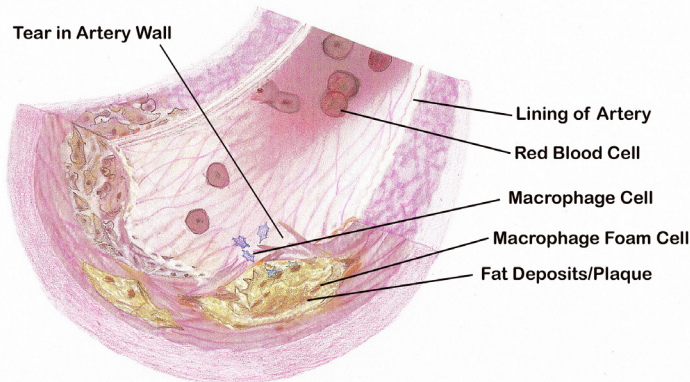


highways. Because the heart is our hardest working organ, its oxygen demands are enormous. As plaques build, the amount of oxygen delivered to the heart muscle declines, which causes a rise in the risk of heart attacks. Atherosclerosis occurring in the heart and nearby neck and chest arteries can also cause ischemic strokes if they obstruct oxygenated blood flow to the brain. Risks increase with poor oral health.

Plaques arise in blood vessels walls as a response to injury. The damage can come from high homocysteine blood levels from either genetic influences or inadequate intake of B complex vitamins, constant high insulin levels found most commonly in people with pre-diabetes, glycation (AGE) damage from eating sugar and other simple carbohydrates, high blood pressure, nicotine irritation from smoking, constant release of the stress hormone cortisol, viruses, or ingestion of trans fats. Gum disease can also accelerate blood vessel damage because it is one of many infections that increase blood levels of C-reactive protein, or CRP. CRPs are addressed more thoroughly below.



SECTION OF PLAQUE-FILLED ARTERY: Plaques do not form in the open channels of blood vessels. They form just beneath the interior arterial walls. To repair damage, low-density lipoprotein cholesterol carriers, or LDLs, bring cholesterol to the site where they aggregate with blood platelets to repair the damage. LDLs tend to oxidize as they flow through oxygen-rich arterial blood so as this complex infiltrates the single cell lining of the artery, or endothelium, white blood cells called macrophages (shown as blue star shapes) engulf them. Unfortunately, these macrophages cannot process oxidized LDLs. Together, they form “foam cells” (shown rust), an important component of the damaging plaques found within the walls of our arteries. These foam cells grow,

rupture, and signal the need for more cholesterol repair, which is brought by more oxidized LDLs in an endless damaging cycle.

Smooth muscle cells and a protective fibrous cap line and thicken the artery wall to keep blood flowing smoothly, but foam cells secrete a substance capable of breaching this protective layer. Debris from the plaques escapes into the bloodstream if the wall ruptures. Clotting occurs quickly. A clot, escaped debris, or both can narrow or block blood vessels. This occurs at the injury site or in smaller downstream vessels.

Active Oral Infections Pour Oil on Inflammatory Fires

CAROLINE: “Claire, a study released to periodontists, or gum surgeons, in 2004 flatly stated, ‘The periodontal (gum) health of patients admitted to the Coronary¹ Care unit due to acute coronary syndrome is unacceptable.’¹ In fact, those with chronic gum disease develop heart disease one and a half times more often than those with healthy mouths.

“As I said, long-term active oral infections initiate a cascade of biological responses. The immune system functions by flooding the body with chemical messenger molecules designed to help your body heal.² These healing molecules help most when they fight acute infections. They are problematic and destructive when they attempt to manage chronic, prolonged infections like gum and heart diseases, the chronic diseases that can begin in childhood, but surface as we age.

“These messenger molecules stimulate the liver to make C-reactive proteins or CRPs. The media most frequently link high levels of CRPs to heart disease. A test for circulating CRPs can give you an indication of your cumulative inflammatory risk from all sources. CRP levels are consistently higher in people with gum disease compared to those with healthy gums.³ Many doctors believe knowing your CRP numbers is as important as knowing your cholesterol numbers.

¹ The term *coronary* pertains to the heart, especially in health issues.

² When gums are infected, tumor necrosis factor alpha (TNF-alpha, Interleukin-1, Interleukin-6, lipopolysaccharides (LPS), and prostaglandins are just some of the messenger molecules that are released.