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Primary Cancer of the Lung

H. R. Corbett, M.D.

WITHIN the past decade, there has been considerable material in the literature, concerning the incidence and diagnosis of lung malignancy.

Improved radiological investigation has furnished the means whereby diagnosis of pulmonary lesions is facilitated, and, in conjunction with bronchoscopic procedures, a definite increase in the number of primary neoplasm has been reported.

Brines and Kenning[1] in the number of new admissions to the Tumor Clinic of the Detroit Receiving Hospital from 1932 to 1936, state that of 936 cases, 73 were found to have primary pulmonary malignancy in contrast to 122 of the stomach, 122 breast. Of 68 cases tabulated for race and sex, 93% were white and 93% males. It was found to be relatively rare in the colored race. The age incidence was highest between 40 and 60.

The same writers, when considering the outstanding symptoms in a study of 73 patients, found, in order of importance, that cough, chest pains, weight loss and frank hemoptysis were present, and duration varied from one month to one year.

Nathanson[2] states that tumors involving apex of lung may cause considerable pain in the shoulder which may be the only symptom complained of. L. A. Hochberg and M. Lederer[3] state that the character of the onset of primary pulmonary malignancy is extremely variable. The disease may start insidiously simulating a prolonged bronchitis, or it may be acute simulating an acute pneumonic process. Epigastric distress is described as an important complaint; it was elicited in 10 of the 13 patients who had no thoracic symptoms. This symptom was more commonly present when the left side was involved and the process was basal.

With the increasing use of roentgenological facilities by physicians, and publicity campaigns embracing cancer and tuberculosis, it is quite obvious that cases of early malignancy may be found before symptoms are sufficiently marked as to cause the patient to consult the family physician.

It is not the purpose of the writer to present a detailed pathological classification of lung tumors. From an X-ray point of view, after giving a positive opinion we can, in most instances decide whether the tumor has arisen from one of the main bronchial divisions or from the lung parenchyma, and state whether the bony thorax or pleura is involved.

The so-called endobronchial tumors by their growth within the lumen of the large bronchi cause a progressive stenosis and eventually atelactasis of that lobe or part of the lobe supplied by the bronchus, and a localized collapse of lung area may be the first indication on the film. There is progressive extension of the growth and if located in the upper lobe, the lower limits of the lesion present a characteristic "S" shaped curve—the convex part of the "S" being the tumor and the concave portion the atelectatic lung substance.

The primary carcinomata originating in the parenchyma or alveolar spaces are distinctly infiltrative, invading the surrounding parenchyma and

obstructing the bronchi later. These infiltrative types, depending on the location and appearance, may assume a hilar, lobar, or a diffuse nodular character.

The hilar type as its name implies originates in the area adjacent to the hilum and is difficult to detect in its incipiency, therefore must be differentiated from mediastinal new growths and tracheobronchial enlargements of an inflammatory nature.

Originating as a small patch of consolidation in the parenchymatous tissue the infiltrative type of tumor will spread to the point where a lobe is involved (lobar type), the extension being usually by means of the lymphatics. Growth may continue until the entire lung structure has been replaced by the tumor but there is very little displacement of mediastinal structures to the opposite side. Invasion of the chest wall with destruction of a rib or ribs is not uncommon especially in growths situated near the periphery.

At any time during the stage of consolidation, the central portion of tumor growth may undergo caseation and necrosis on account of blood vessel destruction and decrease in blood supply. The resulting cavity may simulate a tuberculous cavity or a lung abscess, there is one point of differentiation, that is the walls in a malignant cavity are thick walled, cryptic and irregular. In the final analysis however, laboratory examination of several sputum specimens will be the deciding factor.

When, on radiological examination, a lung lesion suspicious of malignancy is discovered, it is a matter for differential diagnosis and every assistance that the clinician can give to the radiologist is required, if errors are to be avoided.

X-ray interpretation in terms of lung tumor, depend on (a) atelectasis, (b) consolidation, (c) metastatic deposits especially of the thoracic cage.

Atelectasis may be a result of obstruction from within, such as a mucus plug following operations, non opaque foreign bodies and chronic inflammatory disorders. A careful clinical history, lipiodol injection of the bronchial tree for confirmation and localization of the stenosis, bronchoscopic examination and follow up films are needed before the cause can be firmly established.

The appearances of malignant consolidation, to those who have reviewed a number of cases is usually characteristic unless the growth is so extensive as to blot out the entire lobe or lobes. It is well demonstrated in the hilar form where the peripheral limits present sharp projecting margins and the vascular markings of the healthy lung can be made out through the areas of invasion.

Physical examination of the chest will assist in confirming the presence of consolidation when the growth is of sufficient size as to present changes in percussion and auscultation.

Primary sarcoma of the lung is rare. Sante[4] quoting Ewings classification divides this ill defined group into the (a) diffuse (spindle-celled) sarcoma, (b) peribronchial sarcoma, (c) large round celled sarcoma and (d) lymphosarcoma. It is almost impossible to distinguish them radiographically from the common types of carcinoma.

Case No. 1. G. B.

Male; age, 62; laborer.

Referred for radiological examination of the large bowel. This man's complaints were alternating diarrhoea and constipation, and loss of weight. Chest symptoms were not complained of.

The standard barium enema did not disclose any abnormality but a routine fluoroscopic inspection of the chest, a lesion was discovered in right lung. The report on the film reads "In the right lung is a zone of confluent infiltration at a point between the 3rd and 5th ribs, also a dense rounded opacity extending downward from hilum, very suggestive of pulmonary malignancy.

Another film taken two months later revealed a definite increase in extent of the original infiltration which was defined as a rounded consolidation surrounding the right hilus and extending posteriorly. At this date the patient was rapidly losing weight and complained of a distressing cough.

At the end of another three months, film examination disclosed that the tumor mass had increased to the point where it occupied almost two-thirds of the pulmonary field, no appreciable shift of the heart and mediastinal structures. Death occurred six months after the lesion was first discovered.

Autopsy report by Dr. Ralph P. Smith is as follows:

"The lung shows a necrotic cavity in the gross, but the pancreas a definite small secondary nodule. Histological examination reveals the lung to be the seat of an oat cell Ca. with necrosis suppurative changes and cavitation. The secondary in the pancreas is of the same oat cell Ca. type. Primary is usually in the bronchi, but occasionally may arise from the alveolar epithelium.

Case No. 2. M. N.

White male; age, 83; retired.

Complaints: Pain in the left shoulder, duration one month. X-ray examination revealed a circumscribed area of density in left upper lobe measuring approximately 5.5 cm. in diameter and occupying the pulmonary apex down to a point 2 cms. below the inferior border of clavicle. There is no evidence of fibrosis and trachea is in usual position. The above noted picture is consistant with a new growth similar to the so-called superior sulcus tumor.

Recheck examination three months later reveals a slight downward extension of the growth and malignant involvement of the 2nd rib posteriorly, indicative of a spread to the bony thorax.

Summary

(1) The incidence of primary pulmonary malignancy is much greater than was formerly considered.

(2) Improved diagnostic facilities have made possible more accurate and rapid chest diagnosis.

(3) The symptoms of lung carcinoma are variable and may occur late. In chronic pulmonary lesions over 40, it should always be considered in differential diagnosis.

(4) Two case reports are presented for consideration.

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(1) Osborne A. Brines and John Carl Kenning Amer. Jour. Clin. Path. Vol. 7 No. 2 Mar. 1937.

(2) L. Nathanson, Quart. Bull. Sea View Hospital; April, 1939 iv—326

(3) L. A. Hochberg and M. Lederer, Arch. Int. Med. January 1939, LXiii 80.

Diphtheria and its Prevention

Issued by the Department of Public Health, Halifax, N. S., November 12, 1940.

DIPHTHERIA, essentially a disease of human beings, is a specific acute intoxication. The symptoms are due to toxins produced at the site of the lesion, generally located on the mucous membrane of the throat and nose. Children under five years are particularly susceptible to it and more than half the deaths occur under this age. In the five year period 1934 to 1938, seventy-two Nova Scotians died of this cause. Approximately seventy-five per cent of the deaths were in children six years of age and under.

The period of communicability is thought to begin one to three days before the onset of definite symptoms, and to continue as long as bacilli can be found in the nose and throat. Negative cultures from these areas are the basis for considering the infectious period passed. All cases occurring should be promptly reported, to local health departments, by physicians in attendance. Such restrictions as isolation and quarantine are then imposed. It is unlawful to interfere with authority in this regard. Placards must not be removed by persons other than those delegated for the purpose by local health authorities.

Diphtheria is one disease which is definitely preventable and if preventable, "why not prevented?" It is prevented by the use of a preparation known as toxoid. A small quantity of this preparation is injected beneath the skin on three occasions, three to four weeks apart, with little or no discomfort, particularly in infants. It is preferably given during the first year of life when reactions following its use are not met with, in fact reactions are rare in children under six to eight years. Since so many cases of diphtheria occur between the ages of one and six years, immunization should be especially practised in this age group. In fact the time of choice for administering toxoid is between the ages of six months and one year. Parents and custodians of children are requested to unite with health officers and physicians in order that all children, particularly those of preschool age may be given the protection afforded by toxoid of reliable manufacture. In four or more months after the third of the three doses of plain toxoid has been given, a Schick Test should be carried out to determine whether or not immunity has been produced. In this connection physicians are advised not to use the one dose alum precipitated toxoid. This preparation does not give the immunity claimed for it by some manufacturers and salesmen.

It has been found that reactions following the use of plain toxoid are exceedingly rare in children of six years and under and rare in those eight years and under. In a few children over eight years and in a larger number of adults rather marked reactions may occur. These reactions may be local or both local and general. It is therefore recommended that in these groups preliminary reaction and susceptibility tests be made: that is to say, a Schick test and control. For this purpose special packages of material are marketed known as "Schick Test and Control". A positive Schick means that the person is susceptible to diphtheria and a positive control (reaction test) that he may react unfavourably to ordinary doses of toxoid. Following a positive reaction test, a specially diluted toxoid should be used for immunizing purposes.

In all toxoid distributed by the Department, printed instructions will be found. These should be studied carefully before the actual work of immunization is attempted. Remember that diphtheria is largely a disease of childhood, consequently the most important group to protect against it is the preschool age group.

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Candidates holding a United States degree must be registered in the State of New York.

By Order,

W. GOODMAN,

Secretary.

The Prevention of Streptococcal Infection of Wounds*

RONALD HARE, M.D. (Lond.)

Connaught Laboratories and School of Hygiene
University of Toronto

IF the present conflict resembles that of 1914-18 in any respect whatever, a high proportion of the wounds inflicted will become infected, and these infections of themselves may cause the death or serious disablement of many thousands. Many wounds sustained in civil life are similarly infected. It is therefore of extreme importance that we should consider whether, in the light of the knowledge we now possess, these infections may be prevented.

In general, four varieties of organism infect wounds. Firstly, there are the organisms which cause tetanus, secondly those which cause gas gangrene, thirdly the staphylococci and fourthly the streptococci. The sources of the first three have been known for some time. Both the tetanus and gas gangrene organisms can exist in the form of spores and these may be found with distressing frequency in soil and dirt. It may therefore be assumed that the presence of this undesirable commodity in wounds is fraught with tremendous possibilities. Nor do we need to look very far for the source of the staphylococci because these organisms are invariably present on the skin. But the source of the streptococci has always been a subject on which we had no exact information, although there have been theories in plenty, mostly wrong, to account for their presence in a wound.

The streptococci which are of importance in wounds are almost all those known as haemolytic streptococci or *Str. pyogenes*. The other varieties of streptococci, the viridans and those without action on blood, are for all practical purposes negligible. Now the importance of the haemolytic streptococci as a cause of wound sepsis is not always realized. I cannot give you figures for their incidence in the wounds of civil life but I can for wounds of war-time. In the last war, for instance, it was found that no less than 15 per cent of all wounds examined at a casualty clearing station, that is within an average of 12 hours of the infliction of the wound, were already infected with haemolytic streptococci. In another investigation, this time at a base hospital, 23 per cent were infected and in a third investigation on compound fractures of the femur, more than 90 per cent were found to be infected with these organisms. For this reason, haemolytic streptococcal infection of wounds in war is a very serious problem and I have reason to believe that it may be more important in the wounds of civil life than is usually supposed.

It is obvious that if these infections could be prevented we might reduce very considerably the inevitable toll of war and might also reduce the mortality from injuries in civil life. Up to the present we have been unable even to

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approach this ideal by reason of a number of fundamental difficulties. The most important of these is that hitherto it has not been possible to distinguish pathogenic from non-pathogenic strains of haemolytic streptococci. Perhaps it may surprise you to hear that there are non-pathogenic strains of these organisms. As a matter of fact there are a great many and haemolytic streptococci are, on the whole, fairly widespread in nature. They occur quite frequently on the skin for instance, and although we had always suspected that many of these strains were unable to cause infections in human beings, we were unable to distinguish them from strains which most certainly can. But thanks to the work of Lancefield, we are now able to separate those strains capable of causing infection in human beings from those which cannot. The former are, in bacteriological language, known as group A haemolytic streptococci while those assigned to other groups labelled B to M are almost if not quite harmless for man. Some of these strains, I may mention in passing, are highly pathogenic for lower animals. This test has given us an extremely powerful weapon because we can now take any strain and assess its ability to cause infection in human beings.

Sources of Infection.

Employing these methods, let us consider where the organisms causing infection of wounds can have come from. First, we must ascertain whether the organisms can have been implanted in the wound at the time of infliction. This is the explanation for the presence of these organisms which is put forward almost invariably by surgeons. There is, however, a great deal of evidence that this is not the case. Rifle bullets and shell splinters are probably quite sterile while flying through the air because the heat of the propellant would almost certainly kill any organisms on them. Other objects which may cause wounds such as portions of motor car or machinery are almost certainly not infected with these organisms. They cannot live very long in such inhospitable surroundings and even if purposely placed on them would very soon die. I think therefore that we can, in a general way, exonerate the object which inflicts the wound.

The first part of one's person to encounter the object is usually the clothing through which it passes on its way to the tissues and pieces of cloth may even find their way into the wound. Recently we have been carrying out a very extensive examination of the clothing of ordinary normal individuals and can say quite definitely that the clothing of most of us is not infected with pathogenic strains of haemolytic streptococci. Haemolytic streptococci may be and frequently are present but they are not members of the pathogenic group A. There is, however, an exception. This is the clothing of persons who are so unfortunate as to carry the pathogenic strains in the throat and in these individuals the upper part of the trunk, the pocket handkerchief and the pocket in which it is kept may be infected. The trousers generally escape. The clothing of some carriers is apparently quite uninfected, and in those in which it is infected the organisms are for the most part confined to the areas accessible to the nasopharyngeal secretion. Thus, except for the clothing of some carriers we can, I think, exonerate the clothing.

Very much the same remarks apply to the skin. That of normal individuals may have haemolytic streptococci on it but usually such strains are not members of group A. The same exceptions occur too, in the form of nasopharyngeal carriers whose skin in certain parts of their anatomy may be heavily and

persistently infected. These areas include the hair, the skin of the face and the hands but not that of the trunk and legs which are seldom infected. Thus, in general we can acquit the skin.

Now let us consider the dust and the dirt which may find its way into the wound. Nearly everybody thinks that streptococci are present in these substances. But road dust or dirt, the soils of the countryside or the garden are not infected with haemolytic streptococci. We have examined over 50 specimens and while almost any other microbe may be present, pathogenic strains of haemolytic streptococci are certainly not. Tetanus and gas gangrene are undoubtedly due to contamination of the wound with soil but not the particular organisms with which we are dealing. Here again there are exceptions. This is the dust of hospitals in which infected cases are being nursed. This may be heavily infected with pathogenic strains. But not the dust of the road, factory or battlefield.

The only remaining object which finds its way into a wound with any degree of regularity is the air. Exactly as with dust and dirt, the air of the countryside, of factories and well-run hospitals does not contain these organisms. But again there are exceptions. There is now clear evidence that the air of wards in which infected cases are being nursed, scarlet fever and puerperal fever wards in particular, may be and frequently is heavily infected.

Thus the whole of the available evidence suggests that except in rare instances, haemolytic streptococci or pathogenic strains are not present in wounds, even the dirtiest and most contused of them, *at the time of infliction.* If we admit this we must consider what extraneous sources for the organisms are available and the chances that the organisms will reach open wounds.

A fairly extensive survey has been made of recent years of the distribution of pathogenic strains of haemolytic streptococci in nature. As a result of this we know of only two principal reservoirs of these strains, and both of them are in human beings. The first is the nasopharynx of individuals who are either quite well or are suffering from some variety of nasopharyngeal infection, and may I remind you that many of these individuals have the organisms on their clothing and on their hands as well as in their throats. The second possible source is the infected material coming from suppurating wounds, cellulitis, impetigo, otitis media and similar purulent exudates in other patients in the vicinity of the freshly infected wound.

Let us take the first of these, the individual who has the organisms in his nasopharynx. He may, of course, be suffering from a definite infection such as scarlet fever or tonsillitis which everybody knows is due to these organisms. But haemolytic streptococci do not always produce these characteristic clinical entities so that quite a number of mild sore throats or what pass for common colds may be either due to these organisms or to synergism between them and a virus. There is, furthermore, evidence that the organisms may persist for months after such an infection with their owner quite unaware of it. And finally there is evidence that we may become carriers in the absence of any form of infection. However acquired, we now know that on an average, about seven per cent of normal persons going about their normal daily work carry pathogenic strains of haemolytic streptococci in their nasopharynx. In the majority, the organisms are present on the tonsils and not in the nose, but in some they are in the nose as well. I am inclined to think that the latter are the more dangerous to other people. In passing, I may mention that

another ten per cent of the population have haemolytic streptococci in the nasopharynx which are probably not pathogenic.

It may be legitimately asked how these persons coming into contact with an open wound can cause infection in it. Direct experiment has shown that such a carrier, bending over and talking vertically downwards over a blood agar plate can project the organisms on to the medium on which they appear as colonies when it is incubated. Not every carrier can do it, or always do it. Nevertheless a carrier who, unmasked, bends over an open wound and talks—and how seldom do those who attend to wounds remain silent—is evidently running a very great risk of infecting that wound.

But it is not only the breath of the carrier which may directly transmit infection to an open wound, for I have already mentioned that the skin of the face, the hair, the clothing and the hands, particularly the hands, of such a person may be heavily contaminated with pathogenic haemolytic streptococci. If, therefore, he does not prepare his hands with care, or having prepared them, contaminates them by touching himself, he may very easily infect the wound while dressing it.

Now there is every reason to believe that the nasopharyngeal carrier is one of the principal sources from which haemolytic streptococcal infections of all sorts are derived. He has certainly been shown to play an important part in the spread of nasopharyngeal infections such as scarlet fever and tonsillitis, and to be frequently the source of the organisms in puerperal infections. Thus, of a total of 110 puerperal cases studied in four investigations, the source of the infection was found in no less than 65 of them, to be the nasopharynx of one or other of the attendants at the time of delivery. I have little doubt that a similar study of wound infections would yield very similar results and for this reason I think we must assume that the nasopharynx of a carrier is one of the most important reservoirs from which the organisms are derived.

Let us consider the other reservoir of infection. This, strange as it may appear, is the hospital or home to which the wounded man is sent; or rather, the infected cases already there. These may be discharging purulent wounds, abcesses, cellulitis, whitlows and so on. There may also be cases of otitis media, of puerperal infection or that of ulcerating cancer, all of which may be discharging haemolytic streptococci. Any or all of these may be present in the ward or a neighbouring ward, and sometimes in close proximity to the wounded man. It may well be asked how in a well run hospital, an infection of this type could cause infection in an open wound in another person. There is unfortunately a great deal of evidence that this is perfectly possible. It has, for instance, been recently shown that puerperal infections and those of wounds and burns may pollute the atmosphere about them so that haemolytic streptococci may be isolated from the air. And this in a new, modern hospital embodying all the latest architectural improvements. There is also experimental evidence. It can, for instance, be shown that so long as the infected pus or serous exudate on the dressings of such a case are wet, no dissemination of the organisms into the surrounding atmosphere is likely. If they become dry and remain perfectly still, there is likewise no atmospheric contamination. But let the dried material be agitated in any way, mere folding of a piece of lint infected with a dried serous exudate is sufficient, and large numbers of organisms are immediately released into the atmosphere. Even in an almost

imperceptible breeze these particles can be shown to travel at least three metres. In a gale they probably travel far and wide.

This is a danger of which surgeons and nurses seem to be quite ignorant. They will cheerfully nurse infected cases in the same ward as clean, and the astonishing thing is that they usually get away with it. But they do not invariably do so and particularly in the overcrowding and scurry of war-time. For it was shown in the war of 1914-18 that at the base hospital in Etaples in which were collected all the cases of compound fracture of the femur, more than 90 per cent of the wounds were infected with these organisms.

There is also another danger in hospital or home which is far too seldom recognized. In winter it is quite usual for patients in surgical wards to suffer from nasopharyngeal infections. These may be apparently quite trivial but some of them may be connected with haemolytic streptococci. Their presence in a ward along with clean cases is, however, asking for trouble, for these nasopharyngeal infections may also infect the atmosphere about them and so cause infection of wounds in neighbouring beds.

To recapitulate: The organisms present in wounds sustained by carriers may very well have come either from the individual's own throat, his skin or his clothing. But this is certainly not the source of the organisms in most wounds. We must therefore recognize that the vast majority of haemolytic streptococcal infection of wounds are not due to the injection of the organisms into the raw area at the time of infliction. Infection comes sometime later and from two main sources: (1) from nasopharyngeal carriers who attend to the wound and (2) from other infections in the neighbourhood.

Control Measures.

What then can we do? Let us deal with the nasopharyngeal carrier first. Recognition that he may be the source of the organisms shows that anyone who attends a recently inflicted wound, even he who administers first aid on the battlefield or at the roadside, should remember all the time that he may involuntarily be the cause of infection. Ideally, he should wear a mask but of almost equal value is a very simple measure that no one seems to have thought of before. This is merely that he should refrain from speaking (and of course coughing or sneezing) while near the patient or the wound. We do not expel the organisms while merely breathing quietly, it is while speaking, coughing or sneezing that we do so. But if the attendant has any form of nasopharyngeal infection, no matter how trivial, he should, in my opinion, refrain from contact with open tissue until haemolytic streptococci of group A have been proved bacteriologically to be absent from his throat. If this is impossible, and I am quite aware that it often is he should at least take special pains to eliminate the chances of transfer in the way I have already described. A second point in this connection is that he should take particular care over the toilet of his hands. More often than not the washing process is carried out perfunctorily. This is not enough and the correct toilet of the hands is a study in itself. But I would suggest that there should be a much more widespread use of gloves. They are cheap enough and if sterilization of them is a problem, they can be quite adequately sterilized by putting them on, washing them in soap and running water, wiping them over with an antiseptic of adequate strength and leaving it time to act before touching the wound. Lastly, may I point out that in view of the possibility that the clothing may be infected, the operator should be very careful to avoid touching his clothes.

We now come to the prevention of infection in hospital. I think we ought now to lay it down quite definitely that it is a criminal proceeding to nurse an infected case of any kind in the same ward as open clean wounds. I have myself encountered instances in which suppurating wounds were being nursed cheek by jowl with patients who had just had clean operations such as herniotomies. But in addition, every case of nasopharyngeal infection in a surgical ward should be immediately removed, isolated and examined bacteriologically and only when pathogenic strains of haemolytic streptococci are shown to be absent should it be returned to the ward. I know that all this may seriously disrupt hospital routine and will undoubtedly be very unpopular with matrons and superintendents, but in my opinion it is the only safe way.

Thus if we wish to keep wounds free from infection, we must be eternally vigilant to prevent the ingress of organisms over the whole of the period the wound is open. And now that we know the principal sources of the organisms there is no particular reason why the appropriate measures should not be taken.

Treatment of the Wound.

I have said very little about the treatment of the wound itself, principally because this is a surgical problem. There is, however, no doubt that it should receive a thorough surgical toilet as soon as possible after infliction. This should include cleansing, excision, debridement and the like. I am very doubtful whether antiseptics are of value in the treatment of any wounds. Secondly, the wound should be immobilised as far as possible by the use of plaster or suitable splinting. And thirdly, it should be dressed in such a way that the dressings need not be disturbed for long periods. All these measures will tend to diminish the risk of the ingress of haemolytic streptococci and give the tissues the best possible conditions for dealing with the organisms which may be present in the wound. This technique was, in principle, employed by Trueta in the Spanish Civil War and he certainly had very little sepsis.

Of the more specific methods for the prevention of sepsis, I will only refer to sulphanilamide and similar drugs given by mouth as soon after the infliction of the wound as possible. This has not been definitely shown to be successful in human beings but there is experimental evidence in its favour. Every casualty in the British Army is now given prophylactic doses of sulphanilamide for a period of 48 hours after wounding. The total dosage during that period amounts to 17 gm. The results of this measure are not yet available but it is highly probable that they will be encouraging.

In conclusion, may I ask you to reconsider the idea so firmly held by many of us that sepsis in war wounds and in civil injuries is inevitable. Too often we think that because a wound is ragged, dirty and full of foreign material, haemolytic streptococci are already present and that little or nothing can be done to prevent their development. If we would only recognize that these organisms, unlike all the others which commonly infect wounds, are almost certainly implanted afterwards, and possibly while the wound is being dressed, we might succeed in preventing a great deal of sepsis. And if I have persuaded some of you to think in this way, I shall be well content.

For a full bibliography on this subject the reader is referred to a previous paper by the author which appeared in the *Lancet*, 1940, vol. 1, p. 109.

The Medical Congress of Three Rivers

My dear Dr. Schwartz:

At your request, I am sending you my impressions of the Medical Congress of Three Rivers which took place on the 9th, 10th, 11th and 12th of September. Much could be said on a Congress of this kind, but in a short paper such as this, we can only cast a glimpse on the most important aspects of the problems which were studied there. Tuberculosis was a subject of intense study throughout the week; cancer also, particularly that of the lower bowel. Acute abdominal conditions; immunization against infective disease; dagenan therapy; the use of sulphanilamide, were themes of interesting discussions upon several occasions. Epilepsy came to the fore, and a paper presented by Dr. C. A. Gauthier upon the administration of Dilantin in Epilepsy was most practical. In short the papers were exceedingly well prepared and written in a clear, simple style.

The Congress was officially opened on the 9th of September under the Presidency of Dr. C. N. Deblois of Three Rivers. The meeting was public and the auditorium of the "Academie de la Salle" was taxed to its utmost capacity. A thousand persons were present there. The Honorable Henri Groulx, the Minister of Health in the Quebec Cabinet, spoke very eloquently on health matters, stressing the need of health laws and also congratulating the directors of the Congress for having assumed the tremendous task of assembling such a gathering of distinguished men who were here to throw more light upon problems of Health, so essential not to the individual only, but to the whole community at large. "Politicians" said he, "were in the past, somewhat slow to seek medical advice, but now public opinion is wide awake in this matter, and is very conscious of the fact that Public Health is the foundation upon which lies the happiness and the power of state." "Have the best of Kingdoms," said the speaker in the language of Mr. Disraeli, "give them intelligent citizens, prosperous manufacturers, progressive agriculture. If the population remains stationary, if each year it weakens in stature and vigor, the nation will perish and that is why I maintain that the fostering of public Health legislation is the first duty of a statesman."

The lecturer of the evening was one whose name is famous throughout French-Canadian literature: Dr. Leo Pariseau, who gave us a magnificent illustrated lecture on the "History of Blood Transfusion". The author dwelt at some length upon the history of the Circulation of the Blood by Wm. Harvey, followed up the works of the men who studied the circulation throughout the eighteenth and nineteenth centuries up to the present day, and finally entered upon the actual phase of the problem as it is practised to-day in our hospitals. The speaker's grasp of the subject was most fascinating and full of pathos, as he himself had been the victim of a condition which necessitated blood transfusion, the only procedure which could bring him back to life.

We were particularly struck with Dr. Vallin's presentation of his symposium of post operative complications happening in abdominal operations. Thirty-eight years of experience as a surgeon had taught him more and more that such complications would arise, no matter how perfect the technique,

and that we should always be prepared to meet them. The choice of anaesthesia was of great importance; also the *nature* of operation done and the *state* of the *patient.* "Never should we operate", said he, "before we know that the patient is in a fit state to be operated upon." Sedatives were to be used with caution. Acidosis was a complication always to be dealt with in a very cautious way; ileus also. Localized and general peritonitis as usual, are still the worries of the surgeon and are still the conditions which demand the most care and the most attention.

May we mention the very instructive lectures we had from Dr. Bellerose and Bourgeois on the cancer of the intestines? and the most vivid illustrations and statistics they gave us on the subject? "Cancer of the intestines *early recognized*", said the two speakers, "is a problem of surgery with a favorable prognosis." There is no question about that. Dr. Bellerose dealt particularly with the symptoms and the diagnosis of the condition, and also indicated the surgical treatment in a very precise way. Dr. Bourgeois could not emphasize too much the importance of early diagnosis, and he made the statement that the lowering of the mortality of cancer of the intestines rests with the general practitioner, that is, with the *one* who sees the patient first. "Most patients", said he, "come to us *too late.*"

We could go on indefinitely with our comments, but they are all the same and we must not be too tiresome. On the whole, the Congress was a success. It attracted men not only from Canada and the United States, but also from the far off republic of Haiti, down south. Dr. L. P. Mars, was a very distinguished representative from Port-au-Prince, Haiti, and conquered the hearts of all by his magnificent paper on "Culture and Psychiatry".

J. Emile LeBlanc,

West Pubnico, N. S.

CASE REPORTS

Internal Haemorrhage Simulating Concealed Accidental Haemorrhage

Mrs. B. S. Age 22. Para IV. 8½ months pregnant. Admitted to General Hospital, September 13th, 1940, with the following history. At about 6 p.m. seized with sudden severe pain in lower abdomen; no show. Constipated for 3 days. Examination showed uterus to be very hard and tender. Pulse rapid. Patient was apparently in great pain. Upon admission to hospital at 8.40 p.m. patient was very pale and pulse very rapid.

I was called by Dr. J. A. Macdonald about an hour later who gave me the above history and added that there was a great change in her condition since he had seen her and that she was rapidly becoming moribund. When seen she was perspiring profusely and was exsanguinated. She appeared to be in very severe, continuous pain and complained of extreme thirst. There was no external bleeding. The whole abdomen was quite tender and abdominal muscles on guard. Breathing was quite shallow and distressed. Pulse soft, rapid, no volume. Uterus hard but not board like; cervix was not dilated; no fetal movements and heart sounds could not be heard. A diagnosis of concealed accidental haemorrhage was made. In consultation with Dr. J. A. Macdonald we decided, in spite of the fact that we felt the child was dead, that caesarean section offered the only hope[1], for the following reasons:—

1. Cervix was not dilated or thinned out. Labor had not started.
2. The gravity of her general condition.
3. She was still bleeding, as shown by the progressive increase in the severity of her symptoms.
4. If bleeding was not stopped in a very short time, our patient would soon be dead[2]. Bleeding could not be stopped by conservative measures[3] [4].

We are well aware that caesarean section is not always the treatment of choice for concealed accidental haemorrhage but we believe it has a place in selected cases. It is perfectly obvious to us that any other treatment than that carried out would have proved disastrous in this case. Operation was undertaken 6½ hours after onset of symptoms. Abdomen opened to left of midline. There was a thin blood clot covering lower half of uterus. This was removed. Uterus appeared normal. There was no characteristic ecchymosis of interstitial haemorrhage as seen in severe cases of this kind. The abdominal cavity was full of blood, the amount being massive. It was not measured as the suction became clogged as usual when needed most. No rupture of uterus was apparent. As it was impossible to find the source of bleeding the uterus was opened and a still-born child delivered. Placenta was normal. The uterus was closed rapidly. The abdominal incision was enlarged and the liver and spleen found normal as were also the mesenteric vessels. Uterus was lifted out of abdominal cavity. On its posterior surface to the right of midline at about the level of the internal os was found a large plexus of blood vessels[5]. One of these which had ruptured was pumping blood. Five or six deep interrupted sutures were taken, all around this area, which

controlled the bleeding. Blood clots were removed from the posterior cul de sac but no attempt was made to make abdominal cavity dry. The abdominal wall was closed in layers without drainage. She was given glucose and saline intravenously during the latter part of the operation and 550 c.c. of citrated blood immediately after. It does not appear logical to inject blood into such patients while the bleeding point has not been closed or is about to be closed[6]. She was much improved following her return to bed and had the expected elevation of temperature the following two days. On the fifth postoperative day temperature rose to near 100° but gradually subsided. On September 16th., she was given one c.c. of neoprontosil every four hours for three doses and sulphanilamide grains 10 every four hours for seventeen doses. She left the hospital on the eleventh postoperative day in a satisfactory condition.

This case appeared to be of interest for the following reasons:—

First:— The diagnosis of concealed accidental haemorrhage appeared obvious.

Second:—The site of the haemorrhage was definitely on the uterus and not in the broad ligament[7 8].

Third:— In any text at our disposal we have been unable to find any reference to such a varicosity rupturing although Delee[9] remarks that varicosities in the broad ligament have been known to rupture during labor and cause death. Perhaps we are making a fine distinction regarding the site as the vessels are part of the same plexus.

Fourth:—we have been unable to obtain any history of a fight, blow, fall or other accident.

Fifth:— The necessity of emptying the uterus before the bleeding source could be found.

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ERIC W. MACDONALD, M.D.,
Reserve Mines, N. S.

Rupture of Uterus

Mrs. A. W. Age 24. Para IV. Admitted to St. Joseph's Hospital Nov. 9th-39. The previous history is important in that she had a classical caesarean section, for her first delivery, being in labor thirty-six hours, progress arrested, L. O. P. presentation. The second and third child were born normally. Admitted in labor, at term, child L. O. A. Pains became irregular, was not making any progress so was given sedatives. Nov. 10th X-rayed as not making any progress. Dr. Corbett's report was:—"A. P. and lateral films of pelvis show a full term fetus with occiput presentation, head engaged and wedged transversely between extended arms, just below plane of inlet. There is overlapping of the fetal cranial bones, indicative of fetal death. Measurements show sufficient room for head to pass through, however position of limbs complicate matters." At 3.45 p.m. she was delivered instrumentally of a still born child. Her attending physician was unable to expel placenta. Late that evening, Nov. 10th., I was asked to see patient. She was not bleeding, her general condition seemed fair but she complained of feeling very sore and begged to be left alone. No examination was undertaken and her physician was advised to allow her to rest if not bleeding until morning, when if the placenta was not expressed, we would remove it. In the night she complained of weakness but rested well and her condition caused no alarm. Nov. 11th, patient was prepared and anaesthetized. On introducing hand in uterus a rent about 3½ inches long was found. The placenta was not in the uterine cavity. The cord was drawn down, cut off and vagina packed with gauze strip. She was immediately prepared for laparotomy, which was undertaken at noon, twenty hours after the delivery of child. Abdomen was opened through a right rectus incision and considerable blood removed by suction. There were dense adhesions between uterus, omentum and abdominal wall. Lower part of uterus was densely adherent to bladder and abdominal wall. Rent about 3½ inches long was found in uterus towards the left side, plugged with omentum. The edges of rent were rounded and appeared smooth. Rupture evidently had taken place through a thin scar which was not very vascular. Due to the adherent condition of lower segment a hysterectomy appeared too time consuming. About ⅜ of an inch of uterine tissue was removed all around rent. After packing uterine cavity and cervix with iodoform gauze the uterine wall was closed by through and through sutures of No. 2 Chromic as well as by a more superficial continuous suture. Placenta and membranes were found under the liver. Peritoneal cavity was sucked out and abdominal wall closed without drainage. She was given 10 c.c. of prontosil intramuscularly and 1000 c.c. 5% glucose in saline intravenously. Nov. 12th, iodoform gauze packing removed and patient placed in Fowler's position. She went along making an uneventful recovery until Nov. 15th, when she started to cough and expectorate. Her temperature rising from normal to 102.6, she was given prontosil 5 c.c. and expectorants. Temperature gradually fell, sutures were removed Nov. 19th, and she was discharged on Nov. 23rd, twelve days following her laparotomy.

Close questioning of the patient and her physician failed to establish the time when the rupture took place. She had no pituitrin. It may have taken place when forceps were applied or as the child's head was delivered. Rupture of the uterus rarely is symptomless[1]. The symptoms of rupture sometimes make their appearance almost simultaneously with delivery. The fact that the uterus being emptied can contract down upon itself to some extent is a

favourable feature of this type of case[2]. When labor was so prolonged and/or X-ray showed malposition, section should have been considered. Titus says, "Quiet, spontaneous rupture of uterus during pregnancy is best treated by laparotomy and supravaginal hysterectomy. If the edges of this fundal tear are fairly clean as they are when it occurs at the thin wall site of a former caesarean section, there is some justification for trimming the edges of the wound and repairing the tear". Women who have had previous sections should not be allowed to carry on if there is any suggestion of malposition, disproportion or if continuous fairly rapid progress is not being made. Certainly continuous bearing down pains with arrested progress calls for action. Whenever the scar shows definite signs of weakness, the pregnancy should be terminated by a second caesarean section. If the previous operation was complicated by an infection then section should be done before the onset of labor.

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ERIC W. MACDONALD, M.D.,
Reserve Mines, N. S.

Pernicious Vomiting of Pregnancy

Female patient, 28 years of age, complaining of vomiting and weakness. One child 3 years previously. Very ill at that time for 2 months with hyperemesis. Last period 8 weeks previously. Nine days before seeing patient she had begun to feel nauseated and to vomit in the morning. No associated pain. Gradually the symptoms became worse and vomiting continued practically all the time. Patient became weak and nervous.

Examination: Patient had the appearance of having lost weight and was very pale. No evidence of gastric lesion or associated disturbance. Fundus was at height of 6 weeks pregnancy. B.P. 120/80. Urine negative. Pulse 80. Diagnosis of hyperemesis made.

Treatment: Patient was confined to bed and put on dry carbohydrate diet (toast, crackers, popcorn, etc.,); ginger ale, carbonated waters, glucose D tablets. Luminal for sedation. One drachm dilute HCL t.i.d. Theelin in oil 2000 units q. 2 days. For three days improvement noted with very slight vomiting. Then vomiting began again and persisted in spite of home treatment. B.P. 115/80. Urine normal. Pulse 85. Taken to hospital and all foods and fluids by mouth prohibited. Proctoclysis with bromides. Intravenous saline with 10% glucose 1000 cc's t.i.d. Under this intensive regimen there was slight improvement for 2 days but condition again began to show a downward trend. Practically continuous vomiting. Pulse 95. T.100. B.P. 105/70. Traces of diacetic acid and acetone in urine. No jaundice. Patient was kept on intensive regimen 5 days in all but ominous signs were showing. After due consultation therapeutic abortion was considered the life saving measure. This was duly performed and patient's recovery was immediate.

The above report serves to illustrate just how a case of pernicious vomiting may continue to be serious even though intensive measures are carried out

medicinally and dietetically. The watchword is not to wait too long to terminate pregnancy; as if the condition becomes too serious even terminating pregnancy will not prevent a disaster. The majority of serious cases fortunately respond to intensive treatment without therapeutic abortion.

This point was brought out very nicely in the following case which may have been very trying with less intensive measures.

Female patient, 20 years of age, complaining of vomiting. Primipara. Last period 6 weeks previously. One week before she had begun to feel nauseated and to vomit. Could not retain any food. Patient appeared to be very ill. No evidence of gastric lesion. Fundus size of 4 weeks pregnancy. B.P. 100/70. Pulse 90. T. 99. Patient was put to bed on dry carbohydrate diet, ginger ale, etc. Glucose D tablets. Sedation. Kept on this regimen for 3 days with no improvement. Was becoming dehydrated. Hospitalized. Oral fluids and foods prohibited. Intensive intravenous saline with glucose. Proctoclysis. Immediate improvement. Was gradually put on normal diet with no recurrence of symptoms.

RAYFIELD G. A. WOOD, M.D.,
Lunenburg, N. S.

Haemolytic or Acholuric Jaundice

On May 8, 1940, this patient, a white male, aged 29 years was admitted to the hospital, complaining of pain in the lower left thoracic region, and a feeling of general weakness.

Personal History: Negative regarding measles, diphtheria, scarlet and rheumatic fevers. Patient had influenza in 1918 epidemic but attack was not severe. There is a history of gonococcal infection ten years ago, with apparently good recovery. There is also a history of syphilis of ten years duration —under treatment but still active. (This information was given by the doctor who was treating him, not by the patient himself.) Two months ago, the three lower left molars, which were badly infected, causing swelling of left side of face, and of glands of neck, were extracted under general anaesthetic. The patient was sick for several days following this.

Family History: Is essentially negative. Father living and well but has an acquired deafness. Mother living, aged 60, suffers from heart trouble. Two brothers are living and well, another died one year ago as the result of an accident.

Present Illness: Began four weeks ago. After changing from winter coat to a light jacket, the patient developed a cold in the chest and at the same time he noticed a dull pain in the left upper quadrant, just below the costal margin. He went to a doctor and was treated for "intestinal and muscular flu". He kept at work for the next three weeks, the cold and pain already mentioned being still present. During this time he continued to feel weaker and more tired and finally went to the doctor again on May 3, 1940. He was at once put to bed and ordered to rest as treatment for a touch of pleurisy. By May 5th, he felt a little better, but was perspiring heavily at night. The pain in the side was quite improved. Another doctor was called on May 7th, the first one being away, and the patient was admitted to hospital on May 8th with rapid pulse (98), rapid respiratory rate (26) and a temperature of 101 degrees F.

Physical Examination

General Appearance: is that of a fairly muscular but thin, sallow, and somewhat emaciated male, who appears to be of about stated age.

Head: Eyes—pupils round, equal and reactive to accommodation and light.

Ears—hearing good; meatus clear and clean.

Nose—nostrils clear and clean but there appears to be some obstruction in nasopharynx.

Throat—Tongue moist and appears normal. Gums good. Upper left second molar badly decayed. Tonsils normal, pillars slightly injected.

Chest: Movements and contour symmetrical.

Heart: 1. Apex beat palpable in 5th interspace medial to mid clavicular line.

2. There is a visible diffuse impulse over precordium.

3. There is a mitral systolic thrill with a mitral presystolic murmur. The first mitral is diminished and the second greatly increased. All heart sounds are increased in intensity.

Abdomen: Shape symmetrical, flat, with no scars. Liver, kidneys and spleen not palpable. There is tenderness and marked rigidity in left subcostal region and there is a suggestion of a mass in this area but due to the tenderness and rigidity of the muscles this finding is not absolutely positive.

Neuromuscular: Essentially negative.

Skeleton: No visible or palpable deformities. Average height and light build.

Special Examination

Chest—right side: resonant apex to base. Clear vesicular breath sounds apex to base.

left side: dullness in left base, from level of 6th rib to costal margin. Tactile fremitus absent in this area and vocal resonance impaired. Breath sounds and whispered pectoriloquoy absent.

The radiological report states that there is elevation of the left diaphragm, but that the pathology that causes this is not obvious.

Progress and Medication

May 8th, 1st Day: Admitted per ambulance. Had unnaturally pale color in face and was very weak. Appetite poor; restless all that night. He was given ortal sodium gr. 3 and aspirin compound with codeine gr. 1/8 at once.

Blood picture showed: Haemoglobin, 35%. Leucocytes, 6,700. Lymphocytes, 82. Monocytes, 17. Eosinophiles, 0. Basophiles, 0. Neutrophiles, 1.

May 9th, 2nd Day: Patient very thirsty and drank quantities of liquids. He slept poorly but felt somewhat rested. Urine specimen showed specific gravity 1.020 and acid reaction but no positive findings. Patient still weak and tired but no pain. Transfusion (500 c.c.) at 11.30 a.m. Later appetite improved and he felt stronger and looked better. He was comfortable but had a pressing feeling in left thoracic region, following the taking of food, which was relieved by hot drink. He was also coughing lightly and expectorating small amounts of sputum.

May 10th, 3rd Day: Complains of dryness of mouth and throat. Coughing more frequently but feels much stronger. Expectorating yellowish phlegm. Boric solution given as mouth wash. Liver extract: 2 c.c. intramuscularly. Blaud pills: no. 2 t.i.d. pc.

Blood picture: Haemoglobin, 39%. Leucocytes, 3,800. Lymphocytes, 90. Monocytes, 8. Neutrophiles, 2.

May 11th, 4th Day: Appeared weaker, still coughing and expectorating and complained of soreness and stiffness in right arm.

Boric lint compress applied to arm. Ortal sodium and aspirin compound with codeine. Liver extract—2 c.c.—intramuscularly.

Blood picture: Leucocytes, 2,000. Lymphocytes, 84. Monocytes, 14. Neutrophiles, 2. Eosinophiles, 0. Basophiles, 0.

May 12th, 5th Day: Perspiring considerably and complaining of severe pain in arm. Very restless and nervous but not coughing so much.

Ortal sodium and aspirin compound with codeine repeated. Boric lint compresses to right arm.

Blood transfusion (500 c.c.) at 9.30 a.m.
A.P.L. extract (250) intramuscularly.
Liver " (2 c.c.) "
Nuclein " ( ) "

Pain in arm on least movement. At night, stated that left arm was becoming useless.

Blood picture: Haemoglobin, 36%. Leucocytes, 1,900. Lymphocytes, 88. Monocytes, 9. Neutrophiles, 3. R.B.C., 2,100,000.

May 13th, 6th Day: Patient very restless and breathing difficult (Resp. 48). Pulse very weak; perspiring profusely and is very cold. At 3.30 a.m. patient died.

Pathological Report: Post mortem examination (attached).

Ante-mortem Diagnosis—Agranulocytosis.

Post-mortem Diagnosis—Non-familial Hemolytic Jaundice.

J. V. GRAHAM, M.D.

Report of the Autopsy findings performed at the Halifax Infirmary for Drs. J. V. Graham and A. G. MacLeod. Tissue No. 40-3558. P.M. No. 40-29.

General Appearances: White male of approximately the stated age; skin jaundiced; fat has a canary-yellow colour; both arms are oedematous and show some cellulitis.

Pleural Sacs: Nil.

Bronchi: Congested and contain a frothy blood-stained fluid.

Lungs—Right—shows some oedema and congestion (1 piece for section). Left—the lower lobe and lower margin of upper lobe are deeply congested and oedematous and have an almost semi-solid character suggestive of a confluent broncho-pneumonia (3 pieces for section).

Pericardial Sac: Slight excess of serous fluid.

Heart: is moderately enlarged and hypertrophied. There is a healed chronic rheumatic endocarditis of the mitral valve with some stenosis and incompetence. The left ventricle and auricle are slightly dilated and hypertrophied and the right ventricle likewise. The tricuspid valve admits 5 instead of the normal three fingers and shows a terminal incompetence. The right auricle is dilated. The aortic and pulmonary valves are normal. The myocardium shows some fatty degeneration and possibly haemosiderosis. (Pieces for section) also mitral valve for section.

The Bronchial Glands and Ant. Mediastinal Glands are slightly enlarged. (2 for section).

The Peritoneal Sac: Nil.

Spleen—weighs 32 ozs., shows some moderately recent perisplenitis and one section has a dark red beef-steak character but is somewhat friable as well. (Pieces for section and for haemosiderin.) Its appearance is not unlike an acholuric or haemolytic jaundice.

Liver—is somewhat enlarged and has a brownish cafe au lait appearance. (Pieces for section and haemosiderin.)

Gall-Bladder: Nil.

Stomach and Oesophagus are much distended. The stomach shows some petechial haemorrhages into its mucosa but nil else otherwise nor does the oesophagus show any pathological change.

Pancreas is adherent to the spleen at its tail but is otherwise normal. (Pieces for section.)

Adrenals: Nil. (Pieces for section.)

Lymph Nodes along pancreas and retro-peritoneally are slightly enlarged and haemorrhagic especially in the latter situation. (Pieces for section.)

Kidneys are pale and have a brownish yellow appearance. One shows some congestion. (Pieces for section.)

Urinary Bladder: Nil.

Prostate: Nil.

Intestines: Nil.

Bone Marrow (right femur) has a reddish colour and shows a marked erythroblastic reaction. (Piece for section) but no gelatinous degeneration indicative of an aplastic anaemia.

Histological Examination

Lungs: left—The alveoli are filled with red blood cells, some heart-failure cells and oedematous material. There is no true bronchopneumonia. The appearances have the characters of a chronic venous congestion, oedema, hypostatic pneumonia with a diffuse haemorrhage.
right—shows a chronic venous congestion and oedema.

Myocardium—shows cloudy swelling and fatty degeneration, but no haemosiderosis. No accumulations of white cells indicative of leukaemia are seen.

Mitral Valve shows a marked fibrosis and some lymphocytic inflammatory change. No Aschoff Bodies are visible. The condition is that of a chronic quiescent rheumatic endocarditis with stenosis.

Bronchial Lymph Glands show some anthracotic pigmentation, hyper-

plasia of the lymphoid follicles and desquamation of the cells in the sinuses. This appears to be more of the nature of an inflammatory hyperplasia.

Ant. Mediastinal Lymph Nodes show a diffuse lymphoid hyperplasia. In some of the blood vessels numerous white cells of lymphocytic character are seen.

Pancreas—At the tail is a simple chronic diffuse pancreatitis with clusters of lymphocytes and fibrosis. Elsewhere it is quite normal.

Adrenals—show no evidence of any pathological change.

Kidneys—show cloudy swelling, fatty degeneration but no haemosiderosis. There are some small white cells accumulations scattered in the interstitial tissues and a little congestion of the vessels.

Liver—shows round cell accumulations in the portal tract areas throughout the liver, smaller ones in the central vein areas, some fatty degeneration and slight haemosiderosis at the periphery of the liver lobules. These round cells have the character of normoblasts and megaloblasts and are evidently a reversion to the foetal haemopoietic function.

Spleen—shows a marked diffuse congestion with numerous haemorrhages into the splenic pulp, a little diffuse fibrosis and marked phagocytic activity and proliferation of the reticulo-endothelial cells which contain haemosiderin. There is some chronic perisplenitis. The Malpighian bodies are rather atrophic and there are some cells of the granular series diffusely scattered throughout but there is no marked lymphoid hyperplasia such as is seen in the usual case of lymphatic leukaemia, no diffuse fibrosis of a splenic anaemia and no evidence of a true neoplastic or Hodgkin's Disease.

Lymph Nodes (retroperitoneal and from along pancreas)—show congested, some diffuse lymphoid hyperplasia, and proliferation and desquamation of some of the cells lining the sinuses.

Bone Marrow shows marked proliferation of the normoblasts, a moderate hyperplasia of the megoblasts and megakaryocytes which show some phagocytosis. There are practically no myelocytes or myeloblasts seen and nothing to indicate a leucoblastic reaction. The change is thus a normoblastic or erythroblastic one.

Remarks: The appearances of the bone marrow exclude an aplastic anaemia; the appearance of the spleen excludes Hodgkin's Disease, splenic anaemia and a primary neoplasm in that organ; the appearances of the lymph nodes are positively not those of a lymphosarcoma or reticuloma. The lack of leucoblastic response of the bone marrow would exclude a myelogenous leukaemia even of acute or a leukaemic type and also a monocytic leukaemia. The relative absence of any source of the lymphocytes for an ordinary lymphatic leukaemia points to the absence of that condition, and failure to find any true lymphoid deposits in the organs general and proliferation of the lymphoid follicles in the spleen is against an acute lymphatic leukaemia of a leukaemia type. (All such cases recorded have given at least a moderate terminal rise in the total white cell counts.)

Although I can find no references to the appearances found at post-mortem in the spleen in agranulocytosis and although I have not performed a post-mortem on such cases I feel that the appearance of the spleen here with a reversion to the foetal haemopoietic function of the liver and the normoblastic reaction of the bone-marrow and haemosiderosis of liver and spleen

exclude a true agranulocytosis. The clinical history, too, except for the progressive leucopenia and relative agranulocytosis is by no means characteristic. The infection of the lip does not appear severe enough for the agranulocytic Angina type, the absence of a true pneumonia is against the second variety and lastly he does not appear to have been given enough prontylin, sulphanilamide or barbiturate drugs to bring him into the third category of agranulocytosis.

As a result of this process of exclusion and the positive findings in the spleen with the accumulation of red cells in the splenic pulp both inside and outside the sinuses, the marked reticulo-endothelial proliferation and phagocytosis, the haemosiderosis in the liver and spleen, the reversion to the foetal haemopoietic function in the liver, the normoblastic reaction in the bone marrow, the failure of hyperplastic change in the Malpighian bodies of the spleen, the absence of a marked lymphoid hyperplasia in the lymph nodes, and lymphoid or granular cell accumulations in the organs with the marked and profound hypochromic microcytic anaemia, leucopenia, with high relative lymphocytosis and mononucleosis, all points to the condition of a haemolytic or acholuric jaundice possibly of the acquired type which is not benefitted by splenectomy.

Complicating the picture is the chronic mitral rheumatic endocarditis with stenosis and chronic venous congestion, terminal hypostatic pneumonia and oedema of the lungs, and infection of the lip and cellulitis of the arms. The latter, in his condition, I feel, would further depress the activity of the cells of the granular series. Syphilis is not a factor in the cause of death, no evidence of it being found in any of the organs or aorta.

Pathological Diagnosis

Haemolytic or acholuric jaundice (probably of the acquired type) associated with a profound hypochromic anaemia, leucopenia, with a marked relative lymphocytosis and mononucleosis; mitral stenosis and incompetence due to old rheumatic endocarditis with chronic venous congestion; terminal hypostatic pneumonia; cellulitis and Vincent's Angina.

Signed, RALPH SMITH, M.D.,D.P.H.,
Provincial Pathologist.

Empyema followed by Cerebral Abscesses

I. M. R. a 24 year old seaman was admitted to the Halifax Infirmary on March 25, 1940, complaining of cough, pain in the left chest anteriorly and difficulty in breathing. A week previously he had developed a bad cough with blood-streaked sputum, and collapsed while at work a few days later. His medical history was negative.

He was a cyanosed, dyspnoeic well-nourished individual with a temperature of 102.4, a pulse of 104 and with a respiratory rate of 32 per minute. Respiratory movements were diminished on the left side of the chest, which yielded a flat note on percussion. The breath sounds over this area were diminished, but of a broncho-vesicular nature.

The laboratory reported a white cell count of 32,000 with 26 band cells

and 89% neutrophiles. Numerous pneumococci and streptococci were present in the sputum and a trace of albumin was found in the urine.

The patient was placed on sulphapyridine theraphy, thirty grains immediately then grains fifteen every four hours, along with adequate sedation.

Tympanites developed and pain and distress continued. By April 2, when Dagenan was discontinued, the temperature still remained at admittance level. X-ray confirmed the clinical findings of pleural effusion with displacement of the heart to the right. The chest was aspirated and sixteen ounces of purulent fluid, containing many streptococci, obtained. Some measure of relief followed but to obtain adequate drainage resection of the ninth rib in the left midaxillary line was performed under local anaesthesia.

The empyema drained well, appetite and comfort improved, and on April 20th, the radiologist reported diminished fluid with pleural thickening and some air over the upper lobe on the left.

Pain in the lower left chest anteriorly returned and by May 17, a fluctuant mass could be felt under the left subcostal margin anteriorly. This was opened and explored: pus was obtained and a cavity around the tip of the 8th, 9th and 10th costal cartilages was found to communicate with the left pleural sac.

On May 25th, the patient showed improvement of both objective and subjective symptoms, but during June and July the temperature fluctuated between 99 and 103 degrees. The cavities continued to drain and the patient became pale and wasted.

On July 31st X-ray investigation of the left pleural sac with lipiodal revealed a cavity one and a half inches wide lying in the left pleural gutter from the 5th to the 9th ribs. With the object of obliterating this cavity the 7th and 8th ribs were resected at operation on August 10th. Thickened pleura with pale granulation were found, and an hour-glass cavity extending as high as the 6th rib posteriorly. The operation was followed by a course of Prontylin and by August 17th the patient appeared improved, though the temperature remained at 100 degrees.

On September 6th the patient complained of a right frontal headache. This was followed by orbital pain and a convulsive like attack with twitching of the arms. The white cell count was now 20,400 and haemoglobin 70%. Sulphapyridine was administered intramuscularly but neck rigidity appeared within two days. Lumbar puncture revealed a cloudy fluid under pressure with increased protein and cells and decreased chloride.

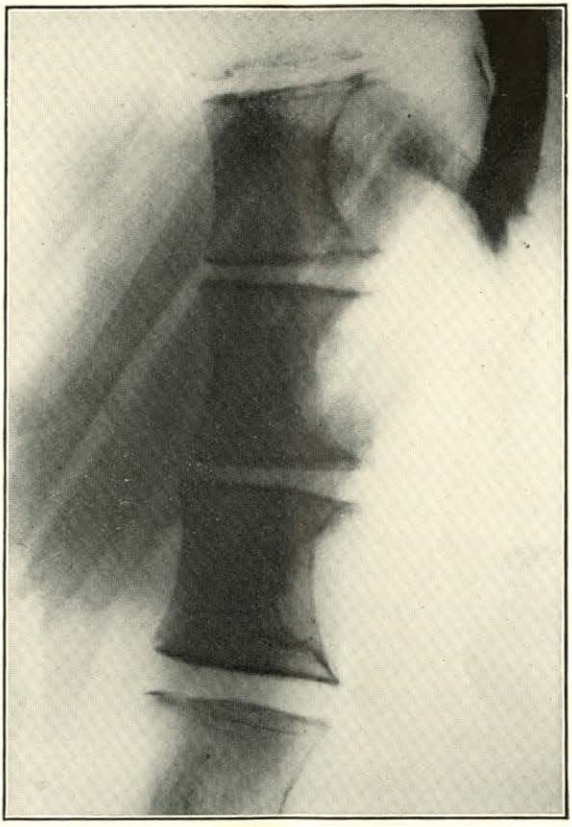
Severe headache and vomiting followed and the patient became weak, rigid and comatose. No abnormality of the eye grounds could be detected.

Spinal tap was repeated on September 14th, the fluid containing many neutrophiles with 400 mg. of protein, 650 mg. of chlorides and diminished sugar. No definite organism could be found.

In spite of intravenous therapy and other emergency measures the patient expired on September 15th, having developed terminally a dilated right pupil and a bilaterally positive Babinski sign.

At post-mortem examination acute cerebral abscesses were found in the left tempero-sphenoidal and right frontal lobes, with a marked purulent basal meningitis. The whole brain was very oedematous and soft. On opening the thorax the left lung was found to be markedly adherent. The cavity outlined by lipiodal was present, though free from pus.

J. W. MacIntosh, M.D.



Showing erosion of the posterior surface of the bodies of the 12th dorsal, 1st and 2nd lumbar vertebrae.

Benign Extra Medullary Spinal Tumour

Miss E. M.—age twenty-one, was referred to me on July 12th, 1940, with the following history.

She had been suffering from low back pain for the past eight years. During this period there had never been complete relief but she had been able to work as domestic at irregular intervals. When attacks of pain were unusually severe, she was confined to bed for two to three weeks. According to her history, menstruation aggravated her complaints. Pain radiated down the posterior surface of the left thigh to the knee and leg. About one year ago she first noticed weakness of the left leg and this has become progressively worse. Dysuria and frequency began five or six months ago. For the past two weeks she has been bed-ridden, suffering constant pain in the back radiating down the left limb, with painful and frequent micturition.

This patient presented a very pathetic picture. Present weight seventy pounds—normal weight one hundred and ten pounds. Extremely emaciated. Large decubitus ulcer over sacrum—complete paralysis of left leg and paresis of right. Severe cystitis, voiding almost free pus.

R. B. C. 2,500,000—W. B. C. 10,250—Hb. 56%.

Kahn reaction negative.

Neurological examination showed a loss of sensation in the left leg corresponding to a lesion at the level of the 12th dorsal segment and complete motor paralysis of the whole limb. There was loss of the knee jerk with an extensor plantar response and paresis of all muscles of the right leg.

X-ray examination showed erosion of regular outline of the posterior surfaces of the 12th dorsal and 1st and 2nd lumbar vertebrae. A diagnosis was made of a spinal cord tumour, and an operation advised. Intravenous glucose and saline were given for two days pre-operatively. On July 15, 1940, under gas, oxygen, ether anaesthesia a laminectomy was performed, the 12th dorsal, 1st and 2nd lumbar spinous processes and laminae were removed. On opening the dura a very soft friable purplish coloured mass filled the spinal canal and apparently confined to the area exposed. The tumour was easily shelled out and all remnants removed as far as possible. Cauda equina was not apparent. Due to the erosion of the vertebrae a rather large cavity resulted. Excessive bleeding was controlled by hot packs and the wound was closed in the usual manner.

Subsequent History.

Following operation the temperature rose to 103, pulse 145, Resp. 40—and condition was rather precarious for three days. Intravenous glucose and saline was administered (2000 c.c.) daily for the first week until she was able to take nourishment by mouth. The temperature, after ten days, dropped to 100 and remained so for the following three weeks due to severe cystitis. The wound healed by first intention in spite of a sacral ulcer. Bladder irrigation and mandelic acid for the cystitis and elastoplast for the ulcer finally cleared up these complications.

Four days after the operation all pain disappeared and her only complaints were due to the uncomfortable positions, lying at first prone and then on either side. Morphine was discontinued and Upnos. 3 drachms, at bed-time substituted.

Bed-side Record.

August 6, 1940—Out of bed in chair. Can lift leg off bed—now voiding voluntarily but requires some pressure over bladder.

September 9, 1940—Walked alone to-day. Reflexes still absent and sensory reactions show little change.

September 21, 1940—Has gained 10 pounds and is able to walk fifty feet alone. No urinary symptoms—sacral ulcer healed—eating ordinary diet and blood picture in Hb. 78%.

Reds 3,360,000; Whites 10,700; Discharged.

Report from Pathological Laboratory, Halifax, N. S.

Tissue—spinal cord tumour. Histological appearance here are those of a very vascular simple tumour arising from the nerve sheaths—the so-called Neurinoma or Schwannoma. There are large areas of haemorrhage in places. I can detect no evidence of malignancy.

(Sgd.) R. P. Smith, M.D.

Extra medullary spinal tumours almost always begin with neuralgic pains. Neuralgia was the only symptom for eight years in a case of Schultze's. Following the neuralgic period progressive motor disturbances developed. An extra-dural position was indicated especially by long-continued Brown—Sequard's paralysis with unilateral root symptoms, followed by unilateral medullary symptoms. Because of the elongated form of many extra-dural tumours, muscular cramps and paralysis are relatively more common than with intradural growths, but owing to the protection of the dura, medullary symptoms may be long delayed (Ewing).

The history of this case extended over a period of about eight years but all the symptoms became progressively worse during the last few weeks. Previous to hospitalization she was treated symptomatically and the underlying cause was not recognized. This is the usual history in all these cases.

L. M. Morton, M.D.,
Yarmouth, N. S.

Issued by the Department of the Public Health November 9, 1940.

It is reported that some of those who have been placed under quarantine in the City of Halifax, as Diphtheria Contacts, have not been maintaining the restrictions imposed. This is no time for dealing leniently with persons who offend in this way. In future those found guilty of breaking the quarantine law should be summarily dealt with and the maximum penalty, provided by Statute, imposed.

When a house is placarded, ingress and egress is forbidden to everyone except the physician in attendance, health authorities and those to whom permission has been given by the health officer. In some instances, and at the discretion of local health authority, the bread winner may be permitted to continue his usual avocation, provided he does not come in contact with the patient or patients. The quarantine period for contacts is (a) In the absence of laboratory control, a minimum period of *seven days from date of last exposure*; (b) If laboratory control is available, until a negative culture has been obtained from the nose and throat respectively. In any event there must be no movement, other than specified, in or out of the premises until the placard is removed by competent authority. The isolation period for the patient is (c) In the absence of laboratory control, until clinical recovery is complete and all discharges from nose, throat and ears have ceased. Minimum period twenty-one days from date of onset. (d) If laboratory control is available, until two successive cultures from site of lesion made at intervals of not less than twelve hours, are negative. Minimum period, ten days from date of onset.

It is realized that quarantine and isolation alone will not bring diphtheria under control. To conquer it, immunization must be practised widely, and especially must the younger age groups be given this protection. There are still many children in the city who have not been toxoided. Parents are urged to send these promptly to their doctors for this treatment. Those who are unable to pay will be given the necessary attention at the Clinic.

Vitamin Advertising and the Mead Johnson Policy

The present spectacle of vitamin advertising running riot in newspapers and magazines and via radio emphasizes the importance of the physician as a controlling agent in the use of vitamin products.

Mead Johnson & Company feel that vitamin therapy, like infant feeding, should be in the hands of the medical profession, and consequently refrain from exploiting vitamins to the public.

Dalhousie Medical and Dental Library

PRACTITIONERS throughout the Maritimes are again invited to make use of the reference service of the library. Borrowers may ask for material on a stated subject, or for special articles, for which they should furnish the name of the publication, number of volume, and date or paging. In asking for material by subject, it is best to be definite in stating it, and to give some idea of the purpose, whether a general review of the subject is desired, or only the most recently published work.

Loans out of the city are for two weeks, and may be extended as required. The postage costs very little, as libraries enjoy a very low rate on books and periodicals which they send out, and this rate includes the return of the parcel if the enclosed label is used, and the same container or wrapping.

Following is a list of some recent accessions to the library shelves:—

MEDICAL SCIENCES:—

Best & Taylor	Physiological basis of medical practice, 2d ed., 1940.
Bodansky & Bodansky	Biochemistry of disease, 1940.
Menkin, V.	Dynamics of inflammation, 1940.
McCollum, E. V.	Newer knowledge of nutrition, 5th ed., 1939.
Solis-Cohen & Githens	Pharmacotherapeutics, 1928.
American Medical Association	Accepted foods.
American Medical Association	Vitamins.

MEDICINE:—

Armstrong, H. G.	Principles and practice of aviation medicine, 1939.
Dunlop, Davidson & McNee	Text book of medical treatment, 1940.
Cecil, R. L.	Text book of medicine, 5th ed., 1940.
Elmer & Rose	Physical diagnosis, 8th ed., 1940.
Ewing, James	Neoplastic diseases, 4th ed., 1940.
Fishberg, A. M.	Hypertension and nephritis, 4th ed., 1940.
Goldberg, Benjamin	Clinical tuberculosis, 2d ed., 2 vols., 1939.
Hill, Leonard & Ellman	Rheumatic diseases, 1938.
Heffron, R.	Pneumonia, 1939.
Houston, Wm. R.	The art of treatment, 1936.
Levine, S. A.	Clinical heart disease, 2d ed., 1940.
Major, R. H.	Physical diagnosis, 2d ed., 1940.
Martini	Principles and practice of physical diagnosis, 1938.
Norris & Landis	Diseases of the chest, 6th ed., 1938.
Savill, A. & Warner, E. C.	Savill's system of clinical medicine, 11th ed., 1939.
Hunter, Donald	Occupational diseases, 1936.
Tuft, Louis	Clinical allergy, 1938.
Wilder, R. M.	Clinical diabetes mellitus, 1940.

SURGERY:—

Bailey & Love	Short practice of surgery, 4th ed., 1938.
Cope, Zachary	Early diagnosis of the acute abdomen, 8th ed., 1940.
Fifield, L. R.	Infections of the hand, 2d ed., 1939.
Scudder, C. L.	Treatment of fractures, 11th ed., 1939.
Vaughan, H. S.	Congenital cleft lip, cleft palate, 1940.
British Medical Journal	War wounds and air raid casualties, 1939.

OBSTETRICS AND GYNAECOLOGY:—

Douglass & Faulkner	Essentials of obstetrical and gynaecological pathology, 1938.
Stone, E. L.	The new-born infant, 2d ed., 1938.

SPECIALTIES:—

Flagg, P. J.	The art of anaesthesia, 6th ed., 1939.
Maxson, L H.	Spinal anaesthesia, 1938.
Duke-Elder, Sir W. S.	Text book of Ophthalmology, 1938.
Guggenheim, L. K.	Otosclerosis, 1935.
Pritchard, Eric	The infant, 1938.
Kugelmass, I. N.	The newer nutrition in paediatric practice, 1940.
Schwartz & Tulipan	Text book of occupational diseases of the skin, 1939.
MacKenna, R. W.	Diseases of the skin, 3d ed.
Cameron, D. E.	Objective and experimental psychiatry, 1935.
Kendig & Richmond	Psychological studies in dementia praecox, 1940.
Henderson & Gillespie	Text book of psychiatry, 5th ed., 1940.

PUBLIC HEALTH:—

American Neurological Association.	Eugenical sterilization, 1936.
Glueck, S., & Glueck, E.	Juvenile delinquents grown up, 1940.
Witmer, H. L.	Psychiatric clinics for children.
Frazer & Stallybrass	Text book of public health, 10th ed., 1940.
Harries & Mitman	Clinical practice in infectious diseases, 1940.
Shrader, J. H.	Food control, 1939.

MISCELLANEOUS:—

Cabot, Hugh	Patient's dilemma, 1940.
Von, Diringshofen, H.	Medical guide for flying personal, 1940.
MacDermot, H. E.	Sir Thomas Roddick, 1938,

CIDER BELT STORIES

The Droll Remark of the Recent Widow

A long, long time ago in the beautiful Annapolis Valley lived a fisherman and his wife. At the time of the episode about to be narrated they were approximately 60 years old. They had no children. From all appearances they did not get along well together. It was a rare year when they spoke to each other more often than twice. If love had ever dwelt in the house, it had long ago departed and banged the door.

One day the fisherman went about his work in company with three other fishermen. In the course of a few hours a fatality occurred. The man who did not love his wife was drowned. His three companions recovered the body and brought it home. One of the men, chosen to break the news to the widow entered the house. The lady was in the kitchen, ironing. After beating about the bush a while, the bearer of bad news finally told what he came to tell.

For half a minute or longer the widow had nothing to say. Then, apparently quite undisturbed by the tragedy, she remarked over her shoulder: "*Put him on the back steps and let him dreen*".

The waters rolled up the Bay of Fundy as they had rolled for centuries, and the murmuring pines and the hemlocks stood like druids of old with whiskers long and pathetic.—From *The Truro News.*

Correspondence

Department of National War Services

Ottawa, Ontario,
October 29th, 1940.

Dear Doctor Routley,

As you must have heard, when the young men called up for military training beginning on the 9th of this month, reached the several military training centres they were again examined by medical officers and, in a number of cases, placed in a different and lower medical category than the young men concerned had been categorized by the civilian examining physicians. I have every reason to believe that the examining physicians, generally speaking, carried out the medical examinations carefully and, except for the hand-writing, turned in good reports, but their categorization of the men did not coincide with the officers in all cases.

Would it be asking too much of you to send out, at your earliest convenience, through your usual channels, a request to all medical practitioners in Canada directing their particular attention to the categorization of the men whom they will examine in the future. A careful perusal of "Physical Standards and Instructions for the Medical Examination of Recruits", copy of which was sent to each examining physician several weeks ago, would assist the examining physicians when they place the man being examined in his medical category.

Our Divisional Registrars are now sending out "Notices—Medical Examination" to young men residing in all parts of Canada; I would very much like to see that the work of the examining physicians meets more closely the views of the military medical officers who will again examine the men when they report to military training centres on the 22nd of next month.

Anything you can do will be very much appreciated.

Sincerely yours,

(Signed) L. R. LaFlech,

Associate Deputy Minister.

Dr. T. C. Routley,
184 College Street,
Toronto, Ontario.

OBITUARY

The BULLETIN regrets to announce the death of Dr. Allan L. McLean, Professor of Epidemiology, Dalhousie Medical School, who died at the Halifax Infirmary on Thursday, October 24th. Dr. McLean took ill with an influenzal infection while attending the meeting of the American Public Health Association at Detroit. When he returned to Halifax, he did not at first seem to be seriously ill. On Wednesday, October 23rd, however, his condition changed for the worse and he passed away suddenly with heart failure on Thursday night, October 24th.

Dr. McLean was born in Toronto, Ontario, in 1896. He began his medical course at the University of Toronto, but transferred to Dalhousie and graduated in 1926. After graduation he went to the United States and took training at the field station of the Rockefeller Foundation which was then at Andalusia and Montgomery, Alabama. He next accepted the position of County Health Officer for Southampton County in the State of Virginia. His work in Virginia soon became known and he was awarded a year's Fellowship at the School of Public Health of Johns Hopkins University. On completion of his studies at Johns Hopkins he was awarded the Certificate of Public Health from that institution. He then returned to the State of Virginia as Health Officer of the County of Henrico, which position he held until 1933 when he came to the Dalhousie Medical School as Professor of Epidemiology. His work in Nova Scotia is known to most of us. He was a sincere teacher deeply interested in research. During the few years he was with us he carried out studies on dysentery in Halifax, a study of tuberculosis in Glace Bay, N. S., a study of tuberculosis at Conception and Trinity Bays, Newfoundland, and at the time of his death he was completing a morbidity and medical service study at Glace Bay and also at Yarmouth, N. S. He will be greatly missed at the University and throughout the Province. He was a pioneer in epidemiology in Nova Scotia.

Personal Interest Notes

DR. J. G. MACDOUGALL, Dr. J. R. Corston, Dr. C. S. Morton, Lieutenant-Commander H. S. Morton, R.C.N., Dr. K. A. MacKenzie and Dr. W. A. Curry, all of Halifax, recently attended the meetings of the Royal College of Surgeons and the Medical Council of Canada held at Ottawa during the last week in October.

The recent epidemic of diphtheria in Halifax has stimulated immunization clinics throughout the Province. So far we have heard of clinics at Dartmouth, carried out under the Health Officer, Dr. H. A. Payzant; at Windsor under the direction of Dr. E. L. Eagles; at Sydney under the direction of Dr. J. K. McLeod; at Pictou under the Divisional Health Officer, Dr. G. G. G. Simms; at Maitland under the direction of Dr. R. G. Wright; also at North Sydney, Florence, Bridgewater, Mahone Bay, Hantsport and at Truro.

Dr. Perley R. Little, who practised for the past five years in Newfoundland, has moved to Truro and has opened up an office on Prince Street.

Dr. and Mrs. T. F. Meahan of Glace Bay were visitors in Halifax during the middle of October.

New Tuberculosis Unit Opened.

On October 15th the new tuberculosis unit which has been built in connection with the Glace Bay General Hospital was opened by the Hon. F. R. Davis, Minister of Health. The unit has accommodation for forty-two patients. The cost of construction was borne by the Provincial Government as part of their programme of tuberculosis control. At the ceremony there were present Angus J. McDonald, President of the Hospital's Board of Directors, Mayor D. W. Morrison of Glace Bay, Claire Gillis, M.P. for Cape Breton South, and Dr. C. J. W. Beckwith, Director of Public Health for Cape Breton Island.

Dr. C. O. Homans, who formerly practised at Hubbards in Halifax County, has been called up to the Royal Canadian Army Medical Service Corps.

Halifax County Home Formally Opened.

The new Halifax County Home and Mental Hospital which has been constructed at Cole Harbour was officially opened on October 23rd by the Honourable F. R. Davis, Minister of Health. Besides Dr. Davis there were present at the ceremony Warden W. J. Dowell, Dr. Murray MacKay, Superintendent of the Nova Scotia Hospital, Mayor W. E. Donovan of Halifax, R. M. Fielding, K.C., County Solicitor, Dr. P. S. Campbell, Chief Health Officer for the Province, and others.

Dr. M. J. Macaulay of Sydney paid a short visit to Halifax during the middle of last month.

Twenty-One Hospitals in Province Approved.

Twenty-one Nova Scotia Hospitals, six of them in Halifax, were included in the 1940 official list of 2,806 approved hospitals in the United States, Canada, and several other countries, which was announced at the 23rd annual Hospital Standardization Conference which opened October 21st in Chicago in connection with the Clinical Congress of the American College of Surgeons.

The six Halifax hospitals listed were the Children's Hospital, Camp Hill Hospital, Grace Maternity Hospital, the Halifax Infirmary, the Halifax Tuberculosis Hospital and the Victoria General Hospital.

Other Nova Scotia Hospitals approved are—

Amherst—Highland View Hospitals; Antigonish—St. Martha's Hospital; Dartmouth—Nova Scotia Hospital; Glace Bay—Glace Bay General Hospital, St. Joseph's Hospital; Kentville—Nova Scotia Sanatorium; New Glasgow—Aberdeen Hospital; New Waterford—New Waterford General Hospital; North Sydney—Hamilton Memorial Hospital; Sydney—City of Sydney Hospital, St. Rita Hospital; Sydney Mines—Harbour View Hospital; Truro—Colchester County Hospital; Wolfville—Eastern Kings Memorial Hospital; Yarmouth—Yarmouth Hospital.

Provisional approval has been given the Highland View Hospital, Halifax City Tuberculosis Hospital, Harbour View Hospital and Eastern Kings Memorial Hospital, because, while accepting the minimum requirements, they have yet to fulfill some details of said requirements. The Victoria General Hospital is also listed as one of those institutions conducting approved cancer clinics.

The number of approved hospitals in 1918 was only eighty-nine. Ten million people—one out of every thirteen persons—were cared for in hospitals in Canada and the United States last year. Three million people were operated upon.

NOTICE

Qualified physicians in Nova Scotia who have not been officially appointed Examiners of Recruits should notify Dr. H. L. Scammell, P. O. Box 1150, Halifax, who will forward their names to Ottawa for approval. Those who desire a further supply of examination forms either in French or English should likewise apply to the above address.

STRAPPED FOR RICKETS

The swaddled infant pictured at right is one of the famous works in terra cotta exquisitely modeled by the fifteenth century Italian sculptor, Andrea della Robbia. In that day infants were bandaged from birth to preserve the symmetry of their bodies, but still the gibbous spine and distorted limbs of severe rickets often made their appearance.

A bambino from the Foundling Hospital, Florence, Italy,—A. della Robbia

SWADDLING was practised down through the centuries, from Biblical times to Glisson's day, in the vain hope that it would prevent the deformities of rickets. Even in sunny Italy swaddling was a prevailing custom, recommended by that early pediatrician, Soranus of Ephesus, who discoursed on "Why the Majority of Roman Children are Distorted."

"This is observed to happen more in the neighborhood of Rome than in other places," he wrote. "If no one oversees the infant's movements, his limbs do in the generality of cases become twisted. . . . Hence, when he first begins to sit he must be propped by swathings of bandages. . . ." Hundreds of years later swaddling was still prevalent in Italy, as attested by the sculptures of the della Robbias and their contemporaries. For infants who were strong Glisson suggested placing "Leaden Shooes" on their feet and suspending them with swaddling bands in mid-air.

How amazed the ancients would have been to know that bones can be helped to grow straight simply by internal administration of a few drops of Oleum Percomorphum. What to them would have been a miracle has become a commonplace of science. Because it can be administered in drop dosage, Oleum Percomorphum is especially suitable for young and premature infants, who are most susceptible to rickets.

Important also to your patients, Oleum Percomorphum is an economical antiricketic.

Oleum Percomorphum offers not less than 60,000 vitamin A units and 8,500 vitamin D (International) units per gram. Supplied in 10 and 50 c.c. bottles, also in boxes of 25 and 100 ten-drop soluble gelatin capsules containing not less than 13,300 vitamin A units and 1,850 vitamin D units.

MEAD JOHNSON & CO. OF CANADA, LTD.
BELLEVILLE, ONTARIO

Please enclose professional card when requesting samples of Mead Johnson products to cooperate in preventing their reaching unauthorized persons.

"What do you make of his name?" asked the chief of police.

"Well," responded the great detective, "from the spelling I should judge he is either a parlour car or a Russian dancer."

The policeman had stopped a motorist who was breaking a rule of the road.

"Now, let's have your name," he said.

"Demetrius Aloysius Hebblethwaite," said the motorist.

"None o' that, now", said the policeman, severely. "It's your name I want, not your family motto!"