medical sciences

Gathered last week from the 66th annual meeting of the National Tuberculosis and Respiratory Disease Association and the American Thoracic Society in Cleveland, Ohio

LUNG CANCER

Checking sputum cells

Subtle changes in the chest often do not show up in chest X-rays. A search for abnormal cells in bronchial secretions, says Dr. D. S. Saksena of the University of Utah Medical Center, can be useful as a backup method for early detection of lung cancer.

He reports that the cytologic examination of sputum accompanying chest X-rays has proved especially valuable in following the postoperative course of patients.

At the university, sputum analysis is done every three months and chest X-rays every six months on high-risk lung cancer patients and those who have already undergone surgery. Dr. Saksena says that newly forming tumors have been detected in eight patients who had normal-appearing chest X-rays.

In one patient, he adds, diagnosis of three different primary carcinomas in different parts of the lung was made by study of the sputum prior to any significant changes in X-ray films. The patient is alive today, four years after his first resection.

The technique is simple and noninvasive, requiring no more than a sample. Samples can, if necessary, be easily prepared for shipment.

TRAUMA

Treating lung complications

With severe chest wounds, there is often a resulting lack of oxygen in the blood, reports Dr. J. R. Hankins of the University of Maryland's Baltimore-based Center for the Study of Trauma. Treating this problem along with treatment of wounds prevented mortality in each of 14 patients with severe chest trauma.

To find oxygen loss, Dr. Hankins performed standard physiologic determinations during treatment of 14 of 17 patients with severe chest injuries, who showed significant loss of oxygen in the blood.

With oxygen lack diagnosed, says Dr. Hankins, respiration was supported early with mechanical ventilators. The bronchi were examined through a bronchoscope and a tube inserted through the trachea to facilitate passing of air to the lungs. As a result, no patient died of pulmonary complications, he reports.

POLLUTION

Ozone and lung disease

The deleterious effects of air pollution on the lungs is increasingly evident. A recent addition to the evidence is a study by a Harvard University investigator who claims that ozone concentrations as low as 0.10 parts per million suppress the capacity of the body to combat infection. Thus oxidant pollution may play a role in chronic and acute pulmonary disease.

Ozone, a pungent, colorless toxic gas, is produced from a photochemical reaction of the solar rays on automobile exhausts. In the study, Dr. G. L. Huber of the Harvard School of Public Health in Boston delivered ozone mixed with room air at levels of 0.10, 0.50 and 1.00 parts per million to 450 mice for 3 hours and withheld it from an additional 250 controls. All animals inhaled *Staphylococcus aureus*.

Results showed that concentrations of 1.00 parts per million markedly depressed the defense mechanism; only about 52 percent of the inhaled bacteria were rendered inactive. With controls, up to 93 percent of the bacteria were inactivated. At levels of 0.10 parts per million ozone, 86 to 90 percent of the bacteria were made inactive and levels of 0.50 parts per million ozone resulted in 69 to 76 percent inactivity.

SMOKING

Agitating defending cells

The harmful effects of smoking on respiratory health are still not clear to physicians. One theory recently expressed by Lt. James O. Harris, M.D., chief of the chest service of the United States Naval Hospital in San Diego, Calif., is that if smoking itself doesn't damage the lungs, perhaps the macrophages, the large mononuclear cells that defend the body, might. These cells are marshalled into combat to protect the lungs against the invading irritating particles in smoke, he says.

A study of five nonsmokers and six smokers showed that chronic smoking elicited a chronic mononuclear inflammatory reaction in which highly energized and agitated macrophages were greatly concentrated.

In the study, Dr. Harris collected from the lower lobe fluid used to wash out the lungs. He found an average of 47 million macrophages per lavage, in contrast to 10 million macrophages of the nonsmokers. Lysosomal bodies with distinctive crystal structures were typical of macrophages from smokers but not from nonsmokers. The lysosomes contain enzymes that could be damaging.

EMPHYSEMA

Links with heredity

Emphysema may be linked to heredity, reports Dr. Paul M. Stevens of Baylor University in Houston.

The Texas physician studied three relatives typifying the familial pattern of emphysema. One was a homozygote, with two defective genes, and two were heterozygotes, with only one defective gene each. All patients, he reports, had varying degrees of deficiency in alpha-1 antitrypsin, a genetically formulated immunochemical that inhibits trypsin.

Trypsin, an enzyme that breaks up food proteins and renders them more easily absorbed into the body, may be released in the lungs from circulating white blood cells. Unless inhibited by alpha-1 antitrypsin, trypsin can destroy the lungs by breaking up certain proteins of the elastic lung tissue in the same manner.

Normal persons with normal genes, says Dr. Stevens, average about 375 milligrams of inhibitor per 100 milliliters of serum. In the Texas study, antitrypsin deficiency ranged from 25 to 60 milligrams per 100 milliliters among homozygotes and 100 to 200 among heterozygotes.