Erythema Nodosum

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ERYTHEMA nodosum is a very interesting, and yet a most perplexing condition. The clinical picture is fairly easily recognizable. Its significance is sometimes of great importance. The etiology and pathogenesis have been the topic of much debate for over a hundred years. There are many points which are not yet settled and much that is not yet known about the subject. It is my purpose to outline the most important facts concerning different aspects of the disease and to give my impressions resulting from a review of the literature and a study of case summaries at the Nova Scotia Sanatorium and the Victoria General Hospital.

Description

As the name implies, erythema nodosum is a nodular erythematous skin eruption. The term was first used in 1798 by Willan of England, a dermatologist, who was discussing different varieties of erythema. Later, in 1860, Hebra, classified the erythemas and his description of erythema nodosum was somewhat different than that of Willan. He designated most of Willan's erythemas in the group Erythema Multiforme, retaining the name erythema nodosum to apply to a restricted group which he described as follows: "light red raised nodules either round-topped or oval in shape, tender to the touch and mainly situated on the legs. In many cases the eruption is preceded by a slight temperature elevation or chills. Often, however, the patient has no previous warning of disease before he sees or feels the nodes. They occur as a rule in various sizes, the smallest the size of a pea, and the largest the size of a closed fist. The individual nodules are usually discrete—at first pale red with a faint gold tinge, at a later stage they turn dark red and livid, and after the redness has disappeared the lesions persist for a long time in the form of yellowish pigmentation. These color changes are similar to those occurring after a bruise, and for this reason the name—dermatitis contusiformis, has been used by some authors." In the main this description is the same as is used today, except that some investigators have failed to note the color changes during the course of the disease in many of their cases, which in other particulars were typical. It seems necessary to supplement Hebra's description to some extent. The pain, fever, chills and so on, may all be absent. However, it is true that as a rule a moderate or even high elevation of temperature, sometimes accompanied by chills and severe malaise occur a day or two preceding the appearance of the eruption. The raised nodules usually occur on the anterior surface of the lower legs—one or two or more appearing, followed by a new crop in a day or two. Frequently as the first nodules are changing color and beginning to fade, fresh nodules appear at a different site. Within a few days following their appearance the oedema subsides and at this stage usually a nodule can be felt under the skin. In addition to the lower legs, which are

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practically always affected, the nodules may occasionally appear on the extensor surfaces of the thighs, arms and forearms, face and buttocks. The duration of the eruption varies considerably—from a few days to several months. However, in the average case it is approximately three weeks. Recurrences are by no means common, a few people have one or more, and one case has been reported where there were ten. It should also be noted, and I think this is very important, that in a significant percentage of persons affected there is a variable degree of joint pain and sometimes swelling. There are no typical or pathognomic blood changes. The white blood cell count may or may not be elevated—if elevated it is rarely over 14,000. The sedimentation rate is pretty consistently elevated to a moderate or a marked degree in the early stages. The condition occurs predominantly in children and young adults. It has been reported in an infant seven months of age, and a few cases have been seen in persons over sixty. For some unknown reason, possibly a hormonal factor, females are affected much more commonly than males.

Etiology

In approaching the question of etiology, I hasten to say that at this point we are wading into controversial waters which sometimes can be very rough. It might be well briefly to trace the changing ideas concerning the etiology down through the past century. Many of the early authors believed that it was a specific infectious and contagious disease, occurring sometimes in epidemics and producing immunity following infection. There are a few who still hold this view today.

As early as 1872 the connection between primary tuberculosis and erythema nodosum was noted, and since that time a tuberculous etiology has been recognized in a large proportion of all cases by most investigators. Throughout Europe the tuberculous theory became popular, but in England and France a considerable number of cases were being related to rheumatic fever, and this theory has been the subject of much debate down through the years—even to the present time. In fairly recent years several authors have noted striking relationship to upper respiratory infections, particularly those caused by the betahemolytic streptococcus. This theory is popular in North America today, and to a somewhat lesser extent in European countries. It is based on fairly convincing evidence. Other well established rare causes are a variety of diseases, including sarcoidosis, coccidioidomycosis (in certain restricted areas in the United States, particularly), syphilis, pneumonia, meningitis, lymphogranuloma inguinale, gonorrhea, septic wounds, measles, pertussis, ulcerative colitis, and a variety of other minor infections. There remains one other group of causes, which include drugs, the most notable being the sulphonamides and bromides.

Now it is necessary to elaborate on some of the evidence brought forth in favor of one or other of the foremost theories. Wallgren of Sweden, in his excellent review of 1000 cases[1] makes a very strong and convincing case of primary tuberculosis in children as the cause in the vast majority of cases. His investigation and that of Ernberg[2] in large numbers of cases, has establish-

ed several significant facts. They have shown, and this has been confirmed by others since, that the condition frequently appears towards the end of the incubation period of primary tuberculosis. This is between three and eight weeks, and at the height of the tuberculous allergy which is being produced. Conversion of tuberculin reaction from negative to positive has been noted in close to 100% of cases in children at this time[13]. Many of these cases subsequently develop manifest tuberculous disease. In well controlled studies it has been clearly demonstrated that children with erythema nodosum show a much higher incidence of definite tuberculous disease than tuberculin positive children without erythema. Further to this Wallgren[1] has studied at least ten epidemics in children in which several cases were traced without any doubt to the source of an open tuberculous person. A very interesting report of an epidemic in this country was made by Brandan, Hardman and Birks.[4] This epidemic took place in a private school where there were 173 boys between ten and nineteen years of age. Fourteen cases of erythema nodosum occurred within a ten week period, all of whom had a strongly positive tuberculin reaction. Twelve of these showed definite or suspicious evidence of active pulmonary tuberculosis. In addition, ten other boys developed definite pulmonary tuberculous disease and five others were suspected, making a total of twenty-eight boys with demonstrable chest lesions. In every case the tuberculous disease was of the primary infection type. The source of the epidemic was in all probability a boy who was diagnosed as an open case five weeks after the first case of erythema nodosum was observed. The disease was probably transferred by direct contact.

Wallgren[1] has been able to isolate tubercle bacilli from gastric washings of children with erythema nodosum in as high as 75% of cases studied. Others have been able to demonstrate tubercle bacilli in the washings of close to 100%. Further evidence in favor of the tuberculous theory is the observation that if a tuberculin positive child is convalescing from an attack of erythema nodosum and is injected with a sufficient dose of tuberculin to produce a general reaction, this will frequently provoke a new eruption, identical to the original lesion.[3] One last point in favor of tuberculous etiology is the observation that the tuberculin reaction in children with erythema nodosum is usually of greater intensity than in others[1,5].

It must be realized that the relative number of cases of erythema nodosum caused by tuberculosis seen by any one or group of investigators will depend on many factors. Firstly, the geographical location has an important influence. For example, the condition has been prevalent in Norway, Denmark and Sweden. In these countries it is thought to be caused by primary tuberculous infection in children in 90 - 95% of all cases. In other ceuntries the tuberculous etiology is considerably less. For example, in England, it is approximately 70%. The reason for this geographical difference has not been adequately explained. Secondly, the incidence of tuberculous etiology would be 100% in those cases occurring in epidemic proportions as a result of exposure of a group to an open case of tuberculosis. Thirdly, the relative incidence would depend to a large degree on the type of patients dealt with by the

investigator. For instance, one would expect to find a high tuberculous etiology in cases reviewed in a Sanatorium for tuberculous patients. On the other hand, patients with erythema nodosum seen in general practice would be expected to have a much lower tuberculous etiology proportionately. The same would apply by a comparative study conducted at the Nova Scotia Sanatorium and the Victoria General Hospital. The records of twenty cases seen at the Sanatorium between 1940 and 1950 were studied. This group included ten in-patients, three staff members and seven seen in the Out-Patient Department. Seventeen were females, three males and eighteen were in the 11-30 age group. Of the twenty cases, eleven were considered of definite and two of highly probable tuberculous etiology (65%). In two others a tuberculous etiology was strongly suspected (10%). In the five remaining cases etiology could not be definitely determined. The study at the Victoria General Hospital consisted of a review of twenty-seven cases admitted during the period 1937 to 1951. Twenty-four were females, three males and twenty-one were in the 11-30 age group. Of these, there were only two definite and three of highly probable tuberculous etiology (18.5%), with a further two cases (7.4%) possibly due to tuberculosis. Six cases (22.2%) were closely related to an upper respiratory infection and were considered due to this cause. Five cases (18.5%) appeared to be related but not conclusively to rheumatic fever and in two others, the etiology may have been either rheumatic fever or upper respiratory infection. In the remaining seven (25%) the etiology could not be determined. In both groups studied several had more than one infection, shortly before or at the time of the appearance of the skin lesion. Trichomonas vaginitis and gonorrhea occurred in addition to tuberculosis or upper respiratory infection and could conceivably have been the cause of the erythema, although this was considered unlikely. It was more difficult to decide on the most probable etiology in a few cases in whom a definite upper respiratory or other infection had occurred recently, yet had a positive tuberculin test and perhaps radiographic evidence of inactive pulmonary tuberculous disease. All persons who were considered to be of tuberculous etiology in both the Sanatorium and the Victoria General Hospital groups were in the 11-30 age group, and in all but two the erythema nodosum occurred at or about the time of the primary infection. This, then, is another consideration in estimating the relative tuberculous etiology. It should be and is higher in children and young adults where primary tuberculosis is encountered more frequently, than in an older age group. Finally, in any study of a group of persons with erythema nodosum the percentage of the total which is attributed to tuberculous etiology will depend largely on the criteria used for such a diagnosis by the individual investigators. Many of the ardent proponents of the tuberculous theory believe that if an individual has a positive tuberculin reaction then tuberculosis is probably the basic etiologic factor regardless of the presence of concurrent infections. They sometimes consider such concurrent infections as provocative agents.[1] If, in addition to a positive tuberculin reaction, there is hilar adenopathy they contend that the etiology is almost certainly tuberculous. Others, and particularly recent American authors,[6] take a different view. They have observed cases with pulmonary adenopathy of short duration. In such cases, with or

without a positive tuberculin reaction, they believe that the pulmonary adenopathy is an associated manifestation of erythema nodosum of non-specific etiology. Others contend that it is the result of drainage from an upper respiratory infection.[7] This question is far from being settled and is one of the many difficult problems which remain to be solved. However, it is difficult if not impossible, to prove that in a tuberculin positive child or young adult the erythema nodosum is not related to tuberculosis.

Now for the rheumatic fever theory. This theory gained great popularity in England in 1886 when Sir Stephen MacKenzie[8] reviewed the records of a number of cases of erythema nodosum in the London hospitals, most of which he had not seen personally, and concluded that there was a very close relationship to rheumatic fever in a large percentage. His conclusions, and those of others since that time, have been based largely on the presence of some joint pain and sometimes the finding of a cardiac murmur in persons who had previously had a sore throat. Very seldom has there been definite evidence of rheumatic carditis in such cases, and those that dispute the rheumatic fever theory contend that the joint pain is not of rheumatic origin but rather an inherent part of the clinical picture of erythema nodosum. Favour and Sosman[7] in a review of 155 cases, of which 102 were adults and 53 children, studied in Boston in 1947, encountered an incidence of migratory arthritis which they considered part of the clinical picture in 80% of adults and 30% of children. They concluded that erythema nodosum is not a form of rheumatic fever, but occasionally may accompany it. Further it is thought that many of the cardiac murmurs heard were probably functional. Granted, there have been a few well authenticated cases of erythema nodosum due to rheumatic fever [1,7,9], but it is probably true that in the great majority of cases in which rheumatic fever was considered to be the cause, the decision was not based on sufficiently substantial evidence. At least there is an increasing tendency in recent years to dispute any direct etiological relationship between rheumatic fever and erythema nodosum except in rare instances.

In 1932 Collis [10] first presented strong evidence for an etiological relation between infection with the beta-hemolytic streptococcus and erythema nodosum. This conception is based on clinical, bacteriologic and immunologic evidence. It has been established that in a large proportion of cases in which tuberculous etiology can be definitely ruled out, that streptococcal upper respiratory infection is the etiological factor. Many of these people have a sore throat a few weeks prior to the appearance of the eruption. A fair percentage of these yield hemolytic streptococcae on culture from their throats. In addition, in most of these cases, a strong skin reaction occurs following the intradermal injection of streptococcal endotoxin, filtrates or vaccine. The skin reaction is of such intensity in many instances to constitute conclusive evidence of a hemolytic streptococcal etiology. This, of course, applies to tuberculin negative individuals. The injection of the streptococcal endotoxin has occasionally provoked a new eruption of erythema nodosum in persons who were convalescing. In persons with positive tuberculin reaction who have a streptococcal upper respiratory infection and streptococcal skin hyper-sensitivity, the

etiology is more difficult to determine. Wallgren[1] and others believe that in the majority of such cases tuberculosis is the underlying etiology and the streptococcal infection merely a provocative factor producing a change in the tuberculin sensitivity state which is responsible for the appearance of the eruption. This again is a debatable question, difficult if not impossible to settle.

The relationship between certain chemical agents—the principal ones being, sulphathiazole, sulphadiazine and bromides with erythema nodosum has been well established. There is, however, a difference of opinion regarding the nature of the relationship. A few writers[1] have thought these agents to be primary etiological factors, the skin eruption being the sign of hypersensitivity reaction in a susceptible individual. Most authors[12, 13] contend that the chemical agents act in a provocative manner, merely increasing a hypersensitivity state in a person previously sensitized to either tuberculin, streptococcal toxin or other allergen.

There remains one other theory, that of erythema nodosum being an expression of a specific infectious disease, probably caused by a virus. This theory was propounded by the earlier writers, such as Hebra, and again in more recent years by Miescher[14] and a few others. This theory is not held by very many and there is little, if any, good evidence to support it.

Outline of Investigation for Determination of Etiology

The age of the individual patient must be considered, remembering that there is a much higher incidence of tuberculous etiology in children and young adults. A family history of tuberculosis may be significant. In regard to the personal history, the following facts should be determined:

(1) Any contact with tuberculosis.
(2) Any previous rheumatic fever.
(3) Recent upper respiratory infection.
(4) Record of previous tuberculin testing.
(5) Previous chest x-rays.
(6) Any previous erythema nodosum.

Physical examination should cover all systems, but particular attention should be paid to the upper respiratory system, the lungs, heart and lymph nodes. Special investigation should include:

(1) Sedimentation rate.
(2) Chest x-ray and serial x-rays at weekly intervals if necessary.
(3) Tuberculin test—Vollmer patch test and if negative or doubtful followed by Mantoux, either old tuberculin or P.P.D.
(4) Sputum tests and gastric lavage. This testing should be done early and repeatedly. It is much more likely to find tubercle bacilli in the early stage. This indicates a recent active disease.
(5) Search for source of infection.
(6) Throat swab for hemolytic streptococcae.
(7) Skin test for streptococcal sensitivity.

(8) Electrocardiogram, if rheumatic fever suspected.
(9) Specific tests for other diseases.

Pathogenesis

The theories regarding pathogenesis are also numerous. The most popular view in recent years is that the condition is a non-specific hypersensitivity reaction in a predisposed individual, produced by any one of several infectious toxic or chemical agents. Ernburg[2], Wallgren[1] and others[8,9,15] have been strong advocates of this theory. Most of the investigative work done regarding pathogenesis has been in connection with cases of tuberculous etiology. It has been clearly shown that erythema nodosum occurs at the peak of tuberculin hypersensitivity in those individuals who have had a recent primary infection. It has also been demonstrated that the tuberculin reaction is usually greater in children with primary tuberculosis and erythema nodosum than in those without. The histology of the erythematous nodules is generally considered to be that of a non-specific inflammatory reaction and this seems to be in favor of it being produced by a non-specific allergy. This theory would seem to apply equally well in any of the infections which have been designated as responsible. The alternative theories do not have a very great support. One of these is that the individual erythematous nodules are produced by bacterial emboli. The difficulty with this is that except in very rare cases no bacteria have been found in the lesions. Tubercle bacilli have been demonstrated in the lesions in a few isolated instances, but it was not clearly demonstrated that their presence there was not merely co-incidental. Wallgren[1] believes that the tubercle bacilli were within the blood vessels and merely constituted a part of the bacillemia which is a common occurrence in primary tuberculosis, and that they did not have any local effect in producing the lesions.

Similarly streptococcae are seldom, if ever found in the erythematous nodules, in cases which are considered of streptococcal etiology.

The only other theory which requires mention is that erythema nodosum is an expression of a specific infectious disease, probably caused by a virus and that it is in fact another exanthem, similar to measles and chicken-pox. This theory seems to be untenable in view of the very substantial evidence in favor of various etiological agents, the most notable being tuberculous and streptococcal.

Pathology

The histological changes in erythema nodosum have been studied extensively. There is general agreement that the histological picture is the same whatever the etiological agent. The sub-cutaneous tissue is principally affected, and within the first few days of the eruption the picture is that of an acute inflammation with hyperemia, oedema and infiltration of polymorphonuclear leukocytes. At a little later stage histiocytes and lymphocytes predominate, and still later the initial signs of acute inflammation subsides, and fibrinoid degeneration of the connective tissue makes its appearance. Finally, non-specific chronic granuloma deposits with giant cells of the foreign body type appear.

Significance and Prognosis

Erythema nodosum serves as a warning of the possible presence of potentially serious disease. With reference to primary tuberculosis, it is very important to recognize this disease early—appropriate treatment carried out promptly may prevent serious progression and complications. When the condition occurs in association with re-infection tuberculosis, it may possibly indicate activity of a lesion which had previously been considered to be inactive.

Wallgren[1] believes that the occurrence of erythem nodosum has no bearing on the prognosis in a child with primary tuberculosis. His studies suggest that the primary tuberculous infection behaves the same regardless of the occurrence of erythema nodosum. Others[16] have found a high incidence of complications, such as pleurisy with effusion, acute miliary tuberculosis and extrathoracic disease, such as tuberculous meningitis in children with primary tuberculosis and erythema nodosum.

Treatment

In any case of erythema nodosum, regardless of the etiology, bed rest is essential until the temperature returns to normal. The sedimentation rate is generally a better indication of the degree of activity. General, symptomatic and supportive measures are indicated, including analgesics and antipyretics. Because of the allergic nature of the condition several cases have been treated in recent years with cortisone. Some of the reports have been quite enthusiastic [17], [18], [19] particularly with its use in an acutely ill patient or when the condition fails to respond to other measures. In such cases cortisone, in adequate dosage, generally reduces the fever, causes the nodules to regress and disappear and brings about an improvement in the general condition. However, there is conflicting evidence that this agent shortens the duration of the disease to any appreciable extent, and there have been reports of prompt recurrence of the nodules following discontinuance of the drug[20], [21]. Because of the known and considerable danger of administering cortisone to a person with tuberculous disease, it is essential that it not be used under such circumstances, unless the indication is very strong and some combination of streptomycin, isonicotinic acid hydrazid and P.A.S. is administered concurrently.

The question arises as to whether or not the incidence of erythema nodosum is declining. A marked decrease occurred in Norway during 1947-1948 during which time a mass vaccination with B C G. was carried out. The effect of the latter was presumably a reduction in the incidence of primary tuberculous infection. Earlier detection and segregation of open tuberculous cases may also be an important factor.

It is interesting to ponder the possible influence which present-day antimicrobial drug therapy for tuberculosis, streptococcal upper respiratory and other infections may be having. It seems quite likely that their wide-spread use may be resulting in a significant reduction in the over-all incidence.

Case Reports

The following are examples which illustrate a few interesting and puzzling aspects of this disease.

No. 1. A.S.—This sixteen year old female started to work as a maid at the Nova Scotia Sanatorium in December 1943, coming in contact with patients frequently. Her chest x-ray on January 7, 1944, was negative and Vollmer patch test on January 20. 1944 was negative. Tuberculin test, 1st strength P.P.D. on February 2, 1944 was positive (+). On February 4th she had a sudden onset of pain in the epigastrium, aggravated by deep breathing and lifting. Dry pleurisy? She also had symptoms of acute coryza. At the same time she had typical erythema nodosum and her temperature was elevated to 102°F. A chest x-ray taken on February 12th showed hilar enlargement and parenchymal infiltration on the left side—primary tuberculous complex. Vollmer patch test on February 29th was positive (+). On February 23rd a pelvic examination was performed because of profuse discharge, P.V., of two weeks duration with diagnosis of (1) acute gonorrhea, and (2) trichomonas vaginalis vaginitis. A chest x-ray on March 9th showed improvement in the parenchymal lesion, but on May 10th another chest x-ray showed extension. Pneumothorax was started on May 15th with satisfactory progress thereafter and she was discharged on August 3, 1944 to continue pneumothorax at home. Diagnosis: Pulmonary tuberculosis—primary infection stage—active, improved.

Comment: This case illustrates the occurrence of erythema nodosum at the time of development of allergy produced by primary tuberculous infection, as indicated by tuberculin conversion. It is possible that the tuberculin test acted as a precipitating factor but this was not considered likely. It is of interest to note that this patient had three other infections: upper respiratory, gonnorrhea and trichomonas vaginalis vaginitis, any one or all of which may have been a provocative agent.

No. 2. J L.—a 28 year old male was admitted to the Nova Scotia Sanatorium on May 21, 1946, with exposure to tuberculosis unknown. History states that he developed streptococcal sore throat on March 12, 1946, and erythema nodosum on March 19, 1946. He was admitted to the Victoria General Hospital, Halifax, where he stayed for four weeks. He had arthritis in the wrists, elbows and knees. X-rays showed marked bilateral hilar gland enlargement and apical parenchymal infiltration on the right side. He was therefore transferred to the Nova Scotia Sanatorium. On admission his temperature was 99°F, sedimentation rate 36 m.m., 1st strength P.P.D. was negative and 2nd strength P.P.D. was positive (+). An x-ray of the chest which was taken on May 21, 1946, and compared with that taken at the Victoria General Hospital showed no change. Sputum and gastric washings were negative for tubercle bacilli. Bronchoscopy revealed secretions from the right and left sides which were negative for tubercle bacilli. Specimens from the trachea were positive for tubercle bacilli. Pneumothorax was initiated on the right side with satisfactory progress. He was discharged in June 1947 to continue pneumothorax. In July 1947 he developed a small effusion on the right side

of the chest which persisted. By January 1948 he had oedema of the legs with massive effusion in the right hemithorax, and repeated aspirations of 1000 to 1500 c.c. He was admitted to the Toronto General Hospital in May 1948. The oedema had reached the upper thoracic level, the liver and spleen were enlarged and the pulse was paradoxical. The systolic blood pressure was diminished on inspiration, 1800 c.c. of fluid was removed from the right side. The liver function was impaired with low serum protein. A chest x-ray showed the hilar enlargement still present. A diagnosis of constrictive pericarditis was made and exploration on June 8, 1948 confirmed this. Most of the tissue removed was typical tuberculous granulation. Histologically a few areas were remarkably similar to Boeck's Sarcoid. Swabs from the pleura and pericardium and two small pus pockets were negative for tubercle bacilli, both microscopically and on culture. Marked clinical improvement occurred. However, oedema and poor health later developed with high fever and fluid on the left side. He was admitted to the Victoria General Hospital in April 1949 and died in June 1949.

Comment: The etiology of the erythema nodosum in this case was considered to be tuberculosis. This was based on the positive tuberculin test, hilar gland enlargement and parenchymal disease process, bronchoscopic specimens positive for tubercle bacilli and subsequent pathological report of tuberculous pericardial granulation tissue. Some doubt was cast on this decision by the pathological report of one area of tissue remarkably similar to sarcoidosis. Could this man not have had this disease with superimposed tuberculosis, the former possibly being the cause of the erythema nodosum? It is to be noted that the tuberculin test was only very weakly positive. The streptococcal upper respiratory infection was probably a provocative factor.

Conclusion

Briefly, Erythema Nodosum is believed to be a non-specific hypersensitivity reaction in a predisposed individual produced by any one of several infectious, toxic or chemical agents. Its greatest significance lies in the relationship to tuberculosis where it serves as a warning and an urgent stimulus to uncover this condition, treat it, and search for the source of infection. Treatment with cortisone may be indicated in acutely ill or stubborn cases of long duration, always accompanied by antimicrobial drugs where a tuberculous etiology is known or suspected.

Summary:

1. The literature has been reviewed in connection with the different aspects of erythema nodosum.
2. Results and conclusions of a study of several case reports encountered at the Nova Scotia Sanatorium and Victoria General Hospital are outlined.
3. Comments are made and impressions given concerning controversial issues.
4. Two illustrative case reports are presented.

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Early Recognition of Mental Illness

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THERE are certain generalities I would like to present to you, before getting directly to my subject. These I believe, have a bearing on the early recognition of mental illness. They are as follows:

(A) It is extremely important to diagnose mental illness early. In most cases the treatment of mental illness is difficult and the earlier the diagnosis can be made the more likely are we to be successful. In some cases, for example, Schizophrenia, delay does not only increase the difficulty but may actually be disasterous for the patient.

(B) It should always be remembered that mental disease is extremely prevalent. In Canada, almost fifty per cent of our hospital beds are taken up with mental patients. It is said that one third of all patients going to doctors do not suffer from organic illness, but from disease of psychogenic origin.

A predecessor of mine, Dr. W. H. Hattie, used to open his lectures to fourth year medicine by remarking that most medical students were interested in difficult surgery such as taking out gall bladders, etc. Dr. Hattie would point out that the new doctor might be in practice for ten years before he would be called upon to carry out such procedures, but that he would certainly see a psychiatric patient within the first hour of his practice.

(C) At the present moment, the public is sometimes ahead of some of our profession in their attitudes towards mental illness. It is not uncommon to have patients say that they had discussed the matter of psychiatric referral with their doctor some months or years before the referral was actually made, only to be told that the physician saw no need for psychiatric referral at that point, because the patient was not "crazy" and therefore, did not need psychiatric consultation.

Many signs of mental illness are neglected because of resistance on the part of medical attendants rather than on the part of the patient.

With this introduction then, let me try to answer the questions regarding early signs of mental illness, Again I would like to start with a generalization that changes in a person's usual behaviour pattern need investigation. In trying to decide how best to describe these changes, it seems to me that there are certain rather characteristic changes at different age periods which need to be stressed. I am therefore, presenting this material in terms of the most likely things that would suggest mental illness at various periods of living. I should stress that I am not doing a complete coverage of these changes, but only talking about the more commonly ones observed.

(A) *Childhood*:

There are two types of problems in this field that are likely to come to your attention during the childhood period.

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(1) *Mental defects*—That is, the child who is deficient in intellectual development most commonly because of defects present at birth, through sometimes the result of brain injury following birth. In general the sort of things that make you feel that the child is mentally defective are the following:

(a) The statement, the child is "a very good baby". I know there is no statement which a mother could make which is any more chilling to the backbone of a psychiatrist than to have her say something like this. The baby who never cries, who never is any problem to mother, may not necessarily be defective, but certainly unless she is a very understanding mother, it is unusual and one of the causes for the baby being so good, may be mental defect.

(b) *The child that does not keep up with the mile-stones of development.*

By and large, children develop in a fairly definite pattern. eg. Sitting up by themselves at about six months, walking and talking somewhere between one and two years. If a child is significantly delayed in these things and in other areas of development, one is always suspicious of mental defect. However, it is an important precaution not to jump to conclusions too rapidly when they are based on the developmental milestones and especially one should not pick out only one area of development and feel that the child is defective because of failure here. This is especially true with regard to talking. There are many other things which will effect development besides intelligence. One may cause unnecessary worry for parents if one immediately jumps to the conclusion that the slow talking child is defective.

(c) *The child fails in school.*

Very often mental defect does not come to anyone's attention until the child has to tackle the curriculum that is laid down by our educational authorities. This is planned for the average child and children both above and below average may have difficulty in adjusting to it. Certainly failure in the first two grades of school should arouse the suspicion of mental defect as one of the possible causes.

(2) *Behaviour problems*

The second type of psychopathology that may present itself during childhood is children who have behaviour problems. These generally depend upon some sort of emotional upset. The first issue to be settled is to make sure that one really has a behaviour problem. One hears many complaints from parents about their children when a problem is not really present. All I am going to talk about here are the things which would make you think that the difficult behaviour which the child is presenting is more than the usual difficult behaviour of childhood and should be referred to some competent person or clinic for survey.

The following points, I think, would suggest that a problem is really one deserving such attention.

(a) When a child exhibits unsatisfactory behaviour as a habitual mode of response. All of us act unsatisfactorily at times. This is especially true of

children. When the usual response is of this type, then we should be concerned about it.

(b) *Behaviour which is quite out of keeping with the age period which the child is in*—Much of the unsatisfactory behaviour that children show to adults is perfectly normal behaviour for their age period. For example, it is perfectly normal for a child of three to wet the bed and for a child of three or four to have temper tantrums. When, however, one finds bed-wetting in a child of ten or twelve and temper tantrums much later than that, this is behaviour which is distinctly abnormal at those age periods and requires attention.

(c) *Behaviour which does not respond to common sense management.*

All children have problems at times and most of these we can deal with by utilizing their common sense. However, when this fails, special help should be sought.

(d) *The chronically unhappy child*

It does not seem to me that any child should be unhappy most of the time and when this situation exists we should look further into it.

(e) *Behaviour which is characterized by running away*

By this I do not mean physically running away but I mean withdrawing from contacts with other people, refusing to struggle any more, staying by one's self, etc. etc. This withdrawing sort of behaviour is the sort of thing which often is not disturbing to adults, In fact, such a child is apt to be well behaved and not make a nuisance of himself and thus to receive adult favor by his withdrawing tactics. However, we know that some of the most difficult mental health problems occur in this child who runs away from his problems. By and large, the child who stays and aggressively fights his problems is apt to be in better mental health than he who runs away. The lonely solitary child then, who seems to have little aggression and who cannot get along with his fellows, is one that should receive mental hygiene attention.

(B) *Adolescence and early adulthood*

The period of adolescence and early adulthood, that is, roughly from about fourteen to twenty-four is a period in which there is a considerable upswing in the prevalence of mental illness. This is borne out by the charts of admissions to mental hospitals. The most common illness occurring in this period is Schizophrenia. It is perfectly true that adolescence is always a stormy period, but it is a safe rule to regard behaviour disturbances as serious during this time, and if there is any doubt at all, to refer to the most competent opinion that is available in your neighbourhood. This warning is especially pertinent to behaviour which is characterized by progressive withdrawal, that is, the child who stays away from his contemporaries and also from adults. He does not socialize well and loses interest in the affairs of real life, such as sports, athletics, his studies, and so on. He becomes more and more concerned with day-dreaming and with preoccupations with religion, philosophy, etc. Gradually, these ideas may get more bizarre and they may assume a paranoid

tinge, that is, he may feel that people are against him. This sort of picture is extremely alarming, and may well be the precursor of schizophrenic illness.

(C) *Adult*

Manic Depressive Psychosis

This is a common type of illness, occurring in adulthood, characterized commonly by gross exaggeration of spirits either in the direction of them being too high or too low. In depression, the patient complains of being in low spirits, he sleeps poorly, waking up early in the morning. He loses weight, feels fatigued, and often has sexual difficulties, such as impotence or amenorrhea. Too often, these symptoms are not recognized as part of mental illness, and are treated as amenias, low blood pressures, etc., until the patient makes a suicidal attempt. Since it is more fashionable to discuss physical complaints in our culture, the patient is apt to stress the physical aspects of the depression and not mention his feelings unless he is encouraged to do so by his physician. This is well illustrated in a case I knew a number of years ago who had a recurrent manic-depressive psychosis and whenever he went into a depressed phase, would complain of pain in the abdomen. On one occasion, he complained to his physician about this, and was told that what he needed was a rest and alkali powders. The rest prescribed was to take a hunting trip, and in the process of taking the hunting trip, he blew about half his skull away with a high-powered rifle in a suicidal attempt. Mild depressions are common and are easily missed unless the physician is on the alert for them. The opposite side of the picture, the manic patient, also may be missed for a time. This is most commonly in the so-called hypomanic phase, when the patient is elated, has grandiose ideas, and loses control of such areas as financial investments, sexual behaviour and so on. During this period, he may simply seem to be more extroverted, more cheerful than usual, and it may be impossible to convince him that he is ill, and almost as impossible to convince his family. The danger of this period is that the patient may get into difficulties which may ruin his whole life. At one time we had a girl being treated for neurosyphilis in our clinic, who originally developed a hypomanic attack, and during this time behaved in a way which was quite foreign to her usual behaviour and thus got herself a syphilitic infection which has ruined her whole life.

(3) *Neurosis*

Adult living is the time when neurotic symptoms are most commonly brought to the doctor's attention. It should be remembered that neurotic illnesses are extremely common. In our culture, there is a tendancy to hide behind a physical diagnosis, and it is very usual to have a neurotic patient complain that he has low blood pressure, that he is amenic, that he is suffering from infection and so on and so on. The point is, that in our society with our modern methods of medical investigation, there are few people that can get through a complete physical examination without showing some signs of physical disability, and if the doctor is ready to ascribe all sorts of symptoms to minor physical changes, the patient will certainly hide behind them as long as he can. The physician should be sure, then, that the physical changes one finds can really account for symptoms. One should also note that it is possible to have organic disease and neurosis existing in the same patient, and that one

does not protect from the other. The diagnosis of neurosis, should always be a positive diagnosis, that is, it should not be based on the mere ruling out of physical disease. If a patient has a complaint and no physical disease, and if he has no positive signs of neurosis, the final diagnosis should be "undiagnoised" and not a "neurosis". The sort of positive evidence that one makes a diagnosis on, is the following:

(a) Signs of anxiety should either be present at the moment or should have been present in the patient's past history. These include such things, as cardiac palpitations, indigestion, diarrhoea, feelings of tension, sleep disturbances, with difficulty of getting to sleep and frightening dreams.

(b) The patient's past history should contain evidence of neurotic manifestations. It is extremely unusual for a person to get neurotic in adulthood for the first time. Usually, one finds a history of neurotic traits in childhood, such as bed-wetting, night terrors, of difficulties in school or work, marriage difficulties or of frequent ill health. A scrutiny of the patient's health history, with inquiry as to why such procedures as appendectomy were carried out, often give you exactly the nformation you need for such a diagnosis.

(c) The patient's previous personality makeup. Usually a neurosis occurs in a patient who has signs of instability throughout his whole personality makeup. Here, you find the compulsive person that always has to follow a routine, the anxious person who always finds something to worry about, and all sorts of other manifestations that we recognize as signs of emotional disturbance.

(d) Precipitating factors. Neurotic illness does not come out of the blue, rather it has a cause, and if one is not able to find some precipitating factor which brought about symptoms of this particular kind, one should be hesitant about accepting them as neurotic.

(e) The family history. Neurosis begins in childhood, and one nearly always finds a story of the type of family background that would produce instability in children. It should be understood that this is not a matter of inheriting neurosis but rather of having unstable parents who are not able to provide the sort of emotional background which makes for adult stability.

(4) *Paranoid developments*

The third type of mental illness that is commonly seen during the period of adult living, are those in which people develop suspicions of other people. These suspicions are often in the field of marital infidelity. Such patients are extremely convincing and unless one is alert to the possibility of such developments, one may find oneself being dragged into family difficulties on the side of the patient, only to find that the story that the patient has told is entirely delusional. As well as getting into marital problems, these patients commonly get into legal difficulties and when one hears a story of persecution by other people one should always be on the alert for this type of development.

(d) *This involutional period.* This is the period roughly from forty-five to sixty-five, which is commonly called a "change of life." This is a period which vies with the adolescent and early adulthood period for the number of mental breakdowns that occur. These ae commonly blamed on the meno-

pause because they occur at the same time, but actually such changes are usually the result of a rather rigid sort of person who has to make changes in his life pattern. In other words, it would be better to talk about change of living than about change of life, and to put stress on these personal patterns and personal problems rather than on endocrine changes. The charasteristic type of illness that one finds during this period is the depression of the agitated type, characterized by low mood, by great tendancy to blame oneself for minor peccadilloes of the past, a great tendancy to guilt feelings, very often physical complaints which are in the nature of delusions, a patient saying that there is something wrong with his stomach or bowels and then going on to say that he has no stomach or bowels anymore. These patients are extremely ill and suicidal and they cannot be argued out of their delusions. On the other hand, they can be helped quickly and effectively with electro-shock treatment, and therefore, it is important to make the diagnosis early.

(E) *The Senile period.* It is rather dangerous to attempt to define the senile period. Roughly, it is the time from sixty on. The characteristic sort of mental illness that one gets during this period is one which depends on an actual structural change is the cerebral cortex, such as arteriosclerosis, senility, general paresis, brain tumor. This type of organic change of the cerebral cortex is characterized by the individual having defects in his recent memory and defects in his judgement. It should be strongly insisted on that the patient who has defect in recent memory and who shows judgement defects has some sort of disease of the cerebral cortex until it is proven otherwise. And this is so, even though the physical examination, the X-Ray and other tests may be negative. It is extremely important that one should be on the alert for this kind of thing, because far too often the symptoms of brain tumor are these behaviour changes and the diagnosis is put off until physical symptoms appear. It is therefore, necessary to stress the fact that behaviour changes occurring after forty should be considered the result of organic cerebral change. However, one should modify this statement by pointing out that other types of mental illness do occur in this period of old age, particularly depressions, and there is too great a tendancy to diagnose all behaviour changes as depending on some sort of cerebral cortical senile change. Only recently we had the opportunity of treating a woman of seventy-four with a depression who has made an excellent recovery with electro-shock treatment. However, she was in a nursing home for seven months untreated because everyone believed that this was a matter of old age, and that no one could do anything for her. It should be stressed that any type of mental illness may occur in old age and while that depending on cerebral cortical changes is most prevalent, yet one should make sure that one is not dealing with some other type of illness which can be successfully treated.

SUMMARY

The above is an attempt to summarize a few concepts which should be helpful in the early recognization of mental illness. It must be realized that the whole field has not been covered and that accuracy has sometimes been sacrificed in the need of making some rather dogmatic statements which the public health nurse can have in mind in the course of her daily work. With these as a background and with the information having become a belief to her that mental illness is common, I am sure that she can be a potent factor in aiding with early diagnosis and in getting the patients to the physicians at a time when treatment can frequently be most effective.

Diabetic Diets*

The Children's Hospital
Halifax, N. S.

THE diabetic diets in use range from values of 800 to 2,800 calories. The principles of calculating each diet are based on the food exchange lists which have been devised by the American Diabetic Association. The number of calories required and the way these calories are distributed between CHO, Protein and Fat can be calculated by following the pattern as set up by the "Diabetic Progression", which gives us the amounts we need for each age and weight, from one to fourteen years.

In arranging these diets, we have allowed for more CHO and higher protein which is more satisfactory for the growth and development of children, while the fat is moderately low.

The scheme of the diet progression is given below, followed by the exchange lists with their values. A sample 1,600 calorie diet with menus is appended.

Diabetic Diet Progression

AGE	WEIGHT	CALORIES	CHO	PRO	FAT
1 year	20	1000	125	80	20
3 years	30	1200	125	60	50
4 years	35	1400	150	75	55
5 years	40	1600	150	70	80
7 years	50	1800	180	80	85
8 years	55	2000	200	85	95
9 years	60	2200	250	100	90
10 years	70	2400	300	100	90
12 years	80	2600	325	110	95
14 years	100	2800	350	110	105

FOOD ALLOWED AS DESIRED

Negligible Carbohydrate, Protein and Fat

Clear Broth
Bouillon
Rhubarb
Sweeta on Sucaryl

*Compiled by G. B. Wiswell, M.D., and used on the wards of The Children's Hospital.

Gelatin, unsweetened
Rennet Tablets
Cranberries
Lemon
Tomato Juice
Pepper
Spices
Vinegar

LIST 1 MILK EXCHANGES

Carb. - 12 gm. Protein - 8 gm. Fat 10 gm. Calories 170

	MEASURE	GM.
*Milk, whole	1 cup	240
Milk, evaporated	½ cup	120
*Milk, powdered	¼ cup	35
*Buttermilk	1 cup	240

*Add 2 fat exchanges if fat free (skim milk)

1 pint of milk = 20 oz = 2½ cups = 3 Meat + 2 Fat Exchanges + 3 fruit
= 2 Bread + 2 Meat + 3 Fat

LIST 2 VEGETABLE EXCHANGES

A—These vegetables may be used as desired in ordinary amounts. Carbohydrates and Calories negligible.

Asparagus
Broccoli
Brussels Sprouts
Cabbage
Cauliflower
Celery
Chicory
Cucumbers
Escarole
Eggplant
'Greens'
Beet
Chard
Collard
Dandelion
Kale
Mustard
Spinach
Turnip
Lettuce
Mushrooms
Okra
Pepper
Rhubarb
Saurkraut
String Beans, young
Summer Squash
Tomatoes

B—Vegetables: 1 serving equals ½ cup equals 100 grams
Carb. 7 gm: Protein 2 gm: Calories 36

Beets
Carrots
Onions
Peas, green
Pumpkin
Rutabaga
Squash, winter
Turnip

LIST 3 FRUIT EXCHANGES

Carbohydrates 10 gm., Calorkes 40

	MEASURE	GM.
Apple	1 sm. (2″ diam.)	80
Applesauce	½ cup	100
Apricots, fresh	2 medium	100
Apricots, dried	4 halves	20
Banana	½ small	50
Berries: Straw: Rasp: Black	1 cup	150
Blueberries	⅔ cup	100
Canteloupe	¼ (6″ diam)	200
Cherries	10 large	75
Dates	2	15
Figs, fresh	2 large	50

Figs, dried	1 small	15
Grapefruit	½ small	125
Grapefruit juice	½ cup	100
Grapes	12	75
Grape Juice	¼ cup	60
Honeydew Melon	⅛ (7″ diam)	150
Mango	½ small	70
Orange	1 small	100
Orange Juice	½ cup	100
Papaya	⅓ medium	100
Peach	1 medium	100
Pear	1 small	100
Pineapple	½ cup	80
Pineapple Juice	⅓ cup	80
Plums	2 medium	100
Prunes, dried	2 medium	25
Raisins	2 Tbsp.	15
Tangerine	1 large	100
Watermelon	1 cup	175

Dried Fruits are High in sugar contents

LIST 4 BREAD EXCHANGES

Carbohydrate - 15 gm., Protein 2 gm. Calories 68

	MEASURE	GM
Bread	1 slice–(½″ thick)	25
Biscuit, Roll	1 (2″ diam)	35
Muffin	1 (2″ diam)	35
Cornbread	1 (1½″ cube)	35
Flour	2½ Tbsp.	20
Cereal, cooked	½ cup	100
Cereal, dry (flake & puffed)	¾ cup cup	20
Rice, Grits, cooked	½ cup	100
Spaghetti Noodles, etc. cooked	½ cup	100
Crackers, Graham (2½″ sq.)	2	20
Oyster	20 (½ cup)	20
Saltines (2″ sq)	5	20
Soda (2 ½″ sq.)	3	20
Round, thin (1½″ diam)	6-8	20
Vegetables		
Beans & Peas, dried, cooked (lima, navy, split pea, cowpeas, etc.)	½ cup	90
Baked Beans, no pork	¼ cup	50
Corn	⅓ cup	80
Parsnips	⅔ cup	125
Potatoes, white-baked, boiled	1 (2″ diam)	100
Potatoes, white - mashed	½ cup	100
Sponge Cake, plain	1 (1½″ cube)	25
Ice Cream (Omit 2 fat exchanges)	½ cup	70

Measure all foods after they have been cooked. Be accurate.

LIST 5 MEAT EXCHANGES

Protein 7 gm., Fat 5 gm., Calories 73

	MEASURE	GM.
Meat & Poultry (med. fat)..... (beef, lamb, liver, chicken, etc.)	1 oz.	30
Cold Cuts (4½" sq. ⅛" thick).....	1 slice	45
Fish: cod, mackerel, haddock, halibut	1 oz.	30
scallops.....	1 oz.	30
oyster, shrimp, clams.....	5 small	45
sardines.....	3 medium	30
Cheese, cheddar, American.....	1 oz.	30
cottage.....	¼ cup	45
Egg.....	1	50
*Peanut Butter.....	2 Tbsp.	30

*Limit use or adjust carbohydrate.

Measure Meat after cooking—remove bones and fat.

LIST 6 FAT EXCHANGES

Fat 5 gm. Calories 45

	MEASURE	GM.
Butter or Margarine.....	1 tsp.	5
Bacon, crisp.....	1 slice	10
Cream, light, 20%.....	2 tbsp.	30
Cream, heavy, 40%.....	1 tbsp.	15
Cream Cheese.....	1 tbsp.	15
French Dressing.....	1 tbsp.	15
Mayonnaisse.....	1 tsp.	5
Oil or Cooking Fat.....	1 tsp.	5
Nuts.....	6 small	10
Olives.....	5 small	50
Avocado.....	⅛ (4" diam)	25

WESTON'S BISCUITS

Number of biscuits that are equal to one slice (30 grams) bread.

Cocoanut Snacks.....	3	Oval Arrowroots.....	3
Crackettes.....	6	Select Tea.....	4
Digestive.....	2½	Saltines.....	6
Peanut Cookies.....	2	Golden Brown Sodas.....	6
Macaroon Crisps.....	1	Vanilla Wafers.....	3
Shortbread Fingers.....	3	Graham Wafers.....	4
Oatmeal Crisps.....	2½	Oatmeal Cookies.....	1

With each substitution of biscuits in first column, omit one teaspoon or five grams of butter from diet.

McCORMICK'S BISCUITS

Number of biscuits that are equal to one slice (30 grams) bread.

Rob Roy.....	2½	Graham Wafers.....	4
Society.....	2½	Sunwheat.....	2
Oval Arrowroot.....	3	Jersey Cream Sodas.....	7

CHRISTIE'S BISCUITS

Number of biscuits that are equal to one slice (30 grams) bread.

Ritz	7½	Pantry Cookies	1½
Cheese Ritz	7½	Digestive	2
Arrowroot	2½	Holland Rusks	1½
Social Tea	4	Premium Sodas	6
Graham Wafers	2½	Sultana	1

Omit one teaspoon of butter when Ritz or Cheese Ritz are used.

GINGERALE AND GRAPE JUICE

¼ cup unsweetened grapejuice (you may buy Welch's or Jordan's without added sugar). Place in glass and fill with chilled diabetic gingerale. Serve with ice cubes, if desired.

Food Value: 10 grams carbohydrates. Therefore this drink is the equivalent of 100 grams of 10% fruit.

LEMONADE

1 tablespoon lemon juice
1 glass water
Saccharin to taste

Serve ice cold—you could garnish with mint. For one glass you do not need to calculate for food value.

ZERO SALAD DRESSING

½ cup tomato juice.
2 tablespoons lemon juice or vinegar.
1 tablespoon onion
Salt, pepper, chipped parsley, horseradish or mustard may be added.
Combine in jar with fitted cap. Shake well before using.

SAMPLE 1600 Calorie Diet

Use only as your Doctor prescribes.

Carbohydrate 150 grms. Protein 70 gms. Fat 80 gms.: Calories 1600.

TOTAL ALLOWANCE OF FOODS FOR THE DAY

AMOUNT	KIND OF FOOD	LIST
1 pint	Skim milk 2½ cups	1
1 pint	Milk 2½ cups	1
Any amount	Vegetable Exchanges A	2A
1	Vegetable Exchanges B	2B
4	Fruit Exchanges	3
3	Bread Exchanges	4
4	Meat Exchanges	5
7	Fat Exchanges	6

DIVIDE THIS FOOD AS FOLLOWS:

Breakfast

0 Fruit Exchange from List 3
1 Meat Exchange from List 5
1 Bread Exchange from List 4
2 Fat Exchanges from List 6
1 cup milk

Lunch or Supper

2 Meat Exchanges from List 5
1 Bread Exchanges from List 4
Any amount Vegetable from List 2A.
2 Fruit Exchange from List 3
1½ cups milk from List 1
2 Fat Exchanges from List 6.

Dinner or Main Meal

2 Meat Exchanges from List 5
1 Bread Exchange from List 4.
Any amount Vegetables from List 2A
1 Vegetable Exchange from List 2B.
2 Fruit Exchanges from List 3.
2 Fat Exchanges from List 6.
1½ cups Skim Milk.

Bedtime

1 cup skim milk from List 1.
1 Bread Exchange from List 4.
1 Fat Exchange from List 6.

SAMPLE MENUS—1600 Calories

These menus show some of the ways Exchange Lists can be used to vary the Meal Plan.

Breakfast
Tomato Juice 4 oz.
Egg 1
Toast 1 slice
Butter 2 teaspoons
1 cup milk

Lunch or Supper
(cheese 1 oz.: ham 1 oz. bread 1 slice: butter 1 tsp.)
Lettuce and Tomato Salad
Zero Salad Dressing (recipe page 9)
Apple 2 small
Milk 1½ cups (12 ounces)

Dinner
Hamburg Patties 2 ounces
Mashed Potato ½ cup.
Carrots ½ cup.
Spinach Green Vegetables.
Bread 1 slice.
Butter 2 teaspoons.
Banana 1 small
1½ cups Skim Milk.

Bedtime
Skim milk 1 cup (8 ounces).
Graham Crackers 2.
Cream Cheese 1 tablespoon.

Breakfast
Tomato Juice 4 oz.
Cereal cooked ½ cup
Boiled Egg 1
Light Cream 4 tablespoons
1 cup milk.

Lunch or Supper
Fish 2 oz.
Radishes, Celery, Lettuce, Tomato.
Potato ½ cup
Salad Dressing
Bread 1 slice
Butter 1 teaspoon
Grapefruit ½ 1 orange
Milk 1½ cups (12 ounces)

Dinner
Tomato juice, small glass.
Roast Beef 2 oz.
Steamed Rice ½ cup.
Green Peas ½ cup.
Mixed Green Salad.
Zero Salad Dressing (recipe page 9).
Bread 1 slice.
Butter 2 teaspoons.
Skim Milk 1½ cups.

Bedtime
Skim milk 1 cup (8 ounces).
Saltines 5.
Butter 1 teaspoon.

No-value liquids as tomato juice, consomme, tea, lemonade, etc. (see list) may be added to above as desired.

Modern Concepts of Physical Medicine

G. J. H. Colwell, M.D.
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THE use of physical agents and methods in the treatment of disease has its origin in ancient times. Primitive man used heat, cold, water and sunlight in various ways to treat his ills. The first man who bathed a painful limb in a woodland stream unknowingly initiated the art of hydrotherapy. The early founders of physical medicine as we know it today on this continent very often were interested in the use of one specific modality such as electricity, ultraviolet light or therapeutic exercises. Electrotherapy was the prime interest of many of these pioneers. In recent years, however, the all-consuming interest in electrotherapy and its innumerable gadgets has given way to a broader interest in the whole field of physical treatment.

Because of this broader application of physical methods in the treatment of a large variety of physical and mental ills, it becomes necessary to define physical medicine and attempt to discover its boundaries in relation to the whole field of medicine.

Physical medicine is concerned with the diagnosis and treatment of disease by physical methods. The physical methods are included under the two main headings, physiotherapy and occupational therapy. Physiotherapy includes the use of infra-red and ultraviolet radiations, cold, massage, heat from many sources, sound waves, water, electricity and therapeutic exercises. We find use for a variety of sources of heat because of special qualities, such as: ease of application, depth of penetration, portability or otherwise, variation of intensity. I suppose our most important physical modality is therapeutic exercise, which is classed as passive, active, active assisted, or active resisted.

Occupational therapy is defined as medically prescribed activity, with a definite therapeutic purpose. In regaining lost function following injury of a limb, there is much that can be accomplished by occupational therapy which could not be done by physiotherapy. The activity prescribed will naturally vary according to the disability treated. The scope of occupational therapy is continuing to broaden with the increasing interest in rehabilitation.

Physical medicine takes a small and variable part in the task of diagnosis. It varies greatly with the interests of the physiatrist, types of cases seen, and the equipment available in the office or hospital department. If a considerable number of traumatic cases are being treated, equipment may be available to provide electrical currents of known duration and intensity, which can be used in diagnosis of nerve injuries, and the differential diagnosis of disease causing muscle paralysis and weakness. Occupational therapy facilities are used in testing work tolerance, and in grading degree of impairment of function, which is of great importance in assessment of pension or compensation cases.

The largest task of physical medicine is in the treatment field. During World War I, because of the number of traumatic cases, the use of physical methods received a great stimulus, and the demand for occupational and

physiotherapists was considerable. After the war, physiotherapy in some instances, was used indescriminately, and without proper indications, and medical men looked askance at hospital rooms filled with machines that were claimed to cure a multitude of ills. Research, teaching and experience over the intervening years have shown the value of certain physical methods, used with the basis of an adequate diagnosis, proper indications, and definite aim of treatment. In some medical schools, departments of physical medicine have been organized, and medical students are introduced to the use of physical methods through each year of their course.

It was particularly during World War II that the whole scope of physical medicine broadened, to take part in the concept of total rehabilitation. Because physical methods are prominently used in the treatment of severely disabled persons, it was inevitable that physical medicine should have a large part to play in physical rehabilitation plans for disabled veterans. Recently, the American Congress of Physical Medicine changed its name to the American Congress of Physical Medicine and Rehabilitation. This, I believe, has led to the misconception that physical medicine has taken unto itself the field of rehabilitation. This, of course, cannot be true because rehabilitation is an aim and a concept that must apply to all fields of medical endeavour, and cannot be taken over by any one medical specialty. However, physiatrists, through their work with disabled persons, have been accumulating valuable information, and have developed techniques for increasing the mobility and level of self-care of such patients. These techniques involve the well known use of braces, crutches, wheel chairs, therapeutic exercises, etc., as well as training by occupational therapists in the problems of daily care, such as dressing, self-feeding, toilet care. Useful appliances have been individually designed to assist those with specific weakness or limitation of motion to overcome problems in self-care or vocation. This has opened up a whole new field for occupational therapy, which is now so extensively used in testing activities of daily living, devising special aids, and training patients to overcome the loss of various functions.

We find physical medicine today taking part in the treatment of many diseases. Most of the patients receiving physiotherapy, for instance, are receiving, or have received, some other form of medical or surgical treatment. In arthritis, exercises graded and varied according to the stage of the disease, are essential in most cases. The Canadian Arthritis and Rheumatism Society has two mobile physiotherapists in the Halifax-Dartmouth area, who visit the homes and assist with exercises ordered by the family physician. Physiotherapy and occupational therapy are used in conjunction with drugs and surgery in the treatment of many neurological disorders of traumatic, developmental or degenerative origin. Plans are being made by the Cerebral Palsy Association of Halifax to open a cerebral palsy out-patient physiotherapy clinic, in association with an academic class for these patients.

Hot packs, hydrotherapy and therapeutic exercises provide mainstays of the early treatmet of paralytic poliomyelitis. The return of function of hemiplegic patients following cerebrovascular accident can be assisted by common-sense measures to prevent contractures; techniques aimed at neuro-

muscular reeducation, by passive, and eventually active assisted exercises, and simple occupational therapy. Straightforward, basic plans for use following trauma such as fracture, are well known, but too often neglected, with resultant prolongation of disability.

In some chest diseases, breathing exercises and postural drainage are being used along with other measures. In many instances, nurses can be trained to carry out effective post-op exercises in chest surgery cases, with resultant decrease in post operative morbidity. In the field of psychiatry, occupational therapy forms an integral part of the inpatient treatment program in many clinics. For many years, ultraviolet has been used effectively in combination with various ointments in the treatment of psoriasis.

In a busy general hospital, physiotherapists may be seen at work with patients from every ward or department. This in itself will give an idea of the scope of physical medicine, and will indicate that exact boundaries of the specialty are impossible to define according to the diseases treated.

I fear that too few doctors know enough of the background training, capabilities and limitations of a physiotherapy technician. The usual three year course leading to a diploma in physiotherapy includes considerable basic training in anatomy, physiology, neurology, and medical physics. Lectures are given by physicians and surgeons from medical school staffs, dealing with diseases commonly treated by physical methods. The students are required to have knowledge of the important signs and symptoms of these diseases, and the recognized methods of treatment. However, it must be emphasized that physiotherapy technicians are not trained as diagnosticians. In order for patients to get any benefit from the skills of these qualified therapists an adequate diagnosis must be provided, and the aim of treatment must be clearly outlined. They are trained to work under the orders of doctors. If the supervising doctor is to get the most from physiotherapy facilities available, he should acquaint himself with the indications for, and limitations of, the types of physical treatment in common use. In this way, valuable time of a skilled technician will not be wasted on worthless treatments.

The demand for facilities for physical medicine is prominent now in Nova Scotia, and in recent years, some facilities have been increased. At present there are about 18 physiotherapists and 5 occupational therapists working in the Province. Camp Hill Hospital has large and busy physiotherapy and occupational therapy departments. The Victoria General has 7 physiotherapists, with one to two of these working fulltime in the polio clinic. The hospital also has 6 physical medicine beds, for patients whose treatment consists mainly of physiotherapy. One physiotherapist is located in Sydney, half her salary being paid by the Polio Foundation, and half by the Sydney City Hospital. St. Marthas Hospital has had physiotherapists on the staff, and I believe there is now an opening for one there. The Royal Canadian Naval Hospital at Stadaconna is well staffed in its physiotherapy and occupational therapy departments.

Plans are going forward for the establishment of a rehabilitation centre in Halifax for treatment of disabled persons of the Province. The organiza-

tion formulating these plans is the Nova Scotia Council for the Rehabilitation of Disabled Persons. Adequate personnel and facilities for physiotherapy and occupational therapy will be located in this centre, as much of the rehabilitation process will take place in these departments.

There is at present a severe shortage of physiotherapy and occupational therapy technicians, and hospital deficits continue to be a problem, but I predict that in the near future many more of our community hospitals in Nova Scotia will install basic equipment for physical treatment, and hire therapists, possibly on a part-time basis, sharing personnel with near-by communities. I also believe that increasing awareness of the use of physical medicine in combination with our other medical skills will be noted among practising doctors, because of changing emphasis in medical school teaching in years to come.

Speaking of Specimens

Bertha Ogilvie Archibald
Halifax, N. S.

MANY years ago a man was sent from Cape Breton to the old Victoria General Hospital. He was supposed to be suffering from appendicitis. Dr. Charles Puttner was at that time an exceedingly busy man, being not only the Assistant Superintendent and Pharmacist, but also the Radiologist. He made a rather hasty X-ray picture, with the patient only partly undressed, and reported no sign of appendicitis. The patient was therefore sent home. He was a very heavily built man, but, returning some months later, was seen to be a mere shadow of his former self. He became exceedingly ill and an operation was performed at once. From the appendix the surgeon removed a black-headed steel pin (a beaded pin) about 1½ inches in length. It was heavily encrusted in a solid mass.

On viewing the X-ray plates taken previously, the doctors noticed a rather unusual spot on the plate. Dr. Puttner then explained that the patient was only partially undressed when the X-ray was taken, and he thought the spot was a 'bachelor's' button, (a nail thrust through the cloth in place of a button). He was as surprised as the surgeons when viewing the specimen.

The patient was removed to the Emergency Room, which was next to the Pharmacy—or Dispensary as it was then called. He made a wonderful recovery and soon returned home. He did not take the specimen. It resided in a receptacle on one of the shelves in the workroom of the O.R. and aroused much curiosity and many comments.

It would no doubt be an unheard-of thing to-day, for specimens from the Operating Room to be sent to the Pharmacy. There were several instances however, when specimens were sent to the Pharmacy in the old Victoria General Hospital.

The Fairbanks Scales were kept in the Pharmacy, in a corner by the Stimulant Cupboard. One day a male nurse from the O.R. burst into the Pharmacy, carrying a very large basin in which reposed a huge cyst. I was requested to weigh the contents of the basin and report same. It seemed unbelievable that such a mass could have come from any human being.

At about this time a rumor circulated (even filtering down to the Pharmacy) to the effect that a visiting Surgeon, opening the door of the Operating Theatre, said to the surgeon who was operating—"What do you think you are doing, Doctor?" The busy surgeon, without lifting his eyes, replied "Oh, just removing this woman from her cyst!" Perhaps that was the specimen that was sent to the Pharmacy to be weighed!

29th Annual Dalhousie Refresher Course

The week of October 24th-28th with the presentation of the 29th Annual Refresher Course of the Faculty of Medicine of Dalhousie University, will, we are sure, offer much of interest and stimulus to the medical practitioners of the Atlantic Provinces.

Contributions by the Faculty are quite varied and several papers will include findings regarding local conditions as brought out in surveys which have been conducted here recently.

Five distinguished medical teachers are participating in our programme this year and we acknowledge their generous contribution gratefully.

* * *

As the John Stewart Memorial Lecturer we welcome Professor Edward G. D. Murray, Canada's senior and best known bacteriologist, who retired this year after twenty-five years as Professor and Chairman of the Department of Bacteriology and Immunology at McGill University. He is now Visiting Professor in Medical Research at the University of Western Ontario. He has served as a member of numerous Canadian, American and International Societies concerned with biology and bacteriology in particular and during both World Wars did considerable research on current problems. Since 1936 he has been a member of the Board of Editors of Bergey's Manuel of Determinative Bacteriology.

Possessed of very versatile interests, he is a keen fisherman and naturalist and has served his city as a member of the Montreal City Council for a number of years.

As a scientist and as an individual Dr. Murray has a highly acclaimed national and international reputation.

* * *

The Guest in Surgery is to be Dr. W. Keith Welsh who will be known to many of you during his years of service with the R.C.N. While in Halifax he also served as Surgical Specialist at Camp Hill Hospital. Receiving his Fellowship from the Royal College of Surgeons (England) in 1930 he returned to Toronto where he practised general and orthopaedic surgery for eight years before the war.

Dr. Welsh now holds the appointment of Surgeon in Chief of St. Michael's Hospital and Associate Professor of Surgery at the University of Toronto.

* * *

Dr. Robert B. Kerr, Professor and Head of the Department of Medicine, University of British Columbia and Head of the Department of Medicine, Vancouver General Hospital has agreed to be our guest speaker in Medicine. Dr. Kerr, who has had a distinguished Army career, was Associate Professor of Medicine at the University of Toronto before he took up his appointment in Vancouver.

* * *

Dr. Edward B. D. Neuhauser, who comes to us from Boston is the only guest from across the border. He is Radiologist in Chief at the Children's

and Infants' Hospitals of Boston and Associate Clinical Professor of Radiology, Harvard Medical School. He is Consultant in Radiology for a number of hospitals, including the International Grenfell Association Hospital, St. Anthony, Newfoundland. A versatile speaker Dr. Neuhauser is renowned for his unusually wide knowledge in all fields of medicine.

* * *

Dr. Arnold L. Johnson is a graduate of McGill University and is at present Demonstrator in the Department of Paediatrics and Lecturer in the Department of Physiology. He has pioneered in the field of cardiac investigation by catheterization and is Director of the Department of Cardiology at the Montreal Children's Hospital. Keenly interested in this field Dr. Johnson has the talent of creating a similar interest in his hearers.

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PROGRAMME

29th ANNUAL DALHOUSIE REFRESHER COURSE - OCTOBER 24th-28th, 1955

Monday, October 24th, 1955.

8.30	Registration.
9.00-10.00	Surgical Clinic.
10.00-10.50	"The Treatment of Pneumonia"—Dr. R. B. Kerr.
10.50-11.00	Intermission
11.00-11.50	"Biochemical Tests and Their Interpretations"—Dr. C. W. Holland, Dr. D. J. Tonning, Dr. F. J. Moya.
11.50- 1.00	Department of Ophthalmology and Oto-Laryngology: "Lid Infections and their Treatment"—Dr. L. G. Holland. "Management of the Acute Ear"—Dr. J. S. Hammerling. "The Watery Eye"—Dr. C. F. Keays "Common Disorders in the Nose and Throat in Children"—Dr. A. G. Shane.
1.30	Luncheon—Courtesy of the Victoria General Hospital.
2.30- 3.15	"Problems in Thryroid Disease"—Dr. R. B. Kerr.
3.15- 3.50	"A Review of Cortisone Therapy"—Dr. W. I. Morse.
3.50- 4.00	Intermission.
4.00- 5.30	Clinical Pathological Conference—Dr. W. A. Taylor.

"TALKS ON TRAUMA".

Sponsored by the Committee on Trauma, American College of Surgeons, Chairman: Dr. A. B. Campbell.

8.30- 9.50

1. The Histopathology of Trauma—Dr. W. A. Taylor.
2. Common Urinary Tract Injuries—Dr. C. L. Gosse.
3. Fractures of the Elbow Joint—Dr. E. F. Ross.
4. Shock in Trauma—Dr. C. C. Stoddard.
5. The Ruptured Abdominal Viscus—Dr. B. K. Coady.
6. The Compound Fracture—Dr. A. L. Murphy.

Tuesday, October 25th, 1955.

9.00-10.00 Medical Clinic.
10.00-10.50 "Management of Severe Haemorrhage from the Upper Gastro Intestinal Tract."—Dr. W. K. Welsh.
10.50-11.00 Intermission.
11.00-11.50 Medical Clinic—"Diabetes"—Dr. R. B. Kerr.
11.50- 1.00 Department of Urology.
"What Can We Expect from Prostatic Surgery?"—Dr. C. L. Gosse.
"Treatment and Expectation in G. U. Tuberculosis"—Dr. F. G. Mack.
1.30 Luncheon—Courtesy of the Victoria General Hospital.
2.30- 3.15 "The Diagnosis and Management of Lesions of the Oesophagus"—Dr. W. K. Welsh.
3.15- 3.50 "The Lipids of the Blood"—Dr. C. B. Weld.
3.50- 4.00 Intermission.
4.00- 5.30 Round Table—"Abdominal Pain—Acute and Chronic".
Moderator: Dr. H. B. Atlee.
Dr. W. K. Welsh, Dr. R. B. Kerr, Dr. J. H. Charman, Dr. R. M. MacDonald.

Wednesday, October 26th, 1955.

9.00-10.00 Dermatological Clinic.
10.00-10.50 "The Diagnosis and Management of Diverticulitis of the Colon"—Dr. W. K. Welsh.
10.50-11.00 Intermission.
11.00-11.30 "The Migraine Headache"—Dr. C. S. Marshall.
11.30-12.00 "Physical Management following Cerebral Accidents"—Dr. G. J. H. Colwell.
12.00-12.30 "Venereal Arthritis"—Dr. J. F. L. Woodbury.
12.30- 1.00 "Diagnosis and Management of Small Bowel Obstructions"—Dr. C. E. Kinley.
1.30 Luncheon—Courtesy Department of Veterans Affairs.
2.30- 2.50 "Evaluation of Newer Drugs in Psychiatry"—Dr. J. F. Nicholson, Dr. S. Hirsch.
2.50- 3.15 "The Contribution of the Electroencephalogram to Clinical Problems"—Dr. W. Leslie.
3.15- 3.50 "The Histological Changes and Clinical Effects of Peripheral Ischaemia"—Dr. J. A. Noble, Dr. N. Nemethy.
3.50- 4.00 Intermission.
4.00- 5.00 Round Table—"Disability Assessment."
Moderator: Dr. L. C. Steeves.
Dr. A. B. Campbell, Mr. H. S. Farquhar, Dr. R. S. Henderson, Dr. C. J. Macdonald.
6.00 Dinner—Lord Nelson Hotel—Open to Physicians Only.
8.00 John Stewart Memorial Lecture.
"Determinate Variability in Bacterial Infections"—Dr. E. G. D. Murray.
(Open to Physicians and Guests)
Facilities of Ward Room Officers' Mess, H.M.C.S. Stadacona available following John Stewart Memorial Lecture—Physicians and Guests.

Thursday, October 27th, 1955.

9.00-10.00 Paediatric Clinic.
10.00-10.50 "Rheumatic Fever"—Dr. A. L. Johnson.
10.50-11.00 Intermission.

11.00-11.45 "X-Ray Findings in Upper and Lower Respiratory Infections"—Dr. E. B. D. Neuhauser.

11.45- 1.00 Department of Obstetrics and Gynaecology.
Moderator: Dr. H. B. Atlee.
"Safe Guarding the Newborn"—Dr. J. McD. Corston.
"Minimal Pre-Natal Care Every Woman in Nova Scotia Should Get"—Dr. M. G. Tompkins.
"Handling The Cancer Patient from the General Practitioner Standpoint"—Dr. W. R. C. Tupper.
"What to do for Unusual Bleeding under 35 years of Age"—Dr. I. A. Perlin.
Each paper will be discussed following presentation by the Departmental Staff.

1.30 Luncheon—Courtesy of the Children's Hospital.

2.30- 3.15 "Cancer of the Breast"—Dr. N. H. Gosse.

3.15- 3.50 "Behaviour Problems in Children"—Dr. F. A. Dunsworth. Staff—Child Guidance Clinic.

3.50- 4.00 Intermission.

4.00- 5.30 Round Table—"Antibiotics and Chemotherapy."
Moderator: Dr. C. J. W. Beckwith.
Dr. J. G. Aldous, Dr. C. A. Gordon, Dr. A. L. Johnson, Dr. E. D. G. Murray.

8.30 Ashburn Golf and Country Club—Social Evening and Dancing.
Sponsored by the Halifax Medical Society.

Friday, October 28th, 1955.

9.00- 9.30 Neurosurgical Clinic—Dr. W. D. Stevenson.

9.30-10.00 "Salk Vaccine"—Dr. A. R. Morton.
Discussion: Dr. C. B. Stewart.

10.00-10.50 "Bone Diseases in Children—X-Ray Findings"—Dr. E. B. D. Neuhauser.

10.50-11.00 Intermission.

11.00-11.45 "Cardiac Problems in Infants"—Dr. A. L. Johnson.

11.45-12.05 "Gastric Feeding in Surgical and Medical Problems"—Dr. D. J. Tonning.

12.05-12.30 "Hazards in Paediatric Anaesthesia"—Dr. A. S. Wenning.

12.30- 1.00 "Immediate Care of the Newborn"—Dr. G. B. Wiswell.
LUNCH.

2.30- 3.15 "X-Ray Diagnosis of the More Common Lesions of the Gastro-Intestinal Tract in Children"—Dr. E. B. D. Neuhauser.

3.15- 3.50 "Problems in the Surgical Care of the Paediatric Patient"—Dr. J. W. Merritt.

3.50- 4.00 Intermission.

4.00- 5.30 Round Table—"Paediatric Problems."
Moderator: Dr. N. B. Coward.
Dr. R. S. Grant, Dr. A. L. Johnson, Dr. E. B. D. Neuhauser, Dr. P. M. Sigsworth.

FIFTH ANNUAL MEETING

CANADIAN PUBLIC HEALTH ASSOCIATION
ATLANTIC BRANCH

Cornwallis Inn – Kentville, Nova Scotia

November 9th and 10th, 1955

Tuesday November 8th.

8.00 p.m. Meeting of Executive.

Wednesday, November 9th

8.30- 9.00 **REGISTRATION:** All those in attendance are expected to register. A fee of $4.00 is charged to cover in part the expenses of the meeting.

9.00- 9.15 **ADDRESS OF WELCOME:**

Miss Phyllis J. Lyttle, R.N., President.
Director, Nursing Service, Department of Public Health.

GENERAL SESSION:

Chairman: Miss P. J. Lyttle.

9.15- 9.45 The Employer's Place in an Occupational Health Program.
Miss Mildred I. Walker, R.N.
Senior Nursing Consultant, Occupational Health Division,
Department of National Health and Welfare, Ottawa.

DISCUSSION: Dr. E. A. Watkinson, Chief, Division of Occupational Health, Ottawa.

9.45-10.15 The Present Status of the Disabled Person's Allowance.
Mr. H. S. Farquhar,
Director of Old Age Assistance and Disability Pensions,
Halifax.

DISCUSSION: Mr. Frank Wellard, Coordinator of Rehabilitation, Halifax.

10.15-10.30 RECESS (Coffee available at 15c per cup)

10.30-12.15 Symposium: The Function of the Local Medical Officer of Health.

Moderator: Dr. C. B. Stewart,
Dean, Dalhousie Medical School.

(a) The Function and Status of the Local Medical Officer of Health.
Dr. John C. Wickwire, M.O.H., Liverpool.

(b) His Relationship to Municipal Councils.
Mrs. Mable Hamilton, Municipal Clerk, Truro.

(c) Can His Function and Status be Improved?
Dr. Sam Marcus, M.O.H., Bridgewater.

(d) Relationship of M.O.H. to Department of Public Health.
Dr. J. J. Stanton, D.M.H.O., Pictou.

DISCUSSION

12.30 LUNCHEON:

Chairman: Miss P. J. Lyttle, R.N.

Special Speaker:

Dr. F. C. Pace, Medical Consultant Special Weapons Section, Civil Defence Health Services, Ottawa.

AFTERNOON SESSION—Chairman: Mr. Frank Wellard.

2.30- 3.00 Federal Health Grant Planning in Nova Scotia.
Miss E. A. Electa McLennan, B.A., A.M., R.N.

3.00- 3.15 Dental Hygienists in Nova Scotia's Health Program.
Dr. W. Gordon Dawson, Director
Division of Dental Services, Halifax.

3.30- 5.00 Sectional Meetings—Programs listed on Page 4
(a) Sanitation—Valley Room.
(b) Nutrition—Card Room.
(c) Mental Health—Cornwallis Room.
(d) Occupational Health—Cornwallis Room.

7.00- 7.30 Reception—Cornwallis Room.
Delegates and Wives, Guests of the Atlantic Branch.

7.30 Annual Dinner—Ball Room.
Courtesy the Department of Public Health of Nova Scotia.
Speaker: Dr. Leonard Miller,
Deputy Minister of Health for Newfoundland.

Thursday, November 10th

9.00-10.15 Annual Business Session—All Members.

GENERAL SESSION—Chairman: Dr. A. C. Guthro,
1st Vice-President, Bras D'Or.

10.15-10.45 What Has the Department of Welfare to Offer the Needy.
Miss Beatrice Crosby,
Ass't. Director of Mothers Allowances, Halifax.

10.45-11.00 Recess (Coffee available at 15c per cup)

11.00-12.30 Symposium on Child and Maternal Health.
Moderator: Dr. E. L. Eagles, D.P.H., Director,
Division of Child and Maternal Health, Halifax.

(a) The part of a Practicing Physician in his Office and Community.
Dr. Henry B. Ross, Ass't. Professor of Pediatrics,
Dalhousie University, Halifax.

(b) The Hospital's Part Within the Hospital and Within the Community.
Sister Peter Claver, St. Martha's Hospital, Antigonish.

(c) The Part Played by the Public Health Nurse,
Miss Maude MacLellan, R.N., Public Health Nurse, Kentville.

(d) The Importance of Good Nutrition and the Part of the Nutritionist in Cooperation with the Hospital, The Public Health Nurse and the Physician.
Miss Hazel Roland, B.Sc., Nutritionist,
Department of Public Health, Yarmouth.

(e) Discussion.

12.30- 2.30 Luncheon Recess.

AFTERNOON SESSION—Chairman: Dr. J. S. Robertson, D.P.H.
Deputy Minister of Health.

2.30- 3.00 Restaurant Sanitation and the Local M.O.H.
Dr. S. E. Bishop, M.O.H., Kentville.

3.00- 3.30 The Present Status of Vaccination Against Poliomyelitis.
Dr. E. M. Fogo, D.P.H.,
City Department of Health,
Halifax.

3.30- 4.00 Erythema Nodosum and Tuberculosis in Children.
Dr. A. H. Barss, Rose Bay and Dr. W. I. Bent, D.P.H., Bridgewater.
Discussion: Dr. R. C. Young, Nova Scotia Sanatorium, Kentville.

4.30 Afternoon Tea—Nova Scotia Sanatorium.
Host: Hon. Geoffrey Stevens, Minister of Public Health, and Mrs. Stevens.

EVENING SESSION—Chairman: Dr. G. M. Smith, D.P.H., Windsor.
Chairman, Medical Advisory Board, N.S.T.A.

SECTION ON DISEASES OF THE CHEST
SPONSORED BY
THE NOVA SCOTIA TUBERCULOSIS ASSOCIATION

7.30- 8.00 Pleurisy with Effusion: Its Significance in Modern Treatment.
Dr. S. J. Shane, F.R.C.P. (Can.).
Medical Superintendent, Point Edward Hospital, Sydney.

8.00- 8.30 The Future of Tuberculosis: Trends in Evidence.
Dr. C. J. W. Beckwith, D.P.H.
Director of Tuberculosis Control for City of Halifax.

8.30- 9.00 Coal Miners Lung Disease.
Dr. W. A. Taylor, Asst. Provincial Pathologist, Halifax.

9.00- 9.30 The Common Cold: Its Potential, Its Prevention, Its Treatment.
Dr. C. B. Stewart, M.P.H.,
Dean, Dalhousie Medical School.

Sectional Meetings—Wednesday, 3.30-5.00 p.m.

Sanitation—Chairman: Mr. R. D. MacKay, Director,
Division of Environmental Hygiene, Halifax.

3.30- 3.35 Opening Remarks by Chairman.

3.35- 3.50 Well Water Pollution—Mr. W. J. Chisholm, Sanitary Inspector.

3.50- 4.00 Discussion.

4.00- 4.30 Symposium on Milk.
(a) Cleansing, Sterilizing and Storage of Dairy Farm Utensils.
Mr. G. MacDonald, Sanitary Inspector.
(b) Dairy Farm Inspection.
Mr. D. Tupper, Sanitary Inspector.
(c) Organoleptic, Chemical and Physical Testing of Raw Milk.
Mr. B. Doane, Sanitary Inspector.

4.30- 4.40 Discussion.

4.40- 5.00 Restaurant Sanitation— (a) A Moving Picture Film.
(b) Discussion.

NUTRITION: Chairman: Dr. Juanita Archibald, Director,
Division of Nutrition, Department of Public Health, Truro.

3.30- 4.10 Nutritional Management of Medical and Surgical Patients.
Dr. D. J. Tonning, Associate Professor of Medicine,
Dalhousie University.

4.10- 5.00 Excerpts from the 2nd Nutrition Conference, Ottawa, July, 1955.

4.10 (a) Current Trends
Miss Mary T. Doyle, Nutritionist,
Department of Public Health, Sydney.

4.30 (b) Looking to the Future.
Miss Barbara R. Robertson, Nutritionist,
Department of Public Health, Halifax.

4.50 (c) Question Period.

Mental Health: Sponsored by the Canadian Mental Health Association, Nova Scotia Division.
Chairman: Mr. Andrew J. Crook, Executive Secretary,
Nova Scotia Division, C.M.H. Association.

3.30- 4.00 The Place of Psychiatry in Industry.
Dr. R. J. Weil, Assistant Professor of Psychiatry,
Dalhousie University.

4.00- 4.30 Mental Health Aspects of Pregnancy and Childbirth.
Dr. H. K. Hall, Assistant Professor of Psychiatry,
Dalhousie University.

4.30: 5.00 Early Recognition of Mental Illness.
Dr. R. O. Jones, Professor of Psychiatry,
Dalhousie University.

OCCUPATIONAL HEALTH:
To be combined with the Mental Health Section—see above.

Reservations for Hotel Accommodations:

These should be made as soon as possible directly with the management of the Cornwallis Inn, Kentville, N. S., giving proposed time of arrival and stating that you are attending the meeting of the Canadian Public Health Association, Atlantic Branch.

Acknowledgements:

The officers and members of the Atlantic Branch gratefully acknowledge the contribution to the work of the Association made by the following:

SUSTAINING MEMBERS:

General Electric X-ray Corporation Limited.
Charles E. Frosst and Company.
T. Eaton Company Limited Maritimes.
Ferranti Electric Limited, X-ray Division.

OTHERS:

Department of Public Health of Nova Scotia.
(Sponsorship of Annual Dinner)
Mr. Geo. C. Robinson (Edwards of Canada Limited)—donation
United Fruit Company.

N.B.—Local Medical Officers of Health are reminded that under provincial legislation expenses incurred by them at this meeting as well as transportation costs are recoverable from their municipalities.

Health Insurance

A NATIONAL Advisory Committee on Health Insurance has been organized by The Canadian Medical Association and the services of this Committee have been offered to the Minister of National Health. It has been recommended that all Divisions of The Canadian Medical Association also form Advisory Committees which will stand ready and willing to advise or assist the Provincial Ministers of Health in matters relating to Health Insurance.

At the Annual Meeting of The Medical Society of Nova Scotia the following members of the Society were appointed to the Nova Scotia Advisory Committee, Doctors D. M. MacRae, H. J. Devereux, H. E. Christie, H. F. McKay, and F. J. Barton. The services of this Committee have been offered to the Honourable Minister of Health for the Province of Nova Scotia.

As the subject of Health Insurance is slated to be one of the items on the Agenda of the forthcoming Dominion-Provincial Conference, renewed interest in this subject is very much apparent. It would therefore appear to be an opportune time to publish the Statement of Policy of The Canadian Medical Association which was adopted at the Annual Meeting held in Toronto in June, 1955.

At the Annual Meeting of The Medical Society of Nova Scotia held in Amherst in September 1955, the Society went on record as affirming and approving this Statement of Policy.

STATEMENT ON HEALTH INSURANCE

(The Canadian Medical Association Statement of Policies and Principles on Health Insurance in Canada, June 1955)

On the question of Health Insurance
The Canadian Medical Association:

1. Strives for the best medical care for all the people of Canada and reaffirms its long established policy of giving consideration to and co-operating in proposals, official or unofficial, that are in the public interest and genuinely aimed at the improvement of the health of the people.

2. Will gladly participate in the formulation of programmes designed to make high quality medical services more readily available and which respect the essential principles of medical practice.

3. Approves of the adoption of the principle of contributory health insurance and favours a plan or plans which will assure the development and provision of the highest standards of health services, preventive, curative and rehabilitative, provided the plan be fair both to the insured and all those rendering the services.

4. Having seen demonstrated the successful application of the insurance principle in the establishment of the voluntary prepaid medical care plans recommends the extension of these plans to cover all residents of Canada, with financial assistance from public funds where this is required.

5. Recommends, where it becomes evident that the voluntary medical care plans cannot achieve adequate coverage, that provincial governments collaborate in the administrative and financial task of extending health insurance to all through the medium of the voluntary repayment plans.

To Maintain Consistent Progress in Health Care:

6. Health is a state of complete physical, mental and social well-being and not merely the absence of disease. Among the factors essential to the achievement of good health are adequate nutrition, good housing and healthful environmental conditions generally; facilities for education, exercise and leisure; and not least, wise and sensible conduct of the individual and his acceptance of personal responsibility for maintenance of health.

7. Each province should be adequately served by a well-organized Department of Public Health providing personal preventive services wherever possible through the practicing physician.

8. The community's responsibility for health services includes not only maintenance of a high level of environmental conditions and the provision of an efficient preventive service, but assurance that adequate medical facilities and services are available to every member of the community whether or not he can afford the full cost.

9. The confidential nature of the patient-doctor relationship must remain inviolate. The patient must have freedom of choice of doctor, and the doctor free acceptance of patient except in emergency or on humanitarian grounds.

10. The granting of a license to practise medicine was designed primarily to protect the public. Therefore it is in the interest of the patient that all who desire licensure to practise a healing art should be required to conform to a uniformly high standard of preliminary education and of training in the recognized basic sciences, as well as to furnish proof of adequate preparation in the clinical and technical subjects.

11. Standards of medical services should be maintained at the highest possible level through:

(a) adequate facilities for clinical teaching in the medical colleges and hospitals

(b) postgraduate training for all medical practitioners at frequent intervals

(c) expanded programmes of medical research.

To Assure Economy and Efficiency in the Provision of Services:

12. Hospitals, health departments, and all other health agencies should co-ordinate their activities so as to provide their services more effectively and economically.

13. Hospitals should be located, and their facilities and size determined on a planned, regionalized basis to assure the availability of hospitals where they are needed, the provision of technical assistance to smaller hospitals by the larger, and the ready transfer of patients as required.

14. An adequate system of institutional facilities and services requires the balanced development of diagnostic facilities, active treatment general hospitals, rehabilitation centres, chronic care facilities (including mental and tuberculosis hospitals) and home care programmes.

15. Lay and professional organizations and government health agencies should participate in community, provincial and federal health planning activities.

In the Application of the Insurance Method to Payment for Medical Services:

16. The opportunity of insuring through a prepayment medical care plan should be available to every Canadian, including dependents.

17. Benefits of a health insurance plan should include preventive, diagnostic, treatment and rehabilitation services, and the services of specialists and consultants should be available as required.

18. The methods of remuneration of medical practitioners and the rates thereof should be as agreed upon by the representative bodies of the profession and the insuring agency. In the provision of personal health services where the usual doctor-patient relationship exists, it is the view of The Canadian Medical Association that remuneration on a basis of fee for services rendered promotes high quality of medical care.

19. The provision of medical services under any plan of health insurance should be undertaken only by qualified and licensed physicians.

In the Event of Government Participation in the Universal Extension of Health Insurance to all Citizens:

20. The introduction of health insurance legislation should be preceded by adequate consultation with the organized medical profession and other groups affected.

21. Health insurance should be administered by an independent, non-political commission representative of those providing and those receiving the services. Matters of professional detail should be determined by committees of the professional groups concerned.

22. Appointments of medical personnel to the Commission and its staff should be made only with the approval of organized medicine in the respective province.

23. The various services should be introduced as benefits by stages, careful planning being given to the order in which each is introduced.

24. Cash sickness benefits, if provided, and the health services benefits should be administered from separate funds.

College of General Practice and General Practitioners Society

A joint meeting of the Nova Scotia Chapter of the College of General Practice and the General Practitioners Society of Nova Scotia was held at the Fort Cumberland Hotel, Amherst, on Thursday, September 8th.

Doctor F. M. Fraser was in the Chair.

After considerable discussion it was decided to continue the General Practitioners Society in nominal form so that doctors who have not yet elected to become members of the College will have an official body to represent their views.

The Executive Officers of the Nova Scotia Chapter of the College of General Practice were re-elected en bloc as follows:

Chairman—Doctor F. M. Fraser, Halifax.
Secretary—Doctor C. H. Reardon, Halifax.
Treasurer—Doctor H. J. Devereux, Sydney.
Provincial Representative to the Board of Representatives—Doctor A. G. MacLeod, Dartmouth.
Chairman of Hospitals Committee—Doctor J. R. Macneil, Glace Bay.
Chairman of Education Committee—Doctor H. I. MacGregor, Halifax.

The Executive Officers of the General Practitioners Society to be elected were:

President—Doctor A. G. MacLeod, Dartmouth.
Secretary—Doctor F. M. Fraser, Halifax.

F. MURRAY FRASER

REGISTRATION

102nd ANNUAL MEETING

THE MEDICAL SOCIETY OF NOVA SCOTIA

September 6th, 7th, 8th, 9th, 1955

Amherst, N. S.

Dr. Wm. O. Coates, Amherst
Dr. Donald F. Smith, Halifax
Dr. G. J. H. Colwell, Halifax
Dr. T. B. Murphy, Antigonish
Dr. W. L. Muir, Halifax
Dr. Fred J. Barton, Dartmouth
Dr. W. M. Grant, Amherst
Dr. T. C. Routley, Toronto, Ontario
Dr. C. H. Young, Dartmouth
Dr. S. S. B. Gilder, Oakville, Ontario
Dr. G. B. Shaw, Halifax
Dr. H. W. Schwartz, Halifax
Dr. D. Drury, Amherst
Dr. M. G. Tompkins, Glace Bay
Dr. J. G. Petrie, Montreal, Quebec
Dr. J. A. Langille, Amherst
Mr. L. W. Holmes, Toronto, Ontario
Dr. F. Murray Fraser, Halifax
Dr. J. E. Park, Oxford
Dr. G. R. Deveau, Arichat
Dr. J. B. MacDonald, Stellarton
Dr. Harvey F. Sutherland, Sydney
Dr. W. E. Hirtle, Sackville, N. B.
Dr. G. McK. Saunders, Amherst
Dr. Arthur L. Murphy, Halifax
Dr. A. G. MacLeod, Dartmouth
Dr. Arthur W. Ormiston, Sydney
Dr. Hugh E. Christie, Amherst
Dr. Arthur J. R. Brady, Halifax
Dr. I. McC. Todd, Advocate Harbour
Dr. W. A. Hewat, Lunenburg
Dr. G. J. LeBrun, Bedford
Dr. Allan R. Morton, Halifax
Dr. W. T. M. MacKinnon, Amherst
Dr. Bruce MacCannell, Amherst
Dr. Norman G. Glen, Amherst
Dr. H. A. Myers, Amherst
Dr. D. J. Tonning, Halifax
Dr. M. E. B. Gosse, Halifax
Dr. Peter O. Hebb, Dartmouth
Dr. H. D. O'Brien, Halifax
Dr. Norman H. Gosse, Halifax
Dr. A. F. W. Peart, Toronto, Ontario
Dr. E. F. Brooks, Toronto, Ontario
Dr. G. K. Smith, Hantsport
Dr. R. E. Price, Amherst
Dr. R. G. A. Wood, Lunenburg
Dr. D. F. MacInnis, Shubenacadie
Dr. D. G. Black, Digby
Dr. R. O. Jones, Halifax
Dr. M. R. Macdonald, Halifax
Dr. S. E. Bishop, Kentville
Dr. B. F. Miller, Halifax
Dr. D. R. Sutherland, Amherst
Dr. J. H. Fraser, Westville
Dr. A. L. Sutherland, Sydney
Dr. J. A. McDonald, Glace Bay
Dr. A. F. Weir, Hebron
Dr. H. J. Devereux, Sydney
Dr. Dan Murray, Tatamagouche
Dr. Gordon Macdonald, Sydney
Dr. K. A. MacKenzie, Halifax
Dr. Edwin F. Ross, Halifax
Dr. W. W. Bennett, Bridgewater
Dr. C. L. Gosse, Halifax
Dr. W. Alan Curry, Halifax
Dr. F. J. Granville, Stellarton
Dr. J. A. Noble, Halifax
Dr. R. A. P. Fleming, Halifax
Dr. C. M. Kincaide, Halifax
Dr. J. H. Charman, Halifax
Dr. D. R. Davies, Oxford
Dr. V. O. Mader, Halifax
Dr. J. McD. Corston, Halifax
Dr. H. C. Still, Halifax
Dr. R. L. Aikens, Halifax
Dr. Charles J. W. Beckwith, Halifax
Dr. Cecil E. Kinley, Halifax
Dr. Ian E. Mackay, Stellarton
Dr. M. F. Fitzgerald, New Glasgow
Dr. E. Hofstaedter, Springhill
Dr. C. G. Harries, New Glasgow
Dr. J. P. McGrath, Kentville
Dr. J. C. Murray, Springhill

Dr. J. S. Robertson, Halifax
Dr. G. MacL. Moffatt, Springhill
Dr. W. A. Murray, Halifax
Dr. J. F. Nicholson, Halifax
Dr. J. C. Wickwire, Liverpool
Dr. Paul Nonamaker, Halifax
Dr. V. H. T. Parker, Stellarton
Dr. A. M. Marshall, Halifax
Dr. J. R. Ryan, Springhill
Dr. A. S. Wenning, Halifax
Dr. A. W. Titus. Halifax
Dr. K. V. Gass, Pugwash
Dr. T. E. Kirk, Halifax
Dr. C. M. Harlow, Halifax
Dr. W. E. Pollett, Halifax
Dr. H. B. Whitman, Westville
Dr. D. M. MacRae, Halifax
Dr. C. B. Stewart, Halifax
Dr. P. E. Belliveau, Meteghan
Dr. C. E. Stuart, New Glasgow

Report of the Nominating Committee at The Annual Meeting of The Medical Society of Nova Scotia, September 8, 1955

President—Doctor R. O. Jones, Halifax.

First Vice-President—Doctor J. R. McCleave, Digby.

Second Vice-President—Doctor A. L. Murphy, Halifax.

Treasurer—Doctor C. H. Young, Dartmouth.

Secretary—Doctor M. R. Macdonald, Halifax.

Legislative Committee—Doctors J. McD. Corston, A. R. Morton, Halifax; J. A. Langille, Amherst; W. A. Hewat, Lunenburg; C. L. MacMillan, Baddeck; P. E. Belliveau, Meteghan.

Cancer Committee—Doctors N. H. Gosse, Halifax; A. W. Ormiston, Sydney; F. J. Granville, Stellarton; R. E. Price, Amherst; H. A. Fraser, Bridgewater.

Public Health Committee—Doctors J. C. Wickwire, Liverpool; C. B. Stewart, Halifax; D. F. Macdonald, Yarmouth; H. F. Sutherland, Sydney; R. A. Moreash, Berwick.

Historical Committee—Doctors K. A. MacKenzie, W. L. Muir, H. W. Schwartz, Halifax; M. G. Tompkins, Glace Bay; Dan Murray, Tatamagouche; L. M. Morton, Yarmouth.

Workmen's Compensation Board Committee—Doctors J. V. Graham, J. H. Charman, C. H. Reardon, Halifax; J. R. Ryan, Springhill; H. B. Whitman, Westville.

Editorial Board Committee—Doctors C. J. W. Beckwith, W. K. House, H. C. Still, Halifax.

Medical Museum Committee—Doctor C. M. Harlow, Halifax and secretaries of the Branch Societies.

Cogswell Library Committee—Doctors A. W. Titus, Halifax.

Medical Economics Committee—Doctors H. J. Devereux, A. L. Sutherland, Sydney; J. B. Tompkins, Dominion; D. M. MacRae, Halifax; T. B. Murphy, Antigonish.

Pharmaceutical Committee—Doctors P. S. Cochrane, Wolfville; J. P. McGrath, Kentville; G. D. Donaldson, Mahone Bay.

Public Relations Committee—Doctors F. J. Barton, Dartmouth; L. C. Steeves and H. C. Still, Halifax (Nucleus Committee); A. W. Ormiston, Sydney; J. A. MacCormick, Antigonish; J. H. Fraser, Westville; L. A. MacLeod, Liverpool; B. J. D'Eon, Yarmouth; J. R. Kerr, Annapolis Royal; J. B. Reid, Truro; G. M. Saunders, Amherst.

Divisional Representative, Editorial Board Canadian Medical Association—Doctor W. K. House, Halifax.

Industrial Medicine Committee—Doctors G. C. Macdonald, Sydney; C. E. Stuart, New Glasgow; J. E. Park, Oxford.

Members of Board of Maritime Service Association—Doctor W. E. Hirtle, Sackville, N. B., (1954-56); Doctor H. E. Christie, Amherst (1955-57).

Tariff Committee—Doctors E. F. Ross, J. W. Reid, Halifax; P. O. Hebb, Dartmouth; A. L. Sutherland, Sydney; S. E. Bishop, Kentville.

Rehabilitation Committee—Doctors W. D. Stevenson, G. J. H. Colwell, J. F. L. Woodbury, Halifax.

Committee on Nursing—Doctors H. F. McKay, New Glasgow; J. C. Murray, Springhill, C. J. W. Beckwith, Halifax.

Traffic Accidents Committee—Doctors A. L. Murphy, Halifax; R. G. A. Wood, Lunenburg; J. A. McDonald, Glace Bay; T. C. C. Sodero, Truro.

Nominee to Canadian Cancer Society—Doctor W. R. C. Tupper, Halifax.

Medical Advisory Board Committee to Lay Organizations—Doctors B. F. Miller, H. D. O'Brien, C. H. Reardon, Halifax; P. R. Little, Truro.

Federal-Provincial Health Grants—

Professional Training—Doctor C. B. Stewart, Halifax.
General Public Health—Doctor J. R. Macneil, Glace Bay.
Medical Rehabilitation—Doctor A. W. Titus, Halifax.
Tuberculosis Control—Doctor W. I. Bent, Bridgewater.
Mental Health—Doctor J. F. Nicholson, Halifax.
V. D. Control—Doctor W. A. Hewat, Lunenburg.
Crippled Children's Grant—Doctor J. C. Acker, Halifax.
Cancer Control—Doctor V. D. Schaffner, Kentville.
Child and Maternal Health—Doctor C. G. Harries, New Glasgow.
Radiological—Doctor H. R. Corbett, Sydney.
Laboratory—Doctor H. C. Read, Halifax.
Public Health Research—Doctor J. A. Langille, Amherst.

Representative on the Board of Governors of the V.O.N. (Canada)—Doctor D. M. Cochrane, River Hebert.