VOL. XXVI No. 6

The Nova Scotia Medical Bulletin

OFFICIAL ORGAN OF THE MEDICAL SOCIETY OF NOVA SCOTIA
CANADIAN MEDICAL ASSOCIATION NOVA SCOTIA DIVISION.

JUNE, 1947

Published on the 20th of each month and mailed to all physicians and hospitals in Nova Scotia. Advertising forms close on the last day of the preceding month. Manuscripts should be in the hands of the editors on or before the 1st of the month. Subscription Price:—$3.00 per year.

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Contents June, 1947

*Experiences With Retropubic Prostatectomy

CLARENCE L. GOSSE, M.D.,C.M.,F.A.C.S.

THE purpose of this paper is to review a series of twenty retropubic prostatectomies and attempt to determine what place this procedure may have in the treatment of benign prostatic hypertrophy.

Shortly after Millin's paper appeared in the Lancet, December 1945, retropubic prostatectomy was first attempted in Halifax. Since that time I have done twenty such operations, and while this number is small compared with the total number of prostatectomies, I feel that some sort of a conclusion can be drawn from the results thus far obtained. One feels that transurethral resection is still the preferred method of treatment in the majority of cases. However, in large glands, particularly in younger individuals, the transurethral operation has its drawbacks and some form of enucleation is to be preferred.

As Millin points out, the usual type of suprapubic operation leaves much to be desired, because of several drawbacks: e.g.

1. High mortality.
2. Uncomfortable and often post lengthy operative course.
3. Considerable blood loss during and after operation.
4. The high incidence of post operative infection.
5. The incidence of secondary infection.

The transurethral operation is also not without some disadvantages: e.g.

1. Difficulty in acquiring proficiency.
2. Blood loss when resecting large glands, both during and after operation.
3. The frequency of persistent low grade urinary infection.
4. The risk of recurrence of obstruction due to incomplete removal —5 to 10%.

However, the big advantage of this operation is the extremely low morbidity and the slightly lower mortality.

In view of these drawbacks and disadvantages, urologists are continually ally seeking a new method or a different avenue of attack in order to find an operation which will satisfy all requirements. The following diagramatic sketch serves to show the retropubic approach to the prostate as used by Millin and followed by ourselves.

(SP) represents the symphysis pubis and (A) the arrow pointing to the retropubic space—behind the pubis but in *front* of the bladder—thru which the prostate is eneucleated. (C) is the arrow pointing to the bladder thru which the prostate is ordinarily removed suprapubically, thus showing the difference anatomically between the retropubic and the suprapubic operations.

Millin has suggested that the retropubic prostatectomy more nearly reaches this goal because it offers, as he claims, the following features:

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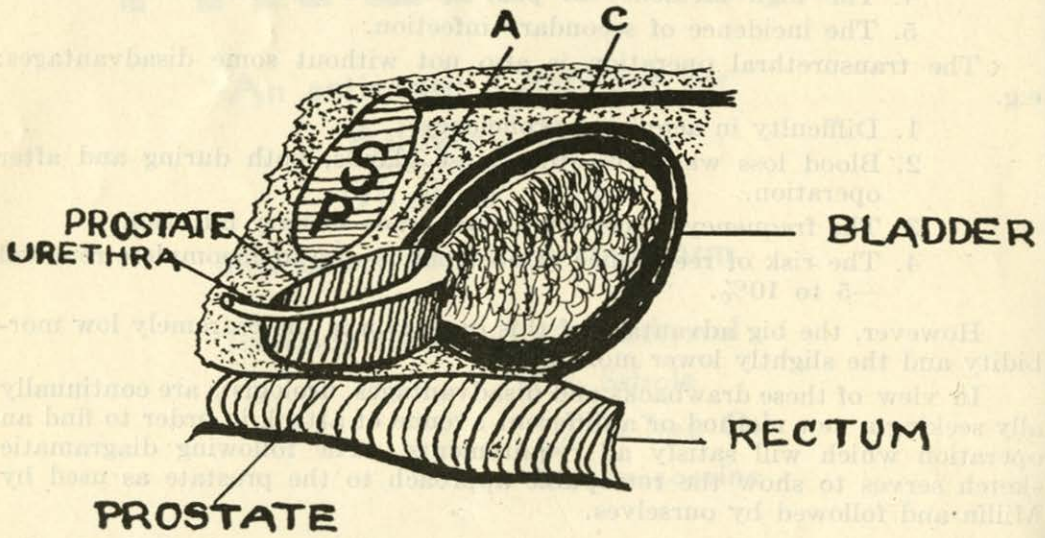
*Paper presented in part at Halifax Medical Society meeting, February 12, 1947.

1. It is an extra vesical gland removed by an extra vesical procedure avoiding supropubic bladder drainage and the risk of slow closing or persistent fistulae.
2. It is applicable to all types of glands.
3. It is a quick operation and relatively shock free.
4. It is anatomically sound—no important organs being interfered with.
5. The morbidity and mortality is low.
6. All the obstructing tissue is removed, thus obviating the possibility of recurrence.
7. The post operative care is easy for both patient and nurses.

In our experience we have been unable in all cases to agree with Millin's first point, viz. the avoiding of the slow-closing suprapubic fistula. Out of our twenty cases, fourteen closed without any suprapubic drainage, but the remaining six cases drained from twelve days to twenty-two days following operation. Three of those cases had remained closed until the fourteenth day when a small sinus appeared, and infected urine flowed freely. These took an average of a week to close. However, this leakage occurred for the most part in the early cases, and with improvement in our technique the latter cases have been much better.



While we feel that this operation is applicable to all types of glands, we do not feel that it is necessarily the best operation for certain types of glands, or for certain patients. We feel that each patient should be judged as an individual, and that the type of operation be done which is best suited to the nature of the gland and the condition of that individual.

In our hands the average operative time is about three-quarters of an hour, which is no less than for most types of prostatectomies. We agree that there is relatively little shock. In nearly all of our cases also there has

practically been no blood loss, and rarely has it been necessary to transfuse the patient during operation. The blood supply to the prostate can be seen after removal of the gland, where it it picked up under direct vision, and hemastasis secured.

The operation, while being anatomically sound—no important organs being interfered with—is technically more difficult than the transvesical type of enucleation. This is due, primarily, to the sometimes troublesome plexus of veins in the region of the bladder neck. However, with a little caution and delicacy this need not cause much concern.

Generally speaking, the morbidity has been low, but not as low as that following transurethral resection. As regards mortality, in the twenty cases there has been one death, but our series is too small to strike a fair rate. This death was early in the series, in a patient who was a poor operative risk for the retropubic procedure. According to our present standard and indications, we would not do this particular operation on that type of patient, but would prefer transurethral resection. The post operative course, from the standpoint of the patient, is relatively simple and easy; and the nursing care, using a Foley 22 F. 75 c.c. catheter for continuous irrigation, is comparatively simple.

The following table shows a breakdown of all the prostatic resections that I have done during the past year. These are shown to give an idea of mortality rate and to show the relative position of retropubic prostatectomy in the overall picture.

	Number	Mortality
Transurethral resections	75	1
Transvesical suprapubic prostatectomies		
2-stage 22		
1-stage 3		
—	25	0
Retropubic prostatectomies	20	1
Total cases	120	2 (1.7%)

One can see from this table that our operation of choice for most glands is the transurethral resection. The 2-stage suprapubic operation, which makes up most of the number of transvesical procedures, was done on cases who were admitted to hospital in poor condition, with renal insufficiency, high blood chemistry, etc. This number of retropubic operations, which amounts to one-sixth of the total done, is, perhaps, not a true picture of what we feel is its real position in operative procedures, as we have done more in the past six months than in the previous six. Nevertheless, I can safely say that in view of our present attitude towards the operation, we are not using it in more than 25%of our cases. However, if the results continue to be as uniformly good as they have been in the more recent cases, then our attitude might well be changed, whereby 50% or even 75% would be handled by the retropubic method.

Conclusion: I have reported a series of 20 retropubic prostatectomies, making up one-sixth of the 120 prostatectomies of all types carried out over approximately a one-year period. There was one death in the series done by the retropubic method, and the overall mortality rate was 1.7%.

At the present time we are doing approximately one-quarter of the cases by this method with the results becoming more and more satisfactory as experience increases.

The blood loss at operation is surprisingly small, and the bladder washings are usually clear well within the first twenty-four hours. In most cases the irrigations are discontinued after twenty-four hours but the catheter is left in for 8-10 days post-operatively. This is done to give the prostatic capsule a chance to heal completely. We have been getting these patients up on the first or second post-operative day, with excellent effect.

We feel that this operation, done on carefully selected patients properly investigated and prepared by all known modern facilities, has much to recommend it. If enthusiasm does not distort judgment and discrimination, and throw the operation into disrepute, retropubic prostatectomy might well find a useful place in the armamentarium of the urologist.

Influenzal Meningitis

A Review of the Literature with a case Report of Recovery in a 14 year old boy

H. C. Read, M. D., Resident in Medicine,
Victoria General Hospital, Halifax, N. S.

A FEW months ago an unusual case of Influenzal Meningitis in a 14 year old boy was treated, with recovery, at the Victoria General Hospital, Halifax, N. S. As it was the first case of its kind to be treated in this institution for adult patients, at least for many years, it is felt that if for no other reason, the publication of this case report is justified. In addition certain interesting features of the case itself make it worthy of presentation.

As the general impression, which is erroneous, tends to be that this disease is a rare one and, as in this disease within the past decade there have been revolutionary advances in therapy and a remarkable decline in mortality rate, a brief resume of the salient features of the disease, with special reference to treatment, is not untimely.

Bacteriology—The causative organism of influenzal meningitis was first isolated by Pfeiffer, in 1889, from fatal cases of epidemic influenza and, since for a long time it was considered to be the cause of the latter disease, it received the name "influenza bacillus" or "Hemophilus Influenzae." As it is now known that no relationship exists between these two diseases, it is unfortunate that this bacillus has retained such a misleading name, for it makes the term "Influenzal Meningitis" not only confusing but also somewhat of a misnomer. However, although it plays only a secondary role in influenza, this bacillus is still an important pathogen and may produce serious infections, especially respiratory and meningeal. Clarification of the bacteriology of this organism came with the work of Pittman (1) in 1929 when she found that the numerous strains of this bacillus were divisable into two groups which, on the basis of morphological and cultural characteristics, she termed the Smooth (S) and the Rough (R) strains. It has subsequently been shown (2,3) that, although the rough (R) strains are not infrequently seen as secondary invaders, especially in respiratory infections, the important pathogenic strains belong to the smooth (S) group and that this group can be divided antigenically or immunologically into six types, called A,B,C,D,E, and F. Pittman also demonstrated (4) that the majority of strains giving rise to meningitis are of the same serological type, namely type B. This last has been substantiated by innumerable reports; for instance, Silverthorne (5) reports that type B organisms were causative in 70 out of 71 cases investigated. Only rarely has one of the other types been implicated (6,4).

Type-specific antisera, as a consequence of these findings, were prepared and introduced as a form of therapy by Alexander in 1939, further mention of which will be made in the discussion of treatment.

Incidence—As has been intimated, this form of meningitis is generally thought to be a rare disease but this is true only in the case of adults. Statistical reports (7,8) on the frequency of different types of meningitis show it to hold from first to third place in meningitis of infancy and early childhood. Hertzog's report (9) on 377 cases of fatal meningitis shows both the frequency and severity of this disease in that it was the commonest cause of fatal meningitis in children under 3 years of age.

The age incidence is especially striking. The disease is rarely seen in infants younger than 2 months and is very uncommon during adolescence and adult life. In fact, about 85% of all cases occur between the ages of 2 months and 3 years. Fothergill and Sweet (10) report that the curve of age incidence begins to rise at the age of 2 or 3 months and reaches a peak between 6 and 9 months after which it falls gradually, levelling off after the age of three and a half years. The explanation advanced for this age-distribution is that, within this age group, the body has very low resistance against this organism. This is based on the observations by Fothergill and Wright (11) that the blood of infants up to 6 weeks of life and the blood of older children and adults possessed marked bactericidal power for meningitic strains of Hemophilus Influenzae, while the blood of infants between 2 months and 3 years seldom possessed this bactericidal property. The relative immunity in the early weeks of life is considered to be a passive maternal transfer immunity, while that in older children and adults is regarded as an active immunity presumably resulting from infections of the respiratory tract.

Route of Infection—Infection of the meninges by this bacillus seems to follow a definite sequence of events in the majority of cases. Analyses (7,9) show that, in nearly all cases, there is a history of an immediately preceding or concomitant respiratory infection. It is to be noted, however, that this respiratory infection is frequently a very minor one so that, with this exception, in the majority of cases the disease appears to start rather suddenly in a previously healthy infant. Invasion of the blood stream or bacteriemia is thought to occur in most cases. Fothergill (7) and Edmonds and Neter (12) report positive blood cultures in 70% and 78% respectively in their series of cases. Thus, the route of infection is presumed to be via the upper respiratory tract to the blood stream, followed by localization in the meninges.

Diagnosis—The diagnosis of this type of meningitis cannot be made by the clinical findings alone, since nothing in the clinical picture clearly distinguishes it from other types of purulent meningitis. The diagnosis can be made certain only by careful examination of the cerebrospinal fluid with the identification of the Hemophilus Influenzae bacillus. Nothing is to be gained even by the chemical analysis and cytology of the cerebrospinal fluid, because the latter is a purulent one showing similar changes to any purulent meningitis. Consequently, identification of the organism is essential. As indicated by the route of infection, complete investigation of a case should include nasopharyngeal and blood cultures in addition to the spinal fluid cultures. The paramount importance of early diagnosis might well be stressed and restressed. Now that effective therapeutic measures are available, the sooner they are applied, the better are the patient's chances of survival with an intact central nervous system. Unfortunately, the diagnosis of meningitis in infants is often a delayed one because of the varied, obscure and often complex clinical picture presented. Inasmuch as this type of meningitis (and also other types) is commonest in infancy and, as the signs of meningitis in this age group are so uncertain and variable, it is to be urged that lumber puncture be preformed early whenever there is any suspicion of meningitis. A bulging anterior fontanel, should be considered a prime and urgent indication for this diagnostic procedure. Although, even before the days of specific therapy, the mortality rate was reported (13) as highest in early infancy, yet since the advent of therapy, a greater disproportion has been noted (8) between the

mortality rate in early infancy (especially under 7 months of age) and that in late infancy and early childhood. All evidence indicates that the latter is largely due to late diagnosis and therefore late institution of treatment. As emphasized, it is the perception of meningeal involvement which constitutes the chief problem, so that only with improvement of our clinical acumen can improvement in results in this age-group be expected.

TABLE I

Types of Therapy in Influenzal Meningitis with their Approximate Effect on Mortality Rate

Approximate Year	Type of Therapy	Number of Cases	Mortality Rate	Reference
....	1. **Non-Specific** (General).....	78	98.0%	10
to		70	98.0%	5
1932		373	97.6%	14
		220	92.0%	13
	2. **Horse Antiserum** (with Human	201	84.6%	7
	Complement).....	50	78.0%	5
1932		18	95.0%	4
	3. **Horse Antiserum + a Sulfonamide** (usually sulfanilamide or sulfa-	20	75.0%	5
to	pyridine).....	? (a literature review)	82-54%	8
1941	4. **Sulfonamides** (usually sulfapyridine).....	10	50.0%	5
1939	5. **Rabbit Antiserum**.....	62	35.0%	15
to				
1946	6. **Rabbit Antiserum + Sulfadiazine**.....	267	29.36%	12
		60	33.3%	12
		15	33.0%	16
	7. **Streptomycin**.....	7	43.0%	17
1944		9	22.3%	18
to		10	20.0%	19
date	8. **Streptomycin** usually + **Sulfadiazine** and **Rabbit Antiserum**....	100	17.0%	20
		25	12.0%	21

Treatment—The types of treatment that have been used in this disease, with their approximate effect on the mortality rate, are represented in Table

1. As can be seen, influenzal meningitis has evolved from a disease with no specific therapy and an almost 100% mortality rate to a disease for which there are now a number of very definite therapeutic measures and from which there is now only a mortality rate in the vicinity of 15%. Variations from this, largely depend upon the promptness and adequacy of therapy.

Immune horse serum, usually combined with human serum for its complement content, was the first specific therapy attempted. This was first prepared and advocated by Ward, Fothergill and Wright (22, 23) of the Department of Bacteriology and Immunology, Harvard University, in 1931. Results from this form of therapy proved disappointing. A few years later, this therapy combined with one of the earlier sulfonamides produced somewhat better results. However, as Alexander (8) found, a review of the literature showed very inconstant results with thelatter therapy, varying mortality rates from 82% to 54% being reported.

In 1939 the use of type-specific rabbit antiserum was introduced. Alexander of the Department of Paedeatrics, Columbia University Medical School, has been its chief advocate and has made the most scientific study of this form of therapy. Results with it have been moderately good and, when combined with a sulfonamide, the results have been even better. Prior to the advent of streptomycin, this combined therapy was considered to be the treatment of choice. Although recoveries from one or the other of these two therapeutic agents have been reported (5,8,15), their relative effectiveness has never been definitely settled because any series of cases is very small in which either one has been used alone. However, Alexander (24) attempted to evaluate them and came to the conclusion that, in order for the sulfonamides to effect complete recovery, it is necessary, firstly, that the infection be a relatively mild one and secondly, that the drug be started early in the course of the infection. It was felt that, only under such conditions, there would be sufficient time for the patient's own defensive powers to produce adequate antibodies to overcome the disease. In more severe infections and in those in which there was delay in therapy, she showed that combined sulfonamide and antiserum therapy was essential and, in fact, rather clearly demonstrated that energetic combined therapy was the wisest procedure in all cases. The commercial anti-serum is called "Anti-Hemophilus Influenza Type B Serum (Rabbit)" and is supplied in vials containing 25 mg. of precipitable antibody nitrogen. Alexander (8) recommends that the dosage of antiserum be based on the sugar content of the spinal fluid (see Table 11) and that the efficacy of this be determined by the daily testing of the patient's blood serum for antibody excess, that is, by testing its ability in a dilution of 1:10 to produce capsular swelling of a suitable suspension of Hemophilus Influenzae (Quellung phenomenon). Failing this, further antiserum therapy is indicated. The intravenous route of administration is the one most favored, the total requirement being diluted in 10cc. of isotonic saline per kilogram body weight and given slowly over a period of two hours. The intramuscular route may be and is sometimes used. The intrathecal route, on the other hand, not infrequently results in undesirable reactions in the early acute stages of the disease, so that its use is advised only in chronic cases showing poor response to other medications (16).

TABLE II

Schedule of Dosage Based on Dextrose Content of Spinal Fluid (8)

Spinal Fluid Sugar Mg. Per 100 cc.	Antibody Nitrogen Indicated Mg.
less than—15	100
15 to 25	75
25 to 40	50
over 40	25

In regard to sulfonamide therapy, substantial therapeutic dosages are recommended. Of the various sulfonamides, sulfadiazine has been shown to be the most effective in this type of meningitis (25,26) and has consequently been the one of choice. More recently, the newer sulfonamide, in a small series of cases of purulent meningitides, has been shown (27) to be equal to sulfadiazine in effectiveness with the advantage that smaller and less frequent doses are required to attain comparable blood levels and comparable results. Further evaluation of this drug is required, however, before it could be acclaimed as the best.

Streptomycin is the latest addition to the medical armamentarium against this disease. Recent literature contains fairly frequent reports of its dramatic powers against the Hemophilus Influenzae. On the other hand, its fellow antibiotic, penicillin, would appear to be of no avail (28,29). The series of cases in which streptomycin alone has been used are small but the results have been good. Even better results have been attained by using streptomycin, sulfadiazine and rabbit antiserum in various combinations depending upon the features of the individual cases.

One of the most comprehensive reports to date on streptomycin therapy in this disease is that of Keefer et al (20) of the National Research Council, in which they present a series of 100 cases with a mortality rate of only 17%. Eighteen of the cases received streptomycin therapy only and made satisfactory recoveries but most of the remainder had either received previous treatment or were given concurrent therapy with penicillin, sulfonamides and antiserum in various combinations. In general, their streptomycin dosages consisted of 0.5 to 1.0 gms. (500,000 to 1,000,000 units) intramuscularly daily (in divided doses every three hours) for a period of 5 to 7 days, the total dosage being 3 to 7 gms. In addition, daily intrathecal treatments in doses of 0.025 to 0.05 gms. (25,000 to 50,000 units) were given for 7 or 8 days, the total intrathecal dosage averaging 0.35 gms. Review of the data on their fatalities suggested that the factor which is most unfavorable for streptomycin therapy is late treatment after other forms of therapy have failed. Their conclusion was that streptomycin is most effective in the treatment of Hemophilus Influenzae infection, but, that undoubtedly, some failures occur, the most probable factors in these failures being: inadaquate dosage, development of resistance ("drug-fastness") to streptomycinin vivo, change in the species of the infecting organism during treatment, and localization of infection in an area inaccessible to streptomycin.

Another comprehensive report is that by Alexander et al (21) of a series of 25 cases with a mortality rate of only 12%. All of their patients had pri-

marily received sulfadiazine with or without penicillin, but these were discontinued when streptomycin therapy was instituted. Their streptomycin dosages were similar to those of Keefer et al. In 12 cases whose infections were considered mild or moderately severe, this therapy was sufficient and they recovered promptly and completely. However, in the remainder, the clinical courses demanded institution of additional therapy in the form of sulfadiazine and type-specific rabbit antiserum.

Their deduction regarding the mechanism of "drug-fastness" is interesting. They support the theory held by most authorities that drug-fastness is dependent upon the existence in a large bacterial population of a small number of organisms with the capacity to survive in the presence of high concentrations of the drug, and that it is not dependent, as suggested by others, upon a change in their metabolism as a result of exposure to the drug. As they state, "the apparent development of resistance appears to represent a selective process which eliminates the sensitive organisms and thereby permits the resistant members to thrive." Thus they suggest that the greater the size of the bacterial population, and therefore the greater the severity of the infection, the greater will be the danger of "drug-fastness." These deductions not only emphasize the necessity of utilizing all available forms of therapy but also emphasize the necessity for larger initial, and also subsequent, dosages of streptomycin. By the latter means, it is hoped that the survival of the small "resistant" fraction in a bacterial population might be prevented and so the numerous failures in the treatment of the severer infections be eliminated. However, until these possibilities are explored, it is considered that streptomycin can not be recommended as the sole treatment of influenzal meningitis. Although it would appear to be adequate in the mild and moderate cases, the wisest procedure to date, would seem to be to treat at least all severe infections with a combination of all three of our present day agents, namely streptomycin, sulfadiazine (or possible sulfamerazine) and rabbit antiserum.

In addition, it is well to realize the possibility and dangers of superimposed infections. Cases are on record (18) in which some form of superimposed infection was the real cause of fatality or in which superimposed infection was controlled and fatality prevented only by the judicious institution of penicillin therapy. The staphlococcus aureus would appear to be the chief offender and it manifests itself most commonly in the production of respiratory or meningeal infections. It is recommended that the nasopharyngeal flora be checked repeatedly throughout the illness and that penicillin therapy be instituted as soon as this organism becomes the predominant one, even before the evidence of invasion is present.

Sequelae—Recovery without sequelae of any kind, occurs in the vast majority of cases. In former years the danger of recurrence from a localization of some of the purulent exudate and subsequent abscess formation was an ever present one, but with the newer forms of therapy this occurence is a rarity. Various cranial nerve palsies and varying degrees of mental impairment are the most common sequelae but even these are fortunately seen only in a small percentage of most series.

Silverthorne's report (5) on the 30 cases that recovered in his series (from 1917 to 1945) is interesting. Only four suffered permanent disabilities (two mentally defective, two deaf) and these did not have the advantage

of our present day "miracle" drugs. All of his cases were followed for a time varying from 4 months to 10 to 13 years. As he states, "certain patients immediately after recovery and after sterilization of their spinal fluid have appeared to have a hopeless prognosis from the standpoint of normal mentality. After observations over months and years, these patients have returned to normal mental and physical health without sequelae." This strongly suggests that, in the early stages of convalescence, sequelae do not denote a hopeless outcome but that the prognosis should be at all times a hopeful, though guarded, one for complete recovery.

Case Report

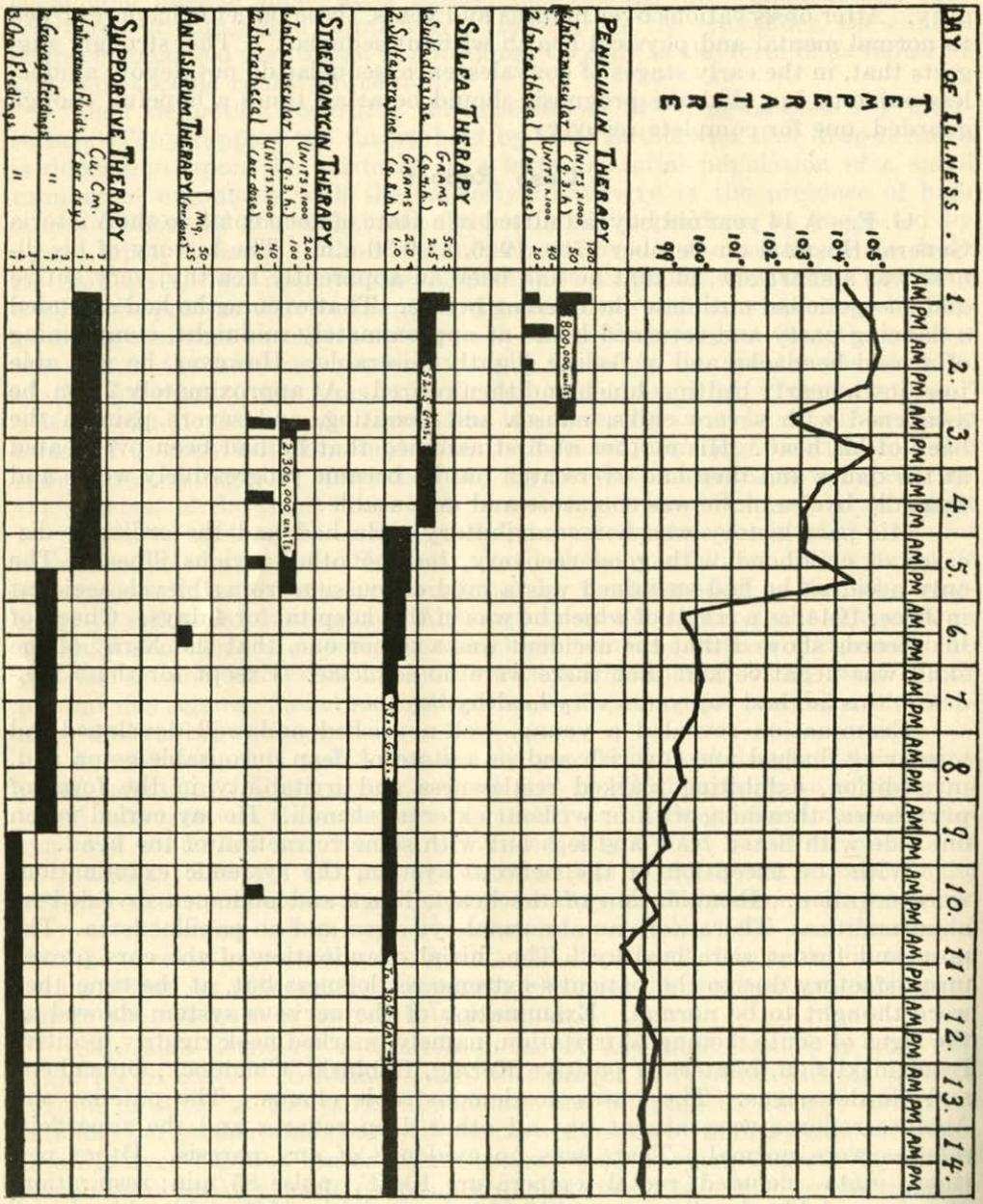
G. F.—A 14 year old boy, admitted in a state of deep coma to the Victoria General Hospital on October 27th, 1946, at 9.30 a.m. The history of his illness was a short one, in that he had been an apparently healthy, very active and energetic lad until late the evening before. That evening he had attended a dancing party and returned home at approximately midnight, complaining of a mild headache and of feeling slightly miserable. However, he was able to enjoy a hearty bedtime lunch and then retired. At approximately 2 a.m. he awakened with severe chills, nausea and vomiting, and severe pain in the back of his head. His mother at first assumed that he had been overheated at the dance and then had over-eaten but he became progressively worse and finanally by 7 a.m. he was comatose and unrousable.

His past history was non-contributory. He had had the ordinary diseases of childhood with good recovery, but no other serious illness. The only accident he had sustained was a mild concussion from a bicycle accident in June, 1944, as a result of which he was in this hospital for 4 days. Check of his records showed that the accident was a minor one, that the X-ray of the skull was negative and that there were no sequelae. Except for these incidents, this lad had enjoyed a very healthy boyhood.

Examination revealed a young, well-nourished and well developed lad appearing flushed and feverish and in a state of deep unrousable coma and, in addition, exhibiting marked restlessness and irritability in the form of purposeless thrashing with or without external stimuli. He lay curled up on one side with flexed back and legs but with some retraction of the head.

With the exception of the nervous system, the systemic examinations were negative. Examination of the heart, lungs and abdomen revealed no abnormalities. There were no abnormal eye signs and no papilloedema. The nose and throat were healthy. The initial examination of the ears proved unsatisfactory due to the patient's extreme restlessness but, at the time, they were thought to be normal. Examination of the nervous system showed all the signs of acute meningeal irritation, namely, marked neck rigidity, positive Brudzinski sign, bilaterally positive Kernig, Babinski, Chaddock, Oppenheim and Gordon signs. There was no demonstrable clonus. The patellar and Achilles reflexes were absent but all other deep reflexes and the superficial reflexes were normal. There was no evidence of any paresis. Other pertinent data included: rectal temperature 104°F.; pulse 85/min; respirations 25/min; red blood cell count 4,910,000; hemoglobin 95%; white blood cell count 28,000; blood pressure 130/90; urinalysis negative. A lumbar puncture was performed and a markedly turbid "watered-milk" fluid was obtained under a pressure of 650 m.m. of water.

Diagram 1.



A tentative diagnosis of an acute pyogenic meningitis of unknown etiology was made and therapy was instituted promptly. The treatment given this case is represented, along with the temperature chart, in Diagram 1.

Pending laboratory diagnosis of the causative organism intensive courses of penicillin and sulfadiazine were instituted. These consisted of penicillin in an initial dose of 100,000 units intramuscularly, followed by 50,000 units every 3 hours and an initial intrathecal dose of 20,000 units; and of sulfadiazine in an initial dose of 2.5 gms. every hour for 3 hours intravenously (in its intravenous form soludiazine), followed by 2.5 gms. intravenously every 4 hours.

After 14 hours the clinical picture was unchanged so the lumbar puncture was repeated. On this occasion, the fluid was found to be less turbid and now only under a pressure of 120m.m. of water but the Queckenstedt test was questionably positive. 40,000 units of penicillin were administered intrathecally at this time. During this period, considerable paraldehyde has been given intramuscularly to control the irrationality, so that examination of the ears was now much more satisfactory and revealed that the right drum was slightly injected and retracted, while the handle of the malleus and the superior margin of the drum were acutely inflammed. The light reflex, however was still present and the lustre of the drum was only slightly dulled. The left drum appeared healthy. The next morning the otolaryngologist was called in consultation as a result of which bilateral paracentesis was considered advisable and immediatley carried out. Cultures taken at that time proved sterile. During this same morning (2nd day), the lumbar puncture was repeated. Again the fluid was found to be less turbid than on previous puncture, and the pressure was only 120 m.m. of water but this time the Queckenstedt test was definitely positive, that is, there was no response on bilateral jugular vein compression. 10,000 units of penicillin were administered intrathecally on this occasion. At this stage of the illness, the general condition of the patient was poorer than on admission. He was even more restless, the head retraction was more marked and the temperature was elevated to 105.2°F. with pulse and respiratory rates elevated to 120 and 40 respectively. With this discouraging clinical picture, associated with an apparently improving spinal fluid but with a positive Queckenstedt test, one was led to the natural deduction that the case was now complicated by intracranial cerebro-spinal fluid blockage.

In the meantime, no help was forthcoming from the laboratory regarding the causative organism. No organisms were demonstrated by direct smears of the spinal fluids, and the special medium (Bordet-Gengou) on which the first cultures were made proved to be contaminated, so that no growth was obtained except that of the contaminant. Subsequent spinal fluid cultures proved completely sterile.

On the assumption that interventricular blockage existed, it was felt that the only hope for this patient was to carry out ventricular puncture, drainage and therapy via this route. Consequently, on the afternoon of the second day, cisternal and ventricular punctures were done and, unexpectedly, a slightly turbid fluid, similar to that obtained by the lumbar route that morning, was procured and it was under similar low pressure. These findings relieved the fear of interventricular blockage but further mystified the diagnosis. Fortunately, however, the mystery did not remain much longer unsolved. Direct smears of the fluid taken by ventricular puncture showed an occasional organism morphologically similar to Hemophilus Influenzae.

The urgent need for streptomycin was then apparent. Unfortunately the need arose before this drug was freely available, so a rush call was put

through to the Streptomycin Control Committee, Montreal, and through the co-operation of all concerned, streptomycin therapy was under way within 12 hours of the diagnosis. The dosages used were 200,000 units intramuscularly initially, followed by 100,000 units every 3 hours. In addition, daily intrathecal treatment was given for 3 days consisting of 40,000 units on the first two occasions and 25,000 units on the third occasion with a final 25,000 units intrathecally 5 days later. As the allotment of streptomycin received consisted of only two and one-half million units, the duration of therapy was short, being approximately only 2½ days.

However, this therapy appeared to mark the turning point in the clinical course of the case because coincident with its termination (5th to 6th day of illness) the temperature took a precipitous drop from 104.5° F. to 99.5° F. Subsequently, the fever remained low-grade and from the 10th day onward the patient was afebrile.

During the period of streptomycin therapy, it was realized that our small quota of this drug was probably inadequate to combat this severe infection so immediate steps were taken to procure some "Anti-Hemophilus Influenzae Type B serum (Rabbit.)"

Although the definite type of the infecting organism was not determined, the high probability that it was Type B justified the use of this type-specific anti-serum. As it so happened, the anti serum was not procured until the 6th day, by which time the patient's temperature and spinal fluid had shown dramatic improvement. The sugar content of the spinal fluid on that day was 62.0 mg. per 100 cc. which is within normal range and, according to Alexander's criteria, did not indicate antiserum therapy. However, as the patient's general condition was still critical, its use seemed warranted so that on that day (6th) 25 mg. of antiserum were given intravenously, diluted in 500 c.c. of physiological saline and administered slowly over a period of 2 hours.

As indicated on the treatment diagram, the sulfadiazine therapy was replaced on the fourth day by sulfamerazine therapy. The reasons for this change were first, because it was desired to try this relatively new drug with such promising reports, and second, because of the advantages it offered in regard to the smaller and less frequent dosages required. The dosages of sulfamerazine employed were 2.5 gms. intravenously every 12 hours for two doses, followed by oral doses of 1 gm. every 6 hours for 4 doses, then 1 gm. every 8 hours up to the 30th day.

Although, as previously mentioned, a marked reduction in the patient's fever took place early on the 6th day at the termination of streptomycin therapy, the general condition of the patient showed little improvement for some time. Throughout the first seven days, the mental state of the patient changed very little. He remained in the same deep coma with the same marked restlessness and moderate head retraction as on admission. On the 8th day he showed less head retraction and was less restless. On the 9th day, he lay quietly, and occasionally opened his eyes to look around with a quizzical, confused expression on his face as though trying, but without success, to attain contact with his surroundings. For the next four days he remained stupified and disorientated, only occasionally demonstrating any signs of contact with his environment and only occasionally answering a question with any evidence of comprehension. On the 14th day he began to show definite improvement, which, over the next few days, was very rapid. He began to

talk coherently and freely, knew his name and address, and rapidly showed an increasing degree of intelligence. After the 20th day, however, the daily improvement became very slight and at this stage he was showing moderate impairment of intelligence, only slight ability to write and slight euphoria. Nevertheless, over the ensuing weeks improvement did continue to take place so that, at the time of his discharge on the 48th day, his family were of the impression that he was "almost the same as ever."

Psychiatric investigation at this time of his mental status showed, on the other hand, that there was still moderate impairment of cerebration, the Binet test revealed that his mental age was 9 years, 10 months (chronicological age being 14 years, 4 months) and his I.Q. was 71%.

The only other evident sequela in this case was a moderate weakness of the left ankle with partial foot-drop. Examination and investigation with galvanic and faradic currents suggested that it was of peripheral, rather than of central, origin. Physiotherapy, during the last three weeks in hospital, produced marked improvement, and it was felt that, if this were continued as advised, it should promote complete restoration of function within a few months.

The patient has been followed and rechecked fairly frequently since his primary discharge from hospital. At the end of one month, his family reported that there has been a daily improvement, both mentally and physically. At that time, the Binet (Revised Standard) test showed his mental age to be 11 years and his I.Q. 79% while examination revealed considerable improvement in the weakness of his left foot. A recent review of the case, approximately 3½ months after discharge from hospital, disclosed marked improvement. There now remained little evidence of the previous definitely organic mental deterioration. The Wechslar—Bellevue Intelligence Test revealed an I.Q. of 103% (normal 90-100) and a mental age equivalent to his chronological age. He gave indication that his I.Q. prior to his illness was possibly somewhat higher than normal, probably in the vicinity of 120 %, and it is hopefully expected that he will re-attain this level with the passage of time. Examination of his left leg revealed equally satisfying improvement. Only slight weakness in dorsiflexion and plantar flexion and a slight limp remained. The boy has been resuming his former energetic activities for some weeks, is feeling fine and he, himself, notes a rapid return of his weakened foot toward normality.

Discussion—Influenzal meningitis is common in infancy and early childhood but rare in late childhood and adults. Consequently, as previously mentioned, its occurence in this otherwise healthy young boy was unusual to say the least and, in addition it provided a case which proved to be an interesting "novelty" as far as this "adult" institution was concerned. Its fulminating onset was equally remarkable and indicates how vicious this ordinary "secondary invader" can be at times.

The difficulties encountered in establishing a diagnosis of the causative organism from the spinal fluids are not readily explainable. Usually the organisms are quite easily demonstrable so that it is an open question in this case as to whether or not the whole answer lies in the failure to demonstrate organisms in the first spinal fluid specimen because of contaminated media, as this would necessitate assuming at the same time that subsequent specimens were sterile due to the rapid action of sulfadiazine and/or penicillin. It

has been suggested that a correlation of these difficulties with the fulminant onset may indicate that this was a case primarily of "ventriculitis". This opens another field for discussion which, for my part, will be left as such. Ventricular puncture was carried out because of the suspicion of interventricular blockage. Although this suspicion was not verified, the procedure nevertheless certainly justified itself in that it provided the diagnostic fluid This experience would seem to suggest the early use of this relatively simple and harmless procedure as a diagnostic aid in any similar confusing case of acute fulminating meningitis.

The causation of a partial foot palsy in this and in many similar cases has never been explained adequately. One may make conjectures regarding the toxic effects of a therapeutic agent, be it sulfa, penicillin or streptomycin. One may argue as to whether or not its toxic factor acts as a general systemic poison with its effects seen in these cases merely as the dragging of a foot; or whether or not it works only when introduced intrathecally where it can attack in high concentration certain fibers of the cauda equina. One is even led to wonder what part is played by a hospital's meticulous method of bed-making, with its neat but tight binding of the bed clothes at the foot of the bed. Certainly, foot-drop is not an infrequent sequela in cases of coma of varied causation but the explanation is still elusive. It may be that no single factor is responsible, but rather a different combination of factors are possibly participant in different cases.

With reference to therapy, it may be admitted that it was satisfying to find that the regime used was in agreement with the most recent recommendations for the treatment of this disease, especially with regard to the utilization of a combination of all available therapeutic measures. Since the pathway of early diagnosis and early institution of therapy was strewn with many obstacles, a dim outlook was for a long time entertained, making the ultimate outcome an even more gratifying one.

Summary—A resume of the salient features of Influenzal Meningitis is presented with special attention being given to the latest methods of therapy and the resultant remarkable reduction in mortality rate.

The frequency of this disease in infancy and early childhood is re-iterated and the urgency of early diagnosis and early institution of therapy is stressed.

A relatively rare incident of this disease in a fourteen year old boy, with a few of the interesting features of the case and his eventual recovery, is reported.

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*Reactions to Injury

M. I. Engel, M. D.

Four cases are presented:

1. B. B.—Causalgia in the foot, following a gunshot wound of the thigh with medial and lateral popliteal nerve injury; almost complete relief obtained by paravertebral sympathetic block repeated once.

2. M.A.—painful amputated finger stump; cyanotic wet, tender stump with mild causalgia.

3. K.—Continuous pain in the calf following acute thrombophlebitis 2 years previously. No edema, normal skin, slight deep tenderness in the mid calf.

4. Y.—Pain and swelling of the knee following a superficial soft tissue injury of the knee, without evidence of internal knee joint damage at the time of injury.

I would like to give a brief discussion of these cases presented in respect of the possible causes of their symptoms and signs, and the physiopathologic processes concerned.

First let me outline some of the present concepts regarding vasomotor innervation and controls.

When a blunt pointed instrument is drawn lightly over the skin a white line appears along the course of the path drawn. This, Lewis states, is due to a tension stimulus on capillaries with resulting contraction. If a more pointed instrument is used, with more pressure, there follows a "red reaction" due to active capillary dilatation. This is not dependent on a nervous mechanism.

If more pressure is used then a flare occurs about the path drawn. This is the result of an active arteriolar dilatation and is dependent on a local nervous mechanism, the axon reflex. It occurs after division of, but not after degeneration of nerves to the area.

If still more pressure is applied then a wheal will replace the reaction. This wheal is due to a transudation of fluid from the vessels concerned in the red reaction. It does not depend on a nervous mechanism but on the presence of flowing blood.

Vasoconstrictor fibres of arterioles:—Arise from the lateral horn from T I to L 3 and via a proximal and distal route reach the arterioles, e.g. in the lower limb proximal fibres go directly from aortic plexus to iliac and thus femoral vessels while the distal part accompanies the peripheral nerves of the part to be given to the vessels further on in the limb-supplying arterioles and capillaries.

Vasodilator fibres to the arterioles:—Carried by 1(the para-sympathetic N. S. i.e. cranial nerves 3. 7. 9, 10 and the pelvic nerves which reach the vessels by proximal and distal routes as mentioned before. 2) also in part by the sympathetic system (this is debated by many)—and 3rd. andidromic impulses. The latter are impulses carried along nerves in the posterior roots in a direction opposite to that in which the sensory impulses travel. Bayliss and Head have shown that these dilator impulses pass along sensory fibres subserving the sensation of pain and for the most part pass on to the vessels of skin and

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*Presented to Staff Meeting at Camp Hill Hospital, January, 1947.

viscera, not muscles. Dilatation in muscle is from the sympathetic outflow but in the main from metabolites.

Regarding the Capillaries:—Evidence for sympathetic constrictor fibres to these has been obtained by many. Sympathetic filaments, extensions of the periarteriolar plexus have been seen ending near, but not on the capillaries.

(Engel postulates that the sympathetic nervous system controls permeability. Perfusing vessels about knee joint with dye-fluid and stimulation of sympathetic nervous system gives rise to an increase, and cutting of sympathetic nervous system to a decrease, of the dye excretion into the joint).

Dilatation of the capillaries appears to be due to a chemical substance liberated at afferent nerve endings. Woolard has found afferent fibres reaching to the arterioles and ending in collateral branches in the arteriole and in the subcutaneous tissues, some with Pacionian corpuscles (deep pressure receptors). These are the fibres probably used in the axon reflex. These fibres also probably convey antidromic dilatory impulses to the capillaries. However no fibres have been seen to end on the capillary itself and as Lewis points out, dilatation is probably due to a chemical substance liberated at these nerve endings. Fibres for antidromic impulses end near the capillaries.

Skin colour is due in part to the degree of dilatation of the subpapillary venous plexus and to the degree of oxygenation of Hb. The temperature is due to the rate of blood flow, controlled by arterioles. (Cold and blue thus means arteriole constriction slowed flow and capillary dilatation). It would seem with these findings, that there must be a close relation between vasomotor control in respect of its antidromic impulses and sensations of pain and deep pressure which are common symptoms following injury. In serious causalgia the disabled extremity is reddened, glossy, edematous, cool, subject to burning pain, sore to touch, intolerant of dryness and intensely sensitive to draughts and jars; bones are atrophied, as are muscles also. These phenomena sometimes occur following injury to large nerves or vessels.

Related to this we have similar states following lesser injuries, such as blows, crushes, fractures, minor wounds or punctures or after thrombophlebitis. Changes which may follow these also included oedema, osteoporosis, Sudeck's atrophy, reflex dystrophy of extremities,chronic segmental arterial spasm, joint disorders, paresthesia and vasomotor disorders (the latter may be the cool, bluish, smooth skin, due to arterial constriction but occasionally vasodilatation may occur). These phenomena may occur together or in various combinations. Thus we find many names attached to the disease, depending on which signs and symptoms are most prominent (post-traumatic dystrophy, Sudeck's atrophy, reflex dystrophy and causalgia).

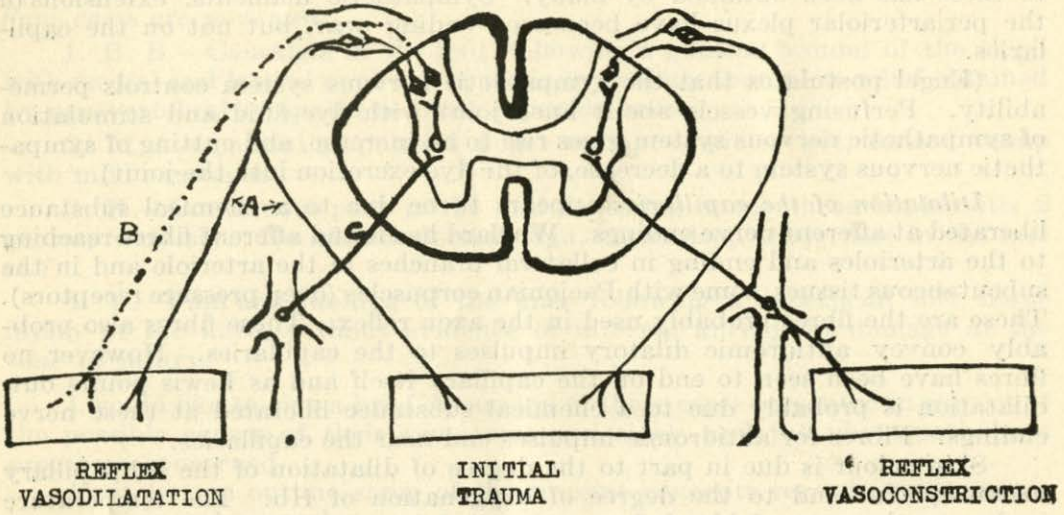
LeRiche states that whatever pain is, it is often abolished by cutting the sympathetic supply of the area.

Causalgic pain does depend on the persistence of an unstable and often easily broken reflex.

The pain in the calf following acute thrombophlebitis, LeRiche attributes to inflammatory reaction about the vessels and is related to the diffuse peripheral spasm.

Homans states further that the reflex changes result from impulses heading centrally in any case.

According to de Takats, trauma of any sort will irritate nerve endings. Normally he claims there are three types of reflexes set up.



(A) is an axon reflex; the afferent impulse does not reach the cord. It acts as a vasodilator impulse to the capillaries and is seen in the flare of the "triple response to injury." This will be absent if the peripheral nerves are degenerated.

(B) is a vasoconstrictor reflex from the sensory stimulus causing afferent vasoconstriction via sympathetic system and gives rise to artery and arteriolar constriction.

In the causalgic state we have an altered reactivity to trauma on the part of the patient due to neurogenic influence

Three stages in this progressive vasomotor disturbance are recognized.

1st.—the extremity is warm, hyperemic, edematous, a severe persistent burning pain and dyesthesia (but no sensory loss or hyperesthesia), skin is warm and dry.

Heat, massage or exercise aggravate the pain. Elevation, cold moist compresses, arterial compression relieve it. Reflexes A and B are then at work B accounts for the strips of hyperemia and dryness in the area. Apparently at this stage we have dilatation of both capillaries and arterioles causing flushing and increased blood flow through the part.

In the second stage the extremity becomes cooler and cyanotic. Hyperhydrosis appears. Due to splinting action of muscles and edema, to changes in endothelial permeability, joints become stiff, synovia thicken and tissue fibrosis occurs. Pain spreads to the roots of the limb and is aggravated by extremes of temperature and jarring. Here the vasoconstrictor element is coming to the fore. Secretion of pain-substances at the nerve endings is responsible for the stocking type of spreading hyperalgesia. The effects can be abolished by a sympathetic block. The vasodilators are cholinergic fibres. P. V. block increases the blood flow by arteriole dilatation and washes away

the pain-substances notably acetycholine. It appears at this stage that we have mainly arteriolar constriction with still some capillary dilatation resulting in a slowed blood flow, thus an increase in reduced hemoglobin in the part with cyanosis and cooling seen. Thus there is presented a further impetus to the continuation of stimuli due to the formation of metabolites and painsubstances.

In the 3rd stage there is atrophy of all tissues and ankylosis of joints. Pain becomes intractable and patients demoralized.

The disease may "burn out" at any stage but factors in the person's autonomic makeup may influence the picture.

Livingston and Mazet state there is an active dynamic influence from the central nervous system contributing to the development of tissue change not only in the extremes of reflex dystrophy and causalgia but also in everyday trauma. There is a neural mechanism causing nutritional disturbance of the extremities. Volkman's paralysis is now deemed to be due to peripheral arteriolar constriction and is a similar phenomenon. One derives the impression that irritation of sensory nerve fibres accounts for all of the changes in the causalgic state—how the irritation is set up and what type of nerve fibre is guilty, is not known.

Some writers say "sympathetic afferents" are supplied to the blood vessel and they may be responsible for the various changes. The pain and hyperesthesia does not conform to somatic nerves. The pain is diffuse, radiates and is poorly tolerated as in visceral pain. They believe that when incoming impulses to the lateral and anterior horus are pathological then the outflow is also pathological and gives all manner of vasomotor, sudomotor and motor disturbance.

Evans (Boston) attributes the cause of "Reflex sympathetic dystrophy" to a prolonged bombardment of pain impulses on the internuncial pool of neurons in the cord and thus is kept alive at this point a constant synaptic activity. Some of these synapses give rise to sympathetic activity and some to motor activity giving cramps and spasms and stimuli to pain travelling up the thalamic tract. The afferent path is in the posterior root. A vicious circle is produced due to the tissue effects of the sympathetic and motor effects which cause more afferent impulses.

Rene' LeRiche in Strasbourg, France, explains the genesis and treatment of hydrarthrosis and traumatic arthritis which appears based on similar concepts. He describes cases of joint pain and/or swelling occurring ten days to two weeks after injury. In one knee joint with effusion (there was no hemarthrosis, fracture or infection) there was a "typical subacute synovitis" found at operation and decalcification of femoral condyles and thickened swollen synovium. Similar findings were seen in a shoulder where also was seen a marked osteoporosis of the upper humerus and at operation a partially detached and partly necrotic thin cartilage. There was much proliferating bone marrow and osteoclastic activity. The changes were as in T.B., yet there was no infection on guinea pig inoculation.

Oscillemetric tests, he states, prove every traumatism in man to be above all a traumatism of vasomotoricity. After injury there is disequilibruim of vasomotor system by a vasodilatation. Normally this is transient but sometimes it persists permanently (probably depends on individual nature of susceptibility of the sympathetic nervous system or on the trauma). After

weeks or months a steady state of vasodilatation with cyanosis occurs and changes in the skin, nails, hypertrichosis, muscle atrophy and bony rarefaction occurs. Bony osteoporosis results from active vasodilatation and hyperemia not functional inactivity, so-called "disuse atrophy."

It is because of hyperemia that we have repair. Post-traumatic hyperemia is met every day but does not have its deserved attention.

LeRiche sums his findings as follows:

(1) Trauma of an articular region produces a hyperemia which if it persists more than ten days causes synovial, osseous and cartilaginous changes.

In the synovia it gives an aseptic synovitis and effusion.

In the bone it gives rarefaction which on reaching the subchondral area causes nutritional disturbances in the cartilage and detachment. Thus we have traumatic arthritis.

If allowed to proceed, permanent changes occur. He is convinced that many cases of arthritis deformans involving a single joint, began as above.

As early treatment for these he suggests (1) avoiding heat, massage, mechanotherapy and every cause of hyperemia. Rest, cold water or even blood letting with leeches is better. Neither plaster immobilization nor normal activity are advised.

(2) In later stages there is no use in forcible mobilizations or massage. Hydrotherapy is better (pouring 1500 litres of hot water over body in a few minutes to cause changes in the general vasomotor conditions).

(3) In serious cases, surgical treatment (early synovectomy). LeRiche has carried out periarterial sympathectomy in traumatic osteoporosis with pain with good results. He plans to try the same in hydrarthrosis. He stresses the fact that bone and cartilage lesions do arise from functional circulatory disorders and suggests new possibilities for sympathetic surgery applied to bone pathology.

Homans states his treatment for minor causalgic states is,

(a) To procainize or excise the "trigger point."

or

(b) Repeated P. V. blocks.

or

(c) Periatyerial sympathectomy.

Sympathectomy should be a last resort. He states that the easiest way is the best way and that resorting to sympathectomy first is laziness, not artistry.

Evans has termed the state concerned here as reflex sympathetic dystrophy, a much broader term than causalgia since in many cases of rubor or pallor and heat or cold, increased sweating and edema, pain may be absent. He also suggests injection of the "trigger point" or the sympathetic pathway repeated if necessary. He has given sympathetic blocks to nineteen patients with such disease as chronic sprain, pes planus, torn ligaments, Scalenus syndrome with Raynaud's, probable herniated disc, old thrombophlebitis and healing or healed fractures, etc. with good but varying relief of symptoms and signs in all but one case. The most effective place to attack the situation would be at the site of the internunical synaptic activity but it is not possible to so so at present.

However in unresponsive serious cases of causalgia one must resort to posterior rhizotomy, or chordotomy, or ablation of the sensory cerebral cortex.

My own impressions are, that too often we look for etiologic factors in disease beyond our own back door so to speak, and do not pay enough attention to functional phenomena which may be at hand to produce the various symptoms-complexes and even pathological findings in disease. One may use LeRiche's term calling these "physiopathic" diseases.

Here we have described the far reaching effects of mild or severe trauma. I do not believe one needs to stretch the imagination too far to relate the findings to such diseases as Paget's disease of bone, or Marie-Strumpell's disease, or rheumatoid arthritis, trigeminal neuralgia, Scalenus syndrome, fibrositis, and a host of others.

In each of the patients presented earlier one can no doubt find similar process at work as a cause for their symptoms.

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Epilepsy

F. A. Dunsworth, M.D., C.M.

Department of Neuropsychiatry, Camp Hill Hospital,

Department of Medicine, Dalhousie University,

Halifax, N. S.

WE feel that our first task in this discussion is to define eqilepsy and we have decided the definition of Hughlings Jackson of "a sudden, excessive and unruly discharge of neuronal cells," is by far the best one for our use even though we realize that it is not too specific. Bing's* definition is also useful " a chronic disease of the brain manifesting itself by a sudden loss of consciousness which may or may not be accompanied by convulsions."

The formal classification into Symptomatic and Cryptogenic epilepsy is much too basic for future studies into this disease and the classification of Penfield* is a marked improvement.

TABLE 1

CLINICAL TYPE	LOCALIZATION
Somatic Motor	
1. Generalized Seizure (Grand Mal)	Complete Motor
2. Jacksonian Seizure (Local Motor)	Pre-rolandic Gyrus
3. Masticatory Seizure	Lower Rolandic
4. Simple Adversive Seizure	Frontal
5. Tonic Postural S. (Decerebrate, Opisthotonic)	Brain Stem
Somatic Sensory (Auras)	
6. Somatosensory Seizure	Post-rolandic Gyrus
7. Visual Seizure	Occipital
8. Auditory Seizure	Temporal
9. Vertiginous Seizure	Temporal
10. Olfactory Seizure	Infra-Temporal
Visceral	
11. Autonomic Seizure	Diencephalic
Psychical	
12. Dreamy State Seizure	Temporal
13. Petit Mal	
14. Automatism (Ictal and Post-Ictal)	
15. Psychotic States (Secondary)	

Symptomatic Epilepsy

Epileptotype seizures may be due to many factors, e.g. cerebral neoplasm, cerebral degeneration, systemic disease, dehydration, adreno-cortical

*Bing and Haymaker:—Textbook of Nervous Diseases, Mosby, 1939.
**Penfield and Erickson:—Epilepsy and Cerebral Localization, Thomas 1941.
***"From Penfield and Erickson's Epilepsy and Cerebral Localization—courtesy Charles C. Thomas, Publisher. Spryfield, Illinois." (Table 1 and 2)

hypofunction, etc., etc. As our knowledge increases the cryptogenic group becomes smaller and smaller as group after group is discovered to be symptomatic of some organic process. In reference to this we mention the syndrome of *Hypoglycemia*. It should not be assumed that this is very common but formerly this was confused with cryptogenic epilepsy. The diagnosis is frequently made by the history. The convulsions usually occur in the early morning or late at night, i.e. associated with a fasting state or perhaps after great exertion. These attacks are often preceded by weakness, sweating, thirst and hunger. It is commonest in younger people. Individual attacks are relieved by carbohydrate ingestion or injection but they are not avoided by a carbohydrate diet, since this stimulates insulin secretion, but by a diet high in proteins and fats. There are several possible causes for this condition, but the possibility of a thalamic neoplasm must be kept in mind.

A few comments are required on *Jacksonian Epilepsy*. This type of seizure is commonly seen in organic cerebral states. It begins as a localized twitching and /or paresthesia, usually in an extremity, spreads up the limb, increasing in scope and may merge into clonic movements and may terminate in a generalized Grand Mal seizure. Observation of the spread may be very useful in localizing the cerebral lesion, and another useful sign is a post-seizure paralysis of one particular region of the body (Todd's paralysis), indicating the cerebral localization.

Post-Traumatic Epilepsy is another type of symptomatic epilepsy. It occurs after *severe* head injuries. It may manifest itself by Jacksonian or generalized seizures; there may or may not be localizing neurological signs, but the Electroencephalogram (EEG) will reveal slow wave activity and paroxysms of waves if seizures occur. Control in these cases may be neurosurgical or anti-convulsive medical therapy. We would like to comment here that one of the best ways to judge the severity of a head injury in the absence of localizing signs is the duration of unconsciousness and also the amount of amnesia, both anterograde (before the accident) and post-traumatic.

Cryptogenic Epilespy

Cryptogenic Epilepsy. We prefer this term to the terms Ideopathic, Essential, etc., and feel that it is more exact since it correctly infers that this is a type of epilepsy whose cause is unknown or hidden.

Cryptogenic Epilepsy is further classified by the type of seizures that occur.

(1) *Grand Mal*—characterized by sudden attecks of unconsciousness, often associated with an aura, with the sequence of a tonic (rigid) phase, then a clonic (jerking) phase. Tongue biting, salivation, voiding, soiling may occur. Following the seizure post convulsive confusion, headache and drowsiness may be present. The essential components are—

(a) Somatic Motor Activity (tonic and clonic)
(b) Viscero-Motor Activity (voiding, soiling)
(c) Loss of Consciousness.

(2) *Petit Mal* (Lennox's classification)
(a) *Pykno* epilepsy. *Short* transient lapses of consciousness. The attack appears to the observer as a slight lapse in the patients' speech and /or activity; is not too common, sometimes ceases at puberty.

(b) *Myoclonic*—A single quick muscular contraction.

(c) *Akinetic*—The attack occurs as a sudden loss of postural control related to narcolepsy.

Common characteristics of Petit Mal Epilepsy—

1. Great frequency
2. Short duration
3. Abrupt onset and termination
4. Maintenance of mentality.
5. Sensitivity Acid Base balance
   O_2 balance
   sugar balance
6. Tendency to recover with age
7. Begin in childhood almost exclusively
8. Refractoriness to ordinary anticonvulsive therapy.

Psychomotor—This type is probably not a pure type, but does not fit correctly into Grand Mal, Petit Mal classifications. (Other terms—epileptic equivalents, equivalent state.) Characterized by states of uncontrolled activity of many types, where patient may wander away, steal or commit arson—the whole phase being amnesic.

This type is very interesting, but must not be considered common. Medicolegally it has often been invoked as a defence in some court cases. Hughlings Jackson considered this type always followed an epileptic attack. This attack may have been so slight and transitory as to have escaped notice. Recent Electroencephalogram studies seem to substantiate this view.

Diagnosis and Differential Diagnosis

(a) The Personality. The epileptic in an Epileptic Institution and the epileptic in civilian life, while having many features in common, differ more than simply in degree. The institution epileptics show a much higher incidence of hereditary transmission; they often develop convulsions earlier in life, have them more frequently, more severely and are often more refractory to treatment. They also more commonly show mental deterioration. Arguments have appeared for years in medical publications concerning this latter statement. Some physicians consider the higher doses of anti-convulsive medication, inadequate diet and institutional life the greatest factors in this deterioration, but we believe that these patients are of a more malignant type, a progressive cerebral deteriorating mechanism. Bleuler describes institutional epileptics as short tempered people, being self-centered, opinionated and unreliable. They also have been compared to psychopaths. I am sure many of you agree that only a small number of epileptics you have seen show these undesirable traits and many we have seen are well behaved, cooperative, intelligent and thankful for treatment and advice.

(b) The Fits: Grand Mal and Petit Mal seizures, as described above.

Hysterical fits may be an isolated feature but, generally speaking, are but a part of the hysteria complex. First we must emphasize that Hysterical fits, while not having the true characteristics of epilepsy are *not* "put on," though they do not arise from an organic condition, they are real and certainly are real to the patient. All sorts of manoeuvers abort these fits. This proves they are not epileptic or organic only.

As stated they are another symptom of the immature, attention seeking hysterical personality and appear as an attempt to escape from an unpleasant situation, or to obtain some gain, i.e. they are an immature attempt to solve a conflict.

On the other hand malingering, NOT as common as is supposed, may take the form of convulsions. The desired object of escape is *very* obvious, the whole pantomine *very* obvious too to the keen observer. The malingerer is a very poor type, usually psychopathic and, fortunately, less common in private practice than in the service or other uncomfortable situations.

(c) Inheritance of Epilepsy. Frankly, the more we read the less we know. We have yet to know, in plain percentages, the familial incidence of epilepsy. The whole question of human genetics would have to be considered before accurate incidence of inheritance could be volunteered. A family history of epileptic parents and/or epileptic siblings is discouraging as an aid in diagnosis. Collateral history (i.e. cousins) is often just as valuable. The rough incidence of a positive family history in an epileptic is probably around 10%, but if asked by an epileptic if he'll have epileptic children we can only guess.

(d) Incidence of Epilepsy. There are *many* epileptics—we are sure of that at any rate. Not to confuse but to illustrate, I quote Lennox "One in every 20 hospital beds in the United States is occupied by an epileptic, and yet only 5% of epileptics are Institutional cases!!" For every frank epileptic there are probably three people who are latent epileptics, i.e. seizures will appear with intercurrent disease, marked stresses, etc. This group, the latent epileptics, are probably the cases that appeared during service, and after discharge often clear up nicely. (NOTE—This is not the same as Post-Traumatic Epilepsy). We wonder too if infantile convulsions may be of latent epileptic origin.

(e) Age—This is very important.

Convulsions occurring in the—

First Year of Life: mostly cerebral birth traumas or congenital brain malformations.

Next four years: plus the above (intra and extra cranial) acute infections commonest.

Until 20 *years*: Ideopathic epilepsy.

Second two decades (i.e 20-40):—Brain tumor.

Later life: renal and cerebro-vascular disorders.

As a rough guide, the above is very useful.

(f) Electroencephalograph. I will not attempt to go deeply into this here. This section is on the plane of what the average physician should know of this instrument. NOTE:—The only available E.E.G. Department in the Maritimes is set up at Camp Hill Hospital. It is available for all cases, Service and civilian, *when necessary* through special arrangement and appointment.

Summary:—The Electroencephalogram and an expert interpretation will give an accurate diagnosis of epilepsy in 90% of cases. This does not mean that one tracing will be sufficient. Repeats, often with special routines may be required, but 90% is the accepted figure where the instructions of the

electroencephalographer are followed, the patient cooperates, the Electroencephalogram technicians and machine are functioning correctly.

One comment—the brain waves of a young child are sometimes poorly organized, so under eight years the results are not quite so accurate. For the rest the following is true—

The Electroencephalogram will diagnose Grand Mal eqilepsy and distinguish most of the Petit Mal variants. However, it does more than that—the expert Electroencephalographer will be able to determine the presence of an organic focus, e.g. tumor or post-traumatic scar and, in some cases, be a very good judge of the indications and prognosis for neurosurgical treatment.

Tracings. Petit Mal has a spike and dome complex—the name derived from the shape of the tracing—a small, fast spike followed by a large (i.e. high voltage, slow frequency) dome. This is the "inherited" type—often diffuse—commonest in children.

Psychomotor is a very high voltage, slow frequency wave, appearing in groups—oftenest diffuse.

Grand Mal is characterized by bursts of high voltage, high frequency waves ("spikes"), diffuse in many—localized in foci of diseased tissue (e.g. tumor or traumatized areas).

Therapeutic Considerations and Treatment

We do not propose to enter too deeply into the mechanism of the actual fit but we would like to assure you that the problem is a very intricate one. The whole field of electroneurophysiology, synaptic transmission, hormones and antihormones required for consideration of etiology is very complex.* We propose, instead, to state the clinical factors that have been known for some time, the variation in which modifies the seizures.

It has been found that several factors contribute to the occurrence of seizures in an epileptic person. These are:

1. Hyperhydration, e.g. the pre-menstrual occurrence present since there is water retention premenstrually.
2. Alkalosis.
3. Faulty elimination.
4. Inactivity.
5. Psychic factors.

I will refer to the first two factors later, but concerning the latter three it is obvious that a sensible way of life is necessary to properly handle these cases. Epileptics do better when they are doing something. Some industries in a very stolid fashion have declared that they will not employ epileptics. If an epileptic's fits are infrequent and the occurrence of a fit will not constitute a danger to himself (e.g. high places) or others (e.g. bus driver), he is certainly employable—there are fewer fits when the patient is working, provided he is not too fatigued.

The significance of proper elimination and diet is obvious. The psychic factors should not be dismissed lightly—firstly the *physician* should under-

*Recent work by Winifred Ashby, St. Elizabeth's Hospital, is worthy of note.

stand the disease, then, the patient's family should understand it too. The proper balance of understanding is required, not too much sympathy and above all not too much pity.

Alcohol is contrindicated in epileptics.

To return to the first two factors—exaggeration of body hydration (pitrissin-hydration test) and raising pH of the blood (through hyperventilation) are useful mechanisms, diagnostically. In treatment, the patient's fluid intake should always be restricted and the diet should be ketogenic (high fat, low carbohydrate) and also ample in calories.

Drug Treatment

A.—Bromides (since 1853).

Bromides still have a small place, but they should be very restricted and should be considered lastly, not firstly.

Bromide is of no use in focal attacks—its efficacy in Petit Mal epilepsy and Grand Mal epilepsy about equal.

Side effects are quite marked in some people.

Its activity is related to chloride content of the cells. The less NaCL ingested the more active the bromides, therefore salt should be restricted with bromide therapy.

Incidentally, NaBr is the drug. Nothing is to be gained in using triple bromides or five bromides.

B.—Phenobarbital (since 1912)

The commonest drug in present use. The efficacy and percentages of results not clear even now and cannot be quoted.

Note: The cessation of phenobarbital medication leads to an increase in the number of seizures in over 70% of cases and status epilepticus is prone to occur.

Note: (1) Most effective in motor type.
(2) Cryptogenic and traumatic epilepsy helped to a greater degree than senile or syphilitic eqilepsy.
10 grains per day maximum dose—6 grains a better limit.

It is better to adjust the drug to the seizure rhythm, i.e. whether fits occur by day or night etc. and the occurrence—e.g. relationship to menstrual periods, etc. Medication should not be discontinued abruptly.

The duration of medication depends (1)on patient
(2) Electroencephalogram evidence.

Side effects—toxic—skin; stomach
—drowsiness (Benzedrine sometimes useful)

Present researches aimed at finding drugs that protect experimental animals from induced convulsions.

C.—Dilantin Dodium (Sodium Diphenyl Hydantoinate).

Merrit and Putnam (1938) found it useful in protecting experimental animals against convulsions and was well tolerated. It is most effective against Grand Mal epilepsy and is more effective than phenobarbital against psychomotor attacks.

The drug is very alkaline and hence patient's stomach is often upset, so medication should be with meals. If used to replace phenobarbital, do not discontinue phenobarbital abruptly but change over gradually.

Side Effects

(1) Hyperplasia of gums (treatment—good oral hygiene and Vitamins B and C)

(2) Toxic effects—(*Not* frequent)

(Tend to clear up spontaneously, but dosage might be lowered for awhile).

(c) More serious toxicity is dermatitis and purpura (perhaps 5%), but apparently uncommon in recent years to judge by the literature and experience.

(a) GI symptoms and appetite and weight loss.

(b) Mild CNS symptoms, e.g. dizziness, weakness.

Occasional patient shows a related fever.

(Medicine should be discontinued in these cases).

Average dosage, grain iss tid.

D.—Benzedrine (and caffeine)

Often helpful in Petit Mal epilepsy, e.g. narkolepsy, akinetic Petit Mal epilepsy.

E.—*Tridione (investigated within past 8 years) (Trimethyloxazolidine dione).

From preliminary reports the best drug in Petit Mal epilepsy.

Abbott Laboratory technicians demonstrated that this drug protected experimental animals against induced convulsions. Has no effect in Grand Mal epilepsy, but effective in Petit Mal eqilepsy in humans. Tridione is somewhat toxic and several cases of aplastic anemia terminating in death have been already reported.

It is not "*a new anticonvulsant*" as advertised, as it has no effect of note in Grand Mal epilepsy.

Side Effects

Skin reactions more frequent than in dilantin and phenobarbital.

Photophobia quite common.

Note:—Under medication { Electroencephalogram tracings improve in Petit Mal epilepsy. / Withdrawal does not seem to produce a flock of seizures.

*F. Mesantoin (Sandoz) (3 methyl 5.5 phenyl-ethyl-hydantoin) (a new drug—not generally in use as yet).

90% reduction in seizures, reported by Kozol, but not as yet substantiated by other investigators.

Types of patients where mesantoin indicated:—

(1) Frequent seizures despite maximum dilantin treatment.

(2) Marked gum hypertrophy and other side effects, i.e. most of cases treated were those who did not respond well to other forms of therapy.

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*Two New Drugs In Epileptic Therapy - Lennox, Wm. G., American Journal Of Psychiatry (Sept. 1946).

*Epilepsy - Treatment with a new drug (Phenantion) - Kozol, Hary L., American Jotrnal Of Psychiatry (Sept 1946).

Mesantoin can be given in *larger* doses than dilantin or phenobarbital.
" has an agreeable taste.
" less side effects.
" low toxicity.
" sometimes produces drowsiness.
" quite effective in combination with dilantin.

Effects—
(1) reduces frequency of seizures.
(2) increases intervals between seizures.

Main effects with Grand Mal eqipepsy,—
less on psychomotor,
(no assessment Petit Mal epilepsy—group too small).

The authors recommend starting with *Small* dosage, i.e. O.I Gm./day (1½ grains).

Surgical treatment

Before surgical treatment is discussed, the pathology of epilepsy must be considered. See Table II (from Penfield).

Table II

A. With Demonstrable Cerebral Lesions	Produced By:
1. Expanding Lesions	Neoplasm, Chronic Abscess, Etc.
2. Cerebral Cicatrix	Trauma, Infection
3. Local Cerebral Atrophy	Compressions, Ischemias, Infections
4. Local Microgyria	Infantile Compression or Ischemia
5. Brain Cyst	Vessel Closure or Hemorrhage
6. Diffuse Cerebral Disease	Degenerations, Infections, Sclerosis
7. Diffuse Cerebral Vascular Disease	Arteriosclerosis, Syphilis, etc.
8. Miscellaneous	Congenital Lesions, etc.
B. Without Demonstrable Cerebral Lesions	
1. Idiopathic (Cyrptogenic)	Abnormal Cerebral Physiology
2. Toxic & Febrile	Extracerebral Causes
3. Hypoglycemic	Extracerebral Causes
4. Miscellaneous (Angioneurotic, Circulatory Arrest, etc.)	

Convulsions arise from an irritation of cerebral nervous tissue. This irritation may be due to many causes—systemic infections, atrophy, hemorrhage, etc., etc. Naturally then as accurate a diagnosis as possible is the first requirement. We will consider that cryptogenic epilepsy is due to "abnormal cerebral pathology" (Penfield).

These people, the cryptogenic epileptics, have a familial tendency suggesting an inherited factor; their pathology might be due to *abnormal physiology of the cerebral circulation.* In this regard we find Electroencephalogram evidence of a diffuse, bilateral abnormality and the view is held that there is a diffuse ischemia that occurs with little precipitating cause. If this ischemia is severe, frequent and of long duration, eventually in many cases small areas of degeneration occur and eventually there is cerebral atrophy. *Obviously this type is not amenable to neurosurgery.*

The "surgical" group includes those cases where there is a *circumscribed lesion* located in an area *approachable with safety,* if this lesion has in its *im-*

mediate neighborhood the epileptogenic focus. Also, to justify the surgical risk, the attacks should be *frequent* and *severe.* Removal of this cerebral segment should *not be likely to cause important disability.*

Naturally with these very important criteria, surgical considerations require the opinions of skilled neurologists and the full use of the Electroencephaloramg and the treatment requires completely trained surgeons and a fully equipped hospital. It is no use sending a case that hasn't a localized approachable lesion to a neurosurgeon and the greatest aid is the Electroencephalogram.

Briefly the types of cases sometimes helped include—

(1) an approachable localized neoplasm.
(2) " " " chronic abscess.
(3) " " " cicatrix.
(4) An atrophic, (post-traumatic) irritating gyrus.
(5) many cases of post-traumatic epilepsy.

The highest incidence of post-traumatic epilepsy comes from the penetrating gun shot wounds of wartime, especially where cerebral damage has been extensive. Adequate debridement after injury with prevention of infection does a lot to prevent seizures. All cases are not satisfactory for surgical treatment, but carefully investigated cases that fulfill the previously mentioned neurosurgical criteria often yield very gratifying results.

Conclusion

Some texts make epileptic treatment sound quite simple, but to treat with success you need a good history, a co-operative patient, a well trained staff, medical curiosity, a neurological knowledge (probably an electroencephalogram) and you should be willing to give individual cases a considerable amount of time. The study is worth it—it has only been through intensive work that the present outlook for epileptics is so much brighter than even a few years ago.

Editor's Note

In last month's issue of the BULLITEN it was announced that a series of articles on Cancer would begin in the June number. Since then we have received from the American Cancer Society a list entitled Cancer Current Literature. Because we feel that the references contained therein may be of interest and value to the doctors of Nova Scotia, we are publishing the list in lieu of an article on cancer. We should like to point out that the list as published does not include the titles of foreign language papers contained in the original. These have been deleted in order to save space, but the original list is on file in the Medical Society office and may be referred to by anyone desiring further information.

M. E. B. G.

CANCER CURRENT LITERATURE

A Periodical Annotated List
Prepared by the Library of
The Medical and Scientific Department

THE AMERICAN CANCER SOCIETY

Vol. I, No. 2

April, 1947

The American Cancer Society

47 Beaver Street

New York, 4

These first issues of **Cancer Current Literature** list complete references to articles in the cancer fields appearing in the medical research journals which have been issued during the previous month.

Cancer Current Literature is intended as a bibliographical record. The American Cancer Society does not have for distribution any reprints of any of the articles listed.

We wish to acknowledge the assistance of Miss Helen Sayer of the Bibliographical Service of the New York Academy of Medicine in covering the journal listings.

A. W. Oughterson
Medical and Scientific Director
Hester Ann Bradbury
Medical Librarian
Chief, Records and Library Section

55. Abowitz, Jacob . . . (
Diagnosis of Cancer of the Stomach, with Special Emphasis on the Pars Cardia. Ann. West. Med. and Surg. 1:35-38 (March, 1947).

56. Abrahamson, Robert H. and Hinton, J. William . . . (Stamford, Conn.).
Gastric Carcinoma: A Comparative Review of the Origin, Diagnosis and End-Results in 583 Patients. Surg. Gynec. & Obst. 84:481-490 (April, 1947).

Poor prognosis and lack of progress between 1918 and 1945 is shown. Early diagnosis has been a failure.

The transformation of benign ulcer to gastric carcinoma is rare.

It is suggested that the following four factors have a definite relationship: (a) age and sex incidence; (b) chronic "gastritis"; (c) endocrine effects on the gastric mucosa; (d) the inception of gastric neoplasm.

A plan is presented for the utilization of mass fluoroscopic and X-ray studies of the stomach, as a means of discovering curable malignant conditions.

30 references.

57. Adair, Frank E. . . . (Mem. Hosp., New York).
The Use of the Male Sex Hormone in Women with Breast Cancer. Surg. Gynec. & Obst. 84:719-722 (April 15, 1947).

Testosterone is not a cure for breast cancer, but its effects are profound and gratifying.

58. Alvarez Zamora, Ramiro . . . (Inst. Urol., Barcelona, Spain).
Sarcoma of the Bladder: A report of four cases. Urol. & Cutan. Rev. 51:88-91 (February, 1947).

8 references.

59. Andrews, Charles G. . . . (Memphis, Tenn.).
The Treatment of Benign and Malignant Neoplasms of the Skin. J. Tennessee M.A. 40:77-82 (March, 1947).

60. Ayre, J. Ernest . . . (Dept. Gynec. & Obst., McGill Cl., Montreal).
Selective Cytology Smear for Diagnosis of Cancer. Am. J. Obst. & Gynec. 53:609-617 (April, 1947).

A new spatula test technique is described providing "surface biopsy" information of pre-cancerous cells prior to their actual desquamation. Tests have been made at the squamo-columner junction of the cervix, and morphologic changes are described here which this author believes to constitute a pre-cancer cell-complex. Death from cancer of the cervix should become highly preventable.

The technique has been found accurate as an indicator of endogenous estrogen by cornification counts.

9 references.

61. Barigozzi, Claudio and Cusmano, Luigi . . . (U. Parma, Parma, Italy).
Chromosome Numbers in Cancer Cells. Nature 159:505-506 (April 12, 1947).

A mitotic figure with a peculiar aspect of the chromosome was observed in 6 epitheliomas of the uterus portio.

5 references.

62. Barlow, H. Cecil . . . (Lincoln Co. Hosp., Eng.).
Malignant Tumour of the Suprarenal Gland with Paroxysmal High Tension. (Case History). Clin. J. 76:53-58 (March-April, 1947).

63. Barnes, J. Peyton and Zarr, L. Lynn . . . (Houston, Texas).
Resection of the Common Hepatic Duct for Papillary Adenocarcinoma. Surg. Gynec. & Obst. 84:427-434 (April, 1947).

Successful resection was obtained. A method of utilizing the ampulla of the gall bladder and cystic duct as a substitute for the common hepatic duct is demonstrated.

3 references.

64. Beck, William C. and Hunter, Albert L., Jr. . . .)
Tumors of the Anterior Abdominal Wall. Guthrie Clinic Bull. (Sayre, Pa.) 16:121-124 (April, 1947).

Two series—of 31 and 56 cases—are summarized.

7 references.

65. Benedict, Edward B. . . . (Mass. Gen. Hosp., Boston).
The Limitations of Roentgenology and Gastroscopy in the Diagnosis of Diseases of the Stomach: An analysis of 53 proven cases. Gastroenterology 8: 251-277 (March, 1947).

The two methods are not competitive but supplementary, and greater diagnostic accuracy is attained when both are used cooperatively . . . If the gastroscopist can get a satisfactory view of the lesion his chances of arriving at a correct diagnosis appear to be greater.

3 references.

66. Bickel, David A. . . . (South Bend, Ind.).
Leukoplakia and Carcinoma of the Vulva. J. Indiana State M.A. 40:338-340 (April, 1947).

Seventeen cases are summarized. Previous studies showing that surgical treatment is definitely important are reaffirmed.

67. Bobbitt, Ray M. and Harwood, Ivan R. . . . (Huntington, W. Va.).
Renal Neoplasms: Review of 48 cases. West Virginia M. J. 43:123-126 (April, 1947).

5 references.

68. Bouton, S. Miles, Jr. and Schanz, Robert F. . . . (St. Mary's Hosp., Rochester, N. Y.).

Carcinoma of Urinary Bladder with Unusual Metastases in a Young Male. Urol. & Cutan. Rev. 51:33-36 (January, 1947).

69. Brailsford, James F. . . . (
Some Experience with Bone Tumours. Brit. J. Radiol. 20:129-144 (April, 1947).

"The purpose of this paper is to set forth the details of a number of cases which were watched throughout their course, during which they exhibited features liable to be misinterpreted as indicating malignancy and the need for drastic surgical measures, yet which resolved without any major surgical intervention. In some, major surgical measures would undoubtedly have been performed but for other features in the cases." Ten case reports.

10 references.

70. Bramhall, Theodor C. . . . (Portland, Me.).
The Problem of Cancer of the Cervix. J. Maine M.A. 38:73-76 (April, 1947).

71. Brown, Willis E., Kraushaar, Otto F. and Bradbury, J. T. . . . (Coll. Med., St. U. Iowa, Iowa City).
Vaginal Smear in the Diagnosis of Gynecologic Cancer. J. Iowa State M. Soc. 37:155-157 (April, 1947).

Technique is being developed. 300 slides from 200 patients have been studied. 3 patients were discovered with unsuspected malignancy in the clinically free group.

72. Brunschwig, Alexander . . . (U. Chic., Chicago).
Resection of Intra-Abdominal Cancer that has invaded the Anterior Abdominal Wall. (13 Case Histories). Surg. Gynec. & Obst. 84:723-726 (April 15, 1947).

73. Burgess, C. M. . . . (Honolulu Clinic).
Carcinoma of the Adrenal Cortex. (Case Report). Proc. Staff Meet. Clinic, Honolulu 13:17-20 (March. 1947).

3 references.

74. Calmenson, Martin and Black, B. Marden . . (Mayo Clinic, Rochester, Minn.).
Surgical Management of Carcinoma of the Right Portion of the Colon with Secondary Involvement of the Duodenum, including Duodenocolic Fistula; data on 8 cases. Surgery 21: 476-481 (April, 1947).

6 references.

75. Carroll, Walter W. . . . (Northwestern U. Med. Sch., Chicago).
Principles involved in Surgical Therapy of "Encapsulated" Fibrosarcoma of Soft Tissues. Surg. Gynec. & Obst. 84:703-709 (April 15, 1947).

Many patients can be salvaged by preventing fatal blood-borne metastasis. This form of metastasis appears to be related to the incidence of local recurrence more than to the incidence of the disease.

Successful outcome will be determined by the procedure used at the time of the first surgical intervention.

13 references.

76. Castilgiano, S. Gordon and Gross, P. Philip . . . (Amer. Oncologic Hosp., Philadelphia).
Master Metal Facial Cast for Swaging Head Masks for the Treatment of Carcinoma of the Skin of the Face. Am. J. Orthodontics 33:319-325 (April, 1947).

Technique for making the masks is described.

3 references.

77. Coman, Dale Rex . . . (U. Pa. Med. Sch., Phila.).
Mechanism of the Invasiveness of Cancer. Science 105:347-348 (April 4, 1947)

Experiments are described testing the adhesiveness and the ameboid movement of cancer cells and the role of hypothetical spreading factors in malignant tumors.

78. Corbus, B. C. and Corbus, B. C., Jr. . . . (Evanston, Ill.).
The Utilization of Heat in the Treatment of Tumors of the Urinary Bladder; A Presentation of Technique. J. Urol. 57:730-737 (April, 1947). Discussion 738-740.

Deep X-ray treatment is recommended as a therapeutic adjunct.

It is suggested that fulgaration in the treatment of bladder tumors be abandoned as a misleading term and as an unjustified procedure.

11 references.

79. Corcoran, William L. . . . (New York).
Melanoma. (2 Case Reports). M. Rec. 160: 211-217 (April, 1947).

6 references.

80. Corscaden, James A. and Gusberg, S. B. . . . (Colum. U. Sch. Med., New York).
The Background of Cancer of the Corpus. Am. J. Obst. & Gynec. 53: 419-431 (March, 1947).

A few observations indicate that a childless woman who is overweight and in comfortable circumstances is a likely candidate for a cancer of the corpus at some time of life. If her menopause is characterized by excessive bleeding, the chances are still greater.

Several fragments of evidence suggest abnormal estrogenic stimulation as one of the growth-stimulating factors. Evidence presented suggests that women destined to have cancer of the corpus are measurably different from other women.

27 references.

81. Covington, E. Eugene . . . (St. Joseph's Hosp. Baltimore, Md.).
Cancer of the Cervix: A Local Recurrence 18 Years after Radium Therapy. (Case Report). J.A.M.A. 133:935-936 (March 29, 1947).

82. Crooke, A. C. . . . (London Hosp., London, Eng.).
Basophilism and Carcinoma of the Pancreas. J. Path. & Bact. 58:667-672 (October, 1946).
"A case is described and 3 similar cases reported in the literature are reviewed. Statistical evidence is produced that this association is not fortuitous and theories of its causation are discussed."
8 references.

83. Davis, John Wyatt, Jr. . . . (Lynchburg, Va.).
The Carcinogenic Action of Hormones. Review of literature since 1935. South. Med. & Surg. 109:63-65; 75 (March, 1947).
4 references.

84. Davis, Kenneth S. . . (St. Vincent's Hosp., Los Angeles).
Problems in the X-ray Diagnosis of Early Cancer. Calif. Med. 66:117.

85. Deming, Clyde L. . . . (Yale U. Sch. Med., New Haven).
Present Evaluation of the Response of Prostatic Cancer to Hormone Therapy. South. M. J. 40:328-332 (April, 1947).
Synthetic estrogen therapy is of equal value and perhaps of more value than castration. Synthetic estrogen treatment should be continued as long as the patient lives, whether he has had a castration or not.
8 references.

86. Dreyfuss, Martin L. . . . (Chesapeake & Ohio Hosp., Clifton Forge, Va.).
Adenocarcinoma of the Vault of the Urinary Bladder. (Case report). Urol. & Cutan. Rev. 51:1-3 (January, 1947).
6 references.

87. Figge, Frank H. J. . . . (U. Md. Med. Sch., Balt., Md.).
Cosmic Radiations and Cancer. Science 105:323-325 (March 28, 1947).
Preliminary experimental evaluation of the possible influence of cosmic radiation on carcinogenesis.
"This remarkable increase in the rate of tumor induction brought about by placing lead plates 3 inches above methylcholanthrene-injected mice was thought to be related to the intensification of the cosmic radiation which results from the production of showers or bursts of ionizing radiations which occur when cosmic rays pass through thin sheets of metal.
According to this hypothesis, carcinogenic substances such as methylcholanthrene induce cancer by converting some of the energy of cosmic radiation into carcinogenic stimuli; in other words, they sensitize the tissues to this kind of energy."
Further tests must be made in an environment free from cosmic radiation.
3 references.

88. Franks, Andrew G. and Barner, John, L. . . . (N.Y.P.G. Hosp., New York).
Basal Cell Epithelioma in a Psoriatic Patch. Arch. Dermat. & Syph. 55:375-378 (March, 1947).
7 references.

89. Gessler, C. N. . . . (Nashville, Tenn.).
The Spread of Carcinoma of the Stomach. South. Surgeon 13:264-269 (April, 1947).
4 references.

90. Glenn, Richard R. . . . (
The Relation of the Porphyrins to the Etiology of Cervical Carcinoma. J. Bowman Gray School of Med. 5:58-61 (March, 1947).
A review of literature.
15 references.

91. Grossman, Samuel L. and Allyn, Russell E. . . . (Harrisburg, Pa.).
Primary Carcinoma of the Ureter. (2 Case Reports). Pennsylvania M. J. 50:715-717 (April, 1947).

92. Harvey, Norman, A. . . . (MC, AUS) (McCormack Gen. Hosp., Pasadena, 2, Calif.).
Kidney Tumors; A Clinical and Pathological Study, with Special Reference to the "Hypernephroid" Tumor. J. Urol. 57: 669-692 (April, 1947).
In a series of 54 pelvic and cortical tumors, 81.5 per cent occurred in the 5th, 6th, and 7th decades of life, and 70 per cent were in males.
The literature is briefly reviewed with special reference to the Grawitz theory and the relations between the renal adenoma and adenocarcinoma. In this worker's opinion, the Grawitz theory is untenable.
62 references.

93. Herrmann, Julian B. and Adair, Frank E. . . . (Mem. Hosp., N. Y.).
The Effect of Testosterone Propionate on Carcinoma of the Female Breast with Soft Tissue Metastases. (6 Case Histories). J. Clin. Endocrinol. 6:769-775 (December, 1946).
3 references.

94. Hieger, I., Henry, S. A., Ross, P. and Winternitz, J. G.
Symposium—Industrial Skin Cancer. Brit. J. Radiol. 20:145-163 (April, 1947).
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35 cases are reported. The age average was 70; 23 cases had metastases, two-thirds of these having pain. Of those with pain, marked relief was experienced within 48 hours after orchidectomy by one-half, with some relief in all. Improvement in well-being and appetite paralleled relief of pain.

The bony metastases showed increased density and became more discrete in the majority of instances, and sometimes also increased in size and number. Improvement in urinary function occurred in only a few cases.

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Society Meetings

Cape Breton County Medical Society

The annual meeting of the Cape Breton County Medical Society was held on May 22nd, 1947, at the Cape Breton Yacht Club, Sydney, N. S. A new slate of officers was elected as follows:

President—Doctor J. A. McDonald, New Aberdeen.

Vice-President—Dr. M.J. Macaulay, Sydney.

Secretary-Treasuer—Dr. F. J. Barton, New Waterford.

Cape Breton Executive—Dr. A. C. Gouthro, Little Bras d'Or Bridge;
Dr. G. C. MacDonald, Sydney;
Dr. J. R. Macneil, Glace Bay.

Representatives on the Executive of The Medical Society of Nova Scotia,
Dr. A. W. Ormiston, Sydney;
Dr. J. S. Munro, North Sydney;
Dr. M. G. Tompkins, Dominion.

The two guest speakers of the evening were Doctor A. E. Blackett of New Glasgow and Monsignor McGillivary, Sydney. Doctor Blackett spoke on the difficulties of using Insulin. Monsignor McGillivary spoke in a very humourous vein on a trip through Europe following the first Great War. Both these speakers were very entertaining. Doctor Blackett's topic was particularly well chosen and contained a great deal of informative material. Following the meeting a buffet supper was enjoyed by all.

F. J. Barton, Secretary-Treasurer.

Important Donation to the Faculty of Medicine

The pharmacutical house of Frank W. Horner Limited has just renewed for two years its annual grant of $15,000 to the Institute of Experimental Medicine and Surgery of the University of Montreal.

Thanks to this grant, the Institute—under the direction of Dr. Hans Seyle—continues its extensive research on hypertension, the number one killer of our times.

At a recent congress in Cleveland, where Dr. Selye represented Canada, it was verfied that more than one half of the whole population of North America will die of sicknesses related to hypertension.

The Institute of Experimental Medicine and Surgery at the University of Montreal was founded in 1945. Its expansion has been such over a two year period that the original quarters established for it at the university are now inadequate and the institute is soon to move into new and more spacious quarters in one of the wings of the university hospital.

An important part of the institute's work is the training of graduate scientists for a career in research. Availing themselves of the facilities of the Institute at the moment are four French Canadian doctors and doctors from Mexio, Brazil, Cuba, France and Greece.

Personal Interest Notes

DOCTOR F. A. Dunsworth of Halifax, accompanied by his wife and two children, left recently for Montreal and Topeka, Kansas, where he will take courses in encephalography and neuropsychiatry prior to taking an appointment at the Menniger Foundation in Topeka.

Five residents appointed to the Victoria General Hospital. Announcement is made of appointment of five resident doctors to the staff of the Victoria General Hospital, which became effective on May 15th. A departure from previous practice is to have a resident in radiology, and appointed to that post is Doctor Peter G. Loder, whose home is in Corner Brook, Newfoundland, and a graduate this year of Dalhousie Medical School. Selected as resident in gynaecology and obstetrics is Doctor A.I. Perlin of Sydney, who previously was in the services and for the past year has been with the Saint John General Hospital. New residents in surgery are Doctor George M. Saunders of Stellarton and Doctor J. Avery Vaughan of Windsor, both graduates this year of the Dalhousie Medical School. Also a graduate of the school this year is Doctor Garfield M. Moffatt of Sydney, who will become a resident in medicine.

Doctor H. I. MacGregor, (Dal. Jan. 1943) announces the opening of an office at 153 Duffus Street, Halifax.

The marriage took place on April 19th of Doctor Donald Sellars MacKeigan, son of the Rev. Dr. J. A. MacKeigan and Mrs. MacKeigan, and Miss Edna Patricia (Pat) Flynn, youngest daughter of Professor A. E. Flynn, Halifax. Doctor MacKeigan graduated from the Dalhousie Medical School in 1944, and is at present doing post-graduate work in Halifax.

The BULLETIN extends congratulations to Doctor and Mrs. E. F. Ross of Halifax on the birth of a daughter on March 27; to Doctor and Mrs. H. I. MacGregor of Halifax on the birth of a son on March 27; to Doctor and Mrs. J. R. Cameron (Constance MacArthur, R.N., of Pictou) of Middle Musquodoboit, on the birth of a son, Roderick Brock, on April 4; to Doctor and Mrs. W. J. Dyer (Regis Palmer of Hamilton) of Halifax, on the birth of a son on April 15; to Doctor and Mrs. E. P. Nonemaker (Vivian Graham) of Halifax on the birth of a daughter on April 15; to Doctor and Mrs. D. F. Smith of Halifax, on the birth of a daughter on April 17; to Doctor and Mrs. E. L. Ramsay (Anne Jeakins) of Summerside, P.E.I., on the birth of a son, John Leigh, on May 3; to Doctor and Mrs. J. E. Worrell of Halifax, on the birth of a daughter, Mary Christine, on May 5; and to Doctor and Mrs. N. J. MacLean (Dorothy Tobin) of Port Hawkesbury, on the birth of a son on May 17.

Doctor Luther B. MacKenzie, alumnus of Dalhousie University, was honoured by the New York University on April 29th, when he received the Alumni Meritorious Service Award for 1947. Doctor MacKenzie graduated from the New York University School of Medicine in 1904 and from the Dalhousie Medical School in 1902. He was chairman of the Medical Centennial Fund in 1940-1, and the Alumni Hall Fund in 1945-6. In 1940-1 he was president of the College of Medicine Alumni Association.

Late in March Doctor F. R. Davis, Minister of Health, announced plans for establishing a division of neuropsychiatry in the Nova Scotia Department of Public Health. His objective will be the development of a mental health programme throughout the province.

The division will be organized by Doctor C. S. Marshall, specialist in neurology and psychiatry, who has been appointed neuropsychiatrist in the Department of Health. In addition he will be neurologist at the Victoria General Hospital, and assistant professor in charge of neurology at the Dalhousie Medical School. Doctor Davis said the department aims to build up a psychiatric service for the entire province in the treatment of nervous and mental illness.

Doctor Marshall was born in Halifax and graduated from the Dalhousie Medical School in 1924. After a year of general practice at Sherbrooke, Guysborough County, he was awarded a fellowship in neurology and psychiatry by the Rockefeller Foundation. He spent two years in Boston, first as voluntary assistant at the Boston Psychopathic Hospital, and then as resident in neurology at Massachusetts General Hospital.

In 1927 he was appointed provincial psychiatrist for Nova Scotia, and helped organize the Nova Scotia Training School at Truro, an institution for mentally deficient children. He resigned in 1930 to teach and do neurological research at Yale University. For the next eleven years he was a member of the Yale School of Medicine. He returned to practise internal medicine in Halifax in 1941.

Obituary

THE death occurred in Halifax on May 7th of Major Karl Kenneth Blackadar after an illness of two weeks. Born on April 4,1880, in Hebron, Yarmouth County, Major Blackadar graduated from Dalhousie Medical School in 1916, following which he served overseas for two years as medical officer to the Dalhousie unit. Following his return he practised medicine at Mahone Bay and Meteghan. In 1927 he joined the Canadian National Steamships and served as surgeon on various "Lady" boats until the outbreak of the second world war. He married Mrs. Edith M. Day of Bermuda in 1931. He joined the R.C.A.M.C. in 1942, and served at Yarmouth, Aldershot and Halifax. Besides his wife Major Blackadar is survived by his mother, Mrs. J. A. Blackadar of Yarmouth, three daughters, and one brother, John, of the Hawaiian Islands.

The BULLETIN extends sympathy to Doctor B. E. Goodwin of Amherst on the death of his wife, formerly Miss Audrey Fox of Windsor, which occurred on June 2nd; to Doctor C.G. MacKinnon of Halifax, on the death of his mother Mrs. Clarence MacKinnon, which occurred recently; to Doctor J.J.Stanton of Canso on the death of his sister Miss Kathleen Stanton, R.N., who died in Montreal late in March; to Doctor R.F. Ross of Truro, on the death of his father, Mr. Murdock W. Ross, which occurred in Halifax on April 18th, and to Doctor R. E. Mathers of Halifax on the death of his brother, Hon. F.F. Mathers, K.C., who died in Halifax on April 14th.

The death occurred in Antigonish on March 26th of Doctor Malcolm Hugh McKay of West Bay, Cape Breton. Doctor McKay was born at Lake Ainslie, and attended public school in Sydney after which he engaged in the teaching profession for a short period. He attended Queen's University in Kingston where he graduated in medicine in 1899, and practised in West Bay since graduation. Possessed of a captivating personality, he was counsellor to many in every station of life. Apart from his professional duties he always found time for social and community affairs and of a jovial disposition, made a worthwhile contribution to these.

He took a keen interest in public affairs. In the 1920 provincial election he was a Conservative candidate for Inverness County.

Twice married, his first wife, Euphemia MacInnis, B. A., of West Bay, predeceased him in 1919; his second wife, Helen B. MacKay, R.N., of MacLellan's Brook, predeceased him a year ago.

Doctor McKay is survived by four sons and three daughters by his first marriage, and four sisters.

Doctor Ebenezer Henry Archibald, a native of Middle Musquodoboit, died in Vancouver on May 6th, at the age of seventy-one. An alumnus of Dalhousie University, where he was the first student to win the Exhibition Scholarship, Doctor Archibald, for the past 25 years, has been head of the Science Department of the University of British Columbia. After receiving his early education at Truro and Halifax he attended Dalhousie, receiving his B.Sc. in 1897, and his M.Sc. in 1898. After leaving Halifax Doctor Archi-

bald was on the staffs of Syracuse and Harvard Universities; and while at the latter place had the Exhibition Scholarship renewed.

During his last visit to Halifax in the early twenties, coming here to attend a Dalhousie reunion, Doctor Archibald and his daughter Helen were stricken with infantile paralysis. Miss Archibald died and her father was crippled, and for over twenty years conducted his duties at the University from a wheel chair.

He is survived by his widow, residing in Vancouver and one son, Doctor Reginald Archibald, Pr. D., formerly with the Rockefeller Foundation, and now head of the scientific research department of Johns Hopkins University in Maryland. Also surviving are two brothers, R. L. Archibald, Hantsport, and G. E. Archibald, Halifax.
