

The Lung as a Mirror of Systemic Disease

PART II—B

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Allergic Diseases

The number of diseases wholly or partly ascribable to hypersensitivity or allergic causes is considerable and the list is growing. It has been estimated that approximately 10 percent of the population suffers from some major form of allergy, chiefly bronchial asthma and hayfever. Of those normally non-allergic, many become sensitized after injections of antitoxin and serum or after treatment with drugs and antibiotics. Brown recently published an **abbreviated** list of 217 drugs and antibiotics which have been found to cause allergic reactions. Obviously allergic diseases represent a sizable segment of human ailments. To appreciate the pulmonary manifestations of this complex group of diseases, a few comments on certain basic aspects is in order, even in a review of this nature.

Allergy is a "condition of altered tissue reaction to substances or stimuli of a physical or chemical nature which in similar amounts provoke no response in the majority of members of the same species" (Urbach). In other words, an allergic individual develops immunity more readily and reacts more violently to a specific stimulus than one nonallergic. An allergic person is said to possess a lower threshold of sensitivity, an ill-defined attribute which varies greatly even in the same individual under different circumstances. Early investigators stressed the humoral component of the allergic reaction especially the role of circulating antibodies. In time the humoral theory gave way to the belief that practically all the fixed tissue cells of the body play a role.

The initial observations on allergy were in experimental animals when it was noted that after injection of a foreign protein a "shocking" or anaphylactic response took place. In 1905 von Pirquet and Schick reported the first clinical observations on anaphylactic reactions in children caused by hypersensitivity to horse serum. The term, allergy, was introduced by von Pirquet a year later to describe the skin reaction following a subcutaneous injection of tuberculin in those infected with the tubercle bacillus. Subsequent studies of allergy continued to center around the immunobiologic phenomena associated with tuberculosis. In 1910 Meltzer suggested that asthma is a manifestation of anaphylaxis, a belief prompted by earlier studies of Auer who noted bronchospasm and pulmonary distention in guinea pigs who died of anaphylactic shock.

The exciting agents responsible for the various manifestations of hypersensitivity may be chemical, physical or living agents and their products. The portal of entry of the allergen does not necessarily correspond to the site of its action, nor is the response constant. The nature of the "trigger" mechanism which sets off the explosive response is still debatable. The current theory stems from the work of Dale and his co-workers who noted that symptoms of histamine poisoning closely resemble anaphylactic shock. The observations of these investigators, as well as of Lewis on the responses of the

blood vessels of the human skin, serve to explain the allergic reaction as follows: when allergens enter the body of certain individuals for the first time they combine with protein molecules and induce antibody formation. These antibodies guard the organism against future inroads of the same antigen, the tissues having become alerted or sensitized to the offenders. Should similar allergens enter the body on later occasions, the specific antibodies combine violently with the invaders on the surface of sensitized cell membranes damaging the latter with the liberation of histamine, a normal constituent of tissue cells. Another theory is that liberation of the toxic substance is initiated by stimulation of parasympathetic (cholinergic) nerve fibers through the mediation of acetylcholine present.

The characteristic allergic reaction is featured by a sudden, often explosive, onset rapidly reaching a climax and a slow resolution. Although the tissue changes, as a rule, parallel the intensity of the reaction, there may be considerable variation. At one extreme one meets with violent functional disturbances, even sudden death, with minimal if any demonstrable tissue changes. This is exemplified in anaphylactic shock. At the other, one meets with reversible inflammatory changes of a temporary character which leave the individual none the worse as a result. Between extremes one encounters instances of permanent tissue damage of a proliferative, degenerative or necrotic nature of varying severity and prognostic implications. As the various elements entering into the allergic reaction become more complex, especially if the irritant is repetitive or of low intensity, and depending on the nature of the shock organs, the tissue changes are apt to lose their classical features. A stage is eventually reached when it becomes impossible to correlate the pathological changes with hypersensitivity, except for indirect evidence in the way of an allergic family or personal history, hematologic or histologic findings, the response to cortisone and other antiallergic treatment and similar suggestive but inconclusive indices.

In line with these remarks, it is clear that hypersensitive lungs may react to an excitant with varying intensity, ranging from a seizure of bronchospasm or transitory pulmonary infiltrations associated with eosinophilia, to rheumatic pneumonia or periarteritis nodosa or a combination of these. Similar tissue reactions may take place in other parts of the body concurrently or consecutively and escape clinical recognition. At times the symptom complexes may express themselves in bizarre syndromes such as eosinophilic granuloma of bone, intermittent hydrarthrosis, Meniere's syndrome, polyneuritis, nephrosclerosis and other conditions suspected of being allergic in nature. The more commonplace, of course, are allergic asthma, hayfever, angioneurotic edema, drug and food idiosyncrasies. In adulthood, the respiratory tract is particularly vulnerable to allergic irritation; with advancing years, the heart and kidneys bear the brunt; at all ages the reticuloendothelial system, composed of a reactive group of cells concentrated chiefly in the lymph nodes, spleen, liver, bone marrow and the loose connective tissue, take an active part.

It might be mentioned at this point that an excess of eosinophils in tissues and/or blood, sputum and other excreta characterizes allergic inflammatory disease. However, the absence of an eosinophilic response does not rule out an allergic cause; nor does its presence necessarily indicate the existence of

allergy since eosinophilic leukocytes in abnormal numbers may be present in association with leukemia, Hodgkin's disease, hydatid and other parasitic infestations, certain skin diseases and, on rare occasion, even malignant neoplasms.

The following conditions, believed to be allergic in nature and at times revealing pulmonary findings, will be described. Those associated with dermal changes will be discussed in the section to follow.

1. Bronchial Asthma
2. Transitory Pulmonary Infiltrations with Eosinophilia
3. Prolonged or Recurring Pulmonary Infiltrations with Eosinophilia, Pleuritis and/or Pericarditis
4. Periarteritis Nodosa
5. Rheumatic Pneumonia
6. Rheumatoid Arthritis

Bronchial Asthma: Bronchial asthma represents a state of hypersensitivity of the respiratory tract to air-borne or blood-borne excitants. As typified in the allergic skin reaction, the histology of which has been studied in detail, hypersensitivity of lung tissue to an irritant is likewise characterized by an outpouring of edema fluid in the bronchi and the establishment of an acute inflammatory reaction around blood vessels. In uncomplicated asthma the vascular response is relatively mild and limited; in hyperergic states the reaction is intense involving especially the small vessels and perivascular tissues. The exudate contains a preponderance of eosinophils; later mononuclear and polymorphonuclear cells appear. The bronchial exudate becomes viscid due to admixture of mucus from secreting glands. In protracted asthma there is desquamation of epithelium, distention of mucous glands, hypertrophy of the elastic fibers and circular muscles of the bronchi and dilatation of the bronchioles and alveoli.

The symptomatology of bronchial asthma ranges the gamut from a temporary seizure of wheezing to a status asthmaticus. The pulmonary manifestations depend on a number of factors especially the degree of damage sustained by blood vessels and bronchi and the obstructive phenomena caused by sputum retention. In addition, the type of asthma, whether it be of the so-called extrinsic or intrinsic variety, has an important bearing.

Extrinsic asthma is caused by inhalation of pollens of weeds, grasses and trees, dusts, animal emanations and molds. The disease appears at a relatively early age in individuals with decided allergic family histories. Additional signs of hypersensitivity, such as atopic dermatitis, allergic rhinitis, drug and food idiosyncrasies are often present. Other significant features include the obtainment of positive cutaneous reactions to offending allergens, minimal evidence of bronchopulmonary infection and usually a satisfactory response to antiasthmatic medication. The prognosis is relatively good. The chest x-ray may reveal an increase in the bronchovascular markings and some degree of hyperillumination of the lung fields, emphysema being the most frequent complication of bronchial asthma. During an acute paroxysm the roentgenogram may show evidence of overdistention of the lungs, as may be seen from the enlarged thoracic cage and depression of the diaphragm. Tenacious sputum impacted in bronchi may cause localized areas of atelectasis which, in obstinate cases, may require bronchoscopy for removal (Fig. 12).

Intrinsic asthma is characterized by its appearance later in life in individuals without an allergic background. A nondescript respiratory infection often antedates the onset of symptoms. After the episode, the individual becomes susceptible to recurring cough and expectoration especially during inclement weather. The symptoms may be temporarily improved by expectorants and anti-bacterial treatment, seldom by antiallergic medication. The picture is more in keeping with a chronic bronchitis, so-called asthmatic bronchitis, than with an allergic disease. Intrinsic asthma carries with it a worse prognosis than the extrinsic variety because of the repeated damage sustained by the lungs during recurring infections and the fact that the resulting fibrosis and emphysema may lead in time to chronic cor pulmonale.

The roentgen findings of advanced bronchial asthma, irrespective of causation, reflect chiefly the effects of bronchial obstruction and infection. As mentioned, the trapping of air by tenacious plugs of sputum in bronchioles gives rise to increasing alveolar distention and emphysema. Air pockets (bullae) may form within the parenchyma and blebs under the pleura. These in turn may cause complications. Air escaping from alveoli may pass along blood vessels and bronchi to the mediastinum giving rise to pneumomediastinum. Rupture of the pleura may give rise to pneumothorax on one or repeated occasions (Fig. 13). Following the development of interstitial emphysema, air may collect under the skin of the neck, chest and abdomen giving rise to variable degrees of subcutaneous emphysema. If the pressure is sufficiently high, air may escape into the abdomen and give rise to retroperitoneum. A more detailed discussion of these and other complications of bronchial asthma is beyond the scope of this presentation.

Transitory Pulmonary Infiltrations with Eosinophilia: This condition is characterized by irregular infiltrations scattered in both lungs, usually of a temporary nature, at times persisting or recurring. The chest x-ray reveals densities of varying configuration and distribution. Upper lobe infiltrations simulate tuberculosis (Fig. 14); lower lobe infiltrations, atypical pneumonia. In a case reported by Elkeles and Butler, a transient ring shadow in the center of an apical infiltration was suspected of being a cavity in an eosinophilic lung. The additional feature which sets this type of pulmonary disease apart from others is the presence of eosinophilic leukocytes in the blood and often in the sputum. In spite of widespread pulmonary involvement, the disease is usually asymptomatic; occasionally, hyperacute.

The association of transitory pulmonary infiltrations and eosinophilia was brought to medical notice by Loeffler in 1932 and the condition is often referred to as Loeffler's syndrome. A number of variants of the original description have since been reported and included under the same heading, the symptom-complex being quite variable. Several of these will be referred to shortly. It might be mentioned at this point that many individuals are now receiving penicillin, streptomycin and other antibiotics, as well as liver injections and drugs (reference was made previously to Brown's list of 217 such allergizing agents) which may cause eosinophilia. An intercurrent atypical pneumonia during such treatment may be taken, rightly or wrongly, as a manifestation of Loeffler's syndrome. The writer has been puzzled by several such experiences.

The pulmonary infiltrations in Loeffler's syndrome represent areas of eosinophilic pneumonia. There are a number of instances on record of pa-

tients who had such infiltrations and who died accidentally. Examination of the organs confirmed the histologic nature of the disease. In four such cases Meyenburg found the lungs to contain eosinophilic leukocytes and giant cells, marked interstitial proliferation and serous exudation. He also noted minute granulomata and vascular alterations, the significance of which will be emphasized in the discussions on rheumatic pneumonia and periarteritis nodosa. A case report with autopsy findings published by Bayley, Lindberg and Baggenstoss likewise revealed lesions consisting of pneumonic exudate containing large numbers of eosinophils. These authors were impressed with the advanced stage of organization of the exudate, the presence of small granulomatous lesions and the occurrence of necrotizing arteritis and phlebitis. Fibrinoid swelling and necrosis of collagen were prominent features and suggested an allergic basis.

Pulmonary infiltrations associated with eosinophilia occurring in asthmatics and others exposed to allergizing agents should be distinguished from a similar picture encountered in, so-called, "tropical eosinophilia." This syndrome, described by Weingarten on the basis of a study of 81 cases encountered in Bombay, India, is characterized by paroxysms of asthma, weakness, loss of weight and appetite and marked leukocytosis. Tropical eosinophilia has since been reported from other parts of the world. In a study of 8 cases, van der Sar was able to demonstrate mites in the sputum, confirming the findings of others to the effect that the condition is probably a form of pulmonary acariasis. Pulmonary infiltrations with eosinophilia have been encountered in individuals infected with various parasites, including amoebae, liver flukes, trichinella, brucella, filaria, cutaneous helminths and other infestations. Although the pulmonary and blood findings are indistinguishable from those of Loeffler's syndrome, in fact some writers believe the two are synonymous, a differential diagnosis is usually possible on the basis of the more protracted course of tropical eosinophilia, enlargement of the spleen, examination of the stools for parasites and response to arsenicals and other medication.

Prolonged or Recurring Pulmonary Infiltrations with Eosinophilia, Pleuritis and/or Pericarditis: As mentioned, a number of variants of Loeffler's syndrome have been described. Dickie and Grimm reported an instance of "Loeffler's Syndrome with Associated Eosinophilic Pleuritis." Zivitz and Oshlag reported one of "Eosinophilic Pleural Effusion and Pericarditis with Effusion in an Allergic Subject." McKinlay reported a case of "Transient Periods of Cardiac Enlargement Associated with Hypersensitivity to Different Etiologic Agents." In a preliminary report of the same case the author entitled his paper "Allergic Carditis, Pericarditis and Pleurisy." In addition, a number of cases have been reported of so-called acute primary pericarditis in which the writers stressed the frequent occurrence of respiratory and pleural lesions which, in some instances, may have been allergic in nature.

In a lucid analysis of the problem, Harkavy interdigitates the several variants of Loeffler's syndrome as representing an expression of the responses in different organs in which primary sensitiveness of the blood vessels, or "vascular allergy," plays a determining role. In Harkavy's opinion there may be pulmonary-myocardial, pleuropulmonary-myocardial or a pleuropulmonary-myopericardial responses. The extension of the process to all serous membranes gives rise to symptoms of polyserositis. Although the

several symptom complexes appear as clinical entities, they are not independent but are merely varying expressions of a state of hypersensitiveness.

The course of the disease in a patient under the writer's observation illustrated graphically the thesis expounded by Harkavy. In the course of slightly more than a year, a man of 37 with a history of chronic sinusitis developed recurrent seizures of pulmonary infiltrations, pleuritis and/or pericarditis. The pleural fluid, as well as the peripheral blood, showed a preponderance of eosinophilic leukocytes. During the acute febrile seizures, which were accompanied by chest pain and pericardial friction rub, improvement followed ACTH and cortisone treatment (Fig. 15).

Periarteritis Nodosa: In 1925, Gruber postulated that periarteritis nodosa is a manifestation of anaphylactic hypersensitivity. The frequency with which bronchial asthma, eosinophilia and other allergic manifestations are found in association with the condition, as well as the demonstration by Rich and Gregory that focal arterial lesions are encountered in tissues after experimental induction of serum sickness, have served to confirm this belief. The morphological changes affect initially the media of small muscular arteries which become necrotic and edematous. There is early intimal proliferation and infiltration with cellular elements chiefly eosinophilic leukocytes. The inflammatory reaction soon extends to the perivascular stroma. Intracanalicular thrombosis sets in and there follows fibrous replacement and occlusion of the lumen of the vessels. Recanalization of thrombi often occurs. The weakened wall of the vessel may undergo aneurysmal dilation leading to hemorrhage. The occluded arteries cause infarction of the affected organs. These changes manifest themselves grossly in nodules of varying size attached to blood vessels, especially of the kidneys, heart, liver, skeletal muscles and nervous system.

For a long time periarteritis nodosa, or panvasculitis since the changes may effect veins as well as arteries, was of pathologic curiosity only because the disease was seldom recognized during life. In recent years the condition is being diagnosed with greater frequency during the patient's lifetime, not only when it affects the heart and kidneys, the organs most often involved, but also the lungs and other organs of the body. Obviously, if vital organs are severely damaged, the disease is fatal sooner or later. But if organs like the lungs, with a large functional reserve are involved without serious impairment of the heart or kidneys, it is not unlikely that periarteritis nodosa may run a relatively benign course and become arrested. The symptomatology of periarteritis nodosa is variable depending on the severity of the disease and the organs involved. If the lungs are also affected, outstanding symptoms are cough, chest pain, bloody sputum and asthmatic symptoms. Dyspnea is usually secondary to asthma or heart failure.

With increasing use of routine chest roentgenography in systemic diseases, instances of periarteritis nodosa involving the lungs are being discovered with greater frequency. The chest x-ray may reveal striking, although seldom characteristic, changes due to the fact that the allergic reaction is part of an acute inflammatory disease involving all elements of the lung. One type of roentgen configuration is represented in pulmonary infiltrations, pleuritis and carditis, much of the lung being obscured by fluid in one or both pleural

cavities including, at times, the pericardial sac. Another manifests itself in irregular nodulations varying in size from a pinhead to a pea assuming a perivascular pattern (Fig. 16). Areas of fibrosis caused by infarction are occasionally encountered. In others one sees dense infiltrations extending fan-wise into the lungs. Cavitation due to necrosis of a large pulmonary infarct may occur.

Rheumatic Pneumonia: From time to time pathologists have drawn attention to the presence of arterial lesions suggestive of periarteritis nodosa in the tissues of patients with rheumatic fever. Others have stressed the fact that in a significant number of patients with periarteritis nodosa one encounters articular and cutaneous manifestations as well as rheumatic heart disease. The intimate relationship existing between these several conditions is supported by the studies of Rich and Gregory who have shown that the basic pathologic features of rheumatic carditis and arthritis can be reproduced experimentally by inducing serum sickness in rabbits. These investigators have also shown that anaphylactic pneumonia caused by hypersensitivity in man has the basic characteristics of rheumatic pneumonia.

The pathology of rheumatic pneumonia is not clear-cut for the reason that by the time the lungs are examined at autopsy the organs are edematous, congested and often the seat of extensive infarction. Indeed, some observers consider rheumatic pneumonia the result of a combination of rheumatism and congestive changes. In any event, it is estimated that rheumatic pneumonia occurs in only about 5 percent of patients with the rheumatic syndrome. Histological features of rheumatic pneumonia are present in thickening of alveolar walls and interstitial tissues, intraluminal exudate, hyaline membrane formation, vascular and perivascular lesions and, on rare occasion, Aschoff bodies, described also as Masson bodies, which are small granulomas in alveolar ducts and alveoli.

The clinical features of rheumatic pneumonia vary widely depending on the nature of the rheumatic manifestations, the competency of the heart and of the pulmonary circulation. The pulmonary symptoms run the gamut from a mild respiratory infection with low-grade fever, moderate cough and expectoration to a hyperacute disease with high fever, severe cyanosis, dyspnea, signs of pulmonary edema and heart failure. In some the occurrence of rheumatic pneumonia does not cause demonstrable increase in the patient's symptoms. There is seldom an upper respiratory infection, chill or sudden pain in the chest such as herald the onset of most bacterial pneumonias. The physical findings are inconclusive as far as differential diagnosis is concerned.

The roentgen features of rheumatic pneumonia are not associated with a sufficiently distinctive pattern to enable one to differentiate the condition from pulmonary congestion which usually coexists to some degree. In the absence of gross evidence of congestive heart failure, the appearance of patchy infiltrations in both lungs, particularly in the upper and midportions, occurring early in the development of the rheumatic state is suggestive of rheumatic pneumonia. The pulmonary infiltrations may assume miliary or nodular configuration. Inasmuch as bacterial pneumonias are rarely encountered in rheumatic fever, roentgen findings such as those mentioned, in patients with active but not decompensated rheumatic heart disease, should make one suspect rheumatic pneumonia (Fig. 17).

Rheumatoid Arthritis: In 1948 Ellman and Ball reported three cases in which pulmonary infiltrations accompanied rheumatoid arthritis. In two, certain features suggested that the pulmonary manifestations were part of the rheumatic disease. As to rheumatoid arthritis itself, it is now generally believed that the condition is a manifestation of hypersensitivity closely allied to rheumatic fever, serum joint diseases and several of the allergic conditions mentioned previously. This view receives additional support from the fact that the administration of cortisone or ACTH exerts significant alterations in the lesions of patients with rheumatoid arthritis. The roentgen findings in the lungs, as reported by Ellman and Ball and several others, consist of bilateral patchy infiltrations of a reticular nature. Pleural effusion may be present. The infiltrations as well as the fluid in the pleural cavity disappear with improvement of the articular manifestations.

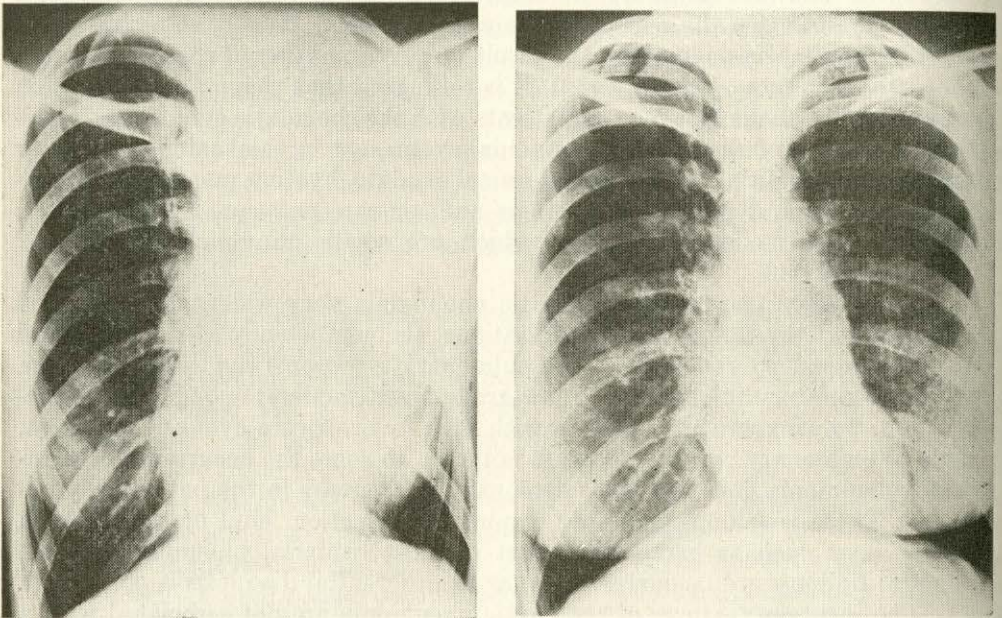


Figure 12.—Massive atelectasis in an asthmatic woman of 36 due to bronchial obstruction by retained sputum. A. Homogeneous density occupying two-thirds of left hemithorax. (Lateral projection revealed collapse of left upper lobe). B. After removal of obstruction, almost complete reexpansion of lung; increase of bronchovascular markings in both lungs. (Similar episode three years previously).

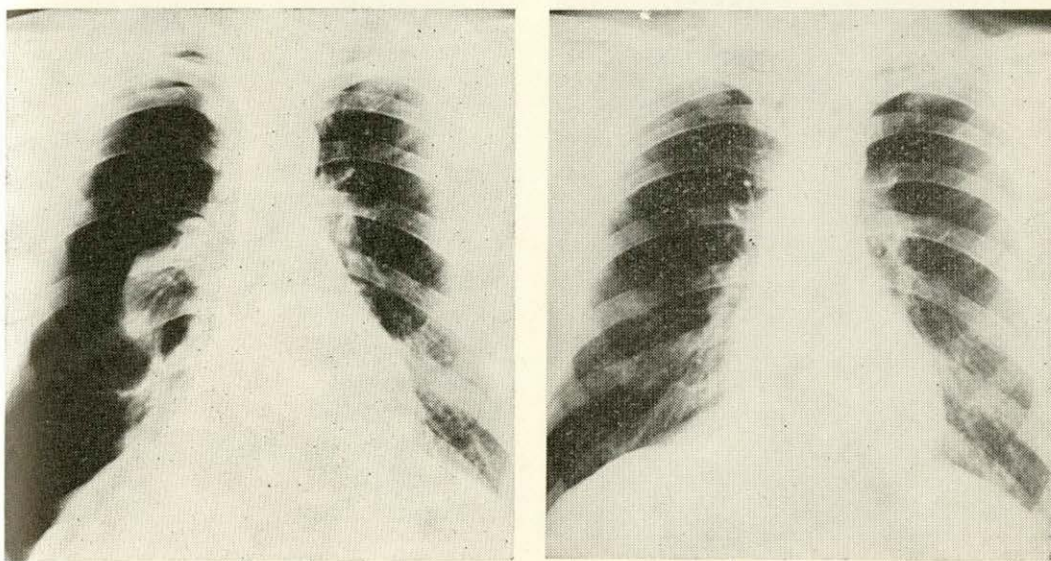


Figure 13.—Spontaneous pneumothorax in an asthmatic of 37. A. Right-sided pneumothorax; lung almost completely collapsed; right diaphragm depressed and flattened showing costal digitations. B. Five weeks later, lung reexpanded; moderate increase of bronchovascular markings in both lungs. (Similar episode six years previously).

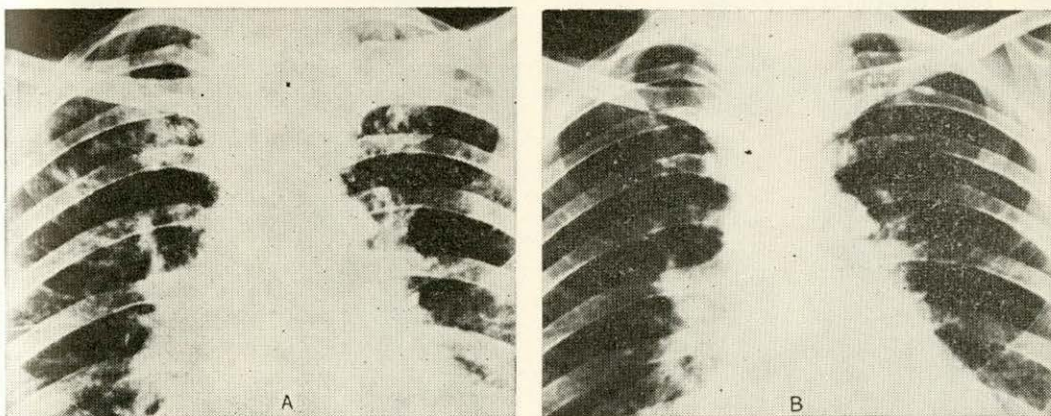


Figure 14.—Transitory pulmonary infiltrations with eosinophilia in an asthmatic woman of 32 (Loeffler's syndrome). A. Irregular, soft densities in both upper lobes. B. One month later, complete clearance of infiltrations with residue of calcific foci in left apex and left hilar region, stigmata of a healed primary tuberculous infection. (Differential diagnosis of active pulmonary tuberculosis; sputum negative for acid fast organisms; eosinophilia 19 percent; rapid resolution of infiltrations).

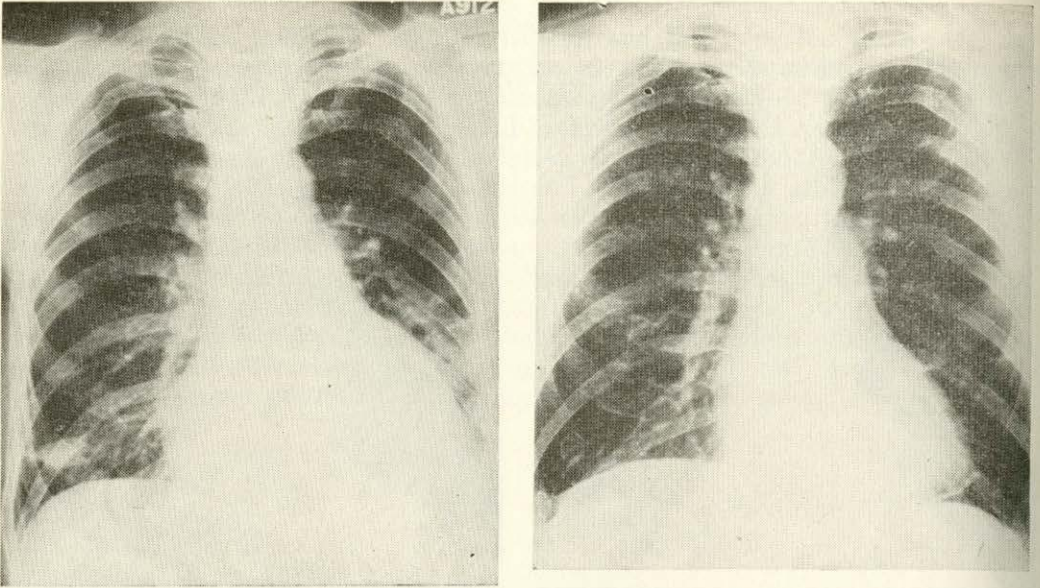
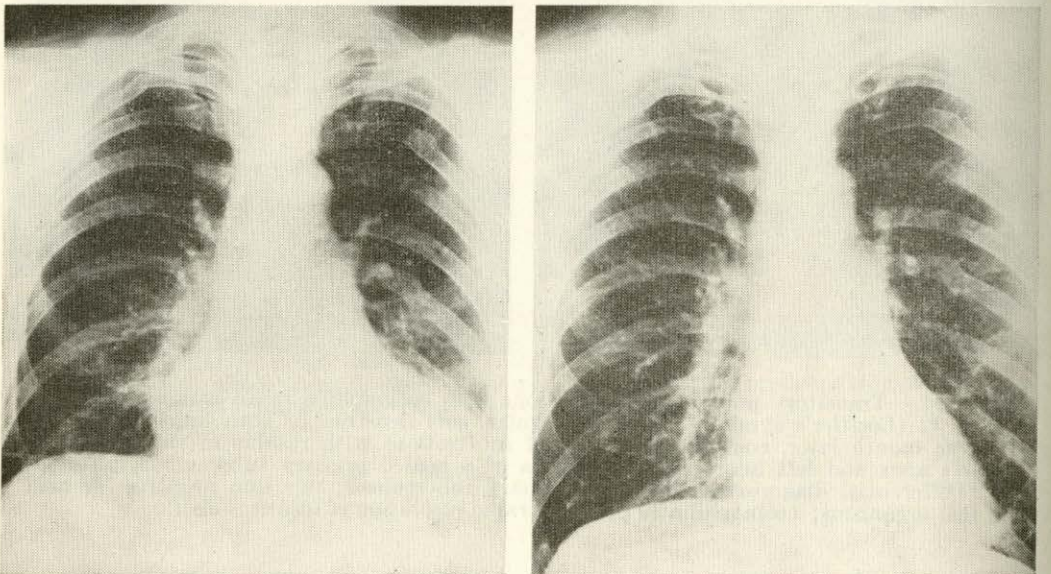


Figure 15.—Recurring pulmonary infiltrations, pleuritis and pericarditis, with eosinophilia in a man of 37. A. Circumscribed infiltration right lower lobe; irregular infiltrations and pleural resection, left lower lobe; enlarged heart; evidence of healed fracture of ribs (R 4, L 5 and 8). B. Three weeks later, decrease in size of heart and regression of pleuropulmonary infiltrations. C. Three months later, heart again enlarged; reappearance of infiltrations and pleural reaction at left base. D. Two weeks later, regression. (In the space of 14 months, 5 seizures of the type shown; clinical and electrocardiographic evidence of recurring pericarditis; fluid aspirated from both pleural cavities showed eosinophilia, 12 per cent; increase in eosinophile count in bone in bone marrow smear and peripheral blood; favorable response to ACTH and cortisone treatment).



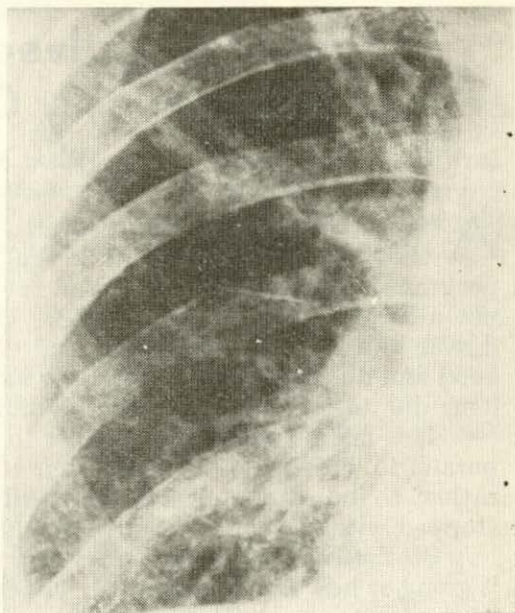
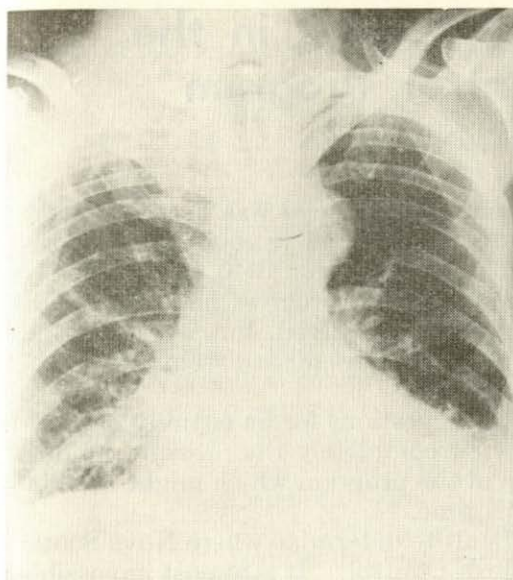


Figure 16—Periarteritis nodosa in an asthmatic man of 61 with pulmonary involvement. A. Nodular and reticular infiltrations in both lungs, especially right; fluid left base; enlarged heart. B. Right lower lobe, showing detail of infiltrations. (Eosinophilia, 22 percent; hypertension, prostate removed because of dysuria showed periarteritis and eosinophilic granuloma; autopsy revealed periarteritic changes involving heart, urogenital organs, muscles and lungs; left lung revealed areas of consolidation and cavitation but no organisms in parenchyma).

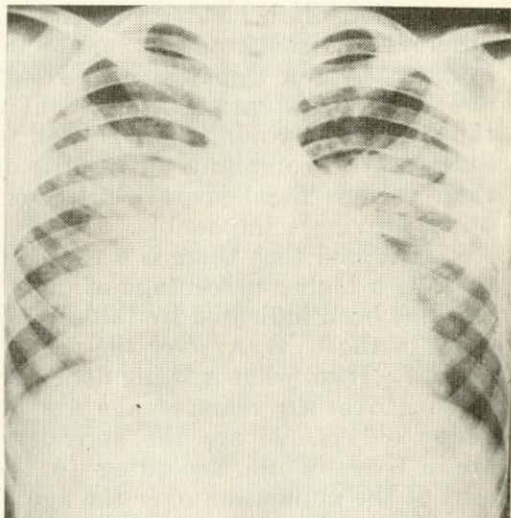
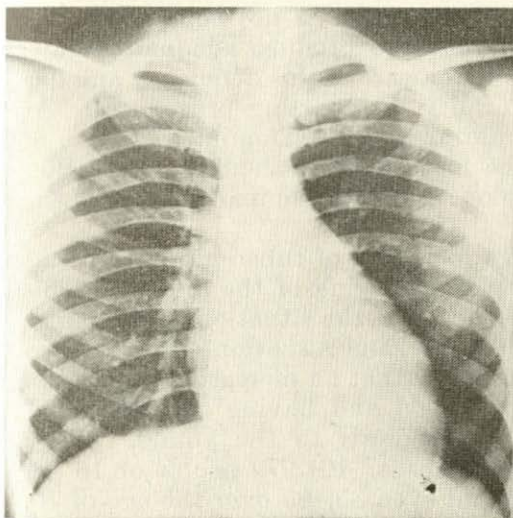


Figure 17.—Rheumatic pneumonia in a girl of 10 associated with acutely progressive rheumatic heart disease. A. Heart moderately enlarged with flattening of left border; lungs clear. B. Two weeks later, fan-shaped densities radiating into mid-portions of both lungs; increase in size of heart. (Total duration of illness seven months; autopsy showed rheumatic endocarditis of mitral and tricuspid valves; congestion and edema of lungs with thickening of septa and mononuclear cell permeation; rheumatic pneumonia).

Some Unresolved Problems in the Tuberculosis Control Program *

J. E. Hiltz, M.D., D.P.H.**

A few weeks ago a senior Health Department official was heard to make the very pertinent remark that Nova Scotia had a good tuberculosis program in operation in 1940 and that, if it was good for 1940, it is probably not altogether adequate for 1953. Of course much progress has been made during the past thirteen years and changes have evolved, but were they all good and did they go far enough, or have we sometimes missed seeing the problems due to the magnitude of the undertaking.

It is not planned in this paper to offer suggestions for an entirely new approach to Tuberculosis Control nor to review completely the present one but, rather, to indicate a few leaks in the dikes of the program which might well be stopped with the finger of initiative at this time.

Statistically, it is sometimes very difficult to determine where Nova Scotia is going or how far it has gone in Tuberculosis Control. It is almost impossible to combine annual reports from the different Divisions and different Hospitals in order to determine how many new cases have been found in the Province each year, or how many of them have been given institutional treatment or what results we have obtained from the treatment routines. During the years when sanatorium beds were in short supply, many cases of minimal or moderately advanced tuberculosis were, of necessity, treated in the home. Did they do better or worse than if they had been in sanatoria? Not only is such information often lacking but that which is available is not comparable for different Divisions and so cannot be combined to produce adequate totals. Fortunately, our Department of Health is well aware of this deficiency and has already taken steps to bring about uniformity of reporting procedures. At the same time would it not be well to conform with the Dominion Bureau of Statistics by compiling all records on the basis of the calendar year rather than various fiscal years which now makes desirable comparisons with the other provinces impossible.

It is said that there is a changing age incidence of tuberculosis and that there is a higher percentage of older people now dying of the disease. This fact has been indicated by tables prepared to show the situation in the larger cities of the U. S. A. over the years. Does the same situation exist in Nova Scotia? If so, what is being done here to assure that an increasing number of persons over 40 years of age are being x-rayed and having sputum tests? These old people are the spreaders of tuberculosis to the young. There are in Nova Scotia, according to the 1951 census, 101,279 people or 16 per cent of the population over the age of 55 and even 35,485 over the age of 70. It must not be forgotten that tuberculosis in these older age groups is very likely to be of the chronic type, which does not kill but spreads to others. If a program calling for a chest x-ray for everyone over 50 could be initiated

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many of our present pools of infection could be found and eliminated. A person cannot move from Canada to the U. S. A. without first having a chest x-ray. Why could it not also be a requisite for all Nova Scotian adults receiving Old Age Pensions and Mothers' Allowances. These benefits are free to the recipient. Why should they not also receive, at no charge, a clean bill of health if they are entitled to one? Our citizens remaining in our country should be just as important to us as those leaving to settle to the 'South of the Border.' "Over 50, an x-ray's thrifty".

When it comes to finding new cases of tuberculosis it is generally conceded that the most productive group comes from the examination of contacts of known cases. This is being rather well done at present. The next most productive group is made up of admissions to general hospitals. In Nova Scotia this is a comparatively new field but one which is developing. At the present time this program is under way in the following hospitals:

Camp Hill Hospital, Halifax
 Colchester County Hospital, Truro
 Grace Maternity Hospital, Halifax
 Halifax Infirmary, Halifax
 Glace Bay General Hospital, Glace Bay
 Roseway Hospital, Shelburne
 St. Joseph's Hospital, Glace Bay
 St. Martha's Hospital, Antigonish
 Victoria General Hospital, Halifax
 Yarmouth Hospital, Yarmouth

and shortly more hospitals are to follow the example of these.

The next most productive group, as far as yielding new cases of tuberculosis is concerned, is made up of those persons who visit a doctor's office because they do not feel well. Are the family doctors being urged to send their patients for x-ray? Most of them work near a hospital where good x-ray facilities are available. Would this source perhaps yield as many or more cases than the present Mobile X-ray Unit or at least more than another Unit—and at less cost. Possibly this could be developed to make a good additional project for the Tuberculosis Control Program.

It is now generally conceded that B.C.G. vaccine gives an appreciable and valuable degree of protection against the development of tuberculosis among those who face an environmental or occupational exposure hazard. It is also generally conceded that the danger to student nurses is greatest during the early days of their studentship upon the wards of the general hospital where they are in training. During 1951, our first year for the General Hospital Admission X-ray Program, only 2,662 persons were so x-rayed, but they included 88 cases of tuberculosis of which at least 20 were proven to be active and would have presented tuberculosis hazards to unsuspecting hospital employees. The student nurse, therefore, should be vaccinated when she first starts to train if she has a negative tuberculin test. At the present time, each of our Nova Scotian training schools for nurses, with two exceptions, has embarked on a B.C.G. vaccination program. All of these, however, do not provide vaccination before the student nurse reaches the wards. It is to be hoped that this situation will be corrected soon.

And further about Schools of Nursing, more education of nurses is needed regarding tuberculosis. The affiliate nursing program at the Halifax Tuberculosis Hospital and the Nova Scotia Sanatorium are not designed to supply free nursing labor to those institutions, but rather to provide the nursing profession with an awareness of the tuberculosis problem, an interest in it, and a freedom from fear of it, which in the past has denied nursing care to the tuberculous, whether in the home or in a tuberculosis institution. Let us sponsor more tuberculosis education of our nurses.

Is it not time, perhaps, that our thinking regarding the tuberculin Patch test was revised. In adults it is definitely inadequate. We can quote many cases from our own Sanatorium records when sputum was positive for tubercle bacilli but the Patch test was negative. In school populations where the incidence of active tuberculosis is very low, it is quite easy to get good correlation between the Patch test and the X-ray, and fairly easy between the Patch test and the Mantoux test, as the skin of children is sensitive and, out of 10,000 tests, only one to two thousand would be positive by either test. It is when we are testing only known positives, that the weakness of this test shows up and especially when applied to the relatively insensitive skins of adults. Should not the Patch test used as a screening process be abandoned unless negative reactors are followed by means of a Mantoux test?

How about the institutional care of patients? Two features that have crept insidiously into our thinking are retarding this program. In the first place, in some cases major surgery has shortened the required period of curing very markedly. Indeed, this has been so dramatic and has happened so frequently, especially following pulmonary resection, that there has developed a general impatience on the part of many non-surgical cases on the cure to a point that they are unwilling to follow curing principles as long as is required to bring about arrest of their disease. Further, even those undergoing major surgery are becoming increasingly impatient and are refusing to convalesce for a sufficiently long period of time to assure that their remaining disease may become thoroughly inactive—and there is always some disease remaining, perhaps on the pleura, perhaps in the resected bronchial stump, or perhaps microscopically in the unresected lung tissue. Surgery is wasted if not effective and will not be effective unless reinforced by adequate bed rest treatment. A readmission to a sanatorium on account of reactivation of disease is a waste both of time and money. Surgery cannot do the job alone. Adequate and prolonged postoperative care are required if readmission rates are to be kept at a minimum. A temporary conversion of sputum is good only so long as it lasts. A breakdown after lung resection may signify the end of the road for that patient. A few additional months of careful curing might have assured firm healing and left readmission beds available for the care of new cases or, better still, left them empty.

Those patients who sign out of a tuberculosis hospital against advice present a real problem, as they are usually persons who are careless about their own health and not interested in the health of others. There seems to be an increasing tendency for patients to sign out. Many of these are patients who have had major chest surgery and it is only a matter of time until some of them will be returned to the sanatorium with empyema or reactivation of parenchymal disease. In what respect are we falling down in regard to them?

In the first place, there should be more intensive education of the patient in regard to the seriousness of tuberculosis, the danger of infection to others, and the need of prolonged treatment to consolidate the healing process once that condition is reached. There is also need for further development of our rehabilitation program in order that they may be provided with diversional activities and entertainment, so that their sanatorium stay may be less trying and more pleasant. We also need further expansion of our Rehabilitation Program in order that more patients may be interested in some vocational goal, and so better able to carry on when fit to go home and, also, while on the cure, may feel that they are accomplishing something worthwhile in addition to the healing of their disease.

Secondly, for those patients who refuse to remain in sanatorium as long as they should, there is need for a more vigorous application of the present legislation governing the apprehension of refractory patients. Recently four patients left the Sanatorium against medical advice, one after the other. It is reasonably certain that each waited to see what was going to happen to the one who departed just before he or she decided to go. The prompt return of the first patient, whether by legislation or persuasion, would probably have prevented the others from even trying to leave. Everything possible must be done to get these people well and protect their associates.

In regard to legislation, it can only be said that it is a necessary evil. Education is always preferable to legislation, but where education fails the law must take over. Once it does so, the matter cannot be treated lightly. A Court Order case is in jail to all intents and purposes, albeit a preferred type of jail with all the comforts of hospital life. But they are still confined. They cannot be permitted away from the sanatorium to which they are confined without the permission of the Attorney General's Department. Should a Court Order case escape he should be brought back by the R.C.M.P. immediately. Next day or next week is not good enough. Recently one of the Sanatorium Court patients ran away and the police did not go after her promptly. She returned of her own volition in 24 hours or so, with the remark "Well, I had my pass anyway!" Following this, as she had got away with it, two other Court Order patients did the same. If contacts are to be protected and a travesty is not to be made of legal procedures, these people must be brought back promptly.

And, speaking of Court Order cases, it is undesirable that sanatorium staff should be required to appear in court against these patients, as they have to live with the patients afterward and look after their needs. The staff, to accomplish anything with them must have their confidence and cooperation. You cannot expect a patient to be very fond of the doctor who has helped to sentence him in court. Indeed, the chances are he will dislike him thoroughly and his hate will be contagious as far as sympathetic fellow patients are concerned, and so the morale of the institution will be undermined. It is desirable to keep the sanatorium staff out of court, whenever possible. Prosecutions are best carried out in an impersonal manner by the Divisional Medical Health Officers.

Another problem that has reared its head on the horizon is the administration of chemotherapy to tuberculous patients in their homes. No one will gainsay the propriety nor the ability of the Divisional Medical Health

Officer or family physician well versed in the knowledge of tuberculosis to supervising properly such treatment in the home. It frequently happens, however, that once treatment is undertaken in the home he may have very little more to say in the supervision of the patient's care until some special difficulty arises such as a drug sensitivity or until the particular form of treatment has been demonstrated to be ineffective.

In the meantime that patient receiving treatment at home may well have been a dangerous source of infection to his contacts and, worse still, has been an attractive example to other patients who might prefer to cure at home and so be led to do so by this poor example. It is well known that chemotherapy can be used effectively for only a relatively short period of time before the tubercle bacilli become resistant to the drug in question.

It is known, too, that there is an optimum time and duration for the use of these drugs and that there should be a synergistic combination of drug, surgery and rest in order to obtain the best treatment result. If patients are to be treated with drugs at home until they become resistant to them, not only may they have infected their contacts but, by the time they reach the hospital, there are bad holes in the umbrella of chemotherapy which may be absolutely essential to cover necessary chest surgery. Another problem which is being reported with increasing frequency is the infection of persons with tubercle bacilli already resistant to the antibiotics. These patients have two strikes against them at the beginning of treatment. Chemotherapy, then, should be used in the home with great infrequency, if at all, and with the utmost care in pulmonary tuberculosis and also in many non pulmonary conditions.

One of our biggest problems at present and one which was not so obvious in the past has been the existence of tuberculosis among the inmates of our County Homes and Mental Hospitals. Our Nova Scotia Hospital has a good tuberculosis control program with B. C. G. vaccination of student nurses, routine x-raying of staff and patients and a separate isolation ward for male and female tuberculous mental patients. The same cannot be said for all our Municipal Homes. During 1951 twenty of the deaths from tuberculosis occurred in Nova Scotian mental institutions and during approximately the first six months of 1952 seven tuberculous deaths are recorded as having similar residence at time of death. Here is a group of patients gathered together in situations where they might be supervised carefully. None of them can refuse examination and yet a goodly number of tuberculous cases must exist in these Homes if they are contributing twenty deaths per year. The mental case is notoriously careless as far as sanitary precautions are concerned. Why should this group be allowed to go on spreading tuberculosis to other inmates and staff and contribute approximately 20 per cent of the tuberculous deaths in Nova Scotia when they are a civic responsibility.

And, finally, the system of reporting deaths by our Dominion Bureau of Statistics has been altered within the past two to three years so that tuberculous persons dying of some condition such as cancer, or coronary disease, or in an accident, are no longer charged against the tuberculosis death rate. This is as it should be, and it is encouraging to see the death rate fall even more rapidly than would be the case if the old system still prevailed, but it should

not be cause for complacency. The cancer case or the heart patient who also has sputum positive for tubercle bacilli is still a menace to his associates and is still our responsibility. Let us leave no avenue unexplored in the control of tuberculosis.

Summary:

There has been presented for consideration the following:

1. A need for more uniform reporting of statistics by Health Units and institutions and also a plea that these be based on a calendar year in conformity with the practice of the Dominion Bureau of Statistics.
2. A need for a special program for the x-raying of persons over 50 years of age. 40 men and 15 women over 50 died of tuberculosis in Nova Scotia in 1951.
3. A need for the further rapid expansion of the Hospital Admission X-ray Program as a case finding measure.
4. A need to further explore as a case finding medium the routine visitor to the doctor's office.
5. A need for expansion of the B. C. G. program among student nurses when they first enter their Schools of Nursing.
6. A need for greater attention to nursing education in regard to all phases of tuberculosis, including rehabilitation.
7. A need to review our thinking in regard to the inefficiency of the tuberculin Patch test.
8. A need to re-emphasize the necessity of adequate bed rest treatment if surgery and chemotherapy are to be effective in the healing of tuberculosis and if its inactive state, once attained, is to be maintained.
9. A need for prompt action in regard to those patients who terminate treatment too early and leave tuberculosis institutions against advice.
10. A need for the prompt apprehension by police authorities of Court Order cases who have escaped from confinement and a plea to keep hospital personnel out of court.
11. A need to limit the administration of chemotherapy in the home to a bare minimum.
12. A need for a more comprehensive and adequate program for the discovery and care of cases of tuberculosis in our municipal institutions.
13. And, finally, a need for the abhorrence of complacency in regard to the rapidly falling tuberculosis death rate. There is still a high morbidity rate which requires all our energy, all our facilities, and our best thought.

Centennial Celebration Notes

DURING the forthcoming festivities connected with our Hundredth Annual Meeting in October it is confidently expected that we, as members of the Medical Society of Nova Scotia, will be hosts to many distinguished visitors and guests. Among them, not the least distinguished, will be the members of the Executive Committee of the Canadian Medical Association, who have arranged to hold their autumn business meeting in Halifax, and so to do honour to our great occasion. To our knowledge this is the first time a division of the C. M. A. has been so honoured, and it would seem only right and proper that the awareness of the honour be demonstrated in the warmth of the welcome accorded to this particular group of people. The fact that a donation of \$1000.00 comes to the Centennial Committee from that source is further proof of the friendliness of their feeling toward us.

In order that the members of the C. M. A. Executive may not come among us as strangers we have thought it fitting to perform a sort of introduction on paper. For this purpose we sought and obtained from Dr. A. D. Kelly, the Deputy General Secretary of the C. M. A., the following brief account of the component parts of the Executive, together with thumbnail sketches of its present members. Dr. Kelly supplied all the personal descriptions except the one applied to himself. There modesty overcame him and he wrote: "The less said the better!" We therefore looked to the Chairman of the Executive Committee to remedy the defect and he prepared the profile on Dr. Kelly. (It is our editorial opinion that, failing in their present vocations, both these gentlemen would achieve success as writers of epitaphs.)

We regret that no photographs could be secured. Some of the faces will be familiar while others will not. It is our hope that when October comes, the names will be enough to identify both the owners and their wives, and to ensure a truly Nova Scotian welcome to those who bear them. M.E.B.G.

The C. M. A. Executive

The Executive Committee of the Canadian Medical Association is, as the name suggests, the executive arm of the policy-making General Council. It acts between meetings of the General Council to conduct the business of the Association. Normally it meets four times a year, but may be assembled at the call of the Chairman. Members of the Executive Committee are elected by the General Council and they consist of—

- (a) the Officers of the Association
- (b) the Divisional Representatives
- (c) the Officials of the Association

The current strength of the Executive Committee consists of the following twenty-one individuals:—

Dr. Norman H. Gosse, Halifax, Nova Scotia

Chairman of the Executive Committee; Chairman of the General Council; Chairman of the Committee on By-Laws and recent Past President

of the Association. Nova Scotia's gift to the C.M.A. A man whose good works in education, organization, economics and cancer require no endorsement to his Nova Scotia colleagues. His stature as a leader of the profession is recognized and acknowledged throughout Canada.

Dr. Charles W. Burns, Winnipeg, Man.

President of the Canadian Medical Association; Professor of Surgery, University of Manitoba; President, Canadian Association of Clinical Surgeons; Past President, College of Physicians and Surgeons of Manitoba; Past President of the Manitoba Division of the C.M.A. Normally busy in the practice of surgery, Dr. Burns has devoted much of his time recently to the affairs of the Association, as host and chief arranger of the highly successful Winnipeg meeting and as visitor in his official capacity to the provincial Divisions in Annual Meeting assembled.

Dr. C. Fritz Strong, Vancouver, B. C.

President-Elect of the Canadian Medical Association; Clinical Professor of Medicine, University of British Columbia. A leading internist of Vancouver, Dr. Strong has served the British Columbia Division as its President and has been Chairman of the Committee on Economics of the C.M.A. Organizer and spark plug of the B. C. Cancer Institute and the Western Society for Rehabilitation. He and his local committees are already at work to insure that the 87th Annual Meeting, Vancouver, June 14-18, 1954, will be a memorable one.

Dr. Edward S. Mills, Montreal, Que.

Honorary Treasurer of the C.M.A.; Professor of Medicine, McGill University; Physician-in-Chief, Montreal General Hospital. Following several years service as a representative of the Quebec Division on the Executive Committee, Dr. (Ed) Mills was elected to control the purse strings of the Association and he has ably fulfilled the duties of Honorary Treasurer since 1950.

Dr. John A. Ganshorn, Vancouver, B. C.

President of the British Columbia Division, whose destinies he has directed during a period of active reorganization and expansion. A busy Vancouver practitioner, John is a new member of the Executive Committee.

Dr. R. M. Parsons, Red Deer, Alta.

A recent Past President of the Alberta Division, Mac brings to the Committee a wealth of experience in economics and all good works. He is the senior member of a small group practicing in Red Deer.

Dr. Fritz E. Werthenbach, Unity, Sask.

Fritz is currently President of the College of Physicians and Surgeons of Saskatchewan and of the Saskatchewan Division. He has served an apprenticeship in the active committees of his province and ably represents the viewpoint of the small-town prairie doctor.

Dr. R. W. Richardson, Winnipeg, Man.

Roy has represented the Manitoba Division on the Executive Committee for three years. He is Chairman of the Committee on Economics, a task of great responsibility and hard work. He has just returned from The Hague where he acted as C.M.A. delegate to the Assembly of W.M.A. While not working for the profession, Roy practices surgery in Winnipeg and is Assistant Professor of Surgery at the University of Manitoba.

Dr. Hugo T. Ewart, Hamilton, Ont.

President of the Ontario Division. In civil life Hugo is Medical Superintendent of the Mountain Sanatorium, Hamilton. Interspersed among a multitude of other duties he finds time to represent the Association on the Canadian Commission on Nursing.

Dr. Roy H. Malyon, Toronto, Ont.

Immediate Past President of the Ontario Division, Roy has served the Association as a member of the Executive Committee for three years. Ask anyone in the Toronto East Medical Association whether he really contributes—they will tell you!

Dr. R. M. Mitchell, Sudbury, Ont.

President-Elect of the Ontario Division, Moe practices surgery in the nickel capital of the world. He brings to his new position on the Executive Committee a wide experience in matters as varied as contract practice and hospital organization.

Dr. J. R. Lemieux, Quebec, Que.

President of the Quebec Division, Reynard is the only member of the Executive Committee whose native tongue is French. He is an able representative of the senior culture of Canada and is a tireless worker for liaison between L'Association des Medecins de la Langue Francaise du Canada and the C.M.A. When not working for the good of the whole profession he conducts a practice in internal medicine, acts as a hospital administrator, and is Profeseur-titulaire of the Medical Clinic at l'Hopital du Saint-Sacrement of Laval University.

Dr. W. deM. Scriver, Montreal, Que.

Walter has represented the Quebec Division on the Executive Committee for several years. He brings abundant experience and wisdom. In his spare time he fulfils the duties of Professor of Medicine, McGill University; Head of the University Clinic and Physician-in-Chief, Royal Victoria Hospital and conducts a consulting practice with special interest in diabetes.

Dr. R. Vance Ward, Montreal, Que.

Immediate Past President of the Quebec Division and Vice President of the Council of the College of Physicians and Surgeons of Quebec. A leader in economic thought in his province, Vance combines an interest in industrial and insurance medicine with the duties of a Lecturer in Medicine at McGill.

Dr. C. L. Gass, Sackville, N. B.

The newly elected representative of the New Brunswick Division, Charlie brings with him the respect and affection of his colleagues in that province. A tireless worker for the advancement of the profession, he was a key figure in the recent Health Survey in N. B. As the head of a first class clinic in Sackville he is medical father confessor to generations of students at Mount Allison. One of his current activities is that of a member of the organizing committee for the College of General Practitioners of Canada.

Dr. M. G. Tompkins, Dominion, N. S.

Dr. Tompkins is Nova Scotia's rather new representative who followed two years of Doctor Blackett. He needs no introduction to Bulletin readers. He is not one of the noisiest members of the Executive, but one does not have to be noisy to be effectively heard.

Dr. W. J. P. MacMillan, Charlottetown, P. E. I.

The veteran representative of P. E. I., Sir William, is either current president or past president of all good works on the Island. He brings to the Executive Committee a wealth of experience and wisdom and is further distinguished by being the only member who can claim to have been Premier of his province.

Dr. John A. Walsh, Manuels.

John was the first representative of the infant Newfoundland Division on the Executive Committee and, after a year's interval, he returns to that post. He is Edinburgh trained, practices in an isolated community and brings his native wit to bear on the problems as they are debated.

Dr. T. C. Routley, General Secretary and Managing Editor.

He is a neophyte indeed who does not recognize in Clarence the man who for over thirty years has been the continuing force of the Association. His recent activities in international affairs with W.H.O. and W.M.A. have demonstrated in a broader field his outstanding abilities and his election in July to the position of President-Elect of the British Medical Association is a crowning honour.

Dr. H. E. MacDermot, Editor.

The editorial sanctum in Montreal contains the man who turns out month for month the Canadian Medical Association Journal. His high standard of literary taste is reflected in its pages and his interest in the history of medicine is also made evident. Ernest made a most useful contribution to literature of medicine by the publication in 1935 of his "History of the Canadian Medical Association."

Dr. A. D. Kelly, Deputy General Secretary.

Doctor Kelly has been the very capable assistant and Deputy to Doctor Routley for a good many years. Next year, when Doctor Routley is translated to a higher level of service, Doctor Kelly will succeed to the position of General Secretary and will bring to it a well established record of service to Canadian medicine. His interest in all departments of the C.M.A. is keen and his knowledge encyclopaedic. His sympathies are broad as medicine. He has shown a fine interest in the movement which has led to the College of General Practice, in the movement to bring into a more satisfactory relationship the various national specialist societies, in Medical Economics and in Public Relations. He has been the virtual father of CAMSI and is father-confessor to its medical student members. Canadian medicine is fortunate in that its most important office will continue to be so ably filled.

Centennial Celebration

SCIENTIFIC AND SOCIAL PROGRAMME

MONDAY, OCTOBER 5th, 1953

- Morning**— Victoria General Hospital, Nurses' Residence Auditorium.
Chairman: Dr. A. E. Doull.
- 9.00—10.00 Surgical Clinic—Carcinoma of Colon, Dr. W. A. Curry.
Ulcerative Colitis, Dr. C. E. Kinley.
- 10.00—10.50 Modern Concepts of the Tonsil and Adenoids.—
Dr. G. E. Tremble.
Intermission—10 minutes.
Chairman: Dr. R. L. deC. H. Saunders.
- 11.00—11.50 Surgical Clinic—Jaundice—Dr. C. F. W. Illingworth.
- 11.50—12.15 "Management of Urinary Lithiasis"—Dr. C. L. Gosse.
- 12.15— 1.00 Psychotherapy in General Medical Practice—
Dr. W. Malamud.
- LUNCH.
- Afternoon**— Ballroom—Nova Scotian Hotel.
Chairman: Dr. H. G. Grant.
- 2.30— 2.50 Cancer Programme for Nova Scotia—Dr. N. H. Gosse.
- 2.50— 3.40 Carcinoma of the Stomach—Dr. C. F. W. Illingworth.
Intermission—5 minutes.
Chairman: Dr. J. W. Merritt.
- 3.45— 4.30 Problem of Degenerative Vascular Disease—
Dr. N. G. B. McLetchie.
- 4.30— 5.15 Nasal Physiology and Nasal Medication—Dr. G. E. Tremble.
- Evening**— Victoria General Hospital, Nurses' Residence Auditorium.
- 8.30 p.m. Committee of Trauma of American College of Surgeons.
Discussion on "Trauma."
Bring your cases and problems for informal presentation.
Moderator: Dr. A. L. Murphy.
Members: Dr. T. B. Acker, Dr. B. K. Coady, Dr. W. A. Curry,
Dr. J. V. Graham, Dr. C. E. Kinley, Dr. E. F. Ross.

LADIES' EVENTS.

All day. Registration. Nova Scotian Hotel.

TUESDAY, OCTOBER 6th, 1953

- Morning—** Victoria General Hospital, Nurses' Residence Auditorium.
Chairman: Dr. T. M. Sieniewicz.
- 9.00—10.00 External Otitis—A Controversial Problem—
Dr. G. E. Tremble.
- 10.00—10.50 Relations Between Organic Factors and Psychic Functions—
Dr. W. Malamud.
Intermission—10 minutes.
Chairman: Dr. M. R. MacDonald.
- 11.00—11.15 Megacolon—Dr. N. B. Coward.
- 11.15—11.45 Subdural hematoma—Dr. R. M. Ritchie.
- 11.45—12.00 Discussion—Introduced by Dr. G. B. Wiswell.
- 12.00— 1.00 Symposium by Department of Obstetrics and Gynaecology.
Moderator: Dr. H. B. Atlee.
Natural Childbirth Methods.
Introduced by Dr. I. A. Perlin.
First Thirty Minutes of Life.
Introduced by Dr. J. McD. Corston.
Carcinoma of Female Genital Tract.
Introduced by Dr. W. R. C. Tupper.
- Luncheon—Victoria General Hospital Cafeteria.
Courtesy—Victoria General Hospital.
- Afternoon—** Ballroom—Nova Scotian Hotel.
Chairman: D. C. C. Stoddard.
- 2.30— 3.00 A Century of Medicine in Nova Scotia—
Dr. K. A. MacKenzie.
- 3.00— 3.20 Pulmonary Emphysema—Dr. C. A. Gordon.
Intermission—5 minutes.
Chairman: Dr. G. G. Simms.
- 3.25— 5.30 Round Table—"Present Status of Health Insurance in
Canada."
Moderator: Dr. C. B. Stewart. Dr. A. D. Kelly, Dr. O. LeRue,
Dr. A. W. Titus.
- 6.00 p.m. Meeting of Defence Medical Association—Nova Scotia Division—Cambridge Library.
- Evening—**
- 9.30 p.m. Centennial Ball—Nova Scotian Hotel.
- LADIES EVENTS.**
- 2.00 p.m. Visit to Uniacke House, Mount Uniacke, Nova Scotia.
(A former home of Richard John Uniacke, one time Solicitor General of Nova Scotia. This excellent example of colonial architecture was built in 1813 and has some fine 19th century furniture.)
- 5.00 p.m. Return to Halifax for tea.
- 9.30 p.m. Centennial Ball—Nova Scotian Hotel.

WEDNESDAY, OCTOBER 7th, 1953

- Morning**— Camp Hill Hospital Auditorium.
Chairman: Dr. T. E. Kirk.
- 9.00— 9.50 Recent Developments in Geriatrics—Dr. W. O. Thompson.
- 9.50—10.10 Long Term Results of Gastrectomy at Camp Hill Hospital.
—Dr. R. M. MacDonald.
- 10.10—11.00— Post-Gastrectomy Syndromes—Dr. C. F. W. Illingworth.
Intermission—10 minutes.
Chairman: Dr. H. D. O'Brien.
- 11.10—11.30 "Surgery of Anus and Rectum"—Dr. E. F. Ross.
- 11.30—11.50 Dysphagia—Dr. E. P. Nonamaker.
- 11.50— 1.00 Round Table—"Management of Peripheral Vascular Disease."
Moderator: Dr. J. A. Noble. Dr. G. W. Bethune, Dr. C. M. Harlow, Dr. L. C. Steeves.
Luncheon—Camp Hill Hospital.
Courtesies—Department of Veterans Affairs.
- Afternoon**— Ballroom—Nova Scotian Hotel.
Chairman: Dr. R. O. Jones.
- 2.30— 3.15 Hormonal Factors in Psychiatric Conditions—
Dr. M. Malamud.
Discussion opened by Dr. W. O. Thompson.
- 3.00— 3.45 Types and Treatment of Hypogonadism—
Dr. W. O. Thompson.
Intermission—10 minutes.
Chairman: Dr. D. J. Topping.
- 3.55— 4.15 Thyro-cardiac Disease—Dr. D. L. Roy.
- 4.15— 5.05 Round Table—"The Menopause".
Moderator: Dr. H. B. Atlee, Dr. M. Malamud,
Dr. W. O. Thompson.
- 9.30 a.m. Executive Meeting of The Medical Society of Nova Scotia—
Dalhousie Public Health Clinic—Lecture Room.
- 1.00 p.m. Luncheon—Executive. The Medical Society of Nova Scotia—
Nova Scotian Hotel.
- 2.30 p.m. Executive Meeting of The Medical Society of Nova Scotia—
Dalhousie Public Health Clinic—Lecture Room.

WEDNESDAY, OCTOBER 7th, 1953 (Continued)**Evening—**

- 6.30 p.m. Reception—The Medical Society of Nova Scotia—Nova Scotian Hotel—Ballroom.
- 7.30 p.m. Buffet Supper—Nova Scotian Hotel—Main Dining Room.
- 9.00 p.m. John Stewart Memorial Lecture—Nova Scotian Hotel—Ballroom.

“SURGERY AND SCIENCE”

Wilder G. Penfield, O.M., C.M.G., M.A., M.D., D.Sc., F.R.C.S. (C), HON. F.R.C.S. (Eng.), F.R.S.C., F.R.S. Professor of Neurology and Neurosurgery, McGill University, Montreal, Director, Montreal Neurological Institute.

Chairman: Dr. A. E. Kerr, President, Dalhousie University. Originated and sponsored by—The Provincial Medical Board of Nova Scotia.

LADIES' EVENTS.

- 11.00 a.m. Coffee Party—Royal Nova Scotia Yacht Squadron.
- 6.30 p.m. Reception—The Medical Society of Nova Scotia—Nova Scotian Hotel.
- 7.30 p.m. Buffet Supper—Nova Scotian Hotel.

THURSDAY, OCTOBER 8th, 1950

Morning—Victoria General Hospital, Nurses' Residence Auditorium.

Chairman: Dr. C. J. W. Beckwith.

- 9.00— 9.40 Medical Clinic—Subacute Bacterial Endocarditis—
Dr. C. S. Keefer.
- 9.40—10.00 Use and Reliability of Miniature Roentgen Survey Pro-
cedures—Dr. W. M. Roy.
- 10.00—10.50 Discussion of Some Common Diagnostic Errors in Medical
Practice—Dr. C. S. Keefer.
Intermission—10 minutes.
Chairman: Dr. C. S. Marshall.
- 11.00—11.50 Neurological Clinic—Dr. W. G. Penfield, Dr. W. D. Stevenson.
- 11.50— 1.00 Round Table—Recent Developments in Therapeutics.
Moderator: Dr. L. C. Steeves, Dr. C. F. W. Illingworth,
Dr. C. S. Keefer, Dr. J. G. Aldous.
- 1.10 p.m. Luncheon—Nova Scotian Hotel.
(Honoured Guests—Executive Canadian Medical Association)

Afternoon—

- 2.45 p.m. Business Meeting—The Medical Society of Nova Scotia—
Room 21, Dalhousie University Arts and Administration
Building, Studley Campus.
- 4.15 p.m. Special Convocation of Dalhousie University—Room 21, Arts
and Administration Building, Studley Campus.
- 4.45 p.m. Convocation Tea—Shirreff Hall.
- 2.30 p.m. Golf Tournament—Ashburn Golf Club.

Evening—

- 6.00 p.m. Reception and Presentation of Prizes—Ashburn Golf Club.
- 8.00 p.m. Buffet Supper and Reunion of Graduates of Dalhousie Medical
School—Lord Nelson Hotel.
- 8.00 p.m. Buffet Supper and Entertainment at Ashburn Golf Club for
ladies and non-Dalhousians.

LADIES' EVENTS.

- 12.30 p.m. Luncheon—Lord Nelson Hotel.
- 3.00 p.m. Golf Tournament—Ashburn Golf Club.
- 4.15 p.m. Special Convocation of Dalhousie University—Room 21, Arts
and Administration Building, Studley Campus.
- 4.45 p.m. Convocation Tea—Shirreff Hall.
- 6.00 p.m. Reception and Presentation of Prizes—Ashburn Golf Club.
- 8.00 p.m. Buffet Supper and Entertainment at Ashburn Golf Club for
ladies and non-Dalhousians.

FRIDAY, OCTOBER 9th, 1953

Morning—Victoria General Hospital, Nurses' Residence Auditorium.
Chairman: Dr. C. W. Holland.

9.00— 9.45 Some Aspects of Diabetes—Dr. W. deM. Sriver.

9.45—10.15 Experimental Nephritis due to Type-specific Streptococci.
Dr. R. W. Reed.

Discussion opened by Dr. C. S. Keefer and Dr. W. deM. Sriver.

10.15—11.00 Medical Clinic—Dr. C. S. Keefer.

Intermission—10 minutes.

Chairman: Dr. W. A. Curry.

11.10—11.55 Surgical Clinic—Dr. C. F. W. Illingworth.

11.55— 1.15 Round Table—Management of Gastro-Intestinal Haemorrhage.

Moderator: Dr. V. O. Mader. Dr. C. W. Burns, Dr. C. F. W. Illingworth, Dr. C. S. Keefer, Dr. E. S. Mills.

Luncheon—Victoria General Hospital Cafeteria.

Courtesy—Victoria General Hospital.

Morning—Ballroom—Nova Scotian Hotel.

Chairman: Dr. C. M. Bethune.

2.30— 3.00 Antibiotics—Some Current Problems in Their Use.—
Dr. C. S. Keefer.

3.00— 3.30 Epidemiology of Peptic Ulcer—Dr. C. F. W. Illingworth.

3.30— 5.30 Business Meeting—The Medical Society of Nova Scotia.

Evening—

6.30 p.m. President's Reception—The Medical Society of Nova Scotia—
Nova Scotian Hotel.

7.30 p.m. Annual Dinner—The Medical Society of Nova Scotia—
Nova Scotian Hotel.

Speakers: The Hon. Paul Martin, Minister of Health.
Mr. W. R. Bird.

LADIES' EVENTS.

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