THE NOVA SCOTIA MEDICAL BULLETIN

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Our Time to Speak

The "witching hour" regarding "Medicare" seems to have come and gone: - so far as can be determined, doctors have not been changed into mice, pumpkins or civil servants. The meeting of Provincial Health Ministers in Ottawa on September 23rd and 24th reiterated the conditions of the Prime Minister's statement of July. "Medicare" is to be run by the provinces. But the method of provincial operation must not violate any of the principles of Comprehensiveness, Universality, Portability or Government-agency operation. It would appear that the "Escape Hatch" of a physician's right to "opt out" and of a patient's right to benefits received outside the scheme *is acceptable* to the federal authorities. It was also indicated that existing **Doctor-sponsored** medical plans could be considered proper vehicles for operation of provincial "Medicare" plans - if their operation was open to government audit and if a minister of government were responsible to the Cabinet and, thus, to the people for the plan operation.

It is not known what recommendation Mr. Frank Rowe's Committee will make to the Nova Scotia government regarding "Medicare". We do know that the Federal people request a decision from the Provinces by the end of December, 1965. It is quite possible that Mr. Rowe will suggest M.M.C. as the "Agency" for "Medicare" in Nova Scotia. It should be pointed out that M.H.S.A. (Blue Shield) is **NOT** considered by the Federal Department of Health as a doctor-sponsored plan and, hence, is **NOT** eligible to be considered as an Agent.

Quite likely, by November 26th, we will know the general decision of the Rowe Committee. The Annual Meeting of The Medical Society scheduled for November 26th and 27th at the Lord Nelson Hotel in Halifax will, probably, be the place and time for release of this information.

This is the first year for **Council** of The Medical Society - but, by no means, will **Council** have everything cut-and-dried - on Saturday, the 27th, Council will report to the Annual Meeting. Decision of Council must be approved by the Annual Meeting.

The time and format of this year's Meeting was altered by C.M.A. Annual Meeting being in Halifax this Spring but the two days after the Dalhousie Refresher Course - November 26th and November 27th - are important dates for you, members of The Medical Society - we urge you to attend and participate in these most important discussions - don't think that the existence of **Council** will stifle debate. All members of the Society are welcome to attend Council and to participate in the discussion - and, at the Annual Meeting on Saturday, can have full say and **vote** on Council's decision. □

T.W.G.

Dalhousie Notes

VIII. 39th REFRESHER COURSE

LEA C. STEEVES, M.D.[1]

Halifax, N. S.

The 39th Dalhousie Refresher Course will be held November 22nd to 25th, 1965. The Faculty will include Dr. Roy G. Holly (Professor and Chairman of the Department of Obstetrics and Gynaecology, The Jefferson Medical College of Philadelphia); Dr. Colin C. Ferguson (Professor and Head, Department of Surgery, The University of Manitoba); Dr. K. J. R. Wightman (Sir John and Lady Eaton Professor, and Head of the Department of Medicine, The University of Toronto); and Dr. C. O. Carter (Director, Medical Research Council, Clinical Genetics Research Unit, Institute - of Child Health, London, England).

Monday afternoon's programme features "Anaemia in the Pregnant Woman", "Rh Problems", "Intra-Uterine Devices".

Tuesday afternoon's programme features Dr. Colin Ferguson discussing "Abdominal Tumours in Children" and a discussion on Abdominal Pain in Children.

The John Stewart Memorial Lecture "Drug Reactions" will be given by Dr. K. J. R. Wightman, Wednesday afternoon. "Peptic Ulcer from a Nutritional Point of View" is Dr. Wightman's topic on Thursday afternoon.

Dr. C. O. Carter will discuss "Genetics and Paediatrics", and Dr. W. A. Cochrane will speak on "Nutrition in Children" Thursday afternoon.

Each morning of the course will be devoted to Small Group Clinics. Several of the topics are: Myocardial Infarction, Asthma, Hypertension, Fevers and Convulsions in Children. How to Lower your Neonatal Mortality Rate.

"Socratic Luncheons" are planned for the first three days of the course and will consist of small groups sitting with a Faculty Member to discuss clinical problems.

The Victoria General Hospital, The Halifax Infirmary, The Grace Maternity Hospital, Camp Hill Hospital, and The Children's Hospital are all cooperating with the Faculty and the Refresher Course Committee to present a really excellent programme for this year's Dalhousie Refresher Course.

The following members of the Dalhousie Faculty of Medicine are among those participating in the programme:

Dr. C. M. Bethune
Dr. R. N. Anderson
Dr. W. A. Cochrane
Dr. R. C. Dickson
Dr. G. H. Flight
Dr. R. S. Grant
Dr. G. R. Langley
Dr. H. N. A. MacDonald
Dr. R. Parkin
Dr. B. S. Morton
Dr. S. J. Shane
Dr. A. S. Wenning
Dr. T. E. Kirk
Dr. S. F. Bedwell
Dr. L. Cudkowicz
Dr. A. A. Drysdale
Dr. S. G. B. Fullerton
Dr. D. A. Gillis
Dr. A. J. MacLeod
Dr. J. F. Nicholson
Dr. E. F. Ross
Dr. J. A. R. Tibbles
Dr. J. F. L. Woodbury
Dean C. B. Stewart
Dr. G. W. Bethune
Dr. P. Cudmore
Dr. F. M. Fraser
Dr. C. L. Gosse
Dr. D. R. S. Howell
Dr. R. M. MacDonald
Dr. R. L. Ozere
Dr. L. C. Steeves
Dr. K. E. Scott
Dr. M. G. Tompkins, Jr.
Dr. S. E. York

[1]Director of Post Graduate Division, Faculty of Medicine, Dalhousie University.

112th Annual Meeting

The Medical Society of Nova Scotia
(The N.S. Division of the C.M.A.)

The 112th Annual Meeting of the Medical Society will take place on November 26th and 27th, 1965 at the Lord Nelson Hotel, Halifax.

The Council of The Medical Society will meet for the first time. This body, representative of all facets of Medicine in Nova Scotia, is authorized by the Order in Council (16th February, 1965) approving the amended By-Laws of the Society (1964). The By-Laws were distributed to all members as an insert in the May 1965 issue of The Nova Scotia Medical Bulletin. The representatives to Council (members of Council) number approximately 100. The By-Laws (Chapter IX) state:-

"The Council shall be the governing body of the Society with its actions subject to the final approval of the Society at its Annual Meeting. It shall report to the membership at the Annual Meeting of the Society and, as warranted, through the pages of The Nova Scotia Medical Bulletin".

It is important to note that members of the Society who are not representatives to Council are welcome and encouraged to attend meetings of Council, but that only members of Council are entitled to vote.

Meetings of Sections within the Society may be held any time during the week at individual convenience. The Chairman of each Section is a member of Council.

It is to be noted that the Annual Meeting (November 26th and 27th) follows the Annual Dalhousie Refresher Course (November 22nd - 25th inclusive). The refresher Course Committee has agreed to have this regarded as the clinical programme for the Society's Annual Meeting.

The Annual Meeting of the Executive Committee will be on Thursday, November 25th. The first meeting of the new Executive will be a luncheon meeting on Saturday, November 27th, 1965.

The President and Executive Committee extend to all members a cordial invitation to attend.

Friday, November 26th, 1965

	- 8.30 a.m.	Registration
9.30 a.m.	- 12.30 p.m.	Meeting of Council
	1.00 p.m.	Luncheon
2.00 p.m.	- 4.00 p.m.	Meeting of Council
	4.00 p.m.	First Session of Annual Meeting
		(a) Report of Nominating Committee and election of Officers, representatives to Executive Committee
		(b) Other business
	7.00 p.m.	President's Reception
	8.00 p.m.	Annual Banquet
	10.00 p.m.	Dancing and Entertainment

Saturday, November 27th, 1965

	8.30 a.m.	Registration
9.00 a.m.	- 12.00	Meeting of Council
	12.30	First Meeting Incoming Executive
	2.00 p.m.	Second Session of Annual Meeting to receive report from Council.

The Ladies will have a Coffee & Sherry Party on Friday and Saturday at 10.00 a.m.

Social Registration fee will cover Friday evening's Social Functions
President's Reception - - Banquet - - Dance

112th ANNUAL MEETING

The Medical Society of Nova Scotia
(N.S. Division C.M.A.)
Lord Nelson Hotel, Halifax,
November 26th – 27th, 1965

Please print the name of the hotel or motel in which you wish to have accommodation:

First choice..... Second Choice.....

Other .....

Date of arrival: ..... Expected time of arrival: .....

Date of departure: .....

Room will be occupied by: Name(s) .....

Address .....

.....

.....

Accommodation required: (please check one)

Single ..... Double ..... Suite .....

Signed .....

Complete and forward to: The Executive Secretary,
The Medical Society of Nova Scotia,
Dalhousie Public Health Clinic,
University Avenue,
Halifax, N. S.

TELEPHONE MESSAGE CENTER

for Annual Meeting 1965

423-6451

This telephone number is made available for all incoming calls received for our members during the convention. Leave this number with your office or home in case a call is necessary.

This message center is located at the Society's Registration Desk in the Main Lobby of the Lord Nelson Hotel.

SUBSCRIPTION RATES

All members of The Medical Society of Nova Scotia receive *The Bulletin* without extra charge. The rate for Medical libraries, Hospitals and others is $6.00 p.a. Medical Students at Dalhousie, $2.00. All correspondence should be addressed to: Subscription Dept., Nova Scotia Medical Bulletin, Public Health Clinic, Halifax, N. S.

FORTY YEARS AGO

From the Nova Scotia Medical *Bulletin*, November, 1925

Sir Arbuthnot [Lane] also spoke [at the Clinical Congress of the American College of Surgeons as reported by Dr. G. H. Murphy] at the opening meeting of the Hospital Standardisation Department. One looks with eagerness to the views of men who can watch the activities of Hospital Standardisation from neutral ground. No doubt there have been many times when some of us wondered if it were all worth while. The principle of Standardisation from the start has been that the hospital is basic and fundamental in any move which makes for greater efficiency in surgery and medicine. The logic for improvement and standardisation of all hospital agencies is therefore inevitable. The plan adopted by the American College of Surgeons was highly commended by the distinguished English surgeon. Himself a representative of the conservative and staid profession of the Old Country, yet he believed fully in going to the public with an educational programme, in order that wide spread intelligence on health matters would form a staunch buttress for the support of better hospitals, better health legislation, and better doctors. □

ERRATUM

Somehow in Dr. Omaboe's paper on "Rheumatoid" Heart Disease (October *Bulletin* p. 257) the caption to figure 1 appeared but figure 1 did not. Our apologies to Dr. Omaboe and his readers.

Editor.

SEARLE ANNOUNCES

the one & only

◉ a new modality in oral contraception...

Ovulen 1 mg.*

◉ a new molecule

"Each tablet of Ovulen 1 mg. contains: ethynodiol diacetate 1.0 mg., mestranol 0.1 mg."

Quick Section Diagnosis

DOUGLAS WAUGH, M.D.C.M., M.Sc., PH.D., F.R.C.P.(C)[1]

Halifax, N. S.

The quick section can be one of the most valuable of all aids to the surgeon. It can provide him in many cases with instant diagnosis as the basis for a one-step definitive surgical procedure, enabling him to avoid delays, risks or excessive surgery. To reach this satisfactory state, a number of vital factors must be harmoniously blended. Everyone concerned must be aware of the capacities and limitations of the method and of the individuals engaged in its application. If we demand of a technique what it is incapable of delivering, the result can only be frustration.

The most fundamental aspect of quick section diagnosis, and one that is all too frequently ignored, is the recognition that it represents a close collaborative effort between surgeon and pathologist. In the ideal situation, the surgeon has advised the pathologist prior to surgery of the type of case with which he is dealing, and has indicated the type of information he hopes the quick section will provide. Except for breast biopsies, advance consultation will often inform the surgeon whether or how quick section will help.

Armed with this information, and having been told of any previous biopsy results, the pathologist ought to be able to give a rapid and accurate answer in most cases.

It is most important that quick section facilities be in the immediate vicinity of the operating suite. If they cannot be placed **close** to the operating room, then there is no value in having them outside of the pathology laboratory itself. If a quick section room is on the O.R. floor, it should be an area that is not used for other purposes. The pathologist performing a quick section is under just as much tension as is the surgeon in charge of the case, and it is unreasonable to ask him to do his work in a room that doubles as a plaster room, interne's lab, or serves some other function. The minimum facilities for a quick section room include:

1. **A cryostat.** This relatively new type of freezing microtome is so superior to the old CO_2 model that it should be available even in relatively small hospitals.
2. A vibration-free dissecting bench, sink, and excellent lighting and a binocular microscope.
3. An exhaust fan or preferably, a fume hood to get rid of odors.

Given the advantages of friendly collaboration with the surgeon and good working facilities, what kind of service can be expected? This will depend to some extent on the ease of communication between the surgeon and the pathologist. Ideally, they should be together in the operating room at the time the biopsy is taken. They may often assist each other in choosing the biopsy site. The pathologist can then incise the specimen in the manner that will be most informative to the surgeon, and can often make a definitive diagnosis on the spot. If circumstances prevent this mask-to-mask contact, then there should be an intercom between the operating room and the quick section laboratory so that questions and information can be easily exchanged.

Provided with these circumstances or a reasonable approximation of them, and given also a depth of understanding of a respect for each other by surgeon and pathologist, the quick section can reach its highest value. There should be agreement first that with rare exceptions the primary (and preferably only) function of the quick section is **to provide information that will immediately influence the course of the surgical procedure.** If the decision in question is that of choosing between a simple and a radical procedure, a quick section is usually essential. Quick sections with microscopic examination are also extremely valuable in checking excision margins for residual malignancy. There are, however, certain neoplastic situations where the quick section has relatively little value. Where it is obvious that an intestinal malignancy has involved the serosa, there is little to be gained from a quick section of the resected end of the bowel several cm. from the tumour - the closest margin to the patient is the serosa, and this is already known to be involved. In lienitis plastica of the stomach, it should be assumed that there will be residual tumour in the non-excised portion, even though the margin may appear free on microscopic examination.

The case of the diagnostic lymph node biopsy presents special problems in quick section diagnosis. It is most important here to realize that however

[1]From Department of Pathology, Dalhousie University and Pathology Institute, Halifax, N. S.

ominous the other clinical features may be, the lesion in question may turn out to be other than neoplastic. For this reason, in almost all diagnostic lymph node biopsies, the surgeon should divide the specimen under sterile conditions, giving half to the pathologist and sending half for culture. It is astonishing how often this elementary precaution is overlooked and an important diagnostic opportunity is missed. Precise diagnosis of lymphomas, although often possible in quick section, should not be expected since special staining procedures are frequently required.

There are some important aspects of the quick section that are unrelated to the capability of the surgeon or to the diagnostic capacity of the pathologist but which, if recognized, can contribute materially to the value of the procedure. If the pathologist's office is remote from the operating room, the nursing staff should keep him informed of the operating schedule and should warn him of changes or delays. Good O.R. supervisors can often anticipate delivery of the tissue with such precision that the surgeon produces it just as the pathologist enters the operating suite. Too often, however, the pathologist spends a great deal of his time drinking coffee in the surgeon's dressing room while he waits for a specimen whose late arrival might have been predicted.

If it is possible for the surgical staff to irritate the pathologist (and thus perhaps impair his judgement) the reverse is equally true. Wherever possible, the pathologist should avoid making an equivocal report to the surgeon. He should nearly always be able to do this if he includes in his diagnostic language the words "I don't know". Too often when the pathologist is uncertain, he confronts the surgeon with a list of differential diagnostic possibilities, each of which may require a different surgical approach and for none of which there is incontrovertable evidence. In this situation, most surgeons would prefer to be advised to close and await definitive sections rather than be invited to add the pathologist's problems to those he already has. In most cases the pathologist should be able to give a firm report that the lesion is "benign", "malignant" or "don't know". Sometimes, however, as with some pancreatic biopsies, he may report "inflammation only - you may be close to the edge of a tumour" - indicating the advisability of a deeper biopsy if this is surgically feasible.

There are certain lesions where accurate pathological diagnosis is difficult and where quick section is of limited value. This is the case with many papillary epithelial lesions such as duct papilloma of the breast, bladder papillomata and polyps of the large intestine. In general, it is safer to await the preparation of paraffin sections for definitive diagnosis in such cases. The pathologist should however be willing to "freeze almost anything" if only to broaden his experience.

Another aspect of quick section diagnosis concerns the surgeon's report to the patient after surgery. In general it is unwise to use a negative quick section as a basis for an optimistic report to the patient, since definitive sections sometimes reveal unpleasant lesions that were not seen initially. Positive quick section diagnosis on the other hand, is much more reliable and, if the pathologist agrees, can be reported to the patient or to a relative.

Although only a few aspects of quick section diagnosis have been dealt with here, I believe the points that have been made are the most important ones. If good technical facilities are available to a pathologist and surgeon who are willing to collaborate closely, they can together accomplish remarkable service to the patient. □

Present Concepts of Hemostasis

JOHN W. STEWART, M.D.[1]
Halifax, N. S.

Introduction

There are few other disciplines of medical research in which the vigor, vitality and complexity of modern medicine are so characteristically displayed than the study of the coagulation of the blood. As pointed out in several recent reviews, this subject has long fostered a feeling of dismay in the novice, who balks at the controversy and confusion in the literature. In the past decade, however, significant attempts have been made at clarification, so that now the ordinary physician is expected to have a sound general knowledge of the subject. The patient with a bleeding disorder can now benefit greatly by a rational approach to his diagnosis and management. This is not to mention the oftentimes life-saving measures available to patients with thromboembolic diseases that have stemmed from the study of anticoagulation and the use of anticoagulants.

As with other biologic processes, the concept of the dyamic state has assumed prominence. No longer are coagulation, hemostasis and anticoagulation considered static processes. It is essential, in the light of current knowledge, to consider these processes as dynamic, with a fine balance necessary to maintain homeostasis. When vascular injury occurs, a smooth chain reaction of unbelievable complexity is set into motion with hemostasis usually resulting. In the "normal" human with "normal" daily activity, it is likely that breaks in the lining of the small vessels are constant occurrences. It is not hard to imagine that coagulation takes place at these sites constantly, with the mechanisms of anticoagulation working simultaneously to prevent the chain reaction from becoming disadvantageous.

It is of paramount importance for the clinician not to be discouraged by the controversies and complexities of the present day knowledge of this subject, but to remember that in the majority of patients he can usually make a diagnosis with an adequate history and physical examination and "routine" laboratory investigations. Often, even the latter are not essential, but only confirm the clinical impression.

Hemostasis

Hemostasis is the arrest of the flow of blood from an injured vessel. The degree to which bleeding occurs following injury varies greatly even among "normal" individuals. There are, however, at least four factors which are generally held to determine the efficiency of the hemostatic mechanism in any individual. A defect in any one, or a combination of these factors may lead to abnormal bleeding response to injury. The concept of so-called "spontaneous" bleeding is a dubious one, particularly in the light of a dynamic state suggesting constant vessel injury at a microscopic level. The first of these factors is the extravascular tissues. Their integrity, or the variations in the resistance they offer to escaping blood may determine the bleeding response of a particular part of the body to injury. The "black-eye" is a well known example. In other situations in which there is "easy" bruising, such as in the elderly, in poor nutrition, in some women, and in conditions such as the Ehlers-Danlos syndrome, it is likely that poor extravascular support is a deciding factor. If combined with a defect in one of the other factors, there may be serious bleeding.

The second factor is the vasculature itself. It is obvious that the integrity of the lining of the vessels, considering the fluid nature of the blood, is vital to normal hemostasis. It is known that the first response to vessel injury is contraction of the vessel itself. The third factor, the size and type of vessel is important in determining the extent of bleeding. A defect at the small vessel or capillary level may cause serious bleeding. There are certain disease states in which such a defect produces abnormal bleeding, (e.g. scurvy, dysproteinemias, certain infections, hereditary hemorrhagic telangiectasia, anaphylaxis, uremia). The common form of bleeding in vascular disorders is purpura. The fourth factor, the coagulability of the blood, is also intimately involved with hemostasis in combination with the other factors. This will be dealt with subsequently.

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[1]Fellow in Medicine, Department of Medicine Dalhousie University and The Victoria General Hospital.

The Platelet

In 1882, Bizzozero described the blood platelets and also incriminated them in the coagulation of the blood, demonstrating their participation in the clot and their adhesiveness. In 1906 Wright discovered the origin of the platelets from the megakaryocytes in bone marrow. Since that time, and particularly in the past decade, there has been intensive study into the function of the platelet and new concepts have arisen. With the use of the electron microscope, the ultrastructure of the platelet has been extensively investigated. The study of the platelets in disease states has resulted in a rewarding relation of structure to function. The normal platelet has a protoplasm bounded by a membrane 60 angstroms thick, which becomes segmented early in clotting. Four well defined structures have been described: Fifty to one hundred round or oval granules of marked density predominate, (granulomere alpha); next appear a few small mitochondria, (granulomere beta); microvesicles and tubules, (granulomere gamma); and granules with clear interiors, (granulomere delta). Platelet factor 1 and 3 activity* has been ascribed to the common granulomere alpha, while platelet factors 2 and 4 are associated with the hyalomere. Soon after venipuncture, the round disc shape of the platelet changes and long pseudopodia protrude, after which the change known as viscous metamorphosis occurs. Further progressive changes result in spreading forms with loss of granular content. The early aggregates in clumps reveal platelet membranes packed together - these later disappear under the microscope. Normal fibrin patterns are geometric and the platelets are seen to form a lattice-like network over the fibrin, acting as focal points for the interlacing fibrin network. The most probable explanation of clot retraction is the contractability of the platelets placed at these focal points.

Braunsteiner has described defects in pseudopodial formation in patients with thrombasthenia. Impairment of platelet process formation and aggregation has been demonstrated in leukemia, pernicious anaemia and tuberculosis. With platelet factor 3 deficiency, platelets were seen to spread excessively and were unable to release their granules. Other qualitative platelet defects have been described in sprue, scurvy and uremia.

Platelet Metabolism

The metabolism of the platelet is amazing. Recent studies have shown it to contain several coagulation co-factors, serotonin, histamine, adenosine triphosphate, contractile protein, inactive factor V, fibrinolytic activity and lipids. The metabolic activity of the platelet is geared to accomplish its hemostatic function. Platelets have been known to consume oxygen, display glycolytic activity requiring glucose, produce lactic acid and have recently been shown to contain large amounts of adenosine triphosphate, which decreases with clotting. This latter energy metabolism has recently been related to ultrastructure and it has been demonstrated that the platelets of patients with so-called thrombasthenia of Glanzmann have markedly diminished adenosine triphosphate content.

When a vessel is injured, there is almost instantaneous constriction of the vessel and aggregation of platelets at the rent. The process of viscous metamorphosis is necessary for an efficient hemostatic plug of platelets. Tissue thromboplastin initiates the formation of a small amount of thrombin, along with plasma clotting factors adhering to the platelets. This thrombin, although minute in amount, is necessary for viscous metamorphosis to take place. Once this occurs, adenosine triphosphate is split by adenosine triphosphatase in platelets and adenosine diphosphate results. This latter compound has been shown to be a further stimulus to viscous metamorphosis. Also, when metamorphosis takes place, platelet co-factor 3 is released, and thrombin is formed via the intrinsic system which also serves to stimulate further viscous metamorphosis.

Recent work on platelet adhesiveness has yielded interesting results. The stickiness is probably related to pseudopodial protrusion. It is not thought to be related to generation of thrombin, and the adenosine diphosphate of injured cells has been incriminated as the stimulus.

In general then, platelet function is related to (1) the ability to form an efficient hemostatic plug; (2) the contribution of a phospholipid (platelet co-factor 3) to the coagulation mechanism; and lastly (3) the relationship between proper platelet function and the integrity of the capillaries.

Clinical Considerations

Clinically then, one must consider both qualitative and quantitative defects which may lead to abnormal function and possible adnormal bleeding. Purpura is once again the common form that this abnormal bleeding takes. As always, an adequate history and physical examination are vital to proper diagnosis. The simple tourniquet test will be positive. The retraction of the clot is another observation of great value. This will be impaired. The number of platelets in the peripheral smear, and their morphology can easily be assessed.

The normal life span of the platelet is 8 - 14 days. However, of importance clinically is the fact that stored whole blood platelet levels fall

*Platelet factor 1 = adsorbed Factor V.
,, ,, 2 = thrombin-fibrinogen accelerator.
P. f. 3 = phospholipid P. f. 4 = anti-heparin effect.

appreciably after three hours and precipitously after twenty-four hours. Fresh platelets transfused will survive normally, unless the patient is bleeding or there is an antiplatelet antibody present.

Finally, with the use of newer radioactive techniques, the thrombocytopenic states have been classified with reference to platelet production and destruction into: (1) Those with primary production defect; (2) Those with predominant production defect; and (3) Those with predominant peripheral destruction. In Idiopathic Thrombocytopenic Purpura, a dual mechanism of peripheral destruction and production deficit has been shown to exist.

Coagulation and the Clotting Factors

"Classic" Hemophilia (Factor VIII deficiency) makes up about 80% of known cases of faulty coagulation. There are, however, pathologic states associated with defects in most of the known coagulation factors. Rather than discuss each factor separately, starting with Factor 1, it is much more useful to describe each factor as it fits into the smooth, dynamic chain reaction of blood coagulation.

In 1955, the concept of "intrinsic" and "extrinsic" pathways of thromboplastin formation was put forth. The extrinsic system consists of tissue thromboplastin and the factors needed to activate it, namely VII, X, V and IV or calcium. The activity of this system is much more rapid in formation than the slower "intrinsic" system. The intrinsic system operates in the absence of the powerful tissue thromboplastin and consists of a suitable surface for contact and Factors XII, XI, X, IX, VIII, V, calcium and Platelet factor 3. Note that factor VII is only required by the extrinsic system and Factors V and X are necessary for both systems. The concept of "intrinsic" and "extrinsic" systems is a convenient way of deciding which factors are affected by which common tests of coagulation. Tests which reflect the activity of the intrinsic system are those that do not involve the addition of tissue thromboplastin and include the whole blood clotting time, prothrombin consumption test, thromboplastin generation test, partial thromboplastin-time test and plasma recalcification time. Those tests which reflect "extrinsic" system activity involve the addition of tissue thromboplastin and include the one and two-stage prothrombin time tests and the stypven time. It should be noted that deficiency of either fibrinogen or prothrombin must of necessity affect all of these tests.

OVERALL SCHEME OF BLOOD COAGULATION

PHASE I	Generation of Thromboplastin
PHASE II	Prothrombin Conversion to Thrombin
PHASE III	Fibrinogen Conversion to Fibrin
PHASE IV	Destruction of Fibrin by Fibrinolysins

Considering now, the current coagulation scheme, one must think in terms of four phases of coagulation in the smooth chain of events. The first phase (Phase I) is the formation of thromboplastic activity by both the "intrinsic" and "extrinsic" systems. Phase II is that concerned with the formation of thrombin by both "intrinsic" and "extrinsic" thromboplastin. Phase III is the formation of fibrin from fibrinogen, and Phase IV is the dissolution of fibrin by the fibrinolytic enzyme system.

Intrinsic Factors

In Phase I of the coagulation scheme, consider first the elaboration of "intrinsic" thromboplastic activity. In the presence of a suitable foreign surface, Factor XII is activated. This factor was demonstrated by Ratnoff et al in 1958 to be the accelerator of clotting, activated by glass contact. It is not activated by contact with silicone tubes. There is no bleeding disorder associated with defective Factor XII activity and recently this has been attributed by some possibly to an associated decreased fibrinolytic activity in the plasma of these patients. Active Factor XII combines with Factor XI in the presence of calcium ions to yield a complex which activates Factor IX. Factor XI or PTA, was first described in 1953 by Rosenthal et al.

Factor IX (PTC or Christmas factor) was described simultaneously in 1952 by Biggs et al and Aggeler et al, thereby ending the mystery of Pavlovsky's finding of two clinical hemophiliacs with mutually correctable blood. Once activated, Factor IX combines with Factor VIII, Factor X and calcium ions to produce an intermediate product. This product stimulates platelet agglutination and viscous metamorphosis with subsequent liberation of platelet Factor 3. Factor X was described in 1957 by Hougie et al in patients thought to have Factor VII deficiency but with an abnormal Thromboplastin Generation Test. They found it subsequently to be present in plasma and serum, absorbed by barium sulfate and reduced by coumarin therapy. Platelet Factor 3 activity, as previously described, has been shown to be present in the granulomere alpha of the platelets, released during viscous metamorphosis. Platelet Factor 3 combines with Intermediate Product 2, which in turn combines with Factor V and calcium to produce socalled "intrinsic" or plasma thromboplastin or prothrombinase. Factor V (proaccelerin or labile factor) was demonstrated in 1947 by Owren to be a distinct entity. It is the factor which decreases in stored blood (unless frozen) and prolongs the prothrombin time in this situation. It is not present in serum.

Extrinsic Factors

Consider now the so-called "extrinsic" system and the mechanism of elaboration of its activity.

With injury to the lining of the vessels, a potent tissue factor comes in contact with the blood. This is the so-called "tissue" thromboplastin. This activates Factor VII. Deficiency of this factor was described by Alexander et al in 1952. This factor is different from Factor V in that it is present in serum and stable in storage. It is absorbed by barium sulfate and decreased by coumarin therapy. It is felt not to be involved in the "intrinsic" system and therefore its deficiency is manifest simply by prolonged one-stage prothrombin time, (Quick's test) with other tests normal. Once activated, Factor VII combines with Factors V, X and calcium to produce "extrinsic" thromboplastin.

Considering phase 2 of coagulation, the "extrinsic" and "intrinsic" thromboplastic activity is capable of converting prothrombin to thrombin. Seegers et al proved the existence of a single substance from which thrombin is wholly derived - namely prothrombin.

Prothrombin is an alpha- 2 globulin, with a molecular weight of 68,000, while Thrombin migrates in the albumen fraction and its molecular weight is probably 31,000. Thrombin is a highly specific proteolytic enzyme. It is necessary in small amounts for viscous metamorphosis of platelets to occur, and its fate is that of neutralization or destruction by antithrombin. In phase 3 of the coagulation scheme, thrombin reacts with fibrinogen to form fibrin, the only directly observable phenomenon in blood coagulation.

It has been shown that the first effect of thrombin on fibrinogen is proteolysis, probably due to disruption of arginyl-glycine bonds. The result is the formation of fibrin monomers and fibrin peptides A & B. The next step is polymerization of the fibrin molecules which apparently take up an end-to-end alignment to form primitive fibrils, or intermediate polymer, and later these fibrils become arranged side to side to form the coarse strands of fibrin seen under the light microscope. This latter step is dependent on a plasma factor (so-called fibrin stabilizing factor) and calcium ions.

Phase 4 of the coagulation scheme involves the fibrinolytic system and this will be discussed subsequently. First, there are a few facts concerning coagulation which are worthwhile mentioning. Generally speaking, there is a relatively large "reserve" of clotting factors. This should be kept in mind when considering results of tests and the clinical expression of an hereditary trait. For example, in Hemophilia, cases with no detectable factor VIII are usually severely affected, and those with more than 5% are almost invariably mild. Fibrinogen is normally present in concentration of 300-500 mgm. per 100 c.c. of plasma, but as little as 100 mgm.% seems adequate for hemostasis. This is so for the other factors and most tests are not able to detect such small quantities. In addition, it should be noted that in the clinically valuable one-stage prothrombin time, apart from prothrombin, factors VII, IX and X are necessary.

Anticoagulation

Mention has been made of the "dynamic" aspect of blood coagulation. It is felt that there is a fine balance between the continuing, slow "natural" coagulation of the blood, and "natural" or physiologic anticoagulation, thus preserving homeostasis and the fluidity of the blood.

After coagulation, thrombin disappears rapidly from the blood, probably due to neutralization by antithrombin. As stated by Biggs and MacFarlane in their text, "The mechanism for neutralizing thrombin in whole plasma must be very powerful," particularly if one considers that if all of the prothrombin in 10 c.c. of plasma were converted to thrombin, there would be enough to clot all of the blood in the body.

There is strong evidence for the presence of a thrombotic-fibrinolytic homeostatic balance in man. This balance is upset by various forms of stress, including exercise, anxiety, trauma, burns, surgery on lung, prostate, uterus and liver. The role of tissue activation of the fibrinolytic system in the walls of arteries as a protective mechanism against thrombosis and atherosclerosis has been postulated. The central enzyme of this system is plasmin. Both plasmin, and its inactive precursor plasminogen are globulins. The action of plasmin is similar to that of trypsin. Although the mechanism of "in vivo" activation of plasminogen is unknown, it is known that certain tissues and urine, as well as milk and tears, contain an activator or proactivator. It is known that streptokinase, and staphylokinase activate plasminogen, and the former has been used with success in the lysis of clots in human patients; also, as an adjunct to thrombectomy. It is known that phospholipids directly affect the clotting mechanism without effect on fibrinolytic activity. The reverse is true of chylomicra and low density lipo-proteins; a factor which may have some bearing on the timing of certain thrombotic accidents. The hypofibrinogenemia of *abruptio placentae* is due to defibrination, or massive clotting initiated by tissue thromboplastin from the placental site. There is no significant fibrinolysis.

As with the other finely balanced biologic processes in the body, there is an intrinsic antifibrinolysin activity as well. This is thought to inhibit plasmin-fibrin interaction due to the presence of substances resembling or containing structural units present in plasmin's natural substrate, fibrin. Active fibrinolysis therefore hinges on the balance between plasmin-fibrin interaction and plasmin-inhibitor interaction.

Heparin

Heparin was discovered by Howell in 1916. In 1933, Charles and Scott of Toronto demonstrated its general distribution in the animal body and subsequently it has been found to be a mucoitin polysulfuric acid, originating in the mast cells. Although it is the most potent and reliable anticoagulant drug, its function in the body is still indefinite. It is also known to have a lipemia clearing effect, and in this respect, the relation of certain lipids to the fibrinolytic system should be remembered. Due to its strong electrical charge, heparin interferes with at least twenty enzyme systems. Its antithrombic effect is particularly pronounced. This is due to activation of antithrombin. By acting on factors V, IX, XI and XII, Heparin blocks thromboplastin generation and neutralizes tissue thromboplastin. Although once considered the natural anticoagulant, this is now doubtful. It is generally felt that it does not exist freely in the blood. Its usefulness in the treatment of numerous thrombotic conditions has been well documented. It is the only drug of value in the urgent situation. There are practically no contraindications. It should be closely watched with impaired renal function. Often, however, one is faced with the treatment of a probably fatal condition for which there is no other drug of equal proficiency.

The Oral Anticoagulants

Along with heparin, the oral anticoagulants have long proven their worth. There is much more controversy in the use of these drugs than heparin, but probably not as much as is commonly stated. Once the decision to use them is made, individualization in management is paramount, along with a comprehension on the part of the physician as to the many physiologic, pharmacologic and pathologic features pertinent to anticoagulant therapy. The coumarin congeners and indandione compounds comprise the oral anticoagulants used today. These agents depress at least four coagulation factors; namely, II (prothrombin), VII, IX and X. These are the factors produced in the liver which are Vitamin K dependent. It is felt that the sequence in which they are affected depends on their individual half lives in the circulation. Thus X declines slowly and IX rapidly The coumarins probably act by blocking synthesis of these factors in the liver, through competitive inhibition. There is wide individual variation in drug response. The patient's nutritional status regarding Vit. K may be one variable. Other factors include the physical and chemical properties of the drugs themselves - dicoumarol is slower acting than sodium warfarin. Substantial lowering of the clotting factors (remembering the large reserve) takes at least 24-48 hours, and it is obvious that urgent situations will not be helped. The gravid female is more resistant and it has been shown that factors VII and X are elevated during pregnancy.

It is remembered that the one-stage Prothrombin Time measures II, VII and X. This probably explains the bleeding sometimes seen when the test results are in the "therapeutic" range, since factor IX has often been found to be low in these cases. The new thrombo-test of Owren also measures factor IX and has received some favorable reviews. Fibrinogen is also important in these tests. As protamine sulphate is used in heparinized patients who bleed, so Vit. K has proven useful with the oral anticoagulants. With small doses some correction is evident within a few hours and full correction usually in 24 hours.

Acquired Coagulation Inhibitors

Mention should be made of acquired coagulation inhibitors. These have been collectively called circulating anticoagulants. Hougie and Margolius et al have divided them into three classes: (1) Those occurring in hemophilia A (AHF or VIII deficiency) and Christmas disease (IX deficient); (2) Those appearing after childbirth, and (3) Those in middle aged people in association with a variety of diseases. Those in Class (1) usually have had blood transfusions. The anticoagulants inhibit VIII or IX. In Class (2) the anticoagulants all arise within a year of delivery and inhibit factor VIII (AHF). Those in Class (3) also inhibit factor VIII. There have also been cases described in which there is inhibition of factor V and PTA. Therapy in general for these disorders is difficult. Unless protamine sulfate will shorten the clotting time "in vivo", a diagnosis of hyperheparinemia should not be made. It is unlikely that heparin is ever a circulating anticoagulant if the patient has not been receiving heparin therapy.

Clinical Approach to a Bleeding Disorder

With a general idea of the coagulation-anticoagulation system, and the other factors involved in hemostasis, the physician should be able to approach a patient with a bleeding disorder in a rational manner, making a diagnosis in most instances. To reiterate, a comprehensive history and physical examination is the first, and by far, most important step. It should be remembered that a positive family history is valuable but not essential for the diagnosis of hereditary defects. Also, the history of major surgery, tonsillectomy or tooth extraction without bleeding virtually eliminates the possibility of congenital hemorrhagic disorders. It also should be noted that although purpura is the typical form of bleeding with platelet defects or vascular disorders, and excessive bleeding after trauma is more likely with coagulation defects, defects in any part of the hemostatic mechanism may cause abnormal bleeding with

surgical procedures. Acquired defects in all parts of the hemostatic mechanism may occur and special note should be made of drug history, and complete functional inquiry. One also should be aware that bleeding is often exaggerated by patients, and on the other hand, mild defects are often extremely difficult to demonstrate, even with laboratory tests.

In the hereditary disorders, Hemophilia and Christmas disease, the inheritance pattern is sex-linked recessive with bleeders almost entirely males. Most other plasma factor deficiencies are autosomal recessive and consanguinity of parents often exists. In the physical examination, evidence of petechiae, purpura, hemarthroses, lymphadenopathy, characteristic telangiectasia and splenomegaly is searched for particularly. In addition, simple tests of value can be carried out at the bedside; namely, bleeding time, tourniquet test, stool and urine for occult blood. The first two mentioned, if abnormal, may be due to platelet dysfunction, thrombocytopenia and/or capillary fragility. The stained blood smear will give some idea of the platelet number and quality and may uncover other disease such as leukemia, pernicious anaemia, etc. The clotting time in glass and silicone, prothrombin time and clot formation and retraction are other tests which can be done routinely as rough screening tests. If the silicone clotting time is prolonged and prothrombin time is normal, then the defect is in the first phase of clotting and TGT and prothrombin consumption may be added as further diagnostic aids. If silicone time is normal and prothrombin time prolonged, then deficiency is in the second phase of clotting and tests to evaluate more specifically defects in prothrombin and factors V, VII and X are required. The therapy of bleeding disorders must be individualized and proper diagnosis is a big step in therapy.

The correction of underlying disease, transfusion of whole blood plasma, platelet concentrates and plasma factors must be coupled with local therapy and general treatment of the patient.

Test	Platelet Disorders		Vascular Disorders	Coagulation Disorders
	Thrombo-Cytopenic	Thrombopathy		
Bleeding Time	I	I	I	N
Clotting Time	N*	N	N	I
Capillary Fragility	I	I or N	I	N
Clot Retraction	D	D	N	N
Platelet Count	D	N	N	N
Platelets on Smear	D	N or Bizzare Forms	N	N
Prothrombin Time	N	N	N	I or N
Thromboplastin Generation Test (TGT.)	N or D	N or D	N	D or N

N = Normal
I = Increased
D = Decreased
* Prolonged only if severe

It must be ovious from the foregoing that the study of the coagulation of the blood and hemostasis in general has come a long way in the past two decades, and has led us to a state of fair comprehension in spite of complexity and still lingering mystery. It should also be clear that the intensity of interest in this field will lead to many new facts in the next five years. The physician should be aware of existing concepts and be ready for additions in the near future. It is, after all, the patient who benefits from new knowledge, and who makes this knowledge worthwhile. □

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A Simple Approach to the Investigation of Hemorrhagic Disorders

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Knowledge of the coagulation mechanism has advanced so rapidly and so greatly that some physicians may feel that it has become too complex for them to grasp. Whenever they are confronted with a possible hemorrhagic state, they tend therefore to limit themselves to three standard tests - namely bleeding time, clotting time and prothrombin estimation - without a clear understanding of the rationale behind these tests or of their limitations.

Bleeding Time - This is a rough and inaccurate measure of capillary integrity and platelet sufficiency. These parameters can also be investigated by the Hess tourniquet test, by examination of a stained blood film, by observation of clot retraction and by a platelet count, with precision at least as high, and certainly with greater specificity.

Clotting Time - All but the last few seconds of spontaneous coagulation are occupied by Stage 1 of the **intrinsic system** and the major portion of this, in the activation of platelets (the formation of so-called platelet factor 10). This activation is dependent on a number of relatively uncontrollable and quixotic factors. In addition contamination by tissue juice has a profound effect, bringing into play the very much more rapid **extrinsic system.**

The red cells contribute nothing to the coagulation mechanism and merely make interpretation somewhat more difficult. It is true that the Lee and White clotting time enables clot quality (fibrinogen) and clot retraction (platelets) to be observed but there are other and better tests for these. The activated partial thromboplastin time (*vide infra*) is a better measure of the total coagulation mechanism.

Prothrombin Time - The one stage test provides an overall measure of the **extrinsic coagulation system.** It reveals with moderate precision a deficiency of either prothrombin, fibrinogen or accessory factors V, VII and X. It is not a measure of prothrombin *per se.*

Activated Partial Thromboplastin Time (P.T.T.) - This is a measure of the intrinsic coagulation mechanism. It represents the clotting time of plasma under controlled conditions. The technique is very similar to the one stage prothrombin test and similarly it will be affected by deficiency of prothrombin, fibrinogen, factor V and factor X. In addition deficiency of factors VIII, IX, XI and XII will prolong it.

Recapitulation

Patients suspected of a coagulation disorder can be divided into three groups on the basis of the results of these last two tests: -

(a) both tests in normal range
(b) PTT abnormal, prothrombin time normal
(c) both tests abnormal

Use of Additive Fractions - The coagulation factors can be divided into two further groups by fairly simple manipulation depending on whether or not they are normally present in: -

(a) aged serum -
(b) fresh plasma which has been adsorbed with certain inorganic precipitates. (TABLE 1)

Thromboplastin Generation Test - This is a somewhat complex and time consuming test in which the components of Stage I are assembled by the combination of additive fractions from the patient's blood and from normal blood in dilutions high enough to inhibit actual clotting of the reaction mixture. Aliquots of this reaction mixture are tested over a period of time for thromboplastic activity by observing their ability to clot normal plasma.

APPLICATION

By intelligent use of the above tests it is usually possible to pin point by laboratory methods the probable deficiency or combined deficiencies in any given case but the clinical findings (which alas! are so often withheld from the pathologist) are of inestimable value.

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TABLE I

ADDITIVE FRACTIONS[1]

	Factors Present								
	I	II	V	VII	VIII	IX	X	XI	XII
	F.	P.	L.	S.	A.H.G.	XMAS	S.-P.	P.T.A.	H.F.
Aged Serum	−	−	−	+	−	+	+	+	+
Adsorbed Plasma	+	−	+	−	+	−	−	+	+

Legend:

F.	Fibrinogen	S.	Stable factor	S.-P.	Stuart-Prower Factor
P.	Prothrombin	A.H.G.	Antihemophilic globulin	P.T.A.	Pl. Thromboplastin Antecedent
L.	Labile factor	XMAS	Christmas factor	H.F.	Hageman Factor

The following investigative regime is suggested: -

(1) HISTORY - Has hemorrhagic state existed from childhood? Is there a family history? Is it apparently sex-linked (hemophilia, Christmas disease) or autosomal (factors V, VII, X, fibrinogen, Glanzmann's disease, hereditary telangiectasia etc). Has there been exposure to any drugs or any recent illness?

(2) CLINICAL PICTURE - What is the type of hemorrhage?

(a) Spontaneous purpura only? (probable platelet or capillary abnormality).

(b) Abnormal bleeding related to trauma or not confined to skin and mucosa? (probable coagulation disorder).

- what other signs or symptoms are present?

(3) PHYSICAL EXAMINATION-Anemia, lymphadenopathy, hepatosplenomegaly, state of nutrition, avitaminosis, jaundice, fever or systemic disease etc.

(4) LABORATORY INVESTIGATION-These cases are usually emergencies and the maximum helpful information should be gathered as soon as possible. It is suggested that tests be grouped as follows: -

First Stage

The first stage of the investigation is concerned with rough classification of the type of abnormality which may be present. It consists of four simple screening tests:

(*a*) Hess capillary fragility test.
(*b*) Complete blood count.
(*c*) Partial thromboplastin time.
(*d*) One stage prothrombin time.

Interpretation

A. (*a*) (*b*) (*c*) (*d*) *all normal.*
Probably no hemorrhagic disorder. Possible excess plasmin activity.

B. (*a*) *abnormal, all others normal*
Probable defect of capillaries (scurvy, hemorrhagic telangiectasia, von-Willebrand's disease, Ehlers Danlos syndrome etc., but specific diagnosis not yet established).

C. (*a*) & (*b*) *abnormal,* (*c*) & (*d*) *normal.*
Defect of the platelets which may be combined with a capillary defect as well (thrombocytopenia, morphologic defect of platelets, pernicious or aplastic anemia, leukemia, septicemia etc.). Specific diagnosis may be possible.

D. (*c*) *abnormal,* (*d*) *normal*
Probable hemophilia or para-hemophilia (deficiency of factors VIII, IX, XI or XII).

E. (*c*) & (*d*) *both abnormal.*
Probable deficiency of fibrinogen or one of the liver dependent factors involved in the prothrombin complex (prothrombin and factors V, VII and X), the presence of so-called circulating anticoagulants or a combined deficiency.
(*c*) *normal* (*d*) *abnormal* - deficiency of factor VII only.

Second Stage

The second stage of investigation involves slightly more complex or time consuming tests and the use of additive fractions.

A. *No apparent abnormality.*
Despite no apparent abnormality in the First Stage tests, if the clinical condition warrants it, further tests as detailed below should be carried out with discrimination.

[1]Additional help may sometimes be gained by a knowledge of the factors present only in fresh normal plasma, plasma from a patient who has recently been placed on short acting coumarin therapy, stored plasma, heated plasma and plasma from cases of known deficiencies, Russell's Viper Venom, heated tissue thromboplastin etc. but we will not concern ourselves with discussion of these in this VERY simple approach.

B. & C. *Probable platelet or capillary abnormality* -
- bleeding time.
- platelet count.
- bone marrow aspiration.
- clot retraction.

D. *Probable hemophilia or parahemophilia* -
Abnormal (*c*) corrected by:
(i) adsorbed plasma but not serum - factor VIII.
(ii) serum but not adsorbed plasma
- factor IX.
- factor X (very rare).
(iii) either serum or plasma - factor XI (very rare).[2]

E. *Deficiency of prothrombin complex, fibrinogen, etc.* -
Abnormal (*c*) & (*d*) corrected by:
(i) adsorbed plasma but not serum - factor V.
(ii) serum but not adsorbed plasma
- factor VII.
- factor X (very rare).
(iii) both serum and plasma together - combined defect.
(iv) neither serum nor plasma
- factor I (clot quality also very poor).
- factor II (corrected by normal, stored or coumarin plasma).
- circulating anticoagulants (corrected only by large amounts of plasma.)[3]

Third Stage

Although the above very simple scheme appears to have solved all possible problems - and in many cases will have done so - results may not be clear cut. They should certainly be confirmed by other tests; but tests ordered with discrimination and not in a "shot gun" fashion.

D. *Probable hemophilia or parahemophilia* -
- Thromboplastin generation test (TGT).
- prothrombin consumption.

E. (a) *Spontaneous prothrombin complex deficiency* -
- TGT (combined defect).
- liver function tests.
- tests for steatorrhea.

(b) *Fibrinogen lack* -
- semiquantative fibrinogen assay.
- chemical assay of fibrinogen.
- liver function tests.

(c) *Inhibitors or anticoagulants* -
- effect of toluidine blue or protamine sulphate on PTT of test plasma.
- effect of test plasma on PTT of normal plasma.

Fourth Stage

Tests in this stage are more fancy and run the gamut from assays of individual factors to clinical microscopy of capillaries and electron microscopy of platelets. We need not concern ourselves with these here apart from the statement that occasionally a defect may be so mild that the simpler tests detailed above will not unmask it. In such cases more complex tests may be indicated.

SUMMARY

Investigation of hemorrhagic disorders is not as difficult as is commonly supposed. Although the hemostatic and coagulatory mechanisms are amazingly complex, a remarkable amount of information can be gained by intelligent use of a very few tests - specifically, a **complete blood count, partial thromboplastin time** and **one stage prothrombin time** together with clinical assessment.

On the other hand the usual order for bleeding and clotting times gives scant specific information and the results may be misleading. □

[2]Factor XII deficiency is not associated with a bleeding disorder but it will show a similar picture which is, however, correctable with heated normal plasma.

[3]It is a law of coagulation that both coagulation factors and inhibitors act in high dilution and in many cases this fact enables differentiation of the two types of disorder. On the one hand if there is a deficiency of a factor this is correctable by the addition of a small aliquot of normal fraction. If the disorder is due to excess inhibitor a large amount of normal plasma will be required.

Appreciation

Dr. A. F. Miller

Arthur Frederick Miller died at his home in Kentville October 5, 1965, twenty-six days before his 88th birthday and eighteen years following his retirement as Medical Superintendent of the Nova Scotia Sanatorium, a position which he had held since January 1, 1910.

Dr. Miller was the last of the pioneers in the treatment of tuberculosis. In the year of his birth the disease was regarded as generally incurable. Five years were to elapse before Robert Koch discovered the tubercle bacillus and it was not until 1884 that the first Sanatorium of any importance on the North American continent opened its doors at Saranac Lake. In the year of his retirement the weapons for the final conquest of the disease were at hand. Largely through the efforts of individuals like himself, the tuberculosis problem was well understood not only by the profession but by the general public. The death rate had fallen markedly and modern therapy was well on its way to full fruition. For the first time in the long battle a drug was proving effective and he was able to supervise the first injection of streptomycin at the Nova Scotia Sanatorium on February 20, 1947. In the year of his death so much progress has been made in the eradication of tuberculosis that we are experiencing the unfortunate trend of fear being replaced by complacency.

Shortly after obtaining his medical degree from Dalhousie University in 1904 Dr. Miller was stricken with tuberculosis. This event, tragic as it was to the young doctor of 1905 proved an inestimable boon to the thousands of Nova Scotians who were to contract the disease in the ensuing forty years.

He left immediately for Saranac Lake where he came under the influence of Dr. Edward Livingstone Trudeau who some twenty years earlier had initiated the first effective treatment for pulmonary tuberculosis at his Cottage Sanatorium in the Adirondacks. As his own disease came under control he realized that he had an unusual opportunity to become an expert in the diagnosis and treatment of tuberculosis and under the guidance of Trudeau, Baldwin and Brown he took full advantage of it. In 1909 he was asked to come to Nova Scotia and take over the small Provincial Sanatorium at Kentville which had been established in 1904. This offer presented a real challenge and urged on by Trudeau he began his life's work in an institution consisting of one building with accommodation for eighteen patients on January 1, 1910. His only tools were his fingers, his stethoscope and his microscope but he began to build and he built well. The story of the growth of the Nova Scotia Sanatorium from eighteen beds in one building in 1910 to an institution of twenty buildings with four hundred beds and all modern facilities at his retirement has been told before. It was a battle all the way but he was equal to it. His was a voice crying in the wilderness but by extensive lecturing, writing and badgering he made that voice heard. He always said that in matters of treatment he was not an experimenter but certainly he was an innovator. We physicians of today with all our equipment must shudder when we visualize the young Miller in 1914 checking a pulmonary haemorrhage by initiating the first artificial pneumothorax in the Maritime Provinces. He had no X-ray, he had no fluoroscope. By physical examination, he diagnosed the source of the bleeding and he had only percussion and auscultation to tell him the extent and the effectiveness of the pneumothorax he had produced. When his friend Edward Archibald suggested that thoracoplasty was of value in some cases of advanced tuberculosis he sent one of his patients to the Victoria General Hospital in 1921 and persuaded Archibald to come from Montreal and himself carry out the procedure. When the then radical approach of pulmonary resection was being advanced he had his own surgical department at the Sanatorium. He gave his blessings to his surgeon, Vernon Schaffner who then carried out the first lobectomy for tuberculosis in Nova Scotia in November 1944.

From 1915 onwards physicians came to the Sanatorium to learn from him and when one reviews the history of tuberculosis in Nova Scotia over the past forty years there are very few doctors connected with it who were not trained by Dr. Miller.

He was a great physician, a superb teacher, a true friend. Good-bye, Dr. Miller. Thank you very much. □

J.J.Q.

Glaucoma

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Glaucoma is an eye disease characterized by an increased intraocular pressure, which if not relieved will result in blindness. It causes typical nerve fibre bundle damage, producing arcuate defects in the field of vision.

The rate of aqueous humor production by the ciliary body and the resistance to outflow of aqueous humor at the angle of the anterior chamber determines the height of the intraocular pressure. Clinically the pressure is estimated by the Schiotz tonometer, as shown in Figure 1.

The average intraocular pressure is 16 mm. Hg. Over 21 mm. Hg. should be considered suspicious and reason for further evaluation while over 24 mm. Hg. is considered abnormal. Figure 2.

Glaucoma is found in approximately 2% of the population over 40 years of age. It therefore affects over 100,000 people in Canada and is more prevalent than diabetes.

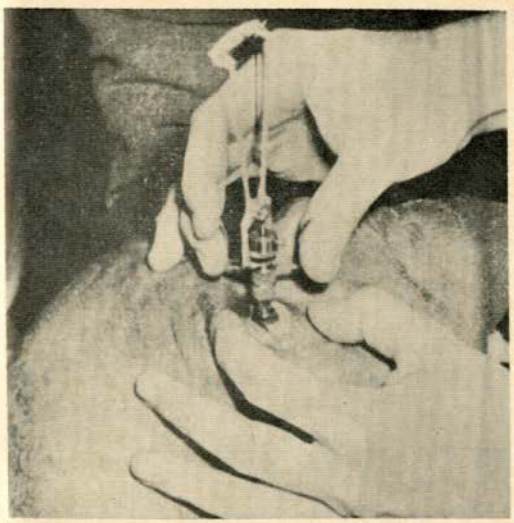

FIGURE 1.

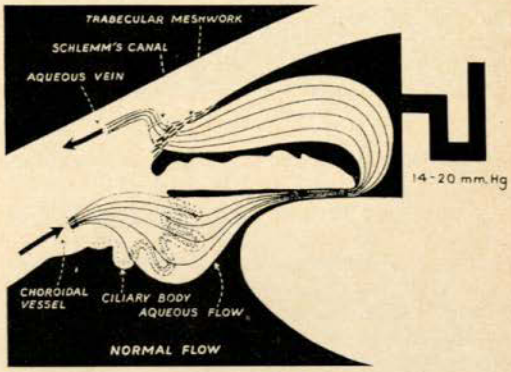

FIGURE 2.

If undetected and untreated it leads to eventual blindness. It is the cause of 12% of blindness in Canada. Because of these statistics, the Federal and Provincial Governments have seen fit to establish glaucoma clinics across Canada, to assist in the diagnosis and control of this insidious disease. There are two glaucoma clinics in the Atlantic Provinces i.e. Saint John and Halifax and another will soon be established at St. John's.

Classification of Glaucoma

(1) Angle-closure or acute glaucoma
(2) Open-angle or chronic glaucoma
(3) Combined mechanism glaucoma
(4) Congenital glaucoma.

ANGLE CLOSURE OR ACUTE GLAUCOMA

(Figure 3)

This glaucoma occurs typically in hyperopic narrow angled eyes which usually have shallow anterior chambers. Tension elevation tends to occur abruptly, causing typical symptoms of halos, hazy vision and ocular pain. A combination of circumstances precipitates the acute attack: a higher pressure in the posterior than in the anterior chamber, a pupillary resistance to the forward flow of aqueous humor at the site of iris contact with the lens, a laxity of the peripheral iris and a possible

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Figure 1, 2, 3 and 4, from Diagnosis and Therapy of the Glaucomas by Becker, B. and Shaffer, R.N. by permission of the Authors.

FIGURE 3.

contribution of vascular factors. The resulting forward displacement of the peripheral iris toward the trabecular meshwork can lead to partial or complete closure of the angle.

Medical therapy usually restores the pressure to normal, but it is imperative to perform a peripheral iridectomy which usually bypasses the pupillary block and normalizes the outflow if the trabecular meshwork is not damaged. This procedure will prevent further attacks of angle closure. In many cases it is necessary to perform the procedure on the fellow eye as investigation may reveal that it also has a very narrow angle which is susceptible to closure.

OPEN ANGLE OR CHRONIC GLAUCOMA

(Figure 4)

In open angle glaucoma the increased intraocular pressure is due to the pores in the trabecular meshwork, becoming smaller for some unknown reason. Symptoms are usually negligible until extensive ocular damage has occurred. This is why it is important to check the pressure of every one over 40 at intervals so the disease will be discovered in an early stage. It is to be remembered that this type of glaucoma cannot be cured, but only controlled.

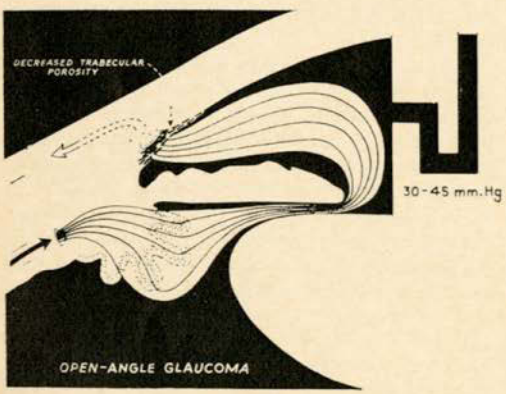

FIGURE 4.

There is a high prevalence of glaucoma (15-20 per cent) among people over the age of 40 who have a family history of glaucoma. It is therefore important that all patients with established glaucoma be informed of the familial aspects of the disease.

Therapy is usually medical using miotics (eg. pilocarpine) carbonic anhydrase inhibitors (eg. acetazolamide), sympathomimetic agents (eg. epinephrine bitartrate). Surgery is indicated only if there is progression of visual field loss on all variations of maximum tolerated medical therapy.

The follow up is very important and patients should be seen at least every 3-4 months throughout their lifetime.

COMBINED MECHANISM GLAUCOMA

This category includes various combinations of angle-closure and open-angle glaucoma.

CONGENITAL GLAUCOMA

This category refers only to these cases in which anomalies of the anterior segment are present at birth. The early signs are watery eye, photophobia and blepharospasm. Late sign are cloudy cornea and enlarged eye.

It should be kept in mind that congenital glaucoma is frequently associated with other congenital anomalies such as neurofibromatosis, Sturge-Weber syndrome, Lowe's syndrome etc.

The treatment is essentially a surgical problem to provide an adequate outflow pathway. □

References

1. Becker, B. and Shaffer, R.N.: Diagnosis and Therapy of the Glaucomas, St. Louis, 1961, The C. V. Mosby Company.

INFORMATION ABOUT CANCER

The National Cancer Institute of Canada is currently distributing the American Cancer Society publication "CA - A Cancer Journal for Clinicians", to all Doctors of Medicine in Canada who wish to receive it. This is a well laid out, easily read journal, which contains up-to-date information which doctors might wish to know in respect to their cancer patients. Complimentary subscriptions may be obtained on request to the National Cancer Institute of Canada, 790 Bay Street, Toronto 2, Ontario.

The Bacteriology and Chemotherapy of Chronic Bronchitis[1]

Drug treatment of chronic bronchitis should not necessarily be delayed until bacteriological reports are obtained. The most important of the microorganisms in this condition is Hemophilus influenzae. In long-term chemotherapy, the tetracyclines are the drugs of choice.

Logical chemotherapy must be based on sound bacteriological principles, but this does not necessarily mean that the bacteriologist must always be consulted before a drug is given. In some syndromes the probability that a particular microorganism is causative is so high that "blind" chemotherapy is permissible. Chronic bronchitis belongs to this category.

The prevalence of *H. influenzae* in patients with purulent sputum seems to remain substantially constant from year to year. In contrast, the prevalence of the pneumococcus varies widely in different years and individual isolation rates have little meaning. Further, it seems that the prevalence of pneumococci in sputum cultures does not provide a reliable guide to the true occurrence of the organism in the lung, and it has been demonstrated that pneumococci are often found in sputum, probably as a consequence of contamination from the throat. The true role of the pneumococcus in chronic bronchitis is not easy to assess but there is no doubt that its main importance concerns exacerbations. Chronic pneumococcal infection of the bronchi without coincident *H. influenzae* is common, and when antibiotics eliminate pneumococci, pus does not always disappear.

The bacteriological principles upon which chemotherapy should be based may be summarized as follows: In patients without acute exacerbation but with purulent sputum, *H. influenzae* is the outstanding pathogen and the pneumococcus is probably of minor importance; in acute exacerbations without pneumonia, the pneumococcus assumes greater importance although its prevalence is variable from year to year; if pneumonia complicates bronchitis, there is the possibility of staphylococcal infection although the pneumococcus is the most common organism; there is no clear evidence that organisms other than *H. influenzae* and the pneumococcus are pathogenic in bronchial infections.

Therefore, the chemotherapy of patients without pneumonia should be directed against *H. influenzae* and pneumococci. Fortunately, neither organism readily develops resistance to antibiotics in common use and thus routine sensitivity tests are unnecessary. However, other problems of chemotherapy must be considered.

Selecting Chemotherapy

Patients whose sputum is always mucoid or contains only eosinophil "pus" derive no benefit from chemotherapy whatever organisms are present. Therefore, it is necessary to be sure that true pus is present before chemotherapy is prescribed. Eosinophil "pus" is most common in patients with an allergic history.

The greatest problem is the recurrence of infection when therapy is stopped. Such recurrences are usually associated with *H. influenzae.* The rapidity of recurrence suggests relapse of the original infection, but could mean a reinfection with a different strain.

The tetracycline group of drugs have come to be considered the drugs of choice for longterm chemotherapy since they can be given by mouth and are relatively free of side effects.

In the last decade several clinical trials have been carried out in an attempt to assess the precise value of chemotherapy. A detailed analysis of all the reports seems unnecessary since a number of general conclusions are apparent. Perhaps the most important is the attention that these trials have drawn to the care needed in interpreting results.

Assessing Progress

The objective criteria most commonly used for assessment of progress were sputum purulence and number and duration of exacerbations. But

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J. Robert May, M.D., *British Journal of Diseases of the Chest*, April, 1965.

[1]Reprinted from the Abstracts of the National Tuberculosis Association, September, 1965.
Printed through cooperation Nova Scotia Tuberculosis Association.

sputum purulence and number of acute exacerbations are not necessarily linked. A patient can recover clinically from an exacerbation sufficiently to return to work although his sputum is still purulent. Likewise, a patient may have lost his pus but be unable to return to work because of increased dyspnea. In general, loss of pus will indicate control of *H. influenzae* - the basic chronic infection. Clinical improvement in an exacerbation may well be due to suppression of a pneumococcus and, since the prevalence of this organism varies from year to year, a given antibiotic may not be equally effective in different trials.

Both intermittent and continuous chemotherapy are valuable forms of treatment if the patients are properly selected. The patient who has occasional purulent sputum but is "mucoid" for most of the time is a candidate for intermittent therapy, while the one whose sputum is purulent throughout the winter *must* be given continuous therapy.

Other Drugs

As regards drugs other than the tetracyclines, trials have covered phenoxy-methylpenicillin, erythromycin, oleandomycin, novobiocin, sulphamethoxypyridazine, and "sulphatriad." Taking account of all the factors, there can be no doubt that tetracycline itself is usually preferred. Sometimes a combination of tetracycline with another antibiotic will succeed where tetracycline alone fails. Tetracycline in concentrations that can be attained in tissues acts by bacteriostasis.

Bactericidal therapy is theoretically possible with ampicillin. It has been found that the highest concentrations appear in the sputum when pus is present, and as the inflammation subsides the level falls until it can often no longer be measured. The crucial factor in treatment is that the infecting organism should be killed before the sputum concentration falls to the ineffective level. As this fall may occur in only a day or so, it is essential that treatment should be directed toward attaining very high sputum levels in the first few hours. It is clear that the sputum as well as the tissue must be sterilized if relapse is to be prevented. Failure of the antibiotic to penetrate into mucoid bronchial secretions may explain the failure of bacteriostatic therapy to give more lasting benefit. If the microorganisms in the secretions are inaccessible to the antibiotic and there are no phagocytes present, there is no reason why they should not remain viable indefinitely and able to cause a fresh infection as soon as the bronchial tissue is freed from protective antibiotic. □

Clinical Staff Conferences

Effective October, 1965

We list below as many regular meetings, rounds, conferences and clinics as we have knowledge of in the Halifax area. This list does not pretend to be complete, but it will be revised as necessary and will be published periodically for the information of members.

All events listed are open to any interested physician, and a cordial welcome is promised in every case.

We hope to publish information of similar arrangements at other hospitals throughout the Province in the future. Chairmen or Secretaries of Medical Staffs are invited to send time tables for publication. Anyone spotting an inaccuracy is also begged to let us know.

THE CHILDREN'S HOSPITAL

Cardiology Rounds	Monday	10:30 a.m.
Neonatal Conference (Grace Hospital)	Monday	11:00 a.m.
Orthopedic Conference	Monday	12:00 noon
Cardiology Conference	Monday	4:00 p.m.
Admission Rounds	Tuesday	8:00 a.m.
Medical Grand Rounds	Wednesday	9:00 a.m.
Admission Rounds	Thursday	8:00 a.m.
Metabolic Conference	Thursday	11:00 a.m.
Neurology Conference (alternates weekly)		
Case Presentation	Thursday	4:00 p.m.
Surgical Conference	Friday	11:00 a.m.
Radiology Conference (biweekly)	Friday	3:00 p.m.
Ward Rounds	Daily	9:00 a.m.

GRACE MATERNITY HOSPITAL

Staff Meeting Luncheon	Monday (Last)	12:00 noon
Obstetrical Conference	Tuesday (Third)	5:00 p.m.
Ward Rounds	Daily	9:00 a.m.
Journal Club Luncheon	Thursday	12:15 p.m.
Prenatal Clinic	Tuesday, Thursday, Friday	2:00 p.m.
Well Baby Clinic	Tuesday, Thursday, Friday	2:00 p.m.
Postnatal Clinic	Tuesday, Thursday, Friday	2:00 p.m.

HALIFAX INFIRMARY

Department of Anesthesia			
Weekly Conference	Monday	2:00- 3:00 p.m.	O. R. Suite
Department of General Practice			
Monthly Conference	4th Thursday	8:30 p.m.	3C Clinic Room
Weekly Joint Conferences - attended by Department members as follows : -			
with the Department of Surgery	Saturday	9 a.m.	
with the Department of Medicine	Thursday	11 - 12:30	
with the Department of Pediatrics	Friday	11- 1 p.m.	
with the Department of Psychiatry	Wednesday	9-10 a.m.	
with the Department of Obs.-Gyn.	Friday	12- 1 p.m.	
Department of Medicine			
Joint Conference with X-ray Dept.	Wednesday	11-12 a.m.	4C Clinic Room
Grand Rounds	Tuesday	12- 1:15 p.m.	4C Clinic Room
Intern-Resident Training Conference	Thursday	11-12:30 p.m.	4C Clinic Room
Department of Obstetrics & Gynecology			
Weekly Rounds	Thursday	11- 1 p.m.	3C Clinic Room
Monthly Meeting	3rd Thursday	11-12 a.m.	3C Clinic Room
Interns Weekly Conference	Tuesday	4- 5 p.m.	3C Clinic Room
Department of Ophthalmology			
Weekly Conference	Tuesday	6:30 p.m.	Outpatient Dept.
Monthly Conference	3rd Tuesday	6:30 p.m.	Outpatient Dept.
Department of Otolaryngology			
Weekly Conference	Tuesday	6:30 p.m.	E.N.T. Department
Monthly Meeting	2nd Thursday	7- 9 p.m.	E.N.T. Department
Department of Pathology			
Clinical Pathological Conference	4th Friday	12- 1 p.m.	Auditorium
Department of Pediatrics			
Grand Rounds	Friday	11-12 a.m.	Pediatric Dept.
Pediatric Conference	Friday	12- 1 p.m.	Pediatric Dept.
Department of Psychiatry			
Case Presentation	Wednesday	9 a.m.	3C Clinic Room
Weekly Conference	Wednesday	9-11 a.m.	3C Clinic Room
Monthly Conference	3rd Wednesday	9-11 a.m.	3C Clinic Room
Daily Ward Rounds		8 a.m.	Psychiatry Dept.
Department of Radiology			
Weekly Conference	Thursday	3:30 p.m.	Radiology Dept.
House Staff Conference	Tuesday	1- 2 p.m.	Radiology Dept.
Department of Surgery			
Weekly Conference	Saturday	9 a.m.	3C Clinic Room
Department of Urology			
Weekly Conference	Thursday	12 noon	Urology Dept.
Monthly Meeting	2nd Thursday	12 noon	Urology Dept.

VICTORIA GENERAL HOSPITAL

Department of Medicine

Cardiac Working Conference	Monday	1:00- 2:00 p.m.	X-ray Conference Room
Grand Medical Rounds	Tuesday	8:30-10:00 a.m.	4th Floor Class Room
Cardiac	Tuesday (1st & 3rd)	1:00- 2:00 p.m.	OPD Conference Room
Pulmonary	Tuesday (2nd & 4th)	1:00- 2:00 p.m.	OPD Conference Room
Haematology	Tuesday	2:00- 4:30 p.m.	3rd Floor OPD
Gastroenterology	Wednesday (1st & 3rd)	1:00- 2:00 p.m.	OPD Conference Room
Haematology	Wednesday (2nd & 4th)	1:00- 2:00 p.m.	OPD Conference Room
Neurosurgery-Neurology	Wednesday	9:00-10:00 a.m.	Pavilion Conf. Room
Rheumatology	Wednesday	9:00-11:00 a.m.	3rd Floor OPD
Metabolism, Endocrinology, Renology	Thursday	1:00- 2:00 p.m.	OPD Conference Room
Neurology	Friday (1st & 3rd)	1:00- 2:00 p.m.	OPD Conference Room
Cardiopulmonary Pathology	Friday (4th)	1:00- 2:00 p.m.	OPD Conference Room
Cardiology	Friday	2:00- 4:00 p.m.	3rd Floor OPD

Department of Surgery

Weekly Conference	Thursday	5:00 p.m.	5th Floor Clinic Room
Ward Rounds			
Surgery A	Friday	8:00 a.m.	6 South
Surgery B	Thursday	8:00 a.m.	6 North
Surgery C	Wednesday	8:30 a.m.	6 South
Surgery D	Saturday	9:00 a.m.	6 North
Orthopaedics	Tuesday	11:00 a.m.	4 West
Out Patients Clinics			
Surgery A	Friday	9:30 a.m.	Outpatient Dept.
Surgery B	Thursday	9:30 a.m.	Outpatient Dept.
Surgery C	Wednesday	9:30 a.m.	Outpatient Dept.
Surgery D	Tuesday	9:30 a.m.	Outpatient Dept.

Department of Gynaecology

Ward Rounds	Daily	9:00 a.m.	5 West
Grand Rounds	Saturday	9:00 a.m.	5th Floor Clinic Room
Pathology Conference	Tuesday (First)	5:00 p.m.	Path. Institute
Tumour Clinic	Tuesday & Friday	11:30 a.m.	Outpatient Dept.
Gyn. Outpatient Clinic	Monday	2:00 p.m.	Outpatient Dept.

Department of Radiology

Therapeutic Radiology Ward Rounds	Thursday	8:30 a.m.	6 South
Diagnostic Radiology Conference	Daily	3:00 p.m.	Radiology Dept.
Proven Case Conference	Thursday	1:00 p.m.	Radiology Dept.
Clinical Conference	Thursday (3rd)	5:30 p.m.	X-ray Conf. Room
Departmental Conference	Friday (Last)	1:00 p.m.	X-ray Conf. Room

Department of Psychiatry

Ward Rounds	Monday & Friday	10:30 a.m.	Pavilion
Seminar	Friday	4:00 p.m.	West Annex Conf. Rm.
Child Guidance Clinic	Thursday	9:00 a.m.	Auditorium
Case Presentations	Monday, Tuesday Friday & Saturday	9:00 a.m.	Pavilion Conf. Room

Department of Urology

Conference	Daily	4:30 p.m.	6 West

Department of Anaesthesia

Conference	Friday (First)	3:30 p.m.	

Nova Scotia Tumour Clinic

Conference	Friday (Third)	12:30 p.m.	Tumour Clinic
Clinics			
Breast	Monday	2:00 p.m.	Tumour Clinic
Lymphomas	Tuesday	2:00 p.m.	Tumour Clinic
Paediatric	Tuesday (4th)	2:00 p.m.	Tumour Clinic
Gynaecology	Tuesday & Friday	11:00 a.m.	Outpatient Dept.
Ophthalmology	Tuesday	2:30 p.m.	Outpatient Dept.
Skin, Soft Tissue & Intestine	Tuesday	11:30 a.m.	Tumour Clinic
Head and Neck	Wednesday	11:00 a.m.	Tumour Clinic
Ear, Nose & Throat	Wednesday	2:30 p.m.	Outpatient Dept.
Urology	Thursday	10:00 a.m.	Outpatient Dept.
Breast	Thursday	11:00 a.m.	Tumour Clinic
Pulmonary & Gastric	Friday	12:00 noon	Tumour Clinic
Orthopaedic	Friday (2nd & 4th)	10:00 a.m.	Tumour Clinic

Personal Interest Notes

FAIRY GOD-MOTHER - COMPUTER STYLE.

One of our recent Medical Graduates was recently thrilled to receive a cheque for over $21,000. for his first three months' services to a Government agency. Tentatively his bank account burgeoned. Behold before the artificial bloom faded, there was a permanent growth of $60. visible remaining from its stay.

ANTIGONISH-GUYSBOROUGH MEDICAL SOCIETY

Late in September the second annual meeting on planning for community health services was held at St. Martha's Hospital, Antigonish with Dr. L. P. Chiasson acting as chairman. Members from all societies and institutions interested in the proposed programme were present from Antigonish, Guysborough and parts of Cape Breton.

The plan was presented to the meeting by **Dr. J. M. Hoc,** medical research investigator Boston College and was accepted by those present. Present at the meeting were Dr. J. J. Stanton, administrator, Department of Public Health in Halifax, Dr. G. Graham Simms, executive director, Hospital Insurance Commission; Dr. Gordon Hatcher, dean of preventive medicine, Dalhousie: and Dr. T. W. Gorman, representing St. Martha's Hospital as well as representatives of the Sisters of St. Martha, and of St. Francis Xavier University, etc.

Dr. Peter Delva, who since his Residency at the Children's Hospital, Halifax has been specializing in Paediatrics at Antigonish, has been appointed Assistant Professor of Preventive Medicine in Queen's University, Kingston, Ontario. His maximum interest lies in the field of Geriatrics.

CAPE BRETON MEDICAL SOCIETY

No. 20, Local Union, Glace Bay, is going to press for the appointment of a resident doctor to the local hospital so that immediate medical care will be available when a patient is brought to the institution, and thus prevent the lag which sometimes occurs unavoidably under the circumstances.

Early in September, **Dr. Hugh Kirkpatrick** was elected as President of the North Sydney Kinsman club for the coming year.

Dr. H. J. Devereux of Sydney was guest speaker at the graduation exercises of Saint Elizabeth Hospital School of Nursing on September 26.

Dr. Herman Sampson, Cheticamp was unanimously endorsed to contest the riding of Inverness-Richmond in the forthcoming Federal Election, as Progressive-Conservative candidate, opposing the Hon. Allan J. MacEachern, (Canada's Minister of Labour).

"**Mental Health Group Plays An Important Role**" is the heading of an article in the Cape Breton Post of September 11 on the Canadian Mental Health Programme and its associated White Cross Volunteers with reference especially to the Cape Breton Centre. Since moving to its new quarters last February, patients are being served in ever increasing numbers. In 1964, more than 4000 interviews were conducted and 630 new cases treated. Services were extended to about 350 patients in general hospitals in the area and visits made to schools, courts and jails. Daily visits to Cape Breton County Hospital were made by Dr. Mian, Dr. Cornelius Donovan and Dr. Eileen McDonagh, along with Dr. Maqbul Mian are the psychiatrists presently on the staff.

HALIFAX MEDICAL SECIETY

A one day refresher course on October 13 arranged by Dr. J. J. Stanton, Chief Inspector of Licensed Nursing Homes in the province was opened by Dr. Stanton with an address on "Nursing Homes in Nova Scotia: Past and Present." The afternoon session was conducted by Dr. Arthur Shears, Medical Director of the Rehabilitation Centre in Halifax, assisted by his staff. It is the first course offered by the Government in this field and has been welcomed with enthusiasm by the Association of Licensed Nursing Home Operators of the province.

Dr. Frank F. P. Malcolm, Dartmouth, was awarded a long service pin for 15 years service to the Halifax County Hospital at a special reception sponsored by the Board of Management of the Hospital for 16 members of the staff.

Dr. David McCurdy, administrator of consultation services for the Department of Health for Nova Scotia says that the province's Emergency Health Service, (the province wide plan that would become activated in the event of natural or war-born emergencies), is "enviable at the present time." All Halifax hospitals have an emergency plan as do all the major ones in the province. At least 80% of the province's general hospitals have been cooperative in their participation in emergency planning measures.

Lunenburg Queens Medical Society

Dr. A. L. Cunningham has discontinued practice in New Germany and has accepted a position with the Nova Scotia Workmen's Compensation Board. **Dr. E. K. Woodroofe** has recently been elected president of the Lunenburg-Queens Medical Society.

All his confrères throughout Nova Scotia join in sympathy with Dr. and Mrs. Howard Creighton of Lunenburg, in the sudden death in a car accident on September 30 of their only son Graham, who was in his second year of medicine at Dalhousie.

Valley Medical Society

Dr. S. U. Anand and **Dr. Saroj Anand** and their young daughter, Getanjali have arrived from England, are guests of Dr. and Mrs. Jodh K. Sangi and have opened a practice in Kentville. Dr. Anand specialises in general surgery and his wife as a general practitioner specializing in anaesthesia. She attended the course held recently in Halifax on Paediatric Anaesthesia.

Dr. and Mrs. Donald Seaman and family have left Guysborough. He is opening a practice of General medicine in Kentville.

Western Medical Society

Congratulations to **Dr. and Mrs. S. W. Williamson,** Yarmouth who celebrated their 60th wedding anniversary on September 13.

University

Dr. C. B. Stewart, Dean of the Medical School, at the invitation of Health Minister, the **Hon. Judy LaMarsh** was a member of the five-man team to represent Canada at the Commonwealth Conference on Medical Education to be held in Edinburgh during October. Topic, "Supply of Physicians in Commonwealth Countries". Dr. Stewart also attended the Lister Centenary Conference in Glasgow during the last week of September, as Dalhousie's representative (Dalhousians do not forget that their first Dean of Medicine was **Dr. John Stewart,** one of Lister's favorite housemen).

Dr. John Stewart was also officer commanding of the **No. 7 Dalhousie Stationary Hospital Unit** which held its 50th Anniversary meeting in Halifax during the last of August with about 60 people coming from all parts of the United States and Canada to bear tribute to what was virtually, the first medical corps of the Canadian army in the First World War. A tea was held at the home of Mrs. J. MacG. Stewart, nephew of Dr. John Stewart, and an anniversary dinner at the Dresden Arms when Mr. Roy MacNutt, presently practising law in the States gave a history of the unit, which went overseas in January 1916. Following the war many of the doctors returned to practise in Halifax, of which **Dr. C. W. Holland** is one, and several of the nursing sisters went on staff in the Victoria General and Camp Hill Hospitals.

Dr. Samuel York, a graduate of U.N.B. and native of New Brunswick, holder of a Dalhousie Medical degree and of Certification in internal medicine from the Royal College of Physicians and Surgeons of Canada has been appointed to the University Staff as lecturer in the department of Medicine.

Dr. J. A. R. Tibbles, M.B., M.R.C.P. has joined the staff of the Halifax Children's Hospital and Dalhousie as a paediatric Neurologist.

Dr. D. M. J. Quastel has been appointed assistant professor of physiology and biophysics. He is a native of Wales and holds both the Ph.D. and M.D. Degrees from McGill with postgraduate work in Sweden and Australia. He has done research on the nerve control of muscle contraction.

Three Pathologists and their families, coming from such diverse places as Buda-pesth, Latvia and London have recently joined the Department of Pathology. **Dr. N. A. Kerenyi,** a graduate of the Medical University of Buda-Pesth, came to Halifax after the Hungarian revolution of 1956, from Buda-Pesth, where he had been first assistant professor of Pathology. He has now been appointed associate professor and head of the division of anatomical pathology for the provincial laboratory.

Dr. O. J. Lucis, a native of Latvia and graduate of McGill joins the department as assistant professor of pathology. His research will continue in the field of basic and applied biochemistry of the sex hormones. His wife expects to complete her work for a Ph.D. from McGill in steroid biochemistry.

A medical graduate of University College London, **Dr. A. J. Lewis,** received his training in pathology at the Royal Army Medical College where he later joined the teaching staff, holds a diploma in tropical medicine and hygiene as well as in pathology. He has specialized in neuro pathology and the pathology of muscle and joins the staff as assistant professor of pathology and as neuropathologist for the pathology institute.

A $1,500 bursary has been awarded to **Dr. W. P. Warren,** now of Toronto, but a native of Halifax and a graduate of Dalhousie with a Bachelor and Master's Degree in Science and his medical degree in 1961. This bursary is awarded by the Canadian Friends of the Postgraduate Medical School of London, England. Dr. Warren will study in the Department of Immunology of the Brompton Hospital for Diseases of the Chest in London, England, a school associated with the Postgraduate Medical School. He then returns to continue to be a member of the staff of Toronto Medical School.

Dr. Edward Grantmyre has been appointed assistant professor of Radiology on the teaching staff of the Department of Medicine of Dalhousie. He is a Cape Breton man, at present on the staff of the Children's Hospital and this year President of the Nova Scotia Association of Radiologists.

Report of Meetings

A meeting of the **Surgical Section** Medical Society of Nova Scotia was held on September 17th, 1965. There were twenty-nine members registered. The program consisted of a clinical session, held in the auditorium of the Victoria General Hospital. There were a total of twelve papers presented.

The business meeting was held at 4 p.m. under the Chairmanship of Dr. E. F. Ross. The officers elected for the coming year were Dr. A. L. Murphy, Chairman; Dr. Arnold Noble, Vice-Chairman; Dr. Charles Graham, Secretary-Treasurer. Other members of the Executive are Drs. E. F. Ross; James Vibert and Allan Sinclair.

A very nice reception and dinner was held during the evening at the Royal Nova Scotia Yacht Squadron which was attended by the members and their wives.

Births

To **Dr. and Mrs. Ian Drysdale,** (née Janet Owen), a son, at the Toronto Western Hospital, on September 29, 1965.

To **Dr. and Mrs. Ernest Johnson,** (née Margaret MacMillan), a daughter, Susan Michelle, at the Halifax Infirmary, Halifax, on October 1, 1965. A first granddaughter for **Dr. and Mrs. Ducan MacMillan.**

To **Dr. and Mrs. J. A. MacPhail,** (née Irene Hatt), a daughter, at St. Rita's Hospital, Sydney, N. S. on September 30, 1965.

To **Dr. and Mrs. Daniel Mehta,** a son, Michael, at the Halifax Infirmary, Halifax, on July 7, 1965.

To **Dr. and Mrs. W. G. Moores,** (née Mary Ann Mitchell), a son at the Grace Maternity Hospital, Halifax, on September 22, 1965.

Obituaries

A former student at Acadia and Dalhousie Universities, **Dr. Travis S. Dougan,** 56, died suddenly at his office in Sussex, N. B. during the latter part of September. A few days before his death he and his wife the former Miss Margaret Coffey of Harvey, N. B. observed their 25th wedding anniversary. Dr. Dougan was born in Harvey, the son of the late Dr. and Mrs. B. Hayes Dougan and took his pre-medical studies at Acadia and his medical course at McGill and Dalhousie University, graduating from the latter in 1938. He practised at Sussex for 24 years. He was a Past President of the New Brunswick and King's Co., N. B. Medical Societies and a member of the College of General Practice of Canada. He is survived by his wife and his brother, Dr. Alfred A Dougan, Lennoxville, P.Q.

Dr. Arthur Frederick Miller, M.D., C.M., F.R.C.P., F.C.C.P., LL.D. died on October 6 at his home in Kentville following a brief illness. He was 88 years of age. He graduated from Dalhousie in Medicine in 1904 and became associated with the late Dr. E. L. Trudeau, medical director of the Adirondack Cottage Sanatorium in New York State, coming from there to become head of the Sanatorium at Kentville, N. S. in 1909. There he remained until 1947, tirelessly educating the public to fight Tuberculosis and winning many honors including an honorary LL.D. from Dalhousie in 1944. The new teaching unit at the Kentville Sanatorium was opened in 1959 and named Miller Hall. To his wife and other relatives we extend our sympathy. An appreciation will be found on page 278.

The **Dr. Perry S. Cochrane Memorial Window** was dedicated on Sunday, October 17 in St. Andrew's United Church, Wolfville. Dr. J. Douglas Archibald of Scotburn was speaker at the service which was conducted by the pastor, the Rev. Robert Mills. Dr. Cochrane, widely known Wolfville physician was active in church and community work for many years, and the window is made possible through friends at home and abroad. □

HAVE YOU MOVED?

Since it takes three weeks to change our mailing lists, members are advised to give us early notice of any change of address. This will ensure continuity of their subscription to **The Bulletin** and enable their Medical Society to give them the best possible service.

As notification of **C.M.A.J.** is not automatic, members are also requested to notify C.M.A. direct. Their address is:

150 St. George Street, Toronto 5, Ontario

Diabetic Meal Planning

The booklet "Meal Planning for Diabetics in Canada" has recently been revised. This booklet has been prepared by the Canadian Diabetic Association in co-operation with the Canadian Dietetic Association, and has proven very useful to physicians with diabetic patients.

At the present time, a number of diabetic "diets" are being distributed by various firms selling diabetic foods and sugar substitutes. Some of the information given is impractical and too costly for use by patients in Nova Scotia. As well, many of these "diets" are based on American exchange lists rather than our Canadian ones.

"Meal Planning for Diabetics in Canada" is a reliable, clearly-printed booklet with illustrations of the sizes of servings, lists of exchanges for various foods, and with a page in the book where the patient's own diet can be filled out.

This booklet is available from:

Secretary
Halifax Branch
Canadian Diabetic Association
5916 Pine Hill Drive
Halifax, Nova Scotia

In quantities up to 50, the cost is 35 cents per copy; in lots of 50 or over, the cost is 25 cents per copy. The Branch states that for an individual patient, physicians may receive a copy free of charge.

BOOK REVIEW

Pathology for the Physician William Boyd 7th edition 1965. Lea and Febiger. Published by MacMillan Company of Canada Limited. 1004 pages, price $20.00.

The physician who tries to keep up-to-date by reading articles in medical journals is always faced with a problem. There are so many articles on so many different topics that it is difficult to separate the wheat from the chaff. For this reason the journals must be supplemented by reviews, year books, monographs, and text books.

Dr. William Boyd's textbook "Pathology for the Physician" has been written specifically for this purpose. The 7th edition must be one of the most clearly written and up-to-date textbooks of pathology available. A surprisingly wide range of recent knowledge is reviewed and kept in correct perspective. For example there are descriptions of electron microscope studies of the kidney in health and disease, the functions of the thymus and pineal are described, and there is an outline of Sir John Eccles work on synaptic transmission. Some aspects of general and surgical pathology are quite properly excluded. For this reason "Pathology for the Physician" is not suitable by itself as an undergraduate textbook of pathology. However it is highly recommended for use by graduate physicians who wish to catch up with the advances in pathology which have taken place in the last few years.

C.P.H.
