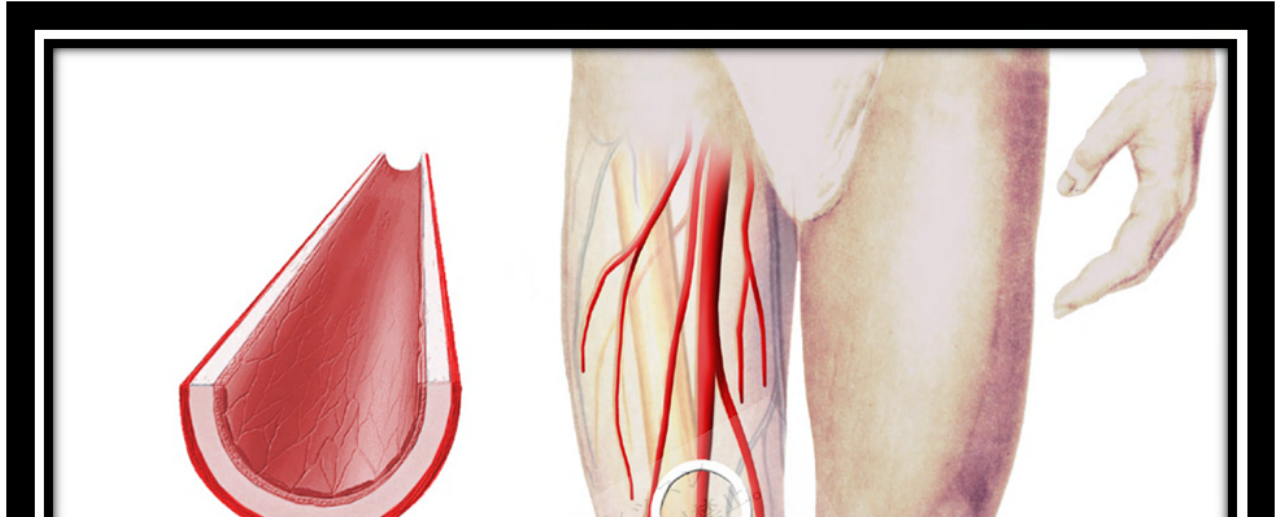


## Non-atheromatous Arteriosclerosis



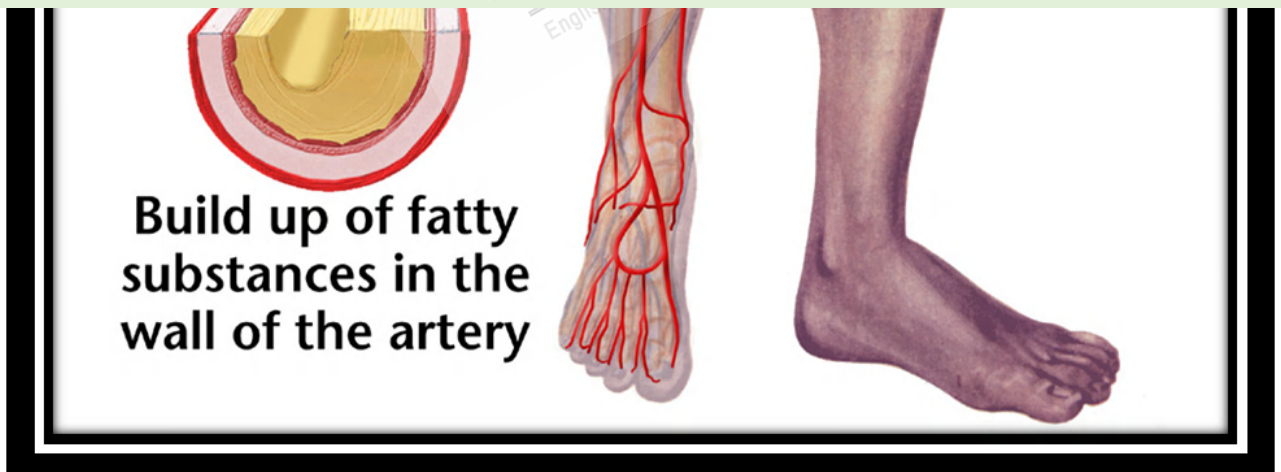
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**Build up of fatty substances in the wall of the artery**

<http://l.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

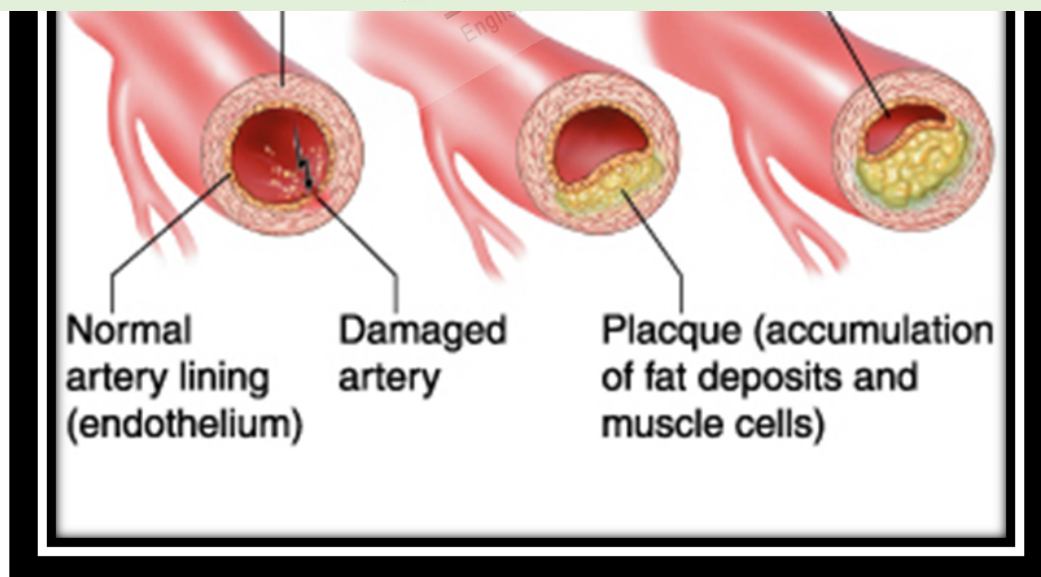
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<http://www.mdguidelines.com/images/illustrations/atherosc.jpg>

**Mönckeberg arteriosclerosis (medial calcific sclerosis)** affects patients > 50; age-related 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>

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