VOL. XL No. 1

JANUARY 1961

The NOVA SCOTIA MEDICAL BULLETIN

EDITORIAL

THE BEST OF TIMES AND THE WORST OF TIMES

From time to time one is asked by promising youngsters about Medicine as a career. Because it has become fashionable to stress to them certain worrying features of the course medical practice may take in future, it is not surprising that some are discouraged. They are briefed on the malevolent machinations of all political parties and told how these are aided in many cases by a fifth column Health Department bureaucracy. The inescapable inference is that this is not a good time to become a doctor.

The fact is, however, that there never was a good time to become a doctor. It was not a good time in the Middle Ages to fight plague with nothing but courage and a few useless simples. Nor was it a good time in the 19th Century to face a lifetime of watching children die from diphtheria. Nor as a surgeon of little more than a hundred years ago to operate without anaesthesia and see half of one's patients die of infection. Coming closer to our own time, few of us would be happy at the prospect of fighting Spanish Influenza with aspirin, poultices and despair. The men who did these things were not influenced by circumstances, good or bad, but did what they had to do, as best they could, because they were doctors.

We would be simple to believe that doctors in all ages have not shared the frailties and wickedness of all mankind in full measure. At the same time there is a common denominator which the doctor of to-day shares with all the doctors who went before him and who will come after him. It is an infelt need, a kind of compassion perhaps, which compels one kind of man to help all other men. This need will manifest itself whether he is ruled by the Pharaohs or the C.C.F. and will outlive both.

To-day, the newest doctor can conquer diseases that twenty years ago were beyond the skill of the greatest physician in the land. Doors locked since the world was born are opening to him on every side. And at the end neither principalities nor powers nor interfering Socialist States nor all Hell can separate a doctor from the patient who needs him.

At last year's Interne graduation one of the speakers remarked to the effect that he had believed that he envied no man on earth but he found that he envied every new doctor standing before him. Few would disagree.

The worst of times? Perhaps. The best of times? Yes.

W.E.P.

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SYMPOSIUM ON OEDEMA

PART VI: NUTRITIONAL OEDEMA*

R. M. Read, M.D.

Nutritional oedema is defined as oedema which has been observed to occur in people suffering from starvation, malnutrition, or undernutrition. These latter terms have themselves been defined in the form of certain arbitrary standards set up in several studies to investigate the effect of food deprivation in humans. (Table 1).

In the Carnegie experiment[1], moderate calorie deficiency was produced in twelve healthy subjects by reducing their normal calorie intake from 3100 to 1700 calories per day on a diet normal in other respects. In five weeks there was a weight loss of 10 per cent with the occurrence of such symptoms as easy fatiguability, diminished libido, diminished resistance to illness, listlessness, depression, but no oedema.

In the Minnesota experiment[2], severe calorie deficiency or semi-starvation was produced by Keys in thirty-two healthy young adults by reducing their normal average intake of 3500 to 1570 calories per day. The diet contained 50 grams of protein, 40 grams of fat, and 300 grams of carbohydrate, with a normal requirement of fluid, vitamins and minerals. There was a 25 per cent loss in weight. Oedema was common after the twelfth week, especially about the ankles, knees and face. It was associated with a fall in the plasma proteins and osmotic pressure but it was not regularly and proportionately related to the extent of these plasma changes. The hemoglobin dropped from 15 to 11.7 grams per cent, serum albumin from 4.3 to 3.9 grams per cent, and B.M.R. to -40 per cent. There was no change in the arterial blood pressure, but venous pressure decreased. The total plasma volume was greatly increased relative to the reduced total body weight; i.e., there was a relative excess of body fluid. In spite of the oedema, polyuria was often a marked symptom associated with a correspondingly high fluid intake. Subjective findings in this severe calorie deficiency included cold intolerance, slow healing, desire for hot foods, postural hypo-tension, inabilty to focus the eyes, slight increase in auditory acuity, muscle cramps, paresthesias, weakness, impaired co-ordination, loss of ambition and libido, and general lethargy. Hunger progressively increased.

Various studies have been made on undernutrition due to famine, malnutrition and man's inhumanity to man. The chief and most detailed studies having been made on newly released prisoners of war at the end of World War II.[2,3] Other studies have been made on special types of undernutrition which can lead to oedema such as kwashiorkor, chronic alcoholism (cirrhosis), scurvy, and beri-beri.

Kwashiorkor[4,6] is a tropical deficiency disease resulting from protein deficiency in the presence of adequate calorie intake. As in other deficiency diseases avitaminosis also plays a major role, in this case deficiency of choline and hence the choline-containing lipids (lecithin, sphygmomyelin) essential to removing *from* the liver the excessive amounts of fat that are transported, in starvation, *to* the liver from the body fat depots. The mechanism of alcoholic fatty liver with resulting cirrhosis is considered to be, chiefly, a similar choline deficiency. In kwashiorkor, the fatty liver and other manifestations

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of protein and choline deficiency result in steatorrhoea, macrocytic anemia, ascites, and oedema. Both beri-beri and scurvy produce oedema, as will be discussed in more detail later, chiefly by means of increased capillary fragility.

Complete starvation,[1] save for intake of small amounts of water, results first in rapid depletion of the liver's glycogen stores, which amount to only several hundred grams. Next, the neutral fat of adipose tissues is mobilized and taken to the liver, conserving until the end the complex intracellular lipids. Next, the body's pool of amino acids is kept replenished by selective breakdown of body proteins of various organs, the least essential organs such as spleen, lymphoid tissue and muscles being depleted of protein most rapidly, the hormones and enzymes having first call and normal blood sugar maintenance by deamination having second call. As the body's proteins diminish, a protein deficiency syndrome develops, including liver damage, increased susceptibility to infection and shock, anemia, osteoporosis, diminished serum proteins, and finally oedema. The last, as mentioned by Sodeman,[4] has always been considered the chief sign of protein deficiency. Starvation leads to impaired function of all organs including increased capillary fragility and impaired renal function. Death usually occurs after a 50 per cent loss in body weight.

These general facts have been presented as a preamble to the more specific subjects of nutritional oedema and its pathophysiology in order to illustrate the multiplicity of adaptive physiological mechanisms brought into play by undernutrition, many of which do or could play a part in the production of nutritional oedema.

The Causes of Nutritional Oedema.

The original theory regarding the cause of nutritional oedema, postulated on the basis of the Starling principle, was that hypoproteinemia, induced by protein loss, led to oedema when the total serum protein fell below about 5 grams per cent. When the serum albumin, the important oncotic molecule, falls below a value variously estimated by different authors at anywhere from 1.6 gm. per cent[7] to 3.8 gram per cent[2], the result is a decrease in the serum colloidal osmotic pressure appreciably below the normal 23 mm. Hg.

However, it has been found that in malnutrition, plasma protein concentration may be normal in the presence of oedema, low in the absence of oedema, or very low in presence of moderate oedema, without demonstrable pressure changes between the tissues and the circulating blood (Sodeman)[4]. In cases of nutritional oedema found among British internees freed from German prison camps at the end of the last war[3], serum albumin in cases with oedema was 2.6 to 3.8 gm. per cent and total proteins averaged 4.9. In individual cases, however, the plasma protein or serum albumin levels were not precisely related to the presence or the degree of oedema. In other words, no critical plasma osmotic pressure level was found to be associated with oedema.[1] Hence, causes of nutritional oedema other than mere hypoproteinemia must be looked for. In malnutrition and starvation there is not merely the usual finding of hypoproteinemia and/or oedema, but, because of general depletion of all the usual nutritional factors such as carbohydrates, fat, proteins, vitamins and minerals, there is a widespread disruption of metabolism with numerous clinical findings, and numerous possible facets to the pathogenesis of nutritional oedema.

The Starling Factors and Perpetuating Factors.

(See diagrams 1 and 2 and table 2)

The Starling principle that exchange of fluids between blood and interstitial fluid depends upon the balance between osmotic and hydrostatic pressure, leads logically to the following local causes of oedema, listed below with the chief clinical types of oedema primarily caused by each. Starling factor 1 increases hydrostatic pressure whereas factors 2 and 3 diminish osmotic pressure. Many observations already discussed by previous authors in this journal indicated that there are other non-local perpetuating factors, hormonal and renal, which must be present in order for oedema to occur.

To see how all these various factors come into play in the production of nutritional oedema we will use a diagrammatic representation similar to the one used by Sheila Sherlock[8] to describe the physiology of the ascites of hepatic cirrhosis.

It can be seen that of the Starling factors, nutritional oedema may be caused by (a) hypoalbuminemia, (long thought to be the only cause) and (b) increased capillary fragility, but not by increased venous pressure, lymphatic obstruction or increased local protein breakdown. As noted in the Minnesota experiment (above) venous pressure actually is diminished.[2]

Diagram I

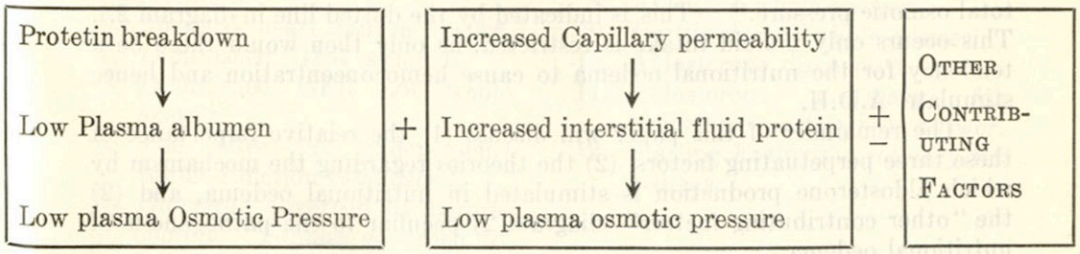
The Starling Forces

	Hydrostatic Pressure		Osmotic Pressure
Venous End	12 mm. Hg.	→	← 23 mm. Hg.
Arteriolar End	32 mm. Hg.	→	← 23 mm. Hg.
Artery	100 mm. Hg.		

Diagram 2

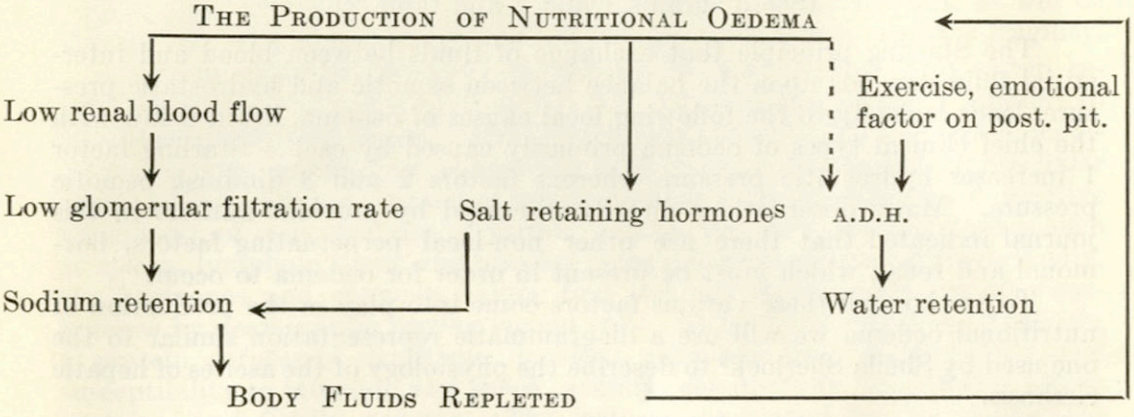
Part 1

The Effects of Undernutrition (Starvation, Malnutrition)



Not increased venous pressure or lymphatic obstruction.

Part 2



PERPETUATING FACTORS:

1. Low glomerular filtration rate. 2. Aldosterone etc.
3. Anti-diuretic hormone.

The lower half of diagram 2 indicated that renal and humoral *perpetuating factors* are involved. These include (a) decreased glomeruler filtration rate (G.F.R.), (b) increased aldosterone and other salt retaining hormones, and (c) increased anti-diuretic hormone (A.D.H.). The low renal blood flow is increased by the tendency of the nutritional oedema and its causative (Starling) factors to lower blood pressure. The mechanism of the stimulation of production of aldosterone and other salt retaining hormones will be dealt with later. The increased production of A.D.H. in undernutrition has been explained and verified by several interesting experiments,[9] in denervated dog kidneys, which duplicated the antidiuretic effect of A.D.H. injection by exercise, anger, fear or almost any noxious stimulation. These effects being prevented proportionately as posterior pituitary tissue was removed. The stress of starvation produces an emotional tone similar to that produced by exercise, anger, fear, etc., and hence probably stimulates the posterior pituitary directly via nervous pathways to produce more A.D.H. The fact that rest diminishes nutritional oedema, without changing serum albumin, may be due to lessened A.D.H. production. A part is played in nutritional oedema by a a more widely known effect of A.D.H. stimulation, namely increased plasma total osmotic pressure.[10] (This is indicated by the dotted line in diagram 2.). This occurs only if fluid intake is restricted, as only then would there be a tendency for the nutritional oedema to cause hemoconcentration and hence stimulate A.D.H.

The remainder of this paper will discuss (1) the relative importance of these three perpetuating factors, (2) the theories regarding the mechanism by which aldosterone production is stimulated in nutritional oedema, and (2) the "other contributing factors" (diagram 2) peculiar to the pathogenesis of nutritional oedema.

TABLE 1

The Effects of Undernutrition (including Starvation and Malnutrition)

1. CARNEGIE EXPERIMENT: Moderate Calorie Deficiency (3100 to 1700) no oedema, 10% weight loss.
2. MINNESOTA EXPERIMENT: Severe Calorie Deficiency (3500 to 1570) oedema in 12 weeks, 25% weight loss.
3. NATURAL UNDERNUTRITION STUDIES
   Wars, Kwashiorkor (choline deficiency), etc.
4. COMPLETE STARVATION results in loss of carbohydrates, then fats and proteins, finally essential proteins. This is followed by the protein deficiency syndrome, includes oedema.

TABLE 2

THE STARLING FACTORS:

1. INCREASE in venous pressure leads to cardiac and cirrhotic oedema.
2. DECREASE in serum protein levels leads to nephrotic and nutritional oedema.
3. INCREASE in intestitial fluid protein caused by
   (a) INCREASE in capillary permeability leads to nephrotic, nutritional, allergic, inflammatory, traumatic oedema.
   (b) INCREASE in lymphatic pressure or obstruction leads to inflammatory oedema.
   (c) INCREASE in protein breakdown leads to traumatic or inflammatory oedema.
4. Local tissue tension and gravity determines distribution.
   Hormonal-renal factors determine Na retention.

TABLE 3

THE HISTORY OF OUR KNOWLEDGE OF THE RELATIVE IMPORTANCE OF PERPETUATING FACTORS IN PRODUCING OEDEMA

1. Oedema dependent upon Na retention.
2. Forward heart failure theory.
3. Diminished GFR studied.
4. Diminished GFR not whole answer.
5. The effect of independent tubule function established.
6. Glomeruli governed by baroreceptors. Tubules governed by by humoral agents.
7. ADH.
8. ADH not whole answer.
9. Corticoids not whole answer.
10. Aldosterone discovered.
11. Aldosterone necessary for oedema; close to whole answer.
12. Starling Factor(s) necessary for oedema.

TABLE 4

THE MECHANISMS OF ALDOSTERONE STIMULATION

1. Diminished volume of fluid spaces (Liddle)
2. Diminished cardiac output.
3. Hyponatremia
4. Hyperkalemia (Laragh)
5. Impaired hepatic degradation of aldosterone
6. Stress

TABLE 5

OTHER CONTRIBUTING FACTORS IN OEDEMA PRODUCTION

1. Fatty liver → ascites → increased aldosterone production
   ↓ ? impaired aldosterone degradation → Oedema
2. Stress reaction → cortical hormones
   ↓ ? increased aldosterone production
3. Emotional stimuli acting on posterior pituitary → ADH production
4. Beri-beri → increased capillary permeability.
5. Scurvy → increased capillary permeability.
6. Protein depletion → increased capillary permeability.
7. Sodium depletion → increased aldosterone stimulation by any potassium intake.

TABLE 6

SUMMARY OF FACTORS PRODUCING OEDEMA

STARLING FACTORS
at least one necessary

PERPETUATING FACTORS
aldosterone necessary

OTHER CONTRIBUTING FACTORS

The Relative Importance of Perpetuating Factors in Producing Oedema (Table 3)

In discussing the relative importance of the various perpetuating factors, we will list and assess very briefly the chief steps in the history of their discovery,[11]. This provides a fairly complete understanding of the forces that perpetuate the various forms of oedema, (including nutritional) once they are established by Starling factors.

(1) The discovery that oedema formation is directly dependent on sodium retention and intake (Starling and Widal, 1900-1910)[12,13]

(2) The forward heart failure theory (Warren and Stead, 1940-50)[14] was the next advance.

(3) This led to the conclusion that diminished glomerular filtration rate is a major factor in other types of oedema as well as cardiac. Thompson and Pitts [15], by placing balloons in the aorta of dogs, showed that normally there is a linear relationship between G.F.R. and sodium excretion. Merrill et al[16] showed that 70 to 80 cc./min. is the critical G.F.R. level above which sodium retention, and hence cardiac oedema, are unlikely to occur.

(4) That diminished G.F.R. was not the whole answer in the production of oedema became apparent when the investigations by Larogh[11] showed sodium excretion does not have a linear relationship to the G.F.R. Experimentally G.F.R. is low in some renal diseases without oedema; nephrotics, cardiacs, and experimental dogs can all have oedema and cirrhotics can have ascites, with normal or increased G.F.R.

(5) Other experiments[17,18] showed that the renal tubules function independently of glomeruli and are a second discrete regulator of sodium and water balance.

(6) Both glomeruli and tubules function in the denervated kidney. The glomeruli are governed by baroreceptors. The tubules are affected, by humoral agents, including A.D.H., aldosterone, hydrocortisone corticosterone, desoxycorticosterone, estrogen, nor-adrenalin and several drugs including the diuretics.

(7) A.D.H. was discovered, its mechanism of action established and later its urine excretion levels were found to correlate well with clinical oedema.[19] The stimulation for release of A.D.H. is emotion, exercise and starvation[9], as well as increased plasma total osmotic pressure.[10]

(8) The levels of A.D.H. does not explain the perpetuation of oedema as a dog, with diabetes insipidus, may get oedema with sodium retention.[20]

(9) Corticoids (known before 1950) are not the whole answer because they are not potent enough and without their presence various forms of oedema with sodium retention can still occur.

(10) Aldosterone was discovered by Leutcher in 1950[21] by inference when he observed that, following the ingestion of the urine from patients with oedema, sodium retention occurred in adrenalectemized rats. In a series of papers, he reported consistently increased salt-retaining activity in the urine of patients with oedema, nephrosis, cirrhosis or cardiac failure. The structure of aldosterone was subsequently demonstrated by Simpson and Tait. Aldosterone has been isolated from the outer shell of the adrenal gland and has been demonstrated to be the most powerful of the sodium-retaining hormones.

(11) Aldosterone has since been established as the most important single factor in the perpetuation of oedema. Stahl and Davis[22] and Howell[23] performed experiments which indicated the necessity of the adrenal cortex being present in order to have oedema. It was also shown that aldosterone was much more important than either diminished G.F.R. or A.D.H. in perpetuation of oedema. The amount of aldosterone in urine correlates well with oedema. Normal 24 hr. urine excretion of aldosterone is 1-4 mcgm., and it is increased to 6-20 mcgm. by a low sodium diet and to as high as 600 mcgm. in oedema, including nutritional oedema.[11]

While causing oedema, aldosterone is making a valuable contribution to the body's economy by preventing the severe hyponatremia and peripheral vascular collapse which would result from the Starling factors if sodium retention did not occur. In other words, the chief function of aldosterone is to help maintain blood pressure by retaining sodium and hence maintaining blood volume. In so doing it perpetuates oedema when Starling factors are present.

(12) Because primary aldosteronism does not cause oedema[24], it is reasoned that at least one Starling factor is necessary for oedema.

Hence, in order for nutritional oedema or any other type of oedema to occur, excluding localized forms like allergic or pulmonary oedema, two factors must always be present—aldosterone plus at least one Starling factor.

THE MECHANISMS OF ALDOSTERONE STIMULATION (Table 4)

The following are the theories regarding the mechanisms by which increased blood levels of aldosterone are produced in various forms of oedema, particularly nutritional oedema. (Parenthetically it should be noted that ACTH has been proven *not* to stimulate production of this particular adrenal cortical hormone.)

(1) DIMINISHED VOLUME IN THE FLUID SPACES. Liddle et al[25] noted the inverse relationship between aldosterone secretion and the degree of hydration. No specific volume receptor has been recognized within the circulation and it is not clear whether the sensitive volume involved is the total body water, the blood volume or the extracellular volume.

(2) A second elaborate, but as yet untested, theory is that a tendency toward *diminished cardiac output*, as caused directly in cardiac oedema, and by the Starling factors in nutritional and other forms of oedema, stimulates some flow-volume receptors in the carotid area which in turn relay a message by a neuro-humoral pathway to the aldosterone-secreting cells of the adrenal cortex. Experimentation may eventually show this and the first theory to be the same.

(3) HYPONATREMIA is the logical stimulant of aldosterone production because the action of aldosterone is to conserve sodium, and a low sodium diet increases urinary aldosterone in normal subjects. However, no correlation was found in experiments in a large number of dogs between serum sodium concentration and aldosterone excretion[11] although a low sodium diet stimulates aldosterone production without lowering serum sodium.[21] Also, while sodium administration reduces urinary aldosterone in oedematous subjects, this theory, alone, does not explain many observed phenomena.

(4) HYPERKALEMIA: That a high serum potassium level is the important factor in increasing blood levels of aldosterone is considerably more in keeping with experimental findings: A low sodium diet, while not lowering serum

sodium does elevate serum potassium (and urinary aldosterone)[26] and makes the serum potassium level (*and urine aldosterone*) extremely *sensitive* to any potassium ingestion[27,28] Also, in a large number of dogs a definite direct relationship was found between serum potassium levels and urinary aldosterone excretion.

(5) Impaired hepatic degradation of aldosterone, rather than an increased production by the adrenal cortex, may be important in determining aldosterone levels. In experiments various forms of oedema, particularly ascites, were produced by artificially obstructing the hepatic veins with resulting hepatic engorgement and concomitant increased aldosterone in the urine. This theory sounds very reasonable until one notes that there is liver congestion or other impairment in some, not all, of the clinical conditions in which aldosteronuria occurs.

(6) Increased aldosterone production, along with other adrenal corticoids, occurs as a part of the "stress syndrome." However, urinary aldosterone correlates poorly with stress as the amount of aldosterone stimulated in this way, if any, is small.

Of the six theories just described the two most compatible with observed experimental findings to date are the first and the fourth, diminished volume of the fluid spaces and hyperkalemia. The potassium ion is intimately related to the state of intracellular hydration and changes in volume may be associated, not only with a change in pressure, but also with significant shifts of electrolyte and water which could escape detection.

Whichever of the six theories is true, it remains to determine definitely whether a hormone exists, which might be called "ACTHa" (adrenocorticotrophic hormone leading to production of aldosterone). Alternately the cortical stimulation may occur as a result of the action of one or more of the mechanisms described above, working through osmotic or electrolyte changes on the cortex. Organs suggested as the possible source of "ACTHa" include the anterior pituitary, the diencephalon and the renal juxta-glomerular cells. These last become granulated and could secrete a hormone during low dietary sodium intake and in adrenalectomized animals.

The manner in which aldosterone, once produced, acts on the tubules to cause sodium retention is discussed fully in other papers in this series on oedema.

In undernutrition sufficient to produce oedema, conditions in the body are such as to make the increased aldosterone of nutritional oedema compatible with any of the above theories. The Starling factors bring about a tendency toward diminished cardiac output, hyperkalemia and hyponatremia, and the undernutrition itself brings about diminished volume in fluid spaces, liver impairment and stress.

Other Contributing Factors (Table 5.)

Finally, one must comment on some of the other contributing factors involved in the production of nutritional oedema. These physiological aberrations, which occur particularly in the special forms of undernutrition, actually contribute to the production of nutritional oedema by means of their effects on the Starling and perpetuating factors.

(1) The fatty or cirrhotic liver of kwashiorkor, chronic alcoholism, severe caloric deficiency and complete starvation can lead to increased aldosterone level if the impaired hepatic degradation is important. This can lead to cirrhotic oedema and ascites with increased aldosterone as described in a previous paper in this series.[31]

(2) The stress reaction in undernutrition may contribute to oedema through an increased output of mineralocorticoids[32], and also of aldosterone. (A hypothesis noted above).
(3) The emotional stress factor of undernutrition may promote ADH production by direct action on the posterior pituitary.
(4) Beri-beri increases capillary permeability.
(5) Scurvy increases capillary permeability.
(6) Starvation resulting in depletion of the cells of the capillary walls, particularly of protein, is the main factor leading to increased capillary permeability.
(7) Sodium depletion makes aldosterone levels extremely sensitive to intake or any small amounts of potassium.

CONCLUSION. (Table 6.)

The chief conclusion of this paper is that nutritional oedema requires two factors, increased aldosterone production and at least one of the Starling factors. In discussing the physiology of nutritional oedema, and as a matter of fact of any other type of oedema, the causative factors can be grouped as Starling factors, perpetuating factors, and other contributing factors peculiar to that particular type of oedema. This is for discussion and organizational purposes only for in the living body the inter-relationship of all these factors is extremely fluid, figuratively as well as literally.

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THE PLACE OF CORTICOTROPIN IN THE TREATMENT OF MYASTHENIA GRAVIS. Freydberg, L. D., Ann. Int. Med. 52:806, (Apr.) 1960.

The use of corticotropin for treatment of myasthenia gravis has been largely neglected for the last seven years, because it is considered to be dangerous. Yet there seems no great reluctance in selected cases to try thymectomy, a procedure which is not entirely without danger. It is the author's conviction that the use of corticotropin is dangerous chiefly for those patients who already have prominent bulbar symptoms. Most patients with myasthenia gravis are troubled mainly by their eye, face or extremity symptoms. In these, the careful use of corticotropin should present a minimum of danger. For those who have difficulty in swallowing or breathing, the outlook is already grave. If other methods of treatment do not bring a remission in these cases, it would seem that a calculated risk may be worth while.

The mode of action of corticotropin in this syndrome is not known. The most likely theory is that the action is connected with the well documented lysing effect of corticotropin on lymphoid tissues. The pathologic source of the presumed myoneural juncture toxin in myasthenia gravis is also not known, but there is reason to suspect the thymus in some, the muscle lymphorrhages in others.

In this study, 5 new cases of myasthenia gravis treated with short courses of corticotropin are reported. Four of the five experienced a beneficial response, some of them repeatedly after each course. One case, previously reported, has been re-treated eight more times with a worth while remission every time.

It is suggested that a re-evaluation of corticotropin therapy be made in selected cases, chiefly those without prominent bulbar weakness. It is important that careful precautions be used.

S. J. S.

THE MANAGEMENT OF THE SMALL BIOPSY

C. D. CHIPMAN, M.D., F.R.C.P.(C)*

In recent years the scope of pathology has been expanded to include the examination of small tissue specimens obtained by needle or aspiration biopsy. In the past, information about the histological changes in tissues in disease was gained from the study of post mortem specimens; now because of recent advances in biopsy technique, and the acceptance of the diagnostic biopsy by the medical profession, the science of pathology includes the histological investigation of the disease process in the living patient. The traditional morphological methods of pathology are used in the examination of needle and aspiration biopsies and in addition, cytological, bacteriological and biochemical techniques may be required in certain cases. Tissues are frequently taken from patients on the Medical services as one part of the investigation of the patient's illness. This sort of study is different from the routine survey for the presence or absence of malignancy in tissue removed at operation; it is really a specialized branch of surgical pathology, a study of the pathology of systemic diseases applied to the living patient to establish diagnosis or control treatment. Obtaining such specimens of tissue is limited only by the slight risk of complications which may follow needle puncture or other minor surgical procedures.

Recently we have noticed an increasing number of these examinations in the Pathology Institute. The tissues come from various sites. Pleural biopsies are useful in discovering the cause of a large pleural effusion when other methods have proved inconclusive. Biopsies of gastric mucosa may provide useful information in cases of gastritis and in some large centers biopsies of intestinal mucosa, taken through tubes, have been used to study the histological changes that occur in malabsorption states. Needle biopsies of the prostate permit diagnosis of localized enlargement of the gland and proper radical surgery can be planned if it is required. The liver is now frequently biopsied and renal biopsies have clarified the etiology of the nephrotic syndrome in many cases. Not only do these procedures enable the pathologist to study lesions in their early or formative stage but such work brings the pathologist closer to the practice of clinical medicine and good use is made of his experience with the histological changes in diseased tissue.

The examination and handling of these specimens require special care on the part of the pathologist and the technical staff in the laboratory. The specimen may have been procured by a procedure not without some risk, or at least discomfort, for the patient. Such risk, slight though it may be, is not worth taking if there is a possibility that the small fragment of tissue may be lost in the laboratory. The following routine has been adopted at the Pathology Institute to prevent loss of specimens and to see that representative sections are cut from the tissue submitted.

Technique:-

The method of processing the tissue is simple and only a slight modification of the routine processing is required in order that the specimen will not be lost. Ten percent formalin is used as the initial fixative. We feel that this simplifies procedure for the operating room staff and secondary fixation may be carried out in the laboratory if it is desirable.

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*From the Pathology Institute, 62 University Avenue, Halifax, N. S.

When the specimen is received in the laboratory it is placed in a second solution of ten percent formalin containing 5 c.c. of 1% aqueous basic fuchsin per liter. After a few hours in this solution the tissue is tinted dark red and can be clearly seen after it is embedded in paraffin. This staining of the tissue fragment does not interfere with the final staining of the section on the slide.

The paraffin block is cut down to the embedded tissue and when the fragment is bright red there is no risk of cutting it away when the block is trimmed. In making sections, serial cuts are taken down to the full thickness of the tissue; the advantages of serial sections in the examination of small specimens are obvious. These sections are stained with hematoxylin and eosin for routine examination. Two more slides are prepared at this time and left unstained; they are available for any special stains that may be needed or for additional hematoxylin and eosin. By cutting the sections for special stains before they are required, there is no danger of losing tissue if the block has to be set up for recutting subsequently.

Results:-

During the past six months this system of handling small biopsies has been in operation at the Pathology Institute and during that time no such specimen has been lost in processing. The following specimens have been examined for diagnostic purposes:—muscle 23, brain 11, prostate 14, kidney 4, liver 20 (including open biopsies), bone marrow 43, pleura 28; total 143. Not included in this list are many biopsies of skin and lymph nodes which have been taken for the purpose of diagnosing a systemic disease; these specimens are larger and easily handled by routine methods.

Interpretation:-

Interpretation of small biopsy material requires close contact between the pathologist and the clinical services. Information from the patient's case record is essential and the exact source of the biopsy must be specified. Often personal consultation with the physician or examination of the patient or his X-rays may resolve a difficult problem. One of the greatest difficulties in interpretation is stretching and distortion of the tissue due to the trauma of removal. Some stretching of the cell nuclei is inevitable at the edges of the tissue but sometimes the whole tissue is distorted and nuclear structure is blurred, particularly if the cutting edge of the needle is dull. This artefact is very obvious when certain undifferentiated tumors with very little connective tissue stroma are sectioned. A sharp needle is the most important factor in preventing stretched and distorted tissues.

Conclusions:-

This has been a short review of the procedure followed at the Pathology Institute for handling the small biopsy specimens that are submitted for diagnosis. The information gained from histological examination of a properly selected tissue specimen may be essential for correct diagnosis and treatment of the patient's illness. We look forward to increasing numbers of these tissues as newer developments in histochemistry are applied to diagnostic histology.

THE SURGERY OF PAROTID TUMOURS*

ARTHUR L. MURPHY, M.D., F.A.C.S.**

Halifax, N. S.

The surgical approach to any tumour depends on the tumour's nature—benign or malignant. The tumours of the parotid are difficult to assess. As an operative guide, the simple clinical signs are best; but between the hard, fixed, obviously malignant tumour and the simple, well-encapsulated, movable, superficial growth, is a wide field of variance.

The common parotid growth is the mixed tumour. It occurs in all the salivary glands, but nine times more often in the parotid. Perhaps the term "mixed tumour" embraces three or four entities. We do not know. The cartilagenous-like material seen in many such growths has been called true cartilage; it has been called pseudo-cartilage. The epithelial elements may closely resemble adenocarcinoma; or they may be strewn throughout the mucoid stroma in the form of cords, again with a malignant appearance. Ewing called these tumours malignant. Even the most ardent proponent of their benignity must admit their tendency to recur within the parotid and, rarely, to metastasize to adjacent lymph nodes. Histologically, it is an insidious step from the simple metastasizing tumour to the mixed tumour with malignancy. The criterion is, of course, invasion.

This much only can we say with certainty about mixed tumours: They present an obscure histological picture. Some undergo, though rarely, degeneration into frank cancinoma, and they have no known microscopic characteristics to distinguish them from their benign relations.

Why does the mixed tumor recur? There is no proven explanation. The most popular theory is based on the fact that the tumor has often minute, finger-like excrescences, easily left behind in a local excision. A second theory is that the growth is multicentric in origin. In a small series of experiments on dogs mixed tumors were produced by tying off the parotid duct. Because small ducts must be tied in a local excision this work suggests another possible cause of recurrence. This could also explain what is, to me, the common association of mild parotitis with neoplasm, the assumption being that the inflammation is primary, the blocking of the ducts secondary, and development of the growth the final stage.

The mixed tumor sometimes undergoes cystic degeneration, again the result, probably, of duct blockage. Rarely, it seems to disappear, and spontaneous cures have been reported. Considering the nature of the growth, these seem unlikely. More probable is that a cyst having absorbed or discharged through a duct, the original growth lies dormant and unnoticed, perhaps for years.

I hope that I have now confused the pathological picture enough to establish the one proper surgical approach to the parotid tumor. Is it benign or malignant? On his own findings, the clinician is uncertain. The next step he will not take is a punch biopsy. It will only perplex him more. For the same reason he will not rely on frozen section at operation, because, like the punch biopsy, it shows only the area examined and tells him nothing of the rest of the tumour which may be quite different. If positive for malignancy,

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*Written from a presentation to the Royal College of Surgeons, Annual Meeting, Montreal, 1957.

**Head and Neck Service, Tumour Clinic, Victoria General Hospital.

it is helpful; if negative, it can be dangerously misleading. Besides, he knows that even the simplest mixed tumor, treated by local excision, often recurs.

It follows that, whatever the pre-operative diagnosis, and whatever the final histopathology, the only sound treatment for a parotid tumour is parotidectomy. Only by this operation can the surgeon be sure that a primary malignancy, or the simple mixed tumour with its excrescences or multicentricities, has been removed.

In all cases of benign tumour and in most cases of malignant, total parotidectomy can and should be done without facial nerve injury. Local excision is good surgery only when the tumour lies completely superficial on the gland, so that if there is recurrence, a second operation is not made more difficult; and in those rarities such as the chronically inflamed pre-auricular lymph node lying under the parotid fascia.

The facial nerve usually passes between the two lobes of the parotid gland as it lies like an inverted collar button. But the two lobes may be one, the nerve passing through gland substance. The parotidectomy, without nerve injury, can still be done.

The psychological approach of the surgeon is most important. His operation consists of a complete dissection of the facial nerve in the parotid region. The removal of the gland containing the tumour becomes an inevitable sequel.

We believe there are no anastamoses between nerve branches. Destruction of a nerve fibre, however minute, results in loss, perhaps equally minute, of facial muscle control.

Even more than usual, the anaesthetist's role is important. He must maintain his patient at a depth where light stimulation of a nerve fibre, by actual squeezing, will produce a response, but sensitivity will not be such as to allow fibrillary muscular twitching from any less specific nerve irritation.

The patient is draped with the full profile exposed, so that the slightest muscle twitch is apparent. The incision is the classical one, extending vertically down in front of the ear, behind the angle of the mandible, and curving forward for full exposure of the gland. Its short arm extends behind the lobule, and the first step of the operation is to dissect the lobule free, and with a sling suture, tuck it up behind the pinna. The scarring from this incision is minimal, and the only post-operative deformity is the hollow of the absent gland. A lesser incision means an inadequate operation and jeopardizes the facial nerve.

Bleeding is not a major difficulty. The superficial temporal artery is ligated as low and as early in the dissection as possible. Tying the external carotid in the neck is rarely necessary.

I have tried all methods of nerve exposure. I have picked up the tiny branches peripherally and followed them back, taking the gland off on the way; I have picked up the nerve at its foramen and then skipped about, finding the cervical branch where it crosses the facial vein approximately a centimetre above the inferior margin of the gland; the middle branch a centimetre above and almost parallel to the parotid duct; and the superior branch running toward the junction of zygoma and zygomatic arch. From behind, from before, or in the middle—there is only one best way to dissect out the facial nerve: Begin at the beginning as it emerges from the stylomastoid foramen, approximately 1.5 centimetres deep to the tip of the mastoid, where the posterior belly of the diagastric muscle is attached. Pick it up here, and with gentle persistence follow it forward all the way.

When the dissection has been carried to an adequate depth, and the nerve is not seen, a sharp pull on the parotid gland, by means of a hook, may bring it, gleaming white, into view. In the early dissection, visualization is enough, but as the branches become minute, other means of recognition are necessary.

A light haemostat (or mosquito forcep) is the solution. All tissue to be cut, often millimetre by millimetre, is picked up between two forceps and squeezed gently, to make certain it does not contain nerve fibres, before the forceps are clamped. The tissue is never cut against one forcep alone, but only between two.

With the nerve completely dissected out and the superficial portion of the parotid removed, the deep lobe may be taken piecemeal, or, often, the nerve can be retracted and the whole lobe withdrawn from beneath it.

The electric nerve stimulator has no place in parotid surgery. It is not delicate enough for the job. For example, a nerve fibre, lying beneath a minute artery to which the instrument is applied, may be stimulated to muscle contraction, in turn stimulating the surgeon to preserve the artery while cutting the nerve filament beneath. The instrument is dangerous.

Sometimes the nerve, instead of being white, is a pretty, parotid shade of pink. This occurs when there is mild inflammation associated with the tumour. Dissection is more difficult. The technique does not vary.

If the tumour be frankly malignant and the nerve invaded, it must be sacrificed. Sometimes, after excision of a segment, anastamosis is possible. More often the patient must accept the deformity. The fascial sling operation in which the ends of the sling are fixed above the belly of the temporal muscle gives a fair cosmetic result and some slight mobility to the affected side of the face.

If the operation is to be combined with radical neck dissection we do the parotidectomy first, and then, from the base of the neck, work up to the parotid area. This is not the most scientific way; it is not a strict "en bloc" resection. But along with the welfare of the patient we must consider the frailties of the surgeon, and to begin the tedious nerve dissection after an hour and a half has already been spent approaching the area from the clavicle, may prove too much for even the most placid nature.

Parotidectomy does not call for great technical knowledge. It does demand a gentle hand and infinite patience. Roughly treated, the nerve will cease to function and the surgeon becomes captain of a rudderless ship. He cannot go back; he is afraid to go on.

Perhaps the most important member of the operating team is the second assistant. His job is to watch the patient's undraped face, without blinking. He must have a clear voice, and be impervious to intimidation by his seniors. The best second assistant I ever had now practises on his native Nova Scotian north shore; and I am sure, on a clear night, his dauntless voice can be heard ringing out from Big Caribou to Judique.

Post-operatively, the patient with a benign tumour must show no evidence of nerve paralysis; or at the most, slight transient paresis.

Summary. The histopathology of the mixed parotid tumour and its relationship to malignancy are poorly understood. It follows that surgical treatment, with a few exceptions, is by total parotidectomy. The surgical approach is by meticulous dissection of the facial nerve, the excision of the gland being an inevitable sequel.

OBSERVATIONS ON INFLUENZA VIRUS VACCINE

A. R. WEBSTER, M.D.

Kingston, N. S.

Some time ago, there appeared in The Canadian Medical Journal a short article reporting the results of observations in an industrial group who had been inoculated against one of the Asian strains of influenza. A control group had also been followed, the latter receiving a placebo at the same time the others received the influenza inoculations. The conclusion was that the inoculations were of little, if any, value. However, the article neglected to mention what type of influenza, if any, was prevalent during the time of the experiment.

During the severe Influenza epidemic of the winter and spring of 1958-59, the writer was practicing in an isolated area and had the advantage of knowing the virus type, symptoms, and what to expect in general before the epidemic became prevalent there. Consequently, one hundred persons (including the author and his family) were inoculated against this type (Great Lakes "B") using the polyvalent type of vaccine, and the results were considered very gratifying. Because of the large demand for Influenza vaccine at that time, no special brand was ordered, and the brands used were produced by Connaught Laboratories and Parke Davis and Co. For all practical purposes they were antigenically the same.

The persons inoculated were either followed during the epidemic, or questioned after the epidemic had run its course. This is not being reported as a proper experimental procedure, since in a busy practice one cannot set up and work with a proper control group.

Observed results for the hundred that were inoculated are recorded below. It includes both sexes and all ages from five years and up.

		Primary Attack	Recurrence	After-effects (d)
Severe	(a)	3	1	1
Moderate	(b)	4	2	2
Slight	(c)	10	0	0
No Symptoms		83		

(a) Prostrated. Temperature over 102°. Severe headache. muscle and bone pain. Intractable racking nonproductive cough. Duration 7-10 days.

(b) Confined to bed. Moderate temperature, headache, muscle and bone pain. Severe cough, controlled by stronger antitussives. Duration 4-7 days.

(c) No temperature. General weakness and malaise. Slight headache and muscle pain. Duration about 3 days.

(d) Severe fatigue and weakness, lasting upwards to 6 weeks. Annoying nonproductive cough.

It should be noted here that each subject was given a course of the recommended amount of vaccine in two doses, two weeks apart. The figure of 83% with no symptoms compares very favorably with Tamm in Current Therapy 1960 who gives a figure of 70%. No figures have been seen in regard to the percentage of the general population who suffered with Influenza during this epidemic, but it is felt safe to judge that it would be far greater than 17%.

From the results recorded with this small group, as well as other figures elsewhere recorded, it is recommended that routine immunization with the polyvalent type of Influenza vaccine be carried out. This holds especially when an "Influenza Year" is expected such as has been forecast for the coming winter.

BOOK REVIEW

ADVENTURE TO MOTHERHOOD. Offer, J. A. Audio Visual Education Company of America, Box 52-6, Miami 52, Florida. 51 pp. $2.95.

Here is a new wrinkle to the standard prenatal gimmick—a glossy little book of colour pictures accompanied by short captions, outlining one woman's adventure to motherhood and showing the author's concept of what this should involve. We take issue with his emphasis on "shot-gun" prenatal capsules, foundation garments, but we are stunned to see a presumably normal delivery conducted by forceps and general anaesthesia and amazed to see the father in a closing sequence congratulating his wife for "the job she's done."

There are already several excellent books available for prenatal instruction. This contribution is unbelievably naive, slick in make-up but sick in outlook.

S. C. R.

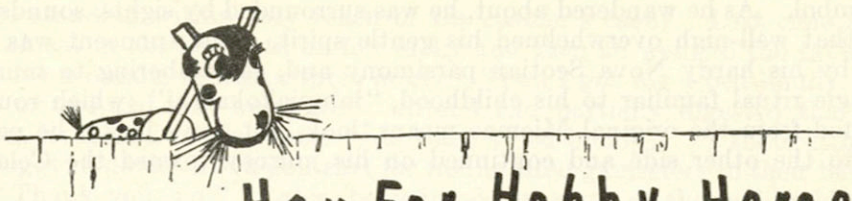
POST-GRADUATE DIVISION, FACULTY OF MEDICINE
DALHOUSIE UNIVERSITY

SHORT COURSES IN HALIFAX

1. INFECTIONS AND INFECTIOUS DISEASES. February 27 to March 3, 1961.
2. OBSTETRICS AND PAEDIATRICS ..... Late April, 1961.

REGIONAL COURSES

A series of six clinical meetings in your local hospital can be arranged on request (priority basis).



Hay For Hobby Horses

Bagdad in the Desert

The week-long meeting of the Society of Amateur Editors was over and I was leaving San Francisco and travelling south-east to Albuquerque, New Mexico for the week-end. Late Friday morning I slipped away from the closing sessions of the society and caught the plane. The "mobile work shops" had introduced the myopic and thin-blooded editors to the wide, wide world. The most coveted assignment of the series "A Biometrician looks at Las Vegas" had been taken from Drs. Novaque, Cole and myself and given to Professor Hees C. Nile of Vancouver. This worthy is the blue-veined, parchment-skinned dean of the Section on Statistics. Armed with an abacus (the mark of a statistician of the old school), chi-square tables, snuff and several hot water bottles, he boarded the Champagne Special, (a flight leaving San Francisco daily at 5 p.m. to return from Las Vegas at 4 a.m.) to do his field work.

Looking back on it now, it is a nice question whether my dessicated colleague was the best choice for this assignment. Biometrics is a trying business, being as it is, the science of statistics applied to biological observation. If the committee had assigned a delegate with any blood in him, would he have been faithful to the statistician's austere art? Could he have avoided the subtle intoxication of gambling fever and the ever-present distraction of the beautiful daughters of Eve? These daughters, faithful to their art, are in Mother Eve's original costume. (Now, there's a euphemism to end all euphemisms. Ed.) If Hees C. Nile toured Vegas, saw the show at the Stardust and was unmoved, I will insist at our next annual meeting that he resign as a *living* member of the society.

We were all envious of Professor Nile but if they had asked me to go in his place, I would have put on my British face, austere but kindly, and said, "Thank you, no. This is not my cup of tea." The truth is that I am a close man with a dollar and when faced with temptation to gamble, I am remorseful if I do and frustrated if I don't. (The same goes for anything else that costs more than two bits. Ed.) As it was, I left San Francisco feeling a glow of virtuous achievement at having put, yet another, temptation behind me until I discovered that the flight to Albuquerque put down in Las Vegas. After agonizing reappraisal I resolved, as a dutiful editor, to put all thoughts of self aside and think only of my readers waiting anxiously at their mail boxes in East Pessary and North Overshoe. I left the plane at Vegas *for my readers sale.*

I was not without consolation for I had a dog-eared copy of Pilgrim's Progress with me and, with Bunyan's vivid warnings in my mind, I scrutinized all passers-by for a glimpse of Mr. Come-by-Chance or Mr. Hate-Life. After the spirit of that classic I could say, "So it came to pass that Innocent was made to go through the Frivolous City and to resist many invitations to gamble

and gambol. As he wandered about, he was surrounded by sights, sounds and tastes that well-nigh overwhelmed his gentle spirit. But Innocent was protected by his hardy Nova Scotian parsimony and, remembering to murmur the magic ritual familiar to his childhood, "lampiakokatchi" (which roughly translated from the original Micmac means 'look, but don't buy'), he passed safely to the other side and continued on his journey toward the Celestial City."

In the small airport terminal the visitor must thread his way through banks of slot machines before reaching the cab stand. I was reminded of Richard Powell's description of them in his lively novel, Pioneer, Go Home. "Along the bar they had half an acre of machines that Blackie said were called one-armed bandits. You put a quarter or a half-dollar or a silver dollar in one of them machines, pull a lever, and the machine spins some wheels and tells you why you can't have your money back. Two fellows was putting in money and yanking the levers. I bet them fellows would have laughed if you had offered them a job in a factory yanking a lever all day at $1.80 an hour, but there they was doing the same work and paying the machines for the chance to do it. Now and then the machines would tease them fellows by giving them a few of their coins back, but it warn't no more than a loan and the fellows paid it back real quick."

I picked an hotel at random from the Yellow Pages and phoned for a reservation. The other half of the conversation went like this. "The Desert Inn would be glad to have you, sir. Yes, you can have the single without grand piano, private pool and personal bar. No one has used it since Elmer Gantry but it can be cleaned up." A cab bore me along the Strip, a two-mile length of Trans-Continental highway on the southwestern outskirts of Las Vegas, where money, energy and imagination have erected a chain of pleasure domes on the desert floor. They stand hip-to-hip in gaudy and electric splendor for several miles. Here are names to conjure with, The Stardust, The Thunderbird, The Tropicana, The Sahara, The Sands, The Silver Slipper, The New Frontier, The Flamingo, The Dunes and the Desert Inn. Waiting to entertain the visitors are such names as: Jimmie Durante, The McGuire Sisters, Lily St. Cyr and Jayne Mansfield, Milton Berle, Peggy Lee, Patti Page, Donald O'Connor, Nelson Eddy, Lena Horne, Dean Martin and the original Folies Bergere, direct from Paris. A bell-hop carried me from the lobby to my motel room in an over-sized golf cart and, spurning a mere half-dollar tip, drove away.

Two hours later your scribe was mingling with an elegant supper-time crowd in the lobby of the Desert Inn. The decor was Nevada Functional, a border of one-dollar slot machines with a central motif of green baize dice tables picked out with the black of dress suits and the white of sable and ermine. Beyond a low partition, a background of mahogany bar was topped by a four piece orchestra. As I wandered about, the chef was setting out hors d'oeuvres and I helped myself to smoked salmon and baby sausages with as much nonchalance as I could muster. Feeling in need of a little medicinal, I sat down in the bar area and accepted a gin-and-tonic from a young thing whose black silk hose seemed to go up to her middle or beyond. My neighbour was a husky specimen in his late twenties. He thrust out a meaty hand and said "Howdy, ah'm Jawn Kosh" then, with hardly a pause, he continued, "Ah'm restin', ah shouldna hit those tables until I felt good. Last night I leaned heavy on them and went down, down, down . . . I had to have my limit raised twice (here he hooked a great thumb at an impassive gentleman in a

tuxedo standing within easy reach of the cashier's cage). Last time ah was here, ah sat in the bar until ah felt lucky and then ah went to the tables and made me a bundle in lessen three hours. Then I got me a bed-buddy, saw a show or two and that was that." After I had partially digested this information, he looked up and said, "you want a bed-buddy?" I thought he said bed-buggy, a device like a golf cart for taking tired gamesters to their beds and said "Thank you, no." Later, he pointed out some of the bed-buddies and they are by no means shaped like a golf cart.

In our conversation, it came out that the show at the Stardust called the Lido de Paree which featured a troupe of English show-girls imported en bloc from Paris, was by all odds the greatest show on the Strip. But I, all ears and anxious, lamented that I didn't have a reservation. My new ally allowed that "The Pit" at the Inn owed him a favour or two and he would see. (The Pit is a pulpit-like eminence from which the casino manager dominates the tables. It is the control center through which that dynamo, Gambling, keeps Las Vegas alive and jumping.) John was back very shortly and said "Go to the Pit at the Stardust. Ask for Leonardo," I could get no more out of him except that a phone call had been made and it was all arranged. (To be continued. Deus Vult and the Editor permitting).

Sincerely, your servant

BROTHER TIMOTHY.

POSTSCRIPTUM. It may soothe the aggrieved reader if I define, as I did last January, the reason for this column. The enjoyment that the writer has while doing it, is the only reason for writing a series like this. My friends complain from time to time that my tastes are partial, even peculiar and that this column does not suit many readers of the Bulletin. That my appeal is not more catholic is no fault of mine. It is true that Hay for Hobby Horses comes in only two flavours, "fun" and "fight." Those who deplore the hectoring tone of some of my "fight" pieces can put me down quite easily and are encouraged to do so. The remedy is simple, illuminate my errors in a letter to the Editor or in a full-dress article for the Bulletin. If the Editor won't give you space, you are welcome to "Hay" for a month. However, even in my fight columns, my aim is a worthy one. In the words of an old hymn;

"My flames will not hurt thee, I only design
Thy dross to consume, thy gold to refine."

In closing, may I repair an omission? At the close of the Pink Champagne Pump, I did not send my readers, many or few, attentive or desultory, partisan or antagonistic, this column's wishes for peace and contentment during the holiday season and in the year to come. I do so now.

Thus, in the third year of its life, I commend my three-year old, Hay, to the nurture and admonition of all.

PERSONAL INTEREST NOTES

Cape Breton Medical Society

November 23, 1960—A very enjoyable social function, in the form of a supper-dance, was held at the Isle Royale Hotel, Sydney. The members and their wives from Sydney and the surrounding districts danced to the music of the Acadians Orchestra from 10-2 a.m. A chicken supper was served by the hotel management at midnight. Over thirty couples were in attendance.

Members of the Sydney medical fraternity are now participating in a bowling league in the new Bowlmore Alleys, Sobey's Shopping Centre, and weekly matches will be held.

Halifax Medical Society

December 12, 1960—At the rear of the Halifax Infirmary's new addition, fire destroyed a construction shed housing tools and building materials. One wall of a new four storey convent suffered damage from the fire, and windows were broken by the heat and portions of the woodwork near the windows and roof were ignited. City firemen fought the blaze during the City's first major snow storm of the winter, and a Halifax Fire Department Captain, Richard J. Kiely, collapsed and died.

The Victoria General Hospital Board Chairman, Mr. Manuel Zive, stated that Dr. Harvey Agnew, Toronto, consultant to the Nova Scotia Hospital Commission, was to visit Halifax, January 17, 1961 to examine plans for the new addition to the Victoria General Hospital. Mr. Zive said no estimate of the starting of construction can be made until after Dr. Agnew's visit.

December 14, 1960—A regular meeting of the Halifax Medical Society at the Halifax Infirmary discussed: group disability insurance, report of the executive meeting of the Nova Scotia Medical Society. A special committee of the Halifax Medical Society chaired by Dr. C. L. Gosse, and including Drs. P. C. Gordon, C. F. Keays, D. F. Smith, and C. H. Young has submitted a very complete study of the report of the special committee (Chairman: Dr. T. W. Gorman) as submitted to the special meeting of the executive committee September 10, 1960, on the structure of the Medical Society of Nova Scotia. This study, in mimeographed form, has been sent to all members of the Halifax Medical Society, pointing out that the Halifax Medical Society Committee feels that Dr. Gorman's committee report, while serving the purpose of bringing the whole problem before the Society, has very little that would improve on the present organizational set-up. Some positive suggestions were made regarding speeding up of the business of the Society, and methods to attract a larger attendance at the annual meetings. This study will be discussed further at the January meeting.

Dr. Harold C. Read, recently attended meetings of the American Society of Hematology in Montreal.

December 3, 1960—A 22 page report made public by the 1980 committee on Health Services proposed the establishment of several small hospitals on the outskirts of Halifax—possibly in the St. Margaret's Bay Road area, and in the Fairview-Rockingham region. Based on an estimated population growth of 5,000 per year, a new 450 bed hospital is required every 10 years. The Committee stated that the maximum size of the Victoria General Hospital must not go beyond 1,000 beds. The new addition to the Halifax Infirmary

will bring its total to 428 beds for active treatment and 31 beds for labour and recovery. It is planned to expand the Grace Maternity Hospital to 100 beds, but lack of land is a limiting factor in their planning. The Children's Hospital, too, is limited so far as expansion to their present site. Extensions and additions being planned and under construction at the Victoria General, Halifax Infirmary, Grace Maternity Hospitals, will increase the total of active treatment beds to 1650—against a required 2035—leaving a shortage of 370 beds to be filled by 1980. Against a requirement of 455 beds for long term patients in 1980, 185 will be available, leaving a shortage in this group of 270. Of the total of 650 additional beds needed, between 200 and 250 of these should be for children, and it is hoped that they may be provided in the Children's Hospital if this is enlarged or replaced. The remaining 400 or more beds will have to be available before 1980 if the Halifax region is to have adequate hospital facilities. The construction of a hospital for the care of convalescents is therefore indicated, for at the present time a considerable number of such patients occupy beds in general hospitals. The committee also gave a detailed study to dental care and the Halifax Area Water Supply. The Committee consisted of Manuel I. Zive, Chairman, Halifax 1980 Committee on Health Services, Dr. C. M. Bethune, Administrator Victoria General Hospital, Sister Catherine Gerard, Administrator, Halifax Infirmary, Brig. A. Atkinson, Suprintendent, Grace Maternity Hospital, C. M. Matheson, Administrator, Children's Hospital, Dr. C. B. Stewart, Dean of Medicine, Dalhousie University, Dr. J. D. MacLean, Dean of Dentistry, Dalhousie University, Dr. A. R. Morton, Commissioner of Public Health and Welfare City of Halifax, Dr. J. S. Robertson, Deputy Minister of Health, Province of Nova Scotia.

University

December 7, 1960—The Students Medical Society of Dalhousie University, announced the beginning of an annual lectureship program, inaugurated this year by Dr. Hans Selye, Director of the Institute of Experimental Medicine and Surgery, University of Montreal. Dr. Selye spoke at the Victoria General Hospital on "Stress and Sudden Cardiac Death" and at the Sir James Dunn Science Building, on "The Philosophy of Basic Research."

December 9, 1960—The annual Dalhousie Medical Ball was held at the Nova Scotian Hotel, Halifax.

Births

Dr. and Mrs. Joseph Cairns, a son, Halifax Infirmary, November 25, 1960.

Mr. and Mrs. F. Ian Gilchrist, a daughter, Erin Jacqueline, Grace Maternity Hospital, Halifax, November 25, 1960.

Dr. and Mrs. Ross MacInnis, a daughter, Mary, Grace Maternity Hospital, December 15, 1960.

Dr. and Mrs. Beverley Trask, a son, Peter Alfred, Grace Maternity Hospital, November 24, 1960.

Coming Events

February 1961—The Canadian Heart Fund will be soliciting financial assistance and support of cardiovascular research throughout Canada during the entire month.

February 20-24, 1960—The American College of Physicians, Post-Graduate Course Number 6, Clinical Patho-Physiologic Conferences at Oklahoma University Medical Centre, 913 N.E., 13th Street, Oklahoma City, Oklahoma.

March 4, 1961—Nova Scotia Division, College of General Practice, Business Meeting with social activities.

March 12-17, 1961—American College of Allergists, Graduate Instructional Course, 17th Annual Congress at the Statler-Hilton, Dallas, Texas. For information write: J. D. Gillespie, M.D., Treasurer, 2141 14th Street, Boulder, Colorado.

March 20-23, 1961—College of General Practice, (Medicine) of Canada, Vancouver, B. C.

June 12-14, 1961—108th Annual Meeting Medical Society of Nova Scotia, at Keltic Lodge, Ingonish, Cape Breton. Host: Pictou County Medical Society.

June 19-23, 1961—Canadian Medical Association, 94th Annual Meeting, Montreal, Quebec.

Obituary

Dr. Edmund Brasset, 53, who had practiced in Antigonish, Canso, and New Waterford, died recently in Saloma, California. He was author of the widely read, "Doctor's Pilgrimage" while practicing in Rhode Island. The book concerned his years as a general practitioner in rural areas of Nova Scotia. Dr. Brasset was born in Inverness, and was educated at St. Mary's, St. Francis Xavier, and Dalhousie Universities. He had been practicing in California for the past 3 years. He is survived by his wife, four sons, one daughter, one brother, and two sisters.

Sympathy

The Nova Scotia Medical Bulletin extends sympathy to Dr. J. A. McDonald, Glace Bay on the death of his youngest child, Paul, aged 6 years, at the Halifax Children's Hospital on December 14, 1960 following a brief illness. Dr. M. S. MacDonald, Dartmouth was the child's uncle.

Post-Graduate News

The Short Course in Psychiatry presented by Dr. R. O. Jones and Staff at the Victoria General Hospital under the sponsorship of the Post-Graduate Division, Faculty of Medicine, Dalhousie University, is being held Monday, January 30th, Tuesday, January 31st, and Wednesday, February 1st, 1961. Detail programs have already been mailed to all practitioners.

"The Week in Infections and Infectious Diseases" is being presented in the Victoria General and Children's Hospitals between February 27th and March 3rd. Guest speakers will include Dr. J. E. Hiltz, Superintendent, The Nova Scotia Sanatorium; Dr. Arnold Branch, D.V.A. Hospital, Lancaster, New Brunswick; and Dr. Edward H. Kass, Boston City Hospital. As this week is followed immediately by the Annual Meeting of the Nova Scotia Chapter, College of General Practice, it has been possible to arrange a contribution by their guest speaker Dr. D. J. MacKenzie of Sunnybrook Hospital. Details of this program will be mailed to all doctors, in early February.

The final Short Course of the 1960-61 Academic Year will be presented in late April or early May by the Departments of Obstetrics, Gynaecology and Paediatrics. Details will be announced in a subsequent issue of the Bulletin.

Annual Meeting, Nova Scotia Chapter, College of General Practice of Canada

The President, Dr. A. W. Titus announces the Annual Business and Clinical Meeting of the College to be held in the Auditorium, Victoria General Hospital on Saturday, March 4th, 1961.

There will be business meetings morning and afternoon, and the Canadian President, Dr. F. M. Fraser, and Executive Director, Dr. W. Victor Johnston will be in attendance.

A clinical program has been arranged in conjunction with the Post-Graduate Division to include a surgical presentation on "Emergency Care of Maxillo-Facial Injuries" by Dr. James Ross; a "Dermatology Quiz" by Dr. D. R. S. Howell. Dr. D. J. MacKenzie, Associate Chief of Service (Medicine) Sunnybrook Hospital will conduct a clinic on "Hypertension" during the morning and present a formal paper in the afternoon.

A social program is well under way with afternoon tea arranged for the wives, and reception and dinner to be followed by an evening of dancing. Dr. W. Victor Johnston will speak at the dinner.

DOCTOR JOHN CAMERON AT DALHOUSIE

An Appreciation

The news of the death of Doctor John Cameron in England recently will be received with regret by a multitude of friends in Canada, the United States, and particularly in the Atlantic Provinces. He spent fourteen years of a very useful life as Professor of Anatomy in Dalhousie Medical School. Every medical and dental student who attended his classes from 1916 to 1930 knew him as he knew them. Outside the University his social, scientific and cultural interests, which were extensive, resulted in cordial and lasting contacts which remained long after he left Dalhousie thirty years ago.

To those who knew him best he was "Jock," and so he will remain in their hearts. Along with Doctor D. Fraser-Harris, Professor of Physiology ("Hypo"), he promoted culture, a love of the humanities, and a respect for the finest traditions of Medicine that made a lasting impression on every student that passed through both their hands.

To describe him to a new generation may seen a futile gesture but a few recollections may awaken pleasant chords of memory in many minds and at the same time convey to those who knew him not, an idea of a lovable character and a remarkable teacher.

I can see him yet, a man of medium height crossing the Forrest Campus. In general appearance he vaguely resembled Sir Harry Lauder, minus the kilt and the eccentric cane. He had a swarthy complexion, quite prominent features, with warmly expressive brown eyes. His hair was black, thinning on top, which he attempted to conceal by cross combing. He invariably wore a salt and pepper grey tweed suit, black brogues, a winged collar and a blue, polka dot bow tie. Outside he covered these garments in winter with a nondescript overcoat and his head covering was a grey Homburg. As a rule he carried a cane and at times an umbrella. His frame was well knit, strong, and the product of early life and work on a Scottish Lowland farm. In the Anatomy Room as well as during lectures he wore a short, thigh length white gown.

He spoke with a well marked Lowland Scots accent, but as his vocal cords were keyed to tenor tones, it did not prove a source of difficulty to his hearers. On the contrary, to mimic Jock was one of the ambitions of his students in their lighter moments. In his earlier years at Dalhousie he lived at Birchdale, the Men's Residence, where Thornvale Avenue is now. Later, in anticipation of marriage he built 47 South Street, the "wee hoose," where he lived until that happy event took place some time later.

In Jock's day the Anatomy Department with its separate cross corridor was on the north end, third floor of the Forrest Building. There was a partition running north and south, and his office was on the front of the building. Later this office, which was very large, was divided, Jock retaining the part next the cross hall, and the north east room becoming part of the Anatomy Room, but mainly devoted to the study of bones.

Associated with Jock in his daily efforts was "Bill" Fry, the laboratory custodian, affectionately remembered by every current student of anatomy. The place for lectures was Room 39, also on the third floor, but south of the staircase, and facing the front of the building. This was also "Hypo's" classroom, and "Bill" Fry kept it in order for both.

Jock was an artist with chalk. He drew "free hand" with wonderful accuracy. As he drew in the "strooctures" he explained, and his explanations

were vivid but simple. A complete set of lectures with his drawings was a text book of Anatomy. Out of these lectures came "Cameron's Regional Anatomy," known to Jock as "The Beuk." It was originally published by The Ross Print on Argyle Street, in Halifax, a fact made known to his students on the first day of lectures. It is no longer used at Dalhousie, but for many years after his departure for England, and perhaps still, it was the text employed by two large medical schools in the United States. It did not have illustrations, as Jock intended it to be used while dissecting, and when not engaged in this task the preface reminded you that "visualization was nature's key to the memory centres."

Later Jock's lectures on bones became "Cameron's Osteology," this time illustrated with his black and white drawings. It was a more ambitious and equally useful work, but it never occupied the place in his affections of "The Beuk."

Some more ambitious students or those who could not draw and could not "visualize" were wont to consult larger and more ambitious works on Anatomy, and in consequence "The Beuk" was once in a while, though rarely, found to be inaccurate or at least inadequate in description. If an "eager beaver" came upon such a flaw it was not the part of wisdom to run to Jock with the news. On one such occasion his remarks were:

"O-O-O-O, so the beuk is wrong; ceartainly the beuk *can* be wrong. When you get pairfect men you get pairfect beuks, an' I've no halo an' harp."

These were delivered in tones which did not indicate the highest degree of approval for the researcher. To criticize "The Beuk" was just not done.

There was another "beuk" in Jock's life and ours. It was the one in which he kept the marks of what he referred to as "quisses." In the first year as soon as you had finished one half of "a part," you notified Jock and he gave you a quiz. Usually it was taken with three other students. In the second year you got a quiz on the entire "part." If you knew it "cold" you asked for the quiz in the morning, but if you had doubts, the proper time was right after the noonday meal. In the first instance, Jock was always "on the beam"; in the latter he was invariably sleepy. In a "sleepy quiss" he would often drop off for ten minutes at a time, and either forget what he had asked and repeat the question, or regard the answer in a far from critical light. Following such examinations the marks were almost invariably low, but Jock was so conscious of his own lack of mental acuity at the time that failures were rare. Just the same all concerned were anxious to know their marks and for this they depended on Bill Fry.

The pass mark on a quiz was 60, and a perfect mark was 80. Jock always held that no student could be more than 80 per cent correct. He gave and later endowed a Prize in Anatomy. Even the student who just "got by" left Jock's classes with a pretty sound knowledge of Anatomy. The final was a written examination and a quizz on the whole body. What you "lost on the swings you gained on the roundabouts." Jock was aware of the extreme tensions involved and treated all oral candidates like a benign Scottish fairy rather than the bogle or the banshee of his more critical priods. He "hit you easy" at first and if you did well the questions increased in difficulty. In spite of average marks in the "Marks Beuk", it was still possible to get distinction."

Before you said "Farewell to Anatomy," it was considered "good form" to get Jock to autograph his two books which had been your daily companion for two years. Those who felt that the event justified a little "apple polishing"

toddled down to Ross Print and bought a new set for the occasion. This, of course, conveyed the subtle implication that the owner had worn his books threadbare with study, but the more direct one that modest sum in royalties had been added to the Cameron exchequer. In any event Jock signed them all, clean or greasy, and gave the owner his blessing.

If this does nothing more, I hope that I have conveyed the following: first that Jock had a profound knowledge of Anatomy; secondly that he was a splendid teacher; thirdly that he was a sensitive and very human individual. For the first two we admired him, for the last we loved him.

Outside interests were numerous, including an active participation in music. He also had activities which accentuated our affectionate, if at times our amused regard. Before coming to Canada he had been associated with Sir Arthur Keith in some anatomical studies on Egyptian mummies. In consequence had had a keen interest in Egyptology. When the tomb of King Tut-ankh-amen was opened in the Valley of the Kings his enthusiasm was boundless. Soon afterwards he had an "at home" in the Munro Room one evening to which he invited all his friends and delivered them a lecture on King "Toot", with refreshments added.

While at Birchdale he purchased a four-oared shell and encouraged young rowing enthusiasts to use it, with he, himself, as an inactive passenger. He thought it would develop their "mooscles." As usual, there was an "apple polisher" or two mixed in with those genuinely interested, and their enthusiasm quickly waned. There is an old Chinese proverb which says, "Man on tiger's back cannot get off," and their fear of Jock's displeasure kept them as unwilling Argonants. Finally, when they were almost in despair one of them accidently broke an oar. This flicked Jock "on the raw." Strangely enough a second oar was broken a few evenings later. Jock told them that they were "too strong," and replaced them with more genuine aspirants.

Although retired and living in England for 30 years, Jock's students never forgot him and their letters to him were invariably answered. He made one last trip to Nova Scotia to see his friends after the War and it was like a royal triumph from Halifax through the Province.

Yes, "Jock" Cameron is gone from this scene of toil, strife and anxiety, but he has left behind a great many people who were wiser and better and happier for his acquaintance. There can be no better epitaph for any man.

H. L. S.

DOCTOR JOHN JAMES MacRITCHIE

An Appreciation

To those who knew him well, "J. J." was the most likeable of men, and for a person in a public office, most singularly free of enemies. He enjoyed hearing and telling a good story—he enjoyed "poorhouse" poker and good companions—and he enjoyed his pipe despite its scratched appearance. His regular appearance at the "kirk" and at his Church Club indicated his belief in the essential goodness of his fellow men. If he had a weakness, it was a belief that Cape Breton, especially the Englishtown area, was somewhat akin to Heaven. It was to Englishtown that he retired after leaving the Department of Public Health in 1955.

With a background of general practice and overseas service in World War I, he was a most popular medical health officer, both with his medical associates and the people of the Province. He pioneered much of the early tuberculosis case finding in the eastern counties, holding chest clinics in halls, churches and hotels—anywhere he could set up a portable X-ray machine. It was not an easy way of life. Many evenings were spent in doctors' offices discussing cases seen during the day.

In later years he restricted his work largely to Halifax County and he gave major assistance to Municipal officials, being a regular attendant at Municipal Council meetings. Even with the most worrisome problems he never seemed to lose his sense of humour—a topical story often relieved tension at critical moments when tempers were high. To appointed officials, patients and the public he was always ready to listen and to assist with wise advice from his fund of experience gained over the years.

"J. J." was most popular with his colleagues in the Department and his retirement did not break this association—he visited his friends and fellow workers when his health permitted. His passing will be regretted by all who knew him.

To his wife and constant companion his passing will leave memories of a husband who was greatly appreciated by his fellow men, who never spoke ill of others and who spent his life in service to humanity.

J. S. R.

Why shouldna he blow?

He's piping hot News.

Dinna ye ken then?

It's our 108th Annual Meeting
of the Nova Scotia Division
of the Canadian Medical Society

So lads and lassies all—will ye nae take the road to The Isle for 12-13-14 June, 1961 when the Pictou Co. Branch is host to The Medical Society of Nova Scotia at Keltic Lodge, Ingonish? The road is completely paved via Baddeck. Make your reservations early: watch for housing application forms in February issue.

Much ground work has already been done for the annual meeting and the date and locale have been decided upon. Under the general chairmanship of President Dr. Fred J. Granville of Stellarton, the Pictou Co. Branch will be host to the Society on Monday, Tuesday and Wednesday, 12-13-14 June 1961 at Keltic Lodge, Ingonish.

Dr. Granville has named the chairmen of various committees, and along with the Executive Secretary, Dr. C. J. W. Beckwith, an organizational meeting was held with these chairmen on 21 November, 1960.

The chairmen of committees are:

1. General Chairman—President, Dr. F. J. Granville.
2. Programme and Entertainment—Dr. C. G. Harries.
3. Registration—Dr. J. M. Williston.
4. Housing—Dr. W. A. MacQuarrie.
5. Medical Exhibitors—Dr. M. F. Fitzgerald.
6. Ladies Committee—Dr. and Mrs. V. H. T. Parker.
7. Publicity—Dr. J. B. MacDonald.

You are asked to watch each issue of the Bulletin from January, 1961, to May, 1961, for information relating to the annual meeting.

Efforts will be made to surpass all previous attendance records and to provide a well considered and worthwhile programme.

For your convenience, as in other years, a housing application form will appear in each issue from February to May, inclusive. These should be completed and forwarded to Dr. C. J. W. Beckwith, Executive Secretary. The application will be acknowledged and confirmation of space sent along at a later date.

A provisional programme will appear in an early issue of the Bulletin and it is planned to have the final programme not later than the April issue.

Suggestions for the meeting or constructive criticism relating to experience at previous meetings will be gladly welcomed by the President, Dr. F. J. Granville, Stellarton.

J. B. MacD.

NOTICE

A machine for measurement of the Basal Metabolic Rate is available, free of charge, to any interested physician.

Please contact—

Mrs. M. Jacobson,
438 Quinpool Road,
Halifax, N. S.

Tel. No. 423-5641

Radiologist Wanted

Radiologist wanted immediately to service two hospitals in the centre of the Annapolis Valley. Hospitals are: Western Kings Memorial Hospital, Berwick, N. S., with bed capacity of 40 and extension under consideration, and Soldiers' Memorial Hospital, Middleton, N. S., a new 68 bed hospital opening in Spring 1961, replacing the present 27 bed structure. These two towns are twenty miles apart, all paved highway. Income commensurate with qualifications. Address reply to:

The Superintendent,
Soldiers' Memorial Hospital,
Middleton, N. S.

INFECTIOUS DISEASES—NOVA SCOTIA
Reported Summary for the Month of October, 1960

Diseases	NOVA SCOTIA 1960 C	NOVA SCOTIA 1960 D	NOVA SCOTIA 1959 C	NOVA SCOTIA 1959 D	CANADA 1960 C	CANADA 1959 C
Brucellosis (Undulant fever) (044)	0	0	0	0	17	14
Diarrhoea of newborn, epidemic (764)	0	0	0	0	4	13
Diphtheria (055)	4	0	0	0	5	2
Dysentery:						
(a) Amoebic (046)	0	0	0	0	0	0
(b) Bacillary (045)	1	0	0	0	219	211
(c) Unspecified (048)	2	0	0	0	64	42
Encephalitis, infectious (082.0)	0	0	0	0	2	5
Food Poisoning:						
(a) Staphylococcus intoxication (049.0)	0	0	0	0	0	0
(b) Salmonella infections (042.1)	0	0	0	0	69	0
(c) Unspecified (049.2)	0	0	0	0	6	149
Hepatitis, infectious (including serum hepatitis) (092, N998.5)	104	0	53	0	530	340
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	1	0	2	0	100	199
Meningococcal infections (057)	1	0	0	0	10	16
Pemphigus neonatorum (impetigo of the newborn) (766)	0	0	0	0	0	0
Pertussis (Whooping Cough) (056)	1	0	14	0	593	863
Poliomyelitis, paralytic (080.0, 080.1)	0	0	2	0	129	519
Scarlet Fever & Streptococcal Sore Throat (050, 051)	317	0	169	0	1074	1302
Tuberculosis						
(a) Pulmonary (001, 002)	0	0	15	0	480	485
(b) Other and unspecified (003-019)	0	5	2	0	59	106
Typhoid and Paratyphoid Fever (040, 041)	0	0	0	0	19	39
Venereal diseases						
(a) Gonorrhoea — Ophthalmia neonatorum (033)	0	0	0	0	0	0
All other forms (030-032, 034)	31	0	39	0	1618	1521
(b) Syphilis — Acquired—primary (021.0, 021.1)	0	0	1	0	208	0
— secondary (021.2, 021.3)	0	0	0	0	0	0
— latent (028)	0	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	1	0
Prenatal—congenital (020)	0	0	0	0	0	0 *
Other and unspecified (029)	2	0	0	0	0	211
(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	2	0
Trichinosis (128)	0	0	0	0	12	0
Tularaemia (059)	0	0	0	0	1	0
Yellow Fever (091)	0	0	1	0	0	0

C — Cases D — Deaths

*Not broken down