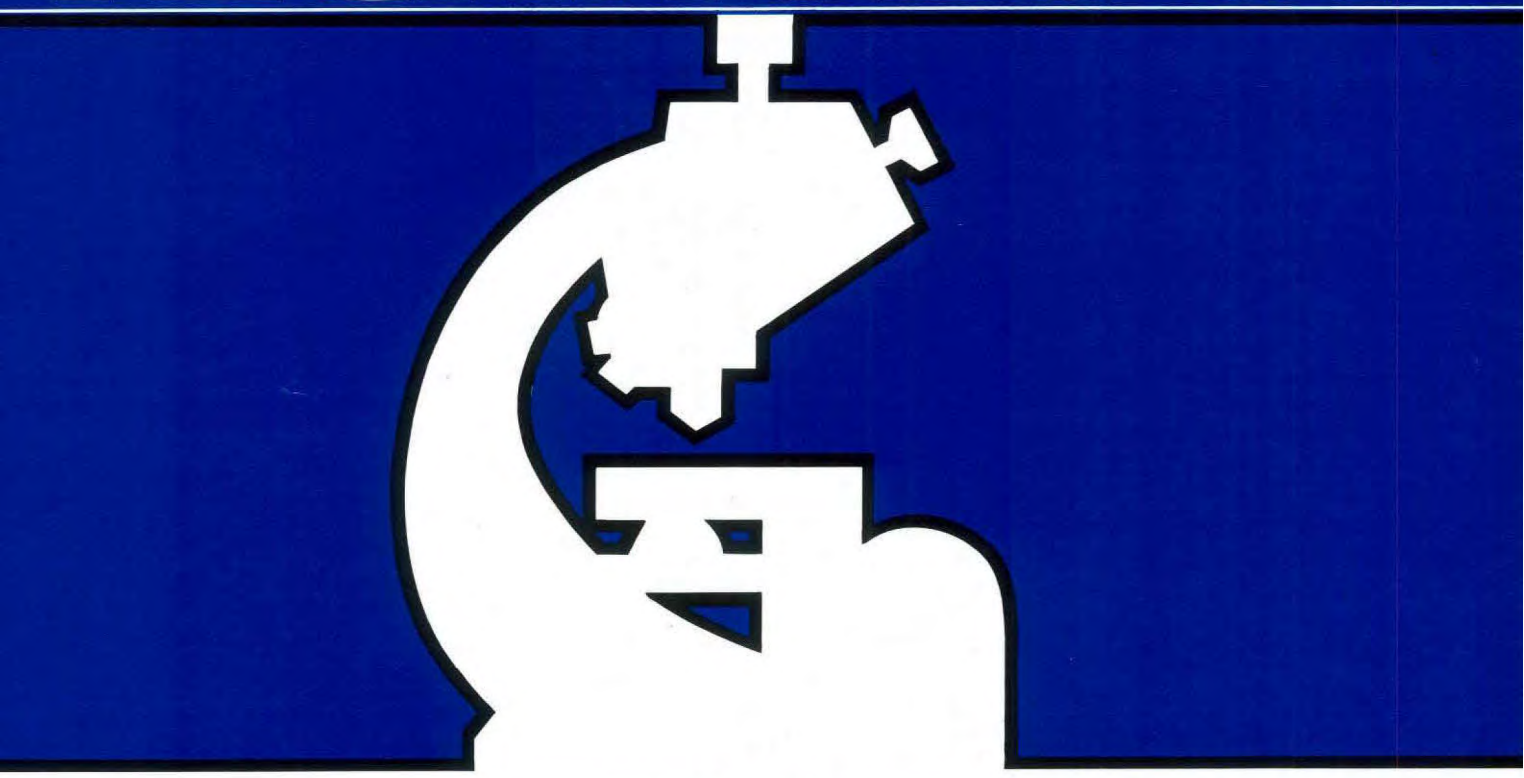
Vol. 38 No. 2 May 1984

ISSN.0028-8349

NEW ZEALAND JOURNAL OF

MEDICAL LABORATORY TECHNOLOGY



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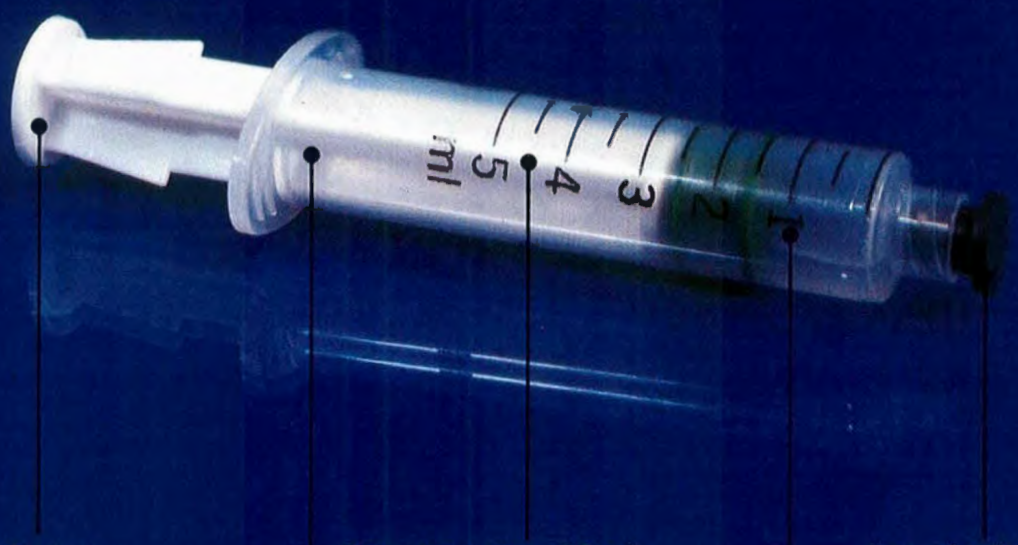

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MEDICAL LABORATORY TECHNOLOGY

Vol. 38 No. 2 May 1984 ISSN 0028-8349

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DATES OF PUBLICATION

The months of publication for 1984 are March, May, August and November.

This Journal is abstracted by: Biological Abstracts, Chemical Abstracts, Cumulative Index Nursing and Allied Health Literature, Current Clinical Chemistry, Hospital Abstracts, Institut nautchnoi informatsii.

Neutrophil Myeloperoxidase Deficiency — is it significant?

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From a paper presented at the 39th Annual Scientific Meeting of the New Zealand Institute of Medical Laboratory Technology, Napier, 18-19 August, 1983.

Abstract:

Evaluation of subjects with myeloperoxidase (MPO) deficiency has been limited by the small number of subjects detected. By using an automated cytochemical analyzer, such as the Hemalog-D[1], for routine haematology work, the chances of detecting MPO deficiencies are very much increased.

This study was initiated to compare results with other reported surveys [2,3,4], and to detect any possible reasons for such deficiencies.

Results show similar findings to the previous large surveys. A short summary of the literature is given. The significance of MPO deficiency could well lie in long-term assessment, as MPO has been shown to have a role in tumouricidal activity[2]. MPO deficiency per se does not seem to contribute to increased infection, as there are multiple neutrophil killing mechanisms.

Key Words:

Myeloperoxidase (MPO) deficiency; Technicon Hemalog-D; cytochemistry.

Historical Review:

Myeloperoxidase (MPO) was reported over 100 years ago, when Krebs published an article in a German medical journal, describing the detection of what we today know as MPO, in pus[5]. In 1941, Agner, a Scandinavian researcher, called the enzyme verdoperoxidase, because of its colour. Agner also reported that the isoelectric point of verdoperoxidase was greater than pH 10[6,7]. The more functional name Myeloperoxidase (MPO) was finally suggested with the finding that the milk peroxidase, called lactoperoxidase, was also green in colour.

In 1970 it was shown that eosinophil peroxidase is chemically distinct from neutrophil peroxidase[8]. Although the eosinophil and polymorphonuclear neutrophil are thought to derive from a common progenitor, the structural uniqueness of eosinophil peroxidase, and its presence in MPO deficiency, indicate that eosinophil peroxidase is under separate genetic control.

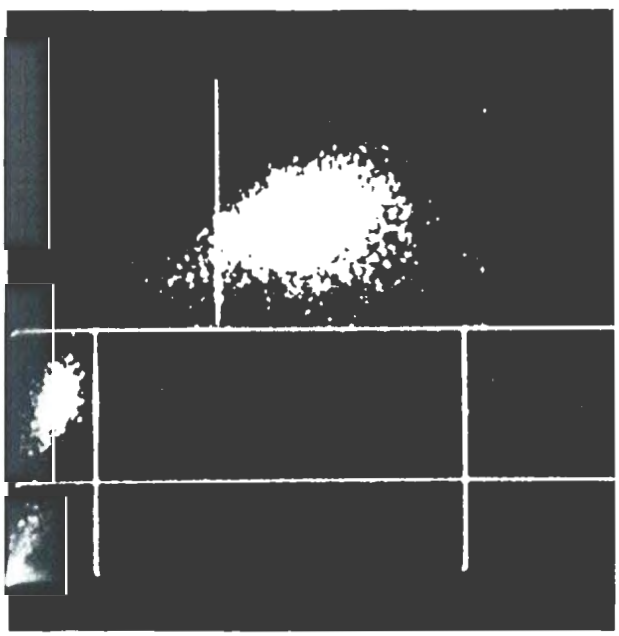


Figure 1. Normal Hemalog-D cell pattern.

In 1972, a group doing preparatory disc electrophoretic separation of the MPO of the normal human leucocyte showed that there were six isoenzymes[9]. Studies of isolated bands suggested that each isoenzyme is a dimeric molecule, arising from three kinds of monomeric subunits, A, B, and C. The isoenzymes are referred to as AA (the most acidic), AB, AC, BB, BC, and CC (the most basic).

In 1978, ultracentrifuge studies carried out by a group from the University of Amsterdam[10] indicated one homogeneous band with a molecular weight of 144,000 — a molecular weight for the purified enzyme has been calculated as 166,000. The carbohydrate content of the enzyme was found to be at least 2.5%, including 1.3% mannose, 0.6% glucose, and 0.6% N-acetyl glucosamine.

Introduction:

The mature human segmented neutrophil contains at least two types of granules[11,12].

a) Azurophilic, formed during the promyelocyte stage, and which contain MPO[13];

b) Specific, formed during the myelocyte stage, and which do not contain MPO. The ratio of peroxidase positive azurophils to peroxidase negative specifics is 1:2. In each cell there are 75 azurophilic granules and 150 specific granules.

Peroxidase positive granules vary in shape — most are spherical (about 500 mμ), but ellipsoid forms containing crystals are also present. In addition, MPO can be seen throughout all the secretory apparatus; i.e. in the Rough Endoplasmic reticulum, in all the Golgi cisternae, and in all forming granules.

MPO, in the presence of hydrogen peroxide (H_2O_2) and a halide, such as bromide, iodide, or chloride, constitutes a potent bactericidal system which is also effective against fungi, viruses, mycoplasma, and mammalian tumour cells[2]. However, despite the presence of large quantities of MPO in normal neutrophils (approximately 5% of dry weight), the relative importance of this killing mechanism is unknown.

In general, a good correspondence exists between the inability of a patient's neutrophils to kill small numbers of bacteria in vitro, and an increased susceptibility to bacterial infection. This has been confirmed in individuals (a) whose neutrophils had abnormalities of specific granules; or (b) a markedly decreased oxidative mechanism. However in contrast to the leucocytes in patients with Chronic Granulomatous Disease, patients with MPO deficient leucocytes are not defective in oxidative mechanism[14].Such patients have usually been free of bacterial infections in spite of the low ratio bactericidal defect of their neutrophils[15,16].

Further evaluation of subjects with MPO deficiency may shed some light on the physiologic importance of this enzyme. In the past, such studies were difficult because of the small number of subjects detected. Now, using an automated cytochemical analyzer for routine haematology work, such as the Technicon Hemalog-D[1], the chances of detecting MPO deficiencies are very much increased.

The Hemalog-D detects different leucocytes by their size and staining characteristics. Neutrophils and eosinophils are stained for MPO activity using 4-chloro-1-naphthol as the electron donor. MPO deficiency is suspected or indicated when the Hemalog-D reports a low neutrophil count and increased numbers of Large Unstained Cells (LUC). There may also be an apparent slight increase in the High Peroxidase activity (HPX), due to the neutrophil cloud moving towards the low absorbance threshold, and leaving normal neutrophils to be included in the HPX percentage. Polaroid photographs of the oscilloscope pattern of the MPO channel show the position of the neutrophil cloud to be shifted towards the low absorbance threshold.

In 1979, a group from the U.S.A.[12] published an article which reported on their performing of approximately 18,000 routine blood

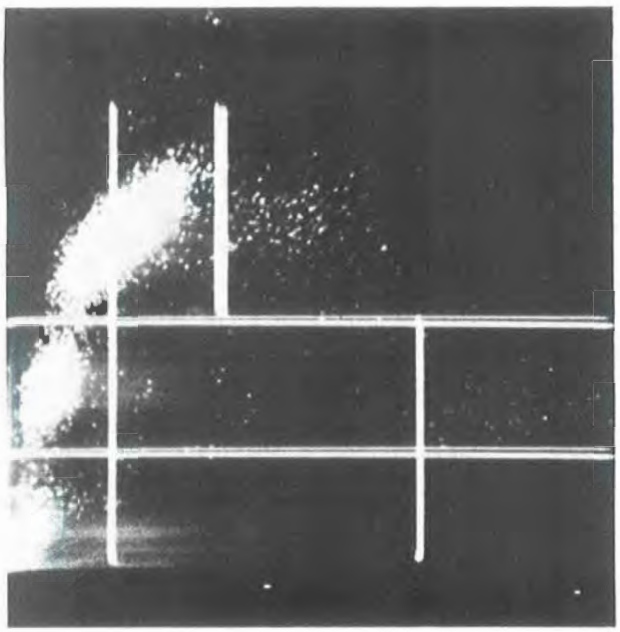


Figure 2. Hemalog-D cell pattern showing a patient with moderately reduced MPO activity — the neutrophil cloud has shifted considerately to the left.

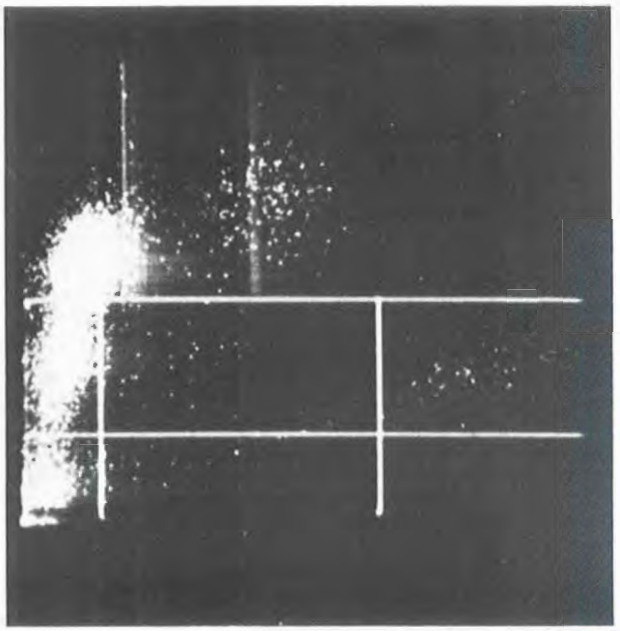


Figure 3. Hemalog-D cell pattern showing a patient with markedly reduced MPO activity — the neutrophil cloud is almost completely within the LUC compartment. Note a few normal neutrophils, which would appear as having HPX activity, due to the marked shifting of the threshold to the left.

samples over a 40 month period. They detected eight persons with partial MPO deficiency, and one with complete deficiency. Subsequently, 120 relatives of these nine subjects were investigated, and a further 23 partially deficient subjects were identified. Numerous electron micrographs of their neutrophils showed that all cells examined had decreased numbers of MPO positive granules. Electron micrographs from the subject with complete MPO deficiency showed no MPO positive granules. Normal numbers of MPO granules were found in the eosinophils from all subjects.

Eleven representative MPO deficient subjects were followed prospectively. Only two have had serious infections. One subject developed three episodes of streptococcal cellulitis, all of which responded to penicillin therapy. This patient also had longstanding lymphoedema secondary to treatment for testicular cancer, which may have predisposed him to these infections. A second subject was hospitalized with aseptic meningitis, but made a complete recovery.

Another survey appeared in 1979[3,4], where again a 40 month period was surveyed, covering 45,000 patients with Hemalog-D counts. Ten partial and ten completely MPO deficient patients were found. Patients were confirmed as deficient by repeated counts and histochemical stains for MPO. Eosinophils stained normally for peroxidase. Functional studies of neutrophils from two partial and two completely deficient MPO patients showed what appears to be fairly typical findings — impaired protein iodination, normal to increased H_2O_2 production, impaired killing of *Candida albicans*, and delayed killing of *Staphylococcus aureus*.

Methods:

In 1982, we decided to do our own survey to check on the frequency of such patients. Over a ten month period we processed approximately 100,000 blood samples through a Hemalog-D, and detected 44 patients with cytochemical indications of MPO deficiency, based on the criteria outlined earlier using the Hemalog-D oscilloscope and printout results.

Results:

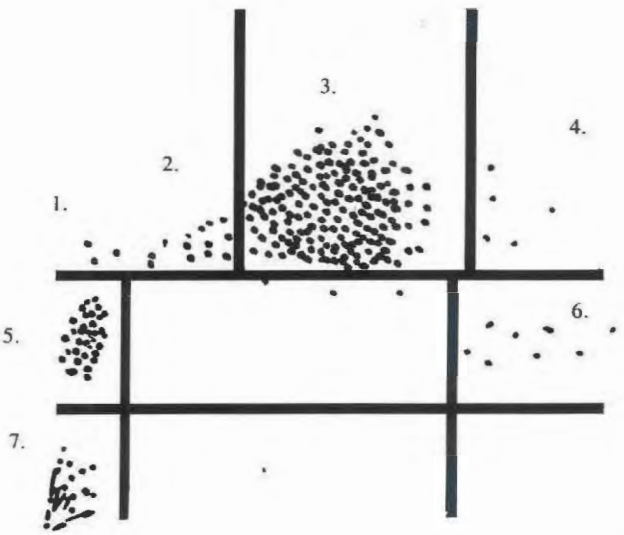
Of the 44 patients thus discovered, there were surprisingly few other haematological abnormalities. Two patients had a low haemoglobin concentration (70 and 94 g/l). A third patient had total white cell counts, on different occasions, of 10.8 and 11.2 x 10^9/l.

The average age for males was 36.3 years, median 23/24 years. The average age for females was 51.2 years, median 45 years. The ratio of males:females was 2:3.

One patient presented with a concurrent urinary infection due to *E. coli*. Three patients, all female, had been diagnosed as having arthritis; another female had polymyalgia. Three females were pregnant. One patient had an elevated glucose, which may be significant, as there have been instances reported of patients with MPO deficiency and diabetes showing susceptibility to infections, especially those due to *C. albicans*, with increased resistance to antifungal drugs[17].

Discussion:

It does not appear possible to define any diagnostic criteria from the ranges of Hemalog-D abnormalities, such as neutrophil percentage, LUC percentage, HPX, or Remainder — there are wide variables to consider, such as calibration of the instrument; apparent slight variation, especially of LUC, on repeating the same sample; and differences on



1. Large unstained cells
2. Monocytes, basophils, LPX neutrophils
3. Normal neutrophils
4. HPX neutrophils
5. Lymphocytes
6. Eosinophils
7. Red cell stroma, electrical noise.

Figure 4. Explanation of cell positions on the Hemalog-D oscilloscope.

separate occasions in percentages in the same individual. Polaroid photographs of the MPO channel on the Hemalog-D have enabled us to suggest a distinction between heterozygous and homozygous MPO deficient subjects.

This report, and those others mentioned, show that MPO deficiency is not a rare disorder. The frequency of around 1 in 2000 that we detected is similar to previous findings.

The molecular abnormality present in MPO deficient individuals has not been well defined. Partial MPO deficiency shows variable expression, which may be accounted for by the variable inheritance of MPO isoenzymes. It is not yet certain whether MPO deficiency represents the absence of normal enzyme, or the presence of an altered enzyme with no functional ability. Different families may have different defects.

Prospective observations of MPO deficient subjects have shown only rare infections, adding support to the concept of multiple neutrophil killing mechanisms.

Three of the University of Utah's original nine subjects have had cancer; two had lymphoma and one had testicular cancer. It would be worthwhile to observe other MPO deficient subjects for a long period of time to see whether or not they develop malignancy, since the MPO-H_2O_2-halide system has been shown to have in vitro tumouricidal activity.

Another group[18] investigated a patient who had 99.2% of his neutrophils deficient in MPO. This patient had an atypical myeloproliferative disorder, characterized by refractory megaloblastic anaemia, and C group trisomy. In composition and function, his peripheral neutrophils resembled those of patients with hereditary MPO deficiency. Because C group trisomy has been detected in other myeloproliferative disorders, it would be of interest to ascertain the peroxidase cytochemistry of leucocytes in such individuals.

Summary:

With expanded numbers of subjects and long term follow-up of these individuals, the importance of MPO may be more clearly defined.

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The Toxoplasma Indirect Haemagglutination Test: A Review

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Introduction:

The Toxoplasma Indirect Haemagglutination Test (IHAT) was originally designed by Jacobs and Lunde in 1957[1]. Along with the Complement Fixation Test (CFT), Indirect Fluorescent Antibody Test (IFAT) and Direct Agglutination Test, the IHAT was designed to replace the Dye Test (DT). The disadvantages of the DT include the safety problem of using live zoites, the use of accessory factor and the heavy demands on time[2]. The IHAT is simple, cheap and quick. It does however have two drawbacks. Sensitised red blood cells have been given a reputation for being unstable and non-specific agglutinations may occur[3].

Key Words:

Haemagglutination; *Toxoplasma gondii;* Serological test; Red Blood Cells; Tanning; Sensitisation.

Method

Haemagglutination red blood cells are prepared as follows:

Red blood cells
↓
Fixation (optional)
↓
Tanning
↓
Sensitisation with antigen
↓
Storage

Choice of Red Blood Cells

Two types of red blood cells, sheep and human, are commonly used in

the Toxo-IHAT. Of these, sheep cells seem to be preferred[1,2,3,4,5,6]. Cells are usually stored in solutions of alsevers, citric acid-dextrose or sodium citrate. Cells are quantified by determining packed cell volume, although Jacobs and Lunde[1] used a nephelometer and strongly supported more standardisation of red blood cell solutions. Chordi, Walls and Kagan[3] were concerned with non-specific agglutination of sheep red blood cells; they found that 87% of DT negative but IHAT positive sera had antibodies directed against the sheep cells. However they also found that IHAT titres of >1:200 were specific. Because of this problem some authors pre-absorb their test sera with normal sheep cells prior to titration[1].

Alternatively human group O, Rh -ve cells which do not usually agglutinate with human sera after fixation, have been used[7,8]. Maloney and Kaufman[7], suggested however that any expired Blood Bank cells can be used. A non-sensitised red blood cell control is therefore not required when using human cells, but was used by Thorburn and Williams[2]. They found that positive Paul-Bunnell (glandular fever) sera were easily identified by their agglutination of pyruvic aldehyde — treated non-sensitised sheep cells. There is also a commercially available Toxoplasma IHA kit which uses turkey cells as the antigen carrier therefore negating the need for prior absorption.

Fixation

Fixation adds considerably to the long-term stability of sensitised cells. Formalin has been used both before[4,6,7] and after[8] sensitisation. Park[6] used an alcohol-formalin mixture for 4 days, replacing with fresh fixative each day. The cells were then washed twice a day for 6 days and sodium bisulphite was added to complex the formaldehyde. The formaldehyde-bisulphite complexes were removed by dialysis for 4 days. The total process took 18 days. Formaldehyde was used by Maloney and Kaufman[7] who stressed that fixation should take place slowly and the cells should be constantly agitated. Jennis[5] and later Thorburn and Williams[2] used pyruvic aldehyde to fix their cells. Jennis[5] originally found that this fixative alone rendered red blood cells capable of adsorbing toxoplasma antigen negating the need for tanning.

Tanning:

Tannic acid is used to render red blood cells capable of adsorbing antigenic material particularly proteins. The majority of authors use tannic acid at a 1:20,000 or 1:40,000 dilution. The time required for tanning varies with different temperature and pH conditions. Temperatures cited in the literature include 4°C[3,9] and 37°C[1,6,7,8]. Tanned cells can be successfully stored, and sensitised with antigen just prior to use. Jennis[5], claimed pyruvic aldehyde treated cells could be stored in 10% v/v glycerol at -15°C for up to 6 months. Maloney and Kaufman[7] also stored their cells at -15°C and Park[6] stored his tanned cells at 4°C for up to 6 months.

Sensitisation and Preparation of Antigen

Antigens for sensitising tanned red blood cells are prepared from *Toxoplasma gondii* zoites which can be cultured in the peritoneal cavities of mice or rats. Peritoneal exudates from infected animals contain a soup of host cells and *T. gondii* zoites. None of the studies reviewed contained any process for removing contaminating host cells. Chordi, Walls and Kagan[3], and Balfour, Bridges and Harford[10] found that some DT negative but IHAT positive sera occur because of the presence of antibodies specific to mouse antigens which have been adsorbed to the red blood cells. Rarely do these reactions exceed a titre of 1:50 and would therefore be of no consequence if a minimum titre of 1:64 was employed. Freeze-thawing to rupture zoites, as a method of antigen preparation, has been used[4,8] but Petterson[11], claimed that this technique was destructive to the antigen. Osmotic rupture is a more popular method for antigen extraction. Zoites are suspended in distilled water which is later made isotonic with an equal volume of double-strength saline[3,6].

Standardisation to yield the correct concentration of antigen is important since antigen preparations of varying concentration give unwanted variability between batches. Jacobs and Lunde[1], quantified their antigen by weight, whereas Fulton and Fulton[12] used a nephelometer. None of the studies reviewed used a Neubauer haemocytometer (probably the simplest method) to count the zoites prior to rupture. In some cases a range of antigen dilutions were tested before each bulk lot of cells was sensitised[9].

Since the IFAT uses whole zoites, it primarily detects antibodies to cell wall determinants. Instead of using just the supernatant from lysed zoites to sensitise their cells Ambroise-Thomas, Simon and Bayard[4] used the whole organism, including fragments of fractured zoite cell walls.

Antigen preparations can be successfully stored at -15°C[1,5] or lyophilised after dialysing[4,12]. Sensitisation may be carried out at room temperature for 15 minutes[1,8] or 30 minutes[7]. A temperature of 37°C for 15 minutes had also been used[3,6] but 56°C for 1 hr was required by the method according to Jennis[5].

Diluent

The diluent for the actual titration of sera whether carried out in tubes or microtitre trays is important. Normal rabbit sera (NRS) that is DT negative is often added to phosphate buffered saline or normal saline to aid the settling of red blood cells. Such diluent, which contains only 1 or 2% v/v[1,6] NRS, gives sharper end points to titrations. Rabbit serum is better than normal human serum for this purpose according to Maloney and Kaufman[7].

Latex alternative to the IHAT

A latex equivalent of the IHAT has recently been introduced and is gaining increasing acceptance. The principle behind the indirect Latex agglutination test is identical to that of the IHAT, except a latex bead is used as the antigen-carrier instead of a red blood cell. Usually latex results can only be read after 24 hours, whereas IHAT reactions can be read after 2-3 hours. Recently, however, a method for obtaining latex results within 1 hour has been developed[13].

Conclusion:

This review gives some insight into the variation in methods employed to prepare toxoplasma haemagglutination cells. There are now methods available for the preparation of cells that remain stable for long periods of time and which do not give non-specific agglutinations. Karim and Ludlam[14] have found the IHAT along with the IFAT (IgG and IgM conjugates) to be the most useful tests for detecting actual cases of glandular toxoplasmosis. They found that the IHAT seroconversion usually follows the IFAT seroconversion by 2-3 weeks. Hughes[15] has found that in immunosuppressed rats the DT titres may be suppressed but the IHAT titres are not affected. Since toxoplasmosis may be reactivated in immunosuppressed patients the IHAT may be of more use than the DT, or the IFAT in the detection of these reactivated infections. The DT is similar to the IFAT in the type of anti-toxoplasma antibodies it detects[14]. In cases of ocular toxoplasmosis the IHAT appears just as sensitive as the IFAT in the detection of seroconversions[12].

The IHAT remains a useful, simple and cheap test for determining patients antibody status to toxoplasmosis.

Acknowledgements

I would like to thank Dr Diana Martin for proof reading this manuscript.

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The Laboratory Diagnosis of Hereditary Disorders of Platelet Function

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Department of Haematology Christchurch Hospital

Treatise presented as part of Fellowship Examination, 1983.

Summary:
This paper reviews some of the methods commonly used in the investigation of suspected platelet function defects. The findings in intra-cellular and platelet membrane abnormalities are discussed. Brief case studies illustrate the results obtained in Bernard Soulier syndrome, Storage Pool disease and Glanzmans Thrombasthenia.

Introduction:
One of the earliest descriptions of platelets appears in an English translation of Gerbers 'General and Minute Anatomy' published in 1842. Gulliver, in an appendix to this translation states that in addition to red cells, "blood contained white globules, nucleated cells and the organic germs of fibrin". He recognised that platelets were included in a fibrin clot but did not feel that they were actively involved in clot formation. By the mid 1870's several workers had shown the involvement of platelets in fibrin formation although the precise mechanism was not understood[1]. Another theory at this time was that platelets were a red cell precursor, capable of growing into a leucocyte before becoming a red cell. It was not until 1906 that Wright demonstrated the relationship of platelets to megakaryocytes, yet as recently as 1920 an article appeared in the literature suggesting that platelets were artifacts precipitated from plasma[1]. It is now known that platelets are required not only for the arrest of haemorrhage following injury but also for the maintenance of normal vascular integrity.

Platelets have the ability to activate certain coagulation factors of the intrinsic pathway and platelet membrane phospholipids are involved in coagulation factor interaction. Platelets also transport both coagulation factors and vasoactive substances either intracellularly or associated with the platelet membrane. At the site of vessel wall injury platelets are exposed to surfaces other than endothelium. Platelets adhere to collagen and release substances stored in the intracellular granules. Some of these substances have the ability to induce platelet aggregation with the subsequent formation of a platelet plug. Following activation, the platelet is involved in the formation of thrombin which converts fibrinogen to fibrin which in turn stabilizes the haemostatic plug. Clot retraction then occurs and is mediated by the platelet contractile protein actomyosin previously known as thrombosthenin.[2] (Fig 1)

The study of congenital platelet function defects has increased the understanding of the biochemistry involved in the reactions of adhesion, aggregation and release, and methods have been devised to measure these in vitro. Although many methods were devised when little was known about platelet structure and function, most are still useful as screening tests for abnormalities. In this review the emphasis will be placed on screening tests such as platelet aggregation, which should give sufficient information for the diagnosis of the majority of known platelet function defects. However in the future it will become necessary to undertake sophisticated biochemical measurements of platelet components to identify the precise nature of the abnormalities.

Causes of Disordered Platelet Function
Acquired disorders of platelet function occur in a wide range of conditions and are often seen in association with other haemostatic defects. The main conditions in which platelet dysfunction may play a significant role are listed in Table 1.

Inherited defects of platelet function may affect any of the steps leading haemostatic plug formation. These disorders may be classified according to the site of the genetically determined platelet abnormality. This classification is set out in Table 2.

This paper will concentrate on membrane and intracellular platelet abnormalities, the environmental disorders are outside the scope of this paper and the May Hegglin anomaly, a disorder consisting of thrombocytopenia and the presence of inclusion bodies in the neutrophil cytoplasm will also not be discussed.

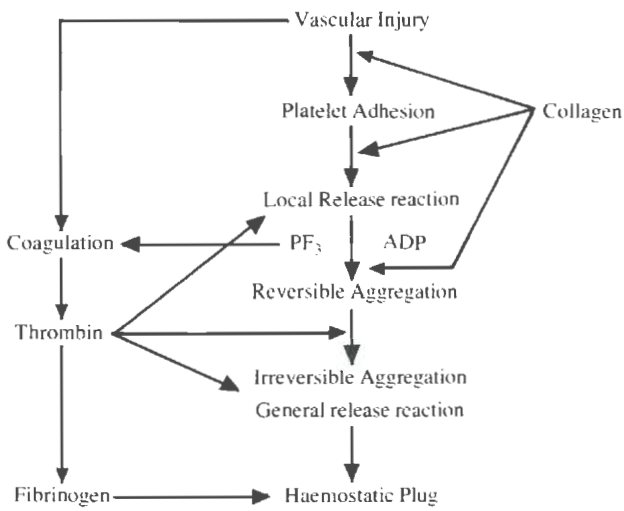

Fig 1
AN OUTLINE OF PLATELET ADHESION AGGREGATION

Clinical Manifestations

Patients with abnormally functioning platelets give a history similar to that seen in a mild coagulation factor deficiency or thrombocytopenia. Spontaneous bleeding may occur from mucosal surfaces especially epistaxis[4,5,6], menorrhagia[6,7], or from the gastro intestinal tract[4,8]. Superficial bruises and prolonged bleeding from small cuts may be noted[4,5,7,8,9,10]. Post traumatic bleeding particularly following tonsillectomy and tooth extraction is often severe[4,5,6,10,11].

The investigation of a patient with a possible platelet function defect should include a general medical history and examination with a meticulous drug history. Direct enquiries about aspirin injestion must be made. A family history should be noted, and investigation should include other family members where possible.

Laboratory Tests

CLOT RETRACTION

The phenomenon of clot retraction was described some sixty years before platelets were discovered. The involvement of platelets in clot retraction was described by Hayem in 1878[1]. This test is widely used as one of a range of coagulation screening tests. However the test is insensitive and tends to reflect platelet number rather than function.

PLATELET COUNT AND MORPHOLOGY

Accurate platelet counts are essential in the assessment of any patient with a haemorrhagic tendency. In addition a Romanowsky stained blood film should be examined with particular note taken of the size and staining characteristics of the platelets. Platelet ultrastructure and function has been studied by both transmission and scanning electron microscopy. These techniques may be used to visualise intracellular organelles as well as morphological changes during adhesion, aggregation and release. The diagnosis of some congenital platelet disorders can be assisted by demonstrating structural abnormalities.

Table 1
CAUSES OF ACQUIRED PLATELET DYSFUNCTION (2, 3)

Uraemia
Myeloproliferative Disorders
Leukaemia
Dysproteinaemia
Liver Disease
Immune or mechanical platelet damage
Drugs

THE TOURNIQUET TEST

This method is time consuming and uncomfortable for the patient. It is more sensitive to platelet number than function and as there have been few attempts to standardise methodology and interpretation, results are difficult to interpret and it is not recommended[12].

THE BLEEDING TIME

The bleeding time is a good test for assessing primary haemostasis but is rather invasive. This test is almost entirely dependant on platelet number and function.

The method most commonly used is that of Ivy[12] or a modification of this method by Mielke. This modification involves the use of a template incision rather than a puncture, thus standardising the wound[12]. A disposable sterile bleeding time device has become commercially available. This method replaces the template and has the advantage of preventing the transmission of Hepatitis. A disadvantage of template methods is that faint scars may remain.

Table 2
CLASSIFICATION OF HEREDITARY PLATELET DISORDERS (2, 3)

Disorders of platelet environment
- Von Willebrands disease
- Afibrinogenaemia
- Ehlers-Danlos syndrome

Membrane Abnormalities
- Bernard Soulier syndrome
- Glanzmans Thrombasthenia

Intracellular abnormalities
- Storage Pool disease
- Release defect
  - — Cyclo-oxygenase deficiency
  - — Thromboxane synthetase deficiency
- α Granule deficiency

Miscellaneous
- May Hegglin anomaly

GLASS BEAD RETENTION

This method involves the passage of blood through a column of glass beads and the calculation of the percentage of platelets which are retained in the column. This test is not a pure platelet function test but is influenced by the haematocrit and Factor VIII Von Willebrand's Factor as well.

There have been a number of methods described but standardisation is difficult as may be seen by the lack of commercial alternatives[13].

PLATELET COAGULANT ACTIVITY

The tests most commonly used to measure platelet coagulant activity are the prothrombin consumption test[14] and the assay of platelet factor 3[15]. Historically these tests preceeded those of platelet aggregation and many patients previously described as having defects of platelet coagulant activity have subsequently been shown to also have underlying defects of platelet aggregation and release[16]. However one patient has been described where the only abnormality was a deficiency of platelet factor 3[11].

The above tests represent the earliest attempts of measuring platelet function. Some remain as essential screening tests especially the bleeding time, platelet count and stained blood film, while others, such as the tourniquet test and clot retraction have been largely replaced by more sensitive and specific tests.

PLATELET AGGREGATION

The phenomenon of platelet aggregation has been studied more than any other platelet activity. Many agents will cause platelets to aggregate in vitro but the most commonly used agents are Adenosine diphosphate (ADP), adrenaline, collagen, thrombin, arachidonic acid (AA) and ristocetin. These substances, with the exception of ristocetin, are all potentially present within the circulation or vessel wall, therefore the platelet response to these agents in vivo will hopefully be reflected by their response in vitro. Ristocetin may mimic some physiological endothelial substance.

Platelet aggregation is generally studied in platelet rich plasma prepared from blood anticoagulated with trisodium citrate and centrifuged to remove the red cells. The platelet rich plasma is removed from the red cells and placed in a plastic tube which is sealed and allowed to stand at room temperature for 30 minutes. Testing should be completed within two hours of blood collection.

Platelet aggregation is usually measured by following changes in optical density. A known volume of aggregating agent is added to platelet rich plasma which is contained in a cuvette in a light recording machine, under conditions of constant temperature and with continuous aggregation. The resulting changes in optical density are usually measured graphically. Typical aggregation curves are shown in Fig 2.

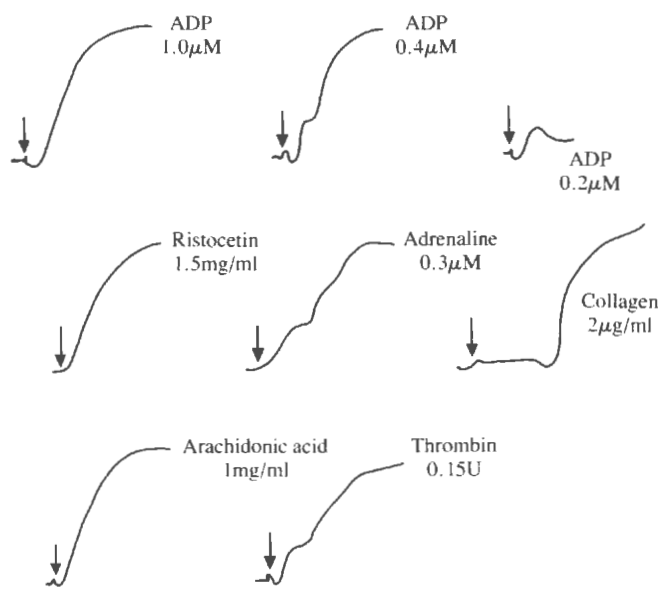

Fig 2
TYPICAL NORMAL PLATELET AGGREGATION CURVES

With most of the primary aggregating agents, such as ADP, thrombin and collagen, aggregation (Fig 2c) is preceded by a transient decrease in optical density (Fig 2b) occurring simultaneously with a reduction in the extent of the oscillations of the baseline. (Fig 2a) The oscillations are due to the alignment of the flat cells as they rotate and the disappearance indicates a shape change into a sphere.

The aggregation induced by low concentrations of ADP (0.2μM) is reversible. At intermediate concentrations (0.4μM) an initial primary wave aggregation is seen followed by an irreversible second wave aggregation induced by ADP released from the dense bodies of the platelets. At high concentration of ADP (1.0μM) it is not possible to distinguish the primary and secondary wave. The aggregation is irreversible.

When aggregation is induced by collagen, shape change occurs following a delay as platelets adhere to the collagen fibres. Aggregation is due to the release of ADP and is therefore similar to the secondary wave aggregation seen with the higher concentration of ADP.

Adrenaline has a direct effect on platelets and induces a primary aggregation response, which is not preceded by shape change. ADP is released from the platelets and secondary aggregation occurs. Thrombin may induce mono or biphasic aggregation which is preceded by shape change[2].

AA invariably induces irreversible aggregation. Although the reaction is not dependant on the release of ADP, release does in fact occur[17]. The platelet aggregation response with labile aggregation stimulating substance (LASS) which consists mainly of thromboxane A_2, may be determined by adding sodium arachidonate to normal platelet rich plasma and incubating this mixture at 37°C for 30 seconds. After this time a subsample is removed and is added to the platelets to be tested in the usual manner[4]. The ability of platelets to produce thromboxane A_2 may therefore be measured in the opposite manner. The metabolism of AA is shown in Fig 3. Ristocetin induced aggregation is concentration dependant (1-2mg/ml) and requires the presence of a plasma cofactor, called Von Willebrands Factor, and a binding site on the platelet membrane. In this instance the platelet appears to be acting passively and agglutination may be a more appropriate term in this situation as aggregation implies the more active participation of the platelet[18].

RELEASE OF ^{14}C SEROTONIN

The platelet release reaction may be measured by ^{14}C serotonin release[19]. Platelets normally contain serotonin derived from the plasma and this is released from the dense bodies at the same time as ADP[20]. ^{14}C serotonin may be incorporated into the platelet during incubation and the amount released following incubation with an aggregating agent such as collagen or AA is measured.

THROMBOXANE SYNTHESIS

A screening test for thromboxane synthesis is the aggregation by LASS as described above. Thromboxane synthesis may be assayed by radioimmunoassay. The levels of Thromboxane B_2 are usually measured as this substance is more stable than Thromboxane A_2 which is the active agent but has a half life of 30 seconds[19].

β THROMBOGLOBULIN ASSAY

β thromboglobulin (BTG) is a platelet protein similar to platelet factor 4 and released from α granules in the release reaction. Plasma and platelet levels of BTG may be measured by radioimmunoassay[20]. The test is of most use as a measure of in vivo platelet consumption.

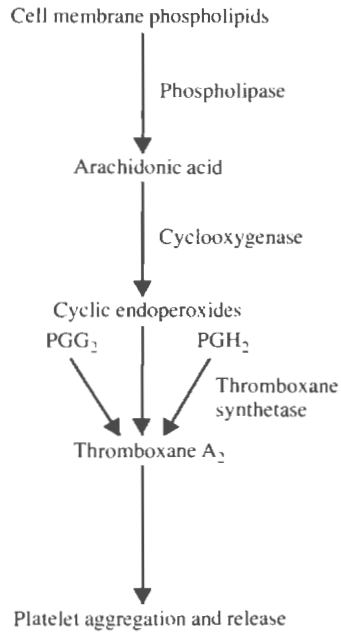

Fig 3
THE METABOLISM OF ARACHIDONIC ACID IN PLATELETS

Membrane Abnormalities

THE BERNARD SOULIER SYNDROME

This disorder is inherited as an autosomal recessive trait. Laboratory abnormalities may be demonstrated in clinically unaffected heterozygotes[7]. The disorder was first described by Bernard and Soulier in 1948 and was characterised by a prolonged bleeding time, normal numbers of platelets with giant forms and defective prothrombin consumption. Subsequent reports have shown mild thrombocytopenia to be an inconsistent feature of the disorder[5,6,7]. Platelet aggregation studies using ADP, Adrenaline, Collagen and Thrombin are normal[5,7]. Bithell and co-workers have shown that Bernard Soulier platelets fail to aggregate with bovine fibrinogen and this was subsequently shown to be a nonreactivity with bovine factor VIII. Bernard Soulier platelets also failed to react normally with the antibiotic Ristocetin[5]. (Table 3)

In contrast to Von Willebrands disease this non reactivity is not corrected by normal plasma[5]. It now has been shown that the disorder is due to an abnormal reaction with subendothelium[6]. Bernard Soulier platelets lack a receptor for Von Willebrands factor which is a necessary co-factor for the interaction of platelets with both ristocetin and subendothelium[21]. The membrane disorder is a deficiency of glycoprotein I complex. Bernard Soulier platelets also contain an increased concentration of dense bodies and α granules[3].

GLANZMANS THROMBASTHENIA

This disorder was first described by Glanzman in 1918 and was characterised by defective clot retraction[3]. The term thrombasthenia is now applied to an autosomal recessive bleeding tendency characterised by a prolonged bleeding time and normal platelet count but where there

Table 3
DIAGNOSIS OF HEREDITARY PLATELET DISORDERS
(3 Modified)

	Platelet Count	Platelet Size	Clot Retraction	Bleeding Time	Platelet ADP	Aggregation Collagen	AA	Ristocetin	^{14}C Serotonin Release
Bernard Soulier Syndrome	D	I	N	I	N	N	N	A	N
Glanzmans Thrombasthenia	N	N	A	I	A	A	A	N	N
Storage Pool Disease	N	N or D	N	I	I°	D	N	N	A
Release Defects									
Cyclo-oxygenase	N	N	N	I	I°	D	D	N	A
Thromboxane synthetase	N	N	N	I	I°	D	D	N	A
Grey Platelet Syndrome	D	I	N	N	D	N	N	N	D or N

D = Decreased N = Normal I° = primary phase
I = Increased A = Absent aggregation only

is failure of platelets to aggregate with ADP, adrenaline, thrombin, collagen and AA. Although the platelets fail to aggregate, their initial reaction to aggregating agents is not impaired and shape change does occur. The platelets also fail to aggregate with LASS but are capable of producing it. (Table 3) It has been shown that Glanzman platelets lack glycoproteins IIb and IIIa on the platelet membrane[22]. The glycoprotein IIb-IIIa complex is thought to be the receptor for fibrinogen. Fibrinogen is known to be essential for ADP aggregation[23]. A varient of Glanzmans disease (Type II) has been described where the disorder is less severe and clot retraction may be nearly normal[3].

Intracellular Abnormalities

STORAGE POOL DISEASE

Storage Pool deficiency occurs in a number of different bleeding disorders; Hermansky Pudlack syndrome, Chediak Higashi syndrome, Thrombocytopenia with absent radii, Wiscott Aldrich syndrome or as a separate entity involving only the platelets. It is to this platelet condition that the term storage pool disease applies[3]. This autosomal dominant disorder is characterised by a prolonged bleeding time and platelets which are normal in numbers and appearance in a stained film.

The platelets have normal primary phase aggregation with ADP, collagen and adrenaline. There is no secondary aggregation, however they usually react normally with AA[3]. ^{14}C release after challenge with collagen, AA or ADP is reduced. (Table 3) Storage Pool platelets contain a decreased amount of intracellular ADP and Adenosine triphosphate (ATP).

PLATELET RELEASE DEFECT

The most common cause of a failure of platelet release in the presence of a normal storage pool is the injestion of aspirin. A number of patients have been reported where these findings appear to be a naturally occurring haemostatic abnormality[4,8,9,10]. Although most of the patients described have a life long history of bleeding there is a lack of family studies and the mode of inheritance of this disorder has not been established.

Defects of platelet release may be distinguished from storage pool disease by the failure of platelets to aggregate with AA[24]. Abnormalities of platelet release may be divided into two disorders — a deficiency of cyclo-oxygenase or thromboxane synthetase. Patients with these two disorders have a prolonged bleeding time. Platelet aggregation with ADP 1-10μM is limited to primary phase aggregation only, which is always reversible. Aggregation with collagen, adrenaline and AA is markedly reduced while ristocetin aggregation is normal[4,9,10]. ^{14}C serotonin release is markedly reduced[9].

The platelet aggregation abnormalities of Cyclo-oxygenase deficiency and thromboxane synthetase deficiency are mutually corrective in platelet mixtures but only thromboxane synthetase deficient platelets will correct the abnormalities of normal aspirin treated platelets[10]. (Table 3)

α GRANULE DEFICIENCY

This autosomal dominant disorder is characterised by a long bleeding time, and a normal or reduced number of poorly staining platelets[25]. The disorder was named 'Grey Platelet Syndrome' because of the peculiar grey colour in a Romanowsky stained blood film. Platelet aggregation with ADP and Collagen is reduced but is normal with AA and ristocetin. ^{14}C serotonin release is abnormal although there is normal uptake[20]. (Table 3) Platelet BTG levels are decreased but plasma levels are normal suggesting that BTG is synthesized normally but because granules which normally store this protein are lacking, BTG is released in the marrow[20].

Conclusion

Advances in the understanding of how platelets function and the development of methods to study it are disclosing increasing numbers of patients whose bleeding disorders are the result of various abnormalities of platelet function. The following section includes brief case studies of three patients investigated recently in the authors laboratory.

The patient with Bernard Soulier syndrome caused no problems in diagnosis. This disorder is a good example of an intrinsic abnormality of platelet adhesion.

Storage Pool Disease comprises a heterogeneous group of patients. It occurs in a number of different hereditary bleeding disorders and may be a feature of several acquired disorders. This patient demonstrates the difficulty of distinguishing between these two groups of patients.

Glanzmans Thrombasthenia has the most pronounced defect of platelet aggregation but may be difficult to distinguish from the release defects particularly when the clot retraction is normal.

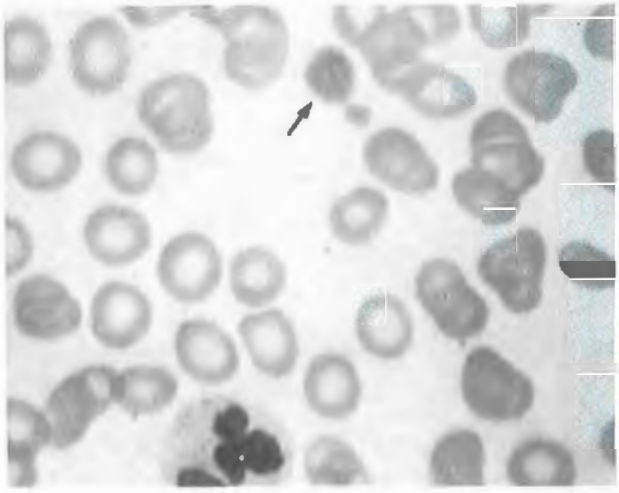

Fig 4

Blood film from patient with Bernard Soulier syndrome. Note large platelets (arrow).

Appendix

CASE STUDY: BERNARD SOULIER SYNDROME

A 22 year old female presented with a life long history of easy bruising and excessive bleeding. A blood screen showed a low haemoglobin, MCV and platelet count. Serum iron was reduced. The blood film (Fig 4) contained hypochromic cells and morphologically abnormal giant platelets. Electron microscopy (Fig 5) showed these large platelets contained an increased number of dense bodies and α granules. Coagulation screening tests and Factor VIII Coagulant (F VIII C), Factor VIII Related antigen (VIII RAG), and Factor VIII Ristocetin Cofactor (VIII RCOF), were within the normal range.

The bleeding time was prolonged. Platelet aggregation with ADP, Collagen and Adrenaline was normal. There was no aggregation with ristocetin or bovine serum. Normal plasma failed to correct the ristocetin aggregation.

A diagnosis of Bernard Soulier syndrome was made. It is reported that Bernard Soulier platelets fail to aggregate with bovine factor VIII, which does aggregate normal platelets. The findings in this patient would suggest that it is in fact bovine FVIIIRAG, rather than FVIIIC, which causes aggregation. Other members of this patients family were not available for testing.

CASE STUDY: STORAGE POOL DISEASE

A 29 year old female presented with a 6 month history of menorrhagia and increasing bruising. Her haemoglobin, white count, platelet count and coagulation tests were normal. The bleeding time was prolonged and the platelets showed a decreased response to ADP. There was no aggregation with collagen or adrenaline. The responses with ristocetin and AA were normal. Prothrombin consumption, platelet factor 3 and ^{14}C serotonin release were all abnormal.

A diagnosis of storage pool disease was made. Although bleeding problems were only present for 6 months prior to presentation, 15 months of follow up investigation have revealed no obvious reason for an acquired defect. Unfortunately other family members are not available for testing.

CASE STUDY; GLANZMANS THROMBASTHENIA

This 18 year old male has a life long history of severe epistaxis requiring transfusion and severe bleeding following tooth extraction. Coagulation investigation performed in 1966 showed that he had a long bleeding time, decreased aggregation with ADP and collagen and reduced platelet retention in a glass bead column.

Recent further investigation has shown that aggregation with ADP (1.0μM) produced reduced reversible primary phase aggregation. ADP at 0.4μM and 0.2μM produced platelet shape change but absent aggregation. Aggregation with collagen, adrenaline and AA were all absent although ristocetin aggregation was normal. His platelets were not aggregated by LASS produced by normal platelets and although his platelets did not aggregate with AA they produced LASS which aggregated normal platelets. ^{14}C serotonin release was markedly reduced. Clot retraction was normal. Originally it was suspected that the platelet abnormality in this case was a release defect, however release defect platelets fail to produce LASS but are aggregated by LASS

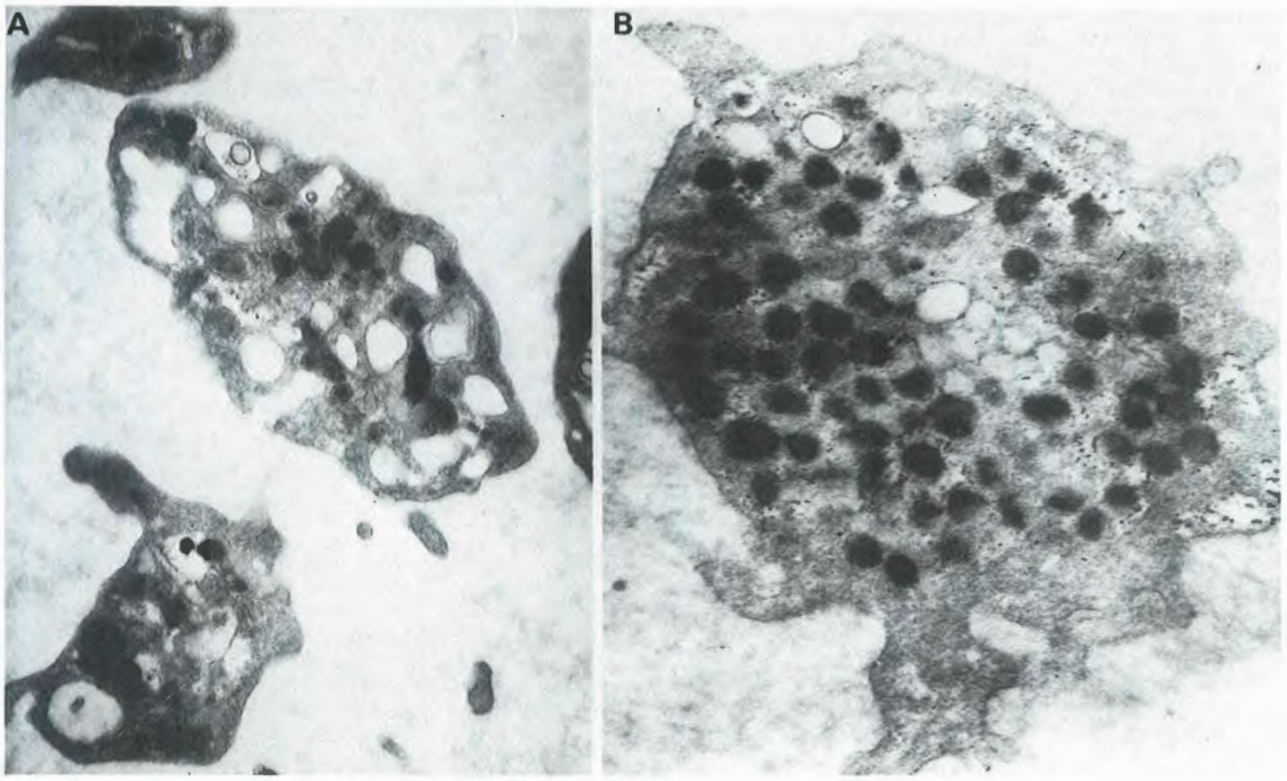

Fig 5

A. Normal platelets (x 20,000)
B. Platelet from patient with Bernard Soulier syndrome (x 20,000) Note the large size and increased content of dense bodies and α granules.

produced from normal platelets.

A diagnosis of Glanzmans Thrombasthenia Type II was made. The patients mother has normal platelet aggregation. There is no family history of easy bruising or bleeding.

Acknowledgements

Special thanks to Drs D. C. Heaton and M. E. J. Beard for permission to report their patients and for helpful criticism and advice on this manuscript. Grateful thanks is also due to Miss Lyndsay McNiven for secretarial assistance.

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CONTINUING EDUCATION

Revision Series in Biochemical Calculations Section I: Units

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"In this issue starts a Revision Series in Biochemical Calculations. There is planned a minimum of seven sections, which will appear in coming issues. Topics covered include Units, Molecular Weights, Titrations, pH Calculations, Spectrophotometry, Use of Formulae and Radioactivity. The series is designed to promote a basic level of calculation skills for all laboratory workers, whether it be a newly recruited laboratory assistant, registrar, science graduate or qualified technologist. No attempt is made to explain the underlying theory in the physiochemical principles involved but adequate references will be provided for the interested worker."

The use in medicine of the Système International d'Unités (SI) was endorsed by the Thirtieth World Health Assembly in May 1977. SI units are an extension and refinement of the traditional metric system. It embodies features which make it logically superior to any other system as well as practically more convenient: it is rational, coherent and comprehensive.

Base units

There are seven base units

Quantity	Name	Symbol
length	metre	m
mass	kilogram	kg
time	second	s
electric current	ampere	A
temperature	kelvin	K
luminous intensity	candela	cd
amount of substance	mole	mol

The base units are very precisely and uniquely defined. All physical measurements made in the laboratory should be traceable back to their base units.

Derived units

By multiplying a base unit by itself or combining two or more base units by simple multiplication or division it is possible to form a large group of units known as SI derived units.

Example of derived units

Quantity	Name	Symbol
area	square metre	m^2
volume	cubic metre	m^3
speed	metre per second	$m.s^{-1}$
acceleration	metre per second squared	$m.s^{-2}$
concentration	mole per cubic metre	$mol.m^{-3}$

The combination of base units to form derived units illustrates one of the main advantages of SI. The formation of the derived unit does not involve any conversion factor other than 1. Such a system of units is said to be coherent. A number of SI derived units have been given special names, most of which are named after scientists who made outstanding contributions to their field of study.

Examples of SI derived units with special names

Quantity	Name	Symbol	Derivation of unit
frequency	hertz	Hz	s^{-1}
force	newton	N	$m.kg.s^{-2}$
pressure	pascal	Pa	$N.m^{-2}$
energy	joule	J	N.m
power	watt	W	$J.s^{-1}$
radioactivity	becquerel	Bq	s^{-1}
temperature	degree Celsius	°C	K
catalytic activity	katal*	kat	$mol.s^{-1}$

*Not formally adopted for use in the medical sciences.

SI prefixes

For many purposes the SI base units and SI derived units are too large or too small for practical use. To overcome this difficulty the SI uses a series of SI prefixes.

Factor	Prefix	Symbol	Factor	Prefix	Symbol
10^3	kilo	k	10^{-3}	milli	m
10^6	mega	M	10^{-6}	micro	μ
10^9	giga	G	10^{-9}	nano	n
10^{12}	tera	T	10^{-12}	pico	p
10^{15}	peta	P	10^{-15}	femto	f
10^{18}	exa	E	10^{-18}	atto	a

Prefixes with restricted use

Factor	Prefix	Symbol	Factor	Prefix	Symbol
10^1	deka	da	10^{-1}	deci	d
10^2	hecto	h	10^{-2}	centi	c

Examples of the use of prefixes

1000 ng	= 1 μg	1000 nmol/l	= 1 μmol/l
1000 μg	= 1 mg	1000 μmol/l	= 1 mmol/l
1000 mg	= 1 g	1000 mmol/l	= 1 mol/l
1000 g	= 1 kg		

Non-SI units

Examples of units presently allowed in conjunction with SI units

Quantity	Unit	Symbol	SI definition
time	minute	min	60 s
	hour	h	3 600 s
	day	d	86 400 s
volume	litre	l	10^{-3} m^3 = dm^3
mass	tonne	t	10^3 kg = Mg
radioactivity	curie	Ci	37 x 10^9 s^{-1}

Catalytic Activity

The present unit of catalytic activity is the "international" unit (U) and is defined as the amount of enzyme which will catalyse the transformation of 1 μmole of the substrate per minute under defined conditions. The activity is expressed per litre of sample i.e. U/l. The proposed SI unit of catalytic activity is mole per second ($mol.s^{-1}$) and is called the katal (kat). The katal has not yet been formally adopted as an SI unit in the medical sciences.

Conversion of "international" unit to katal

1 U = 1 μmol/min
= 1000 nmol/60 s
= 16.67 nmol/s
= 16.67 nkat

Therefore 1 U/l of catalytic activity equals 16.67 nkat/l.

Radioactivity

Two systems of units are in current use for the measurement of radioactivity. The SI unit of activity is the bequerel (Bq) which is equal to 1 nuclear transformation per second. Still widely used is the curie (Ci).

1 Ci equals 37×10^9 Bq

Pressure

The partial pressure of blood gases pO_2 and pCO_2 are presently widely reported in millimetres of mercury (mmHg). The changeover to reporting blood gas results in kilopascals (kPa) has not yet been implemented in New Zealand except for one city. However all new blood gas analysers purchased should have the facility of reporting in either mmHg or kPa.

Conversion of mmHg to kPa

Pressure = Force/Unit Area
= Mass x Acceleration/Unit Area

The volume of a column of mercury 1 mm high by 1 m^2
= $0.001 \times 1 \times 1\ m^3$
= 0.001×10^3 l

However 1 litre of mercury weighs 13.5951 kg

The weight of a column of mercury 1 mm high by 1 m^2
= $0.001 \times 13.5951 \times 10^3$ kg

The pressure exerted by a column of mercury 1 mm high on 1 m^2 (where acceleration due to gravity = 9.80665 $m.s^{-2}$)
= $0.001 \times 13.5951 \times 9.80665 \times 10^3\ kg.m.s^{-2}.m^{-2}$
= $133.3\ kg.m.s^{-2}.m^{-2}$
= $133.3\ N.m^{-2}$
= 133.3 Pa
= 0.1333 kPa

Therefore 1mmHg = 0.133 kPa

Hydrogen ion concentration

Whilst the pH scale is not inconsistent with the SI, it is based on a logarithmic relationship (hydrogen ion concentration (mol/l)= 10^{-pH}. The use of hydrogen ion concentration is preferred by some workers. However at this stage the matter is still under study by international bodies and no definitive recommendation has been made. The unit of hydrogen ion concentration at physiological pH is nmol/l. For example the hydrogen ion concentration at pH 7
= 10^{-7} mol/l
= 100 nmol/l

All new blood gas analysers should be capable of reporting the hydrogen ion concentration in nmol/l independant of the units for pO_2 and pCO_2.

Important rules on the use of SI units

1 A space should be left between the number and the unit symbol. e.g. 5 mg not 5mg
2 A prefix when combined with the unit name is written as one word e.g. millimetre, microgram
3 The symbol for a unit is unaltered in the plural and should not be followed by a full stop except at the end of a sentence e.g. 5 mg not 5 mg. or 5 mgs.
4 The solidus or word "per" should only be used once in each unit. Correct usages are nmol/s.l or $nmol.s^{-1}.l^{-1}$ not nmol/s/l.
5 No commas are used to divide large numbers into groups of three but a space is left after every third digit. If the numerical value of the number is less than one, a zero should precede the decimal sign. In some countries the comma can be interpreted as a decimal sign e.g. 1,200 g is 1.200 g and not 1 200 g as intended.
6 When the symbol for a multiple or submultiple of a unit includes an exponent, the latter applies to both the unit symbol and the prefix symbol.
Example: a litre is a cubic decimetre = $dm^3 = (10^{-1}\ m)^3 = 10^{-3}\ m^3$

References

"The SI for the Health Professions", 1977, World Health Organization, Geneva.

Acknowledgements

The author thanks Dr Michael Lever of the Biochemistry Dept. Christchurch Hospital for his helpful advice in the preparation of this paper.

Section I — Problems (Answers on page 59)

1. How many mm in 1 m?
2. How many g in 1 kg?
3. How many mm in 1 cm?
4. How many mg in 1 g?
5. How many m in 1 km?
6. How many μg in 1 g?
7. How many mmol in 1 mol?
8. How many μmol in 1 mol?
9. How many ml in 1 l?
10. How many ml in 1 dl?
11. How many l in 1 m^3?
12. How many nmol in 1 mol?
13. How many pmol in 1 nmol?
14. Express 1 mg in g.
15. Express 1 mmol in mol.
16. Express 0.06 mmol/l plasma creatinine in μmol/l.
17. Express 0.45 mmol/l plasma uric acid in μmol/l.
18. Express 120 μmol/l serum bilirubin in mmol/l.
19. Express 250 mmol/l urine glucose as mol/l.
20. Express 6220 μmol/d urine creatinine in mmol/d.
21. Express 25 mg/d urine protein as g/d.
22. Express 15 g/dl blood hemoglobin in g/l.
23. Express 10 mU enzyme activity in pKat.
24. Express 100 mg/l in g/m^3.
25. Express 15 μCi in kBq.
26. Express 60 mU/ml enzyme activity in U/l.
27. Express 45 U/l enzyme activity in nkat/l.
28. Express 40 mmHg in kPa.
29. Express 10 μg/ml in mg/l.
30. Express mean cell volume of 84 μm^3 in fl.

TECHNICAL COMMUNICATION

An Evaluation of Solid-phase No Boil Radio-Isotope Assay Kit for Vitamin-B12 and Folate

Marja Hallowes A.N.Z.I.M.L.T., B.F. Postlewaight A.N.Z.I.M.L.T.

Department of Haematology, Auckland Hospital, Park Road, Auckland.

In 1983 Diagnostic Products Corporation introduced a no-boil solid phase (cellulose) assay for the estimation of vitamin B_{12} and folate. The proffered advantages of the new assay were:

1. elimination of requirement for boiling water bath
2. addition of binder and solid phase adsorption medium in one step
3. more accurate estimate of high B_{12} levels in some patients with myeloproliferative disorders with the use of cellulose.

In October 1983 the Diagnostic Products Corporation (DPC) No Boil Solid Phase Kit (NBSP) was obtained for evaluation. The vitamin B_{12}, serum and red cell folate results were compared with those from the DPC Boil, charcoal assay, which is in routine use in this laboratory.

The samples analysed were those referred to this laboratory for routine assay, with some of these samples being especially selected for the trial because they had low or low-normal values by the routine assay. Standards were assayed in duplicate, along with low, low-normal, mid-normal and high value control sera. The principle of the NBSP assay is as follows:

The vitamin B_{12} and folic acid in the patient sample are released from their respective carrier proteins by incubation at high pH (above pH12) in the presence of dithiothreitol and potassium cyanide. Purified intrinsic factor and beta-lactoglobulin are used as binders for the vitamin-B_{12} and folate respectively. Following the denaturation step, the pH is adjusted to pH 9.3 where the intrinsic factor is fully active and folate binder has equal affinity for Methyltetrahydrofolate and pteroyl glutamic acid (the more stable form of folate used in the assay calibrators). The binders are immobilised on microcrystalline cellulose so that in the assay procedure the binders and adsorbing material are added in one step. The microcrystalline cellulose replaces the charcoal which is employed in our routine assay. Following the addition of the solid-phase binder, separation of the bound fraction is achieved by centrifugation and decanting of the supernatant. The bound fraction is held on the cellulose pellet, which is counted in the gamma counter to determine the counts per minute. In this trial, the separation of the unbound fraction (supernatant) was achieved by centrifugation followed by gentle suction being applied rather than decanting. The protocol in the kit states that following the decanting step, the tubes should be inverted and held in contact with suitable absorbent material, so that all of the supernatant liquid is removed. However, this action led to a loss of a small quantity of the fine particles of cellulose and led to unacceptable values being obtained for both the vitamin B_{12} and folate levels.

The correlation data is summarised in the table, where the respective batch means are also provided. The NBSP assay values were treated as being on the y axis.

The NBSP assay gave lower serum and red cell folate values than the routine assay, with comparable vitamin B_{12} values being obtained. The major differences with the folate values occur in the high range of concentrations and is reflected in the slope of the plots (0.5142, 0.5049).

Prior to the assay of red cell folate, the whole blood samples were processed in an identical manner (i.e. dilution in ascorbate 1/30, ascorbate concentration 0.2g/100ml) to exclude any variables not related to the assay procedure.

The results indicate that for both serum and red cell folate, reference ranges would require re-determination since lower values were obtained by the NBSP assay.

With regard to the previously suggested advantages of the NBSP assay, several points require thought. The requirement for a boiling water bath may be a problem for a laboratory where these assays have not been previously performed, but does not present any difficulties for this laboratory. Although alkaline denaturation of the endogenous binders eliminates this requirement for a water bath and the 20 minutes boiling time, 30 minutes is still taken for the denaturation and an extra pipetting step is necessary, so that actual time involved is essentially the same for the two assays.

The addition of binder and solid phase adsorption medium in one step is a definite advantage in relation to the charcoal assay, but is offset in the no-boil system by the additional pipetting and incubation step, as mentioned.

The Research and Development of RIA products Inc. in the United States has produced sound evidence to indicate that charcoal adsorption does not permit accurate quantitation of elevated vitamin B_{12} levels in undiluted sera from patients with chronic myeloid leukaemia, and that the use of cellulose adsorption is superior in this regard. Thus, any CML sera should be diluted 1/10 prior to assay for vitamin B_{12} using the charcoal system for accurate results to be obtained.

In this trial, our laboratory did not investigate this aspect at all. Each assaying laboratory must decide on the appropriate course of action in these circumstances, and perhaps investigate any elevated results by dilution experiment to determine whether their assay gives accurate elevated values, particularly in the myeloproliferative disorders.

In conclusion, the new assay appears to be reliable, giving reproducible results with adequate precision. The clinical validity of results obtained have not been fully assessed and we believe that the proffered advantages of the modified assay over the boil, charcoal method are not sufficient to inspire a change of assay by our laboratory.

Correlation Data

	Serum B12	Serum Folate	Red Cell Folate
No. samples (correlation coefficient)	100 0.9797	100 0.9478	30 0.8034
Slope	1.0171	0.5142	0.5049
Y intercept	+7.9381	+1.74	+194.2588
Batch Mean NBSP	321pmol/l	7.58nmol/l	723nmol/l
Batch Mean (Boil, Charcoal)	308pmol/l	11.36nmol/l	1047nmol/l

THE NEW ZEALAND INSTITUTE OF
MEDICAL LABORATORY TECHNOLOGY INCORPORATED

MEDICAL LABORATORY TECHNOLOGY

Continuing Education Programme 1984

Lift Out

NEW ZEALAND INSTITUTE OF MEDICAL LABORATORY TECHNOLOGY

Course 1.

Annual Scientific Meeting

Theme: Monoclonal antibodies.

Speakers:
Professor R. C. Burton
Professor in Surgical Science at the University of Newcastle, Australia.

Dr C. Philpotts
Senior Lecturer (Histopathology)
Bristol Polytech England.

Dr M. C. Stuart
Assistant Director, Garvan Institute of Medical Research St Vincents' Hospital, Australia.

Proffered Papers.

Centre:
Dunedin 16th, 17th August 1984.

Course 2

Audio Visual Aids

The Institute has a number of Taped Programmes available for loan, together with accompanying course material.

Available from: Mr J. Elliott,
Microbiology Dept.,
Wellington Hospital
Wellington.

All sets have been prepared by the Centers of Disease Control, Laboratory training and Consultation Centre, Georgia, USA.

A full list of material available is elsewhere in the journal.

Course 3

Laboratory Aspects of Diabetes

Theme:
The theme of the workshop is how the laboratory can participate in the investigation and monitoring of Diabetes.

Content:

1. Lectures covering such subjects as population screening, use of blood glucose to monitor Diabetes; the Pathogenesis and epidemiology of Diabetes.
2. Workshops covering HbA_1C and Fructosamine Assays. Home Monitoring of blood glucose levels.

Speakers:
Proposed speakers from Auckland Medical School, Auckland and Waikato Hospital Laboratories.

Centre:
Marion Davis Postgraduate Centre and Nesfield House (Diabetic Society) Grafton Rd. Auckland.

Date:
2 Day Course 19th, 20th June, 1984.

New Zealand Institute of Medical Laboratory Technology Inc.

40th ANNUAL SCIENTIFIC MEETING

DUNEDIN 15-17th AUGUST 1984
VENUE: UNICOL and OTAGO UNIVERSITY
THEME: MONOCLONAL ANTIBODIES

GUEST SPEAKERS:

PROFESSOR R. BURTON — Professor of Surgical Science, University of Newcastle — Australia
Topic — MONOCLONAL ANTIBODIES
Forums — GENERAL, HAEMATOLOGY & HISTOPATHOLOGY

PROFESSOR B. C. FIRKIN — Haematologist, Alfred Hospital, Melbourne — Australia
Topic — COAGULATION
Forums — GENERAL & HAEMATOLOGY

DR C. PHILPOTTS — Senior Lecturer, Histopathology Dept, Bristol Polytechnic — England
Topic — IMMUNOCYTOCHEMISTRY ON RESIN SECTIONS (LM & EM)
Forums — GENERAL & HISTOPATHOLOGY/ CYTOLOGY

DR M. C. STUART - Assistant Director (Scientific), Garvan Institute of Medical Research, St Vincent's Hospital — Australia
Topic — MONOCLONAL ANTIBODIES IN BIOCHEMISTRY
Forums — GENERAL & BIOCHEMISTRY

DR N. SLOCUM — Production Manager — Monoclonal Antibodies Inc. England
Topic — MONOCLONAL ANTIBODIES
Forums — GENERAL & BIOCHEMISTRY

MR A. TAYLOR — Director of Operations — Tuta Laboratories Sydney — Australia
Topic — PRODUCTION OF BLOOD PRODUCTS
Forums — GENERAL & IMMUNOHAEMATOLOGY

PROFESSOR J. M. B. SMITH — Associate Professor in Microbiology, Otago Medical School
Topic — FUNGI
Forum — MICROBIOLOGY

DR G. TANNOCK — Senior Lecturer in Microbiology, University of Otago
Topic — ANAEROBES
Forum — MICROBIOLOGY

DR T. MAGUIRE — Senior Research Officer, Virus Research Laboratory, University of Otago
Topic — VIROLOGY
Forum — MICROBIOLOGY

WORKSHOPS: WEDNESDAY 15th AUGUST 1984

1. Histocytochemistry applied to resin sections (LM & EM) by Dr C. Philpotts
   Cost — $20.00
2. Clinically relevant anaerobes by Dr G. Tannock
   Cost — $20.00
3. Rapid microbiological identificaton by Mr M. McCarthy (sponsored by Carter Chemicals)
   Cost — $10.00

COSTS:

Registration	— $35.00
Hotel Accommodation	— $16.50 bed & breakfast
Dinner	— $4.25
Social Function (Friday)	— $25.00

NEW ZEALAND INSTITUTE OF MEDICAL LABORATORY TECHNOLOGY

Course 4

Allergy Testing — An update.

Content:
This course will be a series of lectures covering a number of topics such as "Allergy, what is it?" Hypersensitivity; currently accepted tests; Bee Venom Sensitivity; current concepts.

Authors:
Dr E. J. MacKay, Immunology Department, Auckland Hospital.
Mr D. Haines, Immunology Department, Auckland Hospital.

Centres:
Auckland: Saturday, June 30th, 1984. School of Medical Technology.
Wellington: Saturday, 15th September, 1984. Lecture Theatre, Wellington Clinical School.
Christchurch: Date to be advised.

Intended for:
The course has been structured so that it will act as an introduction to allergy testing for junior staff as well as providing up to date material for those senior people already involved in allergy testing. It will be of interest to all Technologists in every discipline not just Immunology.

Course 5

Microcomputers — An Introduction.

Outline:
The Topics covered in this workshop will include Hardware modules, how they are used, what they do and how they do it. Also Software Topics including levels of programs.

Content:
A simple run through a restricted subset of basic statements. Attendees will write their own programs and run them using HP 85's, Apple, Commodore and IBM Microcomputers. Demonstration of Word Processing and other Management Packages.

Intended for:
Technologists who are looking for a simple introduction into programming.

Tutors:
Mr E. Johnston, Clinical Chemistry, Auckland Hospital.
Mr J. Atkinson, Biochemistry Dept., Diagnostic Laboratory
Mr J. Webster, Computer Dept., Diagnostic Laboratory.

Centre:
School of Medical Laboratory Technology, Auckland.

Date:
Saturday, 7th July, 1984.
Registrations will be limited to 40, however if there is sufficient interest the course will be run the following Saturday as well.

Registration for all Courses:
Registration brochures will be forwarded to all Charge Technologists prior to each course.

The Pacific Way

Medical Technology in the Cook Islands

The last issue of the N.Z.I.M.L.T. Journal gave an insight into the problems of Medical Technology in Papua New Guinea. In this issue Rennie Dix provides a brief resumé of the situation in the Cook Islands from the 1950's until the present time and Sue Woonton, currently Charge Technologist of the Cook Islands Laboratories provides a description of the present situation.

Rennie Dix is Charge Technologist of the Haematology Department, Princess Mary Hospital, Auckland. Sue Woonton trained at Green Lane Hospital and since qualification has held Staff Technologist positions in the Haematology Department, Green Lane Hospital and the Auckland Blood Transfusion Service.

Rennie writes:

"Laboratory Services in the Cook Islands were at a fairly basic level prior to 1964, two staff had been trained at C.W.M. Hospital in Suva during the 1950's and two more were employed as Laboratory Assistants. The local medical staff made few demands upon the laboratory, other than for haemoglobins, urethral smears and an occasional unit of blood for transfusion. The situation changed with the appointment of Dr Joe Williams, a New Zealand trained Cook Islander as a Medical Officer in the Cook Islands Health Department. He pushed for the appointment of a seconded Medical Technologist to re-organise the existing services and expand the range of tests available. This was in fact my brief when I was appointed to the position in June, 1964.

The Rarotonga Hospital was then situated in Tupapa about one kilometre east of the Administrative Centre of Avarua. The building was old and dated from the early days of the New Zealand administration. Originally the building had central rooms surrounded by wide verandahs but over the years these had been closed in, as extra space was required. The laboratory was in a section of closed in verandah and could only be reached through the X-ray department when the X-ray machine was not in use. Facilities were minimal. They consisted of a sink, several tables and a few pieces of basic equipment, e.g., a Lovibond comparator, a microscope, centrifuge, Sahli haemoglobinometer and a refrigerator. A variety of glassware and sundries left by assorted research projects over the years completed the list of assets.

The Cook Islands Health Department had made funds available to re-equip the laboratory and slowly we were able to acquire such luxuries as a flame photometer, colorimeter, and a piped gas supply. Space was a real problem and it was not until the pharmacy was relocated in a new out-patients building that we acquired our own entrance, (not subject to the whims of the X-ray machine), an office, storage space and a patient area. The laboratory staff had to undertake a large amount of renovation and painting as the Cook Island Works Department was, as always, out of funds and could not provide the staff but they did provide the materials from their store.

Lorna Stewart seconded from Wellington to assist Cook Islands during a suspected typhoid outbreak in early 1964.

In a geographically remote area such as the Cook Islands you become by virtue of your position, a local expert, a sort of D.S.I.R., Public Health Laboratory and Police Forensic Laboratory rolled into one. The majority of the police work was the analysis of illegally brewed beer to check if it exceeded the legal limit of 3% proof spirit. Nearly every Thursday I was in court giving evidence against the unfortunates who let their beer "mature" too long before drinking it.

A training programme was set up for the local staff and recognised by the Cook Islands Education Department, which was necessary to gain pay increases for staff who passed. Jeanette Grey at the Auckland School of Medical Technology forwarded assignment material which we adapted to local conditions.

During my second term the dream of a new hospital came closer with the development of a New General Hospital on the site of the old T.B. sanitorium at Black Rock. The site is magnificent, high on the brow of a hill with panoramic views over the north eastern coast and fringing reef. This redevelopment was unfortunately not completed during my term but was enjoyed by my successors, Peter Moffat and Ted Norman.

Two New Zealand Medical Technologists served in Rarotonga in a relieving capacity during my term. Lorna Stewart from Wellington was seconded to the Cook Islands Health Department during a suspected Typhoid outbreak immediately prior to my arrival in August, 1964 and Allison Buchanan from Auckland, who relieved me for 5 months in 1968 while I was on furlough in New Zealand.

Rennie Dix — the first New Zealand Medical Technologist appointed to the Cook Islands, 1964-1971.

The people of the Cook Islands left a lasting impression on me. I remember with affection and gratitude the original laboratory staff Louis Marsters, John Metua (Long John), Tapo Vakatini (who died tragically after a football accident) and Plumber Nichols who held the fort on the island of Aitutaki and the staff who trained during my term: Vaevae Pare, Papa Aratangi, Ina Papera (who gained the New Zealand qualification at Whangarei), Tama and Timena Robati. My thanks to them all."

Sue Woonton writes:

"I hope this article doesn't sound too much like a travel brochure but it is a lovely place to live.

The Cook Islands group consists of 15 islands scattered over an area of 93,000 square miles. This geography presents special problems in running a health service. Most of the main islands are linked to Rarotonga by regular air or sea services. The largest island is Rarotonga. It is a very beautiful island, 21 miles in circumference, with a flat, narrow coastal belt and mountainous interior. It is surrounded by a reef enclosing the lagoon with several natural harbours.

The hospital is built on the site of the old T.B. Sanitorium, high on a hill overlooking the lagoon and shorefront. There are 100 beds in the hospital, but the occupancy rate is around 50%. There are four wards, medical, surgical, maternity and paediatric. There is also a large outpatient department, X-ray and two theatres where a wide variety of surgery is performed. The Medical staff consists of Cook Islanders and United Nations Volunteers, mostly from Asian countries. We have 3 surgeons, 1 pathologist, 1 cardiologist, 1 O. & G. specialist, 2 anaesthetists and 4 general physicians. Most of the outer islands are staffed by a Medical Officer and Nurses.

Laboratory

The laboratory is housed in the older part of the hospital, in a former ward. Thanks to a chap by the name of Brych, we have a very spacious department. Our Specimens come from the wards, outpatients clinics, village clinics, private practitioners and the outer islands. Only Aitutaki has a one-man lab, which does basic haematology, biochemistry and microbiology. The other islands send their specimens to us via the local air service. Statistics for 1982 show 8,000 patients used the laboratory with over 56,000 tests performed.

HAEMATOLOGY

The haematology section does full blood counts which consist of Hb, P.C.V., M.C.H.C., W.B.C., E.S.R. and differential. In addition each

Alison Buchanan seconded to Cook Islands for 5 months in 1968.

patient is screened for Microfilaria using a wet film. All work is done manually, our staining method is Leishmans. A busy day will see about 35 specimens.

On Rarotonga filariasis has been virtually eradicated, we see only an occasional positive case from the outer islands. Mass surveys of entire island populations are carried out in the outer islands and treatment programmes initiated. Only a few very old cases of elephantiasis are around today.

Platelet and reticulocyte counts are also done, along with coagulation studies, although coagulopathies are uncommon. We have had only two cases of thrombocytopenia this year, one drug induced, the other due to heavy metal poisoning.

The Blood Bank section is lucky to have a Blood Refrigerator donated by the N.Z. Red Cross. As we have no access to specialized blood products, blood for transfusion has to be relatively fresh, although we always have several units on standby. Blood donations come from the family and friends of the patient. Voluntary donation is slowly becoming accepted but people are still reluctant to give unless they know the patient. All donors are screened for V.D.R.L. and Hepatitis B Surface Antigen (Hepatest). Our crossmatch employs saline RT/37°C, enzyme 37°C and Indirect Coombs techniques. An American aid agency, The International Human Assistance Programme (I.H.A.P.) is donating to the laboratory a Mobile Blood Donor Unit which will improve our service immensely as we often have difficulty actually transporting the donors to the laboratory.

BIOCHEMISTRY

Our biochemistry section has recently been pushed up to International Standard with the donation by I.H.A.P. of a Gilford SBA 300 Chemistry Analyser. All biochemistry tests were done manually using expensive kit sets but now the SBA 300 can perform a wide range of tests with the advantage of selective batching, thus saving on reagents. The sample volume is extremely small 5-50μl so it can be used for neonatal and paediatric work. We now have a biochemistry repertoire comparable with larger centres. The tests available here are:

Albumin	G.G.T.
Alkaline Phosphatase	Glucose
Amylase	H.B.D.
A.S.T.	L.D.H.
Bilirubin	Total protein
Calcium	Urea
Cholesterol	Uric acid
CK	Acid Phosphatase
CO_2	Digoxin assays
Creatinine	

The SBA will perform thyroid functions but we do not see the demand justifying setting up the method yet. Electrolytes are done on an IL343 flame photometer. Also performed in biochemistry are Anti-Streptolysin O titres, V.D.R.L.'s and R.A. latex.

MICROBIOLOGY

The microbiology section carries out the usual range of tests and culture methods, although anaerobic culture needs improving.

Parasitology is interesting with Nematodes; Trichuris, Hookworms, Strongyloides and Ascaris. Giardia is also seen occasionally.

Our mycology is varied and interesting but we see surprisingly few exotic tropical diseases. Anything unusual is generally imported by yachties who have been cruising the tropics for months.

There are still new cases of T.B. being detected, mainly in younger people. The laboratory does T.B. culture and Zn stains and any positives are sent to Green Lane Reference Laboratory for identification and sensitivity testing. Microbiology also carry out Food and Water Testing for the Public Health Department, funded by W.H.O.

HISTOLOGY AND CYTOLOGY

This section was established this year as the first Cook Island Pathologist returned home to work. In the past specimens were sent to New Zealand by air, a very costly business.

We were very lucky, thanks to Mr John Sloan of National Womens' to purchase from the Auckland Hospital Board Salvage Store, a secondhand tissue processor microtome and wax dispenser. The first sections rolled off the production line in October, using some techniques unknown to Culling or Carleton, but due to delays in supplies arriving, we had to improvise and make do — but we were proud to send a sample of our H. & E's to Auckland Hospital.

LABORATORY STAFF

The laboratory has a staff of eleven; four Cook Island qualified technicians, six trainees, a laboratory cleaner and myself. The trainees have three years practical experience plus lectures by our Pathologist and myself and are required to sit the Cook Islands Laboratory Technicians Certificate. During their training an overseas course is arranged to allow them to work in another centre. The Pacific Paramedical Training Centre has helped to improve the quality of laboratory workers in the Cooks.

Our oldest member is Louis Marsters, a man in his 60's who has been in the laboratory for over 40 years and can tell some wonderful stories of the old days.

The laboratory is also responsible for the mortuary and carries out any embalming requested. Post mortems are not common here. However we are called upon to carry out forensic work for Police and Coroners cases.

Our other services to the public include supplying distilled water for anything from car batteries to electroplating. Occasionally our talents as taxidermists are called upon, most recently we preserved two Coconut Crabs to send to New Zealand.

The main problem facing us is lack of funds for capital expenditure. As a developing country we rely heavily on overseas aid, especially for the purchase of expensive equipment such as our SBA 300 but there is aid available from many agencies and foreign governments as far away as Holland and Germany. The laboratory has been very, very fortunate this year, apart from the SBA 300 and Blood Donor Vehicle, we have been given a Delphi Portable Hb meter, an Olympus BHS microscope and have had a number of courses for staff members funded.

The second problem here is one of supplies of routine laboratory reagents. We often have to wait months for orders, this makes stock juggling interesting. Occasionally we are unable to do the most basic tests because of delays.

Life in the Cooks is very relaxed. The climate is warm and constant. Traffic is fairly tame and pollution non-existent. Cook Islanders are a friendly happy people with a charming innocence which is refreshing after the intellectual competitiveness and tension that is part of 'civilized' life. They love sport of any kind; rugby, netball, soccer, cricket, and tennis have large followings. Religion plays a major part in the life of a Cook Islander, Sundays are strictly for rest.

The younger people still long for the bright lights of New Zealand and Australia, but many return with qualifications and a determination to help their own country. Many of the positions held by overseas personnel are now filled by Cook Islanders in a move towards complete localization.

My first year has been full of changes, new friends, new skills and a new outlook on life. I have no complaints."

Ted Norman, New Zealand Medical Technologist appointed to the Cook Islands 1974-1980.

MLTB NEWS

New Secretary

Miss Helen Robertshaw who has been the Boards' Secretary over many years, has now been transferred and her position as Secretary has been filled by Miss Jennifer Van Hunen. Although over the next few months Helen will be training Miss Van Hunen, all correspondence, telephone enquiries etc., should be addressed to Miss Van Hunen. At the Board meeting opportunity was taken to thank Miss Robertshaw for her many years of work and service to the Board and I am sure that Laboratory staff around New Zealand will join me in echoing those sentiments and wishing Helen well for the future.

Recognition of D.I.M.L.T. in U.K.

An opportunity has been taken recently to ask the Council for Professions Supplementary to Medicine to state their present attitude to the New Zealand Diploma and its recognition within the U.K. Their reply has stated that they are prepared to accept the diploma as a registrable qualification, but like all other overseas qualifications accepted for registration purposes, holders of the diploma will have to acquire not less than three months whole time training in "approved" laboratories in the United Kingdom and be certified as competent to practice the profession at the end of that time before they can obtain State Registration.

Syllabi for Nuclear Medicine

The new syllabi at both the Certificate and Specialist Level for Nuclear Medicine are now available and will be used for the 1984 examinations.

Handbook

The 1984 edition of the Handbook for Trainees is currently being printed and should be available early in March. Opportunity will be taken to forward copies of the pages where significant changes have been made to Charge Technologists so that they may distribute them to trainees who already have earlier editions of the Handbook so that these can be brought up to date. Opportunity is taken again to remind all Charge Technologists and Trainees that the closing date for exam applications will be strictly enforced and **no late applications can be accepted.** The application forms for these examinations are available at the back of the handbook.

Out of Print Text

We have been notified that the book "Man's Haemoglobin" by Lehman and Huntsman is now out of print. The Board's recommendation for a replacement text is "Human Haemoglobins" by Frank Bunn, published by W. B. Saunders.

Textbooks etc., in Practical Examinations

Although there was some confusion last year about the use of texts and laboratory manuals within the practical examination, the Board takes this opportunity of re-affirming its policy that textbooks and laboratory manuals will be allowed in all practical examinations and this will apply to all disciplines in the 1984 examinations.

Allowable time for Practical Examinations

For the 1984 examinations there will be a minimum of three hours allowed for the practical examinations and there will also be a set maximum time which will be based on the cumulative time required to perform each test independently. This will be notified to all Invigilators by the examiners in each of the disciplines. The Board is additionally introducing a new form that Invigilators will complete for each candidate showing the time taken in the examination and making note of any problems etc., that have occurred.

Times For Practical Examinations

To allow flexibility within the Laboratory so that routine work is disturbed as little as possible the Board will allow the starting time for a practical examination to be set by the local supervisors. However the practical examination must of course be run **on the date** that is stipulated.

Confidentiality on Practical Examinations

The Board has been concerned that some breach of confidentiality may have occurred last year because of the ordering of an unusual reagent from a science supply house. Invigilators and Charge Technologists are reminded that it is essential to retain confidentiality regarding the content of any examination paper and to this end the Board is asking that all senior examiners advise Charge Technologists in plenty of time of any tests that are to be performed by their students in the practical examination, which may require the purchase of a reagent not normally held in the laboratory. It is hoped that this may allow several labs to combine to purchase such a reagent.

Training at Base Hospitals

The Board's present policy for trainees within the smaller centres is that they are to spend an approved amount of time at the Base Hospital during their Certificate or Specialist Level Training. The Board would seriously recommend that all trainees who undertake such training at a Base Hospital should consider sitting the practical examination from that Base Hospital. If examinations are to be undertaken from the Base Hospital it is obviously important that the student and his Charge Technologist make early enquiries with the Base Hospital regarding the feasibility of their candidate sitting the practical examination at the Base Hospital. If candidates need to leave their laboratory to obtain adequate instruction in methods not carried out in their laboratory then there is obviously a disadvantage in sitting an examination in their home laboratory where that method is not currently in use.

Examination Papers

Copies of the examination papers for the 1983 examinations will be distributed in the near future by the Board Secretary to all laboratories. Copies of examiners reports and marking schedules will be available from the Secretary **on request.**

Management

Trainees should be aware that there is a section of management included in all of the Certificate and Specialist syllabi and that these may be examined in any one year. The Board has recently expressed concern over the growing percentage of some papers which is allocated to management and will in the future endeavour to ensure that the amount of management included in any one paper will realistically reflect the percentage of management within the syllabus.

D. J. Philip
CHAIRMAN

Diploma in Medical Laboratory Technology and Limited Registration Qualifyers 1983

ALEXANDER Julie Barbara, ANDERSON Beverley Maree, BARLOW Ian Howard, BRANDSEN Gerardus Marinus Theodorus, BROWN Joyce Danielle, BRYANT Vicki, BULLING Tonia Lily, CALDWELL Rosemary Jane, CAMPBELL Rosemary Ann, CROWTHER Michael William, CURLEY Fiona Willemina, DEROLES-MAIN Jan Rosemary, DIPROSE Janette Louise, DYSON Cheryle-Anne, ELLIS Dianne Karen, EVANS Gloria Evelyn, EWENS Dawn Elisabeth, FLAWS Pamela Claire, GEBBIE Jacqueline Dawn, GEURTS Michael Jan, GLASSEY Fiona Jean, GRAHAM Sandra Marie, HANCOCK Desley Claire, HAWORTH Rosemary Helen, HEARES Fiona Claire Kenny, HILL Cheryl Marie, HOSKEN Leigh Helen, HUYMANS Maria Helena, JACKSON Wendy Miree, JOE Yvonne Dianna, JOHNSON Denise Mary, JONES Bronwyn Anne, KAPE Lynda-Jane, KENNEDY Michelle Louise, LLOYD Gael Margaret, LYONS Elizabeth, MacDONALD Donella Matilda, McCOMB Penelope Winsome, McDOWELL Jeanette Ruby, MACE Anthony Charles, MASON Nicola Anne, MAYES Kathleen May Patricia, MEE Adrian Campbell, MEEK Dianne, MILLER Christopher John, MILNE Kim Alison, MITCHENER Wendy Marie, MUIR Helen Margaret, O'CONNOR Julie Maree, ORTON Heather Brenda, PAYNE Gordon John, PETERS Julie Ann, POLOAI James, RATTRAY Stephanie Barbara, ROBB Leanne Lisbeth, ROSMAN Ingrid, RUSSELL Colleen Dawn, SCARROW David John, SEXTON Sandra Jane, SHERNING Jill Katrina, SILVESTER Barbara Anne, SMITH Sarah Jane, THOMPSON Mark Charles, TOLLEMACHE Carol Joy, TULLOCH Christine Estelle, VAN DAM Caroline Anita, VAN KUYK Joan Elisabeth, VINCENT Clarke Stephen, WAKEM Philip John, WALLACE Dalena Maria, WALLACE Mary Frances, WEDD Kathryn Elizabeth, WHITE Glennis Lynn, WILSON Paul William.

INSTITUTE BUSINESS

Office-Bearers of the N.Z.I.M.L.T. 1983-4

President
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11 Turere Place, Wanganui

Vice-Presidents
C. Campbell
K. McLoughlin

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B. T. Edwards
Haematology, Christchurch Hospital

Treasurer
W. J. Wilson
Blood Transfusion Service, Auckland

Council
M. Young, D. Reilly, J. Elliot, J. Parker, P. McLeod

Editor
D. Dixon-McIver
Biochemistry Dept., National Women's Hospital, Auckland.
or the Editor, P.O. Box 35-276, Auckland, 10.

Membership Secretary
Margaret Young
Laboratory, Waikato Hospital, Hamilton.

Membership Fees and Enquiries

Membership fees for the year beginning April 1, 1984 are:
For Fellows — $40

For Associates — $40

For Members — $30

For Non-practising Members — $20

All membership fees, changes of address or particulars, applications for membership or changes in status should be sent to the Membership Secretary at the address given above.

Members wishing to receive their publications by airmail should contact the Editor to make the necessary arrangement.

EXAMINATION PRIZEWINNERS

Part II

Clinical Biochemistry (Donated by Roche Products (NZ) Ltd.)
Miss C. J. Tollemache, Princess Mary Laboratory, Auckland

Haematology (Donated by Kempthorne Medical Supplies Ltd)
Miss M. A. Janssen, Waikato Hospital, Hamilton.

Histology (Donated by Kempthorne Medical Supplies Ltd)
Mr C. A. Lee, Taranaki Base Hospital, New Plymouth

Immunohaematology (Donated by Technicon Equipment Pty Ltd)
Miss M. J. Collier, Taranaki Base Hospital, New Plymouth.

Immunology (Donated by Hoechst (NZ) Ltd)
Mrs G. D. Lloyd, Auckland Hospital.

Microbiology (Donated by Roche Products (NZ) Ltd)
Miss P. J. Cooke, Auckland Hospital.

Cytogenetics (Donated by Sci-Med (NZ) Ltd)
Miss D. Meek, Dunedin Hospital, Dunedin.

Part III

Clinical Biochemistry (Donated by Sci-Med (NZ) Ltd)
Mrs S. M. Greenwood, Middlemore Hospital.

Haematology (Donated by General Diagnostics)
Miss L. J. Kape, Waikato Hospital, Hamilton.

Immunology (Donated by Hoechst NZ Ltd)
Mrs G. D. Lloyd, Auckland Hospital.

Medical Cytology (Donated by Ortho Diagnostic Systems)
Mr I. H. Barlow, National Women's Hospital.

Microbiology (Donated by Wilton Instruments)
Miss K. A. Milne, Wellington Hospital.

MEMBERSHIP SUB-COMMITTEE REPORT MARCH 1984

Membership

Since our December meeting there have been the following changes:

	March 84	Nov 83	Feb 83
Membership as at 2nd March	1275	1389	1470
LESS Resignations 9, G.N.A. 9	18	6	138
	1257	1383	1332
PLUS Membership Applications 69	69	39	51
	1326	1422	1383

Applications for Membership as at 1st March 1984

Mrs L. Anderson	Wellington
Miss J. M. Andrew	Gisborne
Miss N. M. Ball	Dunedin
Miss L. L. Baran	Christchurch
Mr T. J. Barnett	Nelson
Miss J. H. Bastin	Christchurch
Miss J. S. Beacham	Auckland
Miss J. E. Bolton	Gisborne
Miss C. D. Brown	Auckland
Mrs G. P. Buckley	Christchurch
Miss C. M. Burns	Wellington
Miss J. R. Burrows	Christchurch
Miss P. F. Christopher	Auckland
Miss J. A. Cormack	Christchurch
Miss N. L. Crawford	Auckland
Mrs L. M. Cumming	Auckland
Miss H. W. Davy	Auckland
Miss C. M. Dew	Wellington
Miss M. Dixon	Auckland
Miss L. Donnelly	Kew
Miss R. L. Eagar	Wellington
Miss D. C. Escott	Christchurch
Miss K. J. Familton	Invercargill
Miss M. A. R. Gilbert	Auckland
Miss L. M. Giles	Christchurch
Miss C. A. Goodyer	Wellington
Miss M. Gregory	Whangarei

Miss J. L. Harcombe	Auckland
Miss L. K. Holden	Auckland
Mr D. M. Huege de Serville	Auckland
Miss J. A. Ingle	Wellington
Miss L. M. Jones	Auckland
Miss L. M. Koman	Auckland
Miss D. J. Lindsey	Auckland
Miss B. J. Little	Invercargill
Miss D. J. Loader	Thames
Miss J. D. Main	Whangarei
Miss J. Mayes	Auckland
Mrs A. McAndrew	Auckland
Miss O. M. McCabe	Dunedin
Miss C. A. McClean	Auckland
Miss N. M. McNeil	Auckland
Miss G. L. Miller	Auckland
Miss J. M. Mills	Christchurch
Ms. K. S. Monaghan	Christchurch
Miss K. J. Murray	Auckland
Miss V. M. Neal	Blenheim
Miss S. B. O'Brian	Auckland
Mrs R. A. Oldershaw	Auckland
Miss T. M. Partridge	Lower Hutt
Miss L. A. Pearce	Tauranga
Miss D. M. Peterson	Christchurch
Miss J. M. Robinson	Auckland
Mrs A. M. Sharp	Dunedin
Mrs C. A. Steiner	Hawera
Mrs K. M. Stirling	Auckland
Miss J. L. Stovold	Tauranga
Miss C. Thom	Rotorua
Miss A. L. Tombs	Dunedin
Miss J. M. Abbott	Christchurch
Miss D. M. Aitken	Christchurch
Miss K. M. Anderson	Christchurch
Mrs V. M. Brown	Gisborne
Miss J. H. Carter	Auckland
Mrs L. A. Cooper	New Plymouth
Mr D. Green	Whangarei
Mrs L. Flavell	Auckland
Ms F. F. Foli	Auckland
Ms R. K. Johnstone	Palmerston North
Miss S. L. Stroud	Christchurch
Mr N. Tavati	Auckland
Mr K. C. Taylor	Whangarei
Mr A. L. Thompson	Rotorua
Miss V. A. Woods	Christchurch
Miss M. J. Tresidder	Auckland
Mrs J. I. Trevathan	Dunedin
Miss S. V. Trevatt	Tauranga
Mr P. J. Wakem	Wellington
Miss J. R. Walker	Auckland
Miss D. K. Walsh	Christchurch
Mr R. J. Wharton	Auckland
Mrs J. White	Dunedin
Miss L. M. Woodward	Hamilton
Mr M. I. Wright	Dunedin

Applications for Associateship as at 1st March 1984

Miss L. M. Giles	Christchurch
Mr P. J. Wakem	Wellington

Resignations as at 1st March 1984

Mrs A. J. Williams	Hamilton Med. Labs.
Mrs C. M. Lancaster	Hamilton Med. Labs.
Mrs I. L. Keymer	Auckland Diagnostic Lab.
Mrs L. Hampton	Invercargill
Mrs B. A. Happer	Auckland Diagnostic Lab.
Ms K. Hay	Putaruru
Ms J. Jackson	Christchurch
Ms C. Appleton	Northland Base Hospital
Ms. M. Thomas	Nelson

Gone No Address as at 1st March 1984

Miss C. M. McDonald
Mrs H. M. Kerr
Miss C. A. Crawther
Mr I. D. Wilkinson
Miss R. Mills
Mrs J. S. Salt
Mr D. P. Yep
Mrs F. L. Jarden

Address Given — No Resignation

Mr K. J. Beechey	England

Deceased

Burnie Stevenson	Australia

Education Committee Report for NZIMLT Council Meeting 7th 8th March 1984

The Education Committee met on the 6th December to formulate recommendations to the Medical Laboratory Technologists Board regarding the Massey Degree Course. The recommendations were as follows:-

1. That the Board reaffirms that Massey University is still prepared to offer the course.
2. That a deputation from the Board request a meeting with the Minister of Health.
3. That the Board reply to the Hospital Boards Association making the following points.
   a) That the cost of the course could be reduced by placing trainees on student tertiary bursary while attending block courses at Massey.
   b) That the Board regards international recognition of the qualification of utmost importance.
   c) That the Board remains convinced that a science degree qualification is essential to meet the needs of a modern medical laboratory and accepts the offer of the Hospital Boards Association to jointly undertake a manpower planning study by proposing the establishment of a subcommittee comprising two members of the Medical Laboratory Technologists Board and two members of the Hospital Boards Association. It is proposed that this committee would be responsible to undertake a study of selected hospital and private medical laboratories to establish if there is a need to include a science degree in the registerable qualification of a Medical Laboratory Technologist.

Matters relating to the course from the Medical Laboratory Technologists Board meeting of the 7th December:-

A letter was received from the Hospital Boards Association subsequent to the meeting with a delegation from the MLTB on the 5th October. This reaffirmed the negative stance of the HBA to the degree course for reasons previously outlined.

This was particularly disappointing because the delegation had been well received by the HBA, and left the meeting with some optimism that progress had been made in convincing them that the degree course was the best option. As a result of the letter the Board resolved to take the following action.

1. To send a letter to Massey University to ascertain whether the degree course as previously outlined would be available if the approvals were received.
2. That the Board write to the Department of Health requesting that a cost analysis be done of possible post NZCS education in Medical Laboratory Technology in tertiary institutions.
3. That a manpower planning exercise be carried out utilising the Department of Health management services research unit to ascertain the staffing requirements of laboratories.

Correspondence

Dear Mr Chapman,

Accompanying circular letter (Hosp No. 1983/8) was a new appendix for circular letter No. Hosp 1980/29.

Under this appendix authority is given to Hospital Boards to meet the expenses for staff attending an approved training programme for up to 12 weeks.

As you know this qualifies medical laboratory technology trainees attending block courses at the Central Institute of Technology to receive full pay and a refund of expenses.

Unfortunately, during their final year students are required to attend a course of 14 weeks as two weeks are required for the students' examinations. Unfortunately some employees are experiencing difficulties in receiving pay and expenses for the final two weeks.

This Institute would be grateful if your Department could consider granting authority to Hospital Boards for meeting pay and expenses for the final two weeks in the same way that authority is granted for a block of eighteen weeks for third year orthotists' block courses.

Yours sincerely
NZIMLT
B. T. Edwards
Secretary

Dear Mr Edwards

I refer to your letter of 29 August 1983 about circular letter Hosp. No. 1983/8 and the expenses payable on block courses.

An amendment to the staff Manual giving boards authority to meet expenses for the 14 week block course on the same basis as the 18 week orthotists block course is being made.

I regret the delay in replying.

Yours sincerely
M. J. Chapman
for Director
Division of Hospitals

Congratulations

The NZIMLT congratulates Ron McKenzie on attaining his PhD and Alan Wilson on attaining the Diploma in Health Administration.

AWARDS

Applications are invited for the NZIMLT Scholarship and the Eli Lilly Microbiology Scholarship. Details of these scholarships are set out below.

Eli Lilly Microbiology Scholarship

This award, consisting of $500 kindly donated by Lilly Industries (NZ) Ltd, is to be used **either** for the purpose of funding a research project which cannot otherwise be undertaken **or** to attend an overseas scientific meeting. The scholarship is open to all financial members of the NZIMLT currently working in the field of Microbiology. Applicants for the Scholarship must apply on the official application form available from the Secretary of the NZIMLT.

Acceptance of the Scholarship will require the recipient **either** to prepare an article for publication in the NZIMLT journal relating to that research **or** prepare a full report on the meeting attended for publication in the NZIMLT journal.

Applications close on **1 July 1984** with the Secretary, NZIMLT, Haematology Dept, Christchurch Hospital, Christchurch. The successful applicant will be announced at the Annual Scientific Meeting.

NZIMLT Scholarship

This award, consisting of $500 donated by the NZIMLT, is to be used **either** for the purpose of funding a research project which cannot otherwise be undertaken **or** to attend an overseas scientific meeting. The Scholarship is open to all financial members of the NZIMLT. Applications must be made on the official application form available from the Secretary of the NZIMLT.

Acceptance of the Scholarship will require the recipient **either** to prepare an article for publication in the NZIMLT Journal relating to that research **or** prepare a full report on the meeting attended for publication in the NZIMLT journal.

Applications close on **1 July 1984** with the Secretary, NZIMLT, Haematology Dept, Christchurch Hospital, Christchurch. The successful applicant will be announced at the Annual Scientific Meeting.

Blood Banking School

The Fourth International Applied Blood Banking School is to be held at Ortho Diagnostic Systems Inc. New Jersey USA, October 15-19 1984.

The course is designed for Clinical Pathologists and Medical Technologists who desire to study and work with the latest blood banking techniques. Ortho Diagnostic Systems will provide the services of expert instructors, laboratory and lecture facilities, lunches and local transportation. If you plan to be in the New York area at that time, (AABB Meeting follows in the next week), and would like to attend, please apply as soon as possible, as the courses usually fill quickly — before 15 June 1984.

For further information or applications contact Martin Fraser, Ortho Diagnostic Systems, P.O. Box 9222, Auckland. Telephone 543-735, or 543-755.

NEWS FROM THE HILL

Virology Upgrading

Virology laboratories at Christchurch Hospital are to be upgraded to accommodate equipment included in the regional specialty package announced last year. The North Canterbury Hospital Board has been authorised to proceed with alterations to provide for the relocation of the public health laboratory so virology and serology may be combined in the larger former public health laboratory, Mr Aussie Malcolm, Minister of Health, announced recently.

Mr Malcolm said because the work would have to be carried out while staff were still working in the building, the North Canterbury Hospital Board had decided it was more feasible that it's own staff carry out the alterations.

New Laboratory Pharmacy for Thames

A new laboratory and pharmacy is to be built as an additional floor on the existing physical medicine building at Thames Hospital, Mr Aussie Malcolm, Minister of Health, announced recently. Mr Malcolm said the Thames Hospital Board had been granted approval to call tenders for the project, which would alleviate a long standing problem for laboratory and pharmacy staff, and the Board. "Flooding of former premises has seen staff being scattered over several buildings, often working in less than ideal circumstances. I am grateful to staff for their dedication and know they will welcome this approval as a positive step by the Board to solve accommodation problems," Mr Malcolm said.

Commission Members Announced

The appointments to the Health Service Personnel Commission, were announced recently by the Minister of Health, Mr Aussie Malcolm. The new commission, established under the Health Services Personnel Act 1983, would replace the function of the Director-General of Health in setting pay and conditions for Health personnel under the State Services Conditions of Employment Act 1977.

It would also have a role in respect of other hospital staff whose terms of employment came under the Industrial Relations Act 1973. "Hospital boards will remain the employers of staff however," said Mr Malcolm.

The members of the commission are: Mr J. R. (John) Martin, Deputy Director-General of Health (Administration) Chairman; Mr R. H. (Dick) Kerr, Deputy Chairman; Mr R. A. (Ron) Kelly, a member of the State Services Commission, and Miss R. (Rita) McEwan.

Mr Kerr, the immediate past-president of the Hospital Boards' Association, was chairman of the South Canterbury Hospital Board for 27 years and is a member of the board of Dalgety Crown. Miss McEwan was principal nurse at Porirua Hospital and was a member of the Oakley committee of inquiry. Messrs Martin and Kelly represent the Director-General of Health and the chairman of the State Services Commission as provided for in the Act.

Mr Malcolm said the new commission which begins operation on 1 April 1984, would play a leadership role in promoting and developing personnel policies and standards as well as a national careers structure, within the health service in order to improve its efficiency and economy. "The 60,000 people employed in the Health service are a major asset to this country and the establishment of this commission is a positive measure to promote modern personnel management," said Mr Malcolm.

BRANCH NEWS

Auckland Branch

The Annual General Meeting of the Auckland Branch of the NZIMLT was held at Auckland on December 7, 1983. The following officers were elected:-

Chairman	Mr Ron Law
Secretary	Ms Anne Watson
Treasurer	Mr Mike Brokenshire
Committee	Ms Mary Bilkey
	Ms Fiona Heares
	Ms Vanessa Martin
	Mr Ray Sheldon
	Mr Peter Wyatt

CSU NOTES

Annual and Long Service Leave

It was agreed to refer the proposals of the Institute of Medical Laboratory Technology on annual and long service leave to the Block A Committee for consideration in respect of the next claim.

Actuarial reductions after 40 years service

The following letter of 7 February has been received from the Superintendent of the Government Superannuation Fund:

"I refer to your letters CSU/Super of 17 and 27 January in which you have expressed concern that the retiring allowance of members of the Fund who joined government service after 1 January 1946 may be actuarially reduced on retirement if having completed 40 years contributory service they have not also attained age 60.

At the present time any member of the Fund who entered the service after 1 January 1946 can receive an unreduced retiring allowance from age 60 under the extended provisions of Section 35 (3)(f)(i) of the Government Superannuation Fund Act 1956 without having to complete 40 years contributory service. However, there is no comparable provision to permit the member who has completed 40 years contributory service to receive an unreduced retiring allowance regardless of age before age 60. Consequently, those who retire under the provisions of Section 35(3)(f)(iii), ie with contributory service of not less than 35 years, may find that their retiring allowance will be actuarially assessed.

You will be aware that the Board has discretionary powers under Section 35(7) of the Act as to the application and extent of any actuarial reduction that it may impose on retiring allowances approved under the extended provisions. It could be that the Board may wish to assist those in the area which concern you by taking a more liberal approach as to the extent of the actuarial reduction that may be applied in such cases.

You have now asked that this matter be raised at the next Board meeting and I will arrange to have it included in the agenda.

You have also commented on the operation of the actuarial reduction and asked whether there is some type of identifiable cut-off point beyond which no actuarial reduction is imposed. In today's climate, the degree of inflation is of major significance in determining the size of the actuarial reduction. Recently the Government Actuary remarked that the high rates of escalation in the general level of salaries have resulted in lower actuarial reductions than have previously applied. Over the last 18 months this has changed and actuarial reductions have returned to be a more "normal" albeit higher level. Because of uncertainty of future trends, it could be difficult if not impossible to provide a cut-off point from which no actuarial reduction would be imposed. Also it would be impracticable to ignore salary increases, promotion, inflation and the like which could follow past that point.

While I can offer no practical remedy at this time, I would suggest that members contemplating retirement who are affected by the age 60/40 qualification should write to this office for a quotation at the 40 year point so as to ascertain the extent (if any) to which their retiring allowance could be affected by an actuarial reduction."

N.Z.I.M.L.T. LIBRARY

The following Journals have been recently received. They are available for loan from The Librarian, Mr J. Lucas, Haematology Dept. Dunedin Hospital.

American Journal of Medical Technology 49.7
1. Lab. Diagnosis of Systemic Rheumatic Disease.
2. Current status of Pregnancy Testing
3. I.M. and Syphilis Testing
4. An evaluation of the Novobiocin Disc Diffusion Test for Identifying *Staph. saprophyticus*
5. Simulated C.S.F. for teaching purposes

American Journal of Medical Technology 49.8
1. Therapeutic Drug Monitoring — History
2. Analytical methods of therapeutic drug monitoring
3. Pharmacokinetics
4. Future role of the laboratory in therapeutic drug monitoring
5. *Capnocytophaga:* Literature review

American Journal of Medical Technology 49.9
1. Quality control in Haematology — History
2. Q.C. Program for a computerized, high volume, automated haematology laboratory
3. Q.C. on the Coulter S — Plus 11
4. Q.C. on the Ortho ELT-8
5. Correlation of the haematologic data from the individual patient as a Q.C. tool

American Journal of Medical Technology 49.11
1. Strategies for susceptibility testing of fastidious aerobic bacteria
2. Susceptibility testing of anaerobic bacteria
3. Lab. tests for antimicrobial synergy
4. Assay of antimicrobial agents
5. Selective pigment medium for *Strep. agalactiae*

Laboratory Medicine Aug 1983
1. Automated differentials — review and prospectus
2. New parameters in automated haematology instruments
3. Spurious results from automated haematology cell counters
4. Evaluation of whole blood platelet analyzers
5. Automated differential analysis by flow cytochemistry

Laboratory Medicine Sept 1983
1. Experience with a commercially available kit for determining serum FDP's
2. ARIA II interface with a desk top computer
3. Evaluation of the Replireader for identification and sensitivity determination of Gram negative bacilli
4. Cost of Clinical Chemistry Laboratory Tests
5. Effect of in vitro haemolysis on the ratio of LDH-1 to LDH-2

Laboratory Medicine Nov 1983
1. Non-A, non-B Hepatitis: Etiology and Clinical course
2. Transfusion-associated Hepatitis
3. Hepatitis-B immunoprophylaxis
4. Type A and Type B Hepatitis

Australian Journal of Medical Laboratory Science Vol 4:4
1. Application of Microcomputer in the Blood Bank
2. Rapid Selective Indicator medium for the isolation of methicillin resistant *S. aureus*
3. The use of L.I.S.S. in the routine crossmatch laboratory
4. Immunohistochemical and biochemical correlation of Prostate Specific Acid Phosphatase in prostatic carcinoma

Canadian Journal of Medical Technology Vol. 45:3
1. Evaluation of methods for measurement of plasma ammonia in a paediatric hospital
2. Diagnosis of *T. vaginalis* using acidine orange

AUDIO VISUAL AIDS

Listed below is the complete list of audio-visual material that is available for loan from John Elliott, Laboratory, Wellington Hospital.

CDC Laboratory Update Series

CDC-76-1 Immunodiagnostic Tests for Autoimmune Diseases.
CDC-80-2 Anaerobic Bacteriology in the Clinical Laboratory.
CDC-76-3 The Quality Control of Laboratory Plating Media for Gonococcus and Other Bacterial Agents.
CDC-76-4 Radioimmunoassay. Part I.
CDC-76-5 Radioimmunoassay. Part II.
CDC-76-6 Radioimmunoassay. Part III.
CDC-76-7 Identification of Intestinal Protozoa. Part I.
CDC-76-8 Identification of Intestinal Protozoa. Part II.
CDC-77-10 Leukocyte Morphology in Healthy and Diseased States. Part I
CDC-77-11 Leukocyte Morphology in Healthy and Diseased States. Part II
CDC-77-12 Leukocyte Morphology in Healthy and Diseased States. Part III
CDC-77-13 Isolation and Identification of Streptococci. Part I
CDC-77-14 Isolation and Identification of Streptococci. Part II
CDC-77-15 Isolation and Identification of Streptococci. Part III
CDC-77-17 Identification of *Neisseria gonorrhoeae*. Part I.
CDC-77-18 Identification of *Neisseria gonorrhoeae*. Part II.
CDC-77-19 Biotyping of Enterobacteriaceae in the Clinical Laboratory.
CDC-77-20 Optimizing Spectrophotometric Measurement.
CDC-75-21 Statistical Aspects of Quality Control in Clinical Chemistry.
CDC-80-22 Disc Agar Diffusion Susceptibility Test.
CDC-75-23 Microscopic Evaluation of Red Blood Cell Morphology.
CDC-76-24 Safety Management in the Laboratory.
CDC-80-27 Blood Coagulation. Part I.
CDC-80-28 Blood Coagulation. Part II.
CDC-81-29 Blood Coagulation. Part III.
CDC-78-30 Fundamental Nature of the Antigen-Antibody System.
CDC-78-32 Identification of Helminth Eggs and Larvae.
CDC-78-33 Collection of Satisfactory Fecal Specimens.
CDC-78-34 Evaluation of Techniques for Examining Fecal Specimens.
CDC-78-35 Preparing and Staining Fecal Smears and How to Correct Problems and Errors.
CDC-78-36 Preparing and Staining Blood Films for Diagnosis of Parasitic Infection.
CDC-78-37 Controlling Infectious Aerosols in the Laboratory.
CDC-78-38 Selection and Use of Kits in the Clinical Laboratory.
CDC-78-39 Rubella Screening and Control.
CDC-78-40 Serodiagnosis of Streptococcal Infection (ASO-ADB Tests).
CDC-78-41 Use of the Anaerobic Glove Box.
CDC-80-2 Detection of B-Lactamase in *Neisseria gonorrhaeae* and *Haemophilus influenzae*.
CDC-78-43 Serodiagnosis of Toxoplasmosis.
CDC-78-44 Evaluation of RIA Kits.
CDC-78-45 Human T and B Cells, Basic Concepts
CDC-78-46 Presumptive ID of Anaerobic Nonsporeforming Gram-Negative Bacteria.
CDC-80-47 The Histopathology of Legionnaires' Disease Pneumonia.
CDC-78-48 Demonstration of Legionnaires' Disease Agent in Tissue.
CDC-81-49 Automatic Data Processing in Health Relation Laboratories.
CDC-81-50 The Gamma Scintillation Counter. Part I.
CDC-80-52 Examination of Bone Marrow.
CDC-81-53 The Laboratory Diagnosis of Infectious Mononucleosis.
CDC-79-54 Quantifying Oxygen and Carbon dioxide in Blood.
CDC-79-55 Latex Agglutination Test for *Cryptococcus neoformans* Antigen.
CDC-79-56 Differentiation and Characterization of the Clinically Important Aerobic Actinomycetes.
CDC-79-57 Mycology: Preparation and Reading of Direct Smears.
CDC-79-58 Clinical Chemistry Methods. Part I. Selection and Evaluation.
CDC-79-59 Clinical Chemistry Methods. Part II. Implementation and Quality Control.
CDC-79-60 Identification of *Bordetella pertussis*.
CDC-81-61 Chromosomes and Disease. Part I.
CDC-79-62 Platelet Function and the Clinical Laboratory.
CDC-79-63 Handling and Storing Chemicals Safely.
CDC-79-64 Collection and Preparation of Specimens of Fungal Isolation.
CDC-79-65 Isolation Media Used in Recovering Systemic Mycotic Agents from Clinical Specimens.
CDC-79-66 Amebiasis.
CDC-79-69 Rapid Laboratory Diagnosis of Viral Diseases by Immunofluorescence.
CDC-79-70 Basic Clinical Microbiology. Part I. Host-Parasite Relationships
CDC-79-71 Basic Clinical Microbiology. Part II. Sterilization and Disinfection.
CDC-79-72 Principles of Enzyme Immunoassay.
CDC-81-74 Certification of Health Personnel by Professional Organizations.
CDC-80-76 The Human Complement System. Part I.
CDC-81-78 The VDRL Test. Part I.
CDC-81-79 The VDRL Test. Part II.
CDC-80-80 The Fluorescent Microscope.
CDC-80-81 Isolation and Identification of Streptococci. Part IV.
CDC-81-82 The Human Complement System. Part III. The Role of Complement in Human Disease and Host Defence Against Infection
CDC-82-83 Electrophoresis, Principles and Aplication. Part I. Principles and Theory of Electrophoresis.
CDC-80-87 Standardization of Erythrocyte Suspensions. Part I.
CDC-80-88 Standardization of Erythrocyte Suspensions. Part II.
CDC-80-89 Identification of Some Dermatophytes.
CDC-80-90 Identification of Some Primary Systemic Mycotic Agents
CDC-81-91 Rapid Identification of Genital Herpes virus.
CDC-80-92 Microhemagglutination Assay Methods in the Diagnosis of Syphilis.
CDC-80-93 An Introduction to HLA Typing.
CDC-80-94 Basic Clinical Microbiology. Part III. Specimen Collection and Handling. a. An Overview.
CDC-80-95 Basic Clincial Microbiology. Part III. Specimen Collection and Handling. b. Selection, Collection and Transport of Bacteriological Specimens.
CDC-80-96 Basic Clinical Microbiology. Part III. Specimen Collection and Handling. c. Processing Clinical Specimens in the Bacteriology Laboratory
CDC-80-97 Laboratory Use of Radioactive Material. Part I.
CDC-80-98 The Human Complement System. Part II.
CDC-80-99 Antimicrobial Susceptibility Testing of Anaerobic Bacteriology.
CDC-80-100 Laboratory Investigation of Transfusion Reactions.
CDC-80-101 Alcohol. Part I. Pharmacology.
CDC-80-102 Alcohol. Part II. Laboratory Methods.
CDC-80-103 Laboratory Methods for *Campylobacter fetus ssp jejuni*.
CDC-80-104 Analytical Methods in Laboratory Management. Part I.
CDC-80-105 Nosocomial Infections. An Overview with Perspectives on Disinfection and Environment Control
CDC-81-106 Analytical Methods in Laboratory Management. Part II (a)
CDC-81-107 Analytical Methods in Laboratory Management. Part II (b)
CDC-81-108 Polychlorinated Biphenyl Determination at Parts-Per-Billion Level in Serum.
CDC-80-109 Isolation of *Chlamydia trachomatis* in Cell Culture.
CDC-80-110 Specimen Collection for PKU and Hypothyroidism Screening.
CDC-81-111 The VDRL Slide Test for Syphilis. Part IV. The Qualitative and Quantitative Procedure on Serum.
CDC-81-112 Rocky Mountain Spotted Fever. A Continuing Health Problem
CDC-81-113 Immunohaematology Techniques: Part I. The Direct Antiglobulin Test.
CDC-81-114 The VDRL Slide Test for Syphilis. Part IV. The Qualitative Test on Spinal Fluid.
CDC-81-118 Laboratory Use of Radioactive Material. Part III. Radiation Contamination Surveillance.
CDC-81-124 Preservation of Cells in the Frozen State.
CDC-81-125 Reconstitution of Frozen Cells.

FOR SALE OR SWAP

TECHNICON BLOCK DIGESTOR BD 20/BD40

The Auckland Regional Authority Water Laboratory wants to sell its BD20 unit to buy a BD40. A straight swap would be ideal. Persons interested in buying a BD20, or selling a BD40, or swapping a BD40 for a BD20 should write to ARA Water Laboratory, 1 Wairua Road, Titirangi, Auckland, or **Circle 19 on the Readers Reply Card**

BOOK REVIEW

History of Staining — 3rd Edition
George Clark, Ph.D., Frederick H. Kasten, Ph.D. Published by Williams & Wilkins, Baltimore/London. N.Z. Distributors: Australia and New Zealand Book Co. Pty Ltd, P.O. Box 33-406, Auckland 9. Price $80-95

The third edition of the "History of Staining", though compiled by George Clark and Frederick Kasten, owes much of its content to the efforts of Drs Conn and Lillie. As the title suggests the book is a historical account which outlines not only the development of stains and their uses but who it was that contributed to these advances.

The first chapter honours the work of Dr Lillie, an eminent histotechnologist and histochemist who contributed much to the technique of staining procedures and also the formulation of various stains.

This is followed by a look at many of the pioneers in this field and it is here where the style of the book is set. Historical text often gives the reader little respite from endless attention to detail which is required when recording past events. The authors try to overcome this problem by giving, where possible, an insight into the character and idiosyncrasies of the various people involved in this field of science.

The ensuing chapters alternate between milestones in the development of staining such as the production and use of cochineal, logwood and aniline dyes, and the biographies of those responsible for such developments i.e. Joseph von Gerlach, Paul Mayer, Paul Ehrlich and Gustave Mann. I found this a somewhat disjointed approach to the subject with much repetition of material evident.

The last two chapters are the most extensive and detailed of the whole book. They cover the emergence of fluorescence microscopy and then protein and nucleic acid histochemistry. To any student involved with these subjects they would find much useful and interesting background information.

In conclusion I felt that the reading of this book was like meandering through a museum. One finds some exhibits of particular interest while others require only a polite passing glance. Still they are all of importance and it is only by having an awareness of what has gone before that future developments can be planned with wisdom and skill.
B. L. Dove

Answers to Biochemical Calculations — Section I: Units

1. 1000 mm.
2. 1000 g.
3. 10 mm.
4. 1000 mg.
5. 1000 m.
6. 10^6 μg.
7. 1000 mmol.
8. 10^6 μmol.
9. 1000 ml.
10. 100 ml.
11. 1000 l.
12. 10^9 nmol.
13. 1000 pmol.
14. 0.001 g.
15. 0.001 mol.
16. 60 μmol/l.
17. 450 μmol/l.
18. 0.120 mmol/l.
19. 0.250 mol/l.
20. 6.220 mmol/d.
21. 0.025 g/d.
22. 150 g/l.
23. 167 pkat.
24. 100 g/m^3.
25. 555 kBq.
26. 60 U/l.
27. 750 nkat/l.
28. 5.3 kPa.
29. 10 mg/l.
30. 84 fl.

LETTERS TO THE EDITOR

Sodium and Potassium Estimations on Blood Gas Samples.
Dear Sir,

We were interested to read Bevan Hokin's technical communication, "Electrolyte Determination on Blood Gas Sample Plasma" in Vol 37 No. 5. We concur with his findings but would like to point out that reliable alternatives are available.

This company, through it's New Zealand distributor Thomas Hyde Ltd, will shortly market arterial samplers utilizing dried Lithium Heparin for the measurement of Sodium and Potassium and the simultaneous analysis of blood gas parameters.

Full technical information, as well as papers relating to the dilution effects of liquid heparin may be obtained from our distributors.
Yours faithfully

John F. Rees
Regional Manager, Australasia
Corning Medical Ltd.
Melbourne.

Staffing Survey
Dear Sir,

The only disturbing feature of the annual staffing survey 1983 is the possibility that the results may be used by laboratories to alter technologist training programmes, as is happening in Christchurch.

Before we all succumb to the prevalent delusion that in a few years time the dole queues will be swelled by the inclusion of thousands of destitute and redundant technologists, members should be reacquainted with a similar survey capably conducted by D. G. Bolitho in 1973 and published in the NZIMLT Newsletter May 1974.

After drawing attention to the probability of 38 qualified technologists being without jobs in 1974, the writer goes on to say: "The longer term situation is even worse; assuming that only 60% of those potentially qualifying in 1974-75 actually do qualify then 123 ± 38, 161 qualified

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Chest — type medical freezer with alarm system and recording thermometer.

Extra power compact chest type freezer designed for applications to basic research and for practical use at medical institutes and laboratories. Ridgid polyurethane foam efficiently insulates the useable 227 litre capacity storage space to keep it below -30°C. Also equipped with battery operated temperature rise alarm system, automatic temperature recording thermometer and eight storage baskets.

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- Auto temperature recorder
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technologists will be without posts at the end of 1975, assuming establishments are not increased and that wastage of existing technologists is nil''.

The only conclusion worth drawing from these exercises of this type is that ''The dogs bark, but the caravan moves on''.

In my opinion a more fruitful exercise would be to establish why no new positions are being established: exactly who, in the flourishing hierarchy of medical technology is actually doing the routine diagnostic bench work, and exactly how much longer working scientific professionals can be expected to tolerate a situation in which the demands of increasing workload and expanding technology are not matched by an increase in working medical technologists.

Yours sincerely,
John Aitken
Technologist
Princess Margaret Hospital Christchurch.

OBITUARY

Burnett (Burnie) Stevenson

Burnie Stevenson died suddenly at Geelong, Victoria on 26th January 1984, aged 32 years.

Burnie commenced as a Trainee at the Taranaki Base Hospital, New Plymouth in January 1969. He passed the Basic Training Examination in 1971, Microbiology Part II in 1972 and completed his C.O.P. with Haematology/Immunohaematology Part II in 1973. He successfully passed Microbiology Part III in 1974.

In 1975 Burnie went to Australia where he spent a short time at Mildura Base Hospital and then 3 years at Benalla Hospital as the Senior Technologist. Burnie then moved to the Geelong Hospital in 1978 as the Senior Medical Laboratory Scientist in the Division of Haematology.

Burnie was well respected professionally and well liked socially. He will be sadly missed by his many friends throughout the hospital and our deepest sympathy goes out to his wife Wendy, and children Ryan and Paula.

G. M.
R.E.O.

NEW PRODUCTS AND SERVICES

ABBOTT TDx SYSTEM TECHNOLOGIES

Abbott's TDx employs flourescence polarization which has long been regarded as one of the most accurate and sensitive measurement technologies available. The amount of drug in a patient's specimen is determined by the application of fluorescence polarization technology using direct competitive binding immunoassay techniques.

When a fluorophore is illuminated with plane polarized light, molecules will absorb the light. When the excited fluorophore returns to the ground state, it will emit plane polarized light. If the molecule moves during its excited state lifetime, the emitted fluorescence will be less polarized.

The TDx uses fluorescein labelled drugs as a tracer. This fluorescein labelled tracer absorbs polarized light and emits polarized light proportional to the apparent size of the molecule. Both patient drug and the tracer drug compete for binding sites on the antibody. Those that are bound slow their rate of rotation to correspond with that of the antibody. Because it takes more time to rotate, the polarization reading is high, indicating only a small amount of patient drug is in the solution. Thus the concentration of the patients drug is inversely proportional to the degree of polarization.

The precise relationship between polarization and the concentration of drug in the patient's specimen is calculated by the TDx microprocessor.

The Abbott TDx also employs Radiative Energy Attenuation, an innovative application of one of the fundamental principles of Clinical Chemistry: Beer's Law. The application of TDx REA Technology offers the same wide range of analyte capabilities as conventional absorbance methodologies.

With REA technology, the further light travels through a chromogenic solution containing fluorescein, the more it becomes attenuated. As an example, in a sample containing a low concentration of analyte, light will penetrate further with little attenuation.

In contrast, in a sample containing a high concentration of analyte, there is a high degree of attenuation with TDx REA Technology. There is a direct relationship between attenuation and analyte concentration.

Inside the TDx these principles work as follows: The sample is illuminated with blue excitation light at 485 nanometers. Only the re-emitted green light which has not been attenuated by the chromogen is detected by the instrument at 525 nanometers.

For further information contact Abbott Diagnostics, P.O. Box 40640, Upper Hutt or **Circle 29 on the Readers reply card.**

MONOCLONAL ANTIBODIES INC. RELEASE PREGNASTICK™ URINE hCG KIT — A DIAGNOSTICK™ KIT FOR PREGNANCY TESTING.

Combining a sensitivity of 175 IU/l of hCG, with 100% accuracy in clinical trials, the PregnaSTICK Urine hCG Kit provides reliable detection of pregnancy as early as the first missed menses. Monoclonal antibody specific for beta-hCG subunit and for beta-hCG substantially eliminates the common problem of cross reactivity with urinary LH, FSH and TSH. In this assay, hCG in a specimen is captured between two antibodies; consequently, interference from hCG fragments, protein and haemoglobin is substantially eliminated or minimized.

This exciting new qualitative assay offers procedural simplicity not commonly available in such a sensitive and specific test. It requires no special training, instrumentation or radioisotopes and all reagents are measured with droppers provided with the kit. In just 20 minutes, an easy-to-read colour change provides the answer that you and your patient need.

For further information contact Gibco N.Z. Ltd., P.O. Box 12-502, Penrose, Auckland or phone 593-024, Auckland, or **Circle 41 on the Readers reply card.**

ORTHO DIAGNOSTIC SYSTEMS* KOAGULAB* 40A AUTOMATED COAGULATION SYSTEM

The only computer-controlled coagulation analyzer that performs PT's and APTT's in random order ... directly from collection tubes.

Automated sample pipetting. Only the KoaguLab 40-A automatically pipettes plasma directly from centrifuged blood collection tubes. This feature improves the sample identification system and reduces time and disposables cost.

Random testing flexiblity. PT's and APTT's can be performed in random order ... up to 40 patient plasmas per run, in duplicate. STAT tests of either type are performed rapidly without interrupting pre-set incubation times.

On-board QC program. The KoaguLab 40-A computer provides a statistical quality control program for data analysis and storage. The computer also performs operational functions such as pump calibration, temperature monitoring, and imprecision alert.

For further information, contact Ortho Diagnostic Systems, Phone (09) 543-755, Auckland, or **Circle 25 on the readers reply card.**

ORTHO* ACTIVATED PTT REAGENT

This new product from ORTHO is the first APTT Reagent that is sensitive to in vitro Heparin and factor deficiencies, and at the same time, will precisely define the normal patient range.

ORTHO Activated PTT Reagent is sensitive to deficiences in Factors V, VIII, IX, X, XI, XII, and Fletcher Factor, easily discriminating abnormals from normals, and shows a linear response to Heparin.

This reagent is also extremely stable: on opening, 30 days at 2-8°C or 1 working day at 37°C, single lots are available.

For further information, please contact Ortho Diagnostic Systems, Phone (09) 543-755, Auckland, or **Circle 26 on the readers reply card.**

ORTHO* HBsAg ELISA TEST SYSTEM

ORTHO HBsAg Elisa Test System is a Third Generation test with a sensitivity comparable to radio-immunoassay, but does not require expensive instrumentation for reading, (read visually or in a spectrophotometer).

The method is simple, quantitative, accurate and reproducible, with stable reagents.

For further information, please contact Ortho Diagnostic Systems, Phone (09) 543-755, Auckland or **Circle 27 on the readers reply card.**

ORTHO* RUBELLA ELISA TEST SYSTEM

The complete, standardized test system offers:
Accuracy: linear correlation with hemagglutination inhibition results.
Sensitivity and Specificity: using purified antigen preparation and a monoclonal antihuman IgG conjugate.
Standardized Test System: employs matched components for consistent sensitivity and specificity.

Simplified Procedure: no dispensing or special handling of antigen-coated solid base; no serum preabsorption: nonspecific inhibitors and hemagglutinins do not interfere with the test; photometric end point: for easy-to-read, objective results. Rapid: results in 2 hours with 30 minutes hands-on time.

For more information, contact Ortho Diagnostic Systems, Phone (09) 543-755, Auckland, or **Circle 30 on the readers reply card.**

ORTHO* PAP-IA

ORTHO PAP-IA combines the efficiencies of immunoassay and endogenous enzymatic measurement in a simple spectrophotometric method. The PAP is precipitated by a rapid double antibody technique and acid phosphatase activity of the precipitate is measured with alphanaphthylphosphate, the most sensitive substrate with high specificity for prostatic acid phosphatase. Unlike competitive binding assays, all of the PAP is measured, therefore the standard curve is linear and only a single calibration point is required. With antibody incubations completed in less than fifteen minutes at room temperature, the time to generate results is less than 90 minutes. A robust assay, ORTHO PAP-IA does not require duplicate tests to provide accurate results, reducing time and cost.

For more information, contact Ortho Diagnostic Systems, Phone (09) 543-755, Auckland, or **Circle 28 on the readers reply card.**

LABORATORY SEALING FILM INTRODUCED

Recently introduced to the New Zealand market is an elastic laboratory film called 'Whatman Laboratory Sealing Film'. It moulds and seals itself around culture tubes, flasks, broth tubes, petri dishes and many other instruments and objects, even with irregular surfaces.

Whatman Laboratory Sealing Film is:-

— Heat resistant up to 60°C
— Flexible
— Moldable
— Moisture Proof
— Self Sealing
— Odourless
— Semi transparent
— Thermoplastic

Its high tensile strength (elongation tests showed higher tensile strength against a competitive brand) coupled with its high chemical resistance against common laboratory chemicals such as Sodium Hydroxide, Hydrochloric Acid, Sulphuric Acid, Chloroform and Carbon Tetrachloride, make it ideal for many important laboratory uses and techniques.

A free sample of Whatman Laboratory Sealing Film is offered by the New Zealand agents, Kempthorne Medical Supplies Limited, P.O. Box 1234, Auckland or **Circle 23 on the readers reply card.**

NEW BENCHTOP GC/MS OFFERS BIG-SYSTEM VERSATILITY AND SOPHISTICATED DATA HANDLING

More versatile, faster and even better for capillary GC/MS than its predecessors — that describes the new HP 5995C benchtop GC/MS from Hewlett-Packard Company.

The HP 5995 series, begun by HP in 1979, was the first to offer the chromatographer a benchtop GC/MS at a moderate price. HP 5995s are widely used in chromatography laboratories to help identify compounds in applications such as food, drug, pesticide and petrochemical analysis.

The biggest difference in the new HP 5995C is a high-performance GC/MS workstation that provides data handling and automation. Its interactive CRT simplifies and speeds operation.

When one builds a method, the CRT presents a form. The operator follows the cursor and fills in the blanks; softkeys permit single-keystroke commands. User-built methods automatically can control the entire system, including an optional HP 7672 automatic sampler, in unattended analysis of up to 99 samples from injection through final report.

Other system features such as AUTOTUNE, diagnostics and automatic library search help simplify operation.

New interactive data editing provides capabilities previously available only with bigger and more expensive systems. After a run, using the data-editing features, one can manipulate and display data in a variety of ways that help identify unknowns and enhance presentation.

For example, a portion of a total ion chromatogram can be expanded instantly and displayed in a window on the CRT. The user can overlay spectra and subtract one spectrum from another. Any data on the CRT can be sent to the printer for a hard copy.

There are three standard report formats to choose from: short, long or extended. In addition, if a custom presentation is needed, VisiCalc® can be used. It permits organization of MSD data in spreadsheet or column formats.

With its built-in RS-232 and HP-IB interfaces and optional terminal-emulation capability, the GC/MS workstation is a gateway to a minicomputer such as the HP 1000 where data can be sent for further handling or storage.

The HP 5995C is a completely integrated system with both modules — GC/MS and GC/MS workstation — manufactured and fully supported by Hewlett-Packard.

The proven core gas-chromatograph/mass-spectrometer module is available otimized for capillary or packed-column operation. For superior separation and detection, all temperature zones along the sample path are independently controlled: injector, column oven, transfer line, ion source and quadrupole.

Core-versatility options include:

- a direct insertion probe for solid samples;
- a choice of capillary interfaces, direct or open-split;
- a choice of separators for packed column operation;
- a stand-alone vacuum-pressure monitor; and
- an FID detector with effluent splitter for simultaneous FID/MS detection in packed-column operation.

Three GC/MS workstation modules are available. Each includes computer, disc storage and printer. The standard workstation employs the HP 9000 Series 200 Model 216 (previously HP 9816) computer with black-and-white CRT. The two other GC/MS workstations use the full-color HP 9000 Series 200 Model 236 (previously HP 9836) computer.

When it's not doing GC/MS, the workstation can serve as a high-performance HP 9000 Series 200 technical personal computer that has over 70 software packages including Context MBA™.

For further information, contact the New Zealand distributors for Hewlett Packard:
Northrop Instruments & Systems Limited
P.O. Box 2406, Wellington, Telephone 850-091
P.O. Box 8602, Auckland, Telephone 794-091
P.O. Box 8388, Christchurch, Telephone 488-874
or **Circle 22 on the readers reply card.**

NEW MASS-SELECTIVE DETECTOR INTRODUCED WITH ADVANCED DATA-HANDLING AND AUTOMATION CAPABILITIES

A new mass-selective detector (MSD) from Hewlett-Packard Company offers an advanced GC/MS workstation for ease of use, data handling and automation.

Optimized for capillary gas chromatography, the HP 5970B MSD is both specific and universal. More than any other type of GC detector, it can help the chromatographer make positive, unambiguous compound identification.

This makes the HP 5970B useful in such areas as methods development for target compound identification, drug analysis and identification in overdose cases. It also aids in analysis of pesticides, such as EDB, where it provides specificity comparable to or better than electron capture, nitrogen-phosphorus and flame-photometric detectors.

The MSD produces mass-spectra and ion chromatograms of components eluting from a capillary gas chromatograph. The spectra are stored and comparison with reference spectra, through automated library search, can expedite positive compound identification.

In its selected ion-monitoring (SIM) mode, the MSD selectively can monitor up to 10 groups of 20 ions each with high sensitivity. The user can time-program to monitor different groups at different times during a run. SIM permits analysis for selected compounds at nanogram to picogram levels in very complex mixtures.

The new MSD is supplied in two modules: core and GC/MS workstation. The core-detector module is available in configurations designed to interface with most capillary GCs. This module houses the hyperbolic quadrupole mass analyzer, vacuum system and related hardware. It is easy to maintain and requires no special utilities.

The GC/MS workstation module consists of a computer, disc storage and printer/plotter. It can be the standard GC/MS workstation with black and white CRT, or either of two full-color workstations. All three provide high-speed data acquisition in scanning and SIM modes, with scan rates higher than 1500 atomic mass units per second (amu).

To increase sample throughput, the GC/MS workstation also can control an HP 5890 or HP 5790 gas chromatograph with an HP automatic sampler for unattended analysis of up to 99 samples.

When one builds a method, the interactive CRT presents a form to fill out and leads one through it step by step. Softkeys — up to 10 for each display — permit single-keystroke control of many operations. If the

New, from the leading name in Clinical Instrumentation:

"RADIOMETER"

ABL4 ACID-BASE LABORATORY

Potassium and Blood Gases in less than two minutes

The ABL4 has been designed to meet the ultimate demands in Cardiac Surgery Departments and ICUs.
It will also greatly improve the capability of any stat lab where safe and uncomplicated K^+/ blood gas analyses are required 24 hours a day. Features of the new ABL4 include:

- measurement of K^+, pH, P_{CO2}, P_{O2}, Hb and Bar.
- fully automatic calibration
- built-in-gas mixer requiring only 100% CO_2
- low maintenance commitment
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ABL4 joins the ABL bloodgas family viz:
ABL30 — Low cost-automated
ABL3 — Automated, CRT Display, keyboard control

KNA1 SODIUM-POTASSIUM ANALYSER

Fail-safe STAT analysis

With the KNA1 STAT means much more than just "prompt processing". Thus, the analyzer fulfils the most stringent demands of any hospital ward — in the clinical chemistry lab as well as in all wards responsible for critical care.

- data out in 1 minute — on 125 μl of whole blood, plasma or serum
- ONE sample mode — no risk of losing a sample
- fail-safe operation — no manual intervention
- identical values from whole blood and plasma
- identity to flame-based values for normal plasma. No need for an ISE/Flame mode switch — in accordance with IFCC
- misinterpretations avoided
- dedicated K^+/Na^+ QUALICHECK® plus built-in computer control yielding full quality assurance

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ICA113 Ionised calcium system, fully auto mated, multi-channel.

CLINITUBES High anticoagulant effect and low heparin interference are the alpha and omega of heparinization. Clinitubes fulfil these demands to the letter.

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FOR FURTHER INFORMATION PLEASE CONTACT

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user desires, forms and prompts can be overridden, building methods to meet special needs.

Using the built-in data-editing features after a run, one can edit and display data in a variety of ways that help identify unknowns and enhance presentation of results. For example, a portion of an ion chromatogram can be expanded and displayed instantly in a window on the CRT. One can overlay spectra, subtract one spectrum from another and send any data on the CRT to the printer for hardcopy. Data can be plotted, tabulated and searched against library spectra.

There are three standard report formats to choose from: short, long or extended. In addition, if a unique format is needed, VisiCalc® can be used to organize MSD data in spreadsheet or column formats.

With its built-in RS-232 and HP-IB interfaces and optional terminal-emulation capability, the GC/MS workstation is a gateway to a minicomputer, such as the HP 1000 or 3000, for further data handling.

When it's not doing GC/MS, the workstation can serve as a high-performance HP 9000 Series 200 technical personal computer that has over 70 software packages.

Specifications for the HP 5970B MSD unit include operation on 120 or 240 volts, a mass range of 10-800, a mass-scanning rate greater than 1500 amu/sec and a five-decade dynamic range.

For further information, contact the New Zealand distributors for Hewlett Packard:
Northrop Instruments & Systems Limited
P.O. Box 2406, Wellington, Telephone 850-091
P.O. Box 8602, Auckland, Telephone 794-091
P.O. Box 8388, Christchurch, Telephone 488-874.
or **Circle 24 on the readers reply card.**

NEW CENTRIFUGE TUBE IS SAFER AND EASIER TO READ

Elkay's new 15ml capacity centrifuge tube with screwcap offers laboratories two important benefits.

First, the tube comes complete with a close threaded screwcap which has an inert polyfoam disc as a liner. This provides a leaktite seal when the screwcap is tightened. This safety feature is especially valuable when handling hazardous or pathogenic specimens.

Second, bold easy-to-read graduations are molded into the virgin polystyrene tube at .1ml intervals to 1ml and .5ml intervals thereafter to 15ml. These graduations enable the user to identify sample volume quickly and accurately.

An alternative version of this centrifuge tube without threads and screwcap has also been introduced for use in routine centrifugation procedures.

Elkay Products, Inc. manufactures a comprehensive range of precision molded sterile and non-sterile centrifuge, microcentrifuge and test tubes for scientific, medical, industrial and research laboratories.

For further information contact Medic DDS P.O. Box 205, Wellington or **Circle 21 on the readers reply card.**

ELKAY OFFERS ASTRA USERS SAVINGS UP TO 30%

Elkay Products, Inc. now have a comprehensive line of consumables for use with Beckman Astra analyzers.

These include precision molded pump head tubing, virgin polystyrene sample cups with polyethylene press overfit caps and high resolution trace thermal printer paper.

As an original consumables manufacturer, Elkay is able to offer Beckman Astra users savings of up to 30% in their consumables costs.

In common with all Elkay precision laboratory consumables, these products are guaranteed in writing to equal or exceed the performance of any comparable products.

'Beckman' and 'Astra' are trademarks of Beckman Instruments.
For further information contact Medic DDS P.O. Box 205, Wellington or **Circle 20 on the readers reply card.**

WORK WANTED

Bachelor of Laboratory Technology graduate, seeks appointment as Junior Technician. Will consider any area of medical laboratory science. CV on request.
Please reply to: D. Chiang, 4/F 178 Junction Rd. Kowloon, Hong Kong.

A recently qualified Registered Technologist (general) seeks a position in Medical Laboratory Technology. I am registered with the Canadian Society of Laboratory Technologists and my qualifications have been ratified by the MLTB. I have a Bachelor of Education and Bachelor of Science (Honours) degrees. I was employed part-time in Microbiology and Blood Bank during my training year.

I require a position involving, or related to, one of the following areas: Clinical Chemistry, Haematology, Histotechnology, Immunohaematology or Microbiology.
Replies to: June Davison, 60 Clover Ridge Drive West, Ajax, Ontario L15 3K1, Canada.

A technologist with sixteen years experience in a medical laboratory, the majority of which was in clinical chemistry, seeks a position in Medical Laboratory Technology. My qualifications are the Higher National Certificate in Medical Laboratory Science, Fellowship of the I.M.L.S. in Clinical Chemistry, Certificate in Medical Laboratory Management and Certificate in Medical Information Processing. I am familiar with all forms of routine clinical chemistry investigations and have wide experience of automated analysis and radioimmunoassay work. I am eligible for registration in clinical chemistry.
Replies to: Mr C. D. Jaggs, 13 Bradbury Rd, Howick, Auckland.

SITUATIONS VACANT

MEDICAL TECHNOLOGIST WANTED FOR CHRISTIAN MISSION IN PAPUA NEW GUINEA

The Asia Pacific Christian Mission, an inter-denominational organisation, has a vacancy for a Medical Technologist to be in charge of the laboratory at its rural health centre at Balimo in the Western Province of Papua New Guinea. The health centre has a daily in-patient average of 100, conducts 250-300 confinements annually and has TB, leprosy and malaria case detection, nutrition improvement and general nurse training programs. It is responsible for the health care of 20,000 people.

A new laboratory has just been completed and duties include supervision of a malaria microscopist and a medical laboratory assistant, both Papua New Guineans. The major part of the work done now is haematological together with micro-biological examination of urines, stools, sputums, skin smears and blood films. However, there is scope for developing a bacteriological culturing and identification facility, biochemistry (currently confined to blood urea and glucose) and research interests.

The main duties of the medical technologist in this laboratory are supervision of other staff, maintenance of high standards, setting up and running of unusual tests and maintenance of records and stocks. The person currently holding the position completes his term of service in May, 1984, and so a replacement is needed urgently.

Financial remuneration is through a system of team support, involving the Christian community at levels set by the mission. Periods of service of one year or longer will be considered.

Further information may be obtained from: **The N.Z. Administrator, A.P.C.M., 427 Queen Street, Auckland. Telephone 790-034.**

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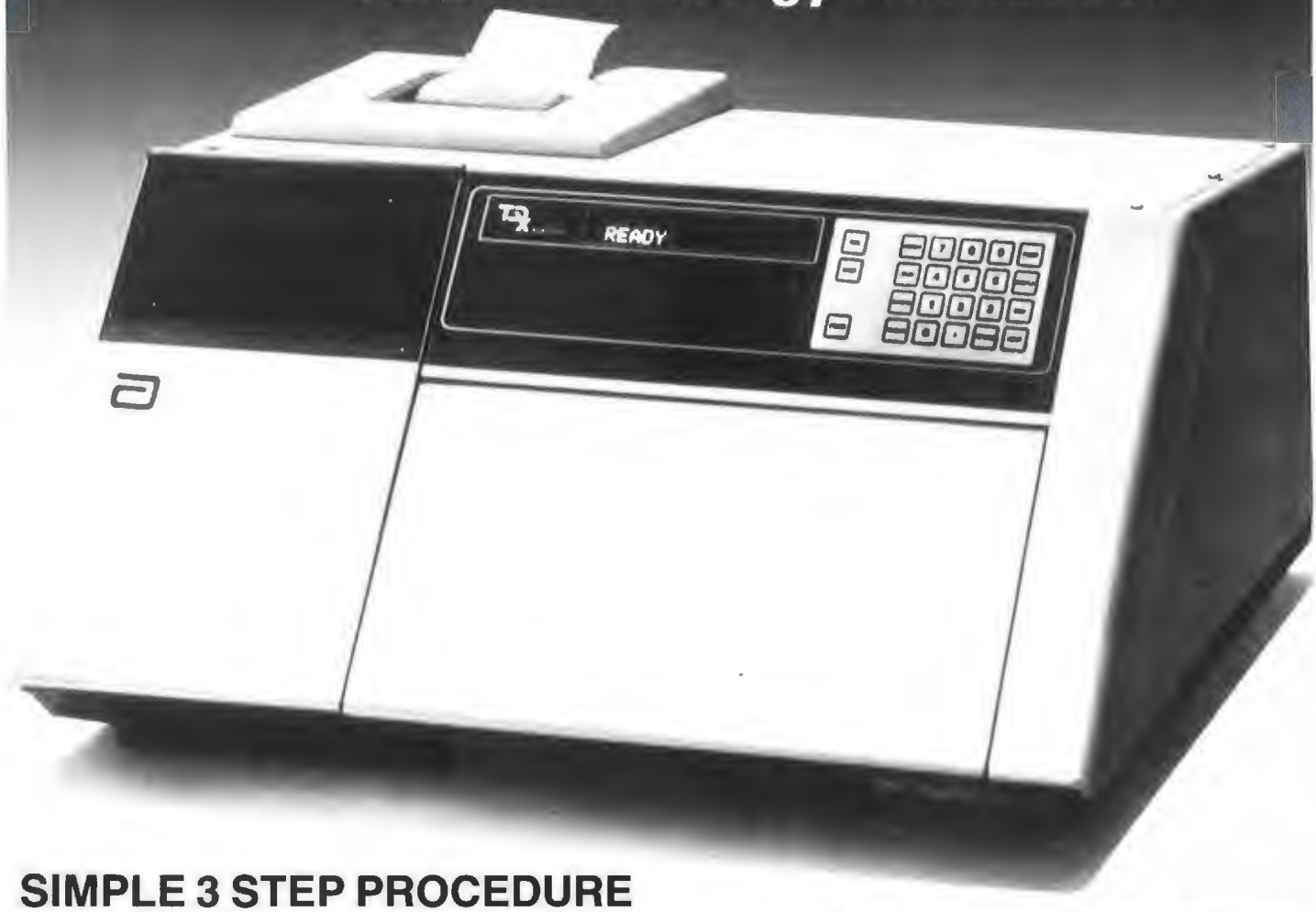
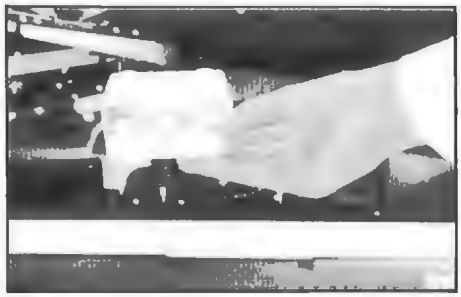
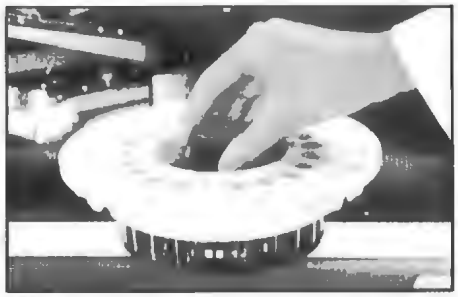


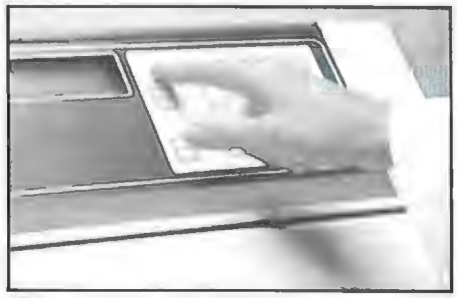
SIMPLE 3 STEP PROCEDURE



1 Insert reagent pack



2 Place carousel in instrument



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