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The BULLETIN

of the

VANCOUVER MEDICAL ASSOCIATION

Vol. XIX. AUGUST, 1943 No. 11

With Which Is Incorporated
Transactions of the

Victoria Medical Society

the

Vancouver General Hospital

and

St. Paul's Hospital

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SEPT. 8, 9, 10, 1943

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THE VANCOUVER MEDICAL ASSOCIATION

BULLETIN

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

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EDITORIAL BOARD:
DR. J. H. MACDERMOT
DR. G. A. DAVIDSON DR. D. E. H. CLEVELAND
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VOL. XIX. AUGUST, 1943 No. 11

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VANCOUVER HEALTH DEPARTMENT

STATISTICS—JUNE, 1943

Total Population—Estimated	288,541
Japanese Population—Estimated	Evacuaetd
Chinese Population—Estimated	5,541
Hindu Population—Estimated	301

	Number	Rate per 1,000 Population
Total deaths	315	13.3
Japanese deaths	---	Population evacuated
Chinese deaths	20	43.9
Deaths—residents only	282	11.9
BIRTH REGISTRATIONS:		
Male, 360; Female, 342	742	29.6

INFANTILE MORTALITY—	June, 1943	June, 1942
Deaths under one year of age	16	24
Death rate—per 1,000 births	22.8	44.9
Stillbirths (not included above)	15	18

CASES OF COMMUNICABLE DISEASES REPORTED IN THE CITY

	May, 1943		June, 1943		July 1 to 15, 1943	
	Cases	Deaths	Cases	Deaths	Cases	Deaths
Scarlet Fever	41	0	31	0	8	0
Diptheria	0	0	---	0	---	0
Diptheria Carrier	0	0	---	0	---	0
Chicken Pox	200	0	211	0	14	0
Measles	691	0	167	0	17	0
Rubella	14	0	11	0	3	0
Mumps	138	0	64	0	10	0
Whooping Cough	64	0	36	2	5	2
Typhoid Fever	0	0	---	0	---	0
Undulant Fever	0	0	---	0	---	0
Poliomyelitis	0	0	---	0	---	0
Tuberculosis	36	0	65	13	15	---
Erysipelas	4	0	5	---	1	0
Meningococcus Meningitis	1	0	3	---	1	---

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

	Burnaby	West Vanc.	Richmond	North Vanc.	Vanc. Clinic	Hospitals & Private Drs.	Totals

Syphilis, Gonorrhœa—Figures not yet available.

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The fibre sources found to be most consistent in their action were cabbage and KELLOGG'S ALL-BRAN. These showed pronounced "bulk"-forming properties and satisfactory laxative action. Fruits and other vegetables gave variable and less pronounced effects.

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FOR ORAL USE

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FOR INTRAMUSCULAR USE

LIVER EXTRACT INJECTABLE is supplied as a purified and concentrated solution in 4-cc. rubber-stoppered vials, and in 12-cc. vials for hospital use.

Note: A change has been made in the packaging of these products. Formerly supplied in packages with yellow or brown wrappers, they are now distributed in blue and white packages. The extract contained in each package is identical with that formerly supplied.

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THE EDITOR'S PAGE

The coming month will be a very important one for the medical profession in Vancouver particularly and British Columbia generally: for two reasons. The first is the Annual Meeting of the British Columbia Medical Association, which will be held in the Hotel Vancouver on Sept. 8th, 9th, and 10th. The programme is published here and in a previous number of the BULLETIN, and there will be no doubt in our readers' minds that it will be a meeting of a greatest value, and not to be missed.

One of the most important features will be the Annual Meeting itself, on Sept. 8th. We shall have with us Dr. J. Sclater Lewis, president of the Canadian Medical Association, and the ever-welcome Dr. T. C. Routley, our General Secretary. There will be a discussion of Medical Economics in the form of a Round-Table—with specially fitted speakers to lead the various phases of the discussion.

Another very important meeting is the Public Meetings to be held in the Banquet Room of the Hotel, where Dr. Howard Spohn, president of the B.C.M.A., will be in the chair. It is hoped that Lt.-Col. D. H. Williams will be there, but in any case there will be a full presentation to the public of the question of Cancer and Venereal Disease.

Everyone should try and attend all the meetings he or she can. There are no fees, and we are all members of the B. C. Medical Association.

* * * *

The second matter, of great consequence to us all, is the Community Chest Campaign, which opens in September. This campaign is part of a nation-wide effort, made annually, as we all know. In Vancouver, it is known as the Vancouver Welfare Federation. It is one of the great projects of the year. No cause is more worthy of our attention, and more, of our active support. We should support it by word and deed. We should urge it on people, and contribute to it as much as we can. It is not any longer merely a charitable organisation: nor do we discharge our duty to it when we contribute a nominal sum, just to salve our consciences, and get rid of the worker who calls on us. It is a great national effort, which contributes immeasurably to the good of the community, and is accepted by all authorities on social service as an indispensable part of our social structure.

The Community Chest needs no justification or vindication. It is an actual necessity: it is a scientific, coherent, and admirably managed method of dealing with a whole series of problems that arise from various social maladjustments. The fact that the best business, professional and scientific men of the community endorse it, and eagerly give their services, unpaid, to the work of the campaign, is a proof, if any were needed, of the fact that it is accepted as a necessary part of our life as a community. And, from a strictly selfish point of view, it is of immense service to us as medical men. Merely read the list of agencies included: the Victorian Order, the Children's Aid, the Family Welfare Bureau, the Crippled Children's Hospital—and many others: and we realise that our work is aided and made far more efficient, by this organisation. From a business point of view, the economy of collection and administration are a sound reason for our support. Of every dollar contributed, ninety-four cents or over are actually applied to the work of the Welfare Federation.

The Community Chest Campaign is not confined to Vancouver. It will be held in every important centre in British Columbia, and we urge our members everywhere to give their strongest support to their local effort.

NEWS AND NOTES

A son was born to Capt. Gordon McL. Wilson, R.C.A.M.C., and Mrs. Wilson at Kamloops Hospital on July 19th.

* * * *

Congratulations are extended to Dr. and Mrs. Hubert Dumont of Vancouver on the birth of a daughter, July 20th.

* * * *

Dr. and Mrs. Howard Mallek of Vancouver are receiving congratulations on the birth of a son, August 16th.

* * * *

Sympathy is extended to Dr. Gerald Burke of Vancouver in his bereavement in the loss of his wife.

* * * *

The whole profession in British Columbia will extend to Dr. Thomas McPherson its sincere sympathy. Mrs. McPherson died on Saturday, August 14th.

* * * *

We regret to announce that the son of Dr. George H. Wilson of Burnaby, an officer with the Canadian Forces, was listed among the fatal casualties in Sicily.

* * * *

A number of doctors in Victoria, officers of the 13th Reserve Field Ambulance Corps, attended Army camp from July 11th to 25th. These included: Capt. A. B. Nash, Officer Commanding; W. H. Moore, G. B. B. Buffam, P. A. C. Cousland, W. A. Trenholm, L. W. Cromwell and V. L. Annett.

* * * *

In the Bulletin of the British Columbia Board of Health, August, 1943, well wishes were passed to Dr. R. J. Macdonald, formerly Director of the Prince Rupert and District Health Unit. Capt. Macdonald is now serving with the R.C.A.M.C. Dr. Roger G. Knipe, who has recently completed a course at the University of Toronto and received his diploma of Public Health, is quoted in the Bulletin as having taken over the "cuties" of Director of this Unit. The mention of this amusing item, which was brought to our attention by an out-of-town member, does not lessen our good wishes which go with Doctor Knipe to his new field of endeavour.

* * * *

Dr. B. T. H. Marteinsson has proceeded to Toronto and is taking a special course in R.C.A.F. training.

* * * *

Capt. N. H. Jones, R.C.A.M.C., formerly of Port Alberni, we understand, is now stationed at Prince Rupert.

* * * *

While Dr. Wilfrid L. Graham and family were vacationing at Mirror Lake in the West Kootenay, the Nelson Doctors held a meeting which was attended by Doctor Graham.

* * * *

Dr. George Young, who at one time practised at Salmo, visited his friends at Nelson recently. Doctor Young is now with the D. P. & N. H. at Calgary.

* * * *

Doctor Shillington of the D. P. & N. H. at Calgary spent a holiday at Kaslo.

* * * *

Dr. A. W. Bagnall of Vancouver was holidaying at Kaslo and on the Kootenay Lake during several weeks.

Dr. and Mrs. E. S. Hoare of Trail had a two weeks' vacation at Willow Creek near Nelson.

* * * *

Dr. Wilfrid Laishley of Nelson has just returned from a fishing trip on the Whatshan Lakes near the Kootenay end of the Monashee Pass road.

* * * *

Flight-Lieut. A. S. Underhill, R.C.A.F., now stationed in Alberta, has been on furlough and visiting in Kelowna.

* * * *

Dr. S. B. Walker, formerly of Manitoba, has entered practice in West Summerland. Since Flight-Lieut. A. W. Vanderburgh, R.C.A.F., entered the Service, Dr. F. W. Andrew has been very much overworked and will welcome the relief afforded by Doctor Walker's arrival.

* * * *

Dr. L. Giovando of Nanaimo has a new boat—a 34-foot express cruiser called "Spindrift."

* * * *

Dr. A. H. Meneely of Nanaimo is spending his vacation at Qualicum Beach.

* * * *

Capt. S. L. Williams, R.C.A.M.C., formerly of Nanaimo, we hear, is in charge of the Venereal Disease programme in the Manitoba District.

* * * *

Dr. W. F. Drysdale of Nanaimo has been ill in Nanaimo Hospital. We are glad to say he is reported to be up and about again.

* * * *

Dr. C. C. Browne will spend his vacation with his family on his yacht the "Ann."

* * * *

Dr. D. J. Millar has returned from his annual trip of exploration in the rivers and lakes of Lillooet. We have no report on the size of the big one that got away.

* * * *

Dr. R. V. McCarley is holidaying in Eastern Canada.

* * * *

Both Dr. A. C. Nash and Dr. Claire M. Onhauser of West Vancouver have managed a vacation.

* * * *

We are glad to report that Dr. A. T. Johnston of West Vancouver recovered from his operation when he lost a troublesome appendix. He is back in practice again.

* * * *

Dr. L. A. Patterson, who has been serving as doctor in the logging area on Moresby Island, is entering the R.C.A.M.C.

* * * *

Major J. Ross Davidson is at Prince George donig Surgery at the Prince George Military Hospital.

* * * *

Dr. K. K. Pump of Williams Lake called at the office. He is extremely busy covering the needs of a wide area.

* * * *

Dr. D. M. King, who has been at Bralorne for some years, is entering the R.C.A.M.C. Dr. John Brown, formerly of Shaughnessy Hospital and more recently at Sooke Harbour on Vancouver Island, is now at Bralorne.

* * * *

Dr. Allan Beech, after considerable period of service with the R.C.A.M.C., has returned to civil life and has accepted an appointment as medical officer in the Industrial Medical Service in the North Vancouver shipyards.

Dr. Earle Hall of Vancouver also spent a short vacation in the Kootenay country.

* * * *

Dr. E. H. Cooke is also doing Industrial Medicine at the West Coast shipbuilding plant. Other doctors involved in the shipbuilding programme of Wartime Merchant Shipping Limited, of which Dr. W. G. Saunders is the Director, are: Doctors J. Scovil Murray, formerly of Calgary, and W. Sager, formerly Health Officer in Burnaby.

* * * *

Since Dr. R. H. Fraser has become a Medical Officer, R.C.A.M.C., Dr. Agnes Black is Medical Officer of Health in Burnaby.

* * * *

Major W. M. Carr, since leaving the Service, has been serving with Wartime Merchant Shipping in the Industrial Hygiene programme, and has now returned to Victoria and will serve the shipyards there.

* * * *

Dr. J. M. Burnett of Greenwood visited Vancouver recently. Greenwood is quite a busy spot with the augmented population due to Japanese evacuees.

* * * *

Dr. C. T. Hilton of Port Alberni called at the office when last in Vancouver. He reports that the doctors are very busy in that area.

* * * *

Dr. Murray Blair and family have been having a well-earned rest at Okanagan Landing near Vernon.

* * * *

Dr. Howard Spohn has been rusticating on a ranch near Cranbrook.

* * * *

Dr. K. D. Panton has not yet reported on the fishing at Buccaneer Bay.

* * * *

Dr. F. J. Buller has been at his summer cottage at Roberts Creek.

* * * *

Dr. Anson Frost has returned refreshed after a few weeks spent at Hope.

LIBRARY NOTES

Recent Accessions to Library—

Surgical Clinics of North America. 1. Symposium on Surgery of the Biliary Tract. 2. Symposium on Surgical Diagnosis.

Diseases of the Nose, Throat and Ear, 8th ed., 1943, Ballenger & Ballenger.

Collected Papers of the Mayo Clinic, v. 34, 1942.

Chemotherapy of Malaria, by James H. Williams.
(Donated by Lederle Laboratories, Inc.)

MISSING FROM LIBRARY

The following books and journals are missing from the Library, no record having been made by the borrowers:

Obstetrics and Gynæcology, 6th ed., DeLee.
Vaginal Hysterectomy, Kennedy and Campbell.
American Journal of Obstetrics and Gynæcology, vol. 43, 1942.
Bulletin of the New York Academy of Medicine, April, 1943.
It is urgently requested that they be returned to the Library at once.

British Columbia Medical Association

ANNUAL MEETING

Sept. 8, 9, 10

IN VANCOUVER,

with Headquarters at

Hotel Vancouver

Speakers:

DR. WILLIAM BOYD

Dr. William Boyd, Professor of Pathology, University of Toronto, requires no introduction. Dr. Boyd will give two lectures, address the Public Meeting and be the Guest Speaker at the Annual Dinner. He is Chief of the Department of Cancer Control of the Canadian Medical Association.

His lecture subjects are:

1. Recent Advances in Cancer Research.
2. Tumours of the Neck.

DR. F. R. FARQUHARSON

Dr. R. F. Farquharson, Assistant Professor of Medicine, and Head of the Department of Therapeutics, Faculty of Medicine, the University of Toronto. Doctor Farquharson (or "Ray" as some of his Toronto contemps call him) was here as Summer School Speaker in 1938, and is well remembered and his return as Lecturer at the September Meeting is welcome.

His subjects will be:

1. Pituitary Syndrome.
2. Physical Manifestation of Emotional Disorders.
3. Anæmia.

DR. D. SCLATER LEWIS

Dr. D. Sclater Lewis of Montreal is President of the Canadian Medical Association and Professor of Therapeutics, Medical Faculty, McGill University. Doctor Lewis will be accompanied by Dr. T. C. Routley, General Secretary of the Canadian Medical Association, and is travelling throughout Western Canada and will be in attendance at the Annual Meetings of the four Western Divisions, all of which are held in September, British Columbia Division at Vancouver, commencing September 8th, 9th, 10th, and then to Calgary, to Regina, and finally at Winnipeg.

Dr. Lewis and Dr. Routley will address the Official Luncheon on the first day and will attend the Annual Meeting on the first evening; will attend a meeting of the Board

of Directors; will address the Public Meeting; will participate in the Round Table Conference on Medical Economics and Health Insurance on the second evening; will be present at the Annual Dinner on the final day of the Meeting. On the lecture programme Dr. Lewis' subject will be "Idiosyncrasies to Drugs."

DR. G. GAVIN MILLER

Dr. G. Gavin Miller of Montreal is Associate Professor of Surgery, Faculty of Medicine, McGill University.

Doctor Miller is the Surgeon in the Canadian Medical Association party which comprises Doctors Sclater Lewis, R. F. Farquharson, William Boyd, Gavin Miller and Clarence Routley.

Doctor Miller's subjects for the Lecture Programme are:

1. Personal Experiences and Conclusions in Gastric Surgery.
2. Recent Experiences in Burn Therapy.
3. Useful Methods for the Diagnosis and Treatment of Ano-rectal Conditions.

DR. T. C. ROUTLEY

Dr. T. C. Routley, the General Secretary of the Canadian Medical Association, brings much helpful information to the Divisions. The British Columbia Division is glad to welcome him once again. The tremendous amount of work which war and post-war planning brings finds him ever willing.

Both Doctor Routley and Dr. Sclater Lewis will address the Official Luncheon on the first day of the Annual Meeting.

Doctor Routley will attend the Annual Business Session on the first evening; will participate in the programme of the Economic Session; will attend the Annual Dinner; will attend the meeting of the Board of Directors and several other meetings.

DR. R. R. STRUTHERS

Doctor Struthers of Montreal is Professor of Pædiatrics, Medical Faculty, McGill University. To find that Doctor Struthers was willing to travel to Vancouver was welcome news. He has held a warm place in British Columbia since he came to Vancouver as speaker at the Summer School in 1941.

Doctor Struthers has had an excellent opportunity to study the results to be achieved by the timely use of the Nurse Kenney method of treatment in cases of Acute Poliomyelitis. He will lecture on that subject.

His second lecture will be of widespread interest.

His subjects for Lecture Programme are:

1. Poliomyelitis.
2. The Obese Child.

Doctor Struthers will give a clinic at St. Paul's Hospital on Friday, September 10th, and will deal also with the subject: "A Classification of Diarrhœal Diseases."

COL. W. P. WARNER

Col. W. P. Warner, R.C.A.M.C., by consent of Brigadier J. C. Meakins, Ottawa, and through arrangement with Col. Wallace Wilson, C.M.O., Pacific Command, will be in Vancouver and participate in the Lecture Programme.

Col. Warner is Consultant in Medicine to the Canadian Army. In pre-war years he was widely known as Associate Professor of Medicine at the University of Toronto.

LIEUT.-COL. D. H. WILLIAMS, R.C.A.M.C.

Colonel Williams will be present at the Annual Meeting and will address the Public Meeting to be held on the second evening. He will also present a paper on the lecture programme on Friday morning.

"Don" not only deservedly holds a warm place in the hearts of the profession in British Columbia but has found an opportunity to make a fine contribution in the service of Canada.

THE PUBLIC MEETING

A Public Meeting will be held on Thursday, September 9, which will be addressed by:

Dr. Howard Spohn, President of the British Columbia Medical Association, who will be the Chairman.

Dr. Sclater Lewis, President of the Canadian Medical Association.

Dr. William Boyd, Head of the Department of Cancer Control of the Canadian Medical Association.

Dr. W. H. Hatfield, Director of the Division of Tuberculosis Control.

Dr. C. E. Dolman, Head of the Department of Preventive Medicine and Bacteriology at the University of British Columbia and Director of Provincial Laboratories.

Lieut.-Col. D. H. Williams, R.C.A.M.C., who has been during several years the Director of the Division of Venereal Disease Control in the Provincial Department of Health.

Dr. G. F. Amyot, Provincial Health Officer, will close the meeting.

The Provincial Department of Health is deeply appreciative of this feature of the Annual Meeting.

Dr. Ethlyn Trapp is in charge of arrangements.

CLINICS

At the Vancouver General Hospital on the first day, Wednesday, September 8th.

At St. Paul's Hospital, on Friday, September 10th, Dr. Struthers will hold his Clinic.

DEMONSTRATIONS

On Thursday afternoon those who are not playing golf will find a warm welcome at the Open-house Sessions at:

1. 2.30 o'clock—The Tuberculosis Division, Chest Hospital, 2647 Willow Street.
   Dr. W. H. Hatfield, Director.
2. 3.30 o'clock—The British Columbia Cancer Institute, 685—11th Avenue West.
   Dr. Ethlyn Trapp, Medical Superintendent.

These three splendid institutions would be glad to have large numbers attend this feature of the Annual Meeting.

At the British Columbia Cancer Institute the British Columbia Medical Association has arranged that tea will be served. Dr. Ethlyn Trapp and the staff at the Institute have kindly agreed to provide this closing refreshment to the afternoon sessions.

ANNUAL MEETING

Annual Business Meetings of the College of Physicians and Surgeons of British Columbia which comprises every registered doctor in this Province and of the British Columbia Medical Association. These meetings should be largely attended.

MEDICAL ECONOMICS AND HEALTH INSURANCE

Thursday evening will be devoted to this important subject.

This meeting will take the form of a Round Table Conference. Questions are invited and should be submitted by the members on the card which has been sent to you.

Such a conference should attract a large attendance. Such a session is timely.

The Committee on the Study of Economics is busily engaged in arranging this Conference. Both Doctors Sclater Lewis and Routley will be available and join the group on the platform.

GOLF

Golf will be played on Thursday afternoon. The Association Trophy and other prizes will be played for. Dr. D. Fraser Murray is in charge of arrangements.

ANNUAL DINNER

The Annual Dinner will be outstandingly good. It will be held on Friday, September 10th. Dr. William Boyd will be the guest speaker and this should not only guarantee a good attendance but assure a very worthwhile address.

LADIES

Mrs. Howard Spohn, the wife of the President, will be supported by a local commitee which will provide a very inviting programme of entertainment for the ladies, who it is hoped will join in this feature of the Annual Meeting.

Mrs. Wallace Wilson has agreed to be General Convener of the Ladies' Local Committees. At this date all arrangements have been made for two large functions in which all the ladies are urged to participate.

On Thursday afternoon at 3 o'clock the ladies will be the guests of the British Columbia Medical Association at a Reception and Tea at the Vancouver Art Gallery. This should be largely attended. It promises to be an affair which all ladies will enjoy.

The Ladies' Dinner will be as well done as usual, and they always have a good time. The Social Suite provides most suitably for such a happy affair.

Mrs. Wilson has kindly arranged a tea at her home on the Wednesday for the wives of Members of Council and of the Board of Directors of the British Columbia Medical Association.

The only request the Ladies' Committees make is that the doctors' wives accompany their husbands to Vancouver and attend these delightful parties.

Dr. Alan Y. McNair, Chairman of the Committee on Programme, and the members of his committee have arranged an excellent programme and your attendance will help to repay them for their efforts.

The 1943 Annual Meeting promises to be the best ever.

We should attend.

It would be well to post at your home and office the phone number of the Registration Desk: MArine 4184.

EXTRACT FROM LETTER FROM THE PRESIDENT OF THE PHARMACEUTICAL ASSOCIATION OF BRITISH COLUMBIA

"Dear Dr. Thomas:

"On instructions of the Council of the Pharmaceutical Association of the Province of British Columbia, I have been asked to bring to the attention of your office a matter that is causing considerable concern amongst the druggists of the Province and would ask your co-operation in this matter.

"Recently benzedrine inhalers were added to the list of restricted drugs under Order in Council 8443 and can no longer be purchased, as you know, other than through a doctor's prescription due to the abuse of this drug. The Council's attention was drawn to this fact that doctors were prescribing benzedrine inhalers or similar products in indiscriminate quantities and to persons who were using them not for their legitimate purpose.

"It was felt that in bringing this to your attention you may be able to advise the doctors of this Province in this regard and to ask their co-operation that this type of drug be not prescribed indiscriminately but only as is required by the patient. You undoubtedly know that this drug is being used by persons who can no longer obtain norcotics and phenobarbiturates, and is being used by such persons as a substitute.

"In bringing this matter to your attention I feel sure that the medical profession of the Province of British Columbia is only too willing to co-operate and that possibly they do not fully realize to what extent the abuse of this particular drug has been put.

"Thanking you in this matter."

COMMITTEE ON NUTRITION

The Committee on Nutrition of the British Columbia Medical Association recommended to the Ration Officer of Wartime Prices and Trade Board that conditions in which extra meat rations may be required would include the following:—

1. Diabetes Mellitus
2. Malnutrition—
   Nutritional Anæmia
   Nutritional Oedema
3. Pregnancy and Lactation.
4. Cœliac Disease—
   (Non-tropical sprue)
   Tropical sprue
5. Certain Food Allergies.

The following extracts from letters received from the Wartime Prices and Trade Board contain certain information regarding meat rations and procedures to be followed in prescribing extra rations of other commodities:—

"At the present time, diabetic and cœliac diseases are the only ones for which extra meat is granted on medical grounds."

Authorizations should contain the following information and be addressed to the Wartime Prices and Trade Board in the district in which the doctor resided. Branches are located in Vancouver, Victoria, Prince Rupert and Kelowna:—

"1. Name and address of patient.
2. Disease from which the patient is suffering.
3. Quantity of extra rationed commodities the patient requires. (The doctor can bear in mind that each patient has a Ration Book, and consequently can procure from this Ration Book one half pound of butter per week; one ounce of tea per week; one half pound of sugar per week; and two coupons of meat per week.)
4. Length of time for which the patient will require such extra rationed commodities."

Extract from letter from Dr. Routley.

RE REPAIRS TO DOCTORS' CARS

"I checked on the situation this morning and I find that there are three classifications set up for car repairs:

(1) The Armed Services;
(2) Essential services such as Doctors;
(3) All others.

"It would appear, therefore, that a Doctor should not have to wait for repairs unless the time of the garage is completely occupied on military cars."

PROVINCIAL BOARD OF HEALTH

The BULLETIN gladly publishes a communication received from Dr. D. E. H. Cleveland, Acting Director Venereal Disease Control of the Provincial Department of Health. The article, which Dr. Cleveland has abstracted, is indeed well worth our careful reading and consideration.

PROVINCIAL BOARD OF HEALTH
DIVISION OF VENEREAL DISEASE CONTROL

Editor, BULLETIN OF THE VANCOUVER MEDICAL ASSOCIATION.

Dear Sir:

In the *Journal of Social Hygiene* (Vol. 29, April, 1943, page 225) an article appeared by a Mr. Alan Johnstone, General Counsel, Federal Works Agency, Washington, D.C., entitled "The Law and Social Hygiene." This was an address given at the Southeastern Conference on Social Hygiene Day, Atlanta, Georgia, February 3, 1943.

I considered this article, which was quite brief, very well done, and it presented most forcibly and picturesquely many viewpoints which the Provincial Health Department holds. I therefore excerpted from this article those which I considered were most valuable and applied most closely to our British Columbia viewpoint, and suggested to Dr. G. F. Amyot, to whom I sent my excerpts, that if the permission of the Editors of the *Journal of Social Hygiene* and the permission of the author could be obtained, it might be published in the BULLETIN.

Dr. Amyot thought this was an excellent idea and I have now obtained the desired permission from the American Social Hygiene Association, Inc., asking that proper credit be given to the Journal of Social Hygiene, and I have also obtained the permission from the author.

D. E. H. CLEVELAND, M.D.,
Acting Director, Venereal Disease Control.

THE LAW AND SOCIAL HYGIENE

ALAN JOHNSTONE
General Counsel, Federal Works Agency, Washington, D.C.

As I came to your great city of Atlanta today to take part in this meeting I thought back twenty-five years. Just that long ago I was sent here by the Government to attack the same problem that we consider now. We were at war then as we are today. Then, as now, the venereal diseases were the greatest cause of disability in the armed forces.

This plague came to the army from civil life. The army cured its soldiers. It taught them the truth about these scourges. It combatted false standards of conduct and health. Men learned to respect their bodies and to think of their future wives and children. I heard a great Commander of that day—Leonard Wood—say, "Do not think of these men in uniform as anything less than the best you have got, and they will rise to the level of your opinion." Under the leadership of two great War Ministers, Baker and Daniels, America organized the cleanest, fittest Army and Navy the world had ever known. And after peace the services kept up their fight for fitness so that in 1939 the venereal rate of the Army reached the all time low of 30 per thousand per year or 3 per hundred per annum. The military authorities made great strides. And so did the civil population—but not so great. When the war clouds gathered again and men came to the army from civil life the rate increased by more than 25 per cent to 42.5 per thousand per year. Since then it has declined and is still declining as military and civil action has quickened.

* * * *

I speak of the role of the law in the eradication of these plagues. The law is the expression of the public opinion and conscience of the people. For a hundred years and until well after the turn of the present century we derived our opinion and schooled our conscience on sex morality and hygiene from two great characters in modern history. They were Napoleon Bonaparte and Queen Victoria.

Napoleon was a great commander and a great lawyer. He conquered Europe and dictated the Code Napoleon—the finest re-statement of the Roman or Civil law. But in sex he took counsel of his lust. And on the hygiene of sex he surrendered his great mind to a complete fallacy. He said that there was a sex necessity in men which made promiscuity inevitable and he formalized the double standard of sex morality. He established a system of public toleration and regulation of prostitution with medical inspection and certification of prostitutes. The first doctrine demoralized modern France. The second poisoned its blood stream. When Abraham Flexner and Raymond Fosdick published their scientific studies on "Prostitution and Police Systems in Europe," they showed what havoc the Napoleonic system had wrought on the police, the courts and the public health. And at the second battle of the Marne, when France nearly fell in the first world war, she had more soldiers in hospital from venereal infection than from battle wounds.

Victoria was a woman of great strength of character. She established a great commonwealth of nations in which the humanities played an important if not the dominant role. She respected order and enforced it. So great was her respect for order and for decency that when disorder appeared which she could not conveniently correct she ignored it. When indecency occurred that baffled her administration she convinced herself that it did not exist. Influenced by these attitudes we established a public silence on these great scourges and spoke their names only in whispers.

Napoleon's system was seductive. It was thought to be smart to believe in it. Victoria's attitude was smug and comfortable. Under it we cleansed ourselves by simply ignoring the dirt.

We in the United States embraced both systems. We passed laws to satisfy our Victorian ideas. We failed to enforce them to satisfy our Napoleonic complex. And so when Lord Bryce examined our institutions and wrote his critique on municipal government he exposed the rot at the core of our great cities. Organized vice was so entrenched that it was big business. Public officials who had the courage to expose it and to suppress it had to run the gauntlet. They were blamed by good people for stirring up scandal. They were attacked by the underworld in the political forum. Some of them were actually indicted in the courts for doing their plain duty.

* * * *

Let me be emphatic if not dogmatic.

The venereal diseases are first rate threats to our safety.

They can be controlled.

They are spread by prostitution and promiscuity.

Men and women who exploit the weakness of their fellows and profit from this filthy business are common outlaws and ought to be stopped in their tracks.

Toleration and attempts at regulation have uniformly failed.

However pitiful the victims of this traffic, they should be apprehended, treated and cured.

We quarantine for smallpox. How foolish to allow carriers of the big pox to roam at large.

The human rights and public safety involved in the enforcement of these laws demand the attention of the best minds in the legal profession and among our public servants.

Our police and our courts need the support of an informed and active public opinion.

* * * *

A soldier and a sailor are precious fighting units. They must not be disabled from community neglect.

We are getting ahead with the war. But we need all our strength to win it.

In the issue lies all that we have.

Our men are dying on land, on sea and in the air. But, God rest their souls, freedom is living!

We shall need all our sagacity and force to secure the peace.

The well springs of the race must not be polluted.

Our liberties are dear to us. Our destiny in the world is great.

The President has said that this generation has a rendezvous with destiny.

We must not fail that rendezvous.

College of Physicians and Surgeons

President ..... Dr. F. M. Bryant, Victoria
Vice-President ..... Dr. H. H. Milburn, Vancouver
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Registrar ..... Dr. A. J. MacLachlan, Vancouver
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MEDICAL SERVICES ASSOCIATION

The employees of the following firms are now enrolled as members of the Medical Services Association. The M-S-A has had a number of inquiries regarding the groups covered and the list below is published for the information of doctors. It is interesting to note the widespread and increasing growth of this service which means that it satisfactorily fills a need.

INDUSTRIAL:

Allard Machine Works Ltd.
Burrard Rivet & Forgings Ltd.
Clarke Bros. Timber Co.
G. H. Cottrell Ltd.
Dominion Bridge Co. Ltd.—
Burnaby Bridge Plant
Dominion Rustproofing Co. Ltd.
Dominion Steelweld Co. Ltd.
Crossman Machinery Co. Ltd.
Electric Power Equipment Ltd.
Graham Electric Co. Ltd.
Hayes Manufacturing Co. Ltd.
M. B. King Lumber Co. (North Shore) Ltd.
Kootenay Engineering Co. Ltd.
Morrison Steel & Wire Co. Ltd.
Pacific Coast Terminals Ltd.
Rat Potage Wood & Coal Yards Ltd.
Arrow Transfer Co. Ltd.
Westminster Canners Ltd.
Heaps Engineering (1940) Ltd.
Lawrence Manufacturing Co. Ltd.
Langley Manufacturing Co. Ltd.
Letson & Burpee Ltd.
Mohawk Handle Co. Ltd.
Mohawk Lumber Co. Ltd.
Pacific Pine Co. Ltd.
Pacific Veneer Co. Ltd.
Patterson Boiler Works Ltd.
Prefabricated Buildings Ltd.
Shell Oil Co. of B. C. Ltd.
Stewart Sheet Metal Works Ltd.
Vancouver Breweries Ltd.
Wartime Metals Corporation—Emerald
Tungsten Project
Westminster Hog Fuels Ltd.
Tyee Machinery Co. Ltd.
Coates Ltd.
McKay & Flanagan Bros. Lumber Mill Ltd.
Wartime Metals Corporation—
Kootenay Florence Project.

MERCANTILE AND SALES OFFICES:

Ash Temple Co. Ltd.
Henry Birks & Sons (B.C.) Ltd.
Canadian General Electric Co. Ltd.
Fleck Bros. Ltd.
Associated Dairies Ltd.
Fraser Valley Milk Producers' Assn.
Galbraith & Sulley Ltd.
International Business Machines Co. Ltd.
Fred C. Myers Ltd.
Powell River Co. Ltd.
Seaboard Lumber Sales Co. Ltd.
George Straith Ltd.
Victor X-ray Corporation of Canada Ltd.

FINANCIAL:

Blane, Fullerton & White Ltd.
Confederation Life Association
General Accident Assurance Co. of Canada
Great West Life Assurance Co.
Montreal Life Insurance Co.
H. A. Roberts Ltd.
Travelers Insurance Co.
Sun Life Assurance Co. of Canada
Retail Credit Co.
Yorkshire & Pacific Securities Ltd.
Marsh & McLennan Ltd.

OTHER:

Crehan, Meredith & Co.
Cowichan School Teachers' Assn.
Creston Valley United School District.
Canadian Association of Social Workers—
B. C. Mainland Branch
Agriculture, Food & Drug Division Employees
Campbell & Smith Ltd.
Dominion Income Tax Employees
Income Tax Specialists Ltd.
News-Herald Ltd.
MacKenzie and Son Ltd.
Metropolitan Health Committee
North Vancouver General Hospital.
Port Coquitlam School Board.
Robertson, Douglas & Symes.
Vancouver Board of Trade
Victoria City Hall Employees.
Roy Wrigley Printing & Publishing Co. Ltd.
Western Sales Book Co. Ltd.
Young Women's Christian Association.

Vancouver General Hospital

A HEART ATTACK IN YOUTH

LIEUT. H. G. WEAVER, R.C.A.M.C.

(Formerly Senior Resident in Medicine, The Vancouver General Hospital)

Mr. G. S., at the age of 27 years, was in good health in July, 1940. He was working hard and keeping late hours. In August, 1940, while working one morning, he had a twinge of pain in the lower left axillary region. He paid no attention to it, as he had had such pain before. After lunch he experienced a peculiar sensation described as a lump forming in his upper abdomen. He says he actually felt as if he might die. A short time after this he suddenly had excruciating pain in both elbows; it was accompanied by a similar but not so severe pain in both knees. His memory of this attack is not too clear, but he had pain in his chest as well. That afternoon he was admitted to a small country hospital, still complaining of substernal pain and pain in most of his joints. The temperature and pulse rate were normal, but they gradually and steadily rose to a temperature of 103° and a pulse rate of 120 in five days. He had a troublesome, mildly productive cough and was dyspnœic. No diagnosis was made so after five days he was sent to a hospital in a nearby city. On admission his temperature was 101.6° by rectum, he was markedly short of breath, and his heart was enlarged, a gallop rhythm being present. No pericardial friction rub was heard. An eltctro-cardiograph was taken in which the T wave of the first lead was inverted with a slightly high take-off, the QRS complex in lead 3 was inverted, and the T wave in the fourth lead was inverted with a high take-off. This suggested the presence of an acute anterior myocardial infarction of recent occurrence, with left axis deviation. A radiograph was taken which showed fairly marked general enlargement of the cardiac shadow, especially in relation to the left ventricle, with increased pulmonary markings. The next day, on the seventh day of his illness, his temperature dropped to normal, and stayed there except for an occasional spike to 99°. The dyspnœa was not present after the seventh day of illness. A white blood count on the sixth day was 22,500 and it remained elevated at 13,000 to 14,000 throughout his six to seven weeks in hospital. History revealed that he had never had joint pains before. He had suffered from frequent colds for many years, during which he would have a mild productive cough. He had measles and mumps when a child. His father, aged sixty-sixx, had a "leaking heart valve." There was an indefinite history of tuberculosis on the maternal side of the family.

After discharge from hospital the patient stayed in bed at home for another six weeks, after which he got up and around by degrees. Since that time he has considered himself a semi-invalid. He moved to Vancouver in July, 1941, and reported to the Out Patients Department of the Vancouver General Hospital. Electro-cardiographs were taken which confirmed the previous impression of anterior myocardial infarction, but there was still a high take-off in lead four and a deeply inverted T wave, which is an unusual finding one year after an attack.

In March, 1942, the electro-cardiograph showed left axis deviation with inversion of the T wave in leads one and four, but the ST segment in lead four had become flattened and this was interpreted as a healing infarction. A radiograph showed marked diminution in the size of the cardiac shadow as compared with previous radio-graphs and the pulmonary markings had returned nearly to normal.

In July, 1942, he was working as a hotel clerk but complained frequently of palpitations and some dyspnœa after exertion. During that month he had an attack of diarrhœa which lasted about one and one-half weeks and he complained of some lower back pain.

On August 10, 1942, almost exactly two years after the first heart attack, he was suddenly stricken with an acute pain in his left anterior chest. It was not similar to the previous attack in 1940. The pain did not radiate and was much worse on the slightest movement. He was admitted to hospital at which time his temperature was 101°, pulse 110 and regular. Examination revealed a heart clinically enlarged, a definite pericardial friction rub was heard over the precordium, which was enhanced when the patient held his breath. The blood pressure was 120/74. The white blood cell count was 21,500 and the sedimentation rate was 3 mm. in three-quarters of an hour; the urine was normal specific gravity, plus 2 albumin, and a few white blood cells. A portable radiograph showed very marked general enlargement of the cardiac shadow and suggested the possibility of pericardial effusion. The pulmonary markings were not increased as in the previous examination in August, 1940. The pain diminished on the second day, the friction rub disappeared after the third day and the pain was entirely absent after five days. The temperature went as high as 102.2° on the fourth day, but dropped to normal on the seventh day and stayed there. The white blooud cell count came down to 8,500 in one week, at which time the sedimentation rate was 41 mm. in three-quarters of an hour. A tuberculin test using 1/1000 old tuberculin was negative. The sedimentation rate was down to 6 mm. in one and one-half months, and the patient was allowed up after a little over two months. A radiograph two months after the attack showed definite decrease in the size of the cardiac shadow and it was quite similar to the one taken in August, 1940, and August, 1942. The electro-cardiograph was interpreted as being a healed anterior infarction.

Since discharge from hospital, the patient has been up and around at home. He feels quite well and has gained weight. He has not yet gone back to work but can walk eight blocks without being dyspnœic. However, when the patient over-exerts himself he feels quite fatigued the next day.

A radiograph taken in April, 1943, shows no essential changes from the previous one. An electro-cardiograph taken in April, 1943, shows some change since 1942. Previously the T wave on the second lead has varied, being upright on several occasions and inverted on at least one occasion. T 1, 2 and 4 are now inverted, the left axis deviation is still present and there appears to be some evidence of possible left ventricular strain.

While this case is not the youngest case of myocardial infarction on record, it is important to keep in mind that such conditions can occur in the younger age group, before arteriosclerosis usually appears. There has been some discussion as to the actual etiology of this particular case. Two suggestions have been offered: (1) That it was due to hardening of the coronary arteries. The patient has had albuminuria and this might indicate some arteriosclerotic change but the radial arteries are soft. (2) That it was due to inflammation of the same arteries. However, at this time it is difficult to elaborate further on these two possible factors.

In summary, the patient, aged twenty-seven years, had an acute heart attack in August, 1940, which was diagnosed as an acute anterior myocardial infarction. The fever was somewhat higher and more prolonged than is customary in such an attack. He complained of substernal pain, pain in his elbows, and in addition had pain in other joints. Two years later, in August, 1942, he had an acute heart attack definitely diagnosed as acute pericarditis. He appears to be recovering from this attack.

EVALUATION OF RENAL FUNCTION

A discussion of physiological applications in the assessment of kidney damage.

By MAX M. CANTOR, B.Sc., M.D., F.A.C.P.

Assistant Professor of Biochemistry, University of Alberta.

(Read to the Summer School Session of the Vancouver Medical Association, Vancouver, June 23rd, 1943.)

Effective therapy depends upon early diagnosis. Tests which show impaired function of an organ before clinical symptoms appear are thus of great value. While many tests for the evaluation of the function of many organs have been described, those of great clinical importance concern the kidney, the liver and the endocrine glands. Tests of function do not always differentiate between organic and functional disease but they do provide a numerical expression of the amount of functioning tissue. No one function test can indicate that alterations have taken place in other functions of the same organ to the same degree—one does expect, however, that good tests of the same function should show reasonable agreement and their value is based on such confirmation.

No discussion of renal function tests is complete without a few preliminary notes on the modern theories of renal physiology which provide the bases for function tests.

The modern theory of renal function evolves from the work of Richards, Marshall, Smith, Shannon and others. It is the theory originally proposed by Heidenhain, modified by Cushny and proven by these observers. The function of the kidney suggests that we are dealing with an extremely complex organ. In reality this is not so. The kidney is composed of about one million units—nephrons—which consist of a capillary tuft or glomerulus attached to an unbranched tubule which may be divided structurally into three distinct parts. These tubules drain into collecting ducts which find exit into the renal pelvis.

Formation of urine begins in the glomerulus and passes into Bowman's capsule and thence along the tubule into the pelvis of the kidney. When the tubule leaves Bowman's capsule it forms a series of convolutions (proximal convoluted tubule) and then descends in a straight line to the pelvis of the kidney (proximal tubule). Here it reverses its direction (Loop of Henle) and returns as the distal tubule to the region of the glomerulus where it forms another series of convolutions (distal convoluted tubule). From this region is joins a collecting duct. Both convoluted tubules form a network about the glomerulus which gave them origin. The organization of the nephrons in pyramidal lobes has no physiological function. The cortex is composed of the glomeruli and convoluted tubules and the medulla represents the proximal and distal tubules and the loops of Henle. From the point of view of function, there is no difference between the convoluted tubules and the partes rectae.

The tubular portion of the nephron is divisible histologically into three parts, the proximal tubule, the thin segment of Henle's Loop and the distal tubule. A complete discussion of the histological differences is beyond the scope of this paper—suffice it to say, however, that each segment has specific functions, the total of the whole being the basis of the modern conception of renal physiology.

In this theory, as the blood courses through the glomerular capillaries, part of the plasma water is filtered out into the capsular spaces of the glomeruli. Along with water, which represents about 20% of that of the plasma, come glucose, salt, urea, uric acid, creatinine and other crystalloids which are present in the plasma. The fluid, however, contains no protein and no colloidal material. These filtered substances are present in the capsular fluid in about the same concentration as they are in the plasma and the pH of the fluid, like the plasma, is about pH 7.4. The blood pressure, 55 mm. Hg. in the glomerular capillaries, is high enough to produce filtration against the osmotic pressure (25 mm. hg.) of the blood and it is generally agreed that the capsular material is a protein-free fluid produced by mechanical physical filtration from the blood by filtration

pressure (30 mm. hg.). The rate of glomeular filtration is about 6 L. per hour or 150 L. in 24 hours.

The capsular filtrate contains in it not only waste products such as urea and uric acid but substances such as glucose, and bicarbonate which the body needs. Furthermore, the water in which these are dissolved and salt are excreted in larger amounts than the body can afford to lose. In order to get rid of waste products yet conserve the essential materials, the cells lining the tubules exert a selective action in reabsorption depending upon the body's requirements.

The re-absorption of water, which is the most important constituent of the filtrate, probably occurs along with the reabsorption of glucose and essential electrolytes and is made possible by the reabsorption of these. Marked absorption of water occurs in the thin segment and some further absorption is effected in the distal tubule. This obligatory reabsorption of water is enhanced by a facultative reabsorption under the stimulus of the antidiuretic hormone. The antidiuretic hormone is responsible for about one-fifth of the water reabsorbed while four-fifths of the glomerular filtrate are reabsobed isosinotically. The effect of the antidiuretic hormone is physiologically and anatomically independent of the obligatory reabsorption mechanism, and when it is deficient, gives rise to diabetes insipidus.

The reabsorption of chloride with its attendant sodium and other inorganic constituents occurs along the whole length of the distal tubule. Glucose is reabsorbed in the proximal tubule, the concentration diminishing rapidly as it moves down the tubule, and it is almost completely absorbed from the filtrate when it is only half way down. The distal half of the proximal tubule can, however, still absorb glucose, but none is absorbed from the distal tubule itself. Glucose is reabsorbed at a constant rate of about 200 mgm. per minute. Raising the concentration in the plasma and increasing as a consequence the amount in the glomerular filtrate, exceeds the capacity of the reabsorptive process and gives rise to glycosuria. This is the basis for the term renal threshold for glucose.

Phosphate is usually concentrated and excreted in the urine. Under certain conditions it may be required by the body, and in those circumstances it is reabsorbed in the proximal tubule in much the same way as glucose.

Urea in mammals is excreted entirely by glomerular filtration. It is, however, reabsorbed to a considerable extent by the tubules.

Apart from the power of selective absorption, the tubules have a reserve function of excretion. While this function is of no special importance and remains inactive in the excretion of natural urinary substances in humans, it is used to handle certain foreign substances which do not pass the glomerular filter. Thus phenolsulfonphthalein is excreted by this mechanism and it forms the basis of some function tests.

The pH of the glomerular filtrate is the same as that of the plasma. Freshly excreted urine has a pH of between 4.8 and 7.4 with an average of about 6. When the pH is lower than 7.4 the kidneys must be excreting acid products which if retained would lower the pH level of the blood. Even at the lowest pH of the urine strong acids are not present. Of the acid products excrete, acid phosphate $H(BHPO_4)$ is the most important. The relative quantity of acid phosphate and basic phosphate $B(BHPO_4)$ among others, determines the reaction of the urine. This adjustment, a process of acidification, takes place exclusively in the distal portion of the tubule, and in a segment nearer its distal end measuring no more than one-fifth of its extent. This segment does not show any outstanding histological features except a widening of the lumen but it can alter the reaction of a buffer from 7.5 to 6.8 in one minute.

From what has been said, it will be seen that the function of the kidney is threefold:

(1) The excretion of:
   (a) Waste products of metabolism.
   (b) Chemical and bacterial poisons which have been rendered innoxious by the liver.
   (c) Foreign substances.

(2) The regulation of osmotic pressure of the blood by the excretion or reabsorption of electrolytes, especially chlorides.

(3) Regulation of the hydrogen ion concentration (the reaction of the blood).

These functions are the basis of more than sixty tests, but only a few of these have been widely accepted. Of these, th Mosenthal test, Volhard and Fahr's concentration and dilution tests, the. P.S.P dye test, Van Slyke's urea clearance test, changes in the N.P.N. components of the blood and tests for pathological constituents of the urine. These tests may be divided into two groups:

(1) Those which measure glomerular function and which include the urea clearance and the blood N.P.N. components, and

(2) Those which measure tubular absorption such as the concentration test or tubular excretion as does the P.S.P. dye test.

Before dealing with these tests it should be emphasized that the performance of renal function tests in the certain absence of proteinuria is a complete waste of time. A routine urinalysis in full detail should be performed in all patients before attempting to assess kidney function.

A. Evaluation of Glomerular Function

1. *Tests of non-protein nitrogen retention* (*N.P.N.*).

These tests are dealt with first not because they are of primary importance but rather since they are commonly performed early in the investigation of renal failure. Actually they have only corroborative value with regard to renal function.

By non-protein nitrogen we mean nitrogen of the blood, tissues, urine or excreta which is not thrown down by the usual protein precipitants. Specifically we refer to the nitrogen in the protein-free filtrate of blood. This amounts to from 15 to 25 mg. % in the post absorptive state and represents ½% of all the nitrogen in the blood. The N.P.N. is composed of a mixture of heterogeneous substances including urea, ammonia, creatine and creatinine, amino acids, uric acid and several others. Expressed as nitrogen, urea accounts for nearly 50% of the N.P.N., the remainder being divided among all the others in amounts ranging from one to four per cent. The estimations most frequently called for are urea, creatinine and uric acid.

Creatinine.—The concentration of creatinine in health and in disease not involving renal function is remarkably constant. Blood retention occurs with gross kidney damage. Most of the extra-renal conditions which alter urea and uric acid have very little influence on creatinine. The chief value of creatinine determinations lies in the differentiation of urologic from medical-nephritic conditions. In nephritis there is a parallelism between the concentrations of creatinine and urea. In purely urologic conditions, such as obstructive lesaons of the lower urinary tract, no such parallelism exists.

Uric Acid.—Uric acid is a substance which has a peculiar fascination for the lay mind and patients often seek to impart information concerning it. There is something about uric acid as there is about alcohol which seems to turn the mildest man into the most heated partisan. Most authorities now regard uric acid and other purines as by-products of metabolism which are harmless in the majority of cases. It has special historical significance in being the subject of the first blood analysis ever attempted (Garrod, 1848). Uric acid in the urine is derived from two sources, exogenous from purine bases in the food and endogenous possibly from the breakdown of cell nuclei. Only part of the uric acid broken down is excreted by the urine, the remainder being destroyed elsewhere in the body. Birds and reptiles excrete uric acid as such. Mammals, with the exception of man and the chimpanzee, convert uric acid to allantoin for excretion. Man occupies an intermediate position: like mammals he forms uric acid from purines, but like birds, much of it must be excreted in the urines. In the process of evolution he has lost the ability to oxidize all the uric acid. This may explain man's peculiar liability to gout and urate calculi in the kidney.

Some physicians lay so much stress on the level of uric acid in the blood that it seems pertinent to spend a few moments on discussion.

Proteins and carbohydrates accelerate the excretion of uric acid while fats produce retention, due possibly to ketosis.

The normal level of uric acid in the blood varies from 2-4 mg. %. It is increased in starvation, exercise, in the last stages of pregnancy and delivery. The blood uric acid content at birth is the same in the maternal blood. It rises during the first three or four days of life, then falls gradually to reach the adult level at about the 8th day. These changes are probably associated with normoblast destruction. High values are also found in leukæmias.

The estimation of uric acid is performed most frequently in gout or suspected gout. Here values rarely exceed 10 mg. %. In advanced chronic interstitial nephritis, the level may rise as high as 20 or 25 mg %. In the lesser grades of nitrogen retention, values of the order of those found in gout are common. It should be recalled that chronic nephritis may co-exist with gout or with other arthritic disease, especially in elderly pateints. Pneumonia also produces a rise in blood uric acid and in susceptible individuals may precipitate an attack of gout.

In nephritis with impaired renal function the uric acid in the blood is at a higher than normal level but the rise shows no consistent relationship to the degree or progress of renal impairment. Because of this failure in consistency, many investigators have abandoned uric acid estimation as an aid in diagnosis of the nephritides. Thus in anuria following bichloride poisoning, there is no increase in uric acid, while in arteriosclerosis and cardiac decompensation without evidence of renal failure it is increased to an extent entirely out of proportion to the retention of other non-proteinnitrogenous substances.

Urea.—Urea ccupies a unique place in the development of human thought. Its synthesis by Wohler in 1828 was the first demonstration of the possibility of preparing artificially a substance elaborated by the living organism. In the philosophy of the last century it had an effect comparable to "Darwin's theory of the origin of the species and Pasteur's demonstration of the parasitic origin of pestilence." It shattered the entire vitalistic conception and laid the foundation for the extraordinary developments in synthetic chemistry—developments which have proven so epoch-making for medicine.

Its importance in the present discussion is threefold:

1. It is the chief end-product of protein metabolism.
2. It is the main metabolite which the kidney must eliminate.
3. It is a natural diuretic—exerting a continual effect on urine flow.

Its high nitrogen content (46.6%), its neutral non-toxic character and ready diffusibility make it peculiarly adapted for the excretion of nitrogen. In normal urine, urea accounts for nearly 90% of the nitrogen. Lowering the protein intake of the diet will decrease urea excretion without altering the level of the other nitrogen components. This illustrates the importance of considering the diet before interpreting the results of excretion tests. In addition to changes in available protein, urea excretion is modified in such conditions as acute yellow atrophy, where the ability of the liver to form urea is diminished, and in ketosis and acidosis where it is converted in part to ammonia for the neutralization of acid.

The concentration of urea in the blood is not especially influenced by mixed meals, but it is altered by great reduction in protein intake. Its blood level, in cases of renal disease on low protein diet, may thus be misleading. In uræmia, it is not unusual to find a high value for urea reduced to within normal limits by protein restriction. The same is true in prostatic obstruction with anorexia. For this reason some advocate the determination of blood urea one hour after administering a 15-gram test dose by mouth (under normal conditions of renal function this should double the blood urea).

One of the difficulties in interpreting the meaning of an increase in blood urea is the exclusion of non-renal factors. Any disease which is complicated by anhydræmia will show an elevation in urea in the blood. The same is true of low blood pressure, slowed circulation and severe bleeding into the gastro-intestinal tract.

The estimation of urea in the blood is a measure of the efficiency of nitrogen excretion and nothing else. Normally the level varies from 8-15 mg. %. This represents about 50% of the N.P.N. Where the N.P.N. is greatly elevated, most of the increase

is due to urea, so that in very high values this may represent 80% of the total. Values below 80 mg. % offer some difficulty in assessment, but there is no doubt about levels in excess of 100 mg. %. The estimation is of value in watching the progress of a case and as a guide to therapy. Taken either as part of the urea concentration test or together with a single estimation of urea in the urine, its value is greatly enhanced.

2. *Clearance Tests.*

Under this heading it is my purpose to deal with urea clearance only since it yields all the information usually necessary for the evaluation of glomerular filtration. It is necessary, however, to say something about the concept of clearance. The term clearance was first used in connection with urea and was defined as "the volume of blood which one minute's excretion of urine suffices to clear of urea." The volume is virtual rather than real, because all the blood flowing through is partially cleared but it is obtained by dividing the quantity of urea excreted per minute by the quantity contained in each cubic centimeter of blood. If "U" is the concentration of urea in the urine, "V" the volume of urine formed in one minute, and "B" the concentration of urea in the blood, then the mathematical expression UV/B describes the capacity of the normal kidney to excrete urea. It should be noted, too, that clearance by these terms also means "the minimum volume of blood required to furnish the quantity of a substance excreted in the urine in one minute's time."

Different substances are cleared at different rates and to different extents. This depends upon the degree of tubular reabsorption of this material. Thus a substance which is present in the glomerular filtrate but which is completely reabsorbed will have a clearance of zero. If tubular reabsorption of a substance is decreased, the substance will appear in the urine until if there is no reabsorption at all, the clearance will equal the rate of filtration (glomerular clearance). If a substance is excreted by the tubules as well as the glomeruli then the clearance will be greater than the rate of filtration—the limit being the amount of a substance which is brought to the kidneys by the blood. We do not know if the kidney can effect complete clearance.

Actually, the efficiency of the kidney in excreting most substances is not great and the clearance is much less than the blood flow. A substance suitable for measuring glomerular filtration should be one which passes through the glomerular filter easily and it should not be absorbed, excreted or synthesized by the tubules. It should be physiologically inert, and it should be one which is capable of being easily and accurately determined. Inulin has been used extensively in such determinations, but for clinical purposes, the facility with which urea is estimated makes its use preferable, although it does not quite meet all the criteria set out.

The excretion of urea in the normal adult is such that 75 cc. of blood are cleared in one minute. The actual amount cleared depends upon the urine flow. Below a certain level which is critical and which is called the augmentation limit (about 1.5 cc. of urine per minute), the urea excretion is related to the blood level in proportion of the square root of the rate of urine flow and UV/B becomes (UV/B) square root of 1/V or U square root of V/B. This is called the "standard clearance" as opposed to the "maximum clearance" where the urine flow is abundant. The mean normal value of the standard clearance is 54 cc. per minute. Expression of results is in terms of % of normal maximum or normal standard clearance, the values 75 and 54 respectively being taken as 100%.

It should be noted that the volume of blood cleared is the same irrespective of the level of the blood urea and regardless of the rate of urea formation. A clearance of 75 does not indicate complete removal of urea from that amount of blood in one minute. Actually this does not happen, but the theoretical conception is a useful measure of renal efficiency—the larger the volume of blood which could in theory be cleared, the more efficient the kidney. It is very much like removing eggs from baskets on an endless chain—the same result can be expected if 100 eggs are removed from one basket in a minute or 10 from each of 10 baskets in the same time.

Interpretation of Urea Clearance.—The urea clearance test is in reality a fractional retention test. Since it includes the determination of the ratio of the urea blood level

to the urea excreted by the kidney per unit of time it actually measures what has been called "the functional elasticity of the filtering mechanism" and gives a reliable picture of glomerular reserve. This ratio varies directly with the volume of blood flow and inversely as the infra glomerular pressure. Any alteration in the rate of flow such as might occur in shock, hæmorrhage, dehydration and cardiac failure, or any change in intra glomerular pressure as might occur in pyelonephritis and ureteral or bladder neck obstruction, will affect the clearance.

In partial destruction of the kidney parenchyma, the total filtration does not vary over long periods of time. This is evident from studies of blood non-protein nitrogen components which remain normal. This is so because the remaining glomeruli compensate by hypertrophy or overactivity. The urea clearance will, however, be diminished because the same amount of blood cannot be cleared per unit of time since there is no glomerular reserve.

The usual range of clearance in man is taken as between 70% and 130% of the average normal. With decreasing clearance in nephritis the variation is not so great. The lower limit of normal is generally taken at 70% although values as low as 30 to 40% of normal urea clearance may be consistent with fair health. There is always a danger in using mathematical terms in interpreting biologic tests since these are nearly always plus (or minus) 20%. The following values are, however, widely accepted:

Over 70 per cent	normal
70-40 per cent	mild deficit
40-20 per cent	moderate deficit
Below 20 per cent	severe deficit
Below 5 per cent	uræmic coma imminent or present

It is difficult to interpret accurately the urea clearance in nephritis in terms of glomerular damage. The proportion of destruction to urea clearance is not direct. The urea clearance falls quite slowly, about 20%, until there is about 50% glomerular destruction. After this the rate is increased rapidly.

B. Evaluation of Tubular Function

Although the tubules in humans are concerned predominantly with absorption of water and essential substances in the glomerular filtrate they have the reserve function of excreting foreign substances. Tests of tubular function are, then, of two types.

1. *Evaluation of tubular absorption.*

The logical test for this function ~~is~~ the ability of the tubules to concentrate the glomerular filtrate by the absorption of water under conditions of stress (water deprivation). The result as indicated by the specific gravity of the urine provides a test of this function.

In order to concentrate, the kidney performs thermodynamic work. It has been calculated that the urea, for example, which is present in 50 L. of blood (12 mg. %) can be concentrated by the normal kidneys in one litre of urine (600 mgm. %). This takes as much work as the compression of 6 litres of gas at atmospheric pressure into 1 litre at 6 atmospheres. Damage to the kidney interferes with this thermodynamic function. Glomerular filtration does not entail such a load (30 mm. Hg.) and it is for this reason that diminished ability to concentrate is often the earliest evidence of damage. For this reason, too, after recovery, persistent low specific gravity may be evidence of residual damage long after the urea clearance and N.P.N. have returned to normal. Thus failure to concentrate is evidence of kidney damage even in the face of normal urea clearance.

Decreased power of concentration, however, in the absence of diminished urea clearance is not as serious as when both are decreased. There are cases in everyone's experience of patients with persistently low fixed specific gravities who have carried on for years because glomerular function was able to compensate for the deficiency in concentration (polycystic kidney). Such individuals, however, are very susceptible to extra-renal influences which may affect their over-compensating glomerular function.

Normally at least one of the samples in the concentration test should have a specific gravity of 1.030 (or at least over 1.025). Values below this indicate failure of tubular function, 1.010 being taken as a uræmic level.

In interpreting concentration tests it should be recalled that the absorptive function is influenced by endocrine, nervous and metabolic changes. The excretion of œdema fluids may lower the specific gravity without indicating renal damage. Diabetes inspidus and an anæmia may also produce a low fixed specific gravity. The presence of protein in the urine will elevate the readings and corrections should be made to the extent of 0.003 for each gram of protein present in 100 cc. of urine.

2. *Evaluation of Tubular Excretion.*

This is generally measured by the excretion rate of phenolsulfonphthalein (P.S.P.) after intravenous or subcutaneout injection. Given intravenously and estimated in the urine at 15-minute intervals, it is said to be as sensitive as the urea clearance. By the subcutaneous route with hourly estimation in the urine it is definitely less sensitive than the urea clearance.

P.S.P. is excreted entirely by the tubules and measures their excretory ability as applied to a substance foreign to the organism. While tubular excretion probably parallels absorption it may not be so under stress. Estimations of the color are open to error and the method is not applicable when hæmaturia is present. So, also, in advanced nephritis the output of dye is so low that the test fails to indicate further change. The chief value is in following the progress of a case. Normally about 40-50% of the dye is eliminated in one hour and 60-80% in two hours. Values below 60 are considered abnormal.

Discussion

From what has been said it seems clear that the urea clearance and concentration tests should be the criteria for evaluating kidney function, the tests of nitrogen retention serving as corroborative evidence.

In acute nephritis a rise towards normal in urea clearance nearly always occurs within four months and is a good prognostic sign. Where no rise occurs, chronicity may be assumed. The return of clearance to normal, however, is no assurance of escape from chronicity so long as the concentration test shows a low specific gravity, and other abnormalities such as hæmaturia, casts and so forth are present.

In other words, a normal renal function test is not a clear indication of absent renal pathology.

Diminished renal function shown by these tests should not be interpreted as evidence of renal damage until extra-renal factors such as traumatic shock, gastro-intestinal obstruction and acute infection are eliminated. Reflex anuria following surgery also fits into this list of exceptions as do cases of obstructive uropathy (ureteral, prostatic or urethral).

A special note of caution should be added. Damaged kidneys are very susceptible to sudden changes in factors which control glomerular activity. In the presence of renal damage, operative procedures and anæsthesia should be chosen with that point in view. Vomiting dehydration following surgery and anæsthesia in a nephritic may cause renal failure and premature uræmic death.

In conclusion I would emphasize that the kidneys possess a great reserve for compensation and recovery. It has been estimated that nearly two-thirds to three-quarters of the kidneys must be functionless before there is evidence of renal failure by the best function tests. However, while our tests may not be absolute, they have great diagnostic and prognostic value. Used as criteria of therapy they are of equal importance.

RANGE OF SOME RENAL FUNCTION TESTS ASSOCIATED WITH VARYING DEGREES OF RENAL DAMAGE

Test	*Mild Deficit*	*Moderate Deficit*	*Severe Deficit*	*Uraemic Coma Imminent*	*Normal*
N.P.N. mg. %	30–50	35–75	45–150	100–450	20–30
Urea N. mg. %	15–30	20–50	30–110	60–350	10–15
Creatinine mg. %	1–2	2–3	3–8	6–35	1–2
Uric Acid mg. %	2–4	3–6	5–15	10–25	2–4
Urea Clearance %	40–70	20–40	10–20	0–5	70–130
P.S.P. % in 2 hours	45–75	25–60	5–45	0–25	60–80
Sp. G. in Conc. Tests	1.015–1.027	1.009–1.020	1.008–1.015	1.008–1.010	1.025–1.035

EVALUATION OF HEPATIC FUNCTION

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For more than twenty-five years attempts have been made to elaborate tests which would measure the functional capacity of the liver. The earlier difficulties concerning the nature of hepatic function have been dissipated by careful investigations on the effects following experimental, partial and total hepatectomy. As a result, tests became more accurate, but it is still necessary to warn that the status of one function provides no indication of the degree of effectiveness of any other function, and that the function tests singly or combined provide no evidence of abnormality until most of the organ is destroyed. This is so because the reserve function of the liver is very great and its recuperative power extensive.

The tests generally used measur only one function and indicate the degree of derangement in that regard only. For purposes of classification these are listed on the basis of what is known concerning liver function.

1. *Bile pigment metabolism and execretion:*
   (Van den Bergh, bilirubin, icterus index and urobilinogen in serum. Bilirubin and urobilin in urine). Bilirubin tolerance test.
2. *Function with respect to metabolism of:*
   (a) *Proteins and deamination:*
   (variations in plasma and excretion of amino acids proteins.)
   (b) *Carbohydrates:*
   (Laevulose and Galactose Tolerance Initial insulin hyper-glycæmia test.)
   (c) *Fat and cholesterol and Fat soluble vitamins:*
   Hemokonia test, cholesterol and cholesterol esters in plasma.
3. *Metabolism and excretion of bile acids:*
   (Bile acids in duodenal contens and in urine.)
4. *Excretory Function with respect to dyes:*
   (Rose bengal and bromsulfalein.)
5. *Function with respect to Coagulation Factors:*
   (Coagulation time, prothrombin time.)
6. *Function with respect to detoxification:*
   (Synthesis of hippuric acid.)
7. *Hematopoietic function.*
8. *Miscellaneous tests:*

   (Takata-ara, Weltmann reaction, colloidal gold test, appearance of erythrocytes.)

From this classification it will be noted that the liver has at least seven distinct functions and tests have been described for their measurement. From the nature of these it is unlikely that any test of one function can provide sufficient information about another function to be significant. By combining several of these tests we are provided with a somewhat clearer picture and such a procedure does assist in diagnosis and in prognosis.

1. *Bile Pigment Metabolism and Excretion.*

Hæmoglobin fro msenile erythrocytes is converted in the resticulo-endothelial system to hæmobilirubin. The chemical nature of this pigment complex has not been determined but it is probably associated with the lipoid globulin fraction of the plasma. It circulates in the blood and is responsible for the indirect Van den Bergh test. This pigment is taken up by the liver cells and is excreted into the bile capillaries as cholebilirubin. The chemical change which is effected is such that it now gives a direct Van den Bergh reaction. It is secreted into and concentrated by the gall bladder, whence it passes into the intestine. Here cholebilirubin is reduced by bacterial action to the chromogen stercobilinogen. Most of the stercobilinogen is excreted in the feces in the

oxidized form stercobilin. This material gives feces their characteristic color. The small amount which is not excreted is absorbed into the circulation. Much of this is returned to the liver and re-oxidized to bilirubin. Small traces remain in the blood and are excreted in the urine as urobilinogen. This is converted to urobilin on exposure to air and accounts for some of the color of urine. Normally then, the urine cantains traces of urobilin but no bilirubin. The feces contain large quntities of urobilin (stercobilin) but no bilirubin. The presence of bilirubin in the feces or urine or the presence of blirubin in the feces or urine or the presence of large quantities of urobilin in the urine and the absence of stercobilin in the feces signify a derangement in the bile pigment mtabolism.

Hæmolytic jaundice is associated with the formation of excessive amounts of hæmobilirubin. To compensate for this the liver excretes larger amounts of bilirubin. This increases the quantity of stercobilinogen formed and absorbed and results in increased renal excretion of urobilinogen. The feces are darkly colored. The blood serum gives an indirect Van den Bergh test.

The determination of serum bilirubin gives as useful information regarding liver function as does the estimation of urea in renal function. Generally speaking the Van den Bergh reaction by which the estimation is carried out differentiates between hæmolytic and obstructive jaundice. The presence of increased serum bilirubin depends on excess production and on the decreased excretory function of the liver cells. Actually pure forms of one or the other types of jaundice must be very rare. There is never gross obstruction to the bile passage without some evidence of injury to the hepatic parenchyma, conversely there is never pure hepatogenous jaundice without injury to or obstruction of the finer bile passages. It is more accurate to say that jaundice is preponderantly hæmolytic hepatogenous or obstructive depending upon whether one is dealing with a blood disorder, a parenchymatous lesion of the liver or an obstruction in the extra hepatic bile duct system. The differentiation is useful, however, for purposes of clinical distinction.

A direct reacting bilirubin is conclusive proof of injury of the liver and rupture of the bile capillaries. Its presence has a quantitative relation to the function of the liver, since its appearance indicates the point at which the derangement is so great that the liver cells cannot handle the pigment and must return it to the circulation. For this reason high indirect values nearly always give way to direct-reacting bilirubin.

It should be recalled that all sera giving direct reactions contain varying amounts of indirect-reacting bilirubin and better correlations with function are obtained by determining both rather than assuming that all the pigment is of the direct-reacting type.

Normal values for serum bilirubin vary from 0.1 to 0.25 mgm.%. The highest values 30-50 mg. are found in severe hepatogenous jaundice and in neoplastic biliary obstruction. Milder forms of parenchymatous damage and intermittent or partial obstruction are associated with values between 10 and 30 mg. Lower grades, 2-10 mg., are usually associated with subsidence of acute intrahepatic forms of jaundice and with the chronic forms of hepatitis which follow biliary obstruction, intrabiliary infection, portal cirrhosis and infectious forms of cholecystitis without gross obstruction.

The level of serum bilirubin is not static from day to day and frequent determinations should be made and plotted on a curve. This procedure may be carried out using the icterus index and provides data of diagnostic and prognostic significance. A rapidly rising curve is the rule in neoplastic biliary obstruction, especially if the gall bladder has been removed or is not functioning. A functioning gall bladder converts this into a slow gradual rise.

Where obstructive biliary cirrhosis complicates stricture or stone in the common duct, a low plateau curve is the rule. Where the liver is not involved extensively (recent neoplastic obstruction) the plateau is a high one. Acute intrahepatic forms of jaundice produce a rapid rise and a rapid fall. Chronic parenchymatous hepatitis gives a low irregular curve, as does also long standing biliary obstruction. If rapid degeneration supervenes this will be marked by sustained rises. Falling curves indicate restora-

tion of bils passage potency and liver repair, the one exception being the very chronic type of biliary obstruction.

The bilirubin tolerance test measures the excretory function of the liver for that substance. Retention of more than 5% of the injected dose (1 mg. per kg. body weight) at the end of 4 hours is considered as evidence of hepatic injury. The test has a high degree of sensitivity where the serum bilirubin level does not exceed 1 mg.%. The test has some serious drawbacks from the technical point of view and on the basis of cost.

Estimation of urobilinogen in the blood is complicated by technical difficulties. Measurement of urinary excretion of urobilinogen in a roughly quantitative manner is as valuable as accurate estimation, since the amount formed varies considerably from day to day. Single examinations have no special diagnostic value. Significance of urobilinogen excretion has already been discussed as has the excretion of bilirubin.

2. *Function with respect to the metabolism of*:

(a) *Proteins and the process of deamination:*

Variations in plasma proteins occur with injury to the hepatic parenchyma. Proteins are manufactured in addition in sites other than the liver but there is a reserve of protein building material, 50% of which is albumin, in the liver. In advanced chronic hepatic lesions the serum proteins are moderately reduced, diminution occurring especially in the albumin fraction. The albumin-globulin ratio is nearly always disturbed. The changes in this ratio take place rapidly and are of prognostic significance in that they indicate failure of the function of the liver to produce protein. The reduction in serum albumin is related to the production of œdema and ascites and contributes to that complication along with portal venous stasis and chronic peritoneal irritation.

Tests depending upon the decreased formation of urea in hepatic injury with coincident increase in amino acids are of some value in the diagnosis of acute yellow strophy. The procedure for the analysis of amino acids is quite involved and analysis is not commonly performed except for experimental purposes. The excretion of urea is modified by extra-hepatic considerations and is generally of little significance as a measure of hepatic function.

The estimation of amino acids excreted following the administration of gelatin has the some drawbacks as those noted for amino acids and does not yield any practical information.

(b) *Carbohydrates:*

One of the most important functions of the liver is to maintain the level of blood sugar at normal. For this reason it might be expected that this function would be the last to be lost. This is the case and also the reason why most tests designed to measure this function are clinically disappointing. Hypersensitiveness to insulin has been noted and forms the basis of a little-used test (test of initial insulin hyperglycæmia) which measures the glycogen reserve.

Laevulose and galactose have been used extensively in the study of hepatic function. The normal liver stores these as glycogen without producing hyperglycæmia or glycosuria.

Tests based on the use of these two sugars have been open to serious objections which modified procedures tend to overcome.

The laevulose tolerance test depends upon the estimation of blood sugar at intervals following a test dose. An increase of more than 30 mg.% indicates damage to the hepatic parenchyma. When laevulose instead of blood sugar is estimated the test is made more sensitive. It has no value in the diagnosis of hepatic disease—it simply measures the extent of hepatic damage and is thus useful in prognosis. A serious drawback is that it takes no account of disturbances in absorption of the sugar from the gastro-intestinal tract.

Galactose tolerance has had wider application. This test is based on the observation that a normal person can assimilate 4 gm. of galactose, losing no more than 2.5 to 3.0 gm. in the urine within five hours of the time of administration. Where hepatic injury

is present, glycosuria above this level occurs and the amount of sugar lost is a measure of the degree of injury. Originally designed to differentiate intrahepatic from obstructive jaundice, it is now used simply as a means of assessing parenchymatous damage since portal and biliary cirrhosis have been found to give consistently negative results and between 25 and 40% of obstructive jaundice give positive results. It may be said, however, that in the acute development of jaundice a positive test provides strong grounds for a lesion which is primarily hepatic. The test is of no value where no jaundice is demonstrated.

It has the same drawbacks as those noted for laevulose tolerance and a recent refinement in technique by which the sugar is administered intravenously and estimated at intervals in the blood shows promise. This modification is free from theoretical objections and evidence is presented that it distinguishes clearly between jaundice due to gross obstruction of the biliary tract without liver cell damage and jaundice due to liver cell damage. It does not detect minor grades of liver damage in the absence of jaundice.

(c) *Fat and Cholesterol:*

In parenchymatous liver disease the individual hepatic cells have an increased fat content. The relationship of liver function to fat metabolism and transport depends on a great many factors. The part played by the liver is difficult to assess in the light of present knowledge. The hemokonia test which measures the increase of fat globules in the blood after a mixed meal is an indication of failure of fat absorption and offers very little of clinical importance.

The estimation of cholesterol and cholesterol esters in the plasma has provided some information of hepatic function. Cholesterol is excreted with the bile and concentrated in the gall bladder. In biliary obstruction the concentration of cholesterol rises. Normally the liver cells esterify about two-thirds of the total cholesterol, *i.e.*, there is a cholesterol to cholesterol ester ratio of 1 : 2. In liver damage the process of esterification is impaired and the ratio is reduced although the total cholesterol may be and usually is increased. When obstructive jaundice is complicated by secondary hepatitis, the ratio becomes altered in accordance with this thesis. It is clamied by proponents of the method that the value for cholesterol esters varies with the degree of hepatic injury and is a guide in prognosis. Independent critical observers are not so optimistic and point to the possibility of extra hepatic influences on cholesterol metabolism about which we know very little. Tests based on the rate of absorption of vitamin A are not well developed.

3. *Metbolism and excretion of bile acids.*

The salts of cholic acid present in the bile are exclusively hepatic products. Formed in the liver and excreted in the bile, they pass into the intestine. Small amounts are excreted with the feces and traces find their way into the urine. The rest are rapidly absorbed into the portal blood and re-excreted in the bile. In obstructive jaundice the quantity excreted in the urine is increased but with continued obstruction they disappear only to reappear again when the obstruction is relieved. The chief value of their estimation in the urine is in jaundice following arsenical therapy or the use of any other drug which is toxic to the liver. Their appearance in the urine in increased amount gives early warning of toxicity. The bile salts are technically difficult to estimate. Simplification of the method might provide a very useful means of evaluating liver function.

4. *Excretory function with respect to dyes.*

Of the numerous dyes used to test hepatic function only two, rose bengal and bromsulfalein, have had general application. Of the opaque dyes used for gall bladder visualization, tetraiodophenolphthalein is the commonest.

The requirement for estimation of the rose bengal test and the rigid routine which must be followed in its performance make this test self limiting. The dye is photo sensitive and the patient has to be protected from sunlight for several hours after the dye is administered. Normally less than 50% of the dye should remain in the blood

eight minutes after it is injected. Greater amounts are associated with varying degrees of hepatic damage.

The bromsulfalein test is simple and as satisfactory a procedure as has yet been described. It measures the ability of the liver to excrete 5 mg. of dye per kg. of body weight by the end of one hour. Retention of dye occurs in over 90% of the cases in which there is evidence of parenchymal injury or moderate mechanical obstruction of the bile ducts of a degree sufficient to produce jaundice. Even retention as low as 10% is considered significant of hepatic disorder. Individual estimations are not of as great value as tests repeated at intervals to evaluate prognosis. The test has no value in frank jaundice. When the serum bilirubin does not exceed 3 mg.% dye retention gives a fairly good estimate of the degree of damage. When the serum bilirubin is normal, the degree of dye retention is most significant. Positive tests are genrally found in chronic liver atrophy, cirrhosis, hemochromatosis, fatty degeneration and so forth. The greater the degree of retention the more unfavorable is the prognosis.

Even moderate hepatic involvement as may occur in malignant metastases will produce some retention, as will also toxic or infectious hepatic lesions.

Any opaque dye such as tetraiodophenolphthalein is excreted into the bile but does not show in the roentgenogram until it is concentrated in the gall bladder. Where the liver is damaged, the dye is not excreted (positive Graham-Cole test). This is also the case in biliary obstruction and damage to the gall bladder mucosa. Jaundice regardless of origin gives a positive test so that its value apart from gall bladder visualization is limited.

5. *Function with respect to coagulation factors* (measurement of the tendency to bleed)

The patient with hepatic disease bleeds because vitamin K is not absorbed from the intestine. This is due to the absence of bile acids. Or it is not stored in the liver because of hepatic impairment. In both cases there is a prothrombin deficiency. Administration of vitamin K in jaundice without parenchymatous liver damage is followed by a rapid and prolonged elevation in the level of prothrombin, in cases where there is extensive liver damage there is only a slight, unsustained rise since the formation of prothrombin is interfered with. A falling prothrombin level in the jaundiced patient with or without associated hæmorrhagic diathesis is indicative of severe secondary hepatic damage.

6. *Function with respect to detoxification.*

Under normal conditions the liver removes from the circulation and renders inert a large variety of noxious substances. The conversion of indole to indican, the conjugation of salicylic acid to form glycuronates, the acetylation of the sulfa drugs are examples of this action. While many tests have been devised to assess this function only one seems to have stood the test of time. This test measures the ability of the liver to synthesize hippuric acid by combining 6 gm. of benzoic acid with glycine (amino acetic acid). Under the conditions of the test and in the absence of renal damage a normal individual excretes about 3 gm. hippuric acid, but no less than 2.5 gm., in the four hours following the administration of 6 gm. benzoic acid by mouth. It is generally agreed that where the blood urea is normal (or the urea clearance normal) the rate of hippuric acid formation by the liver is an accurate measurement of hepatic injury. Values less than 2 gm. are significant. In the surgical types of jaundice values less than 1.5 gm. are judged as of grave prognosis.

The test is simple and reliable, and in the absence of renal damage, gastric retention, dehydration and malnutrition, it provides an excellent means of evaluating prognosis and estimating surgical risks.

7. *Haematopoietic function.*

It is well known that the liver stores and may be concerned with the formation of a substance or substances effective in producing maturation of the erythrocyte. It is well known that liver damage is frequently accompanied by a hypochromic macrocytic anæmia and that there is an alteration in the fragility of the red cells. These findings

Table I.

SYNOPTIC GUIDE TO INTERPRETATION OF LIVER FUNCTION TESTS

Test	*Normal*	*Hæmolytic Jaundice*	*Hepatogenous Jaundice*	*Obstructive Stone*	*Jaundice Neoplasm*	*Indications for alarm in hepatogenous jaundice*	*Indications for surgical interference in obstructive jaundice*	*Indications of liver damage in absence of clinical jaundice*
Van den Bergh reaction	Indirect	direct Ind. to	Direct	Direct	Direct	Direct	Direct	Indirect to direct
Serum bilirubin	.1-.25 mg.%	.5-5.0	30-50	10-30	Up to 50	----------	----------	----------
Daily bilirubin concentration	NONE	moderate fluctuation	rapid rise and fall	intermittent rises	Continual rise no regression	rising bilirubin curve	rapid rise	more than 1 mg.% with increase lin leve
Bile pigments in urine	negative	negative	positive	positive	positive	increased amounts	increased amounts	presence of positive test
Urobilinogen in urine	neg. test	positive	may be pos.	negative	negative	----------	positive	----------
Stercobinogen in feces	always present	increased	none in obstruct. phase	intermittent increase or decrease	none	absent	----------	increase or decrease
Bromsulfalein retention	LESS than 4% 1 hr.	normal	no value	no value	no value	no value	no value	retetion varies with degree of damage
Hippuric acid excretion gm. in 4 hrs.	more than 2.5 gm.	normal	decreased	no change unless damage to the parenchyma		decreased excretion	decreased to less than 1.5 gm.	decreased
Intrav. galact. tolerance:								
½ hr.	40-100 mg.%		100-200	75-150	varies with parenchyma damage	reduced tolerance	reduced tolerance	may show reduced tolerance
1 hr.	10-50 "	normal	75-200	20-200				
2 hrs.	00		50-100	0-20				
Urine in 4 hrs.	2-3 gm.		3-5 gm.	2-5 gm.				
Serum proteins	7.0%	normal	decrease	normal	normal	decrease	decrease	decrease
Alb.-gloþ. ratio	2 : 1	normal	1 : 1	normal	normal	less than 1 : 1	less than normal	less than normal
Total cholesterol	180-240 mg.%	normal	increased	increased	increased	----------	----------	----------
Chol-ester ratio	1 : 2	no change	1 : 1 or less	no change	normal	sudden decrease	sudden decrease	----------
Prothrombin level: % of normal	100%	100%	reduced	reduced	reduced	rapid reduction	reduction during Vit. K therapy	normal

are corroborative evidence of liver damage but do not serve as a test of function. The macrocytes which appear in the blood stream can be explained on the basis of the altered plasma osmotic pressure associated with the decrease in serum albumin.

8. *Miscellaneous tests.*

(a) *The alkaline phosphatase test.* The enzyme *phosph*atase splits organic phosphorus compounds, liberating inorganic phosphorus. It is distributed widely in the body but is found in greatest concentration in the bone, kidney, intestinal mucosa and liver, where it is intimately concerned with the metabolism of phosphorus compounds. Whether or not it is formed in the liver is not known but it is excreted in the bile, so that in disturbances of biliary excretion (extra hepatic obstruction or intrahepatic block) it is increased in the plasma. In our hands the procedure has not justified the time it takes to perform. This has been the experience of other laboratories.

(b) *The Takata-Ara Test*: The cephalin-cholesterol Flocculation test. These tests are designed to evaluate the functional relation between the liver and protein metabolism. The effect on albumin globulin ratio has already been noted. In acute liver damage there is some alteration in the nature of the globulin and the tests depend on the flocculating reaction which these globulins produce in dilute serial solution of mercuric chloride or in cholesterol cephalin emulsions. A similar effect has been noted in a special application of the colloidal gold test. It is claimed that the cephalin-cholesterol and colloidal gold tests are valuable in acute hepatitis and differentiate fibrotic from exudative processes. Judgment in all these tests should be reserved until more knowledge of the mechanisms involved is available.

SUMMARY AND CONCLUSIONS

Liver function tests are extensions of the physical examination and are not intended to supplant it. Their purpose is to corroborate the diagnosis and assist in prognosis. A satisfactory appraisal of liver function entails the testing of as many functions as possible consistent with the comfort of the patient and economy. Most of the dissatisfaction experienced with tests of liver function is due to the tendency to rely on one test.

Any particular liver function test gauges the degree of liver damage only when it is performed repeatedly on the same patient: the results cannot be related to those of another patient. It is important to recognize conditions when certain tests should not be done. Dye tests are valueless in the presence of increasd bilirubin in the serum with a direct Van den Bergh reaction. This is also true of the bilirubin excretion test. The intravenous galactose test can be performed under all circumstances. The hippuric acid test has no value in the presence of renal damage although it may be used together with the urea clearance test and the result re-calculated.

Generally speaking, the tests adopted should have sound physiological bases and should combine ease of performance with accuracy of estimation. They should be planned in such a way that the combination of tests used may provide information of diagnostic and prognostic significance.

1. **TO DIFFERENTIATE HEMOLYTIC FROM HEPATOGENOUS OR OBSTRUCTIVE JAUNDICE.**
   - **(a) The Van den Bergh reaction and the estimation of serum bilirubin.**
   - **(b) Examination of the urine for bile pigments and urobilinogen.**
   - **(c) The color of the stools.**
   - **(d) The blood count and perhaps erythrocyte fragility.**
2. **TO DIFFERENTIATE BETWEEN OBSTRUCTIVE AND HEPATOGENOUS JAUNDICE.**
   - **(a) Serum bilirubin—repeated estimations.**
   - **(b) Examination of urine for bile pigments and urobilinogen and stools for urobilinogen.**
   - **(c) Intravenous galactose tolerance.**
3. **TO DIFFERENTIATE BETWEEN OBSTRUCTIVE JAUNDICE DUE TO STONE AND THAT DUE TO MALIGNANCY.**
   - **(a) Serum bilirubin repeated estimations, charted graphically.**
   - **(b) Examination of urine for bile pigments and stools for stercobilinogen.**
   - **(c) Duodenal drainage.**

4. TESTS OF DIAGNOSTIC AND PROGNOSTIC SIGNIFICANCE:
   (1) Van den Bergh and serum bilirubin at frequent intervals.
   (2) Examination of urine and stools.
   (3) Intravenous galactose tolerance.
   (4) Hippuric acid test and/or bromsulfalein test.
   (5) Plasma proteins.
   (6) Prothrombin level.

5. INDICATIONS FOR SURGICAL INTERFERENCE IN OBSTRUCTIVE JAUNDICE:
   (1) Rapid rise in serum bilirubin.
   (2) Decreased prothrombin during administration of vitamin K.
   (3) Decreased hippuric acid excretion.
   (4) Decreased intravenous galactose tolerance.

6. INDICATIONS FOR ALARM IN HEPATOGENOUS JAUNDICE:
   (1) Rise in serum bilirubin.
   (2) Decrease in prothrombin level.
   (3) Decrease in plasma proteins.
   (4) Increased excretion of amino acids (decreased excretion of urea).
   (5) Cholesterol-cholesterolester ratio less than 1 : 1.
   (6) Decrease in galactose tolerance.

7. ALTERATIONS FROM NORMAL IN THESE TESTS SUGGEST LIVER DAMAGE IN THE ABSENCE OF CLINICAL JAUNDICE:
   (1) Increased serum bilirubin.
   (2) Bilirubin in urine by sensitive tests.
   (3) Bromsulfalein reteniton.
   (4) Albumin-globulin ratio altered or reversed.
   (5) Galactose tolerance test (intravenous).

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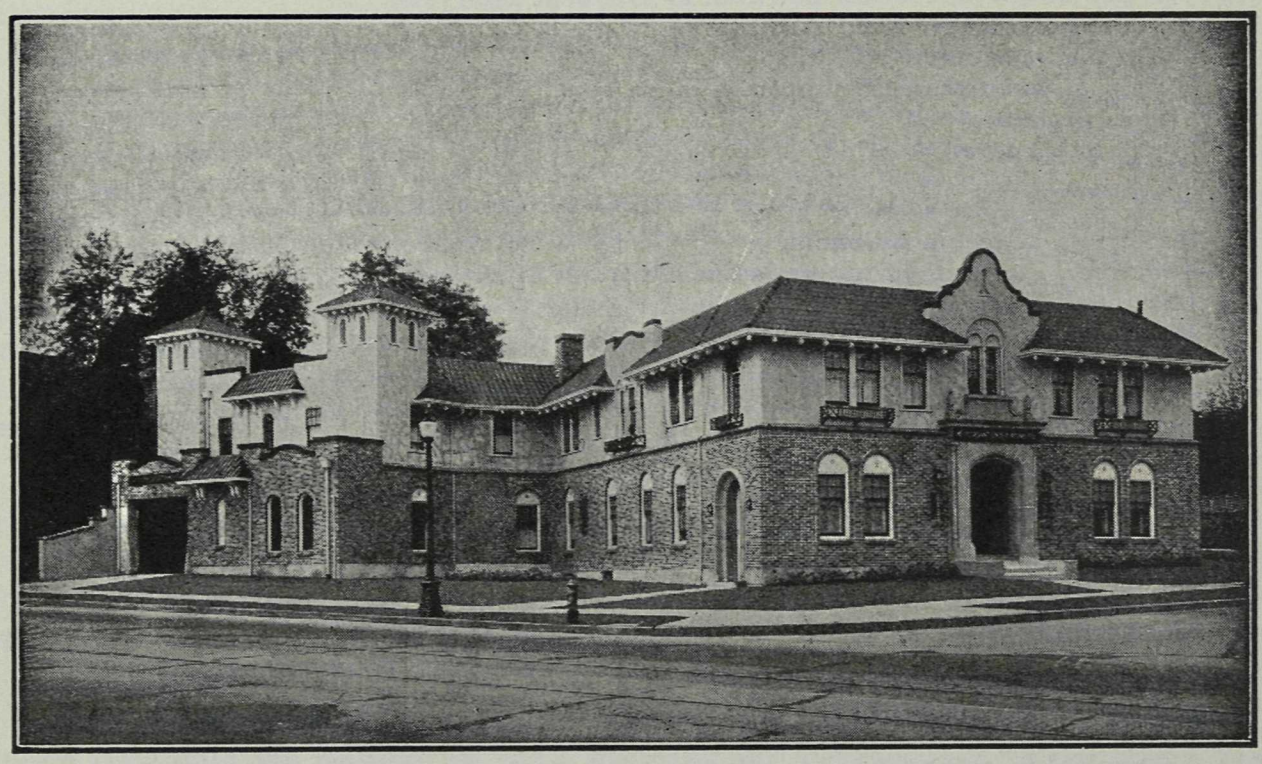

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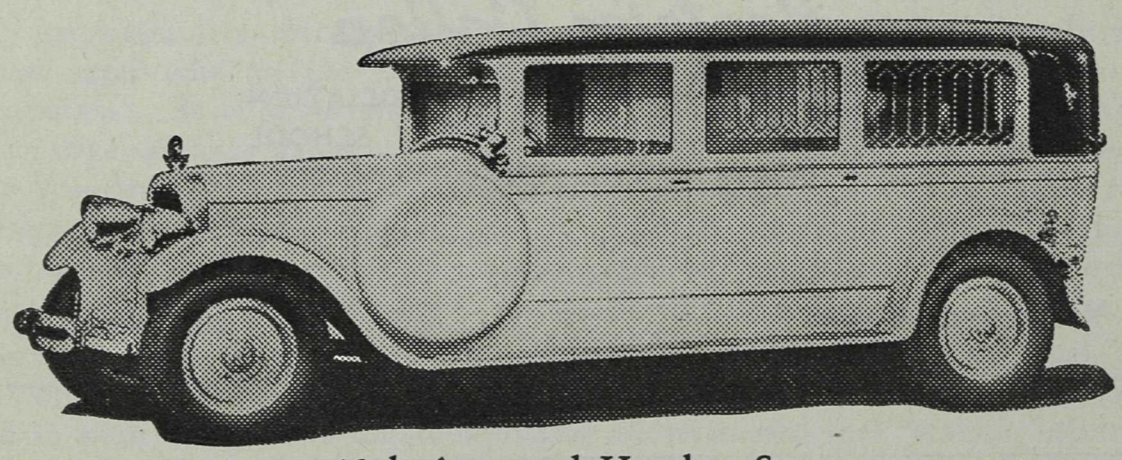

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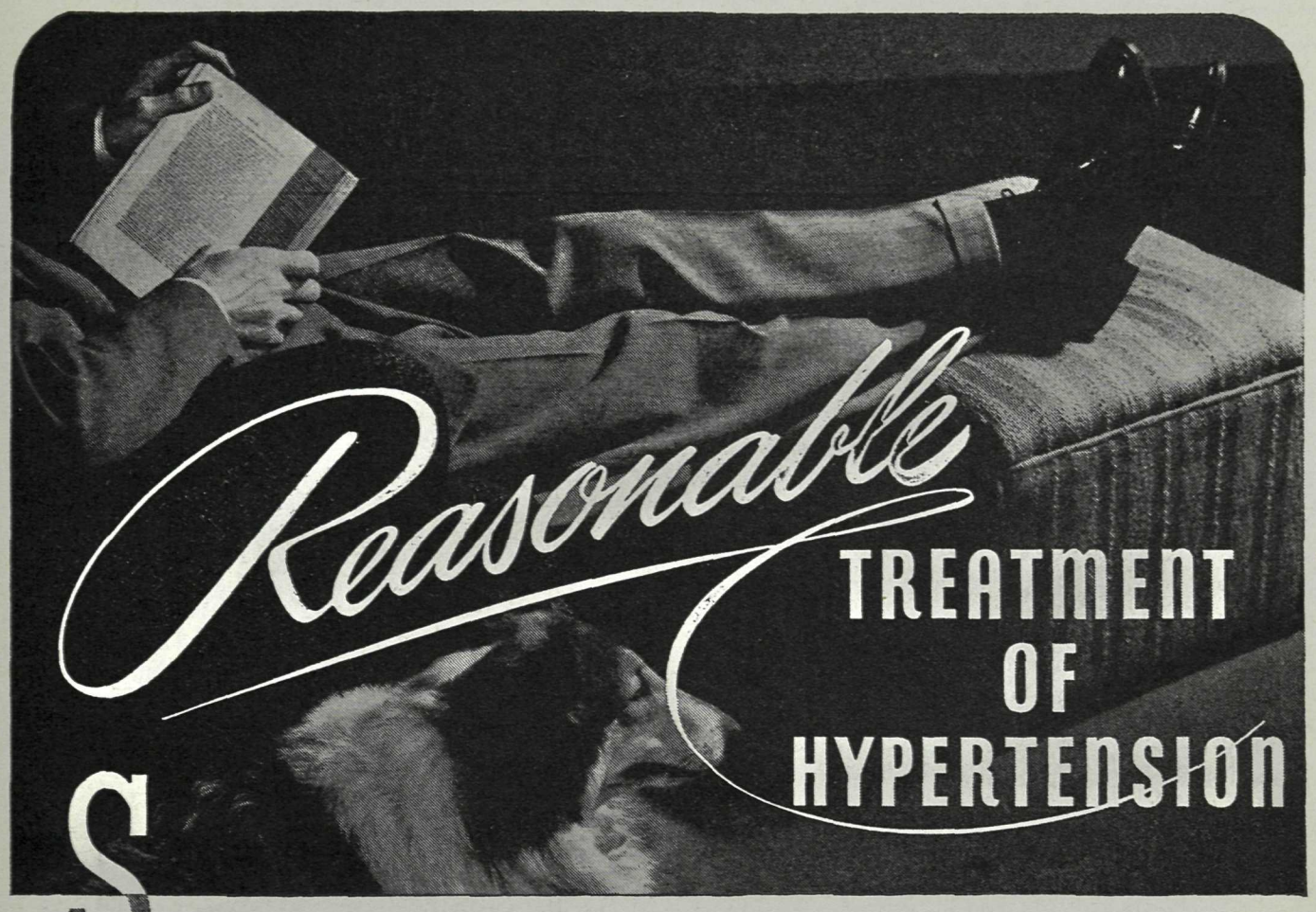

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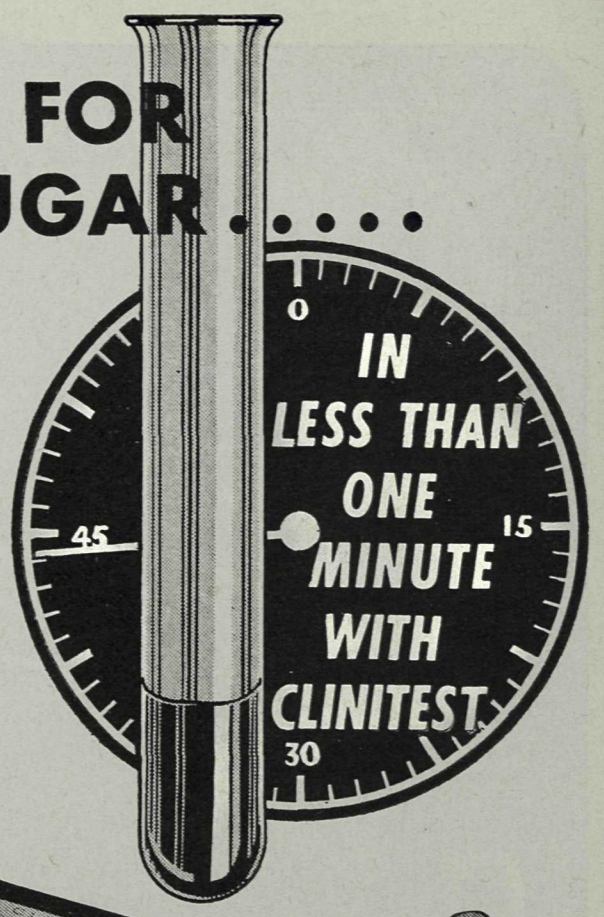

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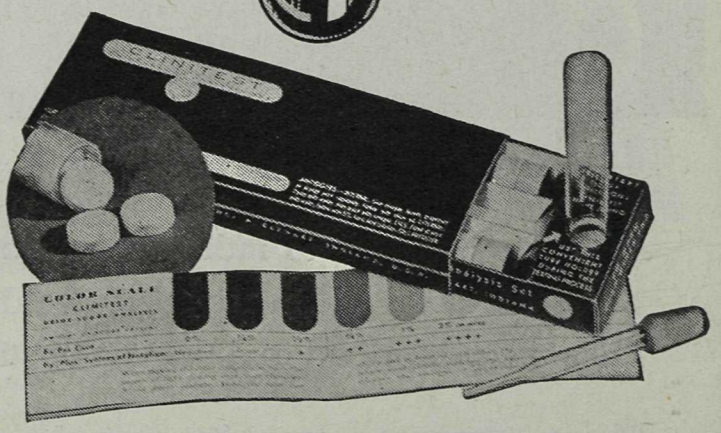

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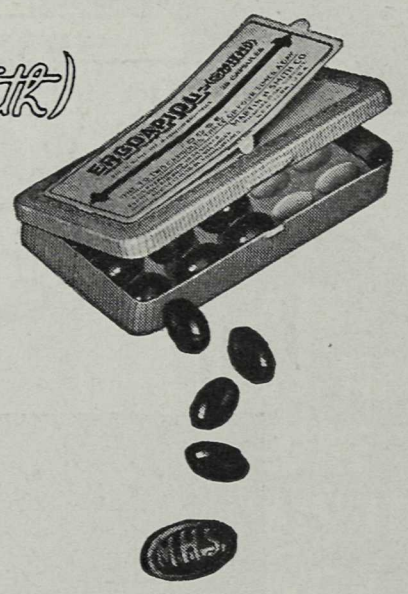

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*Hirsch, M. M., and Novak, M. V.: Evaluation of Germicides with Relation to Tissue Toxicity. Proc. Soc. Exper. Biol. and Med., June, 1942.

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