

*Some Clinical Aspects of Myocardial Infarction and Angina Pectoris

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WITH the introduction of the electrocardiographic examination in different types of myocardial lesion and the straight comparison of these findings with the pathological results, the problem of angina pectoris has been greatly deepened and new aspects of this important syndrome-complex came into view. These culminate in the recognition of the importance of the coronary circulation for the pathogenesis of angina pectoris; for the modern concept of angina pectoris does not stop at the pathological alterations of the coronary arteries alone, because it stresses the functional consequences of such lesions more than the extent of the anatomical alterations of these vessels.

While in normal hearts the coronary arteries may be considered to be terminal (or end-) arteries—almost from a functional viewpoint—we find that in hearts with sclerotic alterations of the coronary arteries, a vast collateral circulation has developed as a result of the obstruction of the normal coronary arterial blood flow. The collateral circulation assumes thus a compensatory function for the ordinary requirements of life.

If this collateral circulation has not developed sufficiently angina pectoris may occur where only minor anatomical alterations of the coronary arteries are found, as angina pectoris may not be present in individuals in whom occlusion of one or even more coronary arteries were found post-mortem, provided that there is a vast collateral circulation.

Wherever conditions arise which increase the demands on the myocardium, the collateral circulation may no longer be able to maintain an adequate blood supply, thus creating a disproportion between the supply and demand of the heart, causing an attack of angina pectoris. The intervention of a spasm of a coronary artery is therefore not absolutely necessary in the mechanism of the production of an anginal attack, since impaired coronary blood flow is proved to be sufficient to cause ischemia or anoxemia, which is considered to be the cause of an anginal attack. This theory has received substantial support by several recent anatomical and experimental investigations.

Angina pectoris and myocardial infarction (or coronary thrombosis—we shall use the more scientific expression “myocardial infarction”—) have many aspects in common. Attacks of angina pectoris often precede an onset of acute myocardial infarction, or anginal pain appears for the first time after such an attack. Pain is almost by definition a part of the syndrome-complex of angina pectoris and is one of the most striking symptoms of acute myocardial infarction, too. But, while we cannot speak of angina pectoris where cardiac pain is absent, we do so in acute myocardial infarction, because the diagnosis of acute myocardial infarction is made from many other clinical manifestations which are symptomatic of this accident. On the basis of the latter, not only

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was the atypical localization of pain recognized to be associated with the clinical feature, but we know also of a great number of cases in which coronary occlusion occurs without pain, and yet a positive diagnosis of such forms can be made.

The clinical aspects of myocardial infarction are so manifold as to call for a critical examination and corresponding classification.

In dealing with coronary thrombosis no distinction was made between coronary thrombosis and *acute* coronary thrombosis or occlusion, and even in the face of the atypical forms of myocardial infarction our attention is directed only to the different atypical symptoms, which may accompany this feature, and not whether these cases are of an acute onset or not. Yet, sometimes, an accidentally taken electrocardiographic or a post-mortem examination may detect in an individual the presence of a myocardial infarction or necrosis for which the history of this case does not give any symptom of a major coronary involvement to be accounted for. Our clinical opinion seemed to be satisfied with the explanation usually given for such cases, namely that the area affected by the infarction was a less vital, a less sensitive one and that therefore myocardial infarction might have occurred without any alarming onset and hence passed unobserved. But when we consider the anatomico-pathological findings in such cases we must admit that such areas often have the same localization of infarcted areas in which the accident has occurred with all the characteristic symptoms, including crushing substernal pain. The question which arises therefore is a double one:

1. What is the mechanism of production of the myocardial infarction in cases of this latter type? And
2. Do these onsets really occur so silently that an early positive diagnosis cannot be made?

Some clinical observations of my own and some new physio-pathological aspects arising mainly from important anatomical observations of Blumgart et al.¹ were the starting point for a new classification of the different forms of myocardial infarction or necrosis recently proposed by myself.² As a result of this classification some clinical and laboratory data are stressed which allow us to separate and describe a new aspect of this important feature of myocardial infarction.

The different forms of myocardial infarction can therefore be classified as follows:

1. A typical form of acute myocardial infarction, with characteristic symptoms;
2. An atypical form of acute myocardial infarction, without all the characteristic symptoms, yet with an acute onset.

Both forms have one aspect in common, namely an alarming and sudden beginning, occurring mostly when at rest.

In addition to these two forms a third one is to be added which is also:

3. An atypical form of myocardial infarction which is not only atypical insofar as its symptoms are concerned, but also in that myocardial infarction or necrosis develops gradually, unlike the other acute forms, and which deserves to be treated as an entity by itself and called "Subacute myocardial infarction or necrosis."

Form 1. With regard to the first form, briefly it may be stated that the most characteristic symptom to attract our attention is a crushing or squeezing sensation of pain over the precordium, accompanied by symptoms of shock and profuse sweating. These symptoms alone are so striking that the diagnosis of acute myocardial infarction can be made by their presence alone, but usually other signs, symptomatic of this accident, are to be found. They are: sudden fall in blood pressure, fever, leucocytosis, increased sedimentation rate, etc.—The electrocardiogram shows, during the early hours after the attack, characteristic changes of the ventricular complex, first described by Pardee.

Form 2. Under the atypical form of acute myocardial infarction we include:

- a. All the cases with atypical localization of cardiac pain, e.g. abdominal form of acute myocardial infarction;
- b. The cases in which cardiac pain is absent or overshadowed by other more striking symptoms; likewise those cases in which a sudden manifestation of nausea with prolonged vomiting occurs, to simulate acute gastritis or acute intestinal obstruction. In these cases, distress over the ensiform is also present sometimes (S. Levine).

The importance which the electrocardiographic examination assumes in the differential diagnosis of cases of this type with an acute abdomen, stands out clearly.

- c. Cardiac pain is found absent in some cases of diabetes. In all these cases symptoms of collapse or shock are always associated with the accident;
- d. In this form we must further include a very important group of cases; so much so, that the statistics of some authors have reached an incidence of approximately forty per cent. These are the cases commonly described as "Coronary thrombosis without pain." Here cardiac pain is considered to be lacking completely, but from the description of a great number of the reported cases, the acute onset of the accident cannot be overlooked. N.S. Davis³, for instance, describes this form characterized "by an abrupt onset of dyspnoea and heart-failure unprovoked by exertion." Cases of subacute myocardial infarction have also been included in this group, from which they deserve to be separated.

Form 3. In accordance with its mechanism of production², the clinical picture of subacute myocardial infarction or necrosis will not show an acute onset like the two forms first described, in which sudden obstruction of the coronary arterial blood flow is responsible and no shock will be manifested. The symptoms will therefore by no means be characteristic. Cardiac pain is often lacking and when present, its character is far from that observed in the acute forms. No definite symptoms can be described. Among the symptoms suspicious of this occurrence I wish to mention: restlessness that develops unexpectedly in cases with manifest signs of heart failure, sweating and weakness accompanied sometimes by fever of unknown origin, or substernal pain of short duration but peculiar in its sensation so much so as to attract our attention to a major coronary involvement. The importance of a properly taken history cannot be stressed enough.

If an electrocardiogram is made in such cases, the first pattern may be even of normal appearance and only successively registered tracings will show definite changes of the ventricular complex of the acute coronary type. In the successive electrocardiograms only minimal alteration may be visible which should be interpreted in the light of the further developments, for the definite changes may not be manifested earlier than five to eight days after the first symptoms were observed by the patient. Besides these delayed changes of the electrocardiogram, a raised leucocyte count is found to be present in most of the cases and may be the only positive evidence of the occurrence where an electrocardiographic examination is not available. Early recognition of this form is of great practical importance, for it may prevent the development of the infarction or almost limit it.

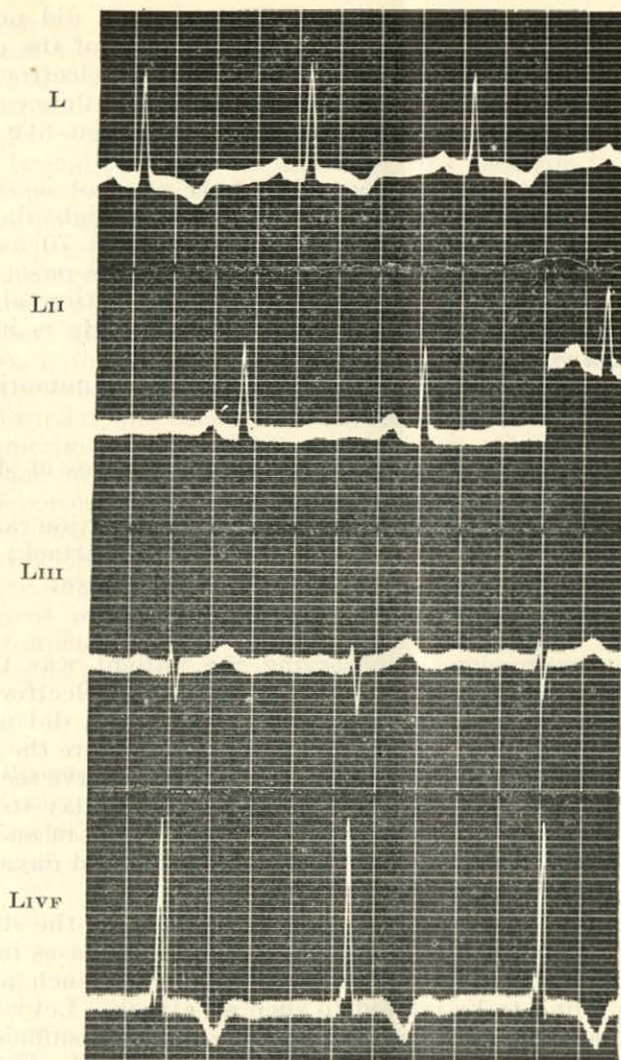
In this connection it remains still to consider the clinical significance of cardiac pain in angina pectoris and myocardial infarction. From the above discussions it is clear that angina pectoris is caused by myocardial anoxemia of a certain area of the heart which on account of its anatomical conditions has become insufficiently irrigated. Cardiac pain would therefore be an expression of the reaction of a partially exhausted myocardium. This means that the pain in angina pectoris would assume a biological significance preventing the ischemic area from undergoing necrosis. In effort-angina, e.g., pain which occurs while walking obliges the individual to rest and so allows the demands on the myocardium to be quickly reduced. In those cases in which pain is found not to be associated with conditions that render the coronary circulation insufficient for the needs of the heart during increased work, namely where myocardial necrosis occurs without the symptom of pain, it is evident that this condition may become fatal to the individual.

The symptom "pain" is therefore in itself not sufficient to indicate the severity of the case. In this regard it has to be borne in mind that while in cases of myocardial infarction pain may be lacking, thereby hiding a more serious accident, there are on the other hand, some cases in which severe and prolonged pain, even with an alarming aspect, may appear, yet without acute myocardial infarction.

The following observation will illustrate this: It concerns a man with hypertensive heart-disease (Blood pressure: 195:135) and a history of anginal pain localized at the precordium and radiating into the left shoulder. The attacks occurred mostly after walking and excitements. He is a man of 50 years of age, of strong bony and muscular constitution, and has been suffering from these anginal symptoms for the last four years.

Of the objective findings I shall mention only that he has an enlarged heart, a systolic apical murmur and the second aortic sound is accentuated. The electrocardiographic pattern shows a few very interesting items, which may be briefly summarized as follows:

The direction of the electrical axis in a man with an hypertrophied heart and hypertension is expected to be between 0 and -90 whereas the direction of the electrical axis in our case shows an angle of plus 23 (the direction of the normal axis lies between 0 and plus 90°). If we add to this fact the aspect of the fourth lead, we might assume that the patient had already one attack of myocardial infarction, although his history does not disclose this. He would appear to have had a lateral infarction (Wood & Wolferth) but I do not wish to discuss this point here. See Fig. I.



J. L. 50 years of age. Electrocardiogram taken on March 26, 1940. All successive electrocardiograms show the same picture. Note particularly: the negative T in Lead I and IVF, the low voltage of R-S in Lead 3.

The interest of the case lies in the course of its history. On my advice he went to bed. I was called five days later, at midnight, to see him because he was suddenly taken with severe prolonged pain at the precordium, extreme weakness, and sweating. He felt dizzy, and was not able to sit up. Shortly before this attack he had an exciting discussion with his friends. I saw him some thirty minutes later, the pain still persisting and very restless. His blood pressure had fallen from 195:135 to 135:65 and was next morning 110:65. The heart action was rhythmic, 90 pulsation a minute, heart sounds distant.

An electrocardiogram taken shortly after the attack did not show any substantial changes from the first examination. In view of the possibility of delayed appearance of electrocardiographic changes, electrocardiographic registration was made in several instances after the onset but none of them were different from the first electrocardiogram which was taken five days before this accident.

There was no temperature, sedimentation rate was not increased, a leucocyte count was unfortunately not taken. In less than eight days the blood pressure returned to 175:100, the heart rate was around 70 a minute, the patient felt well and was allowed to arise 10 days after the onset and attends regularly his activities as usual, complaining from time to time about a heavy sensation at the precordium, especially while walking. He is in apparently good condition now, a year after this onset.

What did happen to that patient? Let us briefly summarize the main items of his history and findings:

1. Sudden appearance of prolonged pain and symptoms of shock;
2. Sudden fall of blood pressure;
3. Absence of temperature and not increased sedimentation rate;
4. Absence of electrocardiographic changes after the attack;
5. Quick return of the blood pressure to previous values;
6. Normal leucocyte-count is also found in such cases.

While the first impression in observing the patient was that of acute myocardial infarction, certain doubts arose after the electrocardiographic pattern, taken in different periods of time after the onset, did not show any changes with regard to the first pattern taken five days before the attack. The behaviour of the blood pressure was also uncommon, but I have seen a case with autopsy in which the blood pressure returned on the fifth day to 200, causing rupture of the left ventricle. A negative leucocyte count in such cases may allow to exclude the presence of infarction already in the first days; for we have here to deal with a special aspect of angina pectoris.

As pointed out above, cases of this type show many of the striking symptoms characteristic of myocardial infarction, but in those cases in which post-mortem examination was performed several years after such an attack, no infarction could be found to be related to such an attack. Levy and Bruenn⁴ think that in such cases minor degrees of acute coronary insufficiency without occlusion have occurred and regard them as intermediate between angina pectoris and myocardial infarction. Blumgard et al.¹ have described the history of two cases in which similar accidents had occurred and in which post-mortem examination three and three and a half years later, could not produce any evidence of an old infarction. The authors call this condition "Coronary failure." "Myocardial anoxemia sufficiently severe to induce even prolonged pain may not necessarily lead to necrosis, if rest in bed and sedatives are utilized. Structural myocardial damage will therefore not be observed."

Our patient was in bed when taken by the attack, rest may have reduced the demands on the myocardium to allow recovery of the anoxic muscle.

I think the history, which I presented to you, is just one more example of "Coronary failure." The diagnosis is secured by the lack of changes of the

electrocardiogram, quick return of the blood pressure to previous values, and by the normal value of the leucocyte-count, a symptom that is stressed by Blumgard et al.¹ In cases in which the patient is seen first and comparison of the clinical and laboratory findings are not available this symptom may be the only positive fact to secure not only an early but also the true diagnosis.

I have brought in the discussion of this case for two reasons: 1. First, because of its practical importance in permitting one to make a differential diagnosis and the prognosis accordingly; 2. This observation demands our special attention also in connection with the above made classification, for we see that his electrocardiogram is very suggestive of subacute myocardial infarction having occurred without evident symptoms sometime earlier.

Let us say, an autopsy is made in this case a year or two later and an infarcted area is found. Evidently this may be connected with the attack of prolonged substernal pain which was mentioned just now, while we have proved that this onset did not occur with myocardial infarction. This may explain why it is sometimes so difficult to give an exact interpretation of such cases. If I had not had an electrocardiogram registered before this attack and observed the onset of coronary failure just mentioned, I would not have been able to give the exact interpretation of the happening either and kept the patient for almost six weeks in bed.

The more chances we have to study certain aspects occurring during the life of a patient with angina pectoris, the better we may be able to interpret certain anatomical findings and obtain more insight into the pathological aspects of angina pectoris and myocardial infarction.

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The Annual Meeting of the Association of Medical Health Officers of Nova Scotia will be held on Tuesday, July 8th, at 10 a.m., at the Cornwallis Inn, Kentville.

Simmonds' Disease

(With report of a case treated with Antuitrin-8)

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Synonyms—Hypophyseal cachexia.

Pituitary cachexia.

Asthenia gravis hypophyseogena.

Simmonds¹ originally described the entity of pituitary cachexia in 1914. Graham and Farquharson² reported the first cases in the American literature in 1931.

It is an extreme form of hypopituitarism with secondary insufficiency of other endocrine glands; it is due to destruction or impairment of the anterior pituitary by various morbid processes including tumours, cysts, emboli, thrombosis, trauma, tuberculosis and syphilis.

Maresch³ first called attention to the fact that the syndrome follows a confinement and suggested that a functional exhaustion of the anterior pituitary was a basis for the etiology. According to Sheehan⁴ most cases of Simmond's disease are due to the late effect of post partum necrosis of the anterior pituitary. He gives a review of these effects, clinical and pathological, based on detailed analysis of 51 published cases and supplemented by information from over 70 other cases in the literature. The original necrosis occurs at a delivery which is invariably complicated by collapse usually as a result of severe haemorrhage. It was originally described as due to septic puerperal infarction of the anterior pituitary (Simmonds) 1914. Females are more often attacked than males; among 65 cases, 47 were females (Calder). Engelbach⁵ maintained that many pituitary cachexias may be due to a transitory functional disorder of the anterior lobe. He thought that it was probable that severe deficiencies of the hypophysis occur as frequently from aneoplastic disorders as from other causes. The autopsied cases summarized by Silver⁶ show that with the exception of a few instances in which the pituitary has been destroyed by tumours, the disease affects chiefly women in the child bearing age, and frequently the onset can be related to a complicated pregnancy. From examination of 59 women dying from other causes during the puerperium, Sheehan found almost complete necrosis in 7 and smaller areas of necrosis in 4 in the anterior pituitary.

Syphilis has been given as a cause, though among 69 verified cases a positive Wassermann reaction was reported in only 7 per cent. It has followed head injury (H. Zondek).

CLINICAL PICTURE⁷

The onset is usually gradual. As it is a multiglandular syndrome, the list of manifestations is long and all of them do not occur in all cases. The most constant and important are: emaciation, asthenia, anorexia, precocious senility, low basal metabolic rate (-40), subnormal temperature, low blood pressure (Systolic 100 mm. Hg.), secondary anaemia, and relative lympho-

cytosis. The skin is dry, often wrinkled, and may be somewhat pigmented; the pubic and axillary hair is absent; the nails may show trophic changes. The genitalia atrophy and amenorrhoea, sterility, impotence and loss of libido are the rule. The urinary excretion of water and especially of salt is small. Mentality is poor, and melancholia and Korsakow's syndrome may follow. Somnolence may be prominent. Constipation is common, and abdominal crises as in Addison's disease, occur. There is sensitiveness to cold, and spontaneous hypoglycaemia has been recognized. Mild and abortive forms occur.

In cases so diagnosed life has been prolonged for 20 and more years, but in such instances the diagnosis has been questioned. Death may be preceded by coma.

Doubt has been cast on the value of a positive diagnosis during life, and especially on the distinction from anorexia nervosa, until the effect of treatment by hormones and feeding and psychotherapy have been tested. It has been stated that a number of patients with anorexia nervosa show signs of pituitary inadequacy (Sheldon). In Addison's disease the pigmentation of the skin is usually much deeper and may occur on the buccal mucosa, and the loss of flesh and weight is not nearly so severe. Sheehan concludes that a severe degree of emaciation is uncommon in Simmond's Disease.

CASE REPORT

A school girl, age 15, height 5' 3", usual weight 110 pounds. She complained of weakness and loss of weight.

Menarche began at twelve years—4-5/21 day type with some pain.

Past history—nothing of note.

Family history—father died following operation when patient was three years old.

History of present illness revealed amenorrhoea for approximately five months before Antuitrin-S was given. There was loss of appetite with resultant loss of weight and weakness. She had lost ten pounds in two months before coming for treatment. This loss of weight preceded her amenorrhoea. She was irritable and very constipated. Naturally she was not irritable. Anorexia had increased progressively to the point where her appetite was practically non-existent. Bradycardia was a feature, pulse being around 50. Somnolence and sensitiveness to cold were marked. Her temperature was subnormal.

Upon examination the patient presented a cachectic, prematurely aged appearance. This aged appearance was so marked that people who knew her said she looked like a little old lady. The skin was scaly, dry, loose and inelastic. The hair of the head was thick but dry, the pubic hair and axillary hair markedly thin and dry. The breasts were atrophied. The eyes were sunken.

Examination of the blood revealed a secondary anaemia. X-ray of the chest on two occasions showed no evidence of tuberculosis.

An adequate diet was prescribed together with a proper amount of rest. She was given iron and vitamin therapy. The diet was of little value simply because the anorexia was so complete that she ate very very little. The patient was taken out of school with the exception of a short time and was kept in bed. (See table). In spite of these measures her condition became worse and for a while it was alarming—loss of weight being at one time 41 pounds

(110 pounds to 69 pounds). After being treated along these lines for four months and three weeks she was given Antuitrin-S (Parke, Davis & Co.), which is prepared from the urine of pregnant women. Antuitrin-S (apoidin) contains anterior pituitary-like sex hormone or anterior pituitary-like gonadotropic principle of pregnancy urine. For dose and progress of patient see table.

Usual weight—110 pounds.

Weight when patient first came under observation—100 pounds.

"	in 3 mos.	91 lbs.	(in bed 1st. 2 mos.)
"	" 3 "	2 wks.	89 lbs. (at school 1 wk.)
"	" 4 "	2 "	79 " (in bed from 3½ mos. to 4½ mos.)
"	" 5 "	69 "	(was getting 100 Rat units Antuitrin-S daily second week)
"	" 6 "	1 "	93 " (100 Rat units Antuitrin-S daily)
"	" 6 "	3 "	103 " (100 Rat units Antuitrin-S daily)
"	" 8 "	3 "	115 " (100 Rat units Antuitrin-S daily for 1 mo. and every second day for 1 mo.)

The patient was allowed to go to school for the week as indicated in the hope that it might give her something to occupy herself. At the time she was very anxious to go to school. When she was not in bed, she was confined to the house most of the time and was for the most part inactive.

On Antuitrin-S therapy her weight went from 69 lbs. to 103 lbs. in two months. She had her first menstrual period in seven months, or two months after being placed on Antuitrin-S treatment. Her menstrual periods have been regular since that time. Her general health improved along with the gain in weight.

Antuitrin-S therapy was discontinued at eight months three weeks' period or after being used for four months. For ten months following this discontinuance of Antuitrin-S her health has been normal.

FEATURES SUGGESTING ANOREXIA NERVOSA

She was very much upset by the marriage of her mother. The marriage took place about one month after she began to lose weight. She was temperamental, thought medicine cost too much and often refused to take what was prescribed for her. She was interested in cooking and wished control of same. If anyone interfered with her as regards cooking, she became very angry. On one occasion she flew into a rage when her brother, home for the week-end, ordered something extra from the store. At this time her mother was away and she wanted no interference with her handling of cooking and food. This suggests behaviour of anorexia nervosa patients described by Rahman, Richardson and Ripley⁸. In a complete study of 12 cases with a symptom complex of anorexia nervosa they stress the compulsive nature and found an interest in diets and cooking was common.

Dr. H. E. Killam saw this case with me in consultation.

DIFFERENT VIEWS IN RELATION TO THE THERAPEUTIC TEST

Bruckner⁹ is of the belief that a response to anterior pituitary or anterior pituitary like substance is evidence against rather than for Simmond's disease. He¹⁰ is still of that opinion. In other words he does not believe in the therapeutic test. He cites two cases in detail, both of which resembled pituitary

cachexia. They both regained normal health by increase in food intake without the aid of hormonal therapy. He cites a third case, similar to the one I report which improved on hormonal therapy. It was at that time treated by some doctor other than himself. Following a relapse the same case in his hands responded favorably to forced feeding.¹⁰

Sheldon¹¹ has intimated that many cases described as Simmond's disease, particularly in early life, may well be instances of anorexia nervosa.

Richardson¹² is critical of the therapeutic test. He reported six cases, three of whom were thought to have Simmond's disease and all of whom proved to have anorexia nervosa. He points out that many of the cases of Simmond's disease reported, treated with anterior pituitary continue improvement after cessation of treatments. He offers this as a point against the effectiveness of hormonal therapy and therefore against the therapeutic test. He says, "the suspicion is when replacement therapy of short duration is followed by permanent cure, the same effect might have been achieved with tablets of lactose or with injections of saline solution."

von Bergmann¹³ states it is impossible to diagnose Simmond's disease without anatomical confirmation of same. In this event it would be impossible to diagnose the disease clinically.

Smith¹⁴ has demonstrated that pituitary substance per os is completely ineffectual in repairing any of the deficiencies produced by hypophysectomy in animals.

Wahlberg¹⁵ states functional insufficiency of the anterior pituitary occurs. Hawkinson¹⁶ says the common misconception still exists that pituitary disorders are due solely to tumour or organic disease of the gland, even though many cases previously reported have been accompanied by disease in this organ. He reports a case of Simmond's disease which improved on being treated by gonadotropic principle of pregnancy urine.

Engelbach⁵ (as noted elsewhere) maintained that many pituitary cachexias as well as other forms of pituitary dysfunction, may be due to transitory functional disorder of the anterior pituitary. He thought it probable that severe deficiencies of the hypophysis occur as frequently from aneoplastic disorders as from other causes.

Sheehan reports substitution therapy is as yet not satisfactory, but if a subsequent pregnancy occurs, the symptoms are permanently cured. Kunstadter¹⁷ reports two cases of what von Bergmann terms pituitary emaciation. They both responded favorably to substitution therapy.

Hicks and Hone¹⁸ conclude there is strong evidence in favor of the curative value of prolan (pituitary sex hormone of Zondek) therapy in certain cases of pituitary cachexia, at all events, when associated with altered circulatory response to effort. In the case they reported, the patient showed a fall in blood pressure on sitting up from a lying position while the pulse response remain unaltered. Following the administration of 100 units of prolan the circulatory response became normal within 24 hours. Instead of a fall in blood pressure there was the normally expected rise.

Baltzan¹⁹ reports a case similar to the one here presented. His patient showed improvement on Antuitrin-S therapy. Among other cases in which substitution therapy had striking results are those of Calder²⁰, Striker²¹, Constantini²², Hurthle²³, McGovern²⁴ and Brougher²⁵. In most of these cases an extract of the whole anterior pituitary gland was used.

The situation is further complicated by the fact that total destruction of the anterior pituitary lobe has been reported at autopsy in patients who had no signs of pituitary insufficiency. Krumbhaar²⁶ reported such cases and cited others.

FACTORS INCREASING DIAGNOSIS DIFFICULTY

Bruckner²⁷ pointed out that in spite of hormone preparations the physician will always attempt to feed his patients as adequately as possible. This would leave in doubt whether or not food alone would have produced the good results. In the case here reported food alone was of no use and the patient was becoming worse.

Psychotherapy can be used without medication, but medication cannot be given without psychotherapy. This is true especially when the medication is given hypodermically as in this case.

SUMMARY AND CONCLUSION

A review of Simmond's disease is given. A young girl of an age when this disease is rare, with marked loss of weight and some features of pituitary cachexia failed to respond to forced feeding and rest with iron and vitamin therapy. She responded favorably to Antuitrin-S therapy and has been in normal health for ten months after discontinuance of same.

In making the diagnosis of Simmond's disease, I do so in the sense that Engelbach believes it may occur, not in the sense of a destructive lesion of the gland. I am also influenced by the effect of Antuitrin-S therapy.

It should be pointed out that much data that would be available if the patient were under observation in a well equipped hospital, is lacking here because the patient was treated at home.

A conclusion to be reached is that a diagnosis during life remains completely uncertain, depending on the value placed in the therapeutic test. The reason is that most if not all the symptoms characteristic of Simmond's disease can be produced by simple inanition. If a case with many of the symptoms does not respond in a reasonable time to forced feeding, Antuitrin-S or a similar preparation should be tried. This is true especially in a case which previously has had a confinement associated with severe haemorrhage.

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* A Doctor Tells His Story

of all that happened in one dreadful night of bombing. His name and the location of his hospital are not published

I HAD just finished an operation and was on my way down to the surgeon's dining-room for dinner when the warning siren sounded. It was about seven. We get everything in readiness as soon as the alert goes, but we don't go to our action stations until the sound of gunfire or bombs dropping tells us that danger is imminent.

I hoped to have enough time to enjoy my meal, but I had just dipped into my soup when I heard the first whishing downward rush of a bomb. We'd had occasional raids before, but somehow I had a premonition that this was going to be a bad one, and as I went to the wall to switch on the yellow action-station lights throughout the hospital, I remember saying to the other surgeons: "Well, fellows, I feel we're going to get it tonight."

My own action-station is to patrol the wards and corridors just to see that everything is shipshape. I walked through the maternity ward from the dining-room and noticed that all the patients had been placed under their beds, with their mattresses over the top of them.

* * * * *

Nurses were wheeling other beds down from the top floors and lining them along the ground-floor corridors, away from flying glass. Since we haven't any underground rooms, that's the best protection we can offer.

I went on up three flights of stairs and stepped out on the flat roof of the main building. I could hardly believe my eyes. All around the hospital grounds glowed literally hundreds of incendiary bombs, like lights twinkling on a mammoth Christmas tree.

Half a dozen small fires had already started in the hospital buildings; flames were licking through the roof of the laundry, and another blaze was going on the roof of the emergency store-room next door to it. From the roof the hospital superintendent was shouting instructions to the hospital's auxiliary fire crew down below, and before long they had their hoses going on both buildings.

As we watched, however, flames leaped from the roof of the main store-room. We were pretty worried by this time, for fires in both storerooms might destroy all our supplies except those on hand in the hospital, just enough for a normal night's work.

I left them fighting the fires and went down to check up on the reception building where the casualties would arrive.

* * * * *

The reception officer, a surgeon who had waited night after night for just such an emergency, was ready to give each incoming patient a preliminary examination before tagging him for the type of treatment required in the wards or operating rooms.

I had just about completed my inspection when the real fun started. First an incendiary fell on the roof of the nurses' home. Fortunately, a workman examining the roof the day before had put his foot through a rotten section and the hole had not been repaired. A nurse passing along the top-floor corridor happened to look up and saw the incendiary perched on the edge of the hole.

She gave the alarm and the fire was put out before it could get hold, but we decided to evacuate the building and bring all the nurses into the main section. Again we were lucky. No sooner had the last nurse left the building than a heavy explosive crashed into it and exploded on the thick concrete top floor. That was our first direct hit.

About 8.30 another shower of incendiaries started fires on top of the men's medical ward, the women's medical ward and the eye ward. With the other surgeons, the orderlies and nurses, and even some of the able male patients, I ran across the open space between the main building and these wards and began transferring the patients.

The nurses wheeled the beds outside while the rest of us hoisted patients on our shoulders and carried them pickaback across to the main hall. There wasn't a murmur from one of them, although some must have been pretty badly hurt with the jogging we gave them.

Providence must have been watching over us. As I reached the door of the main building with the last patient on my back a bomb screamed down and plunged into the men's ward.

We put the patients on stretchers and blankets along the main-floor corridors, which were already so crowded that we had to tread carefully to get from one end of the hospital to the other. Then the casualties started to come in from outside.

From then on everything flashed past me like the action in a speeded-up film. I remember assigning the other surgeons to their theatres; I took the main one on the second floor. We had made elaborate preparations about classifying the patients as they came in, but we didn't have time for detailed examinations. All we could do was to divide them roughly into resuscitation cases and those requiring immediate surgery.

The resuscitation patients were whisked into beds and given electric blankets and oxygen to help them recover from the shock of their wounds. The immediate surgery cases were divided among the three theatres. I suppose I did about 15 operations throughout the night, some of them more intricate than others, but they came too fast for me to keep count. The other theatres handled about 40.

We couldn't work rapidly. Wounds are very tricky in this war of bombs. The majority of cases were lacerations or injuries to limbs.

About midnight the electric power went off but I continued with the operation I was on by the light from two small bulbs run by our own emergency lighting system. By this time I was feeling pretty shaky, I admit.

I wasn't exactly frightened, but the sound of a bomb whistling down from 5,000 to 6,000 feet above you isn't a comfortable one. Every few minutes the nurses and the anaesthetist threw themselves under the operating table as the bombs roared down. I didn't like to follow them, but every time one whistled uncomfortably close I instinctively pulled the knife away and ducked sideways.

Whenever I began to think too much of the bombs, however, I thought of the patients lying all over the hospital, just trusting to luck that they would not be hit. Up on the top floor of the gynaecological ward we had 15 women whom we couldn't move. They stayed in their beds through it all without a complaint, although a bomb that smashed the staff quarters next door covered them with glass from their windows and plaster from the ceiling.

In another wing we had to leave a dozen fracture cases. All night long they lay on their backs, unable to move, hung up on their frames, and watched the Jerry planes cruising about the fire-lit sky through a huge hole that had been blown out of the wall.

The morale was stupefying. Throughout the packed hospital there was not one cry of fear, not one sign of panic. We didn't have a case of hysteria all night long.

The only word of complaint came from a wounded German airman who'd been in the hospital for a few days. He was on the top floor of the main building, and I noticed that no one seemed to want to risk his own life to bring him downstairs. When the orderlies finally went to him they found him cringing in bed and muttering in English, "Too much bomb—too long? Too much bomb!"

* * * * *

By this time the windows in my operating theatre had been blasted out and a bitter cold wind was blowing across the room. It was too cold to uncover the patients and too cold to operate, for I was shivering from head to foot. The windows of the second theatre had also been blown out, so we were forced to move into the ground-floor theatre, the windows of which were protected from blast by an outside brick wall. It was an amazing scene. It looked far worse than the descriptions I've heard of the front-line casualty clearing stations of the World War.

Patients were lying head to toe on every inch of space. The nurses were marvellous. With hurricane lamps and hand torches they moved about among the patients, comforting them and giving them little sips of water. That was about all we could do for them.

It was bitterly cold throughout the hospital. Most of the windows had been blasted out, walls had been blown down and not a door remained in its frame. We issued extra blankets to all the patients, but they kept coming in so fast that we didn't have time to make them comfortable.

By 4 a.m. I couldn't keep a steady hand. I had taken nothing to eat except a sip of soup since lunchtime the day before. Then our emergency lighting failed just as I was in the middle of an operation. We quickly rigged up an automobile headlamp to a battery set and I finished the job. Bombs were still crashing down, but by a great miracle the only casualty was a soldier who was lending us a hand. While he was crossing a courtyard a bomb fell directly on him and blew him to bits.

* * * * *

When daylight finally brought an end to the raid, it was the most welcome dawn I have ever seen—only to be marred when wardens rushed in to report that they had found a delayed-action bomb buried just outside the ground floor operating theatre. All the patients in the main building within range of the bomb had to be evacuated immediately.

But no sooner was this done than we got orders to evacuate the whole hospital full of patients to other hospitals in neighbouring towns. The ambulance and stretcher men, who had been on their feet and out in the debris-littered streets all night long, worked hour after hour. By five that afternoon the last ambulance rolled away from the doors, and I sat down to my first meal in 28 hours.

We were without power and steam. We had lost hundreds of pounds worth of supplies when the storerooms were fired and were certainly not in shape to operate, but we didn't feel that our job was finished. That night we had an emergency casualty station set up in the surgeons' dining-room, with our instruments boiling in a pan on the fire, just in case Jerry paid us another visit.

**The Seventy-Second Annual Meeting of the
Canadian Medical Association will be held at Winnipeg
at the**

Royal Alexandra Hotel, June 23, 24, 25, 26, 27.

A strong scientific programme has been arranged and the local committee are putting forth every effort to see that the visitors have a good time. **Plan Now to Attend.**

Editor's Column

THE NOVA SCOTIA SOCIETY OF RADIOGRAPHERS

ON May 22, 1940, a meeting of X-ray Technicians was held at the Victoria General Hospital and it was decided to form a Nova Scotia Society of Radiographers.

The parent society was organized in Toronto for the Province of Ontario in 1934. At present similar organizations have been formed in every province of Canada and application has been made for a Dominion Charter.

The purpose of these societies is to raise and maintain a high standard among those doing technical work in X-ray. It is realized that radiology is rapidly progressing and becoming more and more exacting.

The technician of to-day must be a student who is ambitious and capable of learning the many new techniques which are being constantly developed.

In order to assist technicians the local branch have held several instructive meetings during the winter. The society is indebted to Dr. Roy, Radiologist, Halifax Infirmary, Surgeon Commander Morton, Surgeon Lieutenant MacLeod and Mr. Hall of the General Electric Corporation for their lectures.

The officers of the Society are:

Hon. President—Dr. A. B. Campbell, President Medical Society of Nova Scotia.

President—A. Perry, Camp Hill Hospital.

Vice-President—Mrs. C. Campbell, Halifax Infirmary.

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

Registrar—Miss W. Flynn, Victoria General Hospital.

Advisory Board—Dr. S. R. Johnston, Victoria General Hospital;
G. E. Harrison, Nova Scotia Hospital.

Provincial Representatives—Sister Regina, North Sydney; Miss A. Appleby, Yarmouth; Miss Cowan, New Glasgow.

Board of Examiners—Dr. S. R. Johnston; Dr. H. R. Corbett; G. E. Harrison.

MEMBERS

Connie Roney, Glace Bay General Hospital, Glace Bay, N. S.

Margaret Gillies, City of Sydney Hospital, Sydney, N. S.

Jean Simpson, Children's Hospital, Halifax, N. S.

Margaret E. Blandford, Colchester County Hospital, Truro, N. S.

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Rev. Sister Anna, All Saints' Hospital, Springhill, N. S.

Rev. Sister Regina, Hamilton Memorial Hospital, North Sydney, N. S.

Winnifred Flynn, Victoria General Hospital, Halifax, N. S.

Violet Toomey, Victoria General Hospital, Halifax, N. S.

Bertild M. Hyland, Victoria General Hospital, Halifax, N. S.
Albert Perry, Camp Hill Hospital, Halifax, N. S.
George E. Harrison, Nova Scotia Hospital, Dartmouth, N. S.
Gilbert G. Harris, Nova Scotia Sanatorium, Kentville, N. S.
Norman Hurst, Victoria General Hospital, Halifax, N. S.
Rev. Sister Mary David, Halifax Infirmary, Halifax, N. S.
Rev. Sister Marie Christene, Halifax Infirmary, Halifax, N. S.

A FEW days ago an interested friend asked me why no mention was made in the BULLETIN of the fact that Doctor so-and-so had secured a high academic distinction—by the hard road of competitive examination. It was an achievement of which all the candidate's friends were very proud. That no paragraph of recognition had appeared in the BULLETIN, the proper repository of all such information, seemed to be regarded as a very unfortunate omission. The Editor heartily agreed that the incident was little short of an outrage and said so in all sincerity. Who was to blame? In the first place the physician himself left the matter to chance with the hope that someone, less interested than himself, would see that it found its way to the BULLETIN. Knowing the man would shrink from anything vaguely suggestive of self advertising it is more than likely that he hesitated to do so himself. There is, however, nothing disreputable about telling our colleagues through our own medium that one has been successful in passing a certain examination and qualifying for an advanced degree. It gives all of us an opportunity to rejoice in the success of an associate and to be stimulated by his achievement. Next at fault were his intimate friends who did not think it worth while to purchase a local newspaper—mark the item and send it along. The last and worst offender is the Secretary of the County Medical Society whose duty it is as local editor to see that just such items along with the report of the meetings of his Society reach the BULLETIN in proper form.

Most of our "personal interest notes" are gleaned, which means to collect with much labour, by the Secretary to the Secretary going down this column and up that of all the newspapers of the Province, whilst the telephone rings and letters are taken down in shorthand between this column and translated and typed after that, and items of chaff referring to doctors of this and doctors of that are winnowed and only items referring to doctors of medicine reach the BULLETIN. What a higgledy-piggledy, haphazard and ridiculous method of news gathering! This catch-as-catch-can spectacle of inefficiency will go on and on until you decide to turn over a new leaf and make the BULLETIN the channel by which we tell one another the personal things and by so doing make the BULLETIN a day to day record of the medical profession in this Province.

H. W. S.

Case Reports

PUERPERAL PSYCHOSIS—TOXIC EXHAUSTIVE TYPE

PSYCHIATRIC SURVEY

Mrs. D.

Examiner: Mr. G. W. McElman, December 16, 1940.

1. EXTRACT FROM ADMISSION FORMS—

The patient is a housewife who was admitted to this hospital from a General Hospital where she had been delivered of twin babies six weeks previously, one child stillborn.

Dr. A. states she was nervous and irritable, talking at random; very violent at times and had to be restrained in a straight jacket; talks very childishly at times, with incoherent ideas.

She had been suffering from acute nephritis for four months previous to delivery; toxæmia of pregnancy; had given signs of mental trouble before delivery.

Dr. B. states she is nervous and irritable, talking at random; violent at times and had to be restrained in straight jacket; has incoherent ideas at times.

She has been suffering from acute nephritis for past four months; six weeks ago delivered of twin babies; had signs of mental trouble before her babies were born.

2. APPEARANCE AND BEHAVIOUR—

The patient is a female, white, about 40 years of age, pale, somewhat undernourished; appears to be clean about person; during interrogation, patient was emotionally unstable, crying a little at times, and continually bringing one hand or the other up to her face or touching her hair.

Attention and cooperation were good, although her memory for recent events appeared to be somewhat hazy at times.

3. ORIENTATION—

Personal—Patient states she is Mrs. D., forty years of age, born September 11, 1900; she is a housewife; she lives at T.; her religious affiliation is Roman Catholic.

Temporal—She states it is December 17, 1940 (Dec. 16); she came to this hospital three weeks ago; she came by car and was accompanied by five others, four men and one woman; she did not know any of them, but she has heard their voices.

Spatial—She states she is in the Nova Scotia Hospital in Dartmouth.

4. MEMORY—

The patient's memory for events occurring since her mental illness began is rather hazy at times.

Family History—Father living and well, 74 years of age; mother living and well, 76 years of age; sisters, two living and well, ages 38 and 36, married; brothers, five, living and well—48, 46, 42, 35 and 33, all farmers.

No history of any familial disease; no insanity.

Personal History—She was born at September 11, 1900, and as far as she knows, her early development was quite normal; no history of convulsions.

Education—She states she did not start to school till she was eleven years of age; this was because she lived so far from school and because she was the oldest girl in the family she had to help her mother; she went to grade 6 and did not miss any grades; says she used to study some at home after she finished school; retention of school knowledge appears to be good.

Economics—Patient is a farmer's wife.

Habits—Her favorite form of amusement is reading, but she never has much leisure; does not use alcohol nor tobacco.

Menstrual History—Puberty at 14 years; regular; no dysmenorrhoea; 2/21 day, using three pads on day of greatest flow; no discharges nor spotting between periods; regular between pregnancies.

Obstetrical History—Eight pregnancies, all living and well, eldest 21 years, youngest 2 months; last pregnancy twins, one stillborn. During last two pregnancies patient has had pre-eclamptic symptoms. States in last pregnancy labor was induced artificially.

Marital History—Patient was married at the age of 18 years; her husband died two years later. Three years after the death of her first husband she married again. Except for the loss of her first husband, she states her married life has been a happy one.

Court Record—None.

Make-up—Patient says she is of a quiet disposition, rather easily upset and somewhat nervous; she does not like to mix with people but prefers to remain at home with her own family; she does not have "blue spells".

Medical—Measles and whooping cough, with good recoveries; no other serious illnesses other than nephritis with her last two pregnancies. No operations.

Previous Attacks—None.

Present Illness—The patient states that two months ago she was delivered at a general hospital. Previous to delivery she had been suffering from "kidney trouble." She says she remembers the birth of her child and that the nurse showed her the child. Her memory for events of the next month is faulty. She remembers she was excited, refused to take her medicine, had to be restrained and on one occasion ran out into the hall where she fell, cutting her head.

5. IDEAS AND JUDGMENT—

The patient had delusions of persecution; she thought the nursing sisters were trying to poison her, and for this reason she refused to take medicine; she also believed they were keeping her letters from her. She also believed

that her people thought she had died and had returned to life; she then asked if this were possible. Judgment does not appear to be much impaired at present. Her insight into her condition is rather poor; she says that she might have been "imagining" things, at least she hopes so.

6. HALLUCINATIONS—*Auditory*—She heard voices speaking to her foretelling the future; she was told that the end of the world would occur Dec. 31st, and after January, 1941, the world would be populated with airplanes, crows and pigs. Since her admission to hospital she has heard voices outside her room.

Visual—At times she would see "fluttering children on the wall," at other times she would see spirits, some of whom she knew and some she did not; those she knew would never talk to her. She saw her first husband who would not talk to her. She states she had a "forerunner" that he did not reach heaven.

7. EMOTIONAL TONE—

During the interrogating, patient was emotionally unstable, crying at times. At times she did not reply readily and again she seemed eager to answer questions. Since interrogation the patient has appeared somewhat more friendly and does not regard me with suspicion as she did previously.

8. MOTOR STATUS—

It is only within the past week that the patient has been able to be out of bed, hence activity is of necessity limited. However, the patient appears to be quiet and well behaved.

ABSTRACT

Patient was admitted to this hospital from a general hospital. After being delivered of twins, one stillborn, she had exhibited a state of excitement which had necessitated restraint, delusions of persecution and hallucinations, both auditory and visual.

PHYSICAL EXAMINATION

Heart—Left border markedly displaced to the left; apex beat palpable in fifth left I.C.S. approximately five inches from midline; a moderately loud mitral systolic murmur, propagated to post-axillary line and a loud pulmonary systolic murmur was heard; no palpable thrills.

Pulse—102/min.; regular, fairly good volume.

No other abnormalities found.

LABORATORY EXAMINATION

Urine analysis negative. Blood RBC 2,810,000; Hb. 35-40%. Kahn negative.

PSYCHIATRIC SURVEY

Previous to admission, patient had been very excited and had to be restrained. She also suffered from delusions of persecution and had both auditory and visual hallucinations. At present her judgment does not appear to be impaired but her insight is poor. Her motor status appears to be normal at present. Her memory is somewhat faulty for the period of her mental illness.

Tentative Diagnosis—Puerperal psychosis, toxic exhaustive type.

This case was presented at the Staff Conference held on January 14, 1941. The family history, personal history, history of previous illness and the present illness were reviewed.

INTERVIEW

At the interview the patient's mental condition appeared to be normal. She had considerable insight. Apparently hallucinations have not occurred since consciousness has become clear.

DISCUSSION

Mr. MacElman—I think she is doing well, but a few weeks will probably be required yet before she is completely recovered. I would diagnose her as a puerperal psychosis—toxic exhaustive type.

Dr. Hopgood—I agree with the diagnosis. She is probably well now, but because of her physical condition I think we should treat her for a few weeks more.

Dr. Murray—I agree with the diagnosis, and she appears recovered.

Dr. MacKay—I agree that she is a toxic exhaustive psychosis as a result of the toxins and strain of pregnancy. Both she and her husband should be informed that it would be very dangerous for her to become pregnant again.

DIAGNOSIS

Toxic exhaustive psychosis, due to pregnancy.

P.S.—Patient was sent home apparently completely recovered a short time following this conference.

INVOLUTIONAL MELANCHOLIA

PSYCHIATRIC SURVEY

Mrs. W.

Examiner: Mr. H. A. Foley.

1. EXTRACT FROM ADMISSION FORMS—

Present illness began about two years ago. She was depressed, irritable and self-accusatory. She has been sleepless, restless and agitated. She took small quantity of lysol on June 5th with suicidal intent.

Dr. A's statement: appearance—anxious. conduct—restless. Conversation—complains of many imaginary symptoms; says she has not slept for two years and thinks that she is burnt inside; she has no reasoning power; has a fair insight into her condition; she thinks she has contracted a venereal disease and spends a great deal of her time crying.

Dr. B's statement: conduct—she is agitated and walks about continuously. Conversation—self-accusatory. Says she has taken poison and says that she does not know why she does things.

2. APPEARANCE AND BEHAVIOUR—

At the time of interview patient appeared depressed and agitated. She was walking up and down the ward, crying continuously. During the interview she remained seated and was very cooperative and talked freely.

3. ORIENTATION—

Orientation was correct in all fields.

Personal—She knew her name, age and religion.

Temporal—She knew the day of the week and what month it was. She also knew how long she had been in hospital.

Spatial—She knew that she was in the N. S. Hospital and that it was in Dartmouth.

4. MEMORY—

Good for recent and remote events.

Family History—Father died at age of eighty; cause unknown. Mother living and well. Two brothers, living and well. One sister, died in N. S. Hospital. Husband died five years ago. Ten children, all living and well; ages ranging from 11 to 30.

Personal History—Born on July 27, 1893. She attended common school and reached the eighth grade at the age of thirteen. She stated that she got along well in school, but had to leave school and go to work. She worked at housework for three years and married at the age of seventeen. Since that time she has been living on a small farm and they managed to make a fair living. Her husband died five years ago, and since that time her son has been working the farm for her.

Marital—Patient stated that she got along well with her husband and that he was a very good husband. There were no other family troubles.

Habits—Patient does not use alcohol or tobacco.

Make-up—States that she always got along well with people. She lived a very quiet life but had always been satisfied.

Menstrual—Began at 13 years and had always been regular and no excessive bleeding until about one year ago. Since that time periods have been more frequent and at present time she menstruates twice a month.

Medical—Patient states that she has always been well. About a year ago she states that she had an operation for haemorrhoids and has never been well since.

Previous Attacks—None.

Present Illness—Began about two years ago. She was depressed, irritable and self-accusatory. She has been sleepless, restless and agitated. She took a small quantity of lysol on June 5, 1940, with suicidal intent.

5 IDEAS AND JUDGMENT—

During interview patient complained of severe burning pelvic pain, and that her bowels "never moved," but on further questioning stated that they had not moved for two days. She also stated that before coming to hospital she had a burning sensation in her tongue and eyes; also that her hands turn blue and feel queer. She stated voluntarily that she had taken lysol. When asked why she did this, she said she did not know. She also believed that she had a venereal disease which she contracted since her husband's death, and it was on this belief that she blamed herself for her present condition.

Insight into present condition was poor. The delusions were fixed and she believed them to be real. She stated that her mind was normal at the present time, but admitted that her mind was not right last winter, at which time she said her nervous breakdown was the result of the suffering that she was having.

6. HALLUCINATIONS—

Denied.

7. EMOTIONAL TONE—

When seen in the ward the patient has always been depressed and agitated. Patient walks continuously and cries a great deal.

8. THOUGHT PROCESS—

Attention—good. Association—normal. Train of Thought—no retardation or flight of ideas. Speech—normal.

9. MOTOR STATUS—

Patient was restless and walks continually.

Diagnosis—Involutional melancholia.

This case was presented at the Staff Conference held on June 27, 1940. The family history, personal history, history of previous illness and the present illness were reviewed.

DISCUSSION

Mr. Foley—"I think she is a case of involutional melancholia. I do not know but what hormones would be indicated here."

Dr. Hopgood—"Involutional psychosis, depressed and agitated type. There were times when she showed much more agitation than she did here. We might try theolin."

Mr. Foley—"The fact that she has been ill for two years makes the prognosis worse."

Dr. Murray—"I agree with the diagnosis, and I think that the idea of Theolin is excellent."

Dr. MacKay—"She has four typical symptoms of involuntional melancholia. I agree with the diagnosis, and with the use of Theolin Therapy, but I am skeptical with regard to the results.

Diagnosis—Involuntional melancholia.

Treatment—Theolin therapy.

P.S.—Four months following Staff Conference the patient was discharged, apparently recovered.

ALCOHOLIC NEURITIS

Mrs. M. W.—White; age 29; admitted to the Victoria General Hospital, September 18, 1940.

Complaint—Severe pain in the feet and legs. Her family history revealed that one sister had "drunk herself to death;" otherwise it was irrelevant. Her present history showed nothing of note.

Inquiry into her habits and mode of life revealed that among other things she was strongly addicted to alcohol, and for the past two or three years had been accustomed to, at least once a month, partake in a drinking bout lasting about seven days. During this she used to consume about two quarts of "hard" liquor per day.

Her present illness had begun two weeks previous to admission, when she developed some pain in the muscle of her thighs. This, at first, was not very severe, but in about a week the pain shifted to her feet and legs, and became agonizing. She noticed also that her legs were quite weak. She was forced to go to bed, and eventually to hospital.

On admission her temperature was 97° F., her pulse 90, and her respiration 30. Examination revealed a pale, undernourished female adult, apparently having a lot of pain. Nothing abnormal was noted in the gastro-intestinal, cardiovascular, or glandular systems. Examination of the lungs showed dullness over the left upper lobe, numerous loud rales all over the left side, and with scattered rales at the right apex. Both feet were markedly tender, red, cold and swollen. Impairment of the sensation of heat and cold were noted all over the feet, and there was marked hyperaesthesia over the same area. The knee and ankle jerks were absent.

Blood pressure 110/60. Urine, negative.

Blood picture: Hb. 70%. Red B.C. 3,900,000; W.B.C. 6,200.

Kahn test positive (four plus). Eagle test positive.

X-ray of chest showed a chronic fibroid tuberculosis in the left lung with some infiltration of the right apex. Three specimens of sputum were positive for tubercle bacilli.

With the positive Kahn test, and the alcoholic history, the pain was thought to be either a manifestation of tabes dorsalis, or alcoholic peripheral neuritis. Lumbar puncture was done and the fluid found to be normal in all respects, leaving the neuritis as the most probable diagnosis.

On this assumption, the patient was given a course of Vitamin B therapy. Betaxin intramuscularly was used, 1000 I.U. per day for ten days, and then 1000 I.U. every second day for ten days.

At the end of this course of treatment the patient showed a marked improvement. The pain became much less, and the limbs markedly stronger. In view of the positive sputum, she was transferred to the Tuberculosis Hospital on October 16th.

The existence of a food factor, present in whole rice, but absent in polished rice, which prevented and cured polyneuritis in fowls was demonstrated by Eijkman in the latter part of the last century. Successive investigators demonstrated its presence in many other foods, studied its therapeutic possibilities and finally isolated it in the crystalline state. Among these men the names of Takaki, Grigus, Funk and Jansen stand out.

This food factor, Vitamin B, or thiamin hydrochloride, occurs widely in nature, particularly in nuts, whole cereals, fresh vegetables, fruits, eggs and liver. A very prolific source is brewer's yeast. It is heat labile, especially in an alkaline medium, but is little affected by ordinary storage, drying, pickling, or brewing. Considerable destruction occurs in the commercial canning processes.

The vitamin in some ways plays a major part in the functioning of the nervous system, and its deficiency or absence produces some form of neuritis.

The best known of these neuritides is the polyneuritis beri-beri, which not so very long ago was so common. The recognition of its relationship to Vitamin B deficiency has practically banished it from the earth.

Alcoholic neuritis is another fairly common condition of hypovitaminosis B. Joliffe explains the relationship by showing that the calories obtained from large amounts of alcohol upset the calory Vitamin B relationship, thus producing relative hypovitaminosis and eventually neuritis. Vitamin B is very efficacious in the cure of this condition.

Many other forms of neuritis have been attributed to Vitamin B deficiency, e.g., polyneuritis of pregnancy; other conditions, e.g., rheumatoid arthritis are also thought by some to be related to hypovitaminosis B. The results, however, of treatment of these conditions with the vitamin are very conflicting. There is not enough evidence on which to conclude that Vitamin B therapy is of much use in these latter diseases.

J. J. QUINLAN, Interne
J. W. REID, M.D.

The telephone bell rang with anxious persistence. The doctor answered the call.

"Yes?" he said.

"Oh, doctor," said a worried voice, "something has happened to my wife. Her mouth seems set and she can't say a word."

"Perhaps she has lockjaw?" said the medical man.

"Do you think so? Well, if you are round this way some time next week, I wish you would look in and see what you can do for her."

CANADIAN MEDICAL ASSOCIATION

BY-LAWS GOVERNING MEMBERSHIP IN AND ATTENDANCE AT ANNUAL MEETINGS

Now that all Provincial Medical Associations have become Divisions of the Canadian Medical Association, the By-Laws for Divisions become applicable to Canada as a whole. It is well, therefore, that all members, both of the parent Association and of Divisions, clearly understand the basis of membership and annual meeting arrangements, including registration.

With that end in view, the following extracts from the By-Laws of the Canadian Medical Association are published:

CHAPTER I

Divisions—A Branch Association may become a Division as outlined in Article V of the Constitution and enjoy all the rights and privileges of a Division in the following manner:

1. By intimating to The Canadian Medical Association in writing that it desires to become a Division.

2. By agreeing to amend, where necessary, its Constitution and By-Laws to place them in harmony with the Constitution and By-Laws of this Association.

3. By agreeing to collect from all of its Divisional Members who desire to be members of The Canadian Medical Association such annual fee as may from time to time be set for membership and remit same to this Association.

4. By agreeing to take such steps as seem proper to the Division to increase membership in The Association.

CHAPTER II (SEC. 9)

Registration at Meetings—No member shall take part in the proceedings of The Canadian Medical Association or in the proceedings of any of the sections thereof, or attend any part of the meeting until he has properly registered. Only members and invited guests are eligible to register and attend an annual meeting.

CHAPTER IV (SEC. 2)

Arrangements for Annual Meetings—When The Canadian Medical Association meets in any province where there is a Branch Association or Division, the meeting of that Branch Association or Division for that year shall be for business purposes only. The local arrangements shall be under the direction of the Executive Committee of The Canadian Medical Association, which may enlist the assistance of the Branch Association or Division or one of its component societies. The Canadian Medical Association assumes full

control of the proceedings of the meeting and of all financial obligations save entertainment.

MEMBERSHIP

Medical practitioners in good standing resident in Canada may become members of The Canadian Medical Association in one of two ways:

By-Laws, Chapter II, Section 1—Ordinary Members.

Every member in good standing in a Division shall be automatically an Ordinary Member of the C.M.A. on payment of the annual fee as levied by the General Council.

By-Laws, Chapter II, Section 2—Members-at-Large.

Any graduate in medicine residing in Canada who is not a member of a Division may be accepted as a member of the C.M.A. provided that, with his application, a certificate of approval from the Executive body of the Division in which the applicant resides be furnished to the General Secretary. In the case of an applicant residing in Canada in a territory beyond the jurisdiction of a Division, the applicant must be endorsed by two members of the C.M.A. Such members shall be designated Members-at-Large and shall pay the annual fee as levied by the General Council.

CONCLUSION

The foregoing extracts from the By-Laws, read in conjunction, make it clear that, applicable to all of Canada save that portion which is outside the jurisdiction of any province, all members of the C.M.A., whether ordinary members or members-at-large, must have the sanction of the Division in which the member resides.

Only members of the C.M.A. may register at a meeting of the C.M.A. and attend sessions.

The annual meeting of the Division, which may be held at the same time and place as the annual meeting of the C.M.A., is for business purposes only; and as this meeting is entirely separate and distinct from the annual meeting of the C.M.A., membership in the Division only qualifies for registration to attend the business meeting of the Division.

Society Meetings

THE HALIFAX MEDICAL SOCIETY

THE monthly meeting of the Halifax Medical Society was held on April 2, 1941, at the Dalhousie Public Health Clinic. The meeting was called to order at 8.45 p.m. with the President, Dr. D. J. MacKenzie, in the chair. Dr. MacKenzie welcomed as guests Dr. E. F. Lampell and Dr. Roseberg. There were twenty-one members in attendance.

The minutes of the last meeting were read and approved.

New Business. The following resolution was read by Dr. W. G. Colwell:

Whereas, the City of Halifax is at present with very inadequate consultant service in tuberculosis, and

Whereas, both the City of Halifax and the Province of Nova Scotia are without a proper consultant in allergy, and

Whereas, this Society feels that Dr. T. M. Sieniewicz would be of much more value to his country in private practice than in the position he now fills in the Canadian Active Service Force,

Therefore, be it resolved, that this Society request the Medical Advisory Committee of the Canadian Medical Association to take immediate measures to have Dr. T. M. Sieniewicz returned to private practice.

It was moved by Dr. Colwell and seconded by Dr. F. V. Woodbury that the above be adopted. On motion it was carried unanimously.

There being no further business the scientific programme was proceeded with which was a presentation on "Some Clinical Aspects of Angina Pectoris and Myocardial Infarction" by Dr. S. T. Laufer. A brief summary is hereby incorporated in the minutes.

During the last ten years a better understanding of the clinical and pathological aspects of cases with coronary disease has been made possible by the closer co-operation of those engaged in experimental, clinical and pathological study.

With regard to myocardial infarction, we can now diagnose not only the typical forms occurring with crushing substernal pain and symptoms of shock, but also many atypical forms of acute myocardial infarctions with atypical symptoms, as, atypical localization of pain or symptoms which may be considered to be equivalent to pain as dyspnoea or vomiting with nausea. In all these cases, the beginning is an alarming and sudden one, occurring mostly while at rest.

The author (Laufer) distinguishes a third form of myocardial infarction which is not only a new aspect of this important feature of coronary disease but which in his opinion should be considered as an entity by itself, having a different mechanism of production and which deserves to be treated separately and called "Subacute myocardial infarction or necrosis." Here, myocardial infarction occurs, unlike the other forms of acute onset, gradually. Its symptoms are uncharacteristic but certain laboratory tests are considered to be of great value in the early recognition of this form. The most important

are: a raised leucocyte count and typical electrocardiographic changes which, begin of the acute coronary type, appear delayed. The histories of these cases with electrocardiograms were shown to illustrate this. He stressed the importance of a perfectly taken history in cases with coronary disease. In view of the different mechanism of production of subacute myocardial infarction, evidence is given that early recognition of this form may prevent formation of an infarct, or, at least to limit its extent. In that, especially, lies the practical importance of the new classification of the different forms of myocardial infarction in the paper.

From the different aspects of angina pectoris, attention was drawn to a very rare form that may occur with symptoms of prolonged substernal pain and shock simulating acute myocardial infarction yet without necessarily being so. In the cases with coronary failure, this aspect of angina pectoris is characterized as follows:

1. Prolonged substernal pain with symptoms of shock.
2. Sudden fall of blood pressure.
3. Absence of temperature, sedimentation rate not increased, and a normal leucocyte count.
4. Absence of electrocardiographic changes after the onset.
5. Quick return of the blood pressure to normal values.

Coronary failure is an expression of acute myocardial ischemia in which the ischemia has not led to necrosis and formation of infarction, because rest and sedatives are usually utilised in time so that the whole process is made reversible.

For the interpretation of the pathological findings of some aspects of angina pectoris and coronary thrombosis a closer co-operation between the clinician and pathologist is necessary because certain pathological findings are found in the absence of clinical symptoms, and vice versa.

During the life of a patient with angina pectoris many occurrences may be interpreted in a different light because of the atypicity of their manifestations. This was shown in a case whose history and clinical causes were given in detail.

Dr. K. A. MacKenzie opened the discussion on the paper and congratulated Dr. Laufer on his very excellent and instructive paper on a subject of interest to everyone. Others taking part in the discussion were Drs. Weld, Lehv, Hal Taylor, R. P. Smith, D. J. MacKenzie and Gosse.

Dr. Gosse expressed the hope that when the programme for next year is being considered, more stress should be laid on the presentation of papers at our meetings.

The President then extended the thanks of the meeting to Dr. Laufer for his very excellent paper.

There being no further business, it was moved by Dr. Colwell, and seconded by Dr. Graham that the meeting adjourn.

K. M. GRANT

Secretary

Abstracts from Current Literature

PLEURAL EFFUSION ASSOCIATED WITH OVARIAN FIBROMA. (Meigs Syndrome).
Harris, F. I. and Meyer, M.A.; Surgery, 1941, 9:87.

In 1937 Meigs and Cass reported seven cases of pleural effusion and ascites associated with a benign ovarian tumor (fibroma), in which the pleural effusion was cured by the operative removal of the ovarian fibroma. Meigs in 1939 had collected fifteen cases, all showing similar characteristics of pleural effusion and ascites apparently due to a benign ovarian tumor. Although no satisfactory explanation of why pleural effusion should occur in a patient with a benign ovarian fibroma has been offered, it, nevertheless, is now well established that it can and does occur.

Harris and Meyer report a case of a woman 67 years of age, who had been known to have a growing pelvic tumor diagnosed as a uterine fibroid for four years. She developed massive pleural effusion in her right chest, which was thought to be due to a malignancy. Abdominal operation and removal of a large benign ovarian fibroma resulted in a complete restoration to health, with no recurrence of the pleural fluid. Based on the experience of this case, the authors believe that an ovarian fibroma producing pleural effusion does not respond to radiation therapy and is best treated by surgical extirpation. Differentiation must be made between uterine fibroids, metastatic malignancy, and ovarian fibromas in the diagnosis of this condition. A correct differential diagnosis may mean restoration to health of a patient who is doomed as helpless.

GENERAL ABDOMINAL LYMPHADENOPATHY WITH SPECIAL REFERENCE TO NON-SPECIFIC MESENTERIC ADENITIS. Wilensky, A. O.: Arch. of Surg., 1941, 42:71.

It has come to be fairly recognized that there exists in children and young adolescents an acute abdominal condition of moderate or severe grade, similar in its general manifestations to appendicitis, diverticulitis, etc., but in which the predominant discoverable observations at operation consist only of an inflammatory enlargement of the mesenteric and retroperitoneal lymph glands in the ileocecal angle. The symptoms are severe abdominal pain which can generally be traced to the right side of the lower part of the abdomen. There is tenderness in the right iliac fossa which may be accompanied by vomiting and rigidity, depending upon the severity of the condition. The temperature is elevated with a leukocytosis. The inflamed mesenteric glands present all types of non-specific inflammation, from the simple hyperplasias and catarrhal exudates to purulent effusions and frank abscess formation. Non-specific giant cells and formations sometimes occur.

Wilensky in this paper has assembled and evaluated all available factual matter from the literature regarding non-specific mesenteric adenitis and compared this type with other forms of known mesenteric lymphadenopathy.

In the non-specific type, the mesenteric lymphadenopathy cannot be clinically connected with any demonstrable preceding or accompanying lesion.

In only a minority of the cases can bacteria be demonstrated in the glands, and the predominating organism is some strain of streptococcus. An unidentified virus has been suggested as a causative agent. The portal of entry for the causative agent for non-specific mesenteric adenitis is only on rare occasions the appendix; this can happen only because of some anatomic abnormality of the lymphatic drainage pathways of the appendix. More commonly the portal of entry seems to be related to catarrhal or throat infections. Most often of all the non-specific mesenteric adenitis is a local effect of absorption from some local non-demonstrable lesion in the ileal segment of the alimentary canal: this includes various forms of transient enteritis, injuries and other forms of physical and chemical trauma.

As in many cases abdominal lymphadenopathy is part and parcel of some larger definite clinical entity, treatment must follow along the lines known to be correct for the original disease. In the presence of non-specific mesenteric adenitis and in the absence of any suppuration or other complication, none but conservative treatment would be indicated if one could so perfectly diagnose the condition that the fear of undiscovered acute appendicitis could be definitely eliminated. Unfortunately, this is not possible in clinical practice at this time, and abdominal explorations are more or less frequently necessary in order to establish the true nature of the intra-abdominal condition.

CHLOROSIS. ESSENTIAL JUVENILE IRON DEFICIENCY ANEMIA. Alsted, G.: Amer. Jour. of Med. Sc., 1941, 201:1.

Alsted reports only six cases of chlorosis observed during the last ten years in the Frederiksberg Hospital, Copenhagen. In certain respects the condition as described here differs from classical chlorosis; the age distribution of the patients is somewhat higher than that found in earlier materials: likewise, menstrual disturbances seem to be more rare than generally reported. The etiology in these cases pointed to a qualitative dietary deficiency. Based on these cases it may be concluded that chlorosis has not as generally assumed, entirely disappeared, but that it does still occur, even if with comparative infrequency. The diagnostic criteria remain the same as ever: hypochromic iron deficiency anemia without any demonstrable cause, occurring in young women with a normal gastric function. All patients responded well to iron treatment. As pointed out, the disease, however, nowadays seem to be common in women of a later age than formerly. As the term chlorosis always preferentially has covered an anemic condition in young girls shortly after puberty, Alsted suggests that it might be better instead of chlorosis to use the denomination essential juvenile iron deficiency anemia.

ERYTHROBLASTIC ANEMIA: REPORT OF A CASE. Wood, B.: Staff Proc. of the Mayo Clinic, 1941, 16:202.

Erythroblastic anemia was first described by Cooley in 1932, and has been frequently referred to as Cooley's anemia. It occurs in members of certain races and is characterized by a peculiar facies, progressive anemia, large numbers of nucleated erythrocytes in the circulating blood, splenomegaly, and lesions of the skeleton.

Wood describes the first case of erythroblastic anemia to be observed at the Mayo Clinic. It occurred in a Greek child, three and a half years old and was characterized by the usual features of this condition including the hematologic and osseous changes.

The condition has been found exclusively among children of Mediterranean ancestry, especially Greeks, and occurs in the first decade of life, most frequently in the first two years. The chief signs are pallor and progressive enlargement of the abdomen. The physical growth is usually retarded if the disease begins in the first year of life.

The skin is pale and muddy yellow in color. The facies is mongoloid, the eyes are prominent and in some cases an epicanthus is present. Small discrete, lymph nodes that are not tender to pressure can be palpated in the cervical and inguinal regions. The spleen is greatly enlarged and is firm and smooth; the liver is larger than normal.

Roentgenograms of the calvarium may reveal proliferative changes involving the external table in the form of perpendicular striations of new bone, having a sunburst appearance, typical of erythroblastic anemia. The long bones and ribs show diffuse osteoporosis, with widening of the medullary portion of the bones and thinning of the trabeculae and cortex.

The blood changes usually shows an anemia with a color index less than one. The number of leukocytes varies from 10,000 to 50,000; most of them are mature polymorphonuclears. The erythrocytes vary greatly in size and shape. Megaloblasts, erythroblasts and normoblasts occur in varying numbers and Howell-Jolly bodies may be seen. The icteric index may be elevated.

Children suffering from this condition rarely attain adult age, as the course is slowly but steadily downward. Death occurs after months or years from intercurrent infection or from acute dilatation of the heart.

Treatment consists of supportive measures, such as improvement of general hygiene, intramuscular injections of iron and liver and transfusions of blood. The transfusions are of decided value but their effects are only temporary. The value of splenectomy is questionable.

E. DAVID SHERMAN, M.D.

Sydney, N. S.

Eighty-eighth Meeting of the Medical Society of Nova Scotia
"Cornwallis Inn", Kentville, N. S., July 9th and 10th, 1941.

PROVISIONAL SCHEDULE

TUESDAY, JULY 8TH

2.30 p.m. Executive Meeting.

WEDNESDAY, JULY 9TH

9.00 a.m. Registration.
9.30 a.m. Welcome by Mayor of Kentville.
10.00 a.m. Paper on Surgery by member of C.M.A. Team.
10.30 a.m. Paper on Obstetrics by member of C.M.A. Team.
11.00 a.m. First Business Session.
12.15 p.m. Addresses by the President and Secretary of the Canadian
Medical Association.
1.00 p.m. Adjournment.
3.00 p.m. Golf Tournament or
Other Entertainment.
7.30 p.m. Annual Dinner.
Presidential Address.
Address by special speaker.

THURSDAY, JULY 10TH

9.00 a.m. Second Business Session.
10.00 a.m. Paper by Dr. A. R. Morton, Halifax.
10.30 a.m. Paper by Dr. T. A. Lebbetter, Yarmouth.
11.00 a.m. Symposium on Tuberculosis, by Dr. A. F. Miller, Dr. V. D.
Schaffner and Dr. J. E. Hiltz.
12.30 p.m. Annual Meeting of the Valley Medical Society.
Adjournment.

Preparations for the Annual Meeting

A meeting of representatives of the Valley Medical Society was held on Friday afternoon, April 25, 1941, at 2.30, at the Cornwallis Inn, Kentville, N. S., to discuss the plans for the next annual meeting of The Medical Society of Nova Scotia. There were present from the local Society, Dr. V. D. Schaffner, the President; Dr. R. A. Moreash, the acting secretary; Dr. A. F. Miller, Dr. J. E. Hiltz, Dr. P. S. Cochrane, Dr. J. P. McGrath, Dr. A. R. Chisholm, Dr. B. S. Bishop and Dr. T. A. Kirkpatrick. Dr. A. B. Campbell of Bear River, the President of the Medical Society of Nova Scotia, and Dr. H. G. Grant, the secretary, also attended. Dr. Campbell presided.

From the Valley Medical Society a committee of three, Dr. J. P. McGrath, Dr. P. S. Cochrane and Dr. J. E. Hiltz, was appointed to act with the President and the Secretary of the Medical Society of Nova Scotia in making arrangements for the meeting. Dr. J. P. McGrath was appointed chairman of the local committee.

As the management of the Cornwallis Inn assured the Secretary that fifty double rooms could be set aside for members of the Medical Society on July 9th and 10th, it was decided to hold the conference at Kentville.

It was agreed to accept the invitation of the Canadian Medical Association to send two speakers to the meeting. On account of the nature of the rest of the programme it was decided to ask for one paper on surgery and one on either obstetrics or gynaecology. The local committee agreed to take care of the arrangements for the golf tournament, the annual dinner and other entertainment. It was decided that the annual dinner be a mixed one, but that no dance be held in conjunction with it. It was also agreed that some well known speaker be asked to give a short address at the annual dinner.

Personal Interest Notes

DR. T. A. LEBBETTER of Yarmouth was created a Fellow of the American College of Physicians at the annual convocation exercises held in the ballroom of the Hotel Statler in Boston on April 23rd.

Dr. and Mrs. Edward DuVernet of Digby returned home the end of April having spent a delightful few weeks vacation in Vancouver. They were visiting their son, Dr. E. O. DuVernet.

A car driven by Dr. J. C. Murray of Springhill was badly damaged in a collision near Pugwash early in May. Dr. Murray was accompanied by his wife and young son, the latter being the only one to receive injuries sustaining two broken ribs.

Dr. E. Dudley Dickie who graduated from Dalhousie Medical School on May 13th has opened a general practice in Digby.

Dr. and Mrs. A. B. Campbell of Bear River motored to Kentville on April 25th where Dr. Campbell attended a meeting of the Valley Medical Society.

Dr. and Mrs. L. J. Lovett and daughter, Edith, of Bear River, are at present enjoying a vacation in Pinehurst, North Carolina.

Dr. S. J. Shane of Yarmouth who graduated from Dalhousie Medical College in 1940, and has since then been on the staff of the Nova Scotia Sanatorium at Kentville, has opened a general practice at Port Maitland.

CO-OPERATIVE MEDICAL PLAN DEFERRED

Called to consider establishment of a system of co-operative and preventive medicine in the Glace Bay district a meeting on April 20th which drew a comparatively small attendance was adjourned until May 17th when efforts will be made to have all interested in the change represented.

Under the proposed system the entire community set-up as known now would be re-organized, chairman John Morrison, U.M.W. Board member for sub-district one, explained.

Various representatives spoke on the matter, pointing out it was necessary to gather a large amount of data, consider financing of the scheme and learn what could be expected from it.

A total of approximately six thousand persons are at present subscribing to hospitals in the area and paying for medical attention through a check-off system to doctors of their own choice.

Several moves to get the proposed system started were made but action was deferred until the May 17th meeting.

Obituary

THE BULLETIN regrets to learn of the death of Captain Robert William Maclellan, M.D., R.C.A.M.C., who died at Cogswell Street Military Hospital, Halifax, on May 6th, at the age of twenty-six, following an illness of only a few days. Dr. Maclellan, a son of Dr. and Mrs. E. K. Maclellan of Halifax, graduated from Dalhousie Medical School in May, 1938. At the University he was prominent in football and social activities, was a member of the Students' Council and of the Pi Rho Sigma Fraternity. The funeral, with full military honors, was held from the home of his father, Dr. E. K. Maclellan on May 8th. Representatives of the army, the medical officers, and a large gathering of friends attended to pay final tribute. The services were conducted by Hon. Captain, Rev. W. C. Anderson assisted by Rev. Dr. Colin Kerr. There are left to survive him his wife, the former Delphine Wallace of St. Andrew's, N. B., two small daughters, Janet and Judith, his parents, Dr. and Mrs. E. K. Maclellan, one brother Lieutenant David Maclellan of the Princess Louise Fusiliers, one sister, Jean, and his grandmother, Mrs. W. E. Maclellan.

We regret to know from an announcement made from Ottawa, May 6th, of the death of Captain William Hazen Embree, M.D., formerly of West Amherst, N. S. On account of military reasons there were no details, but it is understood that Captain Embree's death was due to a sinking in mid-Atlantic. Captain Embree graduated from Dalhousie Medical School in May, 1938, and until the time of joining up he practised in Scotsburn, Pictou County. He is survived by his wife, Mrs. Maru Belle Embree, at present living in Kentville.

War or no War

Come to

THE ANNUAL MEETING

To be held at

THE CORNWALLIS INN

Kentville, N. S.

JULY 9th AND 10th

The programme is not yet complete but things are under way. We expect two guest speakers from the Canadian Medical Association and excellent papers have already been offered by members of our own Society. The Valley Medical Society will be hosts and a committee under the chairmanship of Dr. J. P. McGrath is already at work. The golf tournament will be held as usual and entertainment also provided for those who do not indulge in the ancient game. Bring along the wife and family and make this your holiday for the summer.

The Cornwallis Inn has set aside fifty double rooms for the meeting at usual rates. Make your reservation early either directly at the hotel or through the Secretary.

THE VISION OF DESCARTES

In describing the work of Descartes, Marvin (*The Living Past*, page 181) says his interest in the ultimate utility of well founded and systematic knowledge, especially in the parts affecting human life and health, was equal to that of his great English predecessor "Verulam." The purpose of scientific training was not to be limited by individual advantage. "We shall be able," Descartes says, "to find an art, by which, knowing the force and action of fire, water, air, stars, the heavens and all other objects, as clearly as we know the various trades of our artisans, we may be able to employ them in the same way for their appropriate uses, and make ourselves the masters and possessors of nature. And this will not be solely for the pleasure of enjoying with ease and by ingenious devices all the good things of the world, but principally for the preservation and improvement of human health, which is both the foundation of all other goods and the means of strengthening and quickening the spirit itself."

SUMMER DIARRHEA IN BABIES

Casec (calcium caseinate), which is almost wholly a combination of protein and calcium, offers a quickly effective method of treating all types of diarrhea, both in bottle-fed and breast-fed infants. For the former, the carbohydrate is temporarily omitted from the 24-hour formula and replaced with 8 level tablespoonfuls of Casec. Within a day or two the diarrhea will usually be arrested, and carbohydrate in the form of Dextrin-Maltose may safely be added to the formula and the Casec gradually eliminated. Three to six teaspoonfuls of a thin paste of Casec and water, given before each nursing, is well indicated for loose stools in breast-fed babies. Please send for samples to Mead Johnson & Company, Evansville, Indiana.

What a party it had been! Thompson's wife was away and he'd gathered a crowd of the boys to "celebrate."

Just on midnight, he saw one guest put on his hat and walk uncertainly towards the front door.

"Oi, ol' man," he protested. "You're not going home yet, are you?"

"No," said the other, steadying himself with one hand on the knob. "I—I'm jusht going to mish the lasht train. Be back in a minute."—*Roscommon Herald*, Ireland.

"The recruit was disappointed with his uniform. It seemed to fit nowhere. He was still trying to make the buttons of his tunic meet when he passed the Colonel on the parade ground. He quite forgot to salute.

"Pull yourself together," said the Colonel. "Don't you know you're wearing the King's uniform?"

"Oh, that's it," said the recruit. "I knew it wasn't meant for me."—*Yorkshire Post*.

A COUNTRY DOCTOR DEFINED

If you can set a fractured femur with a piece of string and a flat-iron and get as good results as the mechanical engineering staff of a city hospital at 10 per cent of their fee;

If you can drive through ten miles of mud to ease the little child of a dead-beat;

If you can do a podalic version on the kitchen table of a farm house with husband holding legs and grandma giving chloroform;

If you can diagnose tonsillitis from diphtheria with a laboratory forty-eight hours away;

If you can pull the three-pronged fishhook molar of the 250-pound hired man;

If you can maintain your equilibrium when the lordly specialist sneeringly refers to the general practitioner;

If you can change tires at 4 below at 4 a.m.;

If you can hold the chap with lumbago from taking back rubs for kidney trouble from the chiropractor;

Then, my boy, you are a Country Doctor.—H. W. Davis, in the *Kansas Medical Journal*.

"I have had three personal ideals. One to do the day's work well and not to bother about tomorrow. It has been urged that this is not a satisfactory ideal. It is; and there is not one which the student can carry with him into practice with greater effect. To it, more than to anything else, I owe whatever success I have had—to this power of settling down to the day's work and trying to do it to the best of one's ability, and letting the future take care of itself.

"The second ideal has been to act the Golden Rule, as far as in me lay, towards my professional brethren and towards the patients committed to my care.

"And the third has been to cultivate such a measure of equanimity as would enable me to bear success with humility, the affection of my friends without pride, and to be ready when the day of sorrow and grief came to meet it with courage befitting a man."—*Dr. William Osler*.

After listening (on the side) to an exciting episode of a world wide hunting trip by Theo. Roosevelt to his friend, in the corridor of a New York Hotel, the colored porter tapped him on the shoulder,—“Ted, did yu ever hav th hor'os?”

“No, Sambo, why?” “Den yu aint seen nothin, ner been nowhere.”—*From John Kingborn's Notice*.

The solemn silence at the funeral is suddenly pierced by the voice of our four-year-old nephew: “And where is grandma?”

“Your grandmother is in heaven now,” his aunt hastens to reply.

There is a long pause.

The boy doesn't seem to be satisfied with this explanation, and suddenly he bursts out: “You can't make me believe that!”

His aunt is horror struck: “Why, child . . .”

“There are no angels as old as grandma!”—*Nebelspalter, Rorschach*.

We pay tribute

to the Medical Officers of Health, to the Medical Profession generally, and to those public-spirited citizens, who, by their courage and unremitting efforts are overcoming the inherited prudery and ignorance of generations, by educating the public to the reality that syphilis is a communicable disease which must be treated, yes, and cured.

Quackery and its tragic sequelae are now being completely replaced by proper treatment at the hands of competent physicians.

In his efforts to stamp out this social scourge, the physician has at his disposal two well-tried and proven weapons.

NOVARSENOBENZOL BILLON

The outstanding brand of neoarsphenamine which is recognized as the accepted weapon in the intensive treatment of syphilis by intravenous injection.

Supplied in ampoules of the dry powder in doses of 0.15 gm., 0.30 gm., 0.45 gm., 0.60 gm., 0.75 gm. and 0.90 gm.

NEO-LUATOL

A bismuth compound in oily suspension which assures a high and effective level of bismuth in the blood stream when injected intramuscularly at weekly intervals.

Supplied in ampoules of 2 c.c., in boxes of 12, 50 and 100 ampoules also in bottles of 30 c.c.

Laboratory Poulenc Frères

OF CANADA LIMITED - MONTREAL