and, therefore, may inhibit autoregulatory vasodilatation distal to an area of coronary vascular spasm. Therefore, therapy with propranolol may unmask or potentiate vasoconstriction mediated by $\alpha$-adrenergic receptors.

Bennett concludes that the administration of propranolol may produce coronary vascular spasm. While coronary vascular spasm is now recognized with increasing frequency, there is little evidence to support the hypothesis as presented.

If, as proposed, therapy with propranolol inhibits autoregulatory coronary arterial vasodilatation, it should frequently worsen angina pectoris. This is not the case clinically. We doubt that there is any significant difference in the dissipation of the effects of propranolol therapy from sites which block autoregulatory vasodilatation than from other areas in the coronary arteries.

Thus, we conclude that the postulates of Bennett are highly speculative. The hypothesis appears to have little practical meaning at this time.

**Correction**

To the Editor:

My attention has been drawn to a disconcerting error in the Committee Report entitled "The Pulmonary Response to Fiberglass Dust: Report of the Committee on Environmental Health, American College of Chest Physicians" (Chest 69:216-219, 1976).

In the fourth line from the bottom of the first column on page 216, the sentence should read "thinner than 3.5$\mu$ in diameter" (instead of "greater than").

Paul Gross, M.D., F.C.C.P.
Naples, Fla

Eosinophilic Pneumonia

To the Editor:

Salicylazosulfapyridine (sulfasalazine; Salazopyrin) is widely used for the treatment of chronic ulcerative colitis; it is often used in conjunction with corticosteroids in the treatment of the acute phase of the disease. Side effects are numerous, including nausea and vomiting, jaundice, blood disorders, headaches, skin disorders (including erythema nodosum and erythema multiforme), abdominal pain, dysuria, pyrexia, etc.

This case illustrates an unusual side effect of the drug causing pulmonary eosinophilia; pulmonary lesions due to administration of salicylazosulfapyridine are extremely rare.

**CASE REPORT**

A woman aged 23 years had a three-year history of ulcerative colitis treated with salicylazosulfapyridine (Salazopyrin), codeine phosphate, and ferrous sulfate. Therapy with salicylazosulfapyridine was discontinued during her pregnancy and restarted after her delivery.

In November 1974, the patient developed dyspnea on exertion, cough with mucoid sputum, central chest pain unrelated to effort, loss of energy, malaise, poor appetite, and weight loss. Her general practitioner diagnosed tracheitis, and antibiotic therapy was administered for a few days without any effect. When the patient was referred to the Chest Clinic, she was pale, feverish, and short of breath, and there were some scattered crepitations in both lungs.

A chest radiograph showed widespread ill-defined foci of soft infiltrations in both lungs (Fig. 1). The findings from blood investigations were as follows: hemoglobin, 10.3 gm/100 ml; white blood cell count (WBC) 7,200/cu mm; neutrophils, 57 percent; lymphocytes, 25 percent; monocytes, 2 percent; eosinophils, 14 percent; and basophils, 2 percent.

Although consecutive direct smears of sputum for acid-fast and alcohol-fast bacilli were negative and a Heaf tuberculin skin test was negative, the combination of chest radiographic findings with the evening fever and nearly normal morning temperature, the chill and night sweats, the weight loss, the progressive and eventually alarming dyspnea, and the cough productive of a variable amount of mucoid sputum suggested a diagnosis of tuberculosis. In addition to the previous medications, the patient was given antituberculous chemotherapy with isoniazid, ethambutol, and rifampin, but her condition deteriorated. A new blood cell count showed leukocytosis and eosinophilia with a WBC of 13,000/cu mm and 50 percent eosinophils. The erythrocytic sedimentation rate was 60 mm/hr. A new chest radiograph revealed that the pulmonary infiltrates were migratory.

A diagnosis of pulmonary eosinophilia was made. Tubercu-
lostic treatment and therapy with salicylazosulfapyridine were discontinued. Although corticosteroid therapy was not prescribed, the patient’s condition improved considerably within a few days; the chest symptoms subsided, and the chest radiograph returned to normal. Seven weeks later, the WBC was 7,000/cu mm, with no eosinophils.

**DISCUSSION**

Eosinophilic pneumonia is a pattern of pulmonary reaction that is thought to represent an immune or hypersensitivity response. Pulmonary infiltrates may be self-limited and of less than one month’s duration (Löffler’s syndrome) or prolonged or recurrent, and may or may not be associated with episodic broncho-obstructive disease.

The syndrome can be produced by a variety of etiologic agents, including drugs. Hypersensitivity to salicylazosulfapyridine, although unusual, should be considered in patients with pulmonary lesions who are being treated with the drug.

**REFERENCES**


**Computerized Electrocardiography**

**When and How Do We Go?**

To the Editor:

A prolonged clinical study of many computerized electrocardiographic programs and their hardware is presented. During this period, no system suitable for use in the community hospital or for mass-screening was found.

After millions of dollars and the many man-hours that have been spent on this apparently hopeless project, it would be sensible to admit that, clinically speaking, computerized electrocardiography without fully competent over-read is dead (Wiley Winsor Foundation report and written communication from Travis Winsor, M.D., Feb 4, 1976). As early as 1964, Burch and Depasquele believed that “analysis of low fidelity tracings with complex high fidelity computers appears to be absurd.” These words may well have been prophetic.

The defects detected in these years of exhaustive clinical study could be summed up simply as finding that present programs lack (1) adequate acquisition and transmission, (2) adequate rhythm analysis and printout language, and (3) adequate hardware. The present programs also have other physical defects too numerous to mention but certainly well known to workers in the field.

In view of the previously mentioned findings, we would like to make an appeal and offer some suggestions to the concerned individuals, design engineers, medical electronic personnel, and their companies.

For computerized electrocardiography to emerge as clinically useful and marketable and not to remain just an inadequate tool, the following criteria should be met: (1) the three previously mentioned defects must be corrected; (2) the system must be fast and relatively free of operational defects; (3) it must be determined as soon as possible whether or not digital transmission is desirable, even necessary, for a system to function, and whether the answer lies in computerized analysis of instantaneous vectors; (4) specific comparative testing of large numbers of vectorcardiograms, both normal and abnormal, simultaneously with standard electrocardiographic equipment must be conducted; (5) the package must be sensibly priced and appeal economically to all concerned users; (6) efficiency must be of such a nature and degree that all insurance carriers would gratefully accept the principle, as well as the conclusions of the printout; and (7) the system must be flexible enough to adjust to interchange of hardware.

It would benefit all of us who are interested in computerized electrocardiography and desirous of its widespread use to take a good look at the situation now or prepare for the “wake.” We must have innovative thinking in computerized electrocardiography now or forget it and leave it to someone else or to another generation.

Despite all of this and with a full knowledge that advances have always come slowly in medicine and cardiopulmonary resuscitation, we note that great potential has been shown and specific practical value delineated from computerized analysis of instantaneous vectors and the derived electrocardiogram.

**REFERENCES**

5 Kouwenhove WB, Langworthy OR: Cardiopulmonary

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