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Criteria of Medical Necessity for In-Patient Services in Respect of Long Stay Patients

The following has been released by the Nova Scotia Hospital Insurance Commission to Chairmen of Hospital Boards and Chairmen of the Medical Sub-committees of the Hospital Standards Committee.

The Regulations under the Hospital Insurance Act, Section 4, states "A resident is entitled to insured in-patient services for the period of time following admission during which such in-patient services are medically necessary."

The practical difficulty arises in deciding when in-patient services are medically required. This particularly applies to long-stay cases.

Primarily the authority to make the decision as to medical necessity rests with the physician in charge of the case. However, the Hospital Standards Committee has the duty, under Section 19(A) (iv) of the Regulations, to "—advise forthwith the hospital board if, in the opinion of the Committee, the standard of any service is unsatisfactory or the utilization of any service provided by the hospital is not reasonable and proper."

The Provincial Standards Committee has a duty "—to ensure—reasonable and proper utilization of services in all hospitals of the Province." (Section 19(C) (i). . Moreover, "if, in the opinion of the Commission, any of the services provided to a patient are or were not medically necessary, the patient shall not be entitled to such services as insured services." (Section 6(1). To assist the Commission in arriving at a just decision, the Regulations, Section 6(2), provide "when, in the opinion of the Commission a doubt exists concerning the medical necessity for in-patient or out-patient services in any case, the Commission may appoint and empower a medical review board to report on the case."

While the above provisions of the Regulations, which were reviewed by advisory committees of the Hospital Association and The Medical Society, prior to Government approval, would appear to be reasonable and proper, there would appear to be still some difficulty in defining medical necessity particularly in the borderline long-stay cases.

Before indicating a criterion of medical necessity for in-patient services, it must be stated definitely, in a negative way, that **firstly, nursing home cases or custodial welfare cases cannot qualify and secondly, that economic or social factors cannot be taken into consideration.** The second statement may seem rather hard, but it was on these terms that Nova Scotia, like all other Provinces, had to sign the Federal-Provincial Agreement. The Plan is a hospital plan; there are other Federal-Provincial arrangements dealing with the welfare case.

As a general criterion of medical necessity for in-patient services in respect of long-stay patients, it may be said that **a patient receiving active hospital care is a patient who has a disease, injury or impairment requiring diagnostic and therapeutic Hospital Services (as nursing, pharmacy, laboratory, X-ray etc.) at about the level provided to the average hospital patient.**

G. GRAHAM SIMMS, M.D., D.P.H.,
Vice Chairman.

June 22/59

Hypertensive Encephalopathy*

S. J. Shane, M.D.**

Our understanding of cerebrovascular disease is complicated by the fact that there are almost as many terminologies for different cerebrovascular syndromes as there are workers describing and investigating these syndromes. The following are a few of the titles that have served to confuse the issue for some years: (1) Cerebrovascular accidents, (2) cerebrovascular "episodes", (3) strokes, (4) "little strokes", (5) "slow strokes", (6) "recurrent strokes", (7) cerebrovascular disease, (8) cerebral vasospasm or angiospasm, (9) cerebral infarction, (10) cerebral thrombosis, (11) cerebral insufficiency, (12) intermittent insufficiency of the cerebral arterial circulation; and many others.

It is obvious that such a multiplicity of terms is undersirable for two reasons: (a) because it indicates our lack of detailed knowledge of the subject, (b) because different terms may mean the same thing or the same term may mean different things to different observers.

Fortunately it is not our task here to bring order out of this terminological chaos. Our only function at this time is to discuss one specific, circumscribed cerebrovascular entity, namely hypertensive encephalopathy. Therefore, I should like to discuss this particular condition under the following headings:

1. What is hypertensive encephalopathy?
2. What is **not** hypertensive encephalopathy?
3. What is the mechanism of production of hypertensive encephalopathy?
4. How is the diagnosis made? (a) positively
   (b) negatively or by exclusion
5. Is there more than one type of hypertensive encephalopathy? Yes.
   (a) With diffuse signs
   (b) With focal signs
6. Treatment:
   (1) Antihypertensive agents:
      (a) Sodium nitroprusside—a direct vasodilator.
      (b) Ganglionic blockading agents—e.g., hexamethonium compounds.
      (c) Combined vasodilator and ganglionic blockader—e.g., trimethaphan camphorsulfonate (Arfonad—Roche).
      (d) Less potent antihypertensive agents—e.g., parenteral reserpine.
   (2) Miscellaneous pharmacotherapy—e.g., barbiturates, tranquillizers, preparations to relieve cerebral oedema.
   (3) Treatment of intrinsic causes of the hypertensive crisis—e.g., resection of Goldblatt kidney, resection of pheochromocytoma, resection of carcinoid.
7. Case Reports:
   (1) Case from literature.
   (2) Case from Victoria General Hospital

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* Read at the Week in Medicine, Dalhousie University, Friday, March 6, 1959.
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WHAT IS HYPERTENSIVE ENCEPHALOPATHY?

The term hypertensive encephalopathy is applied to a situation in which the patient has gross hypertension and an acute episode characterized by severe headache, nausea, vomiting, often confusion, convulsions, stupor and frequently coma. One must stress that attacks of hypertensive encephalopathy are directly attributable to a rapid increase of blood pressure as such and **not** to a remote sequel of cerebral vascular disease, if such exists.

One may divide hypertensive encephalopathy into two groups, diffuse and focal. The diffuse form should be restricted to the definition given above. The focal form is a separate and distinct entity, perhaps analogous to post-traumatic epilepsy, in which focal vasospasm seems to be a precipitating factor. These seizures occur in patients with severely elevated blood pressure, but the attacks do not appear to be preceded by rising trends in pressure.

WHAT IS NOT HYPERTENSIVE ENCEPHALOPATHY?

Although the derivation of the term indicates that it may be applied to all forms of cerebrovascular disease associated with hypertension, one must stress that, in practice, and for purposes of clarity, the term "hypertensive encephalopathy" should be restricted to the two situations described above, and perhaps entirely to the first of these. Therefore, the term hypertensive encephalopathy cannot be applied to cerebral haemorrhage, thrombosis, embolism, small or large, slow or rapid "strokes", or any cerebral episode that does not fit the description given above. In other words, the term hypertensive encephalopathy must be interpreted in a highly restricted sense.

WHAT IS THE MECHANISM OF PRODUCTION OF HYPERTENSIVE ENCEPHALOPATHY?

Several theories have been devised to explain the somewhat mysterious sequence of events; and the present-day concept is a synthesis of several of these theories. For example, one theory suggested that it was due to cerebral oedema. Another concept was that it was due to rising peripheral blood pressure, the cerebral arterioles being unable to withstand this pressure. A third suggestion was that this syndrome was due to widespread damage to the cerebral arterioles. A synthesis of these concepts results in a sequence of events somewhat as follows:

(1) From some reason, usually **not** concerned with the brain, there is a rapidly progressive rise in arterial blood pressure. The stimulus to this increase in blood pressure may arise, for example, in the kidney, and may actually have occurred for the purpose of supplying a higher perfusion pressure to that organ. In effect, to use an anthropomorphic concept, the stimulus to rapid, progressive hypertension is a "selfish" one in terms of the "culprit" organ, in this case the kidney. This is the first step.

(2) The second step is intense vasonconstriction throughout the body in order to produce and maintain the "required" hypertension.

(3) Such vasoconstriction may not be harmful to most organs, but the structure of the cerebral capillaries is such that prolonged vasoconstriction of arterioles damages them and permits leakage of fluid and protein into the perivascular spaces, as well as haemorrhage. In other words, cerebral oedema

and pin-point haemorrhages would be the typical morphologic findings in hypertensive encephalopathy of the diffuse type, and it is these morphologic changes that result in headache, nausea, vomiting, convulsions, stupor and coma. It should also be emphasized that cerebral oedema or "wet brain" does not always occur in hypertensive encephalopathy. However, it is generally agreed that persistent widespread cerebral vasospasm may lead to nerve-cell death with secondary hemiplegia or other complications. Botterell et al have also pointed out that vasospasm may result in hypoxia of the brain sufficient to produce serious, if not fatal cerebral oedema; and this is corroborated by other authors.

To summarize, disturbed intra-arterial pressure in an organ such as the kidney elicits a generalized arterial spasm in other regions of the body. This raises the systemic blood pressure and restores the effective arterial pressure to a more acceptable level in, let us say, the kidney. The generalized hypertension then endangers the capillary body of the brain, and the cerebral arterioles respond by further vasoconstriction to protect the capillaries. The hypervasoconstriction damages the walls of the cerebral capillaries peripheral to the arterioles, leading to transudation, oedema and haemorrhage. The damage may be further compounded by cerebral ischaemia due to the vasoconstriction, resulting in cell death and further oedema and haemorrhage. We have, therefore, a synthesis of theories to explain the sequence of events in diffuse hypertensive encephalopathy.

HOW IS THE DIAGNOSIS MADE?

The symptom complex in diffuse hypertensive encephalopathy has already been described. In addition, if cerebral oedema occurs, and has been present for some time, it will be mirrored by an increase in the spinal fluid pressure. Other ancillary features may be of value in arriving at this diagnosis. A history of increasing hypertension; evidence of chronic renal disease such as glomerulonephritis or pyelonephritis, which might result in acute hypertension; evidence of the presence of unilateral renal ischaemia or toxaemia of pregnancy, etc., etc., are all helpful. On the negative side, diagnosis is aided by the exclusion of cerebral haemorrhage, embolism, thrombosis, and other causes of episodic or sustained cerebral "insufficiency". In particular, it is crucial to note that this condition is always associated with **hyper**tension, and can, therefore, never be diagnosed in situations where cerebral symptoms are the result of insufficient perfusion of the brain caused by rapidly developing **hypo**tension. Another point in diagnosis is that we usually see hypertensive encephalopathy in the **young** hypertensive patient. In the older patient, in whom the cerebral circulation has already been damaged by vascular disease, pure hypertensive encephalopathy is less common.

IS THERE MORE THAN ONE FORM OF HYPERTENSIVE ENCEPHALOPATHY?

That this is so has already been intimated, although, up to this point, we have been discussing the diffuse form, which is usually quite simple to diagnose. However, there is an entity which is usually placed under this heading, although perhaps it should not be, described as "focal encephalopathy." In this condition, there is a focus of cerebral irritability the cause of which is unknown. It may be focal oedema, bleeding or even occlusion. The

seizures are not due to occlusion as such, since they have a tendency to assume a repetitive pattern and they are neither induced nor intensified by sudden lowering of arterial pressure. On the contrary, they occur in patients with severely **elevated** blood pressure, but there is not necessarily a rising trend in pressure, nor does lowering of the pressure cause the attacks to subside more readily than does sedation or bed rest. It is important that this condition be differentiated from any one of the forms of organic cerebral vascular occlusion in which the symptoms are intensified by sudden large decreases of arterial pressure, and from the effects of carotid occlusion.

TREATMENT

It goes without saying that, since the stimulus to this form of encephalopathy is generalized hypertension, the treatment par excellence is to lower the peripheral blood pressure. This will, according to present day concepts, result in release of the reactive cerebral vasospasm and put an end to the resulting transudation, cerebral oedema and haemorrhage. For this purpose, one can use several methods of lowering the arterial blood pressure:

(a) Sodium nitroprusside: This is a potent direct vasodilator, which is given intravenously in a way similar to that used for vasopressor agents such as l-norepinephrine or metaraminol. That is, a small quantity of sodium nitroprusside is added to an intravenous drip and the speed of the drip is varied in accordance with the fall in arterial pressure that one wishes to produce—a form of human "titration." Usually the amount of nitroprusside given per minute is extremely small, is measured in micrograms, and varies from 50 to to 400 mcg. per minute; and the blood pressure is maintained at a normal level, e.g., 140/80, for 3 to 4 hours or until the symptoms have disappeared. When the drip is discontinued the blood pressure does **not** usually rise and the symptoms do **not** usually return, or there may be a gradual rise in blood pressure without recurrence of symptoms.

(b) Ganglionic Blocking Agents: The most common of these are hexamethonium salts which are given in spaced intravenous doses of 100 mg. or more.

(c) Combined Vasodilator and Ganglionic Blockader, e.g., trimethaphan camphorsulfonate (Arfonad-Roche): This, like sodium nitroprusside, is given by intravenous infusion. Usually one ampul of trimethaphan camphorsulfonate containing 10 c.c. in a concentration of 50 mg. per c.c. is diluted to 500 c.c. and started at a rate of 60 drops (3-4 mg.) per minute. Frequent blood pressure determinations guide one in determining whether the rate should be decreased to as low as 4 drops per minute, or increased to as high as 100 drops per minute. Note that the dilution is 500 mg. in 500 c.c or 1 mg. per c.c. The blood pressure is maintained at a normal level, as with the nitroprusside, for 3 or 4 hours or more, and usually does not rise to its pre-infusion level.

(d) Other antihypertensive agents, such as intravenous veratrum preparations, pentolinium, etc., have been used but, in conditions where speed is not essential, a very simple and safe procedure is to give reserpine in doses of 1 mg. intramuscularly every 4, 6, or 8 hours, depending on the hypotensive response or the degree of sedation produced.

The above recommendations apply to the diffuse form of hypertensive encephalopathy. In the focal types, there is always some doubt as to whether or not the clinical picture is the result of an organic cerebral occlusion, in which case antihypertensive therapy would be contra-indicated. Furthermore, it has not been demonstrated that antihypertensive therapy in these focal attacks produces any better result than do rest and sedation alone. In these focal episodes, therefore, simple rest with mild barbiturate therapy or the use of one of the ataraxics such as meprobamate, may be all that is required.

Two points must be stressed here, viz.,—(1) If the encephalopathic attack is allowed to persist for a long period of time without antihypertensive therapy, permanent cerebral damage may result or death may ensue. One should, therefore, proceed boldly with this therapy as soon as the diagnosis is established. (2) Despite the cautionary remarks regarding the use of drastic antihypertensives in focal encephalopathy, no harm has been known to result from such therapy where it has been established that these attacks are not due to organic occlusion.

(e) The treatment described above is emergency therapy, and is indicated irrespective of the cause of the hypertensive crisis, e.g. essential or malignant hypertension, chronic cardiovascular-renal disease or some surgically correctible form of hypertension. Naturally, as soon as emergency therapy has been carried out, the patient should be investigated to determine whether the underlying cause is unilateral renal ischaemia, pheochromocytoma or carcinoid tumour etc., and, if found, these should receive appropriate treatment.

CASE REPORTS

In order to clarify the situation as to diagnosis and treatment, I should like to cite briefly two cases, one from the literature, and one that of a recent Victoria General Hospital patient.

(a) Case 1: C. McK., acute generalized encephalopathy. A man of 56 had essential hypertension of about 10 years' duration for which lumbodorsal sympathectomy had been done two years before. His home pressure average was 175/118 during the week ending Sept. 4th, 1956. His pressure increased on Sept. 5th and 6th and on the morning of the 7th he complained of headache, nausea and vomiting which had begun the night before. His blood pressure was 220/140. He was dull, restless and acutely ill. **Sodium nitroprusside (60-130 micrograms per minute) was given intravenously,** maintaining his pressure at about 140/10 for four hours. His symptoms were promptly and fully relieved and did not recur when the infusion was stopped, although his pressure gradually rose to 218/140.

(b) Case 2: L.A., age 14. No prior illnesses of consequence were noted until January 5, 1959. At that time severe right lumbar pain occurred lasting 4 days. There were also pyuria and fever, which improved on penicillin and streptomycin therapy. She was then well until February 15, when severe frontal headache, nausea and vomiting occurred, followed by convulsions. The blood pressure ranged from 150/110 to 204/142. On February 20, she was admitted to the Victoria General Hospital with severe frnotal headache. The fundi were normal and the blood pressure was 190/150. Twelve hours

later there were 2 successive convulsions. Blood pressure remained at levels of 160-190/120-130, and severe headache persisted. She was then given 40 gm. urea intravenously* in 200 c.c. glucose in water. When seen again 8 hours later there was no headache and the patient was mentally clear. This was obviously hypertensive encephalopathy with cerebral oedema as will be seen below.

INVESTIGATION—Urine: 1000 mgs.% albumin; few epithelial cells; no R.B.C. or W.B.C.; S.G. 1.024. B.U.N. 10 gm.%. C.S.F. normal with pressure **335 mm. H_2O.** The test for urinary catecholamines was negative. Phentolamine test while under sedation was positive, but this result was not reproducible on a second attempt. Retrograde pyelogram revealed a hypoplastic kidney on the right with compensatory hypertrophy of the left kidney. The right kidney excreted no urine, the left 1.8 c.c./min. An aortogram revealed no blood supply to the right kidney. In the left kidney, the blood supply to the lower pole was poorly visualized. Subsequent exploration, performed by Dr. C. L. Gosse, revealed almost complete obstruction of the right renal artery, and there was an immediate fall in blood pressure as soon as the renal vein was clamped for nephrectomy. It therefore appears reasonable to suppose that some humoral pressor agent was entering the general circulation from the ischaemic kidney and causing hypertension. During the first two postoperative weeks there has been no recurrence of hypertension or headache.

SUMMARY

1. An attempt has been made to clarify the concept of "hypertensive encephalopathy", particularly with reference to mechanism of causation.

2. It is emphasized that hypertensive encephalopathy should be, by definition, a sharply restricted entity.

3. On the basis of our present knowledge of etiology, system of emergency and specific therapy is outlined.

4. Illustrative case reports are presented.

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* Intravenous urea was provided for experimental use by Dr. Peter Nash, Abbott Laboratories Limited.

The Hemolytic Crises

R. D. Drysdale, M.D., F.R.C.P.(C)

Hemolytic crisis is characterized by fever, pallor, mild jaundice, palpitation, nausea and vomiting and acute abdominal pain. As a clinical entity it occurs in (1) Congenital Hemolytic Jaundice (Hereditary Spherocytosis), (2) Sickle Cell Anemia and certain other hemoglobinopathies, (3) Paroxysmal Nocturnal Hemoglobinuria and (4) Acquired Hemolytic Anemia. Hemolytic transfusion reaction will not be included as an hemolytic crisis in this paper.

RECOGNITION OF HEMOLYTIC CRISIS

In a patient with a moderate to marked degree of anemia and the symptomatology described at the beginning of this paper, the possibility of hemolytic crisis should be considered. This is particularly true if there is progressive anemia in the absence of demonstrable external blood loss.

In addition to falling levels of hemoglobin and hematocrit, a blood film will show signs of active blood regeneration due to the response of the bone marrow to the stimulus of sudden hemolysis. (In Congenital Hemolytic Jaundice this is not always so, as will be seen later). The blood film, then, will show marked polychromasia, nucleated red cells, macrocytes and increased numbers of leucocytes and platelets. Spherocytes are usually seen in considerable numbers in the blood films of patients with Congenital Hemolytic Jaundice and Acquired Hemolytic Anemia at time of crisis.

Indirect serum bilirubin will be elevated and urobilinogen in the urine will be present in increased amounts. On occasion, there may be hemoglobinemia, hemoglobinuria and hemosiderinuria.

A Coombs test should be done in patients suspected of hemolytic crisis. It is usually positive in Acquired Hemolytic Anemia and negative in Congenital Hemolytic Jaundice, but not always.

Specialized investigative procedures will not be dealt with since the above standard laboratory procedures are sufficient to confirm or eliminate the diagnosis of hemolytic crisis.

CONGENITAL HEMOLYTIC JAUNDICE (HEREDITARY SPHEROCYTOSIS)

In this condition, a metabolic defect in the red cell is responsible for its losing its biconcave shape and assuming a spherocytic appearance. It is an hereditary and familial disorder with some degree of acceleration of hemolysis, in excess of normal erythrocyte degeneration, due to the metabolic defect already mentioned. The bone marrow often is capable of stepping up production to a point which compensates for the increased hemolysis and little or no anemia develops.

At intervals, however, there may be "minor" or "major" crises with sudden onset and rapid increase in the degree of anemia. Crisis may develop for the first time in adult life and may be precipitated by intercurrent infection.

Such crises were regarded for many years as being due to a sudden acceleration of blood destruction. However, Owren in 1948, showed that this may not be so and that jaundice, serum bilirubin and urobilinuria may decrease suggest-

ing actual decrease in blood destruction. At present, there is no final conclusion; both hemolytic and aregenerative factors are thought to play a part.

When the condition of Congenital Hemolytic Jaundice has previously been diagnosed, crisis is usually readily recognized. When a patient presents with crisis, the presence of spherocytosis, anemia and negative Coombs test prompt the investigative procedures necessary to prove the diagnosis, namely, tests of osmotic and mechanical fragility and a review of other members of the family.

Treatment of hemolytic crisis in Congenital Hemolytic Jaundice consists of blood transfusions and supportive therapy. Splenectomy is indicated and may occasionally be necessary during the stage of crisis but is better delayed until the general condition of the patient is improved.

ACQUIRED HEMOLYTIC ANEMIA

Occasionally hemolytic crisis occurs in patients with Acquired Hemolytic Anemia. Since the mechanism is related to auto-antibodies, the Coombs test tends to be positive (either direct or indirect). In a very active phase of hemolysis such as occurs during crisis, spherocytes are a prominent feature on the blood film, even though they may be absent during the ordinary course of the illness. These features are of great assistance in the diagnosis. Further investigative procedures in determining the presence or absence of hemolysins and agglutinins will be necessary in such patients but are beyond the scope of this paper.

The management of hemolytic crisis in Acquired Hemolytic Anemia is difficult. Frequently there will be difficulty in cross-matching blood for the patient. He will require transfusion but prior to administration of blood, large doses of intravenous hydrocortisone should be given in an attempt to control the hemolytic process. If the patient's condition improves with steroid therapy, carefully cross-matched blood should be administered cautiously. If, however, these measures are unsuccessful, emergency splenectomy may have to be considered. There are no exact criteria for decision as to splenectomy under such circumstances and good clinical judgement is required.

SICKLE CELL ANEMIA

The electrophoretic identification of various types of abnormal hemoglobins in the so-called hemoglobinopathies has greatly clarified the mechanism of hemolysis in these conditions. Sickle Cell Disease occurs in Negroes with hemoglobin of S S type. In addition, S, type hemoglobin can occur in combination with C, D and other types of abnormal hemoglobin. Hemolytic crisis has been reported in Sickle Cell Anemia (S S), in S C and S D patients and also in patients with Thalassemia exhibiting Sickle Cell trait.

The occurrence of the clinical picture and hematological findings of hemolytic crisis in a Negro would suggest the possibility of one of the hemoglobinobinopathies. In Sickle Cell Anemia, sickling of the red cells may be seen on the blood film or may be brought out by the addition of reducing agents to the blood.

These conditions are rare in Canada but are of considerable clinical importance in certain areas of the United States.

Blood transfusion, sedation and supportive therapy are the measures used in treatment. Splenectomy is of no value.

PAROXYSMAL NOCTURNAL HEMOGLOBINURIA

This condition is relatively of much less importance because of its rarity. The red cells of the patient are abnormally sensitive to hemolysis by normal constituents of plasma (the properdin system). This results in a chronic hemolytic anemia aggravated during sleep by accumulation of CO_2, with passage of dark urine in the morning due to hemoglobinuria.

Hemolytic crisis may occur in this condition and may be precipitated by a variety of causes, including blood transfusion. Therefore, in treatment of crisis, blood transfusion must be used very cautiously. Here again splenectomy is of no value and steroid therapy should be avoided since it may precipitate thrombotic manifestations. Anticoagulant therapy is used in the long term management of this condition in an effort to prevent thromboses. Heparin, however, should not be used since it may accentuate the activity of the properdin system.

SUMMARY:

Hemolytic crisis occurs in several hematological disorders. Recognition of the condition and institution of correct therapy may be life-saving. In addition, since the condition may at times simulate an acute abdominal emergency, accurate diagnosis is necessary in order to avoid unnecessary abdominal surgery.

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Men and women from all walks of life work as volunteers in the welfare organizations supported by your local Community Chest, Welfare Federation or United Appeal.

They gladly give of their leisure time to work in health, recreation and family welfare agencies; they campaign for funds for these services; they serve as committee and board members directing operations. They deserve a friendly welcome from every Canadian.

Diving Emergencies

Surgeon Lieutenant Commander Henry D. Oliver, R.C.N.

In recent years there has been a steady increase in the number of people participating in diving in and around the lakes and shores of Nova Scotia. Most diving is still done by naval personnel, however, the proportion of diving done by other professionals and pleasure swimmers is increasing. Of course, day-to-day diving and caisson operations are usually carried out without any untoward incidents. However, there have been several cases recently, in other provinces, where people have lost their lives or have become permanently disabled through lack of knowledge of the treatment of bends and through improper use of available equipment. It is in an attempt to prevent similar incidents occurring in Nova Scotia that this short article is written. No attempt will be made to cover the treatment of all aspects of decompression sickness in detail, but rather a brief review will be made of the hazards of working under water and of the highlights of treatment.

In caisson workers the most common hazard is, of course, bends. Bends (Caisson Disease) develop as a result of the liberation of gas bubbles from body fluids that are super-saturated with nitrogen when rapid decompression takes place. The bubbles are microscopic in size and form both intravascularly and within the body tissue, particularly those tissues having a high fat content or a poor vascular supply. Bubbles so liberated, may remain in situ producing local symptoms of joint pain, prickling and itching of the skin (formication). Alternatively, bubbles may be swept via the venous circulation to the pulmonary bed where together with locally formed bubbles they hinder the pulmonary circulation, cause local oedema and give rise to the syndrome known as the chokes, the main features of the chokes being cough, substernal pain and dyspnoea. On examination, coarse rales and rhonchi are heard. Bubbles also cause symptoms in various parts of the Central Nervous System which may result in hemiplegia, paraplegia or weakness or paralysis of one or more limbs. Visual disturbances, tunnel vision, diplopia and scotomata also occur. The most common cause of the bends is the failure of the diver or those persons responsible for the diver to adhere to the recommended decompression tables. Either the diver is kept at depth for too long a period, or he ascends too rapidly or makes too many dives in a 24-hour period. Occasionally a rapid ascent is unavoidable due to some other diving accident or emergency. However, all such cases should be treated by recompression and therapeutic decompression as soon as possible if the depth of the dive warrants decompression. In industry, where the time the worker spends in the caisson or under water is the only time that makes money for his firm, it is difficult to impress upon those persons responsible, the need for proper decompression, particularly when four hours work at 70 feet requires approximately four hours decompression.

The main principle in the treatment of bends is recompression. It is impossible to treat caisson disease at atmospheric pressure. The most common error in treatment is failure to recognize the symptoms of bends. A diver who ascends rapidly due to some other rather more spectacular emergency, for example, stomach cramps, rupture of air hose, a fall resulting in a sprain, or a severe cut or burn, may be treated for his immediate complaint with no thought

to the possible development of bends. Another principle to bear in mind when treating bends or cases that are suspect, is speed in commencing treatment. The longer the time interval between appearance of symptoms and treatment, the longer the recompression period will be and, in the case of Central Nervous System involvement, the less chance of recovery. Caisson disease is one of the few conditions where over-treatment does less harm than under-treatment. Failure to treat a serious case adequately results in aggravation of the damage that has already been done and makes proper treatment even more arduous than it need be. Often doubtful cases are not treated by recompression, the symptoms of the bends being ascribed to another condition. In these cases, the only way to make a differential diagnosis is by recompression.

While a recompression chamber is ultimately required in all cases of severe bends, providing the condition of the patient permits and the depth and temperature of the water are suitable, there is no reason why recompression and therapeutic decompression cannot be carried out in the water if another diver can accompany the patient. Controlled recompression in a chamber is to be preferred however, if transportation can be arranged.

Another diving accident, similar to bends, is aeroembolism. Whereas bends are caused by myriads of microscopic bubbles forming spontaneously in intact body tissues, aeroembolism is due to an alveolus being ruptured by expanding gases in the lung with the direct entry of relatively large amounts of air into the pulmonary circulation. The amount of gaseous expansion required to cause rupture of an alveolus and subsequent aeroembolism can occur in a rapid ascent from as shallow a depth as 15 feet. An aeroembolism is a dramatic occurance and is often followed by unconsciousness, hemiplegia, paraplegia or even death. Treatment is the same as for a severe case of bends, the principles being to reduce the size of the gas bubble by as much as possible and to minimize the amount of damage done to the surrounding cells. Even more so than with bends, immediate treatment is essential. In view of the shallow depth at which aeroembolism can occur, more cases of aeroembolism would be expected among aqua-lung swimmers and relatively untrained and unskilled pleasure divers than among professional workers.

Other diving accidents that may require medical attention with or without recompression are, blow-up, squeeze and gaseous contamination of inspired air. Blow-up occurs when a diver in a standard diving suit allows himself to become too buoyant and rises rapidly to the surface. As he ascends, the water pressure decreases and the pressure in the suit remains constant. This causes the suit to blow up, spreadeagling his limbs thereby making it impossible for him to reach his helmet valves and to control the pressure within his suit. The great danger in blow-up is that the suit may burst before the diver can reach the surface and he may fall to the bottom. A blow-up will, of course, require decompression if the diver has been at depth for a sufficient time. Squeeze occurs when a diver descends faster than the air supply can build up in his suit. The situation is the reverse of blow-up. The helmet of the suit is the only rigid part of the suit and the increased water pressure tends to squeeze the diver out of his suit into his helmet. In severe cases, the diver is so firmly wedged into it, that it is impossible to remove the helmet. Squeeze occurs when a diver falls under water either from a staging, or down a slope or from a cliff. Squeeze is often fatal. The symptoms are usually obvious with bleeding from the nose and mouth, maybe a badly swollen face, sub-

conjunctival haemorrhage and the face and neck and shoulders covered with petechia. The imprint of his breast plate may be seen on his chest, back and shoulders.

Contamination of the inspired mixture with carbon monoxide or carbon dioxide is relatively common. In standard diving, the air pumped to the diver by the compressor may be contaminated with exhaust gases from the compressor itself. In diving operations, small amounts of toxic gases can have a very serious effect, as a depth of 33 feet of water is equal to an atmospheric pressure of two atmospheres. An innocuous percentage of carbon monoxide on the surface can produce severe symptoms at depth. In the forms of self-contained diving where the breathing mixture is rebreathed through soda lime, the inability of the soda lime to completely remove the expired carbon dioxide is another common cause of CO_2 poisoning. In addition to the contamination of the inspired mixture, diving at great depths can produce either oxygen poisoning, where oxygen mixtures other than air are used or nitrogen narcosis with air or air mixtures. Oxygen poisoning can occur at any depth greater than 60 feet where pure oxygen is used, Its initial symptoms are mild twitching of the face and fingers, followed soon after by generalized convulsions. Nitrogen narcosis results from a long exposure to air or air mixtures at depths usually in excess of 200 feet. Nitrogen narcosis results in a feeling of well-being, loss of iudgment and subsequent errors or mistakes that may prove fatal. Those people who dive at depths deep enough to give rise to oxygen poisoning or nitrogen narcosis, are usually well-trained and experienced persons and it is only rarely that serious difficulties are encountered. The discussion of these problems is certainly outside the scope of this short paper.

It is regretted that there is no protective legislation to cover divers or caisson workers. British Columbia is the only province whose Workman's Compensation Act covers this industry even though this coverage is neither complete nor up to date. No standards are laid down regarding duration and depth of dives or caisson shift, and no safety code controls the standard of equipment used in any particular operation. Fortunately for us in Nova Scotia, there is a well-equipped and well-staffed therapeutic decompression chamber situated in the Naval Dockyard in Halifax. As this is the only adequate chamber east of Victoria in Canada, the Navy has assumed the responsibility for providing recompression facilities for any patients who may require it. If any practitioner is faced with either an actual or suspect case of the bends or aeroembolism, he will be sure of obtaining advice as to the handling of the case through the Duty Medical Officer in R.C.N. Hospital, Stadacona.

E.N.T. Emergencies in Aviation

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Aviation has made remarkable advances during the 50 years that have passed since the Silver Dart made history as the first heavier than air machine to fly in the Commonwealth. There is little in a modern aircraft to remind us of that frail machine, whose replica was so obviously at the mercy of the elements at Baddeck last Monday. Fighter aircraft today weigh many, many tons, are capable of speeds well in excess of the speed of sound, and operate best 10-12 miles above the surface of the earth. The development of aviation has had as its corollary the development of a group of problems of otolaryngologic significance. Some of these problems are common to both Civil and Military flying, being different only in degree. Today I will confine my discussion to three of these problems.

(1) Effect of Altitude on the middle ear and sinuses
(2) Effect of aircraft engine noise on hearing
(3) Airsickness

(1) Effect of Altitude on Middle Ear and Sinuses

Atmospheric pressure varies with altitude according to a curvilinear gradient. At sea level atmospheric pressure is 14.6 lbs/inch $_2$(which is equivalent to the pressure exerted by a column of mercury 760.0 mm in height). The pressure decreases with increasing altitude until the so-called hard vacuum of space is reached. Some altitude pressure figures are as follows

Altitude	Atmospheric pressure in mm Hg.
Sea level	760.0
10,000 ft.	522.6
20,000 ft.	349.2
30,000 ft.	255.6
40,000 ft.	140.7
50,000 ft.	87.3
Outer space	0

This decreasing ambient pressure with increasing altitude means that if an elastic walled container, such as a balloon, containing a known quantity of air at sea level were carried upwards, the volume of enclosed air would double at 18,000 ft., quadruple at 34,000 ft. and be 8 times larger at 48,000 ft. The reverse would occur with descent. The air within all the bodies air cavities is affected in exactly the same way, but fortunately only in a few cavities, for example the middle ears and paranasal sinuses, is the passage for air exchange so narrow that serious pressure effects may occur. The conditions which result in the ear and sinuses are Otitic Baratrauma and Sinus Baratrauma.

(a) Otitic Baratrauma

The air in the middle ear and mastoid cells is continuous with that of the environment via the Eustachian tube. Each eustachian tube projects

into the nasopharynx as the eustachian cushion; and this eustachian cushion acts as a one way flutter valve. In ascent, the excess air volume in the middle ear system passes down the tube to blow off into the nasopharynx without voluntary movement being required. However, on descent air cannot pass from the nasopharynx into the middle ear, to replace that blown off in ascent, until the eustachian tube is actively opened by contraction of the Tensor Palati muscle. Movements which cause contraction of the Tensor Palati muscle are swallowing, chewing, yawning and the crying of infants. Unless air replacement occurs with every 500 ft. drop in altitude, otitic baratrauma will occur. Otitic baratrauma can occur in any person regardless of his health or physical condition, but factors which predispose to its onset are:

1) Ignorance of the necessity of opening the eustachian tube with each 500 ft. drop in altitude.
2) Falling asleep during descent
3) Flying with a U.R.I., sinusitis, active nasal allergy, or adenoiditis.

These conditions cause a swelling of the eustachian cushions and tubal lining impeding air passage. In mild cases of otitic baratrauma the negative pressure in the middle ear causes injection and indrawing of the ear drum, and the patient complains of mild ear ache, a sense of fullness in the affected ear, a low pitched tinnitus and decreased hearing of the conductive type. In the severer degrees, with a marked negative pressure in the middle ear, the ear drum may become quite red and congestion with a picture not unlike acute otitis media, and serum or blood may be sucked from mucosal blood vessels into the middle ear cleft. The pain is usually quite severe and may radiate about the ear. The conductive hearing loss may be quite marked. Rarely does the ear drum rupture.

Treatment of otitic baratrauma has as its aim equalization of pressures in the ear, and removal of any fluid which has collected in the middle ear. The mucosa of both nasal cavities and nasopharynx should be sprayed thoroughly with a nasal decongestant and this procedure repeated every four hours until symptoms have subsided. In mild cases, the patient may be able to overcome the tubal obstruction after the nose has been decongested, by using valsalva's manoever. In severer cases politzerisation is required using a politzer bag or an inflated balloon, and this should be repeated daily until symptoms have resolved. A handy initial procedure is to puncture the postero inferior quadrant of the ear drum under direct vision through an otoscope, using a bent lumbar puncture needle and a leur lok syringe, and inject air into the middle ear cleft and aspirate any fluid that is present. No local anaesthetic is fully effective for this procedure but 10% Cocaine solution applied to the ear drum for 10-15 minutes is of help.

(b) **Sinus Baratrauma**

Sinus baratrauma occured in about 2% of Canadian aircrew trainees during the 2nd World War, but since then aircraft pressurization has reduced this incidence in civil aviation. This condition does not occur in people with normal and heathy nose and sinuses, but occurs where there is an obstruction of the sinus osteum during either ascent or descent. Predisposing factors are U.R.I., nasal allergy, nasal polypi, acute or chronic sinusitis and structural defects such as a deviated nasal septum, which could narrow the drainage passage of the middle meatus. The frontal sinus is affected more often than the maxillary sinus because of its long and sometimes tortuous duct. The

patient usually complains of severe frontal pain of sudden onset and resembling a bee sting or a blow on the head. The nasal passage on the affected side becomes blocked and epistaxis may occur. X-ray examination shows the affected sinus to be clowdy, and submucosal haemorrhages may be detected in the sinus wall. Treatment has on its aim relief of pain and an unplugging of the sinus osteum. Hot compresses to the affected sinus is soothing. The nasal passage should be shrunk by spraying thoroughly with a nasal decongestant every four hours until the patient is asymptomatic. Proetz displacement with a decongestant solution often gives marked improvement in that it allows some of the vasoconstrictor solution to enter the sinus. Where the middle meatus is crowded by the middle turbinate a useful procedure is to anaesthetise the meatus area with pledgets of cotton batten dampened in 2% Pontocaine, and infracture the middle turbinate towards the nasal septum. On occasion antral puncture is required. The patient must not be allowed to fly again until any predisposing causes which may be present are corrected.

(2) **Effect of Aircraft Noise on the Ear**

Occupational deafness due to noise has been known in industry for many decades. The same type of hearing loss may be caused by aircraft engine noise. The manufacture of aircraft engines with greater and yet greater thrust, has increased the noise level around such engines to such an extent as to endanger the health of individuals exposed to it and present legal problems to airfield authorities. The limit of human tolerance to noise would appear to be in the region of 150 db. (db re 0.0002 dynes/cm^2), yet sound pressure levels higher than this can be recorded close to the newer jet engines. Individuals vary widely in their resistance to noise damage but it is generally accepted that exposure to an overall noise level greater than 85 dbs. may be harmful depending on the intensity of the sound and the duration of exposure. Minor damage causes a cloudy swelling of the cochlear endorgan which may be reversible. Greater noise causes a disruption of the cochlear endorgan affecting the frequencies around the 4,000 cycles/sec. area. Once the cochlear endorgan damage has gone beyond a cloudy swelling the hearing loss which results is permanent. It does not improve and it does not get worse, unless further exposure to noise occurs. There is no known treatment for noise damage to the ears, so its prevention becomes extremely important. Protection depends on the level of the noise and the duration of the exposure, and the following are the criteria suggested for use by the armed forces.

db of noise re. 0.0002 dynes 1/cm^2	duration of exposure	protection required
85-100	Continuous exposure over 4 hrs.	Ear plugs
100-130	Any exposure	Ear plugs
130-150	Any exposure	Ear plugs and ear muffs
Over 150 db.	No exposure permitted	It is considered that people exposed to this level of noise would require total body protection

Dry cotton batten ear plugs are of little use. Paraffin wax impregnated plugs are of some value but rubber ear defenders such as those produced by the Mine Safety Appliance Co. are highly satisfactory. Efficient ear muffs of the type used by the Canadian Armed Forces are not yet readily available for civilian use.

(3) **Airsickness**

According to an analysis made by various airlines, airsickness is the commonest cause of passenger discomfort. Airsickness is due to vestibular stimulation, although the symptoms are often aggravated by a psychic fear of flight. The majority of people will become sick provided the vestibular stimulation is strong enough, but there are a few unfortunate individuals who is particularly susceptible to motion. These individuals had best fly by night when turbulance is usually less, and visual stimulation of psychic fears is minimized. If possible, they should sit near the centre of gravity of the aircraft where pitching and yawing movements are not so marked. Antihistamines have a beneficial effect both because of their specific effect on the end-organs of balance and because of their mild sedative effect. I would suggest Dramamine Bonamine or pyribenzamine 100 mgm. 1 hr. pre flight. To be repeated in 4 hrs. if required.

Gentlemen, my lecture is finished. I have confined the subject matter to those conditions which affect airline passengers, and deliberately avoided the more numerous and more serious conditions which affect military aviators.

The most significant development in welfare fund raising has been the advent of United Funds or Appeals in 42 Canadian cities. The new programme replaces the former community chest and is distinguished by the inclusion of such national organizations as the Canadian Red Cross, March-of-Dimes, C.N.I.B., Canadian Mental Health, St. John Ambulance and societies dealing with dread diseases. As an answer to too many campaigns and as a way of assuring funds for both local and national agencies, the United Fund is being considered in many more Canadian cities.

Acute Gouty Arthritis—Medical Emergency

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For the patient there can be no doubt that an acute attack of Gouty Arthritis is a medical emergency. This is primarily because of the severity of the pain. In other types of Arthritis pain occurs chiefly upon movement of the joint, but in Gout the pain is continuous, though of course aggravated by movement or pressure. The relenting character of the pain adds to the patient's distress. Acute Gouty Arthritis may be an emergency in the eyes of those who are closely associated with the patient, because his irritability and apprehension over his swollen joint makes them uncomfortable.

The impression persists in the minds of some that Gout is a rare condition in North America, but this is quite untrue. The diagnosis has very frequently been missed, a common error being to diagnose Cellulitis of the foot, treat the patient with antibiotics, and feel that one has been right when the inflammation subsides after four or five days, as is common with acute attacks of Gouty Arthritis. What then should lead us to suspect the diagnosis of Gout? First of all it is 20 times as common in males, though it occurs in females particularly after the menopause. The metabolic defect is believed to be inherited as Mendelian dominant, and the appearance of Hyperuricemia usually takes place in males after puberty, but in females not until the menopause. Many men do not have their first attack of Acute Gouty Arthritis until the age of about 40, at which time they may have been Hyperuricemic for 25 years. Similarly, in the female they are apt to have their attacks a good many years after the menopause, that is in old age.

The most striking thing about the acute Gouty attack is its suddenness of onset, which is rarely duplicated in other forms of Rheumatic Disease. Attacks often begin in the small hours of the morning (nocturnal onset was recorded in 62% of attacks in a recent study of 100 cases of Gout (1) and within a few hours the affected joint is usually swollen with reddening of the overlying skin. Later the skin becomes more purplish in colour, and at the end of an untreated attack 50% of patients will note intense itching of the skin and desquamation over the involved joint. High fever accompanies some severe attacks.

We are accustomed to thinking of Gout as occurring very frequently in the metatarso-phalangeal joint of the great toe, and this joint is certainly significantly more frequently involved than any other, but there is no reason why Gout cannot occur in any joint or tendon sheath, and sometimes acute attacks of pain and inflammation occur about Gouty tophi situated outside of joints. The joints of the spine can be involved. The attack may be polyarticular. When Gout affects large joints such as the knee, the characteristic redness may not be present, the pain may be less severe and recovery less swift.

It is therefore important to consider Gout in the differential diagnosis of every case of inflammatory Arthritis, particularly when a single joint is involved, when there is redness, and especially when the attack is of sudden onset. Acute Gouty Arthritis is the commonest type of inflammatory flare-up of a joint 5-10 days after a surgical operation. If tophi are present, for

example in the pinna of the ear, they may be recognizable and may confirm the diagnosis immediately, but they are not present until the patient has had his metabolic disorder for years, and the diagnosis should be scarcely less likely if tophi are not present. A history of complete recovery from previous attacks of acute Arthritis points toward Gout.

Suspecting Gout what treatment should one apply? The use of Colchicine has the advantage that if the joint responds rapidly with relief of pain followed by quick subsidence of inflammation, the diagnosis will be confirmed even if Laboratory support is lacking for a period of months or years afterwards. The drug should be given alone, not in combination with Salicylates, for two reasons; first, the use of an analgesic agent in combination with the Colchicine spoils the diagnostic test; and second, the factor of Salicylate, or other drug, toxicity is added in a situation where it is necessary to give Colchicine to toxic level to be sure an effective dose has been given. Colchicine should be given at the rate of 0.6 mg. every hour or two hours until pain is relieved or the patient feels some symptoms of gastrointestinal upset such as nausea or diarrhea. I usually instruct the patient or nurse to use not more than 15 tablets (9 mgs.) in a course of treatment. Probenecid and Sulfinpyrazone do not affect acute attacks of Gouty Arthritis.

Colchicine can also be given intravenously, and is supplied in ampoules varying from 1 mg. to 3 mgs. I have usually given 3 mgs. intravenously, and if in the course of 3 or 4 hours the patient's pain is not relieved, repeated the injection. Thrombophlebitis is an occasional complication, and, since the solution is an alcoholic one, great care must be exercised that none of the solution gets outside the lumen of the vein. Usually the use of the intravenous route avoids the production of diarrhea by Colchicine, but gastrointestinal reactions do occasionally occur.

Attempts to modify the Colchicine molecule have produced such drugs as Desacetylmethylcolchicine, known as Demecolcine and also as Colcemide. The response of acute Gouty Arthritis to Colcemide has been found to be comparable to that with therapeutic doses of Colchicine, without the undesirable gastrointestinal symptoms, but the drug is believed to possess a potent depressive action on bone marrow when given over a long period, and an occasional patient receiving either Colchicine or Colcemide has developed transient total Alopecia ([2], [3]). For the present it appears wise to stick to the preparation we know best, i.e. Colchicine.

Phenylbutazone (Butazolidin) is as effective as Colchicine if not slightly more so, in relieving the acute attack of Gouty Arthritis, but of course does not have the specificity which makes Colchicine a diagnostic trial of treatment. The avoidance of diarrhea may be valuable. The incidence of toxic effects of this drug in the short period of treatment necessary for acute Gouty Arthritis is small. The administration of a small maintenance dose, such as 100-200 mgs. daily, will control recurrence in patients with frequent acute attacks. In the event that long term prophylaxis is embarked upon, however, one is faced with the necessity of observing ones patient carefully during the period of drug administration, because of the possibilities of Sodium retention, bone marrow depression, gastric irritation (aggravation of peptic ulcers), and serious hypersensitivity reactions such as Dermatitis, which can be fatal.

ACTH or large doses of adrenal glucocorticoids can be used to terminate an acute attack of Gout, but there does not seem to be any particular advantage in using these hormones for this purpose. Administering ACTH and then

stopping it has sometimes resulted in precipitation of acute attacks of Gout.

When the acute attack has been relieved the patient still has a metabolic disorder and a liability to further attacks of Arthritis. It is not the intention of this short presentation to deal with the treatment of Chronic Gout, but too often the patient is neglected after his acute episode is over. What shall we do about the basic problem that this patient produces too much Sodium Urate in his body? First of all, of course, we want to know the level of his Serum Uric Acid and the state of his renal function. If his Serum Uric Acid level is over $7\frac{1}{2}$ mgs.%, or he is having frequent attacks of Arthritis, he should be treated with long-term uricosuric therapy. In practice it may be difficult to persuade an asymptomatic patient to take tablets every day of his life indefinitely. Since, however, he is to some extent threatened with premature Atherosclerosis and damage to his kidneys by the accumulation of Urate, one is justified in advising uricosuric therapy very strongly. The patient who has frequent acute attacks of Gouty Arthritis, or the patient with chronic Arthritis, usually presents no difficulty and should be treated with uricosuric drugs. It is generally thought that there is a danger of an increased number of attacks of acute Gouty Arthritis during the first three or four months of effective uricosuric therapy. For this reason prophylactic Colchicine is usually given over this period in a dose of 0.5 to 1 mg. daily.

The commonly employed uricosuric drugs are Probenecid (Benemid) and Sulfinpyrazone (Anturan). Salicylates have been favoured by some authorities, but it is necessary to give 5 or 6 gms. per day of Acetylsalicylic Acid or Sodium Salicylate to get satisfactory uricosuric action, and many patients experience disturbing side effects at such dosages. In addition the dose consists of a large number of tablets each day or a rather unpleasant liquid medicine in considerable amounts. The use of Cincophen as a Uricosuric drug has been abandoned by most Practitioners because of its high toxicity. Although Phenylbutazone has some uricosuric action when given in large doses, it is not satisfactory as long-term uricosuric therapy.

The dose of Probenecid usually employed is 500 mgs. twice a day or four times a day, depending upon the response. The dose of Sulfinpyrazone is 100 mgs. twice a day or four times a day, based on the same criteria. Probenecid has been proven a very safe drug, causing occasional Dermatitis which can usually be controlled by temporary use of concomitant Antihistamine therapy. Gastric irritation occurs in some patients and can be controlled by giving the drug with food, or by giving an antacid at the same time. Despite the fact that Sulfinpyrazone contains the notorious Pyrazol ring, studies to date have not demonstrated significant toxicity. It would be well, however, if employing this preparation, to warn the patient to report sore throats (agranulocytosis), skin rashes, renal symptoms and dyspepsia. Urinalysis should be done every few weeks, and Hemoglobin, White Blood Cell Count and Differential Count every three or four weeks until we become sure that this is as safe as it appears to be ([4], [5]).

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Recent Advances in the Treatment of Poisoning

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This evening I propose to talk to you about advances which are sufficiently recent, or sufficiently in a state of flux that they could not be described dogmatically in any book that could be published at this time.

The first of these advances is the introduction of what are known as chelating agents. These are agents which are capable of grasping metals, like a claw. They are organic compounds which are able to bind metals in such firm combination that the metals no longer exhibit any of their ionic properties and are, therefore, to a large extent non-toxic.

These chelating agents are used very widely apart from the treatment of poisoning. I suppose their widest use is the softening of water. They are also used to remove traces of metals from dyes, foods, etc. They are used as anticoagulants, as they chelate calcium and render it inactive. They are used a great deal in chemical analysis; some of our newer methods in the laboratory, such as the estimation of serum calcium, are based upon the employment of chelating agents.

These substances, which will seize and sequester a metal so that it no longer has any of the characteristic properties of that metal in solution, have proved their value in the treatment of poisoning. There is one I will only mention for the sake of completeness, because it is far from being a recent advance; that is British Anti-Lewisite or BAL. You will recall that this was developed by British workers during World War II, as an antidote to Lewisite, an arsenical vesicant "gas." British Anti-Lewisite, or BAL is effective in the treatment of a great variety of types of poisoning, especially arsenic, mercury and gold. There is nothing new about BAL. I simply refer to it by way of an introduction.

There is another chelating agent which is somewhat newer. It is often spoken of as disodium calcium EDTA. This is an extremely powerful chelating agent. If EDTA is given rapidly, intravenously, it will chelate calcium so effectively that the animal will die in hypocalcemic tetany. If you give it more slowly it will not lower the serum calcium, but will drain the body of calcium and the calcium will be excreted into the urine as a calcium chelate.

EDTA has been employed without too much success in the management of hypercalcemia, but obviously from the standpoint of the treatment of poisoning, it is of no use and so, instead of employing EDTA, we use the calcium complex of EDTA, that is EDTA which has already chelated calcium. This will still work as a chelating agent because fortunately a great many metals such as lead, for example, have a greater affinity for EDTA than the calcium. So if you give calcium EDTA, the toxic metal will displace the calcium, which does the body no harm, and the toxic metal will itself be chelated by the EDTA. This chelate, like other chelates, is water soluble, is non-toxic, exhibits none of the ionic properties of the metal, and is excreted in the urine.

Therefore you must use calcium EDTA, as the EDTA itself will chelate calcium; the disodium salt is used because it happens to be very soluble in water.

I am not going to talk to you about the details of dosage because they are found in many books published within the last couple of years. This substance can be given parenterally—intravenously, subcutaneously or intramuscularly. It cannot be given by mouth with any great success because only about 3 per cent of the disodium calcium EDTA is absorbed. The value of this agent in the treatment of lead poisoning has, I think, been established. Now you may ask "Why should a new agent be used in lead poisoning?" You are aware that, although we have had fairly potent therapeutic weapons in the treatment of both acute and chronic lead poisoning, we have encountered two problems which have given a great deal of trouble with the older methods of treatment.

One is lead encephalopathy in children, which has never really been conquered, and indeed with disodium calcium EDTA is not very well controlled. However, it is better controlled than it is with the older methods of treatment. The other problem in lead poisoning has been deleading. The common teaching is that the proper thing to do with a patient with lead poisoning is to relieve the acute symptoms by giving calcium and then delead, that is take the lead out of the bones slowly, so slowly that it does not produce any symptoms but so as to get rid of it over four or five years. Anyone who has ever managed a case of lead poisoning knows that such deleading is almost wholly impractical. Either you do not delead enough or you delead too quickly and precipitate acute toxic episodes.

Most people with lead poisoning have gone around for the rest of their lives carrying the lead in their bones, and therefore aways subject to acute toxic episodes. This disodium calcium EDTA gives us an effective method of deleading because it produces a non-toxic soluble lead chelate which is slowly eliminated in the urine. The only difficulty is that is it not very effective by mouth, and a search for other chelating agents is being made. This I will come back to in a moment.

There has been a great deal of interest too in the use of disodium calcium EDTA in poisoning by metals other than lead. There is a fairly long list of metals, poisoning by which can be treated successfully with disodium calcium EDTA. Some of them are radioactive elements.

In haemochromatosis you might expect that disodium calcium EDTA would chelate the iron, and so it does. And you can demonstrate that in a patient with haemochromatosis, in which the basic lesion is excessive absorption of iron over a long period of time, the excretion of iron in urine can be increased by giving disodium calcium EDTA. The difficulty is that it is too slow; one can get rid of the iron faster by controlled venesections. Only the future will tell if there is a more effective chelating agent for the treatment of haemochromatosis.

Many people have asked if disodium calcium EDTA cannot be used to treat argyria. The answer appears to be "No." The silver is in a very inert form and it is not touched by the chelating agent.

There are other chelating agents that are under study. One that has aroused interest in recent years, and has been the subject of many papers, is a compound known as penicillamine. It derives its name from the fact that when you give a patient penicillin, this compound—penicillamine—is excreted in the urine. Most of the interest in penicillamine has been in connection with the treatment of Wilson's disease or hepatolenticular degeneration which, so

far as one can tell at the moment, is chronic copper poisoning. Wilson's disease is a rare disease, and of course all people who are teaching medicine are criticized by their students if they start talking about a rare disease. But sometimes I think students forget that a rare disease is terribly important to the one who has it. These patients with Wilson's disease in an advanced state often end up in institutions.

Penicillamine, unlike BAL and unlike disodium calcium EDTA, is effective by mouth—a tremendous advantage as it is not very practical to treat a condition like Wilson's disease with prolonged intravenous or subcutaneous injections. Penicillamine is quite rapidly absorbed and very encouraging results have been obtained not only biochemically but also clinically with hepatolenticular degeneration. There is a good deal of work going on testing penicillamine orally in patients with chronic lead poisoning with special reference to deleading. It has been tried in haemochromatosis also. It looks as if it were removing a little iron and a little lead, but the amounts are not very great. Penicillamine is not yet on the market.

Then I want to give you another example just to indicate how this field is expanding. I think you are going to find that within the next three or four years there may be ten or fifteen of these chelating agents being employed for the treatment of various types of poisoning, both acute and chronic. Another one of interest is called dithiocarb. This compound is being used in the treatment of poisoning by nickel carbonyl. This is one of the most toxic of the chemicals encountered in industry. It is a gas with no strong or penetrating odour and is met with commonly in industrial processes involving nickel.

Dithicoarb is a chelating agent which is especially effective in chelating nickel, and has given most striking results in the treatment of acute poisoning by nickel carbonyl. It can be given either by mouth or parenterally. Now I do not want you to feel that is the end of the story. I have simply introduced this subject of chelating agents to you. I have told you all I know to date, but I am quite certain that within two or three weeks there will be another paper out on the subject and there will very likely be more chelating agents.

The next recent advance that I want to talk about concerns a group of poisons known as anticholinesterases. These anticholinesterases are organic phosphorus compounds which inactivate cholinesterase, as the name suggests, so that acetylcholine piles up at the nerve endings. I have no intention of describing all the symptoms which result. There is a widespread disruption of the central nervous system and the peripheral nervous pathways, and the mortality may be high.

These anticholinesterases can be met with under three circumstances. First, they are used as insecticides and accidental exposure may occur. Second, they enjoy a rather high reputation as chemical warfare agents. As a matter of fact the anticholinesterases were originally developed by the Germans during World War II as chemical warfare agents; they were one of their "secret weapons," the nerve gases. Third, anticholinesterases are being used in the treatment of myasthenia gravis. As I understand it, you virtually titrate the patient, as it were, with anticholinesterases. You give them the amount which produces the maximum benefit, without giving them so much as to produce poisoning. But frequently you overtitrate, and not uncommonly these patients, who are being treated with anticholinesterases for myasthenia gravis, develop anticholinesterase poisoning and have to be treated for this.

The treatment of anticholinesterase poisoning, by and large, is not too satisfactory. It is largely symptomatic with the emphasis being placed on keeping the airway clear and on artificial respiration. It is characterized

by profound generalized muscular weakness. Of course this is particularly dangerous when it affects the muscles of the chest, tongue and pharynx. Atropine is the remedy that is ordinarily used and in very large amounts. It will keep the airway clear and will have some action upon the central neural effects, but it will not relieve the muscular weakness.

The new advance here is the introduction of a group of compounds known as oximes. There are two of these oximes which have been tried out in human cases, and more are being developed. These oximes do, from a clinical standpoint, just what atropine does not do—they alleviate the muscular weakness. They do not do all the things that atropine does and so, undoubtedly, the recommendation will be to use the oximes along with atropine and all the other measures, but it is very likely that the oximes will remove the need to perform artificial respiration in many of these cases, and help keep the airway clear by relieving the muscular paralysis of the pharynx and tongue. So much for anticholinesterases.

Now I want to talk for a few minutes of another poisoning which is certainly important. From the standpoint of frequency it is the most important type I have mentioned to this point—poisoning by barbiturates. Next to carbon monoxide and ethyl alcohol, barbiturates are the commonest cause of poisoning. (The tranquilizers are changing the situation, since some people prefer to take a bottle of tranquilizers now as opposed to a bottle of barbiturates.) The old controversy about the use of stimulants in barbiturate poisoning still goes on, and is unresolved. It is admitted I think by everyone that stimulants are often over-used and given to mild cases which do not need stimulants, and that in some cases they are given in excessive doses. Everyone also agrees that stimulants play only a small part in the treatment of barbiturate poisoning. There are all sorts of other measures of importance, such as oxygen, airway maintenance, and artificial respiration—all the various symptomatic measures with which anaesthetists are so familiar and at which they are so skilled. But it is still felt, in some circles, that stimulants have their place. This is extremely difficult to prove, and at the Montreal General Hospital we take the view that we have had good results in severe cases with stimulants, and are loath to abandon them.

The recent advance, or at least something that seems to be a recent advance, was the introduction by Australian workers of a compound known as megimide, which they maintained was a specific antagonist to barbiturates. We have found, as have others, that megimide is almost certainly just another stimulant—a potent one, it is true, but not a specific antagonist as for example nalorphine is in morphine poisoning. Furthermore when megimide is given alone, it has both stimulant and depressant effects on the central nervous system, and it would appear that if you are going to use megimide, you should combine it with some other stimulant. Incidentally, megimide is by no means harmless. If given in too high doses, like any other stimulant, it may cause convulsions; also it may produce a toxic psychosis.

The next topic (I admit these are unrelated topics but I am trying to cover what I think one can glean from the literature of the last eighteen months) —the next topic is our old friend carbon tetrachloride. I say our old friend because it has been known as a poison for many years. It has been used on experimental animals for a great length of time to produce liver damage, a fact which has distracted attention from its principal toxic effect on humans, which is not liver damage but renal failure. Everybody knows that it is a poison. Not only does everybody know it is a poison, but most large industries have recognized it as a poison and discontinued its use; they employ other

compounds such as methyl chloroform, trichlorethylene or perchlorethylene as carbon tetrachloride substitutes, which can be used as cleaning and degreasing agents.

Carbon tetrachloride is indeed quite poisonous. About eighteen months ago we reported a series of twelve cases in the Canadian Medical Association Journal. Its toxic effects are greatly increased by ethyl alcohol. A typical history is of the woman cleaning her dress while having a bottle of beer. I think it would be interesting, so as to keep this lecture from getting a little heavy, to tell you that the French have a very practical attitude towards this. A few years ago a paper came out from France, warning that carbon tetrachloride was toxic and that its toxicity was increased by alcohol. Now, you would think they would warn people not to take alcohol when they are working with carbon tetrachloride. But the French are much too realistic for that. They know perfectly well that it is proper to drink wine with meals and so the conclusion of the paper was that work with carbon tetrachloride should not be done after meals.

Finally I want to say a word about poison control centres. I have the record showing that the Children's Hospital here has been way out ahead in the establishment of poison control centres. You are to be congratulated. I want you to understand that I appreciate the tremendous advantage of these poison control centres, but it just happens that I am using poison control centres at the moment as a device to warn you about something. They have a disadvantage. This does not mean, of course, that they should be abolished. It simply means they may be misused. The average general practitioner is going to get the idea that he cannot handle a patient with acute poisoning unless he has a vast amount of information. Although I am naturally in favour of poison control centres and of any educational effort and any attempt to disseminate material about poisoning, I think it is a great mistake if the general practitioner gets the idea that the management of poisoning is a job for a specialist with a card index.

I have been talking about chelating agents. I have been talking about oximes. I have been talking about megimide, which perhaps is not very good, but still has been claimed to be a specific antidote. I have been talking about those because they are recent advances. But I do want now, at the end, to take the emphasis away from specific antidotes and point out that surely we want chelating we want agents, certainly drugs like nalorphine when we can get them. But by and large the management of acute poisoning rests upon certain basic symptomatic procedures which are well known to every general practitioner. It is just as well that it is so because as in all other emergencies, what you do in the first one half to one hour, or even the first few minutes, is far more im- portant than any amount of later treatment with or without specific antidotes.

It is possible to treat poisoning successfully in the majority of cases without knowing what the poison was—by symptomatic treatment. Such symptomatic measures as clearing an obstructed airway, artificial respiration, the judicious use of stimulants, the relief of pain, the control of shock, induction of vomiting, gastric lavage—these are measures which everyone knows about and which everyone can use no matter where he is, whether in a farmhouse or a city hospital, and these still form the basis of the treatment of poisoning.

It is wholly impractical to deal with emergencies by calling a specialist, when so much depends on what you do in the first half hour. Prompt and early symptomatic treatment is, I think, the basis of the management, not only of poisoning, but also of most of the medical emergencies which you are hearing about this week.

Maritime Medical Care Incorporated

Editor's note:—The recent increase in subscriber's rates has led to many inquiries. Typical of such is the following subscriber's letter and the reply of the President. The Bulletin is pleased to publish these letters at the request of Maritime Medical Care, Incorporated.

June 10, 1959.

F. Murray Fraser, M.D.,
President,
Maritime Medical Care, Incorporated,
10 Duke Street,
Halifax, Nova Scotia.

Dear Sir:—

This will acknowledge your letter of May 15th regarding an adjustment of monthly rates. If you will look into my files you will realize that Maritime Medical Care has rendered a great service to me over the past few years. My family has had its share of sickness and the service and security received from your organization has meant a great deal to me. This further increase in rates however, is quite disturbing to me. I am afraid that the main purpose of the organization is going to be defeated. The average wage earner will be pushed out by the high rates and actually, that is the person we want to help.

You say that the Medical Society sponsors M.M.C. and it has the full support of the medical profession. Does this mean that medical doctors agree to accept the rates as laid down by M.M.C.? Also, in this new schedule of rates, and I feel the family rate is too high, will all doctors fees be paid by M.M.C.?

In your letter you mention "ever increasing medical benefits," would you please list these?

Yes, I want to keep my M.M.C. coverage but, I also want to find out more information about M.M.C. I would like to see a financial statement on each year's operation. I want to be a member of an organization that is set up to meet the medical needs of the general public, but I do not want to belong to this organization if it is simply a tool in the hands of the medical profession.

Yours very truly,

J. D. G.

MARITIME MEDICAL CARE INCORPORATED

10 Duke Street,
Halifax, N. S.
June 15th, 1959.

Mr. J. D. G.

Dear Mr. G.:

This will acknowledge your letter of June 10th which raises questions as to the future relationship of Maritime Medical Care, its subscribers and its participating physicians under the new rate structure which will become effec-

tive July 1st, 1959. It is evident that you have devoted considerable thought to this matter and the questions which you wish answered are very pertinent at this time. It is gratifying to hear too of your appreciation of the services which Maritime Medical Care has been able to render you and your family over the past few years.

As you imply, the primary purpose of Maritime Medical Care was and is to make available adequate physicians' services to the general populace of Nova Scotia at as low a cost as possible to the subscriber, and further, to make this coverage as comprehensive as possible in relation to its cost. During the first few years of operation it was quickly found that our subscribers desired much more medical attention than could be regarded as a necessary minimum, that is; the utilization of benefits among those insured became much higher than was the case with the group not covered. This is a common finding of the prepaid health plans in Canada and insurance companies in general.

In addition to the increase in utilization among our subscribers, which resulted in higher medical costs being borne by the plan, there have been, in recent years, many advances in Medicine which have been almost automatically included as benefits. Such advances have been made in all fields of Medicine and at the present time contribute materially to medical costs. As examples of these we may sight advances in cardiac, thoracic and brain surgery; modern rehabilitation techniques; discovery of effective means of immunization against polio, influenza, infectious hepatitis; new methods of study in heart disease; replacement transfusions in the newborn necessitated by Rh factor and more costly techniques in radiology and radiotherapy such as use of the cobalt bomb and radio isotopes.

A third factor necessitating increase in rates is the revision of the Schedule of Fees by The Medical Society of Nova Scotia, which came into effect on January 1st, 1958. At that time the population of the province generally appeared to approve of the increase in fees as exemplified by patient reaction and editorial comment, the previous increase having occurred in 1953. However, because of the "creeping utilization" of the prepaid services previously, the Corporation found it impossible to adopt the 1958 Schedule of Fees and have up to now continued paying the doctors on the 1953 Schedule; so you have the anomaly of the doctors charging private patients on the 1958 Schedule and being paid by the Corporation on the 1953 Schedule! I am sure you will agree that there is no other group rendering service to our community, be he in a "white collar" job or "labour" which would tolerate such a situation for so long.

So to sum up, the increase in subscribers' rates has been necessitated by:—

1. Increased utilization of services by subscriber, of whom the greatest demand comes from the "family" group as is quite natural,
2. Increased services,
3. Adoption of 1958 Schedule of Fees of The Medical Society of Nova Scotia.

The rates which we propose to charge subscribers even now will not be sufficient to pay our participating doctors full fee for their services. During the past, our General Practitioners have accepted eighty-five percent of the scheduled fees as full payment for their services and this pro-ration may be even further increased. We feel that the willingness of our participating physicians to accept this lesser fee from Maritime Medical Care subscribers is the best evidence we can give to prove that we are receiving the wholehearted support of the medical profession.

Maritime Medical Care is sponsored by The Medical Society of Nova Scotia. Although it is an independent organization our Board of Directors consists of medical men who are appointed from the Society and three "lay" members. The present relationship of Maritime Medical Care and its participating physicians will not be altered by our change in subscriber rates. A Specialist physician will still be permitted to bill the patient, in addition to the amount paid by Maritime Medical Care, if he so desires, but in our experience this privilege is being less and less used. The General physician, in certain instances where the subscriber is obviously demanding too much service, will be permitted to bill the subscriber in a limited manner. Other than this, the General physician must adhere to the Fee Schedule as stipulated by The Medical Society of Nova Scotia.

I would support your idea of providing our subscribers with a copy of the Annual Report of Maritime Medical Care. Although this idea has not yet been suggested to the Board, it certainly has a definite public relations value and the adoption of such procedure may be considered in future.

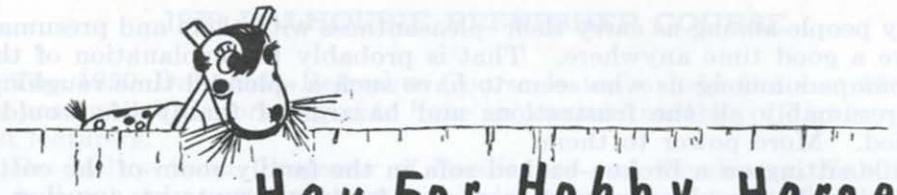
I can assure you that no other scheme can provide the same medical coverage at the same cost as Maritime Medical Care.

I do hope that this reply will clarify the points you raise in your letter, and that Maritime Medical Care may continue to secure you and your family in the future.

Yours very truly,

MARITIME MEDICAL CARE INCORPORATED.

"Contrary to a belief held in some quarters, social welfare is not the exclusive property of social workers. Social welfare is and must be the concern of us all. In this particular field there is no difference between the white collar worker, a member of the trade union, between labour and management—we are all individuals living in a community which we hope will be a good community for ourselves and our children. We must all accept our full responsibility towards determining the needs of the community. Having determined the needs our responsibility is to finance those needs." . . . . . .M. Wallace McCutcheon, Managing Director, Argus Corporation Limited, Toronto.



Hay For Hobby Horses

MIDSUMMER MADNESS:

Let us have a "go" at the matter of vacations. Webster gives the following; "vacation from the Latin vacatio, a being free from a duty, service, etc., also from vacare, to be empty; a respite or a time of respite; an intermission or rest; a period for rest and recreation; a holiday." Let us take the instance of an almost-young couple with three or four children under ten years of age who rent a summer cottage for the month of July. It rains at least half of the month so the children must be entertained indoors. The cottage is uncluttered by any of the amenities such as a refrigerator, indoor plumbing or a watertight roof. The children have not improved during the sixty mile trip from home. They take just as much care and fight just as much as they did at home. Father does not have to go to the office, but he is now stuck with the unfamiliar job of helping around the house and keeping the children entertained. With this type of vacation the possibility of rest or recreation is remote. The other typical holiday might be the same family on their way to Boston at a time of year when even Bostonians desert "Bean Town." They allow themselves to be boiled alive in the Stadium watching two lethargic ball clubs go through the motions, or suffocate in the dim recesses of Filene's basement with the other visitors from out of town.

It is my ill-tempered opinion that the modern vacation is a fraud foisted on the parents of the nation by the travel crowd in the same way that Father's day and Mother's day and other holidays are slipped over on us by greeting-card manufacturers and other clever gentlemen. The bread winners of the nation suffer inconvenience, financial attrition and periodic dislocation of their professional affairs in its name. The mothers of the nation allow themselves to be separated from their gleaming automatic kitchens and are committed to caring for the "mob" in the tent, leaky cottage or farmhouse miles from the milkman, T.V. repair man and their indispensable washers, dryers and frigidaires.

The delusion at the bottom of this nation-wide fraud is the popular fiction about "its good to get away" or "travel broadens." The damndest lie since the one about the apple is the one which says "Buy and Save." A close second is "a change is as good as a rest." For my money the smartest people I know are those who send the kids to Camp Kilcare or to grandma's on Cape Sable Island and travel—travel that is, to the patio in their own back yard. We have been sold a bill of goods by the travel crowd. The bitter truth is that you and I don't change one bit when we travel from Halifax to the fleshpots of Boston, Montreal or New York. We are the same anxious, short-tempered, hag-ridden types down there as we are here. The chief difference is that we pay ten times as much to be hag-ridden in New York as we do in Halifax. John Milton may have been blind, but he was clear enough the day he said: "The mind is its own place, and in itself can make a heaven of hell, a hell of heaven." (Milton: Paradise Lost (1677) Book I, Line 253). And if you happen to be a hell of a fellow, travelling won't improve it very much. Conversely however, the

heavenly people among us carry their pleasantness with them and presumably can have a good time anywhere. That is probably the explanation of these family campers among us who seem to have such a splendid time roughing it when presumably all the frustrations and hazards of family life would be multiplied. More power to them.

While sitting on a broken-backed sofa in the family room of the cottage at Buzzard's Bay and exhausted from my futile attempts at amusing the children, I mused on the origin of the vacation habit. The library at the cottage consists of a copy of Fox's Book of Martyrs, a dilapidated autobiography, "My Fifty Years as a Soldier," by Major-General Scabbard E. Frogge, late of her Majesty's Tenth Punjab Horse. A guest book whose last entry was May, 1940. "There is nothing in Buzzard's Bay except solitude"; and the Ladies Home Journal for the years 1930 to 1934. On such a slender store of reference works my findings on the origin of vacations are not complete, but for general enlightenment here they are.

The majority believe that "vacation" derives from vaccus, a cow, and Webster associates our words vacant and vacuous with this origin. One can imagine our forebearers taking their ease with their loaves of bread and jugs of wine under the shady trees in the ancient groves, passing long hours in bucolic somnolence. If this is the correct interpretation then great changes have been made in our modes of recreation since then.

A less well-known explanation for summertime madness called "vacations," is that vacation comes from an Anglo-Saxon word "vakks," to break down or beat up. The word is attached to a custom common among the Kentish Picts who, after a year of labour and accumulation would break down and use up their goods and energies in a short spasm of riotous living called the "bashh." In ancient times this was followed by a subdued period called the "hun-goffer," and an eight or nine month period of increased activity and fertility followed until the next "bashh" was due. Our current practise favours this theory of the origin of vacationing.

I was unable to pursue the historic aspects of this subject any further and have passed my collected papers over to Dr. J. W. Reid for further research.

Finally, I believe it would be a step toward the good life if the following hypotheses about our vacations were closely examined;

1. Many vacations are not periods of peace and quiet.
2. Vacations are not less work than work.
3. Children do not become transformed to carefree (needing no care), self-sustaining and self-entertaining children of nature upon exposure to sun, sand or water. They do not cease to fuss, fret, scream, natter and bicker just because you move them fifty or a hundred miles to a new locale.

Two weeks with children at the beach especially on a rainy day is an experience best typified by the following story. A young mother with three young children was waiting at a bus stop. She tried to entertain them with a story. "When Mummy was a little girl her Daddy took her to Africa. She wandered away from the camp one day and found herself in a swamp with a great big crocodile in front of her, a big lion behind her on the dry ground, and a large poisonous snake hanging from the tree above her." "What happened Mummy?" "Well boys, the lion pounced on Mummy and the crocodile jumped in too and the poisonous snake fell on her and stung her to death." "But Mummy, you're still alive." "Children, DO YOU CALL THIS LIVING?"

Yours regretfully, as summer passes,

BROTHER TIMOTHY.

1959 DALHOUSIE REFRESHER COURSE

The 1959 Dalhousie Refresher Course is to be held October 26th-29th. This year we will be privileged to have an unusual number of highly qualified guest lecturers.

Dr. Milton Senn, Professor of Paediatrics and Psychiatry, Yale University, will not only participate in the clinical program but will also deliver the John Stewart Lecture. Dr. Senn is an authority on Child Development, and the Refresher Course Committee is convinced that he will make an unique contribution during his stay in Halifax.

Our guest lecturer in Medicine will be Dr. E. J. Wayne, the Sims Commonwealth Travelling Professor, of Glasgow, Scotland. One of his topics will be "Recent Advances in Therapeutics.

In the field of Dermatology our guest will be Dr. R. Roy Forsey, Dermatologist and Syphilologist in Chief, and Chairman of the Department, Montreal General Hospital, and Assistant Professor, Department of Medicine and Clinical Medicine, McGill University.

The field of Surgery will be represented this year by Dr. Bentley P. Colcock of the Lahey Clinic, Boston, whose topics will be of interest to those who do General Surgery.

We will also have with us Dr. John D. Hamilton, Pathologist in Chief, Toronto General Hospital and Dr. J. Clifford Richardson, Assistant Professor of Medicine, University of Toronto, and Consultant Neurologist, Sunnybrook Hospital.

This year the Refresher Course Program will emphasize the clinical approach and it is felt that attendance will be particularly rewarding.

H. K. HALL,
Chairman,
Dalhousie Refresher Course Committee

American College of Surgeons Annual Clinical Congress

The 45th annual Clinical Congress of the American College of Surgeons will be held in Atlantic City, New Jersey, September 28 through October 2, 1959.

More than 10,000 Fellows of the College and guests from all over the world will gather to fulfil the purposes of this Congress: to discover, to inform and to learn. In keeping with the philosophy of the College that the qualified surgeon never stops extending his knowledge and skills, this meeting will present surgical developments through a wide variety of programs, including nine postgraduate courses, panel discussions, symposia, research reports, motion pictures, color closed-circuit telecasts from Bellevue Hospital in New York, nine clinics, and scientific and industrial exhibits.

Dr. Newell W. Philpott, Montreal, current President of the American College of Surgeons, will preside at the opening evening session, at which Dr. Dean Rusk, President, The Rockefeller Foundation, will speak.

Other major addresses will be made by Dr. Owen H. Wangensteen, Minneapolis, incoming president of the College, Dr. R. Arnold Griswold, Louisville, speaking on abdominal injuries, and Dr. David Paton Cuthbertson, Bucksburn, Scotland, speaking in the field of parenteral fluid therapy.

SISTER ROSE ANGELA
AN APPRECIATION

It is an honorable task to express words of appreciation for the life, work and example of the Late Sister Rose Angela.

She was born of Scottish parentage in Antigonish Co., N. S., entered the Sisters of Charity in September, 1918 and served her novitiate at Mount St. Vincent, Rockingham. In 1924, she entered training in the Hamilton Hospital's Nursing School, graduating in 1927. She worked for twenty years in the Halifax Infirmary, most of the time in the Operating Room. In 1947 she returned to the Hamilton Hospital as Supervisor of the Operating Room, later moving to the new hospital, St. Elizabeth, in the same capacity.

The late Sister Rose Angela was a most remarkable person, a woman of great dignity and character, kind and gentle, understanding and tactful. She executed her duties with the utmost efficiency, yet with ease and simplicity. She inspired confidence in patients, nurses and doctors by courtesy and modesty. She was always the same saintly Sister, unruffled by any circumstance.

We mourn her loss, but are grateful for the work she did so well and for the noble life she lived. The fine prose written in the seventeenth Century by John Bunyan in Pilgrim's Progress, depicting the death of Mr. Valiant-for-Truth, expresses our feelings.

"Mr. Valiant-for-Truth was taken with a summons then said he, 'I am going to my Father's, yet now I do not repent me of all the troubles I have been at to arrive where I am. My sword I give to him that shall succeed me in my pilgrimage, and my courage and skill to him that can get it. My marks and scars I carry with me, to be a witness for me that I have fought His battles, who now will be my rewarder. So he passed over and the trumpets sounded for him on the other side."

Sister Rose Angela worked hard and long to arrive where she is. To the ones that follow, she has left her sword forged from an alloy of Charm, Dignity, Modesty, Understanding, Kindness and other Metals that will stand the test of time. Her courage and skill some will get.

Her marks and scars from diligent toil are many—

So she passed over—and all the Trumpets sounded for her on the other side.

K. A. FRASER

INFECTIOUS DISEASES—NOVA SCOTIA
Reported Summary for the Month of May, 1959

Diseases	NOVA SCOTIA 1959 C	NOVA SCOTIA 1959 D	NOVA SCOTIA 1958 C	NOVA SCOTIA 1958 D	CANADA 1959 C	CANADA 1958 C
Brucellosis (Undulant fever) (044)	0	0	0	0	16	0
Diarrhoea of newborn, epidemic (764)	2	0	0	0	6	0
Diphtheria (055)	0	0	0	0	2	8
Dysentery:						
(a) Amoebic (046)	0	0	0	0	0	0
(b) Bacillary (045)	0	0	0	0	57	0
(c) Unspecified (048)	0	0	0	0	8	0
Encephalitis, infectious (082.0)	1	0	0	0	7	2
Food Poisoning:						
(a) Staphylococcus intoxication (049.0)	0	0	0	0	0	0
(b) Salmonella infections (042.1)	0	0	0	0	0	0
(c) Unspecified (049.2)	0	0	0	0	35	0
Hepatitis, infectious (including serum hepatitis) (092, N998.5)	20	0	132	0	387	0
Meningitis, viral or aseptic (080.2, 082.1)						
(a) due to polio virus	0	0	0	0	0	0
(b) due to Coxsackie virus	0	0	0	0	0	0
(c) due to ECHO virus	0	0	0	0	0	0
(d) other and unspecified	0	0	0	0	5	0
Meningococcal infections (057)	0	0	0	0	19	25
Pemphigus neonatorum (impetigo of the newborn) (766)	0	0	0	0	0	0
Pertussis (Whooping Cough) (056)	9	0	34	0	568	570
Poliomyelitis, paralytic (080.0, 080.1)	0	0	0	0	6	6
Scarlet Fever & Streptococcal Sore Throat (050, 051)	108	0	126	0	2422	807
Tuberculosis						
(a) Pulmonary (001, 002)	22	2	23	3	546	548
(b) Other and unspecified (003-019)	4	0	0	0	122	70
Typhoid and Paratyphoid Fever (040, 041)	0	0	0	0	97	32
Venereal diseases						
(a) Gonorrhoea —						
Ophthalmia neonatorum (033)	0	0	0	0	0	0
All other forms (030-032, 034)	33	0	27	0	1175	1080
(b) Syphilis —						
Acquired—ordinary (021.0, 021.1)	0	0	0	0	0	0
— secondary (021.2, 021.3)	0	0	0	0	0	0
— latent (028)	2	0	0	0	0	0
— tertiary — cardiovascular (023)	0	0	0	0	0	0
— „ — neurosyphilis (024, 026)	0	0	0	0	0	0
— „ — other (027)	0	0	0	0	0	0
Prenatal—congenital (020)	0	0	0	0	0	0
Other and unspecified (029)	3	0	2*	0	204*	178*
(c) Chancroid (036)	0	0	0	0	0	0
(d) Granuloma inguinale (038)	0	0	0	0	0	0
(e) Lymphogranuloma venereum (037)	0	0	0	0	0	0
Rare Diseases:						
Anthrax (062)	0	0	0	0	0	0
Botulism (049.1)	0	0	0	0	0	0
Cholera (043)	0	0	0	0	0	0
Leprosy (060)	0	0	0	0	0	0
Malaria (110-117)	0	0	0	0	0	0
Plague (058)	0	0	0	0	0	0
Psittacosis & ornithosis (096.2)	0	0	0	0	0	0
Rabies in Man (094)	0	0	0	0	0	0
Relapsing fever, louse-borne (071.0)	0	0	0	0	0	0
Rickettsial infections:						
(a) Typhus, louse-borne (100)	0	0	0	0	0	0
(b) Rocky Mountain spotted fever (104 part)	0	0	0	0	0	0
(c) Q-Fever (108 part)	0	0	0	0	0	0
(d) Other & unspecified (101-108)	0	0	0	0	0	0
Smallpox (084)	0	0	0	0	0	0
Tetanus (061)	0	0	0	0	0	0
Trichinosis (128)	0	0	0	0	0	0
Tularaemia (059)	0	0	0	0	0	0
Yellow Fever (091)	0	0	0	0	0	0
Pneumonococcal Meningitis	0	0	0	0	0	0
N.S.U.	1	0	0	0	0	0

C — Cases D — Deaths

*Not broken down

Polyvalent Influenza Vaccine

With the kind co-operation of Dr. A. R. Morton and the staffs of the various City institutions and of the staffs of the Halifax Children's Hospital, the Victoria General Hospital and the Halifax Infirmary, the Division of Communicable Diseases' Control, Provincial Department of Public Health, was able to obtain information concerning the efficacy of polyvalent vaccine in the prevention of influenza. The survey was conducted on the nursing staffs only of the above institutions, since it was felt that this group was the one most likely to produce accurate information.

Of 389 nurses surveyed, it was found that 356 had received polyvalent influenza vaccine during the fall of 1958 and 33 had not received any vaccine. Of the 356 who received the vaccine 83 (23.3%) stated that they had an attack of influenza during the season when influenza was epidemic in this area, i.e., during March and April of 1959. Of the 33 who did not receive the vaccine 18 (54.5%) stated that they had an attack of influenza during the same period. When we apply the usual tests of significance to these figures, we find that, from a statistical point of view, the difference is highly significant in favour of the vaccine, i.e., the possibility of this difference being due to chance alone is extremely small. It is realized, of course, that factors other than the vaccine could possibly have contributed to this difference since this was not a well-controlled study. It is felt, however, that the results of this survey are of some value and give support to the view which is widely held that polyvalent influenza vaccine is a very useful agent in the prevention of morbidity caused by the different influenza viruses which it contains.

INFLUENZA DEATHS

Since the Province of Nova Scotia has just come through an epidemic of influenza and since there has been a great deal of publicity in recent months regarding the seriousness of this epidemic, it was thought worthwhile to look back over the records to see if there was, in fact, any thing to indicate that the epidemic of 1959 was very unusual. Since mortality is one indication of the seriousness of a disease, the Death Records for the Province were examined for the calendar years 1950 to the end of June, 1959. It was found that the number of influenza deaths for these years varied between a low of 20 to a high of 223 which occurred in 1951. To the end of June, 1959, there were 92 influenza deaths. During this same period for the years 1950 to 1958, the number varied between 14 and 212 and again the 212 occurred in the year 1951. In all these years except 1957 most of the deaths occurred in the months of February, March and April. In 1957, the Asian Influenza year, the peak occurred in the months of October and November. As for as deaths are concerned it does not appear that there was anything very unusual about the epidemic of 1959. It could well be that the slightly higher number of deaths from influenza in 1959 is partly attributed to changing attitude on the part of physicians to the proper recording of causes of death in recent years.

The influenza deaths were also examined for age groups in which they occurred. In each year, 1950 to 1959, it was found that about half the deaths occurred in the age group 70 and over and about one quarter occurred in the age group under one year.

Since it has so often been reported that influenza cases are complicated by pneumonia, it was felt that it might be interesting to examine the pneumonia deaths also. From 1950 to 1959, the pneumonia deaths were as follows:

1950	1951	1952	1953	1954	1955	1956	1957	1958	1959
249	220	200	179	180	205	193	219	269	154

Unless something very unusual happens, there is no reason to believe that the pneumonia deaths in 1959 will be any higher than in the previous years. In all years about half the pneumonia deaths have been in infants (less than 1 year old). The second highest incidence was in the 70 and over age group and the third highest incidence was in the one-to-four year age groups. From the figures available to date, therefore, we have no reason to believe that the influenza epidemic of 1959 caused any more deaths either directly or indirectly than did influenza in the past eight years.

HOSPITAL INSURANCE INSTITUTE

The N. S. Hospital Insurance Commission is planning an Institute on September 22 and 23 in Halifax.

Recognizing the position of the medical staff in the maintenance of standards of care in the hospital, and the emergence of certain problems in this field, the Commission is requesting that the Chairman of the Medical Sub-Committee of the Standards Committee of each hospital attend the Institute. If the Chairman cannot attend, another member of the Sub-Committee may be delegated in his place.

It has not always been easy for the busy practitioner to study the details of the Hospital Insurance Plan, and familiarize himself with all its ramifications. Nor has he always been able to assess adequately his own relation to the plan. During the course of this Institute, both in general sessions and in those specially designed for discussion of medical problems, representatives of the medical profession will have an opportunity to get the answers to many questions that have been plaguing them, and to discuss with their colleagues and representatives of the Commission the problems of medical care in their hospitals.