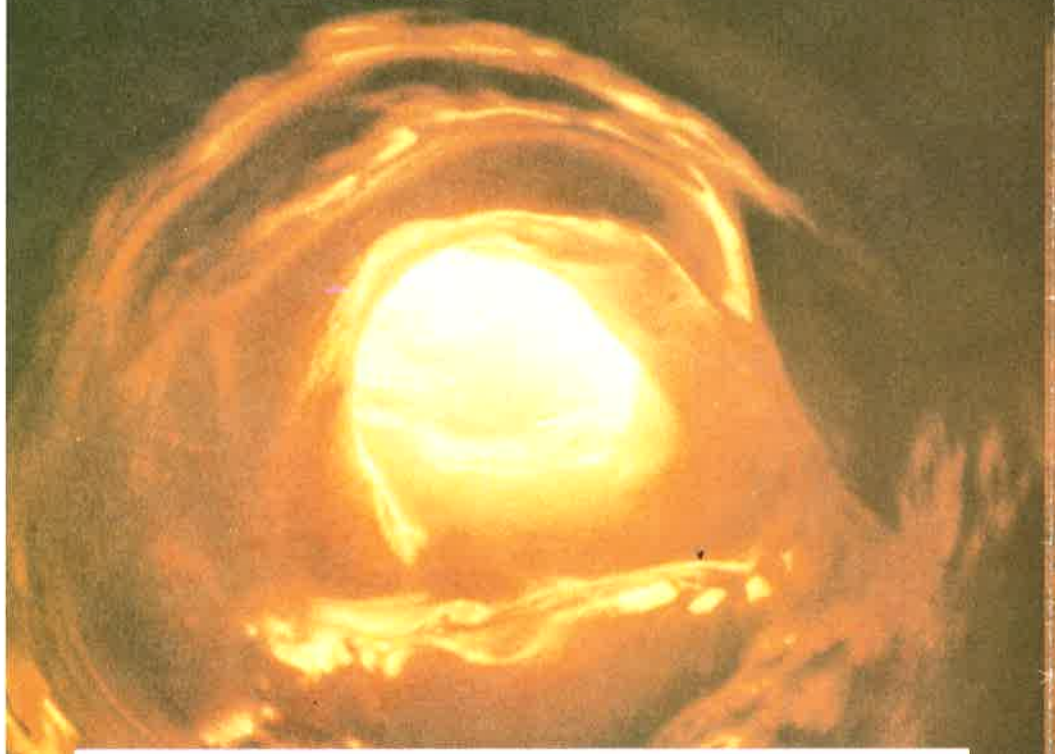
Protecting Your Heart



"Atherosclerosis is widespread and kills many people who have much to give to society, but it continues to be an elusive villain. This silent killer eats with us, sits with us, drinks with us, and smokes with us. One day he makes his presence known—usually with a heart attack or a stroke. Only one in two survive this first encounter."—Lawrence Lamb, M.D., professor of cardiology, Baylor University College of Medicine.

The best answer to heart disease lies in prevention. Obviously the heart is our most vital organ—once it is in trouble, we are in trouble. To reduce such a risk, here are some specific suggestions: Watch your diet and your weight, control your blood pressure, avoid tensions and worry, obtain regular rest and sleep, avoid smoking, get adequate exercise, and have regular medical check-ups.

Your heart is your life—keep it alive!

Photos on pages 1-3 and page 8 of this brochure are by Lennart Nilsson, made available for this *Listen* feature by the American Heart Association (Campbell Moses, medical director) and provided for the programs of the Heart Association through the courtesy of the Ayerst Laboratories. Photos on pages 4-7 are supplied by L. H. Lonergan, M.D., M.P.H., Associate Professor of Tropical Health, School of Health, Loma Linda University.

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LITHO IN U.S.A.

Your Heart

WHAT SMOKING MAY DO

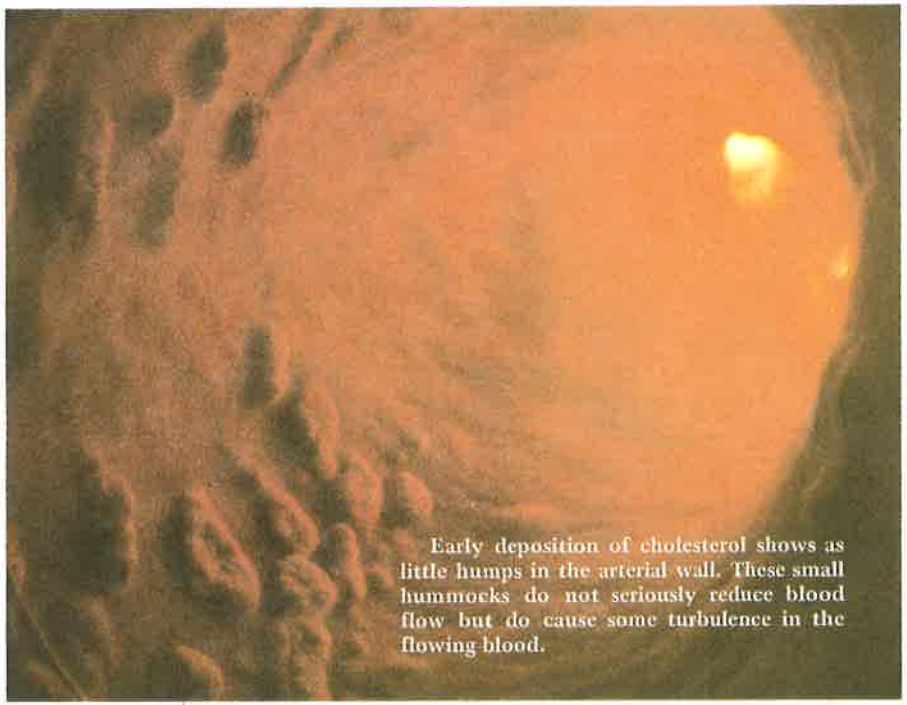
This dramatic inside-the-heart picture shows the normal aortic arch of a healthy heart. Note the smooth inner artery lining.

What can smoking do to this picture?

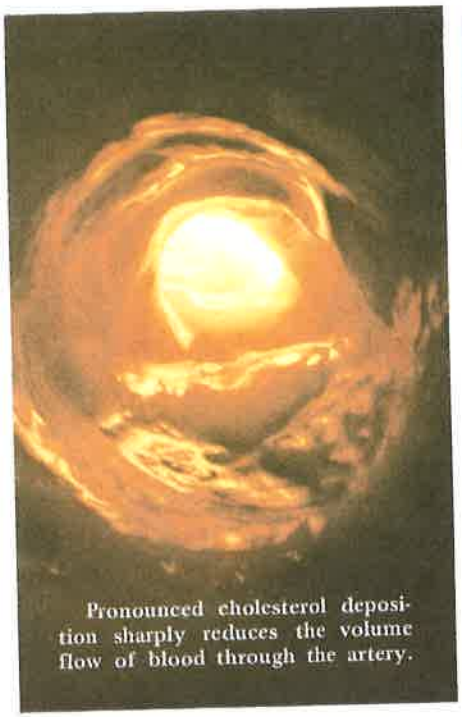
Corrosion of the Inner Lining

These illustrations document the corrosion of the inner lining of the artery with cholesterol and other lipids that occurs in atherosclerosis (hardening of the arteries). This sequence of events results in the obstruction of an artery by a clot or thrombus. The cholesterol deposition reduces the flow of blood through the arteries, and the development of a thrombus shuts off blood flow completely.

Cigarette smoking tends to increase the deposit of these lipids, chiefly cholesterol, within the inner walls of the arteries, in this way making atherosclerosis more severe and gradually clogging the arteries to the heart. In addition, smoking increases the likelihood of blood clot.

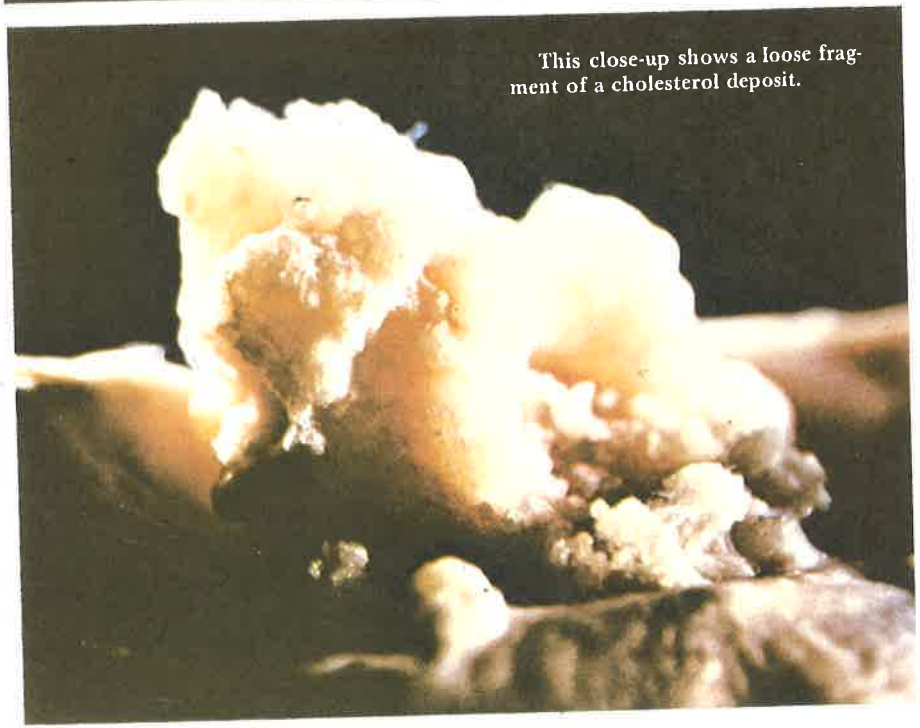


Early deposition of cholesterol shows as little humps in the arterial wall. These small hummocks do not seriously reduce blood flow but do cause some turbulence in the flowing blood.



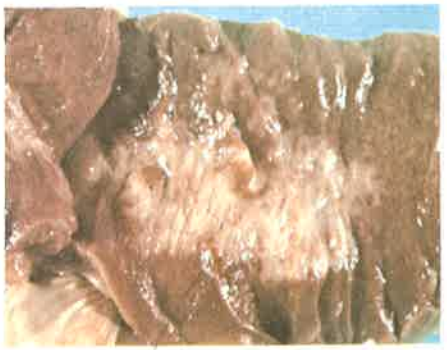
Pronounced cholesterol deposition sharply reduces the volume flow of blood through the artery.

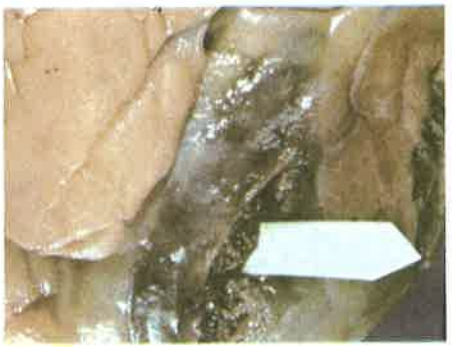
Now there is severe interference with blood flow, with irregular fragments that are loosely attached to the wall of the artery.

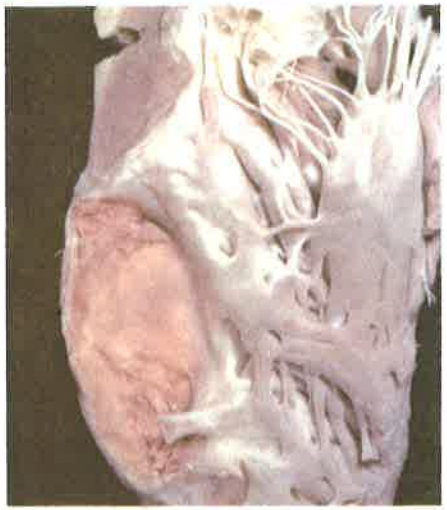


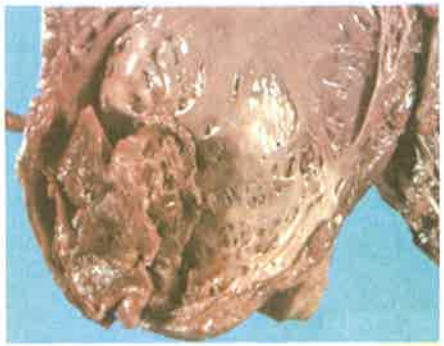
This close-up shows a loose fragment of a cholesterol deposit.

Damage to the Body Pump









Evidence shows that the smoking of cigarettes is a major heart hazard. For smokers the risk of sudden death from heart disease is three times that for nonsmokers. The risk may go as high as five times for those who smoke more than a pack a day.

(Opposite page.) This is a shallow slice into the heart muscle wall of a person who had a coronary occlusion due to atherosclerosis. It shows the condition of the heart muscle wall a few days after obstruction of a coronary artery. The pale yellow area is softened "dead" heart muscle (infarct of heart) which has degenerated because of lack of nutrients and oxygen. The dark red area is hemorrhage in the margins surrounding the infarct.

(Upper left.) The heart wall shows a scar (central, pale area) at site of healed infarct. The dark brown area surrounding the infarct is normal heart muscle. This scar is months to years old, and is the result of an infarct due to coronary atherosclerosis.

(Middle left.) The inner surface and cut section of heart wall at left show thin scarred area, due to old infarct, with normal muscle wall above and below. The scarred area is thin because the inelastic scar stretched and bulged (aneurysm of heart).

(Lower left.) At lower left can be seen a large blood clot (thrombus) adherent to the lining of a dead area (infarct) of the heart. Such a clot is a common result of an infarct. Should a portion of this clot break loose and enter the circulation, it would cause obstruction of some distant artery wherever it might lodge.

(Above.) This shows the outside of the heart at the area damaged by an occluded coronary artery. An arrow has been inserted through the hole in the heart wall where the weakened muscle allowed perforation to occur.

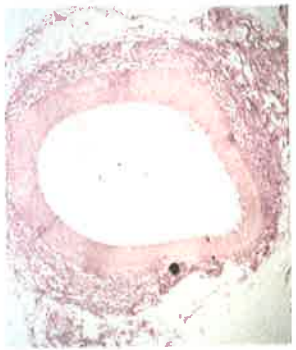
Arteries Get Hurt Too

It now appears that to the smoker, coronary heart disease is a greater threat than that of all other diseases combined. Smoking accelerates the process which leads to blockage of arteries and eventual damage to the heart itself.

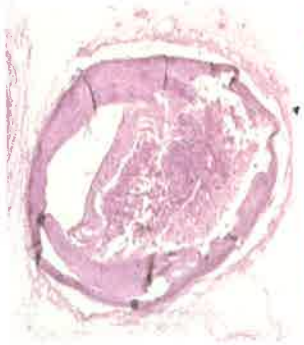
(Upper left.) In these sections of a normal aorta of an eleven-year-old child note the glistening, smooth surface of the inner lining of the aorta. The holes are the openings where smaller arteries branch off.

(Upper center.) This aorta is from a woman who had smoked for fifty years, attaining a rate of forty cigarettes a day. The inner surface of this large blood vessel is covered with plaques of fatty material, some of which show ulcerations and adherent blood clots. The entire lining is very rough.

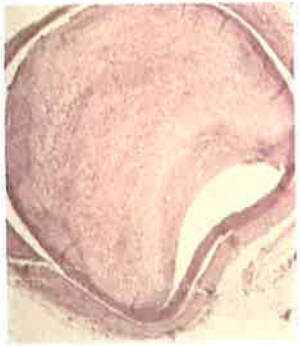
(Upper right.) Here is a cross section from a normal coronary artery. Note the unobstructed channel through which the blood can flow freely.



(Lower left.) Another view of a normal coronary artery indicates that the wall width is one fifth the width of the channel. Here again the smooth inner surface allows free, unobstructed flow of blood.



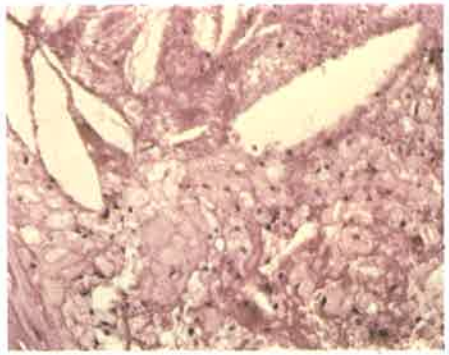
(Lower center.) In coronary atherosclerosis this fatty plaque ("atheroma," cholesterol material) has built up in this artery until the vessel is about three fourths obstructed.

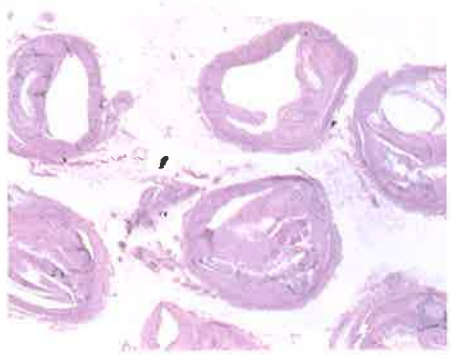


(Lower right.) In extreme coronary atherosclerosis the mushy plaque almost completely obstructs this artery—only about one tenth the normal space remaining through which blood may flow.

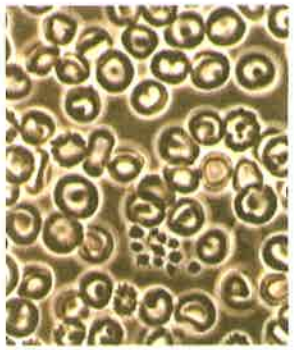
(Opposite page, upper left.) A microscopic view of a typical atheroma obstructing an artery. The elongated blank spaces represent areas where crystals of cholesterol had dissolved out in the preparation of the mount. The circular cells in the lower half of the picture are "foam cells," or phagocytes, full of cholesterol. These are the earliest trace of the deposit of this fatty material.

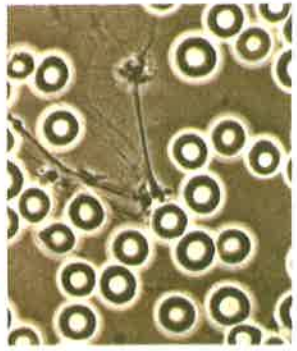
(Opposite, upper right.) This shows five sections from different cases of coronary atherosclerosis mounted on the same slide to illustrate advancing stages of the disease. In the two upper sections, the coronary is narrowed about 10 percent, the one on the right, about 60 percent, and the two lower sections about 80 percent and 90 percent narrowed.



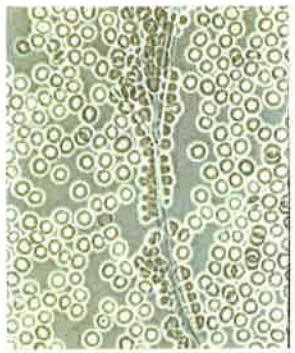


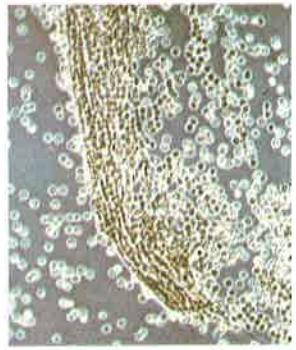
Making of a Blood Clot





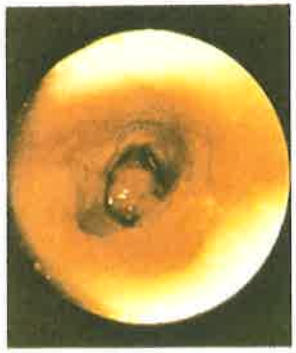
This is a red blood smear (second row, left). The small dark objects collected toward the bottom are platelets, the source of a clotting factor, thromboplastin.





When platelets rupture (second row, right) as a result of tissue injury or turbulent flow, they release thromboplastin. This results in the conversion of a soluble protein in the blood plasma, fibrinogen, into the small strands of fibrin shown here.

The red blood cells quickly adhere to these strands of fibrin (third row, left) in increasing numbers (third row, right) until the artery is completely obstructed (bottom, left) to the flow of blood.



In one of the coronary arteries of the heart (bottom, right) this clot (here shown enlarged) completely occludes or plugs the blood vessel.