VOL. XL No 7

JULY 1961

The NOVA SCOTIA MEDICAL BULLETIN

A GUEST EDITORIAL

THE FAILURE TO CLOT

Peculiar alterations in that the blood may fail to form firm, durable clot have been occasionally noticed in at least four important clinical situations, three of which are associated with pregnancy.

They are:
1. Abruptio placentae.
2. Amnionic fluid embolism.
3. Retention of a dead fetus in utero.
4. Profound hemorrhage—surgical or accidental.

What ever the mechanism, the net effect in these cases, is to produce a deficit in the blood fibrinogen. Insufficient fibrin will form and clotting fails to stem the flow of blood, leading very often to the patient's death. The syndrome is still one of theories, but recent investigations by Pritchard and Wright,[1] following the work of many others, may bring the problem nearer a solution. If the mechanism whereby the blood is defibrinated may soon be known, the problem of clot lysis is much more obscure. The nature of the enzymes, their site of, and stimulus for, production are only guessed at.

Oddly enough there seems to be no way of predicting which case, in the four situations listed above, will develop hypofibringenemia. It behooves those of us who see these cases to be alert to this possibility. Even with no laboratory facilities, one can make a very reliable diagnosis by taping a test-tube of the patient's blood to the bedstead, turning it every five minutes until clotting occurs. After another ten minutes observe the clot for retraction and test its strength by shaking the tube. Where a defect is observed, (the clot fails to form in five to ten minutes and does not retract well or breaks up easily) a deficit of fibrinogen or an excess of fibrinolytic substances can be assumed.

It is vital to anticipate the requirements by using whole blood transfusion to keep pace with loss. The amount of blood required may seem staggering in the severe case. Obviously this should be started long before a clotting defect has occurred in the bleeding case.

(*Continued on Page 193*)

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Where a clotting defect develops human fibrinogen should be given, in amounts of 2 grams (in distilled water) until clotting occurs. If labor commences where a dead fetus has been retained some time, a similar test will indicate the need for fibrinogen.

A word of caution remains. The virus of homologous serum jaundice may be carried by even the excellent preparations of fibrinogen available in this province through the Red Cross Society's Blood Transfusion Service. Deaths have occurred from this infection in rare instances. Before using fibrinogen, one should be certain that the indication is clear and definite.

Elsewhere in this issue, (p. 198) two cases of hypofibrinogenemia are reported which occurred in the province. They are in no way unusual and illustrate exceedingly well the magnitude of the problem which faces the physician burdened with such a case. All who do obstetrics or deal with severe hemorrhage will do well to reflect again on this syndrome.

S. C. R.

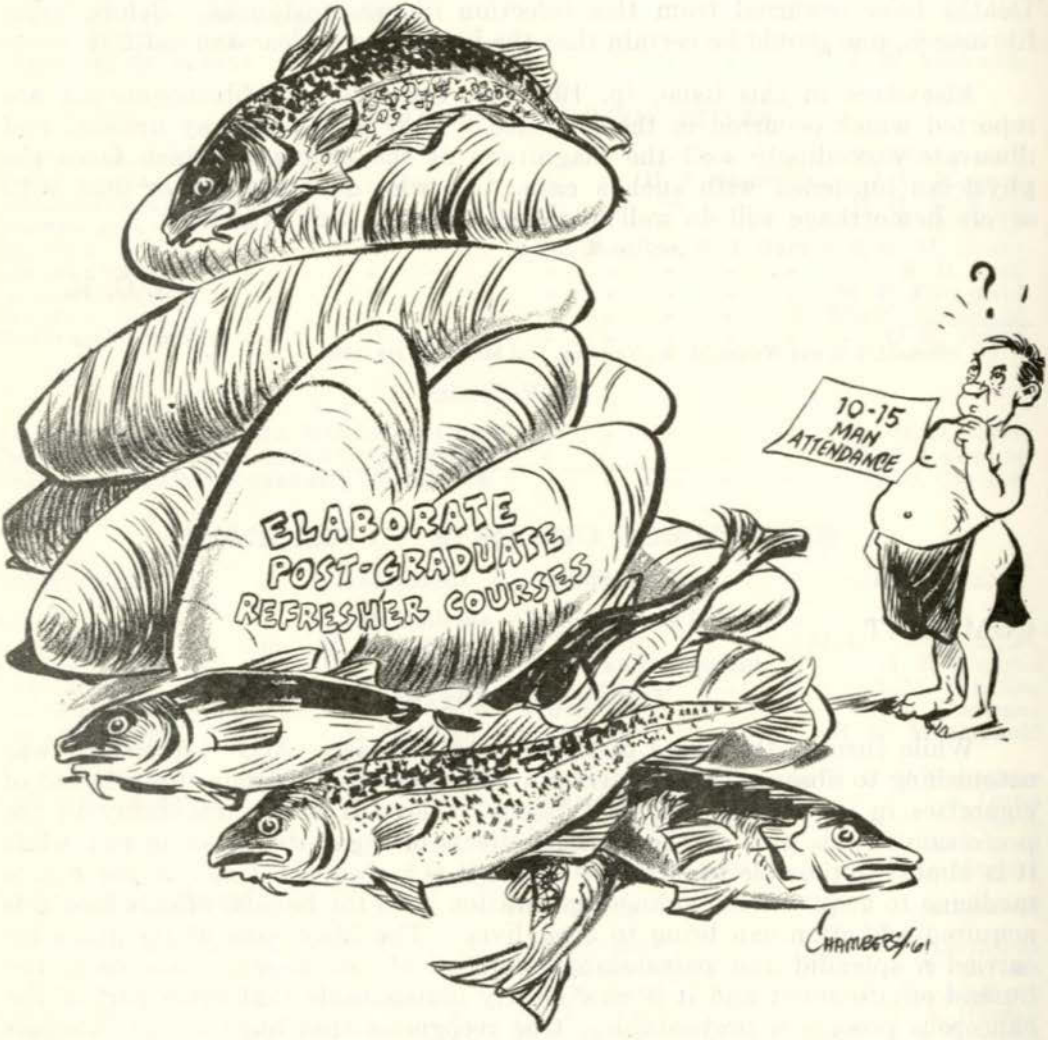
(1) Pritchard, J. S. and Wright, M. R., New Eng. J. of Med. 261: 218, 1959.

COMMENT

Inside the Citadel

While turning the pages of the medical magazine MD (Canada) it was astonishing to observe that it carried a half-page advertisement for a brand of cigarettes in its March and April issues. There is surely unanimity in the profession at this time on the harmful effects of cigarette smoking and while it is almost impossible to cure the addict it is a recognized aim of preventive medicine to acquaint the younger generation with the baneful effects that this acquired addiction can bring to their lives. The May issue of the Bulletin[1] carried a splendid and painstaking appraisal of carcinogenic hazards in the human environment and it is now clearly indisputable that some part of the cancerous process is preventable. One recognizes that huge vested interests will always seek to nullify this aim. But that they should be permitted to snipe from what are ostensibly the ramparts of medicine itself is utterly reprehensible. An editorial lapse one hopes.

1. Roe, F. J. C. (1961.) Cancer Hazards in Our Environment. N. S. Med. Bull. XI. 134.



THE LOAVES AND FISHES BIT - 1961
(IN REVERSE)

HYPO-FIBRINOGENEMIA IN PLACENTAL ABRUPTION (ABRUPTIO PLACENTAE)

A Report of Two Cases

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The pathogenesis of hypo-fibrinogenemia associated with placental abruption still remains an enigma. It has been well known since 1901 (DeLee)[1] that severe hemmorrhage associated with the failure of blood to clot is potentially a disastrous complication of pregnancy. Dieckmann (1936)[2] noted that hemorrhage complicating abrupto placenta is due to a markedly reduced level of fibrinogen. According to Wiener's[3, 4] studies, approximately 5-10 per cent of all cases of abruptio placenta and 1:5,000 pregnancies are associated with this pathological condition. There have been many theories advanced as to the pathogenesis. Some feel that the retroplacental hematoma is followed by degeneration and necrosis of the decidua as a result of which tissue juices and debris are mixed into the blood and thus introduce large doses of thromboplastin into the blood stream. According to Weiner, Hodgkinson[5] and other, the defibrination process is thus related to the circulating thromplastin and this progresses as long as myometrial, decidual or placental damage continues. Pritchard and Wright[6], impressed by the large volumes of blood clot within the uterus of patients with this syndrome and by the observations of Ashworth and Stouffer[10], that these clots appear (histologically) to be quite rich in fibrin, undertook studies to quantitate the amount of fibrin that might be lost from the circulation owing, to coagulation within the cavity of the uterus. They have elaborated an hypothesis to explain the phenomenon, as follows: "The placenta is progressively detached from the site of implantation due to unknown factors. This results in retroplacental hemorrhage followed by clotting of the blood locally. Varying amounts of both the serum (so formed) and the red cells are extruded from the clot. As a result, fibrin-rich clots are found within the uterus. The extruded serum contains no fibrinogen and reduced concentrations of prothrombin, accelerator globulin, platelets and perhaps other factors involved in the process of coagulation. It may contain some active thrombin produced by the process of coagulation or even thromboplastin from the decidua or placenta. This serum could reenter the systemic circulation through the uterine venous system. Such a mechanism of re-circulation would partially correct hypo-volemia. It would also result in a fall in the concentration of circulating fibrinogen and other clotting factors by simple dilution and perhaps by initiating some degree of intravascular coagulation as the result of any thrombin or thromboplastin in such serum. Conceivably, if the degree of hemorrhage during this time was great enough, even further lowering of the fibrinogen concentration might result through the mechanism described, in laboratory animals by Stefanini and Turpini[7]. Some augmentation of circulating fibrinolytic activity is to be anticipated in any stressful situation."

It is not known whether fibrinolysin is present at all times in the blood but it is definitely found in stress and shock. This enzyme is most likely produced by the endothelium of the capillaries and is believed to be under the control of the pituitary, adrenocortical and splenic systems. Hodgkinson,

Luzadre, and Margulis[8] in their studies of obstetrical patients reveal that fibrinolysis was present in most of the patients following delivery, even those who did not have changes ante-partum. Therefore, it almost seems to be physiological that lysis is present post-partum.

The clinical picture of cases of abruptio placenta is vaginal bleeding, abdominal pain, tenderness and rigidity, fetal distress and eventually fetal death. The patient also suffers from blood loss, and may show shock and occasionally renal and pituitary damage. Because of the extreme hazard associated with abruptio placenta, maternal mortality can be reduced if an early diagnosis ia made and proper immediate and follow-up treatment is instituted.

Caillouette, Longo and Russell[9], sensing the importance of this problem, have compiled a very interesting chart which they call a flow sheet and which is used routinely. It will be noted that many bedside procedures can be recorded on this chart. These include clot observation test, qualitative fibrinogen estimation and the determination of circulating heparin-like factor or fibrinolysin level.

Flow Sheet for Hemorrhage, Abruptio Placentae, Coagulation Defect, and Other Disorders

Clot Observation Test.—Five-milliliter sample of maternal blood, in 15-ml. test tube. Gently agitate four or five times. In our experience at this hospital the clotting mechanism is defective if there is no clot within six minutes or if a clot forms which is not solid and lyses within one hour. In other hospitals with other equipment the maximum normal time of clotting may be longer.
Size: After clot retraction a normal clot should occupy 35 to 45% of the total volume of the blood specimen.
Stability: Allow clot to stand one-half hour, then shake test tube a few times. A normal clot will withstand this procedure without breaking.

Bedside Qualitative Fibrinogen Estimation Based on Clot Observation Test.—Clotting time less than 6 minutes: fibrinogen level probably greater than 150 mg.%.
Clotting time more than 6 minutes, with poor clot: fibrinogen level probably 100 to 150 mg.%.
No clot in 30 minutes: fibrinogen level probably less than 100 mg.%.

Bedside Determination of Heparin-like Factor.—Five milliliters of unclotted maternal blood plus 5 ml. of normal control blood. Failure of the combined specimen to clot indicates the presence of a heparin-like factor.

Bedside Determination of Circulating Fibrinolysin.—Five milliliters of unclotted maternal blood plus 5 ml. of clotted normal control blood is held at room temperature. Lysis of the normal clot within one hour indicates the presence of fibrinolysin.

Fibrinogen Replacement.—Four grams of fibrinogen must be given at a rapid rate to the patient with little or no circulation fibrinogen, to elevate the serum fibrinogen above the critical level of 100 mg.%. If the clotting defect is not significantly improved within one hour, an additional 4 Gm. of fibrinogen should be given. Termination of the pregnancy should be expedited.

Treatment of Heparin-like Factor.—Dosage of protamine sulfate: 20 to 50 mg. intravenously. This should be given very slowly and should not be repeated unless persistence of the heparin-like factor can be subsequently demonstrated.

Treatment of Fibrinolysin.—Dosage of hydrocortisone: An initial intravenous dose of 200 mg. should be given, followed by 100 mg. every four hours during the first 24 hours. Following this the dose can be decreased 100 mg. per day, and the intramuscular route can be used.

Height of Fundus.—Measure with calipers in centimeters from symphysis pubis.

Once a diagnosis of coagulation defect is made by this method, proper management must be immediately instituted, namely, (1) obtain matched blood, (2) administer whole blood equivalent to the loss, (3) give fibrinogen intravenously when indicated, (4) institute antiheparin therapy and (5) expedite delivery.

DIAGNOSIS _____ BLOOD GROUP _____

GRAVA _____ Para _____ AB _____ SB _____ Weeks Gestation _____ Date _____

Time am-pm	1st Hour				2nd Hour				3rd Hour			
	15 min.	30 min.	45 min.	60 min.	15 min.	30 min.	45 min.	60 min.	15 min.	30 min.	45 min.	60 min.
Blood Pressure												
Pulse												
Respirations Fetal Heart Tones												
*Height of Fundus												
Uterine Contractions												
Frequency												
Duration												
Intensity												
Cervix Dilation												
Effacement												
Membranes												
Station												
*Clotting Time												
Size												
Stability												
*Fibrinogen, Qualitative												
Quantitative												
*Heparin-like Factor												
*Fibrinolysin												
Est. Blood Loss												
Hemoglobin												
Hematocrit												
Blood Volume												
Intravenous Fluids												
–Whole Blood												
* –Fibrinogen												
–Plasma												
–Other												
Pitocin												
*Protamine Sulphate												
*Acth												
*Cortisone												
Urine Output												
Blood on Hand												
Fibrinogen on Hand												

Surname First Name Middle Name Age Race Hospital Number Ward

Flow sheet for hemorrhage, abruptio placentae, coagulation defect, and other disorders.

Case Reports

Case No. 1: Mrs. M. C. Twenty-four years old. Gravida 5. Para. 5. No previous history of toxemia, previous deliveries normal. First seen October 27, 1957, in thirty-eighth week of gestation. Physical examination revealed excessive p.v. bleeding and tender, painful, contracted uterus. No foetal heart sounds or foetal movements were detected.

Diagnosis: 1) Abruptio placenta with severe uterine hemorrhage.
2) Dead Foetus.
3) Shock due to hemorrhage.

Caesarean Section was performed, after four units of blood were given, under spinal anaesthesia. Immediately after the delivery of a dead male child a large hematoma was found behind the placenta. At the time of the closure of the uterus it was noted that the patient bled readily from all the tissues. At this time blood was taken for clotting and determination of fibrinogen level. Laboratory report revealed total absence of fibrinogen and clotting time was over thirty minutes.

Treatment: The patient received seven blood transufsions, plasma, and fresh blood since fibronogen was not available at this time. Ten hours later fibrinogen was received by 'plane from Halifax and 3.3 gm. of fibrinogen were given intravenously, following which clotting time returned to 4-5 minutes.

Transfusions were continued for twenty-four hours, Levophed drip for six hours, and blood pressure maintained at 110/60 with pulse of 110. Post-operatively there was anuria for the first forty-eight hours when the patient started to excrete urine in amounts of 30 - 50 c.c. a day. The urine was dark red, containing albumin and tubular casts and hemolysed red blood cells. Finally, the patient died thirteen days later.

Autopsy revealed enlarged kidneys typical of lower nephron nephrosis. (Gross. Enlarged or normal in size, cortex pale, swollen deep red pyramids (hemoglobin pigment accumulation). Microscopic. Pigment casts in lower portion of nephron necrosis and degeneration of tubular epithelium, necrosis of basement membrane, interstitial edema and inflammation), the heart was moderately enlarged, the lungs were congested and filled with oedematous fluid. The vessels contained no fibrin.

The final anatomic diagnosis was:

1) Myo-hemoglobinuric nephrosis
2) Uremia
3) Congestive heart failure
4) Pulmonary oedema

This case shows the importance of an early diagnosis and also the necessity of having fibrinogen on hand. The delay of ten hours with renal hypoxia was sufficient to cause tubular necrosis and renal failure.

Case No. 2: 34 years old, Gravida 2, abortus 1. Patient was admitted to hospital on May 3, 1959, at 7:00 p.m., in the thirty-fourth week of pregnancy. She had received prenatal care and was toxic with blood pressure of 160/90, 2 + albuminuria, a few granular casts and 1 + edema. This patient presented a serious problem because of a solitary polycystic kidney, having had a left nephrectomy ten years ago, (at operation a horse-shoe kidney was found on the other side). Four weeks previously, she had an episode of fairly profuse bleeding losing about one quart of blood and was admitted to hospital

and treated until the bleeding stopped. Placentagram revealed no placenta praevia and she was discharged one week later in satisfactory condition. (The toxemia had improved on therapy.)

A few hours before the present admission, the patient developed severe suprapubic pain and p.v. bleeding which gradually increased. Examination revealed marked uterine tone and excessive bleeding. No foetal heart sounds could be heard and the diagnosis of abruption placenta was made. Despite the hypertension during the pregnancy the blood pressure now fell to 120/80. 500 c.c. of Dextran and whole blood were started, but her condition did not improve and a low caesarean section was performed. Complete separation of the placenta was found. It was also noted that bleeding occurred from all areas, including the skin. A tentative diagnosis of a-fibrinogenemia was made and this was confirmed by laboratory findings which were as follows: Hemoglobin 5 gms., P.C.V. 17%, M.C.H.C. 29%. No fibrinogen was found in the blood, no clot formation at sixty seconds, and coagulation time of over ten minutes. Additional laboratory reports stated: "Bleeding time prolonged, platelets decreased temporarily. Capillary resistance diminished. Clot retraction prolonged. Factor VII, factor V in vitro disappears. Prothrombin in vitro disappears".

Immediate treatment of 3,000 mg. of human fibrinogen in 250 c.c. of distilled water was given intravenously. Following this, venous blood coagulated in four minutes. At this time the patient was in extreme shock due to excessive loss of blood, the blood pressure having dropped to 80/50. However, with more blood the blood pressure stabilized to 100/60. It was noted that the bladder contained no urine. The post-operative diagnosis was as follows:

1) Shock due to loss of blood and the operation.
2) Acute renal failure.

Post-operative Course and Treatment: This patient also developed signs of uremia. The dominating features were oliguria, weakness, hiccoughs, nausea, vomiting, drowsiness and eventually, elevation of blood pressure.

Immediately following surgery three bottles of whole blood were administered. Benadryl 50 mg. q 4 h., was given to prevent blood reaction and to neutralize any histaminic substance which might be present in the circulation. The hemoglobin rose to 5.9 gms. The treatment of the patient was directed towards correcting the following:

1) Elevated N.P.N.
2) Oliguria
3) Hyperpotassemia
4) Hypocalcaemia
5) Acidosis
6) Anamia

1) N.P.N.—The patient did not receive any oral feeding up to the sixth day. By this means we were able to keep the N.P.N. as low as 70 mg.%. Uremia was delayed by giving 100 gms. of glucose per day in 50 per cent solution (by polyethylene tube placed in the inferior vena cava) with plain insulin 25 units, which has a maximum protein sparing effect and enables the body to utilize its own fat stores. This was preceded by 500 units of Heparin to prevent thrombosis.

2) Oliguria—The urinary output on the first post-operative day was 0, second day 189 c.c., third day 945 c.c., fourth day 1400 c.c. and the fifth day 3080 c.c. The principle followed in administering fluids was 1000 c.c., in addition to the urinary output daily.

3) Hyperpotassemia—Hyperpotassemia is the most dangerous feature in lower nephron nephrosis causing bradycardia and ventricular standstill. To prevent this, the restriction of food intake, and the administration of glucose and insulin, in order to encourage migration of potassium from the extracellular fluids into the cells was carried out. We also administered sodium-loaded ionic exchange resin by means of daily enemas, and were able to keep the potassium down to 7.4 M.eq./1. Electrocardiograph studies did not reveal any toxic effect from this level of potassium.
4) Hypocalcamia—Repeated studies indicate that in uremia there is a definite hypocalcamia present, associated with acidosis. These findings are explained as follows: Due to retention of phosphate and sulphates and the failure of the diseased kidney to form ammonium, a compensatory reduction in ionized calcium occurs, producing hyperexcitability of the nervous system. In the absence of calcium the beats become smaller and the heart stops in diastole as it does in the presence of an excess of potassium (Mines, Baetjer, MacDonald)[9]. In our cases we found a definite hypocalcemia as low as 4.2 M.eq./1. which was corrected daily by giving calcium gluconate.
5) Acidosis—The retention of waste products soon results in acidosis which in our case was only slight and was corrected by administration of 500 c.c. 1/6 M. lactate.
6) Anemia—In all cases of abruptio, as in this case, there is always a large amount of loss of blood. Every effort should be made to correct the anemia to aid regeneration of the tubules. Blood transfusions should be used, either whole blood or packed cells. In addition we used 100 microgram vitamin B_{12} and cobaltous chloride 250 mg. in divided doses daily. On the eighth post-operative day the hemoglobin was 7 gms. The fibrinogen was normal, the patient started to take food by mouth. Sutures were removed on the ninth day and the patient left the hospital well, on the tenth post-operative day.

This second patient, like the first case, was admitted with premature separation of the placenta. She had toxemia, which was aggravated by her chronic nephritis and one polycystic horseshoe kidney. By prompt diagnosis of afibrinogenemia and correct treatment the patient was carried through successfully.

Summary

It is inviting disaster to make any attempt to deliver these patients before a complete evaluation is made of the blood picture. Death may occur from lower nephron nephrosis. It is imperative to treat the patient conservatively at first with whole blood transfusions plus fibrinogen. Once the blood volume has been restored and afibrinogenemia has been corrected the patient is then ready for delivery, determined by obstetrical indication—vaginally or by Caesarean Section.

The two cases herewith presented indicate that an early diagnosis is essential to the welfare of the patient. Once an early diagnosis is established, have the patient cross-matched and blood and fibrinogen made available immediately. Preparations should also be made for the evacuation of the fetus. The blood pressure of the patient should be maintained at all times so that hypoxia with its ensuing complications will be prevented.

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Mitral Commissurotomy in Pregnancy. Dogliotti, A. M., et al. J. Thor. & Card. Surg. 39:663, (May) 1960.

This paper establishes some criteria that will help the cardiologist and the obstetrician solve the important problem of indications for mitral commissurotomy during pregnancy. They are,—

(1) The knowledge of the cardiac status during previous pregnancies is an indirect aid in the prognosis of the disease during the present situation; a history of previous pregnancies without any sign of cardiac or circulatory failure may orient the physician toward medical rather than surgical treatment. (2) A progressive deterioration of cardiac condition before or during pregnancy, or both, is a definite indication for prompt surgical treatment. (3) The sudden occurrence of an attack of pulmonary edema is a clear-cut indication. In one patient in this report, an emergency operation was performed at the third month during an acute attack of pulmonary edema which promptly ceased at the moment the mitral orifice was dilated.

(4) In cases of combined valvular lesions the problem of surgical treatment is always very difficult *even in the absence* of a pregnancy. If aortic stenosis is associated with mitral stenosis, a combined mitral and aortic valvotomy can be advised and successfully performed. If, on the contrary, a severe aortic or mitral insufficiency is associated, the chances of successful medical treatment of an already failing heart are very small. It is also extremely difficult to evaluate the possibilities of continuing a normal pregnancy and of accomplishing a normal delivery, without danger to the mother.

The writers feel that it is a good policy to perform the operation even in the mild case. The aim of cardiac surgery must be, in fact, not only to cure the established symptoms but also to prevent the occurrence of future manifestations which can appear in the last half of pregnancy, particularly during the delivery. It can conclusively be stated that commissurotomy may be advised and performed without irrational doubts or fears in every case of pure mitral stenosis, the best time being the first months of gestation.

Mitral stenosis is not an indication for a therapeutic abortion.

S.J.S.

PREOPERATIVE ASSESSMENT

HECTOR MacKINNON, M.D.

Fredericton, N. B.

The question of preoperative assessment used to be phrased by the surgeon asking the family physician, or the anaesthetist, "Will this patient stand an anaesthetic?" The implication was, that if the patient could stand the anaesthetic, he could easily tolerate anything the surgeon might choose to do. Nowadays the question might be phrased, "Who is going to do what, to whom, and when?" The stress of operation can be magnified by a careless or clumsy surgeon; or the procedure may have to be modified depending on the patient's tolerance; and the timing of the surgical attack may have to be precise.

There has been some discussion also as to whether the preoperative assessment should be done by a general practitioner, an anaesthetist, or an internist. I consider this of less moment than the general awareness of the examiner. In addition to the general appraisal, one may be asked to put the patient in better condition to withstand surgery, and to give some prognosis as to possible postoperative complications.

The size of the preoperative survey depends on the urgency of the operation. Assessment is nonexistent in such surgical emergencies as acute respiratory obstruction, exsanguinating hemorrhage, and cardiac arrest.

The next category includes conditions in which early operation is necessary, but a few hours may be spared for control of body physiology. It is essential to treat shock, hemorrhage, dehydration, acidosis, and congestive failure, before surgery is started. It is a mistake to think that expansion of blood volume and correction of acidosis can be accomplished concurrently with the operation. This can be fatal, especially in children, in whom even slight acidosis is poorly tolerated.

In the urgent case the patient may be too sick to give much of a history, but extremely useful information may be obtained by asking how the patient reacted to an operation before. He may recall that he had a "clot" the week after his operation, and his convalescence was prolonged. Or he may state that he bled unexpectedly and had to be transfused. In the days when one surgeon had the same patient for repeated operations such knowledge was present in both parties; but in this age of transient patients the question is pertinent. As an illustration of a bleeding abnormality here is the case of a middle-aged female who was diagnosed in the spring of 1960 as having malabsorption syndrome due to pancreatic fibrosis. In August she presented to another doctor with hematuria. This was investigated and the intravenous pyelogram showed filling defects in the right renal pelvis. She was being prepared for a nephrectomy and a consultant was asked to see her. Her prothrombin time showed 39 seconds test time with a control of 14. Obviously her bleeding was due to a deficiency of Vitamin K, and her hematuria disappeared with parenteral Vitamin K_1.

Another question of increasing importance is "What drugs have you been taking?" The list may include digitalis, anticonvulsants, hormones, neostigmine, Vitamin D, tranquillizers, blood pills, anticoagulants, antibiotics. The steroid hormones pose a particular problem as they need to be given in extra dosage to compensate for the stress of operation, as the patient's own adrenal cortex will be unable to do so. Reserpine and the promazines may inhibit the physiological response to stress for some days. Electrolyte imbalance due to the thiazides may require an equal period of time for correction.

The physical examination is the more important in urgent cases as history may be incomplete. It is important to remember no patient is too sick to examine. Apart from the signs of the condition requiring surgery there may be evidence of chronic disease elsewhere in the body, or signs that other systems have been secondarily affected. As the circulatory system is the one that bears much of the immediate stress of operation it is the one to be scrutinized most carefully for signs of congestive heart failure. If there is enlargement of the heart and basal rales rapid digitalization should be initiated. Optimal digitalization is difficult to achieve even when there is plenty of time. It is impossible to digitalize a heart satisfactorily in a few hours, and it has been shown that stress may reveal latent digitalis poisoning. It is better to err on the side of "too little". Induction of anaesthesia may cause increasing degrees of heart-block, ventricular arrhythmias, or cardiac arrest. The most unpredictable patient in this respect is the patient "digitalized" just the day before operation. Diuretics preoperatively to secure "dry" weight may cause side effects of electrolyte imbalance. Demerol and atropine may cause postural hypotension. People who are apt to die suddenly in waking life are the same ones who may expire in the advent of anaesthetic difficulty. These are patients with complete heart block and Stokes-Adams attacks: patients prone to develop arrhythmias: and those with severe angina or recent myocardial infarction. A preoperative electrocardiogram is a very useful test; it will practically always reveal a recent myocardial infarct; usually gives evidence of ventricular hypertrophy and digitalis effect; will show the precise nature of arrhythmias; and may provide a clue to electrolyte imbalance.

In the third category, purely elective procedures, a full study is indicated to assess various diseases and conditions which may influence the response of the organism to surgery and to provide preoperative correction of such if possible; and to predict what complications postoperatively need to be watched for.

In taking the history the same points will be emphasized as noted above for the urgent case but in addition there will be a full functional inquiry of every body system and thereafter a full physical examination.

Under the heading of general condition might be mentioned cases in which there may be chronic hypovolemia. In such conditions as malnutrition; chronic wasting diseases such as tuberculosis, rheumatoid arthritis, malabsorption syndrome, gastrointestinal shunts and fistulas, carcinoma of the gastrointestinal tract, enteritis and colitis; Addison's disease; and recent operations; there exists a diminished blood volume. Such cases are prone to develop shock more easily than the normal under any situation of stress; to expand their blood volume promptly requires the transfusion of blood proteins; a rough guide is fifty milliliters of whole blood for each pound the patient is under ideal weight.

In taking the history an estimate can be made of the patient's personality and of the way this is likely to react. Everyone has some anxiety about undergoing surgery; in some predisposed individuals this may be so intense as to upset the psychic defenses and provoke a post-operative psychosis. It would be useful to be able to predict this in advance, but I know of no way other than a history of such an episode previously. One should be aware of the possible development post-operatively of the sensory deprivation syndrome; this may easily occur in people undergoing cataract operations especially when they are also deaf; anyone with a latent claustrophobia may react to a postoperative situation in a similar fashion.

At this time one can form an opinion about the more subtle signs of early senility such as loquacity, overemotionialism, failing memory, loss of spontaneity and initiative, slowness of response, and fondness for circumstantial detail. These signs may be due to cerebral arteriosclerosis and prompt one to inquire for other symptoms suggestive of organic brain disease, especially the syndrome of insufficiency of the carotid and basilar artery systems. The history of a previous stroke is easy; it may be more difficult to find out about transient vertigo, dysphagia, aphasia, blindness, or sudden change in personality. Patients with arterial insufficiency of the brain may have a severe hemiplegia if the blood pressure drops too low, as from a spinal anaesthetic or heavy sedation. It is useful in such cases, as in all cases with hypertension, to measure the sleeping blood pressure, or the blood pressure on first awakening, when it may be at its lowest; one may then know how low it may safely go, and caution the anaesthetist to try and avoid lower levels that may lead to cerebral ischemia and infarction.

Assessment of the nervous system will include survey for the presence of multiple sclerosis (such cases are better not given a spinal anaesthetic); central nervous system syphilis, with all the problems that this may entail; epilepsy, with the application of careful control of seizures, which sometimes lapses when interest is directed to the surgical condition; and porphyria, which so easily escapes our vigilance until postoperatively, when paralysis of both voluntary and involuntary nervous systems may occur, along with hypertensive crises, personality changes, convulsions, and the telltale urinary changes. The patient with myasthenia gravis does not usually present as a surgical problem, and by the time anything surgical comes up the patient knows all about their disease.

Assessment of the cardiovascular system is so obvious as to need no emphasis. Coronary heart disease, hypertensive heart disease, rheumatic heart disease—these are rarely missed. Cor pulmonale may be overlooked but the pulmonary symptoms of the primary disease will not be. The acute carditis that occurs in many infectious diseases needs to be thought of more often. Electrocardiographic changes occur in fifteen to twenty percent of the respiratory virus infections, and in poliomyelitis the pathological changes may be as high as ninety percent. A recent bout of influenza is an indication for delaying elective surgery even though the temperature has returned to normal. This is another instance in which the preoperative electrocardiogram is helpful. This may also reveal signs of uncommon causes of heart disease which might otherwise be overlooked, e.g., beriberi, which may occur in chronic alcoholics. Cardiac function may also be jeopardized by extra cardiac factors. There are such obvious ones as endocrine abnormalities, especially thyrotoxicosis or myxedema. I think also of electrolyte imbalance, which may be caused in various ways. For example a low serum potassium will make the patient more sensitive to digitalis, will cause changes in the electrocardiogram, and will affect the function not only of the cardiac muscle but also the gastrointestinal tract and the central nervous system. A low serum potassium may occur in chronic renal disease, aldosteronism, prolonged treatment of peptic ulcer with alkalis, prolonged use of laxatives, and prolonged use of diuretics.

Hypercalcemia may act in a similar way, and may occur in people with Paget's disease who are suddenly immobilized. Acidosis is especially dangerous in children. It is also dangerous in complete heart block. Diuretics given to get a dry preoperative weight may result in electrolyte imbalance.

The peripheral circulation needs to be inspected. Varicose veins are a hazard in that postoperative thrombosis and embolism may occur. The arterial circulation needs also to be assessed.

Examination of the lungs these days means more than going over the chest with a stethoscope. Acute and chronic inflammatory disease will not be missed but ventilation and diffusion defects occur at times without very obvious signs of disease. Emphysema, asthma, pulmonary fibrosis, kyphoscoliosis, and pneumoconiosis may cause carbon dioxide retention and oxygen lack. Bronchiectasis may so easily lead to postoperative pneumonia. These conditions are not necessarily contraindications to surgery, but may be a source of difficulty in the postoperative period. It is not necessary to emphasize that all patients should stop smoking two or three weeks before elective surgery.

There are a few points about the gastrointestinal tract and its attendant organs that influence the operative risk. A hiatus hernia may complicate matters by causing digestive symptoms that are aggravated by the necessary recumbency, and may also be irritated by intragastric tube feeding, even to the point of ulceration and hemorrhage. Cholecystitis and pancreatitis sometimes flare up postoperatively in predisposed individuals. The prolonged use of milk and absorbable alkalis for the treatment of peptic ulcer occasionally leads to alkalosis and renal damage, which will then increase the hazard of surgical treatment.

One problem in connection with the gastrointestinal tract that is not easily resolved is the recognition of occult hepatic disease. Occasionally a patient undergoes operation when he is in the incubation period of viral hepatitis; massive necrosis of the liver has occurred in such cases postoperatively. It seems impractical to do Bromsulphthalein retention tests on all patients preoperatively.

Surgery and postoperative care impose quite a burden on the kidneys, not so much in respect of the ability to excrete nitrogenous wastes as on the regulation of body water and electrolyte balance. Retention of water occurs routinely in the postoperative state. The possibility of increased stone formation due to recumbency needs to be considered in people with gout, or hypercalcemia. In Paget's disease of bone, when the stimulus of stress is removed from the osteoblasts, uncompensated bone resorption increases hypercalcemia. A similar process occurs in osteoporosis.

Prostatic hypertrophy may be only moderately troublesome until some preoperative or postoperative medication (such as atropine or ephedrine) increases the difficulty of initiating micturition.

Malfunction of various endocrine glands increases operative hazard. The presence of buccal pigmentation and hypotension in an apathetic individual should prompt further study before submitting the patient to any stressful procedure, even diagnostic. One patient with similar findings was being investigated for diarrhoea; on return from having a barium enema the effort of getting up from the stretcher to his bed killed him. He was also probably depleted from the purging that radiologists require for doing a barium enema.

An occasional cause of concern to the anaesthetist is a sudden marked elevation of blood pressure during the anaesthetic. This is a very good clue to the presence of a pheochromocytoma. Routine repeated checks of the blood pressure by the nurses on the floor may act as a screen for such cases.

Association with tachycardia and marked postural effects may also suggest the possibility. A simple qualitative test for catecholamines in the urine would be most helpful, as there is a wide range between normal levels of catechol excretion and the level present in pheochromocytoma.

Diseases of the thyroid gland should be seen at the first inspection of the patient. Pituitary hypofunction may be occult, as these patients may not look so obviously myxedematous. The ones that may escape detection in a routine survey are cases of Sheehan's syndrome.

Mention should be made of a few blood diseases that can cause trouble. It is so easy to pour in a few bottles of blood to prepare a patient for surgery. We need to be reminded that a transfusion has a mortality comparable to an appendicectomy. One may overlook the fact that Vitamin B-12 deficiency effects other organs than the bone marrow. This is most strikingly apparent in the nervous system and the heart; and transfusion does not correct the weakness here. The mechanics of cross-matching blood may reveal some autoimmune disorders such as hemolytic syndrome, multiple myeloma and dysglobulinemias. Polycythemia is associated with an increased tendency to both hemorrhage and thrombosis. In general, bleeding disorders are best illuminated in the preoperative survey by an adequate history, including a family history. Vitamin C deficiency can often be suspected from an adequate history, and scurvy does survive even among the well-to-do, and will undoubtedly continue into the welfare state. A tourniquet test is easily done while taking the history, and will be positive in scurvy as well as in other forms of purpura.

As has been indicated by the foregoing, the statement that a patient is fit for surgery may require a considerable amount of clinical and laboratory evidence to support it. The above description is not exhaustive, but may suffice to show the size of the answer called for by the simple question, "Can this patient stand an operation?"

DIABETIC NEUROPATHY: A CONSIDERATION OF FACTORS IN ONSET. Ellenberg, M., Ann. Int. **52**: 1067, (May) 1960.

Various situations preceding the onset of neuropathy in diabetes are detailed in this paper. These are independent of the state of control of the diabetes, and hence do not support the contention that a prolonged period of poor diabetic control is essential for the development of neuropathy. The author considers that diabetic neuropathy should be regarded as an essential and integral feature of the syndrome of diabetes mellitus, rather than as a complication of the disease. Awareness of the occurrence of neuropathy as the initial clinical manifestation of diabetes, and the diagnostic application thereof, may help to solve some obscure clinical problems. The diverse character of the factors preceding the onset of diabetic neuropathy suggests that there may be several etiologic determinants. The sequence of neuropathy following stress situations after a relatively constant latent time interval suggests the presence of a toxic or metabolic factor in these cases.

S.J.S.

EXPERIMENTAL EFFECT OF CIGARETTE SMOKE ON HUMAN RESPIRATORY CILIA

JOHN J. BALLENGER, M.D.

The mechanism of the efficient housekeepers of the lower respiratory tract and the nose, the cilia, is interfered with by the smoke from a cigarette, as illustrated in the experiment described. The slackened efficiency of the cilia appears related to the cough of smokers.

The purpose of the experimental work reported in this manuscript is to demonstrate a possible mechanism whereby tar and other combustion products in cigarette smoke might be deposited in the lower respiratory tract of human beings. An impressive body of literature has accumulated to support the thesis that prolonged heavy cigarette smoking is positively related to the increased incidence of bronchogenic carcinoma.

If the combustion products in tobacco smoke contain carcinogenic tars (or arsenic), how does this substance collect in the bronchi and remain sufficiently long to induce a carcinoma? Normally, the respiratory cilia are most efficient in keeping the nose and the bronchial air tubes clean. The cilia of the lower respiratory tract are in constant motion, day and night, sweeping the overlying blanket of mucus toward the upper end of the esophagus. In the nose, the action of the cilia moves the mucous blanket posteriorly to the pharynx.

Failure of the cilia to beat efficiently brings about a stasis of the blanket of mucus and permits injurious material contained in the mucous blanket to remain in contact with the bronchial or nasal surfaces. In health the continuous beat of the cilia is the principal mechanism whereby the posterior two thirds of the nose and the entire lower respiratory tract keep themselves clean.

Measuring Ciliary Activity

The question arose whether or not there was some substance in cigarette smoke that caused a stasis of the ciliary cleansing mechanism. Experimental setups designed to measure the effects of various substances on ciliary activity have always lacked an accurate criterion to measure this activity. The ciliated cells must constantly be bathed in physiologic wetness. Dryness rapidly injures the cilia and destroys the accuracy of measurement. Whatever method is used must accurately provide for humidity control. An attempt to measure ciliary activity in the isolated epithelial strip and in vivo was greatly hampered by this factor.

In the method used by me, minute bits of epithelium taken from the trachea and bronchi of humans are placed in a clot (equal parts of chick embryo extract and chicken plasma) which is adherent to a cover slip. When properly made, the cilia can be seen through the microscope to be beating furiously and will continue beating for two weeks or longer with no further attention. At any time the cover slip and clot may be transferred to a perfusion chamber; the experimental solution may be perfused, and the effect on the cilia observed and recorded on moving-picture film.

Sometimes immediately and sometimes some hours later, aggregates of ciliated cells can be seen though the microscope to have become detached from the main explant to form an irregular rounded mass with the cilia outward. The furiously beating cilia cause the separate aggregate of cells to rotate 6 to 30 times per minute. The rotation serves as a convenient quantitative criterion of ciliary activity.

The New England Journal of Medicine, October 27, 1960.

Cigarettes Mechanically Smoked

The clot containing the rotating groups of cells is first bathed in the basic salt solution, and the speed of rotation of the particular explant selected for experimentation is observed and recorded on moving-picture film exposed at 16 frames per second. A "baseline" rotation speed is thus obtained. Next, perfusion is carried out with the test solution, and the effect on the speed of rotation observed and recorded on moving-picture film exposed at 16 frames per second. A quantitative comparison can thus readily be made between the baseline observations and those after perfusion with the test solution.

The test solution for these experiments was prepared as follows. Two "regular"-sized cigarettes were mechanically "smoked" by intermittent drawing of room air through the cigarettes at a rate of 1 liter per minute. Approximately one and a half to two minutes were required to "smoke" each cigarette. The smoke was allowed to escape under the surface of 100 ml. of the basic salt solution. A sintered glass filter was used to insure good contact of the smoke and the solution. Glasswool filters were inserted in two locations to prevent gross particulate tobacco from reaching the basic salt solution. In successive experiments cigarettes, both with and without "filters," were used.

The basic salt solution changed from colorless to slightly amber. In the solutions tested, the pH dropped from approximately 7.76 to 7.38. All solutions used were at room temperature.

Perfusion with the basic salt solution alone caused no appreciable change in the rotation of the aggregates of ciliated cells. If left undisturbed, rotation would have lasted for 36 to 48 hours.

Perfusion with the "smoked" basic salt solution stopped rotation within five to 28 minutes in 12 consecutive experiments. In some cases if the exposure to the "smoked" basic salt solution was terminated before the toxic effect was marked, return to the baseline speed of rotation could be produced by reperfusion with salt solution alone.

Effect of Tar

It is known that tar from cigarette smoke painted on the skin of a mouse for a third to a half the lifetime of the animal can induce skin carcinoma. It seems reasonable to assume that a similar development could occur on the bronchial mucosa of man if the tar (or other carcinogens) are in contact with the same part of the mucosa for a sufficiently long time. The present experiments demonstrate how the human ciliary mechanism fails in vitro when exposed to smoke in solution from as few as two cigarettes. The failure of the ciliary mechanism was irreversible if great care was not taken very soon to remove the "smoked" basic salt solution. It is suggested that this is one of the mechanisms whereby tars may collect in the lower respiratory tract and come into contact with the mucosa.

If the person concerned is a heavy and persistent smoker, the tars may be assumed to stay in contact with the bronchial mucosa for longer periods. Further experimental work is currently going on to determine if combustion products of petroleum, factory gaseous wastes, and so forth, have similarly deleterious effects on the cilia.

It seems likely that the decreased efficiency of the ciliary mechanism caused by smoke plays a part in the productive cough noted by smokers. If the cilia do not keep the airway clean, the blanket of mucus containing foreign material collects and eventually initiates the cough reflex.

THE DANISH TUBERCULOSIS INDEX

An intensive study of the epidemiology of tuberculosis was undertaken in Denmark by the National Health Service of Denmark and the World Health Organization. A follow-up four years after a mass campaign of tuberculin testing, X-ray, and BCG vaccination showed that all persons with suspicious X-ray lesions and young people with large tuberculin reactions should be followed systematically; others could be ignored.

Tuberculosis eradication programs of the future must depend heavily on the establishment of risk rates in various definable population groups. It is only through a concentration of all resources for screening and supervision of those people most likely to develop tuberculosis that waste motion can be avoided and rapid progress made.

Giant steps in this direction have already been taken in Denmark by means of a mass screening campaign, followed by four years of careful observation. The report of this experience which appeared under the title, "Epidemiological Basis of Tuberculosis Eradication in Denmark," in the Bulletin of the World Health Organization, Vol. 21, No. 1, 1959, is of immediate practical importance to everyone involved in tuberculosis control. The authors were E. Groth-Petersen, Jorgen Knudsen, and Erik Wilbek. The whole study was carried out under an administrative organization called the Danish Tuberculosis Index. It was done as a cooperative undertaking of the National Health Service of Denmark and the WHO Tuberculosis Research Office.

Country-Wide Study Begun

During the period from February, 1950 to December, 1952, tuberculin testing, X-raying, and BCG vaccinating teams covered the entire country with the exception of Copenhagen, the island of Bornholm and a few small communities where campaigns had been carried out previously. The only population group not included was school children aged 7-14 years who were being tuberculin tested and vaccinated in the schools. Over one million persons were examined. A sputum specimen or a gastric lavage was obtained whenever there were suspicious findings on the X-ray.

Among the 795,000 adults examined in the mass campaign, 503 previously unknown cases of active pulmonary tuberculosis were found—a rate of one case per 1,500 examined. Expressed as age specific rates per 100,000 population, there was a range from 36 in men aged 15-24 to 94 in women aged 25-34. The report provides the greatest detail on the cases found during the initial campaign, but the findings in the four-year period of follow-up are striking, indeed, and furnish valuable documentation on risk rates not previously available for any population group in the world.

Four Year Follow-Up

Among the 744,261 individuals judged healthy, so far as tuberculosis is concerned, at the start of the follow-up period, 878 new cases of tuberculosis developed, 742 of which were pulmonary. This is an average annual incidence of 25 per 100,000. Although the rates were somewhat higher for women and for the age group 15-34, the differences are too small to be of much use in defining risk groups. It is only when tuberculin test and X-ray results are considered together that big differences in risk rates become evident.

F. M. Feldmann, M.D., National Tuberculosis Bulletin No. 8, Vol. 46, September, 1960.

In a group of 320,000 unvaccinated tuberculin reactors, the average annual case rates per 100,000 in the age group 15-24, according to size of tuberculin reaction, were as follows: 6-11 millimeters, 24.5; 12-17 mm., 56.4; 18-23 mm., 87.8; and 24+ mm., 72.6. In older persons, the differences by size of reaction were less striking.

X-ray findings at the start of the follow-up period were classified as normal (90 per cent), healed lesions (7 per cent), and suspicious (3 per cent). The corresponding new average annual case rates were 27, 51 and 370, respectively, for all ages. However, the rate was 1,022 for those in the age group 15-24 who had suspicious shadows. The highest case rate—roughly 2,000 per 100,000 persons per year—was in a subgroup of 1,200 persons whose roentgenographic findings were interpreted as definite lesions, probably of tuberculosis origin.

Although the case rates in the vaccinated groups were low, 23 per cent of the new cases arose among them. Since there was no unvaccinated control group selected at random, the effect of vaccination could not be measured.

Conclusions

The report concludes: "Certainly the enormous numbers of routine repetitive X-ray examinations of adults can be drastically reduced and the case-finding nevertheless intensified. Persons in the older age-groups with normal findings on a single photofluorogram, even though they have positive tuberculin reactions, need not be called back for examination year after year. They can be left in peace. But persons of any age with suspicious X-ray lesions and young people with large tuberculin reactions should be followed systematically. These high-risk groups comprise such a small preentage of the total population that continuous and close supervision is both practicable and profitable.

Abstracted by National Tuberculosis Association.
Printed through co-operation Nova Scotia Tuberculosis Association.

Prognostic Factors and Results of Treatment in Pyogenic Pulmonary Abscess. Anderson, M.D., and McDonald, K. E., J. Thor. and Card. Surg. 39:573, (May) 1960.

In this paper, 90 consecutive cases of primary pyogenic lung abscess are reported.

Thirty-two of the 90 patients achieved a good result with medical therapy alone, 21 died while on medical therapy, and 38 patients required operation. Of the 21 fatalities among patients on medical therapy, death was thought due primarily to the abscess in 11 cases; the other 10 patients succumbed to associated diseases.

Certain factors strongly indicated the likelihood of failure of cure by medical treatment alone. In decreasing order of value, these factors were: (1) Size of cavity 6 centimeters or more in diameter, (2) a history indicating onset of disease earlier than 8 weeks prior to admission, (3) staphylococcal infection, and (4) location of abscess in lower lobe of either lung.

S.J.S.

EXPERIMENTS AND OBSERVATIONS ON THE GASTRIC JUICE AND THE PHYSIOLOGY OF DIGESTION. Beaumont, W., M.D.

This book is a facsimile of the original edition of 1833, together with a biographical essay 'A Pioneer American Physiologist' by Sir Wm. Osler. It is an excellent paper bound edition of 280 pages by Dover Publications of N.Y. Price $1.50.

The first thirty-odd pages are devoted to the Osler essay on Beaumont, delivered before the St. Louis Medical Society in 1902. It gives an intimate picture of Beaumont's training and army experience and of the circumstances leading to his opportunity for experiment on Alexis St. Martin. It discloses the difficultues he encountered in retaining the healed but fistulous St. Martin in his employ long enough to carry on his many observations and experiments. An appendix contains interesting letters and side-lights on the lives of both Dr. Beaumont and St. Martin.

The book proper is divided into sections, each of which deals with different aspects of digestion. The first section observes and records the time required for the digestion of various foods. The others consider in sequence: Hunger and Thirst, Satiety and Satisfaction; Mastication, Deglutition and Ensalivation; Digestion by Gastric Juice; Appearance of the Villous Coat and motions of the Stomach; Chymification and the uses of Bile and Pancreatic Juice.

The last fifty pages report the many experiments and observations on the digestive process in detail, together with tables of digestion time of various foods.

This book is printed in excellent type on good paper and should be read or re-read by every medical student and physician.

J.W.R.

SOURCE OF MEDICAL HISTORY—compiled with notes by Logan Glendening, M.D. Dover Publications Inc., New York. 685 pages. $2.75.

This book was originally put together by Dr. Glendening and published in 1942. Dr. Glendening died in 1945. The book has not been re-issued since then, but has now been brought out in a paperback edition by Dover. The book has not been altered or abridged in any way. The format of the book is acceptable and the print clear. The paper is of very high standard and the binding considerably better than that usually found in paperback reprints. The title of the book is indicative of its contents. It sets forth a record of the most important bygone events in medical history chronologically arranged with comments on the times and social customs. As is inevitable in any source books, there are omissions and Dr. Glendening regrets that lack of space and reasons of expediency compelled him to weed out some relevant material. Each chapter begins with a short account of the historical character and works which are to be presented. Most readers would probably find the early chapters rather disjointed and not easy to read. This, however, is not a fault of the book, but is inherent in the reference type of material which is included. The general thread of medical history is coherent and well maintained throughout the entire book. While one would not expect the average reader to work his way through from the first to the last chapter, there is,

nevertheless, something to appeal to every taste. The pathologist can look up the early authentic references to work in his field, as can any other specialist. None would find it necessary to read the whole book.

It is not likely that this Dover edition will have a wide circulation. There are two reasons for this; the first is that the information contained in the SOURCE BOOK OF MEDICAL HISTORY has been available for years, particularly in the same book which was originally published in 1942. The second is that fewer and fewer members of the medical profession are taking serious interest in the classical works of the men upon whose shoulders we all stand. For any one who wants to wet their feet in the fascinating waters of medical history, this book is recommended.

F.D.K.

PRINCIPLES OF PUBLIC HEALTH ADMINISTRATION. Hanlon, J. J. The C. V. Mosby Co., St. Louis, 714 pp. $10.50.

To the public health novice this would be a most formidable piece of reading—to the dedicated public health worker it contains a mine of information—but like all mining, the way to the pay ore is difficult.

There is no doubt that Dr. Hanlon knows his subject but despite this his style of writing is such that the book can be taken only in small doses. His handling of the Introduction is masterful—if written in a popular vein it could do much to tell the public and the Public Health Worker just what is meant by Public Health and the reasons behind the movement.

As a reference book it will be useful but the great need at the moment is for a publication which will be read by the profession and the public—in these days of mass expenditure on treatment, someone should show how smaller expenditures on prevention could prevent a great deal of useless expenditure on treatment.

J.S.R.

ERRATUM

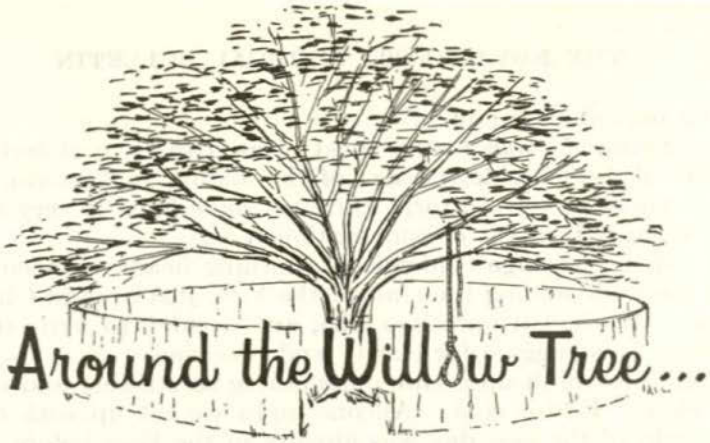
CANCER HAZARDS IN OUR ENVIRONMENT

F. J. C. Roe, D.M. (Oxon).

Page 135 of our May (1961) issue, line 9-13 should read:

'The argument that a cause-and-effect relationship had not been proved and that it is *just as likely* that men who are peculiar, in wanting to work in the nickel industry, are also peculiar in having a high expectation of developing nose and lung cancer, must be regarded as ridiculous if common sense is to play any part in this general field.'

It will be noted that the phrase 'then the statistician concerned' (line 12) has been deleted.



AROUND THE WILLOW TREE

The old order changeth yielding place to new
And GODDEN fulfills himself in many ways.

Yes, dear reader. The little Hobby Horse that has been looking over the fence at us for the past three years has left for greener and richer pastures. For Jack-have-pen-will-travel Godden has left us and with his going there remains a gap in our affections and a vast hiatus on the Editorial Board. No longer can we transport you by his magic carpetry to the banks on the swirling date-palmed Tigris, or spend a vicarious moment tasting with him the forbidden fruits of Las Vegas or sampling the golden sorcery of Champagne. We wish him abundant hay and a pleasant ride through his new fields. And, need we add, he takes with him our appreciation and thanks for all the work he has so freely given for our entertainment and instruction.

It is, then, with a somewhat tempered pleasure, that we offer you our new monthly column entitled "Around the Willow Tree".

We have chosen the willow tree in this age of molecular uncertainty as a symbol of timeless pastoral tranquility; a tree that bends with every storm but never breaks; a fibre beautiful yet strong.

Our *special* willow tree as you know is situated where the five roads meet and where our City Corner gloomily reflects upon the frail mortalness of man and the perverseness of the horses at Sackville Downs last Saturday evening.

The associations of man with the willow have always been pleasant. There is the limpid stream, dimpled perhaps by the rising trout or dace; the gentle rustle of green and silver as the warm, pollen-scented breeze upturns her leaves to the sun; the unruffled decorum of English cricket, for our willow fathers most of her springy bats; and not least our charming new secretary, the willowy blonde who has recently adorned with her presence the aseptic austerity of the office of The Nova Scotia Medical Society. Need we say more?

Our willow tree is no more than a sapling just now, scarcely taller than the fire hydrant beside which it was planted last year. In fact, like this column, it is new and just sprouting its first buds. But soon it will grow and thicken; its graceful fingers will point in silent acclamation to the sky and its branches spread wide with its own special splendour. So, too, we hope our column will grow and blossom and receive its due acclaim.

It may be recalled that the old willow tree in sterner times did service as a gallows for public hangings. The Editorial Board has decided to revive this custom and freely invites any member or aspiring author to hang himself in this column with whatever yarn he may choose to spin. It is in fact *your* column and we invite your contributions whether torpid or tepid, anal or banal, humid or tumid—anything short of "Lady Fourletterly's Lover". And we would like to fill it from every corner of the province.

Returning once more to Jack.

Wiseman, Stammers and I were most pleased to have at last a Bluenose with a blue pencil right beside the seat of the mighty in Toronto. In March we sent in our paper to the Editor. He must have liked it very much for he still has it. What a brilliant consummation it is.

We had noted a strange epidemic of morning headache accompanied by anorexia and lassitude among the staff of the V.G. just around Christmas and the New Year. We had it ourselves, too, and decided to write it up for the C.M.A.J. How we struggled for clarity and precision.

First we had to think up a title—something so striking and original that it would knock the Editor cold. All one night we sat up with our problem and the first light of the new day was already in the East before Wiseman at last came up with it. Triumphantly we banged it out on the typewriter.

"Preliminary Report and Investigation
on
Epidemic Matutinal Cephalgia of the Festive Season."

Wiseman is really bright at these sort of things which is why we put his name first. Then how to start? *You* know how it is done of course. Stammers gave us the lead here. "Currently our attention has been engaged with a series of severely debilitating disorders associated, we think, with some noxium first described by Ginn and Mixe (1) as far back as 1694, and later corroborated by the brilliant eighteenth century Chinese Clinicians Muche and Too Muche (2) and more recently by Neate (3) the Scotch pharmacologist. While not, of course, accepting their hypotheses. . . . . ." By the time we had finished we had one hundred and twenty-seven references. Not that we had read any of them of course. Nor did we suppose that anybody would ever look a single one of them up. But none could deny their decorative value and the air of weighty scholarship that it gave to our contribution to scientific medicine.

We were due to phone Jack this morning abour our paper. He told us before he left he would let us know how it was getting on.

The girl got us through pretty quickly.

"Wiseman et al here, Jack" I said.

"How's it going Al" he said pleasantly.

"No, no. You don't get me. Its about our paper. Wiseman, Stammers et al . . . . ."

"Ah. Yes. I've got it right here."

"When is it going in Jack?" I asked.

"It's not. We're sending it back to you."

"One or two spelling mistakes or something?"

"No. We're mailing it back to you."

"Some re-writing? A few more references perhaps?"

"REJECTED. CAN'T YOU HEAR."

"Rejected? You mean to tell us you don't want it?"

"That's right."

"What may be your reasons" I demanded haughtily.

"Well, take this bit here, for instance—

"We therefore conclude that the responsible noxium is contained in the several diluent fluids commonly added to those spiritous liquors so freely used to add to the gaiety of the festive season. We respectfully suggest that

the triad of symptoms, morning headache, anorexia and a powerful disinclination to work be denominated for convenience "The overhang."

"You don't like that Jack" I asked incredulously.

"We don't believe it."

How obtuse can Editors get.

"Well listen Jack. What d'you suggest."

"Try Reader's Digest."

"Reader's Digest? But this is serious . . . . . .Jack are you there. Hey operator you've cut us off."

There was an interval of buzzing and distant clicking sounds when the girl spoke again.

"He's through with you I guess, Mister?"

"He's hung up?"

"You said it. Bye now."

And she left us in the air too.

So speak no longer with Godden. NOW SPEAK WITH GOD.

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2. Muche and Too Muche. "The cup that runneth over. (1746).
3. Neate, B. Distillery Archives. Vol. 400. p. 1160. (Glasgow 1948).

E.H.E.

NOTICES

PRACTICE FOR SALE

Goldboro, Guysborough Co., N. S.

Unopposed "deep" rural practice 46 miles from Antigonish (paved road). House available for sale. Equipment and office, 100 yards from house, available for rent. Full drug stock for sale at valuation.

Enquire by phone, Goldboro - 11, Dr. Rolf Sers.

FOR RENT

Heated 4 rooms - immediate occupancy. 181 Quinpool Road, Halifax. Over McNeil's Drug Store. Suitable for physician or dentist· Phone 423-5446.

PERSONAL INTEREST NOTES

Impressions and Comments on the 108th Annual Meeting of The Medical Society of Nova Scotia Held at Keltic Lodge, June 12 - 14, 1961

An unusually large number of doctors, many accompanied by their wives, travelled to Keltic Lodge for the 1961 annual meeting. Though some arrived through the rain and fog of Saturday evening, most came through the brilliant sunshine to arrive on Sunday. The executive, it should be pointed out, had been there for several days, to prepare for the business meetings of the Society.

It is difficulty to be sure just why these doctors attended the convention. An unscientific sampling would indicate that there were two main reasons, often intertwined. Most looked forward to an opportunity to get away from the pressures and demands of their regular work, and to enjoy the congenial company of their fellows. Other stated that they attended primarily out of a sense of duty to The Medical Society and to their profession generally, and because they were concerned particularly this year about the thoughts and statements of the people and the political parties which would soon have a direct effect on the practice of medicine, regardless of the thoughts and wishes of the doctors. The more naive members present had the vague feeling that something in the manner of a medical manifesto might come out of the meeting through brilliant oratory, clear thinking and unanimity of opinion, the membership as a whole might indicate their thinking on the subjects of prepaid medical plans, government assistance or management of health care plans, etc. The other more cynical and perhaps more practical members of the organization felt that such delicate and complex matters could not be dealt with effectively at an open general meeting because of the many factors involved, and that our very capable executive had been working on the problem and would continue to work and assist the special research committee to draw up a satisfactory plan for the people and the medical profession of Nova Scotia. A few individuals expressed the opinion outside the meetings that general policy should and would be laid down by the C.M.A. and the provincial divisions would simply add the necessary local color.

At the end of the convention the naive members realized that very few, if any, basic policies regarding our attitude to socialized medicine had been stated by the general meetings and that we would have to depend on the function of the executive committee throughout the next year with occasional referral of certain problems to the branch society meetings to obtain the support or condemnation of the general membership. The more practical members of the Society probably left the convention feeling that their beliefs of the function of general business meetings had been confirmed.

(Editor's Note: Since the actual details of the business meetings will be presented elsewhere they will not be discussed here).

The social functions generally were very satisfactory. The lobster party on the beach was a very popular event and the supply of lobsters held up well. A Tuesday luncheon meeting was addressed by Mr. John Delaney, Board Member of the United Mine Workers, whose subject "Labor Looks at Medicine", suggested a government controlled form of socialized medicine to include drugs, dentistry, preventative treatment, and total rehabilitation. The annual banquet on Tuesday evening, was highlighted by a speech of the outgoing president in which he expressed the ideals of medical practice. Hon. R. A. Donahoe, Minister of Health of Nova Scotia, made a very pleasant speech,

in which he made no contentious statements, avoided all danger areas, and discussed in an entertaining way, the number of doctors who had taken part in politics in Nova Scotia, and I believe he also mentioned that doctors as a group do not know much about politics, although they do get involved occasionally in medical politics. The ladies program seemed quite satisfactory to those concerned, although there was a complaint that no ladies' golf tournament had been arranged. (Editor's Note: may we express our thanks to Dr. G. J. H. Colwell for these notes.)

Antigonish-Guysborough Society

May 27, 1961—Annual meeting of the society followed by a social evening to which the practicing physicians of Inverness and Richmond Counties were invited.

May 13, 1961—Dr. T. W. Gorman addressed the graduating class and alumnae of St. Martha's Hospital School of Nursing at their annual banquet.

Cape Breton Medical Society

Dr. C. Burke has located in Sydney and will specialize in anaesthesia.

Dr. and Mrs. B. V. Earle are now engaged in the private practice of psychiatry in Sydney.

Drs. H. R. Corbett and Malcolm Shannon attended the Annual Meeting of the Nova Scotia Association of Radiologists at the Lord Nelson Hotel, Halifax on May 13, 1961.

Western Nova Scotia Medical Society

Dr. and Mrs. L. M. Morton have returned to Yarmouth after spending five months in Florida. Dr. Morton claims to have had no part in the Cuban invasion.

University

Dr. W. P. Warren has been granted a Canadian Life Insurance Medical Fellowship for research on "Carbohydrate Metabolism in Obesity."

Dr. D. M. MacRae has been promoted to the rank of Professor and appointed head of the Departments of Ophthalmology and Otolaryngology.

Dr. John Yeatman and Mrs. Yeatman of Adelaide, Australia visited Halifax in the course of a six months tour of North America and Britain. Dr. Yeatman is Nuffield Travelling Fellow in General Practice. He is studying continuing Medical Education Programmes for the General Practitioner, Psychiatric Training Programmes for General Practitioners, and the approach of the Medical School toward the training of General Practitioners.

Dr. Yeatman visited with the Dean of the Faculty of Medicine and studied the operation of the Departments of Psychiatry and the Post-Graduate Division. He met with the Chairman of the C.M.A. Committee on Professional Education, with the Special Research Committee of the Medical Society of Nova Scotia, and with local officers of The College of General Practice.

Dr. and Mrs. Yeatman were entertained by The Halifax Chapter of the College of General Practice, and spent the weekend with friends in Port Joli before flying to Boston.

Births

To Dr. and Mrs. Kurt Aterman, Halifax, a son, Grace Maternity Hospital, Halifax on June 10, 1961.

To Dr. and Mrs. J. Randolph Buchanan (nee Jacqueline Singleton) a daughter, Constance Michele, in Victoria, B.C. on June 13, 1961.

To Dr. and Mrs. W. L. M. King (Loudelle MacLellan) a daughter, Hope Madeline, at the Digby General Hospital on June 9, 1961.

To Dr. and Mrs. Bruce Morton, a son, David Bruce, at the Grace Maternity Hospital on June 4, 1961.

Marriages

Dr. Ernest Basil Johnson, Halifax to Margaret Anne MacMillan (daughter of Dr. and Mrs. Duncan MacMillan, Sheet Harbour) in St. Peter's Church, Sheet Harbour, N. S. on June 7, 1961.

Dr. S. Clair MacLeod and Nola Clarke on June 10, 1961.

Congratulations

To Dr. and Mrs. J. E. MacDonnell, Antigonish, on the birth of their daughter, Mary Louise recently. (Ed's Note: We did not have the exact date, so it is not entered in the birth column.)

Coming Meetings

September 25-29, 1961—The annual "Week in Anaesthesia" conducted by the Department of Anaesthesia through the Post-Graduate Division, Faculty of Medicine, Dalhousie will be held in the Victoria General Hospital. Detailed programs will be mailed to all practitioners at the beginning of September. If you plan to attend, please notify the Division at an early date as the numbers to be accommodated are limited.

October 2-6, 1961—47th Annual Clinical Congress of the American College of Surgeons at Chicago, Illinois. Address inquiries to Dr. W. E. Adams, Secretary, American College of Surgeons, 20 East Erie St., Chicago 11, Illinois.

November 13-18, 1961—Canadian Heart Association and National Heart Foundation of Canada, joint annual and scientific meetings in Vancouver, B.C. Address inquiries to Dr. J. B. Armstrong, National Heart Foundation of Canada, 501 Yonge St., Toronto 5, Canada.

June 18-22, 1962—95th Annual Meeting of The Canadian Medical Association, Winnipeg, Manitoba.

October 7-13, 1962—The 4th World Congress of Cardiology will be held at the Medical Centre, Mexico City, Mexico. Address inquiries to the General Secretary: Dr. Isaac Costero, 4th World Congress of Cardiology, Institute N. De Cardiologia, Avenida Cuauhtemoc 300, Mexico 7, D. F.

Obituary

Dr. Roderick James MacDonald, Canada's grand old man of medicine, died at his home in St. Peter's Bay, P.E.I. June 4, 1961, after an illness of several weeks. He was over 100 years old, and received his medical degree from Trinity College, Toronto on April 3, 1888. He practiced in the local community some 69 years, retiring at the age of 99.

Dr. P. S. Campbell - An Appreciation

To many in this province the death of Peter Smythe Campbell indicated the passing of an era in Public Health—an era during which most important advances in the health field in this province had taken place—and "P.S." had an important place in the advance in the field of prevention—to many he was "Mr. Public Health."

A true son of Cape Breton, he never forgot or allowed others to forget this fact. Nor was he less proud of his associations with McGill University and the Montreal General where he took his training—supported largely by his own efforts and determination to be a physician.

Entering the Government service at a time of political unrest, he never allowed his own political beliefs to interfere with his medical judgment nor with what was best for the people of Nova Scotia.

In religion he was a staunch Roman Catholic—all trips to Ottawa on weekends had to be interrupted at Montreal for church attendance, plus a visit to his old haunts of student days. I well remember one of these trips when I accompanied him—due to late rising we went to the nearest church which was French speaking and all announcements were in French. For some reason three collections were taken and I can remember yet the frustrated look on his face as the collection plate was presented to him for the second and third time —he later said this was one time he wished he could understand French.

Under his leadership major advances were made in the field of prevention and public health—most of the present staff were picked and trained by him. Despite an austere countenance he was the most approachable of men and his advice was sound as the years have proved.

"P.S." was intensely interested in his family—two daughters and five sons have justified well indeed the efforts and sacrifices made on their behalf by Mrs. Campbell and "P.S.". One son and daughter in the Church and one son in Medicine carry forward two of his chief interests into the future.

"P.S." was well liked and respected by his associates, which is a recommendation for anyone—his contribution to the welfare of the people of Nova Scotia will not be forgotten.

J.S.R.

INFECTIOUS DISEASES—NOVA SCOTIA
Reported Summary for the Month of April, 1961

Diseases	NOVA SCOTIA 1961 C	NOVA SCOTIA 1961 D	NOVA SCOTIA 1960 C	NOVA SCOTIA 1960 D	CANADA 1961 C	CANADA 1960 C
Brucellosis (Undulant fever) (044)	0	0	0	0	12	8
Diarrhoea of newborn, epidemic (764)	1	0	0	0	4	4
Diphtheria (055)	0	0	0	0	3	1
Dysentery:						
(a) Amoebic (046)	0	0	0	0	0	0
(b) Bacillary (045)	0	0	0	0	179	230
(c) Unspecified (048)	72	0	0	0	119	13
Encephalitis, infectious (082.0)	0	0	0	0	0	1
Food Poisoning:						
(a) Staphylococcus intoxication (049.0)	0	0	0	0	7	0
(b) Salmonella infections (042.1)	0	0	0	0	101	0
(c) Unspecified (049.2)	0	0	0	0	0	54
Hepatitis, infectious (including serum hepatitis) (092, N998.5)	21	0	83	0	923	409
Meningitis, viral or aseptic (080.2, 082.1)						
(a) due to polio virus	0	0	0	0	2	0
(b) due to Coxsackie virus	0	0	0	0	0	0
(c) due to ECHO virus	0	0	0	0	1	0
(d) other and unspecified	0	0	0	0	11	11
Meningococcal infections (057)	1	0	0	0	19	12
Pemphigus neonatorum (impetigo of the newborn) (766)	0	0	0	0	6	0
Pertussis (Whooping Cough) (056)	0	0	11	1	398	631
Poliomyelitis, paralytic (080.0, 080.1)	0	0	0	0	9	23
Scarlet Fever & Streptococcal Sore Throat (050, 051)	86	0	111	0	1445	2559
Tuberculosis						
(a) Pulmonary (001, 002)	21	3	14	1	0	401
(b) Other and unspecified (003-019)	6	0	4	0	0	144
Typhoid and Paratyphoid Fever (040, 041)	0	0	0	0	22	41
Venereal diseases						
(a) Gonorrhoea — Ophthalmia neonatorum (033)	0	0	0	0	0	0
All other forms (030-032, 034)	9	0	33	0	1388	1356
(b) Syphilis — Acquired—primary (021.0, 021.1)	0	0	0	0	0	0
— secondary (021.2, 021.3)	0	0	0	0	0	0
— latent (028)	0	0	1	0	0	0
— tertiary — cardiovascular (023)	0	0	0	0	0	0
— „ — neurosyphilis (024, 026)	0	0	0	0	0	0
— „ — other (027)	0	0	0	0	0	0
Prenatal—congenital (020)	0	0	0	0	0	0
Other and unspecified (029)	0	0	3	1	175*	163*
(c) Chancroid (036)	0	0	0	0	0	0
(d) Granuloma inguinale (038)	0	0	0	0	0	0
(e) Lymphogranuloma venereum (037)	0	0	0	0	0	0
Rare Diseases:						
Anthrax (062)	0	0	0	0	0	0
Botulism (049.1)	0	0	0	0	1	0
Cholera (043)	0	0	0	0	0	0
Leprosy (060)	0	0	0	0	0	0
Malaria (110-117)	0	0	0	0	0	0
Plague (058)	0	0	0	0	0	0
Psittacosis & ornithosis (096.2)	0	0	0	0	1	0
Rabies in Man (094)	0	0	0	0	0	0
Relapsing fever, louse-borne (071.0)	0	0	0	0	0	0
Rickettsial infections:						
(a) Typhus, louse-borne (100)	0	0	0	0	0	0
(b) Rocky Mountain spotted fever (104 part)	0	0	0	0	0	0
(c) Q-Fever (108 part)	0	0	0	0	0	0
(d) Other & unspecified (101-108)	0	0	0	0	0	0
Smallpox (084)	0	0	0	0	0	0
Tetanus (061)	0	0	0	0	3	0
Trichinosis (128)	0	0	0	0	11	0
Tularaemia (059)	0	0	0	0	0	0
Yellow Fever (091)	0	0	0	0	0	0
N.S.U.			8			

C — Cases D — Deaths

*Not broken down

C.D.C. 2