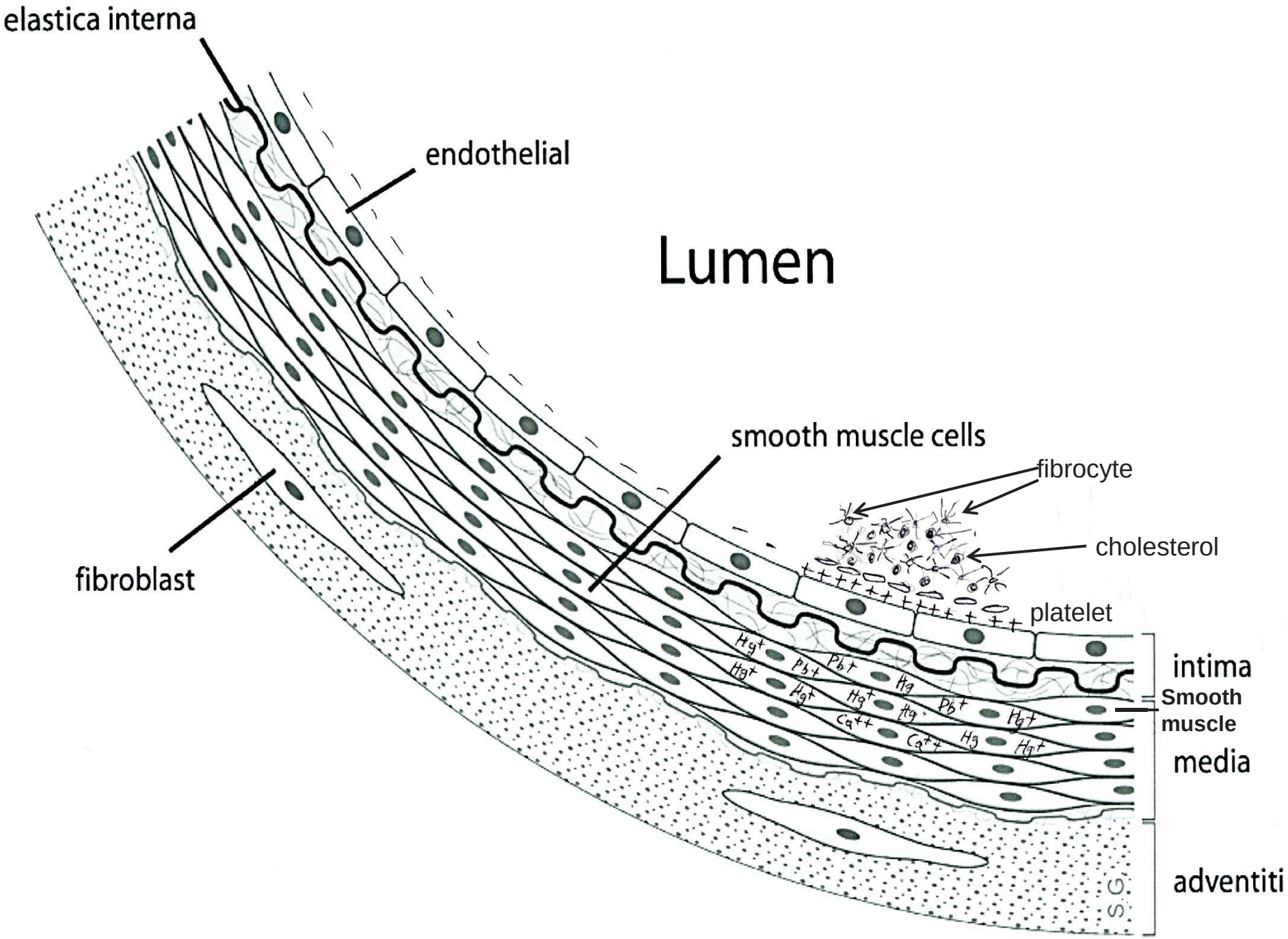
A THEORY OF ATHEROSCLEROSIS

Presented by Dr. Dean Bonlie

An ACAM featured Luncheon Speaker in 1995 was a pathologist from the University of Wisconsin. He examined 120 corpses from heart attacks and microtomed their coronary arteries, examining all slides for any consistent chemicals in all cases. He only found lead and Hg …either one or both, behind every plaque. Where there was NO lead or Hg in the smooth muscles and in the media there was NO plaque.


elastica interna
endothelial
Lumen
smooth muscle cells
fibrocyte
cholesterol
platelet
fibroblast
intima
Smooth muscle
media
adventiti

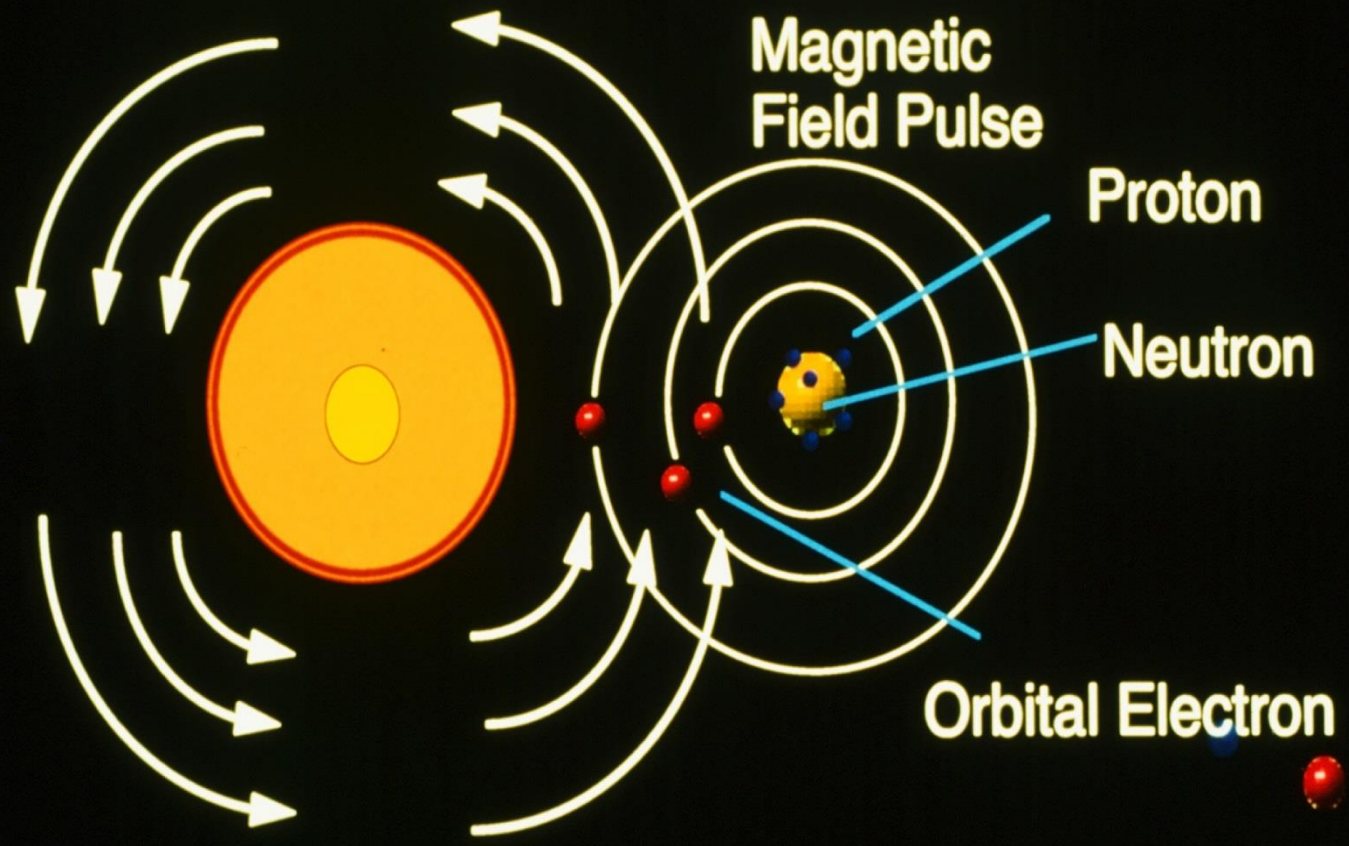
CASE HISTORY

- A 60-year old former Fire Chief from Las Vegas, NV, had been hospitalized with a heart attack. The result was a complete loss of one-third of his left ventricle function from the occluded artery. He was very weak, with extreme angina. He tried EDTA chelation for two months, but with no relief.

- He then tried sleeping on a 20-gauss Magnetico Sleep Pad. He also took 500 mg of DMSA each night at bed time. By the end of one month, he had no more angina. Although, he was still weak from the loss of one-third of the left ventricle muscle. He then treated with the Magnetic Molecular Energizer device for 110 hours, which returned his heart function to normal. He has had no further heart issues since that time.

Body Electromagnetic Forces on Atoms

(MAGNETIC RESONANCE)



Astrocyte

Converts chemical energy to electrical energy

Pulsed D.C. electrical signal to body mitochondria

Overunity due to Resonance

Mitochondria of all targeted cells

Increase in A.T.P. Production

ATP CATALYZES MOST BODY CHEMISTRY

- Including smooth muscles in the media of coronary arteries.
- Smooth muscles are used to open the lumen to increase blood supply to the brain's astrocytes.
- This enhances oxygen needed in electrical production.
- The more fatigued the astrocyte are - the more oxygen needed.

PATHOLOGY

Lack of rest and/or continual stress

Depletes voltage from brain

Weaker pulse

Reduces resonance in the mitochondria

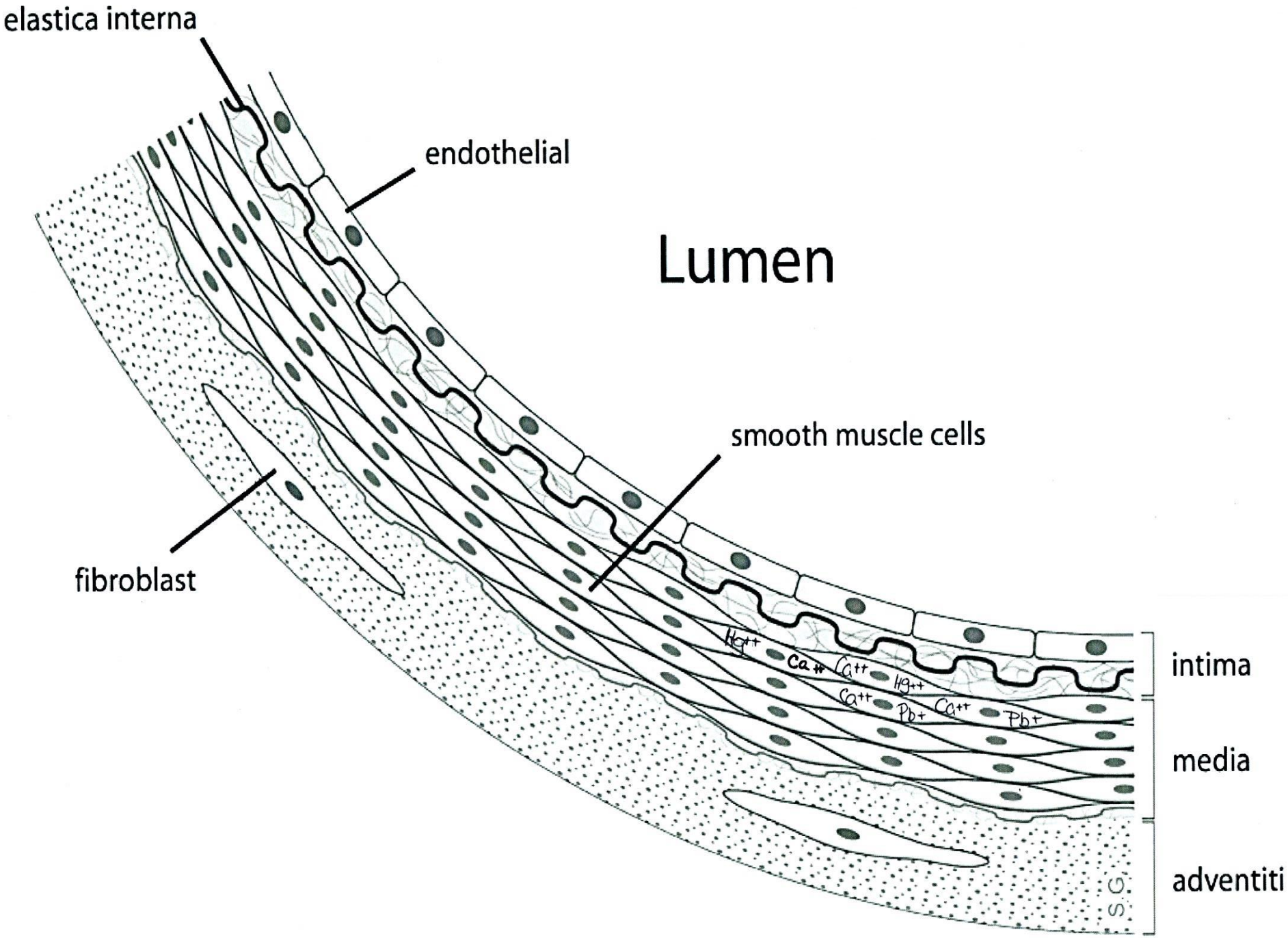
Less A.T.P. production in smooth muscle

Lower intracellular potential

Ca^{++} channels open$\longrightarrow$ Ca^{++} drawn inside cell wall

Increase voltage differential

Increase Ca^{++} channels let in Hg^{+} on Pb^{+} also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Patient gets rest →Voltage from brain increases

More resonance in mitochondria of smooth muscle

Increased A.T.P. production

Na+ / K+ pump increases charge on cell wall

Ca^{++} moves out of cell, but

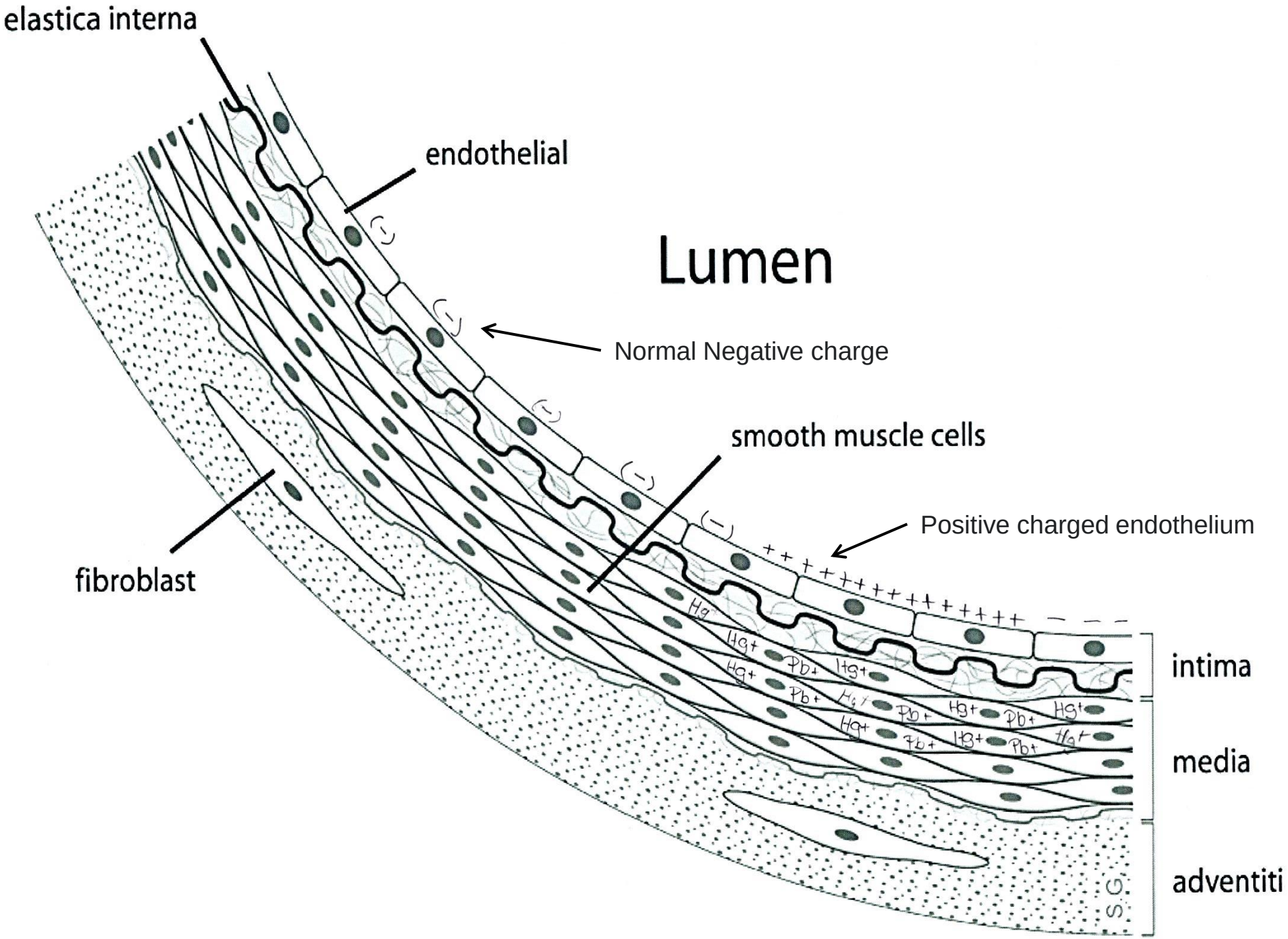
Hg^{+} and PB^{+} stay

It takes 40 millivolt differential on cell wall for Ca^{++} to leave cell

It takes 100 millivolt differential on cell wall for Hg^{+} and Pb^{+} to leave a cell

There is not enough voltage from the brain to resonate the smooth muscle mitochondria to make enough A.T.P. to up-regulate Na/K pump enough to make 100 millivolt potential on their cell wall.

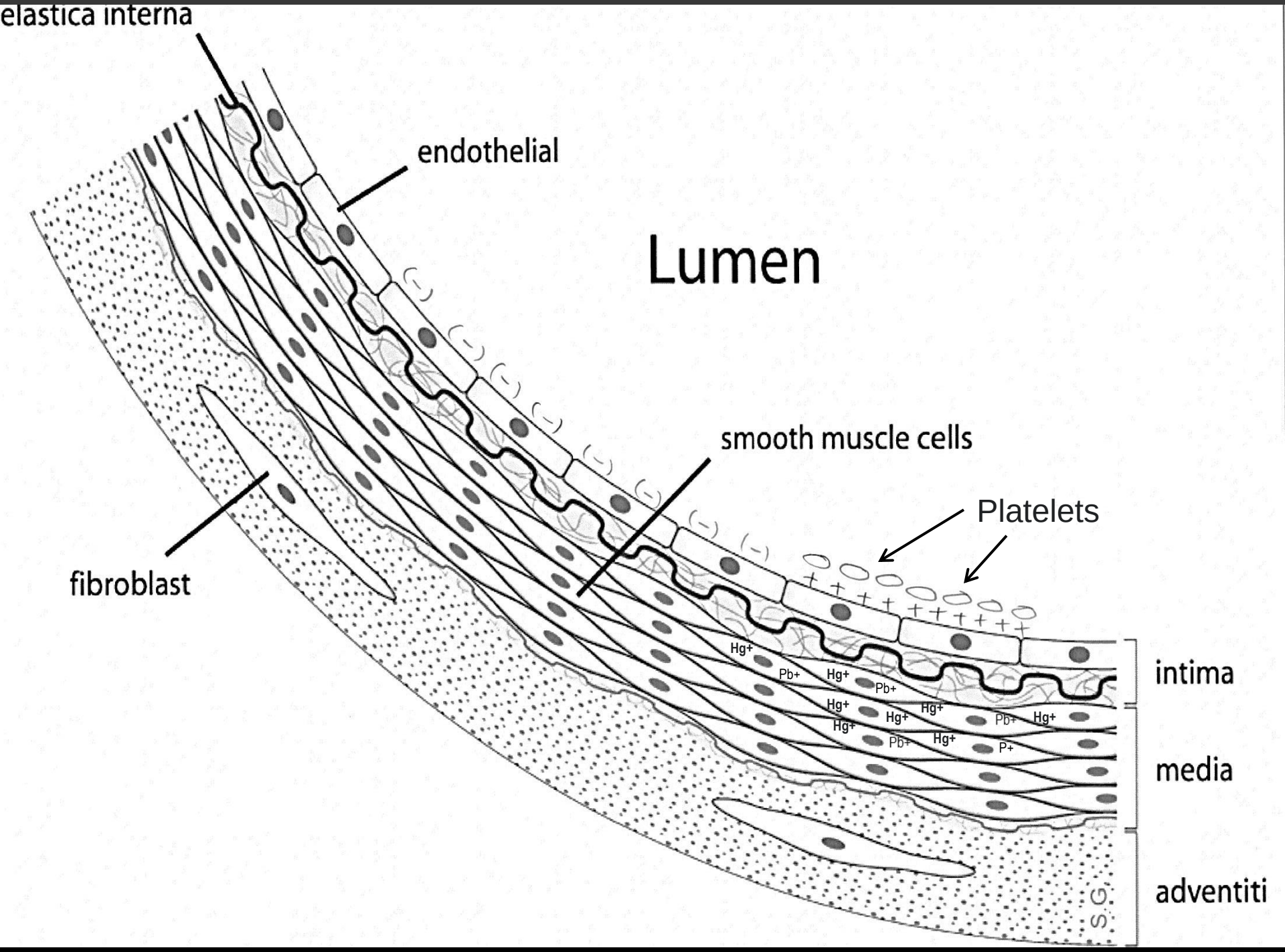
Others have tried to resolve this problem by giving patients high dosages of Vitamin C, Alpha Lipoic Acid, CoQ10 and Glutathion to enhance the Krebs cycle to make A.T.P. – with limited success.


elastica interna
endothelial
Lumen
Normal Negative charge
smooth muscle cells
Positive charged endothelium
fibroblast
intima
media
adventiti

The accumulation of positive ions (Hg+ and Pb+) under the endothelium (very thin) depolarizes it from negative (-) to positive (+)

Positive polarity is the polarity of injury in the body.

This signals platelets to cover the area.


elastica interna
endothelial
Lumen
smooth muscle cells
Platelets
fibroblast
Hg+
Pb+
P+
intima
media
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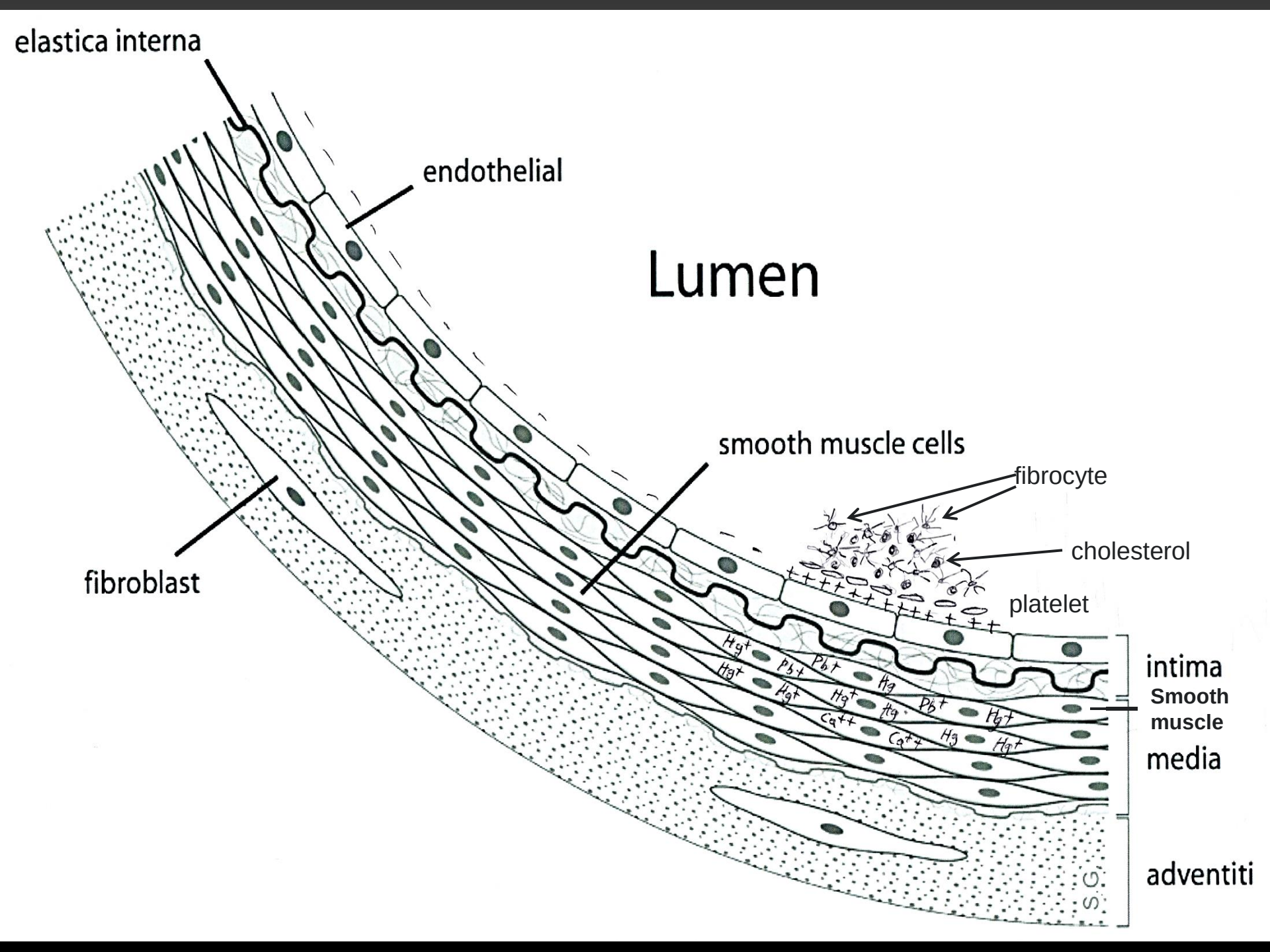
S.G.

Platelets signal fibrocytes to attach to the positive endothelium and make a mesh.

Cholesterol impregnates the mesh and makes soft plaque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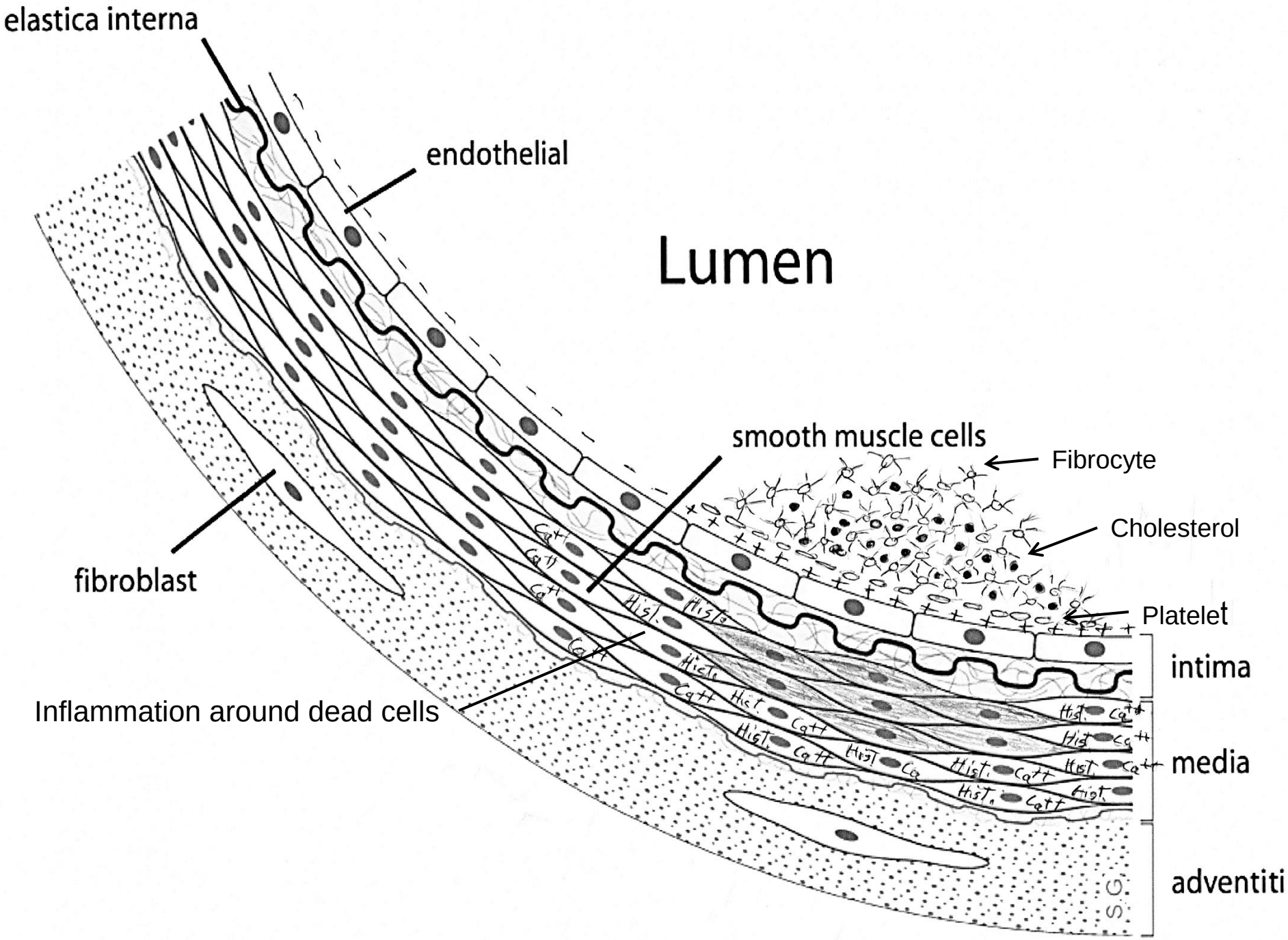
Soft plaque shuts off oxygen and nutrition from underlying cells

Smooth muscle cells die

Releases histamine
(which is very positive)

Causing inflammatory response in media of artery

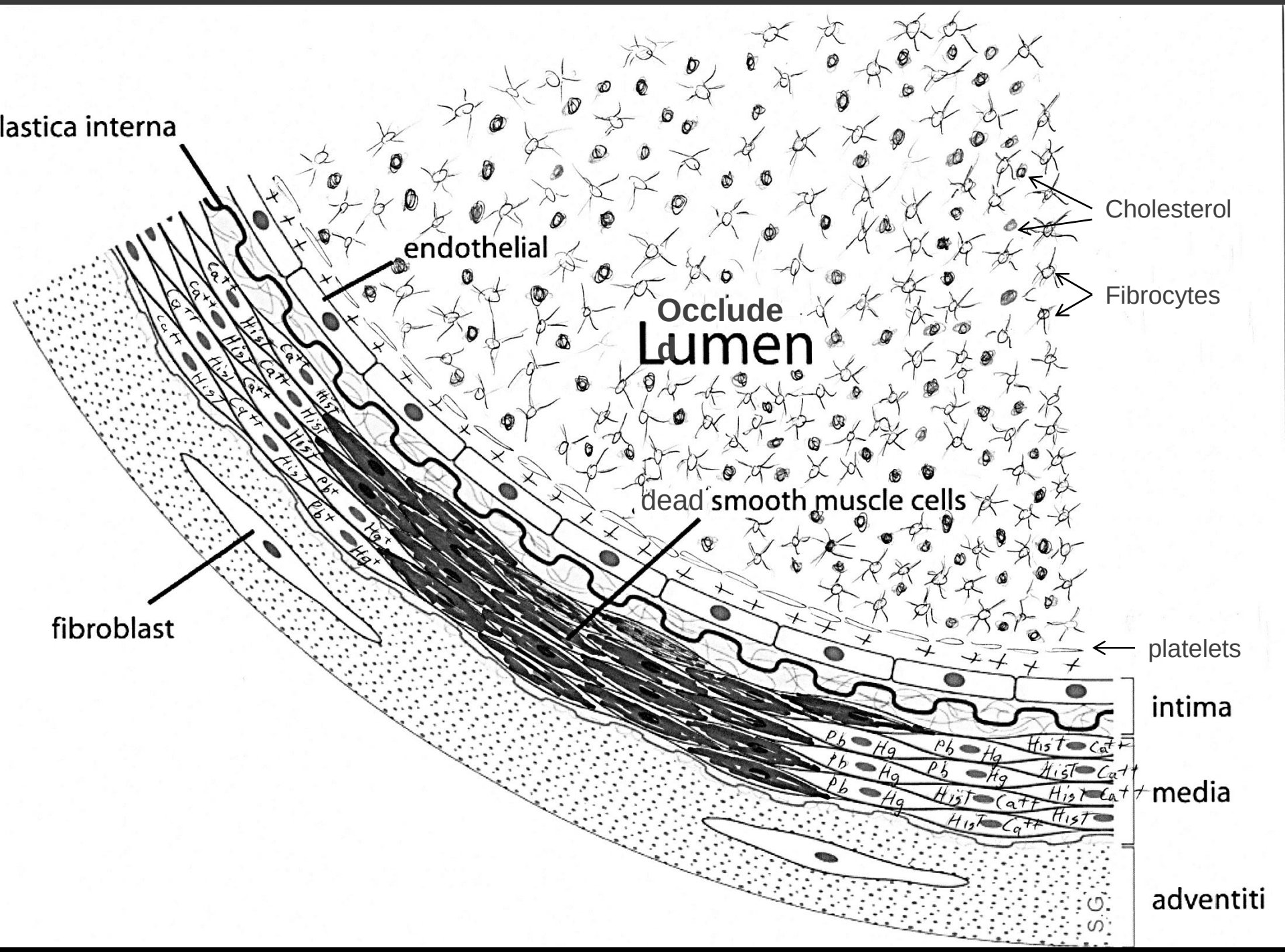

elastica interna
endothelial
Lumen
smooth muscle cells
Fibrocyte
Cholesterol
Platelet
fibroblast
Inflammation around dead cells
intima
media
adventiti

Plaque grows from increased positive charge

Partial occlusion of artery

Angina -
Due to loss of oxygen in heart muscle

Larger plaque → Occlusion of artery


lastica interna
endothelial
Occlude
Lumen
Cholesterol
Fibrocytes
dead smooth muscle cells
fibroblast
platelets
intima
media
adventiti

HEART ATTACK

Ischemia in heart muscle cells

Death of cells

Death of patient