The Management of Acute Myocardial Infarction*

LOUIS WOLFF, M. D.

ACUTE myocardial infarction must be thought of and treated as an emergency. Although many of its victims hardly appear sick, the threat of serious complications, or sudden, unexpected death, is always present. The patient should be put to bed as soon as possible, and if it is necessary to move him for this purpose, he should be transported by stretcher and ambulance. It is generally safe to do this, providing he is not in shock or suffering from severe pulmonary edema.

If complications do not occur, treatment consists in bed rest, relief of pain, anticoagulant therapy, the maintenance of a calm, cheerful, confident attitude in the patient, and good nursing care. Oxygen is administered if cyanosis is present, or the pain is severe. A liquid diet is prescribed for the first 24 or 48 hours, and if a liberal fluid intake by mouth is not possible, and certainly if dehydration is evident, 5% glucose in water is given slowly intravenously. The caloric intake should be moderate, but it is not necessary to restrict salt. B complex and vitamin C are given routinely. There is a tendency to overtreat patients, not only with too many drugs, but with unnecessarily large doses, and often improvement is noted when all medication is omitted. Drugs should be given for specific indications, with a clear notion of what is being treated, and with full knowledge of their therapeutic and side effects.

There is no substitute for narcotics in the treatment of pain. If the pain is severe large amounts of the drug are required, and may be given with safety, unless the patient is myxedematous, of advanced age, or has chronic pulmonary disease. An initial dose of 30 mgs. of morphine sulphate with atropine is reasonable. It is futile to administer sedatives for restlessness, or hypnotics, for insomnia unless the pain is adequately controlled. Barbiturates often increase restlessness or result in mental confusion and disorientation in elderly subjects and their injudicious use is harmful. Chloral hydrate and paraldehyde are effective and harmless hypnotics.

My experience with anticoagulant therapy in the treatment of acute myocardial infarction has been very gratifying, and I am convinced its proper application reduces morbidity and mortality. Dicumarol is the drug of choice for this purpose, and it may be given to all patients whose control prothrombin time is normal, except those wth a hemorrhagic diathesis, liver disease, or renal insufficiency. Its use requires careful supervision, and this is possible only if reliable determinations of the prothrombin time are available day by day. Once the drug is exhibited it should not be omitted until the patient is ambulatory, unless toxic manifestations require its discontinuance.

Undesirable side effects are not often observed when the drug is administered in therapeutic doses under careful laboratory control. These consist exclusively in hemorrhagic phemonena with the uncommon exception of abdominal cramps followed by one or two bulky stools after the first dose of 300 mgs. of dicumerol, and the rare instances of abdominal pain resulting from

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the subsequent smaller doses. Hemorrhagic phenomena, such as microscopic hematuria or guaiac positive stools, are common, but are not serious. While their occurrence does not necessitate withdrawal of the drug, unusual vigilance and readiness to treat more serious hemorrhage are required. In a large series of dicumarol treated cases of acute myocardial infraction I have seen but one type of serious and fatal complication, comprising three cases of hemorrhagic pericarditis. Since we have never observed frank hemmorrhage into the pericardial sac in untreated cases in the absence of myocardial tear or rupture of a coronary vessel, the conclusion appears warranted that dicumarol is related to this serious complication. Our experience with pericardial hemorrhage, small as it is, enables us to formulate a basis for its detection. The unusual persistence of a pericardial friction rub following acute infarction is sufficient to arouse suspicion of oozing of blood into the pericardial space, especially if the prothrombic activity has been unduly depressed, but even if therapeutic levels have not been exceeded. Precordial pain and sudden vascular collaspe in such cases suggest profuse pericardial hemorrhage, and the danger of cardiac tamponade and death. Under these conditions, even though the diagnosis may not be certain, dicumarol is withdrawn, vitamin K is administered intravenously, and plasma or blood transfusions are given repeatedly as indicated by frequent determinations of the prothrombin time. Treatment is discontinued when the tests disclose maintenance of normal prothrombic activity. Pericardial paracentesis may be necessary, and may be a life saving measure.

Opinion concerning the length of time that bed rest is required has changed many times in the past 30 years, and there is still no agreement on this point. There can be no question that unnecessarily long periods of bed rest have been enforced in the past, and the prevailing tendency to shorten this period is a step in the right direction. But, as so often happens when a change is in progress, the new point of view becomes excessive and extravagant. That some patients appear to do well with very short periods of bed rest or none at all, is not a convincing argument, any more than recovery from perforating acute appendicitis without operation is an indication that surgery is not necessary in acute appendicitis. There may be justifiable economic and social reasons for curtailing bed rest, but these do not change the basic medical principles. Also, in occasional cases of mental aberration, and as a rule in the aged, ambulation is permitted earlier than would appear advisable under other conditions.

The harmful and potentially dangerous effects of bed rest are well known: thrombo-embolic complications, pulmonary congestion, impaired bladder and bowel function, muscular wasting and negative nitrogen balance, and the effect on the patient's psyche. Although skillful management can prevent or overcome some of these undesirable ill effects, the list is an impressive one, and is a powerful argument against unnecessarily prolonging bed rest, which, fortunately, is being heeded by the medical profession in increasing numbers. In uncomplicated cases 3-4 weeks in bed is sufficient, and as a rule nearer 3 than 4 weeks is adequate. Then activity is progressively increased, but at a slow pace. Except in the mild case a patient should be considered convalescent for 2-3 months following the onset of the attack, and social, professional and business activities, and travelling are forbidden during this period. Every effort should be made, unless contraindicated by sound medical reasons, to return the patient to his occupation, at first on a part time, then on a full time, basis. Upon termination of convalescence most patients are able to engage in a gainful occupation, and it is the physician's duty to encourage this and

to help the patient select work that is compatible with his health. It is advisable to warn the patient against any activity that produces pain or dypsnoea, against sudden, excessive or unaccustomed exertion, large meals, and emotional tantrums.

Before discussing the important subject of complications I would like to state my point of view concerning the management of angina pectoris of recent origin. This properly belongs here, for as pointed out yesterday, acute myocardial infraction is frequently preceeded by premonitory warnings. Or, to put it the other way, the first appearance of agina pectoris is frequently followed by acute myocardial infarction, within four weeks of its onset. From a practical point of view, therefore, this period must be set off as one in which acute infarction is particularly likely, and a definite attitude in regard to treatment should be formulated. During this early phase in the life history of coronary heart disease, when pain appears for the first time, the proper diagnosis is impending myocardial infarction. It is not possible to foretell which of these patients will develop acute infarction, but it appears more probable when attacks of pain recur in rapid succession, when pain occurs without any apparent provoking cause or is of long duration, when it awakens the patient out of sleep, when it is associated with a rise in sedimentation rate, and changes in the electrocardiogram of a kind which are not indicative of infarction. Nevertheless, not infrequently a patient may have a single short attack of pain, and after an interval of 3-4 weeks of well being, suddenly develop acute infarction.

There can be a reasonable difference of opinion regarding the handling of these patients, since some of them continue their usual mode of life without ill effect, while others are put to bed yet develop acute infarction. But I have a strong impression that the overall results are far better if the patient is taken off his feet, and I insist on bed rest when the manifestations indicate an especially high probability of ensuing infarction, as mentioned above. As for the others, it is desirable that they give up all activity and remain at home, but not in bed, unless unusual hardships are thereby created. There is no medication of proved value in these cases, and nitroglycerine may be harmful in some of them. At any rate it should not be used freely, and not at all if it fails to relieve pain. Demerol is effective and is to be preferred to nitroglycerine for prolonged pain. I have administered dicumarol to several patients in this group but without being able to appraise its value.

The clinical picture in the type of case under discussion is often atypical or bizarre, and may suggest gastro-intestinal or other disease as much, or even more, than coronary heart disease. I have seen a number of such patients develop acute myocardial infarction while in the X-ray room going through the strenuous experience of extensive investigation. It is desirable, therefore, if there is any suspicion whatsoever of impending myocardial infarction, to delay investigation until the situation is clarified, or the critical period has ended. If the gall bladder is being investigated, the dye should never be given intravenously, and pitressin should never be used.

The course of acute myocardial infarction, once the pain is under control, and in complete absence of complications, is singularly benign. The physician continues to see the patient every day, however, keeping a watchful eye for complications, which can occur at any time, but particularly in the first two weeks, and to perform the very useful functions of reassuring him that all is well, maintaining his spirits, and instilling optimism. Complications will be more readily detected if the physician knows which are likely to occur, and

when, in the course of the illness, they are most probable. A drop in blood pressure not reaching shock levels, and a moist clammy skin at the onset, or occuring in the first few days, cannot be considered as complications, but their appearance later, especially after a period of well being, must be regarded with suspicion. Pericarditis may be looked upon as a normal development in the first week, but its advent after this, or its persistence in dicumarol treated patients, as already mentioned, suggests the serious complication of pericardial hemorrhage. Crepitant rales at the left lung base are often noted in patients with acute myocardial infarction, and are not necessarily due to congestive failure, pulmonary infection, or pulmonary embolism. Oliguria and an elevated NPN are common in the first few days, and require no treatment providing the patient is not dehydrated. Mild glycosuria and hyperglycemia do not necessarily establish the diagnosis of diabetes, but indicate the need for continued observation and perhaps a sugar tolerance test. Mild abdominal distention is common in the beginning, but occasionally it reaches such proportions as to seriously embarras circulation and respiraton, suggesting an abdominal complication. Almost without exception, however, enemas, a rectal tube, and turpentine stupes alleviate the condition.

The complications which are commonly seen and which we must be prepared to treat are shock, pulmonary edema, congestive failure, arrhythimas, and thrombo-embolic phenomena. Shock commonly occurs at the onset or early in the course of the disease, and if it is corrected following the injection of caffeine or coramine, which is the treatment employed by many physicians, it is doubtful that the treatment was responsible for the improvement. At any rate, if the process does not reverse itself promptly, more active treatment should be instituted at once. The slow intravenous injection of 5% glucose in water is desirable in the presence of dehydration, in addition to plasma, whole blood, or salt free albumin. Vasopressor drugs which do not increase heart work, such as paradrene or neosynephrin, should be given at frequent intervals. Oxygen is given routinely, and opiates are administered for restlessness or pain. Treatment must be continued without interruption until the desired effect is obtained, or until the patient expires. The possibility that shock is the result of pulmonary embolism, extreme tachycardia consequent to paroxysmal rapid heart action, or pericardial hemorrhage, should be considered and appropriate measures taken if these conditions are discovered.

Pulmonary edema, like shock, may occur at the very onset or at any time in the course of the disease, and may be recurrent. Although it may be self limited, mild, and of short duration, measures should be taken to combat it at once in every case, and in every attack, Oxygen, morphine and atropine, high head rest, and elevation of the head of the bed are often effective. If it persists in spite of these measures, additional treatment is necessary without too much delay. A mercurial diuretic is given intramuscularly, and if wheezing is noted, and perhaps even in its absence, 0.5 gm. of aminophyllin is injected slowly by the intravenous route. Tourniquettes are applied to the extremities, and as a last resort a small venesection may be carried out. All these measures may fail, and improvement will be noted only when the patient is supported sitting with his legs hanging down over the edge of the bed, or sitting in a chair with his legs down. Digitalization is often resorted to, but I am not convinced of its value in these cases. Pulmonary edema may be precipitated by pulmonary embolism, paroxysmal rapid heart action, renal insufficiency, or a large intake of sodium salts, and these possibilities should not be overlooked.

The coexistence of shock and pulmonary edema is not uncommon, and carries with it an extremely grave prognosis. Treatment is difficult, since measures beneficial for one condition may be detrimental for the other. Oxygen, morphine, aminophyllin, and vasopressor drugs are probably harmless, but treatment is not often effective and may do more harm than good. Albumin intravenously may aggravate the pulmonary edema, and if fluids of any type are given by this route the slightest evidence of increasing moisture in the lungs is an indication for immediate cessation of therapy.

Gross congestive failure may appear early in the attack, or any time thereafter; it may become evident for the first time when the patient is ambulated. Slight dyspnoea, cough and minimal wheezing may be the first symptoms, and basal rales, a tender liver, or sacral pitting edema the first signs. Treatment consists in digitalization, low salt diet, and mercurial diuretics.

Paroxysmal rapid heart action seldom occurs before the second and third day, but may occur anytime thereafter, and is often recurrent. If the tachycardia is due to auricular fibrillation it is not necessary to treat it, since the ventricular rate is usually in the vicinity of 110-120, which does not burden the heart or circulation, there are no symptoms associated with it, and the paroxysm is self limited and of short duration. In the occasional cases with ventricular rates of 150 or more, or when the paroxsym persists for days or longer, digitalis is the drug of choice since quindine may induce fixed auricular flutter with extremely rapid ventricular rates. Digitalization is likewise the preferred treatment for paroxysmal auricular flutter. Quinidine, however, is the only drug which is regularly effective in paroxysmal ventricular tachycardia, and properly used, may be life saving. The plan of treatment which I employ, and which has been highly successful, requires close supervision. The drug is given every hour by the clock, in 0.4 gm. doses, until the paroxysm ends. Progressive slowing of the heart rate indicates the dose is adequate. If slowing does not occur the dose is increased with each medication. On this schedule extremely large doses of quindine can usually be avoided. I prefer to give the drug by the oral route but if this is not possible, intramuscular injections are satisfactory. Intravenous quindine is dangerous.

High grade A-V block is a much more common complication than is generally realized. It rarely occurs at any other time than the first week of the attack, and if frequent electrocardiograms are obtained in cases of posterior infarction during this period it will be noted fairly frequently. The diagnosis is missed otherwise, because the ventricular rate in the vast majority of cases, even with complete block, is not slow enough to suggest its presence. Having noted this arrhythmia, those in attendance must be prepared to treat Adams Stokes attacks. Since these rarely occur and the block disappears permanently in hours or one or two days, treatment is not often necessary. If the heart stops beating long enough to produce a seizure, 0.2 to 0.5 cc. of a 1/1000 epinephrine solution is given hypodermically at once, and repeated as necessary. Once a seizure occurs, it is advisable to repeat the injection routinely every two to three hours for 24-48 hours, to forestall a recurrence.

I would now like to summarize what I have said about treatment, and to add a few remarks in conclusion. The doctor should learn to recognize impending myocardial infarction, and treat it by curtailing the patient's activity, or putting him to bed. Extensive diagnostic procedures are to be avoided, and surgery is dangerous during this period. Uncomplicated myocardial infraction, once the pain is controlled, demands little of the scientific skill of the physician, but challenges his ability to practice the art of medicine. The situation is

different when complications occur, as they commonly do, and contribute greatly to morbidity and mortality. Here, it is just as important to know what not to do, as it is to know what should be done, and when. Patients are often harmed by overtreatment. Even when therapy is applied for proper indications, and is apparently effective, we cannot always be certain our interference has turned the tide. Some progress has been made in preventing thrombo-embolic complications with properly applied anti-coagulant therapy, and the treatment of other complications has been discussed.

Morphine, or other narcotics, oxygen, and dicumarol are the mainstays of medicinal treatment. Digitalis properly used is harmless in the absence of shock, but its indications are limited. I believe quinidine is not necessary as routine therapy, and I have even abandoned its use for occasional premature beats. Diabetes should be controlled by dietary measures. and if insulin must be used, great caution is to exercised. Smoking is prohibited during the attack, but I have no objection to it in moderation after the patient has recovered. Minor surgical procedures are occasionally necessary and may be done. Whenever possible surgery should be postponed for at least two months after the attack.

The most important problem in this field is the prevention of acute myocardial infarction, which, fundamentally, is the prevention of atherosclerosis. We know that patients with diabetes, hypercholesterolemia, hypertension, and polycythemia, appear to have a predisposition to hardening of the arteries, and that heredity is vastly important in its development. Anything beyond this is mostly speculation. Our knowledge is still too meagre to warrant an opinion on the role of fat in the diet, or other dietary factors, hormones, and other constitutional elements. The challenge has been accepted by the profession, and it is to be hoped that the great amount of work now being done in this field will be fruitful.

Pathological Conditions of the Ocular Fundus*

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PATHOLOGICAL conditions of the ocular fundus cover such a wide field that it is my intention in the short time allotted to me, to take up the more common ones, which we are more apt to be called upon to deal with, and which consequently are of more interest to us. Namely:

(1) Embolism of the Central Retinal Artery.

(2) Thrombosis of the Central Vein.

(3) Arteriosclerotic Retinitis.

(4) Renal or Nephritis Retinitis.

(5) Diabetic Retinitis.

(6) Retrobulbar Neuritis.

(7) Papillodema.

(1) Embolism of the Central Retinal Artery.

The commonest cause of embolism is endocarditis, and the site of the blockage is usually at the bifurcation. *Signs and Symptoms*: The vision is suddenly lost. On ophthalmoscopic examination the main branches of the artery are found to be very narrow, ill-defined and empty. The disc is pale and there is a pallor over the whole of the macular area. This pallor ranges from a grey-white to a dead-white due to oedema in the retina. The fovea usually carries the characteristic cherry red spot, although this may be absent. After a few days the blood stream may begin to function and it reappears in an interrupted stream. This picture may occur in thrombosis, arteritis or spasm. *Aetiology*: As previously stated, heart disease is the commonest cause the second being arteriosclerosis. *Diagnosis*: The sudden onset and the characteristic retinal picture are indicative. *Prognosis*: Is very bad. A small amount of vision may return from canalization of the embolism, but this is rare. The condition is usually found on one side only, and the area may vary, if one branch of the artery only, is involved, in which case, only the quadrant of the retina supplied, will be affected. *Treatment*: Paracentesis of the anterior chamber, within twenty-four hours of the onset, in an effort to have the embolus passed, into the terminal branch. Amylnitrate should be given and vasodilator therapy, with continuous hot baths should be tried.

(2) Thrombosis of the Central Vein.

This is due to endophlebitis or simple thrombosis, at or proximal to the lamina cribrosa. Retinal arteriosclerosis and high blood pressure are usually present. In young persons who have suffered some infection, such as influenza facial erysipelas or orbital inflammation; it is due to simple thrombosis, but

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generally it is a disease of middle life. *Signs and Symptoms*: The loss of vision is fairly rapid. Veins are intensely engorged, tortuous and dark. There are abundant haemorrhages present throughout the fundus, which are flame-shaped and more evident at the disc. The latter is reddish in colour, usually edged with small exudate, giving the appearance of a blur. Thrombosis of the branch vein only may occur, and in that case, the haemorrhages are in the section of the retina which it drains. *Prognosis*: If the branch vein is involved there may be only a small field loss depending on the area supplied. In a thrombosis of the central vein, however, no useful vision remains. Glaucoma is a frequent complication. *Treatment*: When glaucoma results treatment is non-effective. Where some degree of vision remains miotics should be given as a preventative against impending glaucoma and the general treatment of arteriosclerosis should be carried out.

(3) Arteriosclerotic Retinitis:

This is an advanced stage of retinal arteriosclerosis in which exudate and haemorrhage occur. The disc is ill-defined and the exudates are mostly in the macular area in the form of snow-white rounded spots. The usual signs present in arteriosclerotic degeneration of the retinal vessel are:

(a) Alteration in the arterial calibre of the vessel—the lumen narrows and widens peripherally.

(b) Visibility of the vessel wall is increased, and it appears as a thin white streak on account of the fibrosis and gives the so-called silver wire appearance.

(c) At the arterial venous crossing, the vein which is usually seen through the artery, is obliterated by the thickened arterial wall.

(d) Retinal haemorrhages. These are flame-shaped, as they are in the nerve fibre layer.

Prognosis: The differentiation of arteriosclerotic retinitis from nephritic retinitis is important. The treatment differs and in arteriosclerotic conditions there is a longer life span, death being due, usually, to a cerebral haemorrhage. In nephritic retinitis death is usually from the onset of uraemia. Vision may be effected very little from haemorrhages and exudate, unless they are situated at the fovea. Arteriosclerotic retinitis may be unilateral, but is generally bilateral. *Treatment*: Rest in bed. Restriction of activity. Potassium Iodide. Rutin with Vitamin C and other arteriosclerotic therapy.

(4) Renal or Nephritic Retinitis:

This condition is due to changes in the retina from a toxic factor. It is more common in chronic nephritis. *Signs and Symptoms*: Oedema of the disc is usually present, accompanied by flame-shaped haemorrhages in the central part of the retina. The exudate is of the large cotton wool type, and a fan-shaped figure with bar-like radiation is frequently present and is more or less characteristic of this condition. Papilloedema is sometimes the only retinal sign present, and in this case must be differentiated from a condition of cerebral

pressure, such as seen in tumour. *Prognosis*: The prognosis for renal retinitis is about two years. In pregnancy a rapid and complete loss of vision may occur where no previous nephritis existed. In this case there are no visible retinal changes except slight haemorrhages and these cases make a rapid recovery, in most cases with the termination of the pregnancy.

(5) Diabetic Retinitis

As this condition is not present in the severe diabetes of young people it is, therefore, not a true retinitis, but is found more in middle life where there is some albumenuria and high blood pressure. The characterisitc signs and symptoms are wax-like exudates which have clear-cut margins. The haemorhages are small and rounded as these occur in the deeper layers of the retina. The treatment is that of diabetes as a whole, but unfortunately the haemorrhages and exudates often remain and are unaffected by the disappearance of the sugar from the blood and urine.

(6) Retrobulbar Neuritis and Intra-ocular Neuritis:

This, like any other neuritis, is an inflammation of the optic nerve trunk. It is secondary to infection elsewhere in the body and syphilis is also a causative factor. It is also seen in acute contagious diseases. In the retrobulbar form disseminated sclerosis especially in patients between twenty and forty years of age, is a prominent cause. *Signs and Symptoms*: There are signs of inflammation and pain, and the eyeball and orbit are tender on pressure. Vision is practically lost, and the field is contracted and the blind spot increased. Papillitis is usually present and the condition is followed by optic atrophy. The treatment is antibiotics, heat, salicylates, and removal of toxi foci of infection.

(7) Papilloedema:

Papilloedema, or choked disc, is a sign of intracranial pressure. Brain tumour is present in 70% of the cases. Other causes are cerebral syphilis, tuberculosis, brain abscess, meningitis, sinus thrombosis and subdural haemorrhage. *Signs and Symptoms*: It is remarkable that very little disturbance of vision may be present, but the other symptoms of intracranial pressure such as vomiting, headache, photophobia, are present. With the ophthalmoscope the nerve head is seen to be raised and is white and puffy. Flame-shaped haemorrhages are present. The treatment is that of the condition underlying the intracranial pressure and the prognosis of recovery of vision is good, if this condition can be relieved.

In this connection I would like to give the case history of a man, of about twenty-five years, who engaged in a boxing bout in October last and was knocked out. Some six weeks afterwards he developed a headache, later followed by vomiting. He was sent to the hospital by his doctor for sinus disease and was referred to me. On examination I found the sinuses normal. The pupil was slightly dilated. No fever. There was oedma of both discs, with flame-shaped haemorrhages extending out from the latter. He had received a blow on the left side of his head. The signs were more evident in the left eye, but according to the authorities this has no relation to the site

of the lesion. His field of vision was quite good and he did not complain of loss of vision. A probable diagnosis of a cystic tumor, secondary to a subdural haemorrhage was made, and the patient was sent to Doctor Stevenson, neurosurgeon, for treatment. He was operated upon and a cyst removed from the parietal region. The vision returned and he has been prefectly well ever since.

The interesting points about this case are: (1) That his symptoms did not come on until six weeks after the blow. (2) Loss of vision or other eye symptoms were not what he complained of but simply headache and vomiting, with the result that the connecting history was only obtained after the characteristic eye signs were found.

A Plea for Early Treatment in Carcinoma of the Cervix

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THE routine in the treatment of cancer of the cervix at the Victoria General Hospital has been a combination of X-Radiation and Radium. In December 1941 the radium salt was first made available at this centre and its use supplanted the radon emanations which had been used for several years previously. Since then sufficient time has elapsed for an evaluation of the results. In the hope of obtaining further improvement, a study of these results was recently completed.

The series included all of the cases of Carcinoma of the Cervix treated by the combined efforts of the Departments of Gynaecology and Radiology from December 1941 to December 1944 inclusive. The radium was applied by the Gynaecological Staff only and the X-Radiation given under the direction of the Radiologist.

The total number of patients reviewed was 129. The data on 127 of these is up to date as of December 1949. The remaining two cases could not be located and for purposes of this study have been considered dead.

The classification of cases used is the Clinical one as laid down by the League of Nations. The overall survival rate was 31.7%, and Table I shows the breakdown according to the stages of disease.

TABLE I

December 1941 to December 1944 (incl)

Stage	Incidence		Survival in %
	No.	Percent	
I	24	1 8.4	58.3
II	63	48.7	36.2
III	36	27.9	11.1
IV	6	4.6	0
TOTAL	129	....	31.7

Most of the large clinics reporting have shown a survival rate varying from 30% to 40% so that while our cure figures fit into that category, they do so fairly low down in it. (See Table II)

TABLE II

Institution	Survival in %
Ontario Institute of tadiology (1945 Report)	36.5
Marie Curie Hospital, London (1945 Report)	36.9
J. Heyman (Radium Hemmet)	34.9
University of California	39.9

Comparing this present report with a previous one from this hospital in 1935, considerable improvement is noted. (See Table III.)

TABLE III

1935	1949	Difference
18%	31.7%	13.7%

At present the departments of Gynaecology and Radiology are at work improving technique in the hope of a resultant rise in the survuval rate. It is not, however, the treatment per se that is most important, but rather the earlier the stage of disease at which it is instituted. A glance at Table I shows that 18.4% of the cases were in Class I—the group that had a survival rate of close to 60%. It will also be noted that in Group II, the survival rate was 36.2% and in Group III only 11.1%. If all cases had been in Group I we would have alive today 75 women—instead lf 41—almost twice as many. In other words, if we could get our cases early while still in Group I we would cure about 2 out of every 3 women with Cariconoma of the Cervix.

It is therefore urgent and essential that if we are to offer a real chance of cure to women with this disease, we must keep urging them, no matter what their age, to present themselves immediately they notice any irregular or unusual vaginal bleeding or discharge, and where there is the slightest suspicion of maligancy, send them to hospital for confirmation and early treatment; for in the present state of our therapy there is no other hope for them.

BIBLIOGRAPHY

1. American Journal of Roentgentology—December 1947.

2. J.A.M.A.—October 18, 1947.

3. American Journal of Obs. & Gyn.—November 1949.

Obituary

DR. H. D. CHISHOLM

ON March 12th, 1950, in the twilight hours of the Sabbath, there passed away at this home in Springville, Pictou County, one of the best known of our County practitioners, Dr. Hugh Dan Chisholm. He was in his 69th year.

He was born at Springville. When quite a small boy, the family spent a short time in California, but soon returned to the old home, so Dr. Chisholm received his early education in the one room school there. As did the older Pictou County doctors, he attended Pictou Academy and from thence went to the University. He graduated from Dalhousie Medical College in 1907 and the following year interned in the Victoria General Hospital.

Following this, he practised a few months in Wabana, Nfld., where his father was an official for Dosco. He then relieved a doctor in Glace Bay but his heart was in the beautiful valley of the East River, and he returned to his old home and people where he worked for almost forty years.

During his "horse and buggy" days he led a very strenuous life. Some of his patients lived thirty miles from his office. During the spring, fall and winter, these trips were often very hazardous. Frequently he finished his journey on foot, horseback or snowshoes, but he neve rfailed to reach his patient and if at all fit, he never refused a call.

For forty years he attended his maternity patients in private homes. With only the assistance of a neighbour woman, he met and solved many obstetrical emergencies and never lost a mother—an enviable record.

He was remarkably free from envy and jealousy and had a great capacity for friendship. He was greatly beloved by his patients.

On a beautiful, sunny March day his body was laid to rest in the family plot at Bridgeville. The Church was filled to overfiowing. Very few homes in the East River Valley were not represented. Friends, Doctors and patients came from far and near to pay their last respects to a noble man.

He is survived by his wife, two daughters and one sister. His wife, Adeline Himmelman, is a graduate of the Aberdeen Hospital and in his last painful and trying illness ministered lovingly, faithfully and efficiently to his needs.

Dr. Chisholm will be greatly missed and long remembered.

"He rests from his labours and verily his works do follow him."

R. M. B.

One of the few physicians in Dawson City, Alaska, during the great Gold Rush of 1898, Doctor George Herbert Fulton, died February 11th, at the home of his daughter, Mrs. W. A. Black in Malden, Mass.

Doctor Fulton, a native of Bass River, was born in 1853, and retired a number of years ago. He graduated from Dalhousie Medical College in 1883, and was the oldest living graduate. He began practice in rural communities on the Northern Shore of New Brunswick and returned to his home for a few years before he caught the Klondike fever. With hundreds of other men he

went to the West Coast and then made the long and hazardous trek from British Columbia through the Chilkoot and White Horse Passes into the Yukon Valley to Dawson City. At that time the city consisted of one hundred huts and one muddy street.

He remained in the Arctic for three years and finally returned to a practice in Malden. His father George Fulton, founded the Dominion Chair Company, Limited, at Bass River.

Doctor Fulton is survived by three daughters, Mrs. W. A. Black, Malden; Mrs. M. H. Mellish, Malden; and Mrs. E. B. Mode of Wellesly; one sister, Mrs. Herbert Bentley, Westwood. Seymour A. Fulton, assistant manager of Dominion Chair Company, Limited, Bass River, is a nephew and Mrs. R. B. Lewis of Little Bass River, is a neice.

Funeral services were held February 14th at the Sprague Funeral Home in Malden.

Doctor Bernard Francis of Sydney Mines, died during the weekend of April 2nd after a long period of ill health. Doctor Francis graduated from McGill in 1899 and was a widely known specialist in treatment of children. The funeral procession was one of the largest in the memory of veteran residents. Mourners attended from many parts of the province.

The BULLETIN extends sympathy to Doctor C. J. W. Beckwith of Halifax on the death of his father, W. H. H. Beckwith, D.D.S., at Bridgetown on April 7th, at the age of 75. Doctor Beckwith had been critically ill for the past six weeks from a heart ailment.

PHYSICIAN NEEDED

The village of Port Hood and surrounding community is in need of a medical doctor. Port Hood is on the main highway between Port Hawkesbury and Inverness, and has a population of seven hundred people and a doctor located there would serve around two thousand people. The Government will subsidize a doctor located there to the extent of $1,200. For further information write Mr. A. S. MacPhail at Port Hood.

Doctor S. H. Holloway of St. Andrews, N. B., is leaving there this spring and returning to his home province of Alberta, which will create an opportunity for a doctor to locate at St. Andrews. The practice has grossed close to $7,000 a year for the past three years.

A physician interested in a combined town and rural practice should communicate with Rev. C. R. Harris, Rector, St. George's Church, Parrsboro, N. S.