How is plaque formed in the arteries

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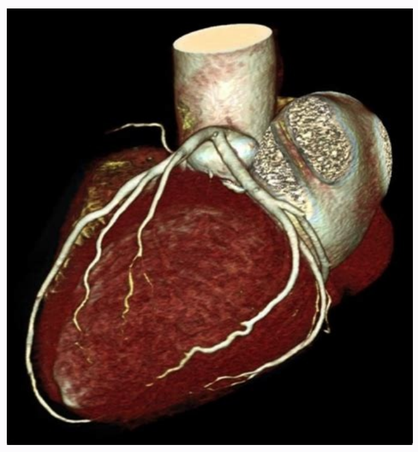
Degree of ICA Stenosis in Doppler US*

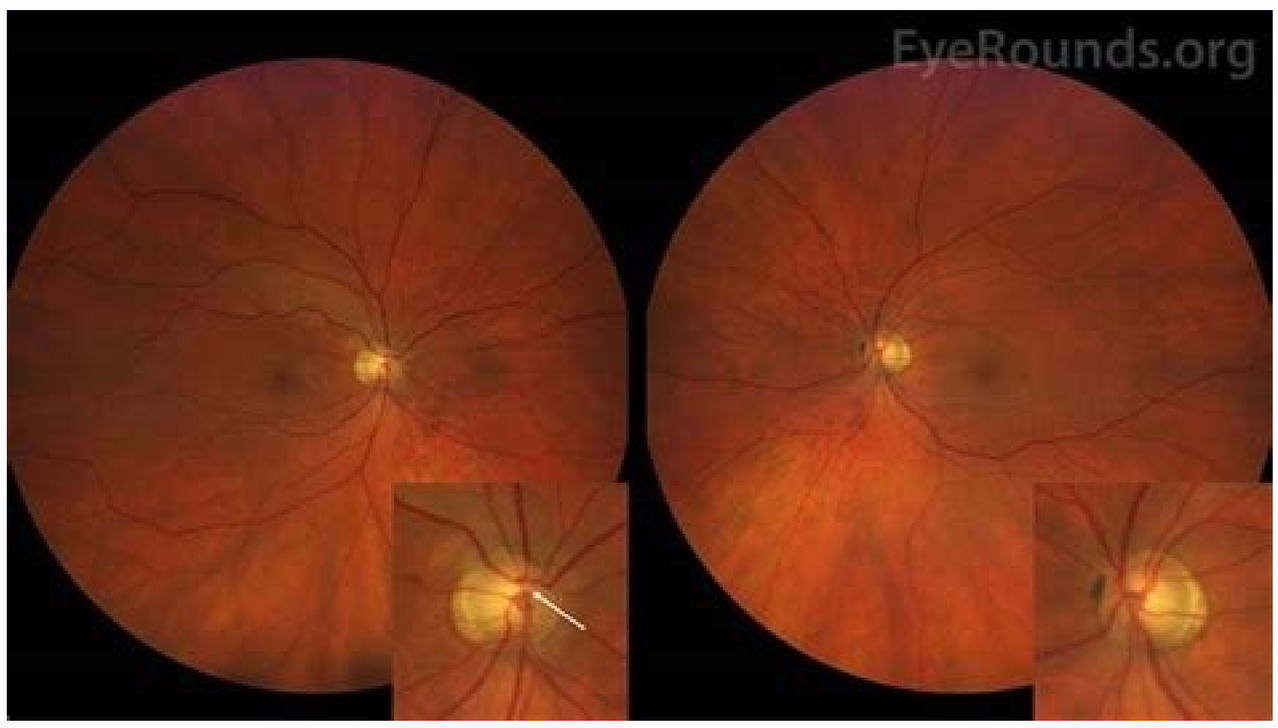
Consensus Criteria – NASCET criteria

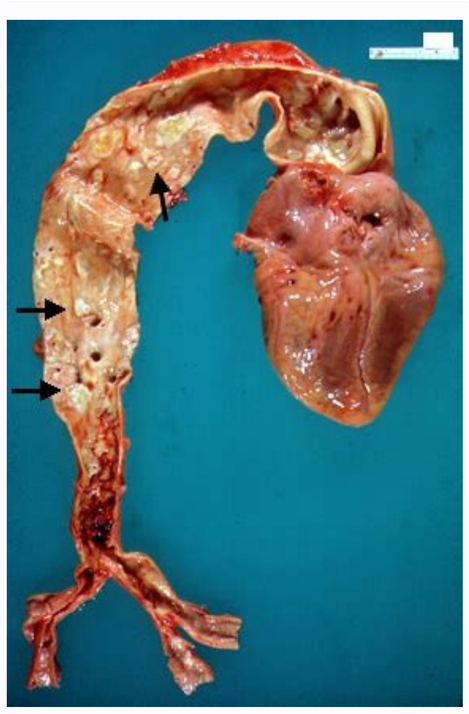
ICA stenosis (%)	ICA PSV cm/sec	ICA EDV cm/sec	PSV ratio ICA/CCA
Normal	< 125	< 40	< 2.0
< 50%	< 125	< 40	< 2.0
50 – 69%	125 – 230	40 – 100	2.0 – 4.0
> 70%	> 230	> 100	> 4.0
Near occlusion	variable	variable	variable
Total occlusion	undetectable	undetectable	not applicable

* Diameter reduction

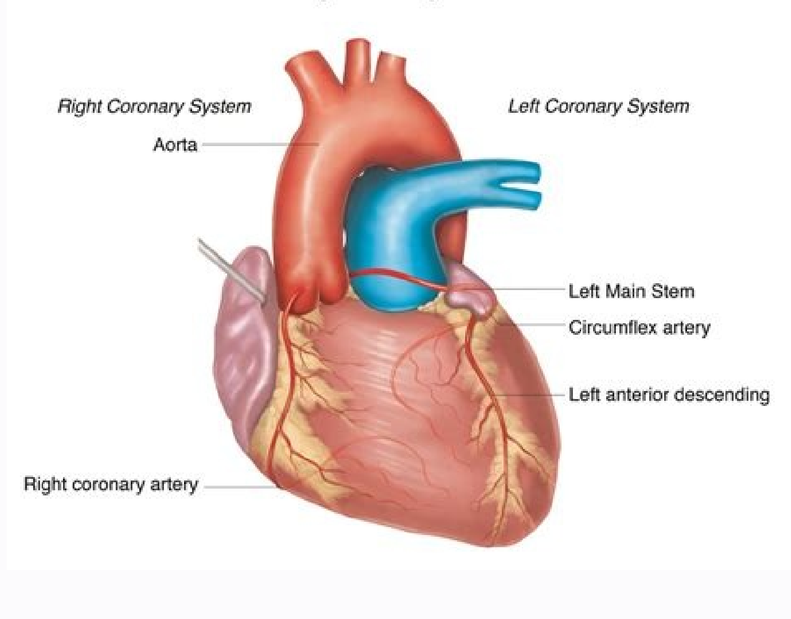
Grant EG et al. Radiology 2003 ; 229 : 340 – 346.







Coronary Artery Disease



How does plaque get in arteries. What really causes plaque in arteries. How does the plaque form in arteries.

Atherosclerosis causes clinical disease through luminal narrowing or by precipitating thrombi that obstruct blood flow to the heart (coronary heart disease), brain (ischemic stroke), or lower extremities (peripheral vascular disease). The most common of these manifestations is coronary heart disease, including stable angina pectoris and the acute coronary syndromes. Atherosclerosis is a lipoprotein-driven disease that leads to plaque formation at specific sites of the arterial tree through intimal inflammation, necrosis, fibrosis, and calcification. After decades of indolent progression, such plaques may suddenly cause life-threatening coronary thrombosis presenting as an acute coronary syndrome. Most often, the culprit morphology is plaque rupture with exposure of highly thrombogenic, red cell–rich necrotic core material. The permissive structural requirement for this to occur is an extremely thin fibrous cap, and thus, ruptures occur mainly among lesions defined as thin-cap fibroatheromas. Also common are thrombi forming on lesions without rupture (plaque erosion), most often on pathological intimal thickening or fibroatheromas. However, the mechanisms involved in plaque erosion remain largely unknown, although coronary spasm is suspected. The calcified nodule has been suggested as a rare cause of coronary thrombosis in highly calcified and tortious arteries in older individuals. To characterize the severity and prognosis of plaques, several terms are used. Plaque burden denotes the extent of disease, whereas plaque activity is an ambiguous term, which may refer to one of several processes that characterize progression. Plaque vulnerability describes the short-term risk of precipitating symptomatic thrombosis. In this review, we discuss mechanisms of atherosclerotic plaque initiation and progression; how plaques suddenly precipitate life-threatening thrombi; and the concepts of plaque burden, activity, and vulnerability.References1. Levy D. Combating the epidemic of heart disease.JAMA. 2012; 308:2624–2625.CrossrefMedlineGoogle Scholar2. Murray CJ, Lopez AD. Measuring the global burden of disease.N Engl J Med. 2013; 369:448–457.CrossrefMedlineGoogle Scholar3. Laslett LJ, Alagona P, Clark BA, Drozda JP, Saldivar F, Wilson SR, Poe C, Hart M. The worldwide environment of cardiovascular disease: prevalence, diagnosis, therapy, and policy issues: a report from the American College of Cardiology.J Am Coll Cardiol. 2012; 60:S1–S49.CrossrefMedlineGoogle Scholar4. Fuster V, Mearns BM. The CVD paradox: mortality vs prevalence.Nat Rev Cardiol. 2009; 6:669.CrossrefMedlineGoogle Scholar5. Nabel EG, Braunwald E. A tale of coronary artery disease and myocardial infarction.N Engl J Med. 2012; 366:54–63.CrossrefMedlineGoogle Scholar6. Yusuf S, Hawken S, Ounpuu S, Dans T, Avezum A, Lanas F, McQueen M, Budaj A, Pais P, Varigos J, Lisheng L; INTERHEART Study Investigators. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study.Lancet. 2004; 364:937–952.CrossrefMedlineGoogle Scholar7. Lim SS, Vos T, Flaxman AD, et al.. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990-2010: a systematic analysis for the Global Burden of Disease Study 2010.Lancet. 2012; 380:2224–2260.CrossrefMedlineGoogle Scholar8. Steinberg D, Glass CK, Witztum JL. Evidence mandating earlier and more aggressive treatment of hypercholesterolemia.Circulation. 2008; 118:672–677.LinkGoogle Scholar9. Ference BA, Yoo W, Alesh I, Mahajan N, Mirowska KK, Mewada A, Kahn J, Afonso L, Williams KA, Flack JM. Effect of long-term exposure to lower low-density lipoprotein cholesterol beginning early in life on the risk of coronary heart disease.J Am Coll Cardiol. 2012; 60:2631–2639.CrossrefMedlineGoogle Scholar10. Bøttcher M, Falk E. Pathology of the coronary arteries in smokers and non-smokers.J Cardiovasc Risk. 1999; 6:299–302.CrossrefMedlineGoogle Scholar11. Go AS, Mozaffarian D, Roger VL, et al.; American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Heart disease and stroke statistics–2014 update: a report from the American Heart Association.Circulation. 2014; 129:e28–e292.LinkGoogle Scholar12. Fowkes FG, Rudan D, Rudan I, Aboyans V, Denenberg JO, McDermott MM, Norman PE, Sampson UK, Williams LJ, Mensah GA, Criqui MH. Comparison of global estimates of prevalence and risk factors for peripheral artery disease in 2000 and 2010: a systematic review and analysis.Lancet. 2013; 382:1329–1340.CrossrefMedlineGoogle Scholar13. Willey J, Gonzalez-Castellon M. Cholesterol level and stroke: a complex relationship.JAMA Intern Med. 2013; 173:1765–

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