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The Surgeon As A Biologist*

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TODAY surgery may be said to be truly international; it has no country of its own, and the American College of Surgeons, with that insight which has always distinguished its founders, has, I consider, done more than any other single body of men to bring all nations and peoples into a common brotherhood for the furtherance of our science and craft and to foster that spirit which works for the benefit of all humanity, and not for any single section. One must hope that the day may yet dawn when, urged by the same lofty principles, the nations of the world will combine to ensure for human beings a stable political world where peace is more or less assured and is no longer a mere interval between successive wars. Science and medicine cannot progress or flourish unless there is security and peace between nations, and the same is true of all useful human enterprises and movements for betterment of the race.

The science of biology is the study of living organisms, the manner in which they arise, the way in which they function, and the methods by which they reproduce themselves. It includes such different subjects as botany, zoology, physiology, pathology, and genetics, and a biologist is one who studies living organisms and strives to discover the truths of life and living things, and in that respect every doctor, who is anything more than a mere craftsman, may legitimately claim to be a biologist, since in his daily work he is constantly called upon to study and observe the most complicated and intricate of all living organisms, the human body.

We may take pride to ourselves that many of the greatest discoveries of the past in the biological field have been made by doctors as the direct result of their observations of the human body in the course of treating patients. I have only to mention Harvey's discovery of the circulation of the blood in the early part of the 17th century, Lister's discovery of the causes of suppuration and the way in which sepsis in wounds can be prevented, Jenner's observation that milk-maids did not contract small-pox and his deductions from those facts which led to the discovery of vaccinia, John Hunter and his intensive search for the fundamental causes of disease and injury, Cushing and Horsley's observations of the functioning of the human brain, and Banting's discovery of the cause of diabetes.

It may be said that every operation a surgeon performs is a biological experiment and affords, or may afford, an opportunity for the discovery of some new biological truth. It is, I think, often not realized by the pure laboratory research workers that unrivalled opportunities are afforded to the surgeon for direct observation of living human tissues, both in health and disease, and for the study of the manner in which such tissues react to trauma and other forms of damage.

The surgeon habitually, as part of his daily work, performs vivisection upon the human body, and in that respect has an advantage over the pure

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research worker, who is necessarily confined to the vivisection of animals. It is true the surgeon cannot experiment in the strict sense, but he has great opportunities of observation and a great deal of our knowledge of living processes is founded upon the accumulated observations of surgeons while performing operations upon living subjects.

We are accustomed to distinguish between pure research and clinical research, but there is no hard and fast line to be drawn between them and the clinician, provided he has the true scientific mind, is as much a pure research worker as he who works in a laboratory. The one is indispensable to the other and the best results are obtained when the clinician and the laboratory investigator are working in close collaboration.

There is, it seems to me, a very real danger in these days that medicine may be too much divided into separate compartments. We have the physician, the surgeon, the pathologist, the pure research worker, and the biochemist, all doing their own work and often hardly aware of what is happening in each others' departments. This division of work into a number of different sections is necessary in these days, if we are to make rapid progress, and there can be no doubt is largely contributing to make medicine a true science, but unless close touch is maintained between the different departments the individuals tend to become over specialized and too much like the machine minders of the mass production factory. The surgeon and the physician should do work in the laboratory and the pathologist, the physiologist, and the research worker should sometimes attend operations and see clinical work at the bedside. I have always made it my practice to attend in the laboratory of my hospital at least once a week and I know I have greatly benefited thereby. I am persuaded that the practice is a good one. The observations that are made in the operating theatre and the laboratory are more correctly evaluated if they are frankly discussed between the surgeon and the pathologist. A fact which may to the latter appear of little importance may be of the utmost significance to the surgeon who knows its clinical bearing, and vice versa.

But if the surgeon is to be a biologist in the scientific sense he must learn to make exact measurements. The essence of scientific observation is exact measurement. It has been said that doctors are not scientists because they cannot express their conclusions in figures. In the past this criticism was to a large extent true, morbidity was stated as the opinion of an eminent person unaccompanied by any kind of data, and was often accepted as fact, but in recent years a great deal has been done to obtain exact data in relation to diseased and abnormal states, and medicine is on the way to becoming an exact science where proof will take the place of opinion.

We can now estimate the state of the damaged heart in terms of the electrocardiograph, the condition of the kidneys in terms of the blood urea, and the water balance of the tissues in terms of the calcium content. It is now often possible to express pathological conditions in percentages and figures.

Neither the surgeon, nor the pathologist, should ever cease to learn new facts or new interpretations of old facts. The art of healing has not yet reached any finality, nor is it likely to do so for a very long while. Surgeons have from time to time fallen into the error of believing that their craft had reached such a stage of perfection that little further improvement was possible.

Even that great surgeon of the seventeenth century, Ambrose Pare, fell into the common error of ceasing to be a humble student of nature and becoming too satisfied with what he had achieved, as is evident from this passage in the

dedication of his work on surgery to King Henry the Third of France: "For God is my witness, and all good men know that I have now laboured fifty years with all care and pains in the illustration and amplification of Chirurgery; and that I have so certainly touched the mark whereat I aimed, that antiquity may seem to have nothing wherein it may exceed us, beside the glory of invention; nor posterity anything left but a certain small hope to add some things, as it is easy to add to former inventions". Pare was undoubtedly the greatest surgeon of his age, but were it possible for him to come back to practice today, he would find that the only thing he knew would be how to reduce a dislocation and how to set a fracture, and even in this respect he would be unable to compete with the most junior house surgeon at one of our big hospitals.

This quotation from Ambrose Pare shows how unwise it is for us ever to assume that what we regard as the great achievements in knowledge and practice of our own time are final or unsurpassable, or that we ourselves have learned all that we can. Nothing is ever final, nor can knowledge ever stand still. The true enquirer, and the true surgeon, must always keep a humble spirit, content to help forward to the best of his ability the practice of his craft in his own time and in the sure hope that those who come after him will find the foundations upon which they desire to build are firm and sure, and have not, as the result of his mistakes, been falsely laid. He must be ready to unlearn much that he has learned and start again from the beginning, not once, but many times.

The changes which have taken place in the practice of surgery even in the comparatively short period since I first became a medical student at Cambridge have been very considerable. Thirty years ago, once an operation had been decided upon, the surgeon proceeded to perform it without any further assistance than an anaesthetist and a house surgeon. No preparatory treatment was deemed necessary and the surgeon was quite prepared for the fact that for many hours, sometimes days, after the operation his patient would be violently sick, due to the ether or chloroform, which were the only available anaesthetics. But today the surgeon requires a regular team of assistants if he is to do first class work. Often elaborate tests have to be made requiring the assistance of skilled scientists, a physician's report on the functioning of the patient's various organs has to be obtained, biopsy specimens have to be reported upon by a skilled pathologist, and the surgeon has to rely upon a number of persons besides himself to assist him in bringing the case to a successful conclusion; in other words, the best surgery now requires team work.

This is now generally realized and is a great tribute to the pioneer work of the Mayo brothers, who first instituted it. One of the results of this development is that much more elaborate equipment for hospitals has become a necessity and that operations cannot now be satisfactorily performed in the patient's own home. Another result is that operations have tended to become more expensive, but any extra expense is well worth the additional safety that is provided by modern team work and modern hospitals.

Admirable as this is, it has always seemed to me there is today a real danger that the young surgeon who is just starting his career may readily believe that surgical operations can be performed *only* under circumstances where such services are available, that he may come so to rely upon the facilities which a modern up-to-date hospital, like those I have seen in this city, affords, that he will be unable to operate successfully under circumstances where they are not available. But it may well happen that he will not have the good

fortune to become a surgeon to one of the great hospitals of this, or any other city, but may find himself in some out of the way part of this great continent, or some other continent, where he will be called upon to perform operations entirely without skilled assistants and with none of the facilities to which he is accustomed, and if he is the man he should be he will not be discouraged, but will so improvise his equipment and reorganize his technique as to be able to deal successfully with the situation. Most of us who had to attend to the wounded during the last war had to learn this lesson, and on the whole, if the results were not as good as we had expected, they were not too bad.

Some years ago I had to go to Nepal, which lies between British India and Tibet, to operate upon the Maharajah of that State, and while I was there I was asked to do a number of operations under circumstances to which I was quite unaccustomed. There were no nurses at all, the patients just "stayed put", if I may use that expression, until I came back, often several days later. They did not seem to have moved at all since I had last seen them, although no doubt they had been fed. The anaesthetists were worse than untrained, they were definitely unsafe, even when giving chloroform, which was the only anaesthetic they knew anything about. I managed, however, to perform a large number of operations with, I am thankful to say, excellent results.

While modern methods are a great improvement and are essential if we are to attain to the standard we have set ourselves, namely one hundred per cent success, a great deal of excellent work can nevertheless be attained without them. The best surgeon is not he who can get good results only when working as part of a team in a modern hospital, or who cannot make a diagnosis without a pathological report, but he who can adapt himself to the circumstances in which he finds himself and still make a diagnosis and operate successfully with the minimum of risk and post-operative discomfort compatible with the circumstances.

Every surgeon who is scientifically minded, and I have little use for the surgeon who is not so minded, must be content to try to add something to the sum of human knowledge. To be content to practise his craft just as he has been taught and to leave it as he found it should not be enough. He should strive to add something for future generations.

It takes a long time to establish the truth of new biological discoveries and only those who are content to wait and are able to put up with constant discouragement and disappointment are worthy to call themselves biological explorers. New truths take time to become established and old customs and old ideas die slowly and with much difficulty, but, in the end, truth can never fail to succeed, and to find its proper place. It may be and often is delayed, but it cannot be entirely or permanently supressed.

Today the surgeon is making more and more use of biological research to assist him in saving the lives of his patients, or in enabling him to avoid procedures which he knows may jeopardize his results. Our recently acquired knowledge of some of the facts with regard to the water and calcium balance of the blood and tissues has proved of the utmost value in dealing with cases of severe intestinal obstruction and in avoiding complications after certain operations. We now have tests such as the "urea clearance test" (Van Slyke test) by which we can often estimate before operation the dangers to which our patient will be exposed and which enable us to so prepare him that the risk can be much diminished or avoided. Operative surgery is gradually becoming a science as well as a craft, and those of you who will be performing operations

during the next thirty years will, if you make proper use of it, have at your disposal the means to reduce many of the risks of surgical procedures that are not available to us today.

At the beginning of this century postoperative surgical shock had taken the place of sepsis as the chief cause of death after surgical operations. A certain degree of shock was expected after all major surgical procedures and the notes of that time revealed it as the most common cause of postoperative mortality. Practically nothing was then known about the cause or physiology of the condition and the treatment was confined to keeping the patient warm and injecting strychnine in large doses. It is now quite obvious that many other conditions, not then known about, were either confused with cases of true shock, or were mixed up with it. Such conditions as dehydration, calcium deficiency, what is often called acidosis, and many other now well recognized complications, were at that time not distinguished from true shock.

Looking back upon my early days at the Queen's Hospital for Children, I remember many postoperative deaths among the children in my surgical ward that I now recognize as having been due to acidosis caused by the chloroform then used as an anaesthetic, and which could easily have been prevented by the administration of sugar or alkalies, had we at that time known what to do.

The commonly accepted pre-operative treatment at that time was to administer a smart purge to the patient the day before the operation and to starve him for twelve hours. After the operation water was given in teaspoonful doses and no food allowed for several days. When we realize that as a result of the use of the anaesthetics then in vogue severe vomiting was the rule rather than the exception after any operation, it only surprises me when I look back to those days that our mortality was not worse than it was, but even then our patients often insisted on recovering despite our efforts! The general principle of treatment in those days appears to me to have been that anything the patient desired *must* be bad for him and should, therefore, be withheld!

It was then that Dr. Crile first published his famous monographs describing the results of his research work into the causes of surgical shock. At about the same time I was carrying out research on the same subject in England, and in 1905 I had the honour of delivering the Hunterian Lectures at the Royal College of Surgeons in England on "The Cause and Prevention of Shock".

I was a follower of Dr. Crile's in that I believed exhaustion of the nerve cells controlling the vasomotor centres, from overstimulation by ascending sensory stimuli, was the primary and essential cause of the condition known as "surgical shock". For a time this theory held the field and was to a large degree accepted as the correct explanation of surgical shock, though it met with considerable opposition both here and in England. But in 1917 when my old schoolfellow, Sir Henry Dale, discovered histamine, opinion began to change. In 1919 when he and Laidlaw propounded the theory that surgical shock was due to a toxic agent (histamine) liberated by trauma of the tissues, opinion swung round to the belief that surgical shock resulted from chemical poisons circulating in the blood as the result of the trauma.

Whether the theory of exhaustion of the nerve centres put forward by Dr. Crile and myself over thirty years ago is correct or not is still unsettled, but I think you will agree with me that the value of any biological discovery must be considered in relation to the benefits to humanity that result: in

fact this might be considered a useful "yard stick" for measuring the value of any advance in knowledge. Now, there can be no question that the theory of shock being due to exhaustion of the vasomotor centres led directly to the application of methods in prevention and treatment which have in recent years almost entirely eliminated surgical shock as an operative risk. I can frankly say that I cannot recollect a single case of true surgical shock having come under my observation during the last fifteen years, and that is not because surgical operations have become less drastic or less formidable than previously, for the reverse is the case.

Whether this theory of the exhaustion of the nerve centres is the correct one, or not, future biologists will no doubt eventually decide, but the benefits to humanity of the publication of the theory have already been reaped. It is interesting to observe in this connection that quite recently the biologists and physiologists have shown a tendency to swing over again to our original theory of nerve exhaustion.

No one has been able to detect any substance in the blood coming from a traumatized limb which will cause shock when injected into another animal. Also Simonart has shown experimentally that if the vessels of a traumatized limb are occluded prior to the trauma but the nerve supply left intact, a typical condition of shock develops, although there is no blood passing from the traumatized limb to the general circulation. It looks, therefore as if the original theory of nerve exhaustion is in the main correct. In any case I feel sure that Dr. Crile, like myself, will be content to leave the truth to be established in the lap of time.

A new branch of biology which has recently engaged the attention of scientists throughout the world is destined, in my opinion, to have a profound influence upon medicine and surgery in the near future. I refer to the science of genetics, the means by which heredity works to transmit characteristics from the parent to the offspring. At first it may seem that this has little to do with the healing art, but it is generally recognized that it is as much the duty of doctors to prevent disease as to cure it and anything which will assist us to understand how diseased conditions arise, or are transmitted from one generation to another, will better equip us to combat them.

There is much yet to be discovered in the field of human genetics, but thanks largely to the brilliant work of Morgan, de Vries, Mueller, Loeb, Maud Slye and their co-workers, a great deal has already been discovered.

In the science of genetics, which may be defined as the manner in which hereditary characteristics are transmitted from parent to offspring, we have, I believe, found the key which will open that door, so long closed to us, behind which is to be found the explanation of the cause of tumors. Ever since human diseases began to be studied scientifically instead of empirically the chief problem that has intrigued everyone has been that of the origin of tumors. A vast amount of time and money has been spent during the last twenty-five years in attempting to elucidate this problem, and, although no definite result has been reached, the enquiry has been considerably narrowed. We now know that tumors occur in all vertebrate animals, that they are species specific and cannot be transmitted from one species of animal to another, that if left alone they continue to grow indefinitely during the life of the organism in which they arise, and that if completely removed they do not tend to recur. We know that by selective breeding, mice can be obtained that are predisposed almost one

hundred per cent to the formation of spontaneous tumors, or are almost entirely immune to the development of such tumors.

The first suggestion that tumors might be explained by a change or mutation in the nucleus of a somatic cell was made by Boveri in 1914. He put forward the theory that a mutation of the chromosomes in a somatic cell was the cause of tumors, but when it was found that the chromosomes in the nuclei of human tumors were normal both in numbers and arrangement this theory had to be abandoned, though it may be true of some of the tumors of plants. Bauer had also suggested that a mutation of the nuclear elements in a somatic cell might be the cause of tumor formation (1928). In 1932 I published the theory that tumors are due to a mutation of the genes in a somatic cell for excessive reproduction.

This theory has the merit of offering a satisfactory explanation of the known facts about tumors which no other theory has hitherto achieved. But in order to establish a theory it is necessary to bring forward experimental proof, and at present this is not possible. The genes are, and must always remain, invisible to the human eye and gene mutation cannot ever be visible. So far no one has succeeded in devising any form of experiment that will prove that a tumor results from a gene mutation in a somatic cell, and at present the theory remains as a logical deduction from known facts of the probable causes which result in the formation of a tumor.

A logical conclusion which explains the known facts, even if it is no more than a stated opinion, promotes the progress of scientific knowledge even if it should subsequently prove to be wrong. At present this theory relies on negative rather than positive evidence, but all the findings of Maud Slye and others on genetic factors in mouse tumors seem to support it.

The remarkable observation made by McFarland and Meade on tumors in uniovular twins seems to me strongly to support the theory. They collected twenty reports on tumors occurring in homologous, or uniovular, twins. In every case the tumor was present in both twins at the same time. The tumor was of the same type and occurred in the same organ. In one case both twins developed sarcoma of the right testicle at the same age and in another case both twins at the same age developed duct carcinoma of the right breast. Since uniovular twins result from the division of the original fertilized ovum into two halves, each of which develops into a complete individual, it must follow that they have an exactly identical genetic constitution.

It seems to me that nature has here performed an experiment for us which proves that tumors are due to some genetic change in the nuclei of certain somatic cells. We know that variations which are permanent and irreversible result from gene mutations in the germ cells, and that by this process, working during millions of years, the various species and varieties of living organisms which inhabit this earth have been slowly evolved. It seems reasonable to conclude that similar gene mutations can also occur in the somatic cells of the adult individual. If such a change takes place for increased rate of growth in a somatic cell then since the change must be permanent and irreversible the ultimate result must be the development of a tumor.

An obvious argument against this theory is that if mutations occur in somatic cells there should be many examples of this, apart from tumors, since it is unreasonable to suppose that only mutations occur which result in excessive growth. But mutations which result in any other change will never be seen. A somatic cell is not like a germ cell in having numerous progeny; when it dies

it is only replaced by another single cell, and if it underwent a mutation to become black instead of white, since only a single cell would be affected, no obvious result would appear. It is only when an increased rate of reproduction is included in the mutations that any result can become obvious.

This theory, while it appears to explain the origin of tumors, does not seem to afford any easy solution to that much greater and more important problem of how to prevent or cure tumors in human beings, but knowledge is strength and human experiences seems to show that when the cause of a disease is known it has generally led to the discovery of some means of prevention or cure, and it may well be that tumors will prove no exception.

I hope that my oration this evening has demonstrated that the surgeon should also be a biologist, that he should not be satisfied merely to practise his craft with dexterity and success, but should strive to make use of his unrivalled opportunities for the observation of human processes in health and disease, to learn new truths, however apparently unimportant, which when checked and rechecked may in the course of time add to the sum of human knowledge and lead in the future to better and surer means of alleviating human suffering.

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More Odds and Ends

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Precision in Diagnosis—How Fast Are We Progressing?

THE solid and instructive piece of work by that fine clinician, Richard Cabot, who compared the autopsy findings at the Massachusetts Hospital, with the medical diagnosis in a great mass of material covering the *commoner medical* diseases, should give every reflecting physician food for thought. That a skilled staff of "internists" should be in error, or at least fail to make a *complete* diagnosis, in practically half their cases even when aided by all the modern improvements seems at first sight incredible.

Let the doubters turn to the surgical side, where greater certainty might be assumed to prevail, and take what comfort they can from statements from the heads of the Mayo Clinic, on the accuracy of their skilled guesses in abdominal conditions, acute and chronic. The degree of precision reached in this type of case when a *complete* and *comprehensive* diagnosis is the standard set, is no more exhilarating. I think it has been put about 40%. To illustrate the point, *complete* diagnosis, a label of mitral stenosis on a death certificate is inadequate, if no mention is made of the little wedge of lobar pneumonia at one base which actually *killed* the patient. And a death from rupture of spleen followed by splenectomy (recent case) requires to complete it, the diagnosis of *thrombosis* in *right upper segment of pons* (*basilar artery branch*) to round it out, and to make the rapid death understandable, when for a few days all seemed going well.

To shed a little light on the question under discussion, let us take the disease which has for years had perhaps the most intensive work and the most money expended on it. The disease which supplies the greatest number of *medical* specialists, pulmonary tuberculosis.

Some twelve years ago, and I admit I cannot find again my reference, though one mastered the data at the time, *and they may be taken as correct*, a Boston clinician published some findings which are striking. To try and solve this point of precision in diagnosis to-day with respect to pulmonary tuberculosis, he examined the autopsy records on a series of cases taken from the two or three leading tuberculosis sanitaria of Massachusetts. All of these cases died in these special institutions after varying periods of residences, and, of course, all died under the assured diagnosis of pulmonary tubercle. Out of *452 autopsy records*, there were found *50 cases* in which there were *no microscopic signs of active tuberculosis anywhere in the body.* A diagnostic error of 11.8%. These deaths were all well within the period of improved X-ray technique, the various tuberculin tests and other aids; were, of course, all under "specialist" care, and, all presumably, had afforded ample time for full observation and revision. The findings gave the following as the chief sources of error:

(a) The pulmonary complications or associations of *cardiac* disease.

(b) The lung scarring resulting from other infective (non-tuberculous) conditions in the lung, chiefly the residual effects of *influenza.*

(c) A miscellaneous small group of comparative rarities, e.g., Hodgkin's Disease, the mycotic group, etc., etc.

When the war ended, a big batch of cases returned to Canada with the label of tuberculosis or *suspected* tuberculosis. All of the military hospitals had their share of these. After revision and varying observation periods, most of these were finally sorted out into a definite tuberculosis group, and a larger group where this disease could be finally excluded, mostly influenzal sequelae. Some of these cases proved difficult, and here and there doubtless even at the last a case was improperly labelled.

One's limited personal experience supports the views I am airing, that mistakes in the diagnosis of pulmonary tuberculosis are not very rare even in the hands of "specialists" in this branch, and that there exist in all of the sanitaria a sprinkling of cases, which are never correctly diagnosed as something that at any rate is not tubercle.

(a) During the war-time a youth, under treatment for tubercle in a sanitorium, consulted me (on his own) as to the state of his heart. I found a clear-cut case of rheumatic endocarditis, with a single valvular lesion—aortic regurgitation. A few anomalous signs in left upper lobe were such as are commonly found in cardiac conditions, but are most constant and most definite in cases of *mitral stenosis*. I thought the evidence (or *lack* of evidence, as tubercle could not be found) justified the exclusion of tubercle altogether, and told him so. This boy left the sanitorium, and, against all advice, managed to enlist in the Canadian Forces and went overseas. Two or three years later he came into my hands again at Camp Hill Hospital, with broken compensation and all the signs of gross aortic regurgitation. He died in hospital, and autopsy confirmed the diagnosis, and also the absence of microscopic tubercle in lungs or elsewhere in the body. This man gave a history of having been for sometime in a tuberculosis war hospital, and of being examined by many doctors before he was finally transferred to a heart hospital.

(b) During the war a case was referred from the Kentville Sanitorium for an opinion on the heart. He had been sent into that institution, I ascertained, by two excellent practitioners. In my office he showed a temperature of 103.5°F. and a weak and rapid pulse. He looked septic. A loud rasping systolic murmur was audible all over the front of chest. Later his blood showed a strong growth of haemolytic streptococcus. The pulmonary signs were suggestive of the circulatory disturbances set up by a primary cardiac condition, and tuberculosis was never demonstrated. A diagnosis of acute infective endocarditis was made. Death followed within two months, and there can be little doubt that the endocarditis was the real and only disease. He gave a history of scarlet fever six years before, with a "weak heart" dating from that attack.

Infective endocarditis on an older mitral lesion with deceptive signs in the lungs. Blood culture gave haemolytic streptococcus. In my young days, it used to be taught in the Edinburgh school that mitral stenosis and pulmonary tubercle were antagonistic conditions, and that their association was a very rare thing indeed. Yet Sir Kingston Fowler showed at the Brompton Hospital, at a clinic, three cases of the combined diseases. Nevertheless the double diagnosis *will* be incorrect in the great majority. The trap consists in the very common occurrence in early congestive failure of mitral stenosis of signs in the left upper zone which are particularly suggestive of tuberculosis,

should they be accompanied by fever, cough and haemoptysis. These signs are impaired percussion note, weak or harsh breathing, and râles which are more variable and less consonating than in tubercle.

(c) In two cases amongst the war material, I transferred patients from the tuberculous to the non-tuberculous group with some confidence, chiefly on two grounds, firstly, the constant absence of tubercle in a chronic case, which showed *gross evidence of nasal obstruction.*

Just before the war, in an analysis of cases admitted to the famous old *Walther Sanitorium* in Germany, it was stated that on an average 2% of cases sent in under the diagnosis of tubercle were rejected for this cause. Chronic mouth breathing, with its loss of the warming, moistening and filtering functions of the nasal passages, causes changes in the right upper lobe especially which may simulate those due to tubercle with great precision. It is most important that every effort should be made to demonstrate bacilli, before a final label is put on such cases.

That great simulator of other diseases, syphilis, can provide many traps for the unwary. In the case of hepatic syphilis referred to in my first article, the patient came up from New York with a diagnosis of tuberculosis, and strange to relate he had been under the care of a personal friend of his who had been (before retirement) a *G.-U. specialist of repute.* Yet the presence of syphilis, the patient denying past infection, had been missed, and a non-existent tuberculosis diagnosed. The late E. G. Janeway of New York related a case, which had been under the care of Dr. Trudeau at Saranac with a hopeless prognosis, the patient left the sanitorium, and was brought to Janeway as a forlorn hope move. Janeway came to the conclusion it was a case of hepatic syphilis, and the patient gained forty pounds in six weeks under mercury and iodide and soon was practically a well man.

Janeway reported five other cases of the same character, where tuberculosis had been diagnosed, yet the prompt response to antisyphilitic treatment proved that lues and not tubercle was the most correct diagnosis.

In 1920 I saw with Dr. Crockett of Glasgow a case which had just been admitted to the Bridge of Weit San. Investigation showed that the true condition was Hodgkin's disease. He showed too much dyspnoea for any reasonably early stage of tuberculosis and X-ray of mediastinum and biopsy of an enlarged neck gland were decisive.

Summary. It would appear then that with all our advances and advantages we have still a long way to travel before accurate and complete diagnoses are reached, even in the commoner medical diseases. For in the one on which most money and most specialist effort is being expended for years past, using such data as I have been able to assemble, there is still much leeway to be made up.

Conceding, and it is generous, 100% increase in precision within the last ten or twelve years (when the Boston article was published), we must assume that in most of our leading tuberculosis sanitaria there is a percentage of cases under treatment, which may fairly be put at 5%, where autopsy would fail to confirm the clinical diagnosis of pulmonary tubercle.

This percentage of patients under useless or positively harmful treatment for the wrong ailment are suffering from cardiac disease, the sequelae of pneumonic and other non-tuberculous lung infections, form a very mixed and varied group of the other simulators of tuberculosis of the lungs.

Hyperemesis Gravidarum

Rayfield G. A. Wood, M.D.C.M., Lunenburg, N.S.

HYPEREMESIS gravidarum or vomiting of pregnancy is a condition of dire importance and one which can creep up on the unwary physician who treats it as as an everyday matter, when great danger may be lurking in the background. With this in mind, I propose to review this so common ailment.

Aetiology and Pathogenesis.

As we all know there have been many theories as to the causation of this distressing ailment and the subject has been and is still being debated very thoroughly with the end not yet in sight. The condition has been described as sensitization of the mother to placental extract; stretching of peritoneum over uterus; to disturbed metabolism resulting from disintegration of the endometrium during implantation of the ovum; carbohydrate deficiency; endocrine disturbances; and finally neurosis.

However the vomiting starts, if it continues long enough there will be a serious diminution of glocogen stores in the liver, which causes liver necrosis, which will set up a toxaemia, because a damaged liver cannot detoxicate metabolic products as it normally should. The toxaemia is aggravated by dehydration consequent on vomiting. Fat metabolism is imperfect and acidosis occurs, which in turn aggravates vomiting. Therefore a vicious circle is established.

Clinical Picture and Course.

In pernicious vomiting of pregnancy the common experience of the patient is to be nauseated in the morning, either on awakening or just before getting up. The stomach is emptied, food is taken, and the woman experiences no further trouble until next morning. In other cases the nausea and vomiting recurs as the stomach becomes empty, to disappear on intake of food. If the case is more severe there is an increase in emesis and no food is retained. Sight and smell of food aggravate the condition.

Loss of strength, nervousness, sleeplessness, and general indisposition ensue. If the patient still keeps on the downward path, pulse becomes rapid, emaciation occurs, and the breath has an acetone like odor. The urine contains acetone, acetoacetic acid, and sometimes bile, is much diminished in quantity and of high specific gravity. Jaundice appears, the condition becomes progressively worse and the patient passes into low muttering delirium ending in death.

Under modern treatment many patients improve; some few cases end in foetal death and abortion, and then the patient's condition becomes usually better. Favourable signs are the following: cessation of vomiting, increase in quantity of urine and a fall of specific gravity, slow regular pulse of good volume, B.P. not too low and normal temperature. No permanent damage is suffered by liver or kidneys, but there is often a tendency to recurrence of the hyperemesis in future pregnancies.

Diagnosis.

The test of a doctor's diagnostic acumen in a case of hyperemesis is to determine in which category to put each patient. The diagnosis of pernicious

vomiting of pregnancy depends on the positive determination of a pregnant state and exclusion of other causes. Onset of morning sickness usually occurs between the fourth and eighth week, and may last six to twelve weeks; rarely lasts throughout gestation. Associate vomiting, i.e. vomiting due to some condition accidently associated with the pregnancy, such as gastric ulcer or cancer, cirrhosis of the liver, tabes dorsalis, cerebral tumour, intestinal obstruction, appendicitis, red degeneration in a fibroid tumour, or an ovarian cyst with twisted pedicle, must be excluded. It should be remembered that the vomitus in hyperemesis gravidarum often contains altered blood, which may lead to mistaken diagnosis of gastric disease.

Diagnosis between the non-toxic and toxic types is not always possible; and a case may pass from the first into the second. A diagnosis of toxic vomiting may justly be made if in spite of treatment the vomiting persists and the patient's condition steadily deteriorates. Also if there is jaundice, or bile in the urine the case is toxic.

Treatment.

Probably the question to be considered most thoroughly is the treatment of the condition as too often disastrous results occur from the fact that the condition has a tendency to pass thru stages of increasing severity and thus *it is better to treat each case as one whose condition may progress to serious illness.* The earlier the recognition and treatment, the more satisfactory the results.

One may treat these patients on the basis of the four factors which apparently enter into the syndrome: nervous exhaustion, dehydration, starvation, and hepatic derangement or disturbance of carbohydrate metabolism. This idea of treatment depends on the general consideration that the condition is one of progressive stages of severity, which although not sharply defined, tend to progress from mild to severe.

Patients with early or mild hyperemesis are usually ambulatory, and dehydration and starvation have produced few, if any, changes. The main features in treatment are:

(1) Correction of associated disturbances.

(2) Reassurance, rest, freedom from nervous and physical stress.

(3) Frequent small feedings of high caloric content. Frequent feedings every 2 hours of easily digested dry foods such as popcorn, dry toast, zwiebach, dry cereals, salted crackers, and soda biscuits. Glucose D is a valuable adjunct here.

(4) Intake of fluid between feedings of sufficient amount to meet requirements of individual and to produce ample output of urine. Water is not well tolerated. Ginger ale, homemade lemonade and carbonated water are useful.

(5) Sedatives in sufficient amount to reduce nervousness and the gastric irritability of the patient. Bromides, phenobarbitol and pentobarbitol are useful.

(6) Enemas are very helpful. Avoid drastic cathartics.

(7) Sometimes achlorhydia is troublesome and ½ to 1 dram of dilute hydrochloric acid in orange juice will control symptoms.

As symptoms decrease on the above regimen a more liberal diet is gradually added.

Moderately Severe Forms.

Whether patients in the mild stage of the condition need further treatment depends in part on the clinical response, the patient's nervous reaction, and

the development of dehydration and starvation or evidence of disturbed or lowered hepatic function. If patient fails to improve or becomes worse it is necessary (1) to have continuance of methods outlined above; (2) isolation; (3) rest in bed; (4) proctoclysis; and (5) increase in sedation.

A physician cannot temporize if a patient is slow to improve, as suddenly the patient may become seriously ill. Therefore in many cases it is best to treat the patient as a severe form.

Severe Forms.

These patients are markedly dehydrated and starvation is plainly evident. A strict regimen must be initiated as follows:

(1) Isolation, preferably in a hospital. The chances of securing adequate isolation in the home are minimal. Control of the patient and the patient's environment, even under the best conditions at home is not as absolute as can be secured in hospital. Good efficient nursing.

(2) Rest in bed.

(3) Sedatives. Bromides, 90 to 120 grains are dissolved in each 1000 c.c.'s of fluid injected rectally. Thirty grains of chloral hydrate may be substituted.

(4) All food, fluid and medication by mouth is best prohibited for 24 to 48 hours.

(5) Avenues for combating starvation and dehydration: (a) Proctoclysis. For the first 48 hours as much fluid as can be administered should be given. (10% glucose in normal saline). (b) Intravenous saline. About 1000 c.c.'s of 10% dextrose solution is given t.i.d. (c) Hypodermoclysis. (d) Duodenal tube. If the condition is far advanced the extra fluid and glucose given by the duodenal tube may mean the difference between success and failure.

(6) Record of progress. Careful check on urine for specific gravity, amount, sugar, albumen, acetone, diacetic acid and urobilin. Record of the daily number of vomits and amount of each. B.P. recorded. Fundi oculi examined for evidence of haemorrhagic retinitis.

One may experiment with progesterone, corticin, eostrone, iodine, HCL etc. in mild cases and in severe cases for a short time; but if the eliminating diagnosis settles on a toxaemia as causative, the patient is scrutinized almost continually to discover the first symptoms and signs of liver, brain or other vital organ damage.

The test of a doctor's prognostic acumen is to determine the time to give up medicinal and dietetic measures and empty the uterus. Overhestitancy to do this when the patient presents ominous symptoms is condemnable. The physician who has been following the individual case carefully, noting the signs of progress and of severity, must use good judgment.

The following may be regarded as indications for terminating pregnancy:

(1) No improvement in a severe case under proper treatment.

(2) Jaundice or bile in urine.

(3) Albuminuria.

(4) Pulse persistently over 100.

(5) Persistently low B. P.

(6) Haemorrhagic retinitis.

The method of emptying the uterus should carry a minimal amount of trauma and shock to the patient and supportive treatment should be continued during the time of emptying the uterus and for a period thereafter.

Pressure Paraplegia in Pott's Disease

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PRESSURE paraplegia with its concomitant features arises occasionally in Pott's disease. Paralysis arises most often when the disease is in the upper and middle thoracic vertebrae. The reason for this, is given in that the spinal cord is narrowest at this point and also because it is difficult to secure complete fixation in this region. The responsibility for the paralysis may be situated in the membranes, in the cord itself or in the bones.

Causes in the Membranes. When the membranes are at fault, the cause is usually a pachymeninigitis with a resulting thickening of the membranes and obliteration of the blood and lymph vessels.

Causes in the Cord. Infection of the nervous tissues of the spinal cord with tuberculosis is claimed to be very rare. However, the cord may be flattened from pressure, or oedematous as a result of vascular stasis.

Causes in the Bones. Angulation of the anterior wall of the bony canal may give rise to paralysis, partial dislocation, sequestrum, extention of tuberculosis granulation into the canal, or a cold abscess may be directly responsible for the pressure paraplegia. More than two-thirds of the cases of paralysis of Pott's disease are the result of abscess formation. The pus is usually collected beneath or anterior to the posterior ligament. If the ligament is perforated, an epidural abscess develops, but even though the pus is in close contact with them, the meninges may escape actual invasion. Pus may or may not infiltrate in a circular fashion to surround the cord.

The paralysis is spastic, with occasional involvement of the bladder and rectum. In a review of several articles, relative to the complications occurring in Pott's disease, one finds that only very rarely are sensory disturbances present.

The following report deals with a case in which the syndrome produced by gradual increasing cord pressure, was present.

A colored girl, thirteen years of age, gave the history that in March of this year, she developed a dull ache in her back. On further questioning, the site of this ache was found to be at the level of the fourth thoracic spine. This aching was more severe at night. It was increased by the patient assuming an erect position It was not sufficiently marked to interfere with her attending school.

In April, the aching disappeared and the mother noticed that the patient began to drag her right foot. Three weeks later dragging of the left foot was first noticed. Within a space of three weeks she found that she was unable to walk, or even stand. This was due to a gradual spread of loss of power, which involved both knees and hips.

I first saw this case in the early part of June. I found that she had a complete loss of power of both lower limbs. Percussion of the spine was painless except in the region of the fourth dorsal spinous process. Deep pressure and side to side pressure of this process produced deep pain. A more detailed examination showed the following bilateral findings to be present, spasticity, increased tendon reflexes, clonus, Babinski, loss of sense of passive movement and sense of position, anaesthesia extended proximally on both sides to the

level of the groins anteriorly and to the upper border of the sacrum posteriorly. In other words she had complete anaesthesia and loss of power of the areas supplied by the segments distal to the first lumbar segment.

One week later the line of anaesthesia had extended proximally on both sides to the lower border of the costal margin, i.e., to the area supplied by the seventh dorsal segment. Never at any time was there any disturbance of the bladder or bowel functions. Atrophy of the muscles of the lower limbs was not present.

It was quite evident that one was dealing with a lesion producing a gradual and equally bilateral compression of the dorsal cord. Antero-posterior and lateral films of the dorsal and lumbar spine failed to show any evidence of a bony lesion. X-ray of the lungs was negative for tuberculosis.

Lumbar puncture was carried out in the fourth lumbar space. The manometric reading was 170 mm. Queckensted's and Amyl nitrate tests showed a complete block to be present. Spinal fluid was cell free. The protein was markedly increased, i.e., 210 mg. per cent. Xanthochromia and spontaneous coagulation were absent. Kahn and Hinton tests of both the spinal fluid and blood were negative.

On the following day, lumbar puncture was again performed through the fourth lumbar space and 5 c.c. of heavy lipiodal were slowly injected into the spinal canal. Upon gradually tilting the X-ray table to about 45 degrees with the patient's head being lowered, the descent of the lipiodal was watched on the fluorescent screen. After fifteen minutes there was a complete stoppage to the further descent of the lipiodal at the lower border of the fourth thoracic vertebra. It, therefore, was quite evident that this site of the spinal block corresponded with the line of the segmental anaesthesia.

The next question that arose was concerning the type of lesion producing the compression. In view of the absence of bone pathology in this region, as shown by X-ray, one felt justified in ruling out the presence of Pott's disease. It should, however, be noted that satisfactory lateral films of the upper thoracic spine are difficult to obtain. Such was the case in this patient.

One's mind next turned towards the possibility of the presence of a neoplasm either primary or metastatic. There was no demonstrable evidence leading one to believe there was a hidden primary with a possible metastatic lesion to the spine.

Spinal cord tumors are uncommon in childhood, and are very rare in infancy. Extradural sarcomas are most frequent in childhood. Most of them arise in the epidural tissues and may extend for several segments along the cord. Others arise in the mediastinum or retroperitoneal space and extend into the spinal canal by the intervertebral foramina.

Syringomyelia and primary spastic paraplegia were considered. However, the positive evidence of spinal block, one felt helped to rule out primary spastic paraplegia and syringomyelia unless there was a rapidly enlarging cyst of the central canal accompanying the latter.

Intramedullary Lesions:

1. Sensory loss begins just below the level of the lesion and is often dissociated.
2. Pyramidal tract signs are late.
3. Lower motor neuron signs are often prominent.
4. Root pains are often absent.
5. Spinal block develops late.

Extramedullary Lesions:

1. Sensory loss begins well below in most cases and extends up to level of lesion.
2. Pyramidal tract signs are early.
3. Lower motor neuron signs are often absent.
4. Root pains are usually the first sign.
5. Spinal bock develops early.

The weight of evidence one thought was more in favor of a rapidly enlarging extradural lesion.

The patient was kept under observation for another week without any definite change in her condition. It was felt that an exploration of the cord in the region of the fifth, sixth and seventh dorsal segments was justified.

Under ether anaesthesia the laminae of the third, fourth and fifth dorsal vertebrae were exposed. When the soft tissues between the laminae of the third and fourth vertebrae were being removed, a definite tuberculosis abscess was opened into. The tuberculosis debris was under marked tension and extruded itself through the opening made between the laminae. Tuberculosis granulation tissue was much in evidence. As much as possible was curetted, and it, together with some of the debris, was later examined microscopically to confirm the type of lesion. A modified Hibbs fusion was carried out, following which a plaster jacket was applied.

At the end of three days the protopathic sensation had reappeared in the skin of the abdominal area. This was followed within a few days by the reappearance of epicritic sensation in this area. At the end of three weeks return of normal sensation had progressed distally to the degree that it had completely returned to the dorsum and planter surface of both feet.

Muscular power appeared at a slower rate, first making its appearance in the thigh muscles, then in the legs below the knees, and finally in the feet. The return of motor power to the flexor muscles preceded that in the extensors. Ankle clonus and the extensor response were the last to disappear.

At the present time, i.e., two months following the operation there has been a complete return of sensation and motor power to the affected parts.

SUMMARY

The case of a young girl in which paraplegia developed within a period of three months. X-ray examination of the spine did not show any evidence of Pott's disease. However, I feel that too much importance was attached to this part of the examination. Physical examination together with manometric tests and the use of lipiodal were instrumental in diagnosing the presence and site of pressure on the cord. The diagnosis as to the causative factor was made only by operative exploration. Liberation of the pressure and proper fixation of the part, has thus far, been attended with a complete return of function.

The Small Hospital

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THE "small" hospital, although well patronized by the majority of people in the community is still frowned upon by the few, who, through ignorance, think that on account of its small bed capacity, it cannot give the service that it really does. For instance, some person of the female species, discovers that she has a small swelling starting in her throat. At an afternoon tea this is diagnosed by her lady friends as goitre and she must go to some famed goitre specialist. This she does and later relates to them how the specialist sent her to a large hospital where she had a very scientific test done—they called it a "test of metabolism". She was given some medicine, told to report to her doctor at home and come back in three months time for another test. She was very much surprised when she visited her doctor at home to hear that the very scientific test is very frequently done at the "small" hospital in her own town. In further conversation she is surprised to hear that, in this same small hospital, most of the major operations are performed in a modernly equipped operating room where there is a lamp, that should the current fail, the light stays on; X-ray and fluoroscopy is done; cystoscopy is done; there is a laboratory equipped for doing urinalyses, blood counts and chemistries; there is an ultra-short wave machine with electro-surgical attachments; the rooms and wards are furnished in very modern hospital style and the rates are very much lower than in the hospital where she was.

The work that is being done in a small hospital surprises one—I think our medical colleagues in the larger centres do not realize it either. I am going to give a review of one week at our hospital which has a capacity of twenty-four beds. The cases cited do not include those in for medical treatment. The medical and surgical staff involved, numbers five. To begin the week on Sunday there were five mothers delivered of five living babies. At seven p.m. there was an operation for acute appendicitis on a girl nine years old. She gave a history of having had two previous attacks of pain in her right lower abdomen which cleared up without medical care. The present attack had commenced ten hours previous to admission. The pain steadily became more severe, she became nauseated and vomited several times. On admission —she had to be carried on account of the pain—her temp. was 99.8; leucocyte count 12,600 and urinalysis normal. A very acutely inflamed appendix was removed and she had a satisfactory recovery. The point of interest in this case is the method of ligating the appendix and mesentery that the writer has been using for some years now. The mesentery is pierced, close to the base of the appendix, by a haemostat. The centre of a ligature is grasped, pulled through and cut, thus giving two separate ligatures. The operator takes both ends of one and the assistant both ends of the other and crosses them. One is tied around the base of the appendix first and then the other around the mesentery. This "interlocking" ligature as I call it prevents at times very bothersome bleeding.

Monday morning after one of the participating surgeons had delivered a 39 year old primipera of a lovely baby—the nurses said it was lovely—we operated on a woman 25 years of age for ureteral obstruction. I was not

definitely sure of the cause but from the history and the way it acted I diagnosed a calculus, opaque to X-ray. She first began to complain of pain in right lower quadrant three years previous to admission. The last year this pain has been more or less constant and the last month she has had frequency of urination, pain in right costo-vertebral angle and several attacks of pain simulating ureteral colic on right side. Her leucocyte count on the day following admission was 7200. A catheter specimen of urine, which is the only one of any value, was negative for albumen, sugar, pus and blood. I made a cysto scopic examination with the following result: the cystoscope passed easily. Bladder capacity normal. No abnormality of bladder wall. No calculi in bladder. Both ureteral orifices looked normal. A catheter was easily passed up left ureter. A catheter could not be passed up right ureter more than one half inch. A flat plate taken then did not show a calculus. I then gave her diodrast intravenously. Three films were taken at intervals. Left kidney pelvis and ureter showed normally. There was a very faint shadow in right pelvis. The dye did not come down the right ureter. As yet I could not make a diagnosis so did another cystoscopic examination the following day. Again I could not pass the right ureteral catheter but while trying to do so she had a severe colicy pain in right lower quadrant and later pain in lumbar region, requiring morphia to relieve. Indigo-carmine came from the left ureter in 4 minutes, but had not come from the right in 19 minutes. I decided she must have a calculus in lower end of the right ureter embedded in its wall and that I had moved it with the catheter. She had several more attacks of renal colic before we were able to operate Monday. I cut down on the right ureter extraperitoneally. It was dilated to the size of her iliac vessels and was very tense. I made a short longitudinal incision in the ureter from which clear urine escaped freely. The hydroureter extended downward to where the right uterine artery crossed it. This artery seemed to be pressing on the ureter. No calculus could be palpated. An ureteral catheter was passed with difficulty downward into the bladder. I then passed a graduated bougie into the bladder dilating the lower end of the ureter to a F. 12. I then tied and cut the uterine artery where it crossed the ureter, thinking it was the cause of the obstruction. Four weeks after I gave her diodrast again. The films show perfectly normal kidneys and ureters, and patient is symptom free.

Tuesday morning we did a posterior gastro-enterostomy for pyloric obstruction. This was on a man 44 years of age. For years he had been treated for indigestion and ulcer of the stomach. For a month or more previous to admission he had almost continuous pain in the epigastrium with vomiting. Being unable to eat he was losing weight rapidly. A barium series was done. This showed a very large stomach, which, when standing, was in the pelvis. A small stream of barium the size of a steel knitting needle could be seen going through the pylorus. At operation a mass the size of a large walnut occluded the pylorus. A posterior gastro-enterostomy was done and a section taken from the pyloric mass for biopsy. Dr. Smith's pathological report was as follows:

"Histological appearances are those of a chronic inflammatory mass. Microscopic appearance does not show malignancy". He made an uneventful recovery. Before discharging him from hospital I gave him some thin barium which under the fluoroscope was seen to pass freely through the new opening.

Wednesday morning we did a cholecystostomy and an amputation of a leg. The former was on a man of 50, very athletic build but of a peculiar

physical makeup. He had been gassed while overseas in the last war, had pleurisy and a partial collapse of his right lung. Since then his pulse has been around 100 as well as his systolic pressure being around the same figure. An electrocardiograph did not show organic heart disease. About a month previous to admission he had a severe attack of pain in epigastrium and became greatly distended with gas. This attack was relieved with an enema and morphia. In the next two weeks he had several slight attacks of pain in epigastrium. One the day of his admission to hospital, he had another severe attack requiring morphia to relieve it. X-ray showed cholelithiasis. He was given bile Salts with Vit K (E.B.S.) and glucose D several days preoperatively. At operation the duodenum was firmly adherent to infundibulum and lower third of under surface of gall bladder. These adhesions could not be broken down. On account of this and the profuse bleeding that occurred when the gall bladder was started to be stripped from the liver beginning at its fundus, I decided to remove the stones and drain. He made a splendid recovery. Before discharging him I took a flat plate which showed no stones remaining.

The other case that morning was on a man 74 years of age. He and John Barleycorn, who were very good friends, went for a car drive and in some unexplained way had a collision with another car. The result was a compound comminuted fracture of tibia and fibula at junction of middle and lower thirds. At open operation we removed the small fragments of bone, put the ends of the tibia in apposition and applied a plaster cast. At the end of two months no callous whatever showed by X-ray and when the cast was then removed, there was no union at all even though he had been taking large doses of calcium. On Wednesday morning we amputated at junction of upper and middle thirds. This healed by first intention and he was discharged in two weeks.

Thursday things were going along quietly when one of the staff brought in an acute mastoid confirmed by X-ray after admission. A specialist was called in who did a mastoidectomy. This man made a very good recovery; was up in two days and back doing mason work in 22 days.

Friday morning we operated on a boy 11 years old for right inguinal hernia. He developed it shortly after birth. He wore a truss for a year and for two years after did not have a return of it. We did a Bassini operation on him. After we dissected the sac to the internal ring we opened it and staring at us was his appendix. We pulled it down and removed it using the interlocking ligature. He made an uneventful recovery.

In the afternoon, a boy aged 9 years was admitted with double Otitis Media. He had been referred before admission to an ear specialist who diagnosed a zygomatic mastoid on the left side. After admission, X-ray showed definite involvement of left mastoid. After we used ultra short wave on his mastoid, his temperature dropped to normal in 12 hours. The discharge stopped in three days. I have used this treatment on several cases the last year with very good results.

Saturday, A. M. we did two T. and A's. In the evening one of the staff brought in a case for caesarian section. She was a primipera aged 25 years. Had no prenatal trouble. She had a breech presentation confirmed by X-ray after admission. Her perineum was very strong and introitus small. Both she and her husband were very anxious for a baby so a caesarian section was decided on. Her baby weighed $8\frac{3}{4}$ lbs. Both mother and baby did well. Thus we came to the end of a week in our small hospital.

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It is to be distinctly understood that the Editors of this Journal do not necessarily subscribe to the views of its contributors, except those which may be expressed in this section.

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HALF-LIGHT

Quickened by eerie night,
Ghostly and shadow thin,
Sheaves in the pale moonlight
Usher the harvest in.

NOT all mortals see an object in the same way and not many see that object in quite the same way on different occasions. It is not, therefore, surprising that many people are not seeing life clearly in the light of present world events. Few there must be who are sure and bold enough to claim that they can see with clear vision through the troubles of these times. Far more, one thinks, are seeing their lives just now in a sort of half-light, unreal and fantastic or perhaps despairingly hopeless as their own particular nervous systems dictate.

Half-lights affect our vision in odd ways, so that an object seen at dawn or dusk may look quite different in the light of the moon, and the clarity with which people perceive the events of the times plays a tremendous part in shaping the morale of nations. Our people may be seeing these events apathetically as they would at dusk, or hopefully as at the break of day, but many are seeing only with fearful unreality in a sort of mental moonlight, and the moon, however full and bright, allows to human eyes the merest travesty of vision.

How easily one recalls the evil threat that emanated from some still shadow by the roadside late at night, accentuated not by the darkness but by the moon and by the hour! That eerie time when sound, perhaps some creaking of a weary tree, takes on the attributes of brutal force and sends its waves to strike to life the silent shadow of an immobile stone! How uncertain in that weird half-light are all the things we see and hear. Even those homely things that are about us every day, by which we plan the pattern of our peace-time lives, take on the sinister attributes of death. The very goal toward which we strive is ringed about with dangers, terrible and real. Hope crys no call of comfort in the night and reason wanders in a blinded agony of fear!

We in our time are living in the most tragic half-light the world has ever known. But whether dusk or dawn or moonlight none can know and each

perceives it in a way peculiarly his own. How very difficult it must be for those people whose imaginations add to this eclipse of humanity and reason the weird distortions of moonlight.

Such may be the manner in which confusion and neurosis combine to undermine the discipline and confidence of great nations.

The dimness of dawn creates no such weird fancies in the human mind. The deepening shadow crouches not to spring, but to give thanks for surely coming light and warmth which soon will cast a purple mantle on the hills and drive the mist herds slowly upward from the vale. The sounds, no longer eerie, cease to warn of death and speak instead the thousand tongues of re-awakened life and hope, till sunshine throws in bold relief the sure and solid purpose of our labors and our dreams!

Those souls are indeed fortunate who see with clear eyes and fine courage the events of these times as prelude to the dawn of a finer and better world. They need no help. They are, indeed, the helpers and physicians should be numbered first among them. The Empire is in need of many more such as they, and of hearing more, much more, from them.

The dusk is melancholy with the dying day and fills the heart with sickly sweet nostalgia for the past. The hour of work is done, and darkness challenged only by the specks of light set here and there by some devout disciple of the truth. The journey ends—the timid make their camps and unresisting wait the coming of some other dawn.

No one cares to think of it, but the possibility remains that for all the world it yet may be the dusk of a night so barren and so black that all the culture of a thousand years may cease to grow in it and ultimately die. The weak will perish with it but the Tallyrands, like rats, will find their way through every lightless cavern of the world to troves of treasure shared out by their kind. Good men there will be who can walk a darkened road, slowly maybe and with a halting step to reach the journey's end. There they will find reward enough for those who dare the road they fear that they may carry the love of freedom and humanity into the light beyond.

Unfortunately the lives and thoughts of hundreds of people are illumined only by the cold and unreal moonlight of fear. For them there is neither the relief of anticipated dawn nor resigned acquiesence to the enveloping darkness of interminable night. They have, in common parlance, jitters. The activities of their day are scheduled, not so much by the common clock, as by the pain of their peptic ulcers and the dictates of the modern gods of the newscast. Before them they sit with clammy hands and palpitating hearts to curse the bad news and disbelieve the good—then rush at break neck speed from where they are to somewhere, anywhere else, to be in time to hear their favorite in his next broadcast! They know the hour and the tendencies of each and find his tiny habitat upon the dial with swift and unerring fingers. When low in mind they seek a gloomy man who will substantiate their worst forebodeings. Should they be gay they seek a man more gloomy still, lest their brief lapse from melancholy bring some calamity upon our cause!

These unhappy creatures are the civil victims of war neurosis, and as such require the constant encouragement and sympathetic management which we, in peace time, were wont to offer cases of this kind.

It therefore behoves the medical profession, first, to take the measure of its own neurosis and keep itself from careless mourning on the times. Whose

words, more than the doctor's, are received and thought upon, and passed with wagging heads and slight embellishment, to neighbormen and friends. The doctor, long a recognized and oft impassioned blower of political glass, must, more than ever in these times, be conscious of his influence, and shape with so much skill the molten mass upon his pipe, that though it may contain all truth, the crassest optimism only will decant from it.

In this way may we be of real service in nullifying the war of nerves and by helping our patients escape the pitfalls of neurosis, do much to maintain the morale of our people.

Personal Interest Notes

DR. and Mrs. A. B. Campbell and son, Jim returned home to Bear River the end of September after spending a month's vacation visiting in Tatamagouche, Cape Breton and Halifax.

The marriage took place at Halifax on September 18th of Miss Gladys Hildred Ritcey, daughter of Mrs. S. Ritcey and the late Clarence E. Ritcey of Halifax, and Dr. Herbert Lindsay Knodell, of Port Hawkesbury, son of Mr. and Mrs. George Knodell, Halifax. Dr. Knodell graduated from Dalhousie Medical School in 1937, and had been practising in Dominion until a few months ago. Dr. and Mrs. Knodell left on a trip to Upper Canada, and on their return will reside in Port Hawkesbury where Dr. Knodell has been practising for the last few months.

Dr. T. I. Byrne, formerly of Dartmouth, and lately residing in Liverpool, has purchased the home of the late Mrs. E. M. Viets in Yarmouth, and Dr. and Mrs. Byrne will in future make their home in Yarmouth.

Dr. E. F. Moore of Canso with Mrs. Moore is enjoying a well-earned holiday in Halifax. Dr. Enid Johnson, daughter of Rev. W. H. and Mrs. Johnson is relieving during Dr. Moore's absence.

The wedding of Miss Marjorie Constance MacArthur, R.N., eldest daughter of Mr. and Mrs. Henry E. MacArthur of Pictou, and Dr. John Roderic Cameron of Sheet Harbour, son of Mr. and Mrs. J. H. Cameron of Grand River, C. B., was celebrated on August 14th, at Pictou. Dr. Cameron graduated from Dalhousie Medical School in 1940. Dr. and Mrs. Cameron are now residing at Sheet Harbour, where Dr. Cameron has been practising since graduation.

Dr. H. J. Pothier and son, Hector, of Weymouth, recently returned from a trip to Maine.

The Nova Scotia Radiographers Society was formed on May 22nd, 1940, for the purpose of improving radiographical work in hospitals of the province.

Officers elected were:

President:—Mr. A. Perry, Camp Hill Hospital, Halifax.

Vice-President:—Mrs. A. Campbell, Halifax Infirmary, Halifax.

Secretary-Treasurer:—Mrs. B. Hyland, Victoria General Hospital, Halifax.

Registrar:—Miss W. Flynn, Victoria General Hospital, Halifax.

Advisory Board:—Dr. S. R. Johnston, Victoria General Hospital, Halifax; Mr. G. G. Harrison, Nova Scotia Hospital, Dartmouth.

Provincial Representatives:—Rev. Sr. Regina, Hamilton Memorial Hospital, North Sydney; Miss Cowan, Aberdeen Hospital, New Glasgow.

Dr. and Mrs. H. A. Creighton of Lunenburg, spent a week motoring in Nova Scotia early in October.

Dr. K. W. Parsons, who graduated from Dalhousie in 1934, and is now practising in Howley, Newfoundland, was a recent visitor in Halifax. Dr. Parsons was spending his vacation motoring in Nova Scotia.

This Man—Churchill

This sturdy little man, with his cigar
This scion of a line that knows no fear,
This son of Britain, whose adventurous Star
In Britain's darkest hour sheds radiance clear:
This prophet of the Truth we feared to face
(And, fearing, turned a deaf ear to his plea!)
This man upholds the Honor of a race
Pledged to protect the world from tyranny.

This man whose bulldog mien is Britain's own,
This man whose dauntless courage nerves us all,
This man who speaks our heart in measured tone,
Whose words sublime our Heritage recall—
This Champion of Liberty who flings
An Empire's gage into the tyrant's teeth...
Inspired by this man's Faith, an Empire springs
To draw the sword for Freedom from its sheath.

This man Beguiles us with no honeyed speech,
Nor with "I told you so" wastes precious breath;
This man would have us gaze on Dunkirk's beach
And know the Sacrifice that conquers Death!...
This man who offers naught but Blood and Tears
By this man led, shorn of all doubts and fears,
An Empire marches on to Victory.

G. L. Creed, R.C.A.F.

(From the *Halifax Herald*, September 25, 1940)

OBITUARY

COLONEL HUGH A. CHISHOLM, M.D.,V.M.G.,D.S.O., retired Quarantine Officer for the Port of Halifax who won high recognition for his services with the Army Medical Corps overseas in the First Great War, died at his home, in Halifax, on September 25th.

Born in Cambridge, Mass., Colonel Chisholm was the son of Duncan and Mary Chisholm, and moved to Linwood, Antigonish County, with his parents at an early age. He was educated at St. Francis Xavier and McGill Universities, graduating from McGill in 1905, and entered the Permanent Army Medical Corps in 1910.

At the outbreak of the First Great War he was the first officer in uniform to arrive at Valcartier Camp, where he was appointed to supervise sanitation. On sailing from Canada the same year he was promoted from Captain to the rank of Major. In 1915 he sailed from Bristol, England, to France where he served with the First Canadian Division until the summer of 1916 when he was appointed Assistant Director of Medical Services of the Fourth Division with the rank of Colonel.

He was awarded the Distinguished Service Order for collecting wounded soldiers in "no man's land" after the retirement of the Canadian Division from the second battle of Ypres, and was mentioned in dispatches by the Commander-in-Chief. He also received mention on two other occasions.

Colonel Chisholm took part in every major engagement until 1917 when he was named Deputy Director of Medical Services at London. He returned to France and was with the Canadian Division as it marched into Germany at the signing of the Armistice.

Returning to Canada in 1920, Colonel Chisholm was Inspector of Health for Nova Scotia until 1928. He entered the Department of Pensions and National Health the following year. He was quarantine officer for the port of Halifax until his retirement last year because of ill health.

Colonel Chisholm was Honorary President of the Red Chevron Club, a member of the Canadian Medical and Canadian Public Health Associations, the North British Society, the United Services Institute and the Ashburn and Waegwoltic Clubs.

He leaves to mourn their loss his widow, Mary Eulalia, two daughters, Ruth, (Mrs. W. W. Porteaus), Victoria, B. C., and Jane (Mrs. J. R. Mote), Boston, two sons, Hugh, interne at the Victoria General Hospital, and Edward, at home; two brothers, R. D. Chisholm, Maplewood, N. J., and W. R. Chisholm, Linwood, Antigonish County, and one sister, Mrs. H. Stewart, West Newton, Mass.

Dr. Victoria Sarah Ernst, retired physician and surgeon, died at her home in Bridgewater, on October 4th, at the age of eighty-four, having been born on July 27th, 1856. A daughter of the late Mr. and Mrs. Caspar Ernst, one of Lunenburg County's oldest families, she was the last surviving member of her family. Graduating from the Provincial Normal College at Truro at an early age she taught for fifteen or twenty years in the Bridgewater and

County Schools. She then became interested in medicine and graduated from Dalhousie Medical School in 1900. She specialized in women's diseases and practised her profession in her native town for about twelve years. On her retirement from active practise she entered the real estate field, and at the time of her death had large holdings in residential, tenement and other buildings in all parts of the town.

A great lover of children she is survived by two adopted sons, Carl F. W. Ernst, 18, and Avard Begin, both of Bridgewater. Another adopted son, William B. Herman, died some years ago. She is also survived by a cousin, Dr. Alice Ernst, retired missionary in India, now residing in the United States, and a nephew, F. Bernal Ernst of Bridgewater.

The BULLETIN extends sympathy to Dr. O. B. Keddy of Windsor on the death of his father, William Keddy, which occurred on October 3rd. Mr. Keddy was ninety-three years of age, and a pioneer of lumber and pulp industries in the province.

The Cod Liver Oil Situation

It is likely that we will experience a shortage of Medicinal Cod Liver Oil unless existing supplies are carefully husbanded. Norway naturally has been eliminated as a source of cod liver oil. To add to our difficulties Iceland which produces much of the finest oil had an exceptionally poor season and their yield of oil is only a fraction of the normal.

By drawing on their reserves and enlisting the co-operation of Newfoundland fishermen, E. R. Squibb & Sons of Canada will be able to supply Medicinal Cod Liver Oil in reasonable quantities of their usual high potency. Squibb Cod Liver Oil is so rich that one 5 cc teaspoonful supplies 9000 international units of vitamin A and 1300 international units of vitamin D (far more than the accepted daily requirements for infants and growing children.

Physicians are urgently requested when prescribing Squibb Cod Liver Oil to order only 1 teaspoonful daily. Where additional vitamin D is required, rather than ordering multiple doses of the plain oil, it will be appreciated if Squibb Cod Liver Oil 10D is specified.
