Vol. IV. OCTOBER, 1927 No. 1

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of the

Vancouver Medical Association

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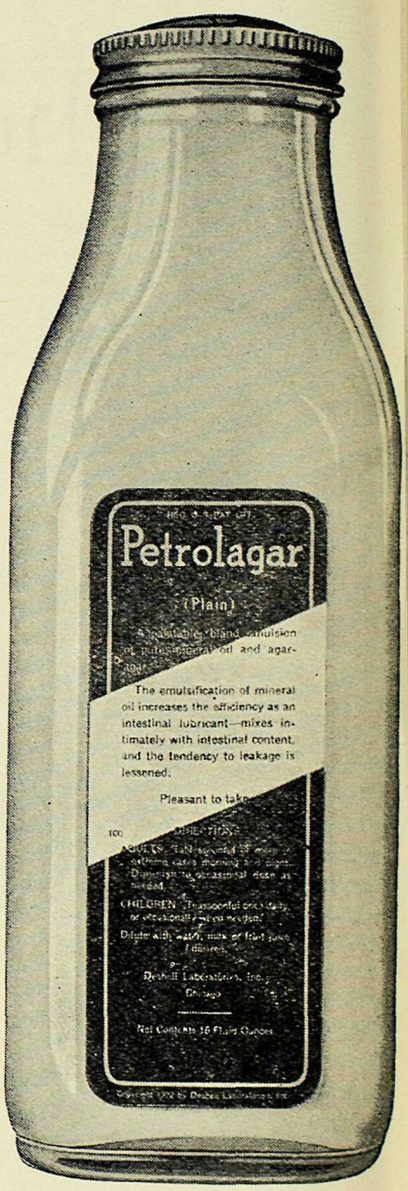

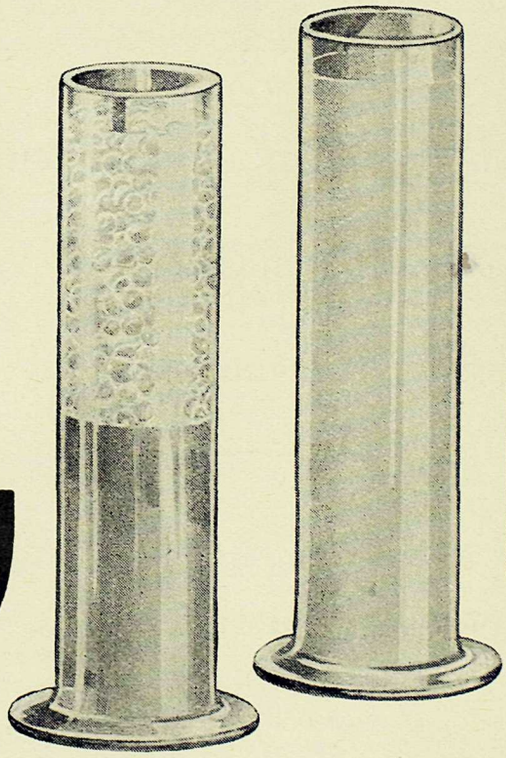

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THE VANCOUVER MEDICAL ASSOCIATION

BULLETIN

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

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529-30-31 Birks Building, 718 Granville St., Vancouver, B.C.

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Volume 4 OCTOBER 1st, 1927 No. 1

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VANCOUVER MEDICAL ASSOCIATION

Founded 1898. Incorporated 1906.

PROGRAMME OF THE 30th ANNUAL SESSION

GENERAL MEETINGS will be held on the first Tuesday and CLINICAL MEETINGS on the third Tuesday of the month at 8 p.m. Place of meeting will appear on Agenda.

1927

Oct. 4th—General Meeting:
Paper—Dr. E. Murray Blair, "A Conservative Treatment for Chronic Endocervicitis."
Dr. D. E. H. Cleveland, "On the Recognition of Syphilis."

Oct. 18th—Clinical Meeting.

Nov. 1st—General Meeting:
Presidential Address, Dr. A. B. Schinbein.

Nov. 10th—Annual Dinner.

Nov. 15th—Clinical Meeting.

Dec. 6th—General Meeting:
Papers—Dr. Chas. Edwin Sears and Dr. Karl Henry Martzloff, of Portland, Oregon. } Subjects to be announced later.

Dec. 20th—Clinical Meeting.

1928

Jan. 3rd—General Meeting:
Paper—Dr. H. H. Pitts, "On the Pathology of the Thyroid Gland."
Dr. T. H. Lennie, "Surgery of the Toxic Goitre."

Jan. 17th—Clinical Meeting.

Feb. 7th—General Meeting:
Papers—Dr. R. E. Coleman and Dr. H. Macmillan } "Relation of Carbohydrate Metabolism to Major Operations."

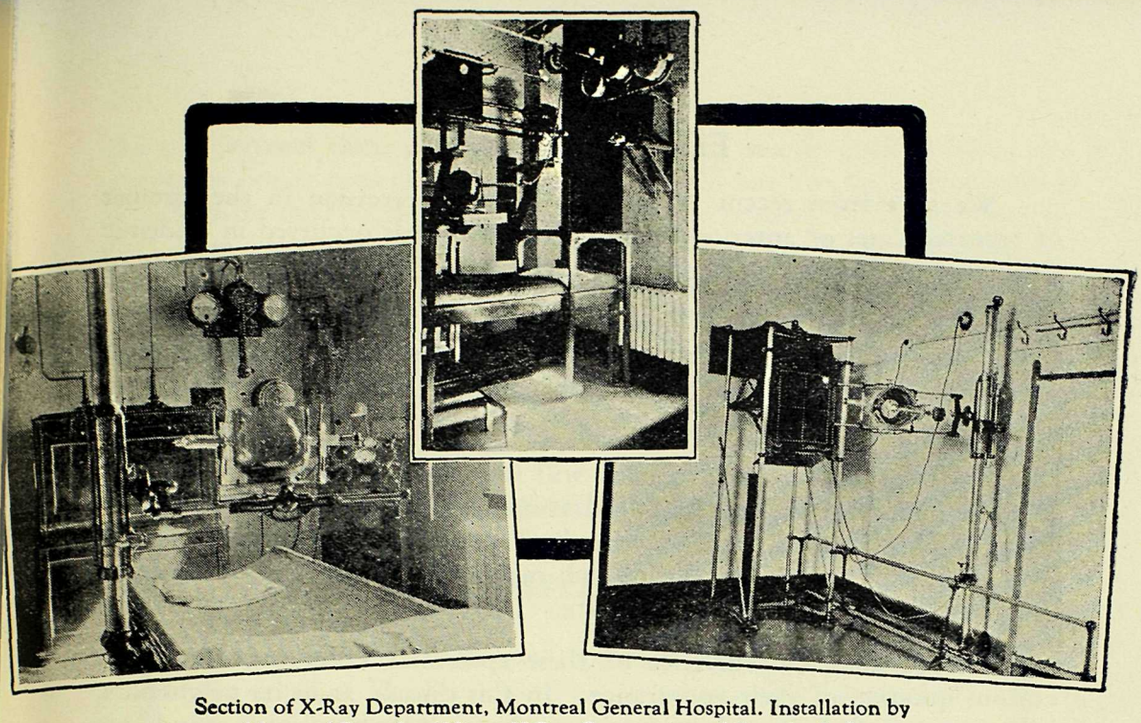
Feb. 21st—Clinical Meeting.

March 6th—General Meeting:
Paper—Osler Lecture, Dr. C. H. Vrooman.

March 20th—Clinical Meeting.

April 3rd—General Meeting:
Paper—Programme to be arranged.

April 17th—Annual Meeting.



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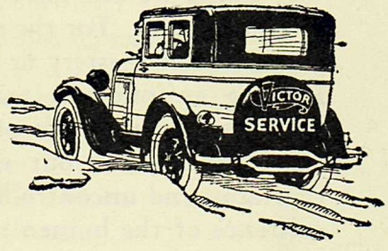

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EDITOR'S PAGE

We note from recent reports the apparent recession in the number of reported cases of anterior poliomyelitis which has occurred in epidemic form in the Western part of the province and the Eastern portions of Alberta.

Presumably the chief incidence of the disease has been among those of school age and has been met by the prompt closure of those institutions.

Doubtless full medical reports will later be available, and details of the immediate and remote effects of treatment be placed before the profession. Unfortunately, the aftermath of the disease will be accompanied by a great deal of the disability which makes this disease one of the most important of the diseases of early life.

The occasional occurrence of these epidemics raises once more the curious question of their appearance. In this disease, as in its neighbours, epidemic encephalitis and epidemic cerebro-spinal meningitis (and one might extend the list in other directions) we have sporadic cases occurring in any community from time to time, the infecting agent, microbic or otherwise, being the same. The isolated case occurs, more or less severe in character, terminating favourably or otherwise and that is the end of the affair.

Special precautions for the prevention of spread are not noticeably energetic and yet no such spread occurs. But at certain times, under conditions of which we have no clear conception and certainly no means of recognizing, this sporadic character is changed.

Another case occurs, and yet another, some in adjoining and some in more distant points and we recognize the disease as having assumed one of its periodical epidemic forms.

Observation and speculation in the region of epidemiology and the sister science of immunology, there have been in plenty and a very fascinating subject it is. The relative value of the two factors of the soil and the seed has been discussed pro and con.

Could we but solve the problem, what relief for suffering humanity it would mean. For the scourge and the pestilence are still with us despite our means of mastery or our control of some of their forms. When such a thing as influenza rears its grisly head and with miasmatic breath penetrates to the ends of the earth slaying in its thousands where even the bloodiest war but slays in hundreds, we are driven to realize the enormous and uncontrolled forces which warn and threaten even the very existence of the human race.

Uncontrolled and uncontrollable today, exhausted and expended on the morrow, what are the conditions which lend themselves to this extraordinary variation?

Is it a question of heightened virulence or of decreased resistance? We recognize that both or either of these factors may and do occur, but

the step beyond that, the conditions producing one or both of these occurrences lies at present beyond our ken. Still less are we able to realize and estimate their vicinity or their degree. At present we can but marvel that these epidemics are not with us more often, and upon the defensive mechanism of the body which, in the course of ages, has been developed as a protective agency against the various forms of pestilence which on all sides threaten to exterminate us.

* * *

OBITUARY

It is with the deepest regret that we have to record the death of Dr. Benjamin H. Champion, which took place in the Vancouver General Hospital on Tuesday, September 13th.

Dr. Champion was born at Alverton, Prince Edward Island, in 1880. He was educated at the public and high schools and Prince of Wales' College, Charlottetown. He graduated in medicine from McGill in 1910 and came to Vancouver where, after a year as Interne at the Vancouver General Hospital, he commenced practice. During the last few years Dr. Champion devoted himself more especially to genito-urinary work in which subject he took post-graduate courses at the Mayo Clinic, Chicago and New York.

Dr. Champion was a member of the Vancouver Medical Association since his arrival in the city and always took a keen interest in the work and was a regular attendant at the meetings. He held the position of Chairman of the Clinical Section for a year, and was afterwards a member of the Credentials Committee. Dr. Champion was also a member of the Staff of the Vancouver General Hospital, serving in the Genito-Urinary Department.

Unassuming in character and quiet in manner, Dr. Champion was one of the most popular men in the profession. Always ready to assist his younger confreres with special knowledge from his chosen field, he will be remembered by many with regret and with gratitude.

Dr. Champion made a hobby of raising foxes and was one of the first to popularize the industry in B. C.

He leaves no children but is survived by his wife.

NEWS AND NOTES

Dr. Underhill, Medical Health Officer, in a note to the Editorial Board, states that the medical profession is not notifying the Health Department of contagious diseases sufficiently promptly. He states that this is more particularly the case with whooping cough and measles. He asks for better co-operation from the doctors.

* * *

Dr. R. B. Boucher has left for the East on a short holdiay. He expects to visit Chicago and New York and will be away from the city for about a month.

* * *

Dr. R. E. McKechnie, who is a member of the Board of Regents of the American College of Surgeons, is atttending the meetings of the college at Detroit, October 3rd to 7th.

* * *

Dr. James Patterson, who has been practising in Vancouver for some years past, has left for the East, and it is understood that he intends to take up practice in New York.

* * *

Dr. A. Primrose and Dr. Duncan Graham, of Toronto, were the speakers at a special meeting of the Association held at the Hotel Georgia on September 7th. Dr. Primrose spoke on "Inflammation and Tumours of the Breast," and Dr. Graham on "Constipation." Full abstracts of these lectures appear in this issue. A joint luncheon meeting of the local and provincial associations was held on the same day at the Hotel Georgia at which Dr. Primrose was the speaker.

* * *

Dr. Schinbein's address as President at the first meeting of the Session on October 4th has been postponed until the second meeting, November 1st. Dr. Schinbein has gone East for a vacation and while away will attend the meeting of the American College of Surgeons at Detroit.

* * *

Arrangements have been made to obtain two Portland men for our meeting on December 6th. Dr. Chas. E. Sears and Dr. Karl H. Martzloff have consented to come to Vancouver and give papers before the Association on that date.

* * *

Dr. Stanley Millar has announced his return to the city after postgraduate study in Eastern centres. Dr. Millar will, in future, limit his practice to X-Ray diagnosis and therapy only.

Dr. W. T. Lockhart has left for the Orient and expects to be away about two months.

* * *

The Annual Dinner of the Association will be held at the Hotel Vancouver on November 10th. This popular event will, in the hands of the present capable Committee, doubtless equal or even excel its predecessors. All members in good standing are expected to attend this function.

* * *

The Treasurer again reminds members that the annual dues for the current year were payable on April 1st last. He urges members who have not yet paid their fees to send in their cheques as early as possible.

* * *

The Committee of the Sickness and Benevolent Fund acknowledges, with thanks, further contributions to the fund from the following: Drs. A. W. Bagnall, N. Allen, J. J. Mason, H. H. Planche, C. E. Brown, Glen Campbell, G. H. Clement, C. F. Covernton, C. Graham, L. G. Griffin, L. Hodgins, W. W. Kennedy, O. S. Large, A. Lowrie, D. M. McKay, A. J. MacLachlan, W. L. Pedlow, F. N. Robertson, R. A. Simpson, G. F. Strong, D. Tompsett, J. S. Conklin, F. W. Lees, P. W. Barker, H. L. Turnbull, J. R. Atkinson, D. L. McLellan, H. H. Milburn and J. A. Milburn.

* * *

Dr. Harold C. White left on September 24th for Toronto, where he will take up a course of special study in public health work, under a grant from the Rockerfeller Foundation. He expects to be away about eight months. During his absence the work of School Medical Officer will be carried on by Dr. Isabel Day and Dr. H. Caple.

* * *

HIGH BLOOD PRESSURE AND ARTERIOSCLEROSIS

An address to the Summer School of the Vancouver Medical Association, June 22, 1927, by *Dr. John Oille,* Asst. Prof. Clin. Med., University of Toronto.

Fifteen or twenty years ago when estimations of blood pressure by apparatus began to be commonly made, the tendency was to overestimate the danger of a systolic pressure of 200 or over. The laity greatly exaggerates its seriousness, and it is probable that the introduction of the blood pressure apparatus has done more harm than good. Even now, one occasionally sees patients who twelve or fifteen years ago were told they had a high blood pressure and were given to understand that something serious was likely to occur within a year or two. The general profession has not yet quite got away from this tendency.

Normal systolic pressure ranges from 105 to 150 for life insurance purposes. One would prefer the figures of from 100 to 145 for men, and five lower for women. It must be recognized that there are quite a few men well and strong, who constantly run a pressure of about 100, and more women with a normal blood pressure down to 90. One must distinguish between normal and average blood pressure. Average blood pressure for men from sixteen to forty is 129; for women 120. Because the pressure is 15 or 20 below the average figure, they should not be told that their pressure is too low. Having excluded tuberculosis, insurance companies get the lowest mortality with a systolic pressure of from 95 to 100. Above this the mortality rises slowly to 145. Above 145 their mortality rises rapidly and is about 200 per cent. at a pressure of 160. Normal diastolic is from 60 to 90. On the whole a high diastolic is more serious than a high systolic. The most serious thing, of course, in connection with blood pressure readings is a low pulse pressure. Blood pressure readings of systolic 100, diastolic 85, are much more serious than blood pressure readings of systolic 200, diastolic 85. Some insurance companies ask for the diastolic reading to be taken at the disappearance of all sound, though the true diastolic is where the sudden and greatest falling-off in the thump occurs. This is from 3 to 6 m.m. above the point of final disappearance in average cases.

The following are some points to keep in mind in connection with taking blood pressure:

1. No one estimation of blood pressure in any case is of any value in showing what the true blood pressure really is. It is a common experience to find the first reading 160 or 170 systolic, especially in one who is a little nervous during examination, or if it be the first examination. Then on making repeated successive observations, it comes down to 140 or 130. In examining university students, who were not thinking at all of blood pressure, quite a number were found with a systolic pressure of 160 or 170. All of these cases on a second examination had a reading 30 or 40 m.m. lower, and one whose blood pressure was first 170 was 100 at the second examination. There are certain people going up for insurance examination who will have a blood pressure of 160 or 170, from that slight anxiety, and previous and later observations on these cases show the blood to be perfectly normal. Then, too, there is another class of patients whose blood pressure varies as much as 60 or 70 m.m. from the mere mention of high blood pressure, or family worry. These cases, whose blood pressure shows such wide variations, if one could chart continuously throughout the day, would show a base line of say 160 or 170, with frequent occurring peaks of 200 or 250, which are due to psychic causes, such as anxiety, fear, anger, or excitement. A clinic on high blood pressure on a case of hypertension has been observed to raise the systolic from 160 to 240 for several hours, and the diastolic 20 or 30 m.m.

2. One should use both the auscultation and palpation method. In average cases the former is 5 or 6 m.m. higher, but in a few cases by palpation the blood pressure reading is 30 or 40 m.m. higher than it is by stehoscope. In either case the high reading is the correct one. There are a few cases in which the reading cannot be made by auscultation. These

are ones with very little difference between the systolic and diastolic, or with exceeding low pulse pressures.

3. In cases in which the thump is heard over the artery all the time, that is, in which the pulse pressure is very high, the diastolic has to be estimated by watching the point where the oscillation of the needle is the greatest. In these cases, however, usually there is a point about 40 or 50 or 60 in which there is a slight diminution in the thump, which is to be taken as the diastolic reading.

4. Blood pressure should be read both sitting and lying. This gives indication of the efficiency of the pressure-controlling mechanism. The standard method is when the patient is lying. On standing, the blood pressure may be 5 or 10 m.m. either higher or lower without any special abnormality. In some cases, however, vascular control is so poor that the blood pressure drops 20 or 30 m.m. on standing up. One case was observed in which on standing the blood pressure dropped from 140 to 90. This was a case of cerebral arteriosclerosis.

5. It is better not to tell patients what their blood pressure actually is, especially if it be up at the first reading. Future readings might be normal. Variations of 20 or 30 points in blood pressure from day to day or from week to week are normal and have no special significance. Patients are prone to attach undue importance to the rise or fall of a few millimetres of blood pressure, and they are better not to know anything about it.

6. Do not betray by word or manner surprise or alarm in taking the blood pressure, and do not make it the important point in the examination of the patient, and do not estimate it too often. These points suggest importance and alarm to the patient. The effect of suggestion is extremely important in the practice of medicine, and it is a very good rule to follow that if one acnnot make a favourable suggestion, don't make any at all, much less one that is depressing.

What are the known facts in connection with blood pressure? We know the normal and its variations. We know that chronic infections such as tuberculosis, Addison's disease, and any condition of weakness, emaciation or anæmia, tend to lower it. As patients become worse in such illnesses as pneumonia the blood pressure falls. We also know that there is a certain group of patients whose blood pressure is constantly 100 or below, and these people have no disease. About 60 or 70 per cent. of such people tire very easily, especially from mental strain; they usually stand physical exertion very well. The most important thing to do with these cases is to reassure them; tell them they will have to put up with their fatigue and will always need longer hours of rest than stronger individuals, but that they will live all the longer on account of their low blood pressure. Encourage them to take plenty of outdoor exercise, daily cold baths if possible, and see that their diet is adequate.

We also know that certain diseases produce elevation of blood pressure. Increased intercranial tension, especially if suddenly brought about, is more effective than anything we know in raising blood pressure. After

an intercranial hæmorrhage from an accident, a progressive rise in pressure is evidence of continued hæmorrhage, and indication for operative interference.

Diffuse glomerulo-nephritis produces a rise of blood pressure in any or all of its stages. In this disease the elevations may be temporary or continuous.

Many cases of thyroid disease, particularly those with a toxic adenoma, have a high blood pressure, and after several years consult a physician for cardio-vascular disease, which is partly the result of thyroid toxicity and overwork, and partly the result of arteriosclerosis, or some irregularity such as auricular fibrillation.

Some cases of aortic insufficiency have a slightly increased systolic blood pressure due to increased ventricular output, reaching 160 or 170. The characteristic point of aortic insufficiency, however, is a low diastolic causing a high pulse pressure.

In myocardial failure with passive congestion, blood pressure may rise 20 or 30 m.m. as increased carbon dioxide causes vaso-constriction. Decreased cardiac output also causes vascular contraction.

Eclampsia produces high blood pressure, which is, however, usually temporary.

Occasionally one sees an odd case with elevation of blood pressure in whom hysterectomy for uterine fibroid, or an operation for hæmorrhoids, or stopping smoking, causes the blood pressure to come to normal. The above facts are about all we know clinically about diseases producing high blood pressure, and in scuh cases the elevated blood pressure is merely a symptom of the disease, just as an elevated temperature is a symptom of a febrile disease. There is, however, a large clinical group of these cases in which no known organic disease is present and in which the systolic pressure ranges from 150 to 200. This group has been recognized as a clinical entity for over fifty years, during which time it has been called by at least a dozen different names, some of the ommon ones being, hyperpiesia, by Sir Clifford Albutt; presclerosis by Huchard, who recognized that it ended in arteriosclerosis; latent arteriosclerosis; the prealbuminuric stage of Bright's disease because a few end in renal insufficiency and uræmia; hypertensive cardio-vascular disease by Janeway, who also used the term essential hypertension; arterial hypertension; benign hypertension, simple hypertension; and simply high blood pressure.

This group of cases must at present be defined as persistent elevation of blood pressure without known cause. As our knowledge increases we will no doubt be able to subdivide this group into the various causal classes similar to those already mentioned.

What is the relation of high blood pressure and arteriosclerosis? Accurate clinical and autopsy observations have proven the following facts:

1. In many cases the blood pressure is persistently high and no arteriosclerosis exists.

2. After high blood pressure has persisted for several years, one finds hypertrophy of the heart, chiefly of the left ventricle and of the media of arteries. Then about the same time or later, there develops arteriosclerosis, which affects chiefly the coronary, the cerebral, the pancreatic and the renal arteries, and the aorta.

3. Some cases develop arteriosclerosis first and high blood pressure later.

4. High blood pressure can develop at any age, even in children, but most cases start in the forties. For a few years it gradually rises, then persists at a maximum height for a few years, after which it may continue elevated throughout the rest of the patient's life, or gradually decline until it reaches normal, and continue so until death.

5. Many cases of general arteriosclerosis, approximately 35 to 40 per cent. of the total number, have low blood pressure throughout their whole life. Often as these cases grow older their pressure gradually declines. There is very little truth in the prevalent idea that blood pressure rises with age.

From a consideration of the above-mentioned facts, we conclude:

1. That arteriosclerosis does not produce high blood pressure.

2. That high blood pressure, by increasing the strain on the vessels, assists in producing arterial degeneration and sclerosis.

3. There is a group of clinical cases who develop arteriosclerosis with high blood pressure early in life, and this form of arteriosclerosis is the most important clinical variety of this disease, because it progresses more rapidly and ends fatally at a younger age than any of the other forms of arteriosclerosis.

Aetiology.—Arteriosclerosis is not one disease; it is rather a sort of common result of several different diseases or causes. No one has improved on the classification of the causes of arteriosclerosis given by Sir Clifford Albutt several years ago. He groups them as under:

1. High blood pressure, recognizing that the elevation of blood pressure is a symptom of an unknown disease and only partly a cause of the accompanying vascular disease.

2. Poisons of different origin:

(a) Bacterial. The most effective intimal or vascular poisons known are toxins of bacterial origin, e.g., diphtheria or or typhoid toxin. Even in cases of severe influenza there is very extensive degeneration of the intima, internal elastic lamina, and inner part of the media of arteries, so much so that thrombosis was fairly frequently seen in the severe epidemic of 1918-19.

(b) Chemical. For many years such poisons as lead have been known to cause vascular degeneration.

(c) Metabolic. This group is a little more indefinite, but is made to include the arteriosclerosis that occurs in such diseases as diabetes and gout. In these diseases the sclerosis is no doubt the result of combined causes; metabolic changes must play a part. The elevated blood pressure in these two diseases also contributes.

3. Senility. Degeneration due to age is a cause of universal application, i.e., it may be the only cause, or it may be a complicated cause in connection with other aetiological factors.

4. Mental and physical strain. Of these two it is probable that mental strain is more important than physical strain.

The above factors might be regarded as the great causes of vascular disease. Syphilis causing vascular disease falls in a special group by itself. Its effect is chiefly seen in the aorta. It is not a cause of general vascular disease. Such things as alcohol, tobacco, over-eating and over-drinking are to be regarded as comparatively unimportant accessory causes.

The discussion as to the causes of hypertension is chiefly negative. When medical men realized that arteriosclerosis does not produce high blood pressure, they began to search for other causes.

It is not due to increased cardiac output. This is normal in high blood pressure. It is not due to hypertrophy of the heart. This is obviously due to increased work due to increased resistance to the emptying of the ventricle.

Careful investigation shows that it is not due to increased blood volume.

Neither is it due to increased viscosity of the blood. It is not due to decreased elasticity of vessel walls; it occurs when vessel walls are normal. When vessel walls become sclerotic the pressure is as likely to go down as up, or remain unchanged.

The blood pressure is controlled by the central nervous system, largely through the vaso-motor nerves altering peripheral resistance in the arterioles and capillaries. The factors, however, that work to maintain the persistent increased tonus are at present unknown to us. Many theories have been advanced as an explanation.

Volhard and Fahr think it is due to renal arteriosclerosis and use the term benign hypertension.

In advanced cases of hypertension, sclerosis of the renal vessels will be present as a part of the general arteriosclerosis.

It has been held that pressure substances have been formed by bacterial decomposition of food in intestinal stasis. The absorption of these substances, which are derived chiefly from proteins, stimulates vaso-constriction. Against this theory is the fact that no such substance has been demonstrated in cases of hypertension. Physiologists tell us that most of

the so-called pressure substances have a double action, namely, in a certain dose they lower blood pressure, and in a different dose they elevate it. Further, the persistence of high blood pressure at a fairly constant level for years, as is so often the case, is incompatible with the toxic theory. If an intestinal intoxication caused high blood pressure one should be able to cure it by purgation and changing the intestinal flora by yeast or buttermilk, or acidophilus. These measures have little or no effect on hypertension.

In the minds of the laity, as well as of many physicians, there is the idea that high blood pressure is due to the eating of meat or to a high protein diet. Mosenthal and others were unable to influence blood pressure in a series of carefully controlled experiments by either a very low or high protein diet combined with meat soups and all the tea and coffee they could get the patient to take. Esquimaux eat 350 to 400 grams of protein a day and are not subject to high blood pressure. This prejudice against meat is handed down from an old tradition. It likely has its origin in the fact that it has been observed that meat makes gout worse. Gouty patients have arthritis and arteriosclerosis with or without hypertension; therefore, in the early history of medicine, patients with any form of joint disease, must go without meat. There is practically no foundation in fact for this theory. Another mistaken idea is that white meat is all right and red meat is harmful.

It has been suggested that hypertension is due to increased suprarenal secretion, because when the suprarenals are destroyed the blood pressure is low. Careful physiologcial tests have shown that there is not an increased suprarenal content in the blood in high blood pressure.

More indefinitely, it has been supposed to be due to some disturbance in the ductless glands, because a certain group of the cases seem to be connected with the menopause, and because the blood pressure may be elevated in hypertension. This theory also lacks confirmation. Only about ten per cent. of a series of five hundred cases of endocrine disturbance had any elevation of pressure, and in most of these the elevations were very slight.

The most favourite theory is perhaps the nervous tension one. It is certainly true that in many cases physic causes, such as anger, fear, excitement, worry, mental strain, or great responsibility produce greater variation of the blood pressure than any other factor we know. This theory, however, will not explain all the cases, as a fairly large per cent. of them have lived quiet lives without overwork or mental strain. Possibly the mental strain theory explains the fact that men develop arteriosclerosis more rapidly than women, and die in greater numbers at an earlier age of coronary and cerebral vascular disease. Yet high blood pressure is about equal in both sexes.

The splanchnic sclerosis theory is based on the fact that ligation of the superior mesenteric artery causes elevation of blood pressure. A more reasonable explanation is that the hypertension is due to sclerosis of the cerebral vessels, particularly those supplying the basal nuclei.

Allan claims that if one lowers the intake of sodium chloride below a certain level, that the blood pressure will come down. This observa- has not been confirmed. The claim is probably only true for cases which have sodium chloride retention due to myocardial failure or renal disease, when other factors enter into the picture to affect blood pressure.

Pathology.—There is no known anatomical lesion associated with this symptom in a causal relationship. The changes that do occur are partly at least the result of the increased arterial tension. They are cardiac and vascular hypertrophy and arteriosclerosis.

Prognosis.—Janeway's idea of prognosis is that a large proportion of these cases do not have their lives materially shortened by the disease but with them in it is an incident of growing old. He estimates that a man's probability of living over fifty-eight is equal to his chance of dying before that age, and a woman's probability of life is three years greater, that is, a woman has an even chance of living to sixty-one. Certainly a large number of cases live to an advanced age. Sir Clifford Albutt followed a case of 250 and over for thirteen years. The writer has in mind a woman who had a severe angina pectoris with hypertension for sixteen or seventeen years, who, six years previous to her death, had several nearly fatal attacks of acute œdema of the lungs, and who lived to the age of eighty-two with a blood pressure of 190 to 220 and finally died of influenzal pneumonia.

The prognosis does not depend on the height of the systolic pressure, but rather on the condition of the arteries of the heart and brain and the function of the kidneys and spleen. A case with a systolic pressure of 150 to 160 might die inside of a year of coronary thrombosis at the age of forty-five, while another with a reading of 250 might live ten years and die at the age of sixty-five. There is, thus, no special relationship between the actual height of the pressure and the condition of the vessels. Other things being equal, a person is obviously worse off with a pressure of 300 than one with a pressure of 170; nevertheless it is true that one estimates how long a patient may live far more accurately by the condition of the heart, brain and kidneys, than by the actual blood pressure figure. One estimates the prognosis in coronary sclerosis in the following way:

1. By how well the heart tolerates exercise. This is found out chiefly by inquiry; one question to ask is "how far do you think you can walk on the level at an ordinary gate without distress?" meaning, of course, distress referable to the heart, such as dyspnœa, uncomfortable palpitation, and cardiac pain. The seriousness is best guaged by the slightness of the exertion that brings on the distress. Find out by inquiry exactly how much exertion it takes to bring on distress.

2. By trial. Develop a standard exercise tolerance test for yourself, such as climbing twenty steps. Observing the amount of distress that this produces is a better gauge than the actual increase in pulse rate and time it takes the rate to return to normal. An exercise test only confirms what you have found out already by inquiry.

Continued on Page 21

The British Columbia Laboratory Bulletin

Published monthly September to April inclusive in co-operation with the Vancouver Medical Association Bulletin, in the interests of the Hospital Clinical and Public Health Laboratories of B. C.

Edited by

DONNA E. KERR, M.A., OF THE VANCOUVER GENERAL HOSPITAL LABORATORIES

Financed by

THE BRITISH COLUMBIA PROVINCIAL BOARD OF HEALTH

COLLABORATORS: The Laboratories of the Jubilee Hospital and St. Joseph's Hospital, Victoria; St. Paul's Hospital, Vancouver; Royal Columbia Hospital, New Westminster; Royal Inland Hospital, Kamloops; Tranquille Sanatorium; Kelowna General Hospital; and Vancouver General Hospital.

All communications should be addressed to the Editor as above. Material for publication should reach the Editor not later than the seventh day of the month of publication.

Volume 2 | OCTOBER, 1927 | No. 2

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* * *

EYKMAN WATER TEST

G. A. Ootmar, M.D., Kelowna, General Hospital Laboratories.

(Editor's Note:—Owing to the final copy of Dr. Ootmar's paper being lost in the mail, two errors occurred in his article. The last sentence in the penultimate paragraph should read):

"In this case bacteria belonging to the colon group are found which lack the property of growing at 46° C. and *others* which belong to a strain which does not originate in the intestine."

and the following was omitted from the end of the article:

"As we saw in the preceding article, sometimes fermentation occurs at 46° C. and sometimes only at 37° C. B. coli *fresh* from the intestine has the property to ferment at 46° C., while B. coli deposited a long time ago in the water has lost this property and only ferments at 37° C. The longer the period that has elapsed between contamination of water with B. coli (fæces) the greater the possibility that typhoid bacilli, if present, will be destroyed by the sun, protozoæ, etc., thus lessening the chance of infection with B. typhosus.

After flaming the neck of the sterile bottle, which contains the water to be tested, put 40 cc. of the water in a U-tube, add 5 cc. of the sterile solution of glucose and peptone, mix well by canting the U-tube and put in an incubator at 46° C. Another U-tube also filled with 40 cc. of the water and 5 cc. of the sterile solution is put in an incubator at 37° C.

After 48 hours if we find:

1. In both tubes gas and cloudiness, then the water is freshly contaminated with fæces and dangerous for use.

2. Only gas fermentation, etc., in the tube incubated at 37° C., then the water was probably contaminated with fæces a long time ago. Thus there is very little danger that the B. typhsus would be still living.

3. Neither of the tubes showing fermentation, etc., then the water is not contaminated with fæces and is safe from a bacteriological point of view."

* * *

HIGH RED CELL COUNTS

Grace Wilson, M.A., Vancouver General Hospital Laboratories.

The following table of red cell counts over the past twenty months is presented for its bearing on a question being asked today by clinical pathologists on this continent. We were all taught that the normal red cell count was five millions for men and four and a half millions for women. Osgood and Haskins, working with normal subjects in Portland, Oregon, have found, for that locality, the normal range is 4.7-6.1 millions for men and 4.3-5.3 millions for women. Working in the Southern states one clinical pathologist claims, from hospital statistics for that locality, the normal red cell count is less than five millions.

The question, therefore, being asked today is, what is the true range and does that range vary with localities other than the classically recognized influence of altitude? Certainly the training rceived by the majority of clinicians makes him look upon red cell counts of six millions or more with suspicion as to the technical accuracy of the determination. Experience in this laboratory, however, had demonstrated that such counts are not infrequent in cases lacking any clinical symptoms of polycythæmia. Though all of these counts have not been repeated, enough have been repeated to leave no doubt as to the general accuracy of the results. In other words, for this locality, at least, there is, even in hospital practice, a number of individuals who show a materially higher red cell count than the text books lead us to expect.

Red Cell Count	Total Number	Hæmoglobin Range
Over 5½ million	54	60—150%
Over 6 million	18	85—150%
Over 6½ million	7	103—150%
Over 7 million	3	120—150%

* * *

THE DETERMINATION OF CHLORIDES IN SPINAL FLUIDS

R. V. Harris, B.Sc., Vancouver General Hospital Laboratories.

"1. In both tubes gas and cloudiness, then the water is freshly con- very desirable that the diagnosis in a case of T.B. meningitis should be made early. The discovery of the tubercle bacillus in the spinal fluid is, of course, the only conclusive proof, but the results of a chemical examination may be extremely useful along with the history and clinical findings.

It has already been established* that the chloride content in these

cases is well below normal and that in other similar clinical conditions the chloride content is either normal or high. In the past few months the chloride content of a number of spinal fluids has been determined with a view to perfecting a simple technique suitable for routine work. The method used was as follows (*loc. cit.*):

One cubic centimeter of spinal fluid (previously centrifuged, if cloudy) is measured out by means of a pipette into a white evaporating dish and diluted with approximately twice its volume of distilled water. Two drops of potassium chromate solution (5%) are added from a dropping bottle and the resulting mixture is well stirred. From a microburette graduated in 0.02 cc. divisions, silver nitrate solution (0.1N) is added, stirring after each addition. As soon as the colour changes permanently from yellow to pale pink, the addition is stopped and the volume of silver nitrate used is read. Multiplying the volume by 0.585 gives the percentage of sodium chloride present. The following table gives the results of twelve fluids examined:

Case	Diagnosis	Chlorides (mg. per 100 cc.
1	T.B. Meningitis	630
2	T.B. Meningitis	730
3	Unknown	740
4	Unknown	760
5	Unknown	770
6	Unknown	760
7	T.B. Meningitis	590
8	Tabes	730
9	Unknown	740
10	Old head injury	740
11	Congenital Syphilis	740
12	T.B. Meningitis	630
	Normal Range	725–740

It will be noticed that in three out of the four cases of T.B. meningitis examined, a low chloride content was detected. The normal chloride percentage shown in case 2 may be an exception to the rule. The patient was a child under 3 years of age.

This work will be continued and it is expected that in time the estimation of the chloride percentage will be included in the regular routine examination, since the clinical inconvenience of obtaining another specimen makes it highly desirable that the first examination be as complete as possible.

*Ministry of Health Report, 1922.

* * *

INADVISABILITY OF KEEPING TRANSFUSION BLOOD

R. E. Coleman, M.B., Vancouver General Hospital Laboratories.

Occasionally conditions arise which induce the clinician to consider the possibility of keeping transfusion blood for some hours in a flask. At such times the opinion of the clinical pathologist may be sought. Under such circumstances, a definitely adverse decision is often difficult because of lack of definite adverse evidence. However, the following extract from a letter written by Charles A. Doan, of the Rockefeller Institute

for Medical Research, bears much weight. The author of this letter has recently reviewed the transfusion problem in the Physiological Reviews, January, 1927 (citing 717 references), so that the opinion expressed probably represents the best thought at present available.

"With reference to the problem of keeping blood for transfusion purposes over a period of twenty-four hours or longer, about which you ask, you will find on pages 45, 56, 57, 58 and 64 of the review, references made to this particular phase of blood transfusion. Rous and Turner (Jour. Amer. Med. Assn., 1915; LXIV, 1980) used a blood-citrate-saccharose mixture which maintained the functional capacity of the red cells for two weeks and prevented hemolysis for one month. O. H. Robertson further adapted this method to the clinical problems facing the A.E.F. and found the method entirely satisfactory. Burmeister 1916, Bigger and Wigham 1922, Torii 1923, and Yourewitch and Teleguine 1925, have all used the same method of preserving red cells and commend it as the most satisfactory procedure known at the present time.

"However, the reason that I did not discuss more fully this particular phase of the subject is because of the very general agreement, I think, that it is a procedure to be used only in such emergencies as were faced during the war, or as perhaps would be faced occasionally by the rural physician, or as in the case reported by Dr. F. C. Grant, ('Autotransfusion,' Annals of Surgery, 1921, lxxiv, 253), in which the surgical procedure of suboccipital exploration always requires a post-operative transfusion. Certainly in the practice of a general hospital, I should think the necessity for a delayed transfusion would very seldom occur; and our present uncertainty as to the nature and extent of the chemical and physical changes which blood undergoes outside the body over a long period of time makes it highly desirable that the blood be received by the recipient in as short a time as possible after its withdrawal from the donor."

* * *

CEMENTING MUSEUM JARS WITHOUT HEAT

Joe Garner, Vancouver General Hospital Laboratories.

The regular museum cement, known as Stockholm pitch, comes in a hard black form, requiring strong heat to melt it. Also, when thus melted it cools quickly upon applying, making it a tedious job to handle.

The following method, however, has proven much more satisfactory: Take the pitch as received, break into small pieces. Put into a wide mouth bottle (I use an ordinary sealer) and cover with Xylol. Close the sealer to prevent evaporation and stir it, with a glass rod, twice a day. In a week's time, it will resemble a black creamy liquid. For use, pour some into a small bottle and allow it to evaporate spontaneously, until it has the consistency of soft putty. Take a small brush, or make a swab with a thin stick and cotton wool and paint around the edges, place the lid on, sealing the jar. Next day it will be set, when an outside coat can be given to the rim. The result is a perfect seal.

Advantages of this method.—A stock solution is always ready. No heat is required. Plenty of time is available to paint, without the cement setting prematurely. There is no objectionable odour. The process is clean and simple. Although slightly slower, the cement sets perfectly.

Continued from Page 16

3. By the course of the disease, i.e., how rapidly it is advancing, how long have the symptoms been present, how far could the patient walk five years ago, one year ago, six months ago, and now.

4. By what you find wrong with heart and circulation on examination. Naturally, a case which has had passive congestion and œdema has a worse prognosis than one that has not. The larger the heart the greater the seriousness, is a fairly safe rule. The character of the sounds and associated valvular disease are not so important as the condition of the circulation and the ability to tolerate exertion.

5. By the response to treatment. A case that grows worse while at rest in bed has obviously a very bad prognosis.

6. By the nervous condition of the patient. One has to estimate the part played by apprehension, fear, sensitiveness, and suggestion, and naturally discounts the seriousness of symptoms in such cases, and is guided chiefly by physical signs.

7. Certain signs are of serious significance. People with pulsus alternans, low voltage of the R wave in the electrocardiograph, branch bundle block, or a negative T in leads one and two, rarely live two years. People with auricular fibrillation often do well because one can do more by treatment for such cases. In a few cases one cannot estimate prognosis and sudden death occurs unexpectedly from unexplained causes. Coronary thrombosis may supervene at any time and end a case apparently doing well.

The prognosis of cerebral arteriosclerosis is more uncertain than that of cardiac cases. One can diagnose cerebral vascular disease, but it is doubtful if a stroke can be foretold. Retinal degeneration (white spots) and retinal hæmorrhages are very unfavourable signs. These are to be regarded as evidence more of serious vascular disease than of renal disease. In high blood pressure cases three simple kidney function tests should be done at periodic intervals in order to estimate danger from this source. The first sign of renal insufficiency is polyuria at night with low specific gravity. The first test is the estimation of the amount and specific gravity of urine passed every two hours by day when on a full diet, and of the total amount and specific gravity of the 12-hour night specimen. The amount should go up after meals, and the specific gravity go down, producing a variation of 8 to 10 points in specific gravity. The night urine should be under 400 or 500 c.c. depending upon whether fluid has been taken at bedtime, and the specific gravity should be over 1020. If the so-called two-hour test is normal, no further test is necessary. If the specific gravity is fixed, or too low, then go ahead with the water and concentration tests. These estimate how the kidney responds to excessive work. After drinking 1500 c.c. of water in the morning without breakfast in the course of half an hour, a patient should excrete 80 to 100 per cent. of the full amount in four hours. The urine is collected, measured, and the specific gravity taken every half hour for four hours. After this period the rest of the urine for the remainder of the twenty-four hours is collected all together. This should be about 1000 c.c. The

concentration test consists in keeping the patient on a dry diet for a day, and finding how high the specific gravity goes. These tests run parallel to the phthalein test and are more important than the presence of albumen and casts on chemical examination.

Symptoms.—It is doubtful whether any symptoms are due to the high blood pressure. Many cases have no symptoms for years and when patients do consult doctors, it is for the symptoms of the associated cardiac or cerebral arteriosclerosis. It is, therefore, the vascular disease which produces symptoms.

Diagnosis.—1. Hypertension. This is made by proper estimation of the blood pressure and by excluding the known causes of elevated blood pressure previously mentioned.

2. Arteriosclerosis. This disease is manifested by local symptoms and signs, that is, it has to be diagnosed by systems.

(a) Coronary sclerosis is diagnosed by surmise when:

1. Signs of myocardial weakness or failure occur in a patient old enough to have vascular disease, having excluded infection, syphilis, anæmia, etc., as causes.
2. Attacks of angina.
3. Attacks of dyspnœa at night.
4. Attacks of Cheynes-Stokes breathing at night.
5. Attacks of cardiac asthma.
6. Attacks of coronary thrombosis.
7. Attacks of acute œdema of the lungs.
8. Pulsus alternans, fibrillation or flutter, any of the blocks, low voltage, and negative T's in the electrocardiogram.
9. Coronary sclerosis should be watched for if the aorta be dilated.

(b) Cerebral arteriosclerosis may be diagnosed:

1. By certain mental changes such as loss of memory, lack of emotional control, change of personality, up to actual dementia. Many so-called neurasthenics over fifty years of age are really cases of cerebral arteriosclerosis.
2. By certain symptoms such as headache, dizziness, failure of the special senses, and Meniere's syndrome.
3. By cerebral "attacks" such as hemiplegia due to hæmorrhage or thrombosis, transient paralyses, or aphasia, temporary loss of consciousness, or disorientation, and occasionally convulsions.
4. By the condition of the retinal vessels and retina, and by an arcus senilis.

(c) Sclerosis of the splanchnic vessels is indicated by ptosis, hypomotility and hyposecretion.

(d) Sclerosis of the leg arteries is diagnosed by intermittent claudication.

(e) General arteriosclerosis is diagnosed by atrophy of the skin, loss of weight, weakness, and thickened tortuous vessels.

Treatment of High Blood Pressure.—The first question that arises when considering the treatment is, when, if ever, should one direct efforts to lowering the blood pressure. Most of us will agree that when arteriosclerosis is advanced, the damage is already done and there is little use in trying to affect the height of the blood pressure. The ideal time to lower blood pressure would be in an early case, either before arteriosclerosis had developed, or when it is slight. If one could predict cerebral hæmorrhage, lowering the blood pressure might avert this accident. An effort in this direction is, however, practically useless, because, while we can lower the pressure temporarily, we have as yet no means at our hand for lowering it permanently. The best measure available at present is rest, both physical and mental. In many cases, especially of early arteriosclerosis, a period of a long rest in bed accompanied by a good diet and good physic treatment will occasionally permanently lower the blood pressure. The usual thing, however, is that the blood pressure comes down considerably during the time in bed, especially if anxiety can be relieved, but that on getting up and going to work again, the pressure gradually goes back to its former height. One has to consider the mental attitude of the patient. In the nervous type, minimize the dangers, point out that the chances are six to one that they will not get a stroke, explain the course of high blood pressure, and mention cases that live to old age. Use all the reassurance and encouragement possible. The most important point of all is not to over-alarm the patient. In odd cases who will not make the necessary sacrifice of time and alteration of habits and rest, tell them frankly the danger and the probable outcome.

There is no drug at present available which is of any permanent value. As time passes new drugs are exploited as cures of hypertension, used a few years, and then discarded. The latest one under trial is sodium sulphocyanate. Christian commences his discussion of the treatment of hypertension in the Oxford System of Medicine by the statement: "In this disease drugs are of no value, and I do not use them." Venesection, electricity and hydrotherapy all have a slight temporary effect. Liver extract aroused hopes a short time ago, but finally proved disappointing. A few observers are still experimenting with extracts, hoping to find something of greater value. It is thus seen that measures directed to lowering the blood pressure are temporary, ineffectual and practically useless. So far, the best we can do is to employ measures directed toward improving the health of the patient, hoping to prevent or delay the development of vascular disease, and that the pressure will be regulated at a lower level. One analyses the patient's life and tries to prevent mental strain. In their work advise against worry, anger, or excitement, and shorten their hours if judged necessary. There is no diet for high blood

pressure; their food must be adequate. It is all right to prevent excess in any respects, but there is no reason whatever for limiting proteins. This is only done when there is serious renal insufficiency. It is often necessary to advise more rest, both mentally and physical. Longer hours of sleep, lying down so many hours per day or possibly staying in bed one day a week. Sometimes vacations should be undertaken, or made longer, or more frequent. Recreations relieve mental strain and enable one to enjoy necessary holidays. Insist on moderate, regular exercise suited to the capacity of the heart. Advise against over-smoking or drinking and excess of all sorts.

By such general measures you will in some cases be gratified to see the blood pressure return to normal and remain so. In many cases the blood pressure will come down with a rest cure and go back up with a return to active life to a point not quite so high as it had been. In many other cases the treatment and advice will not have the slightest effect on the height of the blood pressure, as, perhaps fortunately, this is quite beyond our control.

* * *

THE CLINICAL VALUE OF THE METABOLISM TEST

By *William D. Keith,* Vancouver, B.C.

[EDITOR'S NOTE:—This is the third of a series of articles intended to indicate the *clinical uses,* interpretation and applications of various modern laboratory tests. Each article will be written by a different author, peculiarly familiar with the *uses* of the test treated of.]

You and I are true disciples of medicine only in so far as we adhere to the great traditions of our art. Our first aim should be accuracy in diagnosis; then, with a thorough comprehension of the possible pathological changes taking place in a given case, we can apply the most helpful therapeutic measures available.

An instrument which tells us the metabolic activity going on within a person's body has been found of great aid in differentiating certain diseases and in guiding us in our treatment.

The normal range of metabolism as ascertained *under standard conditions* by the instrument we use lies between a plus 15% to a minus 15% or possibly a minus 10%.

An increase of the metabolism above the standard rate occurs in hyperthyroid disease, in leukæmia, in fevers, and in heart disease. A moderate increase may occur in heart disease. A marked increase often occurs in hyperthyroidism or in leukæmia. The latter is readily enough diagnosed through examination of the blood.

Standard conditions as outlined by Krogh and others.—Metabolism is measured in the morning 12 to 14 hours after the last meal with the patient absolutely at rest in the lying or recumbent position. (Out-patients at absolute rest 20 to 30 minutes after coming to the laboratory

and just prior to taking the test.) The day before the test a low protein diet is to be taken and no unusual activity indulged in.

Basal metabolism estimations are of definite value in hyperthyroidism, as a record of this nature gives an accurate idea of the degree of the toxæmia from which the patient is suffering.

A metabolic rate below the normal points to a hypoactivity of the thyroid. This may be a true myxœdemic state, an indication of asthenia resulting from adrenal insufficiency or certain systemic diseases; or it may be an indication of a hypoactivity of the thyroid gland without any œdema.

Nothing is more valuable in treating hypothyroid states with thyroid extract or thyroxin than the control that repeated metabolism records gives. In this way we learn just how much of the thyroid extract or thyroxin to administer.

Certain conditions of ill health, showing the symptoms or signs which we are apt to associate with hyper or hypothyroidism, fall within the normal as far as the standard metabolism is concerned. These include clinical syndromes of definite abnormal states which have so far eluded classification, possibly on account of their not being associated with any known pathology. There are certain nervous states, disordered action of the heart, effort syndrome, in which the metabolic reading is of undoubted value in differentiating them from hyperthyroidism, for in these conditions the metabolic rate is not increased.

A high metabolic reading of more than plus 60% is as a rule a definite indication to avoid surgery. The help that Lugol's solution gives as a preparatory measure for operation is convincingly shown by a lessening in the metabolic rate which occurs within eight to fifteen days after such medication is begun. The resulting drop in metabolism has been found to reach its maximum within eight to fifteen days, and this is the time to operate, for, with the continuation of the iodine solution, the metabolic rate gradually rises again to its original level or thereabouts in a few weeks' time.

The clinician must realize that, besides certain standard conditions as to rest and diet which must be observed as a preparation for a metabolism test, there are definite errors which may occur with any test. Dubois tells us that three tests in every hundred are wrong because of some error made by the person conducting the test, no matter how skilled that person may be. Then, too, the instrument itself may have a slight defect which has escaped notice; or the patient may not have properly observed the standard conditions. One of my patients who gave a plus 40%, or more, reading, did what I told her regarding rest and no food the morning of the test but drove her car thirteen miles coming for the test. The second test a few days later came within the normal readings when somebody else did the driving for her. Another case, a mother, got up in the night for a couple of hours and took care of a sick child. I could not understand the resulting reading as the clinical symptoms indicated a colloid type of goitre. Even sending the patient to a private room in a hospital over night may be associated with an unusual disturbance of the nervous system. Then there are the errors in the test which result from apprehension on the patient's part as to whether they will be able to

breathe through such an apparatus, or they may often be too zealous or nervous and breathe unnaturally.

No physician is worth his salt who fails to place the proper value on the clinical examination of his case and then duly assesses the laboratory findings.

I think from the above instances of where errors may occur in any reading, it behooves us all to depend on the metabolic finding only after two, or better still, three readings have been made on different days. Then only can we be certain that a repeated similar finding is a definite record of the physiological activity of the thyroid gland. This record is of great clinical usefulness as an aid to diagnosis, or as an indication of the progress of our case or a help in determining the exact amount of thyroid extract or thyroxin to administer in a given case.

Summary.—1. A basal metabolic estimation is subject to errors on the part of the patient, the instrument, or the technician, and the estimation to be dependable should be made *under standard conditions* and be repeated always once and sometimes twice. This is particularly necessary where diagnosis, progress, or operability is in doubt.

2. Basal metabolism estimation is useful in hyperthyroidism as it gives a very accurate record of the clinical severity and progress of the disease. A high metabolic rate of plus 60% or over is a contra-indication to operation.

3. A basal metabolic rate is of great help in differentiating certain nervous states, effort syndrome, etc., from hyperthyroidism.

4. The dosage of the thyroid extract or thyroxin in hyperthyroidism can be regulated by repeated metabolic estimations.

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VANCOUVER HEALTH DEPARTMENT

STATISTICS — AUGUST, 1927

Total Population (Estimated)	137,197	
Asiatic Population (Estimated)	10,576	
		Rate per 1000 of Population
Total Deaths	116	9.95
Asiatic Deaths	16	17.84
Deaths—Residents only	72	6.17
TOTAL BIRTHS	311	26.68
Male 158		
Female 153		
Stillbirths—not included in above	10	
INFANTILE MORTALITY—		
Deaths under one year of age	11	
Death rate per 1000 births	35.37	

CASES OF INFECTIOUS DISEASES REPORTED IN CITY

	July, 1927		August, 1927		September 1 to 15, 1927	
	Cases	Deaths	Cases	Deaths	Cases	Deaths
Smallpox	0	0	0	0	2	0
Scarlet Fever	7	0	4	1	1	0
Diphtheria	23	1	10	1	13	0
Chicken-pox	3	0	1	0	0	0
Measles	14	0	1	0	0	0
Mumps	1	0	3	0	1	0
Whooping Cough	3	0	14	0	1	0
Typhoid Fever	1	1	2	0	0	0
Tuberculosis	14	11	20	14	9	–
Erysipelas	7	0	7	0	3	0
Poliomyelitis	0	0	1	0	3	2
Cerebral-Spinal Meningitis	0	0	1	1	0	0
Cases from outside city—included in above.						
Diphtheria	2	0	4	1	0	0
Scarlet Fever	2	0	2	1	0	0
Typhoid Fever	1	1	1	0	0	0
Poliomyelitis	0	0	0	0	2	1

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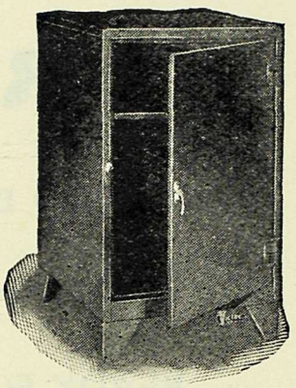

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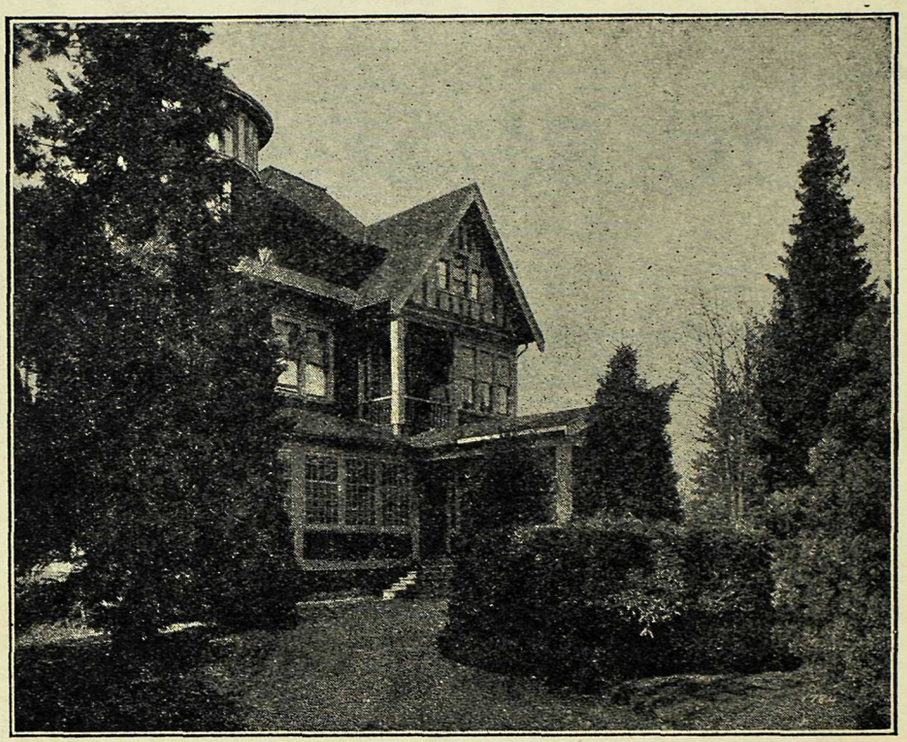

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