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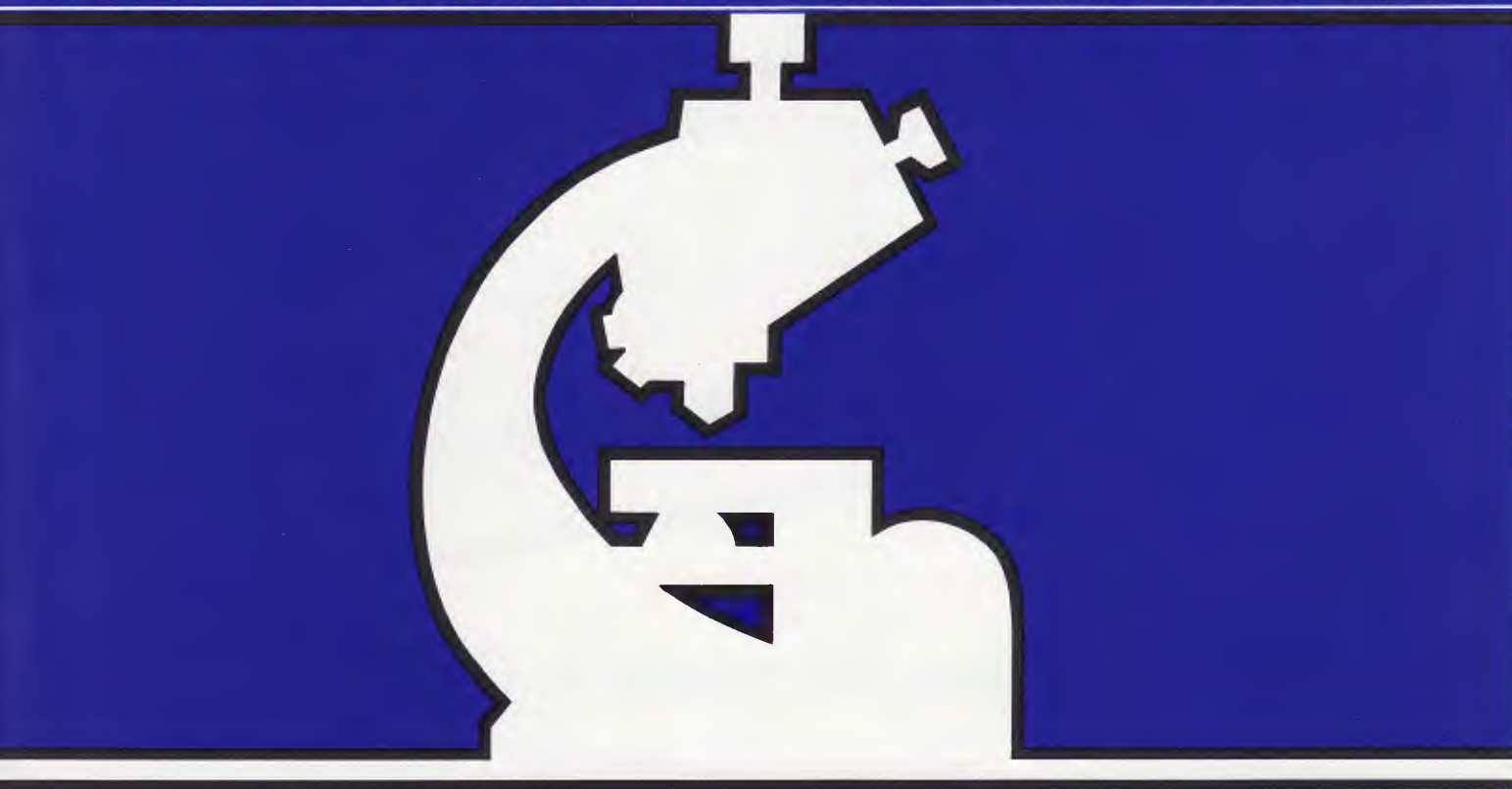

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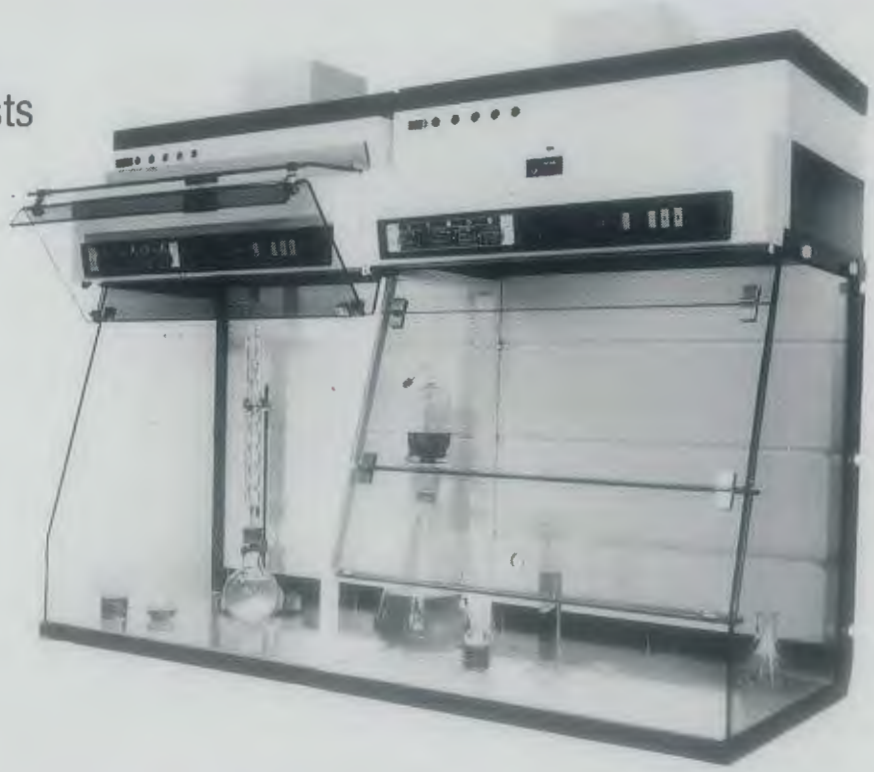

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THE NEW ZEALAND JOURNAL OF
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Filtration of Platelet Concentrates

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Abstract:

Using blood filtered infusion sets produced by 3 manufacturers no significant loss of platelets was observed with passage through the filter. Commonly used filtered blood infusion sets can be used for platelet concentrate infusion.

Key Words:

Platelets, Infusion sets, platelet concentrate.

Introduction:

Debate still exists as to whether filters should be used when transfusing platelet concentrates. The Australian Society of Blood Transfusion[1] states that "conventional blood giving sets have filters which may interfere with platelet transfusion and special platelet infusion sets may be required". However, Arora and Morse[2]in experiments using three different conventional blood transfusion sets found that platelets were not lost during the filtration process.

In New Zealand, platelet concentrates are administered in several ways. Some hospitals do not use filtration, others use modified filtered transfusion sets, while some utilise normal blood transfusion giving sets.

To resolve these different approaches, we have investigated the effect of commonly available filtered blood infusion sets on platelet numbers in platelet concentrates before and after filtration.

Materials and Methods:

Platelet concentrates were prepared from routine units of blood, each containing approximately 430mls of blood and 63mls of CPD anticoagulant, collected in a triple blood pack (Tuta 04-001).

Blood units were centrifuged at 20°C in MSE Coolspin centrifuge for 3 minutes at 1900 G and the platelet rich plasma (PRP) expressed into a satellite pack using a plasma extractor. The PRP was centrifuged at 20°C for 15 minutes at 3000 G and the supernatant platelet poor plasma expressed into the second satellite pack leaving 50-60 ml of plasma and the platelet concentrate (PC) in the primary pack. This pack was left undisturbed at room temperature (20 — 22°C) for 1 hour and then rotated continuously at 5 rpm on an elliptical platelet rotator (Fenwal 4R 4050) at a controlled room temperature of 22°C ± 2 for a maximum of 3 days.

For the platelet filtration experiments, several blood infusion sets were used.

1. Tuta (Cat Nos. 30-000 (D) and 32-001 (C)) blood infusion sets. These are made of medical grade polyvinyl chloride with a monofilament nylon filter of 0.1mm diameter and a mesh pore size of 200μ. Also tested were Tuta 30-500 (E) infusion sets which are recommended for platelet infusion and are modified with a small filter and shortened tubing.
2. Fenwal (Cat. No. 4C-2100) blood infusion sets (B). These are made of medical grade polyvinyl chloride with a nylon 66 filter and a mesh pore size of 170μ.
3. McGaw Ethicals (Cat. No. V2400) blood infusion set (A). These are made of medical grade polyvinyl chloride with a nylon 66 filter and a mesh pore size of 260μ. Also tested was a Haematrol V-1435-60 infusion set (F) with similar specifications.

For the experimental procedure PC suspensions were passed through each infusion set at a rate of approximately 10ml/minute. Six units of PC were passed through each filter consecutively to simulate normal clinical practice. Pre and post filtration platelet counts were obtained on each individual platelet concentrate. Both fresh (1 day old) and expired (over 3 days old) platelet concentrates were tested.

Platelet counts were performed on a Technicon platelet analyser after a 1 in 3 predilution with a 2M Urea.

Results:

The average volume of the platelet concentrate unit was 56.8 ± 11.3ml (1 SD) and the total number of platelet units used was 359.

Pre and post filtraton platelet concentrates did not differ significantly on either fresh or expired concentrates (Table 1) with all filtered infusion sets.

When 6 PC were passed through one filter, there was no signficant difference in the percentage of platelets lost across the filter between the first and last PC (Table 2).

Discussion:

The aim in platelet transfusion is to administer viable active platelets capable of promoting and maintaining haemostasis in patients either thrombocytopenic or with functionally inactive platelets. Because platelet concentrates invariably contain aggregated material and that this can precipitate pulmonary emboli and pyrogenic reactions removal of this material is advantageous. In this study we have attempted only to investigate the likely disadvantages of using commonly available infusion sets for platelet transfusion which will at best only remove macro-aggregrates.

We have been able to demonstrate that the blood infusion sets commonly available in New Zealand are not detrimental to platelet passage and thus can be used for platelet concentrate infusions. These results are not so surprising as the average diameter of a platelet is 2 microns and the pore size of the filters studied ranged from 170 to 260 microns. However it is not only the size difference which ensures no restriction to passage. Platelets when activated aggregate rapidly into micro and macro aggregates and since no reduction occurred across the filters tested we conclude this indicative of the inertness of the filter and tubing material. In a similar study, while investigating the properties of micro-aggregate filters, Snyder et al[3] reported similar findings of insignificant platelet losses during their passage through the microaggregate filters under study. In the same study he noted that platelet function as assessed by aggregometry, biochemical investigation (Beta Thromboglobulin) and cellular morphology was also not significantly affected.

The results of Table 1 are the mean results of all the platelet concentrates passed through a particular brand and type of infusion set. While these results indicate no overall loss across the filters, Table 2 confirms also that there is no accumulated activation during the passage of the six consecutive platelet concentrate units through each filter.

No experiments were undertaken to investigate the effect of pre-loading the infusion set with blood as it is not a recommended practice that infusion of plasma and/or platelets should follow a red blood cell transfusion through the same set. It is useful to also note that in his study Snyder et al[3] found that pre-loading with blood was detrimental to subsequent passage of platelets through the same microaggregate filter. We would be of the impression that a similar result would be obtained with the passage of platelets through a conventional infusion set which had been used for transfusion of red blood cells.

	FRESH PC			EXPIRED PC					
Filter make (see text)	A	B	C	A	B	C	D	E	F
Pre-filtration count (x 10^9/l)	66 ± 20	57 ± 15	58 ± 18	39 ± 11	50 ± 11	40 ± 14	56 ± 15	50 ± 13	38 ± 4
Pre-filtration count (x 10^9/l)	66 ± 21	57 ± 16	57 ± 16	39 ± 11	48 ± 12	39 ± 14	56 ± 14	50 ± 14	39 ± 4
Difference (x 10^9/l)	0	0	1	0	2	1	0	0	-1
Number of filters	10	10	10	2	2	9	9	3	6

Table 1
Effect of filtration or pre and post platelet counts.

	FRESH PC			EXPIRED PC					
Filter make	A	B	C	A	B	C	D	E	F
% Difference between pre and post filtration — 1st unit	3.3	19.0	3.4	2.1	2.0	12.7	3.2	4.0	2.0
% Difference between pre and post filtration — 6th unit	7.8	6.2	3.5	7.8	5.0	5.9	3.2	1.0	2.0
Number of filters tested	10	10	10	2	2	9	9	3	6

Table 2

Effect of filtration on difference between pre and post counts between the first and last platelet concentrate. The percent differences is the mean value of the percentage loss of platelets of each unit of PC passing through each infusion set.

Conclusion:

We could find no detrimental affect on the passage of platelet concentrates through the commonly available blood infusion sets and recommend that to at least assist in the removal of aggregated and other extraneous material filtered infusion sets such as those tested could be used for platelet transfusions. If the platelet transfusion is to follow a red blood cell transfusion then we suggest a new set should be used.

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Lupus Inhibitor: Yes or No?

P. J. Hill, Haematology Department, Middlemore Hospital.

Abstract:

This paper presents a case involving a 70 year old female with Waldenstroms macroglobulinaemia who on histological analysis at post mortem revealed a diffuse malignant lymphoma. This patient exhibited an inhibitor which resembled a lupus type III inhibitor active against Factors VIII, IX, XI and phospholipid. This inhibitor was reduced when the patient was treated by the chemotherapeutic agents Prednisone and Chlorambucil and raised the question, was the inhibitor a true lupus type inhibitor or was it an inhibition of coagulation factors by the presence of macroglobulins?

Key Words:

Lupus inhibitor, coagulation, macroglobulinaemia.

Introduction:

Waldenstroms macroglobulinaemia is a neoplastic proliferation of immature B lymphocytes which retain the capacity to differentiate into large lymphocytes or immature plasma cells and produce large amounts of the monoclonal protein IgM. Similarly lymphocytic lymphomas are neoplasms which originate from lymph nodes, spleen or lymphoreticular tissue, and are composed usually of only one type of malignant cell, the B lymphocyte. A significant number of macroglobulinaemias develop associated malignancies many of them being lymphomas. The opposite sequence of events is also well recognised and monoclonal immunoglobulin components are found frequently in the sera of lymphoma patients (1). Their incidence varies with the type of lymphoma, 14% of diffuse lymphocytic lymphomas have immunoglobulin components, the paraprotein discovered is more frequently IgM than IgG or IgA (2). Whether this patient had, therefore, a diffuse lymphocytic lymphoma producing an associated macroglobulin or a Waldenstroms macroglobulinaemia which became a rapidly advancing lymphoma remains unsolved. What is also a mystery is the unusual coagulation she exhibited.

Methods:

Serum immunoglobulins were quantitated by radial immunodiffusion (R.I.D.) according to the manufacturer's directions (Kallestad).

COAGULATION STUDIES

The activated partial thromboplastin time was performed with 0.1ml plasma, 0.1ml P.T.T. reagent (Dade Actin) and 0.1ml of 0.020M calcium chloride using an incubation of two minutes. Correction studies of patients and normal plasma mixtures were carried out by the above method immediately and after 30 minutes at 37°C.

Coagulation factor assays were performed using factors VIII, IX and XI deficient substrate plasmas (Dade) and were based on the A.P.T.T.

The Kaolin Clotting time (K.C.T.) was performed with 0.2ml platelet poor plasma and 0.1ml Kaolin (20mg/ml) and 0.2ml 0.025M calcium chloride after an incubation of 3 minutes. KCT mixing tests of various patient and normal plasma mixtures were also carried out by this method.

Results:

On admission this markedly anaemic patient exhibited a markedly elevated erythrocyte sedimentation rate (E.S.R.) and abnormal immunoglobulins. Coagulation studies revealed a markedly prolonged activated partial thromboplastin time (A.P.T.T.) (Table 1) and further investigative coagulation studies (Table 2) showed a failure of the A.P.T.T. to correct back using normal plasma indicating the presence of a circulating inhibitor. Specific coagulation tests (Table 3) showed the inhibitor to have inhibitory effects against coagulation factors VIII, IX, XI and phospholipid resembling a lupus type inhibitor. Kaolin Clotting times (K.C.T.) were done on mixtures of patient's and normal plasmas and these results are shown in Table 4.

Discussion

On admission this patient was given a diagnosis of monoclonal

		NORMAL RANGE
IgG	8.2g/l	8.0 — 16.0
IgA	0.7g/l	1.7 — 3.0
IgM	8.2g/l	0.7 — 2.0
Haemoglobin	48 g/l	115 — 164
E.S.R.	Greater than 150 mm/hr	1 — 10
Platelet count	130 x 10^9/l	150 — 400
Prothrombin Ratio	1.2	0.8 — 1.2
A.P.T.T.	65 seconds	25 — 40

TABLE 1. Results of patient on admission.

gammopathy namely macroglobulinaemia, primarily because of the marked increase in the serum IgM concentration and corresponding reduction in IgG and IgA. The elevated E.S.R. also fitted in with a diagnosis of a paraproteinaemia. Because of a bleeding tendency and a subsequent low haemoglobin, a prothrombin ratio and A.P.T.T. were performed to assess the patient's coagulation status. These revealed a markedly prolonged A.P.T.T. of 65 seconds and the need for further investigative coagulation studies.

Bleeding time, prothrombin ratio and thrombin clotting time were all normal. The failure of the A.P.T.T. to correct back upon the addition of normal plasma indicated the presence of a circulating anticoagulant. The pre-incubation of an equal mixture of patients and normal plasma at 37°C did not prolong the A.P.T.T. Rapaport (3) stated that this observation can be used as a single presumptive test to distinguish lupus inhibitors from inhibitors which destroy specific clotting factors, e.g., VIII inhibitors.

		NORMAL RANGE
Bleeding Time (simplate)	5 minutes	1 — 6
Prothrombin Ratio	1.2	0.8 — 1.2
Thrombin Clotting time	6 seconds	< 10
A.P.T.T.	65 seconds	25 — 40
A.P.T.T. (1+1 P/NP)	58 seconds	25 — 40
A.P.T.T. (1+1 P/NP at 37°C for 30)	56 seconds	25 — 40

P = patient N.P. = Normal plasma

TABLE 2: Follow-up Coagulation Studies.

Factor assays indicated the presence of a strong immediate acting inhibitor to coagulation factors VIII, IX and XI. This was shown by the low assay values obtained with undiluted plasma but normal values for these individual factors when the patients plasma was diluted 10-15 fold before testing.

The presence of a normal K.C.T. of 92 seconds, in which there is no phospholipid involved and the prolonged A.P.T.T. which would not correct back suggested the inhibitor also had antiphospholipid activity. This antiphospholipid activity is characteristic of the lupus inhibitor and so K.C.T.'s were performed on mixtures of patients and normal plasma.

The point of interest with these results was that the clotting time

		NORMAL RANGE
A.P.T.T.	65 seconds	25 — 40
P.T.T.K.	96 seconds	29 — 47
K.C.T.	92 seconds	47 — 112

SPECIFIC FACTOR ASSAYS (u/l)

FACTOR	UNDIL.	1 : 10	1 : 15	
VIII	430	1300	N/T	600 — 2000
IX	10	12	675	600 — 2000
XI	84	605	N/T	600 — 2000

N/T = NOT TESTED

TABLE 3: Further Coagulation Tests

% NORMAL PLASMA	% PATIENT PLASMA	K.C.T. (seconds)
100	0	97.0
90	10	133.9
80	20	130.3
50	50	116.5
20	80	104.9
0	100	92.0

TABLE 4: Kaolin Clotting Times.

increased as the percentage of patient's plasma to normal plasma increased but only up to a certain point after which addition of patient's plasma to normal plasma saw a decrease in the K.C.T. Graphing of the results gave the typical representative shape of a lupus type III inhibitor as described by Exner (4) (Fig. 1).

The type III lupus inhibitor shows a more prolonged K.C.T. when patient's plasma is mixed with normal plasma than the K.C.T. of the patient's plasma alone. This effect is thought to be due to the presence of a "lupus co-factor" which when in optimum concentration results in an enhancement of the inhibitors activity, causing a lengthening of the clotting times. This optimum concentration is shown by the peak on the graph and explains the reason why the clotting times decline once this optimum concentration is passed.

The clotting studies had revealed an inhibitor active against multiple coagulation factors and phospholipid which resembled a lupus type III inhibitor. This inhibitor was reduced when the patient was treated with the chemotherapeutic agents Prednisone and Chlorambucil. The reduction of the inhibitors activity coincided with a drop in the level of the serum IgM (Table 5).

DATE	TREATMENT	A.P.T.T. (secs.)	IgM (g/l)
17/11/82		68	8.5
24/11/82	X		
3/12/82		48	4.8
19/12/82	X		
29/12/82			1.1

X = treatment given.

TABLE 5: The effects that the chemotherapeutic agents Prednisone and Chlorambucil had on the IgM concentration and A.P.T.T.

The returning toward normal of the patient's coagulation status when the serum IgM was reduced raised the question: was the coagulation inhibition a result of macroglobulin interference or was it a true lupus type inhibitor that was reduced after treatment with Prednisone? Prednisone is known to depress the action of the inhibitor (4).

The lupus inhibitor appears to block the activation of prothrombin by the prothrombin activator complex of factors Xa, V and phosphlipid (5). It is not unique to patients with S.L.E.; only 5-10% of these patients develop the lupus type inhibitor. In one study of 83 patients with circulating inhibitors, 58 had the lupus type and 50% of these patients did not have S.L.E. but various myeloproliferative disorders and carcinomas (6).

Paraproteins are usually involved in the inhibition of a number of clotting functions. Most commonly they prolong the thrombin clotting time due to interference by the abnormal protein with orderly fibrin monomer polymerisation (Lacher 1970) (7).

However, inhibition of specific factors such as VIII, XI and XII has been documented (8, 9) and two cases of patients who had increased concentrations of IgM in their serum with inhibitors that had phospholipid specificity and so resembled a lupus type (10, 11).

Conclusion

The presence of a blood coagulation inhibitor resembling a lupus type was demonstrated in this patient by the prolonged A.P.T.T. and P.T.T.K., the positive results in the Kaolin Clotting mixing tests, the reduced levels of factors VIII, IX and XI when one stage assays were used but which approached the normal range by reducing the inhibitors activity by diluting it out and the fact that the A.P.T.T. showed no

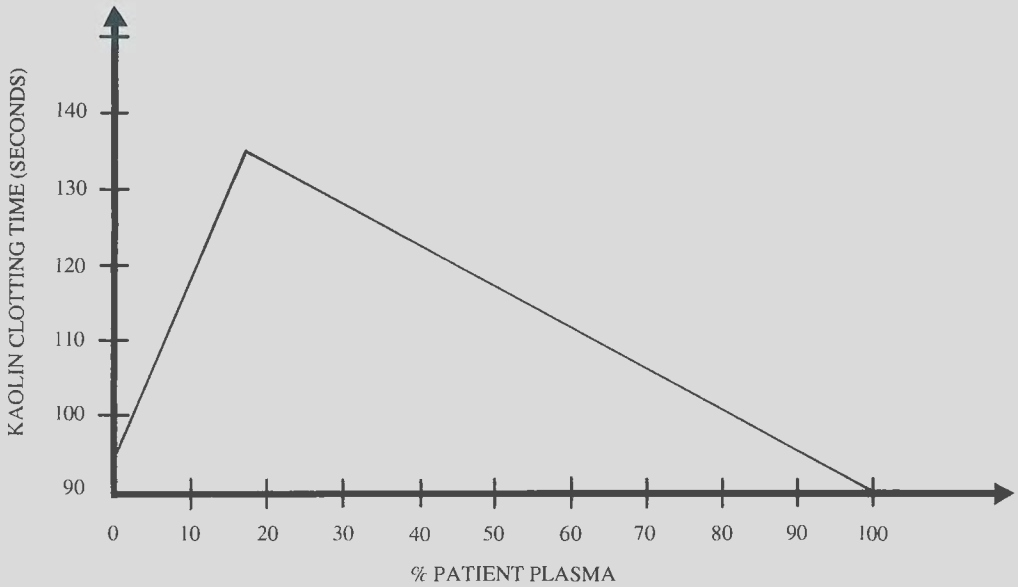

FIG. 1: Kaolin Clotting times of patient and normal plasma mixtures.

change when the patient and normal plasma were pre-incubated for 30 minutes before testing.

Unfortunately we were unable to remove the presence of macroglobulin while the inhibitor was still present and so the question was the inhibitor a true lupus-type anticoagulant or an interference of coagulation by the presence of an abnormally high concentration of immunoglobulin remains unanswered.

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Identification of Monoclonal Immunoglobulins by Immunofixation

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Abstract:

The identification and typing of monoclonal immunoglobulins in this laboratory is carried out by immunofixation on agarose gel. Immunofixation is more sensitive, technically less demanding and less difficult to interpret than immunoelectrophoresis which is the traditional method used for this purpose. The major problem associated with immunofixation is to ensure antigen (Ag) antibody (Ab) equivalence. This often requires multiple specimen dilution. The application of immunofixation to the identification and typing of monoclonal immunoglobulins and its problems are discussed.

Key Words

Immunofixation, immunoelectrophoresis, antibody

Introduction

Immunofixation was originally described by Alper and Johnson[1] as a method for the study of protein polymorphism. Ritchie and Smith[2,3] further described immunofixation on agarose gel and its application to the identification of monoclonal immunoglobulins. Numerous modifications of this method using both agarose gel[4] and cellulose acetate[5] as supporting media have been reported. Immunofixation and

immunoelectrophoresis have been compared in several studies[6,7] and our experience of both methods indicates that immunofixation on agarose gel is superior to immunoelectrophoresis for the identification of monoclonal immunoglobulins.

The purpose of this report is to highlight the advantages and limitations of this technique.

Materials and Methods

Specimens: Serum and urine specimens were obtained from hospital patients for whom either serum protein electrophoresis (SPE) or Bence Jones Protein (BJP) analysis had been requested. Urine specimens were concentrated using a Minicon B15 Concentrating Cell from Amicon Corporation, Danvers Mass., USA to a protein concentration of 25 g/l or 100-fold if this was not possible.

Agarose: Miles Laboratories Pty. Ltd., Goodwood CP 7460, South Africa.

Antisera: Monospecific antisera to immunoglobulins G(IgG), A(IgA), M(IgM), D(IgD) and E(IgE) and light chains kappa and lambda — Dakopaks, Copenhagen, Denmark.

Power Pack: Solsat Industries, Christchurch, New Zealand.

Electrophoresis Equipment: Behringwerke, Marburg, West Germany.

Agarose Bridges: 1.5%(w/v) agarose prepared in SPE buffer according to the method described for the preparation of electrophoresis plates. The under sides of the frame are sealed with pressure tape then filled with agarose. When the agarose is set the bridges are topped up ensuring that there is an even layer of agarose extending from the outside edges of the frame to the inner edges.

Wettex Wicks: Final dimensions 205 x 25 mm. These must be boiled 3 times in distilled water before use to remove any detergent present in the Wettex.

Gel Bond Film: Thickness 7 mil. Sigma Chemical Co., St. Louis, USA.

U-Frame: 205 x 110 x 1 mm.Behringwerke, Marburg, West Germany.

SPE Mask: Marine Colloids, Rockland, Maine, USA.

Tri Partigen IgG, IgA and IgM immunodiffusion plates and IgG, IgA and IgM standards for the quantitation of immunoglobulins: Behringwerke, Marburg, West Germany.

Electrophoresis: This was performed according to the method of Jeppsson et al.[8] with some modifications.

1. SPE Buffer: 0.1 M Tris-Barbital-Sodium Barbital pH 8.6.
2. Fixing Solution: Saturated solution of picric acid in distilled water mixed with glacial acetic acid in the proportion 4:1.
3. Staining and Destaining Solutions: Coomassie Brilliant Blue R.250 Sigma Chemical Co., St. Louis, USA, 7.5 g/l in methanol:acetic acid:water, 9:2:9. The dye is omitted from the destaining solution.
4. Preparation of Electrophoresis Plates:
   (a) Mould: The Gelbond film is cut to give final dimensions of 205 x 115 mm and the hydrophobic side is attached using water to a glass plate (205 x 110 mm). The U-frame is placed on this and overlaid with a second glass plate. The mould is clamped together with Bulldog clips. The Gelbond film overlaps the glass plates by 5 mm forming a lip which aids in pouring the agarose. (b) Agarose: A 1%(w/v) agarose solution in SPE buffer is prepared. The agarose is heated with continuous stirring until boiling to ensure that the agarose is dissolved and that there is no charring. It is then cooled to 60°C and approximately 23 ml is poured into each mould. Any trapped air bubbles must be removed before the agarose sets. The open edge of the moulds are sealed with pressure tape and the plates are stored at 4°C for at least 2 h before use.
5. Sample Application: The glass plate overlying the agarose and the spacer are carefully removed. The Gelbond film is then lifted from the second glass plate. The gel surface is lightly blotted with Whatman 1 paper (240 x 50mm) up to 5 mm from the edge at which the samples are to be applied. The SPE mask is placed in the centre of the blotted strip so that the slits are 30mm from the gel edge.Two μl of undiluted serum or concentrated urine is applied to each slit and allowed to soak in. A Bromphenol Blue-tagged pooled plasma specimen (5 mg Bromophenol Blue/ml pooled plasma) acts as a visual migrating marker on each plate.
6. Electrophoresis: Five ml of distilled water is spread on the cooling plate and the gel plate is carefully placed on this ensuring that there are no air bubbles between the cooling plate and the Gelbond film. Cool tap water (10-14°C) is continuously circulated throughout the cell.

   Contact between the buffer compartments and the gel plate is made by the agarose bridges and buffer-moistened Wettex wicks. The Wettex wicks should extend approximately 5 mm on to the gel surface. Electrophoresis is performed at 250 V constant voltage (approximately 20 V/cm and 100 mA) for 40 min or until the Bromophenol Blue-tagged albumin complex is approximately 25 mm from the anodal edge of the gel plate. The polarity should be

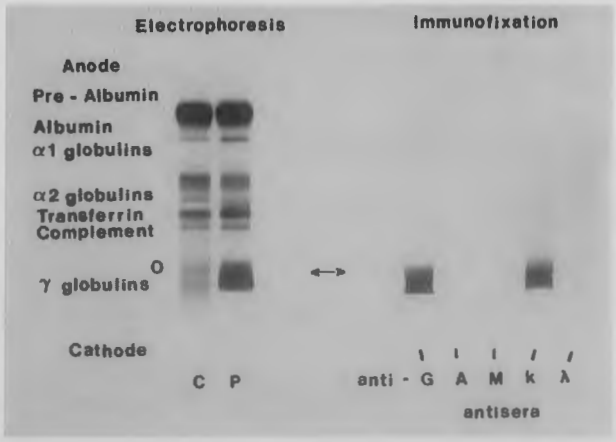

Figure 1: Identification of a monoclonal band in the presence of immunosuppression.
The abnormal band was identified as an IgG paraprotein of kappa type.
O = Origin C = Control P = Patient

Figure 2: Identification of a monoclonal band in the absence of immunosuppression.
a = Electrophoretic strip
b = Immunofixation — specimen diluted 5 fold
c = Immunofixation — specimen diluted 10 fold
The abnormal band was identified as an IgG paraprotein of lambda type.

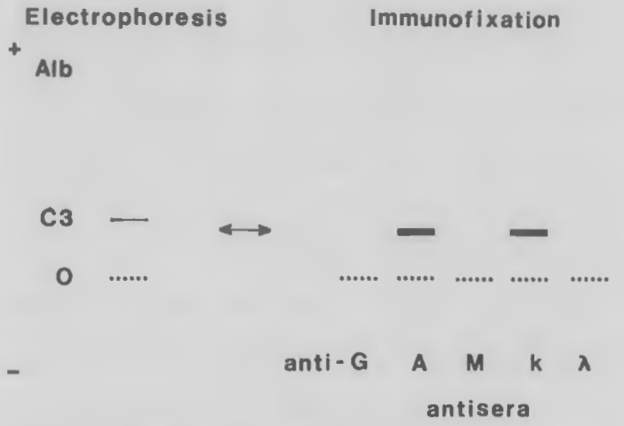

Figure 3a: Identification of a monoclonal band not visible on the initial electroporetic strip due to its low concentration.
Immunofixation was performed on the undiluted specimen (Immunoglobulin levels IgG 5.7 g/l, IgA 2.6 g/l, IgM 1.6 g/l.)
An IgA paraprotein of kappa type was detected just cathodal to complement (C3).

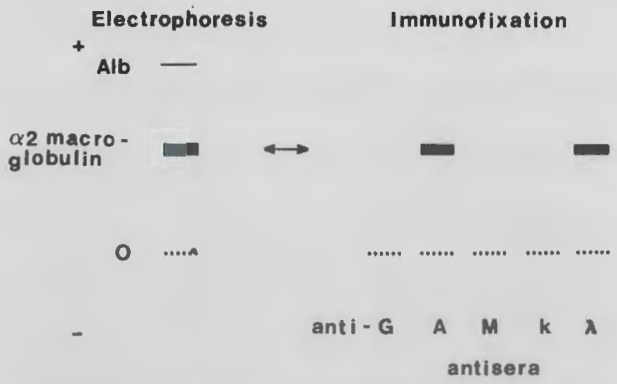

Figure 3b: Identification of a monoclonal band superimposed on another protein band.
An IgA paraprotein of lambda type was detected by immunofixation in the same position as $\alpha 2$ macroglobulin.

reversed after each run to ensure that the buffer composltion is not altered. The electrophoresis buffer and the agarose bridges should be changed after every 12-15 runs and the Wettex wicks should be re-boiled in distilled water.

7. Fixing, Staining and Destaining: Plates are fixed in the saturated picric acid/acetic acid solution for 10 min then overlaid with Whatman 31ET paper, a layer of paper towels and a glass plate, and pressed with a 2kg weight for 10 min. They are then dried at 90°C in a hot air oven, stained for 10 min with Coomassie Brilliant Blue and destained until the background is clear.

Immunofixation: An initial electrophoretic scan on the serum or concentrated urine specimen is routinely performed and any abnormal bands detected are identified by immunofixation. The specimens are re-electrophoresed using appropriate dilutions then immediately immunofixed. Correct specimen dilution is essential and this is discussed in the results section.

Immunofixation was performed using a modification of the method of Ritchie and Smith[2]. Strips of cellulose acetate (80 x 50mm) dipped in antisera and applied to the surface of the agarose are routinely used. The plates are incubated for 45-60 min in a humid chamber. The strips are then removed and the plates are washed with tap water for 30 sec, pressed overnight, then dried, stained and destained as for SPE. Serum specimens are immunofixed using the anti-heavy chain antisera anti-IgG, anti-IgA and anti-IgM and the anti-light chain antisera anti-kappa and anti-lambda. If a precipitin band occurs with either of the anti-light chain antisera but not with the anti-heavy chain antisera, the specimen must be further immunofixed using anti-IgD and anti-IgE antisera before reporting the presence of monoclonal light chains. It should be noted that some monoclonal immunoglobulins do not readily cross-react with the appropriate anti-heavy or anti-light chain antisera and also may not migrate during electrophoresis. In these cases treatment of the specimen with 2-mercaptoethanol (1 drop/0.5 ml specimen for 5 min at room temperature) should be performed before repeating electrophoresis and immunofixation. Gelling of the treated specimen should be avoided. If this does occur use a diluted solution (1:3) of 2-mercaptoethanol.

In some instances monoclonal immunoglobulins exhibit cryoprotein characteristics in which case specimen collection, transport, preparation, electrophoresis and immunofixation should all be performed at 37°C.

It is usually only necessary to immunofix urine specimens with the heavy chain component(s) of the serum paraprotein(s) and both anti-light chain antisera.

Results and Discussion:

For simplicity of presentation, apart from the first figure, electrophoresis and immunofixation results are presented as diagrams with albumin and any relevant abnormal bands only being shown.

It is essential that specimens are diluted to a point where the Ag concentration is approximately equivalent to the Ab concentration. This is demonstrated in Figure 1. A large discrete band with immunosuppression was detected cathodal to the origin by electrophoresis. (The immunoglobulin levels as determined by Radial Immunodiffusion were IgG 21.4 g/l, IgA 10.1 g/l, IgM 0.2 g/l). Immunofixation showing precipitation which occurs when the Ag/Ab

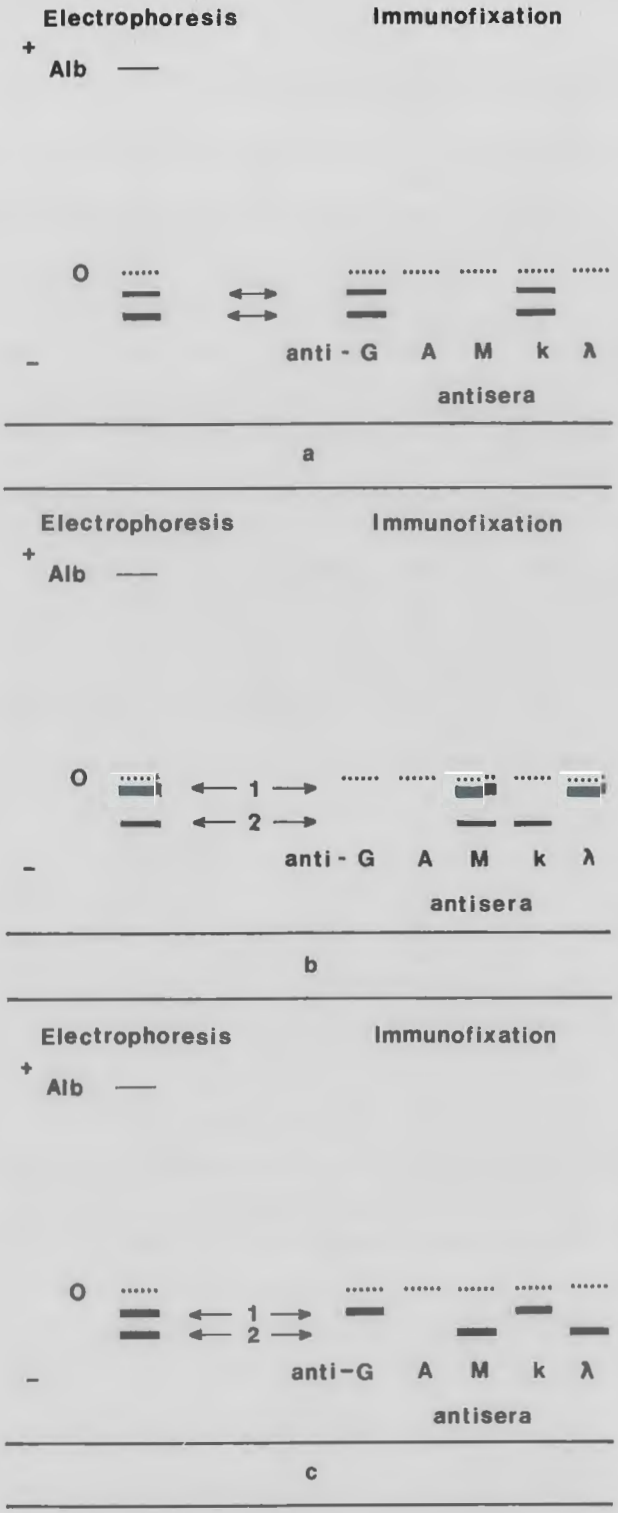

Figure 4: Multiple banding.
(a) Two bands were detected in the gamma region by electrophoresis and were both identified by immunofixation as IgG paraproteins of kappa type.
(b) Two bands were detected in the gamma region by electrophoresis and were identified by immunofixation as an IgM paraprotein of lambda type (Band 1) and an IgM paraprotein of kappa type (Band 2)
(c) Two bands were detected in the gamma region by electrophoresis and were identified by immunofixation as an IgG paraprotein of kappa type (Band 1) and an IgM paraprotein of lambda type (Band 2).

concentrations are near equivalence is shown. No visible precipitation occurred when the specimen is diluted 100-fold. When the specimen is

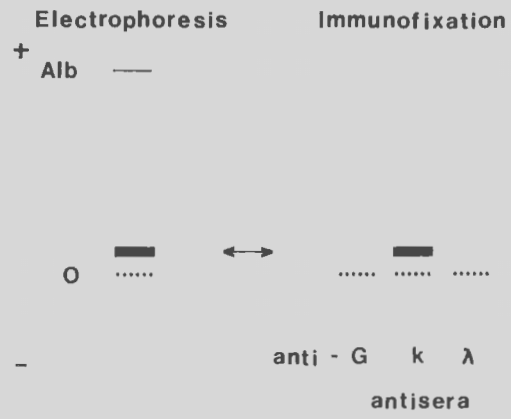

Figure 5: Identification of a single abnormal band in urine.
The single band detected in the gamma region was identified as Bence Jones Protein of kappa type. (An IgG paraprotein of kappa type was previously identified in the patients serum).

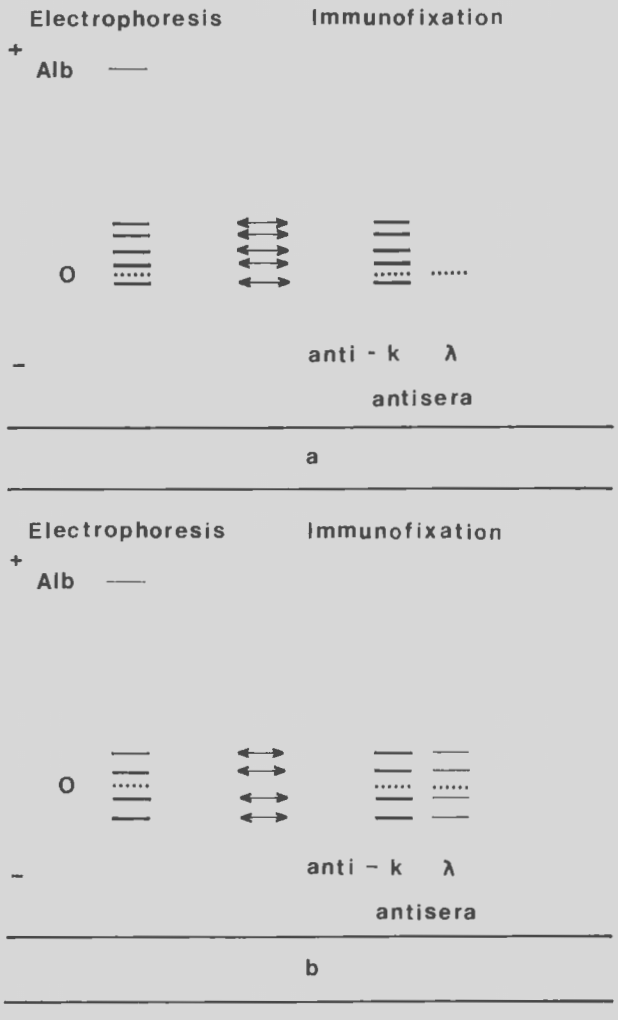

Figure 6: Identification of multiple bands in urine.
(a) The immunofixation pattern indicates Bence Jones Protein of kappa type.
(b) The immunofixation pattern does not indicate the presence of Bence Jones Protein.

not sufficiently diluted the presence of excess Ag results in a 'white hole' on immunofixation.

It is important when typing a paraprotein of greater than 20 g/l or one involving a dilution of 15-fold or more to analyse an undiluted specimen

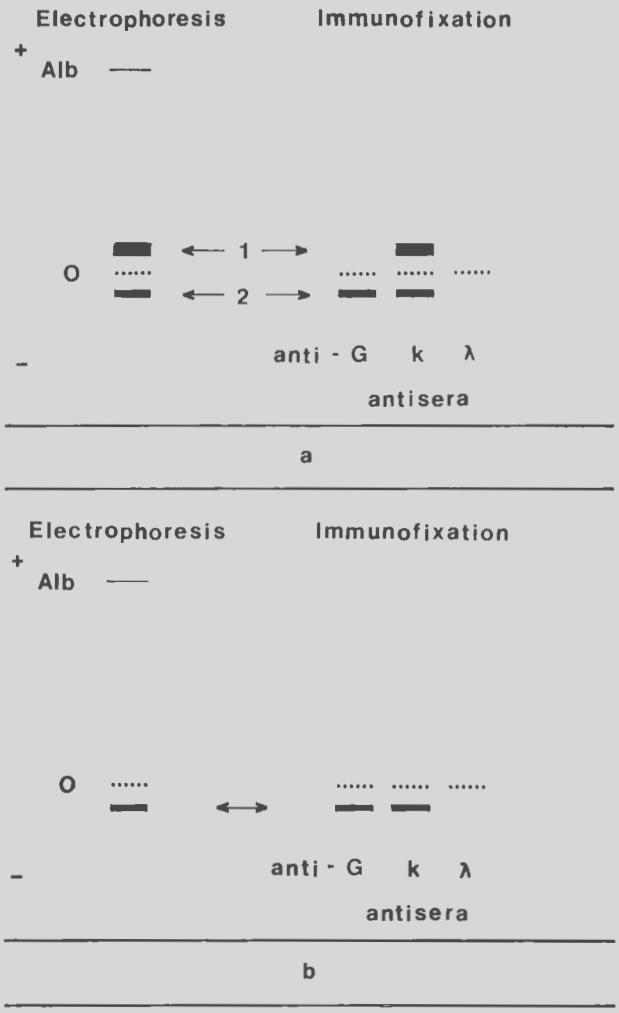

Figure 7: Excretion of paraproteins in urine.
(a) Excretion of a paraprotein and Bence Jones Protein. The two bands detected in the gamma region by electrophoresis were identified by immunofixation as Bence Jones Protein of kappa type (Band 1) and IgG paraprotein of kappa type (Band 2). (An IgG paraprotein of kappa type was previously detected in the patients serum).
(b) Excretion of a paraprotein only.
The single band detected in the gamma region was identified by immunofixation as an IgG paraprotein of kappa type. (An IgG paraprotein of kappa was previously detected in the patients serum).

in conjunction with the diluted specimen. This is to detect any minor bands, not visible on electrophoresis, which would otherwise not be detected by immunofixation in the diluted specimen. Figure 2 further demonstrates the importance of dilution. The initial electrophoresis (Figure 2a) showed a small discrete band cathodal to the origin with no immunosupression (IgG 10.0 g/l, IgA 3.7 g/l, IgM 0.8 g/l). When the specimen was not sufficiently diluted the band was not visible by immunofixation because of interference from the 'background immunoglobulins' and when the dilution was too great the band was diluted out. Correct dilution identified the band as an IgG paraprotein of lambda type (Figure 2c).

As previously stated, not all paraproteins are visible on the initial electrophoretic scan, either because they are not present in sufficient concentration (Figure 3a) or they present in the same position as another protein (Figure 3b). As shown, both of these situations can readily be resolved by immunofixation.

Occasionally a monoclonal protein exhibits precipitation with one anti-heavy chain antiserum but not with the anti-light chain antisera. It is essential that the absence of monoclonal light chains be checked over a wide range of specimen dilutions before making a diagnosis of heavy chain disease. This problem arises in particular with IgA monoclonal proteins.[8]

We have found over the past 2.5 years that 16% of all patients

presenting with paraproteinaemia have more than one abnormal band. Examples of these are shown in Figure 4.

Identification and typing of abnormal bands in urine can present problems in interpretation. This is dependent on the number of abnormal bands detected in the concentrated urine. When a single abnormal band is detected few problems arise in interpretation (Figure 5). However, when multiple banding occurs (Figure 6) extreme care must be taken. Five faint bands were detected in the concentrated urine from two patients both of whom had no detectable serum paraproteins. Immunofixation of the first specimen (Figure 6a) showed five precipitin bands occurring with anti-kappa antiserum but not with anti-lambda antiserum. This pattern can be confirmed as Bence Jones Protein of kappa type. Immunofixation of the second specimen showed five faint precipitin bands with both anti-kappa and anti-lambda antiserum. This pattern, and similar patterns showing faint banding with both anti-light chain antisera, cannot usually be ascribed to Bence Jones Protein. Coexistent Bence Jones Protein of kappa and lambda types have been detected but the pattern is distinctive as evidenced by the relative staining intensity and the position of the bands.

The excretion of a paraprotein in addition to Bence Jones Protein has also been demonstrated in a number of patients (Figure 7a) as has the excretion of a paraprotein only (Figure 7b).

Conclusion:

Immunofixation provides a sensitive method for the typing of monoclonal immunoglobulins and presents few difficulties provided the simple rules outlined are followed.

Acknowledgements:

I would like to thank Dr C. M. André and colleagues for their encouragement and advice.

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Technical Communication

An example of a steroid associated haemagglutinin.

C.G. Storey, ANZIMLT

Blood Bank, Waikato Hospital, Hamilton, New Zealand.

Introduction

To help maintain the integrity of the cell membrane with its antigens and retard haemolysis during their dating period, laboratory panel cells are suspended in special preservative solutions. Solutions such as Alsevers and Rous & Turner (1) have been formulated for short term preservation of red cells. However, manufacturers of commercial reagent red cell panels modify or formulate their own diluent solutions for their products and generally incorporate, in an isotonic solution, antibiotics (2,3) to inhibit the growth of bacteria, purines and nucleosides as preservative agents. Steroids (4) have been reported to inhibit the lysis of red cells in vitro and may also be incorporated in such preservative solutions.

I report an example of an antibody causing red cell aggultination in the presence of such a steroid additive.

Case History

A 53 year old male was crossmatched against two units of blood in preparation for a bladder biopsy procedure.

Tests and Results

The patient's serum was crossmatched and antibody screened against using the following techniques: saline room temperature, one stage enzyme (papain) using a layering technique and saline 37°C — converted to I.C.T. Incubation times were one hour.

No difficulty was encountered during crossmatching but subsequent routine antibody screening tests detected the presence of an antibody which caused strong agglutination at room temperature, weak agglutination by enzyme technique and no agglutination by indirect Coombs test. The antibody agglutinated all cells of the commercial cell panel. However, neither the donor cells nor the patient's own cells (saline suspensions) reacted with the patient's serum. Washed saline suspended commercial panel cells failed to react with the antibody. The donor red cells and patient's own red cells when suspended in the diluent provided with the panel kit, were strongly agglutinated. These results indicated that the antibody was apparently directed against some component of the reagent red cell diluent.

Each antibiotic component of the reagent red cell diluent was prepared separately in isotonic saline at a concentration closely resembling that in the finished diluent. Concentrations were noted from the package insert. Group O cells were suspended in each solution to a final concentration of 2-5% and retested against the patient's serum. No agglutination occurred with any of these cell suspensions.

In order to have the unknown components of the diluent tested, the patient's serum was forwarded to the reagent manufacturer. Confirmation was received that the serum did in fact contain an antibody to a component in their diluent, the steroid additive.

Comment

Mann (4) has described hydrocortisone associated antibodies in human sera. Of twenty-five cases, sixty-four percent were known to have a history of neoplasia. An immunological relationship between the formation of this type of antibody and tumour development was mentioned. It is of interest that this patient was being investigated for a possible bladder tumour although the histology findings were negative.

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Manufacturers Reply

September 2, 1983

Mr C.G. Storey
Immunohaematology Department
Waikato Hospital
Private Bag,
Hamilton, New Zealand

RE: C.J.M. Kamphues
(sample dated 7/18), F-3920
(sample dated 7/14), F-3931
(sample hemolyzed), F-3922

Dear Mr Storey,

We have confirmed your suspicions that the Kamphues serum contains an antibody to a component of the ORTHO red cell diluent. The component to which the antibody is directed has been identified as the steriod which is present in the diluent solution. No reactivity was observed in testing washed reagent red cells or cells to which the other components of the diluent were added. The Kamphues antibody was reactive on immediate spin and after incubation at 37°C; no reactivity was observed when the tests were converted to the indirect antiglobulin test. Additionally a panel of cells tested by the trypsin/Coombs procedure was compatible.

If additional antibody screening is necessary for this patient we would recommend that the reagent red cells be washed free of the diluent prior to testing. Thank you for bringing this problem to our attention and please feel free to contact us if you have any questions regarding our report.

Sincerely,

Norma J. Hock-McKean
Technical Coordinator

CONTINUING EDUCATION

Porphyrins and the Porphyrias

Alison Buchanan

Department of Clincial Chemistry Auckland Hospital

Clinical studies of the porphyrias were initiated in 1874 by a description of a classical case of congenital porphyria. This was misdiagnosed as a typical leprosy. Disorders of porphyrin metabolism have fascinated investigators therefore for more than a hundred years.

Recently many questions have been answered, particularly those relating to aetiology, but many problems remain unsolved amid a wealth of confusing terminology and misleading nomenclature.

Accurate diagnosis is of more than academic interest. Common medications may precipitate life threatening episodes in four of the seven porphyrias. Prompt treatment of these attacks is essential.

Porphyrins

Prophyrins are tetrapyrroles. They arise as a result of oxidation of the various porphyrinogens in the metabolic pathway to haem. They are a byproduct, have no known physiological function, and are normally only found in small quantities in body fluids and tissues.

These compounds are coloured dark red to purple in acid solutions, red to brown in alkaline solution, and absorb light at 400 to 410nm (Soret band). In acid solution they have a strong pink to red fluorescence on exposure to ultraviolet light.

Their solubility in water is influenced by the number of carboxyl groups in the pyrrole substituents. Uroporphyrin has eight carboxyl groups, is the most water soluble and is excreted in the urine. Protoporphyrin has two carboxyl groups, is insoluble in aqueous solutions at physiological pH, but is very soluble in lipid solvents, and is therefore excreted in the faeces.

Coproporphyrin, having four carboxyl groups, has intermediate solubility.

Porphyrinogens are unstable compounds which readily oxidise to

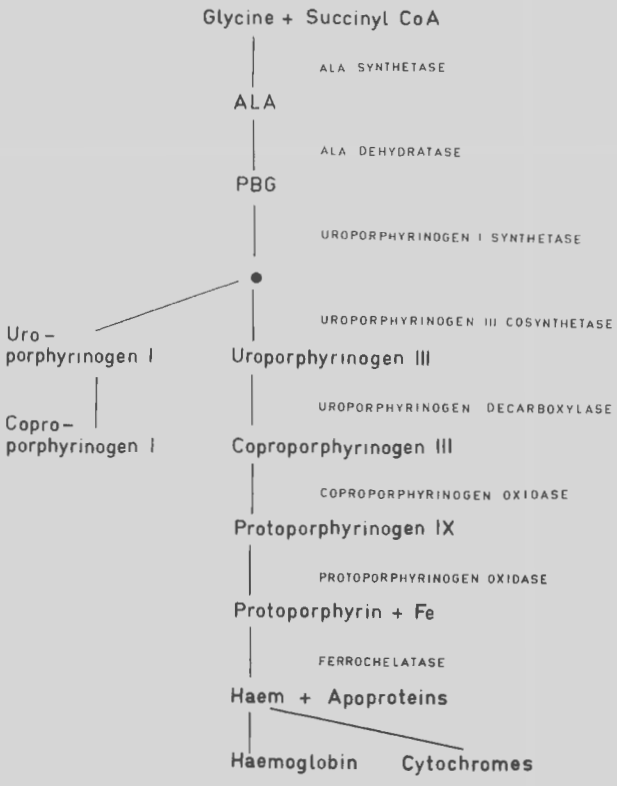

Figure 1: Biosynthesis of haem.

(There are fifteen possible isomers of protoporphyrin. The naturally occurring isomer has been designated IX although it is now known to be derived from precursors of the Type III series).

their corresponding porphyrin. They are colourless and non-fluorescent and therefore must be oxidised before they can be detected. A urine containing increased levels of porphyrinogens will gradually darken on exposure to air and sunlight as the porphyrinogens present oxidise to porphyrins, giving a "port wine" urine.

Porphyrinogens are found in most tissues, but are most abundant in the liver and bone marow. They are involved in oxygen storage and transport (haem), cellular oxidation (cytochromes), and toxin and drug hydroxylation.

Haem Synthesis

The synthesis of haem is outlined in Figure 1 and includes fifteen major enzyme catalysed steps. Only the Type III porphyrinogen isomers are precursors of haem. The Type I isomers have no known physiological function.

Control of haem synthesis is achieved largely by the enzyme delta-aminolevulinic acid (ALA) synthetase. Haem causes a negative feed-back cycle to act on this enzyme.

Uroporphyrinogen I synthetase also has an influence and it is postulated that the level of activity is important in the differentiation of acute and non-acute porphyrias.

In all porphyrias haem production is potentially suboptimal. Where it is reduced, the activity of ALA synthetase and the consequent production of the porphyrin precursors (ALA and porphobilinogen (PBG)) is increased.

In acute porphyrias the activity of uroporphyrinogen I synthetase is normal thereby allowing an accumulation of PBG and ALA. Their increased excretion can be demonstrated.

In non-acute porphyrias uroporphyrinogen I synthetase activity is increased, thus preventing an accumulation of the precursors.

Porphyrias

The porhyrias are a group of diseases caused by a derangement in the metabolic pathway leading to the synthesis of haem. All but one of the seven forms are known to be inherited. They are caused, or potentiated, by abnormally low activity of one or more of the enzymes involved in the haem production pathway.

The oneset of the disease can occur in young children or adults depending on the type of porphyria. The clinical symptoms may include photosensitivity of the skin, persistent abdominal pain, neuropathy and a wide range of other clinical syndromes. The neuropathy may affect the central autonomic and peripheral systems and lead to mental disturbances, motor dysfunction and sensory loss.

Skin photosensitivity and associated lesions occur when high levels of circulating porphyrins are deposited in the skin and are exposed to sunlight. Porphyrins absorb energy at 400nm and it is thought that the "excited" porphyrins damage lysozome membranes in the skin tissues, releasing lysozymes and proteases that cause further local damage to the tissue.

The acute features of abdominal pain and neuropathy are related to an accumulation of the porphyrin precursors (ALA and PBG) in the neural tissues.

Classification

There are several ways in which the porphyrias may be classified, some based on biochemical features and others on the clinical presentation.

The most common classification refers to the site of the metabolic abnormality, that is, erythropoietic or hepatic porphyrias.

ERYTHROPOIETIC PORPHYRIAS

1. Congenital Erythropoietic Porphyria (CEP)
   a. Transmission: Autosomal recessive
   b. Enzyme defect: Probably Uroporphyrinogen III cosynthetase.
   c. Biochemical Abnormality: This is the only porphyria that involves an increase in the Type I isomers.
   Uroporphyrinogen I is increased in the red cells, leading to their premature destruction.
   Urine and faecal uroporphyrin I and coproporphyrin I are also increased.
   d. Clinical expression: The disease presents shortly after birth with extreme photosensitivity and extensive blistering, together with the production of red urine. Teeth and bones may be red-brown in colour and fluoresce under ultra-violet light. There is often associated haemolytic anaemia and hirsutism. There are no neurological symptoms (ALA and PBG levels are normal). There is a theory that these unfortunate individuals with hairy faces and shiny teeth who venture out only at night (to avoid the sun) gave rise to some of the werewolf legends.
2. Erythropoietic Protoporphyria (EPP)
   a. Transmission: Autosomal dominant
   b. Enzyme defect: Ferrochelatase
   c. Biochemical Abnormality: There is an increased concentration of protoporphyrin in the red cells and faeces, but urine levels are normal due to the water insolubility of protoporphyrin.
   d. Clinical expression: The onset occurs in childhood, presenting with features of photosensitivity. Blistering and anaemia do not occur. Liver cirrhosis may develop later in the course of the disease and can be fatal, but this can be reversed if treatment is initiated in the early stages.
3. Erythropoietic Coproporphyria (ECP)

This is a rare disorder resembling EPP but with an elevated red cell coproporphyrin. There has been no apparent liver involvement in the few cases reported. The enzyme deficiency is still unknown.

HEPATIC PORPHYRIAS

Four forms of porphyria have been classified as hepatic porphyrias because it is assumed that the errors of porphyrin synthesis occur predominantly in the hepatocytes.

1. Acute Intermittent Porphyria (AIP)
   a. Transmission: Autosomal dominant
   b. Enzyme defect: Uroporphyrinogen I synthetase (Porphobilinogen deaminase)
   c. Biochemical Abnormality: There are increased levels of ALA and PBG in the urine during an acute attack. PBG readily oxidises in acid conditions and sunlight to porphobilin, therefore urine collected during an attack will darken on standing.
   The faeces show no significant increase in porphyrin levels. During the latent phase urine PBG may be increased but definitive diagnosis at this stage depends on demonstrating a lack of uroporphyrinogen I synthetase activity.
   d. Clinical expression: The disease usually manifests itself during adolescence and is characterised by acute attacks of abdominal pain, peripheral neuritis, and psychotic illness. It is often initiated by drugs (Barbiturates, oestrogen, sulphonamides) or by an acute illness.
2. Porphyria Variegata (VP) (Variegate Porphyria)
   a. Transmission: Autosomal dominant
   b. Enzyme defect: Protoporphyrinogen oxidase and possibly ferrochelatase.
   c. Biochemical Abnormality: There is an increased concentration of ALA, PBG, uroporphyrin and coproporphyrin in the urine during an acute attack (coproporphyrin > uroporphyrin). A disproportionately high level of uroporphyrin may be found and is due to extra-renal conversion of PBG to uroporphyrin. Increased concentrations of coproporphyrin and protoporphyrin are found in the faeces at all times (protoporphyrin > coproporphyrin).
   d. Clinical Expression: The disease manifests itself after puberty and may be associated with acute attacks as seen in AIP. Photosensitivity and skin lesions may occur independently or in combination with the acute attacks. Acute attacks occur more frequently in females than in males and may be precipitated by certain drugs (Barbiturates, sulphonamides, and alcohol). Skin lesions are more common in males and the severity ranges from photosensitivity to blistering and scarring of sun exposed areas.
3. Porphyria Cutanea Tarda (PCT)
   This is by far the most common type of porphyria seen in New Zealand. There appear to be two forms of this disease:
   i) Uncommon and Inherited
   a. Transmission: Autosomal dominant
   b. Enzyme defect: Uroporphyrinogen decarboxylase. The defect occurs in both liver and red cells.
   ii) Common
   a. Transmission: Caused by either inheritance or by chemicals that selectively inhibit the hepatic enzyme.
   b. Enzyme defect: Uroporphyrinogen decarboxylase. The defect occurs in liver cells. The red cell enzyme is normal.
   Both forms of porphyria cutanea tarda show similar biochemical and clinical features.
   c. Biochemical abnormality: There is a marked increase in porphyrins in the urine, mostly attributable to uroporphyrin. Faeces occasionally show increased porphyrin levels.
   d. Clinical expression: The symptoms are variable but usually the patient shows some degree of photosensitivity with blistering and skin fragility in sun exposed areas. Hirsutism and hyperpigmentation are common.

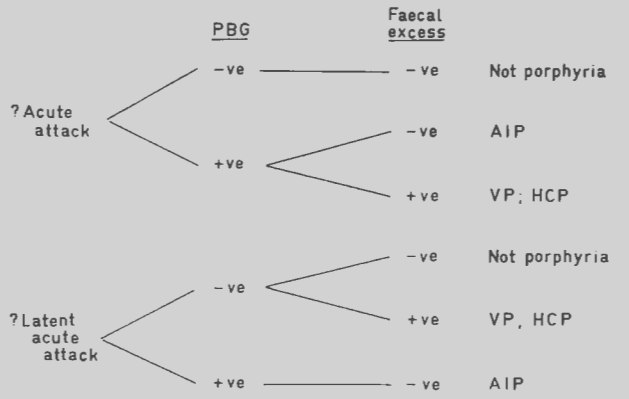

Figure 2: Strategy for investigating suspected acute porphyria.*
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Predisposing factors are liver disease, notably alcoholic cirrhosis, and ingestion of other toxins, (eg, hexachlorobenzene and polychlorinated biphenyls).

4. Hereditary Coproporphyria (HCP)
   a. Transmission: Autosomal dominant.
   b. Enzyme defect: Coproporphyrinogen oxidase.
   c. Biochemical abnormality: Increased levels of ALA, PBG, uroporphyrin and coproporphyrin (coproporphyrin > uroporphyrin) are found in the urine during acute attacks. The faecal coproporphyrin concentration is always increased.
   d. Clinical expression: The disease resembles VP, with acute attacks precipitated by the same drugs. Some cases develop photosensitivity but unlike VP these episodes are always associated with the acute attacks.

LABORATORY INVESTIGATIONS

A diagnosis of porphyria can only be made by correlating the patients clinical signs and symptoms with the biochemical abnormalities detected in the porphyrin metabolic pathway. Laboratory investigations depend on the measurement of porphyrins and their precursors in blood, urine and faeces. Screening tests for total porphyrin levels are easy to perform and often give sufficient information for a diagnosis to be made. A definitive diagnosis may require prophyrin fractionation and in some cases measurement of the appropriate enzyme activities.

Figures 2 and 3 present a simple screening strategy for diagnosing the porphyrias. They make an initial and rapid classification, based on simple screens for excess amounts of urinary PBG, excess amounts of total faecal porphyrin, and increased red cell porphyrins. An added screen for total urinary porphyrins may be necessary to diagnose PCT as faecal porphyrins are not always increased in this condition.

CASE HISTORIES

Following are some brief case histories of patients awaiting diagnosis.

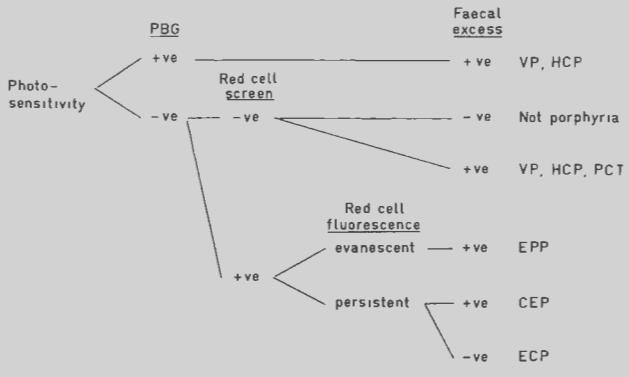

Figure 3: Strategy for investigating photosensitivity. (Redrawn).*
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Attempt to make the correct diagnosis using the preceding information. Answers are to be found on page 22.

Case 1.

A 24 year old woman has been admitted to hospital with colicky abdominal pain. She has been admitted three times previously with suspected appendicitis but no operation has been performed. She is taking no drugs other than oral contraceptive therapy, which she has recently commenced.

Laboratory investigations:

Urine screen:	PBG — Positive
	Porphyrins — Negative
Faecal screen:	Porphyrins — Negative

Case 2.

A 40 year old man, with a history of heavy alcohol intake and a previous diagnosis of alcoholic liver disease, presents with a 6 month history of redness and blistering of the back of his hands and on his face and scalp. He has noticed red urine on several occasions.

Laboratory investigations:

Urine screen:	PBG — Negative
	Porphyrins — Positive
Faecal Screen:	Porphyrins — Negative
Red cell Screen:	Porphyrins — Negative

Case 3.

A 35 year old man presents with a history of photosensitivity from childhood.

Laboratory investigations:

Urine Screen:	PBG — Negative
	Porphyrins — Negative
Faecal Screen:	Porphyrins — Positive
Red cell Screen:	Porphyrins — Positive

Analysis of the faecal porphyrins showed a marked increase of protoporphyrin with normal coproporphyrin excretion.

Case 4.

A 35 year old woman, presents with an acute psychotic illness, abdominal pain, red urine and a history of photosensitivity with skin eruptions.

Laboratory investigations:

Urine Screen:	PBG — Positive
	Porphyrins — Positive
Faecal Screen:	Porphyrins — Positive
Red cell screen:	Porphyrins — Negative

References:

1. Hindmarsh J. T. Clinical disorders of porphyrin metabolism. *Clin Biochem* 1982; **16:** 09-219.
2. Pain R. W. Phillips P. J. Harley H. A. J. Beare J. H. A simple screening strategy for diagnosing the porphyrias. *Med. J. Aust.* 1979: **2:** 474-477.
3. Elder G. H. Sheppard D. M. Salamanca R. E. Olmos A. Identification of two types of porhyria cutanea tarda by measurement of erythrocyte uroporphyrinogen decarboxylase. *Clinical Science* 1980; **58:** 477-484.
4. Ellefson R. D. Porphyrinogens, Porphyrins, and the porphyrias. *Mayo Clin. Proc.* 1982; **57:** 454-458.
5. With T. K. A short history of porphyrins and the porphyrias. *Int. J. Biochem.* 1980; **11:** 189-200.
6. Walmsley R. N. White G. H. A guide to diagnostic clincial chemistry. 1st; Edition Blackwell Scientific Publicatons. 360-380.
7. Eales L. Day R. S. Blekkenhorst G. H. The clinical and biochemical features of variegate porphyria: an analysis of 300 cases studied at Groote Schuur Hospital, Cape Town. *Int. J. Biochem.* 1980; **12:** 837-835.

16th Congress of the International Association of Medical Laboratory Technology, Perth, 1984

The 16th Congress of the International Association of Medical Laboratory Technologists will be held in Perth, Australia from 15th—20th July, 1984.

GUEST SPEAKERS

BARNSTABLE, PROFESSOR C.J.
Department of Neurobiology, Harvard Medical School, U.S.A.,

BERTRAM, Dr. J.F.
Department of Biological Structure, University of Washington, U.S.A.

BURTON, PROFESSOR R.
Professor of Surgical Sciences, University of Newcastle, Australia.

COTTEW, Dr. G.S.
Animal Health Research Laboratory, C.S.I.R.O. Melbourne, Australia.

FINCHAM, MR. W.J.
The Institute of Orthopaedics, Royal National Orthopaedic Hospital, Middlesex, United Kingdom.

GARRATTY, Mr. G.
Los Angeles/Orange Counties Red Cross Blood Services, Los Angeles, U.S.A.

GUST, Dr. I.D.
Virology Department, Fairfield Hospital for Infectious Diseases, Melbourne, Australia.

LAZARIDES, Dr. E.
Division of Biology, California Institute of Technology, U.S.A.

LARKINS, Dr. R.G.
University Department of Medicine, Repatriation General Hospital, Heidelberg, Australia.

LEWIS, Mr. J.
Area Pathology Laboratory, St. Mary's Hospital, Isle of Wight, United Kingdom.

PARDUE, PROFESSOR H.L.
Department of Chemistry, Purdue University, Illinois, U.S.A.

PAVILLARD, Dr. R.
Microbiology Department, The Royal Melbourne Hospital, Melbourne, Australia.

PEARMAN, Dr. J.
Microbiology Department, Royal Perth Hospital, Perth, Australia.

PILKINGTON, Dr. G.
Clinical Immunology and Immunogenetics Unit, The Cancer Institute, MacCallum Hospital, Melbourne, Australia.

VAN DER PLOEG, PROFESSOR M.
Department of Histochemistry and Cytochemistry, University of Leiden, The Netherlands.

POHL, Miss S.
Department of Laboratory Medicine, Good Samaritan Hospital and Health Centre, Ohio, U.S.A.

SEVIER, Dr. D.
Technical Services, Hybritech, San Diego, U.S.A.

TERRY, PROFESSOR R.
Department of Pathology, Albert Einstein College of Medicine of Yeshiva University, New York, U.S.A.

THOMSON, Dr. J.M.
National (U.K.) Reference Laboratory for Anticoagulant Reagents and Control, Withington Hospital, Manchester, England.

SCIENTIFIC PROGRAMME

The 16th I.A.M.L.T. Congress will provide a forum for medical laboratory scientists to enrich their professional life with new technology, new ideas and new concepts. They will be given a penetrating look at some of the latest advances in Medical Technology and an insight into the future.

The SCIENTIFIC PROGRAMME will encompass all disciplines involved in medical laboratory science as well as continuing education and management. Many internationally eminent scientists have accepted invitations to participate in the Congress.

In addition, extensive proffered paper sessions, poster presentations, workshops and laboratory visits ensure a full and rewarding programme.

In keeping with the Congress theme, "The Developing World of Medical Technology", the programme on the first morning will be given over to the rapidly developing biotechnology of the MONOCLONAL ANTIBODY. Dr Graham Mitchell from The Walter and Eliza Hall Institute of Medical Research will be the principal speaker to the morning's theme of "The monoclonal antibody: what impact on the future of medical science and technology?". This theme is designed for all medical laboratory scientists to gain an insight into the effect the monoclonal antibody will have on their profession — as it is happening now and how it may influence the future. No other session will run concurrently to enable all to attend.

Lunch and refreshment breaks between sessions have been designed to allow sufficient time for delegates to visit the trade exhibition, or to extend their discussion on previous papers while enjoying a cup of coffee or a meal.

WORKSHOPS will be held on the three days prior to the commencement of the official opening of the Congress.
LABORATORY TOURS have been organised for interested delegates.

DAY 1
Monday, 16th July

09.30—10.45
The Monoclonal Antibody — what impact on the future of Medical Science?
10.45—11.15
Refreshment Break
11.15—12.30
Application of monoclonal antibodies to the diagnosis and treatment of leukaemia — *Dr G. Pilkington.*
Immunotherapeutic trials using monoclonal antibodies. — *Dr D. Sevier.*
12.30—14.00
Lunch Break
14.00—15.15
Preliminary General Assembly of Delegates.
Concurrent sessions:
- o Clinical Chemisry — proffered papers.
- o Clinical Immunology — proffered papers.
- o Paediatric Haematology - Dr J. Price. — *Dr M.L.N. Willoughby.*
- o Microbiology — proffered papers.

15.15—15.45
Refreshment Break
15.45—17.00
Preliminary General Assembly of Delegates (Cont.).
Concurrent sessions: (continued)
- o Clinical Chemistry — proffered papers.
- o Haematology or Immunohaematology — proffered papers.
- o Histopathology — proffered papers.
- o Microbiology — proffered papers.

END OF DAY 1

Day 2
Tuesday, 17th July

09.30—10.45
Concurrent sessions:
- o Recent advances in instrumentation for clinical chemistry — *Professor H. Pardue.*
- o Recent advances in Immunoparasitology — *Dr G. Mitchell; Dr G. Stewart.*
- o Education or management — proffered papers.
- o Clinical significance of blood group antibodies — *Mr G. Garratty.*
- o Quantitative diagnostic methods for histology — *Dr J. Bertram.*
- o Microbiology — proffered papers.

10.45—11.15
Refreshment Break.

11.15—12.30
Concurrent sessions: (continued)
- o **Energy focus:**
  The application of low energy to hospital planning and its implementation with specific reference to laboratory design — *Mr J. Lewis.*
  Cell quantification in the nervous system — *Dr R.D. Terry.*
- o **Hospital acquired infection (1):**
  New frontiers, new problems — *Dr R. Pavillard; Dr W.J. Fincham.*

12.30—14.00
Lunch Break
14.00—15.00
Concurrent sessions:
- o Recent advances in thyroid studies: the Australian experience — *Dr G. McLellan; Dr M. Dick; Mr F. Watson.*
- o Deciphering cell structure and morphogenesis with immunological probes — *Dr E. Lazarides.*
- o **Energy focus:** an effective cost-saving approach — Panel discussion.
- o External quality assurance programmes in Haematology — *Dr J.M. Thomson.*
- o Histopathology — proffered papers.
- o **Hospital acquired infection (2):**
  Methicillin Resistant Staphylococcus aureus — the organism and its control — *Dr W. Grubb; Mr D. Townsend; Dr J. Pearman.*

15.15-15.45
Refreshment Break
15.45—17.00
Concurrent sessions: (continued)
- o Clinical Chemistry — proffered papers.
- o Clinical Immunology — proffered papers.
- o Education or management — proffered papers.
- o Haematology or Immunohaematology — proffered papers.
- o Histopathology — proffered papers.
- o **Hospital acquired infection (3):**
  Viruses in the hospital environment — *Dr I. Gust; Mr G.P. O'Connor.*

END OF DAY 2

DAY 3
Wednesday, 18th July

09.30—10.45
Concurrent sessions:
- o Taking advantage of monoclonal antibodies in immunodiagnostics — *Dr D. Sevier.*
- o **The Medical laboratory Manager:**
  (Part 1) — In service training or formal educational courses — *Mr G. Smart; Mr A.R Fergie; Mr S. Hayden-Smith; Mr A. Houghton.*
- o An overview of mesothelioma — *Dr K.B. Shikin; Mr D. Whitaker.*
- o Microbiology — proffered papers.

10.45—11.15
Refreshment Break
11.15—12.30
Concurent sessions: (continued)
- o Clinical chemistry — proffered papers.
- o Techniques to characterise the cell surface antigens — *Dr C. Barnstable.*
- o **The Medical Laboratory Manager**
  (Part 2) — Panel discussion with audience participation.
- o Compatibility testing: Past, present and future — *Mr G. Garratty.*
- o **Cytometry:** Quantitation in individual cells — *Professor M. van der Ploeg.*
- o Microbiology — proffered papers.

12.30—14.00
Lunch Break
14.00—15.15
General Assembly of Delegates.
Concurrent sessions:
- o Clinical chemistry — proffered papers.
- o Clinical immunology — proffered papers.
- o Haematology or Immunohaematology — proffered papers.
- o Histopathology — proffered papers.
- o Microbiology — proffered papers.

15.15—15.45
Refreshment Break.
15.45—17.00
General Asembly of Delegates (cont.)
Concurrent sessions: (continued)
- o Clinical chemistry — proffered papers.
- o Clinical immunology — proffered papers.
- o Haematology or Immunohaematology — proffered papers.
- o Histopathology — proffered papers.
- o Microbiology — proffered papers.

END OF DAY 3

DAY 4
Thursday, 19th July

09.30—10.45
Concurrent Sessions:
- o Non-linear data processing methods evaluated for enzyme reactions — *Professor H. Pardue.*
- o Immunological assays in diagnosis — *Professor R. Burton; Professor R. Dawkins.*
- o Education or management — proffered papers.
- o Blood coagulation: recent advances in coagulation factor assays — *Dr J.M. Thomson.*
- o **Histopathology:**
  Lectin analysis of surface saccharides in resident and exudate peritoneal macrophages — *Dr A. Waton.*
  Quantitative studies in alcoholics — *Dr C. Harper.*
- o Microbiology — proffered papers.

10.45—11.15
Refreshment Break
11.15—12.30
Concurrent sessions: (continued)
- o Random access analysers — proffered papers.
- o Immunological techniques in the diagnosis of infectious disease — *Mr N. Hodgen; Mr G. Harnett.*
- o **Medical Technology, a developing profession:**
  The development and future of Medical Technology as a profession — *Mr W.J. Fincham.*
  The management of change in the context of the Pathology laboratory — *Mr J. Lewis.*
- o Third generation automated cell counters — *Dr R. Davis.**
  **Other speakers to be announced.*
- o **Histopathology:**
  Uncertainty in interpretation of experimental data — *Dr A. Lamb.*
  Image analysis of muscular dystrophic nuclei — *Dr R. Howlett.*
  Tissue culture in brain tumours — *Mr P. Jacobsen.*
- o **Newer Mechanisms of diarrhoea:**
  The mechanisms — *Dr M. Gracey; Dr V. Burke.*
- o Aeromonas hydrophilla — *Mrs L. Robinson.*
- o Clostridium dificile — *Mr T. Riley.*

12.30—14.00
Lunch Break.
14.00—15.15
Concurrent sessions:
- o Recent advances in drug analysis — *Mr A. Ukich; Dr R. Nation*.*
- o Clinical immunology — proffered papers.
- o **Current issues in education: (Part 1)**
  Present and future trends in continuing education — *Miss S. Pohl.*
  International portability of qualifications — *Mr J.R. Neal.*
  The need for professional postgraduate qualifications — *Mis B.N. Wilson.*
- o Bone marrow transplanatation: current status — *Dr A. Barr; Mr B. Meyer.*
- o Florescent in situ hybridization — a tool for chromosome analysis and virus detection — *Professor M. van der Ploeg*.*
- o Microbiology — proffered papers.

**Other speaker and title to be announced.*

15.15—15.45
Refreshment Break.
15.45—17.00
Concurrent sessions: (continued)
- o Clinical chemistry — proffered papers.
- o Clinical immunology — proffered papers.
- o **Current issues in education: (Part 2)**
  Panel discussion with audience participation.
- o Haematology or Immunohaematology — proffered papers.
- o Histopathology — proffered papers.
- o The relevance of clinical microbiology in the care of patients — *Dr R. Pavillard; Dr D.B. McGechie.*

END OF DAY 4

DAY 5
Friday, 20th July

09.30—10.45
Concurrent sessions:
- o Recent advances in calcium studies — *Dr R. Larkins; Dr N. Kent.*
- o **Immunological techniques in cell biology:** Cell surface antigens — *Dr C. Barnstable.* Intracellular structures — *Dr E. Lazarides.*
- o Education or management — proffered papers.
- o Serological classification of leukaemia/lymphoma — *Dr G. Pilkington.*
- o **Histopathology:**
  Cytology and histology of condylomatoid lesions of the female genital tract — *Mr V. Williams.*
  Comparative ultrastructural and cytochemical investigations of malignant melanomas — *Mr S. Carello.*
  Freeze etch replicas of coated vesicles and pits — *Mr T. Robertson.*
- o Towards the control of viral hepatitis — *Dr I. Gust.*

11.45—11.15
Refreshment Break.
11.15—12.30
Concurrent sesisons: (continued)
- o Clinical chemistry — proffered papers.
- o **Complement and immunoglobulins:** genetics and relevance ot disease — *Mr T. Cobain; Mr P. Kay; Dr J. Wetherall.*
- o Education or management — proffered papers.
- o Haematology or Immunohaematology — proffered papers.
- o **Histopathology forum:**
  summation by panel of themes presented earlier by panel members — *Dr J. Bertram; Professor M. van der Ploeg; Dr R.D. Terry.*
- o Mycoplasms of Medical and Veterinary interests — *Dr G.S. Cottew*.*

**Other speakers to be announced.*
12.30—14.00
Lunch Break.
14.00—15.15
Concurrent sessions:
- o Clinical chemistry — proffered papers.
- o Clinical immunology — proffered papers.
- o Education or management — proffered papers.
- o Haematology or Immunohaematology — proffered papers.
- o Histopathology — proffered papers.
- o Microbiology — proffered papers.

15.15—15.45
Refreshement Break.
15.45—16.15
17th I.A.M.L.T. Congress — Sweden 1986
Presentation and invitation by Swedish delegates.
16.15—17.00
Closing ceremonies.

WORKSHOPS

Workshops will be held on the 12th, 13th and 14th July, i.e. during the three days prior to the official opening of the Congress.

TRAVEL TO PERTH

To assist members of the Institute (and friends and families) to attend the Congress the following tours have been negotiated:

TOUR No. 1 10 DAYS PERTH

11 July	Auckland/Wellington/Christchurch to Perth (via Sydney) Transfer on arrival
11-21 July	10 nights accommodation — Airways Motels
21 July	Transfer to airport Perth to Auckland/Wellington/Christchurch (via Sydney) COST: $1,389 share twin plus $40 travel tax

TOUR No. 2 10 DAYS PERTH, 3 DAYS SYDNEY

11 July	Auckland/Wellington/Christchurch to Perth (via Sydney) Transfer on arrival
11-21 July	10 nights accommodation — Airways Motels
21 July	Transfer to airport Perth to Sydney Transfer on arrival
21-24 July	3 nights accommodation — Koala Oxford Inn
24 July	Transfer to airport Sydney to Auckland/Wellington/Christchurch COST: $1,498 share twin plus $40 travel tax

TOUR No. 3 10 DAYS PERTH, 3 DAYS SINGAPORE

11 July	Auckland/Wellington/Christchurch to Perth (via Sydney) Transfer on arrival
11-21 July	10 nights accommodation — Airways Motels
21 July	Transfer to airport Perth to Singapore Transfer on arrival
21-24 July	3 nights accommodation — Ming Court Hotel
22 July	Half day sightseeing tour
24 July	Transfer to airport Singapore to Auckland
25 July	Arrive Auckland Auckland to Wellington/Christchurch COST: $1,723 share twin plus $40 travel tax

Costs are subject to final confirmation on receipt of 1984 hotel rates together with currency fluctuations and airfare increases

All tours include: Return travel from Auckland, Wellington or Christchurch
Accommodation in Perth for the Workshops and Congress
All transfers to and from airports
Accommodation in Sydney (Tour 2) or Singapore (Tour 3)

NOTE: Under changes announced in the last budget people are now eligible to claim up to $1,000 tax exemption for attending such a meeting

If you are interested in attending the Congress complete the form below.

TO Mr B. T. Edwards,
Secretary, NZIMLT,
Haematology Department,
Christchurch Hospital,
CHRISTCHURCH

Please send me the following for the IAMLT Congress in Perth

Preliminary Programme and Registration Form ☐

Details of Tour No. 1 ☐

Details of Tour No. 2 ☐

Details of Tour No. 3 ☐

Name .....

Address .....

EDITORIAL

Innovations

Welcome to the first issue of the Journal for 1984. In case you did not notice on the contents page, there will be four issues this year — this March issue, May, early August and November — and copy is required by the first of the month preceding the month of publication, except for the August issue where copy is required by mid-June.

This issue contains two new or improved features. Firstly the new — the reader's reply card which will enable you to obtain the latest information on products that are advertised in the Journal. It will also allow our advertisers to gauge how effective their advertising is. So, if you require any information on any product advertised, fill out the card and post it — a 24c stamp is all that is required. The readers reply card will be in each issue this year, so make use of the service.

Secondly, there is an improved continuing education feature. In this issue the subject is Porphyrins and the Porphyrias, extremely well written by Alison Buchanan. At the end of the feature there are four case histories by which you will be able to assess whether you have understood the article; the answers are located elsewhere in the Journal.

In future issues, the self-assessment may be in the form of multi-choice questions but each feature will have some form of self-assessment. All laboratory disciples will be covered in forthcoming issues.

The continuing education feature is designed not only to bring Technologists in that particular discipline up to date, but also to allow those from other disciplines to broaden their knowledge of laboratory technology in general, and it will also supply students with some very useful information.

I would appreciate some feedback about which topics readers would like to see covered. Any comments at all on the concept would be most welcome.

An Oversight?

Every so often the Editor makes a plaintive plea to members for original articles to be sent for consideration for publication. That he should have to do so is amazing. Are we New Zealanders incapable of producing something original without being cajoled, prompted or pushed? The answer to that question is a very firm NO. One only has to look at our Annual Scientific Meetings to see the quantity and quality of original work that is produced each year, but where does it go? Only a small proportion of it is ever published. It is amazing really, after all the work that authors put in to preparing their material for the A.S.M. In some cases, only a handful of their colleagues are on hand to hear them. At best only 10% of the Institute members have that opportunity. Why deny the other 90% of members their only chance of keeping abreast with new local developments by not submitting the material for publication? Why not get some recognition for your hard work?

So, to those who have presented papers at recent A.S.M.'s and not had them published, take them down from the shelf, dust them off and send them in for consideration for publication. To those who are preparing to give papers at future A.S.M.'s, send them for consideration afterwards.

We have many abbreviations in common usage in the English language. Let us add to the three R's the two P's — **PRESENT** and then **PUBLISH.**

Are there any active branches out there?

From the amount of information that comes from the local branches for all members nation-wide, it would appear that either they have an annual election of officers then hibernate for the rest of the year, or they are already dormant. With the local branches being centred on areas of densest population, there are many technologists who are isolated, in terms of distance from these centres, and receive very limited opportunities to participate. Technologists from Kaitaia to Wellington are interested in what happens from Nelson to Invercargill and vice versa. Branch secretaries, let's hear from you. Keep the whole country informed about what activities you have arranged for the year. There are 1600 avid readers of the Journal out there waiting for news of what is happening.

If your local branch is dormant, bordering on the extinct, get a group of colleagues together and get it going again. Make 1984 the year where the Institute really advances. Give it a high profile locally and show your non-member colleagues that the Institute and the local branches are worth joining and let us make the general public more aware of our profession.

So, to our two P's, add a third P — **PRESENT** and then **PUBLISH** and above all **PARTICIPATE.**

LETTERS TO THE EDITOR

Proposed Degree Course

Dear Sir,

In your October 1983 issue you printed the presidential address given by Mr A. F. Harper to the 39th Annual Scientific Meeting in August. In his speech Mr Harper was critical of the HBA for its unwillingness to support the proposal for the establishment of the proposed degree course in laboratory technology at Massey University. While I do not dispute his right to criticise, I believe that such criticism should be based on full, not partial, information. I would, therefore, be grateful if you would print this letter in the next issue of your journal; a copy has been sent to Mr Harper.

First, I am unaware of any requests by the Medical Laboratory Technologist Board to meet with the HBA Executive having been declined. A suggestion in 1982 for a meeting between the Association and the MLTB resulted in considerable factual correspondence between the two bodies and attendance by an HBA staff member at a meeting of the MLTB Education Sub-Committee. No request for a meeting with the Executive was received until 25 July 1983; this was responded to on 9 August 1983 when an invitation was extended to MLTB representatives to attend the next HBA Executive meeting (5 October). This meeting duly took place. I might add that this request of 23 July occurred after the HBA had given a very full statement of its position to the MLTB (letter dated 9 May) and after an informal meeting between the MLTB and Chief Executive in mid-July.

Secondly, it is not correct to claim that the HBA has obtained information "via the back door". A cross-section of hospital boards was circulated for its views after discussion with appropriate personnel; considerable information was obtained from the MLTB, and comment was received from Massey University. Assessment was then made of the course on the basis of the information received and in the context of the needs of laboratory services and employers' responsibilities.

Thirdly, Mr Harper makes no mention of the HBA's stated position that it supports the view that some review and formalisation of the present post-NZCS course, particularly in tuition methods, is needed, and has expressed its willingness to continue to liaise with the MLTB in encouraging it to examine other options which would be more acceptable in terms of the level of training, the cost to boards, the ramifications for other health groups, and manpower requirements.

In addressing the reasons given by the HBA for opposing the Massey degree proposal Mr Harper does not always present the total HBA viewpoint and reasoning. The Association has stated that international trends in technology education were not, **on their own** considered a justifiable reason for change in the New Zealand context. The Association has no argument with the MLTB view that international trends should be watched and followed if considered relevant to the New Zealand environment. It does not believe, however, that this relevance has been shown; in fact, such a course goes against the moves in other health-related science areas towards technical institute courses for initial training. Nor has it been shown that the level of training required can best be achieved within a university.

The costs of the proposal are not restricted only to the additional staff which would be required to man laboratories while students attend university. There will also be increased costs for paid study leave, course fees, examination fees, accommodation and travel costs. At a time of no financial growth these extra costs are very real problems for hospital boards trying to provide a total hospital service.

Flow-on effects to other health related groups cannot be ignored, again largely because of the possible cost factor. In addition to cost, however, the HBA has not been convinced on the evidence made available to it that the level of training required can by met only be means of a university degree course.

Specialisation of technologists at the senior level is already a problem for some boards, especially those having to roster staff to provide a 24-hour service. While the Massey course may result in specialist training in two subjects rather than one, the question remains as to whether this level of training is appropriate for the laboratory's requirements. In terms of changing technology and increasing automation it is probably too theoretical; in terms of practical laboratory experience it will reduce considerably that component currently achieved by the present system of "in-house" training.

Finally, much laboratory work is now automated, requiring more technical rather than more analytical and theoretical skills. Hence the HBA believes that technical institute or similar training is more appropriate than a university degree which may create unrealistic expectations of research work among trainees or prospective trainees.

The relevance of the university environment to laboratory requirements is again questioned. It may be that only a few technologists need to be trained beyond NZCS to meet the future needs of the laboratory. In this context manpower studies are essential if further discussion on training is to be realistic and productive.

In conclusion it is not the wish of the HBA to enter into acrimonious debate with Mr Harper, the NZIMLT or the MLTB. This letter is written to explain and clarify points made in Mr Harper's address. It has been the Association's approach to this matter to be open with the MLTB as to its position and thinking so that progress can be made towards a solution of what is recognised as being a problem area.

Yours faithfully

T. A. Riddell
Chief Executive

Dear Sir

In reply to the letter written by Mr Riddell Chief Executive of the Hospital Boards' Association regarding my criticisms of the HBA in my address to the 39th Annual Scientific meeting I would like to make the following comments.

As I recall, in 1981 a meeting was held in Wellington comprising representatives of the Medical Laboratory Technologists Board and Massey University. The main purpose of this meeting was to inform the HBA about the proposed Massey course. Representatives of the HBA did not attend the meeting.

As a result of a resolution passed at the 1982 July meeting of the MLTB the Chairman rang the Chief Executive of the HBA suggesting a meeting between the MLTB and the HBA Executive. This was reinforced in a letter dated the 26th July 1982. No meeting took place.

At the 1983 July meeting of the MLTB because up to this time the Board had only had contact with individual members of the HBA and not the full executive it was decided that a delegation from the Board should meet with Mr Riddell. The objective was to attempt to arrange a meeting with the HBA Executive. Mr Riddell undertook to inform the Board if this was possible in due course. During the Annual Scientific meeting in Napier I was informed by the Chairman of the MLTB that he had received a communication from the HBA agreeing to a meeting. This information was given to delegates.

In the fourth paragraph of Mr Riddell's letter he infers that I was withholding the information that the HBA supported the view that some review and formalisation of the present post — NZCS course was needed. In my education committee report published in the same issue of the NZ Journal of MLT as the presidential address this information is given to Institute members.

The other criticisms made by Mr Riddell regarding the proposed changes to Technologist education have either been covered in considerable detail in correspondence from the MLTB or at the meeting attended by representatives of the MLTB and the HBA executive in October 1983.

It is very disappointing to note from Mr Riddell's letter that the HBA continues in the main to disregard the submissions and advice of the two organisations most closely associated with technologist education the NZIMLT and MLTB.

I have no wish to enter into acrimonious debate with Mr Riddell however for the information of readers I feel it necessary to clarify the following points.

We have never said that overseas trends regarding university education are on their own justification for a change. They are however an important factor. Mr Riddell has not answered the question posed in my presidential address, in what way does medical laboratory technology in New Zealand differ from that which is practiced in other countries such as Australia where degree courses are available for technologists?

Formal post-NZCS education, because of the comparatively small number of students and the necessity to provide viable student class numbers would in the main require to be on a block course basis. Costs relating to the Massey course would also be incurred if we use the technical institutes.

Mr Riddell states that the educational proposal goes against moves in other health related science areas towards technical institute courses for initial training. In fact by retaining the NZCS our initial training is in the technical institutes.

Finally automation is NOT as wide spread though all the disciplines as Mr Riddell appears to believe and the amount of automation varies considerably from hospital to hospital.

In addition it is essential for problem solving and method development that we have Technologists who understand the principles, methods, and instrumentation of the tests they perform, and the significance of the results, as well as knowing how to push the buttons.

Yours sincerely

Alan Harper
President NZIMLT
11 Turere Place
WANGANUI

Medical Technology — A registered profession, occupation or just a job — A personal view of registration of Medical Laboratory Technologists in New Zealand.

Dear Sir,

Registration of Medical Laboratory Technologists in New Zealand is now into its second decade. This article is an attempt to put into perspective the amount of progress that has been made for the status of Medical Laboratory Technologists through registration. In doing so, I will start with a brief history of registration of Medical Laboratory Technologists in New Zealand.

On 15 December 1962 ministerial approval for the establishment of an examination board consisting of three members of the New Zealand Society of Pathologists and four from the NZIMLT was given. A full time Secretary was to be employed to establish and maintain a register of trainees.

In July 1963 in an article in the New Zealand Journal of Medical Laboratory Technology entitled 'On improvement in status' it was suggested that a registration board may be established to look after the interests of medical technologists in New Zealand. The multiple functions of the board would include arrangement and conduct of the examinations and also maintenance of a register of qualified technologists. It would replace the then existent examination board. The chief benefit of such a board would be an enhancement of the standing of our profession and on that ground we should welcome the proposal. It was also suggested that it may be more equitable all round and certainly of greater benefit to our prestige if we attempted to secure an act of parliament such as that governing the activities of pharmacists. On reflection it seems a tragedy that such a proposal was not pursued.

In April 1964 nominations for board members were considered and in the communication dated 8 May 1964 the Director General of Health confirmed the membership of the first Medical Laboratory Technologists' Board. The first meeting of the board was on June 5 1964. In those early days the boards main concern was the conduct of examinations and recognition of equivalent qualifications.

In November of 1968 the Institute was approached by the Health Department to look at state registration of medical technologists. In July 1969 a sub-committee was formed. In May 1970 it was said that the purpose of state registration as stated by the Health Department was a matter of protection both to the public and to registered technologists, in that they would prevent unqualified persons from practising. It would seem that once registered there would be nothing to stop a technologist from practising privately, although it would not be possible for him to claim on the social security fund. The eventual aim of the Department of Health is to replace the present Medical Technologists Board with a registration board which will be independent in financial and other respects and which will approve training centres, organise examinations and maintain a register. The secretarial duties will include receipt of applications for registration and the issue of annual practising certificates. The board activities will be financed by the fees paid for registration and all practising certificates and supplemented by funds appropriated from parliament.

In the Presidential address at the 26th conference of Medical Laboratory Technologists it was said that the registration board would have official recognition, public recognition and with that increased strength. In the months that followed there was a lot of lobbying for state

registration and the main point raised was one of the increase in status of our profession. In July 1971 a referendum on state registration was held and of the 560 technologists eligible to vote, 286 submitted votes, the results of which were:

In favour of state registration	255 or 89.2%
Against	13 or 4.5%
Invalid votes	18 or 6.3%

The sub-committee member for registration was commended for his efforts and told to proceed with the new legislation. In January 1973 the Dental and Medical Auxilaries Act 1966 was amended to cover medical technologists. The Medical Technologists regulations 1973 came into affect on 26 February 1973. The inaugural meeting of the registration board took place on 16 August 1973.

The main reason given for state registration was to enhance the standing of medical laboratory technology as a profession and it was my hope and that of a lot of my colleagues that through this increase in status we may one day be able to practice medical technology in our own right. Sadly 10 years registration has bought us no nearer this end.

The Medical Laboratory Technologists Board through registration, has given us a register or persons with qualifications which would enable them to practice medical laboratory technology. It also checks overseas qualifications for reciprocity and also organises the running of examinations. This could however have been done just as efficiently by the New Zealand Institute of Medical Laboratory Technology. Regrettably it appears also that we are no nearer a degree course in Medical Laboratory Technology and with the present political situation, education cuts are not likely to help the situation, indeed they may well sound its death nell. In 1971 when registration was being promoted technologists were told that there would only be a nominal cost involved with registration and indeed when the regulations came into force in 1973 this was the case. Since then however, the regulations have been amended several times to allow increases which in a recent letter from the Minister of Health dated 2 July 1982 was to enable Boards to be self sufficient or to become self sufficient. As the majority of Hospital Boards pay the annual license fees of the technologists they employ, it seems that the Health Department can transfer money from one department to another to achieve this end. Once a Technologist is registered and has paid the appropriate fee there should be no further work involved unless there happens to be a change of address or name or the unlikely event of a person being de-registered. Increases in registration fees to cover new registration should be expected as should increases in examination fees in-line with the rise in the cost of living.

The practice of annual licensing however in order to engage in medical laboratory technology when the Technologist concerned is responsible to another party, be-it the Hospital Board or Pathologist in the private sector seems rather ridiculous. Fair enough if the responsibility of the work done is that solely of the technologist, then we should be licensed, both to protect the public and our profession from unauthorised persons practising. I would like to see the Medical Laboratory Technologists Board take a stand for the people it represents and either give the Technologists the right to practice medical technology in their own right and with that access to the Social Security fund or otherwise amend the regulations by adding a clause to 12 Section 32 of the principle act, and that clause be E, "A registered technologist is responsible to or under the supervision of a member of the Society of Pathologists, Hospital Board or registered Medical Practitioner." This clause would not affect registration of Medical Laboratory Technologists but would sum-up pretty well the status of medical laboratory technology at the present time. One of the reasons for the declining membership of the New Zealand Institute of Medical Laboratory Technology may be an economic one, given the present situation in New Zealand. Having to pay an unnecessary license fee to engage in Medical Laboratory Technology no doubt has had an adverse affect on membership. It may well be time for the New Zealand Institute of Medical Laboratory Technology to become more involved in all aspects of medical laboratory technology.

Yours sincerely

Eric Oudyn
Hamilton.

Canadian Workload Units

Dear Sir,

It is with alarm and regret that I read of the proposal to cease collection of Workload Statistics on a national basis.

Upon introduction of the Canadian Workload Units in 1975 it was stated by the Committee responsible for their implementation that the units were a less than perfect measure of workload but that they felt it was better to implement them then, and work upon their improvement, than to delay implementation. What work was subsequently undertaken to ensure uniformity of application? Surely the real issue is not that some laboratories are theoretically achieving better than 100% productivity, but should have been one of ensuring uniformity of application. A problem which was in fact addressed by the T.A.C. with regard to Immunohaematology.

One of the greatest handicaps suffered by managers in the Health Care field is the lack of a productivity measure, both for internal use and for assessment in relation to like units. In laboratories we are fortunate in having an output that "can" be measured both quantitatively and qualitatively.

To cease collection of workload data under any circumstances is a remarkably retrograde step. To do so because the will does not exist to adapt and properly implement an adequate system is reprehensible.

It is my belief that if our Institute has any claim to credibility as anything but a trade union, then we ought to be vigorously pursuing this matter through the Pathology Advisory Committee and if necessary, to the Minister.

It is way past time that we wiped the sand from our eyes and recognised that laboratory management is at least as important as the scientific aspects of our profession.

Yours faithfully,

Tony A. Smale
Charge Technologist
Oamaru Hospital

ANZAC FELLOWSHIP

Philip Hill, a staff technologist at the Haematology Department, Middlemore Hospital has been awarded an A.N.Z.A.C. Fellowship for 1984.

A.N.Z.A.C. Fellowships are available for study or investigation in Australia and are granted to New Zealand citizens having sufficient background of study or experience to be regarded as likely to make a contribution to New Zealand through their careers. Three A.N.Z.A.C. Fellowships are awarded each year and enable holders to undertake a course or study for periods up to 12 months.

This award will enable Philip to work at the Royal Prince Alfred Hospital, Camperdown, Sydney for 6 months, under the direction of Drs. Ron Trent and Tom Exner.

With Dr Ron Trent, Philip will be working in the haemoglobinopathy unit at the hospital learning the techniques involved and interpretation of gene mapping. Special emphasis will be placed on the prevalence of alpha thalassaemia in the Polynesian, a reasearch project that Middlemore, National Womens and the Royal Prince Alfred are at the moment in collaboration with. By working with Dr Trent, Philip hopes to take a more active role in this project.

The time spent with Dr Tom Exner, a world authority on the identification of the lupus inhibitor, will also enable Philip to gain further knowledge and experience in this specific field.

New Zealand Institute of Medical Laboratory Technology Inc.

40th ANNUAL SCIENTIFIC MEETING

Dear Member,

Orwell wrote that 1984 would herald the end of the world as we know it. Why not live dangerously and come to Dunedin and see the world as it really exists. We are aware that many people do not realise that anything exists below Cook Strait or Christchurch. Be a true Medical Technologist, one with adventure in the blood and an enquiring mind and come to Dunedin and prove the disbelievers wrong.

Your friendly native guides on this adventure hope to have a number of delights for your edification and enjoyment.

Our guest speakers who come from places even further north than Auckland are:

Professor R. C. Burton, Professor in Surgical Science at the University of Newcastle, Australia. Professor Burton is an expert in the field of monoclonal antibodies and will be addressing the General Forum on Monoclonal Antibodies as well as the haematology and Histopathology/Cytology Forum on the more specialised uses of these antibodies.

Dr M. C. Stuart, Assistant Director (Scientific) of the Garvan Institute of Medical Research, St Vincent's Hospital, Australia.

Dr Stuart's main interest lies in the field of Monoclonal Antibodies related to ELISA techniques in Chemical Pathology (Hormones, Steroids etc.) and will be addressing the Biochemistry Forum on two occasions.

Dr C. Philpotts, Senior Lecturer (Histopathology), Bristol Polytechnic, England.

Dr Philpott's expertise lies with the use of resin embedding and he will also be able to discuss histological techniques using monoclonal antibodies.

These are the confirmed overseas speakers and the committee is currently negotiating for a guest speaker dealing with coagulation.

Accommodation for the visit to the deep south is relatively spartan by recent standards. We have obtained hostel accommodation 5 minutes walk from the lecture theatres, which are within the University complex. There are a limited number of rooms with two beds for those bringing their better or other halves. These will be allotted on the biggest bribe received. For the less fortunate however, do not despair, as the memories of certain scenes of debauchery in the Hamilton hostels will be well remembered by some. For the less hardy hotel/motel accommodation is available but will **NOT** be dealt with by the committee.

Being frugal minded Scots in Dunedin we have attempted to keep the cost of this trip of exploration to the minimum. For example the Registration Fee of $35 will include morning and afternoon tea and lunch each day. Your accommodation will cost you $16 a night and that includes breakfast. Dinner at night in the hostel will cost $4.25. Beat that if you can.

No mention has been made in this letter regarding the Conference Dinner (Booze-Up) for the simple reason that we are working on something special and so no final announcement can be made. Do not fret, you will not be disappointed.

The programme for the two days is ready in its skeletal form. It is now ready to be clothed in the muscles and guts of this meeting, i.e., YOUR CONTRIBUTION.

A Scientific Meeting is only as good as the papers presented to it and the committee urges you to get behind it and think about producing papers for it. Although the theme of the guest speakers is on Monoclonal Antibodies we hope for a wide range of papers to fulfil a wide range of members' interests. The committee has done its job of providing a venue, stimulating guest speakers and the chance to relax afterwards, it is now up to you to do the rest.

It only remains for us to invite you to come to Dunedin for the 40th Annual Scientific Meeting. The temperatures may be lower (according to the T.V.) but we hope that you will return home with warm memories of your Southern cousins.

Yours in technology,

The Conference Committee

BOOK REVIEW

Significance of Medical Microbiology in the Care of Patients — 2nd Edition.
Victor Lorian, M.D., Editor.
Published 1983 by Williams and Wilkins, Baltimore/London.
N.Z. Distributors: Australia and New Zealand Book Co. Pty Ltd., P.O. Box 33-406, Auckland 9.
Price $157.50

With Victor Lorian as Editor many prominent and accomplished experts from the field of medical microbiology have contributed towards a stimulating and thought provoking book entitled, "Significance of Medical Microbiology in the Care of Patients". The main purpose of the various authors is to give the reader guidelines on how best the needs of the patient may be met by those employed in medical microbiology. With the ever increasing cost of technology being matched by a decrease in financial resources, informed and intelligent comment on how best the services of a medical microbiology laboratory can be used is most welcomed.

Although those that have contributed to the book are based in the United States of America, the problems they have encountered would appear to be universal i.e., "The final goal of technologists entering the clinical laboratory should be a constant improvement of their understanding of their profession with opportunities to further their education rather than the goal of managerial dead-ends".

The first chapter addresses the relationship between clinical microbiology and healthcare, and emphasises the fact that technology should serve man and not the reverse. It was noted that in the microbiology laboratory staff are detached from the direct care of the patient and thus susceptible to becoming preoccupied with the scientific nature of the work and losing sight of the relevance of the information gained. This is where the book is of great assistance for not only are the technical aspects of microbiology evaluated but their significance is also closely scrutinized. How far and how fast to go with bacterial identification is covered. The significance of isolates from such sites as blood, sputums, wounds etc is extensively evaluated with information from numerous surveys given.

An analytical approach is then given to such pertinent subjects as the predictive value of microbiological diagnostic tests and the systematic means required to conduct technological evaluations. The usefulness of enzyme-linked immunosorbent assays is assessed, as is infection control and the methodology for antibiotic sensitivity testing. Finally there is a very detailed account of the incidence of bacterial types in the United States of America and present trends.

In summary the book provides much useful information for both technologists and physicians and if these two groups can continue to communicate their requirements to teach other, medical microbiology will continue to play a major part towards the care of patients.

B.L. Dove

Current Topics in Inflammation and Infection.
Monographs in Pathology Series
Guido Majno, M.D., Ramzi S. Cottran, M.D., and Nathan Kaufman, M.D., Editors.
Published 1983 by Williams and Wilkins, Baltimore/London.
N.Z. Distributors: Australia and New Zealand Book Co. Pty Ltd, P.O. Box 33-406, Auckland 9.
Price $103.50

The monograph deals briefly with a historical review, but the main emphasis reflects recent developments in the understanding of the inflammatory process and the application of recent knowledge. As the text reviews only selected aspects of research it is not recommended for the uninitiated, however, a comprehensive list of references is provided for the enthusiatic reader.

R.D. Peterson

Answers to Porphyrin Questions

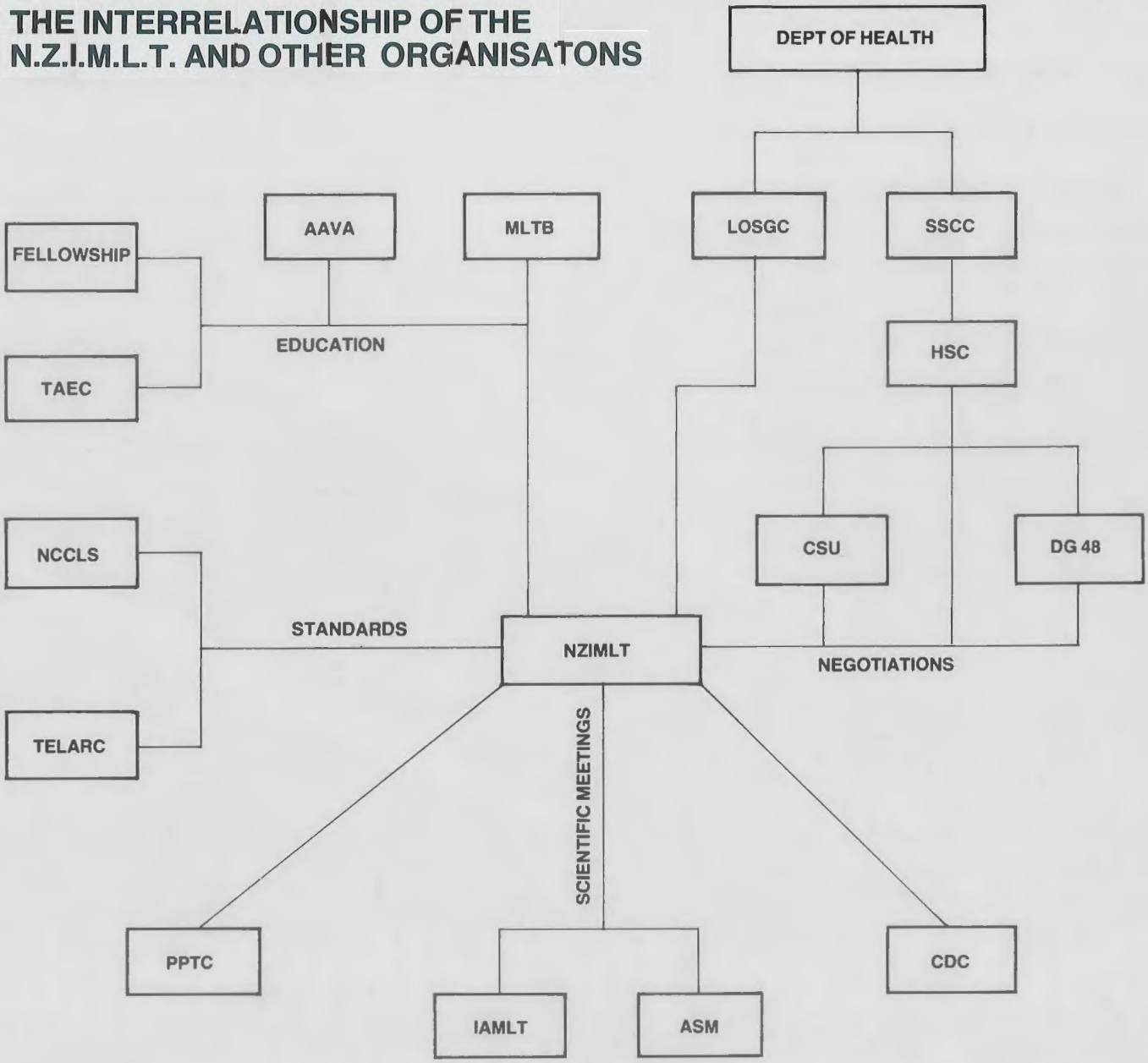
Case 1.
This is a young adult with a history of recurrent abdominal pain with no obvious cause, although the most recent episode is possibly associated with the taking of oral contraceptive drugs. This suggests a possible diagnosis of acute prophyria. A positive PBG screen and normal faecal porhyrin levels confirms that the diagnosis is acute intermittent porphyria.

Case 2.
An alcoholic presenting with signs of photosensitivity suggests a diagnosis of porphyria cutanea tarda. A negative PBG and red cell screen is consistent with this diagnosis, but if the strategy outlined in Figure 3 is followed, a negative faecal screen would suggest that this is not a case of porphyria. Faecal porphyrins may be normal in cases of porphyria cutanea tarda and an elevated urine porphyrin (uroporphyrin > coproporphyrin) would be needed to confirm this diagnosis.

Case 3.
A history of photosensitivity and a positive red cell porphyrin screen suggests erythropoietic porphyria. With increased levels of protoporphyrin found in the faeces and red cells, the diagnosis of erythropoietic protoporphyria can be made.

4. Case 4.
This patient presents with signs and symptoms suggestive of an attack of acute porphyria. This is confirmed by the positive PBG screen, suggesting either acute intermittent porphyria, porphyria variegata, or hereditary coproporphyria. A positive faecal porphyrin screen rules out acute intermittent porphyria. A history of photosensitivity not associated with acute attacks and a faecal protoporphyrin level greater than the level of coproporphyrin confirms the diagnosis of porphyria variegata.

THE INTERRELATIONSHIP OF THE N.Z.I.M.L.T. AND OTHER ORGANISATONS



SSCC	State Services Co-ordinating Committee
HSC	Hospital Services Committee
CSU	Combined State Unions
DG 48	Committee to negotiate standard conditions (DG 48)
MLTB	Medical Laboratory Technologists Board
AAVA	Authority for Advanced Vocational Award
Fellowship	NZIMLT Fellowship Committee
TAEC	Technical Assistants Examination Committee
NCCLS	National Committee on Clinical Laboratory Standards (USA)
TELARC	Testing Laboratory Registration Council of NZ
PPTC	Pacific Paramedical Training Centre
IAMLT	International Association of Medical Laboratory Technologists
ASM	NZIMLT Annual Scientific Meeting Committee
CDC	Centre of Disease Control (USA)
LOSGC	Laboratory Officers Salary Grading Committee

INSTITUTE BUSINESS

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

President
A. F. Harper
11 Turere Place, Wanganui

Vice-Presidents
C. Campbell
K. McLoughlin

Secretary
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, 1983 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.

INSTITUTE CALENDAR

1984	FEB 24	Applications close for Fellowship and QTA examinations
	MAR 7/8	Council Meeting
	APRIL 9	Deadline for sub-committee balance sheets and ledgers to be with the Treasurer
	APRIL 29	Committee Reports for Annual Report to be with the Secretary
	MAY 15/16	QTA examinations
	MAY 18	Proposed Rule changes and Remits to be with the Secretary
	MAY 23/24	Council Meeting
	JUNE 16	Nomination forms for the election of Officers and Remits for the Annual General Meeting to be with the Membership (60 days prior to AGM)
	JULY 3-5	Fellowship examination
	JULY 7	Nomination forms for the Election of Officers to be with the Secretary (40 days prior to AGM)
	JULY 15-20	IAMLT Congress, Perth
	JULY 26	Ballot papers to be with the Membership (21 days prior to AGM)
	AUGUST 2	Annual Report and Balance Sheet to be with the Membership (14 days prior to AGM)
	AUGUST 9	Ballot papers and proxies to be with the Secretary (7 days prior to AGM)
	AUGUST 14/15	Council Meeting — Dunedin
	AUGUST 16	AGM and SGM — Dunedin
	AUGUST 16/17	Annual Scientific Meeting — Dunedin
1985	AUGUST	NZIMLT ASM — Palmerston North
	AUGUST 28-30	AIMLS ASM Launceston, Tasmania
1986	AUGUST 18-22	2nd South Pacific Congress — Sydney

Correspondence

Council sent the following letter to the New Zealand Society of Pathologists regarding TELARC Registration and salaries for private laboratory workers. The Society of Pathologists' acknowledgment has been received.

Dr P. Pedlow
President
New Zealand Society of Pathologists
Royston Laboratory
Knight Street
HASTINGS

Dear Peter

I am writing to you regarding two matters arising out of discussions at our Institute's recent Annual Scientific Meeting.

1. **TELARC Registration**
   It is the feeling of Council that the Department of Health should be requested to eventually require all laboratories (both hospital and private) to have TELARC registration. Before writing to the department I have been requested to seek the views of the Society and ask if they would be prepared to support such a proposal. Council does not see this happening overnight but feels the department should indicate that by a certain date (even eight or ten years hence) all laboratories must be registered.
2. **Salaries for Private Laboratory Workers**
   At a meeting of the staff of private medical laboratories Council was unanimously requested to approach your Society regarding negotiating a minimum salary scale for private laboratory workers. It would appear from that meeting that there is tremendous variation in the salaries paid currently to private laboratory staff. Some laboratories use the hospital salary scales while others are paid either above or below it. It is not the intention of Council to interfere with the rights of employees to negotiate directly with their employers but the meeting felt that there would be some advantage in establishing a minimum scale which could then be used in negotiations with the Department of Health over payment of fees for laboratory tests.

I look forward to hearing from you regarding these two proposals. Kind regards.

Yours sincerely
NZIMLT

B. T. Edwards
Secretary

Mr B. T. Edwards
Secretary
New Zealand Institute of Medical Laboratory Technology
Haematology Department
Christchurch Hospital
Private Bag
CHRISTCHURCH.

Dear Mr Edwards

Your letter of the 30th August to Dr Pedlow has come to me for reply. Your letter has been passed to the secretary of the Society to be discussed by the Executive in due course.
With kind regards.

Yours sincerely

W.L. Kenealy
President
New Zealand Society of Pathologists

Below is a letter received from a Waikato member thanking the Council for assistance in solving a local issue. Council is only too willing to advise and assist members to solve these problems. If you are unable to solve problems locally, do not hesitate to write to Council.

"I am writing to let you know that myself and all others concerned, received back pay for our N.Z.C.S. exams which the Waikato Hospital Board initially refused to pay. I have to thank you for your letter stating you would take it up with the Board directly if no action was taken soon. On seeing this letter, the Board promptly decided to change their minds and have now paid us."

CSU NOTES

ANNUAL AND LONG SERVICE LEAVE

The following letter of 14 December has been received from the Institute of Medical Laboratory Technology:-

"Following the AGM of our organisation and a recent Council meeting I have been requested by Council to refer two remits to you for comment and consideration for negotiation with S.S.C.C.

1. It was resolved "that council be requested to seek an extension of the long service leave entitlement after 20 years service from one month to three months."
2. It was resolved that Council attempt to have the regulations amended for the granting of four weeks annual leave and long service leave so that they are based on accumulated service rather than continuous service.

Applications for Membership as at 28th November 1983

Miss W. P. Duffin*	Mem	Auckland
Miss H. L. Angove	Mem	Waipukurau
Mrs A. G. D. Bennett	Mem	Tauranga
Ms J. V. Cleave	Mem	Napier
Ms H. Dent	Mem	Whangarei
Mrs J. L. Diprose	Mem	Auckland
Miss J. L. Eagle	Mem	Dannevirke
Mrs D. E. Farr	Mem	Auckland
Miss P. M. Frawley	Mem	Tauranga
Miss R. L. Harrison	Mem	Hastings
Miss S. E. Kettle	Mem	Napier
Ms G. Kirkby	Mem	Dunedin
Ms J. M. Knudson	Mem	Saudi Arabia
Mrs L. A. Levenbach	NP	Nelson
Mr G. B. Lovatt	Mem	Whangarei
Miss B. A. McCool	Mem	Napier
Mrs D. J. Nisbet	NP	England
Miss C. J. Norwood	Mem	Napier
Miss A. L. Owen	Mem	Lower Hutt
Miss J. T. Sheppard	Mem	Wellington
Miss G. M. Stevenson*	Mem	Waipukurau
Miss T. J. Tooker	Mem	Dannevirke
Mrs S. Vail	Mem	Napier
Miss A. J. Vass	Mem	Napier
Miss B. Watene	Mem	Auckland
Mrs E. E. Abbott	Mem	Wellington

* Complimentary

Resignations

Mrs C. K. Dimattina	Wellington
Mrs L. Keesing	Hamilton
Mrs K. E. Minnee	Auckland
Mrs S. Sims	Palmerston North
Mr C. J. Wilson	Auckland

Gone No Address

Miss C. Mercer	Auckland

Applications for Associateship

Mrs B. A. Liardet	Lower Hutt
Mr N. G. Wood	England
Mrs Y. M. Wood	England

Membership Sub-Committee Report November 1983

Membership

Since our August meeting there have been the following changes:

	November 83	August 83	November 82
Membership as at 28th November	1389	1383	1464
LESS Resignations 5, G.N.A. 1	6	68	12
	1383	1315	1452
PLUS Membership Applications 28			
Listing Adjustments 11	39	74	18
TOTAL MEMBERSHIP:	1422	1389	1470

Our Membership summary is as follows:

Hospital Laboratories	1012	1000	1050
Other Government	61	60	59
Private Medical Laboratories	174	158	169
Other Employment	27	27	24
Non-Practising	105	100	108
Overseas	39	40	52
Unknown Employment	4	4	8

181 members remain unfinancial (180 last year), and 105 members partly financial (28 last year)

AWARDS AND PRIZES

Examination Prizes

These are all subject to the restriction that the candidate must obtain an aggregate 'B' pass or greater in the Board's Examinations and be a financial member of the Institute.

(1) NZIMLT Examination Prize for Qualified Technical Assistants — $50
Donor — N.Z. Institute of Medical Laboratory Technology
Subjects — Clinical Biochemistry
Medical Cytology
Histological Technique
Microbiology
General

(2) N.Z. Blood Foundation Prize for Qualified Technical Assistant — $50.
Subjects — Immunohaetolgoy
Haematology

(3) Medical Technologist Board Part II Examination Prizes — $50.
Donor —

Clinical Chemistry	— Rochc Products NZ Ltd
Haematology	— Kempthorne Medical Supplies Ltd
Histology	— Kempthorne Medical Supplies Ltd
Immunohaematology	— Technicon Equipment Pty Ltd
Immunology	— Hoescht NZ Ltd
Microbiology	— Roche Products NZ Ltd
Medical Cytology	— Ortho Diagnostic Systems
Nuclear Medicine	— Sci Med (NZ) Ltd
Cytogenetics	— Sci Med (NZ) Ltd

(4) Medical Technologist Board Part III Examination Prizes — $100

Clinical Chemistry	— Watson Victor Ltd
Haematology	— General Diagnostics
Histology	— Sci Med (NZ) Ltd
Immunohaematology	— Medic DDS Ltd
Immunology	— Hoechst NZ Ltd
Microbiology	— Wilton Scientific Ltd
Virology	— GIBCO NZ Ltd
Medical Cytology	— Ortho Diagnostic Systems
Nuclear Medicine	— Ortho Diagnostic Systems
Cytogenetics	— Sci Med (NZ) Ltd

Journal Awards

Restricted to financial members of the Institute. The sum of $200 awarded biennially for the best original or review article in the following categories:

(1) NZIMLT Journal Student Award
(2) Roche Products Microbiology Award
(3) Roche Products Clinical Chemistry Award
(4) McGaw-Dade Haematology/Immunohaematology Award
(5) Hilder Memorial Prize for best technical communication
(6) NZIMLT Journal Prize — for subjects not covered above.

Travel Awards

Application forms are available from the Secretary NZIMLT. Winners are announced at the Annual Conference.

(1) Wellcome NZ Ltd/NZIMLT International Travel Award
$500 donated by the NZIMLT and the balance donated by Wellcome NZ Ltd for single return airfare plus accommodation to assist an NZIMLT Fellow or Associate member to attend the bi-ennial Congress of the IAMLT as official delegate.

(2) The NZIMLT Scholarship
$500 donated annually by the NZIMLT for research or to attend an overseas scientific meeting. Applications close 1st July.

International Award

The Ortho Diagnostic Systems Educational Award. The purpose of this award is to further the education of qualified technologists who are members of the International Association of Medical Laboratory Technologists by sponsoring their attendance at a one-week Case Study Clinic or Applied Blood Banking Course. All expenses, including air transportation and living costs are paid by Ortho Diagnostics.

Further information is available from Martin Fraser, Ethnor Pty Ltd, P.O. Box 9222 Newmarket, Auckland.

MLTB NEWS

Newsletter

At our recent December meeting of the Board it was decided that we would publish a newsletter following each Board meeting. This is in no sense replacement for the formal and official channel by which Board instructions and directives are given — these will still be promulgated through the Secretary's circular which will be published as and when required. Nor is it meant to create a new communication channel to the Board — this should still be through the Secretary, although I am always happy to be contacted about any special problems.

Date of Board Meetings

We intend to alter slightly the dates of the Board meetings and along, with this make minor alterations to what will be discussed at various Board meetings. In particular we would use the February Board meeting to discuss the recommendations in the examiners' reports which are received at the December meeting. Following the February Board meeting, directives will be given to all laboratories setting out the exam procedures which will be used for that year. These instructions will remain in force for the year and no changes (e.g. syllabus change or exam format change) will be made before the next February. The examiners' reports and reports on failed candidates will be sent out as soon as possible after the February Board meeting. The Board feel that the examination system should not only test the candidates of that year but should also be used for educative purposes and the perusal of examiners' comments is one major way in which this can happen. Appended to this newsletter, for instance, is a list of the percentage passes for the recent examinations and we would draw particular attention to the low pass rate in immunohaematology and ask all those involved in training in immunohaematology to be aware of these. Examination results will still be ratified at the December meeting and we would intend that results are posted within a day or two of the meeting.

For the information of laboratories, Board meetings will be held next year on the 7th—8th February, 31st July — 1st August and the 4th—5th December.

Late Exam Applications

This year the Board circulated its policy regarding late exam applications. Because of the rather short notification the policy was not strictly enforced this year but opportunity is taken now to reinforce to the laboratories and training staff that next year **NO LATE EXAM APPLICATIONS WILL BE RECEIVED.** The Boards' Trainee Handbooks have application forms in them and there is no reason why exam candidates should not have their applications in on time and allow the very tight schedule the Board has to work with to flow evenly.

Pre-requisites for Sitting Specialist and Certificate Examinations.

All trainees should note carefully that the certificate (Part II) examination may not be attempted until the N.Z.C.S. (Medical) is totally complete. We often have applications from applicants who have completed all of their N.Z.C.S. except Communications English. This subject is part of the N.Z.C.S. and we will not accept an N.Z.C.S. which is not complete as a pre-requisite for the Part II.

Handbooks

The handbooks for the 1984 intake of trainees will be available very shortly after the February Board meeting. Principal and Charge Technologists are reminded of the need to distribute these handbooks and also the need to ensure that the work log at the back of the book is kept up to date. The Boards' policy is such that we **may** request log books to be forwarded as evidence of work covered.

Massey Degree Course

The request from the Board to the Hospital Boards' Association that they support our application for the Massey Degree Course as a formal means of education for the final two years of our course has, once again, been declined by the Hospital Boards' Association. Although this is a disappointment to the Technologists' Board, we are nevertheless committed to continuing to try and implement a formal course and it is our intention in the New Year to present a statement to the Minister of Health outlining this need for the latter part of our course and setting out the pros and cons of the various tertiary educational

Percentage Pass Rate 1979 — 1983

SUBJECT	1979	1980	1981	1982	1983
Haematology Certificate	90	85	85	81	84
Haematology Specialist	67	82	88	58	80
Immunohaematology Certificate	91	85	73	65	48
Immunohaematology Specialist	70	100	50	83	50
Clinical Biochem Certificate	91	71	70	74	68
Clinical Biochem Specialist	59	71	47	68	67
Microbiology Certificate	84	84	83	93	96
Microbiology Specialist	100	88	81	79	89
Histology Certificate	75	100	100	100	100
Histology Specialist	—	100	0	100	0
Immunology Certificate	50	100	100	100	100
Immunology Specialist	100	100	100	100	50
Nuclear Medicine Certificate	100	80	NC	100	100
Nuclear Medicine Specialist	NC	NC	NC	NC	0
Cytology Certificate	NC	100	NC	100	100
Cytology Specialist	NC	NC	NC	NC	100
Virology Certificate	NC	NC	100	100	NC
Virology Specialist	NC	NC	NC	50	100
Cytogenetics Certificate	NC	NC	NC	100	83
Cytogenetics Specialist	NC	NC	NC	NC	100

NC = Either no examination offered or no candidates.
NOTE: Partial passes have been recorded as FAIL

bodies and their ability to be able to offer a suitable course. The board will use the expertise of the Department of Health and the Department of Education to draw up these comparisons and it will also make use of the Management Services Research Unit to gain information about our requirements for the future.

Annual Licensing

Commencing in 1984 we hope to have the accounts for annual practising certificates out in Mid-February so that registered technologists have plenty of time to pay by the due date — 1st April. On the 30th April, registered letters will go to all licenciatcs who have not paid. On the 31st May those who still remain without a current annual practising certificate will be prosecuted in the terms of the Dental and Medical Auxiliaries Act. There will be no input from the Medical Technologists' Board as such to this programme other than the Secretary advising the Office Solicitor of those people who have not paid. I would strongly urge all licenciates to ensure that they pay in plenty of time to avoid the unpleasantness of prosecution.

General

May I take this opportunity of thanking all those who have contributed to the smooth running of the Board, particularly in the assistance given for the in-house practical examination. I would take this opportunity of wishing all laboratory staff a very Merry Christmas and a very Prosperous 1984.

D. J. Philip
CHAIRMAN

Medical Technology in Papua New Guinea

If New Zealand Medical Technologists are interested in ways and means of assisting laboratories and laboratory personnel in the the Islands of the Pacific, it is important that before aid is given some understanding of the existing conditions and training schemes in each area is necessary in order that the aid offered is directional and acceptable.

Margaret Johnson has written the following article which provides an insight into the problems of Medical Technology in Papua New Guinea.

"I read with great interest, Ted Norman's article on overseas aid. I concur with much of what he says, especially with the statement that people are promoted too quickly or pushed beyond their level of competence. Also because of staff shortages, inappropriate people are selected for overseas courses.

To give some idea of the problems we face in PNG some historical background is necessary.

1. It is government policy to provide a health service to the 3 million people of PNG, with a concerted effort directed towards the rural areas.
   Access to much of the rural area is difficult because of mountainous terrain, the various outlying island groups and/or lack of roads.
   Communication with the bulk of the rural people is also difficult because of the 700 place languages in existence. Consequently these people have only a rudimentary

knowledge, if any at all, of basic hygiene.
The towns and cities contain a small but vocal elite who demand the "sophistication" of the developed world. The growing squatter settlements surrounding the towns also create additional problems.

2. The Public Service is responsible for implementing government policy. Since Independence in 1975, it has grown enormously becoming unwieldy and full of bureaucratic red-tape in the process. There have been efforts in the last year to reduce the overall size of the PS. One way of achieving this has been to make the creation of all new positions as difficult as possible.
3. The Health Department of the Public Service is responsible for providing the health service to the people:
   (a) Health H.Q. is based in Port Moresby and is responsible for the overall planning and co-ordination of this service, together with other related services such as training of para-medical personnel. Often in the past, these two divisions of the Health Department have not worked closely together.
   (b) The everyday implementation of the health care service (including the laboratory services) is the responsibility of the individual 20 provinces into which PNG is divided.
4. PNG has 2 well established, well funded universities, graduating large numbers of academically trained people, who then require the attendant technical back-up. Port Moresby General Hospital is the teaching hospital for the Medical Faculty of University of Papua New Guinea. This Faculty has achieved some international standing, consequently there is pressure to maintain and improve on this, resulting in pressure on Port Moresby General Hospital (including the laboratory services) to provide bigger and better services.
5. Port Moresby General Hospital Pathology is considered the Health Department's reference laboratory for PNG because it has the largest range of facilities. Its reference role is to advise and assist when requested, the provincial laboratories, but not to direct their activities.
   The training of technical personnel has not kept abreast with the demand of the senior clinicians of Port Moresby General Hospital; or for that matter, with the demand of medical officers in the other 19 provinces. For example the Biochemistry Department of Port Moresby General Hospital is carrying out approximately 5,000 tests/month (this includes the usual range of basic quantitative tests — all manual techniques) with a staff of 1 technologist and 3 technicians. The staff also have the added burden of training and supervising students at all levels during their bench practice.
6. The College of Allied Health Sciences (CAHS) conduct 3 courses in Medical Technology which supposedly equate with 3 levels (A, B and C) of the WHO recommended categories for technical laboratory personnel.
   (a) The 3 year Certificate course (WHO category B) has been running for approximately 15 years. Prior to this , a small number of personnel were sent to Fiji for training. All basic subjects and laboratory disciplines are taught by lecturers attached to the CAHS which comes under the Training Division of Health H.Q. (Prior to 1980, the sevice technologists taught laboratory disciplines, until service workloads became too onerous). The first year of the course is composed of full time formal lectures/practicals in basic science subjects. The second and third years are spent on laboratory disciplines with 40% of the academic year spent on formal lectures/practicals and 60% on bench training in the Port Moresby General Hospital Pathology laboratories. The qualifications for this course are preferably Grade 12 (Bursary or Scholarship Level) or alternatively Grade 10 (somewhere between School Certificate and University Entrance Standard, with credits in Maths, Science and English).
   In my view this course produces technicians who are, and will be in the forseeable future, the backbone of the laboratory services.
   To date a total of approximately 65 technicians have graduated. Approximately 25% of these have been lost to the Health department for various reasons, e.g., lack of finance, competition from the University, lack of incentive or the attraction of running a trade store. Many more than this are required to adequately staff the country and to give the necessary back-up service to technologists.
   (b) The 9 months Laboratory Assistants course (WHO category C) was commenced in 1975 with WHO assistance (especially that of Dr N.U. Roo). It was envisioned that these assistants would be trained in an environment away from the "sophisticated" centres, by national tutors; then posted to the smaller hospitals and health centres, so providing a basic service to the rural periphery. For the last few years this course has been conducted in Rabaul and there has been no monitoring of the standard of assistant produced from these courses by experienced personnel from Port Moresby. It has been found that many assistants already posted to health centres have difficulty in creating work for themselves and in remaining motivated because of their isolation. This has resulted in many of them being brought back to the base hospitals and carrying out tests for which they have not been formally trained, e.g., quantitative biochemistry.
   (c) The 2 year Diploma Course (WHO category A) was commenced in 1975. There is an entrance examination and those eligible to sit must have been registered as Technicians for 2 years. This course is not run concurrently because of a lack of candidates that can be released from service commitments and a lack of teaching staff. This course has a 50:50 ratio between lectures and bench training. At present the 3rd course is underway (with a possible 8 graduates for the Health Department). The previous courses produced 3 Graduates; others were attracted to other institutions by better conditions of service. In theory the graduates of this course can localise all expatriate technologists at present in Papua New Guinea.
7. The Health Department have contributed to government policy of reducing the size of the PS, by not having an intake of students to both the Assistants and the Certificate course in 1983. This was done despite critical shortages of technicians throughout the country. It is hoped that the Certificate course will resume in 1984, but as yet there has not been a definite decision made by Health H.Q.
8. The difficulties of improving the standard of the Certificate and Diploma courses are:
   (a) lack of expertise in the use of oral and written English. English is the second or third language of most Papua New Guineans.
   (b) lack of basic science and maths background obtained in secondary schools.
   (c) lack of finance to adequately equip the teaching labs.
   (d) lack of funds to recruit experienced expatriate staff.
   (e) lack of availability of experienced expatriate staff.
9. Often contract and volunteer expatriates are poorly briefed prior to coming to Papua New Guinea. When they do arrive, and during their term, they are not given clear directives as to what is expected of them. (In some cases technologists have tried to introduce methods inappropriate to the country). These expatriates are given "charge" technologist positions with little idea on how to manage staff or a laboratory, sometimes with the added racial problems; (often because of lack of continuity at this level, the labs become totally disorganised, resulting in the sub-ordinate national staff becoming resentful towards new expatriates with their "new ideas"). In many cases because of lack of technical staff, expatriates are used as "an extra pair of hands" at the bench rather than contributing to the overall planning, improvement of standards and training.
   At present there are 4 expatriate technologists out of a total of 7 in the country — 2 Port Moresby General Hospital, (Haematology, Biochemistry), 1 Madang, 1 Lae. In my opinion there should be a minimum of 20 technologists in the service area at the present time.
10. There have not been funds for many years for:
    (a) more experienced technical staff, especially from Port Moresby to travel to the outstations to assist and advise on standards and techniques or (b) outstation staff to participate in refresher course in Port Moresby General Hospital Pathology. Consequently staff moral is very low.

Obviously many of these problems will only be overcome with a well planned policy by the Health Department over a period of many years."

Margaret's suggestions for ways in which assistance could be given were published in a recent edition of the N.Z.I.M.L.T. Journal (Vol. 37 No. 4).

Michael Ballinger O/C Medical Technology at the Papua New Guinea Department of Public Health College of Allied Sciences writes:

"Briefly, Medical Technology is a live and well in Papua New Guinea despite financial restraints. Oversea Aid is welcome, but at this stage more research is necessary to determine our exact needs. However we are short of staff and this is why I am primarily writing. Two positions are vacant now.

SCIENTIFIC OFFICER (MEDICAL TECHNOLOGY) CLASS 2

Qualifications
Diploma in Medical Technology or a Degree in Science with specialisation and experience in Medical Laboratory Technology in hospital particularly in **Clinical Chemistry** (Biochemistry).
Duties
To assist the Officer in Charge of Medical Technology Diploma and Certificate courses in the organisation, planning, implementation teaching/learning and evaluation of courses for Medical Laboratory Technology.
Salary
See below.

SCIENTIFIC OFFICER (MEDICAL TECHNOLOGY) CLASS 2

Qualifications
Diploma in Medical Technology or a Degree in Science with specialisation and experience in Medical Laboratory Technology in hospital particularly in **Haematology** and **Immunohaematology.**
Duties
To assist the Officer in Charge of Medical Technology Diploma and Certificate courses in the organisation, planning, implementation, teaching/learning and evaluation of courses for Medical Laboratory Technology.
Salary
K16,020, plus 24% gratuity p.a. (N.Z. = 28,000) p.a.
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Leave fare every 18 months.
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Beta/pak® contains three differential reagents — ADP, soluble collagen and ristocetin — for the qualitative evaluation of platelet function and the detection of von Willebrand Factor. Each reagent is formulated to the optimal concentration for reliable platelet aggregation studies with platelet rich plasma.

This configuration of reagents is designed specifically for the differential diagnosis of von Willebrand Syndrome and qualitative platelet function abnormalities. The kit precludes the need to assemble these specific reagents from various sources. All three are lyophilized and the contents are readily reconstituted with distilled water. The kit contains one (1) vial of each reagent. ADP and collagen are stable for 30 days when reconstituted and stored at 2-8°C. Ristocetin is stable for 7 days at 2-8°C or may be frozen at -20°C for long term storage. Beta/pak® reagents will perform satisfactorily with any platelet aggregometer.

The reagents are packed in convenient quantities to limit waste and for easy storage in the convenient kit. Detailed package literature facilitates the interpretation of aggregation results for differential diagnosis.

Beta/pak® is clinically useful, economical, convenient and reliable for routine platelet aggregation studies for the differential diagnosis of platelet dysfunction and von Willebrand Syndrome.

For further information on Beta/pak®, contact: Bio/Data Corporation, 3615 Davisville Road, Hatboro, PA 19040 U.S.A., (215) 441-4000 TLX 834482. Please include your title and laboratory affiliation, or **CIRCLE 1 on the Readers Reply Card.**

AUTOMATED SLOPE READER FIRST-OF-ITS-KIND CAPABILITY IN PLATELET AGGREGOMETRY

Bio/Data Corporation has announced that its Platelet Aggregation Profiler® Model PAP-4, four channel platelet aggregometer, features an automated slope reader. This capability is a first-of-its-kind advance in aggregometer technology.

Slope value is an important parameter for the construction or development of a standard activity curve and for the quantification of plasma von Willebrand Factor levels. Platelet Aggregation Profiler® Model PAP-4 is programmed to continuously measure the rate of reaction as it is occurring. The Slope Reader determines the portion of the curve where the reaction is occurring at its maximal rate and calculates the slope for this portion of the reacton. Slope value is automatically printed-out at the end of the test.

This state-of-the-art parameter eliminates the need to manually measure slope, thus minimizing subjective variation from test to test and saving technician time. Model PAP-4 provides slope measurement automatically-microprocessor controlled — so that there are no additional equipment or procedural requirements. Four tests can be run simultaneously.

For further information on the Platelet Aggregation Profiler®, Model PAP-4, contact: Bio/Data Corporation, 3615 Davisville Road, Hatboro, PA 19040 U.S.A., (215) 441-4000 TLX 834482. Please include your title and laboratory affiliation or **CIRCLE 2 on the Readers Reply Card.**

BECKMAN OFFERS KINETIC SYSTEMS FOR UV/VIS SPECTROPHOTOMETER

Three accessory systems for the LDU®-6 UV/Vis Spectrophotometer combine temperature control, data storage and enzyme activity calculations for complete kinetic analysis. Available from Beckman Instruments, Inc., the kinetic systems provide efficient rate analysis with maximum throughput.

The rate is calculated over the operator-chosen time span, using linear regression for high accuracy. Statistical analysis verifies the linearity of the reaction. Because all data are stored, the rate can be recalculated quickly using a different time span. Simply enter a factor, and the rate is converted to enzyme activity.

Kinetics I provides complete rate calculations for a single sample. A water-regulated single cell holder can be used to achieve temperature control.

Kinetics II enables simultaneous rate analysis of as many as six samples. It includes a six-position cell holder with water-regulated temperature control. In addition to rate calculation capabilities, the system enables the subtraction of one reaction from another and the calculation of the rate on the difference. This feature allows blank correction or comparison of two different samples.

Solid-state Peltier electronic temperature control provides accuracy and convenience on the Kinetics III System. As many as six samples can be analyzed simultaneously. Data from all six of the samples are stored and used for the rate calculations described for Kinetics II.

For more information, contact Alphatech, Phone 770-392, Auckland, or **CIRCLE 3 on the Readers Reply Card.**

BECKMAN'S 4-ELECTROLYTE SYSTEM: FIRST WITH SOFT-KEY CONTROL.

Beckman Instruments, Inc.'s System E4A Electrolyte Analyzer utilizes the proven capabilities of the company's ASTRA® Systems and introduces multi-function soft-key control in a compact bench-top instrument.

Five multi-function soft keys incorporated into an easy access touch control key pad puts all System E4A functions at the operator's fingertips. Simple procedures set up routine runs on serum, urine, cerebrospinal fluid or other biological fluids.

The System E4A simultaneously analyzes Na, K, Cl and CO_2. The system automatically processes up to 100 samples per hour and calculates anion gaps to provide as many as 500 electrolyte answers per hour. It has a STAT-interrupt feature that enables the user to interrupt a routine run for a STAT analysis, after which the instrument resumes the routine run.

System E4A requires only 50 μl of sample for all four parameters, making it ideal for pediatric and geriatric testing.

All results appear on the system's digital display as well as on a hard-copy paper printout. Automatic flags identify short samples or out-of-normal-range samples.

The System E4A utilizes the ASTRA time-tested Ion Selective Electrode (ISE) methods for Na and K determinations and introduces an ISE method for Cl as well. The CO2 parameter used the reliable differential pH methodology.

For more information, contact Alphatech, Phone 770-392, Auckland, or **CIRCLE 4 on the Readers Reply Card.**

BECKMAN ADDS CARBAMAZEPINE TO TDM SYSTEMS

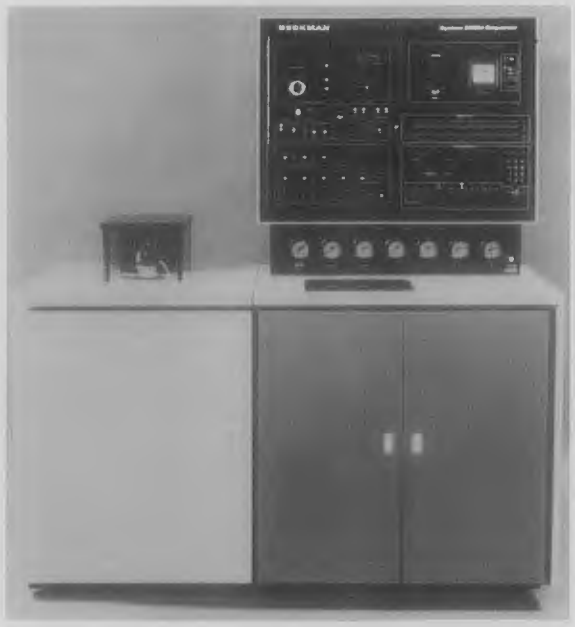
A nephelometric inhibition immunoassay for the antiepileptic drug carbamazepine is now available from Beckman Instruments.

The ICS® Carbamazepine Reagent Test Kit provides total carbamazepine quantitation. It is designed for use on the Beckman ICS II and Auto ICS Analyzers.

The new assay kit can be used for routine monitoring of serum carbamazepine levels or as a STAT procedure for overdose patients. The STAT immunoassay produces an answer in 80 seconds on either analyzer. Correlation coefficients of >0.937 have been obtained with known reference methods.

Other assay kits for therapeutic drug monitoring on Beckman Immunochemistry System include theophylline, phenytoin, phenobarbitol, gentamicin, tobramycin and primidone.

For more information, contact Alphatech, Phone 770-392, Auckland or **CIRCLE 5 on the Readers Reply Card.**



PROTEIN/PEPTIDE SEQUENCER PROVIDES RELIABILITY, VERSATILITY

Beckman Instruments, Inc.'s System 890M Protein/Peptide Sequencer automatically converts ATZ amino acids to PTH amino acids for analysis on an HPLC system. Sensitivity is down to 200 picomoles over 20 cycles. The system offers a wide dynamic range of sensitivity, from micro-sequencing of picomole quantities to the analysis of large nanomole samples.

Microprocessor control provides ease of programming, storage of up to eight programs, precise replication of every program command and linking and switching of programs.

System 890M enables sample application subroutines, reduced cleavage times and double coupling programs. The user can select manual or fully automatic operation.

An integral cold trap efficiently condenses reagent and solvent vapors. This feature eliminates the contamination of vacuum pump oil by volatile vapors.

Chemicals are an integral part of the System 890M's performance. Beckman manufacturers specially purified sequencer-grade reagents and solvents, as well as apomyoglobin protein standard.

An advanced rotary seal electropneumatic vacuum valve has been incorporated into the System 890M for increased reliability. It provides more efficient vacuum operation and single pump configuration to offer efficiency, precision and speed to the system.

For more information, contact Alphatech, Phone 770-392, Auckland or **CIRCLE 11 on the Readers Rely Card.**

EMAIL STAND ON HOT DOP TESTING SUPPORTED BY NEW U.S. RESEARCH

Mr Grahame Gibbs, Engineering manager with the Air Handling Products department of Email Limited, recently disclosed details of new research in the high efficiency air filtration field. Research, conducted at the company's Regents Park Laboratory, placed a question mark against the traditionally accepted relationship between the HOT DOP and sodium flame methods, for testing HEPA (High efficiency particulate air) filters.

Up until now, it was believed that for equivalent performance, a filter must show an efficiency of 99.995% using sodium flame testing, against 99.99% using the HOT DOP method — a 2:1 difference in the degree of penetration. The reason for this 2:1 ratio is the difference in particle sizes generated by the two tests. The sodium flame method generates particles with an average size of 0.6 micrometres (microns), while the HOT DOP particle is half this size, at 0.3 micrometres in diameter.

Email's research had indicated that the difference in the degree of penetration could be as high as 15:1 and not 2:1 as originally thought and

as accepted in British Standard B.S.3928. It should also be noted however that the ONLY testing method acceptable under the more recent local standard A.S.1324 is HOT DOP. As Grahame Gibbs rightly points out, this deficiency will only become apparent when a filter is HOT DOP tested because of the relatively large size of the sodium flame test particles. It should be appreciated at this stage that HEPA filters are often used in critical situations — for example, where toxic or carcinogenic substances are present. Consequently, there can be no margin for error. After all, what we're talking about is the protection of human life.

Research conducted in the United States by Flanders Filters, one of the world's largest manufacturers of HEPA filters, has shown, however, that even Email's figures may be conservative. In fact, Flanders is suggesting 0.3 micrometre particles are around 300 times more penetrating than 0.6 micrometre particles, as can be seen in the table below:

	Particle size	Efficiency	Penetration
HOT DOP:	0.3 micrometres	99.97%	0.03
Sodium flame:	0.6 micrometres	99.9999%	0.0001

The table shows that when a marginal HEPA filter was tested using 0.6 micrometre particles, it registered an efficiency of 99.9999%. However, when retested using 0.3 micrometre particles, the efficiency plummeted to 99.97%

Much of this research has been made possible by the development of new technology — notably the laser spectrometer that can measure particles as small as 0.12 micrometres, which is less than half the size of the HOT DOP particle. Both Email and Flanders have used the laser spectrometer extensively in their research, breaking new ground in the high efficiency air filtration field. Mr Gibbs said that Email would continue its heavy investment in research in order to broaden the appreciate of filter performance.

For further information on this subject please contact Email Industries Limited, Air Handling Products Division; P.O. Box 12648, Penrose, Auckland or P.O. Box 1693, Wellington, or **CIRCLE 12 on the Readers Reply Card.**

TOTAL ACTIVE QUINIDINES MEASURED BY BECKMAN'S TDM REAGENT KIT

The ICS™ Quinidine Reagent Test Kit has been introduced by Beckman Instruments, Inc. It is the first easy to use test for measuring total quinidine activity in human serum.

Quinidine is one of the most widely used drugs for treating ventricular and supraventricular arrythmias. This new immunoassay analyzes for quinidine plus the four clinically active derivatives, Quinidine-N-Oxide, Dihydro-Quinidine, 3-Hydroxy-Quinidine and Quinidine-Diol. Accurate measurement of total quinidine load allows the physician to monitor drug therapy more closely. This is particularly important in elderly cardiac patients with lower average clearances or in children with higher quinidine clearances.

Recent research reported in the literature has shown quindine metabolites possess equipotent antiarrythmic pharmacological activity in the human. The metabolites may also contribute to cardiotoxicity.

The new ICS Quindine Reagent Test Kit was developed as a homogeneous nephelometric inhibition immunoassay for the Beckman Immunochemistry Systems — the ICS II Analyzer and Auto-ICS Analyzer. The single-point assay may be performed as a STAT procedure with a result in 80 seconds or as a routine assay. The procedure uses a centrifugation step in buffer to remove fibrinogen and fibrin, which may occur in the sera of patients receiving oral quinidine.

The Beckman Quinidine immunoassay results parallel HPLC and double extraction fluorescence (DExF) methods for total quinidine. Results are typically 20% to 30% higher then EMIT procedures because of the four metabolites measured in addition to quinidine. With the Beckman assay, typical within-run and between-run CVs for quinidine samples were 7%.

The ICS Quinidine Reagent Test Kit was introduced at the American Association of Clinical Chemists meeting in July 1983. Other therapeutic drug monitoring assays available from Beckman for the ICS systems are theophylline, phenytoin, phenobarbitol, gentamicin, tobramycin, primidone and a new assay for carbamazepine. The Beckman Immunochemistry System also runs 14 specific protein assays and a Rheumatoid Factor test.

For more information contact Alphatech, Phone 770-392, or **CIRCLE 6 on the Readers Reply Card.**

ENZYME MODULES EXPAND BECKMAN'S ASTRA® IDEAL TO NINETEEN CHEMISTRIES

With the inclusion of six new enzyme tests, the clinical laboratory can now perform up to nineteen chemistries on Beckman Instruments, Inc.'s ASTRA® IDEAL systems. The ASTRA IDEAL automated clinical chemistry system joins two complimentary ASTRA 8 analyzers with a communications center known as the ASTRA Link.

The six enzyme tests — AST, ALT, GGT, LD, CK, ALP — are performed on two enzyme modules, now available in ASTRA IDEAL systems and for independent ASTRA 8 analyzers. Three enzyme tests are performed on each module. Enzyme tests can be run simultaneously with other chemistries, or discrete test selections can be made for all chemistries including one or any combination of enzyme tests. The two enzyme modules can be added to most existing ASTRA 8 analyzers or included on the new system.

The ASTRA Link communications center, interfacing two ASTRA 8 analyzers, completes the ASTRA IDEAL automated clinical chemistry system.

The ASTRA Link consists of a 16-bit, Z8000* 6MHz microcomputer, CRT and keyboard, and impact printer, cable assembly and diskette set. It handles all systems functions. From a single keyboard, the Link communications center permits an operator to program both ASTRA analyzer units. The operator can select for each sample any combination of tests or all tests from the up to nineteen available. Instant access for STAT samples is built into each ASTRA system.

The ASTRA Link computer processing unit automatically collates and prints patient results from both analyzers on a single chartable report. The 1 megabyte 8'' floppy diskettes store up to 500 patient results.

Current ASTRA 8 users may expand to an IDEAL system by adding a second ASTRA 8 with a complimentary configuration and a ASTRA Link communications center. Two modules designated to perform four additional chemistries, uric acid, phosphorus, cholesterol and triglyceride are now under development. These will retrofit into existing ASTRA IDEAL systems when available.

Beckman provides complete reagent support for all ASTRA chemistries. The new enzyme test reagents, stored in a cartridge system, are packaged in 50, 100 and 250 test sizes. The cartridge design permits three day reagent stability at room temperature.

Accessories available for the ASTRA IDEAL automated clinical chemistry system are a graphics tablet and stylus and a new ''work-station'' table. Both the ASTRA Link and the graphics tablet, as well as the analyzers, are supported with optional Beckman RAPid Kits. Each RAPid Kit consists of parts designed for immediate replacement in the systems by laboratory personnel.

For more information, contact Alphatech, Phone 770-392, Auckland or **CIRCLE 9 on the Readers Reply Card.**

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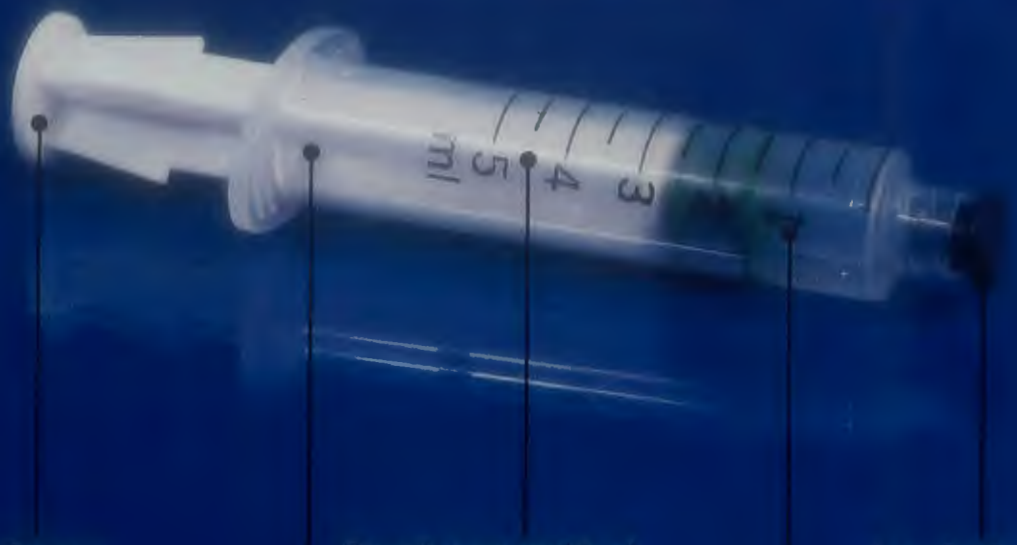

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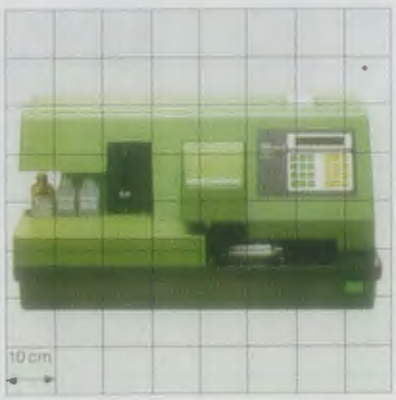

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