula for aCO_2**

The maximum pause reflects aCO_2, according to the already quoted formula $aCO_2\% = 3.5\% + .05*$BHT (here, $aCO_2\%$ is the alveolar CO_2 concentration in %, 3.5% is minimum CO_2 content in alveoli, K=.05 is a coefficient of proportionality, the BHT is breath holding time after quiet expiration, while sitting, after 10 minutes of rest. Those people, who frequently perform breath holds, do the test until the first desire to breathe (due to the "training effect" described in Chapter 3).

This formula was patented during Soviet times (Buteyko, 1986), it was probably considered by Soviet bureaucrats, as a significant achievement of Soviet science. Available Western studies indicate (chapter 1) that both, carbon dioxide and BHT, usually get lower with worsening of health. There were no systematic attempts in western literature to find the connection between usual aCO_2 and BHT or to define the connection between the personal BHT and the aCO_2.

• **The relationship between ventilation and the BHT**

If the official BHT norm is 40 s, it corresponds to about 6 l/min for minute ventilation. Doctor Buteyko's norm of 60 s BHT corresponds to 4 l/min. As we learned in Chapter 1, patients with moderate asthma or heart disease have about 15 s BHT and 15 l/min for minute ventilation. These and other findings suggest that the approximate relationship between ventilation and the BHT is linear. Hence: If your BHT is 30 s, you breathe twice the norm (about 8 l/min for a 70-kg adult).
If your BHT is 20 s, you breathe three times the norm (about 12 l/min).
If your BHT is 15 s, you breathe 4 times the norm (about 16 l/min).
If your BHT is 10 s, you breathe 6 times the norm (about 24 l/min).

5.4 Practical discoveries and their application

Reduction in ventilation and increase in aCO_2 are equivalent to health improvement, while normalization of breathing parameters is equal to health restoration. From a practical viewpoint this Buteyko finding literally saved thousands of human lives and can save millions more.

The main measuring tool of the Buteyko method is the CP (control pause) or BHT. While the term "BHT (breath holding time)" appeared long time ago in Western medical and physiological literature, there are many variations of this test (see chapter 1) so that the results can differ 3-4 times. Russian doctors, who used the Buteyko method, used the term "CP" (control pause). The strict definition of the CP or the method of its measurement is given in the next section. The concept of the CP, together with its relation to symptoms of various diseases, quality of life and other life factors, is one of the fundamental discoveries of Doctor Buteyko. Let us shortly review his findings or what he observed in his patients with asthma, heart diseases, and bronchitis.

When the CP was very short (e.g., about 5 s), indicating very heavy breathing, the patients experienced acute or life-threatening episodes. These periods of acute hyperventilation could be triggered by numerous factors previously described or by allergic responses, as in cases of asthma.

Between acute episodes their CPs were about 10-20 s depending on severity of the disease, the medication recently used, meals, time of the day, posture and many other factors.

When these patients reduced their breathing, using the Buteyko method, and achieved about 25-30 s CP, they were in a safe zone having no symptoms and no need for medication. Should they experience the symptoms of their main health problem, they could use breathing exercises to alleviate these symptoms.

Similar results in relation to symptoms and need in medication were found for epilepsy, sinusitis, primary hypertension, chronic fatigue syndrome, and some other conditions. Generally, cardiovascular and respiratory problems are the conditions that respond quickly to application of the Buteyko method. An exception here is emphysema since these patients need years for restoration of damaged alveoli in their lungs and complete clinical remission.

Buteyko and his medical colleagues in the USSR and, later, Western breathing teachers, also applied the method for various other health pathologies, including hormonal problems (diabetes, thyroid abnormalities, etc.), GI-liver problems (hepatitis, liver cirrhosis, gastritis, ulcers, IBS, IBD, etc.), urinary problems (urinary infections, nephritis, kidney stones, etc.), musculoskeletal problems (arthritis, osteoporosis, etc.), nervous problems (addictions, sleeping problems, phobias, anxiety, depression, initial stages of schizophrenia, etc.), neurological problems

(Alzheimier, Parkinson, motoneuronal disease, etc.), oncological problems (cancers, leukaemia, radiation disease, etc.), skin problems (eczema, psoriasis, etc.), and some other pathologies. Generally, to efficiently solve these concerns, one should have over 40 s 24/7 for a certain period of time often ranging from weeks (for younger patiens) up to years.

The norm of 60 s CP corresponds, according to Doctor Buteyko, to ideal health and absence of medical pathologies related to so called chronic degenerative diseases or diseases of civilization.

These ideas are reflected in Buteyko Table of Health Zones. Discovery of 7 zones of poor health and 5 zones was so important for Buteyko that he filled a patent application for this invention.

Buteyko Table of health zones

Health state	Type of breathing	Degree	Pulse	Rf	% CO2	AP	CP	MP
Super-health	Shallow	5	48	3	7.5	16	180	210
		4	50	4	7.4	12	150	190
		3	52	5	7.3	9	120	170
		2	55	6	7.1	7	100	150
		1	57	7	6.8	5	80	120
Normal	**Normal**	**-**	**60**	**8**	**6.5**	**4**	**60**	**90**
Disease	Deep	-1	65	10	6.0	3	50	75
		-2	70	12	5.5	2	40	60
		-3	75	15	5.0	-	30	50
		-4	80	20	4.5	-	20	40
		-5	90	26	4.0	-	10	20
		-6	100	30	3.5	-	5	10

Table's comments. Pulse – heart rate in 1 minute; Rf – respiratory frequency in one minute (number of inhalations or exhalations); % CO2 - %CO2 in alveoli of the lungs (*or arterial blood if there is no mismatch); AP - the Automatic Pause or natural delay in breathing after exhalation (*during unconscious breathing); CP - the Control Pause, breath holding time after usual exhalation and until first distress); MP (the Maximum Pause, breath holding time after usual exhalation and as long as possible).

/Based on various Russian publications, especially: Buteyko KP, The method of volitional elimination of deep breathing [Translation of the Small Buteyko Manual], Voskresensk, 1994.

* Note about pulse. Not all people have greatly increased heart rates, as it is given by this table, when their CPs are low. Some categories of people with less than 20 s CP can have a resting pulse of around 60 - 70. However, increased heart rate for lower CPs is the feature of, for example, heart patients and patients with severe asthma. During the 1960's, when conducting his research, and later, Buteyko and his colleagues applied the Buteyko breathing retraining program mainly for heart and asthma patients, who were mostly hospitalized with frequent deficiencies in blood cortisol levels.

Dr. Buteyko developed this table during 1960s, after analyzing hundreds of sick and healthy people in his respiratory laboratory, and presented it during his Lecture for the leading scientists at the Moscow State University in 1969. The Table reflects health of his numerous hospitalized and severely sick patients, who started their journey for health at the very bottom of the table and climbed up, sometimes to the very top of the table.

The middle row of the table corresponds to normal health. Below this row are 7 zones corresponding to disease. The borders for these zones are given by 7 rows (from normal down to -6th degree). 5 zones of super-health are above the middle row. Let us start from the very bottom of this table and then climb up.

Terminally sick and critically ill patients during acute stages

The lowest row of this table corresponds to severely sick and terminally ill patients in critical conditions. When people are at the risk of dying, the table predicts over 100 beats per minute for their heart rate, over 30 breaths per minute for respiratory frequency, less than 3.5% CO2 in the alveoli of the lungs. The CP (Control Pause or stress-free breath holding time after usual exhalation) is less than 5 s.

Terminally sick and critically ill patients in more stable conditions

The next row from the bottom corresponds to severely sick and terminally ill patients in stable conditions.

Typical heart rates of such people are above 90 beats per minute (sitting at rest). Respiratory rate (or breathing frequency) is above 26 breaths per minute at rest. A CO2 concentration in alveoli of the lungs is no more than 4%. There is no automatic pause (period of no breathing after exhalation). The Control Pause is less than 10 s, while the Maximum Pause is less than 20 s. (Numerous medical studies confirmed that over 90% patients with chronic diseases indeed die in conditions of severe hyperventilation, while their heart rate and respiratory frequency become much higher than the norms. Quotes and exact numbers from such studies can be found on my website in relation to heart disease, asthma, cancer, and many other conditions.)

These patients usually require numerous types of medication to prevent their multiple symptoms and complaints. Walking is hard and climbing upstairs, due to heavy labored breathing, dyspnea, and low body oxygenation, is often impossible. Most of the time is spent in bed, since even sitting require efforts.

Sleep is dreadful since breathing and symptoms get much worse after transition into horizontal position. Early morning hours (4-7 am) is the time when these patients are most likely to die from heart attack, stoke, asthma attack, or complications from cancer, diabetes, and many other pathologies.

Patients with moderate degree of their disease

The next row ("-4" degree of health) corresponds to patients whose life is not threatened at the moment, but their main concern are symptoms. People with mild asthma, heart disease, diabetes, 1 and 2 stages of cancer, and many other chronic disorders are all going to be in this zone. Taking medication is the normal feature for most of these people.

As we see from the table, heart rate for these patients varies from 80 to 90 beats per minute. Breathing frequency is between 20 and 26 breaths per minute (the medical norm is 12, while doctor Buteyko's norm is 8 breaths per minute at rest). CO2 concentration in alveoli of the lungs is between 4.0 and 4.5%. The CP is between 10 and 20 s.

Physical exercise is very hard, since even fast walking results in very heavy breathing through the mouth, exhaustion, and worsening of symptoms. Complains about fatigue are normal. All these symptoms are often so debilitating that they interfere with normal life and ability to work, analyze information, care about others, etc. Living in the chronic state of stress and being preoccupied with own miserable health are normal, while efficiency and performance in various areas (science, arts, sports, etc.) are compromised. Sitting in armchairs or soft couches is the most favorite posture.

Parameters of these people get worse during early morning hours with corresponding worsening of symptoms. Many sufferers get less than 10 s for morning CP with all effects accompanying the last stage of the disease.

Most modern people

Most modern healthy people have between 20 and 30 s CP. Hence, they are going to be in the third row from the bottom ("-3" degree of health). While there is no need for taking medication in this zone, numerous health pathologies are frequent. This relates to gastrointestinal disorders (gastritis, IBS, IBD, etc.), musculoskeletal problems (arthritis, osteoporosis, etc.), hormonal and metabolic problems (mild obesity, light diabetes), initial stages of cancer, and many others.

Standing for many hours is hard and they prefer to sit for most part of the day. Physical performance after meals is very poor since respiratory and cardiovascular parameters can shift to the lower zone. Level of energy and desire to work physically are low. The over-excited brain easily invents excuses for own laziness.

Morning parameters are much worse (less than 20 s CP) with all effects that present for this zone.

Normal health

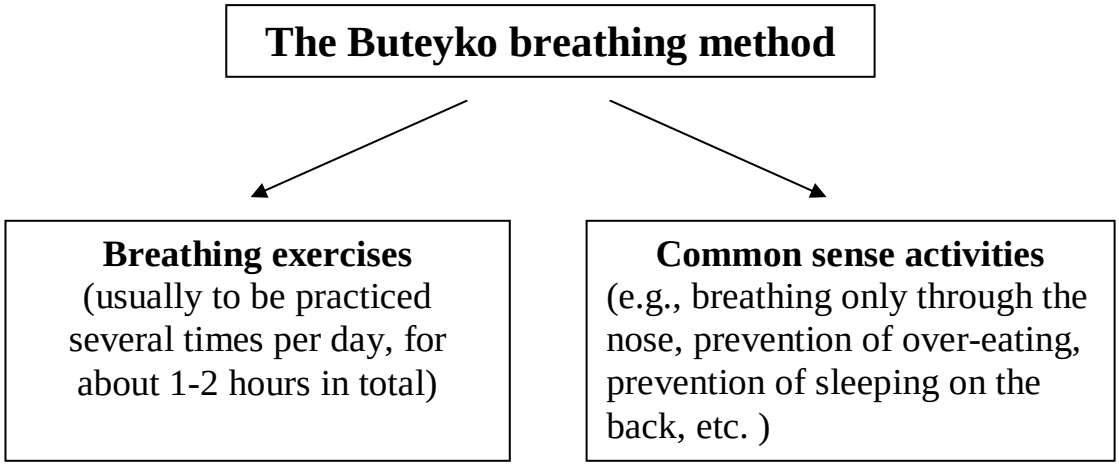
As we continue to climb up the table, we can get the line of parameters that corresponds to norms. The row "minus 2" reflects international norms for breathing: breathing frequency of 12 breaths per minute; 5.5 % for CO2 concentrations in alveoli of the lungs (about 41 mm Hg); 40 s CP and 70 beats per minute for heart rate. People with normal health naturally have so called "automatic pause" or period of no breathing (total relaxation of all respiratory muscles after each exhalation) during their unconscious breathing. The duration of the automatic pause is 2 seconds.

People with normal health and able to run with strictly nasal breathing, safely take cold shower (if they follow certain other rules), have good quality of sleep, and reasonably able to function on the social level (family, community, workplace, etc.).

Development of the Buteyko breathing method

The main practical discovery during 1960's was development of the Buteyko breathing method that is usually tailored for needs of the particular patient. The most poplar version of the Buteyko method includes 2 parts (Fig. 5.1):

breathing exercises to be practiced by the patient and common sense activities that reverse chronic hyperventilation.



There is no uniform understanding about the question: *What does it mean "to learn the Buteyko method?*" Many Russian doctors believed that if you get 60 s CP, then you learned the method. Others were saying that after 2-3 times increase in the CP, patients believe in the tremendous power of the Buteyko method. For many people learning the method meant that they could stop asthma or heart attacks in minutes using Buteyko breathing exercises instead of using medication, like Ventolin or heart pills.

In my view, there are 3 levels of learning the Buteyko breathing method:

Level 1: The student is able to eliminate the main symptoms of own health problems (e.g., asthma, heart disease, epilepsy, sinusitis, eczema, insomnia, constipation, etc.) using the Emergency Procedure so that the Emergency Procedure can substitute the use of medication or significantly reduce the dosage (e.g., twice). Such students have low CP s (usually about 5-15 s), while elimination or reduction in medication is their main practical result. Only about 10% learners or less are satisfied with this level. It takes about 1-2 days to learn.

Level 2: The student partly normalised own breathing until about 25-35 s CP, so that he has no need for medication and does not experience the symptoms of his main health problem, e.g., asthma, bronchitis, heart disease, epilepsy, sinusitis, eczema, etc. However, such learner can still suffer from airways inflammation, allergies, GI problems, hormonal problems, and musculo-skeletal problems. Various medical drugs can be required to control symptoms of these "high-CP" diseases, This is the level achieved by most people on the west. It takes about 1- 3 months to learn.

Level 3: The student has no less than 60 s at any moment of the day. Such CPs provide the guarantee about absence of pathologies and symptoms related to many degenerative health conditions (over 150 so called "diseases of civilization"). While achievement of this level requires will power, self-discipline and determination, there are many students world-wide who got there and none has regrets about the time and energy spent. The final goal of the method for Level 3 is normalization of breathing. Among parameters of normal breathing, according to Doctor Buteyko, are: 60 s CP (breath holding time at rest, after quiet expiration, with no stress at the end of test); 4 l/min ventilation rate (for a 70-kg man); and 6.5% CO_2 (about 46 mm Hg CO_2 at sea level) in alveoli and arterial blood. As we can see here, his parameters are more rigorous than typical medical and physiological norms: 40 s CP; 6 l/min; 40 mm Hg at sea level (or about 5.3%). It often takes 0.5-2 years for an adult to learn.

Note that when discussing the CP here, I mean the minimum CP throughout the day and this is usually (for over 90% learners) is the morning CP. The reasons are described in the next chapter.

There are several factors that define the rate of learning for a student. The most important are:

1. Age of the person (e.g., young children often can progress from, for example, 5 s up to 60 s CP in 3-5 days; teenagers in 2-3 weeks; elderly people in 2 years).
2. Fat reserves (i.e., obesity makes progress slower since obese students, when their practice breathing exercises, first naturally loose weight with moderate CP growth and later their progress normally).
3. Amounts and types of medication used in the past (i.e., those learners who consumed large amounts of medical

drugs during previous years have slower progress rates).

5.5 Advance of the method in the USSR and Russia

According to official statistics, up to 1967, Buteyko laboratory, using his breathing method, successfully treated more than 1,000 severely sick in-patients with asthma, hypertension, and angina pectoris.

Analysing the scope of more than 50 scientific publications written by Doctor Buteyko and his colleagues in 1960's, one can conclude that most of the papers are devoted to CO_2 effects on the cardiovascular system (e.g., tone of arteries, capillaries and veins as a function of aCO_2). Over 15 publications are about different technical improvements in measurements of physiological respiratory parameters using his diagnostic complex. There are also several articles on asthma, its causes and treatment.

Al least 5 Ph.D. dissertations in medicine and physiology were written on respiration, carbon dioxide properties and hyperventilation under Doctor Buteyko's leadership.

Many medical professionals contributed to the development of the Buteyko breathing method, its theoretical foundation, and practical applications. In particular, articles on the Buteyko method and its theoretical foundations were written by the following Buteyko colleagues:

• **Dyomin D.V.**, medical doctor, Ph. D. (Novosibirsk), author of numerous research publications on the relationship between: a) aCO_2 and tone of arterial vessels in hypertension and angina pectoris; b) aCO_2 and blood cholesterol.
• **Genina V.A.**, medical doctor, Ph. D. (Novosibirsk), author of several theoretical and practical articles about the treatment of bronchial asthma using the Buteyko method.
• **Lapa N.A.**, medical doctor (Children's Clinical Hospital No. 8, Novosibirsk), author of publications on the Buteyko method for children.
• **Odintsova M.P.**, medical doctor, Ph. D. (Novosibirsk), author of a of Ph. D. thesis on the aCO_2 connection with the tone of arterial vessels in patients with coronary insufficiency and hypertension, and articles on: a) aCO_2 and its influence on the cardiovascular system in patients with hypertension and angina pectoris; b) hyperventilation and bronchoconstriction of asthmatics.
• **Paschenko S.H.**, research scientist and medical surgeon, Ph. D. (Zaporozshski Medical Institute, Ukraine), author of research articles on: a) CO_2 influence on bone regeneration; b) tissue oxidation processes due to free radicals in CO_2-deficient asthmatics.
• **Samotesova A.H.**, medical doctor, main endocrinologist of Krasnojarsk region (Krasnojarsk), author of publications and research on the Buteyko therapy for diabetics.
• **Souliagin S.S.**, medical doctor (Obskoi Central Hospital, Novosibirsk), author of several practical articles on the Buteyko method (including the influence of focal infections, correct use of physical exercise and other auxiliary methods for health restoration).

By 1990's over 100 Soviet medical doctors completed qualification courses with Doctor Buteyko, while learning how to apply this method in practice.

In 1980's former patients, many of whom were on steroids and with disabilities, literally flooded the Soviet Health Ministry with letters describing their medical histories and success achieved due to the Buteyko method. That resulted in the following practical steps indicating official recognition and approval of the Buteyko method by Soviet medical authorities.

- In 1983 the USSR Committee on Inventions and Discoveries issued a patent with the title "*The method of treatment of hypocapnia* [low aCO_2]" (Author's certificate No. 1067640 registered on 15 September 1983). What is unusual about this document is that it has the priority date of the discovery 29 January 1962. Thus, the discovery was officially accepted more than 20 years later. The patent describes the treatment of bronchial asthma and 40 patients with hypertension and angina pectoris (with over 80 % success rate).
- In 1985, the method was officially approved by the Soviet Health Minister (Burenkov, 1985). The directive revised old note of the Buteyko method for the treatment of bronchial asthma in various medical establishments.
- In 1986 the USSR Committee on Inventions and Discoveries issued another patent "*Method of defining CO_2 content in alveolar air*" using breath holding time (Buteyko, 1986). The formula from this patent was described in the previous section.

Up to now, hundreds medical professionals and breathing instructors adopted the Buteyko breathing method in their work in former Soviet states, while the total number of treated people is over 200,000. Most of these former patients were asthmatics. It was found experimentally that asthmatics usually respond quickly to the therapy. Most other patients had problems with the cardiovascular, nervous and endocrine systems.

More recently, Doctor Buteyko and **Andrey Novozshilov**, chief doctor of the Buteyko Clinic in Moscow, had several interviews published in central Russian newspapers and magazines. The originals and their translations can be found at www.buteyko-clinic.ru.

The advance of the method had and has following challenges. It was not easy to convince ordinary doctors and medical officials that the answer was literally under their nose. Only those medical doctors, who applied and practiced the method themselves, realized the power of this health restoration technique. Therefore, Buteyko was looking and invited those medical professionals, who suffered from asthma, bronchitis, heart disease, CFS, and other disorders, to learn the method. Moreover, Buteyko correctly noticed that, if doctors, after learning the basics of the method, achieved 60 s CP (control pause), then these doctors could become good practitioners or teachers of the Buteyko method. (Up to early 1990's, the Buteyko method was called in the USSR "the method of voluntary elimination of deep breathing".) Hence, Buteyko decided to organize special commissions for certification of breathing practitioners so that they had over 60 s CP at any moment.

These CP commissions produced ambivalent feelings and contradicting effects among participating doctors. Imagine that some people are given a right to come and check your breathing any time, in order to find out if you are good enough to teach the technique. On the other hand, this idea was a logical part of the totalitarian Soviet state based on total control, suspicion, and belief in (partly) evil human nature. The commissions did the job until the collapse of the USSR. Later, the bottled emotions (resentment, anxiety, fear, suspicion, etc.) came out and promoted disintegration of the previously unified movement of Buteyko medical doctors in former Soviet states. However, they are still the most educated and professional teachers of the Buteyko method in the world. Currently there are more than 20 Buteyko medical centres and clinics functioning in former Soviet states.

Apart from medical doctors, in late 1980's and in 1990's Doctor Buteyko trained numerous breathing instructors to apply the method. 30 s CP was one of the criteria for the breathing instructors to be qualified. These instructors worked mainly with patients who had severe or moderate forms of asthma, bronchitis and heart disease. This project was a big success during "Perestroika" since the USSR had huge number of sick people, the method was popular in mass media (TV, radio, newspapers) and breathing instructors with 30 s CP saved lives of thousands and helped them to return to ordinary life with about 25-30 s CP.

In early 1990's the first Russians breathing instructors and a few doctors settled in western counties and started to teach the Buteyko method to western people.

5.6 Advance of the method in western countries

Among first students to learn the method on the west were people from Australia, the UK, New Zealand, and the Netherlands. Alexander Stalmatsky, author of 2 published books "Freedom from asthma" and "Freedom from insomnia", taught thousands of people first in Australia and later in the UK. Many his former students later became breathing practitioners. Both these countries currently (2007) have more than 100 Buteyko breathing teachers each. Some of them are united in professional organizations. The largest are BIBH (Buteyko Institute of Breathing and Health) and BBA (Buteyko Breathing Association). There are also several other, smaller organizations. During first years of the 21-th century the method was actively spreading in the USA and Canada so that now there are over 20 breathing practitioners in these countries. Cuba got about 15 practitioners trained by BIBH about 3 years ago. While the Netherlands has only several practitioners, they are very active.

Most western practitioners (over 90%) teach the method as their part time job, but few (less than 5%) are working full time. About 7-10% have formal medical education. Thus, there are many GPs, nurses and physiotherapists applying the Buteyko method on the west.

Western breathing teachers already saved thousands of lives and helped over 100,000 people to be free from symptoms and medication. Meanwhile, there are great reserves to improve the quality of teaching. These reserves are analyzed later in the chapter about promotion of the Buteyko method.

5.7 Experimental trials of the Buteyko breathing method

• 1968, Institute of Pulmonology, Leningrad, USSR (bronchial asthma, hypertension and angina pectoris)

50 patients with severe bronchial asthma, hypertension and angina pectoris, all of them with many years of heavy medication, most with steroid deficiencies and organic complications; success rate 95% (Khoroscho, 1982).

• 1981, Sechenov's Medical Institute, Moscow, USSR (asthma, with pneumonia, rhinitis, chronic tonsillitis)

52 children (34 in-patients and 18 out-patients; 3-15 years old) with regular asthma attacks (once per day or more); 41 of them had pneumonia, 27 rhinitis, 36 chronic tonsillitis. All had problems with breathing through the nose, palpitations, and were bronchodilator users. In 1-5 days the patients were able to stop the attacks, cough, blocked nose, and wheezing, using the method. Observations in 1-3 months showed considerable improvements (cessation of heavy attacks or a total disappearance of the symptoms) in 83%, some improvement (less heavy attacks and considerable reduction in medication) in remaining 17%. Their average CP increased from 4 to 30 s, aCO_2 from 25 to 36 mm Hg. Higher blood concentrations of IgA, IgM, IgG, and IgE were found, according to laboratory reports. Blood pressure normalised, forced expiratory volume raised over 5 times. Significant increases in lung volume, expiratory speed, and other parameters were found. Average breath holding time (CP) increased from about 3-6 s to over 30 s. For more information on this trial, visit www.buteyko.com/trials.html#children.

• Reports from two conferences in Moscow and Krasnojarsk in 1988 (large variety of health problems)
In addition to these trials, there were about 30 published reports (Buteyko method, 1992) of about 40 Russian medical doctors and health professionals, who met during two conferences in Moscow and Krasnojarsk in 1988 in order to share their practical experience of application of the Buteyko method in over 20 medical hospitals and clinics in Russia. The total reported number of treated people, according to the published conference proceedings (Buteyko, 1991), was over 3,000. Although most of them had respiratory (asthma, bronchitis, rhinitis, etc.) and cardiovascular (hypertension, angina pectoris, ischemia, etc.) problems, hundreds were treated or relieved from arthritis, osteoporosis, epilepsy, ulcers, gastritis, kidney stone problems, hepatitis, different infertility conditions, skin diseases (e.g., dermatitis, psoriasis, eczema), etc. Typical reported results were either some or essential improvement for over 90% patients, while remaining patients were not able to normalise their breathing parameters due to absence of desire or motivation and quitting the method during its initial stages. Thus, those patients who achieved large CPs significantly improved their health state. Normalization of breathing always leads to dissapperance of symptoms and no need for medication.

• 1990, Shevchenko's Central Hospital, Kiev, Ukraine (radiation disease)
50 patients with radiation sickness due to Chernobyl's nuclear plant disaster. 82% patients had considerable improvement in blood analysis, cardiovascular parameters (blood pressure, pulse, etc.), work of the digestive system, and reduction in medication. No cases of side effects or complications due to the breathing exercises were reported (Bebeshko et al, 1990; p.221, Zimchenko & Romanenko, 1991).

• 1991, Kiev Scientific and Research Institute of Epidemiology and Infectious Diseases, Kiev, Ukraine (AIDS)
This trial involved 7 young patients with AIDS, two of them had HIV-infection in the lympho-adenopathic stage (Frolov et al, 1991a). Progression of this disease is usually accompanied by a variety of symptoms and complaints in the digestive, immune, cardiovascular, respiratory, hormonal and other systems. The official documents of the Institute provided information about improvements in clinical symptoms and the patients' quality of life such as emotional stability, irritability, panic attacks, chronic fatigue, insomnia, digestive complaints and some other factors. All symptoms were relieved with no side effects due to breathing retraining.

• 1991, Kiev Scientific and Research Institute of Epidemiology and Infectious Diseases, Kiev, Ukraine (hepatitis and liver cirrhosis)
30 patients, mostly 20-40 years old, diagnosed with acute (6 patients) and chronic (18 patients) hepatitis and cirrhosis of the liver (6 patients) applied the Buteyko method, while continuing to use traditional medication (Frolov et al, 1991b). 28 patients had remissions of their symptoms while 25 showed improvements in their blood test results. The official documents report 93% success rate.

• 1995, Mater Hospital, Brisbane, Australia (asthma)
20 patients with a long history of asthma and significant medication. In 3 months, they decreased use of relievers (bronchodilators) by 96%, preventers (inhaled steroids) by 49%. Minute volume decreased from 14 l/min to 9.6 l/min. The symptoms' score was improved by 71% (Bowler et al, 1998).

• 1997, Perth Academy of Natural Therapies, Australia (chronic fatigue syndrome)

A study by Shellie Gaskin, as a partial fulfillment for a Diploma of Naturopathy, was conducted on 15 people diagnosed with CFS. There were following improvements: fatigue 87%, night sweats 75%, depression 70%, allergies 66%, anxiety 66%, muscular aches 60%, difficulty sleeping 54%, and headaches 50%. After 10-12 weeks all those who continued their breathing exercise regimes reported a 100% reduction in fatigue.

• 1999, Alfred Hospital, Prahan, Australia (asthma)

18 patients with mild to moderate asthma were taught the Buteyko method by a video and compared with 18 control subjects (Opat et al, 2000). The study found a significant improvement in quality of life and significant reduction in inhaled steroid use.

• 2003, Gisborne Hospital, Gisborne, New Zealand (asthma)

In this blinded randomised controlled trial conducted in 38 people with asthma Buteyko Breathing Technique group was compared with control (McHugh et al, 2003). The Buteyko group was taught by a Buteyko practirtioner Russell Stark. As in the previous western trials, the Buteyko group reduced inhaled steroid use by 50% and ß2-agonist use by 85% at six months from baseline. In the conclusions, the medical professionals wrote, *"**Conclusions** BBT is a safe and efficacious asthma management technique. BBT has clinical and potential pharmaco-economic benefits that merit further study."* The instructions for the Buteyko group were provided by Russell Stark.

• 2003, Division of Respiratory Medicine, City Hospital, Nottingham, United Kingdom (asthma)

90 patients with asthma taking an inhaled corticosteroid participated in a randomised controlled trial. The groups were followed in 3 and 6 month periods (Cooper et al, 2003). "*Symptoms remained relatively stable in the PCLE and placebo groups but were reduced in the Buteyko group…The Buteyko breathing technique can improve symptoms and reduce bronchodilator use…*"

• 2003, Glasgow, United Kingdom (asthma)

According to the recent press release (4 December, 2003) of the British Thoracic Society (the UK's professional body of respiratory specialists),

"Nurse, Jill McGowan, led the world's largest clinical trial to measure the effects of the Buteyko method (breathing retraining exercises in conjunction with conventional asthma management). 384 of the initial 600 participants (64%) completed the trial…

Those patients who were taught the Buteyko Institute Method all experienced significant improvement in asthma, with reduced symptoms, reduced medication and improvement in quality of life:

- *asthma symptoms decreased by an average of 98%;*
- *use of reliever inhalers decreased by an average of 98%;*
- *use of preventor inhalers decreased by an average of 92%."*

These results were found after 6 months (McGowan, 2003). This self-funded trial was possible due to heroic and sacrificial, in financial terms, efforts of the Buteyko practitioner Jill McGowan.

• 2005, Foothills Hospital, Calgary, Canada (asthma)

64 patients, all of whom were using inhaled corticosteroids, after 6 months improved their asthma control from 41% to 75% (Proceedings, 2006). Decrease in inhaled corticosteroids was found in 39% patients, elimination of steroids in 21%.

• 2006, Royal Prince Alfred Hospital, Camperdown, Australia (asthma)

The Buteyko method was applied for 12 weeks. Median reduction in usage of relievers was 86%, preventers 50% (Slader et al, 2006).

• 2006, Gisborne Hospital, Gisborne, New Zealand (asthma)

8 children, from 8 to 14 years old, in 12 weeks reduced their average β2-agonist use (salbutamol) by 66%, inhaled steroid (fluticasone) by 41% (McHugh et al, 2006). While 11 courses of prednisone were given 3 months before the trial, only 1 course of prednisone was required three months after the trial.

None of these trials or reports revealed any complications or side effects due to the Buteyko breathing method,

although there are many important practical rules and possible complications in cases of incorrect application of the method.

A note about Soviet medical approbations or trials. The Soviet health care system, for political and social reasons, had always been more centralized than Western ones. Innovations in medicine were usually introduced by the Soviet authorities and health care bureaucracy. New ideas and drugs were often tested in hospitals and research Institutes, which provided higher authorities with reports about the results. In the West, as we know, results of scientific studies or trials are usually published in journals. While the Soviet approach was different, the reliability of their information was comparable. Directors, managers and project leaders of corresponding organizations and departments put their signatures to official reports about such Soviet trials and their approbations. These people were personally responsible for the trustworthiness of the results and their names and copies of the relevant documents, related to the Buteyko trials as well, are still archived in Russia and Ukraine.

Q&A section for Chapter 5

Q: Why did the western clinical trials not revealed improvements in lung function test in those students who learned the Buteyko method?

A: The lung tests reflect presence of inflammation meaning that, while the Buteyko group could reduce medication, their airways, on average, remained inflamed after they practiced the method for 3-6 months (typical durations of last follow ups). Healing would result in better numbers. It is a big physiological change to eliminate inflammation and it needs large morning CPs (about 30-35 s) with no exposure to triggers for some weeks so that the tissues can get healed. Another expected achievement is elimination of allergies leading to full clinical remission of asthma. This is how the method was and is taught in Russia (so that the patient has no inflammation and no allergies).

Many western students, as we know, progress only until about 25-30 s (no medication, better sleep, ability to exercise, etc.). Why do they not progress further? Practice indicates that usually breathing teachers help their students to achieve the same level as they have.

It is not a surprise then that Buteyko demanded 60 s for his doctors in Russia so that the students learn the method till the level of the teacher. High CP teachers, from the very beginning, target their students to Professor's golden health standard: 60 s CP. Russian doctors explain to their students that being stuck, during the healing process, at 40 s is a normal phenomenon due to fundamental changes in the organism. Such information about the known future obstacle (40 s threshold) is important for long-term motivation.

Q: Is CO_2 the only cause of success for the Buteyko method?

A: CO_2 is the most known and investigated factor that relates to breathing and the Buteyko method. There are many other factors that are known to students and practitioners.

The Buteyko method also includes, for example, psychological factors. The students learn how to stop their symptoms and prevent attacks, how to pay attention to stress and other factors that cause hyperventilation. Hence, they acquire a sense of control over their health. Helplessness and depression are no longer the parameters that define the course of their diseases.

Nasal breathing helps the body to use its own nitric oxide that is produced in nasal passages. The roles and some important effects of this hormone have been discovered very recently and there are still many questions in relation to this substance.

Emphasis on diaphragmatic breathing and relaxation of chest breathing muscles should favour elimination of possible abnormalities in regulation of breathing by the autonomous nervous system. Activity of the chest breathing muscles at rest often points to sympathetic dominance since chest muscles get active during both exercise and hyperventilation. While the Buteyko method is not focused on slow diaphragmatic breathing pattern, this pattern gradually appears by itself, for example, during sleep. This effect promotes lymphatic drainage of the nodes located under the diaphragm.

Passive relaxed exhalation during the breathing sessions should also have good effects on the balance between parasympathetic and sympathetic nervous systems. These systems are often out of balance for many diseases, like asthma, heart disease, chronic fatigue, cancer and other health problems.

Deliberate attention to posture and relaxation of body muscles should also influence the autonomous nervous

system. When we relax we again pacify the overexcited sympathetic nervous system which is often too active due to the fight-or-flight mode. Healing and tissue repair are more active when the parasympathetic system is dominant.

Reduced breathing decreases oxygen levels in the lungs and blood creating temporary hypoxia. Such hypoxia is beneficial for various reasons. First, modern air has too much oxygen. Free oxygen in our bodies generates free radicals causing cellular damage and aging. This damage is stronger during hyperventilation. Second, hypoxic training at high altitude has many known published benefits.

It is difficult to tell at the moment what the contributions of these factors are. Clearly, they are individual. Can the various effects of the Buteyko method be separated? Probably yes, for example, using CO_2 injections or CO_2 chambers or submarines with special air. Can CO_2 chambers have similar effects? There are many other interesting scientific questions for further research.

Q: There are many medical studies indicating that acute hyperventilation produces asthma attacks in asthmatics. However, several studies found that acute hyperventilation with CO_2 enriched air also results in asthma attacks. Therefore, as some doctors claimed, low aCO_2 could not be considered as a single cause of asthma. Is this opinion correct?

A: Before being tested with CO_2 enriched air in laboratories, typical asthmatics had many hundreds of times the following course of events. On the background of chronic hyperventilation (all known studies reported presence of hyperventilation for initial stages of asthma), asthmatics experienced the influence of some other triggering factors (like exercise, overeating, oversleeping, allergies, etc.), which resulted in additional hyperventilation and further bronchoconstriction or in further inflammation of airways with the same results: feelings of air shortage (due to airway obstruction), chest tightness, laboured breathing, etc. all signs of an asthma attack. (Sometimes, this airway obstruction could be due to, for example, excessive mucus production or inflammation. That could result in anxiety and panic causing acute hyperventilation.)

In all cases these asthmatics breathed normal air with about 0.04% CO_2 concentration. Thus, before the attacks the following physiological changes were repeated many hundred times: abnormally hard work of the respiratory muscles, increased air flow through the respiratory tract, increased amplitude of pressure variations in internal organs, etc. All these changes, before the attacks, were sensed many hundred times by the millions of nervous cells of the nervous system. Finally, further lowered aCO_2 and some other factors produced additional bronchoconstriction and the attacks.

Now exactly the same asthmatics arrive in the laboratories, where they perform the same acute hyperventilation, which is accompanied by all these described additional features (again sensed by the millions of nervous cells) with one difference, the inspired air is CO_2-rich. Such air has never been experienced by these asthmatics before, but the whole nervous system learned that such situation causes bronchoconstriction. What would be the result now?

The result due to the changed stimulus would be defined by how much of the previous stimulus is left. Low carbon dioxide already created many chronic abnormal changes. Finally, some other triggers which cause the attacks can also be at work when the person deliberately hyperventilates, even with temporary increase in carbon dioxide stores. It was not sudden drop or increase in carbon dioxide stores that causes or prevents asthma attacks, but those chronic changes which affect every cell of the respiratory tract in asthmatics. Therefore, since less than 1% stimulus is absent (low CO_2), while the remaining 99% is left, the reaction would be exactly the same, as for the whole stimulus.

But assuming that the human nervous system is incapable of learning from the previous experiences repeated hundreds of times, and that all these events sensed and recorded by the nervous system did not produce habituation and conditioning, one can assert that low carbon dioxide is not the cause of asthma.

Therefore, even in conditions of artificially increased aCO_2, the influence of so many areas of the nervous system should be more powerful, than that of the breathing centre. Meanwhile, if such tests with CO_2-rich air were repeated many times, the effect of gradual relearning can be observed and acute hyperventilation with CO_2-rich air would not cause bronchoconstriction and the attacks.

Moreover, physiological studies found the confirmations of this psychological effect based on physiology of the nervous cells. It is known that, for example, some breathing manoeuvres (chapter 2), e.g., Valsalva and Müller manoeuvres, or breathing air with the same composition at the end of the breath hold, as in the lungs, extends BHT. Why? All previous life, movements of respiratory muscles resulted in new oxygenated air coming into the lungs. Normally, the nervous system learned millions of times, that such respiratory movements are signs of new (fresh) air flow. When, all of the sudden, the conditions are different, only the breathing centre creates the stimulus to breathe,

while the rest of the nervous system is "happy" and does not contribute to the urge to breathe.

It is now a clear fact, which has been confirmed by all published studies, that development and first stages of asthma are always accompanied by hyperventilation. The situation with medical respiratory professionals and asthma was accurately reflected by Peter Kolb,

"… asthma is a disorder which is investigated by thousands of respiratory specialists with millions of dollars worth of equipment to measure breathing. Yet after more than half a century of work by all these people measuring patients' breathing, they haven't picked up that asthmatics are just breathing too much" (Kolb, private communication, 2001).

Q: Doctor Buteyko claimed that, for example, gastritis is caused by hyperventilation. However, it is known that, poor dietary habits (like eating when not hungry, not chewing food properly, eating spicy and hot meals) can create gastritis without any influence of breathing. How can such facts be explained?

A: Practical studies done by Doctor Buteyko revealed that it was necessary for the patients with GI (gastrointestinal) problems to have low levels of aCO_2 pressure (e.g., less than about 40 mm Hg) in order for gastritis and other GI disorders development to take place. That is probably due to appearance of certain pathological substances generated by affected mucosa of the stomach lining. In practical terms, low CPs (less than 40 s) are required for the progress and existence of the disease. At the same time, the ideal CP of 60 s makes such pathological processes impossible due to normal repair, adequate oxygenation and blood supply of the stomach. The ideal CP and GI disorders are incompatible.

Thus, if we accept 40 mm Hg aCO_2 level (about 35 s MP) as normal (as it is done by official medicine), then GI problems and hyperventilation are independent events. A person can have GI problems, gastritis included, with or without hyperventilation.

If our norm is 6.5% aCO_2 (60 s CP), then gastritis and other GI problems cannot take place, unless this aCO_2 level is lowered. Damage to tissues intensifies respiration making the CP less than 40 s.

Q: Which health conditions, while related to breathing and curable by breathing retraining, are not considered as breath-related by ordinary people?

A: "Breath", in Russian, has the same translation as "spirit". Similarly, other people consider breathing as something immaterial. Hence, when thinking about breathing, many people believe that breath can only relate to respiratory problems, fatigue, and, maybe, asthma. What would be opposite, in our minds, to the volatile and escapable breath? Of course, our strong bones. Hence, it is difficult for many people to make a mental connection between fragile breath and bones. However, musculoskeletal problems respond to the Buteyko breathing method as nicely as heart disease or diabetes. The short summary of the effects of breathing retraining on various disorders is provided in Appendix 7. Russians even published a study about a greatly accelerated rate of bone healing in chickens who were living in air enriched with CO_2.

Q: How can the breathing teacher deal with a student who has some rare disorder or a variety of symptoms related to different diseases? How could one know if the Buteyko method can solve some specific health problems?

A: The names of health conditions, even in official medical literature, often do not have strict definitions. For example, asthma can have wide range of cases with varying degree of symptoms. Many cases of asthma can be close or even diagnosed as COPD, emphysema, bronchitis, etc., by different countries and doctors. Russia, for example, have bronchial asthma, asthmatic bronchitis, etc. Some leading medical authorities claim that the term "asthma" should not be used by medical professionals. The same vagueness relates to many other health conditions, ranging from heart disease to various neurological and GI problems.

This absence of clear criteria in official medicine is based on absence of the understanding of the mechanisms of disease appearance, development, and treatment. However, breathing teachers are armed with understanding of:

- the cause and mechanism of development of various symptoms;
- the method of their treatment.

Doctor Buteyko in his lectures was often going, one by one, through the effects of hypocapnia on different systems, organs and tissues of the body (what happens with cardiovascular system, musculo-skeletal, nervous, GI, etc.). These facts indicate more emphasis on symptoms and specific abnormalities rather than official labels.

It would be logical therefore, to view the "sudden" appearance of various human abnormalities and symptoms with the assumption of increased ventilation. Practically, when a student asks a breathing teacher about possible efficiency of the method for a certain rare health condition ("Can you help me with my …?"), the teacher may ask the

student about particular symptoms and tests' manifestation of the disease, time sequence of their appearance, their severity, and evaluate current breathing (e.g., visually, by voice, posture, and/or CP test). This information could provide the teacher with information related to the likely effects of the method when the certain CP level is achieved (when fatigue is reduced, rigorous exercise is possible, nose is clear, medication can be safely reduced, cold shower can be taken, etc.)

It would therefore make more sense to speak, in many cases, about the same parameters that practically matters: current symptoms, tests' abnormalities (as manifestations of hyperventilation in respiratory, cardiovascular, nervous, immune, and other systems), and current CP.

Finally, let us look at the dynamic of labelling in Russia. The website in Novosibirsk and early Russian doctors used official medical names (Appendix 3). Later, instead of diseases, many websites have been using the names of symptoms (like coughing, blocked nose, running nose, too much mucus, allergies, cold hands, feeling tired, pains in various body parts, sensation of panic, digestive complaints, insomnia, etc.).

The real life teaches us that there is one disease of deep breathing and many symptoms (asthma, heart disease, diabetes, chronic fatigue, etc.) depending on personal factors.

"Q: What is most important in your method?
A: To decrease deep breathing (the volume of inspiration) until the norm. Not to hold breathing, but gradually normalize it. This is difficult, although primitive people and animals breathe like that…" (Buteyko, 1997).

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