The Positive Diagnosis of Neurosis

ROBERT O. JONES M.D.
Halifax, N. S.

INCREASED knowledge concerning bodily symptoms which may arise from disturbed emotions has greatly complicated the problem of diagnosis and treatment of many patients. Despite the complications it has added materially to the understanding and correct handling of these people and has enlarged the doctor's function so that he can no longer be satisfied with the statement that no organic disease is present and let the matter go at that, but must carefully weigh the pros and cons of each case, not finally making a diagnosis of either organic disease or of nothing wrong, but realizing that every patient who has symptoms must have some cause for his complaints in whatever sphere of facts that cause may lie. Vomiting, for example, is just as real and needs treatment as much if it results from a toxemia of pregnancy, from a fear of pregnancy or from a delusion of pregnancy, and all should fall within the sphere of the physician.

What are the requirements for facing the problem presented by such patients? Certainly of most importance is the realization on the physician's part that such a problem exists—that the pain which is so elusive may well be the result of a chronic appendix, but it may also express chronic dissatisfaction or worry. The literature has long been filled with cautions against diagnosing neurosis when organic disease is present. Only recently has the equally good advice begun to appear—beware that one does not diagnose organic disease when actually the condition is on a neurotic basis. It is safe to say that there is far more danger of the average physician falling into this error than the former one. The abdomens of many neurotics bear eloquent testimony to the truth of this statement. Mistakes, of course, must occur on both sides, but with a little more attention to the patient and a little more consideration of the personality factors involved in disease, fewer mistakes would be made.

There is little general realization of the fact that the diagnosis of neurosis is just as positive a diagnosis as is the diagnosis of physical disease. The attitude of the fourth year medical students is characteristic. When asked when to refer a patient for psychiatric study, they invariably answer, "When the patient has complaints and organic disease has been ruled out." An answer of this kind disregards many of the basic facts of twentieth century medicine. In the first place, diagnosis is not an "either-or," thing—a patient has not either organic disease or functional disease. Epilepsy, for example, is certainly a disease with an organic foundation, but one knows with certainty that in many cases the onset of fits is directly dependent on psychogenic causes and that treatment is not adequate, which disregards the personality factor. By the same token, such diseases as peptic ulcer, asthma, and other allergic phenomena are diseases in which organic change is present, but where often the most hopeful line of therapy lies in the realm of personality facts. It then becomes the physician's duty to scrutinize his patient to determine what the important facts are, and what can be done about them, be they mentally determined or physically determined.

Secondly, a negative type of diagnosis is going to result in mistakes on both sides. No one of us is good enough to be able to state finally, "There is

no organic disease in this patient." Too often we are surprised by the appearance of definite physical signs a short time later. On the contrary, the finding of some physical lesion does not necessarily mean that the symptoms complained of are the result of the organic lesion. With our modern method of diagnosis, it is difficult to comb any person from head to foot and not find some evidence of pathology. Merely because we find diseased tonsils, however, does not give the right to say that the palpitation which the patient has is caused by those tonsils. Too many live lives of comparative comfort and ease with diseased tonsils, infected teeth, deviated nasal septums, systolic murmurs, calcifaction in our lungs, and a host of other unpleasant details to make us take too seriously the vast array of symptoms commonly attributed to these. Let us treat them for what they are worth to be sure—no one doubts that general health will be better after any of these things are removed, but let us not expect them to cure bed wetting, truancy, shyness, tremors, insomia and the hundred and one things that are so treated.

It then becomes important to state the positive side of the diagnosis of neurosis. What are the things that can be determined which are essential to such a diagnosis and without which such a diagnosis should be severely questioned? The examination and records of many neurotic patients seem to me to clearly give the answer. There are certain findings which are almost invariably present in the neurotic personality make-up, in the history of a neurotic, and in the examination of the neurotic person. All of these will not be present in one person but several should be present before the diagnosis of neurosis is made. These are found in the complaints, in the past history, in the family history, and in the examination of the patient.

The complaints of the neurotic patient are rarely confined to a single symptom. The patient with pain localized to one spot, without other complaints, is not apt to be neurotic. Typically the patient's complaints spread throughout the organ systems of the body.

The complaints associated with anxiety and consequent autonomic overactivity are present or have been present in the past in virtually every neurotic. Palpitation, shortness of breath, indigestion or diarrhea, tiredness and fatigue represent this action on the cardiovascular and gastro-intestinal system. Headaches at the vertex or as a band around the head are frequent. Sleep disturbences are of a typical nature, the patient lying awake for hours not being able to get to sleep and then dozing off, having a frightening dream to awaken terrified with pounding heart and shortness of breath.

All these things are symptoms of autonomic overactivity—the direct result of anxiety, and every neurotic has had these symptoms at some time in his illness. These anxiety symptoms may be changed into hysterical symptoms paralysis, ansesthesias which follow no anatomical pattern, or fits result. Finally, the anxiety symptoms may be relieved by the development of obsessive compulsive phenomena—insistent impulses to think or do certain things. Unless one of these general patterns can be demonstrated, a neurotic diagnosis cannot be substantiated.

As well as these positive findings, one must look for precipitating factors in the individual's environment. Neurosis do not come without cause—often we are unable to easily determine the important underlying cause which may lie outside the patient's awareness at the moment, but nearly always there are precipitating factors that are easily uncovered if the physician

shows his interest in such matters. One must remember that the importance of such factors vary with the individual's personality—what is sauce for the goose may not be sauce for the gander, but deadly poison. Thus, we must accept such factors in the light of the patient's interpretation and not our own.

In the past history there are nearly always indications of a neurotic personality make-up. This is particularly true in individuals of the later age period—from thirty on. The appearance of clearly neurotic symptoms in an individual, who has reached forty, with a previous good record of adjustment, should arouse strong suspicions of the onset of a psychosis or organic disease. In most neurotics some of the following history will be elicited. In childhood there have nearly always been neurotic traits, night terrors, fears of the dark, prolonged bed wetting, stuttering, or timidity and shyness. The health record is poor, there have been periods of vague illness, time from school or work for such things as being run down, a nervous breakdown, or anemia. All too frequently there is a history of several operations, often with relief of symptoms for a short period and then a return to the same complaint. Because of the above, school and work record may be unsatisfactory. Occasionally this part of the history clearly shows difficulties in interpersonal relationships—not taking part in the usual activities of the school or shifting jobs for wholly inadequate reasons. Occasionally there is resort to alcohol in an effort to improve adjustment. Frequently there is a history of excessive self medication, of being good customers of the patent medicine vendor, and this leads directly to a story of excessive body concern. The personality inventory contains a history of difficulty getting along with people, of feelings of inferiority, of not being able to mix, of feeling lonely or unwanted, or of compensations, such as attitudes of superiority.

The family history frequently lends supporting evidence—a father who was an alcoholic or deserted the family, or a mother who was nervous and perhaps had heart attacks by which she ruled the home. It is no time here to enter into the controversy as to how big a part heredity plays in such matters, but frequently a history of personality eccentricities in the parent may be of as much importance in such a study as a history of ill health.

In the patient's behavior during the interview, strong evidence for a diagnosis of neurosis is also obtained. The patient who talks long and freely of his symptoms, dramatizing everything as much as possible, is apt to be a hysteric. In taking the history of most neurotics, one hits tender spots, topics which perhaps bring tears to the patient's eyes, which bring a blush or cause hesitation in answers, which start the patient twisting his hands or wringing his handkerchief, which do not bring such responses from the ordinary patients. In the physical examination it is common to find evidence of overactivity in most of the physiological spheres—rapid pulse, temporary hypertension, sweating, blushing, and other manifestations of autonomic overactivity. There is as well frequently hyperactive reflexes and possibly vague and incredible sensory changes.

With the direction of attention to this sort of material, the diagnosis of a neurosis becomes a much more tangible thing—something that one can put weight in and believe in. It is difficult to be self confident enough to believe that our physical examination is accurate or complete enough to absolutely rule out any organic cause for symptoms—it is only by finding supporting evidence and psychological causation that we can have faith and guard against

error in the diagnosis of neurotic disturbance. The following cases taken from the consulting work done at the psychiatric clinic of the Dalhousie University Medical School are chosen as illustrative of neurotic and non-neurotic histories:

Case No 1.

A 21 year old single white girl, was admitted to the medical clinic complaining of swellings of her joints and hives, which had been present spasmodically since childhood, but had been worse than ever before, during the past few weeks. The medical clinician noted that she also had attacks in which her heart beat rapidly and dreams of an anxiety sort. She spontaneously mentioned that these latter symptoms occurred at the same time that the hives and swelling were worse.

Physical examination was negative, except for evidence of marked vasomotor instability—blushing, rapid pulse, and dermographia. Because of the absence of physical signs and the indications pointing toward a neurotic personality make-up, she was referred for psychiatric investigation.

In the psychiatric clinic the following history was obtained. She repeated the story of her swellings, saying that they became worse during the past week whenever she went to her work in the candy factory. She had, however, worked there for the past year with no difficulty, and there was no change in the work or in contact with new materials. She had been to a number of physicians previously, without relief. When the swellings came she said, "My whole body swells and I get sick to my stomach. All my veins get dark blue. I get a pain below my heart and my face turns all colors. Oh, it is terrible." She continued her story thus, "The least little excitement gets me down. I just cry and cry and can't control myself. I shake all over and my heart goes fast and I get sick to my stomach." She said she has terrible dreams of people chasing her, which woke her frightened, and with her heart pounding.

Her past history contained the information that she was nervous as a child and could not sleep, "even now I'm frightened to sleep in the dark without the light." At thirteen she had a period closely resembling the present, which had taken her out of school for six months. At first she was unable to account for this, but it finally developed that at this time the father had deserted the family after months of interfamilial strife. She had been moderately well until the present time. She had held her present job for about a year but she had never liked it. She had never been able to mix with girls, and now she was called upon to work in a place employing a thousand. Her education and appearance were certainly better than the average and the rest of the girls made fun of her, calling her stuck up and a snob. When enquiry was directed toward the sex life, she became obviously tense, wringing her handkerchief, and tears coming to her eyes. She rubbed at her hands and arms, with red welts appearing a moment later. She had been going with a boy for some months who all her friends disapproved of. They seemed to delight in finding gossipy stories to tell her about him. She felt there was truth in these stories but cared enough for him to go on. About a month previously he had persuaded her to have intercourse with him. She had yielded against her better principles, and since that time had refused to continue the intimacy. He had wished to go on and it seemed that the affair was going to break up on this point. Since that time she had felt extremely guilty concerning this. She had noted that her symptoms all appeared when she worried over these matters.

Comment: This case presented itself in the guise of an allergic reaction. Modern investigation leaves no doubt that there is a close inter-relationship between such phenomena and psychic events. There were other evidences of instability—the manner of describing the symptoms; the irradiation of symptoms; the anxiety dreams, which convinced the medical clinician that the case required psychiatric investigation. This resulted in further evidence of the neurotic background with a history of nervous reactions since childhood, a previous episode with symptoms similar to the present in a setting of the break up of the family and her father's desertion and a complicated life situation at the moment corresponding with the recent exacerbations of symptoms.

Case No. 2.

A 22 year old girl referred from the Medical Clinic where she had been unsuccessfully investigated and treated for eight months for pain starting in the chin and radiating up both sides of the face, and occasional bouts of fever. Despite complete medical investigation no physical cause had been discovered for the pain. Quite wisely, she was not referred to the psychiatric clinic until definite evidence appeared of neurotic personality make up when it became known that she had a compulsive form of stealing, frequently taking things and then returning them, in the boarding house where she worked. Psychiatric investigation repeated this history in great detail.

The attacks of pain coincided closely with her leaving home and coming to Halifax. She has been sleeping very poorly, getting to sleep late and waking early. She has been having very unpleasant sort of dreams; she complained of shortness of breath, particularly after getting into bed, when she felt that "my breath comes heavily." She has had the feeling that she must take things and cannot seem to do anything about it when the urge comes on. Once before she has had the same sort of sensation—when she was a six year old child in Grade 1 she felt she must steal lead pencils and did this for a short period of time without getting caught. There has been another period of neurotic breakdown two years previously, at which time she had to give up her work because of mild depression, nervousness, poor sleep, and loss of weight. She was at home for a year recovering from this and the present work is the first job she has had since.

Mental status examination revealed an extremely tense girl, always on the verge of tears, twisting her handkerchief, and picking at her lips until they bled. She looked depressed, and said she had been down in the dumps for some time. Her sleep had been poor. She stated she did her best to resist the impulse to take things but that it seemed impossible, and when she fought too hard against it then her pain recurred.

Investigation focused on her personality make up and she appeared to be the typical sort of person that develops such a reaction. She had been raised as a strict Salvation Armyist; always she had been an extremely serious girl with practically no fun in her life. Despite the fact she was 22 and had lived for some time in Halifax and previously in one of the larger provincial towns, she had never been to a movie in her life; she did not dance, did not play cards, and took no interest in any of the usual amusements of her group. A short time previous to the onset of her present symptoms she had been engaged to the only boy friend she had ever had, but he had died suddenly.

Comment: Here there was also adequate evidence for a positive neurotic diagnosis. While the complaints did not fit into any particular neurotic picture it was also extremely difficult to explain them on a physical basis. The personality structure gave adequate evidence of neurotic traits and the history of episodes at the age of six and sixteen provided confirmation of the likelihood of neurotic breakdown. As well there were psychogenic factors at work, for example, the leaving home and the sudden death of her fiance. She was evidently mildly depressed and depression in this type of personality almost always brings out excessive character traits such as she demonstrated. With treatment for the depression, she has made a satisfactory recovery.

Case No. 3.

A 29 year old woman, referred as a neurosis because of attacks of dizziness for which no physical basis could be found. The history was as follows:

She complained of dizzy spells and, on questioning, fatigue for the past month. The description of the spells was as follows. "It seems that everything in the room goes round, I get sick to my stomach and vomit occasionally. Vomiting seems to relieve the dizziness. It happens at any time of the day and often when I am alone. It is worse when I bend over or reach for something." She also gave a history of headache, which had been bad for about two weeks, three to four months previously, but now was not a matter of complaint. This headache was worse at night and frequently kept her awake.

She had gained a good deal of weight in the past few months. There was no gastro-intestinal disturbance; bowel function was good; there was no shortness of breath. She admitted palpitation some ten years previously, but it turned out that this was what the doctor had told her, and something of which she did not complain of herself. She had always been well in the past; there was no history of neurotic traits in childhood and the only significant thing was an illegitimate pregnancy some ten years previously. Physical examination revealed nothing beyond a rather obese woman with dry skin and puffy folds beneath the eyes. Despite the complaint of dizziness, and the completely negative physical examination, it was felt that the history and examination at the present time did not justify the diagnosis of neurosis, and she was asked to return for repeated examinations. She did not keep her next appointment, and it was three months before she returned.

Her complaints were much the same except for some slight increase in dizziness. Examination at this time revealed billateral optic neuritis, weakness of the left leg, marked dimunition of the visual vields and spinal fluid pressure of 300 mms. of water and a protein value of 80 mgms. In other words, the physical examination which had been negative three months previously, now was very definitely that of a cerebral tumour.

Comment: It is of interest to compare this history with those of neurosis already given. Here we have a vague physical complaint for which no organic cause can be found. However, there is no evidence for a positive diagnosis of neurosis. Her symptoms are pretty well limited to one complaint and the only other thing that could be detected, the headaches, were limited in time and were not a cause of complaint when examined.

During the examination there was no evidence of any marked emotional upset except when the illegitimate pregnancy was discussed. The gastro-

intestinal and cardio-vascular systems, which are most susceptible to autonomic over-action and which are practically always involved at some stage in a neurotic process, were completely free of complaint. The past history was innocent of anything suggestive of neurotic disturbance. Sleep was good. Therefore it was felt to be organic and subsequent evidence proved the value of insisting that neurosis is a positive diagnosis and cannot be made on the basis of negative physical findings.

Summary

1. The diagnosis of neurosis is not:
   (a) A negative one based on the absence of organic changes.
   (b) It is not an "either-or" diagnosis. Neurosis and organic disease may coincide.
2. The diagnosis of neurosis must be based on positive evidence if error is to be avoided. This evidence is:
   (a) There must be typically neurotic symptoms:—
      1. Anxiety: palpitations, gastro-intestinal disturbance, insomnia and a host of others. This is the most important grouping and occurs at some time in every neurosis.
      2. Hysteria: paralysis, anaesthesias, loss or exaggeration of function of some kind.
      3. Obsessive compulsive phenomena: persistent worries or impulses.
   (b) The past history must give evidence of previous involvement—neurotic traits in childhood, poor health record of vague diseases or previous breakdown.
   (c) There must be adequate precipitating causes.
   (d) There must be evidence in the examination of emotional over-activity on certain especially tender topics.
   (e) The family history frequently lends supporting evidence, either as an hereditary or environmental factor.

3. If attention is directed to these factors the diagnosis of neurosis can be confidently made or ruled out just as any other medical syndrome can.

BIBLIOGRAPHY

1. Friess and Nelson; Psychoneurotics—Five Years Later: *American Journal of Medical Sciences*—April 1942.
2. Welscher I.S., Diagnosis of Neurosis and Organic Disease. Medical Clinics of North America. November, 1937.
3. Romano J., Diagnostic Errors in the Neurosis—Medical Clinics of North America. September 1940.
4. Moore M., and Abbot J. A., Diagnosis of Neurosis and Psychosis. Medical Clinics of North America. September, 1940.
5. Wilson H., The Neurotics Physical Symptoms; Their Early Recognition. *The Practitioner*. June 1935.
6. Clendening L., The Greatest Problem of the Internist. *Journal of the Missouri Medical Association*. Vol. 27. 1930.
7. Forsyth D., Diagnosis of Neurotic Conditions in General Practice. *British Medical Journal*. February 27, 1932.

Chronic Right-Sided Pain

H. B. Atlee, M.D.

GO into any group of self-respecting surgeons—by which is meant surgeons who question their godhead occasionally—and murmur the words: "Chronic right-sided pain." A look of confusion not untinctured with pain will cross the assembled faces. "What enemy," the expression seems to ask, "is doing this thing to us?" For it is a fact that all we like sheep have gone astray when it comes to a pain in the right side, particularly if the pain happens to be in a woman's right side. Some of us have been led astray through ignorance, some of us through fear, but none of us have escaped, certainly not your present commentator.

Since this is a fact that only the most intransigent will deny, and since in my capacity as a consultant at the Victoria General Hospital I am asked to give an opinion upon this matter more often than upon any other, I propose herewith to set down certain impressions that have come to me along the trying path of error. I set them down in all humility. Forgive me if, from time to time, the didactic note, the ring of conviction, enters my voice; that will not be my better self, only my false front speaking. For in truth I, believe that, despite our long preoccupation with it surgically, the iliac fossa offers still an ample field for research. And since it is a corner of anatomy in which not only the general surgeon, but also the urologist, the gynecologist, the internist and even the orthopedist has a vested interest, truth has perhaps fallen between too many stools. In this last connection let me state in fairness that those with a vested interest are only too willing to pass over their buck to the other vestees. For instance, the surgeon is quite ready to concede that the ovaries are at fault, the gynecologist the appendix, the urologist the pelvic floor, and the internist abdominal ptosis (whatever in Allah's wisdom that is!)

What happens to the case with right-sided pain is as follows—and I merely quote what happens very frequently in the public wards of the Victoria General Hospital (and all other hospitals the world over.) The patient is investigated by the individuals above mentioned. Everything is found approximately normal to gross examination. All the tests are normal. The patient is finally left more or less in the general surgeon's lap and he argues something like this: "If I don't take out that appendix some other fool will . . .and it *may be* her appendix. There have been cases like this where the removal of the appendix caused a cure." Admitting that this is not a scientific reason for an appendectomy, nevertheless it is an artful one—and can we say that even yet surgery is more than an art? So out comes the appendix and—if she is the patient I am talking about—she gets the pain back shortly after she takes up her active life again.

On her next trip to hospital the diagnostic field has been narrowed. If we didn't know it before we certainly know now that she hasn't got, nor ever did have, chronic appendicitis. She therefore now becomes a candidate for the gynecologist. If it isn't her appendix it must be her ovary. What happens to her now depends on your gynecologist. He may be the type (Allah increase his seed) who does not believe in the emergence of chronically painful ovaries

apart from chronic pelvic inflammatory disease. He may be the type who does and who in turn divides into the man who, while holding in abhorrence the thought of a total ovariectomy, has no such feeling towards a partial resection, and the fellow who thinks no more of hoicking out an ovary than little Jack Horner of pulling out his plum. So perhaps our patient loses all or a part of one ovary and still has not lost her pain. (And believe me many such live and have their tortured being).

Perhaps now the urologist has his turn, with a kink of the ureter, or a stenosis of the urethra. So the sounds and the catheters are passed. One says this for the urologist: he does not, as a rule, look upon the abalation of a kidney with the same light-heartedness your surgeion views his appendix and your gynecologist an ovary. But the woman, despite dilatations and catheterizations, still has her pain. (And many such dree their sad weirds)

What shall we do for her now? Can it be ptosis—and should she have a belt? (At last operations are out here). Could it be the uterus?—what about a little hysterectomy, brother?—you know, one of those subtotals, where for a lack of guts you leave half the organ behind. (And often the worst half). Or how about the pelvic floor? There's a wee spot of cystocele when she strains —not much of course—but just enough so you could grab it with an Allis. Could something down there be dragging on the round ligaments that a good old round trip would just fix up dandy? Or—gosh-sakes—could it be a chronic gall-bladder all the time?

Or perhaps by this time, as a result of one thing or another surgically with no relief, our patient is showing signs of what even she herself describes as "nervousness." It might just be worth while letting an internist see her. He might hit on something. The internist goes over her. He gets the hunch it might be a sort of colitis. Or perhaps, recognizing the neurotic elements now in the picture, he throws up his hand scientifically and like the surgeon some time ago now becomes artful with a bromide or barbiturate.

Does this argument sound specious and unreal to you, brother? It is very real to me. It is very real to any surgeon, any gynecologist, any urologist, any internist, any psychiatrist, attached to any large, investigating hospital. The case I have described—a case that has run all the gauntlets described—is too, too common in such hospitals, and in all communities where surgery is done. But I think it can fairly be said that the better the hospital and the wiser the men on its staff the less likely is the patient to have passed so many gauntlets.

With all this sad background in mind, let us now approach closer to the question. Let us talk about the pain in connection with each likely diagnosis.

1. Chronic appendicitis.
2. Painful right ovary, and–or Fallopian tube.
3. Painful right ureter.
4. Constipation.
5. Painful cecum.
6. Such gynecological conditions as torn and eroded cervix, prolapse, retroverted uterus with prolapsed painful ovaries.
7. Chronic right-sided pain as the manifestation of a neurosis.

Chronic Appendicitis: Some surgeons hold that there aint no such animal—just as some gynecologists hold that there's no such entity as a painful ovary. Perhaps the best answer to that is the fact that first class surgeons still continue

to remove appendices on such a diagnosis. They certainly wouldn't do this if the removal of the appendix in some of these cases didn't relieve the symptoms. And while it is not entirely a proof of pathology that removal of certain tissue removes certain symptoms located in the vicinity of that tissue, we can argue that, since surgery is still an art rather than an exact science, one is justified in continuing to perform a surgical procedure that relieves symptoms.

But here is a fact to bear in mind: The better the surgeon the fewer chronic appendices he removes, and he does the removal not as a premier procedure but only after having tried all the other possible therapeusis. And here is another fact: one cannot estimate correctly the result of an operation by the way the patient feels on leaving hospital—or even by the way he or she feels up to the time of going back to active work again. Let me give an illustration of what I mean. Some years ago I used to do a fair number of presacral sympathectomies for dysmenorrhea and chronic pelvic pain. If I had estimated the value of the operation on how the patient felt a month later I would still be living in a fool's paradise; but estimating it, as I finally did through a follow-up on about 40 cases, by the way the patients felt a year later, I discovered that I had cured only 50% In other words here was an operation of some value, but not a panacea.

Why do some of these patients feel better after an appendectomy, only to slip back to pain when they get into their life's work again? Partly it must be due to the psychological effect of an operation. The patient expects to feel better and it takes a little while for her to realize that she is not better. And partly it is due to the effect of the rest in bed and the freedom from stress, worry, etc., in the period of post-operative recuperation.

The impression I get from the number of cases I see who have had an appendectomy done for chronic iliac pain is that only a small number are cured. What happens to those that aren't cured? Unfortunately, too many of them are labelled

Adhesions

I place the damned word purposely there by itself because it is a pariah among diagnoses and should be so pilloried. In fact, I believe that the time has come when we should teach that "adhesions" are never a cause of chronic pain, (although I realize that "never" is a word that should never be used in speaking about human pathology) You make the statement that such and such a thing never occurs in such and such a condition and within a month you run into two or three cases where it does. I used to believe, for instance, that carcinoma of the cervix never occurred except in women who had been pregnant; now I know differently.

But let us look the diagnosis of "adhesions" straight in the face for our soul's sake. Let us first of all ask ourselves why we should postulate "adhesions" for a painful condition that was present before the operation that could have caused them was performed? Why not admit that the operation was a plain failure: it did not cure the pain. And then let us remember the following:

1. That very few abdominal operations are done without some adhesions resulting.
2. That pain resulting in a diagnosis of "adhesions" is present in cases which when reopened are found to have no adhesions at all.

3. That cases in which the lower abdomen is hopelessly matted with adhesions may have no pain whatsoever.

And then let us ask ourselves if we ever relieved pain by releasing adhesions? I don't mean the pain that is sometimes associated with a partial obstruction due to adhesions. I mean the sort of pain we are talking about. Personally, I have never known of a case that was relieved of chronic iliac pain by having adhesions separated. It may happen, but I have never, never seen it. So why do we continue to label falsely and absurdly these cases in which an appendectomy that should not have been done has failed to cure a condition that was not due to the appendix, and is not now due to adhesions?

What type of case can fairly be labeled chronic appendicitis? Being only a gynecologist I tread with diffidence this pathway, acknowledging humbly that I do not speak entirely with the voice of angels. But I would say that chronic right-sided pain in which there was no history of even a mild acute attack of appendicitis or no history of indigestion with pain, is unlikely to be chronic appendicitis, even if the patient is tender over the appendix on palpation. Indeed, I saw in consultation only last week a case of chronic right-sided pain in which there had been attacks with vomiting that sounded acute enough in which the appendix was found at operation to be perfectly normal, and a case yesterday where what I took to be appendix turned out to be a mild chronic pelvic inflamatory disease. The diagnosis of chronic appendicitis should be arrived at then in an atmosphere of the utmost scepticism—and humility. In fact I think it would be a very good rule if all surgeons held a consultation with at least one other surgeon before operating on a diagnosis of chronic appendicitis. It would be a better rule if an internist and a psychiatrist were included.

Painful Right Ovary and–or Fallopian Tube: The history of pelvic surgery is strewn with innocent, ablated ovaries: ovaries removed for a right-sided pain for the most part wrongly diagnosed. I do not say that there is not the rare ovary which, in the absence of any demonstrable pathology in it, causes right iliac pain, but I believe it is extremely rare. In 20 years I have removed not more than 10 such, in two of which cases at least the subsequent story gave the lie to that diagnosis.

This is the sort of picture you get. The woman complains of more or less constant iliac pain which is made worse just before and during the first day or two of menstruation. When you examine her in the interval you find that the ovary is tender to bimanual examination. Such a picture seems to justify the removal of an ovary—and yet in two such cases that I know of removal of the ovary did not so justify it. On the other hand in four that I know of it did. The point I wish to insist on is that right iliac pain due to a tender ovary alone is a rare condition—one that certainly does not justify indiscriminate ovariectomy.

Nor does it justify that commonly performed operation "removal of a cyst on the ovary." Which is something I must say more about at this point. I don't know how many women have told me that they have had done on them the operation of appendectomy plus "removal of a cyst on the ovary", but the number must be close to a hundred. I recall hearing a paper before a local medical society in which the surgeon stated that he had removed over a hundred such "cysts on the ovary". What is the cyst that is removed? I believe it is

nothing more than a ripening Graafian follicle—shall we say a fairly ripe Graafias follicle. I don't think we realize that the fully ripe follicle may be almost an big as the ovary itself, but this is a fact —as I have seen on many occasions. So here is a surgeon who operates on a diagnosis of chronic appendicitis. He finds a normal appendix and, troubled in the conscience, looks further for pathology only to espy this "cyst on the ovary"—this ripening follicle. So he hoicks it out, too. I have a suspicion that if all the ripening Graafian follicles that were removed on a diagnosis of "cyst on the ovary" in the course of appendectomies, were laid end to end they would reach almost to the throne of God. I get along very well without removing them, but when I find one of them in the course of a pelvic operation I always show it to my students and point the proper moral. That the seed of truth thus sown falls upon stony ground I have alas discovered all too often.

It is my considered opinion therefore that ovarian pain, in the absence of demonstrable pathology, is rare. But pain located in the ovarian region—and perhaps actually ovarian—is present in chronic pelvic inflammatory disease—and it is present even where such disease is so small in bulk that it cannot be palpated as a mass on bimanual. On quite a few occasions where I was not able to demonstrate such a mass before operation I discovered the tube and ovary tied up when the abdomen was opened. That the pain is probably mainly tubal is shown by the fact that if only the tube is removed the pain disappears as completely as if tube and ovary are.

Painful Right Ureter: Here again I speak with diffidence, but I have found that the following three conditions can cause right iliac pain.

1. Chronic pyelitis
2. Stone in the ureter
3. Stenosis of the urethra

Chronic pyelitis usually presents itself as a tubercular manifestation and the pain in the side is masked by the great pain on micturition. My face was once made very red because I sent out of hospital as a mild pelvic inflammation what was later proved to be a stone in the right ureter. My own experience with cases of stenosis of the urethra consists in having had the diagnosis made by the urologist in occasional cases of chronic right-sided pain for which I could find no cause. I have always viewed this diagnosis with some scepticism—perhaps because of my foregoing experiences—but I am assured that cure has followed a properly followed-up dilatation.

Constipation: Somebody has said that all women are constipated, and there is just enough truth in this to make it a first-class wise-crack. But it is a fact that surgeons by and large—and this includes gynecologists and urologists—do not take sufficiently into account. For it is my firm conviction that constipation is far more often a cause of right (and left) iliac pain than chronic appendicitis, or chronic oophoritis, or "cyst on the ovary"—or all three lumped together. Dr. S. R. Johnston once confided in me a treatment for these cases that consisted in part of copious colonic irrigations, with which I have caused the chronic right-sided pain to disappear (at least temporarily) in many a constipated maiden. Time and again I have been asked to see in consultation a case of chronic right-sided pain in which the question: "Are you constipated?" was answered: "Yes." And in which the balls of feces in the rectum and sigmoid were so extensive they gave the impression of pelvic masses.

Constipation as a cause of chronic right-sided pain is, I believe, tied up with the next condition in my list:

Painful Caecum: Because of the relative frequency of acute appendicitis, we are inclined to state when we get tenderness over the appendix region that the patient has a tender appendix. Why shouldn't it be a tender caecum? In fact, I believe that most of the tenderness found in the appendix region of these chronic right-siders is due to the caecum rather than the appendix. I don't want to be dogmatic on this point. I may be dead wrong. But it is my distinct impression that what I have said above is the truth.

Why does the caecum get painful? I have postulated to myself as a working hypothesis two factors, both of which probably act in the same way. (1) constipation, by holding up fecal matter in the caecum, causes some localized colitis which is reflected in tenderness. (2) irritating food elements (roughage) and irritating purgatives act in the same way, *and in some cases they actually increase the constipation.*

I don't believe it is generally realized to what an extent roughage and irritant purgatives are responsible for right-sided chronic pain. The whole matter of constipation and its sequelae lies in such a chaos of ignorance that this is no wonder. It is so easy to prescribe a purgative when what is needed is advice on a new set of hygienic rules. It is so easy in these days of radio exhortation for the patient herself to fly to Ex-lax rather than exercise, to the tablet rather than the toilet.

But this irritation of the caecum can also cause an acute condition. Let me recite the story of one such. This was a young woman who used to get attacks of acute abdominal pain, in the course of some of which she would vomit. I saw her in two of them but could not persuade myself she had appendicitis. She moved away to another Canadian city and had another attack. Her appendix was removed. Three months later she had another attack. She finally saw a doctor who discovered that these attacks came on after eating a lot of roughage. He ordered her on to a bland diet and she has never had an attack since—except when she slipped off her diet. Only about six weeks ago I saw a similar case that came on after eating a number of bran muffins. (And let me say here that I believe we should return all our bran to the creatures it was first manufactured for—cattle. It is, certainly for some people, a dangerous food.)

I think it is well worth while trying these people with tender caecums (or would you prefer caeca?) on a bland diet. You will be surprised—as I have been—to find that on such a diet the constipation, which for years had dogged the patient, often improves miraculously. (I won't go into this in more detail here since I intend, if God spares me and Editors of the Bulletin tolerate me, to write an article on constipation one of these days that will elaborate this matter.) But what I will say here is that I believe as axiom Number Two (Ax. 1. having been that no patient should have her appendix removed on a diagnosis of chronic appendicitis without a consultation) we should adopt the following: No patient should have her appendix removed on a diagnosis of chronic appendicitis until she had been put on a bland diet and had her constipation cured.

Such Gynecological conditions as torn and eroded cervix, prolapse, retroverted uterus with painful prolapsed ovaries, etc. A torn and eroded cervix should be cleared

up either by cauterizarion or repair on its own merits alone. Prolapse should be cured as prolapse. Both these conditions can cause chronic iliac pain, but since, as I have stated, both should be treated on their own merits we are not especially concerned with the iliac pain as an entity, unless it persists after the other conditions are cured—in which case it is due to some other cause.

One should also view with a great deal of scepticism chronic iliac pain when it is the only symptom present in a woman who has a retroverted uterus. I think I would go so far as to say that in such a case the pain is not due to the retroversion at all, but to some other cause. There are two real indications for anteverting a retroverted uterus (1) sterility (2) when deep dyspareunia is present. Occasionally in the latter condition there is also iliac pain which clears up after a suspension. But—I repeat —suspension for chronic iliac pain is very bad routine medicine.

Right Iliac Pain as Manifestation of a Neurosis: One hesitates to label a patient with right-sided chronic pain as a neurotic, but it is a fact that patients with this type of pain present many of the stigmata of a neurosis. To such an extent have these been present in some of the cases I have encountered that I wondered if the pain itself was not the result of a mental rather than a physical process. We are so obsessed with the physical. All our training forces us to seek causation through that field and that field alone. But the more I see of suffering humanity the more I am convinced that we will never really do right by a large number of those who consult us until we rid ourselves of this obsession and strike out into the field of mental pathology.

In a world such as ours it stands to reason that thousands of persons fail to adjust themselves to their situation. Out of that maladjustment all sorts of strange human behaviour arises. If a person can become paralyzed in both legs, or lose his sight, or develop areas of complete anesthesia through maladjustment, why can't that person develop pain in the right iliac fossax for the same reason? I have seen, let me repeat, patients whose right-sided chronic pain seemed to me to be due to this cause.

In the unfolding of the subject under discussion I have laid myself open to the criticism that I have not been very constructive. That's O.K. by me. There's a time when criticism should be destructive. And I fear it would take something far more devastating than any words of mine to remove that itch which attacks surgical fingers at the merest suspicion of right iliac pain. But perhaps Allah will cherish me for having entertained the pious hope.

The Relationship of Coronary Disease To The Myxoedema Heart and Thyroid Gland Treatment*

S. T. Laufer, M.D.

THE heart in myxoedema shows some special characteristic features, first described by Zondek[1], namely, dilatation of both chambers, sluggish and slow action, and low voltage of the electro-cardiographic manifestations often associated with inverted T waves in one or more leads. Such findings are not accidental and their occurrence is confirmed by the great majority of the authors. Following thyroid, the heart returns to nearer normal size and so do the other features. George Fahr[2] has added to the above signs congestive failure with cardiac dropsy, chiefly because it is relieved by the administration of thyroid; but this view is not accepted by most investigators. Congestive heart failure in myxoedematous patients is a complication rather than a symptom of the pure myxoedema heart, for the above mentioned features must be considered as a *part* rather than a complication of the disease.

With regard to the occurrence of these signs, there is still some disagreement, probably because milder forms of hypothyroidism are often included under the name myxoedema. For instance, only when the B.M.R. drops to -25, or lower, will abnormal electrocardiographic manifestations be found (Ohler and Abramson).[3]

A second important factor which has prevented unanimity of opinion is that myxoedema is often associated or complicated by coronary disease and heart failure, and as the latter may lead to more pronounced changes in the size of the heart the question may be brought up as to whether such enlargement be considered a complication rather than a part of the myxoedema heart. Coronary disease often complicates myxoedema and the exact relationship between these two conditions has not yet been determined.

I now should like to discuss this point in more detail, as it will enable us to correlate the relationship of angina pectoris and myocardial infarction to the myxoedema heart.

While in untreated cases of myxoedema a few rare reports of angina pectoris are recorded in the literature, its occurrence during treatment is mentioned more frequently, and indiscriminate use of thyroid gland has been held responsible for it and myocardial infarction. Therefore, any discussion of the relationship of coronary disease to myxoedema is intimately bound up with the role which thyroid gland treatment plays in the occurrence of such complications. Thus let us consider these two main aspects:

1. The condition of the coronary arteries in cases of myxoedema;
2. The relationship of thyroid gland to the production and manifestation of angina pectoris and myocardial infarction.

In view of the myxoedematous changes found in the myocardium post-mortem and from experimental findings, one would expect more frequent reports concerning the occurrence of coronary sclerosis and insufficiency in cases of this type. The sparsity of such reports however, can be partially explained as follows:

*Read before the Halifax Branch of the Nova Scotia Medical Society

1. Myxoedema occurs spontaneously at all periods of life, but more commonly at an age when arteriosclerosis may be a concomitant lesion;

2. In untreated cases, the lowered metabolism reduces the work of the heart to so great an extent that cardiac symptoms may not be manifested at all;

3. On account of this latter fact too little attention has been given to the study of the cardio-vascular system and the total number of autopsies in which it has been studied is very small;

4. The difficulty in determining clinically the presence of coronary artery disease at an early stage.

So far, only a few cases of myxoedema in younger people with extensive sclerotic changes of the coronary vessels, have been published (Fishberg,[4] Willius,[5] Keefer)[6]. These reports as well as the occurrence of angina pectoris and myocardial infarction following treatment with thyroid gland, has led to the assumption of a direct relationship between coronary sclerosis and myxoedema. Higgins,[7] for instance, considers coronary artery disease to be *aggravated* by myxoedema and recalls the striking analogy between the occurrence of arterio sclerosis in diabetes and myxoedema. Diabetes is known to lessen the immunity to arterio sclerosis (by its metabolic changes) and hypercholesterolemia is accused by this author of playing an important role in the etiology of the vascular changes in myxoedema. Bartels and Bell[8] think that myxoedema may be a causative agent in the production of coronary disease, while Means,[9] on the other hand, considers myocardial infarction an accidental association of the disease.

From the above results, it is easily understandable, gentlemen, how difficult it is to ascertain the exact relationship between coronary disease and myxoedema. The number of autopsies to date is too small, the importance of hypercholesterolemia in the causation of arterio sclerosis has not been definitely proven, and the few cases of myxoedema in younger people do not allow conclusions to be drawn. However, one fact is evident: The incidence of coronary artery disease appears to be a high one and it constitutes a very important complicating factor of myxoedema, especially in the treated cases. And this leads me to my second point, namely, the relation of thyroid treatment to the occurrence of angina pectoris and myocardial infarction.

With this regard it must be stated that although treatment should be started as early as possible, and this may check further advancement of the disease, it often does *not* prevent the manifestation of an anginal syndrome. For the latter may occur from months to years after the substitution therapy has been started, so much so that it suggests that coronary sclerosis has been present in the individuals, independently of treatment. Bartels and Bell rightly state that "the change in the coronary arteries is not reversible but once started continues to progress."

Evidently early treatment may be able, in analogy to diabetes, to postpone the manifestation of coronary disease. The high incidence of coronary disease in cases of myxoedema, as given for instance by Bartels and Bell of the Lahey Clinic as being 25% of their cases as compared with 3% in diabetes, indicates only that the development of arterio sclerosis is excessive in myxoedema and has become the major cause of death among cases of this type. In consequence we might expect that earlier diagnosis of hypothyroidism and subsequently earlier institution of treatment, will postpone premature develop-

ment of arterio-sclerosis and lengthen the expectation of life of the average case. Moreover, diagnosis and treatment in those cases of hypothyroidism in which coronary disease is a complicating factor, often presents serious difficulties. Even in cases of advanced coronary disease the symptoms of the latter may be overshadowed by the myxoedematous picture and the institution of gland treatment, in cases of this type, often proves to be a *disastrous* experience, for by improving the myxoedema the treatment with thyroid uncovers the underlying coronary disease which manifests itself either in the form of angina pectoris or coronary insufficiency, often ending in sudden death. The thyroid treatment then, unjustly is blamed for causing death. In cases of this type thyroid, in my opinion, may be considered an important diagnostic weapon, in disclosing the underlying coronary disease. It may be compared with the exertion test in angina pectoris, but only is somewhat more dangerous. The heart which has been working at a subnormal level, is elevated to nearer normal activity, thus increasing the blood velocity and cardiac output that creates a condition to which the stenosed arteries cannot respond and angina pectoris or coronary insufficiency ensues.

I have already said that even advanced coronary sclerosis may be overshadowed by the myxoedematous picture. But in rare cases the opposite may be observed, namely, that cardiac failure with dropsy may be so pronounced as to mask the hypothyroid condition, which is the real underlying cause, especially when the latter is associated with coronary sclerosis, as it is commonly seen in cases of this type. The following case report may briefly illustrate this point:

A sixty-eight years old female was admitted to the Halifax Infirmary with generalised anasarca, her feet and thighs being enormously swollen and there was ascites and an enlarged liver. She was very dyspnoeic and often had Cheyne-Stokes breathing. The heart was very enlarged and the heart rate irregular and slow. The Blood pressure was 170/88. The ecg revealed a low voltage of the ventricle complex and inverted T waves in leads one and three, isoelectic T in lead two. There was auricular fibrillation and the heart rate was 62-66 per minute. X-ray examination showed an enormously enlarged heart in all its chambers.

The bradycardia is an unusual finding, because digitalis had never previously been given to the patient. An M.B.R. could not be taken on account of the symptoms of advanced failure. However, digitalis not only was ineffective and was not tolerated, but Salyrgan and Thyroid gland proved to be effective. As a result of this treatment the patient lost about twenty liters of fluid, the oedema almost disappearing, and her general mental condition improving, she was discharged in a much better condition.

This case is an example of cardiac failure in myxoedema, complicated by coronary sclerosis, and the effectiveness of thyroid treatment in such cases.

In conclusion of this first part, it might be said: Our clinical data, autopsies, and statistics, are too few to allow inferences to be drawn upon the causative influence of myxoedema in the production of coronary artery disease and arterio-sclerosis as a whole. The high incidence of coronary disease, however, is striking and it constitutes a serious complication of the disease. With regard to thyroid treatment, we are often between the Devil and the Deep Sea, as to which course to take: To treat the myxoedema and induce anginal distress or let nature take its course.

After these preliminary considerations, I should like to discuss in somewhat more detail, the incidence of angina pectoris in cases of myxoedema and their relation to thyroid gland treatment.

Cases of hypothroidism in which anginal symptoms are relieved by thyroid gland, have been reported on a few occasions and the anginal syndrome did not appear as long as the M.B.R. was maintained at a certain more or less fixed level for each individual case. In the case report of Beaumont and Robertson,[10] for instance, a severe effort syndrome was the only clinical evidence of myxoedema and the B.M.R. around -10 and -17. Over-dosage or under-dosage of thyroid caused a recurrence of pain. In Beach's[11] case, the patient was kept free of angina for several years with a B.M.R. at -2 and -10. The response to thyroid treatment, in cases of this type, suggests that arterial and neuromuscular changes as well, take place in the heart of such cases.

Angina pectoris following thyroid treatment, has been observed and reported by several authors. Its occurrence is not an uncommon finding, especially when the B.M.R. is raised to a higher level than can be tolerated. Here, pain evidently results from an increased flow of the blood through sclerotic narrowed vessels (Blumgart et al).[12]

Myocardial infarction during thyroid gland treatment, has also been described but does not appear to be a frequent complication of myxoedema, if we consider the relatively small number of reported cases. Smyth[13] has reviewed eight fatal cases from the American literature, and added one case. Nichol[14] has reported one additional case and four are mentioned by Bartels and Bell, all of which died, from three weeks to six months after institution of thyroid treatment. To the above is added the following case, which I have still under observation.

The patient, a forty-seven years old male, consulted me first in May, 1941. I am not going to describe in detail his history and shall mention only the following data: His remote history tells of scarlet fever and acute rheumatism while the actual complaints of the present illness, can be dated backward for approximately ten years, when he began to display lack of energy, loss of memory and cold hands. Thyroid was first given four years ago but an M.B.R. was never taken. He took thyroid for a certain time, felt somewhat better and stopped it then for an indefinite period: last year he started to take it again, for a few months, but omitted it because of pruritus that appeared following its use. When I first saw him he stated that he had gained weight again (15 lbs.), he complained of nervousness and headache and that his voice had become hoarse. There was no shortness of breath or signs of cardiac distress.

On physical examination the skin was found very dry and scaly, the face puffy and the extremities cold. The thyroid gland was not palpable and the cardio vascular system apparently normal; blood pressure—130/90; pulse rate slow (60 per minute) and the B.M.R. was —32½. Haemoglobin 80%.

He was started on thyroid (dessicated gland) ½gr. daily and later ½ gr. b.i.d. and lost some 8-10 lbs. and felt better, becoming more alert and interested in his environment. Forty days later, following over exertion (while lifting his car,) he was taken with praecordial distress, which grew in intensity. Seen some three hours later, his blood pressure was 160/90 and pulse rate 60 per minute. The pain persisted but there was no perspiration or symptoms of shock. Next day the ecg confirmed the suspected coronary occlusion, and acute myocardial infraction of the anterior wall of the left ventricle was diagnosed. The blood

pressure fell gradually, the pulse rate went up gradually and the disease took its regular course.

Thyroid gland was omitted only for a short time, for reasons which will be discussed later, and the patient is now actually attending his regular activities. He is taking $\frac{1}{4}$ to $\frac{1}{3}$ gr. of dessicated thyroid U.S.P. daily and his M.B.R. varied from —19 to—25.

As regards dosage of thyroid when myocardial infarction has occurred, while the cases reviewed and the additional one reported by Smyth, received rather high doses, in the four cases of Bartels and Bell and the present one it occurred following small doses. The age at which myocardial infarction occurred was between forty-six and sixty-four years, and the B.M.R. varied from —21 to—41.

On the basis of reported cases, the number with myocardial infarction in untreated cases of myxoedema, appears to be still smaller. But in view of the fact that myocardial infarction may occur without pain (especially in cases with myxododma) one may expect greater number of cases, although it is unlikely that myxoedema can be overlooked now-a-days and remain untreated.

The occurrence of myocardial infarction following thyroid gland has led to warnings as to how to approach treatment in cases of myxoedema and reference often is made to the importance of "careful estimation of the cardio vascular status," before treatment is given.

And here the question arises: Are we able to make an exact estimation of the status of the cardio vascular system in cases of myxoedema? What are the most important elements to secure proper evaluation?

Of the cardio vascular signs found in cases of myxoedema the enlargement and sluggish heart action, both evident by X-ray examination, may be present in advanced myxoedema without coronary disease and then subside after thyroid gland has been given. These signs do not provide any indication of a concomitant coronary sclerosis. Moreover, in the case presented above, the heart was not much enlarged.

More value should be given to the presence of hypertension, for the latter constitutes an important factor in the production or aggravation of coronary disease. But not every case with hypertension necessarily leads to coronary sclerosis. One would expect that the ecg may give more indication as to the presence of coronary sclerosis. But, although it may be considered pathognomonic of true myxoedema, so much so that it can be used to check the progress of thyroid treatment, the ecg has with few exceptions but a limited value; because wherever negative T waves are found associated with a low voltage, the significance of this pattern will be evident, only *after* thyroid treatment has been given for a certain time, and will be of a positive value only if the T waves remain inverted. However, the presence of a high amplitude of the ventricle complexes *with* inversion of the T waves in cases of a definite myxoedematous syndrome can be interpreted as expression of a concomitant coronary sclerosis (Ohler and Abramson). In such cases the T-waves do not disappear following thyroid treatment.

From the above, therefore, it is evident that our clinical and laboratory means are often insufficient to give the requested "careful estimate of the cardio vascular status".

Thus, it appears to me that the best indicator of a concomitant coronary disease in cases of myxoedema, is to be found in the thyroid treatment itself.

It is clear that whenever an anginal syndrome is manifested following thyroid, this indicates the presence of coronary disease and our attention will have to be directed to the minimal complaints of praecordial distress. The control of the myxoedema, in cases of this type, will have to be a limited one, for very often it will be better to keep the B.M.R. low and avoid cardiac complications. This is also borne out from numerous observations in which untowards effects arose from raising the B.M.R. over a certain limit.

In this connection I should like to advance a further warning. Caution with thyroid should be emphasized, not only in treatment of cases with myxoedema and a history of anginal distress, or in previously untreated cases of advanced myxoedema; discontinuation of the treatment and resumption of it, after a period of discontinuation, deserves in my opinion our utmost attention. We know that discontinuation of the glandular therapy causes return of the signs and discomfort of myxoedema.

And I wonder whether or not it is a casual or accidental finding that almost all reported cases of death from myocardial infarction give a history of reinstitution of thyroid treatment after a period of discontinuation, and death occurred shortly after thyroid has been re-instituted. Most authors limit themselves to giving warnings against the indiscriminate use of thyroid gland products, as it is believed by these authors that sudden death was caused by high doses of thyroid. But even small doses of thyroid constitute a trauma for a heart with sclerosed coronary vessels. If thyroid is given in a case of myxoedema the oedema of the inter-fibrillar tissues decreases and returns to its former stage when thyroid gland has been interrupted. It is evident, therefore, that re-institution of thyroid treatment in cases of this type, will constitute a greater trauma to the heart than if it had not been interrupted earlier, or given at all. This fact is confirmed by the histories of the ten cases of literature, and by the present one. In the former, however, high doses of thyroid were given before death occurred;[1] In the four cases of Bartels and Bell, myocardial infarction occurred following small doses, but no history of these cases was given. Therefore, the amount of thyroid gland alone does not appear to play always a causative influence in the production of myocardial infarction, because the latter occurred following both high and small doses. Re-institution of treatment, after its discontinuation for a longer interval, also constitutes an important factor in the production of myocardial infarction, and deserves our attention.

REFERENCES

1. Zondek, H., *cit.* by Higgins.
2. Fahr, G., *Am. Heart Journal*, 1927, 3, 14.
3. Ohler, W. R. and Abramson, J., *Arch. Int. Med.*, 1934, 53, 165.
4. Fishberg, A. M., *J. A. M. A.*, 1924, 82, 463.
5. Willius, F. A., Proc. Staff Meet. Mayo Clinic, 1936, 11, 557.
6. Keefer, C. S., *Cit.* by Bartels and Bell.
7. Higgins, W. H., *Am. J. Med. Science*, 1936, 191, 80.
8. Bartels, E. C. and Bell, G., Trans. Am. Ass. Study Goiter, 1939, 5-15.
9. Means, T. H., The Thyroid and its Diseases, Lippincott, p. 250, 1937.
10. Beaumont, G. E. and Robertson, J. D., *Lancet*, 1939, 1, 682.
11. Beach, C. H., *J. A. M. A.*, 1935, 105, 871.
12. Blumgart, H. L., Gargill, S. L. and Gilligan, D. R., J. Clin. Invest.,1930, 9, 91.
13. Smyth, C. J., *Am. Heart Journal*, 1938, 15, 652.
14. Nichol, E. S., *Jackson Mem. Hosp. Bull.*, 1940, 2, 24.

I wish to thank Doctor H. K. MacDonald for permission to study and use the case report number one, and Doctor R. P. Smith for his aid in the preparation of this paper.

The Annual Meeting

of the

Medical Society of Nova Scotia

will be held

at

"THE ISLE ROYALE HOTEL"

SYDNEY, N. S.

July 8th and 9th

A strong well balanced scientific programme has been arranged. The Canadian Medical Association are sending two guest speakers and there will be two other papers by prominent visitors. The balance of the programme will be given by members of our own Society. The Cape Breton Medical Society will be our hosts and preparations are now in progress for a successful meeting. Everything will be done to make your visit a profitable and pleasant one. Special committees have been appointed for housing and for the entertainment of members and their families. The Golf Tournament will be held as usual. Those who plan to attend the meeting are advised to write now for accommodation to the Isle Royale Hotel, Sydney, or to the Secretary. Full particulars of the meeting with the programme will be published in the June edition of the Bulletin.

Abstracts From Current Literature

SULFANILAMIDE IN THE TREATMENT OF ACUTE GLOMERULAR NEPHRITIS. Williams, R. H.: American Jour. Med. Sc., 1942, Vol. 203:157.

A comparative study has been made of the course of acute hemorrhagic nephritis in a group of 42 individuals treated in essentially the same manner as a group of 108 similar individuals, except that in the former group sulfanilamide was administered. Most of these patients have been followed from 2 to 5 years or longer.

The results indicate that in the subjects receiving sulfanilamide the foci of infection have cleared up more rapidly, the signs of renal damage have disappeared more rapidly, the exacerbations of the nephritis following tonsillectomy have occurred less frequently, the duration of the edema and hypertension have been shorter and the clinical recoveries have been greater.

In the group of 42 patients treated with sulfanilamide there was 1 death in the acute stage. Complete recovery occurred in 15 of 33 cases who returned for observation after 6 months and in 29 of the 39 patients, or in 74.3% of those followed for at least 2 years. Three additional patients who have been followed for only a few weeks are in a quiescent stage. Five of the 39 patients are in the quiescent stage, and 2 are in a progressive stage.

In the control group of 108 patients, on the other hand, there were 12 deaths in the acute stage, and 5 deaths following a progression to chronic nephritis. Of the entire group of 108 patients there are only 56 complete recoveries, giving a percentage of 52; 11 patients are in the quiescent stage, and 24 in the chronic progressive stage of the disease.

There was no evidence that sulfanilamide caused renal damage in any case.

CARCINOMA OF THE LUNG. Betts, R. H.: New Eng. J. Med., 1941, 225:519. (Bronchoscopic Aspects).

Bronchoscopy is the most important method of diagnosis in primary carcinoma of the lung, a disease which now causes from 8 to 18 per cent of all deaths due to cancer. Approximately 75 per cent of such tumors arise in the major bronchi; lesions so situated can be visualized bronchoscopically and specimens can be removed for biopsy.

Bronchoscopic examination is indicated in every patient who is suspected of having a primary pulmonary cancer, with the exception of the patient with obvious widespread metastases. From the bronchoscopic viewpoint pulmonary carcinomas may be conveniently divided into the central and peripheral groups according to the site of origin of the lesion. The central, or stem-bronchus, tumor arises in the larger air passages and includes all those tumors which can be seen by bronchoscopy. Peripheral tumors arise beyond the proximal segment of a bronchus of the third order and cannot be directly visualized by bronchoscopy.

Of 62 patients with histologically verified pulmonary tumors examined by bronchoscopy, 49 were men, and 13 women. The age distribution was from sixteen to sixty-seven years and the average age fifty-two years. The distribution in the bronchial tree was as follows: right upper, 10; right mainstem,

10; right lower, 9; left lower, 7; left mainstem, 5; left upper, 3; and right middle 2.

It is thus seen that 67 per cent of the tumors originated on the right side and 33 per cent on the left. The author concludes that bronchoscopy should be considered in every case of unexplained pulmonary symptoms. He believes that primary cancer of the lung should be favorable for surgical treatment when early bronchoscopic diagnosis is made.

VALUE OF PROCAINE INFILTRATION IN DIAGNOSIS AND TREATMENT OF FIBROSITIS. Moynahan, E. J. and Nicholson, E. S.: Brit. Med. Jour. Vol. 61:65, 1942.

Moynahan and Nicholson subjected more than 80 patients with fibrositis to infiltration of the painful areas with procaine hydrochloride. Only 6 patients failed to obtain relief or cure, in all but 1 of whom the diagnosis was erroneus, 3 had osteoarthritis of the spine, 1 sacralization of the fifth lumbar vertebra and 1 a severe dislocation of the acromioclavicular joint, with fracture of the acromial end of the clavicle, which was mistaken for a deltoid fibrositis. The sixth patient had fibrositis of the shoulder girdle and left arm with no definite tender spots, and "blind" infiltration failed to relieve the condition. After the tender spots are determined by digital palpation they should be marked on the skin, and after the skin is cleansed a wheal should be raised by injecting a small quantity of procaine hydrochloride over the site of the lesion. The needle should be driven down until the tender spot is reached and then 2 to 3 cc. of the solution should be injected. The injection gives immediate relief from pain, and any associated tenderness or muscular spasm disappears. A dental syringe and needle are probably best suited for the infiltration. Some patients experience a return of pain a few hours after the injection, but this pain usually wears off in a few hours. The patient should be encouraged to move the affected part freely and to carry on with his normal occupation. Usually one injection suffices. Every tender area should be infiltrated and the pain eliminated completely at the first sitting.

DEATH DURING SULFATHIAZOLE THERAPY. Lederer, M. and Rosenblatt, P.: J. A. M. A., 1942, 119:8.

Four cases are described in which death attributable to sulfathiazole was associated with necrotic visceral lesions. From these cases, certain clinical conclusions can be drawn:

(a) The indiscriminate and uncontrolled use of sulfathiazole is not without danger.
(b) Examination of the urine during the course of therapy is important, since crystals of the drug in the urine and hematuria are danger signals. In their presence therapy need not be interrupted so long as the fluid intake and output are adequate. Sodium citrate, sodium bicarbonate or some other alkaline salt may be given.
(c) A chill occurring during the administration of sulfathiazole and followed by sustained fever should be regarded as a danger signal, and sulfathiazole therapy should be discontinued at once.
(d) Diminution and suppression of urinary output should be considered a contraindication to further administration of the drug.

(e) Blood concentration of sulfathiazole over 10 mg. per hundred cubic centimeters is undesirable unless the aforementioned precautions are observed.

(f) Sulfathiazole should be used only when definitely indicated and should not be continued longer than is absolutely necessary.

THE KENNY TREATMENT OF ANTERIOR POLIOMYELITIS (INFANTILE PARALYSIS). Report of the First Cases Treated in America. Pohl, J. F.: J. A. M. A., 1942, 118:1428.

As a result of the demonstration carried out at the Poliomyelitis Clinic at the Minneapolis General Hospital for eighteen months beginning in the fall of 1940, it has been stated that Miss Kenny has conclusively demonstrated that spasm is the condition affecting the muscles in the acute stage of infantile paralysis. Spasm is the cause of deformities. Spasm causes mental alienation of muscle, a pseudo-paralysis occurring in the opposing muscles to those in spasm, in which those opposing muscles are divorced from the voluntary motor pattern and cease functioning. Spasm plus mental alienation causes incoordination of muscle action, resulting in further damage to the motor mechanism.

The Kenny method definitely diminishes the crippling after-effects of the disease of infantile paralysis. The Kenny treatment employs the use of moist heat. Wool flannel packs of proper size are immersed in boiling water, wrung twice through a tight wringer at the bedside and quickly applied to the involved area. The pack must accurately cover the entire body of the affected muscle, but joints are left free in order not to give the patient any sense of immobilization of the limb. The moist pack is covered with oiled silk and then with dry flannel. The pack is changed every two hours but may be renewed as often as every fifteen minutes if the spasm is very acute. Packs are continued through twelve hours of the day. No ointments are applied to the skin; burns do not occur if the packs are wrung quite dry. The acute spasm with pain will subside usually within a week if treatment is proper. Tendency of the muscle to remain in a state of contraction or shortening may persist for weeks or months, and especially if treatment is instituted some days or weeks after the onset. Hot packs must be continued until the muscle is able to extend itself completely as evidenced by full range of motion of the joint concerned. A remarkable state of health, tonus and vigor in all the tissue of the extremity affected is preserved by this method, and freedom from circulatory and trophic changes are strikingly noteworthy.

At the end of eighteen months after beginning the Kenny treatment of a series of 26 patients in the acute and subacute stage it can be stated that these patients have all made a far more satisfactory recovery than they would have made by any previously known method. No deformities have occurred, in spite of the complete omission of splinting.

Incorporation of the principles of the Kenny method with those of other methods for the treatment of infantile paralysis would prove unfeasible, as the Kenny method is based on previously unrecognized symptoms of the disease.

The method should be immediately adopted as the fundamental treatment of the disease of anterior poliomyelitis. As the condition affecting the muscles appears with the onset of the disease, it is imperative that treatment be instituted as soon as the diagnosis is established.

An additional series of 28 cases occurring in the fall of 1941, all coming under treatment in the acute stage, have presented very satisfactory progress to date. These cases will show even more remarkable recovery when viewed at the end of the treatment period. The fact that these cases were treated early and that the medical and nursing staff were better prepared to carry out the treatment by virtue of the experience gained with the 1940 series will insure a maximum recovery in the 1941 series.

STATUS OF GASTRODUODENAL ULCER. Editorial:-J. A. M. A., 1942, Vol. 118:1452.

A symposium in a recent issue of the Archives of Surgery reemphasizes the importance of the acid gastric secretion in the causation of gastroduodenal ulcer. Ivy states that destruction of gastric tissues results from the proteolytic action of the gastric juices. He does not wish to imply that the excessive secretion of gastric juice or its retention in the stomach is the cause of gastroduodenal ulcer. He believes, however, that the irritating action of acid and pepsin is the prime factor in the genesis of postoperative jejunal ulcer and is important in the development and perforation of duodenal ulcer.

According to Quigley, hunger contractions through mechanical trauma to the area involved may give rise to distress to the patient with ulcer and may prevent healing. It is desirable to avoid the conditions which tend to exaggerate hunger contraction.

According to Dragstedt, pure gastric juice has the capacity of destroying all living tissue, including the wall of the stomach itself. Animal experiments utilizing a Pavlov or Heidenhain pouch have demonstrated that pure gastric juice has an aggressive action on living tissue as contrasted with the gastric content, which usually consists of a mixture of swallowed food and saliva, gastric juices from the parietal cells of the fundus, mucus and a neutral or faintly alkaline secretion from the pyloric antrum and varying quantities of regurgitated duodenal juices. This gastric content is relatively inert. In all the experiments in which pure gastric juice from an isolated pouch of the stomach is permitted to flow into the lower intestine, the ulcer forms in the intestine rather than in the gastric mucosa. Since the exposure is similar, one must conclude that the gastric mucosa has the greater resistance to digestion. Under normal conditions the gastric wall is not digested away because it is not exposed to pure gastric juice. A continuous gastric secretion occurs which is not dependent on the presence of food. It is slight and its small volume permits its neutralization by the mucus of the pyloric antrum, swallowed saliva and possibly also regurgitated duodenal contents. It is conceivable, Dragstedt points out, that this neutralizing mechanism may fail or prove inadequate and that, as a result, more or less pure gastric juice may accumulate in a stomach empty of food. It seems probable that some abnormality of this type is responsible for most cases of ulcer in man. Anderson and Fogelson reported a relative decrease in the gastric mucin in some patients with duodenal ulcer. Artificially induced continued excessive secretion of gastric juices through the use of histamine pellets has produced ulcers in all the common laboratory animals. Dragstedt advances the opinion that in man a similar excessive secretion of gastric juice occurs and an ulcer begins. The hypersecretion in most cases is probably neurogenic and is abnormal in the sense that it operates when the stomach is empty and in the absence of usual stimuli for gastric secretion.

The problem of ulcer in man, according to Palmer, is one of tissue resistance versus acid attack. Vanzant and her collaborators have found that there was an increase of about 12 units of free acidity in the case of duodenal ulcer. In the case of gastric ulcer the mean free acidity was lower than normal by about 6 units. The incidence of achlorhydria was half that observed in normal persons. Peptic ulcer occurs in persons with a low secretory rate as well as in those with a high secretory rate. Chronic ulcer does not occur in persons with a complete and continuous achlorhydria. Nocturnal secretion, however, exceeds in amount and acidity that observed in normal persons. The presence of acid gastric juice is essential for the production of erosions and ulcers. Pepsin greatly facilitates the progress, but it alone will not destroy the mucosa. The chief protection against the acid attack on the cells of the mucosa seems to be provided by the thin layer of mucus with which they are covered. Thrombosis, embolism and infection are not essential features of experimental ulcer. Palmer calls attention to the fact that ulcers may, and the majority do, heal in spite of the presence of acid gastric juice. This is evidenced by the spontaneous remissions and by the healed lesions encountered in routine necropsies.

There appears to be considerable agreement between internists and surgeons as to the treatment of duodenal ulceration. The wave of enthusiasm for stomach resections which began a quarter of a century ago and was advocated with particular fervor in Germany has now considerably subsided. Thus, Allen states that duodenal ulcer is primarily a medical problem and that apparently 80 per cent of the patients with this lesion respond to conservative measures. This is essentially what Sippy has taught for a number of years: that the surgical indications for duodenal ulcer were complications, namely acute perforation, massive hemorrhage, cicatricial obstruction and intractability. Allen believes, as do practically all surgeons today, that surgical cure for duodenal ulcer can be brought about only by a subtotal gastric resection. The operative mortality from this procedure has been reduced to a level compatible with the results obtained. Wangensteen emphasizes that the most important criterion of an acceptable operation is that it reduces gastric acidity effectually. He feels that the three-quarter resection meets these demands. The only known manner in which the secretion of acid may be diminished effectually is by sacrificing a liberal portion of the gastric mucosa. Excision of antral mucosa is mandatory to insure achlorhydria. The antral mucosa probably contains a hormonal stimulant of gastric secretion other than histamine.

The case of gastric ulcer differs from that of duodenal ulcer principally because of the ever existing danger of malignant degeneration. Walters finds that in 10 per cent of the cases gastric ulcer is malignant. He emphasizes that in many cases of chronic gastric ulcer healing is temporary under non-surgical methods of treatment and recurrence is frequent. The triad which in the past was depended on to insure that the lesion is benign, namely relief of symptoms, disappearance of the niche in the roentgenogram and the disappearance of blood from the stools, cannot be absolutely relied on. Schindler and Arndal stress that it is in the differentiation of benign and malignant ulcer that gastroscopy is most useful and is here superior to roentgenoscopy. They admit, however, that the method had failed to make a correct differential diagnosis in 6 of 113 gastric ulcers. According to Eusterman, gastric carcinoma not only may masquerade successfully as benign ulcer but may react to treatment in similar fashion. Jordan summarized this problem by stating "Neither the size of the ulcer nor the age of the patient nor the presence of normal acid or

hyperachlorhydria should lessen our suspicion of carcinoma, for some of our largest lesions have been benign and some of the smallest malignant. Malignant lesions occur often enough in the young, and benign ulcers often enough in the middle aged and old, and acid is present often enough where the lesion is malignant, so that these three criteria of size of ulcer, age of patient and presence or absence of acid have no actual or practical value in the diagnosis of the individual patient." Proper surgical treatment of gastric ulcer, according to Walters, has been followed by excellent results. In his experience, recurrence has nlt taken place when one-half the stomach was removed. The operative risk should not exceed 5 per cent and in the hands of skilled surgeons should be less than that.

E. David Sherman, M.D.,
Sydney, Nova Scotia.

DALHOUSIE REFRESHER COURSE WILL NOT BE GIVEN THIS YEAR

On account of prevailing war conditions—the rationing of gas—and especially as the staff of the Medical School will be busy with teaching throughout the summer, it has been decided not to hold the Refresher Course this year.

J. V. Graham
Chairman

ASSISTANCE TO MEDICAL STUDENTS

Statement by the Minister of National Defence in the House of Commons

March 19, 1942

1. It is estimated that the Armed Services will, during the ensuing fiscal year, require probably 800 Medical Officers, of which 50 will be needed by the Navy, 600 by the Army and 150 by the Air Force. As a matter of fact, the Army could take on almost at once probably 150 Medical Officers, and the Air Force, I am informed, probably 75 to 100. These requirements are being brought to the attention of the Medical profession throughout Canada and the Canadian Medical Association is co-operating by sending out a questionnaire to all members of the profession.

2. In order to assist in increasing the future supply of Medical Officers for the Armed Forces the Medical Services of the Navy, Army and Air are co-operating in a scheme designed to aid worthy students of Medicine who propose to enlist.

3. This arrangement is the result of a thorough study of the medical needs of the Armed Forces and has been proposed after consultation with the Medical Faculties of the Universities throughout Canada. Briefly the scheme is as follows:

(a) The Universities will be asked to accelerate their courses, while maintaining the same standard of training, by reducing the normal summer vacation periods to a minimum.

(b) In this way a student will be able to cover his full course in a considerably shorter period.

(c) By this acceleration of courses students will be debarred from earning in the long summer vacation. The plan is designed to make up for this loss of income.

(d) It is proposed that the Armed Service will enlist students at any time during the two final years before they qualify for a licence to practise. They will thus, during this period, become members of the Armed Forces and will then be granted leave with pay and subsistence allowance which will enable them to complete their courses and to obtain a licence to practise.

4. This assistance to medical students who intend to join the Armed Forces will be supplemented by the Department of Labour (in co-operation with the Provincial Governments) by extending the present system of scholarships. These scholarships (up to a maximum of perhaps $300) will also be open to other worthy students who because of the acceleration of the courses will be deprived of the opportunity to earn during what would be the normal vacation period.

5. The students who will be eligible for these scholarships will be those who are in need of such assistance, who possess good academic standing and who give satisfactory assurance to serve in the national war effort in the capacity in which they have been trained.

6. There will also be some expenditure involved in connection with additional costs to Universities on account of the accelerated courses.

7. The Department of Labour is now in correspondence with the Provincial Governments with a view to enlisting their co-operation in implementing this extended plan.

8. In the result, medical students who intend to join the Armed Forces will (after the first year) receive assistance from the Department of Labour, in co-operation with the Provincial Governments, by way of scholarships, during the earlier years of their courses, and during the last two years they will receive pay and subsistence allowance as duly enlisted members of the Armed Forces.

Minister of National Defence

Ottawa, March 28, 1942

Dr. T. C. ROUTLEY,
General Secretary,
Canadian Medical Association,
184 College Street,
Toronto, Ontario.
Dear Doctor Routley,

In respect to previous correspondence and the interest you have taken relative the question of assistance to Medical Students, no doubt you have read in the press of my statement in the House of Commons on March 19, 1942.

If it has not come to your notice, I enclose for your information a copy of the statement, and I may say that from comments I have heard it would appear as if it will provide a useful and practical solution in assisting Medical Students who propose to give their services to the Armed Forces.

We would very much appreciate your co-operation in making this plan a success, the objective being, of course, to obtain Medical Officers for the Navy, Army and Air Medical Services and to encourage and support students having that activity in view.

Yours very truly,
(Signed) J. L. RALSTON

Personal Interest Notes

AT the annual session of the American College of Physicians which was held in St. Paul, Minnesota recently Dr. E. David Sherman of Sydney was honoured by election to an Associate.

Dr. B. S. Bishop of Kentville suffered a severe accident recently when the car he was driving left the highway and was reduced to scrap after plunging into a five foot ditch. The accident occurred, it is stated, when the steering gear locked on a curve. The car sheered off two telephone poles before turning over in the ditch. Dr. Bishop was thrown into the back seat of the car, and escaped with only a broken arm.

We are glad to hear that Dr. J. B. Reid of Truro who has been seriously ill is now recovering.

Major Carl R. Trask, Dal. '38, who has been on active service for over a year in England, is home on furlough.

1898 Graduates Have Reunion

There was held recently a reunion of the four remaining members of the Dalhousie Medical School of 1898, Dr. A. McD. Morton of Halifax, Dr. G. G. Gandier of Dartmouth, Dr. Edward M. McDonald of Sydney and Dr. Matthew G. Archibald of Kamloops, B.C., and formerly of Middle Musquodoboit. It all started with a visit to the Maritimes by Dr. Archibald from British Columbia. When Dr. McDonald heard about it, he came to Halifax, and there was nothing to do but to have a reunion and recall old times.

We are glad to learn that Dr. G. A. MacIntosh, the Superintendent of the Victoria General Hospital, Halifax, who has recently undergone a major operation, is making a most satisfactory recovery.

The Doctors of the Pictou County Medical Association at a recent meeting decided that their offices would be closed on Sunday and Wednesday afternoons and evening except by special appointment. Need of getting routine calls in as early as possible was stressed to avoid going over the same neighbourhood several times a day in order to save gas and tires.

Dr. Grace Cragg, Dal. '22, of Boston, Mass. flew to Halifax to attend the graduating exercises of Dalhousie University on May 12th.

Dr. A. R. Morton, the City Commissioner of Halifax, will read a paper on *Health Problems in a Vital Defence Area* at the annual meeting of the Canadian Public Health Association to be held at Toronto, June 1st to 3rd.

The marriage took place on May 8th at Truro of Miss Lillian Marie, daughter of Rev. and Mrs. G. A. Grant, Truro, and Dr. Austin Alexander Macdonald, son of Mr. and Mrs. D. A. Macdonald of Whycocomagh. The bride has occupied the position of public health nurse in Hants and Colchester Counties for the past two years, and the groom is a recent graduate of Dalhousie.

Dr. and Mrs. Robert Wright of Noel, Hants County, are spending a few days at Scotsville, Inverness County, where the trout fishing is reported to be good.

Dr. M. G. Archibald of Kamloops, B.C., is spending his vacation with his brother, J.A. Archibald, and his sister, at Middle Musquodoboit.

Obituary

THE death occurred at Canso on April 27th of Dr. Ernest Fraser Moore, at the age of 72. Dr. Moore was born at Woodstock, N. B., on May 12, 1870, the son of the Rev. and Mrs. E. B. Moore. He received his B.A. from Mt. Allison University, and graduated from Dalhousie Medical School in 1895, and before going to Canso practised in Cheverie, N. S., the Nova Scotia Hospital, Dartmouth, and Camp Hill Hospital, Halifax. He was a veteran of the Great War having served with the rank of major in the Medical Corps. Going to Canso in 1921 he was some time later appointed medical officer to the Western Union Cable Company, Canso, and the Commercial Cable Company, Hazel Hill. He was also port physician and health officer for the town of Canso. Dr. Moore is survived by his wife, the former Anna Hushard, and a sister, Mrs. E. A. Smith of Halifax.

Dr. Lewis Johnstone Lovett of Bear River, died at the home of his brother H. A. Lovett, K.C., at Pinehurst, North Carolina, on April 27th. Dr. Lovett had suffered a rather serious illness during the past winter and a few weeks previously had left for Pinehurst accompanied by his wife and daughter, Edith, and was convalescing there at the time of his death. Prominent in political circles, he was a staunch Liberal and held the federal seat for the Digby-Annapolis constitutency from 1921 to 1925. Keenly interested in the development of his native province he did much to advance the interests of Nova Scotia during his tenure of office at Ottawa. He was an ardent sport enthusiast and for several years held the presidency of the Digby-Annapolis Fish and Game Protective Association. His efforts to develop the Annapolis Valley were many and varied. For several years he was president of the Bear River Board of Trade and also the Valley Medical Society. He did much to develop the community of Bear River as a summer resort. Dr. Lovett was the son of the late Henry Lovett and Annie Lovett of Kentville. He graduated in Arts at Acadia University and went on to take his medical education at the University of New York from which he graduated in 1891.

He is survived by his wife, the former Josephine Marshall of Bear River, and two daughters, Mrs. William Sutherland of Lockeport, and Edith Lovett, R.N., of New York City; two brothers, L. A. Lovett, K.C., of Halifax, H. A. Lovett, K.C., of Montreal, and one sister, Mrs. T. R. Robertson, of Halifax.

An Appreciation

Dr. Lewis Johnstone Lovett was much more than a member of the medical profession to Bear River where he had made his home and had practised for 51 years. He was a leader with the interests of the immediate countryside and of his country at heart.

In half a century, he had helped into this world a whole new generation and had seen them grow up to take their places as useful men and women. His skill and his understanding of human nature combined had helped many of them through crisis in their lives. He was The Doctor.

Tangible expression of the esteem in which Dr. Lovett was held was given many times, notably in the federal election of 1929 when he was elected member

for Annapolis-Digby. In this he was following in the footsteps of a maternal uncle, the Hon. James W. Johnstone, noted figure in Nova Scotia political and juridical life in the Nineteenth Century who sat in the Provincial House for Annapolis for twenty years.

Nor was this the only field in which the Doctor served, for he was President of the Board of Trade and was foremost in the movement which developed Bear River into one of the outstanding resorts of the Province.

Now Death has claimed Dr. Lovett at the end of a long illness. It has taken from Bear River a splendid citizen, a friend, and to-day there is widespread mourning and the deepest sympathy for members of the family. — *The Halifax Herald, April 29-42.*

Dr. John James Roy, one of the most prominent members of the Medical Society of Nova Scotia passed away suddenly on May 13th from heart disease. Dr. Roy was born in Westville on October 11, 1874. He received his early education at New Glasgow and later on entered McGill graduating from there in medicine in 1897. For the past forty years he has practised at Sydney. During that time he has maintained an active interest in the civic affairs of that city and was associated with many of the undertakings launched there for the improvement of civic conditions. He was superintendent of the Dominion Steel and Coal Corporation's emergency hospital and medical officer for the port of Sydney for many years. His affiliations included the Sydney Rotary Club, Board of Trade, Lingan Golf Club, Curling Club, Royal Cape Breton Yacht Club, Cape Breton Medical Society and the Medical Society of Nova Scotia, of which he was president, 1926-27. Surviving are his wife, the former Mary B. Willis and seven sisters. Funeral service was held at his residence in Sydney after which the body was forwarded to New Glasgow for interment there.

The BULLETIN extends sincere sympathy to Dr.V.D. Schaffner of Kentville on the death of his father, R. J. Schaffner of Lawrencetown, which occurred on April 19th: and to Dr. Joseph Hayes of Halifax, on the death of his wife Mrs. Maria Hayes, which occurred at Wolfville on May 18th.