The Riddle’s Solution

A blood vessel that is blocked to a large extent will obstruct the flood flow. A slowly closing narrowing will allow less and less blood to pass. This is known as “critical stenosis”, but in fact, this is not as critical as conventional medicine claims. Heart seizure and heart attacks are by no means an inevitable result. Why is this? The solution to the riddle is that the three coronary arteries are not isolated from one-another; they are not “end arteries” that are not connected with one another, but part of an extensive network of blood vessels that exist in all parts of the heart muscle. Furthermore, the body is capable of helping itself should there be a blockage of the blood flow or in the oxygen supply and it is able to extend this network in a substantial way.

The natural vessel network in the heart muscle

With the aid of a special technique, Baroldi and his coworkers refilled the coronary arteries of numerous corpses. The artificial substance used remains liquid at room temperature. This liquid mass was pumped into the vessel system of the heart with the same rhythmic pressure at which the heart functions when a person is alive. They then warmed the artificial blood to a temperature of 50°C, so that it hardened to form an anatomic model of the vessels. The muscle tissue was then removed in an acid bath. The result of all this careful work disclosed the wonders of nature: “myriads”, “mega” or “giga” numbers of fine vessel branches could be seen in the whole heart muscle.

The following pictures show models of the vessel system of the heart that are based on this technique. Using this method, Baroldi was able to show that the coronary arteries are by no means “end arteries”. In the normal heart muscle there are numerous connecting vessels (“anatomoses” as they are called in medical language) between the three large coronary arteries and also between the individual branches of one and the same artery. These communication paths between the individual trunks, branches and twigs of the coronary “vessel tree” exist from birth and develop in harmony with the growth of the organism.

Fig. 1: The blood vessel network in a healthy heart; from the front, (left) and from behind (right)
The extensive network of connections between the coronary vessels in a healthy human heart is nicely shown in the above photos (16). To compare, it is good to take a look at the sketches in the section “Classical Concept”, with which nearly every heart patient is acquainted and which are shown to patients again and again during their rehabilitation. Nowadays, the whole world has it drummed into them that the coronary arteries are “end arteries”, meaning that they are isolated from one-another – this is obviously incorrect. We are cheated out of this wealth of vessels.

Now, we come to the self-help system in the body. The way in which the natural connections between coronary arteries expand, if necessary, their network is clearly shown in the following figures (16).

![Fig. 2: Extended network of links between the coronary arteries](image)

In this figure on the left, a thickening of the heart wall requires an increased blood supply; the figure on the right shows the heart of a patient with pronounced chronic anemia. The “anastomoses”, the links between the large vessels, react by growing, by increasing in diameter and by extending. New vessels do not grow, but the existing network is extended and expanded.

“Collaterals”: the life-saving natural bypass system

Dramatic growth is seen in the connecting links when the blood flow in the coronary arteries is obstructed by severe stenosis.
Such a severe stenosis (arrow) in the right coronary artery can be seen at the left border of the above figure. Nevertheless, the blood supply to the heart is obviously unaffected as the right coronary artery is also well filled with blood beyond the stenosis, far into the periphery. The blood supply to the whole of the heart muscle is perfect. There is no defect in the sense of scarring or of an acute heart attack (16).
Figure 4 shows why this is so (16). The expanded and extended bridges between the great arteries and between their branches and twigs build an extensive diversion around the blocked part of the vessel. These extensions are called “collaterals”. The expansion of the collateral network is stimulated by the slow or sudden increase in coronary artery stenoses. **This natural bypass system develops proportionately to the severity of the stenosis of the vessel, so that the blood supply to the heart muscle is at no point damaged by the development of arteriosclerotic stenoses and blockages.**

In this figure we see what Baroldi called “satellite collaterals“, a bundle of fine vessels that make a direct diversion around a blocked vessel (16).

**Fig. 5: A bundle of collaterals create a diversion around a blocked vessel (arrow).**

**Blood flows through the collaterals around the blockage**, supplying the muscle and finally finding its way back into the blocked artery. The blocked vessel is supplied with blood by the pathways emerging from all sides of the tissues; the blood fills the vessel from beyond the stenosis and ascends to the narrowing. This explains why the right coronary artery in Fig 3. is so splendidly full of blood beyond the severe stenosis, far into the finest branches.

Baroldi is not alone with his theory. Since the 17th century (Richard Lower: “Tractatus de corde”, 1669, Amsterdam), this network of fine vessels in the human heart muscle has often been described by pathologists. 50 years ago, several researchers experimented with techniques similar to those of Baroldi, trying to display the blood supply to the heart muscle by way of anatomic models.
William Fulton (19), from Scotland, was also able to show the existence of the extensive network of blood vessels in the human heart. In Fig. 6, one sees the numerous links between the two coronary arteries in a normal heart, and below, the large network of extended “collaterals” as a result of a blockage in the vessel (23). At least as much blood flows through these collaterals as would normally flow through the unblocked normal artery. The heart muscle therefore does not suffer from a blood deficiency due to the coronary stenosis.

Collaterals grow at a great pace

How quickly do collaterals grow? If one were cruel enough to completely bind up the left coronary artery of a dog, there would be an acute shortage of blood in the heart muscle, despite the existence of the “anastomotic” network. A heart attack would occur. However, if one were to proceed differently and only induce a critical stenosis, i.e. incomplete blockage of the left coronary artery, then at first nothing would happen. In an experimental study, such a critical stenosis of the coronary artery was changed into a complete blockage after 7 days. This remained without consequences – neither a heart attack nor threatening rhythm problems resulted. And it has been shown that the growth of collaterals is at such an advanced stage after 1 week that even a total blockage of the coronary artery can no longer seriously endanger the heart muscle (20). In a very similar study, within 4 days the rapidly growing interconnecting vessels had extended sufficiently to protect the heart (21). In the past years, modern techniques have made it become possible to visually display the early and rapid growth of collaterals (22).

There are individual differences in the capacity and speed with which the collateral system expands. Arteriosclerosis is a chronic, and as a rule, slowly progressing disease. The arteriosclerotic alterations in the vessel wall develop over months and years. It is therefore to be expected that the natural development of the collateral bypass system generally keeps pace with the development of the coronary stenoses.

A short film on this website shows an excerpt from a typical “angio”, a coronary angiogram, which most heart patients will be acquainted with. During this examination a catheter is entered into the body, usually through the groin, and is pushed upwards through the aorta to the heart. Then a contrast medium, a liquid substance visible in an x-ray, is injected under pressure into the left, then into the right coronary artery.
In the “heart catheter” film the right coronary artery is injected and a severe stenosis is visible in the middle section. To make the most important points in this quick procedure clear to you I have included a series of chronological individual photos so that you can study the procedure at leisure. The stenosis is wonderfully displayed; the collaterals can hardly be seen. However, according to this series of photos there is no doubt that the collaterals do exist and that they function effectively. From this it becomes clear that the heart suffers no damage due to the stenosis.

A heart catheter examination omits decisive information: the fine network of collaterals, vessels that enable blood to bypass the stenosis, is not encompassed with this method. This results in a serious misinterpretation with regard to the significance of coronary artery stenoses.

The heart catheter protocol given to the patient in the film after the angiogram has been completed, could look like this:

This natural network of interconnecting vessels between the two great arteries and between the branches and twigs of one and the same artery, this network, in which collaterals develop when required, is not included in the protocol. The existence of the collaterals is totally overlooked. The coronary arteries appear, as traditionally accepted, as “end arteries”. The severe stenosis therefore appears to be a risk factor for heart attacks. Our patient will be encouraged to undergo further catheterization to widen the stenosis with a balloon and a stent will be inserted.
Such an intervention allows the blood to again flow unobstructed through the right coronary artery; the collaterals will then degenerate and disappear as quickly as they had developed. If an “in-stent” thrombosis now develops, a blood clot within the stent, which is not rare when “drug-eluting stents” are used, a heart attack occurs. An acute blockage of a coronary artery within a stent, without any protection by collaterals, is fatal. To minimize this risk, patients who have had drug-eluting stents inserted must take two blood thinners for one year after the intervention. However, this increases the risk of bleeding, either in the stomach or in the brain after a fall. Would it not have made much more sense to accept the existence and effectiveness of the collaterals, to trust the self-healing capacities of nature and not to touch the stenosis?

The angiogram usually convinces the patient. Without knowledge of the collaterals, the pictures of severe stenoses are of course very worrying. Here are three typical photos. However, when studying your own angiogram, do not only concentrate on the stenoses, but note that the artery is full of blood beyond the stenosis, and the circulation in the heart muscle is not disturbed. The more impressive the angiogram, the greater it proves the effectiveness of the collaterals and the inefficiency of the stenosis. So please, remain calm!

Fig. 7: Prominent stenoses with pronounced blood supply beyond the stenosis.

Conventional medicine does of course know of the existence of the “anastomoses” and the “collaterals”. For many decades it was thought that anastomoses either did not exist or they did not function sufficiently. Generations of practitioners of conventional medicine denied that collateral circulation could balance or even compensate for the effects of critical stenosis, or even a complete vessel blockage. In exceptional cases this could be possible, but not as a rule. The coronary arteries were understood to be “end arteries”.
Crucial information neglected by catheterization

When evaluating the blood flow to the heart, conventional medicine bases its findings solely on what is shown in the “angio”, the vessel models made by the pathologists in earlier days are outdated. The coronary angiogram distinctly portrays the great coronary arteries that surround the heart and the alterations to the vessel walls, stenoses and blockages. The fine network of interconnecting vessels within the heart walls is largely omitted by the x-ray contrast medium. The many fine vessels with a diameter of not much more than 0.1 mm are not shown. This technique only shows collaterals with larger diameters. Another reason that the fine vessel network is not displayed in the angiogram is that the contrast medium is injected in bursts and quickly mixes with the blood and is thinned down. And finally, as normally the coronary arteries are only filled selectively, one at a time, either from the left or the right, any flow from the other side is not shown at all.

The coronary angiogram does not give precise information on the extent of the network of blood vessels in the heart muscle and in particular the extent to which the collaterals bypass stenoses. The assessment “present” or “absent” with regard to the collaterals, based on the angiogram, has little or no meaning and is very often irrelevant.

Fig. 8: Different forms of collaterals; fine network (left), roughly enlarged collateral (right), in similar stenoses of the coronary arteries.

Fig. 8 shows two anatomic models made by Baroldi (16). The photos are unfortunately not very clear. He describes comparably severe stenoses in the left coronary artery, which are indicated by two bars. On the left we find a wealthy, dense network of fine collateral vessels. In contrast, on the right we see a row of pronouncedly enlarged and corkscrew-like extended collateral vessels. These enlarged vessels would also be seen in an angiogram, so that this patient is said to be well protected by good development of the collaterals. In the other case, where the angiogram does not display the network, one would assume that this patient has no collaterals worth speaking of. This has a serious impact, as this patient would also be advised to have a stent inserted or a bypass operation, although the stenosis does not endanger the heart due to the extensive development of fine collaterals.
Greatly widened collaterals are found especially in patients with several severe coronary artery stenoses, or after a heart attack. This is generally more often the case in older patients. In young heart attack patients, fine vessels are more frequently found (16). The current idea that heart attacks are so severe and threatening in young patients because these have hardly any collaterals is unsupportable. **Because these vessels cannot be seen in the angiogram does not mean they do not exist.**

Current research into “anastomoses” clearly shows that “angio” is inaccurate, even poor when reporting on the collaterals (23). But like Baroldi, and the others who have tried for decades, these experts also find no acceptance of their results. Their voices are ignored in the daily routine

**An empty magic formula**

During the past years, however, there has been a change of mind in cardiological science with regard to “anastomoses” and “collaterals”. Today, the patients’ complaints are taken as a guideline. Meanwhile, all stenoses of the coronary arteries, even severe ones, in patients without complaints, are regarded as having an adequate, compensatory collateral system. This is a large step towards the acceptance of collaterals. If a patient has symptoms or complaints, for instance, tightening in the chest when climbing the stairs, it is still assumed that this is caused by a stenosis. It is no longer insisted that there is a severe stenosis, but in the case of complaints, a moderate narrowing of the vessel may also be the cause. Nowadays, the magic formula is “symptomatic stenosis”.

This view is untenable. As long as the patient in our short film is free from complaint, the effectiveness of the collaterals is accepted. If the patient has complaints, the stenosis is declared to be a severe risk of heart attack; although there is no doubt that the stenosis does not affect the blood supply to the heart muscle. As a large proportion of people in industrial countries have stenoses of the coronary arteries, in patients with heart complaints one will nearly always find a stenosis that is then stamped as the culprit. The concept of “symptomatic stenoses” developed because conventional medicine automatically links every heart seizure exclusively to vessel factors. **It would be better to accept that most heart complaints and attacks cannot be explained by the classical vessel theory; hence one should search for other causes.**

**A stenosis and a heart attack are two very different things**
The development of collaterals is a result of severe coronary artery stenoses. The development of a heart attack is not related to this. One last picture from Baroldi’s work to illustrate this (Fig. 9). In this anatomic model of the vessels one sees a break in the right coronary artery just after it has branched off from the aorta, marked by a red arrow above left. This is complete blockage of this artery. This whole area of the heart supplied by the right coronary artery has a good collateral network; the circulation is good and intact. The area circled at the bottom of the picture is almost free of vessels. This is the scar from the heart attack. This heart attack has affected the area served by the descending branch of the left coronary artery, which makes a wonderful downward swing. This coronary artery is free of critical stenoses or blockages in its whole course (16).

Why have I taken all this trouble to clarify these facts? Every serious coronary artery stenosis possesses its natural “bypass”, or to be more exact, its many natural bypasses. This gift of nature protects the heart muscle against restrictions in the blood supply. If one trusts this self-healing process, one should as a patient be freed from much of the fear experienced when told one has one or more severe coronary stenoses.