The Diagnosis and Treatment of Coronary Insufficiency*

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According to the Standard Nomenclature of Diseases and Operations, published by the American Medical Association, arteriosclerotic heart disease is a generic term used to designate coronary arteriosclerosis when it produces cardiac symptoms such as angina of effort without evidence being present of gross heart damage.

With this in mind I looked over our records of 928 cases seen at the Winnipeg Clinic from 1945 to 1951 and carefully analyzed 200 of these.

Coronary insufficiency is sometimes known as the *acute* phase of angina pectoris, or the *premonitory* phase of myocardial infarction. However, I wish to emphasize that it is a complete entity, conforming to a particular pattern with its own physiology and pathology and providing a characteristic electrocardiogram. If we diagnose it early and treat the patient adequately we can prevent the calamitous onset of myocardial infarction.

Definition

The simplest definition of coronary insufficiency is that it represents the disproportion between the nutritional requirements of the myocardium and the actual coronary flow.

Of prime importance is the fact that the damage caused to the heart muscle in these cases *can be reversed.* The lesions in coronary insufficiency, if present at all, are usually disseminated, patchy, and often microscopic. Often there is only an ischaemia of the sub-endocardial layer; in fact the sub-endocardial region, particularly the papillary muscles, may be the only area involved while in myocardial infarction there is serious through and through necrosis.

Etiology

The commonest *coronary* affection is arteriosclerosis and as you know, the precursor of this is ordinary *atheroma*, a very old Greek word, meaning meal or porridge; it consists of softening, with yellowish fatty (cholesterol) areas in the intima of the vessels. Fibrosis, calcification and formation of cholesterol abscesses are the result. The cause of atheroma of the coronary arteries, as well as that of arteriosclerosis in general, is still unknown. Despite all the research being carried on to determine the significance of serum cholesterol and other blood lipids, we have no positive proof that the lipoprotein molecules cause atherosclerosis. (1)

Dr. Blumgart(2) has recently pointed out that the older view of arteriosclerotic heart disease as a simple progressive narrowing of the coronary arteries with final closure due to thrombosis, is no longer tenable. It is now known that the narrowing of a vessel predisposes to the formation of anastomoses. The balance between the rate of development of arteriosclerosis

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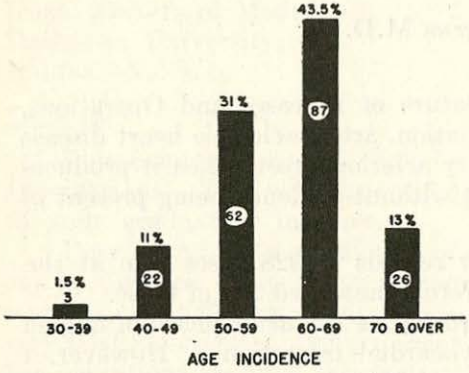

Fig. I—There were only 2% or 4 more cases of coronary insufficiency in the seventh decade than in the fourth

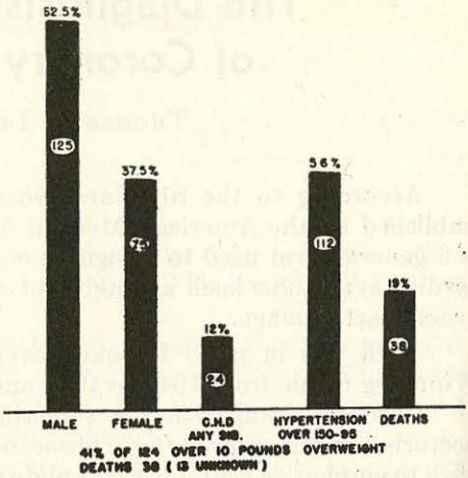

Fig. II—**Sex ond other relevant information.**

C. H. D. any Sib.—Coronary heart disease, any sibling (brother or sister).

Deaths — 40-50 years — 3; 50-60 years — 12; 60-70 years — 17; over 70 years—6.

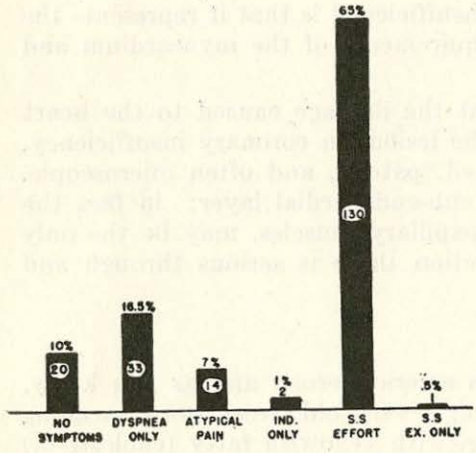

Fig. III—**Symptoms**

Atypical pain—i. e. not in the chest.

Ind. only—Indigestion only.

S. S. effort—Substernal pain on effort only, the classical textbook type of pain.

S. S. ex. only—Substernal pain (classical type) on excitement only.

The above figures show that 35% of patients did *not* have the usual symptoms, and because the diagnosis is so frequently missed in this latter group, it is of particular interest to us as a diagnostic challenge. Some of these patients, with a normal resting electrocardiogram, may be dismissed as normal, whereas further investigation, for example the "2-Step" electrocardiogram may reveal coronary insufficiency.

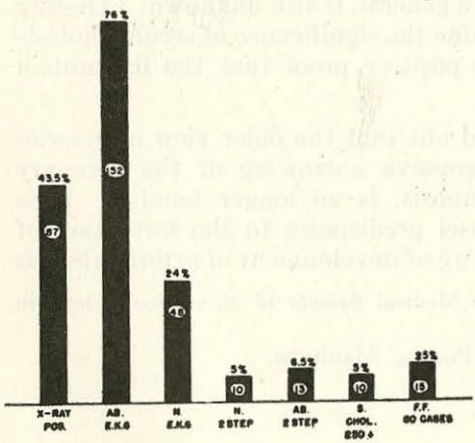

Fig. IV— **Results of X-ray and laboratory investigation**

X-ray pos. — enlargement of heart confirmed by barium and fluoroscopy and flat films.

Ab. E. K. G.—abnormal resting electrocardiogram.

N. E. K. G.—normal resting electrocardiogram

N. 2 Step—normal "2-Step" electrocardiogram.

Ab. 2 Step—abnormal "2-step" electrocardiogram.

S. Chol. 250+—Serum cholesterol abnormal, i. e. over 250 mg.%.

F. F. 60 cases— Flicker fusion tests were done on 60 patients, and 15 of these had an abnormal reading, i. e., above 60 flashes per minute.

and the rate of formation of anastomoses will decide what will happen. If the oxygen requirement can be held down by rest, many fibers which might have become necrotic, if forced to contract more frequently and more forcibly, may survive. If coronary insufficiency is diagnosed early, and the patient is treated adequately, myocardial infarction may be delayed. Experiments on animals have demonstrated that large collateral channels may be produced in response to marked narrowing of the coronary arteries; that 2-5 days are required for the ample development of these new channels, and that the latter not only protect the heart from serious damage, but also enable survival following subsequent complete occlusion.

Causes

There are three important causes of coronary insufficiency or failure, and a combination of all three is probably the most common.

1. Increased Cardiac Work

This is a result not only of physical or emotional stress (by far the most common), but also extremes of temperature, over-indulgence in food, tobacco and alcohol, tachycardia, hypertensive or hyperthyroid crises, acute infectious states, and drugs such as adrenalin and pitressin.

2. Diminished Coronary Flow

This may result from shock, hemorrhage, operation and trauma, and as such is of utmost importance to the surgeon. Master (3) points out that it is insufficiently realized that morbidity and mortality in hemorrhage often are produced by ischaemia or necrosis of the left ventricular myocardium. Other causes of diminution in coronary blood supply are hypotensive crises which occur in spinal anaesthesia or sympathectomy and reflex vaso-constriction in acute abdominal conditions; gall bladder disease, complicated hiatus hernia and pancreatitis. Pulmonary embolism and peripheral thrombosis or embolism, also the hypotensive crises of Addison's disease and orthostatic hypotension, are additional causes of lack of coronary blood supply.

3. Impaired Oxygenation of the Blood

This occurs in acute anaemia, pulmonary embolism or insufficiency, asthmatic attacks, and during anaesthesia or in carbon monoxide poisoning, (where it may be more important than respiratory or cerebral involvement).

Clinical Symptoms

In assessing clinical symptoms it is necessary of course to exclude extra-cardiac conditions which cause chest pain; such as spondylitis, herpes zoster, pneumothorax, pleurisy, gall bladder disease, peptic ulcer, hiatus hernia, and esophageal spasm. I need not tell you that pain in the chest is NOT necessarily angina, any more than a sore throat is diphtheria!

Clinically patients with coronary insufficiency usually experience attacks of substantial pain lasting 20-30-40 minutes. It is usually the upper waistcoat button location, and is described by the clutching hand. You can make a correct diagnosis and be right very often, if you take a careful history. MacKenzie used to say, "Listen carefully to his story he is telling you the diagnosis". The presence or absence of organic heart disease neither proves or disproves anything. From his story, you learn that this particular

patient, (he may be 25 or 95), is having attacks of substernal distress induced most often by effort or emotional upsets and relieved by rest, nitroglycerine, whiskey or amyl-nitrite. As Lewis says, "It's the pain of a given grade, which recurs with the repetition of a particular act." Some particular cause then, does not produce it occasionally; it produces it regularly, almost inevitably, and while we have seen that there are other causes, the most common cause is walking, out-of-doors, particularly against a cold wind, after a heavy meal. The discomfort may start anywhere in the thorax, sometimes it starts at the periphery and ends at the sternum and the radiation is proportionate to its intensity. The attack may simulate coronary occlusion at its onset but does not resemble it in the electrocardiographic pattern.

In interrogating these patients it is wise to avoid leading questions, say rather, "What brings on the discomfort?" Not, "Do you have any chest pain on walking?" People vary a great deal, both in their description of pain and their tolerance to pain, and we should consider this in our evaluation. They may think of pain as something like an abdominal cramp, therefore a mild discomfort in their chest is either entirely forgotten or considered anything from heartburn to neuralgia. The ability of patients to describe pain is dependent on their sensitiveness, education, imagination and facility of expression. They may describe the discomfort of breathing faster on effort as their only symptom and you must differentiate dyspnea from pain although both may be present, and the patients may over-emphasize the dyspnea.

In angina of effort you do not have a true dyspnea, it's rather an immobility of the chest. The patient holds his breath because he is afraid to breathe.

When examining middle-aged patients for conditions remote from the coronary circulation, surgeons should inquire regarding the presence of chest pain on effort. It is a wise pre-operative precaution. Patients often do not mention it voluntarily unless it becomes severe and crippling.

Coronary disease with pain does not usually proceed in a smoothly or even irregularly progressive manner from mild to moderate to severe, as one might expect IF pain were simply a reflection of a gradual narrowing of the coronary bed. Dr. S. Proger (4) has pointed out that excluding the influence of extra-coronary factors, the course of coronary disease with cardiac pain is usually characterized by one or more NEW episodes and each new episode may appear in a different guise. It is likely that each new episode results from a new narrowing or occlusion.

The pain of coronary insufficiency may linger on resembling a severe attack of angina pectoris or it may progress to a full blown myocardial infarction.

The incidence of atypical coronary pain has been reported by the Mayos as ranging from 20-30%. In our series it was 33.5%. One of Heberden's original patients in 1772 had pain only in his left arm from the age of 60 till his death at 75. Albutt described a patient whose pain was only in the palms of his hands, Osler described a case whose pain was exclusively brachial, and Mackenzie reported one case where pain was confined to the left little finger. (5).

Other Clinical Signs

There is usually no fever, the heart sounds remain normal, and there is usually no change in the blood pressure except that it may rise during an attack of severe pain and fall if shock is co-existent. Ordinarily there are no arrhythmias present, although certain of these may pre-exist and precipitate an attack. Moderate leukocytosis and elevated sedimentation rate are only found in the course of severe and prolonged attacks.

The diagnosis, resting on the pattern of distress elicited from a carefully detailed history, is confirmed by significant findings in the electrocardiogram. *Here is where it differs from angina pectoris.* The characteristic pattern is RS-T depressions and T wave inversions; Q waves are rare. The former are often absent in the resting tracing. Unlike acute myocardial infarction there is no reciprocal relationship between leads 1 and 3 and unlike acute infarction the changes found in coronary insufficiency are of short duration and reversible. This is because in coronary insufficiency there is ischaemia of the subendocardial layer, not the serious through and through necrosis found in the myocardium in infarction.

It becomes extremely difficult at times to differentiate coronary insufficiency from the so-called "premonitory phase" of coronary occlusion. Both should be put to bed and serial tracings done. If Q waves appear in 8 to 24 hours, a definite diagnosis can be made. Acute pericarditis, may, at times, add to the confusion.

Therapeutic doses of nitroglycerine are helpful in diagnosing doubtful cases but nitrites relax smooth muscle of the bronchi and biliary tract too, therefore relief of pain by them is not always presumptive evidence of angina of effort.

The resting electrocardiogram may show changes in T waves and RS-T segments, but 24% of our series were normal. The 10% oxygen anoxemia test and the double "2-Step" exercise electrocardiogram, are of assistance in doubtful cases, often showing changes not found in the resting tracing. The anoxemia test is positive in only 20-30% of patients with organic coronary disease. (6)

Master (3) has emphasized that we will all be open to criticism if we give a clean bill of health to a patient with a questionable pain, because the physical examination, x-ray and resting electrocardiogram, show no abnormalities. We should not be satisfied until a single or double "2-Step" electrocardiogram rules out all traces of coronary artery disease.

In the "2-Step" test the patient ascends and descends two 9 inch steps a variable number of times (according to a table based on age and weight), in $1\frac{1}{2}$ minutes with electrodes attached. A "double" test means that the patient does this for 3 minutes. Three tracings are taken, the first immediately after, then 2 minutes after, and 6 minutes after.

Before proceeding with the "2 Step" test we look at the direct writing resting tracing while the electrodes are in position. IF no significant abnormalities are found in the resting tracing, then we proceed with the "2-Step" exercise test.

If the resting tracing shows signs of coronary artery disease, it is unwise to proceed further, and may be dangerous. Other contra-indications to the "2-Step" electrocardiogram are an enlarged heart and evidence of congestive heart failure. Of course it should never be done if one suspects the presence of an acute myocardial infarction.

It is only necessary to do the three standard leads and V4 and V5, the latter being more sensitive. The criteria for a positive single or double "2-Step" include RS-T segment depressions over 0.5 mm. below the iso-electric P-R level; partial or complete inversion of a T wave in any lead except lead III; and transient cardiac arrhythmias, bundle branch block patterns, etc.

If both the standard and double "2-Step" tests are negative, coronary insufficiency may practically be excluded and we should look elsewhere for the cause of the discomfort. It must be emphasized however that there is a *functional* or *neurogenic* coronary insufficiency which should be differentiated from an organic disease. We know, for example, that anaemia and fatigue, as well as rheumatic or congenital heart disease, may produce changes in the electrocardiogram on exercise.

The co-relation of the tracing with the clinical history is the all important factor. One must not read too much into the tracing, and if doubts exist, serial tracings often help clarify the diagnosis.

Flicker Fusion Test

Dr. A. C. Ivy and his associates in the Department of Clinical Science at the University of Illinois invented a machine for estimating the response of the flicker fusion threshold to nitroglycerine. (7) This, of course, is a physiological measurement. It showed hypertonus of the retinal arteries in 98% of patients with existing hypertension or coronary arterial disease.

They first estimate a patient's normal response by averaging several tests. Then they give the patient grains 1/150 nitroglycerine and see what happens in 2, 4, and 6 minutes.

A patient with arterial hypertonus sees better, or shows an increase in the number of flashes after nitroglycerine, on account of the vaso-dilatation which occurs; while a patient with normal retinal vessels sees worse, or shows a decrease in the number of flashes on account of the hyperemia which has resulted from the vasodilatation. The second dose of nitroglicerine should increase the changes one way or the other, the normal patient seeing worse and the abnormal better.

Dr. Ivy and his associates feel that in view of the intimacy of the embryological development and vasomotor supply of the heart and the retina, that vasospastic tendencies in the retinal vessels can be correlated with changes in the coronary circulation.

We obtained a machine and did the test on 60 patients in our series. Of this number 15, or 25% were abnormal, that is, they had a reading above 60 flashes. Five of these 15 patients had no typical pain, and 4 of them had negative electrocardiograms, both resting and "2-Step".

Treatment

Specific measures for treating coronary insufficiency frequently depend on the causal factor present; for example, in cases of severe anaemia the

coronary symptoms are relieved by iron therapy, in toxic goitre the symptoms are relieved by thyroidectomy; and in gall bladder disease by cholecystectomy. These facts are worth re-emphasizing because we should search out suspicious causal factors and not confine our investigation solely to the cardiovascular system.

In shock and hemorrhage, restoration of the circulating blood volume, blood pressure and hemoglobin is necessary by intravenous infusion of fluid, plasma and blood. It is better to risk the occurrence of heart failure induced by excessive fluid, than to allow the patient to lapse into irreversible shock. Spinal anaesthesia may be used but care should be taken to prevent the blood pressure from falling below 100 mm. hg., if it does occur, blood should be given freely. In diabetic patients receiving insulin it is important to prevent episodes of hypoglycemia which often precipitate episodes of coronary insufficiency. Where increased cardiac work is the cause, physical and emotional stress must be eliminated and coronary vasodilators are indicated, also sedation.

Undue physical or mental strain, intense enough to produce severe anginal symptoms, with the possibility of irreversible changes in the deeper subendocardial layers, requires at least a week to 10 days in bed.

In hospitalized cases, where careful evaluation of prothrombin time can be done accurately, anticoagulant therapy with dicumarol or tromexan must be considered as a therapeutic measure. This, plus bed rest for 2-3 weeks and repeated serial tracings to observe the progress is the ideal treatment in selected cases, with sedation by sodium amytal, three times a day.

In his latest edition of Heart Disease, (8) Paul White says that nitroglycerine is the drug of choice while an adequate collateral circulation is being established and during these weeks or months when all undue strain must be avoided he has found the regular use of nitroglycerine the best procedure of all. One seldom, if ever, encounters serious complications from its liberal use. If a patient expects pain from an act, he should put a nitroglycerine tablet under his tongue *beforehand*, i. e. before a game of golf, a bath, or a hurried walk to the bus or car.

Just telling patients to avoid worry and anxiety is both trite and useless. It is necessary to take time to talk to these patients and ask them about their work, their family, their home life, and their leisure time. Have they learned to relax, or to enjoy a good book in a tranquil restful atmosphere? As many cases are doctors it may be necessary to suggest some radical changes in their daily habits, but it is wise to remember that idleness may induce hopelessness and a happy worker is safer than an irritable idler.

Mackenzie's dictum is a good general rule. "Patients with symptoms can do with safety whatever they can do with comfort."

If the patient has any peripheral vascular involvement or if there is an increase in the heart rate of more than 25 beats per minute after smoking one inhaled cigarette, smoking should be discontinued. Spirits in moderation are most helpful.

It is also advisable to talk to the family and see that the patient's affairs are in order, and arrange for him or her to return at frequent intervals for a

recheck. If you can inspire the patient's intelligent co-operation, then you can be optimistic about the results.

I tell my depressed doctor patients to remember that Sir James Mackenzie and Sir Thomas Lewis lived and worked for 17 years after their original attacks.

An explanation of Selye's General Adaptation Syndrome (GAS) can be helpful for some patients. In his book "STRESS" (9) Selye explains that this syndrome includes the three phases of body reaction to long-continued exposure to systemic stress, namely: 1. alarm reaction, 2. stage of resistance, 3. stage of exhaustion.

Investigation of the effect of chronic nervous irritation on the cardiovascular system has not been completed, but so far it is known that chronic damage can result from such nervous stimuli.

We may wonder how it is possible to eliminate conditions of stress in these hectic, atomic years. Apropos of this, Selye has written an interesting dedication to his wife, stating that she understood he could not and should not be cured of his stress but merely *taught to enjoy* it.

And isn't that exactly what Osler had in mind when he told us many years ago, to enjoy longevity by getting a chronic disease and taking care of it?

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Morbidity and Mortality in Prostatic Surgery

A Brief Analysis of 500 Consecutive Prostatectomies

CLARENCE L. GOSSE, M. D.

A quarter of century ago mortality rates in prostatic surgery ranged from 10-25%. At the end of the last war the rates were still as high as 5-10% in the better centres. Today to have a patient die after prostatectomy is not only a rarity but a matter of grave concern to the surgeon, causing him to review most critically his handling of the case, as if something unusual and unnecessary had occurred. Furthermore, a few years ago with the now extinct two-stage operation it was not unusual to see patients lying around hospital for two or three months after operation. To-day one does not anticipate a longer hospital stay than two weeks following operation. True, a general reduction in mortality and morbidity rates has been enjoyed in practically all forms of surgery, but as comparatively few common procedures carried the high mortality rate of prostatectomy, few have shown such a large reduction.

The following is a synopsis of the last five hundred cases in which prostatectomies have been performed in the Victoria General Hospital. No attempt was made to "select" risks for operation. All patients who, it was felt, would be helped by prostatectomy, were given the benefit of operation, with the rare exception of a severe cardiac or almost moribund patient, and these comprised less than 1% of such admissions.

The following table shows a breakdown of the cases under discussion:

Type of Operation	Number	Average post-op. hospital stay	Mortality
Transurethral	202	15 days	0
Retropubic	276	16 days	0
Suprapubic	22	17.5 days	0
Total	500	16 days	0

The average age of this group was 69 years. The youngest was 37—operated on for calculus prostatitis, and the oldest 92.

One might ask why some of these patients did not succumb to pulmonary embolism or to coronary artery disease. One might also ask why the morbidity in terms of hospital stay was so low. Furthermore, anyone seeing these patients go through their post-operative convalescence might also ask why the post-operative discomfort was so minimal in almost all cases.

I do not believe that the answer lies in any unusual surgical technique. We perform pretty much the same operations on the same class of patient and in more or less the same manner as elsewhere. We conform quite rigidly, however, to the principal of "selective prostatic surgery", believing very definitely that the prostatic surgeon should have several types of operation at his disposal, so that he may choose the operation to fit each particular patient, rather than make all patients conform to one operative procedure.

We believe, however, that a more important part of the answer lies in an organized prostatic "set-up". This revolves around a well-trained operating room and floor nursing staff, who by virtue of their practice and experience,

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not only facilitate rapid surgery—so important in this age group—but also recognize instantly the first signs of impending post-operative complications. This early recognition provides the time element so necessary to ward off such complications or to treat them before they become irreversible. To avoid mortality in prostatic surgery, one must avoid complications. Many elderly patients, particularly the poorer risks, give the surgeon only one chance. If this chance is missed and a major complication ensues, the possibility of a fatality is very much increased.

Our cystoscopic and operating room nurses and the nurses who are responsible for the first vital twenty-four hours of post-operative care are of necessity full time in the Department of Urology and of considerable experience and seniority. Each is a specialist in her phase of the work and each a member of a nursing team of experts. Such special training and team work are absolute essentials and are mainly responsible for the mortality and morbidity figures in this series at the Victoria General Hospital. It is to these nurses that the aging males of Nova Scotia owe their hope and thanks for a freer flowing future and a longer life in which to enjoy it.

While we give to our nursing staff a major portion of the credit, there seem to be worthy of mention certain other contributory factors. To state a few, we would include our highly competent and efficient interne-resident service, the extremely high standard of anesthesia, and thirdly, the physical arrangements within the hospital for immediate post-operative prostatic care—the so-called quiet rooms. There all patients are sent for the first twenty-four hours after operation, to be retained in our own department rather than in the general recovery room. Thus they are within easy reach and under constant surveillance of our urological nurses and our interne-resident group.

The anesthetic requirements are simple. Spinal anesthetic, usually novacaine crystals, is used almost routinely. The importance of maintaining a constant blood pressure and the administration of oxygen to prevent cerebral anoxia, are the constant concern of the anesthetist throughout the operative procedure. So well has this been handled in this series of cases that no post-operative complications which might be attributable to anoxia were noted.

Two other factors which are by no means the least important and to which we owe a great deal, are the earlier diagnosis and earlier treatment. The credit for this is given to those engaged in general practice in the province, who, because of their awareness of the danger of surgery in the elderly, whenever possible, advise these patients to submit to treatment while their cardiac condition is still good and before their kidneys are irreversibly damaged by obstructive back-pressure. This awareness alone has accounted in no small measure for the lowering of both morbidity and mortality rates.

The post-operative morbidity in this series has shown a tremendous improvement. With the exception of occasional bladder spasms, which occurred in a comparatively small percentage of cases, the post-operative course of these patients was not accompanied by a great deal of discomfort. In patients in whom it was thought that the pain threshhold might be lower than average, consideration was given to performing an operation where no abdominal incision would be used—in other words, a transurethral resection of the gland.

In obese patients, where one might expect tension on the sutures to be greater than normal and thereby to predispose to wound infection, one might again choose the transurethral operation. When that procedure was not thought feasible, because of the size of the gland, a transverse incision rather than a vertical was frequently employed. It was our aim to avoid where possible, opening of the bladder or putting sutures into the actual vesical musculature, knowing that such a disturbance of the bladder wall produces varying degrees of painful post-operative spasm. Because of this the retropublic operation results in far fewer post-operative bladder contractions than the transvesical prostatectomy and has therefore been our choice in the majority of open operative procedures.

Post-operative sedation was used as sparingly as possible, though there was no hesitation in its use if actual discomfort was sufficient to warrant it. We are convinced that it is important to keep these patients alert and active from the time of operation and early ambulation is the keynote of our post-operative program. We feel that it is because of early post-operative activity that pulmonary complications and venous thrombosis have practically been eliminated.

Antibiotics and urinary antiseptics as a prophylactic measure were used routinely, the drugs being confined chiefly to Penicillin and Sulfonomides. All other antibiotics were used when indicated as a therapeutic measure only. In severe or resistant infections, the responsible organism was cultured and sensitivity tests carried out. This allowed treatment of the infection with the maximum efficiency and minimum time lag.

Summary

Five hundred consecutive prostatectomies involving the principle of selective prostatic surgery, operated on without mortality and with an improved morbidity, have been presented. Factors accounting for the success of this series include early diagnosis and treatment, a well-trained urological operative and post-operative nursing service, a competent interne-resident staff, an efficient department of anesthesia, and the grouping of all such patients within the hospital in such a way that essential and constant post-operative supervision is easily facilitated.

Case Reports on Endometriosis

By Hugh J. Martin, M. D.,
Sydney Mines.

Having had two cases during this September in which the tissues sent to the Pathological Laboratory definitely determined the lesions to be Endometriosis, I thought it might be interesting to report them, because both cases had a clinical picture that in no way resembled the classical picture of this condition, if, indeed, there is such a picture. The diagnosis is far more frequently made at the operating table than by clinical examination.

So that we might start away together, it might be in order to briefly review the subject of Endometriosis.

The most common variety we see may develop in the ovary and give rise to a chocolate or tarry cyst.

The extragenital endometrioma of similar structure may occur in the recto-vaginal septum, the pelvic colon, caecum, appendix, small intestine, omentum, bladder, umbilicus or in the scar of a gynaecological operation wound.

Like many other conditions, even in this enlightened age, the origin of ovarian and extragenital endometriomata has been disputed, but the best theory so far proposed is the so-called "spill theory of Sampson", who believes that they grow from fragments of uterine mucosa carried in a reflux of menstrual blood outwards along the Fallopian tube and implanted, while still viable, upon some peritoneal surface.

Briefly the arguments of Sampson are:—

1. During operation on menstruating women blood has been seen dripping from the fimbria of the Fallopian tube.
2. Endometrioma occurs only in women, and only during child bearing period.
3. The extragenital tumour tissue, observed through bladder mucosa, becomes swollen and vascular during menstruation.
4. If the patient becomes pregnant, the glands of the tumor undergo a deciduous change.
5. Endometrioma is never found in the upper abdominal organs.
6. The only abdominal operation scar in which endometrioma is known to occur is the scar of an operation upon uterus, ovary or tube.
7. In cases of endometrioma of the pelvic colon a chocolate cyst may be found in the left ovary; in endometrioma of the caecum or appendix a chocolate cyst may be found in the right ovary.

MacLeod has a good argument against this theory, although he accepts the theory of "spill", but suggests it is carried indirectly by way of lymphatic channels. He holds that if direct "spill" were to blame, the broad ligament would be affected most commonly, while, actually, endometrioma of the broad ligament is exceedingly rare.

Now to present the cases.

Case 1.—A mother of five children age 36, began to bleed after six weeks amenorrhea. For the previous year or two it was not uncommon for her to go as long as 40 days, once 44. She complained of slight pain across lower abdomen, but more so on left side. She was the type of individual where assessment of pain was difficult. After a few days bed rest bleeding ceased. Four days after I left for the Yarmouth convention she had a severe attack of menorrhagia. She was sent into hospital in a state of shock, but was revived with plasma and a tranfusion of whole blood. On my return about five days later, the bleeding had lessened, but her general condition required more transfusions. When her general condition improved sufficiently, she was taken in for D & C. The cervix was hard and difficult to dilate. There was very little free bleeding to suggest a miscarriage. Uterine scrapings showed no evidence of products of conception. Her condition continued to improve. It was when she felt good and was up around that I was pleased to have her complain of pain on her right side, as it made it easier to suggest a Laparotomy, having had Ectopic in mind now for some time, while waiting for the best time in her steadily improving condition to break the suggestion to this "nervous person". At Laparotomy we found what appeared to be a typical case of Ectopic Gestation. The left tube was removed. Her appendix, my secret persuasive weapon, was sub-acutely affected and bound down, retrocaecally.

The pathologist's report on the tube showed Ectopic Gestation although he said further sections were necessary to be finally sure. The report three days later stated there was no evidence of products of conception and that the diagnosis had to be changed to Endometriosis.

Case 2.—A mother of three children, age 36, who had her left Fallopian tube removed about 8 years ago for Ectopic Gestation, began to have rather severe attack of menorrhagia in June. The bleeding would stop for a week, then start again. As it was difficult to persuade her to come into hospital I put her on Menagen. She told me that the condition was steadily improving. So for the rest of the summer she had little trouble. In September she and her husband came to see me. She then admitted that she had considerable bleeding still. She was persuaded to come into hospital. She did not complain of pain at any time, although there was now some definite tenderness on deep palpation over the left lower abdomen. I decided on Laparotomy, as I felt she might have uterine fibroids, although P. V. and bimanual examination, which were unsatisfactory, revealed no evidence. At Laparotomy I found a large chocolate cyst of the left ovary which was removed, on the side where I had removed the tube previously for Ectopic. On the right side the Fallopian tube was distended and curled up at its distal portion and contained a reddish serous fluid. The uterus was normal in size and no fibroids could be felt. The right tube was removed and the uterus suspended.

The pathological findings reported nothing remarkable on the right tube. On the chocolate cyst of the left ovary the diagnosis was Endometriosis.

You will notice that in the beginning I stated that I had two cases in which the "*Pathological Report*" definitely stated the diagnosis to be Endo-

metriosis. As far as I am concerned with Case I., I am convinced it was extra-uterine pregnancy regardless of the pathological report, the lack of the usual clinical signs of pregnancy, the state of the uterus, and our own gross examination of the blood clot in the tube, which showed no evidence of gestation.

In Case No. 2, the gross and microscopic findings fitted in with the picture of Endometriosis. With the chocolate cyst of the left ovary, the right tube resembled an early pyosalpinx, only that the reddish fluid, not clotted blood, but free and serous looking, was easily discernible through the unthickened walls of the tube. Certainly there were no inflammatory changes in the walls of the tube, as confirmed by the pathological report. This phenomenon may fit in with Jackson's theory that the reflux retrograde flow of menstrual blood became trapped in a sealed-off tube.

Symptomatically in case No. 2 the clinical picture was atypical, Menorrhagia instead of Dysmenorrhea. Absence of pain, in spite of the pathological conditions found.

From this presentation several conclusions may be drawm.

1. The point, that pathologists themselves stress, that their findings as well as all other laboratory findings, should be read in the light of the clinical findings.
2. How easily one can be misled in the assessment of pain in different individuals.
3. The importance of Laparotomy in case of pelvic disturbances, where physical examination fails to provide a clue. In Case I a preliminary D & C revealed nothing. In Case 2 I did it, to freshen the uterine mucosa, following the Laparotomy, rather than before, as I was quite sure she would require a Hysterectomy.
4. That it is true that Endometriosis is found more frequently at the operating table, than on clinical findings.

These observations in a sense might illustrate the importance of the magnificent presentation of Dr. Moore of Harvard Medical School, a relatively insignificent personage among the illustrious great who attended the latest convention of the American College of Surgeons at New York this fall. Apparently he "stole the show" with his presentation. Before becoming a surgeon, because he liked the agressive approach to a cure, he spent many years studying the physiological and biochemical processes of the human mechanism in their reaction to trauma, which includes operative procedures. He stressed the importance of knowing the physical stamina of different individuals and how they must be prepared physically and mentally to undergo such procedures successfully. I realize that my presentation is only remotely connected with his observations, the only connection being the best time for operative procedures in these two patients. But because it is so important in the general sense, and presented so dramatically recently, I just had to get it in somehow. I liked the notation under his picture, "He tried to bury an old saw, namely: the operation was a success, but the patient died."

Clinical Course of Tuberculosis Correlated With Pathological Concepts

IT is important for the clinician to attempt to visualize in each patient the pathological character and potentialities of the tuberculous lesions upon which prognosis and treatment depend.

The interval of time between the entrance of the tubercle bacilli and the appearance of tissue hypersensitivity, as manifested by reaction to tuberculin, varies considerably (extremes of 19 and 110 days have been reported) but usually averages from four to six weeks. During this period it ordinarily is not possible by clinical or roentgenologic examination to determine whether or not infection has taken place. Symptoms seldom appear until the hypersensitive state has become established.

Tuberculosis Infection

Nondemonstrable Primary Lesions. The initial lesion of primary pulmonary tuberculosis is so small that it is not demonstrable clinically; it is only after its size has increased by direct extension and adjacent endobronchial dissemination that it becomes manifest. Even then, it usually goes unrecognized at the time of its occurrence; ordinarily it is detected only by chance or in specially studied groups. Symptoms often are entirely absent or are so few, or so mild and fleeting, that they are unnoticed or are attributed to other causes. There are usually no detectable abnormal physical signs. A tuberculin reaction may, by inference, indicate the presence of lesions, but in the large majority of cases these are too small to be roentgenographically demonstrable. The vast majority of first infections heal, and the only clinical evidence of their presence is a tuberculin reaction; later their site may be revealed roentgenographically by the appearance of densities of calcification in the tracheobronchial lymph nodes or in the lung parenchyma. Experience has shown that, of all those who react to tuberculin, it is possible to demonstrate a pulmonary or lymph node focus roentgenographically in less than 25 per cent.

Demonstrable Primary Lesions. In children, gross and readily demonstrable pulmonary and lymph node lesions of primary tuberculosis may likewise produce only slight constitutional symptoms in their early phase. Absorption from the lesions may then be sufficient to cause a low or moderate afternoon fever for a few days or weeks; this may soon subside or may recur at intervals. Loss of weight or failure to gain may be noted. There may be irritability and listlessness, but one is often struck by the good general physical condition, brightness, and liveliness of the child. There is a marked disparity between the extent of tissue involvement and the abnormal physical signs, which usually are few or lacking. If the lesions are large and advanced, signs of pulmonary consolidation and of a mediastinal mass may be detected. Roentgenograms, repeated at frequent intervals (every two to four weeks), show best the changes which occur in these unstable lesions. Hemoptysis is an uncommon symptom of ulcerating lesions at this time. The cough, if any, may

be productive of sputum, but young children swallow any discharges which they raise. Consequently, specimens to be examined for tubercle bacilli must be obtained by a laryngeal smear or gastric lavage. Bacilli are most likely to be found in the presence of a sloughing parenchymal focus, but sometimes they may be traced to necrotic tracheobronchial lymph nodes, ulcerating into a bronchus.

Massive enlargement of the tracheobronchial lymph nodes occasionally results in compression of the main bronchi or trachea, giving rise to labored, loud, asthmatic breathing and cyanosis; both breath sounds and cough may be stridulous. The secondary effects of bronchial narrowing may appear as obstructive emphysema, obstructive edema, or nontuberculous bronchopneumonia. Rapid, complete obstruction leads to atelectasis.

Because of the usual peripheral location of the pulmonary focus, local fibrinous pleurisy may develop and, occasionally, pleurisy with effusion, from which it is sometimes possible to recover tubercle bacilli.

A mild clinical course characterized roentgenographically by marked retrogression of the parenchymal and lymph node foci is the most common course in children. There may be resolution without a roentgenographically demonstrable scar, or the lesions may become encapsulated by fibrosis, decrease in size, and gradually become calcified.

Increased resistance is associated with a healed primary tuberculosis even though the evidence of hypersensitivity may eventually be lost. In most of those who acquire primary tuberculosis, the lesions heal and clinical pulmonary tuberculosis never develops. However, in this group, i.e., those who have healed primary tuberculosis, reinfection may occur.

In adults, gross and readily demonstrable pulmonary lesions of primary tuberculosis are indistinguishable clinically and roentgenographically from reinfection lesions.

Clinical Course Related to Age. Pathologically, primary tuberculosis is fundamentally the same at all ages in that it consists of a parenchymal focus with involvement of the tributary broncho-pulmonary lymph nodes. There are, nevertheless, certain differences in the roentgenographic pattern, the clinical course, and in the prognosis at different periods of life. Generalized tuberculosis and meningitis, more common under the age of six and especially before the age of three, are relatively rare in older children and in adults. In young children the lymph node swelling in tuberculosis, as in some other infections, is a prominent feature, whereas in adults it is usually not demonstrable clinically or roentgenographically. Healing of the parenchymal and lymph node foci by calcification, commonly seen in children, is less often observed in adults.

Progressive Pulmonary Tuberculosis

Progressive Primary Tuberculosis. In some, the primary infection is progressive from the outset, the pulmonary and lymph node foci continuing as active tuberculous processes, i.e., progressive primary disease. When the lesions slough into the bronchi and endobronchial disseminations occur, the patient rapidly develops the signs and symptoms of acutely spreading pulmonary tuberculosis. Young children rarely survive them. Older ones may drift into chronic disease and live for years. In adults the clinical manifestations are indistinguishable from those of reinfection disease.

Exacerbation of Primary Tuberculosis. The parenchymal focus may not heal completely but may remain clinically stationary for a time, only to progress at a later date and persist as a well-established pulmonary lesion. The lymph node component may not heal and, because of its close proximity to a bronchus, extension of the lesion through the bronchus may give rise to endobronchial dissemination. This condition is seen more commonly in infants than in older children and adults. In children the lymph node enlargement is a prominent feature, but in adults the lymph nodes usually are not sufficiently enlarged to be roentgenographically demonstrable.

Because of the tendency for disseminated lesions to occur in primary tuberculosis, search for evidence of dissemination by way of the lymph from necrotic lymph nodes, and by way of the blood stream from necrotic parenchymal lesions, should be instituted in all such cases, particularly in individuals under twenty-five years of age. The consequences of discharge of tubercle bacilli into large lymphatics or into veins or capillaries depend largely upon the dosage and the frequency of entrance of the bacilli. A single light seeding of the blood stream may result in a few small lesions in various organs without clinical manifestations. These lesions may heal completely, or some, e.g., those in the bone or kidney, may progress slowly and give rise, at a later date, to serious disease. More abundant seeding may result in a variety of lesions: splenic enlargement, single or multiple crops of tuberculids of the skin, and a light seeding of the lungs. These may heal completely or may persist in a chronic state. Recurrent seeding of the blood stream may lead to protracted and, after a few months, fatal hematogenous tuberculosis. Abrupt overwhelming seeding of the blood stream terminates rather rapidly in generalized miliary tuberculosis. Meningeal tuberculosis may result from any of the foregoing types of hematogenous dissemination.

The clinical picture of serious necrotic lesions of primary tuberculosis and widespread disseminations from them is observed more often in infancy than in later life and more frequently in Negro than in white persons.

Early Infiltrate. The early pulmonary infiltrate, precursor of most of the chronic and fatal tuberculosis in adults, may appear at any age but most frequently between eighteen and thirty or thirty-five (in Negroes a few years earlier). It may be either a primary infection or a new exogenous infection (reinfection). Early pulmonary infiltrates developing in adults have much the same clinical and roentgenological characteristics, whether due to primary infection or reinfection, and it is mpossible by such methods of examination to distinguish one from the other.

In adolescents and young adults the early infiltrate, in the majority of instances, is a primary infection acquired by inhalation of tubercle bacilli a few months to a year previously. In certain instances it may be due to the sloughing of necrotic residues in an unhealed parenchymal focus of a primary lesion, acquired some years previously, which had remained latent. Less often, at least in a large part of the United States today, it is a reinfection.

In older persons the early infiltrate is usually a reinfection. It may be, as in adolescents and young adults, a primary infection. This possibility, however, is naturally reduced in communities where most of those over forty years of age have previously acquired and healed a primary infection.

Symptoms and Signs. The onset may be symptomless, devoid of abnormal physical signs, and denoted only by the roentgenographic appearance of a softly mottled or cloudy patch, a centimeter more or less in diameter, situated usually in the upper and dorsal portion of any pulmonary lobe but most frequently in an upper lobe. Symptoms, when present, are constitutional: mainly fatigue, loss of energy, and small loss of weight; when more severe they suggest grippe, and the lesion may resemble that of bronchopneumonia. Abnormal physical signs are elicited in less than half of the cases at the onset, and in these may consist merely of a few rales heard after expiratory cough in an area scarcely wider than the bell of the stethoscope. If the lesion has not ulcerated, there is usually no sputum. As a rule there is an unidentifiable ulceration in this lesion and, if a mere fleck of mucopurulent sputum is raised by the patient, a specimen of it carefully collected and examined may yield tubercle bacilli. Often this minute discharge is unconsciously swallowed and, in the majority of cases, cultures of fasting gastric contents are positive for tubercle bacilli. To demonstrate the tubercle bacilli, numerous examinations by culture may be necessary, and animal inoculation may have to be made.

The early infiltrate is always an exudative and unstable lesion. It may heal completely by resolution or by fibrosis, or there may be a rapid, slow, or intermittent progression with necrosis, liquefaction and excavation. There is a greater tendency of the lesion to progress in youths, in Negroes, and in persons under unfavorable environmental and constitutional circumstances, e.g., uncontrolled diabetes. These small, exudative infiltrations are always potentially dangerous.

Local symptoms of cough, expectoration, and hemoptysis are indicative of necrosis and sloughing of the lesion into the bronchi, i.e., cavity formation. The finding of tubercle bacilli in the sputum has the same significance. Sloughing marks the beginning of endobronchial dissemination of pulmonary tuberculosis, and prevention or closure of cavities is the most important single requisite for clinical recovery. Otherwise, eventual contamination of other parts is inevitable. If an early infiltration becomes necrotic and liquified and rapidly sloughs its contents into the bronchi, much of the material may be aspirated into healthy parts of the lungs and thus bring about massive infection by tubercle bacilli. The patient then may abruptly develop all of the signs and symptoms of acute and uncontrollable caseous pneumonia, or the period of profound toxemia may last only for several weeks, at the end of which resolution and absorption of some of the inflammatory exudate may occur. Similar changes frequently occur as the result of hemorrhage from a pulmonary cavity.

As a rule, the constitutional symptoms of pulmonary tuberculosis are related closely to the rate and degree of extension of the lesions. A disparity of symptoms, however, is sometimes observed. Some patients are not aware of symptoms even in the presence of advancing disease. At times, progressive excavation of a pulmonary lesion may be demonstrated while the fever lessens and the patient gains weight. Perhaps this is because the products of tissue necrosis are drained off by the bronchi and, consequently, there is less absorption of toxic substances into the blood stream.

Tuberculous lesions of the trachea and bronchi, by their interference with aeration and drainage, often modify, and may profoundly influence, the course and prognosis of pulmonary tuberculosis.

Symptoms are often related to mechanical causes. Patients with extensive pulmonary tuberculosis may recover but may be left more or less disabled with dyspnoea, cyanosis, and other evidence of anoxemia. The explanation for these disabilities may be found in extensive fibrosis, emphysema, pleural adhesions, and retraction of the mediastinum.

The innumerable clinical episodes of tuberculosis all have an organic basis, and the explanation should always be sought.

Artists, Photographers Invited To 9th Physicans' Art Salon

All Canadian physicians and medical undergraduates with art or photography as a hobby are invited to exhibit some of their work at the 9th annual Physicians' Art Salon, to be held at the Royal Alexandra Hotel, Winnipeg, from June 15 to 19, in conjunction with the Convention. All entries in the division of fine arts, monochrome photography, and colour transparencies will be displayed on the convention floor and judged for awards by a panel of outstanding Canadian artists. Members of this panel are: Mr. J. M. Duncan, Mr. Alvin C. Eastman and Mr. Bert Hunter.

By popular request there will be a slight modification in judging of fine art. Entries will be subdivided into traditional and modern works, and prizes awarded in each category.

Again sponsored by Frank W. Horner Limited, the salon is expected to attract a large number of enthusiasts in the various media. Organized orginally to foster restful pursuits in the profession, the Physicians' Art Salon has aroused widespread interest across the Dominion and has become a forum at which artistically gifted physicians can exhibit the produce of their leisure hours before an interested medical audience.

In addition to awarding prizes the Horner Company will reproduce winners in the 1954 Physicians' Art Salon calendar. A copy of this attractive full colour desk model can be obtained by writing Frank W. Horner Limited.

To Enter

Anyone interested in entering work is urged to notify the sponsor, P. O. Box 6139, Montreal, who will furnish full details and the necessary entry form. A short note or postcard will do. All expenses, including the transportation of exhibits to and from Winnipeg, will be borne by the sponsor.

Deadline

Entry forms must be completed and in hand before May 25th to ensure proper listing of exhibits in the catalogue. Exhibitors are also asked to ship entries far enough in advance to allow for the inevitable delays in express and parcel post. Full shipping instructions appear on the entry form.

CANADIAN MEDICAL ASSOCIATION CONVENTION WINNIPEG: JUNE

Members planning to FLY to the above meeting are reminded to establish their travel plans as early as possible! Trans-Canada Air Lines offers a *discount* of approximately 10% from the regular one-way or return fares for parties of ten or more, travelling *together on the going journey.* TCA cannot accept your individual reservation requests at the special rates, and you are therefore urged to co-ordinate your flight dates through the Secretary's office (phone 3-8848) if you wish to qualify for the reduced fares.

As a matter of information the regular TCA fares between Halifax and Winnipeg are $132.90 one way and $239.20 return. It will be worth your while to save 10% by informing the Secretary if you wish to travel with a group (together) to Winnipeg. It is quite permissible to return independently, make stop-overs, etc. according to your individual plans on the *return* flight.

POST-GRADUATE TRAINING

The Halifax Tuberculosis Hospital offers a year of post-graduate training* in Tuberculosis and diseases of the chest starting July 1st, 1953. This hospital has 120 beds for the medical and surgical treatment of Tuberculosis. There is an out-patient service for the diagnosis and follow-up of pulmonary disease particularly Tuberculosis. This hospital is the centre for the Tuberculosis control programme for the City of Halifax and provides all services in case finding, diagnosis, treatment and B.C.G. Vaccine.

Salary $1,800.00 and found.

Address application in writing to Medical Superintendent, Halifax Tuberculosis Hospital, University Avenue, Halifax, N. S.

*This hospital is approved by the Royal College of Physicians and Surgeons as one year's training toward certification or fellowship in Internal Medicine.

THE AMERICAN COLLEGE OF ALLERGISTS

The annual conclave of The American College of Allergists will be held this year at the Conrad Hilton Hotel in Chicago April 24 to April 29.

The first four days will be devoted to instruction under the tutelage of recognized authorities and the last three to a discussion and reporting of recent advances in the field of allergy by the investigators themselves. For detailed information write The American College of Allergists, La Salle Medical Building, Minneapolis 2, Minnesota.

ASSISTANTSHIP WANTED

An assistantship is wanted in the Halifax area. For further particulars apply to the Secretary of The Medical Society of Nova Scotia.