"THEY ASKED ME HOW I KNEW"

Smoke Gets in Your Lives

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In the regional Emphysema Clinic we see hundreds of patients each year who are referred for diagnostic studies or therapeutic guidance because of chronic pulmonary disease. Thirty years ago such a clinic would have been unnecessary. Furthermore, the Georgia Regional Medical Program has asked us to aid other health facilities in providing similar services. Why?

In each of the last two decades, there has been a doubling of mortality from "chronic obstructive bronchopulmonary diseases" (COBPD), primarily emphysema and chronic bronchitis. Why?

Although initially this rise was a problem affecting men, recently this same rise has begun among women. Why?

Chronic obstructive bronchopulmonary disease is now the second most common reason for Social Security Disability payments in the United States (Cardiovascular diseases being first). Why?

Everyone who sees patients with chronic obstructive bronchopulmonary disease is struck by the fact that almost all (close to 98%) are or were heavy smokers. It is interesting to correlate the rise in cigarette smoking with that of COBPD. Begun on a small scale in the early 1900's, smoking became a common practice and a status symbol of adulthood and sophistication among men in World War I. Among women, smoking was rare until World War II. The rise in COBPD thus follows that of cigarette smoking with a thirty year lag ("You've come a long way, baby," say the folks at Virginia Slims).

Voluminous medical literature summarized in the Surgeon General's report, "Smoking and Health," clearly links cigarette smoking and chronic obstructive lung disease, as well as lung cancer and several other health problems. Yet we are still surrounded by advertising which claims "Never a rough puff" (Kool advertisement showing a sail boat on a clear blue sea), or "Spend a milder moment with Raleigh" showing an outdoor-type young man building a log cabin in the woods. This same strong male outdoor type will show up thirty years later as a pulmonary cripple.

A typical patient seen in our Clinic or in a physician's office will be a man in his late 50's or 60's, just at his time of peak ability and earning power. Frequently, he has had a "cigarette cough" and slowly progressive shortness of breath on exertion for many, many years. Yet he himself often has not known that these are symptoms denoting abnormality and danger. Even after consulting a physician, he may not be diagnosed and is often left without benefit of specific testing for functional impairment such as pulmonary function tests. Even if diagnosed, he is rarely given the benefit of a complete and integrated treatment program, including specific advice on cigarette smoking and environmental control, infection prevention, recognition and treatment, bronchodilator therapy, and physical therapy and rehabilitation. Why?

Perhaps more rapidly than necessary, the patient's symptoms become disabling and he loses his job and often his insurance coverage and retirement benefits. In addition, he becomes a burden to society. The enormous and tragic problems faced by the patient personally, by his family, and by society as a result of chronic illness can only be fully understood by those in a close contact with these patients.

Of course, other factors besides cigarette smoking enter into the pathogenesis of chronic bronchitis and emphysema. One of great public concern at the moment is air pollution, particularly that produced by the automobile. Several convincing studies have shown a link between heavy short-term exposure to air pollution and excess mortality in patients with heart and lung disorders, as well as a link between chronic lung disease and residence in high pollution areas on a long-term basis.

A third factor is the possibility of genetic predisposition to these disorders. Familial pulmonary emphysema was first described in Sweden in patients with alpha$_1$ antitrypsin deficiency. Since then it has been shown that patients with homozygous antitrypsin deficiency almost universally develop emphysema, usually more severe in the lower lobes occurring equally in men and women, and causing disability at an early eage, i.e., the thirties. The occurrence of a heterozygous state among the population of COBPD patients as a whole has been variously reported to be between 10-60%.

In the future, genetic counselling for affected individuals will become more commonplace. In addition, large-scale childhood screening for alpha$_1$ antitrypsin deficiency may become possible. Affected individuals could then be counseled in regard to residence, environmental control, jobs, and personal respiratory care.

Compared with the leadership exerted by the medical profession in fighting such health problems as polio, measles and rubella, the efforts exerted to control the much larger health problems produced by such factors as automobile accidents, alcoholism, and cigarette smoking seem very feeble. As physicians we must strive, both on a personal and an organized level, to control genetic factors, air pollution and most of all, the strong personal air pollution secondary to cigarette smoking. In particular we must counteract the all-pervasive advertising *which tries to correlate cigarette* smoking with youth, pleasure, naturalness, the good life, and fresh air. We are so surrounded by coccoons spun by advertising's silver lies that we do not even notice any longer the utter preposterousness of their claims.

In the Emphysema Clinic we will continue to serve those patients already affected by respiratory problems, but the future in conquering these disorders lies in prevention; and the most obvious factor in this regard is cessation of cigarette smoking as common practice. □

Living (?) Proof

write-up by Dick Dussia, EUSM '75
of a patient seen at Grady Memorial Hospital

INCOMPLETE PROBLEM LIST

5/6/72 Problem No. 1. Coronary Atherosclerotic Heart Disease
- a. Anterior Myocardial infarcation. Age undetermined.
- b. Inferior myocardial infarcation. Age undetermined.
- c. Coronary incident. (5/72)
- d. Angina Pectoris; progressive.

PROGRESS NOTE

Pre-Cardiac Catheterization Note

11/2/72 Problem No. 1d. Angina Pectoris; progressive

Subjective--the patient is a forty year old white male with an eight month history of retrosternal exertional "indigestion." There were three episodes of prolonged discomfort for which he was hospitalized twice, five and three months ago. Now he experiences nocturnal pain, post prandial pain, and pain at rest in spite of intensive medical therapy. He has had to cease work as a salesman and is limited to a bed and chair existence because the slightest effort brings on his pain.

Objective--Cardiovascular exmination: BP 110/70 in both arms; pulse 80 and regular; JVP's are not seen at 30 degrees; carotid upstroke normal; chest clear to auscultation; late systolic parasternal lift palpable on anterior precordium; S1 normal intensity and S2 splits normally; S4, but no S3 present; no murmer; all peripheral pulses are present and of normal intensity; no xanthomata are present.

ECG: Normal sinus rhythm with inferior and anterior dead zones; non-speoific ST-T change.

Chest Film: Cardiomegaly with no intracardiac calcification or obvious ventricular aneurysm; equivocal pulmonary venous hypertension is present but there is no obvious interstitial pulmonary edema.

Assessment--severe disabling angina pectoris; an area of abnormal chest wall movement--questionable ventricular aneurysm.

Plan--Coronary angiography and left ventricular cineangiogram to look for any lesion that may be surgically treatable such as isolated segmental coronary disease and/or ventricular aneurysm.

Post Cardiac Catherization

11/2/72 Problem No. 1d. Angina Pectoris; progressive

Subjective--

Objective-- Left and right heart catheterization with left ventricular cineangiogram in RAO and LAO position with cine-coronary angiograms done without complication.

-- *Tentative Findings*

Left ventricular aneurysm of anterior wall.

Diffuse three vessel disease. Diffuse disease of both branches of the left coronary artery and complete obstruction in the proximal one third of the right cornonary artery.

Assessment --
Plan-- pending final evaluation of data.

Although the final cath report was not available at the time of this writing, the most pertinent piece of data obtained was that the patient's ejection fraction, or that portion of the blood in the left ventricle at the end of diastole that is ejected during mechanical systole, was 0.17. That means that only 17% of the ventricular volume was ejected. The normal ejection fraction is about 0.66 or 66%. The patient's coronary "risk factors" were listed as obesity (5'4", 190 lbs.) and smoking (1-1 1/2 packs per day for 25 years). □

"Maybe someday cigarette smoking in public will be like (breaking wind)." -Wayne Hardwick EUSM '75

ANLAGE

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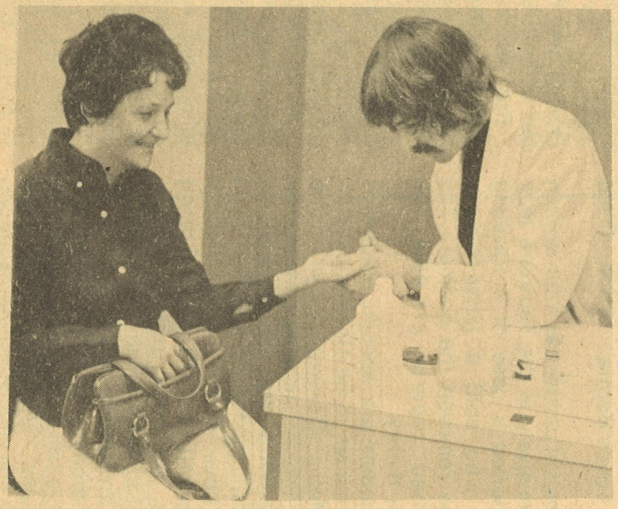

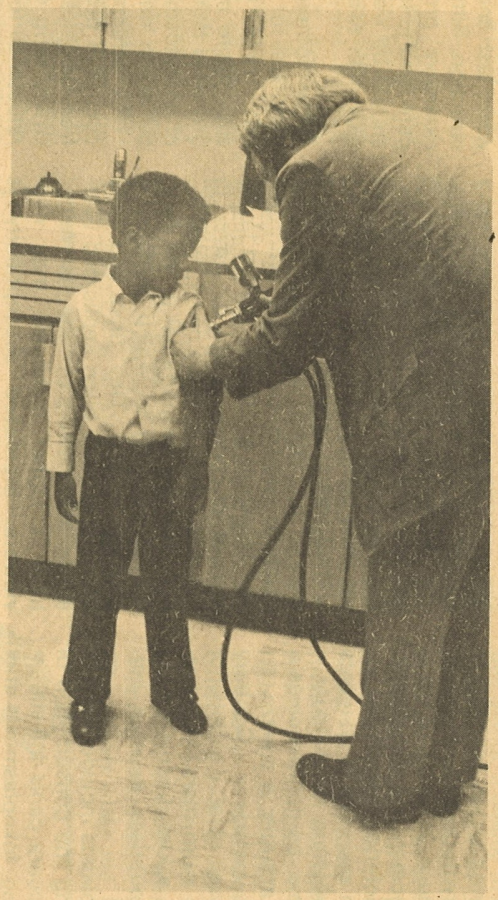

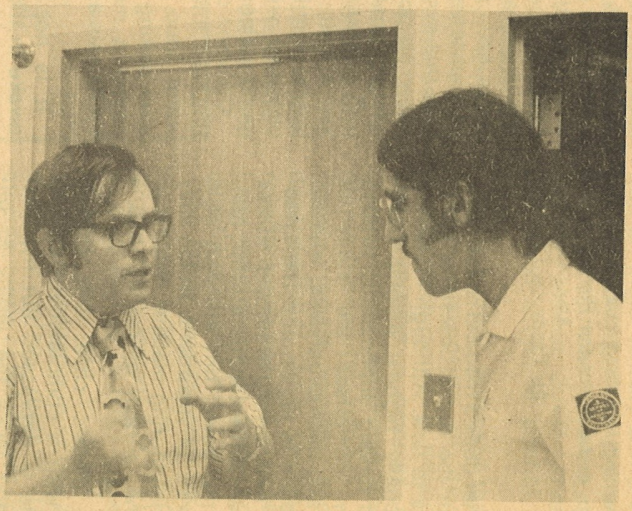

Scenes of the Newton County Health Fair, organized by Emory medical students. *See page 5*