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EDITORIAL

THE SPECTRE OF MALPRACTICE

This issue contains an abstract (page 51) dealing with malpractice. Among the skeletons in the medical cupboard, perhaps, this one is most commonly rattled in public.

The press irregularly but with disturbing frequency, reports instances of judgments handed down against physicians. Articles appear, from time to time, in lay periodicals indicating a growing frequency of malpractice suits and a tendency to larger settlements. Many factors are involved in this growth. We are living in an increasingly compensation-minded society. The publicity such lawsuits receive is another element in the increase. The deteriorating relationships between the medical profession and members of the public is both a cause and an effect of malpractice suits. The releases in the lay press emphasizing the many advances in medicine, inevitably lead a patient to expect more of the physician, working with such advanced facilities, than was expected of his predecessor.

Mr. Duncan K. MacTavish, K.C., whose article is abstracted in this issue, indicates that if the practitioner provides "a fair and reasonable standard of care and competence," he is unlikely to be found guilty of malpractice. The importance of well kept records, as proof of such care and competence cannot be over-emphasized.

Unfortunately, a fair and reasonable standard of care and competence, does not protect from innocent involvement in legal actions. With this sobering thought, I leave you to read the abstract and the entire Annual Report of the Canadian Medical Protective Association, and to reflect on the value of membership in this body.

L. C. S.

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THE TIDE IS RISING! WHERE ARE THE REST OF THE SHIP-BUILDERS?
The ARK of PROFESSIONAL FREEDOM
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THE BIOCHEMISTRY AND PHARMACOLOGY OF ORALLY-ACTIVE DIURETIC DRUGS

J. G. ALDOUS, Ph.D.*

I. Biochemical Considerations

During the last 20 years our concepts of normal kidney function have undergone radical changes, which, in large part are due to observations arising out of abnormal metabolic conditions produced by drugs. Twenty-five years ago our picture of kidney function was very much colored by Cushny's tenet that all the final constituents of the urine were already present in the glomerular filtrate. Quantitative changes only took place (reabsorption) during the passage of the urine through the kidney tubule.

The years 1937-38 will be remembered as the age of the new "wonder drug" sulfanilamide, during which wide use was made of this compound in the control of bacterial infections. Clinical experience with sulfanilamide early indicated that its use frequently resulted in the production of a metabolic acidosis and an accompanying alkaline urine.

In 1941 Davenport and his associates, in an extension of pioneer work done in collaboration with Roughton, observed that renal tissue contained large amounts of the enzyme carbonic anhydrase. This enzyme had almost 10 years earlier been shown to catalyze the reaction $H_2CO_3 \rightleftharpoons H_2O + CO_2$ and had already been assigned an important role in the elimination of CO_2 from the blood in alveolar tissues. Sulfanilamide was known to be an inhibitor of carbonic anhydrase, and Davenport suggested that the metabolic acidosis resulting from its use was merely the result of an inhibition of $NaHCO_3$ reabsorption. Thus, carbonic anhydrase was assigned a role in one of the reabsorptive mechanisms of the kidney tubule. It is to be noted that this suggestion in no way violates Cushny's postulate.

However, in 1945, Pitts and Alexander reported that when acidotic dogs were infused with phosphate buffer, they excreted more H^+ in the urine than was potentially present in the glomerular filtrate. In other words, H^+ had been added to the glomerular filtrate during its passage down the tubule. These authors went on to postulate that H_2CO_3 was the source of the hydrogen ion secreted by the kidney tubule; and this of course implicated carbonic anhydrase. The final proof of the role of this enzyme came with the demonstration that sulfanilamide decreased the titratable acidity of the urine.

Work done since 1945 has served not only to fill in the finer details of the picture, but to emphasize the fact that whereas we formerly tended to look upon the kidney as an organ of conservation, one of its very important functions was that of hydrogen ion excretion. The importance of carbonic anhydrase in this respect may be appreciated by referring to the accompanying diagram (page 39) which has been adapted from Gilman (1) and Goodman and Gilman (2).

The mechanism for bicarbonate reabsorption is a beautiful illustration of a self-regulating biological device. First of all, it will be noted the slightly alkaline glomerular filtrate contains, besides other ingredients, sodium and bicarbonate ions. From this mixture the sodium ion is reabsorbed by the tubular cell (a process regulated by secretions from the adrenal cortex) and,

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*Professor of Pharmacology, Dalhousie University.

in order to preserve electrical neutrality in the cell, a hydrogen ion is secreted into the lumen of the tubule. Carbonic acid is thus formed from what was originally sodium bicarbonate. As this process continues, the urine becomes more acid, and as a result the carbonic acid dissociates into carbon dioxide and water. Carbon dioxide now diffuses rapidly back into the tubular cell where, through the action of carbonic anhydrase, it is hydrated to form carbonic acid and finally, bicarbonate. Bicarbonate can also arise as the result of hydration of the carbon dioxide from metabolic reactions of the cell. Thus the sodium bicarbonate originally filtered through the glomerulus is returned to the extracellular fluid (E.C.F.). Note however that the urinary product resulting from these reactions is water, whose H^+ came originally from hydrated carbon dioxide (carbonic acid) and that carbonic anhydrase is necessary for the efficient function if this Na^+ - H^+ exchange. The beauty of this mechanism lies in the fact that the cation ion exchanging for Na^+ is one which acidifies the urine. Were this not so, and should another cation like K^+ have exchanged for Na^+ no dissociation of bicarbonate would have taken place. This in turn would mean that bicarbonate would be lost from the body fluids.

Hydrogen ion excretion is also involved in the elimination of phosphates as their acid salts, where one hydrogen atom in the molecule of NaH_2PO_4 is derived through an exchange for Na^+. In somewhat the same manner, and involving the Na^+ -H^+ exchange, ammonia arising from protein metabolism is eliminated as NH_4Cl. Here the greater the concentration of H^+ and Cl^- ions in the tubular fluid, the greater will be the diffusion of NH_3 from the tubular cell. The two latter mechanisms (phosphate and ammonia excretion) are not nearly so dependent upon carbonic anhydrase activity, suggesting that dissociation of bicarbonate in the tubular cell can supply an adequate amount of H^+.

In order to appreciate fully, the actions and limitations of the drugs to be discussed, a further point concerning bicarbonate reabsorption should be emphasized, namely the influence of p CO_2. If, through failure to eliminate CO_2 from the lungs, a respiratory acidosis occurs, the kidney attempts to compensate for this by conserving alkali. Thus a high p CO_2 is associated with a high rate of sodium bicarbonate reabsorption, which, through the mechanisms already discussed, results in a high rate of excretion of H^+.

In that type of metabolic acidosis, however, when the $BHCO_3$:H_2CO_3 ratio has been altered by an excessive loss of alkali, the lung normally compensates by excreting bicarbonate as CO_2 and this results in a deficiency of both cation and anion. Because of this deficiency, the total amount of alkali reabsorbed by the kidney tubule is considerably lower than normal, and this in turn means that the Na^+-H^+ exchange is not nearly so active. Gilman (1) has produced evidence of this state of affairs as a result of experiments carried out in dogs. Whereas the normal excretion of H^+ per 24 hours amounts to about 4600 m Eq, in experimentally induced metabolic acidosis, the H^+ excretion is reduced to 2850 m Eq. It does not necessarily follow that under these circumstances, the urine will be less acid, because in metabolic acidosis, organic acids, such as acetoacetic, may make their appearance in the glomerular filtrate, and since these are much stronger acids than H_2CO_3, the net effect will be an acid urine.

Mention was made earlier of the fact that sulfanilamide induced a metabolic acidosis which was accompanied by an alkaline urine. This state of affairs arises by virtue of the fact that the anion which is excreted (bicarbonate) is weakly acidic. Inhibition of carbonic anhydrase in the tubular cell immediately reduces the source of H^+ with which Na^+ normally exchanges. The consequences of this are two-fold: (a) Na^+ reabsorption is reduced, and (b)

because of this the glomerular filtrate does not become acid enough to produce a dissociation of bicarbonate into CO_2 and H_2O. The final effect is therefore a loss of Na^+ and HCO_3^- which together, result in an alkaline urine. It is to be noted that in these circumstances there is always a loss of K^+, and this has been interpreted by some to indicate that to a limited extent, K^+ may replace H^+ as the cation exchange for Na^+.

II. Pharmacological Considerations

The connection between the above discussion and the phenomenon of diuresis should now be evident. Carbonic anhydrase inhibitors reduce the amount of available H^+, thereby limiting the reabsorption of Na^+ and finally that of bicarbonate. The tubular fluid is therefore richer than normal in electrolytes which by virtue of exerting osmotic pressure, prevent the reabsorption of water which takes place in the more distal regions of the tubule. Thus, "obligatory water" is excreted along with these electrolytes.

Shortly after the rather weak diuretic action of sulfanilamide was appreciated, a search was undertaken for compounds possessing a more powerful action in this respect. The first of these to receive any extensive clinical trial was acetazolamide (Diamox) which proved to be a very powerful inhibitor of carbonic anhydrase and a fairly good diuretic. The search for such a compound led to the conclusion that in order to possess carbonic anhydrase-inhibiting properties, the drug must possess an unsubstituted sulfamyl group in the molecule. We might therefore visualize the general chemical structure of this group of drugs as: $R\text{-}SO_2\text{-}NH_2$, where R is an aromatic or other cyclic structure.

Despite the fact that all the newer "thiazide" diuretics possess this chemical structure and are therefore powerful inhibitors of carbonic anhydrase under *in vitro* conditions, it is extremely doubtful whether they owe very much of their diuretic action to this mechanism. Indeed, the only true anti-carbonic anhydrase diuretic to receive extensive trial is acetazolamide.

Interest in this group of drugs as diuretics has lain chiefly in the fact that like their parent compounds, the sulfonamides, they are readily available following oral administration. They thereby offer an advantage over the mercurial diuretics which must be given parenterally, if one is to obtain a predictable action. The troublesome side actions associated with incipient mercury toxicity are also obviated; but these advantages are not gained without paying the price of caution in other directions. In fact, when advantages and disadvantages of the "thiazide" drugs are weighed against those of the mercurial diuretics, it turns out that their only real advantage is that they may be given orally.

Apart from such readily reversible side reactions such as skin rashes and depression of bone marrow activity (herein resembling sulfanomides) the chief disadvantages to a drug such as acetazolamide lie in the diuretic refractoriness which is often observed to develop and in the hypokalemia which may accompany its continued administration. Both these undesirable characteristics are a direct expression of its mechanism of diuretic action; for by referring to Figure I, it may be seen that continued inhibition of carbonic anhydrase, leads to a loss of Na^+ and HCO_3^- (i.e. a metabolic acidosis) and it is this particular condition which finally leads to a very much reduced Na^+ -H^+ exchange, i.e. the need for carbonic anhydrase activity is greatly reduced. Thus the diuresis induced by acetazolamide is always self-limiting. The important consideration which immediately follows is that this refractoriness is not nearly so amenable to correction as is the refractoriness which develops to mercurial diuretics. In the latter case, where the mechanism of diuretic action depends

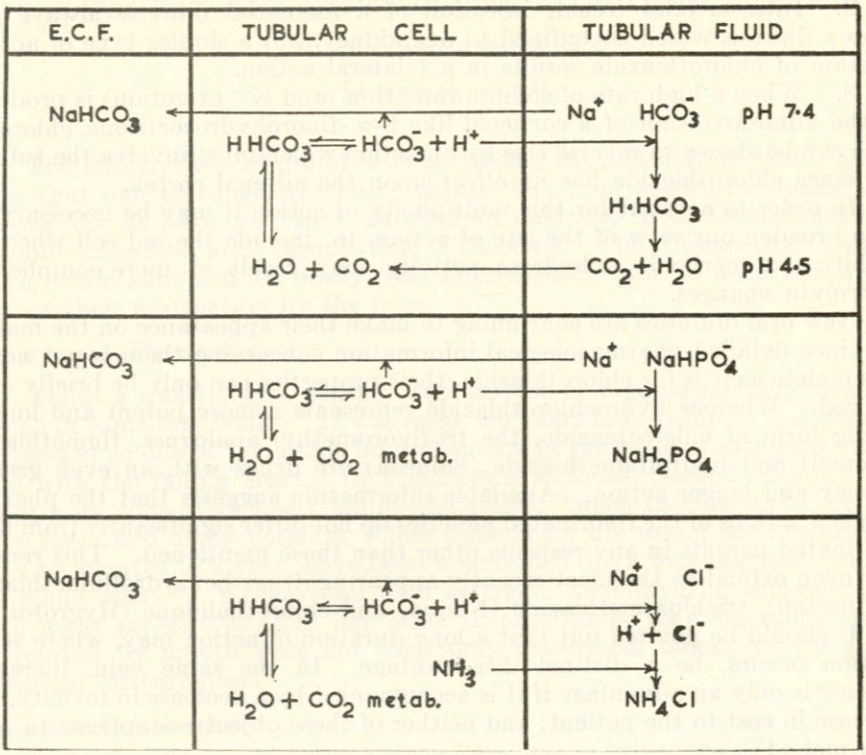

FIG. 1. RENAL FUNCTIONS WHICH INVOLVE EXCRETION OF THE HYDROGEN ION.

upon the continued availability of chloride, refractoriness may be corrected by administration of ammonium chloride. In acetozolamide-induced diuresis, it is not desirable to administer $NaHCO_3$ and so the only alternative becomes one of waiting until the metabolic acidosis has corrected itself.

The second undesirable side effect of acetazolamide lies in the hypokalemia which results from the urinary loss of potassium. Unless steps are taken to replace this cation alterations in myocardial irritability occur and these may be particularly serious when the patient is receiving concomitant digitalis therapy.

The search for more suitable drugs in this class had, as its prime objectives, an increase in potency and an increase in duration of action (acetazolamide is administered t.i.d.) and a reduced tendency toward potassium loss. In present day representatives, all these objectives have been met, but unfortunately the manufacturer's claims to the third objective tend to minimize the importance of the potassium losses which still occur.

Despite the fact that drugs like chlorothiazide (Diuril) and hydrochlorothiazide (Hydrodiuril, Esidrex) are potent inhibitors of carbonic anhydrase *in vitro*, a glance of some of the pharmacological characteristics of the diuresis they induce leaves one with the impression that their mechanism is very much more complicated (3):

1. Chlorothiazide-induced diuresis results in the excretion of large amounts of Na^+ and Cl^- and, to a lesser extent of K^+ and HCO_3^-. In the elimination of Cl^- the action resembles that of mercurial diuretics, but whereas the latter compounds produce an acid urine, chlorothiazide produces an alkaline urine; and in this respect it resembles a carbonic anhydrase inhibitor.

2. Intra-arterial (renal) injection of a mercurial diuretic always produces a diuresis which is confined to one kidney, but a similar type of administration of chlorothiazide results in a bilateral action.

3. When a high rate of sodium retention (and K^+ excretion) is produced by the administration of a corticoid like 9-α -fluorohydrocortisone, chlorothiazide can be shown to reverse this by an action which must involve the tubular cell, since chlorothiazide has no effect upon the adrenal cortex.

In order to account for this multiplicity of action it may be necessary for us to broaden our view of the site of action, to include the red cell where an inhibition of carbonic anhydrase activity may result in more complicated electrolyte changes.

New oral diuretics are continuing to make their appearance on the market and since detailed pharmacological information concerning them is not nearly so complete as it is for chlorothiazide, their properties can only be briefly summarized. Whereas hydrochlorothiazide represents a more potent and longer-lasting form of chlorothiazide, the tri-fluoromethyl analogues, flumethiazide (Ademol) and hydroflumethiazide (Saluron) are drugs with an even greater potency and longer action. Available information suggests that the pharmacological actions of the fluorinated moieties do not differ significantly from their chlorinated parents in any respects other than those mentioned. This remark may even extend to the most recently-appearing drugs benzydroflumethiazide (Natureton), trichloromethiazide (Naqua) and chlorothalidone (Hygroton).

It should be pointed out that a long duration of action may, where sensitization occurs, be a distinct disadvantage. In the same vein, increased potency is only an advantage if it is accompanied by a decrease in toxicity, or a decrease in cost to the patient; and neither of these objectives appears to have been met (4).

Perhaps of more concern to the general practitioner is the manufacturer's repeated insistence on the lessened need for attention to potassium loss induced by their products. The need for critical appraisal of these claims cannot be over-emphasized. To quote from a recent survey (4) "Despite the evidence that effective doses of chlorothiazide produce greater loss of potassium and bicarbonate in *normal* animals and man than do comparable doses of the new analogs, in *edematous* patients clinically significant differences in potassium loss with the various thiazide derivatives have not been demonstrated. Losses of potassium with any thiazide are likely to be influenced less by the structure of the derivative than by the duration and total dosage of thiazide therapy and the nature of the disease being treated. For example, some patients with severe congestive heart failure or cirrhosis of the liver, and in patients depleted of sodium by dietary or diuretic measures (such as the thiazides themselves) there is often excessive secretion of aldosterone causing accelerated loss of potassium and greatly curtailed loss of sodium. Patients are sometimes advised to liberalize salt intake with these diuretics in order to suppress the potassium loss due to hyperaldosteronism, but since the major usefulness of the drugs is to rid the body of sodium, it would seem better to impose modest restrictions of sodium intake and to use smaller doses of drug together with potassium supplements."

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THE IMPACT OF THE NEWER DIURETICS ON GENERAL PRACTICE

JOHN C. WICKWIRE, M.D.

Liverpool, N. S.

The effectiveness of diuretics in correcting the oedematous state, particularly that due to congestive heart failure, has been known for many years. Among the earliest used were ammonium chloride, urea, theobromine with sodium salicylate and theophylline (Theocin). The therapeutic result of these, when administered orally, was unpredictable due to uncertain absorption or their destruction by the liver.

Mercurial Diuretics

About a quarter of a century ago the use of organic mercury was mentioned in some text books. It was some ten years later before it was generally recognized as one of the major improvements in the relief of symptoms associated with the failing heart. In Friedberg's textbook (second edition) in the chapter on congestive heart failure, he states, "since the mercurial diuretics have been used many patients, whose outlook for life would not average more than six months to a year, have been maintained in various degrees of comfort for many years." We have all used salyrgan, mercuhydrin, thiomerin, etc., with great satisfaction, and like ether in anesthesia, they are still excellent. Mercurials by mouth were not commonly prescribed, largely due to gastrointestinal disturbances.

Thiazide Diuretics

The need for a more reliable oral diuretic was recognized. Here the research clinics in conjunction with the large drug houses have recently supplied us with a considerable selection of effective drugs: chlorothiazide, (Diuril), hydrochlorothiazide, (Hydrodiuril), benzydroflumethiazide, (Naturetin), flumenthiazide, (Ademol), benzothiadiazine, (Naqua), and others. These have heralded a great advance in the treatment of various oedematous states—cirrhotic, steroid, nephortic and premenstrual; also the oedema of pregnancy, oedema due to drug therapy, as that associated with rauwolfia and phenylbutazone (butazolidine).

I have also used these preparations occasionally in patients who suffered from periorbital and leg oedema, the etiology of which was not clear. I recall a lady, 52 years of age, who had a hysterectomy twenty years ago. There was no evidence of kidney, heart or thyroid disease, yet an effective diuresis and weight loss were obtained with an oral diuretic followed by the disappearance of the physical signs of swelling and an improvement in her sense of "well-being."

Water as a Diuretic

Water has long been known to have a diuretic effect. The use of high water intake was first proposed by Schemm.[1] At the same time it was recognized that its effectiveness was enhanced by low sodium consumption.

Biochemical Factors

Factors controlling the exchange of water and electrolytes in the presence of a low sodium—highwater intake are poorly understood. However, it has

been shown that the kidneys play a fundamental role in the regulation of salt and water in the organism. It would appear that the diuretic effect of the mercurials and the sulfonamide derivatives is due to the toxic action on the renal tubules, thus reducing their capacity to resorb salts and consequently water.

The kidneys are influenced by the anti-diuretic hormone of the posterior pituitary gland—A. D. H. and the adrenal hormones especially aldosterone. A. D. H. apparently acts on the distal tubules of the kidneys and controls the reabsorption of water. With dilution of the plasma by increased consumption of water, the osmotic pressure of the plasma is decreased which specifically affects the osmoreceptors of the internal carotid, and less A. D. H. is produced. As a consequence diuresis occurs.[2]

Many workers have found that in the oedematous patient there is an increased production of aldosterone, which is presumably responsible for the excessive reabsorption of sodium and thus the retention of water. It may be that on congestive heart failure haemodynamic changes occur which influence the adrenal gland and thus the production of its hormones.

Some workers consider that the principal factor in the secretion of aldosterone is not only the electrolytic status but the changes in the volume of body water, particularly the extracellular volume. "Acute depletion of sodium accompanied by a loss of water causes an increased production of aldosterone. If changes in the volume of body fluids are avoided by administration of water, only minor alterations in the production of aldosterone are observed."[3]

The Anti-Hypertensive Effect of Low Sodium Diets

The anti-hypertensive effect of low sodium intake has been known for many years. In 1920 Allen recognized the need of sodium restriction. In 1944, Kemptner proposed the rice-fruit diet. When salt intake is reduced to 0.5 - 2.0 gms a day there is usually a considerable drop in blood pressure. However, as most of us have experienced, few patients will continue on such an unpalatable diet for any great length of time. With the introduction of the new natriuretic agents, it was found that an amount of salt that would make food palatable could be permitted and a reduction of blood pressure obtained.

Theories Concerning the Anti-Hypertensive Effect of the Thiazides

Plummer and Yorkman state "it may reasonably be suggested that hydrochlorothiazide exerts its anti-hypertensive effect, at least in part, by combating the tendency for an accumulation of fluid and electrolytes in arterial walls, thus reducing their response to the constricting influence of catecholamines and other pressor substances.[4] Raab observed that high intracellular sodium potentiates the vaso-constrictor action of extrinsic and intrinsic catecholamines and that a decrease in intracellular sodium weakens this action. During the initiation of thiazide diuresis, there is a fall in body weight and an associated loss of extracellular fluid volume and plasma volume. It would appear that the initial fall in blood pressure is the haemodynamic consequence of dehydration. With chlorothiazide, Conway et al, in a group of 83 patients, found a reduction of 10% in systolic and diastolic pressure in 55 patients, i.e. 66%[5] With chlorothiazide, Meltzer et al reported a 10% reduction in blood pressure in 45% of patients.[6] The thiazides may enhance the effectiveness of other hypotensive drugs, as well as that of splanchnicectomy.

Diuretics in the Presence of Angina Pectoris

One would be remiss without mentioning the benefits that may accrue to the patient from the use of diuretics in the presence of angina pectoris, with and without hypertension. With careful investigation the physician may find a few rales at the lung bases, slight pitting oedema of the legs, undue shortness of breath, possibly of the nocturnal type. In such patients adequate diuresis will frequently effect a dramatic relief of symptoms. Sodi-Pallares[7] and others in the August 6, 1960 issue of the C. M. A. J., stress the importance of low sodium, high water and high potassium intake and chlorothiazide in the management of angina pectoris. There is an occasional patient in whom hypertension and physical signs of congestive failure are lacking, yet with diuretic drugs some diuresis may occur with relief of anginal symptoms and the incipient failure. One would presume that subclinical oedema is present.

I have in hospital at this time a 62 year old man who has suffered from hypertension for several years and angina pectoris during the past two years. Eighteen months ago he had a mild stroke. He also has arrested pulmonary tuberculosis with a right thorocoplasty. Rales have been present in the right chest as long as I have known him—about four years. There was no peripheral oedema or liver enlargement. Three weeks ago, he developed coronary thrombosis. Last week, on two consecutive mornings, he developed attacks of acute dyspnoea at 6 a.m. Hydrodiuril was given, whereupon diuresis occurred. There has been no return of the dyspnoea and he tells me that he now feels better than he has for several days.

If the serum electrolytes, haemoglobin, existing hypertension or congestive failure and infection are well controlled, the need for coronary vasodilators such as iproniazid or nitroglycerin will frequently be reduced, or possibly they will not be required at all.

Toxic Effects of the Sulfonamide Derivatives

With profuse or prolonged diuresis and in chronic congestive heart failure, electrolytic disturbances may occur—hyponatremia, hypoklemia, hypochloremia and a lowering of the serum bicarbonate level. With the newer diuretics electrolyte reduction with symptoms is less likely to occur. If the response is poor, before increasing the dose of diuretic one should carefully check the blood electrolytes as well as urinary sodium and potassium. Hypopotassemia occurs in 40% of patients taking the thiazides.[8] Blood levels below 4.2 mEq/L are considered by Sodi-Pollaris to represent hypokalemia.[9] Symptoms of potassium deficiency include apathy, drowsiness, anorexia and extreme weakness. Objectively one may find paralysis or muscular flaccidity, hyporeflexia, paralytic ileus, hypotension and shallow respirations.[10] Electrolytic disturbances may occasionally be detected in the electrocardiogram. In hypokalemia there may be a lowering of the T waves, a change from pointed to rounded T waves, depression of the S-T segments and an increased magnitude of the U waves. Arrhythmias may occur. In hyperkalemia the most conspicuous feature is the tall, pointed and symmetrical T waves. Reversal in direction of T waves may occur. The QRS interval may be measurably longer. Arrhythmias may occur. In acidosis also the T waves may be tall and peaked. Alkalosis may result in lowering of T waves and lengthening of the QT interval.[11]

Symptoms and Signs of hyponatremia

With the newer diuretics, sodium depletion with symptoms is less likely to occur. One should avoid blood sodium levels below 130 mEq/L. Symptoms of hyponatremia include:—

(1) Gastrointestinal—anorexia, nausea, vomiting.
(2) Neurologic—apathy, drowsiness, restlessness, confusion, convulsions, coma.
(3) Muscular weakness, cramps.
(4) Tachycardia, shock.[12]

The clinical features of hypochloremic alkalosis resemble those associated with hyponatremia.

Danger of Diuretics in the Presence of Liver Disease

In the presence of serious liver disease or long continued steroid therapy, hypokalemia may occur earlier than in the usual case, since the cation exchange with chlorothiazide is chiefly sodium, and where it is not present, the next available cation, potassium, will be excreted in fairly large amounts.[13] In such cases where the response is poor and urinary sodium output is low, one can presume that aldosteronism is a factor. On the other hand, in the presence of liver disease, patients with a good diuretic response and a high urinary sodium output, hypokalemia may occur, which may not be controlled with potassium supplements. Hepatic precoma or actual coma may develop.

Toxicity in the Presence of Kidney Disease

Far advanced renal disease is likely to restrict the clinician in his use of thiazides. Actual deterioration of the renal status may occur, with a rise in the B. U. N. With a drop in the blood pressure, it would appear that the arterial pressure head is not adequate to maintain the glomerular filtration rate. This is mainly true in patients with hypertension secondary to renal disease and who are in need of effective anti-hypertensive therapy.[14] Hypokalemia has a deleterious effect on the kidneys and renal function.

Other Dangers of Diuresis

Rapid diuresis with haemoconcentration and concomitant lowering of blood pressure predispose to coronary and cerebral thrombosis. Chlorothiazide tends to produce a rise in uric acid in the serum. Cases of gout have been reported. Hyperglycemia has also been reported in patients with incipient diabetes. In the digitalized patient, rapid diuresis may lower blood potassium and produce symptoms of digitalis intoxication.

Summary

Several new oral diuretics that have proven useful in the treatment of the oedematous patient and in the control of hypertension and angina pectoris have been discussed. The role of water as an effective diuretic is again emphasized. Theories concerning mode of action of the thiazides and water have been recorded. The thiazides frequently potentiate the effectiveness of other anti-hypertensive drugs. Potential toxicity of these drugs, especially in the presence of kidney and liver disease, is stressed. Hyperglycemia and hyperuricemia have been reported. Cardiovascular and cerebrovascular accidents may be precipitated by incautious use of these agents.

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Some Quantitative Observations on a Methamphetamine-Phenobarbital Anorexic Compound in Obese Outpatients. Simkin, B., and Wallace, L., Am. J. Med. Sci. 239:533, (May) 1960.

Many physicians feel that weight reduction is merely a matter of will power on the part of the patient and that the physician and adjuvant drugs are essentially of psychologic importance. An opposing school of thought supports the thesis that anorexic compounds are necessary for sustained adherence to a basically difficult and unpleasant regimen of dietary restriction.

In this study the value of a methamphetamine-phenobarbital anorexic agent was evaluated in 101 patients by the single-blind and double-blind method The over-all loss of weight achieved by patients taking the active medication was significantly greater in both the double-blind and single-blind groups than that achieved by patients on placebo medication. The greatest effect of drug therapy was observed from the 5th to 16th week of therapy, after psychogenic influences had largely ceased to influence the degree of weight loss.

The close similarity of results in the double-blind and single-blind studies suggest that single-blind methods are as suitable as double-blind when an objective indicator such as weight loss is used to evaluate effectiveness. The absence of a discernible difference in the influence of the physician's personality on the results under constant conditions further strengthens the validity of the observed drug effect.

S. J. S.

INTRAVENOUS LIDOCAINE WITH SUXAMETHONIUM IN GENERAL ANAESTHESIA

(A Small Clinical Study)

C. B. BURKE, M.B.

North Sydney, N. S.

The administration of local analgesic solutions by the intravenous route has been an accepted procedure for many years. Intravenous procaine used alone to provide analgesia for painful procedures and as a potentiating analgesic in general anaesthesia is well reported.

Since the advent of lidocaine, this agent has also been given in intravenous solution in place of procaine with good results. In addition to its use in general anaesthesia intravenous lidocaine has also been found to be beneficial in the relief of pain due to carcinomatosis and in the relief of labour pains. Advantages claimed by those using the drug include the relatively low toxicity in comparison with procaine, the prolonged post-operative analgesia, the low incidence of post-operative vomiting, the absence of respiratory depression and the early return to consciousness. Consequently it may be said that lidocaine constitutes a useful drug in the anaesthetist's armamentarium of intravenous agents in addition to its properties in the field of local and regional analgesia.

Procaine and suxamethonium halides are both broken down by pseudocholinesterase and competition exists between them. If procaine and suxamethonium are administered together the depolarisation block produced by the suxamethonium is prolonged, for this reason it is better not to give both to the same patient. The anti-cholinesterase activity of lidocaine compared with procaine however is 1 to 10 and consequently combination of suxamethonium and lidocaine is not only possible but possesses certain advantages.

Following a personal communication (Ruddell, J. S., 1960.) the writer decided to investigate the use of an intravenous mixture of suxamethonium and lidocaine in general anaesthesia. The results of an initial series of 100 cases are now complete.

The intravenous solution used consists of lidocaine 1000 mg. (50 c.c. of 2% solution), Suxamethonium chloride 1000 mg. (20 c.c. of 5% solution). Dextrose 5% in water to 1,000 c.c. Anaesthesia is induced with Sodium thiopentone $2\frac{1}{2}$% using a 'Sleep Dose' only. Preliminary relaxation for intubation is produced by suxamethonium 40 mg. The suxamethonium-lidocaine infusion is then started. Anaesthesia is maintained using Nitrous oxide 2 litres / minute and Oxygen 2 litres / minute in a closed circuit with a slight leak. The writer uses a Water's canister for Carbon dioxide absorption.

The rate of the intravenous drip varies between individuals but may be easily adjusted to provide complete apnoea and relaxation for controlled respiration with manual inflation. It has been found that a flow-rate of about 60 drops per minute is adequate for most patients and provides satisfactory operating conditions. In any case the total quantity of the mixture given should not exceed 500 c.c. in the first hour and may be reduced during the second and subsequent hours of anaesthesia. During the early minutes of the operation it is useful to slow down the infusion in order to recognize those few individuals who show hypersensitivity to suxamethonium. As soon as respirations start to return the drip-rate can be increased until apnoea is again complete.

This anaesthetic procedure has been used in a variety of different cases which comprise the work of a busy general surgical department.

Results

Relaxation has been highly satisfactory. In the majority of cases this has been produced by a smaller quantity of suxamethonium than would be expected from previous trials of continuous suxamethonium alone. The solution is discontinued during skin closure, and in all except four patients respiration returned before the last suture was inserted. Four patients, who did not respond immediately, ventilated normally within five minutes of termination of the solution.

The anaesthetic gases are turned off when respiratory tidal exchange is considered adequate. All patients recovered consciousness and opened their eyes within two to three minutes, most of them were able to converse intelligently before leaving the operating room. Post-operative vomiting seemed much less than with other Nitrous Oxide / Oxygen / Relaxant techniques although no statistics are available yet. Post-operative analgesia was excellent and further medication was not needed until much later in the post-operative period than usual. There were no complaints of muscle pain following operation. No signs of twitching indicating overdosage with lidocaine were observed, but care must be taken to avoid giving more than the recommended dosage, for, presumably, lidocaine induced convulsions would be masked by the suxamethonium.

A total of 100 patients have received lidocaine with suxamethonium during anaesthesia. These cases have included; appendectomy—21 cases; intestinal obstruction—8 cases; resection of bowel—3 cases; gastrectomy—4 cases; lumbar sympathectomy—1 case; hysterectomy—8 cases; cholecystectomy—7 cases. Patient's ages have ranged from 5 years to 84 years.

Conclusion

Despite some variance of opinion it would appear that lidocaine possesses anti-cholinesterase properties and hence potentiates the action of suxamethonium, but to a lesser degree than procaine. Intravenous administration of a lidocaine-suxamethonium mixture has been shown to produce:

1. Potentiation of myoneural block.
2. Potentiation of analgesia, allowing low concentrations of anaesthetic gases to be used.
3. Rapid recovery of consciousness after the operation.
4. Prolonged post-operative analgesia.
5. Reduced incidence of post-operative vomiting.

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IMPENDING CARDIAC INFARCTION AND NON-SPECIFIC ELECTROCARDIOGRAPHIC ABNORMALITIES

G. W. MANNING, M.D.*

It has been known for many years that myocardial infarction is frequently preceded by premonitory symptoms. These prodromal symptoms occur as chest pain and frequently are accompanied by non-specific electrocardiographic changes. A number of case reports have appeared in the literature with regard to this prodromal syndrome and a number of terms have been applied to it such as "coronary failure," "acute coronary insufficiency," "intermediate coronary syndrome" and "slight coronary attacks." Various series[2,3] have reported prodromal pain with a frequency of from 30 to 50 percent of all cases experiencing cardiac infarction. The onset is usually mildly acute or subacute in nature. The patient, from his state of relatively normal health or good health, suddenly finds himself unable to walk more than a few yards without some chest pain or he may have had a prolonged attack of angina, at rest, particularly after food. The pain is often relieved by nitroglycerin and there is no fever, leukocytosis, sedimentation rate increase or increase in transaminase level. The blood pressure in these cases does not fall. There is no friction rub to be heard and no other evidence of infarction. An electrocardiogram may show nothing or may show ischemic depression of the RS-T segment or non-specific T wave changes. The pathological basis of this syndrome is quite variable. However, in view of its rather acute onset, it may be considered to represent coronary thrombosis but without accompanying myocardial infarction. The outcome is also quite variable. The patient may return to a normal state soon after the episode; he may go on to myocardial infarction or he may continue with angina of effort. The fatality rate in this group of patients with prodromal symptoms has been reported from 15-43 percent by different observers.

Soon after the introduction of anticoagulants, several cardiologists suggested that these agents might be used to prevent myocardial infarction in those patients who presented with prodromal symptoms indicative of impending infarction. From our knowledge of the pathology of ischemic heart disease, it is apparent that infarction is usually a process which occurs over a variable period of time with increasing narrowing of the coronary vessel due to gradual thrombus formation. It would seem reasonable that anticoagulants might prevent further thrombus formation and thus avoid complete occlusion and myocardial necrosis.

However, the situation is not as simple as it appears, for the diagnosis of impending infarction (before it has occurred) is by no means easy. Furthermore if we are to treat all suspected cases, there is danger of treating many individuals who do not have ischemic heart disease. Some 10 years ago I sought an opinion regarding this from my former teacher Sir John Parkinson. He said it would be of great value if we could forecast with a high degree of accuracy which patients were going to develop infarction for it would be quite reasonable to use anticoagulants in the hope of preventing the catastrophe. However, in his opinion, this may well encourage "unwarranted heart disease"

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*From the Cardiovascular Unit, Victoria Hospital, London, Ontario, Canada. Delivered at H.M.C.S. Cornwallis, Dalhousie Post-Graduate Division Clinical Day, May 18, 1960.

and if we taught this approach we might well create a far greater incidence of coronary disease than actually existed.

We have no difficulty in the diagnosis of the typical or classical case of cardiac infarction in which the clinical and laboratory findings, including the electrocardiogram, all confirm the diagnosis and our treatment here is fairly straight forward. It is with the atypical cases that we have difficulty, those in which the sedimentation rate, white blood count, transaminase, etc. does not reveal significant alterations and the electrocardiogram shows either no abnormalities or non-specific changes (particularly T wave variations). A considerable number of these atypical cases occur and I am sure that many have the premonitory or prodromal symptoms of cardiac infarction. In a considerable number the typical E.C.G. changes of infarction develop over a period of days to weeks. An example of this is well illustrated in a recent case, in which the patient complained of anginal-like attacks for some time prior to consulting his physician. The patient was anxious, fatigued and in a state of general malaise and unhappiness, complaining of vague chest pain. The electrocardiogram revealed non-specific T wave changes but none of the other laboratory findings indicated myocardial necrosis. Eventually this patient developed (in spite of anticoagulant therapy) the findings of myocardial infarction, with the typical E.C.G. pattern of a posterior cardiac lesion, over a period of some weeks.

These patients are usually middle-aged males who present with substernal or other chest pain, often of a vague nature, which may or may not be associated with exertion. The patient often complains of undue fatigue, lassitude, general malaise, and often presents himself on the advice of his wife and is frequently embarrassed or somewhat reticent about consulting you at all. This is particularly true if the victim is a physician. From experience I have come to the conclusion that we should not dismiss these cases lightly but should regard the atypical case with care and caution. Observe him at rest for a number of days, usually on anticoagulants and with serial electrocardiograms until you are sure that infarction has not occurred. I can recall a case of a few years ago in a physician who presented vague symptoms suggestive of cardiac ischemia. The electrocardiogram, however, was completely normal and a Two-Step Exercise Stress Test, carried out two or three days prior to the time that he developed cardiac infarction, was normal.

A number of reports have appeared in the literature claiming the prevention of cardiac infarction by the use of anticoagulant drugs during the prodromal stage of infarction. (Wood,[4] Beamish[1]). This is very difficult to assess for the case may have never infarcted even if anticoagulants had not been used. I would feel, however, that the most practical approach is to use anticoagulants in ischemic heart disease as follows: (1) Cases of proven cardiac infarction. (2) Cases of previous myocardial infarction who, having been free of angina, complain of obvious anginal symptoms including those cases who have experienced some angina and suddenly develop a marked increase in symptoms. (3) Selected cases with definite prodromal symptoms and non-specific E.C.G. changes. If there is a reasonable suspicion of impending infarction, even though the E.C.G. is normal, anti-coagulant therapy could be initiated and, after a short period lf observation including serial electrocardiograms, discontinued when no evidence is forthcoming to support the diagnosis.

Non Specific E.C.G. Changes

It is of considerable importance to view the E.C.G. in the light of the clinical findings. When non-specific abnormalities occur in the presence of prodromal symptoms suggestive of ischemic heart disease, we have useful evidence of a myocardial effect likely due to ischemia or impending infarction. On the other hand, when such E.C.G. changes are encountered in a fit individual on a routine examination we should search very carefully for physiological or environmental factors which explain the abnormality, rather than accept it as indicative of ischemic heart disease. In this regard, I would suggest to all who are using the E.C.G. in the course of their practice that, in cases presenting the non-specific E.C.G. patterns and often only minor abnormalities, the assistance of a cardiologist familiar with all phases of electrocardiography be obtained in these bizarre and often difficult E.C.G. problems.

Summary

Impending cardiac infarction has been briefly reviewed, indicating the historical development of its recognition, the difficulties of its diagnosis, the importance of electrocardiogram findings, and the necessity for prompt anticoagulant therapy. The ease of over-diagnosis, particularly when minor E.C.G. abnormalities are not correlated to clinical findings, has been emphasized.

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(Dr. Manning's paper was illustrated by informative lantern slides of electrocardiograms which could not be reproduced satisfactorily on our small page format. We regret our inability to include them. The Editor).

OUR LEGAL ADVISER LOOKS AT MALPRACTICE

DUNCAN K. MacTAVISH*

(This abstract gives the practicing physician some insight into the frame of reference in which the law judges the quality of medical practice. The entire annual report of the Canadian Medical Protective Association should be carefully studied by every physician, especially if he is not a member of the Association. The Editorial Board is indebted to Dr. T. L. Fisher, the Secretary-Treasurer of the Association, for permission to reprint this material. The Editor).

Does it seem that the law is cold and unrealistic and impersonal, imposing on you an unreasonable and unanswerable burden which leaves you completely at the mercy of any unscrupulous patient who wants to take the trouble to institute suit? If so, let me assure you that such is not the case.

You are not an insurer. You do not guarantee a cure or even that the treatment you prescribe will succeed. What you are expected to bring to the treatment of each case is that degree of skill and care which a normally skilful practitioner in your particular field or specialty can reasonably be expected to exercise in a similar case or in similar circumstances. The lowest level of competence will not suffice but, by the same token, you are neither expected nor required to possess the highest possible degree of skill known to your profession. What the law requires of you is simply a fair and reasonable standard of care and competence. But this does not mean that all doctors will be judged by the same absolute standard; those of you who hold yourselves out to the public as specialists having a specialized skill and training must be able to demonstrate such a specialized skill and you will be judged on the basis of what may be fairly and reasonably expected of men with your background qualifications.

The English case of Roe v. Minister of Health which was decided in 1954 on facts which arose in 1947, probably sums up as well as any the modern attitude of the courts in malpractice cases. In that case two patients underwent operation on the same day and in both cases a Dr. Graham, a specialist anaesthetist, injected nupercaine by means of a lumbar puncture. Following operation both patients became permanently paralysed from the waist down and it was agreed that this was the result of phenol having contaminated the nupercaine. The nupercaine, in glass ampoules, had been stored in a solution of phenol which apparently leaked into the ampoules through invisible cracks or molecular flaws. In any event the evidence disclosed that the ampoules had been examined before use and no cracks or other damage was apparent. Both patients sued the doctor and the hospital for damages. The Judgment of Lord Justice Denning in that case reads in part as follows:

> "I approach this case, therefore, on the footing that the hospital authorities and Dr. Graham were called on to give an explanation of what happened. But I think they have done so, they have spared no trouble or expense to seek out the cause of the disaster. The greatest specialists in the land were called to give evidence. (Lord Justice Denning then reviewed the facts as brought out in the evidence and continued) That is the explanation of the disaster, and the question is; were any of the staff negligent? I pause to say that once the accident is explained, no question of res ipsa loquitur (the thing speaks for itself) arises. The only question is whether on the facts now ascertained anyone was negligent. Leading

*General Counsel, Canadian Medical Protective Association, Ottawa, Ont.

counsel for the plaintiffs said that the staff was negligent in two respects: (1) in not colouring the phenol with a deep dye; (2) in cracking the ampoules.

I will take them in order: (1) the deep tinting. If the anaesthetists had foreseen that the ampoules might get cracked with cracks that could not be detected on inspection they would, no doubt, have dyed the phenol a deep blue; and this would have exposed the contamination. But I do not think that their failure to foresee this is negligence. It is easy to be wise after the event and to condemn as negligence that which was only a misadventure. We ought always to be on our guard against it, especially in cases against the hospitals and doctors. Medical science has conferred great benefits on mankind, but these benefits are attended by considerable risks. Every surgical operation is attended by risks. We cannot take the benefits without taking the risks. Every advance in technique is also attended by risks. Doctors, like the rest of us, have to learn by experience; and experience often teaches in a hard way. Something goes wrong and shows up a weakness, and then it is put right. That is just what happened here. Dr. Graham sought to escape the danger of infection by disinfecting the ampoule. In escaping that known danger he, unfortunately, ran into another danger. He did not know that there could be undetectable cracks, but it was not negligent for him not to know it at that time. We must not look at the 1947 accident with 1954 spectacles.

The judge acquitted Dr. Graham of negligence and we should uphold his decision.

One final word, these two men have suffered such terrible consequences that there is a natural feeling that they should be compensated. But we should be doing a disservice to the community at large if we were to impose liability on hospitals and doctors for everything that happens to go wrong. Doctors would be led to think more of their own safety than of the good of their patients. Initiative would be stifled and confidence shaken. A proper sense of proportion requires us to have regard to the conditions in which hospitals and doctors have to work. We must insist on due care for the patient at every point, but we must not condemn as negligence that which is only a misadventure."

Reprinted from the Annual Report of the Canadian Medical Protective Association 1959.

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).

REFLECTIONS ON THE NATURAL HISTORY OF ERROR

It is a highly dubious honour to be asked to review a paper of the quality of "The Natural History of Error". It is depressing to realize the many neatly classified types of error which doctors have initiated, subscribed to and perpetuated. The inevitable conclusion would appear to be that if to err is human, practitioners of the art of medicine have eminently proved their place in the forefront ranks of humanity. The author, Dr. William Bean of Iowa City, accepts this but he is indulgent of the frailties inherent in the nature of man and quotes Milton to the effect that "Truth never comes into the world, but like the bastard, to the ignominy of him that brought her forth; till Time, the mid-wife, rather than the mother of Truth, hath washed and salted the infant and declared her legitimate."

Among the many types of error, Dr. Bean illustrates the Error of Judgment on the use of the French waterproof celluloid truss designed for hernia patients who wish to swim. The incident in question concerned a man who, having enjoyed his immersion in the Mediterranean, was drying himself in his beach hut when he backed into a lighted cigarette on the edge of the table on which he had placed it. The celluloid burst into flame and the victim shot out of his hut at a velocity that would have excited the envy of all Cape Canaveral. He tore through the amazed spectators on the beach and emitting large quantities of smoke and steam, entered into the natural saline bath awaiting him. When the local gendarmerie on the beach demanded that he submit himself and his infernal machine for investigation, he refused with a modesty rather rare in the history of the French.

While reading Dr. Bean's most interesting article, my thoughts were led by his many illustrations to another course. By the nature of their relation, doctors and patients continually confront each other with unusual situations and much of the fascination of the practice of medicine must depend on this. My own experience began in a very modest way when I first started practice in a Nova Scotia village. One of my first patients was a lady with part of a needle buried in her hand. After injecting a local anaesthetic and searching for a long time, I finally got the end of the broken needle in the jaws of an artery forceps. Then, with a triumphant expression and a dexterous twist of my not particularly muscular wrist, I flicked the needle out. It sailed up in the air and then down into the top of the front of the lady's dress and into the mysterious depths of the regions below. Being by nature a peaceful man and shy to boot, I did not pursue my search further.

To cite another example, I remember being called to the Victoria General Hospital to see an Indian boy of about 18. Reposing high up in his rectum, with the sphincter tightly closed below it, was a large coca-cola plastic glass bottom end down. When I asked him how it got there, he told me his best friend had put it there when he, the patient, was sleeping in his bunk. I had heard of removing turnips by obstetrical forceps, wooden objects with the aid of a gimlet and glasses with the open end down by filling with wet plaster of Paris bandage, allowing to set and pulling on the ends of the bandage left hanging down. None of these methods being feasible, I had to break the plastic glass with forceps and remove the pieces separately with my fingers. The following day he was none the worse physically but I am sure, from his thoughtful expression, that he now realized a boy's best friend was his mother, after all.

Sometimes the experiences one remembers evoke feelings that are poignant rather than Rabelaisian. I remember that in my first job as House Surgeon in an English hospital, I was required to give anaesthetics. The patient on one occasion was a boy of abour four or five years and as I placed the mask on his face he said, "Could I say my prayers first?" He then recited the usual "Now I lay me down to sleep" and when he came to the peculiarly appropriate "If I should die before I awake", he seemed to realize its special significance. I remember his complete composure when the prayer was finished and how easily he took the anaesthetic.

Every doctor meets and has met many unusual situations. Perhaps we might induce Dr. Shane to offer a prize (a troupe of performing tubercle bacilli, perhaps?) for the most unusual experience encountered by a reader of the Nova Scotia Medical Bulletin.

Finally, since we have come a long way from Dr. Bean, we should return to him for a last word. He states, "Our aim, then, must not be to deny error but to learn from it." His article is to be found in the A.M.A. Archives of Internal Medicine 105:2, Feb. 1960 and is very well worth the trouble of seeking it out.

W.E.P.

LETTER TO THE EDITOR

THE SOCIETY OF AMATEUR MEDICAL EDITORS
SOUTH WESTERN DIVISION

"Bull's Height,"
R.R. No. 2. Independence,
Missouri, U. S. A.
January 17, 1961

To the Editor:

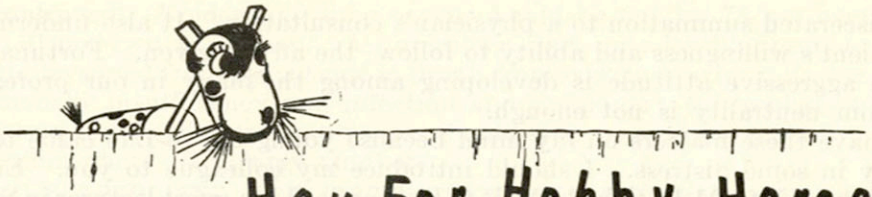
Professor Hees C. Nile of Vancouver and Dr. Willis Fafnir of Toronto have each sent copies of the December, 1960 and January, 1961 issues of your journal. Both have protested, in the strongest terms, the reports of our San Francisco meeting which appeared in these issues. I have taxed Benge and Jim Tallgrass for the lapse in judgment that sent Dr. Frater to our meetings.

I have conducted a telephone poll of the Executive of the Society and received unanimous agreement to the following opinion: You as editor, on pain of expulsion from the Society, are required to restrain Dr. Frater from making any further account of the affairs of this Society, especially material relating to the study groups devoted to the topics "The Biometrician Looks at Las Vegas" and "The Cultural Values of Classical American Burlesque." Professor Nile and Dr. Fafnir have generously agreed to withhold a formal complaint against you but deplore, as I do, the superficial way in which their investigations and our meetings were reported. If your readers indicate a genuine interest in our affairs, by a significant number of letters to you requesting further reports of the S. A. M. E. studies, we will consider release of a report from this office.

More in sorrow than anger, I am

Editorially yours,

Pierre Parti-Pax, M.D.
Executive Secretary.



Hay For Hobby Horses

THE VANITY OF MEDICAL WISHES

"Wishing don't make it so . . . . ."

From a popular song.

I swore off the reform kick, years ago, because I believe, like Lincoln Steffens, that reformers are a dangerous crowd and that it is better to pay the professional his percentage and get an efficient job of work done. I am sceptical of any educational programmes except those that are persistent and thorough-going, particularly when the public seems to love the fool's gold of modern advertising more than the cold comfort that serious education offers them. Samuel Butler spoke for many when he said "The older I grow, the more convinced I become of the folly and credulity of the public, but at the same time the harder do I see it is to impose oneself upon that folly and credulity." If the physician wants the patient to conserve his health he must do much more than 'advise' him.

At one time I said that, as far as health education went, my job was to give information and my patients, or yours if they are within range of my voice or my pen, could take my advice or leave it. That is a comfortable position to hold and I wish my conscience always would let me stay there but I am bedeviled, now and then, by an echo of other times and, perhaps, of another morality that says "to whom much is given much shall be required." This is the gadfly that torments us. Must we, as a profession, provide the *leadership* in the solution of health problems that have a socio-economic basis, as well as produce the knowledge that makes the solution possible? If so, what is the minimum individual action consistent with our professional responsibilities in the face of this or any other definite relationship between behavior and serious human disease?

A rueful, if not sarcastic, remark is going the rounds among the tobacco buffs to the effect that "The physician who takes the cigarette out of his mouth while he is advising his patient not to smoke considers himself to be *an impartial expert in the field of Tobacco and Health.*" Some of you may bridle at this caustic reminder that he who smokes teaches the smoking habit, but no better example of our problems in leadership lies so close at hand nor one that has so many ramifications in the areas of social custom and commercial expedience. The individual physician does not have to be a tobacco buff or even to have stopped smoking to see this problem. There are other obvious but less pointed examples and all turn on the *duty of example.* In the past, a distinguished member of our profession said "the physician is a signpost pointing the way to health, he is not the leader of the column proceeding thence" (Almoth Wright—1895). But from time to time, it must occur to the most hardened of us that the advice "do as I say and not as I do" is a weak

and eviscerated summation to a physician's consultation. It also undermines the patient's willingness and ability to follow the advice given. Fortunately, a more aggressive attitude is developing among the many in our profession for whom neutrality is not enough.

I have these matters on my mind because young Sayve-Life came to me recently in some distress. I should introduce my colleague to you. Ernest Wood Sayve-Life M.B., Ch.B., M.R.C.P. (London) is a guest lecturer in Social Medicine at the Medical School. I get along well with Woody because he is well trained and is a sound and practical physician. His views on general medical topics reflect expert opinion although he is in advance of the average and the orthodox. I have to sit on him, from time to time, because in medico-social matters he expects too much, too soon. However I do not lose any sleep over Woody because I believe, as does Thoreau, that "any man more right than his neighbours is already a majority of one." In any case, Woody was asked to be principal speaker at the forthcoming annual dinner of the women's division of the Health League of Canada. He was given the topic "The Women's Share in Health Maintenance." Woody had mother wit enough to try to refuse, but once trapped, he went to work on the assignment in his usual energetic fashion. When he came to me he was oscillating between exasperation and depression. "Frater, old man, the average citizen is an innocent babe in health matters. He will believe any rag-tag of magic but is thrown into confusion by the plain truth. If I get up and tell them the truth, they will become anxious and disturbed. What's more their family doctors will send me to Coventry. What can I do and remain honest?" I calmed him down and said "Relax, Woody, after a few years you will get tired, as I did, and stop trying to teach the multitudes anything. You can do something with the public, one by one, in your office if you take the time to do a thorough-going way but you can do very little with him in job lots. Why not just give them the same old stuff everybody else does with the occasional dramatic fact or statistic tossed in, as raisins in the pudding." Of course, Woody couldn't do this and I am afraid he will be quite uncomfortable, over the whole matter, to no real purpose.

After he had gone I amused myself by jotting down a dialogue between young Sayve-Life, the medical innocent, and The Devil's Advocate. In this, the theme is *what could be* and the refrain *what is*.

SAYVE-LIFE: The people value good health and will do anything to get it and keep it.

DEVIL'S ADVOCATE: Health is the last thing people want to think about when they are well. The average man's philosophy is far better summarized by the phrase "eat, drink and be merry." If you want an indication of how people regard health, examine the priority given to pre-paid medical insurance in the average Canadian budget.

SAYVE-LIFE: A positive program of health maintenance based upon a disciplined way of life including regular exercise, the avoidance of obesity, abstinence from addicting drugs and pollutants would reduce our losses in cardiovascular disease, chronic pulmonary disease and cancer within two decades.

DEVIL'S ADVOCATE: The average man is psychologically incapable of taking action *now* to prevent disease *later*. He will resent and, by various conscious or unconscious strategems, resist your attempts to save his life in advance, particularly if it means more work or less pleasure for him.

SAYVE-LIFE: Tobacco-related diseases could be reduced by a substantial amount if the cigarette were made safe or the habit of cigarette smoking were

discontinued. Epidermoid lung cancer would be cut by 75 per cent in men and 50 per cent in women. An unknown, but considerable, number of lives would be saved from reductions in death from coronary artery disease, chronic pulmonary insufficiency and infection and pulmonary tuberculosis of the re-infection type. Tobacco companies should stop repaying their best customers by killing them off in large numbers.

DEVIL'S ADVOCATE: This example proves my case better than any other. These facts have been widely known to the public for five years or more. An intensive education campaign in Edinburgh last year on the relationship between cigarette smoking and lung cancer achieved negligible results. The panders who sell cigarettes on television are among the best paid of any performers in this medium. (I objected to this word but Frater points out to me that the dictionary defines pander as 'a minister to the passions of others'. Ed.) If the hypothesis, that compulsive cigarette smoking is nicotine addiction, is true then many adult smokers cannot help themselves. In any case, too many Canadians profit from the tobacco business to expect any serious opposition to the advertising and sale of cigarettes even though the business is becoming more costly. It is estimated that, of U. S. children now in school, one million will die of epidermoid carcinoma of the lung before they reach the age of seventy.

SAYVE-LIFE: In school health studies and in public health work in venereal disease, it is found that many young people contract these diseases or become an unwilling partner to an illegitimate pregnancy chiefly through ignorance. In a British study, 36 percent of the prostitutes were in the 15-20 year age group, of the teen-age prostitutes 2.7 percent had syphilis and 49 percent had gonorrhoea. They lacked even the most rudimentary knowledge of sexual hygiene. Sex education in our schools, beginning well before the age of leaving for the most backward members of the class, would save many of these young people.

DEVIL'S ADVOCATE: Could be, but the "better people" in our communities repeatedly refuse the offer of public health agencies to conduct such training in our high schools. They are sure their own children don't need it and are afraid of attracting attention to this disturbing subject.

This exercise would have been lost on Dr. Sayve-Life and physicians of his type. May God prosper them! He would look at me sharply to see if I was serious, shrug and say that we have no idea how effective health education will be in the prevention of disease until we give it a serious try. If I batted an eye, he would rattle off a summary of the few good studies on the effect of health education to keep me at bay. I may kid Woody and his crowd but they have the right idea. Think of the vast opportunity for continuing health education, presently neglected, in our offices, in school health services, in our hospitals (where education as a considered part of treatment is wellnigh incomprehensible to the disease-oriented physician) and, God forgive us, in our homes. I wonder too how much we would increase our total effectiveness, as physicians, if we again put on the ancient mantle of authority and backed our advice with our augmented personalities. Did the awe-inspiring physician of the good old days, when pharmacy was puny but the personality was powerful, have any more luck with his obese patients than do I?

Here's to authority as an article of therapy.

BROTHER TIMOTHY

BOOK REVIEW

INFECTIOUS DISEASES OF CHILDREN. Saul Krugman and Robert Ward. Second Edition. The C. V. Mosby Company, St. Louis, 1960, 381 pp. $13.00.

The authors modestly state in the preface to the first edition that "the purpose of the book is to provide a concise and handy description of certain common infectious diseases of children." This is, indeed, a modest description of a most comprehensive and detailed reference book for the practising doctor. A few important diseases such as tuberculosis, syphilis and malaria, and several rarer entities are omitted, because as the authors say of their "limited personal experience." We regret the omission, but accept their reason.

The book is written for a wide public; for, while being practical enough to suit the busy doctor who wants a quick reference in differential diagnosis, it has also a considerable amount of bacteriological, virological and pathological detail to suit the more scientifically minded. The chapters are arranged alphabetically by common disease names—a down-to-earth approach—and as there is thus no special sequence, chapters can be read at random satisfactorily. The coloured plates, of which there are seven, and a chart are beautifully reproduced and useful for differential diagnosis. As well as many excellent chapters on the well-recognized disease entities such as measles, chicken-pox etc., there are several on the other viral infections, encephalitis, entero-viral infections, and acute respiratory infections, including laryngitis, croup and laryngo-tracheo-bronchitis. These chapters give much up-to-date information of the "newer" diseases with many recent references. One feels that differentiation clinically is not quite so simple as implied but the discussion is at least helpful. The need for adequate regional laboratory facilities is emphasized and we are fortunate in this Province in having such facilities readily available.

From the public health point of view the techniques in management of certain types of cases are well-covered, e.g. salmonella and shigella infections. The regulations for dealiug with a case of smallpox are listed, and although the disease is very rare in this country, the rapidity of air-transit makes its recognition important. The tricky question of rabies immunization is discussed, as well as all other preventive immunizations.

The final chapter written by Dr. Morris Greenberg is concerned with the control of communicable diseases and contains a great deal of material suitable for nursing teaching programs. Care both in hospital and in the community is covered.

I feel that this book would be a most valuable addition to the library of any doctor who has in his practice a large number of children.

M. H. R.

PERSONAL INTEREST NOTES

Cape Breton Medical Society

Dr. Alex Guthro, Little Bras d'Or was a patient in St. Elizabeth Hospital, North Sydney.

Dr. Humbert Giovannetti is now improved and has returned home after having been a patient in St. Rita's Hospital.

Cumberland Medical Society

Dr. David Kernohan, Parrsboro, was visiting his relatives in Ireland recently.

Dr. H. E. Christie recently attended the meetings of the Medical Economics Committee of the Canadian Medical Association as the representative for Nova Scotia.

Dr. George Saunders recently attended the Royal College of Physicians and Surgeons meeting in Ottawa and then went on to the American College of Surgeons convention in Mexico City.

Dr. Norman Glen, Amherst, is attending the Victoria General Hospital for three months as a part-time resident in anaesthesia.

Halifax Medical Society

Dr. Ronald M. Ritchie, paedetrician, recently removed his office to 276 Robie St., Halifax.

The two local medical fraternities held their annual initiation banquets and Annual Balls recently, Alpha Eta of Phi Rho Sigma on January 12 and 13, 1961 and Nu Sigma of Phi Chi on January 18 and 20, 1961.

Dr. John Cameron MacDonald, (Dalhousie 1951) has recently begun the practice of radiology in Windsor, Ontario.

Births

To Dr. and Mrs. Cyril W. Bugden (née Margaret Barrett) a son, Halifax Infirmary, January 4, 1961.

Dr. and Mrs. Craig Campbell (née Ann Ashley) a daughter, Leslie Ann, Aberdeen Hospital, New Glasgow, January 9, 1961.

Dr. and Mrs. Richard H. James, a son, Geoffrey, Grace Maternity Hospital, December 26, 1960.

Dr. and Mrs. Anthony Lamplugh, a son, December 9, 1960.

Dr. and Mrs. Lester Wiseman (née Isabel Marshall) a daughter, Lesley Ann, Dawson Memorial Hospital, Bridgewater, on January 4, 1961. A sister for Barry.

Congratulations

To Dr. and Mrs. Walter K. House, Halifax on the marriage of their son, Rhys David, to Shirley Elizabeth Walker at Halifax on January 14, 1961.

To Dr. and Mrs. Robert Matheson, Sydney City Hospital, on the birth of their son in December 1960.

To Dr. and Mrs. Patrick J. Gouthro, Sydney, on the birth of a daughter.

To Dr. and Mrs. Louis W. Christ, Sydney, on the birth of their son in January, 1961.

(Editor's Note: These last three were not listed under the births section, because we did not have available the exact dates.)

Coming Events

March, 1961—The Canadian Red Cross Society is soliciting assistance for its welfare program during the month.

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.

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

May 4-6, 1961—A Symposium on Paediatric Surgery at New York University Medical Centre, 550 1st Avenue, New York City, under the direction of Dr. Donald A. Davis, Professor of Clinical Surgery. For information write: Office of the Associate Dean, New York University, Post-Graduate Medical School, 550 1st Avenue, New York 16, New York.

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. Arthur Ernest Doull, 56, Halifax, died suddenly of a heart attack on January 7, 1961. He had gone to the Halifax International Airport to greet his father returning from Montreal, where he had been visiting his other son. Dr. Doull, was born and educated in Halifax and was a Dalhousie Medical School Graduate of the Class of 1928. After a year of post-graduate training at Harvard Medical School, he returned to Halifax to become associated with his father, Dr. A. E. Doull, Sr., and Dr. R. E. Mathers in the practice in Eye, Ear, Nose and Throat. He subsequently became Professor of Ophthalmology and Otolaryngology at Dalhousie University and a Consultant to the Schools for the Blind and for the Deaf in Halifax. He was past-president of the Nova Scotia Provincial Medical Board, the Halifax Medical Society, and of the Medical Staffs of the Victoria General and the Halifax Children's Hospitals. He is survived by his wife, three daughters, one son, and his father, and one brother.

Sympathy

To Dr. C. M. Bethune, Medical Superintendent of the Victoria General Hospital, Halifax, on the death of his brother, Robert J. Bethune, New Glasgow.

To Dr. and Mrs. R. L. Aikens, Halifax, on the death of Mrs. Aikens' mother, Mrs. E. M. Rees, on January 14, 1961.

(Editor's Note: There follows a list of successful Fellowship and certification candidates for 1960 examinations of the Royal College of Physicians and Surgeons of Canada, recently completed. Since the total number for Canada includes 174 successful Fellowship candidates and 497 successful Certification candidates, we have included only those who are local graduates, local residents, or who are known to have taken some local training.)

CERTIFICATION

SPECIALTY	NAME	UNIVERSITY GRADUATION	ADDRESS
Anaesthesia	BLAKE-KNOX, Peter E. A.	Trinity College, Dublin 1950	Bedford, N. S.
	DRYSDALE, Alan A. MacD.	Dalhousie 1955	Halifax Co., N. S.
	SMITH, John Arnold	Dalhousie 1955	Windsor, N. S.
Int. Medicine	CAIRNS, Joseph May	Dalhousie 1954	Armdale, N. S.
	EPSTEIN, Hans Harold	Dalhousie 1956	Lancaster, N. B.
	MACDONALD, Austin Alexander	Dalhousie 1942	Victoria Co., N. S.
	SPURRELL, Pierce Augustus	Dalhousie 1953	Minnesota, U.S.A.
	TRASK, Beverley Campbell	Dalhousie 1955	Halifax, N. S.
	VINCENT, Merville Oulton	Dalhousie 1955	Guelph, Ont.
Paediatrics	AFTAHI, Farhang	Tehran Univ. Iran 1955	Halifax, N. S.
	PARKER, Kenneth Ross	Dalhousie 1956	Charlottetown, P.E.I.
Pathology	KERENYI, Norbert Andrew	Budapest, 1952	Halifax, N. S.
Phys. Med. & Rehab.	BASHOW, Lynn Elwyn	Dalhousie 1943	Fredericton, N. B.
Psychiatry	RADO, Eva	Manitoba 1956	Woodside, N. S.
	STACEY, Donald Leslie	Dalhousie 1956	Dartmouth, N. S.
Diag. Radiology	GRANTMYRE, Edward Bartlett	Dalhousie 1956	Armdale, N. S.
	MACDONALD, John Cameron	Dalhousie 1951	Windsor, Ont.
Diag. & Ther. Rad.	MERRIAM, Richard Kerr	Dalhousie 1952	Red Deer, Alberta
Orthopaed. Surg.	COWAN, John David Earle	Dalhousie 1951	St. John's, Nfld.
Urology	BONNELL, George Elmer	Dalhousie 1953	Lachine, P.Q.

FELLOWSHIP

Paediatrics	EDDY, George Everett	Dalhousie 1954	Edmonton, Alberta
General Surgery	INGLIS, Frederic Graham	Dalhousie 1955	Montreal, Que.
	KOVEN, Irving Herschel	Dalhousie 1954	Toronto, Ont.
Obs. & Gyn.	MOORE, Donald Charles	Dalhousie 1953	Don Mills, Ont.
Plastic Surgery	ROSS, James Frederick	Dalhousie 1951	Halifax, N. S.
Urology	CHESLEY, Arthur Evans	Dalhousie 1955	St. John, N. B.

Housing Application Form

108th Annual Meeting
The Medical Society of Nova Scotia
Keltic Lodge, Ingonish, N. S.
Monday, Tuesday, Wednesday, June 12, 13, 14, 1961

Dr. C. J. W. Beckwith,
Medical Society of Nova Scotia,
77 University Avenue,
Halifax, N. S.

Please reserve for me the following:—

A. Main Lodge

( ) Double room with bath—twin beds—including meals $14.50 per day.

B. In Cottage

( ) Cottage with sitting room and two twin bedded bedrooms—including meals $14.50 per person per day.

I WILL EXPECT TO ARRIVE JUNE ..... A.M. ..... P.M. .....

I WILL EXPECT TO DEPART .....

Names of persons who will occupy above accommodations:

Name .....

Address .....

In view of the attendance expected, no single rooms will be available at the Keltic Lodge, unless cancellations permit. If coming alone please check here.....if you are willing to share a room. If you have a preference for some party to share a double room with (or couple(s) to share cottage with) please insert name(s) below:

I would prefer to share accommodation with

Name .....

Address .....

Name .....

Address .....

Signed..... Date.....

Let's talk about our June affair.

What — a June wedding? A grand idea.

Let's make it a date and lead her down the ISLE — and all unite at KELTIC LODGE, Ingonish, 12 - 13 - 14 June for the 108th Annual Meeting of The Medical Society of Nova Scotia.

Dr. C. G. Harries, Chairman of the programme committee for the 108th annual meeting, 1961, announces that Dr. Walter C. MacKenzie of Edmonton, Alberta, will be one of the guest speakers at Ingonish. Dr. MacKenzie is a distinguished alumnus of Dalhousie, a graduate in medicine of the class of 1933. This class also included Dr. Ronald A. Thompson, President of the Royal College of Physicians and Surgeons, and Dr. F. J. Granville, the President of The Medical Society of Nova Scotia. Dr. MacKenzie is Professor Emeritus of Surgery of the University of Alberta and Dean of the School of Medicine. He is a native of Baddeck and this trip to Keltic Lodge will be in the nature of a homecoming.

J.B. MacD.

INFECTIOUS DISEASES—NOVA SCOTIA
Reported Summary for the Month of November, 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	5	7
Diarrhoea of newborn, epidemic (764)	0	0	0	0	1	1
Diphtheria (055)	0	0	0	0	9	1
Dysentery:						
(a) Amoebic (046)	1	0	0	0	1	0
(b) Bacillary (045)	0	0	0	0	150	146
(c) Unspecified (048)	50	0	0	0	159	41
Encephalitis, infectious (082.0)	0	0	0	0	1	2
Food Poisoning:						
(a) Staphylococcus intoxication (049.0)	0	0	0	0	0	0
(b) Salmonella infections (042.1)	0	0	0	0	47	0
(c) Unspecified (049.2)	0	0	8	0	38	81
Hepatitis, infectious (including serum hepatitis) (092, N998.5)	89	0	31	0	448	227
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	18	82
Meningococcal infections (057)	0	0	0	0	2	16
Pemphigus neonatorum (impetigo of the newborn) (766)	0	0	0	0	0	0
Pertussis (Whooping Cough) (056)	0	0	0	0	365	721
Poliomyelitis, paralytic (080.0, 080.1)	0	0	1	0	57	139
Scarlet Fever & Streptococcal Sore Throat (050, 051)	104	0	104	0	763	1940
Tuberculosis						
(a) Pulmonary (001, 002)	0	0	18	3	235	409
(b) Other and unspecified (003-019)	16	3	3	0	24	105
Typhoid and Paratyphoid Fever (040, 041)	0	0	0	0	15	19
Venereal diseases						
(a) Gonorrhoea —						
Ophthalmia neonatorum (033)	0	0	0	0	0	0
All other forms (030-032, 034)	28	0	39	0	960	1092
(b) Syphilis —						
Acquired—primary (021.0, 021.1)	0	0	0	0	0	0
— secondary (021.2, 021.3)	0	0	0	0	0	0
— latent (028)	0	0	2	0	0	0
— tertiary — cardiovascular (023)	0	0	0	0	0	0
— „ — neurosyphilis (024, 026)	0	0	0	0	0	0
— „ — other (027)	4	0	0	0	0	0
Prenatal—congenital (020)	0	0	0	0	0	0
Other and unspecified (029)	0	0	1	3	141	138*
(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	1	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

C — Cases D — Deaths

*Not broken down

C.D.C. 2

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.