Dear readers. I hope the translation is correct. I wrote the text in German and translated it by an AI.

Today I would like to tell you about our daughter. Now she is 22 years old and has been through an endlessly long ordeal. To say it in advance, she is doing WELL today, given the circumstances!

It will be a long report in which I will also go into medical values.

But I think it's worth writing AND reading this report. And I hope that not only those affected will read this report. I hope that medical professionals will also read this report and perhaps take our experiences as inspiration for a possible new approach.

Our daughter has been diagnosed with many things. ME/CFS, hEDS (hypermobile Ehlers-Danlos syndrome), fibromyalgia and post-Covid.

Her ordeal began when she was 8 years old. She had an inflammation of the growth plate in her heel due to sporting activities. At that time, the pain began to spread and ended up all over her body. Years of suffering and a medical marathon began.

She had several stays in a pain clinic for children, but this only alleviated the symptoms to a limited extent. When she was about 12 years old, she contracted influenza, herpes zoster and mycoplasma pneumonia within a year. Then she lost a significant 8 kg (18lbs) and only weighed 44 kg (97lbs).

Her complaints became more and more pronounced over the years. She could barely walk 300 meters. She spent further stretches in a wheelchair.

At the age of 19, ME/CFS was diagnosed on the basis of the symptoms and subsequent positive laboratory values:

anti-ß-1-adrenergic antibodies

anti-ß-2-adrenergic antibodies

anti-Muscarinic Cholinergic Receptor-3 Antibodies

anti-Muscarinic Cholinergic Receptor-4 Antibodies

A human genetic report was also prepared for her in 2019 (genetic family tree analysis over three generations). All genes were examined for a defect or change. A mutation of the TPM2 gene was detected. This could indicate nemaline myopathy type 4, which could not be confirmed, but would fit our daughter's symptoms. At the same time, she was diagnosed with hEDS (Ehlers-Danlos syndrome of the hypermobile type).

In 2020 and 2021, our daughter underwent endometriosis surgery.

Presumably due to her hEDS, she was diagnosed with ligamentous stenosis of the coeliac trunk. This was operated on in September 2022 with robotic assistance, laparoscopically (Dunbar operation).

In the following weeks, these symptoms were extremely pronounced:

Pain throughout the body

Migraine

Shortness of breath

Palpitations

Cardiac arrhythmia

Palpitation (feeling the palpitations)

POTS (postural tachycardia syndrome)

Muscle twitching

Muscle cramps

Physically unstable and always exhausted

THE UNEXPECTED PATH TO IMPROVEMENT:

In recent years, we have seen a number of seemingly inexplicable phenomena for which no doctor (and we have asked many) had an explanation.

In 2018, the pain all over her body was once again unbearable, so a doctor friend of ours infused our daughter with cortisone in doses of 500-250-250 mg. Our daughter was almost pain-free for about a week. A repeat treatment some time later with oral cortisone at a much lower dose was unsuccessful. (Phenomenon 1)

After her Dunbar operation in September 2022, our daughter had to inject Clexane for 10 days post-op to prevent thrombosis. A few days after the last injection, she told us that her body pain was as good as gone under Clexane and had now returned. As we still had a few syringes of Clexane left, we tried it ourselves and injected her with Clexane once a day again. The pain was gone again. After a report to the GP (he had also diagnosed ME/CFS), we "tried" an oral antithrombotic medication. It was not successful. Another attempt at pain relief with Clexane was successful. (Phenomenon 2)

It was a Saturday in November 2022, I was sitting in the kitchen with my wife when our daughter came out of her room complaining of severe shortness of breath. She was physically exhausted.

As my wife and I both come from a medical background (she is a nurse and I am an emergency paramedic), we are well prepared for medical emergencies.

So I measured our daughter's oxygen saturation (SpO2). The value was completely normal at 100%

But since I also have medical oxygen for emergencies, I tried a little experiment and had her inhale oxygen through an inhalation mask.

We were amazed at the result. It took 10 minutes for our daughter to seemingly transform into a new person. She took the mask off her face and said: "And what are we going to do now?" She wanted to go shopping. Her acute symptoms, the shortness of breath and exhaustion, were suddenly gone.

I then immediately bought an oxygen concentrator and installed oxygen lines in our house. She then inhaled oxygen through a nasal tube for hours every day, which alleviated her symptoms. (Phenomenon 3)

So far we have had 3 phenomena that no doctor has been able to explain to us. Neither in the coagulation outpatient clinic nor in the cardiology department of the nearby university hospital. We have asked countless well-known doctors, but no one had an explanation.

Many of the complaints she has had in the meantime match many of these illnesses.

ME/CFS

Post-VAC

Post-Covid

Fibromyalgia

hEDS

and not only these illnesses have the symptoms described. Post-traumatic stress disorder (PTSD) patients can also have such symptoms.

The solution:

To put it bluntly. Not all symptoms have disappeared, but many have.

And the solution began in January 2023.

Our daughter, now 20 years old, was lying on her bed watching TV in the afternoon. I joined her and we lay quietly next to each other. At some point, I noticed that she was breathing much faster than you normally do at rest. Unnoticed, I counted her breathing rate. It was 20 breaths per minute. Normal is 12-14 breaths per minute.

The pathological breathing pattern of acute hyperventilation was sufficiently familiar to me in terms of appearance and symptoms due to my profession. But our daughter was not agitated. She was lying completely relaxed on the bed.

Up to this point, we had countless lab results. If our daughter was really hyperventilating, then the PH value of the blood would have to be pathologically altered, or so I thought. There was nothing conspicuous in any of the findings.

As I work in the emergency services myself, I was able to borrow a patient monitor, which I took home with me. This allowed me to measure the etCO2, the end-expiratory CO2 value of the exhaled air. This is normally between 35-45 mmHG.

Our daughter's value was 28mmHG, clearly too low.

So I started to find out on the internet whether there is such a thing as chronic hyperventilation. And what I subsequently researched left me almost speechless.

In addition to what I already knew from my training, I will describe my findings below. (It's getting a bit medical now, sorry):

The respiratory rate is controlled by the respiratory center in the medulla obpongata.

The main sensor for respiration is controlled by the CO2 content in the blood.

The blood is kept at a pH value between 7.35 and 7.45 by breathing. If we breathe out too much CO2 (carbonic acid), the blood becomes alkaline, we speak of respiratory alkalosis (pH > 7.35). If we breathe too little, the blood becomes too acidic, we speak of respiratory acidosis (pH < 7.35). Respiratory=from breathing

Our body has developed a number of compensation options that enable it to influence the pH value of the blood.

The quickest way is to breathe. During sport, the body produces a lot of CO2 through muscle work. By breathing faster, the CO2 is exhaled and the pH value remains constant. If we produce less CO2 during sleep, for example, our breathing becomes calm and slow. But what happens if we breathe (slightly) faster on a permanent basis?

An adult at rest has a breathing rate of approximately 12 breaths per minute. We then inhale approx. 500 ml per breath. That is about 5-6 liters of air per minute that flows into our lungs. If you are constantly breathing faster due to chronic pain or constant mental stress, you can quickly reach a breathing rate of 20 breaths per minute. This is known as ***tachypnoea***. The depth of breathing may decrease slightly. However, with a breathing volume of 400 ml and 20 breaths per minute, this is now 8 liters of air that are inhaled per minute. Each deep sigh increases this value considerably. For an adult, this can be an additional 1-2 liters more *per sigh*.

The body exhales CO2 with every exhalation. Our blood should actually become alkaline (pH value > 7.45) due to faster breathing and the increased exhalation of CO2.

However, our body has created another way of compensating for this very situation. If this is the case, the bicarbonate buffer system comes into action.

A bicarbonate buffer is a buffer system that consists of bicarbonate and prevents the blood from becoming too alkaline or too acidic.

A blood gas analysis (BGA) can show whether the buffer system is being increasingly used. *Base Excess (BE) is* the corresponding marker here. Too *little CO2* means a *negative base excess*, too much CO2 in the blood means a positive base excess.

One way of measuring the CO2 content in exhaled air is capnometry. Since it depends on the last bit of air during exhalation, it is called the *end-expiratory ("at the end of exhalation") CO2*: **etCO2**

The etCO2 reflects the CO2 content in the blood.

The etCO2 value of a healthy person is between 35-45mmHg. If you chronically breathe too quickly, this value falls below 35mmHg, as too much CO2 is exhaled. This is referred to as respiratory alkalosis or respiratory hypocapnia.

(Our daughter's initial value was 28 mmHg.) However, this was never noticed in the laboratory findings. (pH value normal, base excess not known)

But what happens in our body when the CO2 level in our blood is (chronically) too low?

In the following I would like to look at the following body regions:

- Blood
- Headache/migraine
- Heart - tachycardia (rapid heartbeat), palpitation (palpitations), cardiac arrhythmia, tightness in the chest
- Shortness of breath
- Spine - hunchback (hyperkyphosis) and hollow back (hyperlordosis)
- Gastrointestinal tract
- Musculature (in the entire body)
- "Mysterious" mechanisms of action of drugs (phenomena1-3)

Blood:

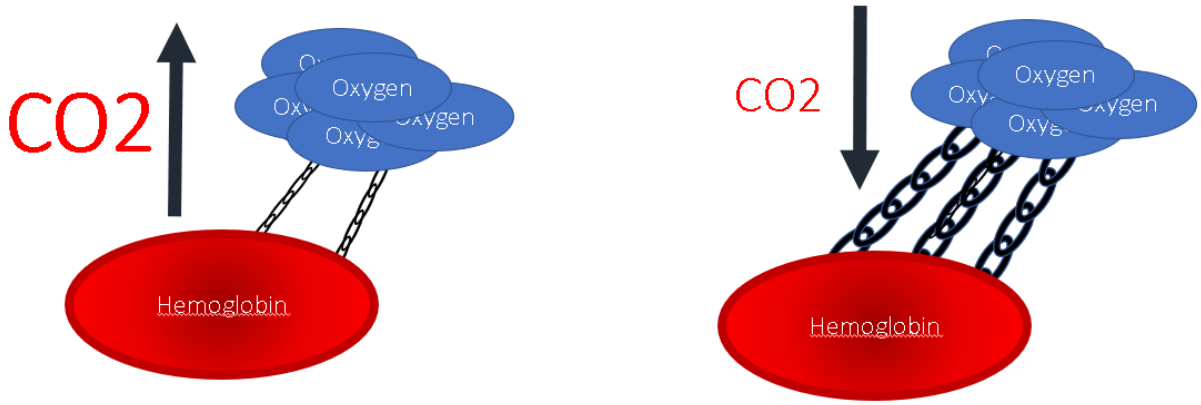
Our blood transports oxygen (O2) to our body cells and the metabolic product CO2 from the cells back to the lungs, where it is exhaled.

Haemoglobin, a protein compound, makes up around 90 percent of our red blood cells (erythrocytes). It consists largely of iron and thus gives our blood its typical red color. Haemoglobin is therefore also known as the red blood pigment.

The oxygen binds to the hemoglobin for transport through the bloodstream.

The *Bohr effect* (after the Danish physiologist Christian Bohr, 1855-1911)

Describes the reduction in the affinity (binding capacity) of oxygen to hemoglobin when the pH value falls (=increased CO2 concentration).



If the CO2 concentration falls, the affinity increases. As a result, the hemoglobin can no longer deliver oxygen to the cells as well

Head:

Hypocapnia (low CO2 level) causes a narrowing of the blood vessels in the head, a so-called cerebral vasoconstriction.

This results in reduced blood flow to the brain, cerebral hypoxia (lack of oxygen in the brain)

In addition, as described, there is the increased affinity of oxygen to hemoglobin.

The result: less blood flows through the brain. And the oxygen that is present in the blood is delivered to the brain cells more poorly due to the increased binding to the red blood cells. This leads to hypoxia (oxygen deficiency)

The result appears to be headaches or even migraines, drowsiness can also be a symptom, rapid irritability, mental tension or even panic and sleep disorders

This is because a lack of oxygen in the cells causes hypoxic pain.

Heart:

Despite 100% oxygen saturation (SpO2) throughout the body, the increased affinity of oxygen for red blood cells causes a certain degree of hypoxia (oxygen deficiency) in the cells, including the heart.

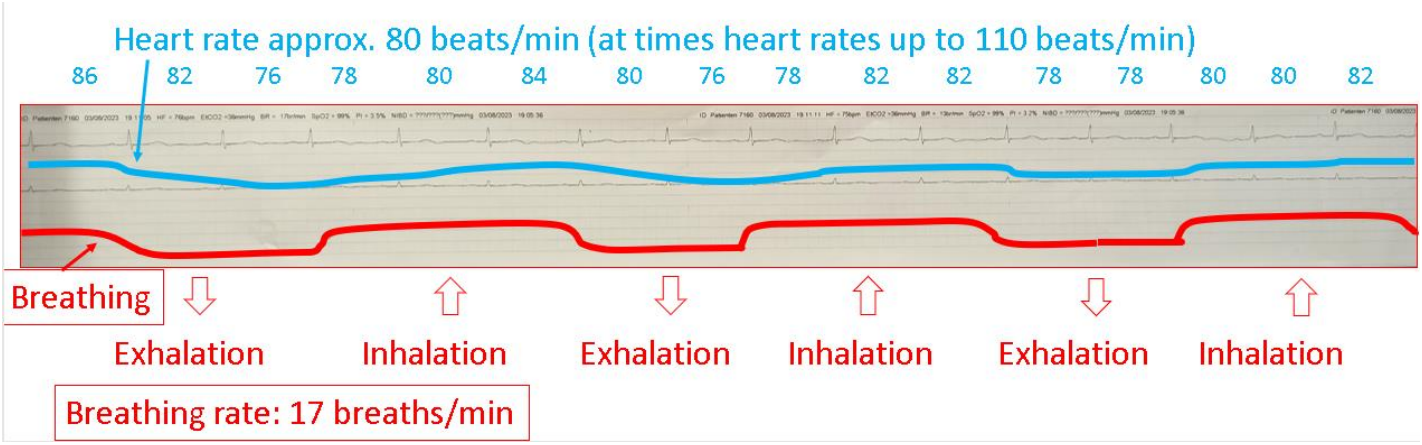
The heart reacts with an increase in heart rate, the heart becomes tachycardic. You can feel your own heartbeat (palpitation). Oxygen deficiency in the heart is manifested by a feeling of tightness, a feeling of pressure in the chest.

Some patients report that their heart is stumbling. The results of a cardiology examination were unremarkable.

In this case, it could be a physiological (natural) respiratory sinus arrhythmia.

ECG rhythm: Respiratory sinus tachyarrhythmia

When inhaling, the pressure in the thorax increases and the increased pressure on the heart increases the heart rate. When you exhale, the pressure decreases and the heart slows down again. This irregularity is felt as stumbling.



The rapid heartbeat at rest causes inner restlessness and patients breathe even faster... a vicious circle...

Despite 100% blood oxygen saturation, the partial pressure of oxygen (proportion of dissolved oxygen) in the blood can be increased by administering oxygen via an inhalation mask to such an extent that the symptoms of oxygen deficiency in the cells are noticeably reduced. (Phenomenon 3)

Breathing:

The cellular oxygen deficiency triggered by hypocapnia signals to the body that there is an acute oxygen deficiency.

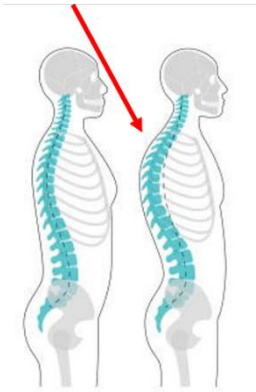
The body reacts acutely with the symptom of shortness of breath.

Those affected often sigh and have to take a deep breath.

Hunched back and hollow back:

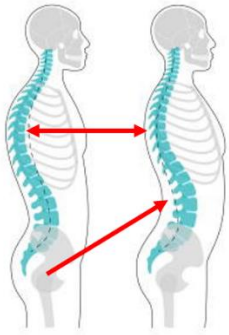
The breathing movement is nothing more than muscles contracting and relaxing again. At rest, our diaphragm is the muscle that moves most of the air. The intercostal muscles are only used when we inhale a lot and more deeply.

If a muscle is used a lot, it will be trained. And what does a trained muscle do if you don't stretch it? It shortens. If the intercostal muscles are constantly trained through increased breathing, they become "stronger" and shorten. As a result, the space between the ribs on the front of the body becomes smaller. This in turn results in a hunched back, a hyperkyphosis, on the back of the body.



The bent forward posture in the thoracic vertebrae could affect the entire spine and develop a hollow back (hyperlordosis) in the lumbar spine.

The lumbar spine presses into the abdominal cavity.



The resulting consequences in the abdomen could be compression of various vessels.

Gastrointestinal tract:

As already described, the chronically low CO2 content in the blood results in a chronic lack of oxygen at cellular level. This also applies to the gastrointestinal tract.

If the pathological change in the spine also causes blood vessels in the abdominal cavity to be constricted or partially compressed (vascular compression), this leads to circulatory disorders in the affected organs and a lack of oxygen in the cells of the organs. The transport of nutrients from the digestive organs is also impaired.

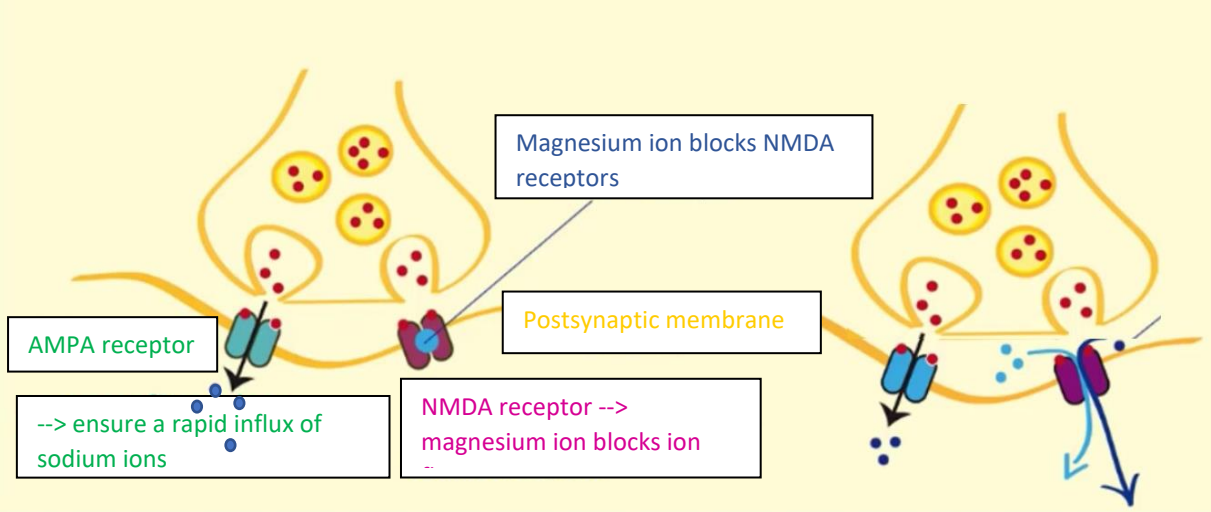
This could all be a cause of frequent nausea, but also of a possible lack of weight gain despite apparently sufficient food intake.

Another symptom appears to be indigestion in the form of constipation.

Musculature:

Muscle cells are excited by the receptors receiving a stimulus. This stimulus causes sodium to flow into the cell through the AMPA receptor, which is located in the cell membrane. By increasing the potential of the sodium in the cell, sodium and calcium flow through the NMDA receptor. This NMDA receptor is also located in the cell membrane. The CO2 deficiency (hypocapnia) changes the permeability of this cell membrane, which results in increased excitability of the muscles.

This chronically increased muscle activity is noticeable through tingling, muscle cramps and pain in the muscles.



The phenomenon 1 -cortisone:

A few years ago, we had already had a medical marathon, we made a small "attempt" with a doctor friend (out of sheer desperation...)

Our daughter was given a high dose of cortisone intravenously (into the vein) (500mg→ 250mg→ 250mg).

And the result: she was significantly less painful for a few days.

No doctor could explain this mechanism of action (and there were many, many we asked)

Another trial with a lower dose (50mg→ 25mg→ 12.5mg→ 5mg) of oral administration (tablet form) was without the desired effect.

If you now look at what cortisone does, among other things, then this could be an explanation for the pain relief.

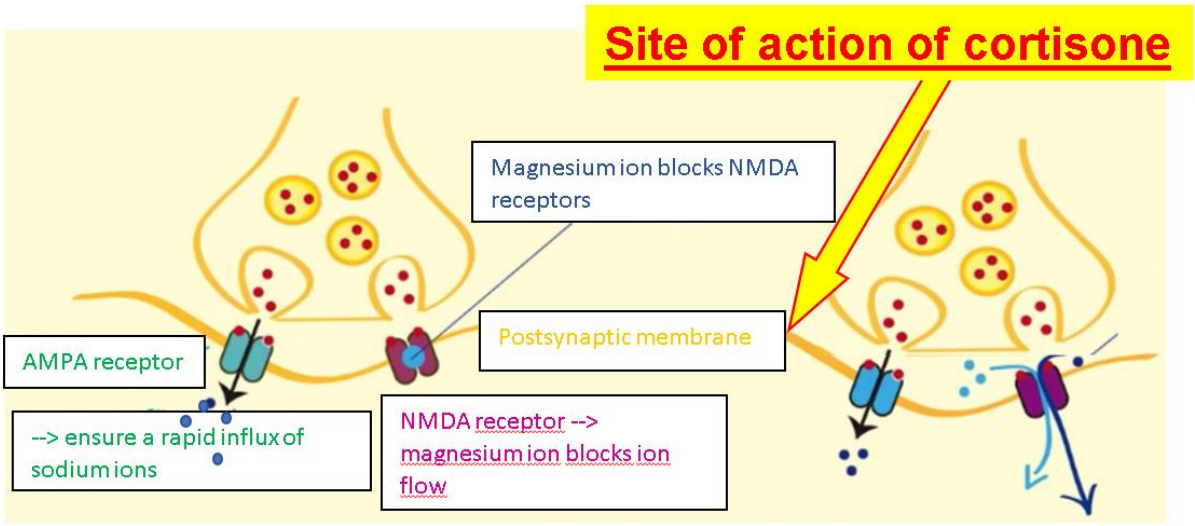
Cortisone causes (cell) membrane stabilization. The change in the permeability of the cell membrane caused by hypocapnia, and the associated excessive influx of sodium and calcium into the cell, could be stabilized again by cortisone. The result: the influx of sodium and calcium into the cell is normalized and the muscle cramps, pain and tingling are stopped.

But why did the oral administration of cortisone not have the desired effect?

The "non-genomic effect" of cortisone was probably responsible for this.

This effect occurs within seconds to minutes after administration of high glucocorticoid doses (> 200-300mg prednisolone equivalent) and probably leads to membrane stabilization or

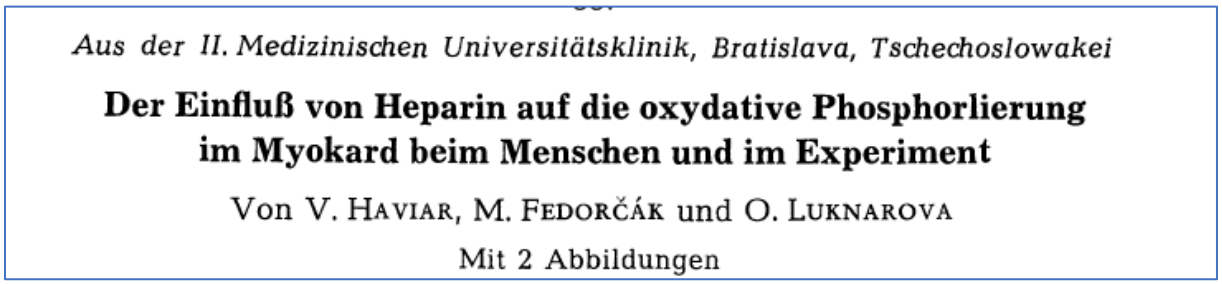
reduced cellular excitability via a non-specific physico-chemical interaction with the cell membrane (intercalation) or binding to specific membrane proteins. Rapid non-genomic mechanisms may explain acute onset effects due to high glucocorticoid doses administered in clinical emergencies.



In our "attempt" to reproduce the "cortisone effect", the dose was simply too low...

Phenomenon 2 -Clexane (heparin)

After endless research on the Internet, I came across a study from 1969 by chance:



Aus der II. Medizinischen Universitätsklinik, Bratislava, Tschechoslowakei

Der Einfluß von Heparin auf die oxydative Phosphorlierung im Myokard beim Menschen und im Experiment

Von V. Haviar, M. Fedorčák und O. Luknarova

Mit 2 Abbildungen

"from the 2nd University Medical Clinic, Bratislava, Czechoslovakia

The influence of heparin on oxidative phosphorylation in the myocardium in humans and in experiments.

By V. Haviar, M. Fedorcak and O. Luknarova"

Quote: "In humans, the ADP/O ratio was found to increase after heparin administration in our experimental conditions. The average quotient of ADP/O was 1.21 in non-heparinized patients and 1.61 in patients after administration of heparin. Therefore, in humans, the ADP/O quotient increased by more than 30% compared to controls"

ADP/O quotient = ratio of how well the phosphate atom and oxygen bind.

We remember: lack of oxygen causes pain --> more oxygen in the cells --> less pain

Muscle work requires energy.

Adenosine triphosphate, or ATP for short, is a chemical molecule that provides energy in every cell of a living organism. This energy enables all work processes such as locomotion or material transport. An ATP molecule contains three phosphate atoms.

During oxidative phosphorylation, ADP (adenosine diphosphate) is converted into ATP. (cellular respiration)

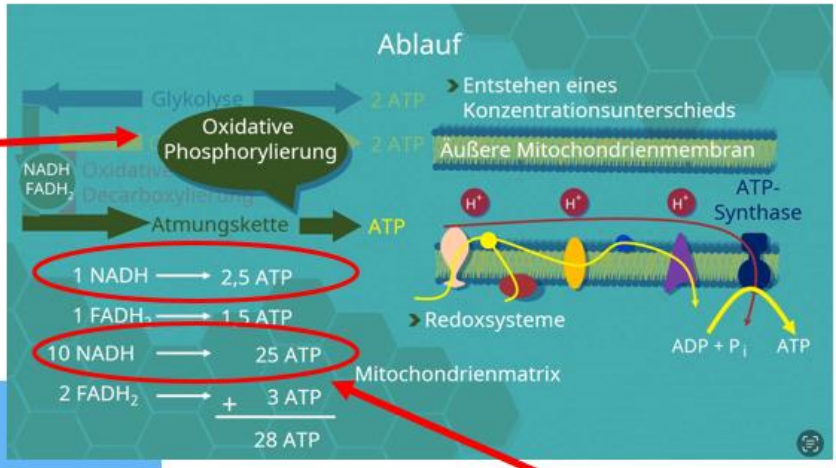
Using the example of a heart muscle cell, the study described that in patients who were administered heparin, the available blood oxygen is oxidatively phosphorylated up to 30% better in the cell.

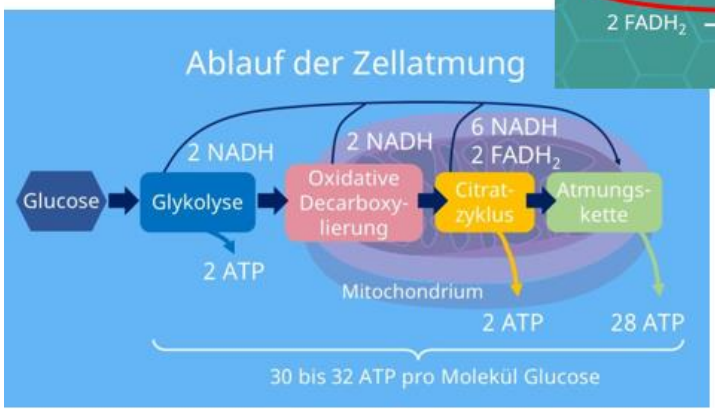
If one assumes that the mechanism of action, as shown in the study, is not only transferable to the heart muscle, but to all (muscle) cells, then this would explain why heparin worked against pain.

If the pain was caused by the lack of oxygen in the cells due to low CO2 levels (CO2 deficiency→ increased oxygen affinity at the red blood cells→ cellular oxygen deficiency→ hypoxic pain), then the conclusion could be that the effect of heparin compensates for the lack of oxygen and thus the hypoxic pain is absent. The sublimation of NADH could possibly have the same effect

Therapeutic approach:

According to the study, heparin increases Oxidative phosphorylation →increases the supply of ATP →Cellular respiration





Perhaps the sublimation of NADHhas the same effect and equally increasesthe supply of ATP →Cellular respiration

Therapeutic approach: NADH

According to the study, heparin increases oxidative phosphorylation→ increases the supply of ATP→ cell respiration

perhaps the sublimation of NADH has the same effect and equally increases

the provision of ATP→ Cellular respiration

Conclusion

The many complaints seem to have a common origin. Chronic hyperventilation. This can be triggered by various causes. Whether infections, chronic pain due to endometriosis, compression syndromes, micro-injuries due to connective tissue weakness or even long-term psychological stress. Post-traumatic stress disorders could also be a cause.

Over the years, all these different underlying illnesses often develop common features: Their subsequent symptoms such as migraines, palpitations, gastrointestinal complaints, pain, sleep disorders, exhaustion and lack of energy.

And possibly triggered by chronic hyperventilation.

The aim must be to get the respiratory center used to a normal CO2 value again. This is approximately 40mmHg (etCO2) in the exhaled air. The aim of respiratory therapy must be to normalize the respiratory rate and the respiratory volume per minute.

As the body's ability to compensate is very good, it takes a long time for everything to normalize.

One therapeutic approach could be respiratory therapy.

The Buteyko method seems to me to have a good approach here.

Konstantin Pavlovich Buteyko (1923-2003) was a Russian doctor and scientist.

The Buteyko method is about increasing the CO2 content in the exhaled air again and thus eliminating the symptoms.

We have now looked for a respiratory therapist who works according to the Buteyko method. And we have found one. It took about three months before we could see any success. The breathing rate has normalized. A new measurement of the CO2 content of the exhaled air (etCO2) showed a value of 36mmHg.

And what has changed in terms of the symptoms?

The shortness of breath is gone

The pain in the arms and legs is significantly less

The muscle twitching and muscle cramps are less

The headaches are significantly less

The cardiac arrhythmia and palpitations can no longer be felt

Postural tachycardia syndrome (POTS) has disappeared

The permanent physical exhaustion has disappeared. Our daughter can lead a normal life in line with her age. (Her underlying disease hEDS is not yet curable). She works full-time and can also be active in her free time.

Important: I BELIEVE THAT CHRONIC HYPERVENTILATION CAN ONLY BE DIAGNOSTICIZED BY MEASURING THE END EXPIRATORY LEVEL. WHEN MEASURING THE BLOOD VALUES, THE COMPENSATION POSSIBILITIES OF THE BODY ARE NOT MEANINGFUL ENOUGH. AT LEAST NOBODY RECOGNIZED IT IN OUR DAUGHTER.

I hope that my report will encourage other sufferers and perhaps find their way to those who are looking for a medical solution. Perhaps chronic hyperventilation is the key to alleviating many complaints.

https://www.healthrising.org/blog/2024/12/24/better-breathing-for-better-health-solve-mes-inspiratory-breathing-study-scores/

And if you read through this report, then this form of therapy could not only be muscle training, but could also influence the CO2 concentration in the body in the same way as Buteyko breathing.

I thank everyone for reading and wish you all the best.

Best regards

Norbert Müller