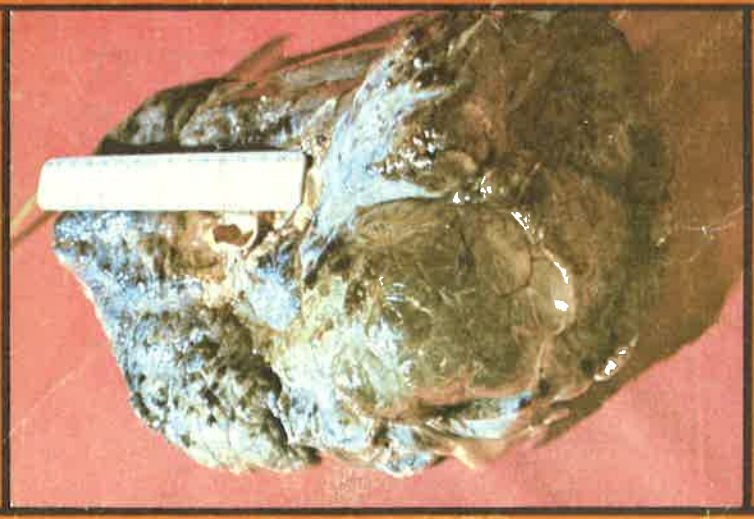


An enlarged, inflated emphysematous lung fills the lung cage of the chest and at the same time has little ability to exchange oxygen and carbon dioxide gases. Note the large bleb at the lower right. The destruction of the air sacs as seen here in these balloon-like blebs occurs throughout both lungs causing increasing difficulty in breathing.

Litho in U. S. A.

If you smoke -- here's what your doctor may see.

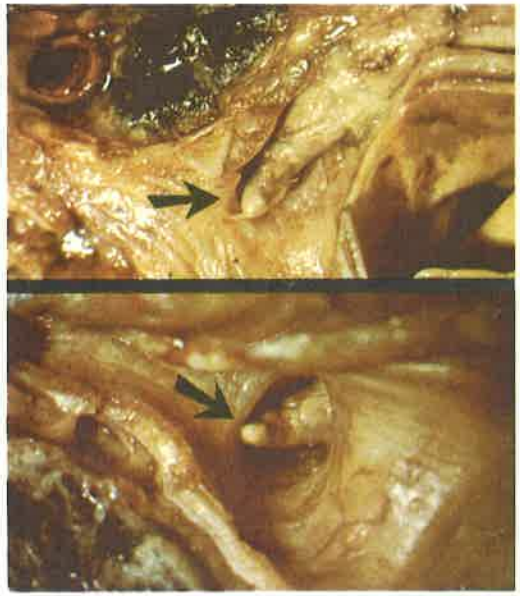
A pathologist's view of the effects of smoking on the human body

FOR SMOKERS, the lung is target for the constant irritation of tars and chemical agents from cigarette smoke which may result in the development of lung cancer, here shown by the whitish area in an otherwise blackened lung.

FOR NONSMOKERS, here is an inflated, essentially normal lung. This comes from a city dweller forty-seven years old who has breathed the usual amount of smog. The black spots are caused by carbon particles from air pollution and should be distinguished from the tar deposits on the walls of the breathing passages which lead to cancer in a smoker. "The effect of smog is nil" in causing cancer, says the Surgeon General's Report.

C. L. Dale, M.D., pathologist, Hinsdale Sanitarium and Hospital, Hinsdale, Illinois 60521

Photography by G. T. Hewlett



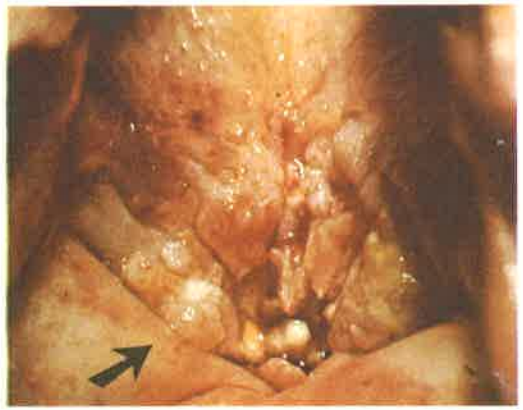
This cancer is developing in a major breathing passage and is caused by chemical cancer agents in the tar of tobacco smoke. Note that it begins in the inner wall of the breathing channel and then spreads into surrounding lung tissue. Blackness in the upper left indicates the large amount of smoking resulting in this cancer.

Lung Cancer

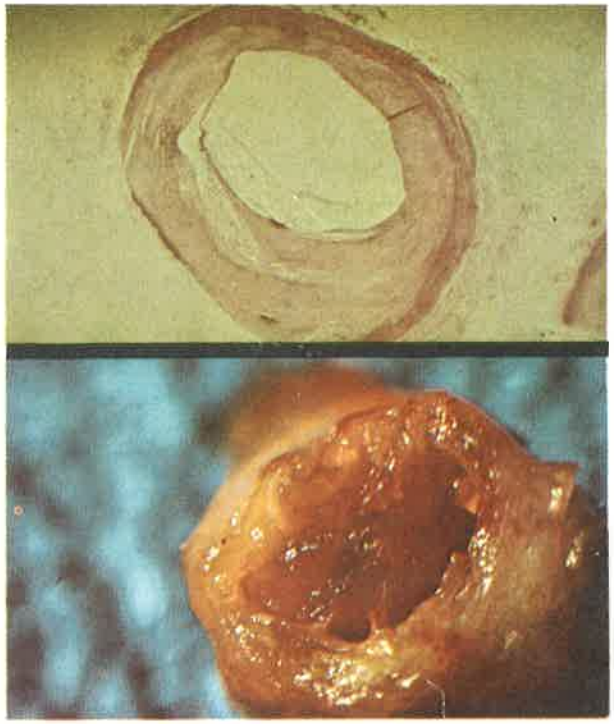
When a smoker takes a typical long drag on a cigarette, he follows with a deep inhalation that pulls the smoke into the farthest recesses of the lungs. It is as if every one of the hundreds of thousands of air sacs is clamoring to be filled with the tar-bearing, nicotine-laden, gaseous mixture.

In this process, the sticky tar with its many chemical constituents, including several cancer-causing agents, is deposited on the mucous membrane of the entire bronchial system—air passages of the lungs. The combined tar, chemicals (some cancer-causing agents), and gases irritate the mucous membrane. The cilia, or hairlike structures that sweep foreign matter from the air passages, are paralyzed. The tar continues to collect on the mucous membrane, and after a variable number of years the mucosal cells become altered. Many flatten and increase in number. The nuclei become larger, the cells heap up and then begin to grow inward. This produces a cancer in many individuals after a period of years.

The cancer spreads into the surrounding lung structure and invades lymph and blood vessels. Groups of the cancer cells separate and are carried by the lymph and bloodstream to other parts of the body to develop into metastatic tumors.



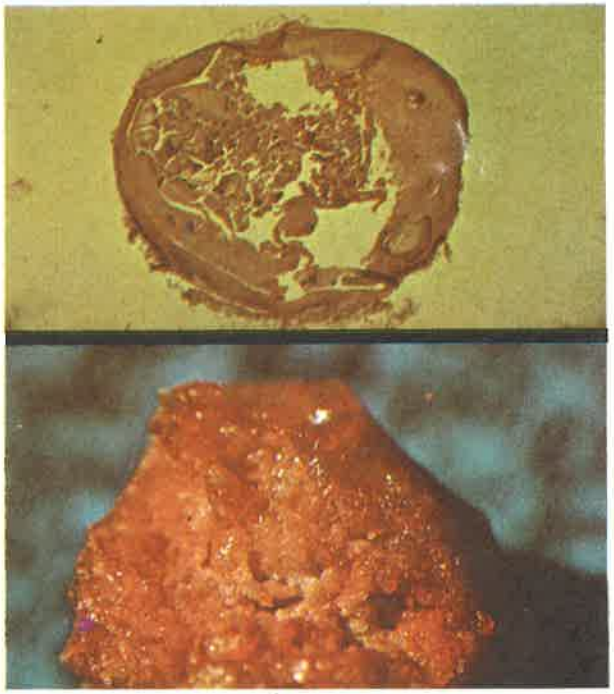
As the doctor views the voice box, he sees that one vocal cord has been destroyed by cancer. The other vocal cord is being invaded.



This is a cross section of an average coronary artery, showing some cholesterol deposit.

This coronary artery of the heart has thickened walls with resulting decreased interior diameter of the blood vessel. Note the deposit of cholesterol in the inner wall which has partially obstructed the artery. One action of nicotine is to constrict the small to medium-sized arteries such as this one, causing further decrease of the blood supply to the heart muscle.

Arteries Get Clogged Up!



This is a coronary artery showing excessive deposits of cholesterol.

This end view of a coronary artery shows an almost complete obstruction by cholesterol deposits. This prevents the free flow of blood.

This inner wall of a healthy artery shows branches leading off from the main blood vessel. Note smooth surface with lack of cholesterol deposit.

Two arteries—the upper from a 32-year-old high-fat eater, the lower from a 34-year-old low-fat eater. Note the cholesterol deposits in the inner wall (upper). Evidence now indicates that nicotine increases the cholesterol deposit.

What About Cholesterol?

This close-up of an artery with heavy cholesterol deposit (arteriosclerosis), indicates that ulcerations result, also calcification or hardening.

Severe arteriosclerosis brings about destruction of the artery. This one has been replaced by a nylon graft.

Smoking and Arteriosclerosis

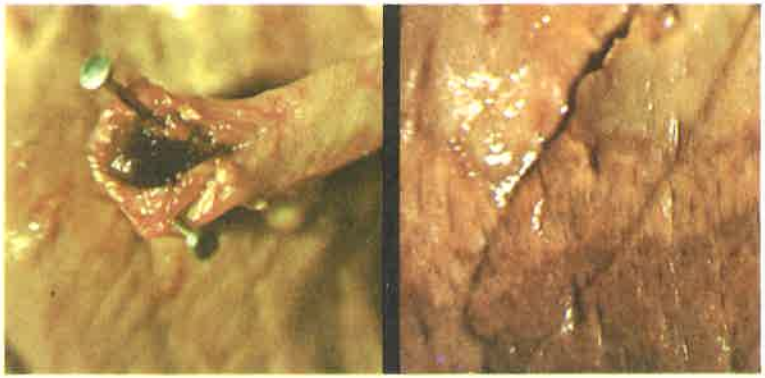
Hardening of the arteries (arteriosclerosis) today causes some 54 percent of deaths in the United States. It is widely known that this disease is related to high-fat diet.

There is now increasing evidence that nicotine, and possibly other chemicals absorbed from various forms of tobacco, enhance the deposit of this fatty material, chiefly cholesterol, within the inner walls of the arteries. The medium-sized arteries supplying the heart, brain, extremities, and other organs with blood become increasingly less able to furnish enough blood to these organs because of the decreased size of the lumen, or inner diameter of the blood vessel. Tissue damage results, often producing death by heart attacks, strokes, et cetera.

The absorbed nicotine also causes constriction of these same arteries, combining with the effect of partial fatty obstruction. This results in more severe heart attacks and strokes, which also occur earlier when tobacco is used. People who have had heart attacks, strokes, vascular problems of the extremities, and similar conditions should definitely stop smoking.

Pinned to a heart muscle, a part of a coronary artery is opened to show its thickened walls and a blood clot (thrombus) that has stopped the flow of blood. This is so-called "arteriosclerotic coronary thrombosis" which causes many of the serious heart attacks.

Damaged heart muscle has a mottled appearance. The lighter shade indicates greater damage. This is "myocardial infarction," which if extensive enough causes the patient's death or if it heals leaves a white scar.



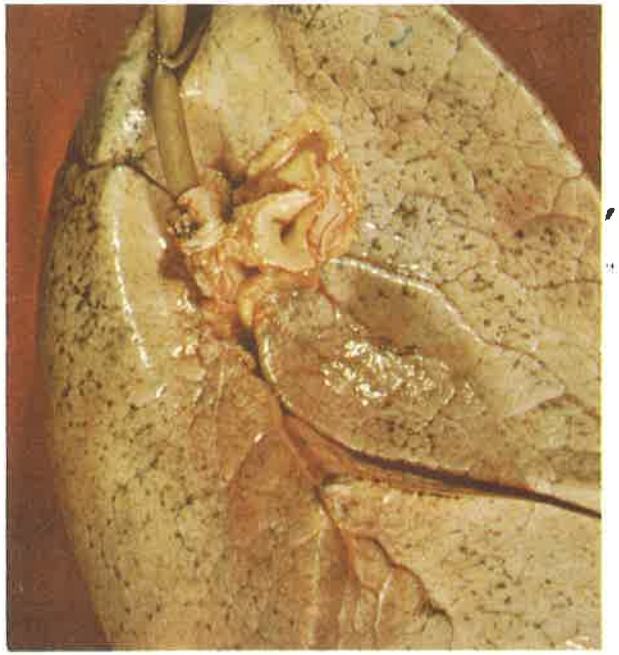
Smokers May Suffocate

Pulmonary emphysema is a disease that is increasing rapidly. The severe cases are most often found in relatively heavy cigarette smokers. Emphysema and lung cancer have much in common as to cause—changes in the bronchial mucosal epithelium—surface cells of the air passages of the lungs.

In pulmonary emphysema the heaping up of the epithelium produces partial obstruction of the tiny air tubes of the bronchial tree. The air is able to enter the air sacs more readily than it can exit. The result is gradual distention of the air sacs with eventual rupture of many sac walls, resulting in larger and larger balloon-like sacs. These cause blebs, or distentions of the surface. The process occurs throughout both lungs. The air sac walls have also been damaged by losing their elasticity as a result of the chemicals in the gases and tars.

Nicotine and other chemicals in the cigarette smoke produce arterial changes that help to cause damage to the air sac walls. The emphysematous lungs do not absorb oxygen and expire carbon dioxide sufficiently to enable the victim to carry on necessary functions of normal life.

Outer view of an emphysematous lung. The clearly visible blebs, balloon-like distentions of the surface of the lung, are the results of emphysema caused by smoking. Compare the color, shape, and size of this lung with the near-normal lung. The blackness is carbon particle deposits which do not cause the emphysema but do indicate the amount of prolonged smoking over years of time.



Inner view of the lung shown on page 1. The tube was used to inflate the lung. Note the color, shape, and general appearance of this lung. The small dark spots are carbon deposits acquired through breathing city air or smog.

From Healthy Lungs to Broken Air Sacs

The inner tissue of an emphysematous lung degenerates into stringlike shreds, the remains of the walls of the alveoli (small air sacs). The continuing dilating and rupturing of the alveoli produce the large combined blebs and useless lung structure.

Lung tissue similarly destroyed by emphysema shows the remains of air-sac walls. The bleb cavities are nonfunctional even though they are distended with air, since the oxygen and carbon dioxide are not exchanged as in the normal action of air sacs.