VOL. XXX No. 5

The Nova Scotia Medical Bulletin

Official Organ of The Medical Society of Nova Scotia Canadian Medical Association Nova Scotia Division.

MAY, 1951

Published on the 20th of each month and mailed to all physicians and hospitals in Nova Scotia. Advertising forms close on the last day of the preceding month. Manuscripts should be in the hands of the editors on or before the 1st of the month. Subscription Price: $3.00 per year.

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Iron Deficiency Anemia and its Treatment with Saccharated Iron Oxide

By William I. Morse, II

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MANY periodicals currently draw our attention to a new iron preparation suitable for intravenous use in large doses, namely saccharated iron oxide. There has been considerable study of this agent in the last 3 years [1, 2] and it seems timely to emphasize some aspects of iron deficiency and to define tentatively the indications for and the contraindications to intravenous saccharated iron oxide.

The normal male excretes very little iron. The additional loss in the female occurs through menstruation and childbirth. Because of the negligible excretion, the organism normally controls its iron balance by regulating iron absorption from the intestine.

Etiology of Iron Deficiency Anemia. One will rarely have to resotr to a diagnosis of ideopathic hypochromic anemia if all possible causes of iron deficiency are investigated in the patient. In many cases more than one factor contributes.

Hemorrhage is the outstanding cause. A careful search for blood loss should be instituted in any patient showing an anemia with the features of iron deficiency. In the older age group a malignant tumour is not infrequently demonstrated. Genital blood loss probably accounts for the higher incidence of iron deficiency anemia in the female. The blood loss may be hard to detect as in certain patients with hiatus hernia or intermittently bleeding hemorrhoids. It should be emphasized that the major part of the blood loss may have occurred months or even years before the patient is seen in some instances. The contribution of repeated blood donations is important in those individuals predisposed to iron deficiency for any other reason. The danger of harm from properly spaced donations of blood is minimal if the donor is not losing blood abnormally by other routes and the causes of iron deficiency to be discussed are not present.

Pregnancy contributes to iron deficiency because of fetal requirements and some blood loss at parturition. The infant of an iron deficient mother often becomes anemic. *Growth* increases iron requirements and, therefore, contributes in the above instance.

Dietary Deficiency of iron is a factor in some cases. This used to be common in the first year of life. The following foods are considered good sources of iron: liver and other organ meats, muscle meats, brewer's yeast,

1. Nissim, J.A. Lancet 2: 49, 1947.
2. Slack, H. G. B. et al. Lancet 1: 11, 1949.

molasses, brown sugar, egg, whole wheat flour, oatmeal, whole grain, cornmeal, spinach, turnip, beans and peas.

The role of *iron absorption* from the gut in iron deficiency is still confusing. Attesting to its importance, however, is the fact that patients are occasionally encountered in whom the anemia persists for no apparent reason when adequate iron is provided in the food or even following large doses of ferrous sulphate. In figure 1. several of the factors are depicted which we know can influence iron absorption but the relative importance of these and probably additional factors remains obscure.

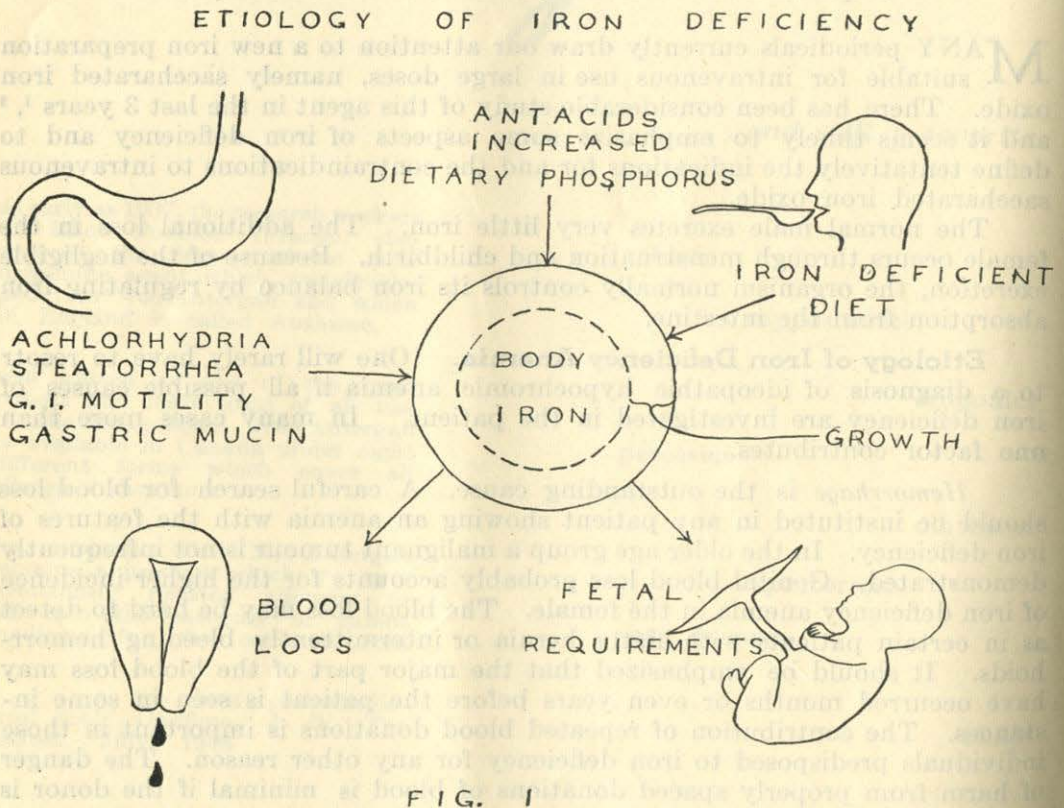

FIG. 1

Iron absorption is chiefly at the duodenal level, but some absorption can occur lower down in the intestine. It seems likely that hydrochloric acid in the stomach aids considerably in changing iron from food sources into a soluble form.* It is then reduced by substances in the food and chiefly absorbed as the ferrous ion. Hydrochloric acid also retards transformation to insoluble iron salts in the intestine. There is some evidence that hydrochloric acid is necessary for the full benefit of therapeutic oral iron preparations [3], [4], but this

3. Mettier, S. R. et al. Am. J. Med. Sc. 181: 25, 1931.

4. Bethell, F. H. et al. J.A.M.A. 103: 797, 1934.

*C. V. Moor et al, have recently provided evidence that absorption of iron from food sources is not diminished by achlorhydria or ingestion of aluminum hydroxide, and is not increased by iron deficiency. These startling findings await confirmation and further study.

is controversial. The intestinal mucosa has a very limited capacity to absorb iron, so that in most situations a dose of 15 grains (1 gram) daily of ferrous sulphate certainly provides an excess of ferrous ions (with or without achlorhydria). It is well known that most people with achlorhydria are not anemic, indicating that they obtain sufficient iron from their food. Therefore, the above considerations only apply in those instances where an additional factor predisposes to iron deficiency.

Steatorrhea is being emphasized currently in the etiology of iron deficiency anemia.[5] This situation may occur with various intestinal operations and abnormalities and also following partial gastrectomy. The stool fat content may be increased in the absence of an abnormal bowel habit or an abnormal appearance of the stool. The anemia may be refractory to oral therapy.

Diagnosis of Iron Deficiency Anemia. In making an etiological diagnosis of the anemic patient one would direct inquiry to the above mentioned causes of iron deficiency. The so-called "epithelial" signs and symptoms would be checked, i.e. smooth tongue, angular stomatitis, brittle or spoon shaped nails and dysphagia. It is hazardous to place much reliance in the color index derived from a single hemoglobin determination and red cell count. Even experienced workers cannot avoid a large error in these determinations when done by the usual techniques. The most reliance will be placed in the examination of the blood smear. More than slight hypochromia almost always indicates iron deficiency. This is also associated with considerable anisocytosis, poikilocytosis and decrease of the average cell size. The association of iron deficiency and pernicious anemia or related forms in one individual is quite unusual, but in such an instance the cell size might not be decreased. Mediterranean anemia (or the inherited trait without actual anemia) is an unusual cause of hypochromic red cells. Confirmation of the diagnosis can be obtained by serum iron determinations along with the iron binding capacity of the serum. but these are seldom necessary.

Saccharated Iron Oxide. Indications. Although oral iron therapy is very effective in the great majority of iron deficiency anemias, there are three groups of iron deficiency where recourse to this new intravenous agent should be considered:

1. Significant intolerance to oral iron therapy in the form of nausea, diarrhea or abdominal cramps unrelieved by symptomatic measures.

2. Instances where a rapid response of the anemia is very desirable, e.g. the last trimester of pregnancy.

3. Those few cases of true iron deficiency anemia refractory to oral iron therapy.

Claims have been made for saccharated iron oxide in the hypochromic anemia associated with theumatoid arthritis[6] and other anemias secondary to an infection. We and others are studing this interesting observation in greater detail, but any statement favoring saccharated iron as a treatment

5. Peeney, A. L. P. et al. Lancet 2: 387, 1950.

6. Sinclair, R. J. G. et al. Brit. M. J. 2: 1257, 1950.

for this group of anemias at this time would be premature. Furthermore, the agent is not free of dangers and its indiscriminate use is to be deplored.

Unique Properties and Disposal by the Body. Prior to 1947, parenteral iron therapy was impractical using the available agents in safe doses, and dangerous in the larger doses which could offer significant benefit. Therefore, saccharted iron oxide represents a definite advance in the therapy of iron deficiency states.

The special feature permitting its use in larger doses concerns the manner in which the iron is bound within the molecule. Toxic symptoms develop when the organism is exposed to circulating iron in an ionized form. The blood plasma, however, contains a specific protein ("the metal combining globulin") which binds iron specifically so that it is no longer ionized. This protein is always partly saturated with the iron used in the metabolic processes of the body. Only a small additional amount of readily ionizable iron can be injected into the blood stream before the protein is completely saturated. When this small amount is exceeded, toxic symptoms occur, usually in the form of flushing, paresthesias, weakness, tachycardia, nausea, abdominal or low back pain, precordial distress, fall in blood pressure or fever.

Saccharated iron oxide is unique because it undergoes very little dissociation in the blood stream and very little ionized iron is released at any one time. Therefore, in the recommended doses the metal combining globulin does not become saturated.[7] (See figure 2).

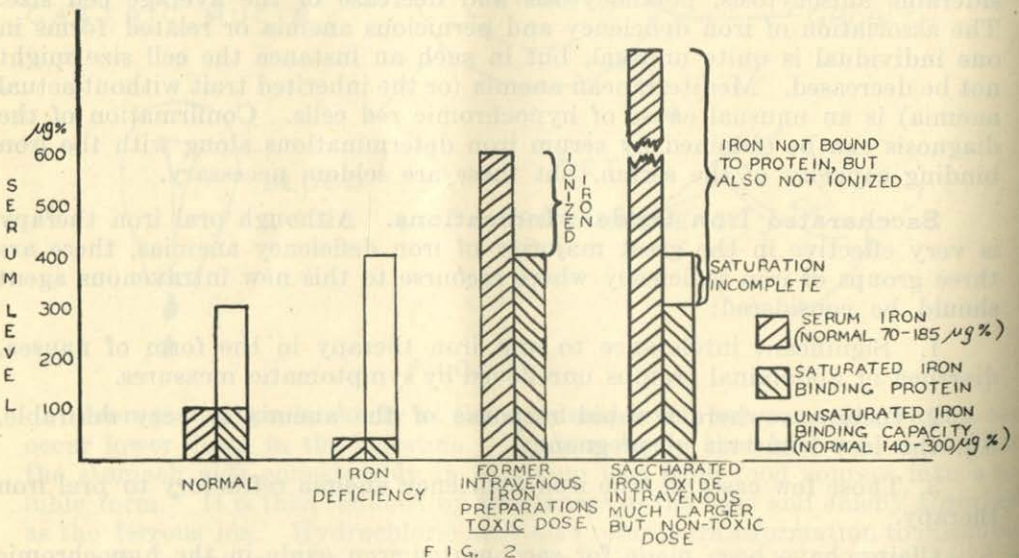

FIG. 2

The saccharated iron is removed from the circulation rapidly—presumably by the reticulo-endothelial cells—so that the iron concentration in the plasma 24 hours after injecting 200 mgms. of iron in this form has returned to the pre-injection value. Although the form of storage which occurs in the

7. Cameron, D. G. et al. Canad. M.A.J. 64:30, 1951.

reticulo-endothelial cells is not a normal one, the iron is subsequently released and can be used quantitatively for hemoglobin formation.

Dosage Calculation and Method of Administration. The most widely used proprietary preparations of saccharated iron oxide contain 20 mgms. of metallic iron per c.c. The dosage prescribed is calulated from the total body hemoglobin deficit. Therefore, one must consider the blood volume which can be roughly calculated as 37 c.c. per pound of body weight. In the formula below we also require the average hemoglobin concentration per 100 c.c. for normal persons which allows for some debate. For this purpose we may consider it as 16 gms. for the male and 14 gms. for the female. To transfer total body hemoglobin deficit to iron deficit we must know the fraction of hemoglobin represented by iron. This fraction is 0.0034. Finally, in replacement therapy we must consider the iron stores of the body because they are depleted in iron deficiency anemia. Their dimensions are not well defined, but 20 per cent of the total body iron is an approximate figure. The allowance which several authors have made for the iron stores is 50 per cent of the hemoglobin iron deficit, so that in our equation we multiply by 1.5

$$\text{Total dosage in grams of iron} = \frac{\text{Normal Hb\#} - \text{Initial Hb\#}}{100} \times \text{Blood volume} \times 0.0034 \times 1.5$$ [8]

Assuming a blood volume of 5000 c.c. for an adult of average size one can multiply $\frac{5000}{100}$ by 0.0034 by 1.5 and obtain the factor 0.255 The formula is then simplified to become:

Total dosage in grams of iron = (Normal Hb# – Initial Hb#) x 0.255.

The injections of saccharated iron are made intravenously using the solution supplied without dilution. Because of the irritating properties of the material the injections are made slowly (about 2 c.c. per minute) and care to avoid extravasation is essential. As a precaution against idiosyncracy the first injection is limited to 50 mgms. (2.5 c.c. of the solution). If uneventful, the second injection may be raised to 100 mgms. and subsequent injections (not oftener than one each day) given at the 200 mgm. level until the calculated requirement has been satisfied. A requirement in excess of 3 grams would be very unusual.

Therapeutic Response. Response with intravenous saccharated iron oxide has been uniformly good in iron deficiency anemias. The rate of hemoglobin formation is increased by comparison with oral therapy, so that with a good response the concentration is raised by 0.2 grams per 100 c.c. or more each day. Some cases have approached 0.3 grams per 100 c.c. per day which is much more than one expects on oral iron therapy. The following patient who was clearly unable to tolerate iron by mouth is presented as a typical therapeutic response.

E. D. (V.G.H. 101171) a 48-year old spinster was admitted to the surgical service of the Victoria General Hospital on Oct. 13, 1950 and subsequently

8. Brown, E. G. et al. J.A.M.A. 144: 1084, 1950.

Hemoglobin is given in grams per 100 c.c. Because of variations between instruments, the per cent "of normal" hemoglobin concentration should be transposed into gra

transferred to the medical service. She complained of diarrhea, vomiting, abdominal cramps, fatigue, and exertional dyspnea of several years duration. There had been no gross blood in the stool, but the stool was observed to have a fatty appearance at times. Menses ceased 10 years before admission following bilateral salpingo-oophorectomy. Oral iron therapy had previously precipitated marked diarrhea and abdominal distress. Examination revealed marked pallor, a rather smooth tongue, spoon shaped brittle nails, and slight enlargement of the liver and spleen. On rectal examinations a stricture was felt which on further examination proved to be non-malignant. The colon showed some dilation proximal to the stricture radiologically. Occult blood was repeatedly demonstrated in the stool. Gastric analysis showed up to 10 units of free acid. The hemoglobin was 5.5 grams per 100 c.c., packed cell volume 24% and red cell count 4.36 million. The blood smear showed typical hypochromia, microcytosis and marked variation in size and shape of the red cells.

Saccharated iron oxide injections were given daily between Nov. 1 and Nov. 10, total dosage being 1950 mgms. On several occasions there was momentary local pain during the injection due to venous spasm, but no other unpleasant effect. The hematological response is illustrated in figure 3.

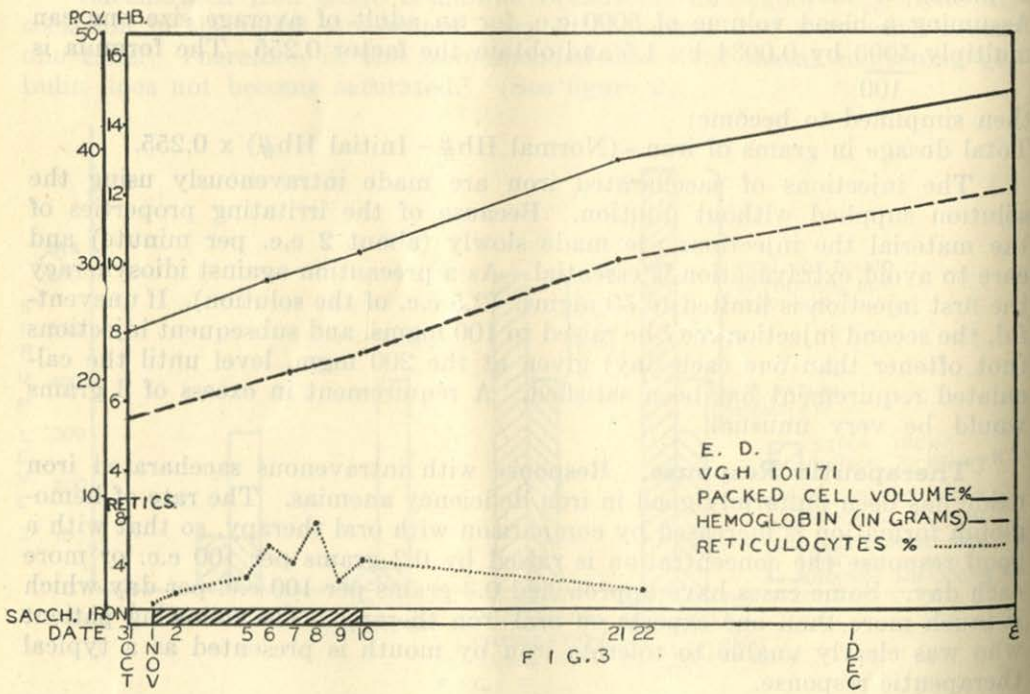

FIG. 3

The rate of hemoglobin formation from Nov. 1 to Nov. 21 was 0.22 gms. per day. On Nov. 28 a superficial phlebitis developed in one leg and subsequently in the opposite leg. Varicosities had been present for some time and the phlebitis is not regarded as being related to the intravenous iron therapy.

Colostomy and subsequent corrective surgery were delayed because of the phlebitis. It was interesting to observe the normal configuration of the new nail growth following correction of the anemia. The anemia in this patient may have been caused by impaired iron absorption in addition to the chronic blood loss.

Early Toxicity. Immediate toxic effects are unusual except for local pain due to venous spasm. The latter is not unfrequent during the performance of the injection and can be remedied by using a slower rate of infusion. This pain occurs less often when larger veins are available, but in any case has not posed much of a problem. A local phlebitis has been an unusual occurence.

Other toxic signs reported include hot flashes, weakness, tachycardia, nausea, pain in the lower back or extremities, precordial distress, faintness or syncope, fever, cough and subjective dyspnea.[9] These have usually been associated with doses in excess of 200 mgms. a day and simulate the toxic effects of circulating ionized iron. Very recently a case was reported which was considered to be an iron encephalopathy associated with convulsions, coma, hemiplegia and long standing psychic changes.[10] The saccharated iron oxide used in this instance was prepared locally instead of being obtained from a pharmaceutical house. Nevertheless, this report strengthens the premise that at this time intravenous iron is only indicated in situations where oral iron will not do the job.

Overdosage. Lastly, one must consider the possible undesirable effects of overdosage with this agent. An excess of iron in the body is the most outstanding feature in hemochromatosis and it seems reasonable that the impairment of hepatic, pancreatic and cardiac function noted in this disease are related to the abnormal iron storage. We now know that a similar disability can occur following receipt of very numerous blood transfusions, again being associated with very excessive iron storage. With saccharated iron oxide we have a readily administered agent which in sufficient dosage could swell the iron stores of the body to the same dimensions as we have observed in the above mentioned disabling diseases. It is, therefore, requisite that we use this substance as a therapeutic weapon with discretion, reserving it for those patients where it is clearly indicated and then only in the dosage required to correct the iron deficiency. Insidious damage might take its toll only after 10 or 20 years.

Summary

Some important points are reviewed in the etiology and diagnosis of iron deficiency anemia. A new therapeutic agent, saccharated iron oxide, is now available and for the first time permits adequate iron replacement therapy by the intravenous route. Tentative indications for this therapeutic agent

9. Horrigan, D. L. et al. J. Lab. Clin. Med. 36: 422, 1950.
10. Birch, C. A. et al. Brit. Med. J. 1: 62, 1951.

are stated. The properties which make it suitable for intravenous use are discussed. Methods of dosage calculation are presented. A typical case is included to illustrate the response which may be expected in iron deficiency anemia. Immediate toxic effects are unusual but iron encephalopathy has been observed. It is very possible, that over dosage may cause considerable disability several years later. It is, therefore, requisite that we use this substance as a therapeutic weapon with discretion, reserving it at this time for anemias clearly due to iron deficiency and then only when iron by mouth is unsuitable.

9. Horrigan, D. L. et al. J. Lab. Clin. Med. 36: 422, 1950.
10. Birch, C. A. et al. Brti. Med. J. 1: 62, 1951.

Medical Ophthalmology

J. LAND, M. D., Glace Bay, N. S.

REVIEWING the ophthalmic literature of the past ten years, I encountered numerous articles dealing with specific medical diseases and their relation to ophthalmology. Having made some notes on important points during this review, it occurred to me that possibly some general practitioners, who have less time than specialists to read, might pick up a point from this review—with this in mind the following is being written.

Probably the most common complaints that medical practitioners hear, are headaches and head pains of the most varied and endless description. The ophthalmologist is daily called upon to rule out or confirm, eye anomalies as the source of headache. Apart from acute glaucoma, iridocylitis, etc., all of which are easily recognized, there are many other anomalies which are not apparent upon the first examination. Latent hypermetropia, muscular imbalances and prodromal glaucoma are examples of such. Therefore, it is important that he make a thorough examination of refraction, muscle imbalances and visual field studies. Myopic's patients seldom complain of eyestrain. An accurate history is important, If a headache has no relation to close work, but independent of it, the symptom should be suspected to be of extra-ocular origin. One must never forget the possibility of headaches of dental origin. Headaches in children are seldom of ocular origin. When patients over 40 complain of headaches, one should think of prodromal glaucoma. This is one condition frequently overlooked. History of this is characteristic. At first, the headaches are rare, but after a period of several months, they tend to increase in frequency and severity.

During summer months they are often entirely absent; (due to the large amount of light and sun, keeping the pupil contracted) while during winter months, headaches are more frequent. These headaches often occur when in a theatre, or following excitement or worry.

Ophthalmology, as a medical science has not taken too kindly to the concept of psychosomatic medicine. This is probably because of the precision of diagnostic and therapeutic procedures, available to ophthalmology, which has encouraged too accurate particularization of diagnoses; but of late many authors are stressing these conditions such as: blepharospasm, convergence spasm, asthenopia, photophobia, hysterical amaurosis, amblopia, ciliary spasm and central angiospastic retinopathy.

It is natural that the greater part of an article under this heading shall be concerned with the pathological changes in the fundus oculi; and it is well to remind ourselves that the retina is a composite structure, entirely unique in the body from the point of view of clinical observation. One can imagine that general physicians and surgeons would consider it a great advantage if he could examine the lung or liver under living conditions under the microscope. In the eye, it is exactly this that is available to us. The fundus changes are so many and so varied, that we are often unable to clarify them.

Quoting Wilmer in his introduction to the Atlas Fundus Oculi he states: "The reproductions of the pathological lesions in the fundus are of unusual value, both to the ophthalmologist and to the clinican; for there is, perhaps,

1. Wagener, Cuisick and Craig: From trans. Am. Oph. Society—75th Annual Meeting pgs. (379-394).

no structure in the body that reflects so frequently and so definitely such a variety of diseases that affects the body generally. One may see in this small space, the head of the optic nerve, a meshwork of arteries and veins, as well as irregularities in the disposition of the pigment. Inflammatory reaction, secondary to infection affecting distant portions of the body, are prone to involve the highly vascularized tissues of the retina and choroid, while intoxications that may not produce a visible result elsewhere may cause changes that can be detected in the fundus.

Suppurative processes in distant parts of the body may be accompanied by profound changes in fundus oculi, as hemmorrhages and optic neuritis; embolism of central retinal artery in infective endocarditis. In syphilis, the eye is so frequently affected that we must regard the careful inspection of this organ as of equal importance with the examination of the skin, heart and nervous system. The Ophthalmoscope offers a remarkable opportunity to study the detailed structure of the blood vessels. Even in the early instances of essential hypertension, it is possible sometimes to see the compression and nicking of the veins when they are crossed by the turse arteries. This may occur without visible disease of arterial walls. In arteriosclerosis, we can follow, step by step, the progressive alterations that involve the vessels of the Fundus.

The general practitioners that wish to use the ophthalmoscope can dedelop their ability to examine the fundus with the pin hole without drops—but where more thorough and detailed examination is desired—in elderly people a few drops of 5% Ephedrine solution will give sufficient dilatation—following the examination a drop of 1% pilocarpine. In young people a few drops of 5% Euphthalmine can be used in adddition to the Ephedrine, or 1% Paredrene can be used.

Diabetic Retinitis

The occurrence of retinopathy in diabetes is well known. It occurs in 30 to 40% of diabetics; sometimes in association with generalized hypertension and arteriosclerosis, but also in patients with normal blood pressure and no changes in the larger blood vessels. The only factor which seems to influence the incidence of pure diabetic retinopathy is the duration of the disease. There has never been proved any relationship between blood sugar levels and retinopathy. The clinical picture of diabetic retinopathy consists in small round hemorrhages deep in the retina and sharply outlined, usually shiny white deposits. Decreased vision depends upon extent the macula is involved. The more important type is proliferating retinopathy, because it often leads to complete blindness due to hemorrhages into the vitreous, and organization of these hemorrhages into veils of connective tissue which remain in front of retina, and are penetrated by new vessels from vitreous itself.

Retinitis is the most discouraging vasuclar complication in diabetes. Perhaps this is so discouraging because incidence is so high among young people. Dr. Priscilla White has recently found that 85% of young persons with diabetes of over 20 years duration showed evidence of retinitis. There is an undeniable relationship between the retinitis and duration of the diabetes.

Vascular Changes

The basis for most of the vascular conditions in the eye and brain is a sclerosis of the blood vessels. The most common types are (a) arteriosclerotic changes of the arteries and (b) those resulting from hypertension and (c) arteriolar sclerosis.

Arteriosclerosis is primarily a fibrosis and hyaline degeneration of the muscular media. The intima may be affected secondarily with fatty deposits and endothelial cell proliferation of the vessel—leading to thrombosis and occlusion of vessels.

Essential Hypertension may be symptomless regarding vision—but later exudates and hemorrhages may affect vision, depending how extensive and what part of retina is involved.

Arteriosclerosis, 3rd type, affects retinal arteries of all sizes, but especially the smaller arterioles whose visibility and tortuosity are increased—the 3-2 ratio of veins over arteries is increased. One finds widening refiex, irregular tortuosity, arteriovenous constriction with tapering of veins, sheathing of vessels. Later hemorrhages and exudates and finally papilloedema.

The general physician who uses an ophthalmoscope should examine the arteriovenous crossings. When an artery and a vein cross in the retina, it is normal for artery to be in front of the vein. The vein is neither cut into, nor compressed by the artery, nor is its course diverted. When the arterial walls become thickened, not only is it impossible to see the vein where it is under the artery, but it is also invisible for a short distance on either side of it. The vein on both sides often comes to a point known as tapering. If the pressure continues the vein developes a sigmold twist which is pathognomonic of arteriosclerosis. As lumen in arteries is reduced. various attacks of fleeting blindness due to spasm occur. As the kidney disease progresses in severity, so does the blood pressure become increased. and there is an increase in frequency of retinitis. The ophthalmic changes are divided into exudates, oedema and hemorrhages, and one often sees the star figure due to hyaline exudate in outer malecular layer of retina.

A careful estimation of the grade of the sclerotic changes in the retinal arterioles is of value in determining the suita' ility of patients for surgical treatment in hypertension. Wagener et al' write that the proportion of good results drop from 40% without retinal arteriosclerosis to 9% in those with marked retinal sclerosis; and failures rise from 20% in patients without arteriosclerosis to 82% in those with marked retinal arteriocslerosis.

M. P. Salenes[2] gives his results pertaining to the fundus picture follow up. The author reaches the following conclusions:

1. During the first few weeks after the Smithwick operation in cases of Hypertension an increase in narrowing of the retinal arterioles, which accentuates the "oedematous" papilloretinal lesions, is observed.

2. Within a few months the angio spasm (localized constrictions and uniform narrowing) of the arterioles remaining. These, according to investigators who have kept the patients under observation for years after operation, also tend to disappear in time.

2. M. Puig Salenes GAC. Med. de Mexico. 77: 16 (Feb.), 1947.

3. The alterations in the retinal vessels in the patient after operation are independent of the effect produced by the operation on the arterial tension, and the general health of the patient.

4. It is supposed that similar alterations occur in other circulatory regions of the organism, especially the brain.

Considering hypertension further, it must be remembered that vast majority of hypertensive cases show no lesions of the fundi. It often takes years for suggestive changes to appear. At first, one may only see slight narrowing of the arteries, with or without indentation of the veins, where they are crossed by arteries. Later on, these signs are intensified; and still later the definite signs of retinopathy as hemorrhages, exudates, and increased involvement of the arteries. Towards the end, more hemorrhages with cotton wool patches, and evidence of neuro retinal oedema appear.

In a late article by Dr. Arthur Bedell,[3] the author asks "Just when in this progressive disease should the ophthalmologist recommend operation, or even sanction it?" He answers this by saying "If the surgeon concluded that the patient's condition warrants operation and the ophthalmologist has found no signs of retinopathy, there should be no delay." I would now like to quote verbatim, the summary of Dr. Bedell's article for those practitioners who have no access to this article.

"If the patient has no serious involvement of the heart, kidneys or cerebral vessels and no retinopathy, the ophthalmologist can sanction operation. If a retinopathy is present, with cotton wool patches, hemorrhages and exudates, approval may be given only when the results of physical and laboratory tests seem to warrant an operation, with the expectation that life will be lengthened.

If the patient has definite arteriosclerosis as indicated by white-walled vessels, if he has round, deep, red, granular retinal hemorrhages, if there is considerable retinal odema, as evidenced by a decrease in the visibility of the retina, or if there are intense, widespread oedema of the retina, and obscuration of the margins of the disk, with or without newly formed vessels on or about the disk, operation is contra-indicated. If he has had recent closure of a retinal artery, with the typical white oedema in the region supplied by the closed vessel, or sudden occlusion of the central vein, operation should not be attempted.

If there is a marked papilloedema with narrowing of the arteries, fullness of the veins, operation should be opposed, unless there are extenuating circumstances, at which time the surgeon assumes the responsibility and the patient understands the risk, for it is improbable that life will be materially prolonged by surgical intervention.

In addition to these sharply and clearly defined exceptions, there are many patients with little retinal oedema, but with large, greatly indented veins and uneven lumen of the vessels—such patients are poor risks.

Finally, if the patient with hypertension is to be made comfortable his anxieties must be relieved and his fear of imminent disability or death dispelled."

3. Bedell, A. J. Reduction of high blood pressure, Arch. Opth. Nov. 1948.

Hallum [4] has shown the great importance of the eyegrounds in the toaemias of pregnancy. He has shown that the eyegrounds are probably the best indication of the progress and severity of the toxaemia, and the most consistently reliable single guide in determining when pregnancy should be terminated.

Eye Changes in Disease of the Thyroid

The thyroid patient is often primarily an eye patient, and the frequency with which these patients are overlooked, warrants a constant watch for thyroid disease, whenever the anterior position of the eye is advanced. Patients who are nervous and highstrung, and cannot get used to their glasses, are often candidates for toxic goitre. Many patients with muscle instability and head pains are relieved only when their thyroid state is improved.

Graves listed three cardinal signs of hyperthyroidism—namely tremors, tachycardia and exophthalmos. Since then others have been added—as increased B.M.R., loss of weight, irritability etc. Widening of the palpebral fissure is observed more frequently than exphothalmos and usually precedes the latter.

Many patients with hypothyroidism suffer from severe ocular disturbances. They usually present themselves because of headaches and fatigue, and are not made comfortable because of glasses. The headache is sub-occipital in region, but ocular in origin. It is due to weakness of convergence accomodation. In an attempt to offset the eye muscle weakness, the patient uses his neck muscles, so that pain developes at the site of insertion of muscles along the superior and inferior nuchal lines of the skull, and the sternocleido-mastoidea at the tip of the mastoid. The abduction-adduction ratio may drop down as low as 1 to 1, although 1-3 is usual rate. Externally the outstanding feature in cases of hypothyroidism is oedema of the upper and lower lids, similar to that seen in long standing toxic goitre and in some cases of hypertension. The oedema is worse in the morning, but usually persists throughout the day.

N. Fenichel [5] collected a group of 20 patients with distressing headaches in which B.M.R. was below normal. Administration of thyroid extract obtained gratifying relief. Obly one exhibited moderate myxoedema with obvious clincial evidence of hypothyroidism. Headache usually occipital, but may be frontal. The cephalgia varied in intensity from dull, annoying distress to severe throbbing ache.

Most of the disturbances of the optic nerve are related to diseases of the retina, for the nerve is fed by same vessels, shares intimately with vascular pathology of this tissue. Anaemia and hyperaemia of nerve is accompanied by same condition of retina. Embolism and thrombosis of central retinal vessels, is more usually an affection of optic nerve that the retina in strictest sense of the term, for the usual place for the artery or vein to be occluded is at the optic disk where the lumen of the vessel is constricted by the tissues of the lamina cribrosa. Two important conditions of the nerve must be considered; namely hemorrhages into the intervaginal space and papilloedema.

4. Hallum, A. V. Change in retinal arterioles associated with the hypertension of pregnancy. Arch. Ophth. 37- 472 (April) 1947
5. N. Fenichel Annuals of internal med. Vol. 29. No. 3. Sept. 48. pgs. 456-61.

Since the subdural space is only a potential space, the more important communication is the sub-arachnoid space. Sub-arachnoid hemorrhages are divided into the traumatic and spontaneous. The traumatic are due to (a) rupture of basal aneurysm (b) spontaneous cerebral haemorrhage and (c) meningeal haemorrhages in diease such as diabetes, renal disease, etc. In the above, diagnosis is clinched by finding blood in cerebro-spinal fluid. Ocular signs in meningeal hemorrhage may be entirely lacking, but in a large number of cases, whether hemorrhage be traumatic or spontaneous, its extension into the sheaths leads to fairly constant clincial picture; the manifestations are papilloedema and retinal hemorrhages, to which are often added ocular palsies. These symptoms are usually bilateral.

Papilloedema is a passive oedema of nerve due to raised intra-ocular pressure without primary inflammatory changes and often without disturbance of function; and optic neuritis can be described as a swelling of the disc associated with inflammation and loss of function.

Brain tumors are most common cause of papilloedema—about 60% show it. In cerebellar tumors the papilloedema developes very rapidly. Brain abscesses only show papilloedema in about 20-25% of cases.

Regarding the clinical picture of papilloedema, one of the earliest signs is indistinctness and blurring of the disc. The physiological cup becomes filled up and the tissues veiled. Another early sign is absence of venous pulse at disc. Later the cup is filled in, and blurring of edges has passed around the cup. If oedema is increased, small linear hemorrhages frequently appear; if oedema still increases, the disc often bulges mushroom-like measuring as high as 6-8 diopters of swelling. Veins are usually engorged; vision not affected early, but later atrophy sets in. The earliest sign is enlargement of the blind spot. The value of Perimetry should not be underestimated. The fields of vision belong to the realm of the opthalmologist, but the G. P. can roughly test the fields by the confrontation test. Place the patient 1 meter away from you so that he faces you directly and his eyes are on a level with your eyes. Close your right eye. Direct the patient to cover his left eye and to fix your left eye with his right. Midway between you and the patient carry a 3mm. white-beaded pin from various parts of the extreme periphery of the filed (from above, below, nasally and temporally and then along meridians between these points) towards the centre. Direct the patient to indicate just where the object becomes visible. Compare the limits of the field of vision of the patient's right eye with those of your left eye (assuming your own fields are normal.)

The value of fields of vision is demonstrated by a case, male age 47, referred to me September 29 by Dr. A. Green of Glace Bay. History of headaches and some vomiting for 4 weeks. Eye grounds practically normal—except some blurring of the left disc above and nasally and the physiological disc quite filled up. The fields as plotted on perimeter showed a right homonymous hemianopia with sparring of maculae—which meant involvement of the visial pathway somewhere between the optic radiation at the level of the posterior end of the lateral ventricle and the occipital cortex. This case was sent to Dr. Cone of Montreal Neurological Institute the same day as feilds were done (due to certainity of diagnosis)—the operation for this occipital tumor was performed 36 hours after arrival in Montreal.

Optic neuritis clinically manifests itself essentially by loss of vision—typically a central loss of vision, irrespective of ophthalmoscopic appearances, and some pain in and behind the eye. Symptoms come on, suddenly, last 1-4 weeks and recovery of vision may be complete. Loss of vision usually precedes ophthalmoscopic changes, which usually are oedema and hemorrhages. Multiple sclerosis is commonest cause of optic neuritis. The typical cases of optic neutiris associated with disseminated sclerosis occur unilaterally in young women under 35, with acute onset of complete central scotama without ophthalmic change. The most typical field defect is a dense and absolute unilateral central scotoma, varying in shape and size. When the lesion affects the chiasm or tracts, hemianopic or quadrantic scotomta appear. The central scotoma is usually fleeting—but some defects of field and some degree of atrophy usually remain.

According to Gevan and Walsh [6] subdural hemorrhage is a common occurence in infants. In 37 cases only one above 2 years of age. The adult group consists of 54 patients. Nineteen of the 37 infants (one child of age 8) and 49 of 54 adults had a history of trauma. It is more difficult to assess value of trauma in infants.

Convulsions were present in 28 infants. This is in contrast to adult group, only 2 of whom had convulsions. In infants, a convulsion was frequently first evidence that anything was wrong. Drowsiness with retained irritability to stimulation was present in 20 infants to a pronounced degree. Only 2 of the infants were alert. In adults, drowsiness, with or without irritability, was an extremely common symptom. Twenty-six of the 28 infants with an open fontanelle exhibited building. Neurological signs were practically absent in both groups. The ocular signs of importance were pupillary dilatation, retinal hemorrhages or papilloedema. These signs are quite different in children and adults.

These writers feel the papilloedema although occuring in 20 to 50% of cases is a late development. In children pre-retinal hemorrhages are constant and absent in adults. Careful observation of pupils of great importance, particularly in adults. It is less significant in infants because in them lesion is usually bilateral. In adults one pupil may be dilated and fixed. This usually signifies the side of the hematoma. The explanation of this dilation has been by Reid and Cone; [7] as due to her herniation of the hypocampus or the medical aspect of the temporal lobe and midbrain through the tentorium. As a result of such herniation, there is a pressure on the homolateral 3rd nerve with development of an internal ophthalmoplegia, which may be associated with ptosis.

Ocular Changes in Head Injuries

One often finds cranial nerve involvement in head injuries—the 6th nerve being the largest intracranial nerve, and as it passes over the petrous portion of temporal bone it is the one most often involved; causing paralysis of external rectus.

6. Gevan-Walsh: Subdural Hematoma. Arch. Ophth. June 1947.
7. Reid, W. L. and Cone W. V.; The mechanism of fixed dilatation of the pupil, resulting from Ipsilateral cerebral compression.

Papilloedema, when found, strongly suggests the possibility of a subdural haematoma, or other expanding lesion. The occurence of an unilateral papilloedema or presence of more oedema in one eye than the other, may indicate the side on which the haematoma exists. A unilateral dilated pupil indicates same. Visual fields done often is important. From a therapeutic standpoint, examination of fundus and visual fields offer best imformation. Relief of increased intracranial pressure by a decompression operation seems to present complete loss of vision.

PHYSICIANS' ART SHOW

at

AMERICAN MEDICAL ASSOCIATION MEETING

ATLANTIC CITY

The American Physicians' Art Association will have an art exhibit, as usual, during the A.M.A. convention at Atlantic City, N. J. June 11 to 15, 1951, inclusive. Any physician in the United States, Canada and Hawaii desiring to participate in this show should communicate with the secretary for particulars.

J. Henry Helser & Co., Inc., Investment Managers with offices on the Pacific Coast, are the new sponsors of the American Physicians' Art Association and will award 200 trophies besides a special Helser Trophy—a large decorative cup depicting Yankee Ingenuity. This cup is to be awarded for art work done in any medium. Also the large Popularity Trophy will be awarded to the owner of the art piece receiving the most popular votes during the A.M.A. convention. Over 4000 members of the American Physicians Art Association will receive shortly, entry blanks, shipping labels and rules about this fourteenth art exhibition.

The Annual Art Banquet will be held Tuesday evening, June 12 at the Malborough-Blenheim Hotel, Atlantic City, N. J.

F. H. Redewill, M.D., Sec'ty.
American Physicians' Art Association
760 Market Street
San Francisco 2, California

Minutes of a Special Meeting of The Medical Society of Nova Scotia, 1951

A SPECIAL meeting of The Medical Society of Nova Scotia was held at the Lord Nelson Hotel, Halifax, N. S., Wednesday, April 4, 1951, at 2.00 p.m.

Doctor J. J. Carroll, the President, was in the chair. There was an excellent attendance of 102 members present. Doctor Carroll opened the meeting with a few introductory remarks regarding its purpose. The Secretary then read two motions which had been passed at a special Executive meeting held on March 20, 1951. One was that the present contract with the Department of Welfare of the Provincial Government be discontinued; the other that negotiations be opened with the Department of Welfare for a new and more satisfactory contract.

Doctor H. J. Devereux, the chairman of the Committee on Economics, then told of the work of that committee for the past few months. He said they had had four meetings, that they had interviewed Mr. Harold Connolly, Minister of Health, in an attempt to have the payment to the doctors increased from 75c to 85c per month, and that up to the date of the meeting all efforts had been unsuccessful. As a final effort a meeting had been arranged with the Premier and the Minister of Health for the morning of the day of the meeting. After a discussion lasting from ten o'clock until about noon the Premier promised that he would call the members of the Government together and if any change in their attitude was arrived at he would notify Doctor Devereux. Before coming to the meeting Doctor Devereux said that he had been notified that the Government had held a meeting that morning and they offered to pay 83c per month for old age pensioners and to continue to pay 75c per month for mother's allowance and also for the blind.

Doctor Devereux then moved and it was seconded by Doctor H. F. Sutherland that the new offer of the Government be accepted.

Then followed a very lively discussion most of which was out of order. Mr. D. C. Macneill, General Manager of Maritime Medical Care Incorporated, gave the meeting the information that there were 21,000 old age pensioners, 8,000 dependents on the mother's allowance act and 1,000 blind pensioners, making a total of over 30,000. The question of mileage was referred to, and it was brought out that the present rate paid by the Workmen's Compensation Board is 60c. Doctor W. H. Eagar asked for special information on many points dealing with the present arrangement. Doctor Carroll stated that Maritime Medical Care at present received about $18,000 for administering the scheme for The Medical Society. Doctor R. A. Moreash asked what percentage of the total cost was paid for mileage and in reply Mr. Macneill stated that the monthly accounts ran between $38,000 and $44,000, and the cost of mileage per month from $14,000 to $16,000. Doctor D. M. Cochrane thought that a new contract should be drawn up, and that the pensioner should pay something, preferably towards the mileage, although he thought

that the present mileage of one dollar was a bit too generous. Doctor C. H. Reardon suggested that all of the discussion was out of order. Doctor N. H. Gosse, stated that the cost of operating Maritime Medical Care was roughly 10%, and that it might be possible to reduce the charge made by Maritime Medical Care for administering the medical care scheme for pensioners. He said that Ontario was able to administer at a very low rate because they were using their Medical Society personnel to do it. He also said that Ontario was paying from 80c to 85c to the doctors on a basis of a 25c and a 50c mileage. He was in favour of accepting the new offer by the Government. Doctor J. W. Merritt felt that before doing anything we needed the help of a financial expert and he moved an amendment that a proper insurance actuary be obtained to advise the Economics Committee. This was seconded by Doctor G. R. Douglas. Doctor C. K. Fuller thought that the pensioner should be required to pay part of the charges. Doctor A.R. Morton then moved that the proper procedure was that the two motions of the Executive should be voted on, and this was seconded by Doctor C. H. Reardon. As a consequence Doctor Devereux withdrew his original motion and Doctor J. W. Merritt withdrew his amendment. The Secretary then read the notice of March 22nd which went out to all members giving the action of the Executive in which it was decided to discontinue the present contract with the Department of Welfare, and also in which the Committee on Economics was empowered to continue negotiations for a new contract. The Executive also went on record as having continued interest in the medical care of the welfare group. The President then called for a vote and both of these motions were adopted.

Doctor H. J. Devereux then moved that the new offer of the Government of 83c per month for old age pensioners and 75c per month for mother's allowance and also for blind pensioners be accepted, and this was seconded by Doctor H. F. Sutherland. In the discussion Doctor R. A. Morcash asked what percentage would be paid by Maritime Medical Care for February, and Mr. D. C. Macneill replied 40%. Doctor C. K. Fuller asked how much of the money went to the old age pensioners. Mr. D. C. Macneill answered that 65% of all the monies goes to the rural areas. Doctor J. W. Reid felt that since there was now no contract with the Government that first a new scale of fees for this work should be drawn up, and that nothing should be done until such a scale was drawn up. He moved as an amendment that the vote be deferred until such time as a new scale of fees was drawn up and a new contract written and clarified. The amendment was seconded by Doctor G. W. Turner. On the vote Doctor Reid's amendment was lost and Doctor H. J. Devereux's motion carried.

There was an then intermission of ten minutes.

Following the intermission Doctor Carroll announced that Mr. A. J. Sollows, City Supervisor of the North American Life Assurance Comany, was in the lobby and would be glad to see anyone who wished to sign up for the group insurance scheme.

Doctor J. H. Devereux first spoke about the old agreement. He suggested that the administration charges should be reduced, also that mileage should be reduced to 60c, and said that if these changes were accepted, and that if the office calls were reduced to $2.00 and the house visits to $3.00, with no

extras, that things would be more satisfactory and that there would be a more equitable distribution of funds. He finally moved that the Committee on Economics be empowered to negotiate with Maritime Medical Care for a charge of 4% for administration. This was seconded by Doctor M. G. Tompkins.

Doctor A. R. Morton moved an amendment to the effect that the Society appoint a committee, which with the assistance of Mr. D. C. Macneill of Maritime Medical Care would draw up a new agreement. He suggested that in this agreement the pensioner pay for the first call, and that the matters of mileage, office calls, administration be carefully looked into. Doctor Morton's motion was seconded by Doctor G. W. Turner. Doctor H. G. Grant spoke against the motion pointing out that it practically threw our Economics Committee overboard.

Doctor A. R. Morton explained that he did not mean a new committee.

Mr. D. C. Macneill stated that he knew Maritime Medical Care could not administer our funds for 4%, but perhaps they might for 5%.

Doctor D. M. Cochrane suggested a fee of 5% instead of 4%.

Doctor J. W. Reid moved an amendment that a new committee be appointed to draw up an agreement with the Government which would clarify the service which the doctors would give to the people.

Doctor P. R. Little thought the whole matter should be dropped and expressed the opinion that both amendments were out of order.

The motion to the effect that the Committee on Economics negotiate with Maritime Medical Care to administer our affairs for 5% was passed.

Doctor H. J. Devereux then moved and it was seconded by Doctor H. F. Sutherland that mileage be reduced from $1.00 to 60c.

Doctor J. C. Wickwire stated that the Lunenburg-Queens Medical Society had voted that the mileage fee should be one dollar.

Doctor R. A. Moreash spoke against reducing the mileage from one dollar to sixty cents.

Doctor J. W. Reid suggested that instead of reducing the mileage it be prorated to a lower figure.

Doctor D. M. Cochrane thought that the mileage should vary according to the district, and that there were in the Province from thirty to forty doctors who should receive special consideration.

Doctor P. E. Belliveau felt that the mileage should be the same as that presently paid by the Workmen's Compensation Board. The motion that mileage be reduced from one dollar to sixty cents was defeated.

Doctor J. W. Reid moved that a committee be appointed to clarify the medical services to be rendered to the pensioners and also to draw up a schedule of fees for contract purposes.

Doctor N. H. Gosse seconded Doctor Reid's motion. He felt that that committee should be given instruction to provide machinery to deal with men who were not playing the game. He felt that there had been a lot af abuse.

Doctor D. M. Cochrane thought there should be a thorough house cleaning; that the profession had a big responsibility and that every doctor should be circularized and told what his responsibilities were.

Doctor H. G. Grant spoke against Doctor Reid's motion stating that he thought the committee to carry on was the Committee on Economics.

Doctor J. W. Reid then moved that the present Committee on Economics be instructed to draw up a new contract, and this was seconded by Doctor N. H. Gosse.

Doctor D. M. Cochrane stated that in British Columbia under the new scheme patients are now required to pay for the first few days in hospital, and that something like that should be brought into our new contract.

Doctor H. E. Kelly felt that the country doctors should get one dollar for mileage, and that sxty cents was plenty for city doctors. Alse he felt that there were many calls made that were not necessary.

The decussion dealt with the new agreement and Doctor H. J. Devereux expressed the opinion that if the Committee on Economics had authority to draw up the new contract they also should have authority to sign it.

Doctor J. W. Reid moved that the Committee on Economics should report back to the Executive and this was seconded by Doctor J. P. McGrath.

Doctor H. G. Grant informed the meeting that the present contract was in force until next July, and that if this motion were passed there would have to be a special meeting of the Executive to receive the report.

Doctor D. M. MacRae moved an amendment that the Economics Committee report back before the present contract expires, so that the Executive could draw up a new contract.

Doctor H. G. Grant did not feel there should be very much detail in the contract.

Doctor H. F. Sutherland moved an amendment that a committee of two be appointed this afternoon by the meeting to draw up the new contract, and that the committee be made up of Doctor J. W. Reid and Doctor C. H. Reardon. He stated that the 83c would be retro-active to the first of March.

Doctor J. P. McGrath felt the Society should ratify the agreement and that the Economics Committee should carry on.

Doctor J. A. Langille asked if the maximum charge for office consultations were $2.00 and for home visits $3.00 and whether Doctor Devereux intended to put that in the form of a motion.

Doctor N. H. Gosse moved an amendment that the Economics Committee be empowered to draw up this contract and sign for the Society and that they get legal advice on the terms of the contract.

Doctor J. W. Reid suggested that Doctor Gosse's amendment should state that the new contract be brought before the Society as a whole before it is dealt with.

Doctor H. J. Devereux, the Chairman of the Committee on Economics, said he would have nothing to do with the signing of the contract.

Doctor N. H. Gosse then changed the wording of his amendment to state that the Committee on Economics be empowered to draw up a new contract and get legal advice and when the contract was drawn up it should be brought back to be signed by the President and the Secretary.

Doctor W. W. Bennett thought it should be sent to all the Branch Societies before it was signed.

Doctor H. D. O'Brien said that we had accepted the new contract and that we had the authority to sign a new contract, and could see no reason for prolonging the discussion.

Doctor Gosse's amendment was carried.

Doctor M. A. Macaulay made a motion that $2.00 should be the fee for office visits and $3.00 for home visits. This was seconded by Doctor H. J. Devereux and carried.

Doctor D. M. Cochrane moved that a rate of 75c for mileage be accepted, and this was seconded by Doctor J. C. Wickwire.

Doctor R. A. Moreash moved that the fee for mileage be one dollar. This was seconded by Doctor A. G. MacLeod.

Doctor J. W. Reid asked if Doctor D. M. Cochrane would change his motion to read that the mileage be pro-rated to seventy-five cents.

Doctor A. A. Macdonald moved that the mileage fee for welfare cases be pro-rated seventy-five cents on the dollar. This was seconded by Doctor D. M. Cochrane. Carried.

It was moved that the meeting adjourn at 6.50 p.m.

Post-Graduate Course in Surgery

The Victoria General Hospital

Halifax, Nova Scotia

A Post-Graduate course in Surgery will be given by the Victoria General Hospital, Halifax, Nova Scotia. It is designed to assist those writing Fellowship or Certification examinations, and for surgeons generally desiring further Post-Graduate Training.

The didactic lectures will be held from October first to October twenty-seventh inclusive.

The course will include lectures in Anatomy, Pathology, Basic Sciences, Clinical Medicine and General Surgery.

The fee for the course is One Hundred Dollars.

Applications should be addressed to The Chairman, Post-Graduate Course Committee, The Victoria General Hospital, Halifax, Nova Scotia.

RESIDENT DOCTOR WANTED

The inhabitants of Spryfield, which is five or six miles from Halifax, are desirious of having a physician locate there. It is a populous district and there should be a good practice for an active physician. Further information may be obtained from Mr. J. W. Stevens, Manager, Balcom-Chittick Limited, Spryfield, N. S.

Society Meetings

A meeting of The Nova Scotia Medical Society of Opthalmology and Otolaryngology was held on Wednesday, November 15th, 1950 at the Victoria General Hospital, Halifax, N. S.

The programme opened with clinical presentations by the Halifax members of the Society at the out-patient department of the Victoria General Hospital.

This was followed by a business meeting, the question came up of the advisability of changing the time of our annual meeting and after some discussion it was moved by Dr. McKean and seconded by Dr. Davidson, that no change be made at the present time.

An application for membership was received from Dr. C. F. Keays, Halifax, on recommendation of the Executive the application was passed by the meeting.

Dr. Pullins of Moncton speaking on behalf of the New Brunswick Specilaists Society invited the members of our Society to consider a joint meeting in Moncton, some time in May, 1951.

It was regularly moved by Dr. McGrath, seconded by Dr. Fuller that our members attend this meeting to be held the early part of May, 1951.

The Vote was carried unanimously.

The meeting then adjourned for Luncheon at the Lord Nelson Hotel.

The afternoon session consisting of presentation of papers and films was held in the auditorium of the Victoria General Hospital, Halifax, N. S.

Dr. H. R. McKean presented a paper on "Glaucoma—A Review of the years literature."

The paper showed that Dr. McKean had spent much time in reading and preparation to present such a full discussion of the subject. There was considerable discussion by the following, Drs. Holland, Stoddard, Doull, Jr. Pullins, D. M. MacRae, Kirkpatrick and Davidson. Dr. Schwartz suggested that Dr. McKean forward the paper to the Bulletin for publication so that it would be available to all the members.

Dr. H. F. Sutherland then presented a paper—"Some interesting lesions of the tongue."

Discussions of the paper and comments were made by Drs. Kirkpatrtick, McGrath and Schwartz.

Dr. J. P. McGrath presented a paper—"Impressions of the 1950 Chicago meeting of the American Academy of Ophthalmology and Otolaryngology."

Dr. D. M. MacRae and Dr. E. I. Glenister added comments of their impression of some of the papers and the meeting at Chicago.

Two technicolor films through the curtesy of the Imperial Optical Co., Ltd. were shown: "Cateract Techique" and "Keratoplasty" by Dr. Raymon Castroviejo, New York.

The meeting adjourned on the motion of Dr. McGrath, seconded by Dr. Davidson.

E. I. Glenister, M.D.
Secretary-Treasurer

Obituary

Doctor Albert Meldrum Arbuckle died at his home in Pictou on April 22nd. He underwent an operation at the Royal Victoria Hospital, Montreal, in February, and since his return home his condition gradually worsened.

He was born in Pictou, July 10, 1896, the younger son of Mr. and Mrs. I. T. Arbuckle. He was educated at Pictou Academy, Acadia University, from which he obtained his B.A., and graduated in medicine from McGill University in 1928. Before commencing his medical studies he taught at Acadia Villa Seminary, Hortonville, and Rothesay Collegiate, Rothesay, N. B.

Following graduation from McGill he served as house surgeon at the Strong Memorial Hospital and the Rochester Municipal Hospital, Rochester, N. Y,. and later was resident physician at the New York Polyclinic Hospital and Medical School. From 1931 to 1935 he was resident physician at the Waldorf-Astoria Hotel, New York City, and director of the Waldorf-Astoria Industrial Clinic.

In 1936 he served as resident roentgenologist at the City Hospital, Quincy, Mass., then began specializing in chest work. He was assistant physician at the Rhode Island State Hospital for Tuberculosis, Wallum Lake, R. I., in 1937, then for three years assistant medical director of the tuberculosis division of the Belmont Hospital, Worcester, Mass. A few years ago he was made a Fellow of the American College of Chest Physicians.

When Foundation Maritime Limited established an industrial clinic at the Pictou Shipyards during the war years, Doctor Arbuckle was appointed director, and continued in that position until the closing of the Yard.

Since his return to Pictou, Doctor Arbuckle took an active part in community affairs, giving willingly of his time and effort in any movement for the betterment of the town. He was a member of the Board of Trade, an active worker on several committees of the Pictou-North Colchester Exhibition, and Past President of the Men's Service Club of the Community Centre, and a past president of the Pictou Horticultural Society in whose work he took a deep interest. He was also a member of the Lobster Carnival Association, and of the New Caledonia Curling Club, a member of The Medical Society of Nova Scotia, and the Canadian Medical Association, the National Tuberculosis Association, and the American Association of Industrial Physicians and Surgeons. He was a member of the First Presbyterian Church and of New Caledonia Lodge, No. 11, A. F. and A. M. He was unmarried. He is survived by his parents, and a nephew, Stewart Arbuckle, of Pctou. His only brother, J. Stewart Arbuckle, died in Montreal in 1938.

The funeral, under Masonic auspices, was held on Tuesday, the 24th, a short service at his home followed by a service at the First Presbyterian Church. Interment was at Haliburton Cemetery.