VANCOUVER MEDICAL ASSOCIATION
1898
NON SIBI SED OMNIBUS

The BULLETIN

of the

VANCOUVER MEDICAL ASSOCIATION

Vol. XV. AUGUST, 1939 No. 10

AUG 8 1939

With Which Is Incorporated
Transactions of the

Victoria Medical Society

the

Vancouver General Hospital

and

St. Paul's Hospital

In This Issue:

COMPULSORY PASTEURIZATION OF MILK

NEWS AND NOTES

BRITISH COLUMBIA MEDICAL ASSOCIATION ANNUAL MEETING, SEPTEMBER 18, 19, 20, 21

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THE VANCOUVER MEDICAL ASSOCIATION

BULLETIN

Published Monthly under the Auspices of the Vancouver Medical Association in the interests of the Medical Profession.

Offices: 203 Medical-Dental Building, Georgia Street, Vancouver, B. C.

EDITORIAL BOARD:

DR. J. H. MACDERMOT

DR. G. A. DAVIDSON DR. D. E. H. CLEVELAND

All communications to be addressed to the Editor at the above address.

Vol. XV. AUGUST, 1939 No. 11

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VANCOUVER HEALTH DEPARTMENT

STATISTICS, JUNE, 1939

Total population—estimated	263,974
Japanese population—estimated	8,891
Chinese population—estimated	7,728
Hindu population—estimated	389

	Number	Rate per 1,000 Population
Total deaths	228	10.5
Japanese deaths	6	8.2
Chinese deaths	9	14.2
Deaths—Residents only	194	8.9
BIRTH REGISTRATIONS:		
Male, 206; Female, 184	390	18.0

INFANTILE MORTALITY:	June, 1939	June, 1938
Deaths under one year of age	5	10
Death Rate—per 1,000 deaths	12.8	29.3
Stillbirths (not included in above)	9	3

CASES OF COMMUNICABLE DISEASES REPORTED IN THE CITY

	May, 1939		June, 1939		July 1st to 15th, 1939	
	Cases	Deaths	Cases	Deaths	Cases	Deaths
Scarlet Fever	10	0	19	0	3	0
Diptheria	0	0	0	0	0	0
Chicken Pox	61	0	28	0	9	0
Measles	7	0	3	0	1	0
Rubella	1	0	0	0	1	0
Mumps	27	0	21	0	4	0
Whooping Cough	112	0	86	0	19	0
Typhoid Fever	0	0	0	0	0	0
Undulant Fever	0	0	1	0	0	0
Poliomyelitis	0	0	0	0	0	0
Tuberculosis	37	15	30	18	11	0
Erysipelas	3	0	1	0	1	0
Ep. Cerebrospinal	0	0	0	0	0	0

V. D. CASES REPORTED TO PROVINCIAL BOARD OF HEALTH, DIVISION OF VENEREAL DISEASE CONTROL.

	Burnaby	West Vancr.	Richmond	North Vancr.	Vancr. Clinic	Hospitals, Private Drs.	Totals
Syphilis	0	0	0	1	58	----	58
Gonorrhœa	0	0	0	1	84	----	84

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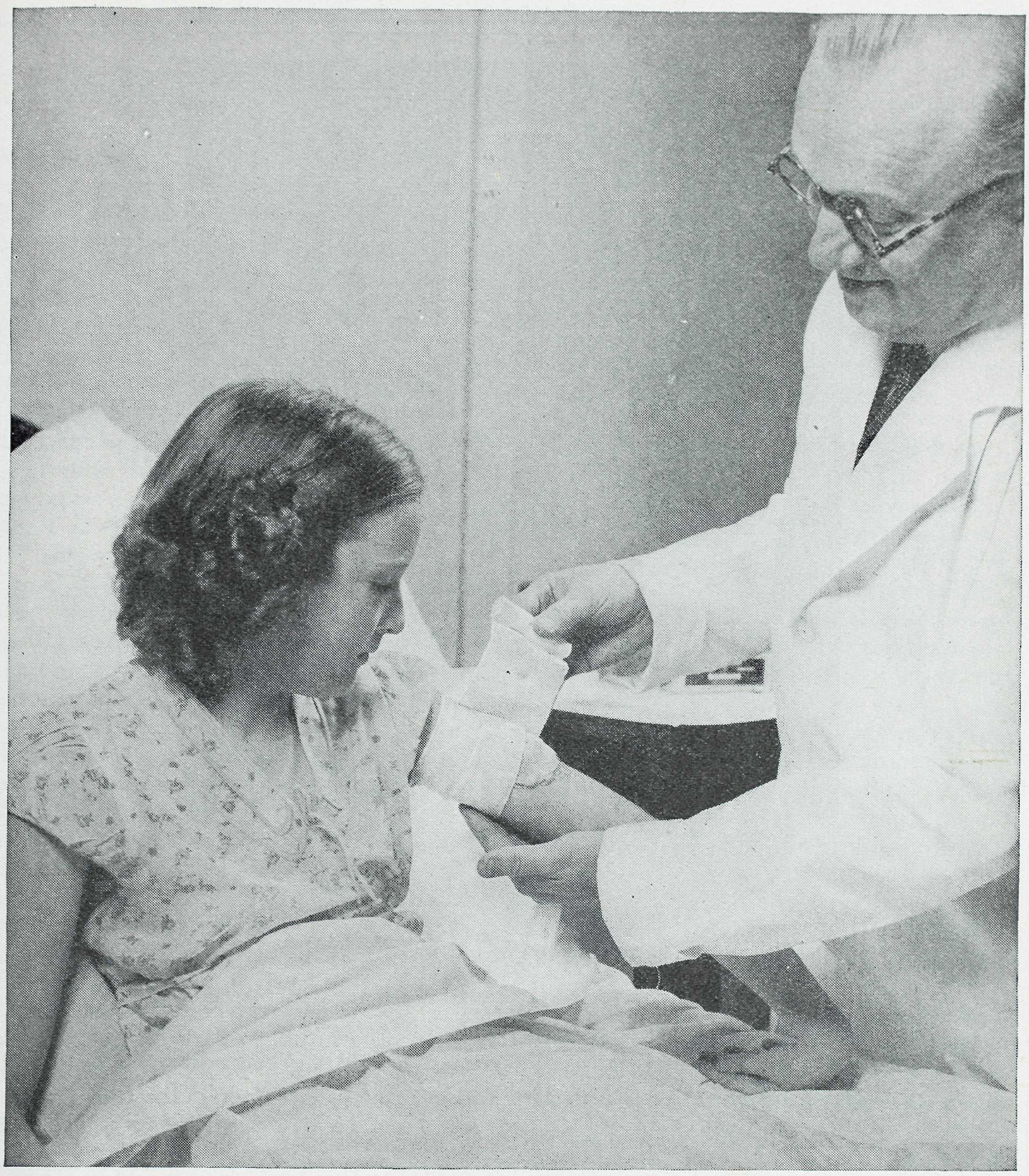

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Sodium Bicarbonate -	*5 grains*

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THE EDITOR'S PAGE

The "next item on the programme" is the forthcoming Annual Meeting of the B. C. Medical Association next month. This bids fair to be a very noteworthy affair. A foreword from the pen of Dr. M. W. Thomas, Executive Secretary, appeared in the July BULLETIN, and there will be more details in this number—but there are two or three matters to which we should like to call special attention.

As our readers know, there are two sides to this meeting—the scientific and the business sides. With the former we shall deal later on this page, but while we shall all derive great benefit from the papers and addresses that have been arranged, we should not forget that this is the opportunity of the year for us to meet as an organized profession, to take stock, to examine into our business affairs, to take counsel together about the future.

There is perhaps a very naural tendency to scamp this duty of ours. We tend to leave it all to the Executive and various Committees, while we go off and play golf, or have a good time listening to a disquisition on the latest work on the acid-ion. This is not altogether fair to our elected officers and representatives. Business meetings are not necessary evils—they are very necessary, and will do us all good, if we will only attend them.

This year they should be especially interesting. As the last Annual Meeting of the Canadian Medical Association, in June, the Council and Executive took the very wise step, after hearing the report of the Committee on Economics, presented by Dr. Wallace Wilson, the Chairman, of appointing Mr. Hugh Wolfenden as Consulting Actuary to the Association, to work with the Committee, and guide them and the Association at large, on sound actuarial lines, and to survey the situation in Canada as a whole.

Mr. Wolfenden needs very little introduction to us in British Columbia. He is perhaps the leading actuary in Canada, and is regarded all over the continent as an unimpeachable authority. He has headed the leading Actuarial Associations in both Canada and the U.S.A. We shall all remember the address he gave on the Financial Implications of Health Insurance last summer; this was reported in full in the BULLETIN. He is scientifically dispassionate in his approach to a problem—and his mental integrity is as far above suspicion as his ability in his chosen profession. We are very fortunate indeed to have such a man associated with us. He will attend the Annual Meeting of the B.C.M.A. next month, will give an address, will be available to answer questions, and will confer at length with the Executive on various matters.

The meeting has been lengthened to four days, on purpose that Mr. Wolfenden may be given ample time to do all these things, and that the membership may have ample opportunity to avail itself of his services. A questionnaire is being prepared for circulation throughout the province, and members are asked to bring with them any questions they may wish to have answered.

A glance at the programme shows an excellent list of speakers. There will be eighteen lectures, and various round table conferences. There will be sectional meetings, as we now have four Sections. There will be clinical demonstrations, a symposium on Public Health, and we hardly like to refer to the Entertainment Programme—for fear that someone might think there will be no time for the Scientific Meeting at all. Provision has been made for the visiting ladies, for Golf, for Luncheons and a Dinner, and, in fact, there is something for everybody. We feel that a perusal of the Programme will make it quite unnecessary for us to urge anyone to come—the man who is unfortunate enough to be unable to do so will have our sympathy and commiseration.

Last, but not at all least, there are the Business Meetings. Reports are being condensed this year, and it will be arranged that they will be disposed of before the meeting. Copies of them will be sent to all the members, so that there will be no need to sit through long wearisome readings of reports, and much time will be saved.

Do not forget, too, that the Annual Meeting of the College of Physicians and Surgeons of B. C. will also be held, and the College will be giving an account of its stewardship at this convention.

NEWS AND NOTES

NEW HOSPITAL FOR VANCOUVER

When St. Vincent's Hospital opened its doors to the general public on July 19th, another milestone in hospital progress was set up.

St. Vincent's is located at Thirty-third avenue and Heather street, and is conducted by the Sisters of Charity of the Immaculate Conception of St. John, New Brunswick. It is ideally located for a hospital, elevated and in a quiet residential section of the city. Every window commands a view of the beauty of Vancouver.

His Excellency Archbishop Duke blessed the building and performed the opening ceremony. Dr. R E. McKechnie, Chancellor of the University of British Columbia, spoke of the need of further hospital beds and extended best wishes. Dr. A. K. Haywood, superintendent of the Vancouver General Hospital offered his congratulations and assured the management of his close co-operation and support.

The key to the building was then delivered to the Archbishop by the architect, Mr. Gardner.

Dr. Edward J. Gray, the chairman, introduced the various officials and speakers.

About 30 doctors evinced their interest in the hospital situation in Vancouver by their presence and some 500 people attended.

The hospital contains 103 beds, which are divided into Medical, Surgical and Obstetric departments. There are private rooms, two-bed semi-privates, and wards limited to four patients. The hospital has all the latest modern equipment. The X-ray is of the latest design and is under the direction of Dr. C. T. McCallum. The laboratory is supervised by Dr. R. Wilson. Dr. Jordan, late of St. Paul's Hospital, is the anæsthetist and resident interne.

The hospital is available to all qualified physicians and surgeons, and during the next few months a medical staff will be organized.

Following a visit through the building, beautiful in its efficient simplicity, the visitors were guests of the Sisters at tea.

* * * *

Dr. J. R. Parmley of Penticton has returned to practice after a bout with the flu and a short holiday.

* * * *

Dr. J. G. McLeod of Blakeburn was a recent visitor to Penticton and called on Dr. W. H. White.

* * * *

Dr. Green of Edmonton has been holidaying at the home of Dr. R. P. Borden of Penticton. Dr. Green is on the teaching staff at the University of Alberta.

* * * *

Congratulations are extended to Dr. and Mrs. N. J. Ball of Oliver on the safe arrival of their first born.

* * * *

We are pleased to report that Dr. D. W. Davis of Kimberley is recovering satisfactorily from an operation, in July.

* * * *

Dr. G. A. McLaughlin is associated with Dr. E. A. Martin in North Vancouver.

* * * *

Dr. D. J. Miller of North Vancouver has returned from a ten day fishing trip in the Cariboo.

* * * *

Congratulations are extended to Dr. and Mrs. W. R. S. Groves of Port Renfrew on the birth of a son.

Dr. F. B. Jordan will be resident physician at St. Vincent's Hospital, Vancouver.

* * * *

We offer congratulations to Dr. John A. Macdonald of Kincolith on the occasion of his marriage on June 5th, to Miss Sybil Berry of Westport, Ontario.

* * * *

Dr. J. H. Moore of Victoria has been holidaying in Hawaii and is expected home shortly.

* * * *

Dr. H. E. Ridewood of Victoria is enjoying a holiday in California.

* * * *

Dr. W. E. M. Mitchell of Victoria is on holiday with the Alpine Club, mountain climbing in the Rockies.

* * * *

Dr. G. B. Helem of Port Alberni returned from his holiday on July 18th. He reports that there is only one place hotter than Rochester, Minn.

* * * *

Dr. Herbert Gale, formerly at the Vancouver General Hospital, is now located at Copper Mountain.

* * * *

Doctors R. McCaffrey of Chilliwack and P. S. McCaffrey of Agassiz were seen chatting with Dr. R. E. McKechnie on Georgia street. The latter appeared to be attempting to enlist the interest of the Doctors McCaffrey in the purchase of the old hotel.

* * * *

Dr. E. Howard McEwen and Dr. S. Cameron MacEwen of New Westminster are on vacation.

* * * *

Doctors J. McNichol and A. W. Hunter found a good fish-pond in the Cariboo last week.

* * * *

Dr. R. D. Coddington of Ocean Falls called at the office when last down.

* * * *

Dr. Eric W. Boak of Victoria was in Vancouver on the day of Mr. Howe's funeral. He called at the office and reported Victoria as a good summer resort.

* * * *

Dr. A. Howard Spohn has left for an extended trip to Europe. He sailed directly for Sweden, whence he will go to Helsingfors, Finland, where he will visit his friend, Dr. Yllpo. Dr. Spohn will also spend some time in England, and hopes to visit in Vienna before returning to Vancouver, about September 12th.

* * * *

Dr. Wallace Wilson, G. F. Strong, A. H. Spohn, D. E. H. Cleveland, R. D. Kinsman and S. Hobbs were among the Western contingent which attended the annual meeting of the Canadian Medical Association.

* * * *

The best wishes of the profession are extended to Dr. A. R. Wilson of Chilliwack, who was married in June to Miss Mildred Paskins, who has been on the teaching staff of the high school in Chilliwack for some years. Dr. and Mrs. Wilson are making their home in Chilliwack.

* * * *

Congratulations are extended to Dr. and Mrs. Russell A. Palmer, on the birth of a daughter on July 22nd.

British Columbia Medical Association

(Canadian Medical Association, British Columbia Division)

President ..... Dr. D. E. H. Cleveland, Vancouver
First Vice-President ..... Dr. F. M. Auld, Nelson
Second Vice-President ..... Dr. E. Murray Blair, Vancouver
Honourary Secretary-Treasurer ..... Dr. A. H. Spohn, Vancouver
Immediate Past President ..... Dr. Gordon C. Kenning, Victoria

Acting Honourary Secretary-Treasurer ..... Dr. Roy Huggard, Vancouver
Executive Secretary ..... Dr. M. W. Thomas, Vancouver

1939 ANNUAL MEETING
VANCOUVER

HOTEL VANCOUVER

SEPTEMBER, 18, 19, 20, 21

FOUR FULL DAYS

Make Your Plans and Reservations Early

SCIENTIFIC SPEAKERS

DR. R. FRANKLIN CARTER, New York City; Associate Professor of Clinical Surgery, Post-Graduate School, Columbia University.
DR. W. G. COSBIE, Toronto; Senior Demonstrator in Obstetrics and Gynæcology, University of Toronto.
DR. H. B. CUSHING, Montreal; Emeritus Professor of Pædiatrics, McGill University.
DR. ALEXANDER GIBSON, Winnipeg; Associate Professor of Clinical Orthopædic Surgery, University of Manitoba.
DR. ROSCOE R. GRAHAM, Toronto; Assistant Professor of Surgery, University of Toronto.
DR. F. S. PATCH, Montreal; Professor of Urology, Head of the Department of Surgery, McGill University.
DR. E. P. SCARLETT, Calgary; Internal Medicine.

REPRESENTING CANADIAN MEDICAL ASSOCIATION

DR. F. S. PATCH, Montreal, President.
DR. T. C. ROUTLEY, Toronto, General Secretary.
MR. HUGH H. WOLFENDEN, Toronto, Consulting Actuary to Committee on Economics, Canadian Medical Association.

MONDAY, SEPTEMBER 18th

8:30 a.m. Registration.
9:30 a.m. 1. Dr. W. G. Cosbie—Cancer of the Cervix.
2. Dr. Roscoe R. Graham—The Surgeon's Responsibility in Cancer.

3. Dr. Frank S. Patch—Renal Infections.
4. Dr. H. B. Cushing—
Indications for and the Results of the Removal of Tonsils and Adenoids.

12:30 Luncheon—Dr. Frank S. Patch, Dr. C. T. Routley, Mayor, President Vancouver Medical Association.

2:00 p.m. Clinical Demonstration—Vancouver General Hospital.

8:00 p.m. Annual Meeting—College of Physicians and Surgeons of B. C.
" " British Columbia Medical Association.

TUESDAY, SEPTEMBER 19th

8:30 a.m. Round Table—Obstetrics, Dr. J. W. Arbuckle.

9:30 a.m. 1. Dr. H. B. Cushing—The Principles of Artificial Feeding of Infants.
2. Dr. E. P. Scarlett—
Angina Pectoris and Coronary Thrombosis, a clinical study of 100 cases of each condition.
3. Dr. R. Franklin Carter—
The Treatment of Cervical Lymphadenitis in Children.
5. Dr. W. G. Cosbie—Maternal Mortality.

12:30 Luncheon—

12:30 Luncheon—Mr. Hugh H. Wolfenden—
"The Development of Health Insurance Throughout the World and Its Bearing on Medical Economics in Canada."

2:00 p.m. Venereal Disease Demonstration—Place to be announced.

8:15 p.m. Medical Economics—Round Table Conference, Mr. Hugh H. Wolfenden.

WEDNESDAY, SEPTEMBER 20th

8:30 a.m. Round Table—Orthopædics—Dr. Murray Meekison.

9:30 a.m. 1. Dr. Alexander Gibson—Fracture of the Neck of the Femur.
2. Dr. Frank S. Patch—
The Importance of Early Diagnosis in Urinary Tract Tumors.
3. Dr. Roscoe R. Graham—
Cæcostomy, a simple and safe measure in diseases of the colon.

12:30 4. Dr. E. P. Scarlett—Peptic Ulcer—New variations on Old Themes.

12:30 Luncheon—Meeting of Board of Directors.

2:00 p.m. Public Health Demonstration—Place to be announced.

8:15 p.m. 1. Dr. Alexander Gibson—Fractures of the Forearm.
2. Dr. R. Franklin Carter—
The Diagnosis of Appendicitis in Children in the Formative Stage. The Results of Our Attempt to Reduce the Mortality in Appendicitis.

THURSDAY, SEPTEMBER 21st

8:30 a.m. Round Table—Nutrition and Gastro-Intestinal Diseases—
Dr. H. A. DesBrisay.

9:30 a.m. 1. Dr. Alexander Gibson—Mechanism of the Spine.
2. Dr. R. Franklin Carter—
Selection of Cases with Gall Bladder Disease for Surgery.
3. Dr. F. P. Scarlett—
Common Fallacies in the Diagnosis of Cardiovascular Diseases.

12:30 Luncheon
Golf

7:00 p.m. Annual Dinner—Hon. R. L. Maitland, K.C., M.L.A.
Distribution of prizes.

SPECIAL FEATURES

The Committee on Programme submits for your approval the proposed programme as above detailed. A close study of the programme will reveal many features which should prove interesting.

GENERAL SESSIONS

The General Sessions include seventeen lectures on well varied subjects.

ROUND TABLE CONFERENCES

Four Round Table Conferences on "Obstetrics," "Orthopædics" and "Nutrition and Gastro-Intestinal Diseases" at 8:30 on three mornings and of one hour's duration.

MEDICAL ECONOMICS

The conference on Economics will be held in the evening—is open to all and should be well attended.

MR. HUGH H. WOLFENDEN

Please note that Mr. Hugh H. Wolfenden, who will address the Luncheon on Tuesday on a subject of interest to every member of the profession, has been retained by the Canadian Medical Association as Consultant to the Department of Economics.

Mr. Wolfenden is attending the annual meetings of the Provincial Associations in the four western Provinces. He will be prepared, at the Round Table Conference on Medical Economics on Tuesday evening, to deal with selected subjects. His reputation precedes him and we predict that he will make a valued contribution to these sessions dealing with economic questions.

A questionnaire has been circulated to the members requesting submission of questions which they wish discussed at the four Round Table Conferences. It is urged that the members avail themselves of this opportunity to assist in providing a programme of their own choosing.

CLINICAL DEMONSTRATIONS

Monday afternoon will be devoted to Demonstrations held at the Vancouver General Hospital. These have proven a popular feature of our Annual Meetings.

PUBLIC HEALTH

An open session for free discussion of the programme of Preventive Medicine of the Departments of Health will be held on Wednesday afternoon. This should be attended by all. It will be confined to *Doctors Only* so that there may be untrammelled interchange of opinion.

SPECIAL FEATURE contributed by DIVISION OF VENEREAL DISEASE CONTROL

Dr. D. H. Williams, the Director, has secured an outstanding film which will be shown in one of the motion picture theatres. This will be open to the whole convention. Further announcements later.

OFFICIAL LUNCHEON

On the first day, September 18th, the Official Luncheon will be devoted largely to the Canadian Medical Association when, following the welcomes by His Worship the Mayor and the President of the Vancouver Medical Association, Dr. Frank S. Patch, President, and Dr. T. C. Routley, General Secretary, will address the members. The Official Luncheon will be set up for a large gathering.

SPECIAL LUNCHEON

On the second day, September 19th, Mr. Hugh H. Wolfenden will be the chief speaker. See programme above.

MATERNAL WELFARE

The Committee, of which Dr. C. T. Hilton is the energetic chairman, will interest itself in the Round Table Conference conducted under the leadership of Dr. J. W. Arbuckle, and in the special lectures provided by Dr. W. G. Cosbie. The Committee will meet during the Annual Meeting.

ANNUAL MEETINGS

The Annual Meetings of both bodies—the College of Physicians and Surgeons of B. C. and the British Columbia Medical Association—will be held on Monday evening. Important business will be presented to the profession at both meetings and a large attendance is predicted in that matters ofinterest to each member will be dealt with.

ATTENTION! CHAIRMEN OF STANDING COMMITTEES

Reports of all Committees must be handed in at the office before August 10th. This year it is proposed to publish and circulate the reports prior to the Annual Meeting. This should expedite business.

ARRANGEMENTS FOR ANNUAL MEETING

Chairman of Committee on Programme and Arrangements—Dr. G. F. Strong.
Chairmen of Sub Committees:

Registration and Reception—Dr. H. H. Milburn.
Publicity, Press and Publication—Dr. J. H. MacDermot.
Arrangements—Dr. Roy Huggard.
Clinical Demonstrations—Dr. R. A. Palmer.
Entertainment—Dr. J. R. Naden.
Golf—Dr. J. P. Bilodeau.
Transportation—Dr. J. R. Neilson.
Commercial Exhibits—Dr. W. T. Ewing.

ANNUAL DINNER

The Annual Dinner will be held in the Banquet Room in the (new) Hotel Vancouver. The Honourable R. L. Maitland, K.C., M.L.A., will be the guest speaker. Golf prizes will be distributed at this closing function.

GOLF

The British Columbia Medical Association Trophy will be again competed for. The past holder was, 1937, Dr. D. Fraser Murray; the present holder is, 1938, Dr. G. R. F. Elliot.

Dr. J. P. Bilodeau is Chairman of the Committee on Golf and that means that the arrangements will be satisfactory. Joe seems to have a way of providing prizes for everyone. It is important that you return your golf registration card which you will receive sometime before the Annual Meeting.

COMMERCIAL EXHIBITS

During the past two years our many friends who deal in pharmaceuticals, electrical and X-Ray equipment and surgical supplies have supported loyally our Annual Meeting as exhibitors and contributors. This is much appreciated and we again ask the membership to reciprocate by showing interest in our commercial friends.

PROGRAMME OF ENTERTAINMENT FOR THE LADIES

Doctors are requested to inform their wives that an attractive programme for the entertainment of visiting ladies has been developed by the local members of the Ladies' General Committee. The ladies always enjoy themselves at our meetings and we are glad to have it so. We find that some ladies bring their husbands.

TEA

Tuesday, September 19th, at 4:00 o'clock. Tea for all ladies to be held at the Jericho Country Club.

SCENIC DRIVE

The Tea will be preceded by a drive for the visiting ladies, cars leaving the hotel at 2:00 o'clock.

LADIES' DINNER

A capable and enthusiastic Committee is already arranging for the Annual Dinner at 7:00 o'clock, Thursday, September 21st. Ample accommodation will be provided and it is hoped that the dinner will again be largely attended.

LADIES' REGISTRATION

The ladies are asked to register. A Reception Committee will be in attendance at the hotel and will be happy to assist the visitors and give information regarding the many things that lady-visitors to Vancouver will be interested in.

Public Health Section

[We are glad to have the opportunity of publishing this brief, but very succinct, address of Dr. Dolman's. It was broadcast recently as part of a programme given by the Greater Vancouver Health League.

We think our readers will gladly have at finger's reach this summary of the arguments that are constantly being brought up *seriatim* by those who, for one reason or another, attack compulsory pasteurization of milk. That word "compulsory" seems to stir the hackles on most of our necks, though we cheerfully submit to all sorts of compulsion daily —but it has always seemed to us that this resentment is the only reason one *can* have for objecting to this most necessary measure for protection of the public health, and it is not a very worthy or at all well-founded reason. Dr. Dolman takes up each objection in turn, and answers it, as we think, irrefutably.

Speaking for ourselves, we are glad to have this to refer to. It is not always easy, off-hand, to find an adequate answer to objections that we feel are unreasonable and mistaken, and a *vade-mecum* of this sort will, we feel sure, prove to be of great value. While it was addressed primarily to a Vancouver audience, it applies everywhere in the Province, and so we felt that it should be printed, so as to reach all those who might find it useful.—Ed.]

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COMPULSORY PASTEURIZATION OF MILK

C. E. DOLMAN, M.B., B.S., M.R.C.P., D.P.H., PH.D.

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Everybody knows that cow's milk is an excellent food. It is easily digested, a cheap source of bodily energy, rich in certain vitamins and in those calcium salts which are so necessary for tooth and bone formation. Your family doctor, and public health officials, have been telling you this for some years now; articles in the weekly and monthly magazines, and the daily press, have urged you to drink more milk; while even the milk producers and vendors (whenever they can spare a few moments from their curious pastime of squabbling amongst themselves) will unite in extolling the health-giving virtues of their product. So I shall not take up time urging you to drink *more* milk. But I do want, no matter how much or how little milk you consume, to persuade you to drink only *safe* milk.

The need to stress the safety of milk supplies arises, of course, from the fact that milk is an excellent food, not only for human beings of all ages, but also for many species of bacteria, including those micro-organisms which produce such human disease as tuberculosis, typhoid and paratyphoid fevers, dysentery, septic sore throat, and undulant fever. I do not contend for a moment that these diseases are always milk-borne in origin. But it is certain that bacteria capable of causing these diseases in man may gain access to cow's milk, either directly from an infected cow, or indirectly through the milker of the cow. It is equally certain that, once present in the milk, such microbes will survive and multiply therein, unless the milk be pasteurized or boiled; and it has been established beyond a doubt that numberless lives have been lost, and still are being lost, in epidemics due to consumption of raw cow's milk containing disease-producing microbes. Moreover, there is emphatically no evidence of a milk-borne epidemic ever having been due to properly pasteurized milk. If all this be true (as indeed it is), does not the question of compulsory pasteurization of all milk supplies clearly merit dispassionate examination?

I mentioned just now that boiling would kill disease-producing microbes in milk. But boiled milk is in certain respects inferior to pasteurized milk, and pasteurization of all milk supplies before delivery is advocated in preference to boiling after delivery. If you cannot get pasteurized milk in your district, then boiling it at home is a wise precaution.

Just what is meant by pasteurization? It is a process, named after the great Louis Pasteur, whereby milk is rapidly brought to a temperature of 145 degrees Fahrenheit, held there for thirty minutes, then rapidly ccoled, and filled, under aseptic conditions, into sterile containers. When properly performed, pasteurization does not significantly reduce or impair any component or property of milk which is of importance to health. Let us consider for a moment some of the fantastic accusations levied against pasteurization by many of the raw milk producers and consumers.

There are some twelve points commonly raised in defense of raw milk, and I intend to refute each one of them. While certain of these claims are intrinsically of a trivial nature, they may loom large in the minds of otherwise quite unprejudiced persons. For instance, pasteurization is said to change the cream line in the milk bottle. But the cream line is much more affected by such variables as the breed of cow yielding the milk, and the time elapsing between bottling and delivery, than it is by pasteurization. In any event, the cream line is not a reliable index of the cream content; and even if it were, on should remember that more than half the full value of cow's milk, and nearly all its body-building properties, are retained by the generally-despised "skim" portion. The significance of the cream line is a fallacy deriving from the relatively high commercial value of the butter-fat content of milk.

A second claim often made is that pasteurization spoils the flavour of milk. Now, in recent years at the University, a large class of Bacteriology students has taken part in an annual tasting competition. The class results have always shown that raw milk cannot be differentiated from pasteurized on the basis of flavour alone. Similar tests conducted elsewhere, on a larger or smaller scale, have given similar results. I suggest, if you are addicted to raw milk because of its taste, that you try out a similar experiment in your own household, with an unprejudiced neighbour as referee. Some raw milks have indeed a special flavour and aroma; but this is due, as a matter of fact, to absorption of stable smells; and can be restored to a sample of pasteurized milk by addition of a drop or two of an infusion of cow manure.

Thirdly, you may have been told that pasteurized milk rots on keeping, whereas raw milk merely sours. This statement is false. Whether a milk sours or putrefies on keeping depends upon the relative numbers of souring and of putrefying bacteria initially present. Thus, if putrefying bacteria preponderate in a raw milk sample (as they may do) it will rot on keeping; while if souring bacteria preponderate in a pasteurized milk sample (as they usually do) it will sour on keeping. Kept under parallel conditions, pasteurized milk remains sweet longer than raw milk. If kept long enough, either raw or pasteurized milk will end in putrefaction, just as does meat, and many another foodstuff.

Fourthly, the vitamins in milk are often said to be destroyed by pasteurization. Now milk is an excellent source of vitamin A, and contains useful amounts of vitamin G. The vitamin A content of milk is absolutely unaffected by pasteurization, while the vitamin G content is reduced only very slightly. True enough, pasteurization may destroy up to 20 per cent of the vitamin C content of milk; but of what moment is this when, as every modern mother knows, even raw milk, even mother's milk, is an inadequate source of vitamin C for infants, who depend upon orange or tomato juice for their main supplies of this vitamin.

Fifthly, the valuable calcium and phosphorus content of milk is allegedly reduced, or rendered unassimilable by pasteurization. This contention is categorically untrue; and even if as much as one-half the calcium salts in cow's milk could be rendered unabsorbable by pasteurization, such milk would still contain nearly twice as much calcium as does human milk.

Sixthly, you may have heard that the general nutritive value of milk is impaired by pasteurization, the argument having perhaps been illustrated by a pathetic picture of an emaciated calf with the caption "Fed on Pasteurized Milk" alongside a portrait of an

extremely robust bullock entitled "Fed on Raw Milk." The words "Posed by Professional models" are carefully omitted. The truth is that numerous studies at first-class universities have established beyond a doubt, that even calves thrive better on pasteurized cow's milk than upon the raw product.

Seventhly, much noisy argument has centred around the point that pasteurization encourages the pooling of dirty and of clean milk. This argument misses the real point, that only pasteurization will render *any* milk *safe*. Five hundred typhoid bacilli in a quart of milk from a clean environment are immeasurably more dangerous than 500 million harmless bacteria in a quart of milk from an æsthetically dirty source; and pasteurization alone could remove the menace to public health of the former sample. Moreover, under a system of compulsory pasteurization, the usual channels of advertisement would still be open to the enterprising and conscientious dairies which really had fine herds, barns, and equipment to place before the public eye.

Eighthly, it is claimed that compulsory pasteurization is not feasible, and that no means are available for ensuring its proper carrying out. But many cities in the United States have enjoyed the benefits of compulsory pasteurization for years; and in the city of Toronto, for example, these benefits have been so impressive that the Provincial Government has enacted province-wide compulsory pasteurization. Why should British Columbia deny itself the benefits which Ontario will reap from this policy? As for checking on the pasteurizing plants, a very delicate test is available for determining whether milk samples have been properly pasteurized; and such tests could be regularly performed by your Provincial Laboratories.

The ninth argument to be refuted is that there are better alternative methods available, such as tuberculin testing, for ensuring a safe milk supply. But even if the tuberculin test were being done regularly, and were fool-proof enough to ensure elimination of all tuberculous cattle from dairy herds, tuberculosis is only one of many milk-borne diseases. For instance, local attempts to eliminate the risk of raw milk-borne undulant fever by means other than pasteurization recently proved a complete failure. Moreover, even if a perfectly healthy herd could be secured by some culling process, pasteurization would still be required to guard against contamination introduced between milking and delivery.

The tenth untruth is that expert opinion is divided as to the desirability of compulsory pasteurization. Yet this measure has been officially recommended not only by the Vancouver and B. C. Medical Associations, but also, among others, by the Canadian and the American Medical and Public Health Associations, the United States Public Health Service, the Ministry of Health of Great Britain, and the Health Organization of the League of Nations. What right has any layman to pit his prejudices against the united opinion of such authorities?

The eleventh point to dispose of, an eleventh-hour plea, as it were, from the raw milk addicts, is that the conscientious objector to pasteurization should be permitted his daily quart of raw milk. But in matters affecting the public health so closely as does this question of a safe milk supply, there ought to be no compromise. All raw milk distributed represents a potential menace to your health, and to your children's health; while most of Vancouver's raw milk is repeatedly exposing those who drink it to a definite risk of contracting undulant fever.

The twelfth and last contention of the proponents of raw milk is that pasteurization is a costly process, and increases the price of milk. How odd, then, is the fact that most raw milk sells in Vancouver today for at least 11 cents a quart, while the just as nutritious, just as palatable, and much safer pasteurized product sells for a dime.

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Vancouver General Hospital

CONVULSIONS IN INFANCY AND EARLY CHILDHOOD

Dr. G. O. Matthews

A good deal of clinical research, as well as considerable animal experimentation, has been devoted to the study of convulsions, and this work has brought us a certain small, although far from complete, understanding of the convulsive state. Although the entire mechanism of the seizure and the whole truth regarding the precipitating factors is not known, certain features are now generally accepted.

According to Kennedy and Foster,[1] most convulsions are probably initially due to a cerebral anæmia, which is the tonic stage of the seizure, and which is quickly followed by a cerebral congestion and œdema which gives us the clonic stage of the seizure. This statement is well borne out by the work of Leonard,[2] who showed that pressure on the carotids and vagi in humans produced clonic spasms and loss of consciousness, and Pike finally showed that cutting off the cerebral and medullary circulation in cats caused unconsciousness and tonic convulsions, whereas a sudden return of the circulation gave clonic seizures. Direct observation by Forster, during a convulsion, of the human brain exposed at operation, showed that just prior to the attack the brain shrinks in volume and becomes pale, and then later there is an enormous increase in brain size, accompanied by cyanosis.

The significance of cerebral œdema in epilepsy has been known since the time of Hippocrates and has been substantiated over the past fifteen or twenty years by half a dozen different investigators. The knowledge of the œdema has been used with some success in the dehydration treatment of epilepsy by Dandy and later by Peterman."

Possibly the successes of the ketogenic diet may, partly at least, be due to the dehydration it produces. Certainly Wilder[4] of the Mayo Clinic showed that the ketosis *per se* was not the only successful factor. However, the peculiar susceptibility of the idiopathic epileptic to the periodic recurrence of his seizures, which are usually of the same character, has not as yet been adequately explained. In other words, although we know that certain definite and known changes occur within the cerebral cavity prior to and during a seizure, we do not know just what the stimuli may be that cause these changes. Whatever these stimuli are, we must not conclude that they alone are always capable of producing an attack, as the *individual susceptibility to convulsions varies greatly.* Whether a typical seizure will result depends on the height of the so-called "convulsive threshold," which varies tremendously in different individuals and at different times in the same person.

The high incidence of convulsions in childhood, particularly between the ages of six and thirty-six months, has been the subject of much speculation. A great deal has been written about this very striking susceptibility in young individuals and their low threshold to convulsions. It is manifest not only in functional conditions of a toxic or infectious nature, but also, *to just as high a degree,* in organic lesions. For example: First, the convulsion that ushers in lobar pneumonia in a child is so common that it can almost be likened to the initial chill in an adult; second, in juvenile paresis or in meningitis, convulsions are much more common in the child than in the adult.

There are many theories as to why this is so, such as "incomplete development of the cortex," or "incomplete myelization of the nerves," etc., but all one can say, apparently, is that the convulsive threshold in infants and young children stands at a lower level than in the adult, and we can accept the view that any external factor or any pathological lesion which may bring on a convulsion is more likely to do so in a child than in the corresponding adult. Certain authors regard this view as paradoxical, but they, I think, fail to distinguish between convulsions and susceptibility to convulsions. They feel, apparently, that this question of increased susceptibility in early life was partially solved by the finding of an extremely high incidence of birth injury with some slight cerebral hæmorrhage, as pointed out by Ylppo and Swhartz, as well as more recently by Howard Smith of Portland.

It is quite true, then, that this work on birth injury cleared up the cause of a goodly number of infantile convulsions that has been obscure, but at the same time this fact of a high susceptibility in childhood cannot be solved by merely pointing out a new high incidence of one type of organic lesion.

This point is well summarized by Peterman, who says: "A convulsion at any age is a pathological episode; but in young children, because of the late and slow development of their central nervous systems, there exists a physiological predisposition which makes it easier for a convulsion to appear in response to a given cause."

It does also happen that the infant and young child are subject, because of their age, to a great many diseases which in themselves are or can be the exciting factor of a convulsion. When this age period is past, the incidence of convulsions is greatly lowered. To me, then, there must be two definite and distinct factors. First, an increased susceptibility, and, second, a greater number of possible exciting factors. Certainly it is not alone the incidence of infections or febrile diseases, as later in life no convulsions accompany them. As to why febrile diseases cause seizures, Peterman feels, and no one else has expressed any diverse opinion, so it may be said to be unanimous, that thse convulsions are probably due to a response of the patient to an invasion of the blood stream or meninges by micro-organisms, or to an upset in the water balance which so many infections produce (this might cause the initiating cerebral œdema).

So much for the cause of convulsions. To repeat: The cerebral pathology prior to and during the seizure is pretty well known and the exciting factors can be enumerated, but it can only truly be said that a child is such an easy prey to a seizure *just because he is a child*.

Convulsions can be best classified under two main groups.

First: Convulsions of organic origin such as occur in acute or chronic disease of the central nervous system.

Second: Symptomatic convulsions—and this group must be subdivided into two divisions: (a) Incidental or functional seizures such as accompany febrile diseases or such as psychogenic seizures (here I mean seizures in psychopathic children from very little cause); (b), convulsive diseases, namely, idiopathic epilepsy or tetany.

Peterman in 1934 gave us a very practical study on the cause and types of convulsions in childhood. He carefully analyzed five hundred cases. First of all, he took a very careful history, paying particular attention to the birth and to the new-born period, and to any familial incidence of seizures in other members of the family.

His cases were all hospitalized and thoroughly investigated. All had Kahns done, blood calcium and phosphorus estimated, X-rays taken of the skull, and stool and urine examination, plus, of course, a careful physical examination. Spinal fluid was examined in all except those of definite spasmophilia. Encephalograms were done in doubtful cases.

The most striking part of this study to me was the fact that he made a definite diagnosis in 94% of all his cases. He also found that there are certain diseases peculiar to childrood that are the cause of most of the convulsions (measles, streptococcic infections). While he found that certain children may be said to be particularly susceptible to seizures, there is usually a physical basis for this susceptibility (such as a lowered blood calcium) and one which may be amenable to treatment.

Every convulsion produces a certain amount of cerebral injury, and Peterman thinks that one seizure increases the child's susceptibility. This I heartily agree with, and for this reason I feel that each individual convulsion must be given a very painstaking and careful study in order to prevent a recurrence if possible. Only such a course is adequate treatment.

It is unfortunate that even today one finds occasionally an older physician who considers convulsions a more or less necessary evil of childhood, and who merely advises the parents that the child will outgrow his tendency. Such a child probably will, but just as a succession of blows spoils the sharp edge of a knife so a succession of convulsive seizures may irreparably impair a child's mental alertness. Although such trivial things as worms or teething may set off a seizure, they are *never* the real basic cause, as in all cases some underlying factor such as a mild spasmophilia can be proven to exist if searched for. The thymus, whatever it may do, never causes convulsions.

Convulsions in childhood are just as serious as in adults and there is no justification in minimizing the symptom because of age, or in believing that time alone will remove the cause.

I give you herewith a short abbreviation of Peterman's classification of his 500 cases:

Idiopathic Epilepsy	34.4%
Acute Infections	21.4%
Spasmophila or Tetany	16.0%
Cerebral Birth Injury or Sequelæ	13.1%
Cause unknown	6.7%
Perhaps hyper-or hypo-glycæmia, perhaps allergy with a resultant brain oedema.	
Miscellaneous	8.4%
Acute Gastro Enteritis.	
Traumatic Brain Injury.	
Pertussis with slight cerebral hemorrhage.	

Naturally this percentage classification will vary greatly in the different age groups. For example: From birth to 1 month, over 50% of seizures were due to birth injury or hæmorrhagic disease; from 1 month to 6 months, only 20% were due or incidental to birth, 30% were infectious and 16% spasmophilia; from 6 months to 36 months, infections accounted for 30%, spasmophilia 23%, and epilepsy was now the cause of 20%; from 3 years to 10 years, epilepsy was the cause of 60%.

It is to be remembered that some time may elapse, months or even years, before a case of birth injury will have its first seizure, but such convulsions will often be of the localized Jacksonian type and a careful history of the new-born period will give valuable diagnostic help. Very often, too, as you know, these babies will have other signs of birth injury.

Idiopathic Epilepsy

This is a convulsive disease, the essential feature of which is its gradual but persistent progressiveness to which exceptions are rare. Once an epileptic, always an epileptic, is practically true. The onset very often occurs in early childhood. There are statistics that show from one-third to one-half of all adult epileptics suffered from convulsive seizures in their first year of life. On the other hand, Peterman's series showed only a mere 5% under 2 years that were true epileptics, and I think this work carries a great deal of weight. It is, however, fair to say that idiopathic epilepsy is no great rarity in early childhood, but certainly no convulsive seizure in a child under four should be labelled epilepsy without a most painstaking and thorough search for a more likely cause.

There are, of course, the two types of seizures—*grand mal*, with or without aura, injury during the fall, biting of the tongue and fixation of the pupils, and the minor *petit mal* attacks which, as you know, are mere lapses lasting only a few seconds.

Sometimes various forms of seizures occur in the same patient; for example, after a *grand mal* attack the child may go for months with merely a few *petit mal* spells, or vice-versa, but in general *petit mal* attacks will come more frequently than *grand mal*, and the great majority of *petit mal* cases will, if untreated, and unfortunately all too often even if treated, be replaced eventually by typical *grand mal* seizures.

Attacks can and often do occur at night when the patient sleeps, and so are often missed or misinterpreted. Perhaps certain older children who had been quite trained and then begin to wet the bed periodically may in reality be epileptic, and the bed wetting incidental to the seizure.

Findings on physical examination are meagre. In fact, if there are any positive neurological signs they probably rule out epilepsy as the diagnosis. A small group of children have been reported to have periodic recurrences of nightmare. These children would awake screaming and in a confused state, and the so-called nightmares were gradually replaced by typical epileptic seizures.

I do not for one minute mean to imply that many of our bed wetters, or children having nightmares, are epileptics, but only that, when such histories are related to you, epilepsy should at least be thought of.

The mental deterioration suffered by epileptic children depends a great deal more on

the frequency of the attacks than on their severity. In fact, mentality is only slightly affected unless the seizures are quite frequent.

The prognosis of epilepsy in a child is, of course, very grave; and the nature of the attacks is no measure of the course it may take. Indeed, the outlook for true *petit mal* is just as bad as for *grand mal,* and an optimistic outlook, no matter what the type, and no matter what the treatment, is unwise.

However, although it is true that no other convulsive state has such a poor outlook, it is also and equally true that there is no other condition where errors of diagnosis can so easily be made. In the worst age group only 60% of all convulsions can be epileptic.

Symptomatic Convulsions

Here we are dealing with seizures that are similar to *grand mal* epileptic attacks and which occur in connection with many causative factors, but which never lead to a true epileptic state, and which at most may reappear due to the original cause or a similar one.

The most frequent and common convulsions of this type are those which precede or accompany the onset of an acute infectious disease. As these convulsions so often occur at the beginning of the prodromal period, their significance is often obscure for several days.

Just what the exact mechanism of these initial convulsions may be is not known. Many of them, of course, are tetanic in origin, the metabolic changes that occur as a result of the illness being sufficient to change a latent tetany phase into a manifest one. We know how easily this can be done, as so often we find a blood calcium of 8 or even 9 in a child that has suffered a series of seizures from some comparably minor ailment. Such a blood calcium is not greatly lowered, but it is down sufficiently to give a lowered convulsive threshold and almost any upset may start them off. It is this type of thing that Peterman stresses so strongly and which he found present in the great majority of his series that had convulsions from such initiating factors are worms, constipation, teething ,etc.

Apparently a rise of fever, as such is not the exciting factor, as many children will have a full-sized convulsion with moderate fever, and then, a week or two later, no seizure accompanying a much higher fever. During the course of febrile conditions, such as pyelitis or influenza, convulsions may occur, but not nearly so frequently as at the onset of the infectious diseases or lobar pneumonia. In pertussis, also, convulsions occur, but here they are seldom purely symptomatic or tetanic, but more often caused by minute cerebral hæmorrhages due to spasmodic coughing. Terminal convulsions in young children often occur a few hours before death, usually accompanied by hyperpyrexia. In these cases, at autopsy little is found except cerebral œdema, and only perhaps something such as a small bronchial pneumonia, or even nothing, to account for the fever. I have lately had two cases that died within twenty-four hours of the onset of their illness—both had fever of 109° towards the end, and had convulsion succeeding convulsion right up to their death. Their postmortem examinations were almost identical. Well-nourished, well-developed babies with minute signs of early bronchial pneumonia and extreme brain œdema. Their infection was very overpowering, but later I hope to point out how somewhat different treatment might have helped put out even these two wild fires.

Differential Diagnosis of Convulsions

No matter what the age, it must first be decided whether the seizure is organic or symptomatic. In the first few days of life, even in the first few weeks, the great majority of convulsions are, of course, organic, from birth injury or hæmorrhagic disease. After this new-born period, the age group is of only relative help.

Seizures of the Jacksonian type are, of course, organic and relatively simple of diagnosing, but, in my own experience, seizures of this type in early childhood are extremely rare. I may be open to correction, but I feel that, in young children, even the definitely localized lesion is more apt to give a generalized seizure than a true Jacksonian one, perhaps because of the diffuse œdema that is usually present.

Tetany is uncommon under 6 months, although it may even occur in the new-born.

As the child becomes older, the percentage of seizures that are epileptic increases and at puberty that percentage reaches its maximum of 60%. I wish again to stress the important fact that at no time in childhood can the stigma of epilepsy be attached to more than half of all children having even periodic convulsions.

However, a history of recurrent seizures during both summer and winter in a child on good dietary regime, whether or not there is any family history of epilepsy, makes one very suspicious, and if the attacks last for four years, or if they cease only to recur after an interval of years, in all probability that child is epileptic. It may also help if it is remembered that in true epileptic seizures the pupils are fixed and widely dilated and that the attacks often occur during sleep.

There is also, of course, the defective child—the Mongol, the microcephalic idiot, the low-grade moron—or perhaps merely the child with an extremely low intelligence quotient. These children have convulsive seizures very, very easily, but even here the attack needs some trigger to start them off. Mild fever, severe scoldings, even frustration may be sufficient cause, as, although not due directly to the psychopathic state, such children have an extremely low convulsive threshold. Such children can be spoken of as a primitive type and are inclined to respond to their surroundings by explosive reactions, because they lack entirely or in part the power of thoughtful or deliberate reasoning. To a lesser degree the so-called neurotic child, living in all probability in a high tension atmosphere, is so likely to explode into a temper tantrum or even a convulsion from very little cause. Fortunately, such types as a rule are easily recognized, and as these neurotic youngsters have a lowered blood calcium so much more often than the normal, placid child who is in tune with his surroundings, calcium therapy is sometimes helpful.

There is little that is new in the treatment of convulsions. Its fundamental basis is threefold:

1. Prevent harm to the patient.
2. Stop the seizure.
3. Find the cause and prevent a recurrence.

A convulsion is always, in the eyes of the family, an immediate and urgent emergency. There is nothing that will upset and frighten the ordinary family more than a real generalized convulsive seizure. Usually they come out of a more or less clear sky without warning, and although we know that most convulsions will cease by themselves in a comparatively short time, advice and orders that are clear and didactic, and that will keep all the members of the family busy as well as prevent injury to the patient, must be given over the telephone. Don't forget the stick between the teeth. This telephone advice must, as I have said, prevent further harm to the patient, but it really won't help very much in terminating the seizure. The usual things, such as the hot bath, perhaps with mustard, and the enema, do no harm, and it is good to keep everyone busy until you arrive fifteen or twenty minutes later. As soon as the seizure has ceased, its cause must be determined and, of course, further convulsions prevented.

In my opinion the one drug of choice for this purpose is phenobarbital; give it freely in large doses, by mouth if possible, or if necessary by hypo in the form of the sodium salt. It will usually work. I have had no occasion to use the bromides, paraldehyde or chloral hydrate, as, if luminal won't work, neither likely will they.

Magnesium sulphate, of course, is very valuable. It may be used in various dilutions intramuscularly, has a strong depressing action, and will perhaps in some cases work faster and more surely even than phenobarbital. It must be always remembered, however, that magnesium sulphate should not be used in conjunction with sulphanilamide therapy.

If a convulsion does not, however, subside within a short time—say thirty minutes—I feel that chloroform should be resorted to more often than is customary, for two reasons: First, the longer the seizure the more possibly cerebral damage will result; and secondly, and perhaps more important, until the seizure is terminated its cause can hardly be diagnosed, and it is also equally difficult to treat the fundamental cause adequately during a convulsive state.

As previously stated, it has been my recent experience to find at autopsy, in several children dying soon after severe convulsive seizures, an almost unbelievable amount of cerebral œdema. They were in all cases children with high fever, but the cerebral œdema was practically the only autopsy finding. They had not been ill long, and undoubtedly suffered from an overwhelming infection of some sort, and it may well be that this œdema resulted from the infection and was the initiating cause of the seizures and the hyperpyrexia rather than the result of them. I am not satisfied however, that had I controlled

the convulsions quicker (and they were either uncontrollable or very slow in being controlled with drugs, as can always be done almost immediately with chloroform), and then instituted hypertonic intravenous therapy, at least a percentage of these children might not have lived. Therefore, in future, I intend to use chloroform much sooner than I have done in the past and, where diagnosis is obscure, follow its quick sedative action with hypertonic intravenous therapy.

Of all our diagnostic aids I resort last to the lumbar puncture. The history and type of convulsions will usually tell whether or not it is indicated, and it is seldom of much value in merely controlling the seizures. If time is taken for lumbar puncture, valuable time may be lost in doing other things. Certainly I do not now feel, as I once did, that all convulsive cases must have or need an early lumbar puncture either for diagnosis or for treatment. This applies most particularly, perhaps, to the new-born baby who is twitching, but to older children as well. Instead of running for the lumbar puncture needle in these new-borns, run for the blood syringe. This opinion is far from unanimous, early drainage, etc., but I think the less handling the better—sedatives. Later, in two or three days, if the baby is still alive and your bleeding has stopped, is the proper time, in my opinion, to do the lumbar or cisternal taps.

As the treatment of epilepsy, I wish merely to emphasize one or two points. Here, again, I feel that phenobarbital is the drug of choice. It needs to be given in big doses, sufficient to control the seizures, and continued over a long period of time. The child must be kept free from infections and there must be no dietary excesses. They should lead a calm and regular life, free from excitement and all over-stimulation. Children's parties and movies are much less suitable for the epileptic than for the normal child. In my own limited experience, phenobarbital is more effective in preventing *grand mal* attacks than *petit mal* lapses. I have no experience of mebaral, which is supposed to be as effective as luminal and not so stupefying. The ketogenic diet is unquestionably of great value, but outside of hospital is extremely difficult to maintain. Not only is it expensive, but in my few attempts with it I have had to contend with gastro-intestinal upsets from its high fat content. When it is used, it must always be preceded by a two to three-day starvation period and fluids must be limited to 1000 cc. per days.

In ordinary practice the epileptic is well handled if the following rules are followed:

First: Restrict carbohydrate and protein somewhat and give as much fat as the child and parents' pocketbook will stand. Second: Limit fluids to 1000 cc. per day. Third: Phenobarbital in sufficient dosage to control seizures.

Lastly, a paper such as this should not omit mentioning the fact that certain poisons may in children set off convulsions. Such substances as santonin, atropine, strychnine, oil of chenopodium, camphor, bad mushrooms, phosphorus, lead, and lastly, arsenic, can all initiate severe and lasting seizures. I remember very vividly a child that had extremely severe and prolonged convulsions from arsenic, which was being given in the course of anti-luetic treatment. The child was aged seven, and syphilis was only suspected because of keratitis, so I feel sure that the seizures were due to the treatment and not to the disease. These convulsions were finally and dramatically relieved by the intravenous use of sodium-thio-sulphate, but not before I was gravely worried for the life of the child. Lead is the only other poison that I have had any experience with, and convulsions from it as well as from arsenic are probably due to encephalitis. The above case showed a few blood cells in the spinal fluid, and is mentioned in Jean's book as an unusual complication of arsenic therapy and called encephalitis hæmorrhagica arsenicalis. His cases all died, but he advocated the use in future of sodium-thio-sulphate, and it certainly worked in my one case. It was also interesting that further arsenical therapy was made possible by the giving of sodium-thio-sulphate by mouth during its further courses of treatment.

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PRINCIPLES OF POST-OPERATIVE TREATMENT

Dr. T. H. Lennie

[We publish the attached lecture by Dr. T. H. Lennie as an example of the type and standard of lectures given to the Internes at the Vancouver General Hospital.—Ed.]

This subjct covers a very large field and is one in which I find great difficulty in preparing subject matter which would be of sufficient authority to designate as a lecture.

There is a remarkable diversity of opinion and practise among surgeons of equal competence with little variation in their results. It should be inferred from this that the rules of convalescence should be flexible and adopted to the individual patient. In other words, one should not feel that his school has said the last word, but should approach each problem with an open mind. For myself, I find as age increases the less dogmatic I become.

It seems to me that the first principle of post-operative care is a careful and adequate preoperative preparation. The handling of hyperthyroid cases is the outstanding example of this, and I feel that, apart from immediate emergencies, many of the means applied in this connection might with advantage be used in surgery generally. I refer particularly to adequate rest, the instilling of confidence in the patient regarding this major epoch in his or her life, and I would like to emphasize this point, and the bolstering of resistance to a surgical attack by adequate fluid and carbohydrate intake.

It has been said that every surgeon should undergo an operation, as only in this way can he appreciate the pain and suffering which an operation entails. It is true that memory for pain and physical discomfort in the average individual is short, but I can recall that my first thought after coming out of the anæsthetic some twenty years ago following a simple appendectomy was that I had opened my last abdomen.

As was said before, the individual patient must be studied. No two people are entirely alike, and the ability to withstand pain varies in races and sexes. The frail little woman will often put the robust male to shame. Post-operative comfort is therefore all important.

As to dressings, it is probably sufficient to say that they should adequately protect the wound from contamination and irritation. In addition, the dressing should lend support to the abdominal wall, but this does not mean the application of large strips of adhesive so tightly as to interfere with abdominal respiration and thereby add to the risk of pulmonary complications.

As a rule, a clean wound does not require dressing until the stitches are removed. If, however, there is the escape of blood or serum, the dressings become uncomfortable and should be renewed. The modern tendency is away from all but necessary drainage, but I would qualify that by saying that when in doubt, drain. By all means one must drain when frank pus is encountered. It naturally follows that when drainage is instituted dressings must be changed frequently. Just a word about the escape of infected material on the abdominal wall. The skin may be protected by sterilized vaseline, but, better still, if bowel contents are escaping, a paste made up of egg albumin and cornstarch makes a very satisfactory protection.

The immediate post-operative orders should provide for: (1) Quiet and relief of pain; (2) stimulation if necessary, and (3) the administration of fluids.

Morphine, unless there is some idiosyncrasy for this drug, is by all means the narcotic of choice. This should be given in doses large enough to assure rest, and if the respirations are depressed should be combined with atropine. My choice is $\frac{1}{6}$ morphine and $\frac{1}{150}$ atropine, repeated in four hours.

Stimulants, except in long and severe operations, are seldom necessary. Caffeine sodium benzoate 3-5 grains does very well.

Fluids, either by mouth, rectum, interstitially or intravenously. We seem to have adopted the intravenous method of giving normal saline and glucose, as this gives the quickest response. It has been estimated that within four hours after a major procedure the patient loses 1000 cc. of water and that the average adult requires from 3000 to 4000 cc. of fluids in 24 hours. One word of warning about salines. The above amounts of fluids, if given in this form, would be far in excess of the normal daily requirement, and "pushing" normal salines may actually do harm by imposing an impossible load on the kidneys. Thus it is wise to give some of the fluids in the form of 5% glucose.

Nausea and vomiting may be due to the anæsthetic. The more skilful the anæsthetist the less likely is the patient to vomit. The proper preliminary preparation and absence of trauma during operation will lesson the amount of nausea and vomiting. Of course individual susceptibility varies. Warm water with the addition of small amounts of soda will often serve as a natural lavage. In protracted cases the stomach tube may be necessary. I believe it was one of the Mayo Brothers who remarked that an interne should carry a stomach tube around his neck rather than a stethoscope.

Distention may occur after any operation, but is more likely to follow abdominal surgery, particuarly in the upper quadrant. It may vary from a few gas pains to a fatal complication. It may be avoided largely by gentle handling of the abdominal contents. Distension usually manifests itself on the second post-operative day. Loosening of the bandage will frequently give relief. The passage of a rectal tube and enemas, with the possible addition of heat to the abdomen, will usually suffice.

By the third day the patient should be on the road to recovery. He should be on a full liquid diet and possibly some softs. A cathartic is usually given on the night of the third day and he should be returned to his normal diet as soon as possible.

Complications

The average surgical case gets by with remarkable smoothness. It may with some truth be said that the operator's skill may be measured to a large extent by the absence of post-operative complications. The majority of complications develop as a result of pathology in some system or organ not actually concerned in the original disease process; but some serious complications may ensue as a direct result of the operation itself.

Infection: Leaving out the ordinary wound infections one must always recognize the possibility of septicæmia developing either from the original infection or from infections introduced by a break in the surgical technique. Here one must increase the fluid intake, particularly by giving glucose intravenously, and, if there is an accompanying blood destruction, blood transfusion. The new drug, Sulphanilamide, will likely prove a potent addition to our armamentarium.

Cardio-renal and pulmonary complications may be looked upon as medical and, therefore, will not come within the purview of this paper.

Abdominal Complications: The most important of these are ileus of whatever type and acute dilatation of the stomach. Lewis states that "paralytic ileus has almost disappeared as a post-operative complication due to the perfection of aseptic technique." To this can be added the importance of gentle handling of tissues. Any abdominal distension after operation may be regarded as a mild grade of ileus. It is common after surgery of the biliary tract, less likely to follow stomach operations. Large ventral hernia repairs are prone to be followed by distension of a serious nature, but it may also follow a simple appendectomy. Early recognition is imperative and treatment should be instituted immediately. It is important but difficult to differentiate between paralytic and obstructive ileus. In the former peristalsis should be stimulated, in the latter this may prove fatal.

In paralytic ileus the following measures may be employed: enemas, hot turpentine stupes, pituitrin by intramuscular injections, hypertonic salines, acetylcholine, continuous suction and possibly jejunostomy. Spectacular results have been reported following the administration of a spinal anæsthetic. In paralytic ileus, due to general peritonitis, one should not stimulate peristalsis—jejunostomy is the procedure of choice.

Acute dilatation of the stomach also requires early recognition, and treatment. No patient should die from this condition. Gastric lavage is the treatment. In fact I believe the more routine use of continuous suction has in several instances prevented the development of these two distressing conditions. One need hardly point out the rapid development of a grave toxæmia which develops in all obstructive lesions. The object of continuous suction, of course, is to rid the intestines of the toxic material which rapidly collects therein. It is extremely important that the chlorides lost be replaced by normal saline immediately.

Hiccoughs: This distressing condition is most likely to follow upon extensive operations of the gastro-intestinal tract where large peritoneal surfaces are involved. Next in

order are operations upon the brain and spinal cord. Thus it is difficult to explain the etiology. The actual mechanism, however, is a spasm of the diaphragm. Lasting for more than a few days it should be looked upon as a very serious complication and may end fatally. Practically every drug in the pharmacopœia has been advocated for the treatment of this condition. This alone would indicate their ineffectiveness. The usual attack is self-limited, so that the drug last used gets the credit for the cure. An anti-spasmodic such as atropine is indicated. In protracted cases gastric lavage followed by large doses of morphine should be given and this repeated at intervals. The rest thus obtained is very beneficial.

Inhalations of carbon dioxide have also been advocated. Blocking of the phrenic nerve with alcohol may occasionally be indicated.

Shock: I shall not attempt to define this condition. There is no unanimity of opinion as to the exact changes which occur in producing and maintaining shock. It is generally recognized now that this condition depends upon a dilatation of the smallest channels in the arterio-venous tree, thus normal arterial pressure cannot be maintained.

The symptoms and signs are subnormal temperature; irregular, rapid and compressible pulse; cold, pallid, clammy skin; shallow and irregular respirations; consciousness maintained at a low ebb and mentality weak. The sphincters are relaxed. There may be marked restlessness or even delirium. The onset is always rapid. Treatment should include the following: heat to the entire body, elevation of the lower part of the body and lowering of the head, bandaging the lower extremities, adrenaline for its temporary effect. Blood volume must be increased immediately. Transfusions of whole blood, if available, are by far the most efficacious. As a substitute slightly hypertonic solutions should be used, preferably glucose.

Naturally I realize in this short paper the subject of post-operative care has been very inadequately covered. Abdominal conditions have been stressed, as it was felt that the surgery of the specialties could be discussed much more effectively by representatives from these departments.

* * * *

STREPTOCOCCIC MENINGITIS
in the Vancouver General Hospital

Dr. T. F. H. Armitage

Streptococcic meningitis is not a common disease, only thirty-three cases occurring in this Hospital in the seven year period, 1932 to 1938, diagnosed by positive culture from the spinal fluid. The disease up until two years ago was almost universally fatal. The majority of these cases have arisen from mastoiditis.

There was thought to be a relationship between the incidence of scarlet fever and the incidence of mastoiditis, but the statistics on the whole do not bear out this relationship. There were twenty-five cases of mastoiditis in the seventeen hundred and ten cases of scarlet fever in the period 1932 to November, 1938.

The following figures show the concurrence of these diseases and the results of the treatment of streptococcic meningitis with sulphanilamide. There were no cases up to the beginning of 1937 treated with sulphanilamide, and all cases since that time *have* been treated with sulphanilamide either in the form of prontosil or prontylin or both.

	Number of cases of Scarlet Fever	Number of reported cases of Scarlet Fever with Mastoiditis
1932	110	1
1933	280	0
1934	595	9
1935	190	1
1936	173	8
1937	167	5
Nov., 1938	185	1
Total	1710	25

	No. of Cases of Mastoiditis and Mastoiditis with Otitis Media	No. of Cases of Streptococcic Meningitis	Mortality. d.	%
1932	84	3	3	100%
1933	137	1	1	100%
1934	114	1	1	100%
1935	108	4	4	100%
1936	213	11	10	91%
1937	220	10	7	70%
1938, Nov.	82	3	3	100%
Totals	958	33	29	

In 1936 there were 11 cases of streptococcic meningitis, 3 male and 8 female, their ages ranging from 11½ to 28 years. The original sources of the infection were as follows:

Otitis media and mastoid	5 cases
Streptococcic pharyngitis	1 case
Bronchopneumonia	1 case
Not stated	4 cases
	11 cases

Ten died and one was discharged well. Mortality rate 91%. This case that recovered was a male Chinese, age 10, treated with scarlet fever antitoxin and acriflavine intravenously. None of these was treated with sulphanilamide.

In 1937 there were ten cases of streptococcic meningitis, 4 male and 6 female, their ages ranging from 7 to 78 years. The original sources of infection were as follows:

Otitis media and mastoid	2 cases
Scarlet Fever	1 case
Not stated	1 case
	10 cases

Seven died and three were discharged well. All these cases were treated with sulphanilamide.

Up to November, 1938, there have been only three cases of streptococcic meningitis, 2 female, ages 1 and 7 years, and 1 male, age 62 years. The original sources of infection were:

Otitis media and mastoid	2 cases
Streptococcic pharyngitis	1 case
	3 cases

All these patients died.

These thirty-three cases in the Vancouver General Hospital constitute a series in which there is no selection and the analysis of which does not take into consideration the condition of the patient when treatment was started. In such a group as this, individual cases should be studied in considering the final results. For instance:

Case 1. Reported in November, 1938: Female, age 7 years, had had definite signs of meningitis ten days previous to admission and improved slowly for three days on treatment with sulphanilamide, but died with a bronchopneumonia.

Case 2. Female, age 1 year, admitted April 4, 1938. Patient's condition was poor on admission; there were frequent convulsions and twitchings. Patient was treated with scarlet fever antitoxn and prontosil but developed bronchopneumonia and died May 5, 1938. Source of infection was bilateral suppurative otitis media.

Case 3. Male, age 62 year, admitted September 9, 1938. Patient had chronic otitis media and condition on admission was poor. Prontosil was given but condition became worse and patient died September 11, 1938.

Up until the time that the use of sulphanilamide in the treatment of streptococcic meningitis became general this disease was almost 100% fatal but now a definite number

can be cured, a great deal depending on the early diagnosis and early treatment. In a comparative series of seventeen cases treated with sulphanilamide by Neal and Appelbowan in New York, four of the seventeen cases died and thirteen recovered, the mortality rate being 24%.

REPORT ON CASES OF PERNICIOUS ANÆMIA TREATED IN THE OUT-PATIENT DEPARTMENT

R. D. MacLaren and B. M. Fahrni

At a recent staff meeting of the Vancouver General Hosptal a review of cases from the pernicious anæmia clinic of the out-patient department was presented by Drs. J. E. Walker, S. H. Sievenpiper, E. F. Christopherson, R. D. MacLaren and R. M. Fahrni. Only those cases attending since November, 1936, were considered, this date coinciding with the appearance of the more concentrated liver extracts. It was found that there were thirty-eight cases which we could conclusively prove were pernicious anæmia. The data obtained was from these thirty-eight proved cases and the conclusions drawn, as well as parts of the data which we feel would be of value to the general practitioner, will be given below:

Diagnosis: The importance of satisfactorily establishing the diagnosis of pernicious anæmia in a new patient by ruling out the other causes of macrocytic anæmia cannot be overestimated. The inconvenience, expense and psychic trauma caused by committing a patient, especially a relatively young patient, to the sentence of weekly or bi-weekly liver injections for the remainder of his life, are not to be treated lightly.

No symptoms are in themselves diagnostic, but the history of sore tongue, weakness, and such neurological symptoms as numbness, tingling of exremites and difficulty in walking, are always suggestive. These cases demonstrating the triad of achlorhydria, typical blood picture, and subacute combined secerosis of course present no difficulty. Twenty per cent. of our cases showed no neurological signs or symptoms on admission. The following procedures were carried out, as a routine, in all cases:

1. Full blood count and colour index.
2. Gastric analysis for free HCl. with histamine preceding the test in cases of doubt, bearing in mind the danger of the use of this drug in elderly patients.
3. Gastro-intestinal X-Ray—three cases of gastric carcinoma have been picked up during the past eight years, one of which was under treatment with liver extract for two years before admission.
4. Stool examined for occult blood—this helps in the diagnosis of malignancy and less serious causes of chronic blood loss.
5. Examination of stool for parasites—in the last eight years there has been one case of tapeworm infestation picked up at our clinic.
6. Van den Bergh may be done as further confirmatory evidence, though it is not of specific value.
7. Sternal puncture as a rule is unnecessary, unless one is in considerable doubt as to the diagnosis, and especially where aleukæmia may be considered a possibility.

Another reason why we labour the point of adequate investigation when the case is first seen is because we not infrequently admit patients who have been on liver treatment for varying lengths of time, and who have had little or no initial investigation, sometimes not even a blood count. In such a case, liver treatment has obscured the original blood picture and the only way left to make a definite diagnosis of pernicious anæmia may be to discontinue liver therapy and allow the patient to relapse.

Maintenance Dosage: The majority of those who attend our out-patient department are maintenance cases, those who relapse being hospitalized. As with insulin in diabetes, there is no standard dosage of liver in pernicious anæmia. Our cases were maintained on from 1 to 5 cc. of concentrated liver extract weekly injected intramuscularly, the dose in most cases being 2 cc. per week, although 2 cc. every two weeks was sufficient for many cases (1 cc. of the extract used is equivalent to 100 grams of fresh liver (3).

The importance of keeping the red cell count well up to the normal figure, both to prevent the development of neurological lesious, and more especially in those cases where signs of postero-lateral sclerosis are already present, is well recognized. This is demonstread by the fact that two of our cases developed these signs after irregular treatment. Four million red blood cells per cubic millimetre and 80% hæmoglobin is not sufficient. The figures of five million red blood cells and 95% to 100% hæmoglobin should be aimed at in every male case, with the count slightly lower in women. It is inexcusable for any patient to develop serious incapacitating neurological signs while under treatment for pernicious anæmia; it means simply that insufficient liver was given—the count was not maintained at sufficiently high level.

To cases were satisfactorily maintained on autolyzed liver extract by mouth, the doses being drs. 1, t.i.d., and q.i.d.

Inhibiting Factors: The amount of liver required to maintain the red cell count at normal level is greater in the presence of any condition to which inhibits hæmopoiesis. In our patients, as might be expected in their age group, the commonest of these factors was general arteriosclerosis. Second to this cause was infection, most commonly in the oral activity or respiratory tract, such as carious teeth, septic tonsils, or chronic bronchitis. The presence of diabetes, heart faliure, duodenal ulcer, pulmonary T. B., and menopausal symptoms, also appeared to act as inhibiting factors.

Cases in Relapse: (These findings were based in indoor patients). The object of the review of these cases was to decide what constitutes a satisfactory dosage of liver in this stage of the disease. During the past twenty months, twenty-three cases were found suitable for study, the remainder having had insufficient investigation for us to be sure of the diagnosis, or insufficient data to enable the course of recovery to be closely followed. The cases were divided into three groups, depending upon the blood count on admission: group one constitutes cases below one million, group two between one and two million, and group three between two and three million. The complicated and uncomplicated cases of each group were separated.

Group I: According to Osgood, a satisfactory response in group one entails a reticulocyte count peak of 20% to 70%, with an average of 40%, and according to Minot, a R. B. C. rise of one million in two weeks. In this group were two uncomplicated cases.

Liver Dosage up to time of peak of reticulocyte count	Retic Response	Increase of R.B.C. (millions) and time recorded
[1] 1 cc. daily for 3 doses	30%	.95 in 2 weeks
[1] 4 cc. in 1 dose	20%	.94 in 2 weeks

The remaining four cases were complicated by arteriosclerosis, active lues, or pneumonia, and larger doses of liver than those stated above gave poorer responses.

Observations: A total dosage of more than 3 or 4 cc. of concentrated liver extract (1) should apparently be given in cases in this group and daily doses appear more satisfactory than a single larger dose.

Group II: Theoretically, in this group, a satisfactory response entails a reticulocyte peak of 10% to 60% with an average of 25%, a red cell of one million in two and a half weeks. Six cases were uncomplicated and are listed below:

Liver Dosage up to time of peak of reticulocyte count	Retic Response	Increase of R.B.C. (millions) and time after starting treat
[1] 4 cc. in one dose	17%	.91 on 16th day
[1] 2 cc. daily for 2 days	27%	.90 on 10th day
[1] 3 cc. daily for 5 days	39%	.73 on 15th day
[2] 2 cc. in one dose	30%	1.21 on 20th dya
[3] 5 cc. daily for 5 days	25%	1.3 on 15th day
[4] 5 cc. daily for 5 days	26%	2.2 on 15th day

The four complicated cases in this group suffered from arteriosclerosis, cardiac decompensation, peripheral neuritis, and pneumonia, and responded similarly to the complicated cases of Group I.

Observations: The length of hospitalization necessary in Group II varied but little, while dosages varied as much as 400%. From the results it would seem that the largest

dose, 15 cc. ([1]) appeared to do little but increase expense, while the smallest single dose, 4 cc. ([2]), was hardly sufficient, and again daily smaller doses gave a better response.

Group III: Here a satisfactory response ranges from 5% to 33% reticulocytes with an average of 11%. Four uncomplicated cases comprise this group and are recorded as in Group II.

[1] 1 cc. twice	10.8%	1.0 on 20th day
[1] 2 cc. twice	11. %	.3 on 5th day
[1] 2 cc. daily for 5 days	17. %	
[2] 4 cc. once	8.2%	.61 on 27th day

The complicated cases responded as in other groups. Complications here were arterioslcerosis, chronic nephritis and essential hypertension.

Observations: In this group the smallest dose, 2 cc. ([1]), given evoked a satisfactory response.

General Observations: Following the reticulocyte peak in these cases it was found bi-weekly injections caused a continued satisfactory response up to the stage where maintenance dosage was commenced. In regard to the complicated cases the necessary dosage was noticeably larger, and to some extent dependent on the severity of the complication.

Accessory Treatment: After reviewing the results of the accessory treatment given our out-patient department cases of pernicious anæmia, we find our views much the same as those stated by some of the eastern clinics, i.e., in regard to the use of iron, hydrochloric acid, transfusions, and vitamin B.

Iron: It is believed that in pernicious anæmia the breakdown products of red cells are stored in the body, and are available at once for the manufacture of hæmoglobin, should normal blood formation recur as the result of a natural remission or liver therapy. In the patient who presents himself with a one million red cell count, it is not uncommon for the colour index to fall below one, temporarily, during the rapid rise expected from adequate liver therapy, i.e., for the red cell rise to be greater in proportion than the rise in hæmoglobin. But it is usual that following this initial rapid rise the colour index again becomes greater than 1.0. However, in a few cases the iron storage may not be sufficiently great to prevent the C.I. from remaining below 1.0 for a considerable period, as the blood count rises. Under such circumstances we feel that iron is indicated. In three of our cases we noticed almost specific effects from iron when given in addition to liver therapy. For example, one case of ours, who, when first seen, had a count of 1.5 million, improved as expected on liver therapy until a count of 3.3 million was reached. No further increase was noted, even though 6 ccs. of concentrated liver extract were given each week. Iron and ammonium citrate (90 grs. a day) was then started and the count rapidly rose to 5 million, where it has remained, the patient requiring 2 cc. of liver extract a week to maintain her count at this level. In 50% of our cases iron was not used and in 40% of the remainder was of no apparent benefit, so we conclude that the average case of pernicious anæmia does not require iron, with the exception of the above stated conditions, or when the case is complicated by hæmorrhage from any cause. Previously large doses of iron were thought to benefit neurological symptoms, but this has been shown many times not to be the case, and certainly caused no improvement in our cases.

Hydrochloric Acid: In regard to hydrochloric acid we consider the only indication in pernicious anæmia to be in the treatment of digestive disturbances—certainly some cases of dyspepsia which are not alleviated by specific liver therapy are kept well in check by the use of ½ to 1 drs. of dilute hydrochloric acid taken with meals. Among our cases the incidence of those given hydrochloric acid for the control of gastro-intestinal symptoms was just below 25%. Two cases obtained more relief when pepsin was added. The achlorhydria of pernicious anæmia does not appear to greatly affect the absorption of iron from the gastro-intestinal tract.

Arsenic: None of our patients in this series received Fowler's Solution; it is generally agreed that arsenic is of no benefit in the treatment of pernicious anæmia.

Transfusions: While formerly transfusions were frequently given to pernicious anæmia patients with hæmoglobin of 30% or less, it is now felt that unless the patient is suffering from the effects of insufficient oxygen carriage, at bed rest, this procedure is

not indicated. Transfusion has no beneficial effect on blood production in pernicious anæemia and it has been shown that giving 500 cc. of citrated blood affects very little in the end the time required to bring the count of a patient from one to four million, when adequate liver therapy is given. In contrast to hypochromic anæmia there is an added deficiency element often not relieved by elevating the blood count alone, and frequently do not disappear until some of the deficiency factor in the form of liver is supplied. Since clinical improvement in our cases began three to four days after injection of liver, and in two of our cases no improvement in symptoms was noticed following transfusion, we believe that liver extract rather than blood is always the primary need. It must not be forgotten also that serious transfusion reactions are more apt to occur in the severely anæmic patient than in one with a hæmoglobin of 60%. So we consider the chief indications for transfusion to be the following:

1. In the presence of some severe inhibiting factor, such as infection (notably pneumonia), or arteriosclerosis.
2. Where surgery is contemplated, several transfusions may be given in addition to liver therapy to repidly elevate the blood level.
3. Where there is definite doubt as to the diagnosis, a small transfusion is the safe and conservative course.
4. Where the case is complicated by hæmorrhage from any cause.

Vitamin B: The role of vitamin B in the treatment of pernicious anæmia is at present uncertain. In 1932, Castle believed that the extrinsic factor deficient in this anæmia was vitamin B.2, but this has been shown not to be so. With the recent flair for vitamin B.1 in medical treatment, it was suggested that all cases of pernicious anæmia with sensory changes in the extremities be given this vitamin. Consequently, six of our cases were put on what was considered adequate dosage of vitamin B.1 with improvement in paræsthesias in only one case. The few cases reported improved in the literature were uncontrolled, and like our cases do not constitute significant evidence that vitamin B is of any value in the treatment of pernicious anæmia.

Legend:

[1] Lederle's Concentrated [2] Anahaemin [3] Connaught [4] Campolon

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UPPER ISLAND MEDICAL ASSOCIATION

The members of the Upper Island District Medical Association gathered for a Dinner Meeting at Qualicum on Wednesday, June 21st. While not as largely attended as the Annual Meeting, which takes place in the Fall, it was not lacking in enthusiasm.

Those present included: Dr. P. L. Straith, Courtenay, the President; Dr. T. L. Briggs, Courtenay, Secretary; Dr. G. K. MacNaughton, Cumberland; Doctors C. T. Hilton and R. W. Garner of Port Alberni; Doctors C. C. Browne, W. F. Drysdale, S. L. Williams and E. D. Emery of Nanaimo; Doctors J. McKee and H. A. L. Mooney of Courtenay; Doctors H. A. DesBrisay and M. W. Thomas, Executive Secretary, visiting from Vancouver.

A short business session preceded the papers of the evening.

Dr. C. C. Browne of Nanaimo dealt with Separation of the Tibial Tuberosity, discussing fully etiology and treatment, proving that the general outlook in these cases was good. He was able to show a series of interesting films illustrating case reports.

Dr. H. A. DesBrisay of Vancouver presented in a very interesting way a Review of Newer Trends in Modern Medicine, stressing particularly recent therapeutic advances and showing wherein they were very practicable and based soundly on our knowledge of changes in disease. He told of useful tips in diagnosis which could be easily used outside of hospital practice.

Dr. M. W. Thomas, Executive Secretary, told the members of the work in the office of the College of Physicians and Surgeons and finally reminded all of the excellent programme being prepared for the Annual Meeting to be held in September.

Doctors Hilton and MacNaughton presented a vote of thanks to those who had provided the programme and such an interesting meeting.

POSSIBILITY OF FAULTY DIAGNOSIS OF DIABETES IN PATIENTS TAKING THIAMIN CHLORIDE

RUTH S. HART, M.D., AND LOUIS E. WISE, PH.D,, WINTER PARK, FLORIDA

Reprinted from J.A.M.A. Feb. 4, 1939, p. 423.

The first morning specimen of urine collected from the patient, Mr. L. W., showed very marked reduction of Benedict's solution. The following morning the fasting blood sugar content was determined and found to be 90 mg. per hundred cubic centimetres. The following day, after a breakfast high in carbohydrates, the urine was again examined and showed no reduction. At intervals thereafter, with a continued daily dose of 4 mg. of thiamin chloride, the urine either showed traces of reducing substances or no reduction whatever. The medication of the patient, begun a month prior to the first examination, was two tablets of thiamin chloride prior to each meal (i.e 6 mg. a day), three 1½ grain (0.1 Gm) tablets of theophylline with ethylenediamine and 3 grains (0.2 Gm) of phenobarbital daily. Neither theophylline with ethylenediamine nor phenobarbtal reduced Benedict's solution.

On the other hand, 10 mg. of pure thiamin chloride in 1 cc. of distilled water reduced Benedict's solution only slightly, giving an atypical yellow precipitate.

Whether or not thiamin chloride alone was responsible for the reducing substance found in the urine originally examined still is problematical.

Morning specimens from three other patients who were on at least 6 mg. of thiamin chloride daily were examined and showed no reduction of Benedict's solution. Of these, one patient was taking in addition to thiamin chloride an uncertain amount of morphine. One other was taking 40 grains (2.5 Gm) of acetylsalicylic acid daily.

The following possibilities in this case must be considered:

(a) Storage of thiamin chloride with marked elimination at intervals.

(b) Elimination of oxidizable degradation products of thiamin chloride.

(c) Elimination of oxidizable degradation products of theophylline with ethylenediamine or phenobarbital or both.

(4) Lowering of the renal threshold to dextrose through the agency of thiamin chloride.

The question arises of the accuracy of Benedict's test for measuring dosage of insulin with patients taking thiamin chloride.

CONCLUSIONS

1. Pure thiamin chloride will reduce Benedict's solution in vitro.
2. Urine of one patient receiving 6 mg. daily of thiamin chloride reduced Benedict's solution despite a blood sugar of 90 mg. per hundred cubic centimetres of blood.

[We reprint this as what we consider rather an important item, in these days of extensive use of vitamin B. Our attention was drawn to the matter by Dr. Hebb of the medical staff of the Vancouver General Hospital who, quite independently, had made the discovery that the administration of thiamin chloride, in two cases, led to reduction of Benedict's solution by the urine of the people concerned, though neither was diabetic. One large manufacturing firm assured him his ideas were nonsensical, but another, perhaps more up-to-date, was able to locate for him the short article reproduced above, of which Dr. Hebb knew nothing. We feel that this constitutes an important warning in the treatment of diabetes and others, and thank Dr. Hebb for letting us know about it.—Ed.]

St. Paul's Hospital

REPORT OF A CASE OF PRIMARY CANCER OF THE LIVER

By C. H. Vrooman and A. Y. McNair

Those who attended the clinic of Doctor Holman at St. Paul's Hospital during the Summer School session will remember the case of a Chinaman with large hæmorrhagic ascites, in which the diagnosis was discussed but nothing definite arrived at. We are now able to present the post mortem report.

HISTORY

Chinese cook, aged 48, seen first on April 13th, 1939, complaining of lumbago. Nothing was noted in the abdominal examination at that time. He was seen a second time on May 26th when he complained of acute swelling of the abdomen of only two weeks' duration. The abdomen was greatly distended, and the patient was sent to St. Paul's Hospital, where about 1000 cc. of blood was aspirated. Even after aspiration the liver could not be well defined, though it was thought it had slightly roughened edges. The man was again aspirated at the clinic, and pure blood was again found. He gradually got worse, and died on June 9th.

Doctor McNair did a post mortem, and reported as follows:

Post mortem examination of Chew Lee, age 39. Apparent age of this Chinaman was 45, fairly well developed and poorly nourished. Two small trocar holes, from which bloody, peritoneal fluid exuded, were seen in the lower abdomen, which was markedly discolored from subcutaneous extravasation of blood. No marks of violence were seen on the body.

Peritoneal cavity contained approximately one quart of blood, with some increase in peritoneal fluid. Liver was enlarged, particularly the right lobe. Stomach was small and contained some "coffee-ground" material. Spleen, esophagus, and gastric wall were negative. Gall bladder and cystic duct were negative except for some biliary sand. Duodenum was normal. Ampulla of Vater and pancreas were normal. Kidneys were negative except for mild arteriosclerotic changes. Intestines from end to end showed nothing remarkable.

Liver showed many adhesions to surrounding structures, weighed approximately 1800 grams, with a very marked enlargement of the right lobe. On section there was a very large tumour mass involving right lobe and practically replacing three-quarters of it, with numerous other smaller areas of malignancy scattered through the left central portion. Portal vein and splenic vein were opened in situ. These two vessels were filled with malignant thrombi throughout with the large tumour growth in the liver. In the liver these thrombi were fairly firm, and very densely adherent to the vessel walls. Tumour in the liver on section was grayish-yellow in colour, very soft, and central portions undergoing liquefaction.

No secondary growths were found anywhere in the body. Lungs showed terminal broncho pneumonia. Heart was rather small, very lean, and had the appearance of a heart of starvation.

Microscopically, the chief point of interest was the liver. Sections of the liver show very definite cirrhosis of the unilobular type, with very heavy, fibrous trabeculi separating this into small islands of various sizes, and containing from one to three lobules. Portal areas show a good deal of round cell infiltration, and many bile ducts compressed and scarred. Liver cells themselves stain rather diffusely, and their cell margins are indistinct. Central vein areas are moderately engorged. Tumour varies considerably in density, with many areas of softening and degeneration, and other areas show growth to be the outstanding feature. In many respects these cells have the appearance of liver cells. Many of them are arranged in cords, are quite hyperchromatic and hyperplastic. Many tumor giant cells are seen, with active mitoses, and complete loss of normal lobular arrangement.

Tumour is quite vascular and in places tumour cells line venous sinuses. Extravasation of blood into the tumour mass is quite marked. Search for parasites in the liver and duodenum was negative.

This is a malignant hepatoma, and has been superimposed on a liver showing moderately well advanced cirrhosis, with malignant thrombus formation filling portal and splenic veins, with terminal broncho pneumonia and concealed hemorrhage.

CANADIAN MEDICAL ASSOCIATION

The Seventieth Annual Meeting has passed into history and Montreal excelled itself both in weather and the success of the convention. 1085 registrants enjoyed a fine programme.

Dr. T. H. Leggett, Chairman of General Council presided, with an attendance of 88. The Council adopted the consolidated Constitution and By-Laws for which a Committee with Dr. R. I. Harris as chairman, is to be commended.

Federation: New Brunswick has applied for status as a Division and it now remains for Manitoba to take similar action arising out of its Annual Meeting in September to bring the nine provinces into Federation.

Arising from recommendations in the splendid report of the Committee on Economics, of which Dr. Wallace Wilson of Vancouver is chairman, Mr. Hugh H. Wolfenden has been engaged as consultant to assist the committee in its work during the coming year.

Ten Senior Members were elected and included Doctors Harvey Smith of Winnipeg, R. D. Rudolf of Toronto, W. C. Galbraith of Lethbridge and I. Glen Campbell of Vancouver.

In 1940 the Annual Meeting will be held in Toronto and in Winnipeg in 1941. In 1942 the meeting will come west to Alberta. There is a suggestion that it may be held at Banff or Jasper, in which case the British Columbia Medical Association would be asked to hold its meeting at that time.

The needs of refugee doctors and their admission to practice in Canada were sympathetically studied. It was felt, however, that more than sufficient doctors are graduating from Canadian Medical Schools to provide for the needs of the Dominion.

Among the distinguished visitors to the meeting were Sir Arthur MacNalty, Chief Medical Officer, Ministry of Health for Great Britain, London, England; Professor Edward Proven Cathcart, Professor of Physiology, University of Glasgow; Dr. Thomas S. Cullen of Baltimore, fraternal delegate of the American Medical Association; Dr. Allen O. Whipple of Columbia University, New York, who gave the Lister Lecture.

Montreal did herself well.

Dr. G. F. Strong of Vancouver was elected to the Executive Committee of the Canadian Medical Association as representative from British Columbia, which position was capably filled by Dr. Gordon Kenning of Victoria during last year.

IMPRESSIONS OF THE ANNUAL MEETING OF THE CANADIAN MEDICAL ASSOCIATION, JUNE 19, 1939

F. Sidney Hobbs, M.D.

Was the Annual Meeting of the Canadian Medical Association, held in Montreal, a success? Emphatically yes. From the time the first committee meetings were held until the final wind-up with the Golf Tournament and Golf Dinner, there was never a dull moment. If any criticism was to be made it would be that there were too many good things on at the same time, so that often one had to miss lectures which one would like to have heard.

The programme began with the meeting of the General Council on Monday morning. Drs. Wallace Wilson and G. F. Strong flew to Montreal to be present. Tuesday was set aside for meetings of the General Council and the Council were guests in the evening at a dinner given by the Montreal Medical Chirguical Society and La Societé Medical de Montreal. The dinner was very well attended and by that time a great many of the

doctors were present who had come for the general meetings, so it was pleasant to renew old acquaintances among teachers, classmates and friends.

The scientific programme got underway on Wednesday and was without a doubt one of the best that has ever been presented. The round table conferences were well attended, but what a struggle it was to be on time at 8:30 a.m. when one had been well, perhaps, too well entertained the night before! To give a detailed account of each meeting is out of the question but mention should be made of the papers presented by the doctors from British Columbia. Dr. D. E. H. Cleveland gave a well received paper on "The place of Allergy in the diagnosis of skin conditions," and Dr. Kinsman spoke on "Some aspects of intra-cranial birth injuries."

One of the best events of the meeting was the Lister Oration, presented by Dr. Allen O. Whipple of New York. His subject was "A consideration of recent advances in surgery in the light of Lord Lister's studies." Other professors from outside of Canada were Professor E. P. Cathcart from Glasgow and Dr. Norman Miller of Ann Arbor, Michigan. The latter gave a particularly good talk on treatment of dysmenorrhœa, which suggested many new angles of attack on this problem.

British Columbia was honoured by the election of Dr. Glen Campbell to Senior Membership in the Association.

The scientific exhibits were well arranged and well attended. Dr. Norman Kemp was busy at the Ayerst, Harrison and McKenna booth, describing all the latest hormones that his company have brought out. He forgot business however, when he suggested a trip to room 704, where with glass in hand, he asked all about his old friends in Vancouver. It wasn't hard to see that his thoughts often strayed back to Vancouver.

And what of the entertainment provided? To say the least, it was excellent. The dance held at the Chalet on Mount Royal was a great treat, as was the dance held on the Normandie Roof of the Mount Royal. The golf dinner on Friday night wound up the meeting. The ladies' entertainment was well looked after—one doesn't need to worry about entertaining one's wife at such a meeting—that is all looked after. How hard it must be for the ladies to come down to ordinary living after being entertained at Beaconsfield and Mount Bruno!

The weather man was kind to us, too, the only rain being on the afternoon of the golf tournament.

And so, on Saturday morning the hotel lobby was deserted, and the medical profession of Canada was returning home, but with happy feeling of having learned a little, and renewed a great many old friendships. One and all resolved that when next year rolls around, instead of buying tickets for Montreal, they will be for Toronto, where just as many old friends will be on hand to greet us again.

The officers of the Canadian Medical Association elected for the coming year are:
President—Dr. F. S. Patch, Montreal.
Chairman of Council—Dr. T. H. Leggett, Ottawa.
Honorary Treasurer—Dr. D. Sclater Lewis, Montreal.
Editor—Dr. A. G. Nicholls.
General Secretary—Dr. T. C. Routley.
Associate Secretary—Dr. G. Harvey Agnew.
President-elect—Dr. D. Graham.

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Announcement has been made that the Fellowships of the John Simon Guggenheim Memorial Foundation have been extended to Canada and Newfoundland. These Fellowships were established in 1925 by the former U. S. Senator and Mrs. Simon Guggenheim in memory of a son John Simon Guggenheim. By them during the past 15 years, 840 artists, poets, novelists, composers of music, biologists, physicists, economists and workers

in all fields of the mind and spirit have been assisted, and now six stipends, normally fixed at $2,500 a year will be awarded annually to assist scholars and artists from Canada and Newfoundland to go to the United States to do research and creative work in their various fields.

For information please write to Henry Allen Moe, Secretary General, John Simon Guggenheim Memorial Foundation, 551 Fifth Avenue, New York City, U.S.A.

A circular regarding the above is now in the library, which gives some further information and also gives a list of those scholars of Canadian origin who have already been awarded one of these scholarships.

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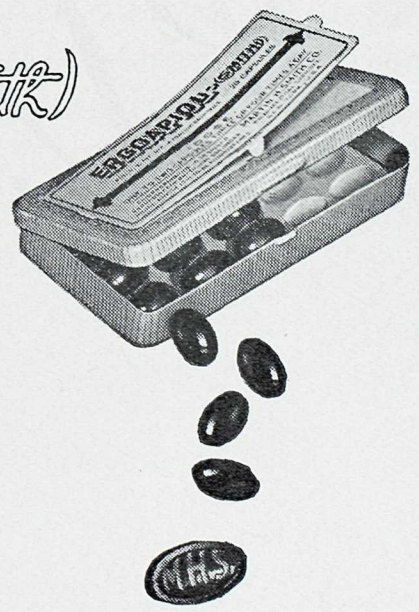

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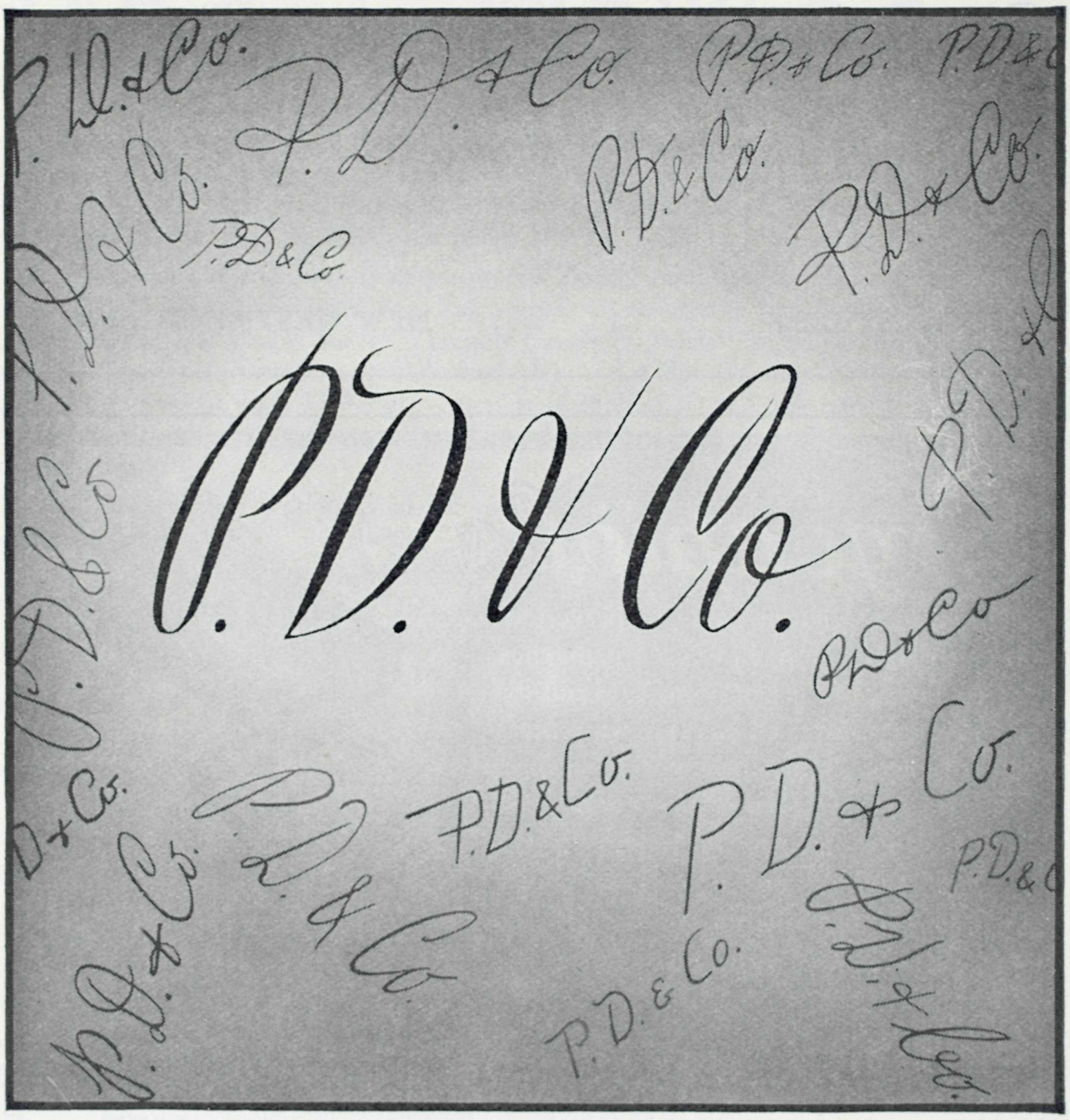

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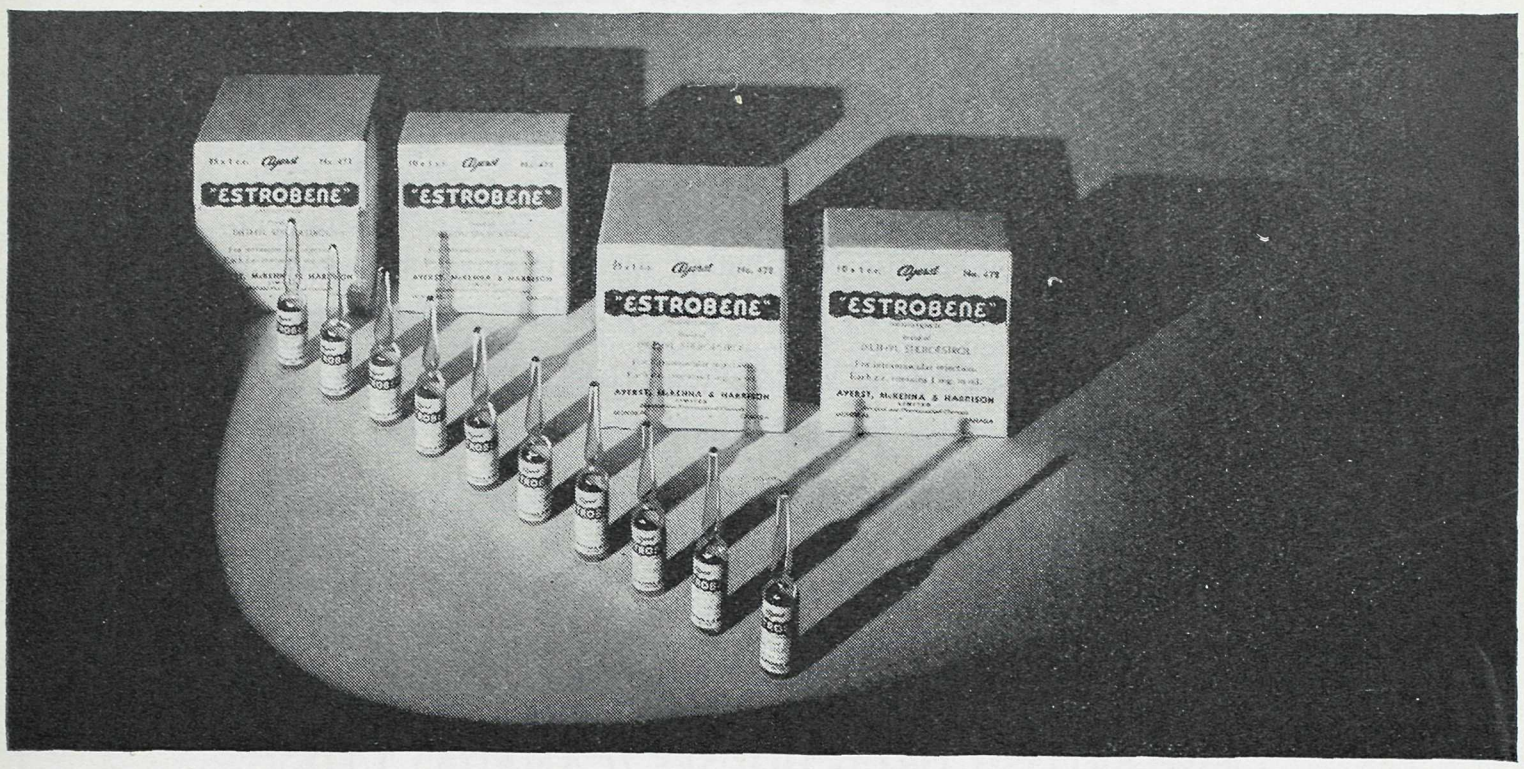

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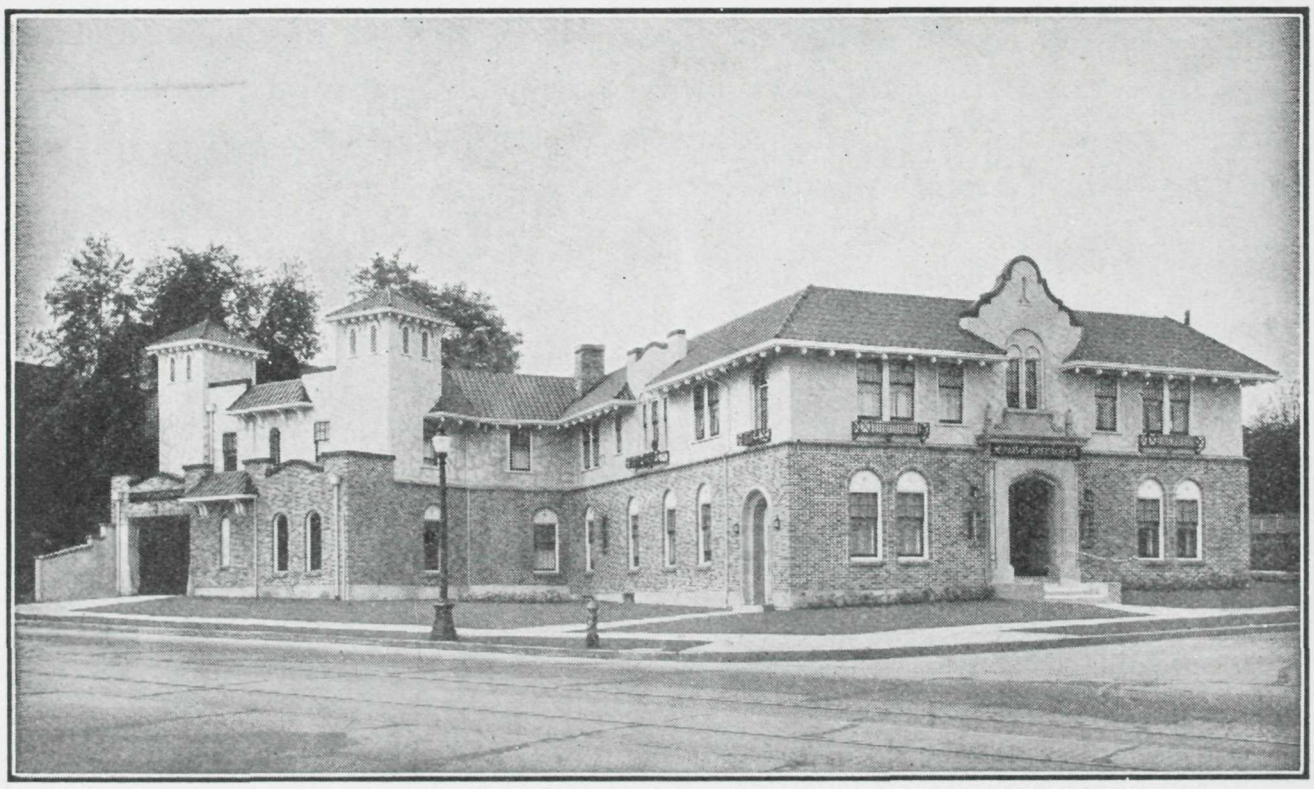

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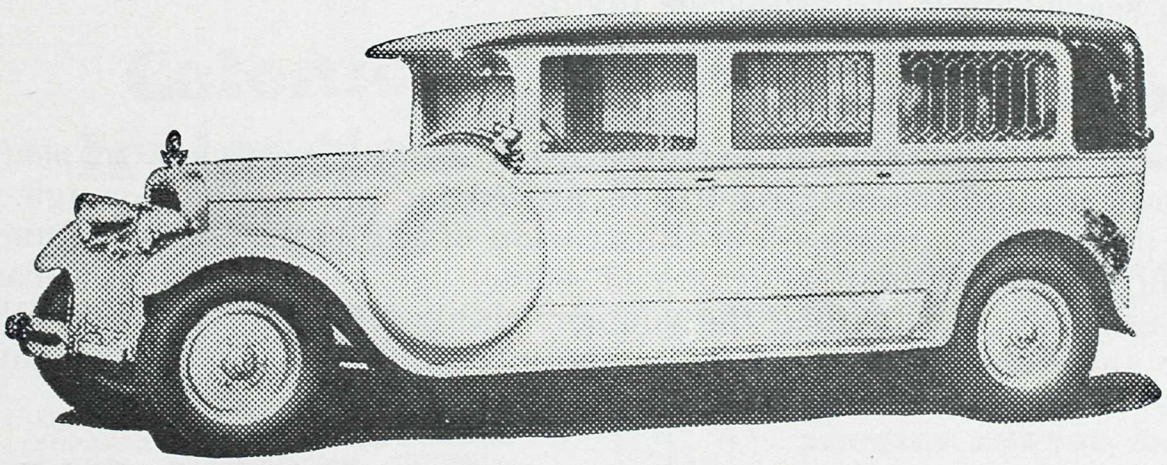

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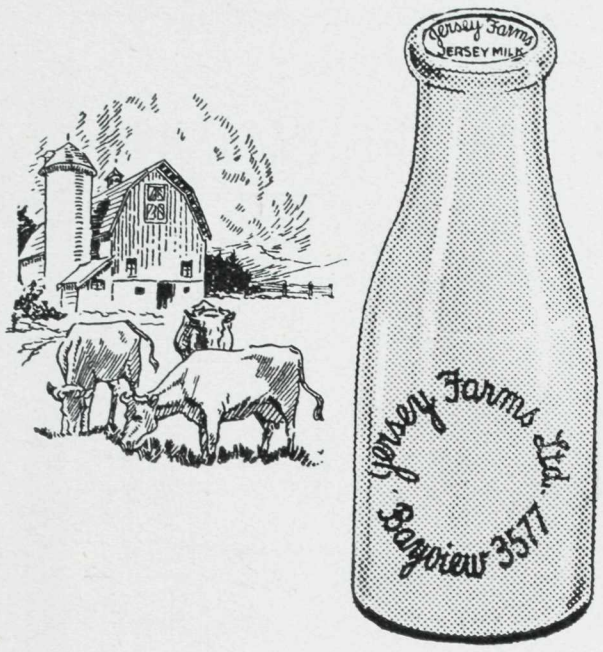

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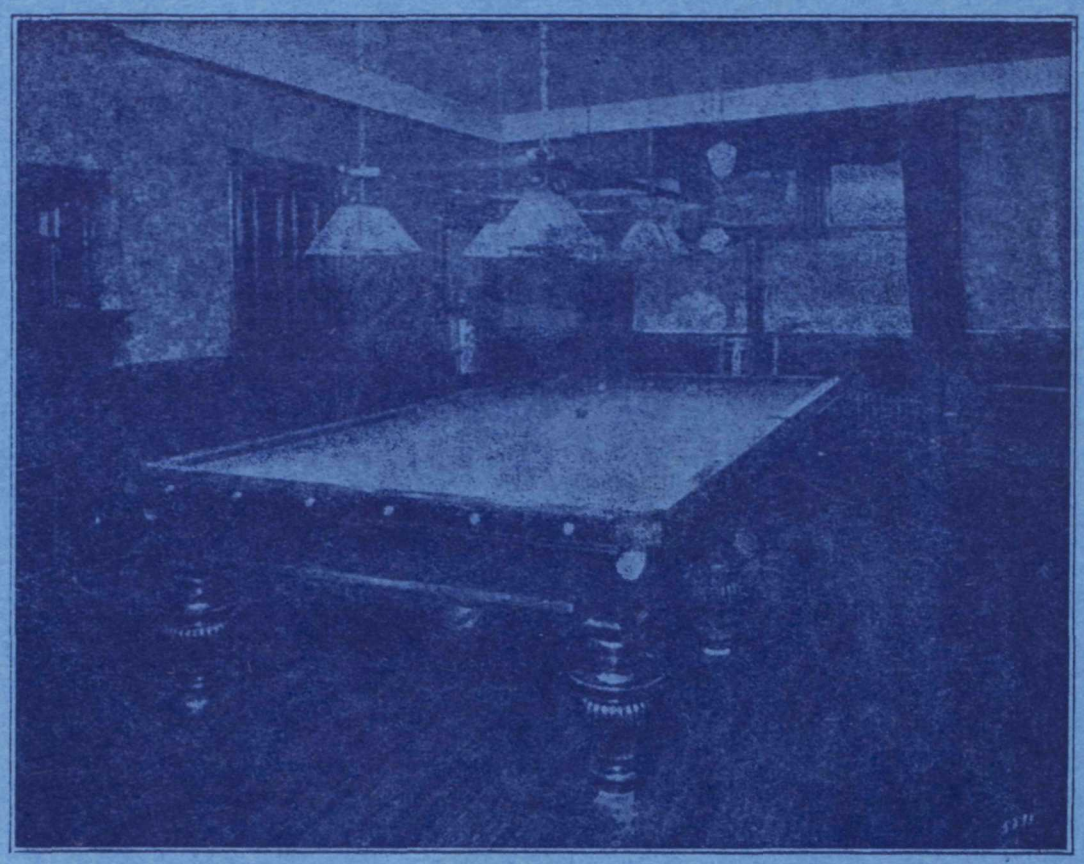

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