

Acute blockage

When a coronary stenosis tears apart, a thrombosis develops to close the injury. **The prevailing concept is that an acute blockage of a coronary artery due to a “thrombosis”, a blood clot, is the main cause of heart attack.** This generalized view must be vigorously contested. During autopsies, striking observations have been made. Again, it was Baroldi (16) who put forward the most well-founded and decisive criticism on this subject.

Objective Data and Doubts

Doubt: Complete acute blockage of a coronary artery is found in only about 50% of heart attacks. Acute thrombosis, a blood clot that blocks an artery, can therefore only be the cause of every second heart attack. This, of course, limits the significance of the classical approach.

Doubt: The greater the size of the heart attack, the more frequently thrombosis is found. If acute thrombotic blockage of a vessel is a constant cause of heart attacks, then such blockages should cause attacks of varying severity. There are not only severe heart attacks, but also more moderate ones. A clear relationship between the size of the infarction and the occurrence of thrombosis contradicts the assumption that a coronary thrombosis is a primary event in the development of a heart attack. On the contrary: the greater the area affected by the heart attack, the greater the blood congestion in the coronary artery supplying this area. The greater the congestion, the greater the tendency of the vessel wall to injury and the more frequent the formation of blood clots. This supports the idea that blood clots are a secondary event, resulting from a heart attack.

Doubt: The longer a patient had survived following an acute heart attack, the more frequently thromboses are discovered during autopsies. A certain proportion of thromboses therefore develop after the event. This also contradicts the classical theory and supports the idea that, in general, blood clots develop as a result of heart attacks. The longer the interval after an attack, the longer the blood congests in the vessel and the greater the risk of thrombosis.

These connections strongly support the idea that acute thrombotic closure of a coronary artery is normally a secondary effect that is caused and triggered by a heart attack. First the heart attack, then the thrombosis, particularly in the case of severe heart attacks, and this is more frequent, the longer a patient survives following a heart attack.

Accepting thrombosis to be a secondary event means the end of the classical theory in its all-encompassing validity. Thrombosis as a secondary event implies that heart attacks develop independent of the coronary arteries. New explanations must therefore be sought.

Pathological studies exist in which in over 90% of the cases, an acute thrombosis was found in patients who had had heart attacks. As a rule, these studies are structured in such a way that usually only heart attacks of a certain size are included, that is to say only severe attacks, which are associated with higher rate of thrombosis. **In a very thorough long-term study carried out in Heidelberg, Germany, over a period of 10 years at the beginning of the 70's, in over 1000 autopsies a thrombosis rate of 20% was found after heart attacks (24).** A study carried out in 1998, using modern methods, showed a quota of **49%** (25); Baroldi (16) reported on 200 patients and found a quota of **41%**. If one, as I do, assumes a rate of about 50% of thrombotic vessel closure in patients with heart attacks, this figure is certainly not too low.

Catheterization is misleading

Conventional medicine has largely ignored the view of the pathologists on this subject, now relying fully on the information from the heart catheter examination and the “angio”. Heart catheter examinations carried out immediately after a heart attack almost always shows a complete blockage of the vessel leading to the area of the heart attack. This is exactly what conventional theory demands. However, when one takes a closer look, one finds that these closures have a large tendency to disappear again after a short time. Here are a few statistics: in severe heart attacks acute blockage was found in 87% of the cases in the first 4 hours after a heart attack. Between 6 and 12 hours after the attack the figure was only 68% (26). The body requires much more than a few hours to dissolve a thrombosis. In addition, heart attack patients are obviously very much restricted in their body’s capacity to dissolve a blood clot.

There can be no doubt that a large proportion of vessel blockages found by angiography during the acute stage of a heart attack are “spasms” (vessel cramps). There is always the possibility of vessel cramps during catheterization due to the pressure of contrast medium injection. This happens far more often when the blood in the artery leading to the area of the heart attack is congested and there is already a certain tension in the walls of the vessel. **A vessel cramp (or spasm) is not a “thrombosis”, not a blood clot.** A spasm releases after a short period. A spasm does not trigger a heart attack, except in rare exceptions, which are disputed among scientists.

In the case of moderate heart attacks, the figures present a different picture. In a representative study, the figures on complete blockages found in the coronary angiogram carried out on patients following a heart attack were as follows: during the first 24 hours **26%**, between 1 and 3 days **37%**, and after 7 days **42%** (27). These figures show that acute thrombosis cannot be the cause of a heart attack. 26% is not sufficient to declare an acute blockage as the general cause. The correspondence between the frequency in thromboses and the increasing time interval suggests that the thromboses are an after-effect of heart attacks.

Danger due to thrombosis?



Does thrombosis have dangerous consequences when it is an after-effect? What risk does a complete closure of the coronary artery pose when it is a subsequent, secondary event? Vessel blockages caused by thromboses usually only develop as the result of an injury in a severe coronary stenosis that is generally circumvented by many collaterals. Nowadays, one would say “So what?” when such a stenosis becomes blocked. The small amount of blood passing through such a narrowing is nothing in comparison to the large amount that finds its way through the collaterals surrounding the stenosis (28). One could compare the situation with a highway: when a 3-lane highway with a lot of traffic is suddenly reduced to one lane, due to work on a bridge for instance, then a large traffic jam results. When it becomes known that the building work will last for some time, an increasing number of drivers will take alternative routes via minor roads. If the police have been so kind as to put up diversion signs, and some of these side roads or “collaterals”

may even be two-lane roads in the right direction, then the traffic will flow quickly via these

diversions. Only very few drivers will pass through the bottleneck at snail's pace. If, one day, a complete closure cannot be avoided, what then? Then a few more cars will use the "collaterals" without causing any major problems.