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The NOVA SCOTIA MEDICAL BULLETIN

EDITORIAL COMMENT

THE COMPROMISE WITHOUT

Toynbee introduces his *Study of History* with the premise that the "intelligible unit of historical study is neither a nation-state nor (at the other end of the scale) mankind as a whole, but a certain grouping of humanity which we have called a society." We believe that this panoramic concept must also be applied to the understanding of medicine as a profession within society. This is the age of the giant corporation, the efficient but faceless organization, the "grey-flannel suit" . . . in short, the "organization man." The science of medicine, it is said, has become too broad for one man to maintain a reasonable and up-to-date knowledge of everything, hence specialization. Today we present to the lay public a picture of a group that is apparently at odds within itself, which (according to a recent study by Maritime Medical Care) seems to have great difficulty in defining what a specialist is, and which, in its new rulings, leaves itself obvious loopholes to vary the definition according to 'local circumstances.'

It is this rather interesting variation according to local circumstances that may lead the profession into more difficultues than the compromise surmounts. The variation in payments by several insurance companies—not only as between general practitioners and specialists, but also as between different types of specialists—all based on sound business policies by the insurance carriers and yet seeming to split the doctors into those whose fees are just and those whose fees are unjust; the family doctor who may hospitalize and treat patients in one town but not in another, or only under rigid controls; the payment of specialist fees to certain dubiously-qualified specialists in one area but not in another; the apparent Horatio-at-the-bridge effort of the radiologists and pathologists, while most of the remainder of the profession sleeps on; the oft-heard statement that "we must be practical"—i.e., "we must compromise — all these inconsistencies lead to the attitude "Am I my brother's keeper?" and if we are not careful, will lead us to founder on the rock of self-interest.

The ultimate aims of man have been discussed at great length by various groups in European society, and by the "angry young men" group in England but apparently warrant little concern on this continent. The lethargy of the medical profession, supposedly one of the better educated and one of the more financially secure groups of our society, is frightening to behold. As Professor George Grant pointed out in a recent address to us, "while the medical profession has been wallowing around in a sea of technical details, the legal profession has kept secure its participation in traditions and its deep sense of responsibility to human meanings."

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As this goes to press there is a concerted effort by the Halifax general practitioner group to have more so-called "open" beds on the wards of the new wing of the Victoria General Hospital (see Personal Interest Notes). While the advice to the government pays lip service to the necessity of beds for 'teaching purposes' we have yet to hear a sincere *open* debate with *both* sides presented to the medical profession. We feel that the interests of society as they relate to medicine, will not be well served by back-room politics and yet, if the profession as a whole does not take a lively interest *now*, there will be little hope for the future.

May we sink to the mundane and point out that our own Nova Scotia Medical Bulletin is markedly lacking in that rather strong department so noticeable in British and American journals, namely, Letters to the Editor. These may be written at your own leisure and will be seen by all. While we are aware of the present generation's desire not to take strong public stands (particularly in writing) on any subject, we feel that only if such stands are taken will we maintain Adam Smith's concept of "a liberal economic society in which regulation is by competition and the market and not by the state, and in which each man, thrown on his own resources, labours effectively for the enrichment of the society."

J. H. Q.

The Nova Scotia Medical Bulletin notes with pleasure the appointment of Dr. Donald C. Graham of Toronto to the post of Editor of the Canadian Medical Associaton Journal, effective July 1, 1960. His predecessor, Dr. S. S. B. Gilder has raised the Journal to the level of a weekly, and the post therefore requires a man of no mean ability.

While our personal contact with Dr. Graham was one of teacher-pupil and of rather brief span, the broadness of his approach to most subjects and his inherent wit were obvious to all. We hope these attributes of the new editor will shine through the pages of our senior Canadian medical publication, which sometimes (sad to say) seem weighted on the side of bureaucracy.

J. H. Q.

CURRENT CONCEPTS OF CORONARY SCLEROSIS AND THROMBOSIS*

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During the past 40 years, deaths from ischaemic heart disease have been increasing, just as deaths from cancer of the lung have done, and in the same sex and age group. Epidemiological studies have made it quite clear that both are penalties of civilization and that coronary artery disease goes hand in hand with a high standard of living and leisure. Its highest incidence is in the United States of America, with Canada following closely behind. As a cause of death it is more important than cancer, and like cancer, has proved equally elusive to all attempts to pin down a single cause.

Nature of the Disease

One of the most difficult features to grasp about atherosclerosis is that there is no natural history, as there is in every other lesion, with progression from an early to a late stage with complications. The simple fatty streak in the intima does not necessarily progress to the fibrous plaque, nor to the fatty necrotic ulcerating lesion. There is apparently no definite relationship between these three variants of the atheroma. Furthermore, the consequences of this disease are not uniform, nor predictable. We are all familiar with ths patient, too often a friend in a good state of health, who suddenly collapse and dies in minutes in his car, on the street or at home. We say he has had a heart attack, and yet if an autopsy is done the pathologist will not find an occluding thrombus in more than 30% of the cases. What he does find is a marked to moderate degree of coronary atherosclerosis, with one or more points of stenosis in the proximal portions of the right or left coronary artery. The mechanism of death is said to be acute coronary insufficiency.

On the other hand, those individuals who have one or more clinical attacks of infarction, will show at autopsy two or more old occlusions or stenoses or both, with marked sclerosis.

Sometimes, it is true, in the young man in his twenties we find a thrombus on an isolated area of atheroma, while at the opposite end of the scale there is the old man, dead of heart failure, who has sclerotic arteries without occlusions and without infarctions, and only scattered microscopic areas of myocardial scarring.

These clinical and pathological observations have led some investigators to suggest that, because atherosclerosis may at times be extensive and symptomless and discovered only at autopsy, while at the other times it is almost nonexistent in the presence of death from a fatal thrombosis, the disease may be different in the old and the young, the causes of atheroma being distinct and separate from those of thrombosis.

Theoretical Concepts of Etiology

One of the first theories about atheroma was developed by Rokitansky[15] over 100 years ago. He thought that the thickening of the vessel wall was due

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*Read at the Eastern Regional Meeting, Royal College of Physicians & Surgeons of Canada, Halifax.

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to precipitation of blood elements, the intimal lesions being the end result of mural thrombi. Virchow, the father of pathology, who has much to answer for, discarded Rokitansky's mural thrombosis concept without producing a reasonable substitute. In the last few years Duguid[6] has revived the mural thrombus theory and, despite tremendous opposition from the Americans, this is now gaining ground. Duguid's evidence is morphological and is convincing.

Duguid has not given reasons for the localisation of lesions, but many others have. Duff[5] and later Texon[16] showed that lesions developed at points of stress, for example just distal to the ostia of the intercostal arteries where the wall of the aorta is firmly attached to the vertebral column, and similarly in the abdominal portion where it is also fixed. Texon felt that the factor in localisation was not the fixation and resulting tension on the vessel wall but the haemodynamics, with a relative negative pressure just distal to irregularities in a rapidly flowing stream, or deviations in direction of flow, such as might occur at points of branching. In the coronary arteries no one has produced any satisfactory explanation for the localisation of the lesions to the exposed portions of the vessels, where they are embedded in fat, but perhaps it is related to the lack of support by cardiac muscle. In the experimental animal one finds the distribution of atherosclerosis in coronary arteries the same as that in man, while in the aorta it differs in being more proximal. This is probably related to posture.

One of the most interesting theories about localisation of lesions was first put forth by Winternitz[17], namely that intramural vessels ruptured, and that the hemorrhage damaged the wall, with thrombosis as a sequel. Paterson[14] demonstrated intramural hemorrhage in relation to thrombi, and, as a result, said that elevation of blood pressure as in emotional excitement or exercise, would cause rupture of delicate intramural capillaries. So far, however, Paterson has not been able to convince most of us that there are normally vessels in the intima, and secondly, if they *are* there, that they may rupture and thus produce the atheroma. Both Paterson[13] and Duguid consider that the fatty and necrotic material present in an atheroma arise from disintegration of blood clot.

Other views regarding the vessel wall are supported by Florey[7], who believes that abnormalities of the lining endothelium may form a basis for the precipitation of blood elements. In support of this is the observation that there are abnormalities of the lining endothelium over atheromata.

In summary of the foregoing, nearly everyone believes that the pattern for distribution of atheroma is determined by factors affecting the vessel wall, but what these are is not clear. There may be alterations in haemodynamics, in tension of the wall, or in the endothelium.

In considering those factors in the blood that may determine localisation of lipids in the vessel wall, there are three or four principal theories.

Ever since Ignatowski[9] fed eggs to rabbits, and Anitschkow[2] proved that cholesterol-fed rabbits developed atherosclerosis, there has been a very large body of opinion that emphasized the cholesterol itself and the amount of cholesterol ingested. Since 1914 hundreds of investigators have fed thousands of rabbits cholesterol and produced intimal deposition in large vessels, but beyond that no one has progressed very far. First of all, the fat in the atheroma is mixed and in the same ratio as it occurs in the plasma, with free and esterified cholesterol, phospholipid and neutral fat all present. Attempts to relate atherosclerosis to the level of blood cholesterol have not been successful,

although recently Adlersberg[1] has been able to show that, in the general population, there are varying degrees of elevation of the blood cholesterol, many people having a tendency towards hypercholesterolaemia, but that this may be modified or obscured by other factors. The pattern of cholesterol levels follows that of the blood pressure in the general population, and this level is in turn genetically determined. In those people with the tendency to elevation of cholesterol, the incidence of ischaemic heart disease is greater.

Attempts to relate the level of plasma lipids to other lipids in the diet have been somewhat more successful, with more emphasis today on the importance of lack of unsaturated fatty acids, linoleic, linolenic, and arachidonic acids, and the presence of phosphatides, notably ethanolamine. The influence of these two substances is antagonistic, with the unsaturated fatty acids tending to lower blood lipid levels, while the phosphatides tend to raise blood lipid, and to modify the clotting mechanism.

The physical state of the lipids in the blood stream has been claimed by Gofman[8] to be of major importance in determining whether or not they settle in the intima. His technique is ultracentrifugation. The larger molecules of lipid in combination with protein, the beta-lipoprotein complexes, are said to be unstable and tend to break up with consequent precipitation in the tissue of the subendothelial layer.

Just to confuse the issue of the blood lipids and the role they play, Mustard[12] believes that the phospholipids and the beta-lipoprotein complexes that they form, tend to enhance the precipitation of platelets and thus to initiate blood clotting. The substance in the diet said to be most influential in this regard is the phosphatidyl ethanolamine that occurs in butter and egg yolk, but principally the latter.

Other factors that interest the biochemists particularly are the proteolytic enzymes that dissolve fibrin. These are called fibrinolysins, but, like everything else in the blood stream, do not exist in the active state. There is a precursor that must be activated, and, to counter-balance this, there is an inhibitor. At present most interest centres on the inhibitor.

General Factors

Atheroma is more frequent in males, but the female is just as susceptible once she is past the menopause. The oestrogenic hormones appear to have a protective effect on the arteries.

While it is true that atherosclerosis increases with advancing age, the disease does occur in the young and also in middle aged people. I have mentioned before that in the very young the disease may consist of isolated lesions only in the coronary arteries and that thrombi may develop on them. In later years it is the environmental factors that seem to play a more important role in determining the degree of atherosclerosis that develops. By the environmental factors I mean the diet, and degree of activity, and the presence or absence of hypertension.

Everyone is familiar with the way in which hypertension enhances the development of atherosclerosis—so much so that the majority of people with hypertension die of coronary thrombosis or ischaemic heart disease or both. This enhancement of the process is not related solely to the increased stress and possible haemodynamic factors in the evolution of the atheroma. It is more complicated than that and may be related to constitutional and genetic factors.

In the diabetic, disturbed carbohydrate metabolism may affect the vessel wall and its permeability and consequently there may be an enhancement of localising factors there. It is also true that in the diabetic there is a disturbance of fat metabolism and lipemia is not uncommon. Joslin[10] maintains that those juvenile diabetics who have been in a state of physiological control since the discovery of their disease do not show an enhanced development of atherosclerosis.

Insurance figures have shown that people who are overweight die earlier, and that most of them die of heart disease. There is no question, as stated above, that obesity predisposes to a higher incidence of atherosclerosis. The high caloric intake that is associated with it is one of the factors that is quoted so often in the United States of America. Recently Dock[4] has been writing extensively about the finding of atheroma of marked degree, often with thrombosis, in the coronary arteries of young men in their twenties, autopsied during the Korean War. He ascribes this unusual degree of atherosclerosis and coronary thrombosis in these young men to a diet containing a high percentage of fat, with in fact 40% of the calories derived from animal fat. He says that the milk bar, the milk shake, butter and chocolate are responsible.

Some of the most interesting work in recent years has been produced by Morris and his collaborators[11] in England, where an analysis of bus drivers and bus conductors, letter carriers and letter sorters was carried out. In both these groups of workers one member has a sedentary occupation in that he is sitting or standing all day long with little physical activity, while his opposite number for example the bus conductor, is continually on the move up and down in the double-decker buses. In the very large series of figures analysed, it was quite evident that ischaemic heart disease was more prevalent in the individual who did not engage in physical activity. There has been additional work done in the United States on the subject, and it has been found that the increased caloric intake is apparently not a factor in elevating blood lipids nor in enhancing atherosIcerosis, if the individual engages in physical activity to such an extent that the added calories are burned up.

It is often said that diminished thyroid activity as in myxedema is associated with an increased development of atherosclerosis. In our own Department[3] we have not been able to verify this, although we have found that degenerative changes do occur in capillaries and possibly in the media of some of the large vessels.

A great deal has been written and there are many clinical trials under way at present on the effects of long-term anticoagulant therapy. The results of this are, of course, especially interesting to those who believe that disturbances of the clotting mechanism may be principally responsible for both atherosclerosis and thrombosis. On the whole the results are encouraging, although from a theoretical standpoint one may question the efficacy of dicoumarol, as it is active against Christmas factor and tissue thromboplastin only. The administration of dicoumarol is not without danger because there may be bleeding episodes without reduction or impairment of platelet disintegration and platelet thrombus formation.

Heparin has been found to be much more active than other drugs in clearing the blood of lipemia. All this means is that the milky character of the plasma may be changed by the administration of heparin, and the activation of liposes. This alteration in the appearance of the plasma is not accompanied by a change in lipid concentration, but simply an alteration in its physical state.

The high molecular weight macromolecules of lipoprotein complexes are dispersed and smaller molecules are formed. Gofman states that this reduction in molecular size and dispersal is beneficial because it is the high-molecular-weight substances that contribute to atherogenesis. The administration of heparin, however, may have importance in another direction too, because heparin is active against factor 7 in blood clotting and against a similar substance which is present in the vessel wall and which may promote precipitation of clot. There are many reports in the literature about the beneficial effects of administration of heparin to individuals with angina pectoris.

Conclusions

Despite the tremendous volume of publications on the causes of atherosclerosis we do not appear to be much nearer a solution of the etiology than we were many years ago. I think however, that certain aspects of the problem are becoming clearer. There is undoubtedly, for example, a factor in the vessel wall that determines the localisation of the lesion to a particular site. Some of these factors are related to points of stress or tension and to alterations in the character of the blood flow at these points. In relationship to the blood lipids there is also no doubt in my mind that a high caloric intake without burning up the calories through physical activity will tend to enhance the development of atherosclerosis. Whether the atheroma, however, is the result of the increased lipid in the blood, or dependent upon the nature of the lipid, one cannot say. It is just as possible that the kind and nature of the lipid in the blood modifies clotting mechanisms so that blood elements become deposited on the vessel wall. It may also be possible that enzymatic removal of precipitated elements is at fault. Lastly, one cannot deny that lipid may accumulate in the vessel wall through imbibition, or through disturbances of intercellular substance in the intima, or both.

In conclusion, I would suggest that several etiological factors, not necessarily related, may cause atherosclerosis.

REFERENCES

1. Adlersberg, D. Hypercholesteremia with Predisposition to Atherosclerosis. Am. J. Med., **11**: 60, 1951.
2. Anitschkow, N. On the Changes in the Rabbit Aorta in Experimental Cholesterol Steatosis. Beitr. z. path. Anat. u. z. allg. Path., **56**: 379, 1913.
3. Baker, S. M. and Hamilton, J. D. Capillary Changes in Myxedema. Lab. Invest., **6**: 218, 1957.
4. Dock, W. Editorial. Research in Arteriosclerosis—the First Fifty Years. Ann. Int. Med., **49**: 699, 1958.
5. Duff, G. L. and McMillan, G. C. Pathology of Atherosclerosis. Am. J. Med., **11**: 92, 1951.
6. Duduid, J. B. Mural thrombosis in Arteries. Brit. Med. Bull. **11**: 36, 1955.
7. Florey, Sir H. W. Lecture delivered at the University of Toronto, June 1956. (Unpublished)
8. Gofman, J. W. et al. Blood Lipids and Human Atherosclerosis. Circ. **5**: 119, 1952.
9. Ignatowski, A. On the Influence of Animal Proteins on the Aorta and Parenchymatous Organs of the Rabbit. Virch. Arch. f. path. Anat., **198**: 248, 1909.
10. Joslin, E. P. The Treatment of Diabetes Mellitus. Philadelphia, Lea and Febiger, 1946. p. 481.
11. Morris, J. N. et al. Coronary Heart Disease and Physical Activity of Work. Lancet **2**: 1053 and 1111, 1953.
12. Mustard, J. F. *In Vitro* and *in Vivo* Effect of Different Fat Preparations on Blood Coagulation. Canad. M.A.J., **79**: 818, 1958.
13. Paterson, J. C. Vascularisation and Hemorrhage of Intima of Arteriosclerotic Coronary Arteries. Arch. Path., **22**: 313, 1936.
14. Ibid. Relation of Physical Exercise and Emotion to Precipitation of Coronary Thrombi. J.A.M.A., **112**: 895, 1939.

15. Rokitansky, C. A Manual of Pathological Anatomy. Trans. by G. E. Day. Philadelphia, Blanchard and Lea, 1855. Vol. 4, p. 205.
16. Texon, M. A Hemodynamic Concept of Atherosclerosis with Particular Reference to Coronary Occlusion. A. M. A. Arch. Int. Med., 99: 418, 1957.
17. Winternitz, M. C., Thomas, R. M. and LeCompte, P. M. The Biology of Atherosclerosis. Springfield, Ill., Chas. C. Thomas, 1938.

HODGKIN'S DISEASE ASSOCIATED WITH OTHER MALIGNANT TUMORS AND CERTAIN NON-NEOPLASTIC DISEASES. Razis, D. V., Diamond, H. D., and Craver, L. F., Amer. J. Med. Sci., 238:327, (Sept.), 1959.

The coexistence of Hodgkin's disease with other malignant neoplasms, other lymphomas, and certain non-malignant diseases is reported and discussed in this paper.

Other malignant neoplasms were found in 2.2% of the Hodgkin's disease patients as compared with 3.9% in the lymphosarcoma group and 2.3% in the leukemia group. Whether this association is higher than in the general population is not known.

Lymphosarcoma was found to be associated with Hodgkin's disease in 2 patients. In 2 other patients, Hodgkin's disease was associated with monocytic leukemia. In contrast, 7.6% of the lymphosarcoma patients developed leukemia.

The existence of more than one histological variety of malignant lymphoma in the same patient is due, in the opinion of the authors, either to a process of transformation or to their independent origin. The termination of cases of mycosis fungoides and giant follicular lymphosarcoma as reticulum cell sarcoma probably represents a metamorphosis. The appearance of lymphocytic leukemia in patients with lymphosarcoma may be explained by transformation or by extension of the disease to the bone marrow and peripheral blood. The coexistence of Hodgkin's disease with lymphosarcoma or leukemia probably represents an example of associated diseases of independent origin, although the opposite view may be supported by rare cases.

Active tuberculosis was associated with Hodgkin's disease more often than with lymposarcoma, *and this difference was highly significant statistically.*

Secondary amylodiosis was found in 5 patients with Hodgkin's disease but in no patients with lymphosarcoma and leukemia. This seems to exonerate nitrogen mustard as a causative factor of the amylodiosis, as this agent is used widely in the treatment of lymphosarcoma. Furthermore, one patient with Hodgkin's disease and secondary amyloidosis did not receive nitrogen mustard.

The association of Hodgkin's disease with sarcoidosis, the so-called "collagen diseases," idiopathic thrombocytopenic purpura, diabetes mellitus, and syphilis is also reported.

S. J. S.

COXSACKIE VIRUS INFECTIONS

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The virus of Coxsackie infection was isolated in 1947 from the stools of two children in Coxsackie, N. Y., who were suffering from paralytic poliomyelitis. The infection with Coxsackie virus was coincidental and probably had nothing to do with their symptoms. For a considerable period, clinicians had been aware of febrile illnesses having neurological signs which resembled those of mild poliomyelitis but with a benign outcome and no residual paresis. Such illnesses had been classified as "non-paralytic poliomyelitis" and though undoubtedly this was true in some cases, the isolation of a completely different enterovirus made it obvious that the disease was most frequently not poliomyelitis.

Since then the Coxsackie viruses have been subjected to much study by modern virological techniques and it has been shown that they may be divided into two groups "A" and "B", distinguished clinically by the symptoms produced—though there is some overlap—and in the laboratory by the nature of the lesions they produce in suckling mice. Each group is further subdivided into several types—25 or more in the case of Group "A" and 5 in the case of Group "B." Clinically the only significance of this is that certain types seem to be related to fairly clear-cut syndromes. The virus is spread by droplets from the throat, by faecal contamination and possibly by flies. The diseases usually occur as family infections or among intimately associated groups. The incubation period may be from one to fourteen days—with a mean of three to five days.

Distribution of Coxsackie Viruses in Tissues

Throat secretions, faeces and cerebrospinal fluid may yield virus in the acute phases of illness. Virus may be recovered from stools for several weeks. Subclinical infections are frequent in contacts. Some 1 to 7 percent of normal persons harbour Coxsackie Group "A" viruses in faeces; excretion is commonest in Summer and Fall. In contrast Group "B" viruses are rarely isolated from the throat or faeces of persons other than those ill with epidemic myalgia, aseptic meningitis, pericarditis, orchitis, etc. A detailed description of methods used in the collection of virus specimens is given in an appendix to this paper.

There is now reliable evidence that the following conditions are attributable to Coxsackie virus infection.

Aseptic Meningitis

This condition is caused by Group "B" Types 1—5, also (rarely) Group "A". The illness may have a sudden or gradual onset—sometimes with a prodromal phase lasting 2-6 days during which time virus may be recovered. The symptoms include fever, headache, vomiting, drowsiness and dizziness, with pain and stiffness of the back and neck. True muscular weakness is not common and actual paralysis must be quite rare—though it does occur. The cerebrospinal fluid shows an increased cell count—rarely exceeding 500 cells/-

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*A/Surgeon Captain, R.C.N.

cu. mm and consisting mostly of lymphocytes. Sometimes a minor increase in protein is noted. Occasionally a polymorphonuclear pleocytosis will occur at first and may cause confusion with bacterial meningitis.

The symptoms are rarely severe and recovery is almost invariably complete, though convalescence may be protracted. Confusion with poliomyelitis is of course, all too easy in the early stages and the two diseases may be indistinguishable if no paralysis occurs. Coxsackie virus and poliomyelitis virus may coexist in the same patient though this has rarely been proven in the case of Group "B" viruses which are far more important in the causation of aseptic meningitis.

Epidemic Myalgia (Bornholm Disease—epidemic pleurodynia)

This illness, caused by Group "B" only, types 1,3,4,5, was first fully described (in 1934) by Sylvest who described an epidemic of cases on the island of Bornholm, off the Danish coast. Earlier, if less detailed, descriptions of what is almost certainly the same disease exist particularly in the Scandinavian literature. The onset is usually quite sudden with fever and myalgic pains. The latter may occur in any muscle groups but characteristically in the thoracic muscles. The pain may be very severe and may be described as intrathoracic or retro-sternal—usually, however, it is at the sides of the thorax and is markedly accentuated by breathing movement—or any movement. Pain may be primarily in the abdominal wall—particularly in children in whom an abdominal emergency may be simulated. Neck stiffness is common and probably due to myalgia though meningitis may of course coexist. The affected muscles are often tender to the touch. Clinical laboratory investigations are usually negative and the white cell count normal or low. Symptoms usually last only a few days (occasionally one to two weeks). Recovery is invariable and complete. *Orchitis* may rarely complicate the disease usually *after* the acute symptoms have subsided.

Herpangina

This is another variant of the Coxsackie virus infection caused by Group "A," Types 2,4,5,6,8,10. The onset is usually abrupt with fever, anorexia, dysphagia, sore throat and perhaps vomiting. The characteristic lesions are found on the pillars of the fauces, soft palate, uvula, tonsils or, less commonly, fhe tongue. They start as a papule which rapidly becomes a grayish-white vesicle 1-2 mm in diameter and surrounded by a red areola. When the vesicle ruptures a shallow ulcer is left rarely more than 5 mm in diameter. The whole condition lasts 4 - 6 days. There may be perhaps 5 - 15 vesicles per patient. There are no lesions on the gums which prevents confusion with Vincent's Angina. This is a benign, self-limited illness—though *parotitis* has been described as a complication. The clinical laboratory findings are normal unless secondary infection occurs.

Myocarditis of Newborn

This grave manifestation of Coxsackie infection is caused by Group "B" —Types 3,4, have been definitely implicated—others are probably involved. In 1955, ten new-born infants died with evidence of myocarditis during an outbreak of epidemic myalgia in South Africa. At autopsy Coxsackie B3 virus was recovered from the heart muscle. Similar incidents have been described in Holland and on this continent. It now seems apparent that new-

born babies—like new-born mice—are particularly susceptible to Coxsackie infection and in these patients the infection is extremely serious and almost invariably fatal. It is thought that this is a neonatal infection from an infected mother or attendant and not one transmitted transplacentally, or at the time of birth there is a sudden onset—about 8th or 9th day of life—when a hitherto healthy infant shows evidence of tachycardia, dyspnoea and cyanosis. Clinically the heart is enlarged. The illness characteristically takes a fulminating course and death is rapid. Post mortem examination shows an acute necrotising myocarditis and virus may be grown from the muscle. It is not known why new-born children and animals are liable to this particularly grave manifestation of what is normally a benign disease. It has been suggested the high levels of circulating corticoids which are known to occur in the first few days of life—perhaps as a protection against the trauma of the birth process—render the subject liable to a more severe viral illness. It is known for example that adult mice cannot normally be infected with Coxsackie virus but that if they are also given corticoids, infection does occur and can have serious manifestations including myocarditis. The important thing is to remember that new-born babies are particularly liable to develop serious Coxsackie infections and that persons with any suspicion of such infection should be rigorously excluded from the new-born invironment. This prohibition would include the mother herself if she had such an illness.

Pericarditis

The condition known as idiopathic benign pericarditis has been known for many years but until recently its etiology has been quite obscure. It was described initially as occurring particularly in adults of middle age but it is now recognized that the condition may develop in children and in fact, a number of such cases occurred in Canada in 1958. The pericarditis usually occurs as part of a febrile illness and the chief symptom is pain which is often severe, retro-sternal and may closely simulate that of myocardial infarction. Pericardial effusion probably occurs in a majority of cases but is rarely gross. However if it becomes excessive it will seriously embarrass the heart action and may cause death from tamponade. The very few recorded deaths from idiopathic benign pericarditis—about 5 or 6 in all—were nearly all due to this complication, which went unrecognized in life. Needle aspiration of the pericardial sac may be life saving in these circumstances.

For some time it had been suspected that this condition was of virus origin, but it was not until 1958 that a virus was isolated and a significant rise in neutralizing antibody demonstrated. Since then these findings have been duplicated in Canada both in Toronto and Halifax and a Coxsackie virus Group "B" implicated. Apart from the danger of tamponade, the disease appears to be benign and no convincing evidence of myocarditis has as yet been demonstrated in adults. However, the convalescence from this disease is often protracted and undue dyspnoea on exertion and a high pulse rate may persist for a long time. It is probably wise to insist on a very gradual resumption of normal activity.

Summary

These five varied clinical manifestations have been described in this paper as separate entities. It must be remembered that more than one may be present during the illness and that any combination may occur. Perhaps

other manifestations of Coxsackie infection will be demonstrated. In the meantime, it is wise to be alert to the known features and to remember that serious sequelae may result from infection, particularly in young infants.

Appendix

Collection of Specimens

Coxsackie viruses are extremely sensitive to environment and will die promptly unless specimens are correctly collected and handled. The following instructions from the Nova Scotia Provincial Virus Laboratory are included for the guidance of those sending specimens from outside the Halifax metropolitan area. Local material must be delivered to the Virus Laboratory *as soon as possible* and certainly within one hour. If delay cannot be avoided the specimens must be frozen as shown below.

Throat Swabs or preferably physiological saline garglings or throat secretions aspirated in infants, which travel better and do not dry up like throat swabs. These should be transported to the Laboratory, *frozen in dry ice.*

Rectal Swabs Collected as usual and placed in a sterile screw-capped test tube and transported *frozen in dry ice* to the Virus Laboratory.

Stools. About 50 grams should be picked up on a wooden tongue depressor and placed in a *sterile* glass screw-capped jar. It must be forwarded to the laboratory *frozen in dry ice.*
N.B. If jars are filled more than 1/3 or 1/2 full, breakage may occur from the fermentation gases produced in the stool specimen. Decontamination of the outside is appreciated. Sterile jars and phials will be supplied on request.

Cerebrospinal Fluid. First exclude meningococcal or other pyogenic bacterial infection of the brain and meninges by appropriate bacteriological examination. In all other cases of suspected meningeal involvement a specimen of CSF should be sent to the Virus Laboratory for attempted isolation of virus. During the past year it has been the experience of this laboratory that specimens of CSF showing pleocytosis 40 to 400 lymphocytes per cu/mm have frequently yielded virus on monkey kidney or human amnion tissue culture. This strongly suggest that clinicians would be well advised to request virus culture of all specimens of CSF where lymphocytic pleocytosis is present. Occasionally CSF yielding virus has shown a preponderance of polymorphs, but this could be a phase in the cellular reaction as we have noted in the case of ECHO 9 infection. We would like to stress that whereas in several ECHO and Coxsackie "A" and "B" types of infection virus is recoverable from the CSF, this is *NOT SO* in poliomyelitis where the virus is usually *NOT* found in CSF in any stage of the disease. The virus of Poliomyelitis is, however, found in the stools of over 90% of acute cases of poliomyelitis and in carriers in poliomyelitis infected areas. CSF should be sent to the Virus Laboratory in a sterile screw-capped phial, *frozen in dry ice.*

Blood-Serum. In all suspected viral conditions, it is necessary to obtain two-phase sera, one collected in the EARLY FEBRILE ACUTE STAGE (Preferably first 24 hours) of illness, and the other in the CONVALESCENT STAGE of illness—say three weeks later. It is most important to have these *two* specimens of serum.

If a virus can be identified from the throat secretion, stools or CSF of the patient in the acute stage and be readily identified serologically, diagnosis is made and there is no problem.

Unfortunately, however, a positive culture may not be obtained. In this case, the identification of the patient's illness rests on attempting to demonstrate a rise in antibody level in two-phase sera by neutralization tests against known strains of virus. Conversely, a virus may be grown from the patient which cannot be typed by the use of known specific antisera. In this case the aetiological relationship of the suspected virus to the patient's illness can only be adduced by demonstrating a rise in antibody titre in the patient's own serum against his own virus.

Transmission of Serum. Serum should be separated with sterile aseptic technique, *packed in dry ice* and sent to the Laboratory promptly. *WHOLE* blood should never be frozen for serological tests—only the serum. Plasma is of no value.

Air Shipment. It would facilitate the operation of the Virus Laboratory if a telegraphic message were sent in advance intimating the flight by which the specimen was due to arrive at Halifax.

Diabetes and Tuberculosis. Banyai, A. L., Dis. Chest, 36:238, (Sept.), 1959.

The material presented in this paper was gathered from the author's experience with 200 patients who were afflicted with diabetes mellitus and complicating pulmonary tuberculosis. Concepts are reviewed which deal with the cause of increased susceptibility to tuberculosis of patients suffering from diabetes mellitus. Some peculiarities of the clinical substrate of pulmonary tuberculosis in these individuals are discussed. Therapeutic precepts applied in the treatment of the two diseases are outlined. The writer feels that in well-controlled diabetes, adequate antimicrobials and adjuvant measures are bound to bring about a recovery rate from pulmonary tuberculosis which compares favorably with that of tuberculous patients without diabetes. After proper institutional treatment, diabetics with pulmonary tuberculosis may become eligible for out-patient care, provided facilities and personnel are available for competent medical supervision. Under such circumstances, their chances for complete socio-economic rehabilitation are favorable.

S. J. S.

SPECIAL PROBLEMS OF THE TEACHING AND REFERRAL HOSPITAL

J. McD. CORSTON, M.D.

Halifax, N. S.

Because of its special facilities over fifty per cent of the patients in a referral hospital are sent by their own physicians as problem cases. The newest diagnostic procedures, some of which may take considerable time, are instituted. The most specialized therapeutic measures may be indicated and put into operation.

The "bad risk" patients, such as the elderly or the sufferer of a chronic medical complaint, are sent to such a hospital for major surgery. These frail individuals must first be treated by internists and their condition improved to the maximum possible before surgery is embarked upon with the help of the anaesthetic staff. Post operatively their convalescence is of necessity slower than the younger, healthy "good risk" patients. All this adds to the overall hospital days of the institution but the mortality rate is kept to a minimum and major surgery is made possible.

Likewise, patients admitted for cardiac surgery must first be fully investigated by the cardio-vascular unit team. Desperately ill head injury cases are admitted to the neurosurgical unit. As a result of great advances in treatment, quadriplegics, who formerly died, now live and their average hospital stay is about three months before they can be transferred to another institution for rehabilitation. Spinal fusion cases which are becoming more common require about two months on a frame before discharge from hospital.

Cancer patients at the rate of fifteen hundred new cases per year are admitted. About sixty per cent have to remain as in-patients while undergoing radiotherapy. This therapy usually consists of ten minute treatments per day up to three weeks. These unfortunate people are meanwhile occupying a bed even though they may be, and usually are, ambulatory.

Because a large number of patients in a referral hospital come from long distances they must be kept in hospital a few days longer following operations before they are ready to travel home than if they lived in the immediate environs.

From the foregoing remarks it is obvious, I believe, that in a referral hospital a large number of patients must be kept in hospital longer because of the nature of the time consuming investigative procedures which are essential in diagnosis and treatment. This factor in turn militates against the number of beds available for the commoner diseases and, therefore, the waiting lists grow longer. One essential of a referral hospital is that special investigative cases be allotted to an investigative floor containing beds separate from the active treatment floors. In this way beds needed for cases of the commoner diseases will not be occupied by long-stay patients. By the same token a teaching and referral hospital must have more actual beds in proportion to population than the non-teaching hospital.

Clinical research must also be energetically pursued in such an institution because without research, clinical advancement is slow and mental stagnation follows. Freedom of action for those pursuing new knowledge and breaking new trails into the unknown is essential. They must not be fettered by red tape and lack of facilities. Though this may represent the expenditure of con-

siderable sums of money, it may represent a small investment for a much larger return in terms of improved health of the people. Welfare states such as Russia, Scandinavia and Great Britain are notoriously liberal with facilities and money for the research worker. This hospital insurance scheme of ours in Nova Scotia is a great socialistic advance. Let us adopt a more courageous and realistic attitude in supporting clinical research.

The teaching hospital is responsible for the medical education of medical students, doctors and nurses. It may be necessary to keep patients in hospital slightly longer to be used as interesting teaching material. Also, these patients may be submitted to a few more tests in order to complete the "work-up." In the non-teaching hospital these tests may not be quite so numerous but although some may not be absolutely essential, it is important for the trainees to be instructed in thoroughness.

Finally, to sum up, it is a self-evident fact that more beds are needed in proportion in the teaching and referral hospital because of the peculiar needs in its proper functioning. Because of its duties and specialized work in the community, this type of hospital should be looked upon by the Hospital Insurance Commission and the Government as being in a special category as regards budget and freedom of action of the medical personnel.

Etiology of Cardiac Infarction. Yudkin, J., Arch. Int. Med., 104: 681, 1959.

This editorial by the Professor of Nutrition and Dietetics, Queen Elizabeth College, University of London, points up the many illogical assumptions that increasingly becloud the few facts that we have available in this field. A particularly relevant paragraph is as follows: "With our recent preoccupations with diet and dietary fat, we have tended to give less attention to other etiological factors. There is reasonable evidence that one factor is smoking, and an increasing amount of evidence that another is decreased physical activity. Conflicting evidence appears concerning mental stress, obesity, and physical build. About the only generality which can be applied is that Cardiac Infarction is associated with what we have come to call "high living standards" and "cardiac infarction thus seems to be due to a variety of causes. Some of these probably act after a long latent period, and may be called predisposing factors. There are others such as no doubt occurred during the war, which act over a relatively short period, and may be called precipitating factors." Regarding therapy, he closes by saying "Let us be ready to acknowledge that present therapy is based on expediency rather than science, and let us receive with open mind more logical measures as soon as they become available."

L. C. S.

ATHERO: GENESIS TO EXODUS*

When I was born, my mother's milk
Was all the food I had.
It seemed to taste quite good enough,
But now I find that precious stuff
For me was very bad.
For it contained much butterfat,
And we all know the harm in that —
It fills our arteries with plaques,
Which predispose to heart attacks.

The years went by, my cravings changed,
My appetite increased.
I ate ice cream, and butter too,
I had not learned the modern view:
To stay away from grease,
I kept on eating eggs and ham;
I cared not for my cardiogram.
This fatty intake took its toll —
I had a high cholesterol.

Adulthood came — then I reformed—
For I had seen the light.
I gave up eating greasy food,
And even in a hungry mood,
I curbed my appetite.
I lived on rice and fruit alone,
No rich dessert did I condone.
As I grew thinner, day by day,
I felt my plaques would melt away.

But now I learn, to my regret,
That diet was in vain.
The food I ate when I was young,
In form of pork, or smoked beef tongue,
Will cause me future pain.
So when my T-waves go astray,
I'll shout with anger and dismay:
"I have been foiled by Nature's plan—
The child is father of the man".

EDWIN H. MERZ, M.D.

*Reprinted with Permission.

THE MEDICAL SOCIETY OF NOVA SCOTIA

ANNUAL MEETING—1960

The Annual Meeting, 1960, will be at White Point Beach, Queen's County, Monday, Tuesday and Wednesday, June 27, 28 and 29th. The general meeting will be preceded by a regular meeting of the Executive Committee on Saturday, June 25th, and the annual meeting of the Executive on June 26th.

The programme for the general meeting is attractive. Dr. R. M. Parsons, now President-Elect of the C.M.A., will have taken over the office of President at the annual meeting, June 13-17th, in Banff. We are very pleased that Dr. and Mrs. Parsons have found it possible to be present at our annual meeting. Dr. A. F. W. Peart, Assistant Secretary, C.M.A., will also be present.

Dr. Harry S. Morton of the Royal Victoria Hospital, Montreal, who is well known in Nova Scotia, has accepted an invitation to give two clinical talks during the meeting.

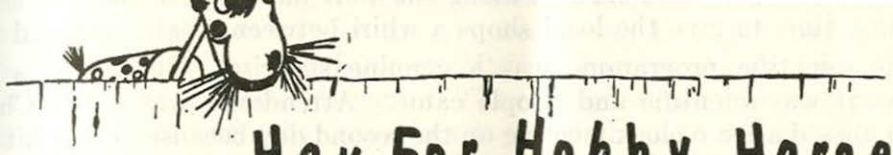
At least one panel discussion will be on the programme, which will deal with intra-professional relationships.

The B.B.C. film, "On Call to a Nation", which has been described as an "excellent documentary film" of the story of nationalized medicine in the United Kingdom, will be shown. Other films dealing with clinical subjects will be available.

Business sessions will be held each day and are expected to be interesting and informative. The reports to be presented to the annual meeting will be distributed prior to the meeting to those who have made application for housing accommodation. The purpose is to provide an opportunity to those members attending the annual meeting to be familiar with the reports so as to discuss them more freely during the business sessions.

Entertainment for guests, members and their wives will equal or surpass the well-known South Shore hospitality. The Lunenburg-Queens Branch, being the host Society, plans to lay on entertainment which will take full advantage of local talent, the natural beauty and attractions of the White Point Beach, including the nearby golf links. Wednesday, the last day of the meeting, will be free from noon. Golf Tournament and other attractions will be followed in the evening by the President s reception, annual banquet and dance.

All in all, the meeting promises to be very pleasant with a balanced programme of business, clinical and entertainment features.



Hay For Hobby Horses

Gone to Wentville

A Fantasy

As I slept I dreamed a dream. I dreamed my Everloving and I were invited to attend the first meeting of the Nova Scotia Society of Lumpers and Dividers which was held at the Cornpone Inn in Wentville March 4 and 5, 1960. The original name proposed for the society which hosted us was the Nova Scotia Society of Interminabilists but the public kept getting them confused with internists and interns so the Nova Scotia Society of Lumpers and Dividers was born. (They are now being confused with the somnabulists. Ed.)

The trip was begun in some trepidation because Rube Hornstein had promised 5-7 inches of snow for that weekend. I had more anti-blizzard gear in the back of the station wagon than I had luggage. A rough count showed one bag of coarse salt, one box of sand, one storm coat, one pair high overshoes, one non-collapsible aluminum shovel and one collapsible male vertebral column. (My wife does all the snow shovelling at our house in deference to Lea Steeves' advice.) All unnecessary of course, but in our house as Rube goes so goes the nation. We followed Hal Hyshine and Will Burry from Avonport into Wentville arriving one half hour before the opening meeting.

The Cornpone Inn looked a bit forlorn on that March afternoon, much like a dowager caught without her crinolines. It is an imposing structure with battlements, cornices, ambuscades, gargoyles and rhododendra all in the best Rhineland tradition. In truth, the Cornpone should have been set down amid vast estates and would look best approached in a leisurely way along a winding-tree-shaded drive. (Daphne DuMaurier does this sort of thing much better. Ed.) Instead the C.P.R. piled it up on an intersection in the very heart of Wentville with the commerce of this bucolic hamlet lapping at the very doorstep.

The N.S.S.L. & D. must have confused me with someone of importance because they gave us the red carpet treatment. We shared a suite with Huey Neurone, the bland secretary of the Society and his charming wife Mary. I found it easier to phone the Neurones than walk the half-mile from our corner of the suite to theirs. No press attache of the Nova Scotia Med. Bull. was ever treated better. I wait with dread to discover who the N.S.S.L. & D. want me to murder in return for the elegant weekend. (If the Society expects Brother Timothy to be silent it will take more than a weekend to do it. Ed.)

I have no idea what the girls did during our meetings but they seemed to have a lovely lazy time. Heaven knows they all deserve it and the sensation of sleeping late and dining without preparing the meals must be priceless to our harassed better-halves. They had a coffee party at which everyone compared hats, alternately lamented upon or mooned about their children, gossiped in a nice ladylike way and had their pictures taken. Lib. Howl had eight hats

plus one that doubled as a beach bag. My Everloving was having too much fun to notice that Lib had brought along one more hat than she did. The girls also found time to give the local shops a whirl between meals, teas and talk.

The scientific programme was a genuine surprise. There was a programme, it was scientific and people came. Attendance was good, Charlie Gorgon missed a ten o clock meeting on the second day because he was hit by a Flying Breakfast. The latter is a terrifying specialty at the Cornpone and I never got accustomed to scraping my breakfast off the wall. The opening programme was chaired by Estie Loafer and favoured us with John Dogberry in a sprightly, if involved, paper on "Ham Fat and Bar Grease in Oyster-toe Arthuritis." Then Earl Stiltz jarred us into wakefulness by insisting that there was yet a Crisis in Phthisis. (Damn it, just as we were getting comfortable about the disease. Ed.)

I slept through the business meetings. On the second day Eric Cincinnati lifted our post-party depression with some hopeful words about a new pep pill. The highlight of the day was a confusing exchange between two earnest young scientists, Huey Neurone and Johnny Balloon, who got their slides mixed up. Many of the gathering were most disgruntled when they awakened too late to catch the bare facts of Dr. Neurone's slide. Dr. Neurone championed the cause of the dividers by introducing two new sub-groups into the class of convulsive disorders, known as non-convulsive adiodiodyspraxic hallucinoses. The Leprechaun syndrome was illustrated by the case of a young man who when stimulated with an ancient Irish remedy, Bushmill's Elixir, would perceive green men dancing on his chest and attempting to communicate with him. Dr. Neurone advised the patient to listen carefully but not to answer the green men, especially not out-loud. The second case, now called Neurone's disease, was that of a 26-year old small car enthusiast who had a "revving-up" type of tinnitus and was obsessed with the notion that he was an M.G. This man's hallucination was due to a lesion localized in the frontal uncus of the co-axial connections in the rear gyrus of Hillman-Minx. The young man has started a positive ablation therapy to be carried out in three stages. He has cut out cars, he has cut out women, drinking and smoking and soon, Dr. Neurone expects, he will be cutting out paper dolls. Dr. Balloon's talk entitled "Rigid Structures 1960" was a very learned and arresting dissertation. I was disappointed to hear many ribald comments on this outstanding contribution, such levity does not become an august group like the N.S.S.L. & D.

A good time was had by all. If every postgraduate effort were at once as nearly painless and as highly profitable as the weekend in Wentville, the task of our director of postgraduate education Lee Sheaves would be much easier. Nearly everyone was there. Except for the Chief, Walthar Looslie, Pappy Netherlands and Garnitt Coldwall the capital city was denuded of medical consultants. There was a pleasant relaxed air over the whole proceedings and I had time to talk to all sorts of delightful people—Sham Sane, who has done so much to put the bull back in the Bulletin., Dense Howl who can see farther into the woods than most men, Arem MacDee and his charming wife—Dan Tuneing and Mrs. T., our beloved physician-philosopher-essayist Jim Tallgrass "what come ye forth into the wilderness to see, a Reid shaken in the wind?" On the first evening Jim presided over a pleasant group seated before the main fireplace which included the Art Scissors, the Balloons and the Leander Achins. Bill Morsel and Rugger Doye and their Jeans organized a

hiking trip on the breezy uplands of South Mountain on Saturday afternoon. None of us went along but we all envied them their enthusiasm. Among the 'think-physicians' from outside the city were Doug Dentist (Foxvale), George Vapour (Plunderville) and Downey Muffin (Parrtown) and from Wentville itself Earl Stiltz and Helen Huggin. The closing ball was the high point of the weekend. Space and other considerations forbid me to mention all the good men and charming ladies who dined on fillet mignon and champagne by candlelight that evening. (He put these items on his income tax so he has to claim them here. Ed.) John Dogberry moved among the happy throng with his trusty Leica recording all for posterity (and extortion? Ed.) A private showing of this interesting documentary may be had on application to Dr. Dogberry.

It was wonderful, Dear think-physicians, please ask us again.

Buoyantly yours,

BROTHER TIMOTHY.

Letters to the Editor

To the Editor:

Re: Blood Donor Situation

With reference to my article "What Do You Know About Your Blood Transfusion Service?" appearing in the March Issue of the Bulletin, I feel obliged to clarify the ambiguity present in the fourth and fifth last lines of the article. This ambiguity was apparently created by either typographical error or editorial change and missed on proof-reading. In my original article I had stated that only "5% of our population donate blood" while the article states that "15% donate;" the ambiguity is that this could be read as meaning either 15% of the population or 15% of the potential 35%. The truth is that 5% of our population or only one out of seven (approximately 15%) of the potential donors (with respect to age and health) actually donate.

Those of us concerned with donor procurement surely wish that the implication, "15% of the population donate", were true; perhaps our wishful thinking was in some way projected and so responsible for this ambiguous sentence.

If you concur, I would appreciate having this clarification brought to the attention of our profession so that no misunderstanding will exist regarding the true blood donor situation.

Yours very truly,

H. C. Read, M.D.

BOOK REVIEW

THE TRIUMPH OF SURGERY. Jurgen Thorwald. 454 pp. Illust. McClelland Stewart Ltd. Toronto, 1960.

This book is a successor to the author's *The Century of the Surgeon*, and covers that great era of surgery which grew out of the general acceptance of antiseptic surgery in the late nineteenth century and led to the establishment of modern surgery in the twentieth. All the giants appear—Paget, Charcot, Billroth, Mikulicz, Murphy, the Mayos, and a score more. Even Freud finds a place, not as a psychiatrist, but for his pioneer work in the use of cocaine.

A good medical historian should be, first of all, a student of people. With a knowledge of medicine alone as his background he is in danger of becoming a mere chronicler of scientific advancement. Jurgen Thorwald, born in the Rhineland in 1916, is an editor, journalist and historian; but before being any of these, he studied medicine. He is thus well trained for his task, and his characters emerge with their eccentricities and accomplishments, their personal charms and antipathies, as the living humans they were.

His story of the Crown Prince of Germany, dying of an ill-treated cancer of the larynx as he was crowned Kaiser Friederich III, is one of the most sordid, yet colorful, dramas in medical history. The villain of the piece, Morell Mackenzie, English laryngologist, called to Berlin by Friedrich's English wife (Queen Victoria's daughter), who lacked faith in German surgeons, was a master of quackery and deceit. Through bluff and chicanery, he succeeded in discrediting the German surgeons who had correctly diagnosed the Prince's condition in an early, operable stage, and so completely won his patient's confidence that the German surgeons were dismissed. Only in the terminal stages of the disease was Mackenzie content to work deftly out of the scene, leaving his German contemporaries to bear the onus of the Kaiser's death. Time exonerated the Germans, in part at least; but Mackenzie had long since been knighted by Queen Victoria for his distinguished service to her "beloved son-in-law".

Surgical developments in America are not given the space or the enthusiasm of those in Europe. This is, in part, because they were not as important through this era, in part because the Atlantic ocean limits the author's horizon.

As in *The Century of the Surgeon*, Thorwald uses a fictitious eye witness, Dr. Hartman, confidante or, at least, acquaintance of all the surgical great, and a constant traveller, to give the reality and intimacy of personal contact to his story. Dr. Hartman succeeds with this nicely. He also intrudes with his personal activities, annoyingly at times, slowing up the live action of the story; and his fictitious dialogue sometimes becomes confused, in the reader's mind, with documented quotations.

The large, well documented bibliography is good indication of the broad knowledge the author has brought to his task. For the student of history or the casual reader, the book is highly recommended.

A.L.M.

PERSONAL INTEREST NOTES

Cape Breton Medical Society

Dr. Frank Kelly has recently opened an office in Sydney for the practice of the specialty of surgery. A graduate of Sydney Academy, St. F. X., and McGill Medical School, he obtained his training in surgery in the Eastern United States, having been a member of the surgical staffs of the Lahey Clinic and Bellevue Hospital in New York.

Dr. Albert Prossin, Westmount, has been recently appointed as Civil Defence Officer in charge of health services for Cape Breton County. He is the present health officer for Cape Breton County, South. Dr. Prossin was recently awarded the Schering Bursary of the College of General Practice of Canada for the Province of Nova Scotia 1960.

Halifax Medical Society

April 13, 1960—6th Regular Meeting of the Halifax Medical Society at the Nova Scotia Hospital, Dartmouth. A unanimously passed resolution at this meeting proposed that the society recommend that 50% of the Victoria General Hospital Beds, after the enlargement, should be classed as "preferred beds" which could be used by patients to receive treatment at the hands of their own physicians, instead of staff doctors. This arrangement would allow some ward beds to be used in the preferred class, all of which are presently so-called public beds.

It was pointed out by Health Minister Donahoe in the Legislature that the decision on the ratio of preferred (semi-private and private) to ward accommodation was a job for the Insurance Commission, while "the decision on whether wards in the Victoria General should remain opened or closed, as they are now, is one for the Victoria General Hospital Commission." In addition, Mr. Donahoe pointed out that the Medical Staff of the Victoria General Hospital worked without remuneration from the patients in ward beds. However, in the last several years, he said, that they have been permitted to bill insurance companies in cases where a ward patient was insured. Individual doctors did not collect these payments, he said, but they were pooled and used generally for such purposes as "attending refresher courses." The Victoria General Staff men had suggested that an honorarium system might be established which would give the highest payment to the specialists who were the shortest time in practice, and diminish the payment as the length of practice grew.

A rather interesting copy from the Calgary Herald which appeared in the April 11, 1960 issue of the Mail-Star, stated that "a visiting Russian doctor recently told the College of General Practice Meeting in Montreal, that G.P.'s are outmoded and should be replaced entirely by specialists. This attitude, of course, is entirely in line with Communist philosophy; the state is everything, the individual is nothing; hence the treatment is everything, the patient is nothing. In North America, however, there is a definite and very welcome place both for the individual and the G.P. The best concept is still that of the patient and his family, the General Practitioner and the Specialist in the background on call with his highly specialized training and skills. Fortunately, people in this part of the world are not yet machines and medical men have not yet reduced themselves to mechanics."

Dr. Robert Parkin has recently given up his practice at Moser's River and will begin post-graduate training in psychiatry. He is presently living in Dartmouth.

Valley Medical Society

Drs. J. P. McGrath and V. D. Schaffner of Kentville attended the recent meeting of the American College of Surgeons in Boston.

Western Nova Scotia Medical Society

Dr. W. C. O Brien recently had the misfortune to fall and injure his knee. He was treated in the Yarmouth Hospital.

Nova Scotia Chapter of the College of General Practice

March 27, 1960—The Halifax-Dartmouth Branch held a meeting, preceded by a buffet supper at the home of Dr. C. H. Reardon. Twenty-one members attended.

University

Dr. and Mrs. Duncan MacMillan, Nottingham, England, arrived in Halifax, March 17, 1960 from Montreal. Dr. MacMillan spoke to the post-graduate students in the psychiatry department of the faculty of medicine. Dr. and Mrs. MacMillan were entertained by Dr. and Mrs. R. O. Jones, Halifax, and left for New York on March 20, 1960.

National Cancer Institute grants were awarded totaling $24,790.00 to Drs. W. I. Morse, S. D. Wainwright, and J. A. McCarter.

The Victoria General Hospital, which presently has a total of 558 beds, of which 57 are private, 155 semi-private, and 346 public ward beds, will soon be expanded to a total of 853 beds—of which 92 will be private, 205 semi-private, and 556 public ward beds, it was announced recently by Dr. J. S. Robertson, Deputy Minister of Health. The editorial columns of the Halifax Mail-Star pointed out that this reduces the preferred bed category from a present 39% to a new 35%.

It is rather interesting that a lay publication should point out there are two basic considerations, "those who wish to preserve the status quo at the Hospital state that the public or teaching bed system must be maintained for training purposes; on the other hand, those who seek a change point out that patients, especially those who wish to pay the preferred accommodation rate, ought to be granted the right to choose their own medical practitioners.

In view of the heated debate, inside and outside medical circles, it is apparent that the issue will not be solved satisfactorily by present means. For this Reason, we advocate an inquiry into the matter by a special committee of the legislature, or some other outside group to collect facts, opinions, and make recommendations."

Births

Dr. and Mrs. Allison Barss, Rose Bay, a son, March 11, 1960.

Dr. and Mrs. J. F. Boudreau (nee Peggy Meade), a daughter, Halifax Infirmary, March 22, 1960. A sister for Tommy, John, Robie, Mary Louise, and Jimmy.

Dr. and Mrs. Allen A. Drysdale (nee Myria McCully), twin daughters, Carolyn Leigh and Susan Christine, Vancouver General Hospital, April 6, 1960.

Dr. and Mrs. T. B. Hall (nee Betty Lou Norman) a daughter, Halifax Infirmary, March 17, 1960. A sister for Heather and Pamela.

Dr. and Mrs. A. E. Johnston (nee Bette Wilton, R.N.) a daughter, Leslie Margaret, Grace Maternity, Halifax, April 1, 1960.

Dr. and Mrs. R. L. Ozere, a son, Thomas Vincent, Grace Maternity, Halifax, April 7, 1960.

Dr. and Mrs. D. M. Robb, a son Douglas Fraser, Halifax Infirmary, March 31, 1960.

Congratulations

To Dr. C. B. Weld, Head of the Physiology Department of Dalhousie University, on his recent election as a Fellow of the Royal Society of Canada. His work in recent years has been concerned with fat metabolism and the function of the small intestine in digestion. Dr. Weld was also recently elected President of the Waegwoltic Club.

Coming Meetings

May 31-June 2, 1960—Canadian Public Health Association—48th Annual Meeting, Halifax, N. S. (See Medical Care Section Programme outlined on page 168.)

June 13-17, 1960—Canadian Medical Association—93rd Annual Meeting, Banff, Alberta.

June 27-29, 1960—Medical Society of Nova Scotia—107th Annual Meeting—White Point Beach, N. S.

Canadian Public Health Association—48th Annual Meeting
Medical Care Section—Programme

Tuesday, May 31

General Session

Chairman, Dr. J. S. Robertson.

10.40-12.30 *Trends in the Organization and Financing of Health Care and their Implications for Public Health Services.*

Moderator: Dr. K. C. Charrón, Director of Health Services, Department of National Health and Welfare, Ottawa.

Participants

Dr. F. B. Roth, Deputy Minister of Health, Saskatchewan.
Dr. A. D. Kelly, General Secretary, Canadian Medical Association.
Dr. E. H. Lossing, Principal Medical Officer, Health Insurance, Department of National Health and Welfare.
Mr. Stanley Martin, President, Canadian Hospital Association.
Miss Elizabeth Reed, R.N., Acting Director-in-Chief, V.O.N.
Mr. Edward Goldberg, Research Officer, United Steelworkers of America.

2.30- 5.00 *Sectional Meeting.*

Chairman, Dr. G. G. Simms.

2.30 *Control of Standards and Utilization—Principle and Practice.*

Dr. G. G. Simms, Executive Director, Nova Scotia Hospital Insurance Commission.

3.00 *Management of Long Term Illness.*

Dr. A. H. Shears, Medical Director, Nova Scotia Rehabilitation Centre, and Assistant Professor of Medicine, Dalhousie University.

3.30 *The Children's Health Plan in Newfoundland.*

Dr. Leonard Miller, Deputy Minister of Health, Newfoundland.

4.00 *Business Meeting.*

Wednesday, June 1

3.00- 5.00 *Sectional Meeting.*

Chairman, Dr. Leonard Miller.

3.00 *Progressive Patient Care.*

Doctor E. J. Thoms, Administrator, Manchester Memorial Hospital, Hartford, Connecticut, Special Consultant, United States Public Health Service.

3.40 *Maritime Medical care—A Physician Sponsored Program.*

Dr. F. M. Fraser, President, College of General Practice of Canada; President, Maritime Medical Care.

4.20 *Diagnostic Groups for Hospital Statistics.*

Gordon H. Josey, DSc., Research Officer, Department of National Health and Welfare.

INFECTIOUS DISEASES—NOVA SCOTIA

Reported Summary for the Month of February, 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	2	3
Diarrhoea of newborn, epidemic (764)	0	0	0	0	2	3
Diphtheria (055)	0	0	0	0	5	2
Dysentery:						
(a) Amoebic (046)	0	0	0	0	0	0
(b) Bacillary (045)	0	0	0	0	172	73
(c) Unspecified (048)	0	0	0	0	54	1
Encephalitis, infectious (082.0)	0	0	0	0	3	2
Food Poisoning:						
(a) Staphylococcus intoxication (049.0)	0	0	0	0	0	0
(b) Salmonella infections (042.1)	0	0	0	0	0	0
(c) Unspecified (049.2)	0	0	0	0	53	54
Hepatitis, infectious (including serum hepatitis) (092, N998.5)	88	0	23	0	604	591
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	23	8
Meningococcal infections (057)	3	0	1	0	17	14
Pemphigus neonatorum (Impetigo of the newborn) (766)	0	0	0	0	4	0
Pertussis (Whooping Cough) (056)	18	0	1	1	497	393
Poliomyelitis, paralytic (080.0, 080.1)	1	0	0	0	31	2
Scarlet Fever & Streptococcal Sore Throat (050, 051)	244	0	147	0	4057	2189
Tuberculosis:						
(a) Pulmonary (001, 002)	19	4	15	2	359	280
(b) Other and unspecified (003-019)	4	0	4	0	94	0
Typhoid and Paratyphoid Fever (040,041)	0	0	1	0	28	7
Venereal diseases						
(a) Gonorrhoea—						
Ophthalmia neonatorum (033)	0	0	0	0	0	0
All other forms (030-032, 034)	42	0	25	0	1126	997
(b) Syphilis—						
Acquired—primary (021.0, 021.1)	1	0	0	0	0	0
—secondary (021.2, 021.3)	0	0	0	0	0	0
—latent (028)	4	0	2	0	0	0
—tertiary — cardiovascular (023)	0	0	1	0	0	0
— ,, — neurosyphilis (024, 026)	0	0	0	0	0	0
— ,, — other (027)	0	0	0	0	0	0
Prenatal—congenital (020)	0	0	0	0	0	0
Other and unspecified (029)	1	0	1	1	155*	140*
(c) Chancroid (036)	0	0	0	0	0	0
(d) Granuloma inguinale (038)	0	0	0	0	0	0
(e) Lymphogranuloma venereum (037)	0	0	0	0	0	0
Rare Diseases:						
Anthrax (062)	0	0	0	0	0	0
Botulism (049.1)	0	0	0	0	0	0
Cholera (043)	0	0	0	0	0	0
Leprosy (060)	0	0	0	0	0	0
Malaria (110-117)	0	0	0	0	0	0
Plague (058)	0	0	0	0	0	0
Psittacosis & ornithosis (096.2)	0	0	0	0	0	0
Rabies in man (094)	0	0	0	0	0	0
Relapsing fever, louse-borne (071.0)	0	0	0	0	0	0
Rickettsial infections:						
(a) Typhus, louse-borne (100)	0	0	0	0	0	0
(b) Rocky Mountain spotted fever (104 part)	0	0	0	0	0	0
(c) Q-Fever (108 part)	0	0	0	0	0	0
(d) Other & unspecified (101-108)	0	0	0	0	0	0
Smallpox (084)	0	0	0	0	0	0
Tetanus (061)	0	0	0	0	0	0
Trichinosis (128)	0	0	0	0	0	0
Tularaemia (059)	0	0	0	0	0	0
Yellow Fever (091)	0	0	0	0	0	0
N.S.U.	0	0	1	0	0	0

C — Cases D — Deaths

C.D.C. 2

*Not broken down

ANNUAL MEETING—1960

HOUSING APPLICATION FORM

The Medical Society of Nova Scotia

White Point Beach Lodge,
Queen's Co., N. S.
June 27th., 28th., & 29th, 1960

DR. L. A. MacLEOD,
Liverpool, N. S.

Please reserve for me the following accommodations—

A. **Main Lodge**
Double room with bath—including meals—$10.00 a day per person.

B. Cottage with single bedroom for two people—including meals—$10.00 a day.

C. Cottage with two bedrooms for four people, including meals—$9.00 a day.

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

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

Name of persons who will occupy above accommodations:

Name (Dr. and Mrs.) .....

Address .....

In view of large attendance expected, no single rooms will be available at White Point Beach Lodge unless cancellations permit. If coming alone and willing to share a room please check here..... 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 .....

This form valid until May 15, 1960. After that date the committee assumes no responsibility for rooms.

Confirmation of Accommodations

Dr. and Mrs.....have reservations as follows for White Point Beach Lodge.

Cabin No.....

Room No.....

Date .....

PSYCHOSIS AND ADDICTION TO PHENMETRAZINE (PRELUDIN). Evans J. Lancet, 2, 152, 1959.

Preludin, a drug similar to amphetamine and ephedrine is a sympathomimetic agent with euphoriant stimulating qualities. It was first used in 1954 in the treatment of obesity. Since then it has been given in asthma, in parkinsonism and as a euphoriant. This drug may be taken initially as an aid in weight reduction and then continued for its stimulating effect. Twelve cases, ten females and two males are reported in this paper. All found that the number of tablets taken had to be increased in order to obtain the same effect. All the patients except one had taken over ten tablets a day for many weeks. The amount taken did not appear to have a close relation to the severity of the illness. These twelve cases developed addiction to this drug producing a psychotic illness. The clinical picture cannot be distinguished from amphetamine or bromide psychosis, or alcoholic hallucinosis. Restlessness, pressure of talk, disturbance of mood, delusions and hallucinations are present without clouding of the consciousness. It is the author's opinion that preludin should be placed on a restricted list so that its abuse can be prevented.

J.O.G.

THE NATURAL HISTORY OF ERROR. Bean, W. B.: Arch. Int. Med., 105: 184, 1960.

This after dinner presentation to the Association of American Physicians abounds with the amusing yet thought provoking remarks characteristic of this writer, e.g. "who has not wondered at man's marvellous talent for making mistakes and then perpetuating them." After illustrating in a wide variety of moods; speculative errors, errors in the use of symbols, in translation, observer errors, the errors of mythical maladies, the error of forgetfulness, and several others; he concludes with remarks such as the following: "Our path is strewn with error, for only by error can we learn. It is error persisted in and perpetuated, error mistaken for truth, or error enthroned in the bleak robes of authority, which damages and destroys."

L. C. S.