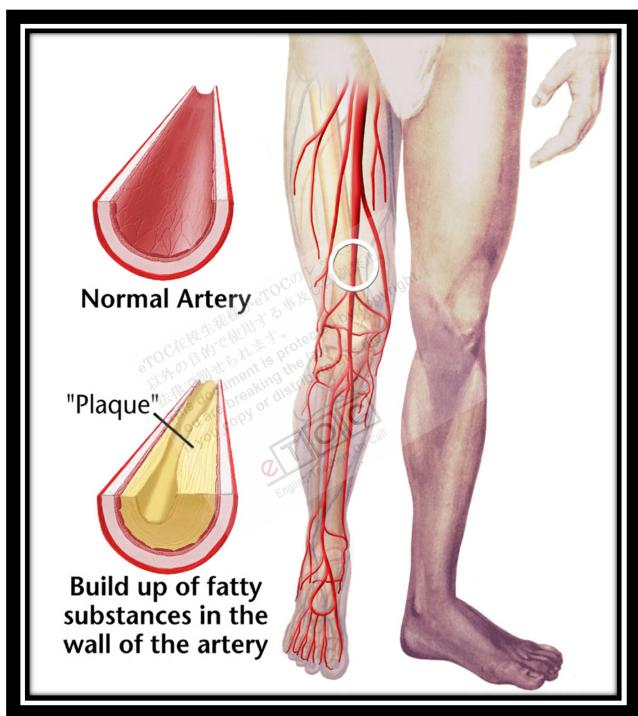
Non-atheromatous Arteriosclerosis

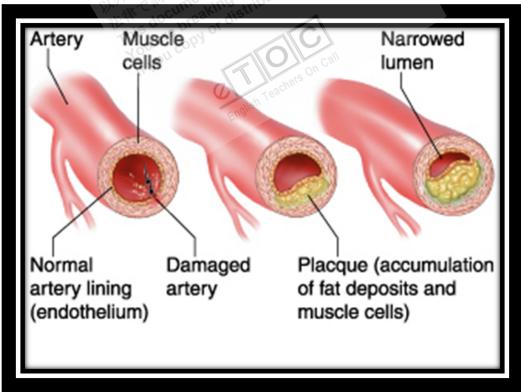


http://i.lifelinescreening.com/en-us/RichTextboxImages/Image/media-library/arteriosclerosis.jpg

Non-atheromatous arteriosclerosis is age-related fibrosis in the aorta and its major branches.

Non-atheromatous arteriosclerosis causes intimal thickening and weakens and disrupts the elastic lamellae. The smooth muscle (media) layer atrophies, and the lumen of the affected artery widens (becomes ectatic), predisposing to aneurysm or dissection. Hypertension is a major factor in development of aortic arteriosclerosis and aneurysm. Intimal injury, ectasia, and ulceration may lead to thrombosis, embolism, or complete arterial occlusion.

Arteriolosclerosis affects distal arteries in patients with diabetes or hypertension. Hyaline arteriolosclerosis affects small arteries and arterioles in patients with diabetes; typically, hyaline thickening occurs, the arteriolar wall degenerates, and the lumen narrows, causing diffuse ischemia, especially in the kidneys. Hyperplastic arteriolosclerosis occurs more often in patients with hypertension; typically, laminated, concentric thickening and luminal narrowing occur, sometimes with fibrinoid deposits and vessel wall necrosis (necrotizing arteriolitis). Hypertension promotes these changes, and arteriolosclerosis, by increasing arteriolar rigidity and increasing peripheral resistance, may help sustain the hypertension.



http://www.mdguidelines.com/images/Illustrations/atherosc.jpg

Mönckeberg arteriosclerosis (**medial calcific sclerosis**) affects patients > 50; agerelated medial degeneration occurs with focal calcification and even bone formation within the arterial wall. Segments of the artery may become a rigid calcified tube without luminal narrowing. The diagnosis is usually obvious by plain x-ray. This disorder is clinically important only because it can greatly reduce arterial compressibility, causing extremely but falsely elevated BP readings.

Reference: http://www.merckmanuals.com

