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Contents January, 1947

The Toxaemias of Pregnancy

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I HAVE been very much honored as a member of the teaching staff of this University to speak at this time. I appreciate the honor and opportunity very much.

I have been asked to speak on the toxaemias of pregnancy. The state of pregnancy is a physiological condition in a normal healthy female, but the border line is often very indistinct, and the strain of pregnancy is felt by every system of the body so that it is not surprising that in the presence of well established systemetic lesions liable to exacerbations that recurrences occur, but more disturbing is the fact that perfectly healthy women may develop serious and even fatal lesions during the course of their pregnancy. These lesions are grouped together and called for want of a better name the toxaemias of pregnancy.

They are usually grouped into the toxaemias of the early months and the toxaemias of the late months. The subject is a large one, far too large to attempt to discuss in full in the time available to-day and so with your permission I will confine my remarks to those of the latter months of pregnancy. If I am able to bring out one good point for each one listening to me I will be satisfied as I remember one of the dictums of a former teacher of mine was just that "not to talk too long and be sure to teach one point well."

I think it is an agreed fact that the underlying cause of these conditions is not known, the varied manifestations of symptoms and signs and the clinical course leads one to believe that perhaps it is not an entity, at times the toxin may have a selective action and the resulting lesion may be confined to one system in great measure, so that the nervous system will be affected and the patient will have marked irritability of the central nervous system with irrationality, stupor, convulsions, etc., and the effect on the circulatory system as shown by increased blood pressure, oedema, etc., be minimal, or the converse be true, marked oedema, high blood pressure, albumenuria to an alarming extent with seemingly little effect on the central nervous system.

So it is hard to classify these toxaemias, but as a guide for my talk I am going to use the classification of The American Committee on Maternal Welfare:

Group A. Diseases not peculiar to pregnancy.

1. Hypertensive disease.
   (a) Benign (essential) mild, severe.
   (b) Malignant.
2. Renal disease.
   (a) Chronic vascular nephritis or nephrosis.
   (b) Glomerulonephritis, acute or chronic.
   (c) Nephrosis, acute or chronic.
   (d) Other forms of severe renal disease.

Group B. Diseases dependant on or peculiar to pregnancy.

1. Pre-eclampsia.
   (a) Mild.
   (b) Severe.
2. Eclampsia.
   (a) Convulsive.
   (b) Nonconvulsive.

Group C. Vomiting of pregnancy.

Group D. Unclassified toxaemias.

It is to Group B that we will direct our thoughts.

In considering the pathology of these conditions, and just here may I make a point that I think is important in our thinking, that pre-eclampsia and eclampsia are one and the same condition in different degrees, I always teach this to my students as I notice among the students there is a tendency to pass a pre-eclampsia off rather lightly and to become very serious if it reaches a degree where a convulsion occurs, so the pathology of the two conditions can well be considered at the same time. We can think of it under the he adings of

1. The toxin itself.
2. The effect on the placenta.
3. The effects on the maternal organs.
4. The effects on the foetus.

I stated earlier that the cause of toxaemias was not known, and that watching the effects of it, it seemed to me that it was not an entity because of its protean manifestations, as opposed to that, last November The Canadian Gynaecological Travel Club were guests for a day at the Women's Free Hospital at Brookline, Mass., and we were shown some work that was being done by Dr. George Van Smith and his associates. At that time they had produced a toxin artificially by injecting turpentine into the pleural cavities of dogs, which would produce on being injected into pregnant dogs all the clinical conditions of toxaemia. I have watched the literature since but have not as yet seen anything published, it would seem they are very close to isolating the toxin of toxaemia.

Some very fine work has been done in England on the effect on the placenta, they are pleased to consider it in three stages, first there is a thickening of the large and small vessels of the villi right down to the terminal capillaries with a narrowing of the intervillous spaces, the next stage is a state of obliteration of these spaces with a beginning loss of definition of the structure of the villi, and finally the whole structure becomes homogenous infarcted area.

The effect on the maternal organs are most characteristic in the liver where we see petechial haemorrhages and also pale areas, microscopically near the portal vessels are distended capillaries with often infarcted areas, and central necrosis. The kidneys are large, pale near the cortex and congested at the medulla, microscopically the essential lesion is a thickening of the basement membrane of the glomericilar capillaries with swelling of the cytoplasm; the brain's most common lesion is haemorrhage either petchial or massive. The lungs become oedematous and the heart engorged.

The organs of the foetus show none of the definite changes of the maternal organs but the nourishment of the baby is interferred with by the placental changes and the baby is most often undernourished and premature or deadborn.

In spite of the fact that we have previously stated that the toxin causing toxaemia has not been isolated just here we could for a few minutes review the latest trends of investigation that are going on as to the causes under aut-ointoxication, anaphylaxis, endocrinology, and deficiency diseases.

From Hippocrates until the last century it was considered a disease of the nervous system, then Lever's discovery of an associated albumenuria changed the disease into the class of renal disease where it remained for about forty years. Now for the last fifty years it has been the subject of intensive aboratory and clinical research without any certain conclusion as to the origin being reached.

A number of inferences have been drawn from clinical observations, there appears to be a seasonal variation being more common in cold weather and being less frequent in warm countries, again part of this has been crediited to the diet. During the first Great War the incidenec fell in large cities in Europe where the food was mostly vegetables and less protein, another observation has been that when there is a deficiency in Vitamins A and D and in the calcium content the incident of toxaemia increases. Blood analysis does not support this as the blood calcium remains normal, the variation may take place only in the ionized calcium.

Many people have thought that there was an underlying endocrine cause of toxaemias, the theory that a thyroid deficiency was responsible has not been proved. The para-thyroids control the calcium metabolism and a deficiency in their secretion would cause a tetany in pregnant women, no definite relationship has been established as yet to show any relationship in eclampsia. Recently it has been suggested that hyper secretion of the posterior pituitary gland of pressor and antidiuretic substances might cause hypertension and oedema, but as yet this has not been proven to be the cause of eclampsia.

Pre-eclampsia, Mild:

Here the three cardinal symptoms of hypertension albumenuria, and oedema usually occur after the twenty-fourth week in varying degrees, there is often a sudden gain in weight before external oedema is evident, this is often the very first evidence of a beginning change in the metabolism, then a mild degree of palpable oedema, a beginning rise in blood pressure 130-140 systolic and 80-90 distolic, with a trace of albumen in the urine. These symptoms are four or five times as frequent in plural pregnancies. Blood chemistry is disappointing as an aid to diagnosis. Later in advance stages the uric acid goes up some, the CO^2 combining power goes down, etc., but in the early stages these are not much changed. In the pre-eclampsia severe cases the symptoms and signs are simply increased and are present in a greater degree, with beginning changes in the retinal arteries.

Eclampsia:

When the toxaemia reaches this stage, it can be considered under three classes, (1) The slow type developing gradually from a pre-eclampsia, (2) The

fulminating type, and (3) The type in which coma develops rapidly without convulsions.

The first is probably the most common, where in spite of or without treatment a progressive pre-eclampsia goes on to convulsions, these so often occur in an illegitimate mother that is hidden away by her people and has no pre-natal care or observation, and then when she develops convulsions a history is obtained of swelling of the extremities, headache, etc. For a case under observation there is very little excuse for allowing this to happen.

In the fulminating type the onset if different, the first symptoms may be a convulsion. I have had this happen to me in practice, to have a woman near term be checked in the office and be normal to all tests and be given instructions to report back in one week and several days later before her week was up to have a convulsion, or to appear in the office in one week as instructed with an impending eclampsia in full bloom.

The third type I know from reading only, they do occur according to the literature, they are called "Eclampsia without Convulsions." Here the toxaemia is so overwhelming that the patient goes into coma and usually dies without convulsions, and the diagnosis is made on the microscopic examination and kidneys and liver, etc.

I do not believe it is necessary to describe an eclamptic seizure, but there is nothing more terrifying or that causes a usually very attractive female to look more like an animal.

I think just here would be an excellent place in this talk to say a word about differential diagnosis between these pre-eclampsias and eclampsia versus chronic nephritis and other renal and hypertensive diseases. These are something most difficult or even impossible to differentiate at the time. The symptoms from the renal diseases as a rule appear earlier, often in the first trimester of pregnancy instead of the third, unless of course the amount of kidney damage is small and the kidneys have enough reserve to carry on without symptoms until later, then again the history helps us as there is often a history of kidney deficiencies available in these early cases or even a known diagnosed kidney lesion. The blood chemistry may help in the early cases showing a definite retention of urea, etc. in the blood with a lowering of the CO^2 combining power, etc. Very often it remains until the peurperium to complete the differential as a true toxaemia will, if it is not complicated by any other lesion most often clear up rapidly, the blood pressure dropping rapidly, the oedema disappearing as rapidly, and the urine become clear of albumen, whereas the chronic nephritic will go on with the symptoms somewhat improved but present for a long while or permanently. In the so-called essential hypertension the only symptom may be increased blood pressure, if one is aware of this before pregnancy it helps, if a woman appears with an abnormally high blood pressure as her only symptom, she may carry on quite well. I have one such patient, who gave me considerable concern as a few hours after delivery the last time she had an acute cardiac failure during which she nearly died, and death is so final.

Treatment of Pre-eclampsia;

Prophylactic: I really believe that a great deal of the pre-eclampsia seen can be prevented by proper pre-natal care as follows, when the patient is first seen, she should be given instructions regarding proper care of her diet

and her elimination. Her diet should be a balanced diet containing an adequate amount of carbohydrates, fats and proteins. As a rule she will eat enough of the first two, but she must be warned to have sufficient protein. To this should be added extra calcium and Vitamins A and D. I believe that it is good precautions to keep her salt low right from the start and then make sure that she has daily adequate elimination from her bowels. I follow all these routines in my practice and I have very few toxaemias to deal with. I also make sure and insist that she has plenty of water as many women are poor water drinkers. Then her general health is improved by insisting that all foci of infection, such as teeth, tonsils, etc., be cleaned up.

Curative: The essential treatment is early diagnosis, this overlaps our prophylactic measures, the urine of a pregnant woman should be examined every four weeks up to the last three months, then every three weeks in the seventh month, every two weeks in the eighth month and every week in the last month. At these examinations her blood pressure should be taken and her weight carefully recorded. Often it is the sudden retention of water in the tissues with a sudden excessive gain in weight that will be the first symptoms of oncoming toxaemia. When these symptoms are found in even minimal amounts then is the time to institute treatment. I believe that rest is so important here, restrict her salt, give an adequate amount of fluids depending on the oedema, a lowered, but still sufficient amount of protein, clean out the bowel well and follow up with a moderate saline cathartic, the total twenty-four hour specimen of urine should be measured and recorded and the amount of albumen estimated.

In most cases with rest and the above treatment the albumen will disappear and the blood pressure will come down and the oedema disappear, the patient is then rested and gradually has her activities and diet increased, but however, if there persists a blood pressure above 140 cystolic, 80-90 diastolic and persistent albumen for a period of over two weeks, or if the symptoms increase in spite of rest and treatment then the question of interruption of the pregnancy must be considered as a preventative in one case of permanent kidney damage and in the other of approaching eclampsia.

Treatment of Eclampsia:

Again may I repeat that the pro-phylactic treatment is most important. In the last thirteen years I have only had one case in my private practice that was under my care having pre-natal attention that went into convulsions, and she was a fulminating type that was in my office and to all the usual tests normal, and five days later had a convulsion at home, but even then the history was that she had headaches and oedema for three days and did not call me as is the usual instructions to patients at that time of their pregnancy. We have likewise to the best of my knowledge had only one woman develop convulsions that was under prenatal care at our Outdoor Clinic. So that most eclampsia can be prevented, but when we are presented with a case we must then do more than argue that it should not have occurred. To me the underlying principles of treatment of eclampsia are as follows:

1. To protect the central nervous system from damage.
2. To improve the elimination through both the kidneys and the bowel.
3. To protect the liver from damage that is beyond repair.
4. To get a live baby if possible.

Stroganoff in Russia in 1911, published a series of 600 cases treated by conservative methods, with a maternal death rate of 8%, which was an improvement over the then popular radical method of prompt Caesarean Section done on all cases as soon as the first convulsion occurred, with an average maternal death rate of 18%, which in turn had been an improvement over the previous method of treatment of the condition as a nervous disease and treated by sedatives with a maternal death rate of from 33%-50%.

Stroganoff's method has been the basis of most of the conservative methods of treatment, it has been modified many times, and added to but is still the underlying basis of most of our treatment. First the patient should be in hospital if at all possible, she should be isolated in a darkened room, and kept protected from other external stimuli than light such as noise and rough handling, that protects her central nervous system, which is further protected by adequate sedatives, he used first morphia hypodermically with chloral and later chloral and bromides rectally, later magnesium sulphate has been substituted for the chloral, etc., with good results. I do not believe that any one or two drugs are the only ones that can be used, but we must give adequate sedatives to prevent if possible the recurrences of convulsions, as each convulsion predisposes to paralysis of the respiratory and circulatory centres, to cerebral haemorrhage, to pneumonia and possible late mental disturbances.

To improve elimination the bowel should be well washed out and then some magnesium sulphate either given as the patient becomes conscious, or in those cases where the stomach is washed left there when the stomach tube is removed. This will improve the elimination through the bowel, if the patient is very cyanotic and, has signs of right sided congestion, I believe it is a great help to remove by venesection from 5 to 800 cc. of blood, at the same time some hypertonic glucose solution in the vein will often stimulate the output of urine and at the same time supply some carbohydrate to the liver which is a protection to the liver cells. The patient must be kept warm, warmth stimulates dilatation of the peripheral vessels, facilitates the course of the circulation and the action of the heart, and perspiration helps in the elimination of toxins. Intravenous glucose is given by me in the effort to protect the liver, this can be given in hypertonic solution to induce diuresis or in 5% solution if the patient is not oedematous.

We must keep in mind that as soon as we begin to get improvement in symptoms that we must plan our type of labor. Most often we will find the cervix softened and open some and then a simple rupture of the membranes will throw the patient into labor, I have at times used a bag, I do not like them, I try to get away with rupture of the membranes and some times a medical induction; the question of the place of Caesarean Section in the treatment always comes up at this stage of any discussion, I believe that it has a place but the occasion is very rare, certainly the eclamptic is a poor operative risk. I can imagine a fulminating type of eclampsia, in a primigravida, with a long hard cervix and no response to treatment where a section would be indicated and one might get a good result, but I would fear that it was a sort of a long chance. The second condition that warrants a Section is in the presence of obstetrical difficulties, disproportions, etc., that would ordinarily be indications for a Caesarean Section.

In the presence of a convulsion there are several things that are worth remembering, have a mouth gag ready and as all eclamptics should never be left alone, as soon as the convulsion starts she should be protected from injuring her mouth and tongue, often times a little anaesthetic will control the convulsion. The clothing should be loose, and the bed kept against the wall so that the patient will not throw herself out the other side from the attendant. After the convulsion, if the patient remains cyanosed, she should be given oxygen. During the convulsion the head should be kept on one side to avoid inhaling saliva, and after the mouth should be cleaned.

What Is Good Response to Treatment:

In our pre-eclamptics we get a gradual lowering of blood pressure, the oedema disappears, and the urine becomes free of albumen, then the patient can be carried on to term under close observation, or if the symptoms and signs do not clear up well it is wise to induce labor as the foetus may perish if infarction goes on too long in the placenta.

In the eclamptics a good response is indicated by the cessation of fits, ample secretion of urine, good perspiration, lowering of blood pressure and a return to consciousness. Sometimes if the foetus dies, the patient improves rapidly, again all these improvements may occur with the foetal heart still good. I do not believe that it is wise to go on with the pregnancy in these so-called "intercurrent eclampsias", the risk is too great of a recurrence. I believe it is wiser to induce labor.

Now about the prognosis of these cases, in the pre-eclampsias the prognosis depends on the response to treatment, in those cases where the response is good and we believe that it is a true pre-eclampsia the patient may escape scott-free. In those cases where there is a persistence of albumen we probably have a degree of chronic nephritis or at least what Stander called a low reserve kidney, there is some further damage done by each succeeding pregnancy. Accidental haemorrhages occur and the foetal mortality is high. In the fulminating type of eclampsia the prognosis is so often good, they have a better and more complete recovery than the protracted albumenuric type; so many of those will have succeeding pregnancy with no sign of toxaemia. There are many lessons to be learned from a discussion of this type, several points appeal to me:

(1) Many cases of toxaemia can be avoided by proper pre-natal care, which first prevents the appearance of toxic symptoms, secondly recognizes the early signs and symptoms and prevents them going on to a serious stage, and salvages many babies. This is shown by the low incident of cases in private and clinic practice.

(2) No case of pre-eclampsia is to be regarded lightly, every toxaemia is potentially dangerous, we have only imperfect methods of judging the amount of damage being done.

(3) In eclampsia the immediate maternal mortality varies; from 10 to 25%, subsequently develop essential hypertension or chronic nephritis with all their life shortening sequelae.

*"Inoperable" Carcinoma of The Gastro-Intestinal Tract (Exclusive of The Rectum)

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IN reviewing the history of medicine ands urgery of the past two decades one cannot help but be thrilled with the tremendous accomplishments. One disease after another has been made less dangerous, or more easily cured, by the discovery of new, and epoch-making, therapeutic agents. New methods for the control and prevention are, in many instances, reducing the incidence of large and disabling groups of diseases to a minimum. Operations considered impossible fifteen to twenty years ago are now frequently done with complete success. For instance, during the last war, bullets and other foreign bodies were surgically removed from the chambers of the heart, leaving the patient no worse off either as the result of his serious wound or operation. Congenital abnormalities of the heart such as patent Ductus Botali and the more serious Tetralogy of Fallot are now either cured or their symptoms controlled by surgery.

Numerous advances in all fields of medicine and surgery could be enumerated and elaborated upon, but certain fields remain that do not present so pleasing a picture. Carcinoma of the stomach and large bowel remains one of the most discouraging of all surgical problems. A patient suffering from such disease has very little more chance of survival than he would have had twenty or thirty years ago. In one year in the United States approximately 30,000 people die of carcinoma of the stomach and another 27,000 of carcinoma of the large bowel. Carcinoma of the stomach accounts for about 20% of all cancer deaths in the United States and carcinoma of the colon (exclusive of the rectum) accounts for 11% of all cancer deaths.

In Canada there are approximately 4,573 annual deaths from carcinoma of the stomach and large bowel. In Nova Scotia according to the last statistics 277 persons died of carcinoma of the stomach and bowel in one year.

The prognosis is universally bad. Pack, in discussing the subject at a meeting of the American College of Surgeons, pointed out that of 100 patients admitted to hospital with carcinoma of the stomach only 25% are found operable. Of this operable group at least 25% die in the hospital following operation. Of the remaining fifteen patients or so, only four are alive at the end of a five-year period. A four per cent survival rate presents a pretty dismal picture indeed. The figures for the colon are nearly as bad.

It is obvious from the start that the vast majority of patients suffering from malignant disease of the gastro-intestinal tract cannot be *cured* by surgery, but, in my opinion, this does not mean that they cannot be *helped* by it, and this large and unfortunate group of "incurables" should not be labelled "inoperable" and left to die in a rapid and miserable manner. Very often the application of radical surgery for the removal of the primary growth will result in an extended and ever so much happier life. Practically all text books, most physicians, and many surgeons would have us believe that if

*Read at the Annual Meeting of Cumberland County Medical Society, Amherst, September 11, 1946.

definite metastases can be demonstrated, operation of any radical nature is contra-indicated. This I do not believe to be so and a few illustrative cases will be presented later to support this view. For some time now we have been resecting primary malignant growths of the stomach and colon, even with the presence of extensive liver metastasis, with what we consider justifiable results. Carcinoma of the rectum is not included as it presents an entirely different problem from carcinoma of the stomach and colon.

Before proceeding to a discussion of the so-called "inoperable" group, it is perhaps advisable to discuss very briefly carcinoma of the stomach and colon in general.

The unsatisfactory status of gastro-intestinal carcinoma can be attributed to three main causes. Firstly the nature and progress of the lesion itself accounts for the development of many incurable cases. Unfortunately in many cases the growth produces no symptoms whatsoever until extension has occurred. Small non-symptomatic growths may be found in both the stomach and bowel with widespread secondaries. It would seem that little can be done to reduce the number of this group. They would no doubt be missed even with routine periodic health examinations, unless these included careful gastro-intestinal X-ray examinations. Such universal and complete examination is rather a forlorn hope! Secondly in many instances, the patient himself, can be blamed for his own hopeless condition. His symptoms may be so mild, or so slowly progressive, that he does not seek medical aid until it is too late. Intensive lay education along the lines of cancer control would do much to impress upon the public the necessity of seeking early advice concerning vague gastro-intestinal disturbances. Thirdly, the medical profession itself can be directly blamed for many late diagnoses, and this is a very regrettable fact. All too frequently a patient will present himself at a doctor's office with vague and slight gastro-intestinal symptoms only to be told that nothing abnormal is to be found on ordinary physical examination. A diet and some medicine is prescribed and the patient may be told to report back at a future date. Unfortunately, early carcinoma of the gastro-intestinal tract responds symptomatically to dietary and medical treatment for a while. Such improvement reassures both the patient and doctor until hopeless advancement has occurred. No physician has the right to prescribe any treatment for gastro-intestinal symptoms complained of by patients over forty years of age without complete and competent X-ray examinations.

Carcinoma of the gastro-intestinal tract can occur at any age but rarely under thirty years. Sixty-five per cent of cases occur between the ages of fifty and sixty-nine years. It has long been believed that carcinoma develops on, or from, pre-existing gastro-intestinal lesions such as ulcers, gastritis, benign tumors, polyps, etc., but this is far from a proven hypothesis. It is true that cancer cells are sometimes found in the edges of chronic gastric ulcers or in polypoid growths of the colon, but it is equally true that carcinoma may, and probably does, in the majority of instances, develop on sites, not previously affected by disease of any kind.

Time does not permit the discussion of the pathology of gastro-intestinal carcinoma beyond that which is of practical importance to the surgeon. Most carcinomas of the gastro-intestinal tract are adeno-carcinomas of varying degrees of malignancy. In the stomach all types of carcinoma exist from the

rapidly disseminating medullary and colloid ulcerative growths, through the scirrhous, to the least malignant papillary growth. Their degree of malignancy can frequently be rather accurately determined by observation and palpation. In the colon the vast majority of malignant tumors represent some variety of adeno-carcinoma. About two-thirds occur in the left colon and one-third in the right. The tumors are largest in the caecum and become progressively smaller along the large bowel from the caecum to the rectum. Scirrhous obstructing carcinoma occurs more frequently in the left colon than the right and, as a consequence, produces symptoms earlier but are not more amenable to radical cure. Carcinomas of the sigmoid produce metastasis earlier and more frequently than do carcinomas of the caecum.

The spread of a primary cancer may occur in any one or more of five ways, namely: (1) lymphatic emboli to regional lymph nodes; (2) direct extension and infiltration; (3) lymphatic permeation; (4) blood-borne emboli to various parts of the body; (5) transplantation. Demonstrable metastases may be looked for in the liver, left supraclavicular gland, left axillary glands, mediastinum and lungs, inguinal glands, and pelvic peritoneum. A point of practical importance in regard to operative technique is that direct extension of a stomach cancer seldom involves the duodenum, even if low in the pylorus, but often extends directly into the lower oesophagus. The upper limit of resection therefore has to be carefully selected while resection through the upper part of the duodenum is satisfactory.

In regard to symptoms and diagnosis, it may be stated at once that there are no characteristic symptoms of early gastro-intestinal carcinoma. It should also be emphasized that any gastro-intestinal symptoms occurring for the first time in an individual of over forty years of age should be regarded as carcinoma until proven otherwise, and any change in the character of symptoms of the chronic dyspeptic should also be regarded as carcinoma until proven to arise from other cause. Any gastro-intestinal symptoms in any age group deserve proper investigation including competent X-ray opinion before any type of therapy is prescribed or suggested. Considering carcinoma of the stomach and colon as a whole, the average symptomatic duration is about six months and the average time elapsed before hospital admission is one year, thus explaining, in a large measure, the extremely low cure rate.

Often the first symptoms complained of in carcinoma of the stomach are: (1) epigastric discomfort, (2) disturbances of appetite, and (3) slight nausea. The late symptoms are all too well known and need no repetition. They may be due to either the presence of an advanced primary growth or to its metastasis or a combination of both. The earlier symptoms of carcinoma of the bowel result mostly from bowel irritation and disturbed bowel function. Patients may complain of mild pain, diarrohea, or constipation and gas consciousness. Blood or mucus may appear in the stool. The location of the site of pain in one particular quadrant does not necessarily indicate the site of growth is also there. It is not unusual for carcinomas of the splenic flexure to produce pain and some tenderness in the right lower quadrant due to distension of the caecum. Obviously the removal of an appendix (which has been done) under such circumstances does not improve the patient's chances of recovery from his real disease. Again the symptoms of advanced obstructing lesions of the colon are too well known to need further discussion.

The investigation in suspected cases of carcinoma of the gastro-intestinal tract should include (1) complete fluoroscopic and X-ray examination by one competent to do such examination, (2) complete blood studies and (3) complete stool examinations especially repeated examinations for occult blood, (4) gastro-scopic examination when possible, and (5) sigmoidoscopic examinations in all cases where the growth is suspected to be in the lower bowel.

The prognosis of carcinoma of the stomach, as stated above, is poor, there being only about four per cent five-year survivals in those presenting themselves to hospital for treatment. With carcinoma of the colon the survival rate is decidedly better, being about twenty-five per cent five-year cures.

The treatment of early carcinoma with either no metastases, or only regional ones, is obvious, namely radical resection. However, we have all observed, far too frequently, a surgeon open an abdomen, discover a carcinoma, and some large regional lymph nodes, or even distant metastasis, and then pronounce the case inoperable and proceed to close the abdomen or do some palliative, simple operation such as a colostomy. In cases of regional metastasis such procedure is definitely not justifiable and in cases of distant metastasis very doubtfully so. Very large glands may exist in the drainage area of a carcinoma and be only inflammatory in nature. This is particularly true if the growth is ulcerative.

The real problem is when indisputable metastases are found at a distance from the growth, such as deposits in the liver, distant lymph nodes, lungs, etc. These have been pronounced "inoperable" almost universally in the past, but there are many reasons why this opinion may change in the future. We have all seen carcinomas removed without the knowledge of the existence of secondaries. These have been discovered at a later date and yet these patients have lived comfortably for long periods of time, some of them up to ten years. It is also quite true that if it is technically possible to remove a primary carcinoma of the gastro-intestinal tract, in spite of relatively high operative mortality, that patients so operated upon live a great deal longer and ever so much more comfortably than if they are left to die from the disturbed function or obstruction caused by the primary growth. A patient with advanced carcinoma of the stomach may be admitted, anaemic, thin, dehydrated, and vomiting. Liver metastases are obvious from the start. If no operation is attempted, the patient will die in a very short time. If resection is possible, and done, nutrition is again restored. There is an immediate gain in weight and strength and the misery of the primary growth disappears. Such patient may live a normal life up to three years or more and finally die in a relatively comfortable manner.

Unless a patient is obviously near death when admitted, we feel resection of the primary growth is justifiable even in the presence of widespread metastasis, provided this is technically possible.

Statistics of 37 "inoperable" cases presented and slides demonstrating specific cases.

1. Total number of cases of gastro-intestinal carcinoma with clinical or operative demonstration of metastasis (1940-1946)-37

Stomach	18 or	48.6%
Bowel	19 or	51.4%
	37 or	100.0%

2. Procedure in total group of 37:

1. No operation	11 or	29.7%
2. Laporotomy only	5 or	13.5%
3. Colostomy only	5 or	13.5%
4. Resection	16 or	43.2%
Total	37 or	100.0%

3. Results in total cases with demonstrable metastasis.....(37)

Died in hospital	7 or	18.9%
Lived under 6 months	12 or	32.4%
Lived 6 months to 1 yr.	4 or	10.8%
Lived 1 to 3 yrs	12 or	32.4%
Unknown	2 or	5.4%
Total	37 or	100.0%

Note: Of the 12 that died under 6 months 11 were so far advanced that no operation advised or carried out.
Of this group 9 or 24.3% are still alive and well.

4. Long time survivals from resection (1 to 3 years).
Stomachs resected.....18
4 have lived 1 to 3 years or 22.2%.
Bowels resected.....19
8 have lived 1 to 3 years or 42.1%.

Note: This group experienced markedly improved health and were *free of pain* for the survival term.

5. Number of cases still alive and free of pain and distressing symptoms..... 9 or 24.3%

Case Examples

(1) *Mrs. N. C., age* 44—1081.
Complaints: 1. Epigastric distress—not related to meals.
2. Loss of appetite.
3. Weakness.
4. Nausea.
5. Loss of weight—30 lbs.

History of one and a half years.
Physical Findings: 1. Emaciation.
2. Dehydration.
3. Anaemia.
4. Hard tender mass in the epigastric region.

X-ray—carcinoma stomach.

Metastasis: 1. Direct extension gastro-hepatic omentum.
2. Superior glands.

Operation: *Resection—Total*: Two carcinomas found; one pyloric scirrhous; one fundal ulcerative of spheroidal cell type.

Response to Operation: Excellent. All symptoms disappeared. Marked gain in weight and strength. Lived a little over two years. Died of mediastinal metastasis.

(2) *E. V., age* 49—8844.

Complaints: 1. Abdominal pain.
2. Loss of strength.
3. Loss of weight—15 lbs.
4. Nausea.

History: Periods of "indigestion" for years. Epigastric distress 1½ hours to 2 hours p.c. relieved by food and soda. Change in character and severity two months previously. Constant pain and nausea.

Physical Findings: 1. Anaemia.
2. Evidence of loss of weight.
3. Hard tender mass in epigastric region.

X-ray: Carcinoma stomach.

Metastasis: Glands of lesser omentum and around aorta.

Operation: *Gastric resection*: Large mucoid carcinoma, lower half of stomach involving all coats. Stomach wall ½" thick.

Response to Operations: Excellent. No symptoms. Gained weight and strength. Still alive one year after operation and working every day as a stationary fireman.

(3) *H. T., age* 73—6638.

Complaints: 1. Vomiting.
2. Loss of weight and strength.
3. Pain in epigastric region.

History: One week.

Physical Findings: 1. Anaemia.
2. Dehydration.
3. Evidence of loss of weight.
4. Marked distension upper abdomen.
5. Marked tenderness in upper abdomen.

X-ray: Carcinoma first part of jejunum—obstruction.

Metastasis: 1. Retroperitoneal glands.
2. Liver (rather extensive).

Operation: Resection scirrhous adeno-carcinoma.

Response to Operation: Excellent. Alive 2½ years after operation. Active and has no symptoms.

(4) *E. S., age* 58—5039.

Complaints: 1. Abdominal pain.
2. Vomiting.
3. Loss of weight and strength.

History: Years of epigastric pain after eating. Change of symptoms of eight months' duration.

Physical Findings: 1. Anaemia.
2. Loss of weight.
3. Dehydrated.
4. Tender hard mass in epigastric region.

X-ray: Obstructing carcinoma stomach.

Metastasis: (1) Direct extension on both the lesser and greater omentum.
(2) Mesenteric and retroperitoneal glands.

Operation: *Resection*: Healed ulcer at pyloric ring. Adeno-carcinoma of whole lower half of the stomach.

Response to Operation: Good. No symptoms. Gained weight and strength and returned to a full day's work as a farmer which he continued for 18 months following operation. Suddenly developed obstructive jaundice and died within two weeks.

(5) *H. V.*—5788.

Complaints: 1. Abdominal pain.
2. Nausea and vomiting.
3. Loss of weight and strength.

History: Two years.

Physical Findings: 1. Anaemia.
2. Loss of weight.
3. Hard mass in epigastric region.

Metastasis: (1) Direct invasion of lower oesophagus.
(2) Regional glands.

Operation: Laparotomy only.

Result: Died in a few weeks. Requested not to attempt removal unless cure possible. Case no less operable than others cited.

(6) *Mrs. G. O. L., age* 62—6095.

Complaints: 1. Pain in right lower abdomen.
2. Loss of weight.
3. Periods of diarrhoea.

History: Six months.

Physical Findings: 1. Slight anaemia.
2. Occult blood.
3. Tenderness in R. L. Q.

X-ray: Carcinoma caecum.

Metastasis: Mesenteric and retroperitoneal glands.

Operation: *Resection*: Papillary adeno-carcinoma.

Result of Operation: Excellent. No symptoms. Alive and well nearly three years after operation.

(7) *A. M. F.*

Complaints: 1. Increasing constipation.....3 months
2. Abdominal cramps.....2 weeks
3. Abdominal distension.....2 weeks
4. Vomiting.....1 week

History: Three months.
Physical Findings: Abdominal distension—general.
X-ray: Flat plates. Large bowel obstruction complete splenic flexure.
Metastasis: Mesenteric.
Operation: (1) Decompression (caecostomy).
(2) Resection (scirrhous adeno-carcinoma).
Result: Excellent, but too recent to evaluate (under one year). Returning to usual occupation.

(8) *J. K., age* 66—6045.

Complaints: 1. Loss of weight and strength.
2. Pain in the abdomen.
3. Frequency and nocturia.
4. Urinary obstruction.

History: Urinary symptoms for several years. Abdominal symptoms one year.
Physical Findings: 1. Anaemia.
2. Loss of weight.
3. Mass in epigastric region.
4. Large hard nodular prostate with lateral infiltration.

X-ray: Carcinoma of the stomach—pylorus.
Operation: None on account of presence of carcinoma prostate.
Result: Died in about six weeks from continued vomiting.
Comment: Stomach growth probably resectable. Prostatic growth might have been controlled by castration and stilbesterol (therapy unknown at time of investigation).

(9) *Mrs. J. C., age* 68—7647.

Complaints: 1. Increasing constipation.
2. Abdominal pain.
3. Distension of the abdomen.

History: Several months.
Physical Findings: 1. Anaemia.
2. Loss of weight.
3. Occult blood.
4. Abdominal distension.
5. Hard mass in epigastrium.

X-ray: Carcinoma—hepatic flexure.
Metastasis: Mesenteric nodes and retroperitoneal glands.
Operation: *Resection*: Adeno-carcinoma scirrhous.
Result of Operation: Excellent. No symptoms. Alive and well 21 months following operation.

Fat Absorption and Lipemia

C. B. WELD*

THE mechanism of the absorption of fat from the intestinal canal is still far from clear despite intensive investigation. The classical view has been that hydrolysis of fat has been a necessary preliminary to absorption, and this view is still the only one presented in most texts of physiology. According to this view the generally accepted sequence of events is something as follows. The fat is split in the small intestine into fatty acids and glycerine which are absorbed into the mucosal cells where they are resynthetized into neutral fat. This is passed through into the lacteals and carried in the lymph to the systemic blood. Despite several gaps in our knowledge this concept seems to be well supported by facts. There is no doubt than fat is digested in the small intestine, that globules of neutral fat are found in the mucosal cells [1] and that these are passed through to the lymphatics. Furthermore there is histological evidence that the earliest globules found in the mucosal cells during fat absorption are fatty acids whereas later they are neutral fat.[2] However it has been pointed out that even if one accepts this not too certain histological evidence without reservation, it still does not prove that resynthesis of fat has occurred; it could be a mere matter of timing, absorption first of fatty acid and later of fat. Not much attention has been paid to this possible interpretation because of the firmly ingrained belief that fat had to be hydrolysed before absorption, a belief well grounded in the common knowledge that if there is no fat digestion there is no absorption. Yet, recent evidence has made it clear that it is possible for undigested fat to be absorbed. If it is first emulsified to a particle size of less than one micron, even paraffin is readily absorbed.[3] There is an apparent paradox to be resolved.

Frazer and his co-workers in England have been publishing evidence and arguments for the last few years in support of their theory that fat is chiefly absorbed in the undigested emulsified state.[3] These workers have examined the emulsifying capabilities of a large number of substances which might be present in the intestinal lumen and pointed out that any of these to be of physiological significance would have to be effective in a slightly acid medium because the normal upper intestinal contents are usually neutral or acid in reaction. This is important as many substances are useful emulsifying agents in alkaline media, probably aided by soap formation, which are quite ineffective in acid media. An acceptable dispersing agent would have to cause the fat to be rapidly and spontaneously emulsified to a particle size of less than 1 micron even at a pH of 6.5. No single emulsifying agent was found with these qualifications and indeed only one combination of substance of physiological significance was found which did have these properties. This was a combination of (a) bile salts, (b) fatty acid, and (c) a monoglyceride. The source of the monoglyceride and the fatty acid in the upper intestine would lie in the partial digestion of fat.

This gives us the basis of Frazer's argument. Hydrolysis of some fat is admittedly necessary but instead of being an essential preliminary to absorption, in Frazer's view, its chief function is to provide monoglyceride and fatty acid, which then, together with bile, rapidly emulsifies the rest of the

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neutral fat to such a small particle size that it is readily absorbed without hydrolysis. In corroboration of this thesis Frazer points out (a) That fat is emulsified in the upper small intestine to this degree of dispersion. (b) That though the initial stages of lipolysis may be rapid, the rate soon declines and almost comes to a stop before 30 per cent of the contained fatty acids are liberated.[3] (c)That even if fat were completely hydrolysed there is not enough bile salt in the intestine to bring about solution of all the fatty acid.[4] (d) That despite a seeming insufficiency of lipase and bile, fat absorption is rapid and complete. Furthermore he shows that the route of absorption of unhydrolysed fat may differ from that of fatty acid [5] and that the visible opacity of the, plasma which develops after a fat meal and which we call alimentary lipemia, is due to neutral fat only. [6] Some of these points will be elaborated later but on the basis of the above it seems clear that Frazer's view has much to commend it. We are more likely to contest the quantitative aspects of the theory than its essential correctness. The classical conception that any fat to be absorbed must first be hydrolysed, seems disproven. On the other hand, Frazer's demonstration that the absorption of emulsified neutral fat is a common process, does not therefore make it the only method of absorption. The two methods almost certainly operate together and the relative importance of the two will probably not be constant.

Lovatt-Evans [7] gives a modified view of the classical conception. He reports that for the rapid absorption of fat three factors are essential, (a) the fat must be hydrolysed, at least in part, (b) bile salts must be present, and (c) phosphatase must be present. Note that he adds the phrase, "at least in part" to the need for hydrolysis. This is a concession to Frazer's evidence which has already been discussed in part. The need of bile is well recognized. In its absence absorption of fat does not occur. Bile may facilitate the digestion of fat but even if fat is hydrolysed, absorption fails in the absence of bile. One reason for this is the ability of bile to dissolve fatty acids, the hydrotropic action of bile. A 5 p.c. solution of bile salt is capable of dissolving 0.12 p.c oleic acid. In the presence of 0.8 p.c. lecithin the solubility of oleic acid is raised to 4.5 p.c.[7] This is a considerable improvement but it is still not enough to account for the rapid and complete absorption of fatty materials which actually takes place. The importance of phosphatase in the intestinal mucosa is shown by the almost complete cessation of fat absorption when the phosphatase is poisoned by iodoacetic acid. The enzyme is shown by radioactive phosphorus experiments[10] to cause phospholipid formation in the intestinal mucosa and elsewhere. Some of the fatty acids may thus be absorbed as phospholipid directly.

No matter which form the fatty material is in during absorption the mechanism of the absorption is obscure. During the absorptive period, in the gut lumen there is a mixture of globules ranging in size from the coarse to the ultramicroscopic, consisting of both neutral fat and a variety of fatty acids. Some of the fatty acid may be brought into true aqueous solution, possibly with the aid of bile or possibly as a phospholipid, and be absorbed without difficulty. Such material may be said to be in a state of molecular dispersion. As for the rest, even if emulsified to a particle size of only a fraction of a micron, each particle consists of vast numbers of molecules and is of such a size that it is hard to understand how it can get into the mucosal cell and,

once in, how it gets out again. That it does is indisputable; especially prepared histological sections of the mucosa taken during fat absorption show minute globules of lipid material within the cell.[8] None are seen in the outer margin of the cell but the presence of a "Brush Border," with fine radial lines in this region, suggests canaliculi to some workers. Within this margin the globules appear and as they move towards the back of the cell they tend to coalesce, and may become quite large. They can also be seen to become extruded through the base of the cell and pass into the lacteal. The pumping action of the intestinal villi probably assists in this process and propels the particles along. The particles do not appear to pass between the cells and the old theory that they might be carried by the lymph cells has been discarded.

These minute particles or globules of chyle then pass to the systemic blood and in the plasma they are called chylomicrons and appear under dark field illumination as tiny bright specks a fraction of a micron in diameter. They may be counted and the doing of serial counts on the serum after a standard test meal is known as a "Chylomicrograph."[9] When lipemia is not marked the chylomicrograph method is perhaps the most practical for following changes in the degree of lipemia. If precise counts are to be made the technique is exacting but quite rapid when mastered; if only rough estimates are needed, it is relatively easy. If the lipemia is marked, simple photometric measurement of the opacity of the serum is much easier and probably conveys the same information, especially if a quick check is made with the microscope of the type of particles present.

During the period of fat absorption, chemical analyses show increases in the plasma of neutral fat and phospholipids chiefly and of cholesterids to a lesser degree. It is only the neutral fat fraction which forms the chylomicrons and causes the opacity or milkiness of the plasma.

Fat emulsions, even those dispersed to a particle size of 0.5 micron are unstable unless they are protected by a soap or protein molecular film at the oil-water interface. The emulsion in the intestinal lumen seems to be of this type; at any rate it is readily broken down by protein precipitants and not by fat solvents. On the other hand the chylomicrons in the plasma and lymph are more resistant to protein precipitants but they are destroyed by a lecithinase such as perfringens toxin. It has been shown that the incorporation of a trace of phospholipid in the surface film of the oil-water interface will cause just this change.[3] Hence it is surmised that during absorption a trace of phospholipid is added to the protecting film surrounding the fat particle. The importance of phosphatase in the intestinal wall in fat absorption has already been mentioned; this is another clue to its full action. It has long been known that after double adrenalectomy fat absorption is greatly curtailed and it has been suggested that failure of phosphorylation in the intestinal cell is the cause. Direct proof has been given however in the rat, using the radioactive isotope P^{32}, that phosphorylation is not interrupted by adrenalectomy.[10] Frazer reports ([3]) that adrenalectomy greatly reduces the absorption of neutral fat, the absorption of fatty acid being practically normal. The absorption is largely restored by salt administration and the disturbance is probably due to an ionic imbalance rather than to an abnormal phosphorylation mechanism.

Frazer has also by his chylomicrograph technique, reopened the question of absorption into the portal system.[5] It has long been known that the arithmetic of fat absorption is unbalanced. The amount of fat recovered from the thoracic duct is never more than 60 p.c. of that absorbed from the intestines, and it may be much less than this. The alternative route has never been found; the portal route has been denied by many workers on the basis of chemical methods. However the expected changes would not be quantitatively great and accurate measurement of portal blood flow with concurrent fat estimations are difficult to obtain under physiological conditions. Hence the results cannot be quite conclusive. Frazer reports that with a small needle, repeated small specimens of blood may be taken from the portal vein for chylomicron counts, without physiological disturbance to the blood flow. He finds that when a stained (Sudanized) fat is fed, there is a marked systemic lipemia and the stained fat enters the fat depots. If oleic acid is fed this does not occur but the portal blood shows a chylomicron increase and the liver becomes filled with fat. Furthermore these workers point out that a systemic alimentary lipemia can be prevented or stopped by feeding extra lipase.[11] They conclude that ordinary lipemia is the result of the absorption of emulsified neutral fat by the lymph route while fatty acid is absorbed into the portal blood. Their results are impressive and it seems reasonable to assume their general validity. However there are a variety of fatty acids and it is not yet proven that all pass equally easily into the portal blood; it may be that the distinction between the two routes of absorption is not as sharp as they suggest.

To summarize, it seems to be necessary to revise the old idea that fat must be digested to fatty acid and glycerine as a preliminary to absorption. Some at least, and perhaps the major portion, may be absorbed as neutral fat in a finely emulsified state. However, in order to produce this finely emulsified condition some fat hydrolysis is necessary. Some clinical implications are evident. Biliary disturbances with loss of bile salts would greatly curtail absorption of either fatty acid or fat. Pancreatic lesions on the other hand would have little effect as long as bile could still enter the duodenum. Even with complete loss of pancreatic lipase there might well be enough lipase of gastric or intestinal origin to initiate the emulsification process and allow a large measure of fat absorption. The alternative routes of absorption, and the phosphorylation mechanisms, and the relation of the adrenals, and the use of the chylomicrograph, also are fruitful fields for speculation and further study.

The lipemic state, however produced, is also interesting from another point of view. It is a finely divided emulsion of negatively charged particles and it is an active adsorbing agent.[12] For example, it adsorbs insulin and reduces its hypoglycemic action, it reduces agglutinin titres by about one half, and it also detoxicates useful amounts of diphtheria toxin, tetanus toxin, cobra venom and other poisons by adsorption.

None of the foregoing discussion has touched on the great field of the intermediary metabolism of fat, its deposition and its mobilization and transfer from one part of the body to another. Yet, lipemia can be produced by toxic agents affecting the liver, and sometimes by starvation. Hence though there is no place in this short review of fat absorption to cover these wider problems, they should be mentioned. Only one aspect will be stressed. All modern

studies with tagged elements and molecules such as deuterium and radioactive isotopes have shown the great mobility of the body fat. Absorbed fat is deposited in the depots in much the same form as the ingested fat. Then, during the next few days there is a redistribution of the fatty acid components so that soon the fat in the depots has the characteristics of the host species. Even after this state is reached there is a continual deposition and dissolution of the fat molecules.[13] It is a mistake to consider the fat to be parked in storage against some distant need. It provides a mechanism for a minute to minute adjustment in metabolic needs and is in a continual state of flux. Indeed it is calculated that in mice under static weight conditions there is a change over of one half the depot fat every week.[14]

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Society Meetings

The Pictou County Medical Society

The Pictou County Medical Society held a dinner on December 11 at the Norfolk Hotel, New Glasgow. The guest speaker was Dr. Eric MacDonald, Glace Bay, who gave an informative talk on carcinoma of the large bowel.

W. A. MacQuarrie
Secretary-Treasurer

The Colchester East Hants Medical Society

The semi-annual meeting of the Colchester East Hants Medical Society was held in Truro on Dec. 6th. Dinner was served at 6.30 following which a professional program was enjoyed by fifteen of the medical men. They were as follows, Dr. D. F. MacInnis, Shubenacadie; Dr. H. B. Havey, Stewiacke; Dr. R. A. McLellan, Rawdon; Dr. Daniel Murray, Tatamagouche; Dr. J. B. Reid, Dr. S. G. MacKenzie, Dr. H. R. Peel, Dr. P. R. Little, Dr. H. R. McKean, Dr. E. M. Curtis, Dr. T. C. C. Sodero, Dr. J. A. Muir, Dr. D. S. McCurdy, Dr. R. F. Ross, and Dr. J. B. Reid, Jr., all of Truro.

Dr. D. F. MacInnis, President, introduced the special speaker of the evening, Dr. E. F. Ross, F.R.C.S., Halifax, who spoke on fractures of the leg. Dr. Ross drew from his extensive army experience overseas and presented his subject in an interesting, practical way under the following headings.

1. Plaster immobilization with or without padding. Sometimes the fragments slip in plaster and so 5 to 10 pounds extension is necessary with the plaster. Padding under the plaster is necessary until all swelling has subsided, but if padding is not used, the patient must be under constant supervision to detect pressure.

2. Kirschner wire.

3. Roger Anderson splint—the popularity of this splint is declining as in the army non-union and infection developed.

4. Straider splint—which is not being used much to-day.

5. Metal appliances—plates, and screws without plates.

6. Compound fractures—after debridement a member of these wounds may be closed. Dirty wounds should be packed widely open—put open plaster and in 5 to 7 days through an open window in the plaster a secondary suture operation is done to close the wound. Screws and plates should not be used unless the wound is considered clean.

Following a discussion on leg fractures Dr. D. S. McCurdy, of Truro, showed moving pictures of inguinal hernia, anatomy and surgical procedure. This method of teaching is pleasant and efficient and is appreciated by groups as part of a program.

D. S. McCurdy
Secretary-Treasurer

Correspondence

135 St. Clair Avenue West
Toronto 5, December 31, 1946

TO THE SECRETARIES OF DIVISIONS

Dear Doctor:

Re $20,000 Grant from D.V.A.

It will be recalled that, in June, 1945, under P.C. 3999, the Department of Veterans Affairs made a grant of $20,000.00 to the C.M.A. to be used in a "counselling service to returning Medical Officers." Inasmuch as this service was carried on by the respective Divisions on a voluntary basis, it was not found necessary to utilize any of the grant and, therefore, on December 19th, the Association returned the full amount to the Government.

We have received an acknowledgment from the Minister of the Department of Veterans Affairs, the Honourable Ian Mackenzie, a copy of which is enclosed for your information.

Yours sincerely
T. C. ROUTLEY
General Secretary

DEPARTMENT OF VETERANS AFFAIRS

Office of the Minister
OTTAWA
December 27, 1946

Dr. T. C. Routley
General Secretary
Canadian Medical Association
135 St. Clair Avenue West
Toronto 5, Ontario
My dear Dr. Routley:

Mr. Woods, my Deputy, has referred to me your letter of December 19th with which you returned a cheque in the sum of $20,000.00, constituting a refund of an amount voted to your Association under P.C. 3999 in June, 1945.

You state that the services for which this sum was intended, namely, to provide honoraria to members of your Association who rendered rehabilitation advice to discharged physicians from the Forces, have been donated gratuitously. May I say how deeply I appreciate this action on your part. It is in the highest tradition of your profession that the members of your Association would prefer to give their services to those who served us so well during the war years.

On the next appropriate occasion I would appreciate it if you would extend my deep appreciation to your members for this cooperation and their splendid contribution.

Wishing you the Season's Greetings, I remain,

Sincerely yours
"IAN MACKENZIE"

Personal Interest Notes

Dr. John F. Quinlan, a member of the medical staff of the Nova Scotia Sanatorium since May 15, 1941, has been appointed assistant medical superintendent according to an order-in-council dated December 5. Dr. Quinlan succeeds Dr. J. Earle Hiltz who was granted leave in July, 1944, to take over the duties of acting superintendent of the Victoria General Hospital, Halifax, and who was later appointed superintendent of Roseway Hospital, Shelburne.

Dr. Henry Kenneth Hall, until recently assistant in psychiatry at the Dalhousie Medical School, is in New York beginning a three-year course in psychiatry at the Payne-Whitney Psychiatric Clinic in the New York Hospital of Columbia University. After graduating from Dalhousie in 1944, Dr. Hall served with the R.C.A.M.C., holding the rank of captain until his discharge in January, 1946. He then joined the staff of the Medical School, and also gave part of his time to the service of the Nova Scotia Hospital.

Dr. Miller Ballem, son of Dr. J. C. and Mrs. Ballem, New Glasgow, was listed by McGill University, in recent appointments in their medical school, as being appointed Bristol-Myers Research Fellow. Dr. Ballem is a graduate of Dalhousie and following his graduation enlisted with the R.C.A.M.C. He served overseas for several years.

Dr. Sidney Gilchrist has left to return to his missionary work in Portuguese West Africa after an absence of five years, part of which time he served overseas in Italy.

Deadline

May 1, 1947, is the deadline for entering the $34,000 prize art contest on the special subject of "Courage and Devotion Beyond the Call of Duty" (on the part of physicians in war and in peace). This contest is open to all M.D.'s in the Western Hemisphere. The exhibition will take place in conjunction with the A.M.A. Centennial Session at Atlantic City, June 9-13, 1947. For complete information, write or wire now to Francis H. Redewill, M.D., Secretary, American Physicians Art Association, Flood Building, San Francisco, California, or to the sponsor, Mead Johnson & Company, Evansville 21, Ind., U. S. A.

Obituary

DR. CHARLES FENWICK WYLDE, a native of Halifax and for many years a prominent member of the medical profession in Montreal, died at his Montreal home, Crescent Street, on Nov. 24, 1946 in his 80th year. Dr. Wylde, who had a distinguished record as a doctor and as a soldier, was well known in Nova Scotia. In the early part of his career he practised in Westville and for many years spent his summers in Chester.

Dr. Wylde, who was the son of the late Charles J. and Mary Wylde of Halifax, is survived by his wife, the former Kate Napier Budden and by a daughter, Kathleen. A son, Napier, who was a veteran of the First Great War, predeceased him in 1928. Dr. Wylde was born in Halifax in 1867 and received his early education here. He went to Montreal to study medicine at McGill

University and he graduated in 1888 before he had reached his 21st birthday. After graduation he worked for the C. P. R. near North Bay, Ont. for a short time, and then returned to Nova Scotia where he practised at Westville for several years.

He went to Montreal in 1895 and had since been a resident of that city.

Dr. Wylde represented the Montreal district at the coronation of King George V. On the outbreak of the First World War he volunteered for service and went overseas with the 1st Contingent. He became commanding officer of the No. 1 Canadian General Hospital and later Assistant Director of Medical Services in the London area. He was awarded the C. B. at the end of hostilities, the highest award made to a Canadian medical officer.

At the outset of the Second World War, Dr. Wylde again volunteered his services and was appointed chairman of the medical board of his district.

In his early years of professional activity he was very interested in children and was physician to the Montreal Foundling Hospital. Later he was out-patient physician to the Montreal General Hospital and demonstrator in medicine at McGill University. He was also for many years the librarian of the medical library at McGill.

The BULLETIN extends sympathy to Dr. H. A. Payzant of Dartmouth on the death of his father, L. A. Payzant, which occurred December 9, 1946.

NATIONAL RESEARCH COUNCIL (CANADA)

Division of Medical Research

GRADUATE MEDICAL RESEARCH FELLOWSHIPS

The National Research Council offers a limited number of Fellowships for promising graduates in Medicine who wish to devote themselves to an academic career in medical teaching and research in Canada.

The Fellowships are designed to provide a mental discipline in research and to enable those showing an aptitude for research and experience in one of the basic medical sciences necessary to qualify them for medical teaching and research. Fellowships will not be awarded to graduates in Medicine for the purpose of providing practical training and experience in the clinical branches of Medicine.

Fellowships of the value of $1,000.00 to $1,200.00 per annum will be open to graduates in Medicine with high distinction in scientific study after completion of one year in post-graduate training.

Fellowships of the value $1,500.00 to $2,400.00 per annum will be open to graduates in medicine who have had experience in research work and have shown distinct evidence of capacity for original research. The value of the Fellowship will depend on the candidate's past record in research and the evidence given of capacity to conduct independent research.

In general, the conditions relating to qualifications, the submission of applications and the conditions governing tenure of these awards, will be found in the regulations governing Medical Research Fellowships awarded by the National Research Council.

Copies of the above regulations and application forms, may be obtained from the Dean of the Faculty of Medicine, or upon application to the General Secretary, National Research Council, Ottawa.

March 1st is the Final Date on Which Application May be Made.

S. P. EAGLESON,
General Secretary,
National Research Council.