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Steffen Gloekler, MD; Christian Seiler, MD

Blood to the heart muscle is provided by the left and right coronary arteries, which arise from the aorta and then branch into increasingly tinier vessels. Each coronary artery supplies blood to its own area of muscle (Figure 1). In humans, very small, hairlike vessels (capillaries) are often the only interconnections between the coronary arteries and their service areas. Sometimes, however, larger vessels interconnect the supplied areas. These vessels are called “collateral vessels” or “natural bypasses.” Only these collaterals, not the capillary network, are capable of delivering an adequate amount of blood to the heart muscle. The extent of these natural bypasses varies from person to person and depends on individual conditions, such as hereditary factors, the degree of physical activity, and the existence of constrictions in the main coronary arteries, called “coronary artery disease.”

Myocardial Infarction Is a Major Cause of Death
In industrialized countries, coronary artery disease is a major killer. The disease is characterized by a general inflammatory process of the entire coronary artery system, with development of local deposits (called “plaques”) of lipids, cells of inflammation, connective tissue, and calcium. This can lead either to progressively slow narrowing of the affected artery (which is typically sensed as angina pectoris) or a sudden rupture of a plaque (Figure 1) caused by its instability. In the latter situation, the downstream blood flow is abruptly blocked (which is the classic “heart attack” or myocardial infarction; Figure 1).

Factors That Determine the Size of an Infarct
As a consequence of such a blockage, the section of heart muscle normally supplied by the vessel lacks “fuel,” that is, oxygen and nutrients, and it finally dies. If the patient survives, the final stage is a loss of cardiac pumping strength and a scar that replaces the former muscle. The size of an infarction is not the same in every case: It is mainly influenced by the size of the blocked artery (that is, a larger artery supplies a larger area of muscle, the so-called area at risk), the duration of the blockage (that is, the damage is smaller when rapid reopening of the artery occurs), and importantly, the extent of blood flow through collaterals from neighboring vessels (that is, the more that collaterals can “step into the breach” to supply the starving muscle tissue in need, the smaller is the size of the infarction; Figure 2). In the ideal case, a very good collateral supply to the area at risk can make the infarct size shrink to zero when there is a sudden blockage of an artery. In the worst case, when no or a very low collateral flow to the starving area of infarction is available, the infarct involves the entire area at risk of the supplying vessel, which is blocked.

Established Treatments for Coronary Artery Disease
Besides treatment with drugs, constricted vessels can be widened by inflating a balloon on a catheter (angioplasty) and, in most cases, also placing a wire tube (stent) to keep the vessel open. In an infarction, this is the superior therapy, because blood flow to the heart muscle is restored as quickly as possible, and damage can be limited. Coronary artery bypass grafting (CABG; pronounced “cabbage”) operation is needed if numerous severe constrictions near the outlets of the coronary arteries are present. Alto-
together, these technologies have led to enormous progress in the treatment of the disease in the last 30 years.

**Treatment Limitations**

As in every catheter-based intervention or surgical operation, there are risks related to the procedures. Apart from that, approximately one fourth of all patients are not suitable for these revascularization procedures at all. These procedures do indeed repair the local problem of narrowings, but they do not alter the natural progression of the disease. Alternative treatment practices are therefore required to alter the course of coronary artery disease, alleviate angina pectoris, and thereby decrease the number of deaths due to infarction.

**How to Turn the Capillary Network Into Collaterals**

When there is increasing traffic volume on a highway, it may make sense to make the highway into a larger freeway to allow a higher traffic volume. In short, the same happens to the coronary arteries: When blood flow is increased, the inner layer of vessel cells (endothelial cells) sense this necessity and start the process of enlarging from capillaries into genuine collateral vessels. In response to endurance exercise training (such as running, bicycling, swimming, and hiking), blood flow is increased, which leads to a conversion from capillaries into collaterals. This is a very elegant treatment everybody can accomplish. It reduces the chances of the occurrence of angina pectoris, myocardial infarctions, and death. Beyond the interventional, surgical, and medical treatments against coronary artery disease, this collateral training is a natural and valuable therapy that many patients can apply by themselves, for themselves, if only they are aware of it.

**Key Points**

- As a pump, the heart muscle is critically dependent on the coronary arteries for sufficient blood supply.
- Narrowing of the coronary arteries by deposits (plaques) can starve the heart muscle of oxygen and nutrients. This results in chest pain (angina pectoris).
- A heart attack results when a diseased coronary artery is blocked completely. As a consequence, the portion of the heart muscle normally supplied by this artery dies (myocardial infarction).
- After a myocardial infarction, a certain mass of muscle is lost forever. Depending on infarct size, quality of life and life expectancy can be shortened.
- Collateral training results in a better blood supply of the heart and thus demonstrably fewer heart attacks, less severe loss of valuable heart muscle, better quality of life, and higher survival rates.