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1



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Leading Article

Laboratory and clinical aspects of measuring serum eosinophil cationic protein (ECP) in asthma. *S Holt, R Siebers*.3-4

Review Article

Thrombocytopenia – A Review. *K Potluri, C Kendrick*.....5-7

Original Article

Production of defined-haematocrit plasma-reduced blood for exchange transfusion. *SR Khull*.....8-9

Regular Features

Advertisers in this Issue9
Awards34-36
Council News27-31
Instructions to Authors.....2
Leukemia & Lymphoma Workshop.....20
New Products and Services13-14
NZIMLS Membership Application Form24
NZ Medical Laboratory Science Trust17
QTA Examination Liftout.....21-24
South Pacific Congress.....32-33
Special Interest Groups15-16

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Laboratory and Clinical Aspects of Measuring Serum Eosinophil Cationic Protein (ECP) in Asthma

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NZ J Med Lab Science 1999; 53(1):3-4

Introduction

Eosinophils are granular leukocytes, forming about 1% of the circulating white blood cell population. Their role includes mediating inflammation and they can secrete several highly basic cytotoxic proteins. Four principle proteins have been isolated and studied: Eosinophil Cationic Protein (ECP), Eosinophil Peroxidase (EPO), Major Basic Protein (MBP) and Eosinophil Protein-X (EPX) which is also called Eosinophil-Derived Neurotoxin (EDN) (1). There is evidence that secretion of these eosinophil granule proteins can damage the respiratory epithelium, causing an acute and chronic inflammation and bronchial hyperreactivity (2).

ECP was initially purified from human myeloid cells and subsequently identified as an eosinophil granule protein with a pI of pH 10.8 (3). It is a heterogeneous protein with different variants reflecting in its molecular weight range of 16-24 KDa. The biological activities of ECP interact with other immune cells, and with plasma proteins predominantly those of the coagulation pathway. Measurement of ECP in biological fluids is useful as an index of eosinophil turnover and in-vivo activity.

ECP has been studied more extensively than the other proteins, having been hypothesized to be a clinical marker of asthma severity. Levels of ECP have been measured in serum, sputum and bronchoalveolar fluid (BALF) (4). This paper focuses on serum ECP measurements only as serum is easy to obtain and more likely to be the fluid sampled in clinical practice.

Laboratory Measurement of ECP

Methodology

ECP is generally measured by double antibody radioimmunosorbent assay (RIA), detection limit <2.0 µg/L (5,6); or by fluorescent enzyme immunoassay (FEIA), detection limit <0.5 µg/L. Both methodologies are commercially available as a kitset from Pharmacia & Upjohn, Uppsala, Sweden. The FEIA method results in slightly lower Serum ECP levels compared to the RIA method, with a correlation coefficient of 0.98 between the two methods (K. MacLachlan, Pharmacia & Upjohn, personal communication as quoted in ref. 7).

Sample requirement

Serum is the preferred sample source. The reason for this is that eosinophils actively release further ECP in-vitro during the clotting process reflecting the state of in-vivo eosinophil activation. This amount of released ECP, together with ECP already present in blood, gives a total picture of in-vivo ECP and of eosinophil activation. Thus the higher the measured serum ECP levels, the greater the propensity of eosinophils to release the highly cytotoxic ECP when attracted to the site of inflammation, such as in the lung in asthma.

As the turnover (elimination rate) of ECP in-vivo is about 65 min⁶, blood samples collected in plain Vacutainer tubes are left to stand for 60 min (±10) at room temperature before centrifugation.

The collected serum is then re-centrifuged to ensure no eosinophils remain in the serum, and this sample is then stored deep-frozen in plastic (not glass) tubes before ECP measurement. If gel-containing Vacutainer tubes are used to collect the blood samples the re-centrifugation step can be omitted and the blood samples thus collected can be left for up to 120 min before centrifugation. Haemolysis is to be avoided as this potentially damages eosinophils.

Reference range

Peterson et al (6) analyzed serum samples from 101 healthy individuals aged 20-63 years using the RIA method and obtained a geometric mean of 6.0 µg ECP/L with a reference range (90%) of 2.3 - 15.9 µg/L. No sex or age differences were found. However, Marks et al (7) found higher serum ECP levels in men. Furthermore they found that serum ECP levels were related to age with decreases in serum ECP on average by 6.4% for each 5 years of age over 20.

Current smokers have on average 15% higher serum ECP levels after adjusting for age (7). This is similar to the findings of Jensen et al (8) and is consistent with findings of higher eosinophil counts in smokers (9). Serum ECP levels are moderately reproducible in individuals over time. Fifty-seven subjects had two blood samples taken for serum ECP four weeks apart, showing an intra class correlation coefficient of 0.62 (7). Serum ECP levels stored at -70°C for 27 months showed a mean ECP ratio (pre- and post- stored) of 1.03 (90% range: 0.95 - 1.10).

Serum ECP and asthma

Eosinophils play a major role in the pathogenesis of asthma, which is defined as "chronic inflammatory disorder of the airways in which many cells and cellular elements play a role, in particular, mast cells, eosinophils..." (10). It has been hypothesized that serum ECP is a measure of eosinophil activation in peripheral blood induced as a consequence of inflammation in the airways and that it may indicate the degree of airways inflammation in asthma. This has led to the proposal that measurement of serum ECP may represent an objective measure of asthma severity and that its interpretation may guide the clinician as to the most appropriate treatment. It is proposed that this may be of particular use in young children, in whom symptoms may be an unreliable guide to the severity of their asthma and who may have difficulty undertaking lung function measurements. It has been suggested that measurement of serum ECP may also have a role in monitoring asthmatic subjects and in determining corticosteroid requirements in this group. Many studies have used serum ECP measurements when looking at the effects of drugs in asthma, such as corticosteroids, anti-histamines and β₂ agonists (11-13).

Is it useful?

It is undisputed that asthmatics, as a group, have higher serum ECP levels, although considerable overlap exists between asthmatic and

non-asthmatic subjects. However, there is much conflicting evidence in the literature as to whether or not serum ECP is a sensitive and specific marker of asthma severity.

First of all raised serum ECP levels do not appear to be specific for asthma. Levels can be raised in other conditions such as atopic dermatitis (14), rheumatoid arthritis (15), ulcerative colitis (16), ankylosing spondylitis (17), bronchiectasis (18) and even acute bacterial infections (19). Therefore, raised serum ECP levels seem to be indicative of many inflammatory processes and are not necessarily diagnostic of asthma.

Within a population of asthmatic subjects, some studies show that serum ECP correlates significantly with currently used measures of asthma severity, such as FEV1 and symptom scores (20-22). However, other studies have failed to find such correlations (23-25). Most of the studies have different designs and the results are not consistent, therefore it is difficult to draw conclusions.

Another way to examine its diagnostic value is to determine the sensitivity and specificity of serum ECP for detecting symptomatically active asthma. This was undertaken by Vanto et al who found the sensitivity to be 54% and the specificity to be 71%. They noted that in several patients, the serum levels of ECP actually increased, despite clinical improvement of the asthma. As a result of this they concluded that serum ECP measurement was of limited utility in an individual patient⁽²⁶⁾.

Overall, although serum ECP is increasingly being measured, the clinical application of the results has not been clearly demonstrated. In particular, it is uncertain whether serum ECP levels provide additional useful data, over and above more standard tests, as a one-off test or as repeated tests in an individual. It may prove to be a useful tool under certain circumstances, but currently has no role in routine clinical practice.

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Thrombocytopenia – A Review

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Introduction

In 1808 William first classified purpura into: simplex, haemorrhagica, urticans or contagiosa. In 1887 the role of "haematoblasts" (platelets) in "purpura haemorrhagica" was described by Krauss and Denys and later Hayem [1895] described thrombocytopenia as the cause of the non-retractility of the blood clot. Duke [1912] was the first to demonstrate the prolongation of the bleeding time in thrombocytopenia (1). Thrombocytopenia is a quantitative disorder of platelets in which the peripheral blood platelet count is less than the normal range. Of the many diseases that either cause or contribute to haemostatic failure, thrombocytopenia is the most frequently encountered in man. The associated clinical symptoms of spontaneous skin purpura (petechiae), mucous membrane haemorrhage "wet purpura" and prolonged bleeding or bruising after minor trauma (2) is related to the degree of thrombocytopenia.

Pathophysiology

In humans clinical thrombocytopenia develops following; 1. deficient platelet production, 2. accelerated platelet destruction, and 3. abnormal distribution or pooling of platelets within the body (3).

Platelet production is reduced in bone marrow failure associated with leukaemia, myelodysplasia, aplastic anaemia, HIV infection, myelofibrosis, megaloblastic anaemia, uraemia, multiple myeloma and marrow infiltration. Selective megakaryocytic depression caused by drugs, alcohol, chemicals and viral infections also lead to low platelet production and peripheral blood thrombocytopenia. The hereditary platelet syndromes of May-Hegglin, Wiskott-Aldrich, Bernard-Soulier and others depress platelet production causing variable levels of thrombocytopenia.

Accelerated platelet destruction is most frequently the result of an immune response or a consumptive coagulopathy. In Immune Thrombocytopenia Purpura (ITP), thrombocytopenia follows the formation of antibodies to structures on the platelet membrane. Allo or autoantibodies sensitise the platelets causing a decreased peripheral blood survival. ITP is most commonly associated with systemic lupus erythematosus (SLE), chronic lymphocytic leukaemia (CLL), bacterial and viral infections, post bone marrow transplantation, post blood transfusion, and following some drug therapies.

Consumptive coagulopathies as a cause of thrombocytopenia include disseminated intravascular coagulation (DIC), microangiopathic processes such as haemolytic-uraemic syndrome (HUS), thrombotic thrombocytopenic purpura (TTP), extra corporeal circulation and giant haemangioma [Kasabach-Merritt Syndrome] (4).

In the third group an abnormal distribution of total platelet numbers can lead to a peripheral blood thrombocytopenia. This is most commonly associated with splenomegaly. Transient or dilutional thrombocytopenias are a feature of massive blood transfusion.

Laboratory diagnosis

The laboratory diagnosis of thrombocytopenia follows the finding of a patient in whom the platelet count is below the lower limit of the nor-

mal range (150-450x10⁹/L). The diagnosis of immune and non-immune forms establishes treatment protocols. No single laboratory test is able to provide a clear discrimination. Recently Brighton and colleagues showed the Monoclonal Antibody Immobilisation of Platelet Antigens (MAIPA) assay to have a greater specificity than the Platelet Associated IgG assays (PAIgG) for this purpose (9).

In the absence of a reliable laboratory procedure to differentiate between immune and non-immune ITPs, George and colleagues (6) states that the diagnosis of ITP should be based on patient history, physical examination, complete blood count and examination of a peripheral blood smear.

According to Hoffbrand and Pettit (7), ITP can be diagnosed when the platelet count falls to between 10-50x10⁹/L in the presence of a normal Hb and WBC. The finding of large platelets in the peripheral blood film is a common feature and the bone marrow aspirate shows a normal to increased number of megakaryocytes. Cahill and Newland (8) suggest that electron microscopy may provide worthwhile diagnosis in patients with larger platelets to distinguish refractory ITP from Bernard Soulier syndrome and Epstein's syndrome. In some the presence of serum anti-nuclear antibody (ANA) may support the finding of thrombocytopenia caused by SLE.

Colman and colleagues (3) support the use of a bone marrow aspirate examination in the evaluation of thrombocytopenia. The finding of increased numbers of bone marrow megakaryocytes are seen in destructive thrombocytopenia with a reduction or absence seen in acquired or congenital amegakaryocytic thrombocytopenic disorders. MPV and plasma glyocalicin and platelet RNA measurements may also be helpful in the diagnosis.

Drug-induced thrombocytopenias can lead to very low platelet counts (<10x10⁹/L). Often drug dependent antibodies can be demonstrated in the patient. The finding of low platelets, a microangiopathic blood film and platelet anisocytosis are strong indications for the diagnosis of TTP. Commonly the RDW, MPV and PDW are all increased. A biopsy confirmation of the presence of capillary thrombi is also useful (8). The cord blood platelet count and the presence of platelet antibodies present in the maternal serum are helpful in the diagnosis of neonatal thrombocytopenia.

Classification

Immune Thrombocytopenia Purpura: According to Hoffbrand and Pettit (7), ITP is the commonest cause of thrombocytopenia in women aged between 15-50 years in whom there is no anaemia or neutropenia. Clinical features of the disease are the onset of petechial haemorrhage, easy bruising and mucosal bleeding with rare intracranial haemorrhage. ITP follows the production of allo or auto antibodies to glycoprotein (GP) IIb/IIIa and the GP Ib/IX membrane complexes. Increased sequestration of IgG sensitised platelets by Fcγ bearing splenic macrophages occurs faster than the bone marrow response. ITP occurs alone or sometimes accompanied by Evan's syndrome (1, 8). It is also seen with greater frequency in SLE, HIV and CLL patients.

In a survey of the UK practise of ITP diagnosis, Bolton-Maggs

and Moon (9) state that ITP is benign in nature, despite its low platelet count and suggests a greater attention to the clinical picture in the classification of ITP. Corrigan states that in ITP the risk of spontaneous bleeding can not be predicted by the platelet count (10). To date there is no laboratory test that supersedes the clinical assessment of ITP at the bedside.

Brunner-Bolliger and colleagues (11) also suggest that ITP may also be caused by a passive transfer of anti-P1(A1) alloantibody through plasma transfusions. Absorption of alloantibody on to the recipient's platelets has led to severe thrombocytopenia. Autoimmune thrombocytopenia is common in children after infection and vaccination due to formation of immune complex and complement depositions on platelets (6).

Drug Induced Thrombocytopenia: It is seen in patients on treatment with quinine, quinidine, heparin, para-aminosalicylate, sulphonamides, rifampicin, stibophen and digitoxin. Thrombocytopenia develops following the formation of antibody directed against the drug. The drug complexes with plasma proteins which act as foreign antigen stimulating an immune response. The immune complexes are absorbed onto platelets (6) which are then removed in a manner similar to that in ITP.

Thrombotic Thrombocytopenic Purpura (TTP): Moschowitz first described TTP in 1924. He considered it a diffuse disease of the micro-circulation, which followed the wide spread deposition of micro-vascular thrombi of fibrin and platelets. Hoffbrand and Pettit (2) state that TTP is a fatal disorder of thrombocytopenia with confluent purpura, fragmentation haemolysis and wide spread ischaemic organ damage of the brain and kidney. According to Koultz (13) TTP is an enigma that has been attributed to primary platelet activation occurring either in the presence of a plasma activating factor or the absence of an inhibitor of platelet activation. Other suggested causes include defective endothelial cell production of prostacyclin or plasminogen activator and the over production of the high molecular weight forms of von Willebrand's factor. The blood picture in TTP is characterised by the presence of a microangiopathic haemolytic anaemia, thrombocytopenia. Neurological and renal abnormalities are common clinical symptoms. TTP is most common in females aged between 20-50 years of age.

Other thrombocytopenias: Congenital thrombocytopenias are due to inherited or pathological process. Infection or exposure to toxic substances during intra-uterine life leads to a failure in platelet production or increased consumption or destruction of platelets (1). Seidman and colleagues (14) states that neonatal and antenatal alloimmune thrombocytopenia is caused by the placental passage of maternal antibodies directed against platelet specific fetal antigens. This disease is analogous to Rh haemolytic disease of newborn and may be complicated by intracranial hemorrhage. The most common cause of neonatal thrombocytopenia is the finding of ITP in the mother during pregnancy.

Thrombocytopenia due to splenomegaly follows the increased 'pooling' of platelets. Moll and colleagues (15) documented an example of thrombocytopenia seen in a case of wandering spleen.

Thrombocytopenia as a complication of human immunodeficiency virus (HIV) infection is an important and common haematological finding (16). Although this condition is often asymptomatic, it may manifest clinically as a spectrum of bleeding problems including petechiae, ecchymoses, epistaxis, or menorrhagia or as hemorrhage of the gingivae, gastrointestinal tract, or CNS. Thrombocytopenia may present at any stage of the immunodeficiency, with spontaneous remission occurring in some.

Reports of haemophagocytic histiocytes causing thrombocytopenia in immuno-competent patients with sepsis syndrome or septic-shock (17) have been reported. Richards and colleagues (18) report the finding of thrombocytopenia following orthotopic liver transplantation. They suggest the cause of the increased rate of platelet con-

sumption to be associated with thrombin generation, reflecting the magnitude of the liver transplant surgery.

Treatment

Spontaneous recovery occurs in less than 10% of patients with chronic ITP. Treatment of ITP usually commences with high dose corticosteroids to reduce autoantibody formation and destruction of sensitised platelets. The mechanism of the steroid action is not clear but it may act to block the Fc γ receptors on splenic macrophages or to inhibit antibody synthesis. This can be useful during pregnancy for the control of bleeding episodes preoperatively, and in the management of ITP in infants (8). In patients who do not respond to steroid therapy splenectomy may be indicated. More recently high dose intravenous immunoglobulin (IVIg) as reported by Colman and colleagues (3) has proven useful in ITP, HIV associated thrombocytopenia, and infectious mononucleosis induced thrombocytopenias. In most cases IVIg is able to restore the platelet count rapidly through the saturation of splenic Fc γ receptors.

The use of immuno-suppressive drugs eg. vincristine, vinblastine, cyclophosphamide, azathioprine or cyclosporine are used on patients refractory to steroids and splenectomy. Other refractory cases may be treated with danazol, Vitamin C in large doses, colchicine, anti-Rh(D) immunoglobulin and α -interferon. Platelet transfusions are used to treat life threatening bleeding episodes.

Thrombocytopenia induced by drugs usually resolves with the cessation of the implicated drug. Extremely low platelet counts (<10x10⁹/L) may be encountered in these cases. The transfusion of platelet concentrates until the bone marrow response is mounted is often required.

Kaushansky (19) and Helleberg and colleagues (20) state that haematopoietic growth factor such as thrombopoietin treatment be used on patients who do not respond to steroids, splenectomy and/or intra-venous IgG treatment. Regulation of megakaryocytopoiesis, with the use of these cytokines may be useful in reducing the use of platelet transfusion therapy.

Conclusions

Thrombocytopenia is a significant cause of morbidity and mortality and the commonest cause of abnormal bleeding of patients on presentation. The laboratory investigation of cases suspected of having a platelet or blood vessel abnormality should commence with a routine CBC and blood film examination. A review of the patient history and a physical examination together with the CBC are essential parts of first line investigation. In confirmed thrombocytopenia a bone marrow biopsy is frequently indicated to establish aetiology prior to the selection of patient treatment. The results of the biopsy may indicate: generalised bone marrow failure or failure of megakaryocytopoiesis alone; provide support for an immune or consumptive thrombocytopenia. Laboratory tests useful in the definitive diagnosis of ITP are not part of the routine screening in most laboratories. Despite the limitations of platelet antibody tests such as PAIgG and the MAIPA assay each may still prove to be useful in the confirmation or exclusion of ITP as the cause of thrombocytopenia.

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1998 Bayer Essay

This year the subject for the Bayer Essay for Massey University students completing Haematology in the final year of their studies toward the BMLS degree was 'Thrombocytopenia'. The cash prize and Bayer products was won in 1998 by Kusuma Potluri who spent her time on clinical laboratory placement at Middlemore Hospital. Congratulations to Kusuma and thanks to Joanne Paton and Bayer NZ. Ltd. for their involvement and support.

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Production of Defined-Haematocrit Plasma-Reduced Blood for Exchange Transfusion

*Sheryl R. Khull, MNZIMLS, Charge Technologist,
Transfusion Medicine Department, Palmerston North Hospital.*

NZ J Med Lab Science 1999; 53(1):8-9

Abstract

The objective was to develop a practical method of production of plasma-reduced whole blood for exchange transfusion of neonates, which resulted in a unit with a haematocrit consistently within the ideal range of 0.5 to 0.6.

The volume of plasma to remove from each unit of whole blood in order to achieve the desired haematocrit in the final component is calculated from each donor's haemoglobin and the weight of the collected unit of blood. The results of these calculations for each acceptable donor haemoglobin and donation weight were converted to a chart for daily use.

Plasma-reduced whole blood produced using this procedure consistently has a haematocrit within the desired range. Our experience indicates that simple calculations and procedures can be used to produce a blood component with a consistent haematocrit, to meet clinical requirements.

Keywords

Blood processing, exchange transfusion, plasma reduced blood.

Introduction

Although the transfusion needs of infants make up a relatively small proportion of routine Blood Bank workload, babies in neonatal units are among the most intensively transfused of all hospital patients. Their small blood volume as well as their physiological immaturity gives them special transfusion needs, such as irradiation of blood components to prevent Graft-versus-Host disease, "fresh" components to avoid overloading with plasma potassium, or aliquotted units for several small transfusions over a period of time while limiting donor exposure. Exchange transfusion of neonates has particular issues because it is a relatively massive transfusion. Exchange transfusion was traditionally indicated for babies suffering from haemolytic disease of the newborn, but is becoming more common for babies who are serologically normal but metabolically critically ill.

The blood component generally recommended for exchange transfusion of neonates is plasma reduced red cells with a haematocrit in the range of 0.50 to 0.60 (1). The method for the production of such a component is given very little treatment in the literature. The removal of 100ml of plasma from a unit of whole blood is sometimes used. If the raw materials used for production were standard, then removal of a standard volume of plasma could produce standard products. However since the haematocrit of normal blood donors and therefore of collected whole blood can vary significantly within acceptable limits, this practice can lead to the production of components intended for exchange transfusion with haematocrits which vary widely and which may be less than ideal.

We aimed to develop a procedure, which would produce red cell components with a haematocrit which was reliably consistent as well as within the ideal range of 0.5 to 0.6.

Method

Calculation of donor haematocrit.

The haemoglobin of volunteer blood donors is measured immediately prior to donation. This was used to calculate the donor's haematocrit. O'Connor and colleagues reported that, providing the Mean Cell Volume is close to normal, there is a strong linear correlation between the haemoglobin and the haematocrit, and that the haematocrit can be calculated by multiplying the haemoglobin by 0.003 (2).

Since donors are a selected healthy population, their MCV should not be significantly abnormal.

Calculation of initial haematocrit of collected unit.

The anticoagulant in the blood collection pack dilutes the collected blood to a lower haematocrit than that of the donor. This dilution factor was calculated as the volume of the unit minus the volume of anticoagulant and divided by the volume of the unit.

Calculation of weight of plasma to remove.

The desired volume of the final component was calculated as the initial unit haematocrit multiplied by the initial volume of the unit and divided by the desired haematocrit. A desired haematocrit of 0.55 was used for these calculations, being the midpoint of the clinically indicated range for exchange transfusion blood components.

The volume of plasma to remove from the whole-blood unit was therefore calculated as the initial volume of the unit minus the desired volume of the unit. The plasma was removed after centrifugation by weighing it on to a balance, so the volume of plasma to remove was converted to the weight of plasma to remove, using a specific gravity of 1.03.

The calculations were entered onto a computer spreadsheet and run with varying figures to cover the common range of haemoglobins and blood unit weights, in practical intervals. The results were converted to a chart for ready reference in the blood processing laboratory.

Results

The ready reference chart is reproduced as Table 1.

The number of grams of plasma to remove from each unit is obtained by simply reading the value shown at the intersection of the column showing donor's haemoglobin with the row showing the tared weight of the unit (i.e. the total weight of the collected blood and anticoagulant without the weight of the bag).

This procedure is now in routine use in our blood processing laboratory. Quality control samples taken from all fourteen such blood components prepared over the last month have shown haematocrits of between 0.52 and 0.58.

Discussion

The clinical demand for a blood component for neonatal exchange transfusion that has a defined haematocrit is widely documented in the literature and confirmed by the expressed preferences of our

Table 1:

Grams of plasma to remove in preparation of plasma reduced blood.

		Haemoglobin (g/L)							
		125	130	135	140	145	150	155	160
T	490	196	184	173	162	150	139	128	117
a	500	199	187	176	164	152	141	130	118
r	510	202	191	178	167	155	143	132	119
e	520	205	192	181	169	158	145	133	121
d	530	207	196	183	171	160	147	135	122
	540	211	199	186	174	162	148	136	124
W	550	214	201	188	176	163	149	137	125
e	560	217	204	191	178	165	152	138	126
i	570	220	207	194	180	167	155	140	127
g	580	224	210	197	182	169	156	141	128
h	590	226	212	199	184	171	158	144	129
t									

neonatologists. The production of such a blood component requires that the volume of plasma removed during production be adjusted, to take account of the haematocrit of the blood donor and the volume of blood collected. The procedure we have developed achieves the production of components under routine conditions with haematocrits that are well within the clinically desirable range.

The weight of plasma required to be removed in order to produce a component with a haematocrit near 0.55 ranges from 117 to 226 grams. It is clear from these results that the common practice of removing a standard 100ml volume will seldom produce the ideal component for exchange transfusion of neonates. The origin and scientific basis for such a practice is not well documented but it may be based on convenience for the blood centre rather than the drive to meet customer requirements.

Since blood components for exchange transfusion of neonates are usually produced in relatively small numbers each day, it is feasible to individually tailor the production process in order to produce components with a haematocrit within the defined range, without excessive disruption to the work flow in the blood processing laboratory. We have found this to be a simple and practical procedure that produces units with a consistent haematocrit within the desired range. The

neonatal consultants in our hospital are pleased they can feel confident in the reliability of the product we offer.

The blood component which we produce by this procedure is not a standard product as defined in the Minimum Standards (3). The product defined in the Minimum Standards as 'Whole Blood, Modified, Paediatric' is an aliquot of a unit which has had no more than 100ml of plasma removed and which has a final haematocrit of no more than 0.60 but no lower limit defined. It is clear that most donations which have no more than 100ml of plasma removed will have a final haematocrit of well below 0.60, and indeed predominantly below 0.50. These calculations are derived from first principles and can be readily adapted for any laboratory's specific needs.

Conclusion

Consultation with the clinicians, computer-aided calculations and a simple procedure can lead to the development of a practical production scheme for components that have enhanced clinical acceptability.

Acknowledgements

The author wishes to thank Kate Marson, who initiated this project as a fourth year BMLSc training project, and to Sharon Sims, Quality Manager, for aid and encouragement.

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Advertisers in this issue

Abbott Diagnostics	17
Beckman Coulter	11
Biomerieux	26
Biorad	12
BMG Associates	14
Corinth Medical	14
Johnson & Johnson	25
Med Bio Enterprises	7
Medica Pacifica	13
Roche Diagnostics	18-19 & Outside Back Cover
SCIENZ Corporation	Inside Front & Back Covers

Index to Volume 52, 1998

Subject Index

Biochemistry

Implementing the RHA draft national quality standards for medical laboratories for glucose near patient testing.

Clare Murphy.....6-9

General

TH Pullar Memorial Address. Training for paradise.

Michael Lynch.....3-5

Alternative site testing: a solution for the isolated country hospital. *Lorraine Craighead, Martin Watts*83-4

Biochemistry in paradise. *Elisabeth Stephenson*.....109

Haematology

von Willebrand's disease – A review.

J Anastasi, C Kendrick.....59-60

Evaluation of specimen handling protocol for neutrophil alkaline phosphatase analysis. *Greg Pennell*85-9

Immunohaematology

A simplified IgG anti-D quantitation method.

Jandhe Carter, Robert Coleman, Graeme Woodfield, Steve Henry45-6

The quantitation of anti-Rh(D) in New Zealand blood donors used for the manufacture of anti-Rh(D) immunoglobulin.

Robert Coleman, Jandhe Carter, Anna Pryde, Graeme Woodfield, Steve Henry.....61-2

Microbiology

Salmonella – Not your average enteric pathogen.

Annamarie Clarkin.....43-4

An evaluation of the 'mouse model' in the study of the virulence of coagulase-negative staphylococci isolated from sheep subclinical mastitis. *Angeliki R Burriel*.....90-4

Editorial

Write me a (research) letter. *Rob Siebers*44

Letters to the Editor

The roomful of straw. *John Aiken*110

Reports

NZIMLS Annual Scientific Meeting survey.

Tony Mace19

South Island seminar report. *JB Kitto*47

New Zealand laboratory science trust.....75

NZIMLS President's report. *Shirley Gainsford*96

Minutes of the 54th AGM of the NZIMLS. Palmerston North, 2 September 1998.....95-6

Abstracts of papers presented at the ASM of the NZIMLS. Palmerston North, 1998.....112-24

Book Reviews

Standardised reporting of haematology laboratory results.

HSIG of the NZIMLS50

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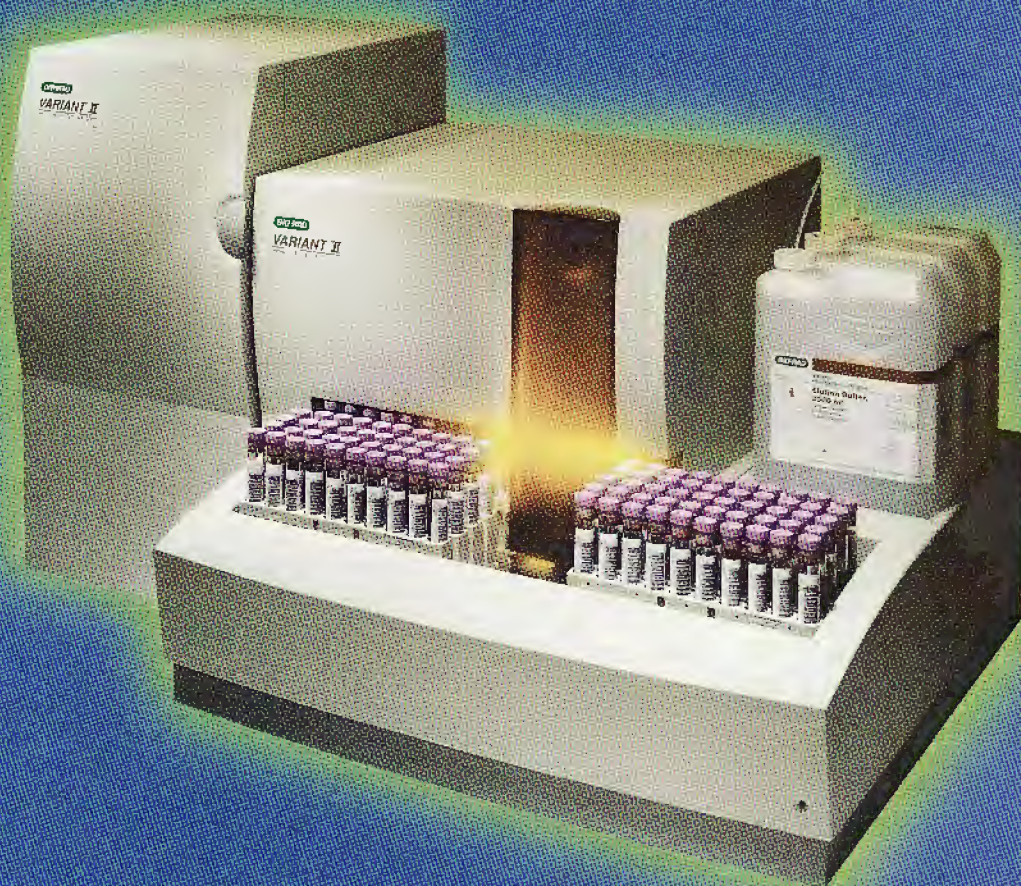


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Bayer Strengthens Position in Clinical Diagnostics Market

A US\$1.1 billion plus buy out will further strengthen the Bayer Group's position as one of the world's leading manufacturers of clinical diagnostic technology.

On November 30, 1998, the Bayer Group and the Californian diagnostics company, Chiron Diagnostics Corporation, finalised their agreement for Bayer to acquire Chiron.

Bayer and Chiron technology is widely used in New Zealand medical and pathology laboratories, particularly those working in the areas of biochemistry, histology, haematology and immunology.

Based in Leverkusen, Germany, the Bayer Group is a \$US32 billion company involved in the research, development and manufacture of pharmaceutical products, healthcare technology, crop protection and animal health products, and raw materials such as polymers and organic chemicals.

Bayer is possibly best known as the company which in 1897 brought the world Aspirin®.

Chiron Diagnostics, headquartered in Emeryville, California, is a leading biotechnology company employing thousands of people in facilities on four continents.

The Bayer Group is represented in New Zealand, which has its head office on Auckland's North Shore.

Mr Tony Nagle, General Manager of Bayer New Zealand's Healthcare Division said the acquisition of Chiron would have a significant and beneficial impact on New Zealand laboratories and medical facilities, especially those currently using Bayer central laboratory technology.

He said that central laboratory diagnostics was in a constant state of development with technologies becoming increasingly sophisticated, requiring more specialised technical support.

"Given the diverse nature of Bayer's business base we believe it is in the best interests of customers to integrate our Central Laboratory Business Unit into Chiron's New Zealand distributor, SCIANZ Corporation Ltd, a company specialising in central laboratory diagnostics. Most business unit staff will be moving across to SCIANZ, which pretty much means business as usual for Bayer customers when it comes to people relationships. Our objective is to make this transition as seamless as possible.

"While proud of the achievements of the Central Laboratory Business Unit, the fact remains we are a diversified company whereas SCIANZ is able to offer a greater depth of human and technical resources in this highly specialised field."

Tony Nagle said he felt that users of Bayer and Chiron technology can confidently look forward to continuing high standards of service and the release of major advancements in central laboratory diagnostics.

Subject to final arrangements, the integration of Bayer's Central Laboratory Business Unit with SCIANZ Corporation Ltd will occur on January 1, 1999.

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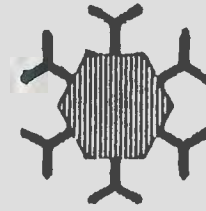
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As Convenor of the ISIG, I would like to invite anyone with interests relating to the fields of Immunology/Serology to our 1999 Seminar to be held in Blenheim on May 29th/30th. The team at Canterbury Health Laboratories are well organised and look forward to your response. (Please refer to the programme presented in this issue of the Journal).

It is our intention this year to reintroduce a regular newsletter to our members and to that end, I would be delighted to receive correspondence from members and intending members, particularly addresses, phone/fax numbers, Email addresses so that we can compile current contact lists to distribute around. If you would like to receive a copy of our Newsletter please let me know. Also gratefully received, any questions or articles (no matter how small or seemingly trivial) to include in these newsletters.

A further intention of the ISIG, under the guidance of David Haines - Medlab Auckland, is the establishment of an Immunology Test Directory. More information on this will be available in the ISIG Newsletter. Anyone wishing to contribute to this project please contact David directly or myself.

It is our intention to see the ISIG alive and kicking in 1999 but we need your help.

Finally below is a summary of the recent ISIG Seminar held in September. Anyone wishing to seek more information on these brief summaries please feel free to contact the various speakers.

Immunology Special Interest Group Weekend Escape, Rotorua, September 19-20 1998

The annual ISIG meeting was held on the weekend of 19-20th September at the Manary Lodge Rotorua. Located 7kms out of town on the beautiful shores of Lake Rotorua. The meeting was well attended with over 30 delegates mainly from the North Island but with Nelson, Christchurch and even Dunedin represented. Good food and company combined with the relaxing setting helped make this a very enjoyable weekend for all.

The aim of the conference is to provide a forum for information sharing and updating knowledge of new and existing areas of work. This agenda was certainly met.

The ISIG kicked off in earnest with morning tea – quickly followed by a welcome from David Haines (co-organiser and ISIG convenor at the time).

The first speaker, Paul Austin (Immunology Auckland Hospital), repeated his award winning talk, simply entitled Dengue, given at the recent NZIMLS conference. A comprehensive story of Dengue fever was discussed: the what is it, where it comes from, how to test it and the interpretation of serological results. About two years of data, from the Auckland and Pacific experience, was neatly summarised. One key point emerged that Dengue often showed a delayed humoral

response requiring doctors to order follow up serology. This does not often happen, which Paul thinks leads to under-reporting of Dengue in New Zealand. Paul finished with a case study and answered several questions from the floor.

Stewart Smith (CHLabs – ChCh) followed up with a talk on the autoimmune disorder SCLERODERMA the clinical and laboratory features followed by two case histories from Christchurch and concluding with new evidence for Alloimmunity from Nelson and Branchi et al (USA) as a potential cause of this rather debilitating disease.

Rubee Yee from Wellington Hospital spoke next about Nuclear Lamin antibodies. She presented a patient whom had nuclear lamin ANA of 320, was RHF positive, CRP (199), dsDNA negative when she came into hospital. Antiphospholipid Syndrome (APS). Rubee then presented what she knew about APS and Nuclear Lamin antibodies.

Another pure immunology talk followed titled Sjogrens and heat seeking missiles (Dianne Siegenthaler). Dianne detailed the history of a patient over a number of years and proved the importance of laboratory input into result interpretation and patient management.

After lunch Jane Humble from Wellington Pathology presented an evaluation of three commercial kits for analysis of EBV infection: Organon, Gull method, Pan Bio, Jane had commented on the difficulty in the interpretation of some unusual results. Experiences from the floor were soon forthcoming and Jane seemed relieved that she was not alone.

Linda Smith presented a case history on Troponin I. The patient had normal cardiac markers (including TnT) except for repeatedly raised TnI by Abbott method. TnI by ACS-180 was normal. Why (and what) is causing this discrepancy?

Heterophile antibodies and the eating live mice were a couple of the more obvious causes for these strange results. Abbott has sent this sample to the USA for further testing.

Guru Arunasalem from Diagnostic Lab in Auckland presented a short talk on the value of external QAP. He passionately pushed the virtues of RCPA external AP and the need for user feedback. Without this feedback (positive and negative) they cannot improve their service or know when they are doing it right. Feedback please to the following Email address: RobertMcEvoy@flinders.edu.au.

Lynley Smith from Auckland Hospital presented a conference report on the Immunology papers presented at NZIMLS meeting in Palmerston North. Topics included: Ultra sensitive CRP testing, Evidence based diagnosis, Medical Liability, IANZ, Zenotransplantation.

David Haines discussed the pros and cons of automated ANA/ENA/dsDNA methods. Also raised the issue of standardisation of EIA, RIA and immunofluorescence assays.

He expanded on the dsDNA EIA methods where low affinity antibodies can be removed with 8 M urea solution. He compared various EIA methods and they all compared well with Farr assay.

It was suggested that RCPA QAP sent out treated and untreated samples to be run. This may help solve lack of standardisation in this area.

David's second talk was quite short where he proposed the establishment of an Immunology/Serology test directory. It was felt in discussion that this was a reasonable thing to do and David will be sending out a form in the ISIG Newsletter.

The last talk was Rodger Linton from Medlab South on the future of the ISIG. This was a good topic as it was felt the ISIG was left

to drift last year. Important issues decided were that Rodger Linton is to be the new ISIG convenor. Rodger is to collate information provided by members around the country and re-introduce the newsletter. Rodger was at pains to point out that it needed a good level of support for the newsletter to continue. Addresses could be sent to Rodger care of Medlab South for mailing purposes.

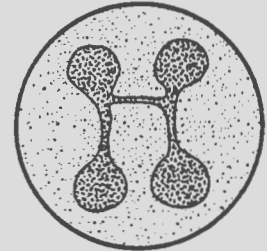
There was also general discussion on the value of the meeting. There was general consensus from everyone present that they are an

extremely useful forum in which to discuss issues of concern, new developments etc.

On behalf of all those attending a special thanks goes out to David Haines and Penny Newton for their extreme effort in organising this highly successful meeting. We look forward to seeing you all next year.

Haematology

Special Interest Group



Haematology's Liver transplant experience

Liver transplantation evolved rapidly in the 1980s. Today it is the therapy of choice for many patients with chronic, advanced, irreversible liver disease. Also liver transplantation has now become a therapeutic option for acute fulminant hepatic failure.

The New Zealand Liver Transplant Unit expects to do 66 transplants over three years at Auckland Hospital. The budget for a transplant is \$120,000 per patient over three months. After the three months any follow-up care is provided by their Regional Health Authority (RHA).

The donor organ is assessed mainly at the donor's hospital. Liver function tests, viral serology and ABO grouping are already known. At this stage the recipient and staff involved in the transplant, including the Haematology Laboratory, are notified. However, it is not until the liver is examined by the donor team that it can be confirmed as suitable for transplant, i.e. colour, shape, and size. The most common reason for a transplant not to proceed when a liver becomes available is because of ABO incompatibility.

The Haematology laboratory has an on call roster system to cope with the transplants occurring during the night and the weekend. After hours there are not enough rostered staff working to handle the workload of a transplant. The nature of the operation means that one person has to be dedicated to the sample received. When the person is called in their job is to register, process the sample for a PR and fibrinogen, and phone the theatre with the results. By that stage there is usually another specimen on the way. For consistency the PR and fibrinogen are measured using the Behring fibrintimer, a manually operated system which handles markedly abnormal results well, should they occur.

The liver transplant has 5 phases; preparation, induction to hepatectomy, anhepatic phase, reperfusion and post reperfusion to intensive care.

The liver transplant protocol has guidelines for when bloods are to be taken off.

- Baseline at induction
- Hourly until
- 5 min before anhepatic phase
- 10 min into anhepatic phase
- Hourly until
- 5 min prior to reperfusion
- 10 min post reperfusion
- 30 min post reperfusion
- Hourly until close

On average we receive 10 specimens over 4-6 hours with an average turnaround time of 20 minutes. Which is well under our 30-minute target.

The difficulty with this operation is that the liver produces the majority of the coagulation factors. The pre-existent poor synthetic ability of the liver compromises coagulation. Which is obviously exacerbated when the liver is removed. This is the stage where normally the PR rises and the fibrinogen drops. Also there is a tissue plasminogen activator (tPA) and tissue plasminogen activator inhibitor (tPAI) imbalance. These two factors help regulate the fibrinolytic pathway. tPA converts plasminogen to plasmin, which in turn breaks down cross-linked fibrin in a clot. In liver failure tPAI is not produced in normal amounts and does not hold tPA in check. Thus the patient is hyper-fibrinolytic which predisposes to the patient's wounds spontaneously rebleeding after clotting. Once the new liver is reperfused, it produces the coagulation factors depleted during the operation.

Blood bank support is a critical area of the transplant. On average 8.1 units of red cells and 10.3 units of fresh frozen plasma are transfused.

Transfusion of blood products is done as per the following protocol.

Red cells. As required to maintain the haemoglobin at 90-100g/L

Fresh frozen plasma. As required to maintain intrinsic factor levels. (30% of normal is usually adequate. If there is no active bleeding then an INR of <2.5 is adequate)

Platelets. Bleeding may be associated with quantitative and qualitative platelet defects. It is rarely necessary to transfuse if the plt count is >100 and the plt count of 50 is usually sufficient in the absence of fibrinolysis.

There are a few clues that the new liver is working post reperfusion; decreased acidosis, lactate, potassium, and glucose levels and spontaneous correction of the coagulopathy.

There have been eleven transplant recipients between February and October 1998. At present all are alive and well.

Thanks to all the Liver Transplant Unit Staff, especially Kerry Gunn, and the Haematology Staff at Auckland Hospital.

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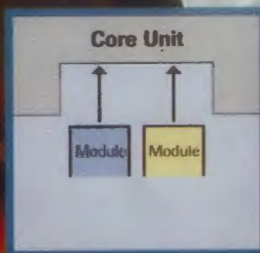
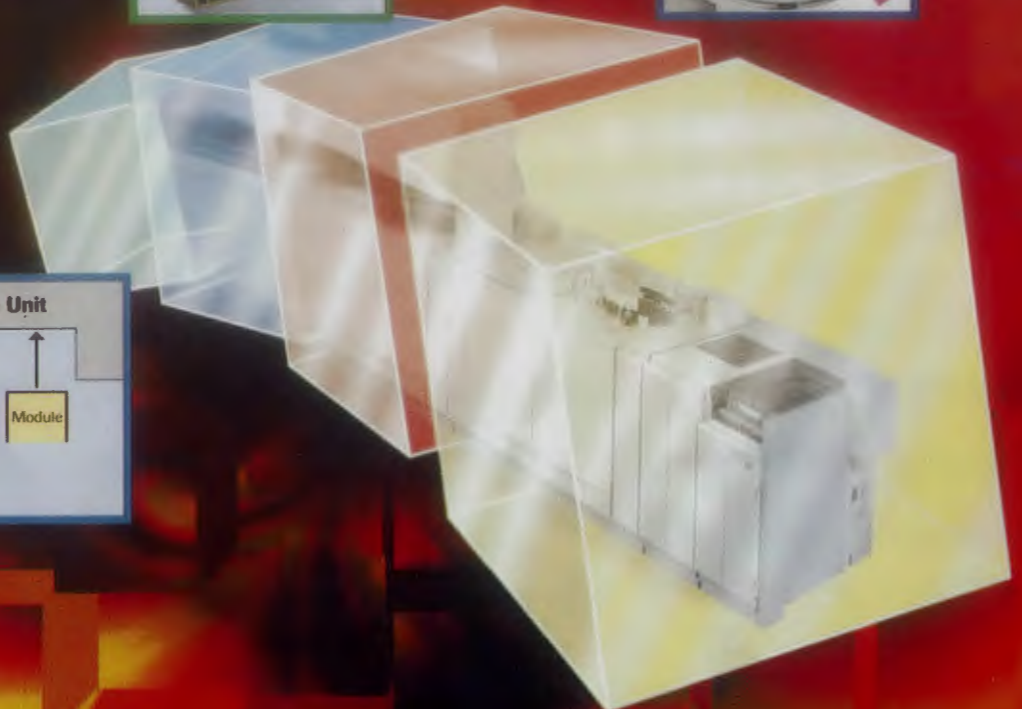
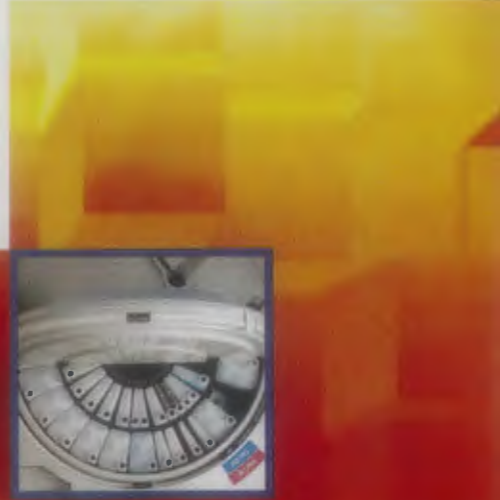


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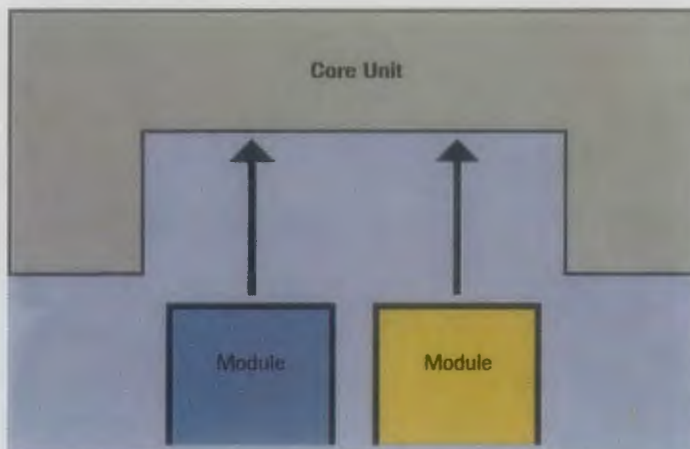
MODULAR ANALYTICS is just one part of the new **MODULAR SYSTEMS** platform which combines Pre-analytics, Clinical Chemistry and Immunology.

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New laboratory solutions must optimize the return on investment. A new system has to combine a proven reliability and a superior menu performance to ensure that laboratory efficiency is improved.

The results produced must be not only reliable but also standardized across STAT and satellite laboratories.

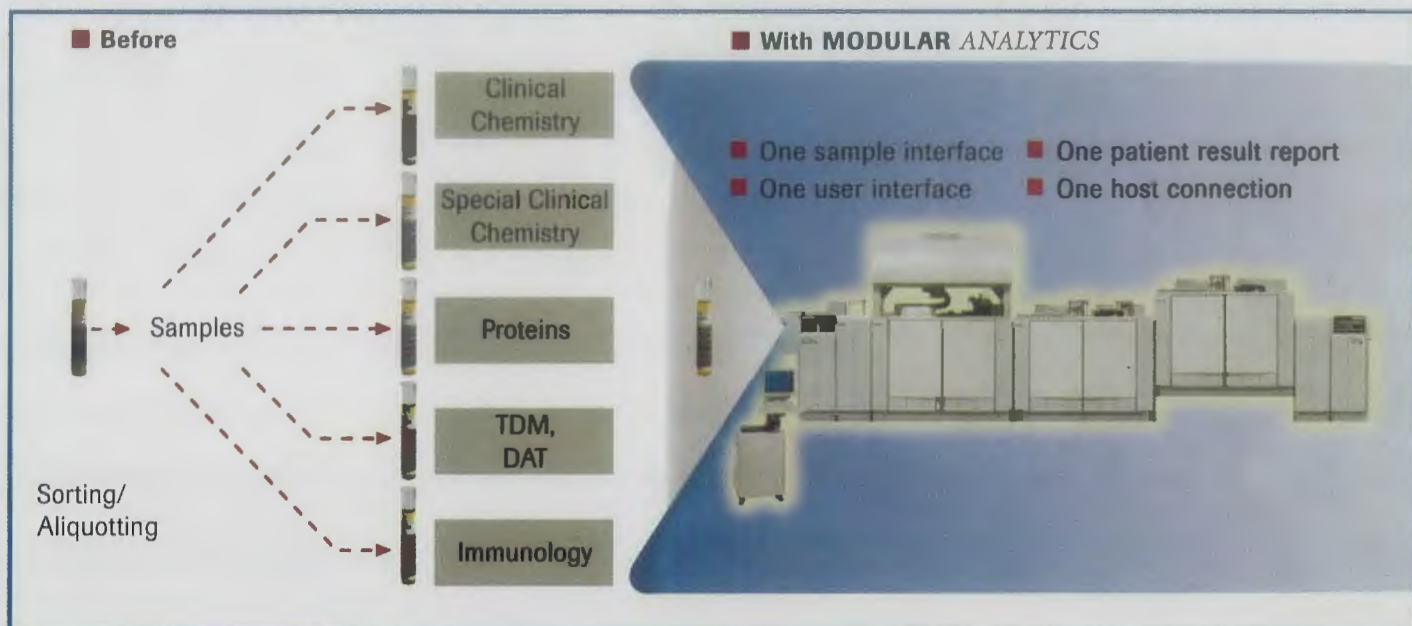


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The system consists of the Core Unit analytics with its built in "Intelligent Process Management" combined with a wide variety of different analytical modules to meet the needs of your laboratory.

MODULAR ANALYTICS successfully combines Clinical Chemistry and Immunology on a single, easy-to-use workarea.

MODULAR ANALYTICS simplifies work processes on to a single workarea.



**THE SOUTH PACIFIC CONGRESS AND THE HAEMATOLOGY
WORKSHOP COMMITTEE WISH TO INVITE REGISTRATIONS
FOR**

***The Leukaemia and Lymphoma
Workshop***



WHERE: Christchurch Convention Centre
South Island, New Zealand

WHEN: 23rd - 24th August 1999

(in conjunction with the SOUTH PACIFIC CONGRESS)

CONTENT: The workshop aims to provide an update on Leukaemia and Lymphoma, incorporating all the complimentary disciplines, to provide an overall classification package. This will be based on the proposed W.H.O classification. Organisers have been fortunate to secure *Professor Richard Brunning* from the University of Minnesota as our expert guest speaker. Specialists from each of the complimentary disciplines of Cytogenetics, Immunophenotyping, Molecular Biology, Histopathology and Cytochemistry will provide an overview of their techniques and role in helping to establish a diagnosis.

REGISTRATION FEE :

NZ\$180.00 for NZIMLS and AIMS members

NZ\$225.00 for non-members

Please direct registration and accommodation enquires to:

The Conference Secretariat
Executive Events
P.O Box 78
Rangiora
New Zealand

Tel: 64-3-313 4761

Fax: 64-3-313 2098

**THE NEW ZEALAND INSTITUTE OF MEDICAL
LABORATORY SCIENCE [Inc]**



EXAMINATION LIFTOUT

**QTA Regulations
QTA Examination Application Form**

NZIMLS Membership Application Form

The New Zealand Institute of Medical Laboratory Science offers to medical laboratory assistants the qualifications known as the Certificate of Qualified Technical Assistant (QTA).

All correspondence relating to Fellowship and the QTA examinations should be addressed to:

**Executive Officer
NZIMLS
P O Box 3270
Christchurch
Tel: 03 313 4761
Fax: 03 313 2098
Email: NZIMLS@exevents.co.nz**

THE NEW ZEALAND INSTITUTE OF MEDICAL LABORATORY SCIENCE [Inc]

CERTIFICATE OF QUALIFIED TECHNICAL ASSISTANT

EXAMINATION SUBJECTS

Clinical Biochemistry
Haematology
Histological Technique
Clinical Cytology
Immunology

Transfusion Science
Transfusion Science - Blood Products
Clinical Microbiology
Clinical Mortuary Hygiene and Technique

PREREQUISITES

1. Candidates for the examination must be employed as medical laboratory assistants in an approved laboratory in New Zealand and have worked continuously in the subject for 18 months prior to the examination or accumulated not less than 18 months practical experience in the examination subject. Upon completion of two years continuous or accumulated practical experience in the subject, the certificate of Qualified Technical Assistant will be awarded.
2. Candidates who have passed a Qualified Technical Assistant examination and who wish to sit a second Qualified Technical Assistant examination must fulfil the above criteria but need only to have worked continuously or accumulated experience of one year in the examination subject.
3. **Candidates must be financial members of the NZIMLS at the time of sitting the examination and be a financial member or have submitted a valid membership application form at the time of applying to sit the examination.**

SYLLABUS

Copies of the syllabus are available from the Executive Officer of the NZIMLS, P O Box 3270, Christchurch.

EXAMINATION

1. The examination will be held annually in New Zealand in November.
2. Candidates must complete the application form and forward this, complete with examination fees, to the Executive Officer of the NZIMLS before the closing date. **No late applications will be accepted.**
3. Candidates must be financial members of the NZIMLS at the time of sitting the examination.
4. The examination consists of one written paper of three hours duration. Candidates for the Clinical Cytology examination are also required to complete a practical examination.
5. To pass the examination candidates must obtain an overall mark of 50%. Clinical Cytology candidates must pass the practical and theory examinations.
6. The results of the examinations will be announced by the NZIMLS. Successful candidates will be awarded the NZIMLS QTA Certificate in the appropriate discipline.
7. The candidate's script will be returned upon receipt of a written request by the candidate. No copy will be retained and no correspondence relating to the marking of the script will be entered into.
8. Candidates who have disabilities or injuries at the time of the examination may request the Examinations Committee of the NZIMLS to allow them a scribe. Details may be obtained from the Executive Officer of the NZIMLS.

**THE NEW ZEALAND INSTITUTE OF MEDICAL
LABORATORY SCIENCE [Inc]**

**APPLICATION TO SIT THE EXAMINATION OF QUALIFIED TECHNICAL ASSISTANT
3 November 1999**

SECTION 1 - TO BE COMPLETED BY THE CANDIDATE

Title: Mr, Mrs, Miss, Ms Surname:.....First Names:.....

Of, Laboratory:.....

Laboratory Address:.....

.....

Subject: (Haematology, Microbiology etc):.....

EXAMINATION FEE: \$125 (GST Inclusive)
The full examination fee must be paid with the application.



SECTION B - TO BE COMPLETED BY THE PATHOLOGIST OR CHARGE SCIENTISTS

Date candidate commenced work in examination subject:.....

"I certify that the above candidate meets the requirements of the QTA Regulations"

Signed:..... Designation:.....

Please state the name and address of the person responsible for receiving the papers and supervising the Examination in your laboratory or centre.

Name:.....

Laboratory Address:.....

.....



APPLICATIONS CLOSE FRIDAY 21ST MAY 1999

Please forward application forms accompanied by fees to: NZIMLS, P O Box 3270, Christchurch

NO LATE APPLICATIONS WILL BE ACCEPTED

Special Note to Applicants

If no already members of the NZIMLS applicants to sit this examination **must** submit a valid membership application along with this examination application.



For Office Use Only

Date received:..... Cheque number:.....

Bank:..... Branch:.....

Drawer:..... Amount:.....



THE NEW ZEALAND INSTITUTE OF MEDICAL LABORATORY SCIENCE (INC)

PO Box 3270, Christchurch, New Zealand Tel (03) 313 4761 Fax (03) 313 2098

APPLICATION FOR MEMBERSHIP

(Please print clearly AND Tick appropriate Box)

Title: Dr, Mr, Mrs, Ms, Miss Surname: First Names:

Of, Laboratory:

Address:

Address for Correspondence if not as above:

I hereby apply for membership of the New Zealand Institute of Medical Laboratory Science in the category selected below (please see membership categories near bottom of page):

- Member (\$101.40)
- Associate (\$48.10)
- Non Practicing (\$44.20)

I am employed as:

In the Speciality Department of:

Highest Professional Qualification: Year obtained:

Specialised in:

Nominated by: (Current Financial Member NZIMLS)

Signed: Date:

- I enclose the completed membership application form and payment of: \$ (NOTE - Examination candidates must pay 1st years subscription in full)
- OR
- By Salary Deduction on a fortnightly basis: Please sign the deduction form below. We will advise your employer.

CLARIFICATION OF MEMBERSHIP CATEGORIES:

- Member:** Any person who is registered by the Medical Laboratory Technologist Board.
- Associate:** Any person engaged in Medical Laboratory Science who is not eligible for any other class of membership eg QTA.
- Non Practicing:**
 1. Any person who has been a Fellow, Member or Associate, but is no longer engaged in Medical Laboratory Science and who elects to become a non practicing member.
 2. Any person not engaged in Medical Laboratory Science.

Thank you for your membership application

AUTHORISATION FOR DEDUCTION FROM SALARY

Name:

Employer:

I authorise the above-named employer to deduct current subscriptions to the New Zealand Institute of Medical Laboratory Science (Inc) from my salary to pay the amount to that Institute. This authority is to remain in force until canceled in writing by me.

Signed: Date: NZIMLS Computer No:

mmb/1/98

“Time is on your side”



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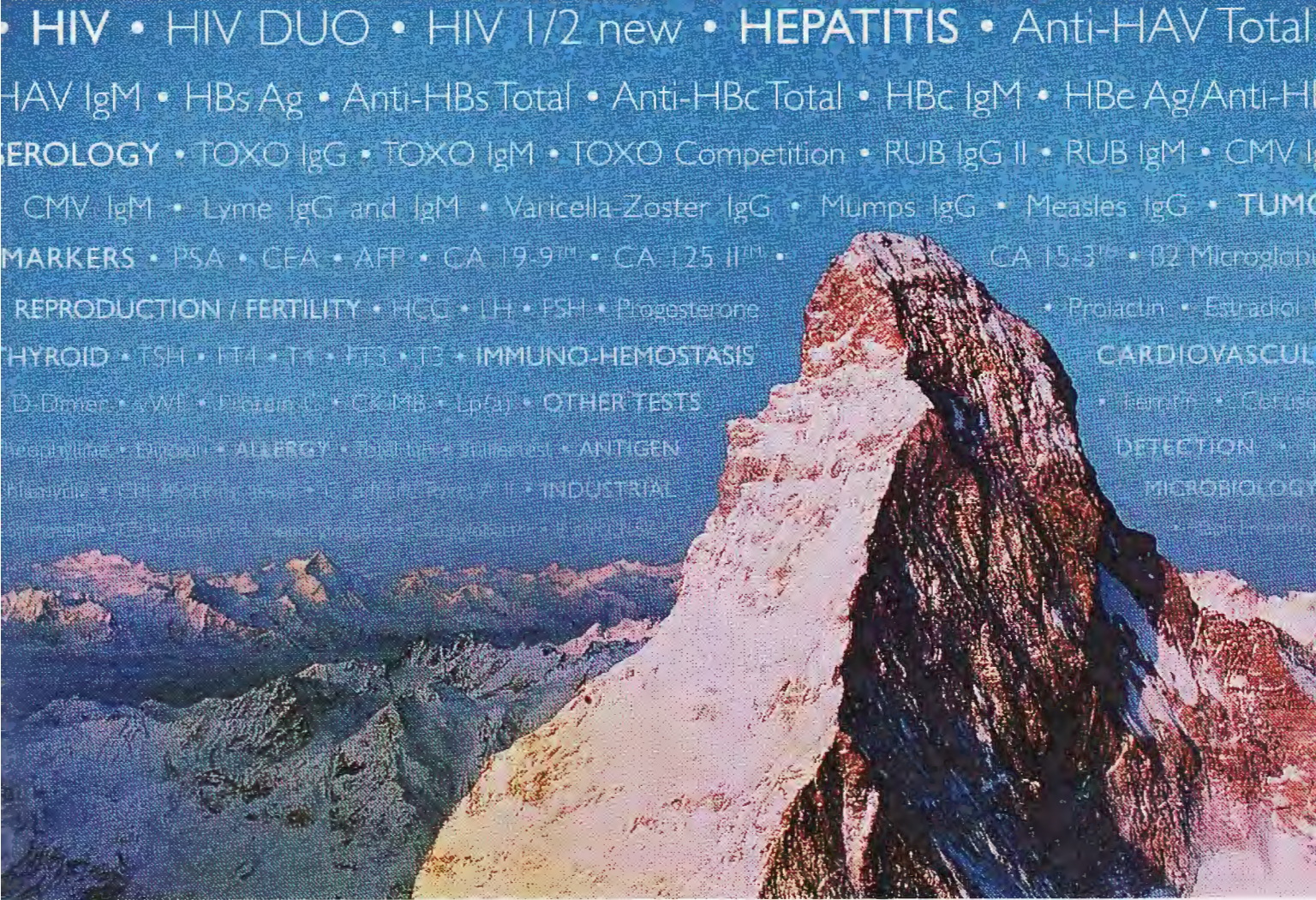
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 D-Dimer • vWF • Fibrinogen • CK-MB • Lp(a) • OTHER TESTS • Ferritin • C-reactive protein
 Hepatitis A • Hepatitis B • ALLERGY • Total IgE • Specific IgE • ANTIGEN • DETECTION •
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NZIMLS COUNCIL NEWS



Needs Analysis

A Needs Analysis for future Medical Laboratory Scientist requirements will be undertaken this year. Information is currently being gathered from both Educational Institutions and potential employers. It is hoped that all concerned will co-operate for the benefit of the profession.

1998 QTA EXAMINATIONS

Successful QTA candidates for 1998

Biochemistry

Louise Robertson
Lynette Clarke
Leonie Dutch
Taryrn Shapcott
Christine Hall
Catherine Ormerod
Denise Shaw
Hilary Addison
Selina Fa'asolo
Rachel McDaid
Rachael Goulin
Sean O'Neill
Sara Thomas *

Cytology

Regan Kendrick *
Iskra Citkuseva

Haematology

Misiona Nicholas
Autonina Volikova *
Zora Feilo

Histology

Anton Schollum
Alex Frolov
Kirstie Roff
Bharathi Cheerala *
Cara Mackle
Leanne Coote

Immunology

Varsha Jeram
Sashi Rao
Maree Chambers
Helena Thompson
Kerryn Wilton
Marita Smit *
Nicole Jones
Belinda Grossman
Lynda Jayet

Microbiology

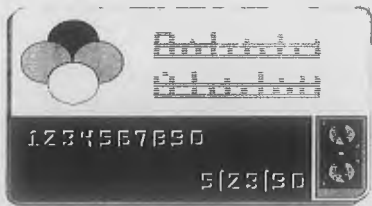
Shirley Aickin
James Brokenshire
Fay Cobbett
Paula Davidson
Deborah Wetherall *
Gaylene McKenzie-Parker
Erin Hart
Emily Nicholas
Linda Reynolds
Ryan Hunter
Marie Hogg *
Donna Doreen *
Kyla Hughson
Nicola Wilson

Mortuary Hygiene

Robert Peters *



* Identifies top candidate



A Visa option of payment will be available this year with your annual membership subscription invoice.

FELLOWSHIP

Remember! Only one (1) year left for this window of opportunity.

Medical Laboratory Scientists who hold a Specialist Certificate are exempt from sitting the Part I examination. They may proceed directly to the Part II Dissertation of 3-5,000 words.

The final date for applications under Clause 3.12 of the Regulations is March 31st 2000. Contact Executive Office.

GENERAL

- ♣ Financially, the NZIMLS has recovered from it's woes of two years ago. Council are now reviewing ways in which services can be delivered to members. Continuing Education and underwriting the efforts of the SIGs will remain a primary focus.
- ◆ Council have requested that SIGs place any recommendations for nominees before Council for consideration for the following:
 - ◆ MLTB - Medical Laboratory Technologists Board
 - ◆ Life Member - NZIMLS
 - ◆ Honorary Member - NZIMLS
- ♠ It appears User Group meetings with good scientific and/or technical content are eligible for MOLS points with the MLTB. The MLTB should clarify this soon. Obviously any social content/time would be excluded.
- ♥ The NZIMLS has examined the need to change BMLS courses because of the introduction of core laboratories in New Zealand. The NZIMLS will not be recommending any additional options or changes in the structure of the Bachelor of Medical Laboratory Science degree courses.

A general science base, plus learning in all disciplines addressed in the 3rd year is a strong base for a Registered Medical Laboratory Scientist wherever they work. Fourth year specialisation in two disciplines completes the degree course with the option of further post graduate qualifications in any of the other disciplines.



The degree courses were specifically designed to provide the competency and flexibility required for mobility within our profession and workforce. This includes core laboratory, small laboratory and specialisation in a large laboratory employment. Each laboratory accredited by IANZ will have its own ongoing competency and training programme specific to it's service requirements.



1999

Thursday 15 April 1999

Biomedical Science - the Key to Control of Diabetes

2000

Date to be advised.

Infectious Diseases (Immunology, Virology and Microbiology components)

Start thinking how you can promote our profession NOW for the year 2000:

Which public will you choose? Medical/general public.

How will you approach the topic of Infectious Diseases?

- ✧ prevention
- ✧ detection
- ✧ epidemiology

This is our opportunity to promote our profession

1998 Conference Revisited.....



Winner of the 1998 Industry Display Award
BioRad Laboratories
Shirley Gainsford, Simon Pratt & Elizabeth Bonagura



Our 'flash' Conference Convenor
Chris Kendrick



The docking of the 'Lochness Monster'
Special Guest Dr Ian Wright from Scotland
in the dock with Jan Deroles-Main, Bronwyn
Sheppard and Ann-Louise Weaver



No Comment !!



Cheers Fellas!!



Line Dancing Concentration

NZIMLS WEBSITE DEVELOPMENT

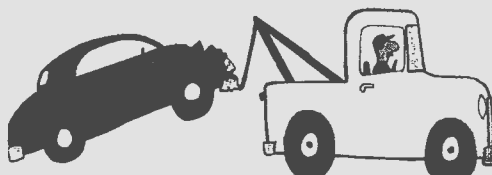
In 1998 the NZIMLS Council investigated the potential for the use of the World Wide Web (WWW) for Institute activities. At the November meeting of the Council it was decided to proceed with the development of stage one of an NZIMLS webpage. Details of costings and webste structure are still being finalised between the project coordinator Chris Kendrick and the website developer, KC Multimedia (www.kc.co.nz) of Palmerston North.

Once registered it is hoped to use the URL (www.nzimls.org.nz) which will be linked to all commonly used web search engines. The site is to be dynamic (web talk for updated frequently) and will be maintained by the executive office. As mentioned the exact features of stage one are yet to be decided upon but ultimately it is hoped that the site will provide:

- calendar of MLS events
- advertising and promotion of the ASM and SIG activities
- improved communication between the Council and its members
- promotion of the NZIMLS and Medical Laboratory Science as a career
- cover and contents page for each publication of the NZIMLS journal
- electronic membership application
- membership status queries
- annual subscription payments
- electronic examination application fees payment
- MLS links

The site will utilise a re-stylised NZIMLS logo and at this stage it is hoped to be online by the middle of 1999. Any comments, suggestions, should be directed to Chris Kendrick C.J.Kendrick@massey.ac.nz or your regional NZIMLS representative.

If Microsoft Made Cars....



At a recent Computer expo (CONDEX?), Bill Gates reportedly compared the computer industry with the auto industry and stated: "If GM had kept up with technology like the computer industry has, we would all be driving twenty-five dollar cars that got 1000 miles to the gallon."

In response to Bill's comments, General Motors issued a press release stating: If GM had developed technology like Microsoft, we would all be driving cars with the following characteristics:

- For no reason whatsoever your car would crash twice a day.
- Every time they repainted the lines on the road you would have to buy a new car.
- Occasionally your car would die on the freeway for no reason, and you would just accept this, restart and drive on.
- Occasionally, executing a manoeuvre such as a left turn would cause your car to shut down and refuse to restart in which case you would have to reinstall the engine.
- Only one person at a time could use the car, unless you bought "Cars95" or "CarNT". But then you would have to buy more seats.
- Macintosh would make a car that was powered by the sun, reliable, five times as fast, and twice as easy to drive, but would only run on five percent of the roads.
- The oil, water temperature and alternator warning lights would be replaced by a single "general car default" warning light.
- New seats would force everyone to have the same size butt.
- The airbag system would say 'Are you sure?' before going off.
- Occasionally for no reason whatsoever, your car would lock you out and refuse to let you in until you simultaneously lifted the door handle, turned the key, and grab hold of the radio antenna.
- GM would require all car buyers to also purchase a deluxe set of Rand McNally road maps (now a GM subsidiary), even though they neither need them nor want them. Attempting to delete this option would immediately cause the car's performance to diminish by 50% or more. Moreover, GM would become a target for investigation by the Justice Department.
- Every time GM introduced a new model car buyers would have to learn how to drive all over again because none of the controls would operate in the same manner as the old car.
- You'd press the 'start' button to shut of the engine.

5th SOUTH PACIFIC CONGRESS

Profiles of some invited speakers

Dr Vaughan Clift

Received his Medical Degree from the University of Melbourne, past the entrance examination to the Royal Australasian College of Physicians at 27, and practised clinical medicine at the Royal Melbourne and Royal Children's Hospitals for 8 years. He began his research career as the Fellow in Endocrinology at the Royal Children's Hospital and began a PhD at the University of Melbourne. He developed a non-invasive blood glucose sensor which was patented and the project was transferred and sold to Novo Nordisk, Denmark. He worked for one year as a Senior Research Scientist in Novo Nordisk's, Medical Sciences Division, Copenhagen, to transfer the technology and remained a member of an International Scientific Advisory Board for Novo until 1995.

In 1991, he was invited to NASA's Johnson Space Centre in Houston, by Dr Carolyn Huntoon, Director of the Centre. As an Australian citizen, he was unable to work as a US Civil servant and was employed through a Government contract held by Lockheed Martin Corporation. As the Chief Scientist, Medical Sciences Products group, Lockheed Martin for the five years he managed a research team studying medical problems associated with space flight and developing innovative technologies for in flight research. This grew rapidly to half a dozen projects and \$US1 million annual budget.

In 1993, after only 8 months of development, he had his first experiment flown on board the Space Shuttle Columbia and subsequently has had numerous experiments flown on board the shuttle. He has completed over 700 parabola on NASA's KC 135, with a total of 3 hours in microgravity.....all in 30 second bursts. In September 1997, had his first experiment as a co-inventor of a technology being flown on board the Russian Space Station, Mir.

In March 1997, he formed a new company, DBDC Inc., to transfer the blood/serum separation technologies to the commercial sector and signed a Space Act Agreement, with NASA to continue to support shuttle activities, Mir experiments and development of research studies and technology for the International Space Station beginning construction late 1998.

Professor Richard Brunning, University of Minnesota

Dr Brunning is currently Professor of Laboratory Medicine and Pathology and Director of Haematology Laboratories, University of Minnesota and is Deputy Chairman of the Department of Laboratory Medicine and Pathology.

Professor Brunning is internationally known for his work on morphology, cytogenetics and immunological features in Leukaemia and Lymphoma. He has been involved in laboratory medicine since 1965 and is a lecturer and tutor of Neoplastic Haematopathology. He has been involved in the new WHO classification of Leukaemias and Lymphomas as a guest lecturer for the European School of Haematology 1998. He has authored over 130 papers and numerous chapter publications for Haematopathology.

Dr Peggy Whitson

Received a Bachelor of Science Degree in Biology/Chemistry from Iowa Wesleyan College in 1981, and a Doctorate in Biochemistry from Rice University in 1985.

From 1981 to 1985, Peggy conducted her graduate work in Biochemistry at Rice University, Houston, Texas as a Robert A Welch Predoctoral Fellow. Following completion of her graduate work she continued at Rice University as a Robert A Welch Postdoctoral Fellow until October 1986. Following this position, she began her studies at NASA Johnson Space Centre, Houston, Texas, as a National Research Council Resident Research Associate. From April 1988 until September 1989, she served as the Supervisor for the Biochemistry Research Group at KRUG International, a medical sciences contractor at NASA-JSC. In 1991, she was also invited to be an Adjunct Assistant Professor in the Department of Internal Medicine and Department of Human Biological Chemistry and Genetics at University of Texas Medical Branch, Galveston, Texas.

NASA Experience

From 1989 to 1993, Peggy worked as a Research Biochemist in the Biomedical Operations and Research Branch at NASA-JSC. In 1990, she gained the additional duties of Research Advisor for the National Research Council Resident Research Associate. From 1991 - 1993, she served as Technical Monitor of the Biochemistry Research Laboratories in the Biomedical Operations and Research Branch. From 1991 - 1992 she was the Payload Element Developer for Bone Cell Research Experiment (E10) aboard SL-J (STS-47), and was a member of the US-USSR Joint Working Group in Space Medicine and Biology. In 1992, she was named the Project Scientist of the Shuttle-Mir Programme (STS-60, STS-63, STS-71, Mir 18, Mir 19) and served in this capacity until the conclusion of the Phase 1A Programme in 1995. From 1993 - 1996 Peggy held the additional responsibilities of the Deputy Division Chief of the Medical Sciences Division at NASA-JSC. From 1995 - 1996 she served as Co-Chair of the US-Russian Mission Science Working Group. In April 1996, she was selected as an astronaut candidate and in, August 1996, began two years of training and evaluation. Successful completion of initial training qualified her for various technical assignments leading to selection as a mission specialist on a Space Shuttle flight crew.

Peggy is the recipient of many awards/honours and has a prestigious list of publications and presentations.



Speaker Profiles cont...

Dr Annette Trickett

Annette initially studied medical science in the UK, specialising in Haematology and Blood Bank. After working in the UK for a couple of years, she worked in South Africa and Saudi Arabia before going to Australia in 1985. Her first job in Sydney included Leukaemia Immunophenotyping and Bone Marrow Transplant technology.

Annette obtained a Masters degree with a thesis on purging autologous bone marrow in 1992, and completed her PhD thesis on cryopreservation and culture of autologous lymphocytes for patients with HIV last year.

Currently, Annette runs the Bone Marrow Transplant Laboratory at St George Hospital in Sydney.

Dr Ian Morison, Dunedin

Ian Morison trained in Clinical Pathology (Laboratory Medicine) at the University of Washington, Seattle. In the Department of Laboratory Medicine, he had particular interests in haematology, molecular diagnostics, clinical immunology and enzymology. He returned to Dunedin where he completed his FRCPA in Haematology.

Ian now combines careers in diagnostic and research Haematology. His diagnostic work is performed as a Haematologist/Clinical Pathologist with Southern Community Laboratories, where he is particularly interested in the difficulties of establishing reference ranges and communicating the data that they contain.

As a Senior Research Fellow in the Cancer Genetics Laboratory, he has research interests in childhood ALL, familial thrombocytopenia, genomic imprinting and overgrowth disorders.

Dr Lester Levy

Lester Levy is an entertaining, provocative and inspirational speaker with a wealth of practical and applied experience. He is best known for leading a number of successful business transformations in both the private and public sectors.

Lester is an accomplished communicator whose speaking style results in the audience carrying long lasting visual images in their mind which can be applied when the opportunity arises ... and he does all this with humour. It is extremely common for people who have been in Lester's audience to return to work the next day and immediately apply some of the principles and fundamentals at the core of his message.

Lester's key attributes include:

- leadership
- track record of management achievement
- strategic planning expertise
- major success in organisational restructuring
- marketing and financial expertise
- wide experience in technology implementation
- experience in managing large organisations
- experience in both the private and public sectors
- political astuteness

Lester graduated with a degree in medicine in South Africa. After specialising he altered his direction to take up a career in business, graduating with a Master of Business Administration degree.

He moved through the multi-national environment with 3M, Beecham Research Laboratories and SmithKline Beecham in a range of senior management positions. From the multi-nationals he moved into the public health sector with a desire to bring to the sector a stronger commercial and customer focus. Lester was General Manager in the Bay of Plenty, from where he was seconded to the Department of the Prime Minister and Cabinet in 1992.

In May 1997 Lester resigned his position as Chief Executive

Dr Tom Exner, Gradipore Limited

After graduating from Sydney University in 1967, Thomas Exner went on to do a PhD in Biochemistry at the University of Waterloo in Ontario in 1972. He has considerable experience in developing new clotting tests, in particular those based on snake venom enzymes, in the expectation that such tests may simplify the laboratory diagnosis of bleeding and clotting abnormalities. He had 10 years experience in Haematology at Royal Prince Alfred Hospital followed by 10 years as Principal Hospital Scientist at Westmead Hospital in Sydney. He has written over 100 publications and given numerous presentations. Much of his work has been to investigate circulating inhibitors of coagulation and standardise blood clotting methods, but more recently he has been investigating simplified methods for thrombophilia screening in private enterprise at Gradipore.

of South Auckland Health which he had held since early 1993. South Auckland Health is focussed on health delivery and has revenue of \$200 million and employs 3,200 staff. It has had the most significant turnaround of any of the 23 Crown Health Enterprises. Commencing three years ago with an operating deficit of \$23 million, South Auckland Health is one of only two of the 23 not in deficit situation and is the most profitable. South Auckland Health is also well advanced on a major futuristic facilities modernisation programme.

In 1994 Lester was awarded a prestigious Kings Fund International Fellowship from the Kings Fund in London. These fellowships are awarded every two years to the 25 outstanding healthcare managers internationally.

On leaving South Auckland Health Lester began a new career as an investment banker with the Calan Group, specialising in the health sector and also took on the position of Chairman of Comunicado, an independent film and television company.

Gilbert Enoka

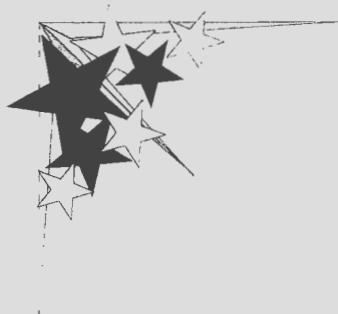
Gilbert is a highly skilled practitioner who has worked in the area of enhancing performance for the past 10 years. He is a practising consultant to a wide range of corporate and sporting organisations where he facilitates a process of personal improvement through the development and refinement of psychological skills. He encourages individuals to take ownership of their direction and to improve their ability to control the many variables that inhibit optimal performance.

His most recent campaign has been as part of the management team of the 1998 champion super 12 rugby team, the Canterbury Crusaders and he has recently accepted a contract with NZ Cricket up until and including the 1999 World Cup. He continues to work with numerous sporting and corporate groups and acts as a mentor to a number of NZ's high flying Chief Executives.

NZIMLS

1999

AWARDS



**BOEHRINGER MANNHEIM MEDAL
AND TRAVEL GRANT IN
CLINICAL BIOCHEMISTRY**

Medal plus \$1,000 travel grant for the best Clinical Biochemistry paper presented at the

**5th South Pacific Congress
Christchurch
23rd - 27th August 1999**

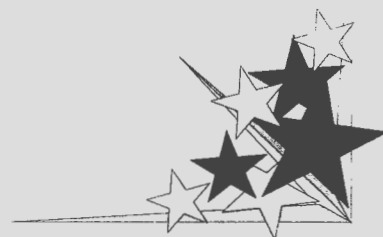
*All fellows, members and associate members
of the NZIMLS are eligible.*

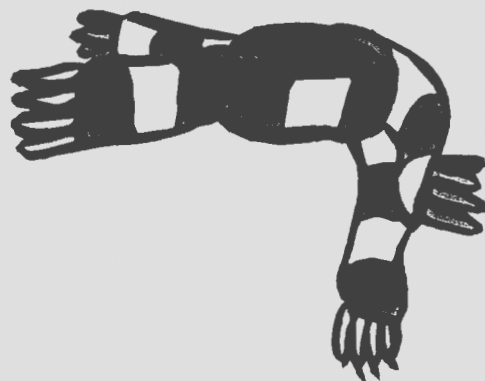
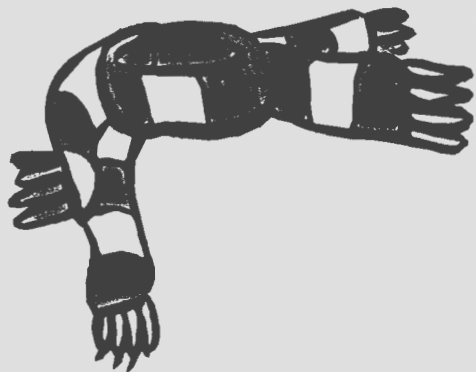
Criteria

The best paper in the field of Clinical Biochemistry presented at the 5th South Pacific Congress.

Recipient

Of the travel grant is requested to prepare the paper for publication in the New Zealand Journal of Medical Laboratory Science.





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APPLICATIONS CLOSE 30TH JUNE 1999

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Is an **award** to the value of **\$150** for each issue of the NZIMLS Journal. It is offered three times a year.

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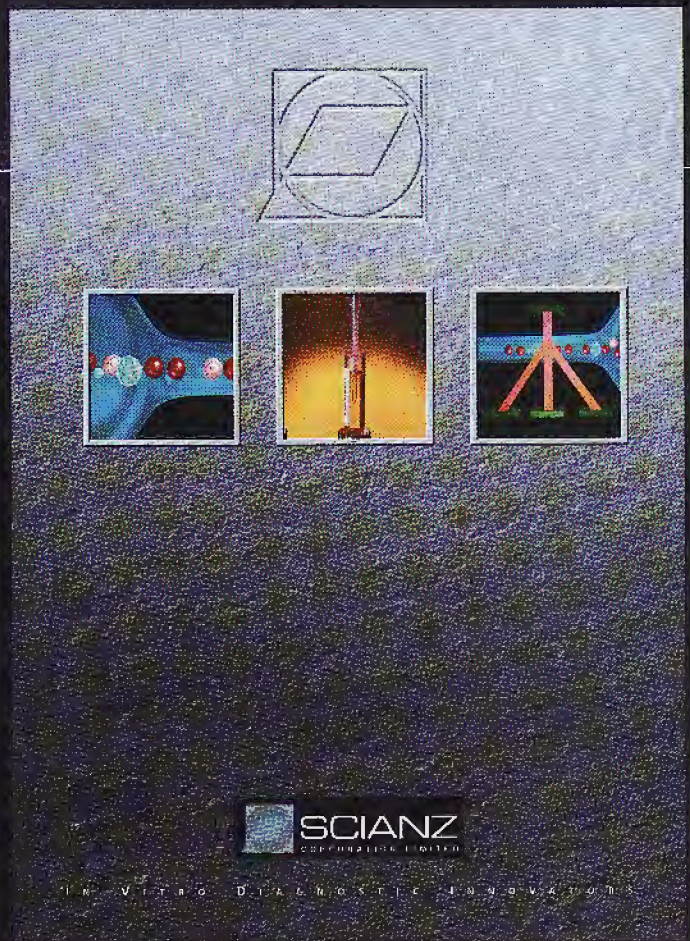


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