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EDITED, PRINTED AND PUBLISHED FOR THE ASSOCIATION

BY

DOUGLAS WHILLANS

CONTENTS

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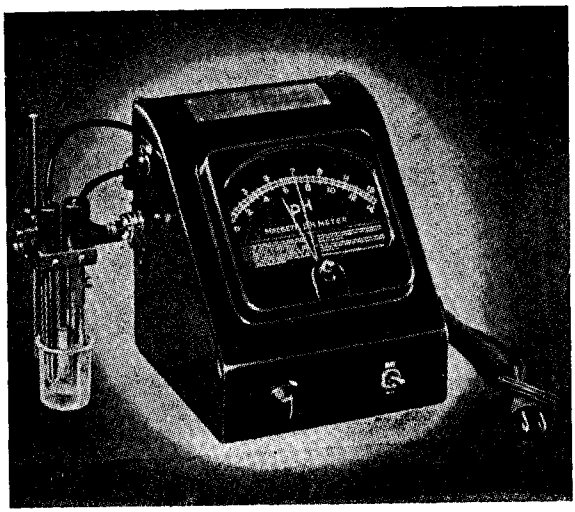

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JOURNAL

of the

NEW ZEALAND ASSOCIATION OF BACTERIOLOGISTS

Editors: D. Whillans, Auckland,

H. T. G. Olive, Wellington and M. O. Ekdahl, New Plymouth.

Vol. 4 - No. 3 JULY, 1949

EDITORIAL

As the Annual Conference 1949 draws nearer, it behoves one to think of this prospect, commencing with the Latin root "conferro—I bear together." In order that the literal translation of the root may have greater significance to us, it is proposed that this conference shall have an even wider coverage than those of other years.

A visit to the Dominion Physical Laboratory has been arranged, where a demonstration of the Electron Microscope is promised and many matters of physical and theoretical interest to the Bacteriologist will be discussed.

Similarly, a morning will be spent at the Animal Research Station, Wallaceville, where the practical application of our Science has a Veterinary bias.

A question hour has been included in the programme where the various angles of the medical application of our laboratory work will be discussed. To this session members should bring along their individual problems so that the solutions may be brought nearer them and that all may learn therefrom.

Having learned, let us return to work refreshed in our search for the ultimate scientific facts that will the sooner bring relief to the sufferer whose cause we have espoused.

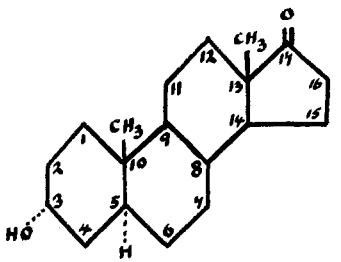
LABORATORY INVESTIGATIONS FOR THE ASSESSMENT OF ADRENAL CORTICAL AND GONADAL FUNCTION

J. B. BROWN

(*From the Department of Pathology, Public Hospital, Auckland.*)

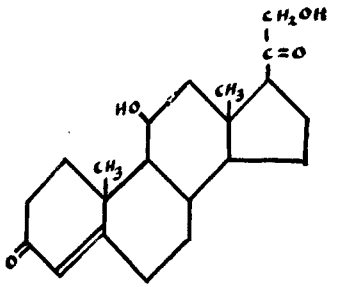
The adrenal cortex and gonads are closely related, both in their embryonic origin and the chemical similarity of the hormones produced by them. They both secrete hormones of the steroid type.

The adrenal cortex influences many body processes which include sodium retention by the kidneys, glyconeogenesis or the conversion of protein to sugar, the distribution of body hair, skin texture, bone formation and the destruction of lymphoid tissue to release antibodies in times of infection. The gonadal hormones can replace the adrenal hormones in a number of these functions. The structural formulae of two typical steroid hormones are given below.



Androsterone

A 17-Ketosteroid i.e. contains a Keto group at the 17th carbon atom



Corticosterone

An 11-oxysteroid i.e an oxygen atom is attached to the 11th carbon atom.

ADRENAL CORTEX:

Electrolyte Metabolism: The adrenal cortex elaborates a number of steroid hormones without which the kidneys are unable to conserve sodium. The chief of these is desoxycorticosterone. In the absence of these hormones sodium and therefore chloride is excreted in the urine at the expense of blood sodium and chloride. When this loss cannot be replaced by salt intake the blood chloride and sodium drop. This is an important feature of the condition known as *Addison's Disease* which is a state of *adrenal cortical* insufficiency, due to chronic tuberculosis or atrophy.

An untreated patient with Addison's disease will show a low blood sodium and plasma chloride with still appreciable amounts of sodium chloride present in the urine. Patients with normal adrenal function who have lost chloride by other causes such as continued vomiting will conserve what sodium chloride they have, and although the blood sodium and chloride are low, the urine chloride will be practically zero.

Addisonian crises occur when salt deprivation becomes acute. Here we find a compensatory increase in serum potassium (i.e., compensating for the lack of sodium) and markedly lowered serum sodium and plasma chloride values. Since a fall in blood sodium is invariably accompanied by a fall in plasma chloride, the estimation of chloride is usually sufficient in obtaining all the information required in these cases and the estimation of serum sodium and potassium, though interesting is seldom of more than academic interest.

In cases of suspected Addison's disease, this abnormal excretion of sodium has been made use of by *Cutler, Power and Wilder*, in their well-known sodium withdrawal test. Here the patient is placed on a sodium deficient diet rich in potassium. After a few days on this regime a patient with Addison's disease will show a definite lowering of blood sodium chloride, while appreciable amounts are still found in the urine. Normal patients will show a low excretion of sodium chloride. This is a reliable method for testing adrenal electrolyte function, but in positive cases may precipitate a crisis. The test must, therefore, be performed under careful medical supervision.

Kepler, Robinson and Power have recently introduced a test which depends upon the fact that patients with adrenal cortical insufficiency do not experience prompt diuresis after the ingestion of large amounts of water. The patient drinks nothing after 6 p.m. All urine passed between 10.30 p.m. and 7.30 a.m. is collected and the volume compared with those of hourly specimens passed after the ingestion of a large volume of fluid. A normal patient will excrete at least one larger hourly specimen of urine than the night specimen. The Addisonian subject, suffering from delayed excretion has not eliminated excess fluid by 9.30 p.m. and passes a large night volume, while the hourly morning specimens are smaller in amount. When this occurs, the authors have introduced a further refinement which includes a specimen of blood taken at the completion of the test. They have devised a formula (see later) where the three ratios involved are lower in cases of Addison's disease than in normal subjects.

The Kepler, Robinson, Power water excretion test is a simple and safe screening test. It is, however, sensitive to renal damage (c.f. Specific Gravity fixation test) and positive cases should be confirmed by more reliable data.

Carbohydrate Function: The steroids of the adrenal cortex which influence carbohydrate function ("S" Hormones of the adrenal) have an oxygen atom attached to the eleventh carbon atom and are known as 11-oxysteroids. Corticosterone is a member of this group. The "S" hormones are necessary for the conversion of protein to glucose (glyconeogenesis) by the liver, and play an important part in the regulation of the fasting blood sugar. The production of "S" hormone is increased in trauma or infection (the alarm reaction of Selye). There is evidence that increased circulation of "S" hormone leads to increased breakdown of lymphoid tissue with the release of immune globulin. This is an important defensive mechanism. Patients with Addison's disease lack this defensive mechanism and are therefore susceptible to even mild intercurrent infections.

"S" hormone production is assessed by the Glucose Tolerance Test and the Insulin Sensitivity Test. There are more direct chemical and biological estimations, but these are at the moment outside the scope of the routine laboratory.

Excess production of "S" hormone, such as is found in Cushings disease, leads to excessive destruction of body protein to glucose and an insulin resistant diabetes. The glucose tolerance test shows a raised fasting blood sugar and a decreased glucose tolerance, yielding a typical diabetic curve.

Decreased production of "S" hormone, such as is found in Addison's disease, leads to a low fasting blood sugar and an increased glucose tolerance the glucose tolerance curve being flat. The insulin sensitivity curve usually shows a normal or sometimes delayed initial drop in blood sugar with a delayed return to normal levels, i.e., the patient is hypoglycaemia—unresponsive due to diminished glyconeogenesis.

The glucose tolerance and insulin sensitivity tests are valuable confirmatory tests in the diagnosis of Addison's disease.

Androgenic Function: The adrenal cortex elaborates a number of steroid hormones with male hormone like properties. These are known as the androgenic or "N" hormones of the adrenal. An index of their production is given by the twenty-four-hour excretion of 17-ketosteroids in the urine. These hormones play an important part in the growth and distribution of body hair and are powerfully anabolic, in that they stimulate nitrogen retention by the body and promote muscle formation.

Over-activity of the adrenal cortex leading to excess production of "N" hormone is the basis of the "androgenital syndrome."

Adrenal hyperplasia occuring in the female foetus is an important cause of pseudo-hermaphroditism. In boys it causes precocity, and in women and girls masculinization is the result.

Here the full syndrome consists of increase in body weight with masculine distribution of body hair, florid complexion, deepening of the voice and loss of menstrual function. Increase in production of "N" hormone is due to either simple hyperplasia of the adrenal cortex or to an adrenal cortical tumour. In both cases, the 17-ketosteroid excretion will be high. Exceptionally high values are usually only found in the presence of a tumour. Fractionation of 17-ketosteroids into α and β fractions (α and β refer to the spatial configuration of the 3-carbon atom) will differentiate between hyperplasia and tumour. If the increase is due mainly to the β fraction the existence of a tumour can be assumed. Surgical removal of this if practicable, is usually followed by a rapid return of the patient to normal. In the case of hyperplasia surgical intervention has given disappointing results.

In the female the whole of the urinary 17-ketosteroid is derived from the adrenals (average about 9 mgms. in 24 hours), while in the male a portion is also contributed by the testes (about 5 mgms. in 24 hours). Female patients suffering from Addison's disease therefore have a low to zero excretion of 17-ketosteroids (less than 1.0 mgms. per 24 hours) since the adrenal no longer functions, while the male patient will excrete 2-5 mgms. of testicular origin.

Gonads: The gonads are closely related to the adrenal cortex in that they secrete hormones of the steroid pattern.

In the male, follicular stimulating hormone of the pituitary (F.S.H. which is readily measured by a modification of the Aschheim-Zondek test, reaction 1) stimulates the germinal epithelium and spermatogenesis. The interstitial cells which excrete the male hormone are under the influence of the luteinizing hormone of the pituitary. Their function is measured by the excretion of 17-ketosteroids in the urine (about 9 mgms. by the adrenals and 5 mgms. by the testes). Primary testicular failure gives an excretion of 17-ketosteroids which falls within the female range. Testicular hormone (testosterone) normally tends to inhibit pituitary function. The absence of this inhibition leads to the presence of increased amounts of F.S.H. in the urine. Secondary testicular failure due to lack of pituitary stimulation leads to a similar 17-ketosteroid excretion, but the urinary F.S.H. is negative. We can, therefore, by means of the F.S.H. estimation, determine the site of testicular failure (i.e., whether primarily testicular or secondary to pituitary) and treat accordingly.

We find a similar picture in the female. Under the influence of F.S.H., Graafian follicles mature in the ovary to secrete oestrogenic hormone which can be estimated only with difficulty either biologically or chemically. After ovulation and rupture of the follicle, the corpus lutem grows under the stimulation of the lutein-

izing hormone of the pituitary and secretes progesterone. This is partly eliminated in the urine as pregnanediol and can be estimated as such. In the absence of pregnancy the corpus luteum retrogresses with the onset of menstruation. The menstrual cycle with its functional reactions is still our most sensitive index of ovarian activity. Primary ovarian failure (the menopause), like primary testicular failure, leads to a raised F.S.H. in the urine, while secondary failure due to lack of stimulation by the pituitary leads to a zero urinary F.S.H.

Pituitary: We cannot consider the function of the gonads and adrenal glands without considering the function of the pituitary. The pituitary elaborates specific hormones, protein in nature, which stimulate the function of the other endocrine glands; for instance, thyrotrophic hormone stimulates the thyroid and adrenotrophic hormone stimulates the adrenals. Under this stimulation the glands secret their own hormones which, in turn, tend to inhibit the production by the pituitary of the hormones which are stimulating them. A fine balance is therefore set up. Hyperfunction and hypofunction of the pituitary therefore cause secondary changes in the other endocrine glands. Hyperfunction is usually selective; for instance, we find excess thyrotrophic hormone in hyperthyroidism and increased growth hormone in gigantism and acromegaly. Hypofunction of the pituitary, however, usually affects all the other endocrine glands. For instance, pituitary myxoedema is usually found associated with Addisonian symptoms and atrophy of the sex glands. This condition of generalised atrophy of the endocrine glands is termed *Simmonds disease* or *panhypopituitarism* (pan-all). This is a not uncommon disease, eleven proven cases having already been investigated in Auckland. It is more common in women than in men and is frequently associated with a history of severe haemorrhage at childbirth. Cases suffering from Simmonds' disease show a low Basal Metabolic Rate and raised blood cholesterol due to thyroid hypofunction, the full syndrome of Addison's disease, sexual impotence in men with amenorrhea in women, a 17-ketosteroid excretion below 1 mgm. in 24 hours and a negative F.S.H.

Anorexia Nervosa is a condition which can closely resemble panhypopituitarism and is due to a psycho-neurotic abstinence from food. Young unmarried women are the chief offenders. Lack of high-grade protein due to "dieting" causes a depression of pituitary activity and a loss of appetite. Unless food is forced to promote the return of appetite, the condition may ultimately be fatal. Laboratory and clinical investigation show varying degrees of panhypopituitarism, but the excretion of 17-ketosteroids is usually not so low as that found in true panhypopituitarism. Rapid clinical improvement follows an increased intake of high grade protein without any other treatment.

Instructions for the carrying out of the less weil-known laboratory tests.

Cutler Power Wilder Sodium Withdrawal Test. Ref. Mayo Clinic, 1938, 13, 244.

The patient is put on a diet containing vegetables, fruit, salt-free bread and butter, cream, milk, eggs and lean beef providing 0.9-1.0 gm. of chloride, 0.5-0.6 gm. of sodium and 4 gm. of potassium for three days. Fluid intake on the first day is not restricted. During the afternoon of the first day and morning of the second extra potassium is given as potassium citrate in a dose of 0.9 gm. of potassium citrate per kilogram body weight. Fluid intake is restricted to 40 cc. per kilogram body weight on the second day, and on the third day 20 cc. per kilogram are administered before 11 a.m. The examination ends at noon on the third day. Urine is collected from 8 a.m.-12 noon on the third day. If the concentration of chloride in this specimen exceeds 225 mgms. % (as chloride ion) Adrenal insufficiency is strongly suggested, if it is less than 125 mgm. % adrenal insufficiency is unlikely. If values lie between these figures, the test should be continued until more significant results are obtained.

At the close of every examination, an intravenous injection of 1000 c.c. solution containing 50 gm. dextrose, 10 gm. sodium chloride, 5 gm. of sodium citrate and 20 c.c. of an active preparation of cortical extract should be given. This solution is always kept on hand in case of emergency since this regime may precipitate an Addison's crisis.

Kepler Power Robinson "Water Test"

The day preceding the test the patient eats three ordinary meals, but omits extra salt. He is requested not to eat or drink anything after 6 p.m., but up to this time he may drink water as desired. At 10.30 p.m., he empties his bladder and the urine is discarded. All urine which is voided from them on, up to and including 7.30 a.m. is collected. The volume of this urine is measured and the urine saved for chemical analysis. Breakfast is omitted and the patient is asked to empty his bladder again at 8.30 a.m. He is then given 20 c.c. of water per kg. body weight (9 c.c. per lb.) which should be drunk within 45 minutes. The patient should empty his bladder at 9.30, 10.30, 11.30 a.m. and 12.30 p.m. The volume of the largest of the four specimens is measured, and compared with the volume of the 10.30 p.m.-7.30 a.m. night urine. If the volume of any single hourly specimen voided during the morning is greater than the volume voided during the night, the result is negative, indicating the absence of Addison's disease.

If the volume of the largest hourly specimen voided during the morning is less than the volume voided during the night, we go

on to the second stage of the test.

Blood is drawn from the patient any time between 9.30 a.m. and 12.30 p.m., and the plasma is analysed for urea and chloride. The specimen of urine voided during the night is also analysed for urea and chloride. The four determinations, and the volume determinations of the first stage are applied to the following equation.

$$A = \frac{\text{Volume of largest hourly day specimen of urine (c.c.)}}{\text{Volume of night urine (c.c.)}} \times \frac{\text{Plasma-chloride (mg. \%)}}{\text{Urine chloride (m.g. \%)}} \times \frac{\text{urine-urea (mg. \%)}.}{\text{plasma-urea (mg. \%)}.}$$

If A is greater than 30, the patient probably does not have Addison's disease.

If A is less than 25, the patient probably has Addison's disease, provided that nephritis can be excluded.

Insulin Tolerance Test (Insulin Sensitivity Test).

Ref. Fraser, Albright and Smith, J. Clin. End. 1941, I, 297.

A glucose tolerance test is performed as a preliminary and the patient is kept on an adequate carbohydrate diet for at least three days before the test.

The patient, fasting from the night before is given intravenously 0.1 units of ordinary insulin per kg. body weight. Capillary blood samples are taken at 0, 20, 30, 45, 60, 90 and 120 minutes. Clinical symptoms of hypoglycaemia are carefully noted. These usually consist of faintness and dizziness starting at about twenty minutes, followed by a feeling of warmth and perspiration. The perspiration, if present, reaches its maximum between 30 and 35 minutes and appears to herald the commencement of the return of the blood sugar to normal. Symptoms usually disappear by 45 minutes and the patient often lapses into a state of lassitude and may sleep soundly. In cases of Addison's disease or panhypopituitarism, hypoglycaemic symptoms may persists for some time and until these have disappeared the patient should be watched carefully and should be kept awake. Clouding of consciousness is a grave sign and may be detected by the inability of the patient to answer questions intelligently or to carry on a conversation in the usual manner. In this case, the test should immediately be interrupted by the oral administration of 50 gms. of glucose dissolved in a cup of water or by the administration of intravenous glucose.

In cases suspected of having panhypopituitarism with a flat glucose tolerance curve, one should start with one-third the calculated dose of insulin and symptoms should be even more carefully noted since these patients are often abnormally sensitive to insulin, a standard dose eliciting a dangerously low blood sugar level.

After the test is concluded, the patient is given a high carbohydrate meal.

A normal result shows an initial blood glucose drop to about 50% of the fasting value in 30 minutes with a return to 80% of the fasting values in 60 minutes.

Summary of results in two typical cases, one of Addison's disease and one of panhypopituitarism.

Addison's Disease:

Case: Miss L.A.P., aged 17. Pigmented skin, B.P. 115/70, X-ray shows extensive calcification in the region of the suprarenals on both sides. Irregular menses.

Kepler Robinson Power Water Excretion test—

Volume of night urine 290 c.c., volume of largest morning specimen, 115 c.c., urea content of night urine 1600 mgms. %, chloride content of night urine 920 mgms. % (as NaCl), plasma urea 30 mgms. %, plasma chloride 556 mgms. % (as NaCl).

A = 115/290 x 556/920 x 1600/30 = 13.

Glucose Tolerance test: Fasting sugar 72 mg., ½ hr., 96 mg., 1 hr. 90 mg., 1½ hr. 88 mg., 2 hr. 68 mg. per 100 c.c.s. blood.

Insulin Tolerance test, (0.1 units IV/Kg.) values expressed as % fasting sugar level.

Fasting 100% (80 mg. %), 20 min. 61%, 30 min. 57%, 45 min. 57%, 60 min. 61%, 90 min. 76%, 120 min., 86%.

Serum sodium, 308 mg. %.

Serum potassium, 18.5 mg. %.

17-ketosteroids, 0.5 mg. per 24 hours.

Panhypopituitarism:

Case H.W., aged 43. Pituitary tumour removed at age of 21, followed by symptoms of hypopituitarism.

Small man with wrinkled, hairless face, female distribution of fat, poorly developed external genitalia and loss of pubic hair.

B.P. 118/80. B.M.R. —40%, blood cholesterol, 230.

Kepler Robinson Power water excretion test:

Volume of night urine 140 c.c., largest day urine, 66 c.c., night urine chloride, 830 mg. % (as NaCl) urea 600 mg. %, plasma chloride, 530 mg. %, plasma urea, 30 mg. %.

A = 66/140 x 530/830 x 600/30 = 6.

Glucose Tolerance test: Fasting sugar level 112 mg%, ½ hr., 120mg. %, 1 hr., 114 mg. %, 2hr, 111mg. %.

Insulin Tolerance test: (0.033 units, IV. Kg.) values expressed as % fasting value. Fasting sugar 100% (108mg%), 20 min. 49%, 30 min. 41%, 45 min. 47%, 60 min. 48%, 90 min. 47%, 120 min. 47%.

17-ketosteroids—zero excretion in 24 hours.

F.S.H. negative to 6.6 mouse units excreted in 24 hours.

THE IDENTIFICATION OF S. BOVIS MORBIFICANS INFECTION IN NEW ZEALAND

By S. W. Josland,

Animal Research Station, Wallaceville, N.Z.

During the past year two cultures of salmonella that have been submitted to this Station for investigation have been identified as S. bovis morbificans. One culture (1948) was from an adult patient in Wellington, and the second (1949) was from an infant in Auckland Hospital. Confirmation of the identification of the Wellington strain was obtained from both the Kentucky and Adelaide Salmonella centres. As the Auckland organism, which was submitted by Mr. J. Callaghan, of the Auckland hospital laboratory, possesses an identical antigenic structure, the serological examination of this strain will be described.

A broth culture of the organism, which was motile, was tested in the water bath at 55° C, against polyvalent salmonella sera P.S.A., P.S.B. and P.S.C. Strong agglutination immediately occurred with P.S.B. serum.

Slide agglutination against colonies emulsified with a little saline was then carried out against the more commonly occurring somatic group sera and strong agglutination occurred against factor VI., VII. and VI., VIII sera. Saline suspensions prepared after alcoholisation were then tested for 24 hours at 37° C, against factor VI., VII., VI., VIII., VI II XX (S. kentucky) sera and agglutination was obtained against all three; to titre against the VI., VIII serum. Agglutination was also obtained with a pure VIII. serum prepared from S. newport serum (VI. VIII) by double absorption with S. oranienburg (VI. VII).

The somatic groupings were therefore VI., VIII.

For the identification of the flagellar antigens, formalinised broth cultures were tested in the water bath at 55° C against those specific sera which are included in the polyvalent P.S.B. serum with which agglutination had already been obtained. The sera used where those for factors e n x, e h, r, y and z.

The only serum with which agglutination occurred was factor r to a titre of 1/1600. Slight cross agglutination occurred also with specific factor i serum. The r-i cross agglutination relationship has been established by Kauffman and by Bruner and Edwards.

The culture was therefore in phase 1. By growing the organism in semi-solid agar to which factor r serum had been added, phase 2 was readily obtained and agglutination occurred with 1, 5 and single factor 5 sera. The antigenic groupings were therefore

VI, VIII; r, 1.5, which is that corresponding to S. bovis morbificans. It was further shown that the organism was capable of exhausting by absorption these antigenic factors from somatic and flagellar sera containing them.

DISCUSSION:

S. bovis morbificans was first isolated by Basenau at Amsterdam in 1893 from the carcass of a cow that had aborted and had developed metritis. Cultures of Basenau's bacilli were kept and studied by a number of workers, including Bruce White (1926), who worked out its antigenic structure.

The next recorded isolation of this salmonella was by Sladden and Scott (1927) from human faeces examined during an outbreak of food poisoning in Swansea, pressed meat being incriminated as the cause of the disease.

In 1937, Henning and Greenfield established the antigenic structure of an organism isolated from pork, the consumption of which led to an outbreak of food poisoning in Capetown and showed that it was S. bovis morbificans.

That this organism can be pathogenic for sheep was shown by Stewart (1940), who isolated S. bovis morbificans from the mesenteric and colic lymph glands of affected sheep in a mortality of some 200 sheep in New South Wales. That S. bovis morbificans is not uncommon in Australia is shown by the identification of 15 strains, 11 from children and 4 from adults with gastro-enteritis by Atkinson et al (1947) from a total number of 156 salmonella strains examined. On the other hand, Bruner and Edwards (1949) in the United States, who examined over 12,000 salmonella cultures during the years 1934-47, found but 3 strains of S. bovis morbificans from human sources.

At this Station, we have not as yet found S. bovis morbificans in any case of salmonellosis of animal origin that have been investigated.

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COUNCIL MEETING

This was held in Kelvin Chambers, Wellington, on Saturday, 14th May, 1949, at 10.30 a.m. There were present Mr. N. J. Ellison (chairman), and Messrs. M. O. Edkahl, G. W. McKinley, E. L. F. Buxton, H. Olive and S. O. Jarratt. Apologies were received from Messrs. D. Whillans and D. H. Adamson. The resignation of Mr. J. A. Samuel from the Council owing to his taking up a position in Samoa, was accepted with regret.

It was resolved that copies of remits to the Salaries Advisory Committee and the Pathologists' Conference be published in the Journal (see later).

Miss A. L. Bennett, of Christchurch Hospital, was appointed Overseas Corresponding Member in England.

The following new members were accepted as members of the Association:—Miss Jeanette Grey (New Plymouth Hospital), Miss Lois Evans (Christchurch Hospital), Miss L. J. Grey and Miss Honor L. Price (Invercargill Hospital), Miss Barbara A. Wright (Napier Hospital), Mr. D. E. Parfoot (Timaru Hospital), Miss Joan Mattingley and Miss Joan White (Wellington Hospital). These were all junior members of the Association.

It was resolved that letters of congratulation be sent to successful candidates for the Final Examination.

Mr. E. L. F. Buxton and G. W. McKinley were appointed delegates to the Pathologists' Conference held in Christchurch on May 25th, 1949.

Remits were also sent to the Director-General of Health re the Intermediate Examination, the Diploma Examination and the holding of examinations in both North and South Islands.

COPY OF LETTER AND REMITS

Miss M. B. Howard,
Rt. Hon. the Minister of Health,
Ministry of Health,
Parliament Buildings,
WELLINGTON.

Dear Madam,

We are deeply grateful for your encouragement and interest in our efforts to secure a more appreciative financial recognition of our services. Through the tedium of interviews, communications, remits and negotiations, we have always felt we have had your sympathetic attention. It has inspired within our association a sense of justice in the fairness of your administration.

The Hospital Employment Regulations, 1948, Amendment No. 2, 1948/192, met with general approval. The anomalies which do exist we are leaving to the individuals concerned to bring before the Salaries' Advisory Committee when this body meets again.

However, in reviewing the Regulations in Council, the Association is submitting several remits for your consideration. These are also being forwarded to the Salaries Advisory Committee, to the Hospital Boards' Association and to the Director-General of Health.

Yours faithfully,
S. O. JARRATT,
Hon. Secretary.

The following remits from the Executive of the New Zealand Association of Bacteriologists (Inc.), are being submitted to the Salaries Advisory Committee for sympathetic consideration.

Hospital Employment Regulations, 1948, Amendment No. 2, 1948/192.

Remit No. 1. *Laboratory Assistant Salary.*

Part IV. "Hospital Bacteriologists, Bacteriological Trainees, and Laboratory Assistants."

Page 4. Under Section "47."

"3" Laboratory Assistant.

'(c) After the eighth year of service or earlier on the recommendation of a Pathologist, a maximum amount not exceeding £500 per annum, as approved by the Minister in the special circumstances."

Remit No. 2. Overtime Payment.

Page 5. Section "52." Overtime. Second half of paragraph 1.

Provided that the total overtime in respect of any year ending on the 31st day of March, plus the salary for that year shall not exceed £670 and provided also that the worker shall have the option of time off in lieu of overtime."

Remit No. 3. *Annual Leave.* January 2nd as a Public Holiday.

Page 6. Section 53. Paragraph 4.

"That January 2nd be also recognised in the regulations as a Public Holiday. As legislation has latterly been passed declaring January 2nd a Public Holiday, the Association awaits clarification of the position.

MEMORANDUM: Pathological Conference, May 25th, 1949.

(1) INTERMEDIATE EXAMINATION

My Association views with concern the delay in implementing the Intermediate Examination.

We understand that acting on the recommendation of the Advisory Committee of Pathologists, the Department has already agreed in principle to the institution of the Intermediate Examination. Candidates have been preparing for this examination in a number of laboratories and this has entailed a considerable amount of work on the part of candidates and bacteriologists concerned. My Association was under the impression that the first year examination was to be held at Auckland in May of this year. Surely whatever alteration may be made in the conditions or status of the qualifying examination for Hospital Bacteriologists, there will still be a need for an Intermediate Examination to cater for the requirements of the staff in smaller hospitals and for those who are not able or anxious to attain the standard required for the qualifying examination. It is the hope of the Association that it will be a prerequisite of the final examination in the future and that a candidate who passes, shall be entitled to sit for the final examination after a further two years spent in a laboratory controlled by a Pathologist.

My Association deplores the lack of any official information on this matter.

(2) TITLE OF CERTIFICATE

We have not as yet received any official information concerning the institution of a Diploma Examination under the aegis of the University authorities. We would like our representatives to be advised of the situation in this respect.

(3) CERTIFICATE EXAMINATION

May we enquire whether it is the intention of the Department to hold the final examination in both the North and South Islands.

(Sgd.) S. O. JARRATT,

On behalf of the Association.

3rd June, 1949.

Mr. S. O. Jarratt,
Secretary,
N.Z. Association of Bacteriologists,
C/o Public Hospital,
PALMERSTON NORTH.

Dear Sir,

INTERMEDIATE EXAMINATION FOR HOSPITAL LABORATORY TRAINEES

1. Referring to your letter of 17th May, I write to inform you that the Intermediate Examination for Hospital Laboratory trainees was given full consideration at the Conference of Pathologists, held at Christchurch Hospital, on 25th May. After receiving the submissions of your two representatives, Messrs. Buxton and McKinley, the Conference decided that the examination be instituted this year on the basis recommended by the Advisory Committee, with certain amendments and additional provisions agreed to by the Conference.

The terms and conditions of the examination as finally determined are as follows:—

Terms and Conditions—Intermediate Examination for Hospital Laboratory Trainees.

(1) *Pre-Entry Educational Qualifications.*

Candidates are required to have passed the School Certificate Examination at the commencement of the three-year training period.

(2) *Syllabus*: As attached.

(3) *Training Centres*:

Hospital Laboratories under the control of either a pathologist or qualified Hospital Bacteriologist and approved by the Director-General of Health, will be recognised as training centres for the examination. Trainees in the laboratories of Private Pathologists will also be eligible for the examination.

(4) *Period of Training*:

The training period shall be not less than 3 years in an approved laboratory, except for a qualified nurse, in which case it shall be not less than 2 years.

(5) *Examination*:

On completion of the prescribed period of training and subject to suitable recommendation from the pathologist or hospital bacteriologist under whom training has been undertaken, the trainee may sit for the examination. It will consist of written practical and oral portions and will be conducted by the Department of Health.

Examiners:

Of the two examiners, one will be a pathologist, the other a qualified senior hospital bacteriologist. It has been decided that the examining pathologist will be one who does not also examine for the Certificate of Proficiency Examination.
Dates of Examination: The examination to be held at not less than six monthly intervals, commencing October, 1949, and thereafter in May and early November of each year. *The first examination in October, 1949, to be held at Auckland Hospital.*

(6) *Eligibility to sit Qualifying Examination for Certificate of Proficiency.*

In the case of trainees who commence training in a District Hospital Laboratory and qualify in the Intermediate Examination, they will be eligible to sit the Examination for the Certificate of Proficiency, provided that on passing the Intermediate Examination, they undertake two years' training in a base training laboratory.

Hospital Boards which maintain laboratories under a Pathologist or Hospital Bacteriologist, also Private Pathologists, are being advised of the examination and will be supplied with copies of the terms and conditions for the information of trainees.

(2) *Examination for the Certificate of Proficiency in Bacteriology and Clinical Pathology.*

The Conference decided that as the suggestions put forward by the University of Otago concerning the institution of a Diploma in Bacteriology would not be practicable and that in order to meet requirements, endeavours be made to adopt the present examination.
It was resolved:—

(a) *Title of Certificate*: That this be changed from its present title to "Certificate of Proficiency in Hospital Laboratory Practice."

(b) *Syllabus*: That the Advisory Committee of Pathologists draw up and submit a revised syllabus for the Certificate of Proficiency Examination.

Yours faithfully,

T. R. RITCHIE,

Director-General.

SYLLABUS—INTERMEDIATE EXAMINATION FOR HOSPITAL LABORATORY TRAINEES

1 *The Operation and Maintenance of Laboratory Equipment*:
All types of sterilisers; the microscope; centrifuges; stills; thermo-regulated apparatus; filters; hydrogen ion apparatus (visual); colorimeters; balances.

2 *Preparation of Glassware and Reagents*:
Types of laboratory glassware, including graduated glassware; the cleaning of glass; the disposal of cultures and specimens; the preparation and sterilisation of:

(a) Specimen containers; (b) Pasteur pipettes; (c) Culture media; The preparation and use of routine stains and solutions.

3 *Bacteriology*:
An elementary knowledge of the classification and nomenclature of bacteria; an elementary knowledge, including practical recognition of the following:—Staphylococci, streptococci (including S. pneumoniae and enterococci), the Neisseria group, C. diphtheriae and diphtheroid organisms, H. influenzae, the typhoid-dysentery-food poisoning group, the coliform group, Br. abortus, M. tuberculosis, Vincent's organisms; The routine bacteriology of water, milk and milk products.

4 *Urinalysis*:
Deposit; bile; specific gravity; acetone; sugar and albumin (qualitative and quantitative); urea (hypobromite).

5 *Antibiotics*:
The storage and dispensing of antibiotics. Standard methods of testing sensitivity of organisms to antibiotics.

6 *Haematology*:
The collection of specimens, excluding venipuncture; an elementary knowledge of the origin and development of cells; the technique of the complete blood count; a knowledge of the differential count, excluding opinion on anaemias, leukaemias, etc., but ability to recognise such abnormalities; reticulocyte and platelet counts; haematocrit technique; sedimentation rate; coagulation and bleeding times; blood grouping and compatibility test; icterus index.

7 *Examination of Puncture Fluids*:
Cytology of such fluids; bacteriology and chemistry as under separate sections.

8 *Biochemistry*:
Blood; sugar, T.N.P.N., C.S.F.; estimation sugar, chlorides, protein, Faeces; Occult blood, Gastric Analysis.

9 *Miscellaneous*:
A knowledge of first-aid treatment and antidotes to cover accidental injuries and poisoning in the laboratory. A thorough knowledge of aseptic technique as applied to laboratory work and to personal safety; the packing of specimens and the postal regulations.

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THE NEW ZEALAND ASSOCIATION OF BACTERIOLOGISTS (INC.).

Election of Officers for the Year 1949-50

The following are the nominations of members standing for election as Officers of the Council of the New Zealand Association of Bacteriologists (Inc.) for the year 1949-50.

The election will be held at the Annual General Meeting to be held in the Wellington Hospital on July 29th and 30th, 1949.

Members unable to attend this meeting should vote by proxy, Rule 13 (g) 1, and are urged to do so in order that a representative vote be obtained.

PRESIDENT (One Vacancy)

E. L. F. Buxton (Wanganui Hospital); N. J. Ellison (C/o Dr. P. P. Lynch, Wellington); and G. W. McKinley (Waipawa Hospital).

VICE-PRESIDENTS (Two Vacancies)

E. L. F. Buxton (Wanganui Hospital); N. J. Ellison (C/o Dr. P. P. Lynch, Wellington; G. W. McKinley (Waipawa Hospital), and D. Whillans (Auckland Hospital).

SECRETARY (One Vacancy)

S. O. Jarratt (Palmerston North Hospital); G. W. McKinley (Waipawa Hospital).

TREASURER (One Vacancy)

D. H. Adamson (Christchurch Hospital); S. O. Jarratt (Palmerston North Hospital); G. W. McKinley (Waipawa Hospital); H. T. G. Olive (Wellington Hospital).

ORDINARY MEMBERS OF COUNCIL

(Three Vacancies)

D. H. Adamson (Christchurch Hospital); H. G. Bloor (Blenheim Hospital); Miss J. Byres (C/o Dr. Fowler, Auckland); L. G. Eccersall (Waikato Hospital); M. O. Ekdahl (New Plymouth); B. Gibson (Christchurch Hospital); V. J. Hawke (Nelson Hospital); F. Hilder (Christchurch Hospital); S. O. Jarratt (Palmerston North Hospital); Miss B. McKenzie (Christchurch Hospital); G. W. McKinley (Waipawa Hospital); H. T. G. Olive (Wellington Hospital); K. B. Ronald (Oamaru Hospital); I. W. Saunders (New Plymouth Hospital); H. Ward (Timaru Hospital); D. Whillans (Auckland Hospital).

As several names appear more than once, members should give their proxies a list of preferences.

THIS IS *NOT* A VOTING PAPER.

S. O. JARRATT,
Hon. Secretary.