

What causes a heart attack?

Let's leave the coronary arteries and say goodbye to the classical approach – to the theory that acute blockage of the coronaries is the crucial cause of a heart attack. What next?

When searching for alternative explanations one must try to find characteristic factors that differentiate heart attack patients from the rest of the population. Such factors do exist. One such important factor is a chronic reduction in the control function of the PNS in heart attack patients. The PNS has been described in detail in Section 2 “What is the relationship between stress and heart attacks”.

Defective heart control due to blockade of the PNS

What does this mean – a defect in the control function of the PNS? The PNS activity can be measured. This will be discussed in detail in Section 11. To put it briefly: every time a person breathes in, the pulse quickens, and when they breathe out it slows. This constant variation in the rate of the pulse in harmony with breathing is controlled exclusively by the PNS. Examining the difference between the highest frequency when breathing in and the lowest when breathing out is a good measurement of the influence of the PNS on the heart.

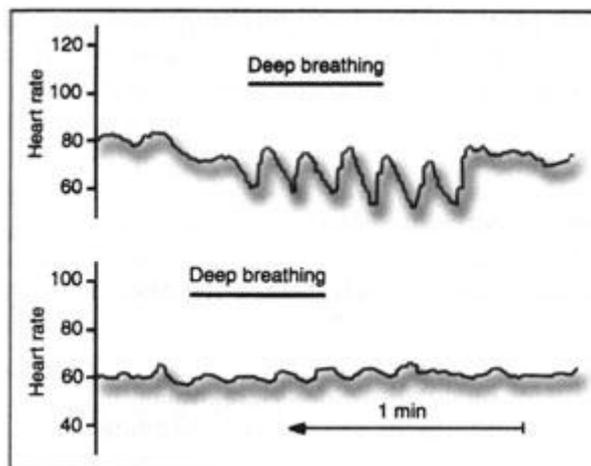


Fig. 1: Defect in the “PNS” expressed by an almost “rigid pulse rate”

This simple illustration provides surprising information. The upper curve is that of a healthy person, the lower that of a person with coronary heart disease. The upper curve shows a typically normal wave-like pattern during 6 deep breaths. **The lower curve shows an almost “rigid pulse” (33). The above graph is a very good example of the defective PNS function in a heart patient.**

Using various mathematical methods, a whole series of studies has shown that **this reduction in PNS heart control is a typical characteristic of heart patients.** There is a direct connection between this defect and heart seizure, “angina pectoris”. This is clearly demonstrated in the following graphs.

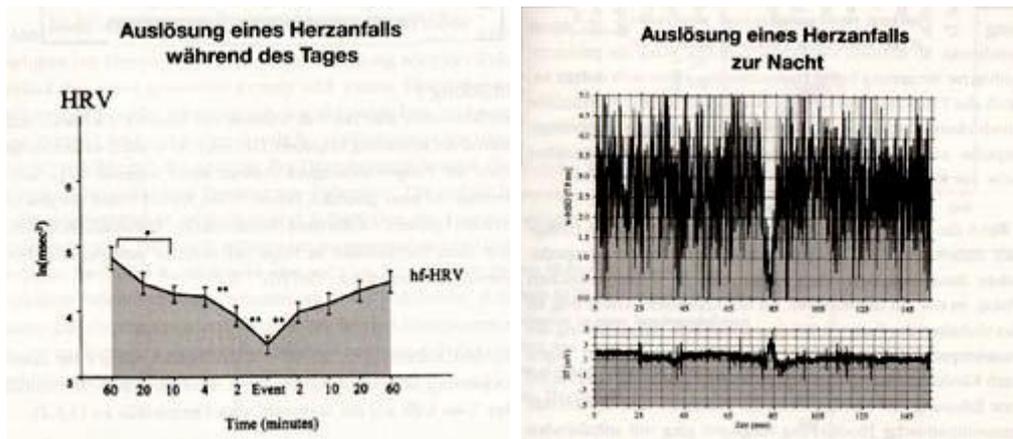


Fig. 2: Heart seizure: during the day (left), at night (right) due to blockade of the “PNS” heart control

In the **left graph** the “hf-HRV” (high frequency heart rate variability) represents the actual PNS activity level; “**event**” represents a heart seizure in normal every day life. One can see that PNS activity decreases continually during the hour before the seizure. In the last 4 minutes before the seizure, the reduction becomes more acute. At the lowest level of PNS the “event” takes place (34).

The graph on the right shows a seizure during sleep at night. The sudden drop in the PNS impulses on the heart, causing “angina pectoris”, could not be displayed more dramatically (35).

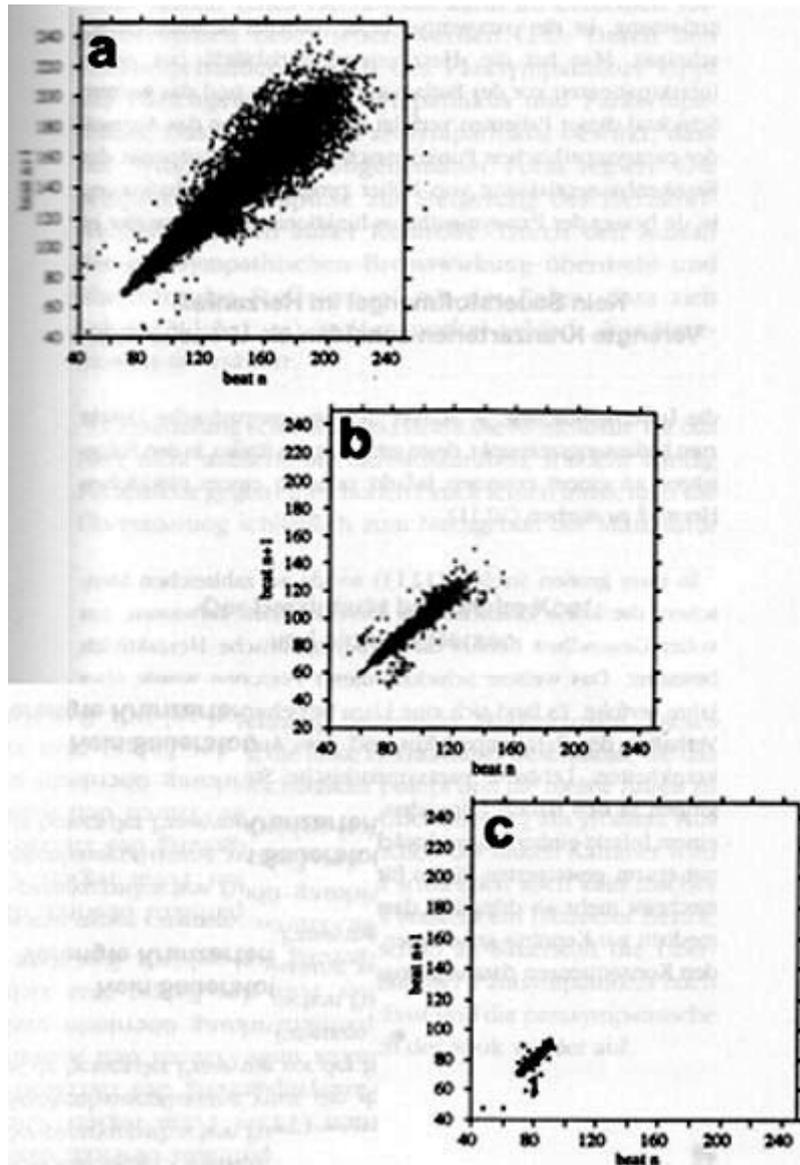


Fig. 3: Healthy, weak, and blocked “PNS” activity

In this figure, PNS activity is shown as a “scatter plot”. The charts show: a) a healthy 49-year-old female b) a 62-year-old male heart patient and c) the same patient as in b) during a heart seizure. The breadth of this structure, which normally takes the shape of a baseball bat, corresponds to the strength of the actual PNS activity. This is a good illustration of **PNS weakness and blockade in heart patients compared to healthy persons** (Sroka and Peimann, 1995).

Based on my own research and on a dozen international studies, the relationship between PNS reduction and heart seizure is presented in my comprehensive article “**On the genesis of myocardial ischemia**” (17). According to these studies, about $\frac{3}{4}$ of all heart seizures are triggered in this way.

Loss of inner balance

PNS weakness does not appear overnight. As already mentioned in Section 2, several factors must occur simultaneously in order to chronically weaken the PNS.

An example: Male, over 60, slightly overweight, little movement, living alone with few social contacts, suffering from chronic stress with a negative psychological tendency – this constellation could have a lasting negative effect on the PNS system over a period of time. If, in addition, repeated hurtful arguments with a close neighbor, who our patient finds it difficult to stand up to, are added to this burden, sapping at the man's strength, then this chronic weakness of the PNS system will eventually turn into a total blockade.

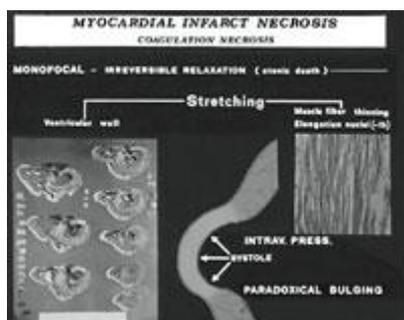
As long as the PNS heart control remains intact, then no acute physical effort or psychological tension can harm the heart muscle. An intact PNS takes the edge off any great increase in sympathetic (SNS) activity. A total block in PNS activity means there is a risk that the SNS is gets out of control. **Uninhibited SNS impulses on the heart, when the PNS brake is blocked, present a constellation, which results in heart seizure and heart attack (17).**

From defective control to a heart attack

The effects of this constellation on heart metabolism can be studied well in experiments by applying adrenaline, the prime substance in SNS activity, to the isolated, well-supplied and vital heart without any PNS protection. This leads to immense acidity (“acidosis”) of the heart muscle (32,33,34), whereby more recent work on this subject has been able to explain precisely why this occurs (35).

Acidity is dangerous to the heart muscle. When one oversteps one's limit while practicing some kind of sport, when jogging for example, then acidity develops in the skeletal muscles and the legs tire. The legs can't carry you. Acidity blocks the muscles in the legs, as well as in the heart. The heart muscle loses the strength to contract. Acidity weakens and lames the heart muscle. **In the left chamber of the heart, which is under the most tension and does more work than any of the other cavities of the heart, acidity is at its greatest. Here, blocked, weakened and lame areas develop in the walls of the heart.**

One can stop jogging, but one cannot simply stop the heart beating to allow it to recover from acidity. The heart keeps pumping at high pressure to supply the body with fresh blood. The weakened, lame areas of the left heart chamber are naturally subjected to this high pressure.



It often happens that such areas in the heart wall bulge out under the high inner pressure and overexpand, as shown in this sketch by Baroldi (“paradoxical bulging”) (16). As an example of overexpansion, just imagine a broad rubber band. Overexpansion increases the tension, the pressure on the tissue. When the pressure in the tissue is greater than the pressure of the pulsing arterial blood, then the blood supply ceases. No fresh arterial blood can enter and the used venous

blood is pressed out of this area. **A bloodless (“ischemic”) area then develops.** Please note that such a bloodless area does not arise as a result of circulation problems due to blocked coronary arteries. **This bloodless area is the result of an acute overexpansion of parts of the heart muscle, ultimately the result of a defect in PNS heart control.**

Such bloodless areas often cause pain and are the basis of attacks of angina pectoris. When the PNS blockade is released after several minutes, the scare is not yet over. The tension is released and the arterial blood flows into this area of the heart muscle. The renewed blood flow in the previously lame area is not solely beneficial; the oxygen supply resumes,

but this puts the area in question under great stress. This is officially called “**refilling trauma**”, as many so-called “oxygen radicals” are produced, which have a strong destructive influence on the tissue of the heart muscle. The “free radicals” are discussed in Section 6 on “oxidative stress”.

It is not rare for a lame muscle area to remain dysfunctional or lame for hours or days following acidity and refilling trauma. This area is referred to as “**stunned**”. Such an area is obviously particularly vulnerable to further injury. It does not require the unpleasant neighbor to reappear and act threateningly; a change in the weather from warm to cold (warmth stimulates the PNS, cold downregulates the PNS) is enough to trigger a new seizure.

With every repeated lack of blood, the acidity in the tissue grows; after every refilling trauma the destructive attacks of the radicals increase. When finally, the cell walls start to give way and begin to disintegrate, then the so-called “**point of no return**” is reached. The damage to the cells cannot be repaired and the whole muscle area dies. This is a heart attack.

This is roughly the way a defective PNS heart control results in a heart attack. The circulation in the coronary arteries plays no causal role in this scenario. The heart attack does, however, have consequences for the coronary arteries and their stenoses. The blood in the coronary artery leading to the area of the heart attack congests and often leads to tears in the narrowed passages. These tears lead to thromboses in about 50% of heart attacks. The larger the area of infarction, the more often this occurs. The longer a patient survives the acute stage of a heart attack, the more frequently thromboses develop.

But the process is the same in 50% of heart attacks with thromboses and in 50% of the heart attacks without thromboses. During a heart attack, the heart muscles die in an acid, lame and overextended condition, in a bloodless area, with or without thrombosis.

Many roads lead to Rome

Many roads lead to Rome. Vessel factors explain the development of a small percentage of heart attacks. It is unlikely that all other heart attacks can be explained by a PNS control defect. Another known cause is the “Tako-Tsubo syndrome”, in which the heart remains in “hypercontraction”, i.e. in a total cramp, resulting in heart seizure and heart attack. In this type of heart attack, the left heart chamber resembles the shape of a Japanese octopus trap (“Tako-Tsubo”), which explains the name. These patients usually suffer from high emotional excitability; the PNS and the coronary arteries do not appear to be involved. The origin and frequency of the syndrome is unclear, as it is often overlooked as a possible cause. There are probably even other roads to Rome; perhaps a diet lacking in essential nutrients for instance, or other factors that cause heart attacks, which nobody has yet thought of and which wait to be discovered.

To obtain more clarity, it is urgent necessary that alternative possibilities are considered and the fixation on the coronary arteries is overcome. **The blinkers must be removed and the view broadened to take in other possible causes of heart attacks.** Ultimately, more clarity on this subject would have important consequences for everyday clinical practice and benefit patients by improving their chances of healing.