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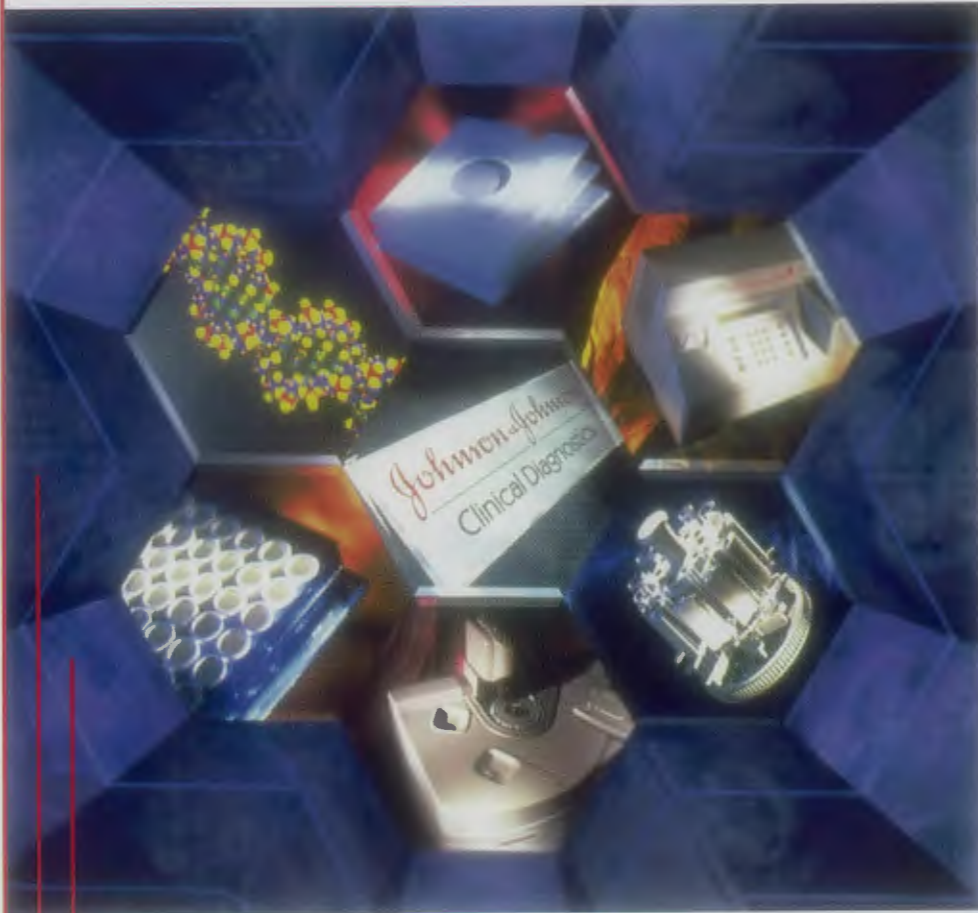
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EDITORIAL

Medical Laboratory Technology – A Regulated Profession?

In 1972 the Medical Auxiliaries Act 1966 was amended to include medical laboratory technologists. Since then those who practise medical laboratory technology and use the title medical laboratory technologist must be registered by the Medical Laboratory Technologists Board (MLTB) and hold an annual licence.

In 1996 the Ministry of Health (MOH) undertook a review of this regulation along with other health occupation regulations. The NZIMLS was asked to discuss a number of issues with the MOH and to make a written submission on the following.

- 1) Should Medical Laboratory Technologists (MLTs) be regulated?
- 2) If so what form should this regulation take. Should it be licensing, certification or registration?
- 3) Could the Medical Practitioners Act 1995, act as a model for regulation of MLTs should regulation remain?

The NZIMLS submitted a response to these questions and in November 1996 the MOH published a draft report titled "Review of the Arrangements for Licensing Podiatrists, Medical Radiation Technologists and Medical Laboratory Technologists."

Some of the main points in the draft report are discussed below.

1. The need for regulation.

The question asked was: "Would the absence of statutory regulation for medical laboratory technologists result in significant harm to consumers or significantly endanger public health and safety?"

The Ministry supported regulation for podiatrists and Medical Radiation Technologists (MRTs) because they deal directly with the public and may cause harm by using ionising radiation in the case of MRTs and infection or reaction to anaesthetics in the case of podiatrists. However, they stated that "In comparison to MRTs and podiatrists the risks posed by the work of MLTs is not great." They then presented arguments for and against the continued regulation of MLTs.

A response was made to the draft report, in particular to the arguments against regulation for MLTs. The main points are summarised below with the statements in italics being the ministry's arguments and the NZIMLS response in normal type.

a) *"Generally MLTs do not interface directly with the public. The risk to the public comes, from the medical practitioner who requests the laboratory test and who may give incorrect treatment rather than the direct action of the MLT."*

We have explained that medical practitioners act on our results, they rely on them to be correct and in some instances immediate action is necessitated by the results, for example results on CSF and blood gas results to name two. The obvious example of incompatible blood transfusion causing direct harm has already been made.

b) *"Employers currently take responsibility for ensuring quality of their laboratory tests and there is no evidence to suggest that without regulation employers will employ unqualified incompetent persons."*

We have countered that if there is no regulation there is no guarantee that an unscrupulous employer will not emerge, or that

with current commercial pressures employers will not be tempted to reduce costs by employing unskilled labour. Employers do not always have the ability to decide who is competent. The MLTB has the ability to do this and has built up expertise in this area particularly in assessing overseas qualifications.

c) *"There is little overseas evidence of reduced consumer safety where MLTs are not regulated."*

This is true but may be because most countries which are similar to New Zealand ie. Canada, Britain and the USA have regulation. Australia does not but is proceeding towards regulation.

d) *"The NZIMLS could maintain standards without the need for regulation."*

We have replied that the NZIMLS is a professional society whose prime purpose is to benefit its members to whom it is accountable. The MLTB is there for the protection of the public and is accountable to the Minister of Health. The NZIMLS does not have the resources to monitor the BMLS courses or continuing competency, which are most important functions of the MLTB, ensuring that MLTs receive appropriate training. Most importantly the NZIMLS has no statutory power and practitioners of medical laboratory technology do not have to belong to it. Therefore maintaining standards would be voluntary.

2. What form of regulation should we have?

Currently we are subject to a licensing regime, which means that only those who have obtained a licence after meeting certain standards prescribed by the MLTB, are able to legally practice medical laboratory technology. Medical laboratory technology is defined in the act and we have exclusive rights to the title "medical laboratory technologist."

The Ministry suggests that the Medical Auxiliaries Act be amended so that MLTs are subject to a certification regime (they recommend the same for podiatrists and MRTs). This means that a person must reach a certain standard to be certified and restricts the use of the title MLT to those who are certified. It removes the need for defining medical laboratory technology and does not restrict the right of others to practise medical laboratory technology.

The Ministry think that this would ensure that unqualified people do not practice medical laboratory technology.

The NZIMLS is not convinced of this argument and still supports licensing. We have also asked for a name change to medical laboratory science and for protection of the titles Medical Laboratory Technologist and Medical Laboratory Scientist.

3. The Medical Practitioners Act 1995 as a model for regulation legislation.

The NZIMLS agrees that the Medical Practitioners Act 1995 would be a suitable model for the regulation of medical laboratory scientists.

The main changes to the current regulations would be:

- a) Competence.

Revised legislation would provide for reviewing MLTs

competence. The MLTB is already piloting a continuing competency programme, the MOLS programme.

b) Discipline

Registration and discipline which are currently both carried out by the MLTB would be separate. The MLTB would have a registration function, while discipline would be first investigated by a complaints assessment committee then prosecuted before a disciplinary tribunal. The MLTB and the disciplinary tribunal would have registered MLT and lay representation. The disciplinary tribunal would have legal representation and it would have much wider powers than the MLTB has at present, including the ability to remove people from the register, impose a fine, or impose supervision or conditions for practise.

c) Structure of the Board

The Board would be more independent, able to set its own fees, determine its own procedures and manage its own affairs. It has been recommended that Boards become bodies corporate providing more flexibility in the management of their business. However, this does mean an increased exposure to financial risk.

The Ministry is revising the draft report on the review of the regulation of podiatrists, MRTs and MLTs. The revised report will be widely circulated asking for more comment than a final report will be put to the government.

It is expected that the issue of regulation and the new Act will be completed by the end of 1997.

I believe that the regulation of medical laboratory technologists should remain, so that the standard of medical laboratory technology remains high in New Zealand and the public are assured of a safe service.

The Council will keep you informed of further developments.

1. Registration: Requires all practitioners to be on a register. Does not require a qualification.

Certification: Practitioners must reach a set standard and have exclusive right to a particular title. Does not restrict the right of others to carry out the occupation.

Licensing: Restricts the practice of an occupation to those who hold a licence. Must meet prescribed standard to gain a licence.

Shirley Gainsford
Valley Diagnostic Laboratories
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Paroxysmal Nocturnal Haemoglobinuria – A Review

Andrea Lun, BMLS student, Chris Kendrick, MNZIMLS, Lecturer – Massey University, Palmerston North

Paroxysmal nocturnal haemoglobinuria (PNH), formerly known as Machiafava-Michaelis syndrome,²⁷ is a rare acquired defect of marrow stem cells which produces an abnormality of the red cell membrane. This renders it sensitive to lysis by complement, causing chronic intravascular haemolysis⁶. PNE is estimated to affect 1/500,000¹⁵.

Genetic Defect

The defect lies in the phosphatidylinositol glycan²⁶ (PIG) anchor which attaches 15 or more proteins to the red cell membrane⁶. These proteins include:

1. complement defence proteins - eg. decay accelerating factor (DAF or CD55), homologous restriction factor (HRF or C8bp), membrane inhibitor of reactive lysis (MIRL or CD59)
2. receptors - eg. Fc gamma receptor III (CD16) and lipopolysaccharide receptor (CD14)
3. enzymes - eg. acetylcholinesterase, neutrophil alkaline phosphatase (NAP), ectonucleotidase
4. proteins of unknown function

About 10 genes are involved in PIG anchor biosynthesis but in PNH patients studies so far, all show somatic mutation in the pig-A gene¹⁴ which has been mapped to the X chromosome (Xp22.1)^{26,1,16}. Yamada *et al* studied Japanese patients with PNH and discovered there is no mutation hotspot in this gene ie. the mutations are entirely random and include single base changes through to frame shift mutations. Pramoonjago studied Thai patients with PNH and discovered the type of mutation varies between ethnic groups. In Thai patients, multiple base deletions are more common than in British and Japanese patients where the latter tend to have more substitutions. By 1995, 84 mutations had been identified in 72 patients¹⁶. Two other genes, pig-h and pig-f, (position unknown) can also contribute to PNH but these are autosomes and require defects in both alleles to produce the disease¹⁶. This form of PNH is rare. The cause of the pig-A gene mutation is unknown.

Gene defects have been shown to produce two populations of red cells, one sensitive to lysis and a normal population. The cells can be classified as:

- Type I which have normal sensitivity
- Type II with medium sensitivity ie. 3-5 times more sensitive than normal cells
- Type III very sensitive with 10- 15 times more sensitivity than normal⁴.

It has been suggested that the two populations are derived from a single BFU-E²¹. However, some patients develop two clones of PNH cells a result of two different mutations¹. Evidence that the PNH defect is expressed at the pluripotent haemopoietic stem cell is supported by the findings of Dessypris *et al* who found PNH CFU-M express increased complement sensitivity as seen in PNH red cells. The question of how the PNH clone can dominate so much has been asked. Bessler suggests that since PNH does not possess invasive neoplastic features, the PNH clone must have a growth or survival advantage where normal haemopoiesis is decreased. Yamada states that the PNH clone expansion stabilises in the early stages of the disease and this expansion is not determined by the mutation in the pig-A gene but by other factors ie. some selective mechanism where

normal haemopoietic cells are suppressed while the PNH clone expands.

PNH also affects granulocytes, platelets and lymphocyte² cell lines supporting the theory that the defect originates in the bone marrow resident stem cell.

Clinical Features

There are four basic components to PNHs pathophysiology⁶

1. Hyperhaemolysis
2. Bone marrow (BM) failure
3. Infection
4. Thrombosis

PNH patients are classified as having either a haemolytic form, where red cell destruction dominates the clinical picture, or an aplastic form where pancytopenia and its consequences are the main findings⁹. Increased haemolysis is due mainly to the deficiency in complement defence proteins ie. DAF, which accelerates the rate of destruction of erythrocyte-bound C3 convertase, HRF (which inhibits complement-mediated membrane channel expression) and MIRL (which regulates the assembly of polymerised C9 in the membrane attack complex)^{16,16}. CD59 (MIRL) is of great importance accounting for most of the lysis and may be responsible for thrombosis as absence of CD59 on platelets leads to a hypercoagulable state¹⁶.

Infection is due to the qualitative and quantitative abnormalities of the neutrophils⁹. There is a deficiency of Fc gamma receptor III on PNH phagocytic cells causing an increase in the number of circulating immune complexes and susceptibility to bacterial infections¹⁶.

Absence of urokinase plasminogen activator receptor (UPAR) on monocytes leads to more stable clots⁶. As a result, monocytes cannot bind urokinase which activates plasminogen to plasmin and cleaves the fibrin in clots. Ronne found that uPAR is elevated in the plasma of PNH patients but is deficient on affected monocytes and granulocytes. The binding of pro-uPA to uPAR in the plasma may interfere with binding of pro-uPA to cell-bound uPAR leading to inhibited cell associated plasmin generation and fibrinolysis. Venous thrombosis is also attributed to increased platelet activity. In PNH, platelets are abnormally sensitive to activated complement fragments which cause platelet aggregation. This may be induced by ADP released from lysed red cells¹⁶. Some patients may develop Budd-Chiari syndrome, a severe condition where progressive, diffuse hepatic vein thrombi develop²⁷. Thrombi also occur in other unusual places including the abdomen and cerebrum. Magnetic resonance imaging (MRI) can be used to diagnose and differentiate acute haemolytic attacks from abdominal venous thrombi which are very serious and often life threatening¹⁰.

Other clinical features include haemoglobinuria although it is only seen in a minority of cases; renal function abnormalities where the kidneys are enlarged with cortical infarcts, cortical thinning and papillary necrosis; and neurological problems due to small venous occlusions²⁷. In women, one third of pregnancies are successful but abortion and venous thromboembolism are common. Life-threatening complications are rare²⁷.

Patient history in examples of PNH show few commonalities

suggesting a cause for the malignant phenotype. Patients are not exposed to ionising radiation although some have developed the disease following chloramphenicol induced aplastic anaemia¹⁶.

Laboratory Diagnosis

The two most commonly used diagnostic tests are Ham's test and the sucrose lysis test. However, these are qualitative tests and cannot distinguish the phenotypes of PNH erythrocytes or determine their proportions. The complement lysis sensitivity (CLS) test developed in 1966 by Rosse and Dacie was able to do this but it is not used routinely for diagnosis. Other methods for PNH diagnosis and distinguishing phenotype have been sought including the use of flow cytometry (FC) and monoclonal antibodies (mAb). The measurement of DAF has been investigated using flow cytometry³ and by ELISA using anti-DAF antibodies¹¹. Schubert describes immunophenotyping of PNH cells using mAb against CD48, CD55 and CD59 on leukocytes. This may be a more superior method than conventional laboratory tests especially in differentiating PNH and aplastic anaemia. Today some laboratories are using FC and mAbs against CD59, CD16 and CD14 as well as conventional tests.

By114 is a specific mAb for the 90kD component of CD66 found on neutrophils²³. It is a valuable marker for detecting PNH before the Ham's test becomes positive. So far, it has proved to be a more sensitive detector of deficient PIG-anchored proteins than other mAbs. It can also distinguish between PNH and AA.

Schichishima *et al* compared the CLS test and two-colour FC using mAb to DAF and CD59. The two methods correlated well and indicated that two-colour FC is a method equal or superior to standard tests.

In patients undergoing bone marrow transplantation, the detection of PIG-anchored proteins using mAb against monocytic CD 14 and CD16 (found on granulocytes, NK cells and macrophages) is preferred over Ham's test as the results are often conflicting if the patient has had repeated blood transfusions¹².

Ronne reported uPA is elevated in PNH but further research is needed to determine the significance. A decrease in serum haptoglobin in PNH patients correlates with decreased haemoglobin levels. This may help in the initial diagnosis and with evaluation of the severity and prognosis of PNH³.

Association with other Haematological Malignancies

PNH has been associated with aplastic anaemia (AA), myeloproliferative syndromes (MPS) and acute leukaemia⁴. The link with AA is particularly close as they have similar findings of moderate to severe pancytopenia, decreased number of white cells and platelets without increased peripheral destruction⁶. By using immunophenotyping techniques, it has been shown that a large proportion of AA patients have cells deficient in PIG anchored proteins³⁴. In one German study, 22/40 patients with PNH had AA³⁵. It has been estimated that 58% of patients with PNH develop AA (1995)³⁶ compared to the figure of 15% stated by Bowlen in 1989. It has been reported that the evolution of PNH in AA patients treated with immunosuppressive therapy (antithymocyte globulin and cyclosporin) is as high as 10-13%⁶. There is epidemiological evidence of increased prevalence of PNH in areas where acquired AA is frequent eg. Thailand^{9,14}. Bowlen reported that half of PNH patients have marrow aplasia at some stage of the disease.

PNH and myelodysplastic syndrome (MDS) has also been investigated. Longo followed the case of a patient with PNH who developed MDS and found MDS arose from within the PNH clone. He regarded PNH as a pre-leukaemic lesion. However, in a case described by van Kamp²⁴, MDS did not arise from a PNH clone but

was a separate clone from injured marrow. This patient's abnormal red cells disappeared with the onset of MDS.

2-3% of PNH patients progress to develop AM¹⁶. According to Longo occurrence of leukaemia in a patient with previous PNH could be due to either the patient's marrow being generally prone to somatic mutation or to the PNH cell population having an increased risk of leukaemogenesis.

Treatment & Therapies

Treatment for PNH is supportive. Bowlen lists these as:

1. dextran to decrease haemolysis
2. androgens to combat anaemia
3. anticoagulants to avoid thrombosis
4. steroids to suppress haemolysis
5. blood transfusions
6. bone marrow transplantation (BMT)

Luzzatto also suggests iron for anaemia due to recurrent haemolysis, iron chelation in cases of iron overload due to repeated blood transfusions, and other drugs to limit complement activation and/or stimulating haemopoiesis and splenectomy (although surgery is not recommended due to thrombotic complications)⁷.

If blood transfusions are to be given, the blood should be filtered or the red cells washed to minimise white cell and platelet alloimmunisation and subsequent immune transfusion reactions.

BMT is a possible cure for PNH⁹. Myeloablative conditioning using BUS (busulphan) or FTBI (fractionated total body irradiation) is advantageous for BMT in PNH⁷. Perez-Oteyza *et al* described a case where a 20-year-old male was successfully treated for BM failure with an allogeneic BMT (from a sibling) using leukocyte depleted blood & blood products.

Autologous BMT may be possible if subpopulations of stem cells exist which express DAF and CD59. These normal cells (CD34+, CD38-, DAF+, CD59+) are capable of self-renewal and could possibly be used in autologous BMT following the eradication of CD34+, CD38-, DAF-, CD59- cells.

Experimental therapies include prolonging the life span of PNH erythrocytes by incorporating DAF into the cells¹¹. The mean red cell life span (MRLS) was 13.6 days before DAF- incorporation but extended to 30.9 days after DAF-incorporation. The researchers concluded that DAF could partially cure the shortened red cell life span but correlation of other membrane defects might be required for complete amelioration.

Okada²⁰ described treatment with purified 20kD HRF (HRF20) plus DAF may provide resistance to lysis by homologous complement. Since HRF20 has been cloned, the possibility of using recombinant HRF20 exists.

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Bayer essay prize

Bayer NZ Ltd. this year provided a cash prize for the best review of "Paroxysmal Nocturnal Haemoglobinuria" by students undertaking Haematology as an elective in the fourth year of the Massey University BMLS degree. This was the first year that this prize has been offered and was contested by a small number of presentations of a high standard. The essay took the form of a review of the recent literature and was restricted to approx 1500 words. The reviews were graded by Chris Kendrick from Massey University and Cindy Lincoln from Auckland Healthcare Ltd.

The winner of the competition was Andrea Lun who completed her placement in the Haematology laboratory of Canterbury Health Ltd in the first semester of 1996. Congratulations Andrea and best wishes for your career. My thanks go to Cindy, Joanne Paton and Bayer NZ Ltd for their help and support.

Potential for Clinically Misleading Susceptibility Results due to Extended Spectrum Beta-lactamases.

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Abstract

Aims. To report on *Enterobacteriaceae* producing extended-spectrum beta-lactamases (ESBLs) giving misleading in vitro susceptibility results.

Methods. Isolates of *Escherichia coli* and *Klebsiella* species recovered from blood cultures in the first six-months of 1994 from five major hospitals in the Auckland region were tested for the presence of ESBLs. To detect transferable resistance, ESBL-positive isolates were subjected to conjugation experiments. Prospectively, *E. coli* and *Klebsiella* spp. isolates from Auckland and Green Lane Hospitals were monitored for the presence of ESBLs. Clinical details of patients with isolates producing ESBLs were recorded.

Results. ESBLs were present in 3 of 152 *E. coli*, but none of 20 *Klebsiella* spp. blood culture isolates tested retrospectively. Transferable resistance was demonstrated in two of the three *E. coli* tested. Prospective testing detected four *E. coli* and three *Klebsiella oxytoca* isolates with ESBLs, giving a total of 10 patients with such isolates since January 1994. On routine breakpoint susceptibility testing for third generation cephalosporins, many isolates appeared susceptible to ceftriaxone (5), ceftazidime (6) and aztreonam (6). Three tested susceptible to both ceftriaxone and ceftazidime, and two tested susceptible to all three agents. In two patients, both of whom were neutropenic, death resulted from overwhelming sepsis caused by isolates producing ESBLs.

Conclusion.

Enterobacteriaceae producing ESBLs have emerged in Auckland. In two *E. coli* isolates resistance was transferable, making them the first such reported strains in New Zealand. Extensive use of broad-spectrum cephalosporins applies a selective pressure for the emergence of these mutants. ESBL-mediated resistance has important implications for prescribing habits, therapy and infection control practices. Isolates producing ESBLs may not be detected by standard susceptibility testing methods. Procedures specifically designed to detect their presence should be used in medical laboratories.

Introduction

Bacteria continue to develop resistance to antimicrobial agents. β -lactamases, enzymes that bind and destroy β -lactam antibiotics, are the commonest mechanism for resistance against this class of antimicrobial agents.¹⁻³

Plasmid mediated β -lactamases, e.g. TEM- I and SHV- 1, confer resistance to ampicillin and the first generation cephalosporins. TEM- I and SHV- I are widespread in *Enterobacteriaceae*.^{1,2} Third generation cephalosporins are not degraded by these enzymes but in some strains, the genes encoding TEM- I and SHV- I β -lactamases have undergone point mutations. These mutations have led to key amino-acid changes at the active sites of these enzymes resulting in an increased hydrolytic spectrum which includes both third generation cephalosporins and monobactams, e.g. aztreonam.² TEM and SHV derived extended spectrum β -lactamases (ESBLs) are usually transferable plasmid mediated enzymes which are inhibited by clavulanic acid. ESBLs have little effect on cephamycins, e.g. cefoxitin or carbapenems, e.g. imipenem.³

Since their first recognition in 1982 ESBLs have now been identified in all continents.¹ This paper presents the results of retrospective and prospective studies to detect ESBL-producing members of the *Enterobacteriaceae*, particularly *Escherichia coli* and *Klebsiella* spp., from patients in Auckland Healthcare.

Methods

Retrospective study. In July 1994 laboratories from five Auckland Hospitals provided blood culture isolates of *E. coli* and *Klebsiella* spp. from the previous six month period. Participating hospitals were Green Lane-National Women's (GLH), Middlemore (MMH), North Shore (NSH), Auckland (AKH) and Auckland Children's 'Starship' (ACH) hospitals. Isolates were screened for ESBLs on agar plates containing 1 mg/L of ceftazidime (CAZ), ceftriaxone (CTO) or cefotaxime (CFT). Isolates which grew at this concentration for any of these antibiotics had minimum inhibitory concentrations (MICS) performed by the NCCLS broth microdilution method⁴ for ceftazidime (CAZ), ceftazidime + clavulanic acid (CAZ+CLAV), ceftriaxone (CTO), ceftriaxone + clavulanic acid (CTO+CLAV), cefotaxime (CFT) and cefotaxime + clavulanic acid (CFT+CLAV). Reduced susceptibility to at least one of the third generation cephalosporins along with demonstration of synergism with clavulanic acid was considered confirmation of ESBL-production.

Resistance transfer. Successful transfer of ESBL-mediated resistance from isolates producing ESBL (donors) to *E. coli* J62-1 or *E. coli* J53-1 (recipient strains) was taken to indicate plasmid-mediated resistance.⁵

Prospective study. Between September 1994 and July 1995 all blood culture isolates of *E. coli* and *Klebsiella* spp. from patients at AKH and ACH were tested for ESBL-production by the double disk synergy test⁶. Isolates from other specimens were tested only if they had reduced susceptibility to CTO, CAZ, amikacin or gentamicin, in routine break point testing.⁴

Between August 1995 and January 1996 *E. coli* and *Klebsiella* spp. isolated from all sites from patients in AKH and ACH were screened for ESBL by inoculation onto plates containing 1 mg/L of CAZ and CTO. Isolates growing at these screening concentrations were further tested using double disk synergy test to confirm ESBL-production⁶. Between March 1995 and January 1996 blood culture isolates of *E. coli* and *Klebsiella* spp. from patients in GLH were tested for ESBL by the double disk synergy test.^{6,7}

Results

Source, time frame and distribution of *E. coli* and *Klebsiella* spp. isolates tested during retrospective and prospective studies are summarised in Table 1.

Retrospective study. A total of 191 *E. coli* and 44 *Klebsiella* spp. isolates were submitted. A number had died, leaving 152 *E. coli* and 20 *Klebsiella* spp. available for testing. The number of *E. coli* and *Klebsiella* spp. isolates respectively from the five hospitals were: ACH, 8 and 0; AKH, 80 and 9; GLH, 8 and 2; MMH, 45 and 6; and NSH, 11 and 3. Screening detected three *E. coli* isolates with ESBLs. No *Klebsiella* isolate possessed ESBL. MICs of ceftazidime, ceftriaxone

and cefotaxime with and without 2mg/L of clavulanic acid to the three *E. coli* isolates are shown in Table 2. The addition of 2mg/L of clavulanic acid resulted in ≥ 64 fold reduction in the MIC of at least one of the antibiotics tested, demonstrating clavulanic acid-susceptible ESBL production.

Table 1. Isolates tested for ESBL production

Hospital	Time period	Source	<i>E. coli</i>	<i>Klebsiella</i> spp	Total
Five hospitals*	Jan 94-July 94	blood	52	20	172
APH and ACH	Sept 94-July 95	blood	154	45	199
APH and ACH	Aug 95-Jan 96	all sites	931	139	1070
GLH	March 95-Jan96	blood	11	3	14
Total			1248	207	1455

* AKH, ACH, GLH, MMH, NSH

Table 2. MIC (mg/L) of three *E. coli* isolates with ESBL

Isolate Number	CAZ	CAZ + CLAV	CTO	CTO + CLAV	CFT	CFT + CLAV
27	8	≤ 0.25	16	≤ 0.25	16	0.5
233	>128	1	4	≤ 0.25	1	≤ 0.25
240	32	≤ 0.25	32	≤ 0.25	32	≤ 0.25

CAZ = Ceftazidime, CTO=Ceftriaxone, CFT=Cefotaxime, CLAV=2mg/L Clavulanic acid

Resistance transfer. Plasmid-mediated transferable resistance was demonstrated in two of the three ESBL producing *E. coli* isolates tested. Repeated attempts to transfer resistance from the third *E. coli* isolate were unsuccessful.

Prospective study. At GLH between March 1995 and January 1996 a total of 14 blood culture isolates of *E. coli* and *Klebsiella* spp. were tested for ESBL production. None of these possessed ESBLs. At AKH and ACH prospective testing detected 4 *E. coli* and 3 *Klebsiella oxytoca* isolates producing ESBL-mediated resistance. In the two periods where different testing methods and criteria were used, September 1994 to July 1995 and August 1995 to January 1996, one and six ESBLs were identified respectively. Thus, from January 1994 to January 1996, 1455 isolates of *E. coli* and *Klebsiella* spp. in the Auckland region were tested and 10 (0.7%) ESBL producers were identified. Two patients had infection or colonisation with these organisms on two separate occasions giving twelve episodes in ten patients.

Routine susceptibility results. Using the NCCLS interpretative criteria⁴ all ten isolates were susceptible to aminoglycosides (gentamicin and amikacin), quinolones and imipenem; nine isolates were susceptible to cefoxitin; all were resistant to amoxicillin; and four isolates were resistant to trimethoprim and trimethoprim-sulphamethoxazole. Of the third generation cephalosporins and

monobactam tested: five appeared susceptible to ceftriaxone; six appeared susceptible to ceftazidime, six appeared susceptible to aztreonam; three appeared susceptible to ceftazidime; and two appeared susceptible to ceftazidime and aztreonam.

Patient Details. Clinical details and outcomes of the ten patients with these isolates are summarised in Table 3. Sources were blood, urine, tracheal aspirate and a pancreatic drain. In two patients with underlying haematological disorder, ESBL-producing *E. coli* isolates caused fatal neutropenic sepsis. The duration of hospitalisation before the isolation of ESBLs ranged between 2 to 8 weeks for nine of the ten patients. In one patient, however, the isolate was recovered within 72 hours of hospitalisation.

Discussion

Emergence, spread and increasing prevalence of ESBL-producing *Enterobacteriaceae* has been recognised world-wide over the past decade¹. The ability of these enzymes to hydrolyse the third generation cephalosporins limits the therapeutic efficacy of these otherwise useful agents in treating infections caused by this group of bacteria.

Detection of three *E. coli* isolates producing ESBLs during our initial retrospective survey encouraged us to incorporate additional testing methods, specifically designed to detect ESBLs, into our routine protocol at APH, ACH and GLH. During the initial one and a half years of testing at APH and ACH, i.e., retrospective and initial period of prospective testing, four ESBL-producing strains were identified. However, a further six ESBL producers were identified in the following six months. Although this increase could partly be explained by our altered testing methods, there appears to be an increasing prevalence of these isolates.

In New Zealand different ESBLs have been reported in the South Island. Smith et al. reported seven ESBL-producing *Klebsiella oxytoca* isolates from Dunedin and Christchurch.⁸ For these isolates, however, resistance transfer experiments were unsuccessful and the resistance genes were thought to be chromosomal.⁸ Among our isolates, plasmid-mediated resistance was demonstrated in two of three *E. coli* strains tested, making them the first such strains reported in New Zealand. In our third isolate the resistance genes were probably chromosomal. The remaining seven isolates were not tested for transferable resistance.

On routine breakpoint susceptibility testing, using NCCLS interpretative criteria,⁴ five isolates would have been reported as susceptible to ceftriaxone; six to ceftazidime; six to aztreonam; three to ceftazidime and ceftazidime; and two to all three of these drugs. These false susceptibility results are due to low-level expression of the mutant enzymes.⁹ This low level resistance is, however, clinically important because it results in therapeutic failures.¹⁰ It is recommended that ESBL producers be reported resistant to all third generation cephalosporins irrespective of their apparent susceptibility results.³

The need to detect ESBL production has led to the introduction of various novel supplemental methods of testing. Double-disk synergy testing, Vitek automated panels, and special E-test strips all rely on demonstrating synergy between third generation cephalosporins and clavulanic acid to detect the presence of ESBLs.³ We have found initial screening to detect this low-level resistance using 1mg/L CTO and CAZ followed by double-disk synergy testing of isolates a simple, effective approach.

Genes encoding resistance to other antimicrobial agents, especially aminoglycosides and trimethoprim-sulphamethoxazole, are often present on plasmids encoding ESBL production. Therefore finding aminoglycoside resistance in *E. coli* or *Klebsiella* spp. has been

Table 3. Details of patients with isolates producing ESBLs

No.	Age, Sex	Underlying Condition(s)	Therapy 4 weeks of ESBL isolation	Organism	Source	Clinical relevance	Therapy	Outcome
1	41 yrs M	AML*, allogenic bone marrow transplant	Ceftazidime	<i>E. coli</i>	Blood	Gram-negative sepsis. Neutropenic enterocolitis	Ceftazidime then imipenem	Death due to sepsis
2	6 mths M	SCID†, haploidentical bone marrow transplant	Cefuroxime	<i>E. coli</i>	Blood	Central line sepsis	Amoxycillin, ceftazidime	Recovered
			Ceftazidime, amoxycillin	<i>E. coli</i>	Urine, x 4	Coloniser (bladder stab – negative)	Cefactor for chest infection	Well
3	63 yrs M	Burkitt's lymphoma	Nil	<i>E. coli</i>	Blood	Neutropenic sepsis	Gentamicin, amoxycillin-clavulanic acid	Recovered
			Gentamicin, amoxycillin-clavulanic acid, ceftriaxone, metronidazole	<i>E. coli</i>	Blood	Septic shock	Piperacillin, flucloxacillin then ceftriaxone, metronidazole	Death due to sepsis
4	44 yrs M	Hepatic fibrosis. Primary myeloproliferative disorder	Ceftazidime, imipenem, cefuroxime	<i>E. coli</i>	Tracheal aspirate	Coloniser	Ceftazidime	Death, unrelated to infection
5	2 mths F	Prematurity	Nil	<i>K. oxytoca</i>	Urine	UTI‡	Gentamicin, ceftazidime	Recovered
6	2 mths M	Repaired congenital lumbar myelomeningocele	Amoxycillin-clavulanic acid	<i>K. oxytoca</i>	Wound	Coloniser	None	Well
7	80 yrs F	Dementia, malnourishment, recurrent UTI	Cefuroxime, amoxycillin-clavulanic acid	<i>K. oxytoca</i>	Urine	UTI	Trimethoprim	Recovered
8	86 yrs F	AF§, on warfarin. Rectal bleeding	Nil	<i>E. coli</i>	Blood	Transient bacteremia	Amoxycillin-clavulanic acid	Recovered
9	74 yrs F	CVA¶. Carcinoma colon recurrence	Amoxycillin-clavulanic acid	<i>E. coli</i>	Urine	UTI	Norfloxacin	Recovered
10	70 yrs F	Acute necrotising pancreatitis	Imipenem, amoxycillin, ceftazidime	<i>E. coli</i>	Pancreatic drain	Abdominal sepsis	Cefuroxime, then amoxycillin, gentamicin, metronidazole	Recovered

*Acute myeloid leukaemia, † Severe combined immunodeficiency, ‡ Urinary tract infection, §Atrial fibrillation, ¶ cerebrovascular accident

used as a marker to alert the laboratory to employ additional testing. All our isolates, however, were susceptible to gentamicin and amikacin. Traditionally ESBL enzymes do not confer resistance to cephamycins, e.g. cefoxitin, but one of our isolates was resistant to cefoxitin. Resistance to both third-generation cephalosporins and cefoxitin, mediated by other β -lactamases, e.g. CMY-1, MIR-1, or porin deficiency has been reported.¹¹⁻¹³

ESBL production limits the choices for treatment of infections caused by these strains as does the frequent co-existence of genes encoding resistance to other antimicrobials on the same plasmid. Although β -lactam-inhibitor combinations and cephamycins are stable to these enzymes in vitro, their efficacy in vivo is controversial.^{3,14} On the other hand carbapenems, e.g. imipenem, are stable to these enzymes in vitro and are clinically effective against these organisms.

All ten patients with ESBL isolates were from AKH and ACH. One patient had just returned from Australia after a bone marrow transplant, suggesting acquisition overseas. Among the 12 episodes where ESBL-producers were isolated there were eight episodes of clinical infection: four bacteremias, three urinary tract infections, and one episode of intra-abdominal sepsis. In three of the episodes the isolates were thought to be colonisers, and in one the isolate caused a transient but true bacteremia. In two patients, both with underlying haematological disorders, these organisms caused fatal septicaemia. One patient with *E. coli* bacteremia recovered with ceftazidime therapy, but change of central venous line contributed to this favourable outcome. The three patients with urinary tract infections treated with third generation cephalosporins and aminoglycosides or other agents were cured.

Previous therapy with third generation cephalosporins, applies selective pressure for the emergence of strains expressing mutant enzymes.¹⁵ Due to co-existence of other resistance genes on the same plasmid as ESBL genes, use of antibiotics other than β -lactams may also play a role in selection of these organisms. Of our ten patients, two had received third generation cephalosporin therapy, three had not received any antimicrobial therapy, and five had received a combination of β -lactams and other agents. Nine of the ten had been in hospital between 2 to 8 weeks before recovery of their ESBL isolate. Similar findings have been reported before⁶ and extended periods of hospitalisation have been associated with outbreaks.¹⁵ Long hospitalisation is often associated with exposure to multiple antibiotics and also carries with it the increased risk of nosocomial acquisition of resistant strains following breakdown of infection control measures.

In overseas hospitals a number of nosocomial outbreaks have been reported due to ESBL producing strains.⁷ High level usage of extended-spectrum cephalosporins was often associated with these outbreaks. These were thought to result from the spread of a single resistant strain or spread of a plasmid among different species. Naumovski et al. reported an outbreak due to ceftazidime resistant *K pneumoniae* and *E. coli* among paediatric oncology patients who received empirical ceftazidime monotherapy for febrile neutropenia.¹⁴ Molecular studies revealed that all the isolates possessed the same plasmid encoding TEM-26 enzyme.¹⁶ Because they thought ceftazidime therapy exerted selective pressure on emergence of these mutant enzymes they changed their protocol to the combination therapy of amikacin, azlocillin and nafcillin. No further cases were identified over the following 18 month period. Venezia et al. recently reported an outbreak in a neonatal intensive care unit due to ESBL-producing *Klebsiella oxytoca*.¹⁷ Using DNA typing methods they found the majority of infants were colonised by the same strain. Cross-infection following sporadic breakdowns in infection control practices was thought to play an important role in this outbreak.¹⁷ At present our testing to detect the presence of ESBL is confined to *E. coli* and *Klebsiella* spp., Although ESBL-producing strains have predominantly been found in *Klebsiella* spp., especially *Klebsiella pneumoniae*, they have also been found in *E. coli*, *Serratia marcescens*, *Enterobacter* spp., *Citrobacter* spp., *Salmonella* spp., and *Morganella morganii*. It has been suggested that it is a reasonable practice to confine monitoring for the presence of ESBLs to *E. coli* and *Klebsiella* spp. isolates when the prevalence of ESBL isolates is low. At present, the prevalence of ESBL producers among our isolates is low, < 1%. We will, however, have to extend our testing to encompass all *Enterobacteriaceae* if this prevalence increases.

Conclusion

The arrival of ESBL producers with their potential to cause outbreaks of nosocomial infection serve to remind us of the importance of infection control measures to prevent cross-infection within hospitals. Judicious use of broad spectrum cephalosporins should help curb the selection and spread of these mutants but all antimicrobial agents should be used selectively and for the shortest possible duration. Finally Microbiology laboratories need to employ methods designed specifically identify these isolates. Failure to do so will lead to the issuing of clinically misleading susceptibility results.

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Leading Article

Laboratory Work, Pipetting and Musculoskeletal Ailments

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It is well known among experts and laymen that heavy physical loading can cause musculoskeletal disorders. Outside the world of occupational health professionals it is perhaps less well known that also light physical loading covariates with ailments and problems mainly in the upper part of the body.

Laboratory technicians who are working with pipetting in laboratories are often highly specialized. This may lead to repetitive and monotonous work in the sense of repeating the work tasks over and over again, in research settings or in routine work. Tasks with so called light physical loading in industrial settings are known to have an impact on the prevalence of musculoskeletal disorders. Several factors must be taken into consideration to estimate physical work load; type and design of equipment, level of musculoskeletal loading and organization of the work. These factors will be discussed in the article.

To our knowledge, only a few studies have been performed to investigate the work situation of the laboratory technicians and the prevalence of ailments in shoulders and hands, but the findings are concordant.

During recent decades there has been a change in the design of laboratory pipettes, with glass pipettes being replaced in favour of plunger-operated pipettes. They are maneuvered by a tangent for one or more fingers or the thumb. The handling of these pipettes demands a high degree of precision and a considerable amount of repetitive movements of the fingers and static work for the muscles of the hand, arm and shoulder, which is known to be harmful for the muscles, and show covariation with musculoskeletal ailments of different kind.

Fredriksson¹ provides a detailed description of the physical strain upon the shoulder, arm, hand and fingers when pipetting, in a study performed among the laboratory staff at a research department in a pharmaceutical company. She calculated the average percentage of maximal voluntary capacity (MVC) of the thumb muscles, used when depressing the tangent in pipetting for the female subjects. The result was compared to acceptable average stress levels for muscles and Fredriksson¹ stated that the level of strain can be unsuitable high for women. She also confirmed that the neck and shoulder muscles have to work statically when the head and neck are bent forward more than 30° and the arm is elevated without support for lengthy periods.

Björkstén et al² performed a study to evaluate the prevalence of hand and shoulder ailments among female laboratory technicians. A "nested case control" study in the cohort of subjects was performed to study the prevalence in relation to the "dose" of pipetting and in relation to some psychosocial factors. E.g. those with a lower dose were compared with a higher dose. Forty-four percent of the technicians reported hand problems, 58% shoulder problems

and 44% neck problems. A dose of more than 300 hours per year (high exposed) was found to be associated with a five-fold increased risk of hand ailments. This annually high exposed group also had a two fold risk of getting shoulder ailments compared to the annually low exposed group (less than 300 hours per year). Based on the assumption of 44 work weeks per year, 300 hours per year corresponds to 1-2 hours of daily pipetting. There was no difference in prevalence of hand (fig.) or shoulder ailments among those low exposed and a control group of female state employees in general.

Shoulder ailments also covariated with psychosocial factors at work. Repetitive, monotonous work, in addition to the direct physical strain, is often experienced as boring and mentally inactivating, which workers interpret as poor "work content." Those of the laboratory technicians who characterized their work content to be less satisfactory, had an eight-fold risk of reporting shoulder ailments compared to those more satisfied with their work content.

In a clinical examination performed by Sassarinis Jansson³, among those reporting hand ailments in the inquiry study, many of the laboratory technicians complained about a feeling of weakness in their hands. A strength test confirmed that they on a group level had a lower hand strength in the dominant hand than a control group.

Those who were available 4 years later were followed up by Sassarinis Jansson^{4,5} and re-examined. The conclusion from that follow up study was that many of those with earlier hand problems and still pipetting had changed their work organization to a pipetting time closer to 300 hours a year. Many also had changed pipettes to models better fitting their hands. The hand strength was tested and showed lower values for the right (dominant) hand compared to the left hand, which indicated a trend with weakening of the hands.

The conclusion from these studies is that laboratory technicians to a rather great extent experience ailments mainly in the hand and thumb but also in the shoulders.

Suggestions to diminish the load and improve the work situation of the laboratory technicians include both the design of the pipettes and change of the work organization.

Fredriksson¹ suggested: – mechanizing and purchase of diluting machines should be considered, if series become large, – the producers should be influenced towards the development of pipettes for different hand sizes and with as little resistance of the buttons as possible – pipetting work should be organized according to biotechnological/ergonomical needs. She also advised short breaks. The relevance for use of short breaks, to diminish the physical loading of the muscles, without limiting total time of exposure is under discussion and is not proved. Referring to Björkstén et al² we can advise that the daily exposure time of pipetting should not exceed 2 hours altogether, to meet ergonomical needs.

% reporting ailments

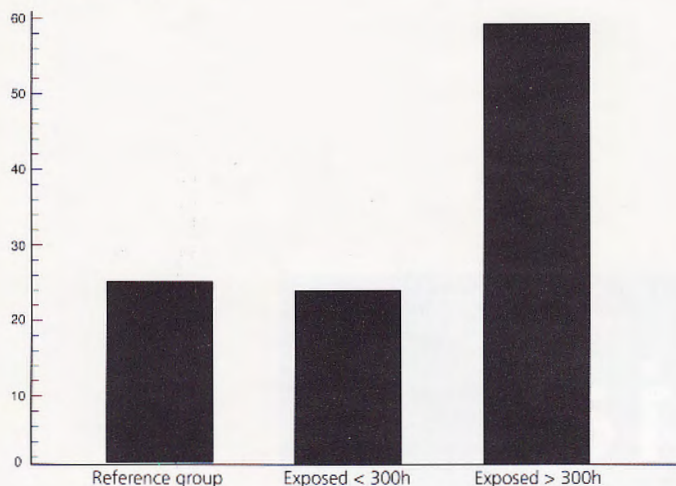


Figure – Percentage reporting hand ailments among female state employees (N = 25 378), female laboratory technicians pipetting less than 300 hours/year (N = 56) and more than 300 hours/year (N = 58)

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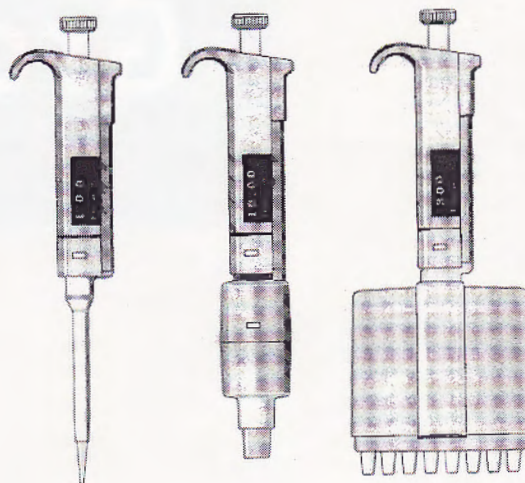


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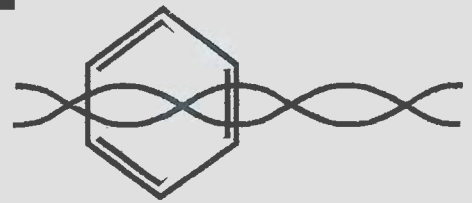
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NZIMLS Continuing Education

Special Interest Groups



Convenor: Alison Buchanan
Clinical Biochemistry
Main Building
Auckland Hospital
Ph: (09) 307 4949
Ext: 7553
Fax: (09) 307 4939



Meeting of the Biochemistry Special Interest Group, Tuesday 27th August 1996 at the Ellerslie Convention Centre

Measurement of Myocardial Proteins in Ischaemic Heart Disease.

Dr. John French, Cardiology Dept. Green Lane Hospital

The measurement of myocardial proteins from plasma samples to confirm the diagnosis of myocardial infarction has been widespread for several decades. The enzyme activities of proteins such as lactate dehydrogenase, aspartate transaminase and creatine kinase has been measured. Results of these assays aided diagnosis, contributed to the assessment of infarct size, but did not generally influence acute management.

Prior to the introduction of coronary care units (CCU) in the 1960s the hospital mortality from myocardial infarction was approximately 30%. The CCUs in the 1970s primarily aimed to treat arrhythmias which resulted in an approximate halving of hospital mortality from myocardial infarction. The landmark work of De Wood et al in 1980, which showed that most patients within hours of myocardial infarction had an occlusive intra-coronary thrombus, provided the conceptual basis for thrombolytic therapy. Thrombolysis has subsequently been refined and recently it has been shown that primary angioplasty is an equally efficacious method of coronary reperfusion. The rapid achievement of complete reperfusion, known as TIMI-3 flow, is related to prognosis. However, the use of coronary arteriography to detect reperfusion is limited by the lack of widespread access.

If reperfusion could be detected by measuring non-invasively parameters such as myocardial protein release, this may suggest the need for further reperfusion therapy, either re-administration of thrombolysis or rescue angioplasty to be undertaken. The myocardial protein markers commonly used to measure reperfusion have been troponin

T, myoglobin and CKMB mass. In addition measurement of troponin I and cytosolic fatty acid binding protein has been suggested though these proteins have not been measured in large studies.

In order that results are available promptly for the clinician there is a need for rapid assays such as the spot bedside test, currently available for troponin T. The challenge will be to identify perhaps even smaller cytosolic myocardial protein markers of less than 15 Kd that are rapidly released with minor myocardial necrosis. Additionally troponin T levels on admission have been shown to be of prognostic importance, initially in unstable angina and more recently in myocardial infarction. Elevated Troponin T levels at presentation in these acute ischaemic syndromes have been associated with a poorer prognosis.

In summary myocardial proteins were originally used by clinicians as makers of myocardial infarction. More recently, these proteins have evolved to become tools for measuring reperfusion non-invasively and also predicting prognosis.

CKMB

Sarah Simmonds, Biochemistry Dept. Middlemore Hospital

What is CKMB?

Creatine Kinase exists as a dimeric molecule containing two subunits M and B. These combine to form 3 different Isoenzymes CKMM, CKBB and CKMB.

CKMM is found primarily in Skeletal muscle tissue

CKBB is found primarily in Brain tissue.

CKMB is found primarily in Cardiac tissue.

The quantitation of CKMB in serum is the best known marker for diagnosing Myocardial infarction.

CKMB Measurement

CKMB is measured by several techniques. they fall into two main categories, one measuring activity and the other measuring mass.

Methods such as immunoprecipitation and immunoinhibition measure activity.

Immunoassay methods measure mass.

Assays based on monoclonal antibodies that measure CKMB mass are rapid, highly sensitive and specific, and are now the analytical method of choice.

CKMB evaluation on the AxSYM at Middlemore

Interbatch precision

Sample	Number	%CV
Patient 1	6	6.7
Patient 2	6	4.9
Abbott Low	10	9.3
Abbott Medium	10	7.8
Abbott High	10	5.6

Linearity

Linear to 300 ug/L

Correlation with the Stratus

Y (AxSYM) = 1.27 (Stratus)

Lower limit of detection

Minimum detection limit = 0.16ug/L

Use of the Algorithm

An Algorithm is used to interpret CKMB results. This is so the CKMB is related to CK.

To use this calculate the Relative index

Relative index = $\frac{CKMB}{CK} \times 100$

If CKMB is < 4ug/L

- Do not calculate relative index
- Report interpretive comment "Results do not suggest Myocardial damage."

If CKMB is > 4ug/L and CK < 400U/L

- Do not calculate relative index
- Report interpretive comment "Results suggest Myocardial damage."

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Troponin T

Don Mikkelsen, Health Waikato Laboratory

Acute Myocardial Infarction (AMI) is a common medical emergency and usually of overt presentation requiring little or no diagnostic input from the Laboratory. In the cases of atypical presentation where a diagnostic test is required, the Laboratory provides a less than optimal service because the markers that are detectable in blood result from cell death which is the event that treatment is designed to avoid. Cell death occurs outside of the window of opportunity for the most effective treatment, which is thrombolytic therapy aimed at re-vascularisation. Markers of AMI are now available, which are cardiac specific (nearly) and are uncomplicated to interpret in the presence of skeletal muscle injury. Cardiac Troponin T is one of these.

TnT is present in the tropomyosin complex of muscle and binds the troponin complex to the myosin strand. The cardiac form of Tnt (cTnT) has a distinct amino acid sequence and is different from that found in skeletal muscle. It is present in very high concentrations in the cytosol, approximately three times that of cTnI.

Interest in cTnT has now focused on its apparent ability to risk stratify patients with angina into groups that do, and do not, require treatment. This is based on the minor myocardial damage hypothesis which attributes the small rise in cTnT seen in at risk patients due to small amounts of cell damage from vascular insufficiency. cTnT is considered effective because of its high cytosolic concentration.

There are problems with the cTnT assay due to the specificity of the assay and reversion to embryonic protein forms in some diseases.

Assays for cTnT are currently only available from Boehringer Mannheim and are offered quantitatively on the ES systems and the new ElecSys as well as a Trop T rapid test dip stick technology.

Troponin I

Karen Glover, Clinical Chemistry Dept., Auckland Hospital

Introduction

Troponin I is the contractile regulatory protein complex of striated muscle.

It exists as three different isoforms, the cardiac isoform being found only in the myocardium.

It is therefore a more specific marker of myocardial injury than CKMB and the levels remain elevated for 7 to 10 days.

Troponin T has a similar early release, remains elevated longer than Troponin I, is specific for cardiac damage but increased levels have been seen in renal disease.

Assay

The assay described is the Stratus automated two-site immunoassay produced by Dade-diagnostics

The second antibody is labelled with alkaline phosphatase and the reaction rate measured is proportional to the concentration of Troponin I.

The method takes approximately 15 minutes, is very user friendly and, on the limited number of assays performed, appears to be very precise.

Clinical Trial

We are performing the assay as part of a study separating unstable angina patients into treatable high, medium and low risk groups. This study does not now start until September so the results I had hoped to show you have yet to be produced.

Maternal Serum Screening for Neural Tube Defects and Chromosome Abnormalities –

Why it isn't just another lab test

Dianne Webster; Mary Stuart, Dennis Dixon-McIver; Bruce Knox, Jennie Giles, Dale Gilbert and Alistair Roberts, Auckland Healthcare Services Ltd.

Screening for neural tube defects (NTDs) and chromosome abnormalities as it is done in Auckland involves analysis of maternal serum at 15-20 weeks gestation. Tests performed are α -fetoprotein (AFP) to indicate NTD risk, and free α and β -subunits of hCG, unconjugated oestriol (uE_3) and AFP which, together with maternal age, are used to calculate a risk of chromosome abnormality. Women found to be at increased risk are offered appropriate diagnostic tests (level-3 ultrasound or amniocentesis and karyotyping).

This isn't just like any other lab test because:

- Results are estimates of risk, not values of analytes.
- Well women come into the program which creates, and must care for, worried women.
- Well women are screened so there are no clinical details to help interpret results, most of which are normal.
- Doing lab tests and getting mostly normal results is boring and it is difficult to maintain quality in this situation.
- Pregnancy outcomes of screened women must be monitored to allow alteration of screening parameters if recall rates are too high or too many cases are missed.
- The implications of fetal abnormality and possible termination of pregnancy must be dealt with when offering the test.
- Gestational age information is critical in interpretation of test results.

The authors of this abstract are clinical chemists; genetic counsellors; managers and clinicians.

Urinary Catecholamine Measurements and the Diagnosis of Pheochromocytoma

Alan McNeil, Departments of Clinical Chemistry, Auckland Hospital and Molecular Medicine, University of Auckland.

Introduction

Pheochromocytoma is an uncommon tumour of the adrenal medulla that secretes catecholamines and can cause dangerous rises in blood pressure. Urinary catecholamine measurements are widely used for the diagnosis of these tumours though the interpretation of the results is often difficult.

Aim

To review our experience with urinary catecholamine measurements in the diagnosis of pheochromocytoma over the last 5 years.

Methods

The outcome of patients with elevated urinary catecholamines over a 14 month period was investigated by case-note review. The range of urinary catecholamine values was also determined for all patients diagnosed with pheochromocytoma between 1990 and 1996.

Results

The majority of the patients with pheochromocytoma had elevated urinary noradrenaline excretion either alone (4/18) or

in combination with adrenaline and/or dopamine (12/18). One patient had normal urine catecholamines and one had a pure adrenaline secreting tumour. Over a 14 month period 1% of all urine catecholamine results were elevated at least twice the upper limit of normal. Of the patients with these results 11% (5/46) had phaeochromocytoma, 48% (22/46) were normal with repeat testing and 7% (3/46) had unresolved abnormalities. Using a cutoff value of twice the upper limit of normal, the sensitivity of urine catecholamine results in the diagnosis of phaeochromocytoma was 83%. Of concern, 35% (16/46) of the patients with high urine catecholamine results had not been investigated any further.

Conclusions

In this series urine catecholamine values above twice the upper limit of normal, particularly noradrenaline, were seen in most, but not all, patients with phaeochromocytoma. Repeat urine testing was probably the most useful initial investigation for patients with one high result as the second result was often normal. The follow-up of some patients with abnormal urine catecholamine results is probably inadequate.

Sweat Testing – A Clinical Perspective

Alison Wesley, Director of Cystic Fibrosis clinic, Starship Children's Hospital.

The diagnosis of cystic fibrosis is one with serious implications for both the child and its family. The diagnosis should be accurate and should be achieved as soon as possible whenever the diagnosis is suspected. Despite the availability of molecular diagnosis the sweat test remains a major diagnostic tool for the confirmation of cystic fibrosis.

Because of the identification of greater than 600 mutations in the CF gene it remains impractical in the clinical laboratory to use the detection of these in the diagnostic process of a significant number of patients.

The categorisation of these mutations into a number of classes has shown a correlation of between the sweat chloride level and some classes of the CFTR gene mutations. Those CFTR gene mutations that produce a cAMP – responsive channel with reduced conductance are associated with significantly lower, intermediate sweat chloride values. These patients frequently also have retained pancreatic function. There has been at least one mutation present in either the homozygote or heterozygote state i.e. 3849+10 kb C to T where sweat chloride values are normal.

A recent report from Australia describes the delayed diagnosis of children with an uncommon genotype (dF508/R117H). These children were identified initially as a result of a newborn screening program but were not diagnosed as having cystic fibrosis because their sweat chloride values ranged from 40 to 58mmol/L.

Two recent reports have advocated reducing the lower limit of a borderline sweat chloride to 30-40mmol/L particularly in infants less than 6 weeks of age at the time of the sweat testing.

Sweat electrolyte analysis remains a very important part of the diagnostic process of cystic fibrosis. Measurement of the chloride level is preferable because there is less overlap between populations with and without cystic fibrosis.

Sweat Testing – A Clinical Perspective

Colleen Bassett, Snr Technologist, Special Techniques, Clinical Biochemistry, Auckland Hospital.

Most journal articles will tell you –

“The sweat test is not a difficult test to perform but is fraught with the possibility for error.”

“It is reported to have unacceptably high false positive and negative rates attributable to inaccurate methodology, technical error, patient physiology.”

“Comprehensive guidelines addressing the collection of sweat and the quantitative analysis of electrolytes need to be set.”

“It should only be performed by fully trained, experienced personnel.”

“Both the sodium and the chloride should be measured and when the results fall within the equivocal/borderline range particular attention should be paid to the sodium/chloride ratio and the summation of the 2 ions.”

“The diagnosis of Cystic fibrosis should never rest on the sweat test alone. It should be considered with the clinical findings and the laboratory evidence of pancreatic dysfunction.”

I will show the findings of a retrospective study of our results from November 1992 to May 1996 looking at:

- The sodium vs chloride argument
- The sodium; chloride ratio and summation of the 2 ions.
- The effect of age.

Pancreatic Elastase 1

P Rowe, Chemical Pathology, Middlemore Hospital, Private Bag Otahuhu, Auckland

Introduction

Pancreatic elastase 1 is a digestive protease synthesized and secreted by the pancreas. It

is an absolutely pancreatic specific protein which shows an extraordinary high intestinal stability. Thus the faecal concentration clearly reflects the exocrine capacity of the pancreas. The test is used for the diagnosis and staging of chronic pancreatitis and may be used for monitoring cystic fibrosis patients.

Faecal Pancreatic Elastase 1 Assay

The assay uses two monoclonal antibodies to human pancreatic elastase 1 and is based on the ELISA sandwich technique using a 100 mg stool sample which has been extracted and diluted.

The control and patient samples are compared to a six point standard curve run with each batch.

Results	Reported in µg/g of wet faeces
Greater than 200	Normal
100-200	Mild to moderate exocrine pancreatic insufficiency
Less than 100	Severe exocrine pancreatic insufficiency
Imprecision	+/- 20%

Evaluation done at Middlemore Hospital

Imprecision:	Interbatch CV	9.2%
	Interbatch	11.6%
	Control CV	4.9% at 200µg over a 12 month period (the control is not extracted or diluted)
Patients results over 12 months:	66.5%	>200
	11%	100-200
	22.6%	< 100 (with 81% of these <50)

Clinical Evaluations

Clinical trials have compared the measurement of Elastase 1 in stool samples with the Secretin – pancreozymin test (the gold standard), the pancreolauryl test and faecal chymotrypsin.

The Elastase test has been described as simple, accurate, more sensitive than faecal chromotrypsin and more specific than the pancreolauryl test. It shows excellent correlation with the invasive Secretin – pancreozymin test where the activities of amylase, lipase and trypsin are measured in basal and 30 minutes post IV secretin in duodenal juice.

Advantages of using the Faecal Pancreatic Elastase 1 assay

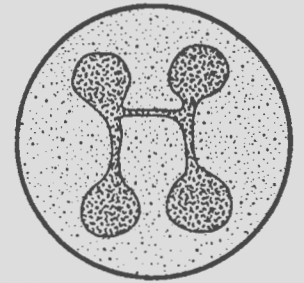
- absolute pancreatic specificity
- non invasive
- good correlation with the secretin – pancreozymin test
- not influenced by replacement therapy
- not affected by extrapancreatic disease
- approximately 90% sensitivity and specificity for chronic pancreatitis.



Haematology

Special Interest Group

Convenor: Ross Anderson
c/o Diagnostic Laboratory,
Symonds Street,
AUCKLAND



Conference

The high standard of papers presented and the selection of speakers at the HSIG seminar complimented the overall success of the NZIMLS 50th Anniversary Conference held in Auckland. The well attended session enabled technologists from all over New Zealand to revisit old friends, make new acquaintances, catch up on gossip and equally importantly reevaluate their Laboratory procedures in view of new information or technology made available to them at Conference.

A wide range of haematology specialities were addressed. Papers presented covered, heparin like anticoagulant in a patient with carcinoma, near patient testing of INR's, new method for the evaluation of HbA2 & HbF, presence of Barts in neonates, a panoramic view of Haemophilia, Acute Leukaemia and Lymphoproliferative Disorders. Something to suit every interest.

For those unable to attend Conference, and in recognition of the time and effort required to produce these excellent papers, we will be actively encouraging speakers to forward their

presentations for publication in the Journal. After all, there are financial rewards and it will contribute to MOLS! Have you caught your MOL yet? More on this in the next issue.

Between a Rock and a Hard Place

Today, in the laboratory, we have a wealth of information available to us from increasingly more sophisticated analysers and interconnecting computer systems.

In our desire to provide the clinician with the best possible service, the researching of data and follow up tests required to either aid, clarify or find a diagnosis for a patient have been undertaken as matter of course.

Today all medical/hospital departments operate under considerable financial and staffing constraints, while at the same time still trying to maintain professional standards. Requests for laboratory tests are now more carefully scrutinised and rationalised. The focus in the laboratory is increasing on rapid turn around

times and throughput of samples with less time and fewer experienced staff available for monitoring and evaluating of results. If an abnormal result requires further investigation, who bears the responsibility for its initiation and what are the potential effects on the patient.

For example, you have been monitoring a patient on heparin and as a matter of protocol you take off an FBC to check for HITS. Platelets are fine but the neutrophil count has been slowly dropping over the previous week (to $0.9 \times 10^9/L$.) No FBC had been requested. Fortunately the change was noticed, the patient didn't develop septicaemia and corrective measures were taken, more by good luck than good management. Would the Laboratory have been at fault had this not been detected?

Similarly, should follow up tests for haemolysis, haemoglobinopathies, HITS, DIC, Glandular Fever etc continue to be requested by laboratory staff? Who will bear the cost? (especially if the results show the Laboratory may have been a little over zealous).

Patient health versus financial health, that is the dilemma currently facing us all.

Book Review

Bone Marrow Pathology

By Barbara J Bain, David M Clark, and Irvin A Lampert. Blackwell Science, Oxford. Second Edition 1996. Pages 328 hardback

The book is written by a Haematologist and two Histopathologists with cytological and histological assessments being presented. They look to place bone marrow abnormalities within the broader context of clinical and peripheral blood findings. The aim being an integrated approach to diagnosis.

The chapters, ten in all, are comprehensive, well written and easy to read. They are supported by an extensive set of coloured photomicrographs of blood, marrow and trephine biopsies that cover morphology, histology, histochemistry and immuno-histochemistry. There is very little cytochemistry included in this book. One criticism is that in a number of photomicrographs the cells appear faint and pale. Some pictures lack the clear, defined crispness found in some of Bain's other publications. Each chapter is referenced with a modest bibliography.

The first chapter deals with the normal marrow in detail with tables of the cellular elements at all ages. It contains a useful section

on artefacts that may be found in the aspirates and trephine biopsies. Chapter two is on infection and reactive changes and encompasses the changes and infections found in AIDS. The lymphoproliferative disorders are covered in chapter five and given special attention. A comparison of the Kiel, European-American and Working Formulation classifications is usefully set out. Chapter ten, the last, is a good account of Diseases of Bone that may be seen when examining the trephine biopsies.

This book achieves its aim and will appeal to all groups involved with reporting bone marrow pathology. The set of photomicrographs it contains, alone, is a very worthy reference collection to have on one's shelf.

G.J. Green
Haematologist
Wellington Hospital

Interview with the Editor

The Council members of the NZIMLS will be profiled in the 1997 issues of the NZIMLS journal. As well as a short biography of their career, they will be asked a few questions about themselves. We begin with the PRESIDENT.

Name: Shirley Gainsford.
Present position: Charge Scientist Microbiology, Valley Diagnostic Laboratory, Lower Hutt.
Training: Wairau Hospital, Blenheim. Wellington Hospital.
Previous employment: National Heart Hospital, London. Wellington Hospital.

Elected to the Council in 1987 as Region 3 representative. Subsequently served as Secretary/Treasurer and Vice President. Has represented the NZIMLS as Chairperson of the Advisory Committee of the National Diploma Medical Laboratory Science at the Auckland Institute of Technology and reviews of the BMLS courses in New Zealand and Australia on behalf of the Medical Laboratory Technologists Board.

What are your main interests in Microbiology?

I am a jack of all trades in clinical microbiology. At Wellington Hospital my section was responsible for routine microbiology of about one third of the wards plus the STD clinic, mycology and identification of gram negative bacilli. At Valley Diagnostic laboratory I have become reasonably adept at parasitology and mycobacteriology and retain mycology as my special interest.

What do you consider to be the highlight of your career?

Isolating *Prototheca wickerhamii* from a gentleman's cerebrospinal fluid and performing combined sensitivity on it by checkerboard titration.

What has been the worst moment of your career?

The weekend 5.00 – 10.00 pm shift covering all departments at Wellington Hospital in the late 1970s. There was considerable distance between the departments, I never seemed to be able to perform a bicarbonate with the Van Slyke apparatus without having mercury all over the bench and one night I shut myself in the tearoom of the blood donor area when raiding the blood donors biscuit box.

What do you think is the main issue facing medical laboratory scientists?

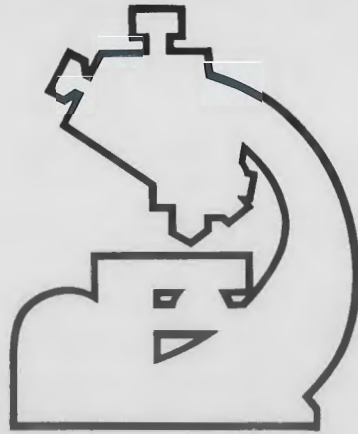
Facing the competitive health environment by becoming more efficient and at the same time trying to maintain the high standard of medical laboratory science in New Zealand.

During your term as president of the NZIMLS what do you hope to achieve?

I would like to improve continuing education opportunities for members and we hope to build up a library of CD Roms and have access to the Australian Institute of Medical Scientists programmes. I would like to convince more laboratory workers to support their professional society so that we can ensure we have a voice in the political health arena and in the education and training of medical laboratory scientists.



Shirley Gainsford and Hugh Bloore at the 1996 ASM.
Mr Bloore was Shirley's first boss at Wairau Hospital, Blenheim and is past-president of the NZIMLS.



NEW ZEALAND INSTITUTE OF

MEDICAL

LABORATORY

SCIENCE

EXAMINATION LIFTOUT

Specialist Certificate Regulations
Specialist Certificate Examination Application Form
Q.T.A. Regulations
Q.T.A. Examination Application Form
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The New Zealand Institute of Medical Laboratory Science offers to medical laboratory assistants the qualification known as the Certificate of Qualified Technical Assistant (QTA) and to medical laboratory technologists the qualification known as the Specialist Certificate.

The Examinations Committee is based in Christchurch and all correspondence should be addressed to:—

Executive Officer
N.Z.I.M.L.S.
P.O. Box 3270
Christchurch
Phone/Fax (03) 313-4761

**NEW ZEALAND INSTITUTE OF MEDICAL LABORATORY SCIENCE
SPECIALIST CERTIFICATE EXAMINATION**

EXAMINATION SUBJECTS

The examination is offered in:

Clinical Biochemistry
Haematology
Histology
Cytogenetics
Virology

Clinical Microbiology
Transfusion Science
Medical Cytology
Immunology

PREREQUISITES

1. Candidates for the examination must be registered as a Medical Laboratory Technologist by the New Zealand Medical Laboratory Technologists Board and have completed one years practical experience in the examination subject in a laboratory in New Zealand.
2. **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.

EXAMINATIONS

1. The examinations will be held annually in New Zealand during November.
2. Candidates must complete the application form and forward this, complete with examination fees, to the Executive Officer of the Institute 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 two written papers each of three hours duration.
5. To pass the examination candidates must obtain an overall mark of 50%.
6. The results of the examinations will be announced by the New Zealand Institute of Medical Laboratory Science. Successful candidates will be awarded the NZIMLS Specialist 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. Enquiries should be made to the Executive Officer of the NZIMLS.

NEW ZEALAND INSTITUTE OF MEDICAL LABORATORY SCIENCE
Application to sit Specialist Certification Examination
12th and 13th November 1997

SECTION A — TO BE COMPLETED BY THE CANDIDATE

Name: Mr
Mrs
Miss (Surname) (First Names)

Laboratory

Laboratory Address

.....

Examination Subject

I certify that I am registered in New Zealand as a medical laboratory technologist and have completed a years practical experience in New Zealand in the examination subject post registration

Signed

EXAMINATION FEE: \$400 (GST Inclusive)

The full examination fee must be paid with the application.

SECTION B — TO BE COMPLETED BY THE PRINCIPAL OR CHARGE TECHNOLOGIST

"I certify that the above candidate will meet the requirements of the
Specialist Certificate Examination"

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

Address

.....

.....

APPLICATIONS CLOSE FRIDAY 23 MAY, 1997

Please forward application forms accompanied by fees to: Executive Officer, NZIMLS, PO Box 3270, Christchurch.

NO LATE APPLICATIONS WILL BE ACCEPTED

Special Note to Applicants

If not already members of the NZIMLS applicants to sit this examination **must** submit a valid membership application along with this examination application.

**NEW ZEALAND INSTITUTE OF MEDICAL LABORATORY SCIENCE
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.

EXAMINATIONS

1. The examinations 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 Institute 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 New Zealand Institute of Medical Laboratory Science. 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.

NEW ZEALAND INSTITUTE OF MEDICAL LABORATORY SCIENCE
Application to sit the Examination of Qualified Technical Assistant
5th November 1997

SECTION 1 — TO BE COMPLETED BY THE CANDIDATE

Name: Mr
Mrs
Miss (Surname) (First Names)

Laboratory

Laboratory Address

Subject (Haematology, Microbiology, etc)

EXAMINATION FEE: \$80 (GST Inclusive)

The full examination fee must be paid with the application.

**SECTION B — TO BE COMPLETED BY THE PATHOLOGIST OR CHARGE
TECHNOLOGIST**

Date candidate commenced work in examination subject

“I certify that the above candidate meets the requirements of the Q.T.A. 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

Address

.....

.....

Office use only

APPLICATIONS CLOSE FRIDAY 23 MAY, 1997

Please forward application forms accompanied by fees to: Executive Officer, NZIMLS, PO Box 3270, Christchurch.

NO LATE APPLICATIONS WILL BE ACCEPTED

Special Note to Applicants

If not already members of the NZIMLS applicants to sit this examination **must** submit a valid membership application along with this examination application.

THE NEW ZEALAND INSTITUTE OF MEDICAL LABORATORY SCIENCE (INC.)

Application for Membership (For use with Examinations only).

(Please Print Clearly and Tick Appropriate Box)

I, _____
SURNAME _____
MR, MRS, MS, MISS _____
INITIAL(S) _____
FIRST NAME(S) _____
OF, _____
WORK ADDRESS _____

Hereby apply for membership of the New Zealand Institute of Medical Laboratory Science in the category of:

Member Associate

AND Certify That I Have:

Not Previously Been a Member Previously Been a Member (State Category: ____)
 Resigned (Date:) _____ Did Not Resign

I am employed as: _____

in the Speciality Department of: _____

Highest Professional Qualification: _____ Year Obtained: _____

Nominated By: _____

(Current Financial Member N.Z.I.M.L.S.)

Please forward payment with Application for Membership, to the Executive Officer, NZIMLS, P.O. Box 3270, Christchurch.

Current Membership Subscriptions are:

MEMBER \$101.40 (GST incl.) ASSOCIATE \$48.10 (GST incl.)

Member — any person who is registered by the Medical Laboratory Technologists Board

Associate — any person engaged in Medical Laboratory Science who is not eligible for any other class of membership.

The appropriate membership subscription must accompany this application for this to be a valid application.

New Zealand Institute of Medical Laboratory Science



NBC Conference 1997

Wellington

VENI
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VELLINGTON

Plan to participate in this August event

26th August to 29th August, 1997

- Full scientific programme
- General forum on work/family relationships
- Social event . . . "WHEN IN ROME"

INVITATION TO ATTEND

CALL FOR ABSTRACTS

TENTATIVE PROGRAMME



“Friends! Romans! Technologi! Lend Me Your Ears!!!”

Ever since Shakespeare recorded these famous words of Marc Anthony, some 1600 years after they were uttered to catch the crowd's attention at Julius Caesar's funeral, they have been used by orators of many generations for the same effect. (Spike Milligan has been seen on numerous occasions carrying a large sack full).

Ears are an integral part of Conference attending paraphernalia, along with eyes, a brain, and a reasonably functional liver. The organising committee invites you to bring all these along when you attend the 51st Annual Scientific Meeting of the NZIMLS being held in the old (but beautifully refurbished) Town Hall of Wellington.

As our profession moves into its prime years, it becomes increasingly important, if our hard won professional status is to be maintained and enhanced, to continue supporting efforts made to advance scientific knowledge through information sharing and active discussion, both formal and informal. While acknowledging the strain under which we, like all other Health professionals, have been asked to function under in recent years, we cannot allow this to drain our enthusiasm, pride, or competence in our chosen fields of interest. The Conference then, *Nil Bureaucrati Carborundum*” encapsulates the spirit whereby we as a viable profession can face the future with tenacity, effectiveness, and even humour (never let bureaucrats grind you down).

This Conference will address issues of mutual concern in a constructive and supportive way, as well as continue the well established record of good scientific interchange. Its success is dependent entirely upon those who choose to support, participate and attend. The format will allow greater interaction with Industry representatives, and will be presented in an Absolutely, Positively Wellington style.

Plan to participate, plan to be there..... your future may well depend on it, for without an Institute and its annual conference, our profession will fragment and fade. Contracts, with or without Union involvement, are not enough, if we wish to be regarded and paid as professionals then we must continue to function as a profession, and be prepared to put in as well as take out.

Come along and Veni Vidi Vici in Wellington!

Gerard R Verkaaik
Convenor, NZIMLS 51st Conference 1997



ORGANISING COMMITTEE

Convenor	Gerard Verkaaik
Scientific Convenor	Rob Siebers
Exhibition	Geoff Day
Treasurer/Sponsorship	Gerard Verkaaik
Social	Philippa Holdaway

SECRETARIAT

Fran van Til
 Executive Events
 P O Box 78
 Rangiora
 Tel/Fax: 03 313 4761

VENUE

The Wellington Town Hall is the venue for the NZIMLS 1997 Conference. This elegant Edwardian building has been completely renovated and upgraded, faithfully restored to its original state of elegance. Delegates can step outside the Town Hall to stroll or relax on the plaza, visit the stunningly designed Public Library, the children's science and technology museum or the City Art Gallery.

ACCOMMODATION

We offer accommodation in the following categories:

Plaza International:

Luxury rooms	\$213.75
Executive Suites	\$331.90

Terrace Regency:

Business standard	
Twin Share	\$101.25

Trekkers:

Motels (Sleep 1-5)	\$99.00
Back-packers (double)	\$19.00
Back-packers (single)	\$17.00

All accommodation is within easy walking distance of the Wellington Town Hall.

ABOUT WELLINGTON

Variety and centrality are the key to Wellington's success as a conference venue. Wellington is the country's centre of politics and commerce. But there's also a fun side to Wellington's centrality - its cultural and social centre, boasting the best nightlife in the country. It is also the centre for static and performing arts.

SCIENTIFIC PROGRAMME

Will focus on comprehensive discipline sessions, business and stress related issues.

Further details follow in the tentative programme format. Keynote speakers will be profiled in the final announcement brochure.

EXHIBITION

Large-scale exhibitions are easily accommodated in the main auditorium of the Town Hall. Exhibiting is a powerful extension to a company's advertising, promotion, public relations and sales function.

If you are interested in exhibiting, please contact HISANZ, telephone 09 486 3000

SPONSORSHIP OPPORTUNITIES

We offer a variety of sponsor categories from single items to specially tailored packages which set out benefits to the sponsors. Full details of sponsorships available on request to Secretariat 03 313 4761.

SOCIAL PROGRAMME

The social programme has been designed to give delegates a taste of *Absolutely, Positively Wellington!*

▶ **Tuesday 26th August**

Jim Le Grice Icebreaker/Opening of the Industry Display. Drinks, nibbles, entertainment and more.

▶ **Wednesday 27th August**

Wellington is known as New Zealand's *cafe capital*. In a single 500 metre stretch you will find more than 100 bars, cafes, restaurants and night clubs. A recommended list will be available for you and your friends to choose from.

▶ **Thursday 28th August**

The CONFERENCE DINNER at a *Mystery Destination!!* Experience fine wine, cultural cuisine and a blast from the past. **It's a secret but it will follow the theme ... 'When in Rome...'**

CAR PARKING

Is available in car parking buildings in the immediate area of the conference venue.

CALL FOR ABSTRACTS

Abstracts are invited for oral and poster presentations. Prospective presenters will receive notification in writing of the acceptability of their proposed presentation.

Oral Presentations

10 minutes duration with 5 minutes for questions.

Posters

1 metre x 1 metre.

Abstracts**CLOSING DATE 6 JUNE 1997**

Structure your abstract under the following four headings:

- ▶ Introduction
- ▶ Methods
- ▶ Results
- ▶ Discussion

Please submit your abstract on a computer disk and **one** hard copy in an IBM compatible format (WordPerfect or Microsoft Word).

Approximate length 250 words on A4 page. Please include:

- ▶ The Presentation Title
- ▶ Authors with the **presenter's name in bold and underlined**
- ▶ Contact address.

In a covering letter please state your preferred session and indicate whether a poster or oral presentation.

Abstracts formats will be standardised for publication.

Please post to:

Fran van Til
Secretariat
Executive Events
P O Box 78
Rangiora

Enquiries to:

Rob Siebers
Tel: 04 385 5999
Fax: 04 389 5725

REGISTRATION FEES

Early Bird

(before 30 June)	Member	Non Member
Full Registration	\$300	\$400
Day Registration	\$100	\$130
Half Day Registration	\$ 50	\$ 65

After 30 June

Full Registration	\$360	\$460
Day Registration	\$120	\$150
Half Day Registration	\$ 75	\$ 90

Registration forms will be available in the May issue of the NZIMLS Journal or by contacting the Conference Secretariat.

QUESTIONS YOU WANT ANSWERED**Who should attend?**

Anyone who:

- ▶ is associated with Medical Laboratory Science
- ▶ wants to keep up with the latest developments in Med Lab Science
- ▶ wants to be at the forefront of their profession.

Why should you attend?

- ▶ Major changes are happening in the work place in science, technology and employment. This conference is addressing these changes.
- ▶ Discover how others are coping with the many changes that have happened and are going to happen in the working environment.
- ▶ Share new ideas with colleagues.
- ▶ Networking and socialising.

I don't have time to attend!

- ▶ Time spent now may save valuable working hours later.
- ▶ Half day registrations will be available.

I can't afford to go!

- ▶ You cannot afford not to go!
- ▶ It is in your best interests to attend, and the best interest of your employer for you to continue your education and competency.

Same old thing

- ▶ The scientific fora has been designed to meet today's needs and changes, and a look at what maybe around the corner both in Medical Laboratory Science, the work environment and the home front.
- ▶ Some of the more *mature* professionals will be sharing with us, the way they have dealt with the changes which have occurred.
- ▶ Extended lunch-times will give delegates more time to converse with the exhibitors.

NEW ZEALAND INSTITUTE OF MEDICAL LABORATORY SCIENCE

NBC CONFERENCE WELLINGTON

TENTATIVE PROGRAMME

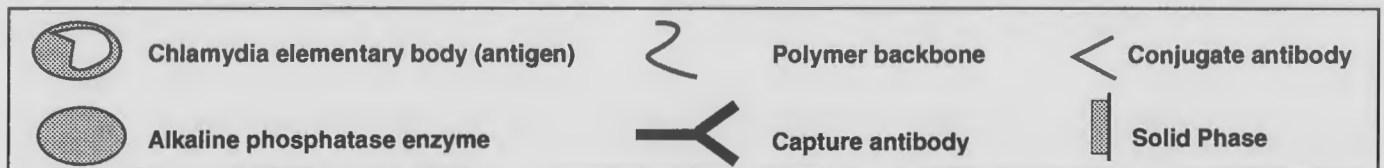
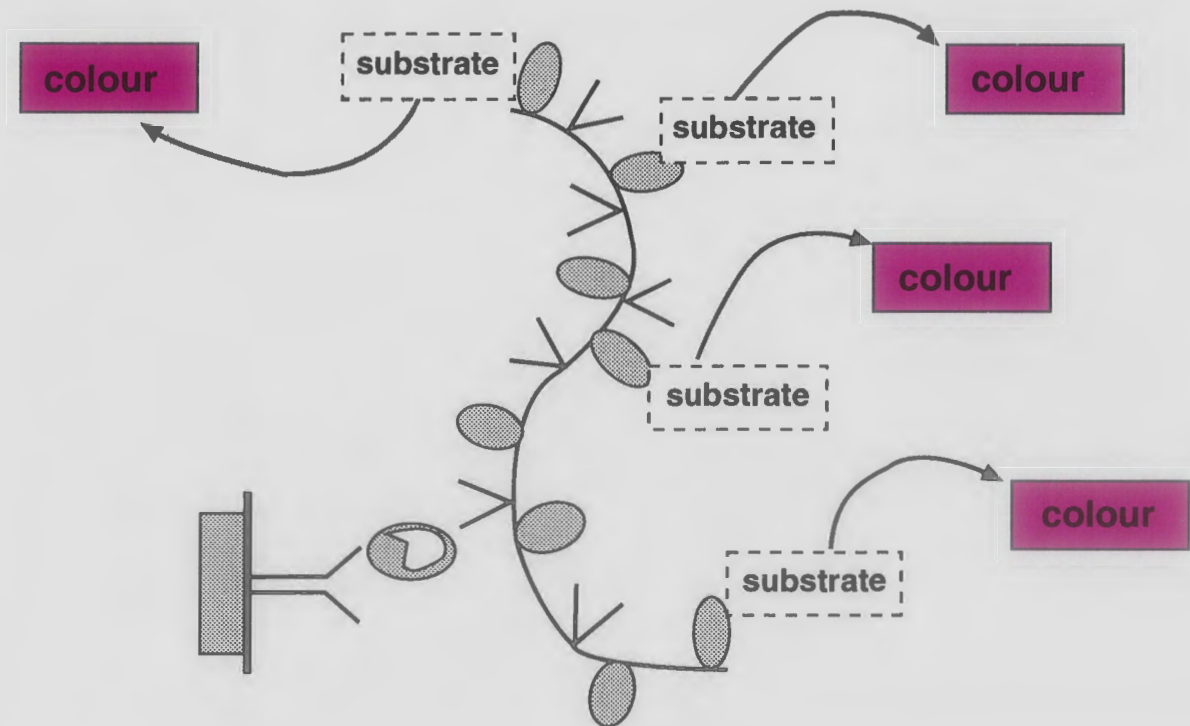
Time	Tuesday	Wednesday	Thursday	Friday
8.00am		Registration	Registration	Registration
9.00am		Welcome T H Pullar Address	Special Interest Groups ▶ Concurrent Sessions	Workshops ▶ Internet ▶ Performance Appraisal ▶ How to Give a Presentation
10.15am		Morning Tea	Morning Tea	Morning Tea
10.45am		Health Service Directions	Special Interest Groups ▶ Concurrent Sessions	Workshops ▶ Internet ▶ Performance Appraisal ▶ How to Give a Presentation
12.00		Lunch	Lunch	Lunch
1.00pm				Guest Speaker/ Entertainer Closing
2.00pm		Laboratory: Business or Public Service?	Special Interest Groups ▶ Concurrent Sessions	Happy Hour
3.00pm		Registration commences	Surviving the Bureaucracy	
3.30pm		Regulation: MLTB Transport Health & Safety	Afternoon Tea	
4.00pm		Afternoon Tea	Special Interest Groups	
4.30pm	Annual General Meeting	▶ Concurrent Sessions		
Evening	Jim Le Grice Icebreaker Opening of Industry Displays	Casual Dining	Conference Dinner at a Mystery Destination	

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- ◆ Excellent clinical performance
- ◆ Convenient economical testings
- ◆ Easily adaptable to open automated systems



Clinical and in-house trials show very high sensitivity and specificity on male and female urines. Results from all specimen types compare very favourably with probes, PCR and other amplification methods.

T.H. Pullar Memorial Address

*Marilyn M Eales FNZIMLS
Laboratory Services Middlemore Hospital, Auckland*

What a great occasion this is, the 50th Anniversary of the New Zealand Institute of Medical Laboratory Sciences (NZIMLS). A time to celebrate, a time to reflect and a time to ruminate and predict the future.

For me it is a time to feel both humble and honoured to be invited to give this address. Humble because I am deeply conscious of my forbears the prominent Pathologists and Medical Laboratory Scientists who have delivered this address before me. Honoured because a woman has been asked to deliver this address, only the third to do so in the 30 years since the inaugural address!! Honoured also because this address is dedicated to a man who contributed so much to the gradual build up of professional laboratory standards throughout New Zealand during the first 20 years of this Organisation.

Tomorrow the 29th of August is the 30th Anniversary of the death of this man. I refer of course to Dr. Thomas Pullar in whose memory this address has been given at our Annual Conferences since 1967. Thos Pullar was a true friend, teacher and supporter of the fledgling Organisation that we know today as the NZIMLS.

This address does not need to have a title but in keeping with the conference theme I have chosen to entitle it "Going for Gold" for two reasons. The first being that never before in the history of our Organisation have we been challenged like the Olympic athletes in their quest for gold medals to become assertive and competitive. Our quest is not a gold medal, but financial gain [gold] in the market place. Every aspect of our working practices are being analysed, taken apart and examined by Managers with, [dare I say it] multi-skilled backgrounds to see if our production line [laboratory tests] can be turned into a leaner meaner machine and return a profit to the organisations for which we work.

Some of you probably feel that you have been taken apart so many times over the past five years in an effort to seek financial gain "cutting off the flesh" they call it that there is now nothing left but "the bones to rattle".

The second reason for the title reflects on the 50th Anniversary, fifty golden years of our Organisation. Whilst we go for gold in the materialistic sense; in our confusion as we are re-engineered to do so; let us not forget the GOLD of the past. What must be avoided before it is too late is :

- The loss of professional knowledge and experience
- The lack of opportunities to develop and maintain expertise
- Loss of "Common sense" - that something that all of us have, but regrettably seldom use.
- Deterioration in our standard of work which directly relates to patient care.

It has been said and I quote [lest I be accused of it] that 95% of Managers today say the right thing but only 5% do it.

Fifty years to the very young seems an eternity; to those of us who are older, 50 years does not seem very long at all. In the context of history as it unfolds into the future our 50 years as an Organisation will certainly be viewed as our formative years, our metamorphosis phase, our evolution as a mature Organisation.

Currently we are bouncing around in a sea of uncertainties as budget driven management, compounds our feelings about our self worth; where the buzz words are redundancies, attrition, multi-skilling, re-engineering of staff and equipment and "down-sizing".

Where external surveys [usually done by Professional Accountants] relieve Managers by stating phrases like "your staff is surplus to supply" or "staff reductions are required as planning targets are exceeded". In short where the total emphasis is on cost control measures. In this environment it is difficult to find our self-esteem and respect both personally and professionally. We know we have a wealth of professional experience behind us which is gradually being eroded. We have concerns about our work quality and that multi-skilling will lead to less expertise and inferior trained staff.

What in bemusement we ask ourselves has happened to our comfortable working environment where we had, we believed, mostly a caring system.

Now that caring system has been replaced by a dollars system and it's uncomfortable.

For a few moments this morning I invite you to look behind with me to the future. To look back to our achievements - Nuggets of gold to be found there - polish and cherish them and take them into the future with us.

Winston Churchill once said "The further backward you can look the further forward you are likely to see".

The year we have travelled back in time to visit is not 1946 but 1896. One hundred years ago, Queen Victoria is on the throne of England, hospital boards in New Zealand are being pushed by doctors to set up Pathology Departments.

To understand the reason for this we need to glance even further back at what had been happening in medicine in Europe during the 19th century. Doctors had been seeking answers to questions and had used their intelligence to ask the right questions about possible causes of disease. Several had set up their own primitive research laboratories in the hospitals in which they worked. The outstanding characteristic of 19th century medicine was the correlation of discoveries in these primitive laboratories and autopsy rooms with observations at the bedside.

Alexander Virchow was one of these early pioneers fully utilising microscopic studies in the complete autopsy and he set the pattern for the full development of Pathology as a distinct speciality.

Others who were medical pioneers and contributed greatly during this century were;

Semmelweiss and Lister for their observations on cross infection and antiseptic technique.

Robert Koch for his work on the causative organism of tuberculosis. Edward Jenner as a pioneer of vaccination.

Paul Ehrlich as a founder of chemotherapy.

Knowledge of pathology was passing from the realm of general information to that of immediate practicality.

Samples of tissue a few millimetres wide could be obtained for microscopic study from a wide variety of organs and the interpretations of pathologists could determine not only the diagnosis but the course of therapy and ultimate prognosis.

Techniques were cumbersome and took many days to process, a far cry from the frozen section of today. In the primitive laboratories, disease processes were generated in laboratory animals to provide information on the basis, nature and therapy of similar conditions in humans.

Is it little wonder then that at this time one hundred years ago doctors were pushing to set up Pathology Departments in New

Zealand. Wellington Hospital appointed a Dr. Fyfe as its first Bacteriologist. Christchurch Hospital had set up a corner of the dispensary as a laboratory and any member of staff was free to work there if he was competent in the technique. Few were apparently and specimens were sent to Dunedin where there was more expertise at the Medical School.

Christchurch Hospital then appointed a Bacteriologist, a young Dr. Louisson who had been acting Bacteriologist at Guy's Hospital, London. You will note that I said young as many years later as a General Practitioner in his late 60's he had the honour of bringing me into the world. Later I too, was to work at Guy's Hospital. Such is the link in history.

In 1900 the Health Department in New Zealand was formed. It arose from a rushed piece of legislation with the threat of bubonic plague which had appeared in Auckland. It was given wide powers and when the urgency passed the powers remained and have steadily increased. In the shadow of *Bacillus pestis* the Health Department was born. By 1910 it had a firm grip on hospital control, a grip it retained firmly for over 80 years.

Coming a little further forward in time and working towards 1946, the birth of our Institute, what events were taking place to hasten this birth?

In 1912 the first full time Pathologist Dr. A.B. Pearson was appointed in New Zealand. Dr. Pearson started in a laboratory that contained one incubator, one autoclave, one microscope and an iron saucepan for sterilising articles. From 1913 onwards skilled technical people were imported from the United Kingdom, a trend that continued for some considerable time.

My esteemed Chairman today, Mr. John Case was one such import to Dunedin Hospital.

In fact the occasional import from the United Kingdom is still happening, particularly in the area of Blood Transfusion Science where we are woefully short of experienced people in New Zealand.

Major happenings in the scientific world that affected the development of laboratory science in the early part of this century were: the discovery of the major blood groups the arrival of the electron microscope, the birth of antibiotics, thanks to Alexander Fleming's astute observations on the action of penicillin.

During the 1930's many qualified nurses went into training in laboratories in New Zealand and once trained were transferred to smaller hospitals as bacteriologists. Perhaps the greatest influence on developing laboratory technology in New Zealand was the advent of the Second World War 1939-1945. The skilled technicians went overseas and trained replacements were impossible to find. Married women, nurses, medical orderlies, retired typists and volunteers all worked as technicians. Many of the skilled technicians who joined the Armed Forces became Commissioned Officers, a recognition of their expertise.

The early 1940's saw the development of Blood Transfusion Services. Inoculations, vaccinations and blood grouping of soldiers were done by Pathology Departments.

Penicillin was available for civilian use in 1944 and the distribution of this was rigidly controlled and entrusted to Pathologists. Prior to this time it was never permissible for a technical person to take blood from a patient by venipuncture. The Pathologists generally did not like doing it and viewed it as a formidable procedure. Is it little wonder that as the Pathologist's duties increased, the delegation of this procedure was given to the growing numbers of technical people in the laboratories? The Health Department introduced water and milk supply analysis and tests involving the use of live animals increased.

Towards the end of the 1940's one era was closing and another beginning. The war was over, the skilled technicians returned from war service and with them many others who had gained

laboratory experience in Military Hospitals overseas - wider experience than the volunteers and nurses who had filled the void while the war was on.

Test numbers and test variety had increased. Pathology Departments were beginning to break up into specialities. The scene was thus set for the birth of the New Zealand Association of Bacteriologists that would bring together the diverse technical people from all over New Zealand. An Organisation where professional issues would be improved, where knowledge would be shared, where training programmes would be planned and developed and National Examinations established.

Dissemination of information was one of the primary aims of the New Zealand Association of Bacteriologists who swiftly moved to produce a Journal, conduct formal examinations and stage an Annual Conference.

The need for ongoing training was recognised from the beginning and was voiced in the Editorial of the January 1947 Journal. I quote "Senior members do not just happen, they grow from Junior Members and it is an urgent and essential duty to train our Junior Members now and in the best possible way so we will be prepared for the expansion that must come".

From the enthusiastic beginning of this Organisation the Journal became our professional voice. Despite a paucity of articles being proffered and Editors pleas for Journal material it has survived. That it has done so is a tribute to the foresight, intelligence, stamina and determination of all the editors over the past 50 years.

To those of you who have complained at times about the lean content of the Journal I say "look first to yourselves, what did you do to improve the content or more pertinently what are you doing today to ensure its survival into the 21st Century?". Is this one of the nuggets of gold we should seriously be looking at to take with us and preserve?

One person who quietly financially supported our Journal in the 1940's when it was struggling to become established was Thomas Pullar. He was not only a true friend, mentor and guide of our fledgling Organisation but gave financial assistance as well.

When I joined the New Zealand Association of Bacteriologists in the 1950's Senior members of staff were people alluded to earlier, war veterans who had returned after working in Military Hospitals in Italy or the Middle East and nurses who had trained as bacteriologists. The examination system had been established and the requirements at that time were to sit an Intermediate Certificate after three years training to be followed by the Certificate of Proficiency in Hospital Laboratory Practice two years later. A five year course. Since then the examination system has gone through many phases. The New Zealand Certificate of Science as the basic training examination followed by O and A levels in specific specialist topics.

These were later called Part II and Part III and later again Certificate and Specialist Levels, all incorporating both oral and practical examinations. The oral and practical examinations were later phased out, to be followed by the introduction of log books for practical assessment.

After 50 years of metamorphosis, our examination system has thankfully emerged with a Bachelor of Medical Laboratory Science (BMLS) now firmly established for Technologists. Over the years many people worked extremely hard at different periods of our history to achieve this and I say today a special thank you to all those who contributed to this long struggle to keep the professional hope alive. Your efforts have at long last been professionally realised.

A training programme and examination system pioneered by the invaluable School of Medical Laboratory Technology in Auckland was established for Laboratory Assistants. The Qualified Technical Assistants (QTA) examination still continues today for Laboratory Assistants.

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Many here today will recall the days when practical examinations were held in the laboratories at Massey University. Students and examiners all travelled to Palmerston North for the occasion. Students were not only under the expense and strain of travelling there but also often under the threat of "If you pass - we pay. If you fail - you pay".

Examiners had the nightmare of trying to keep cultures viable, reagents potent and have sufficient equipment and additional reagents available to allow for any variations on the theme of a method that a wayward candidate might embark upon. After arriving at Massey with all this clobber, examiners might then find themselves gathering stones from the Massey driveway to hold down the plastic racks that insisted on floating in the water baths or sitting down and cleaning 30 microscopes so that visibility was a "little less than foggy".

But I digress, teaching and training is another nugget of gold which must be cherished, polished and taken with us as we journey onwards.

The formation of the Special Interest Groups and their challenge of continuing education must be encouraged to grow and develop further.

In the evolving years of our Organisation the New Zealand Institute of Medical Laboratory Sciences has firmly established or assisted the following to be achieved often in conjunction with the Medical Laboratory Technologists Board (MLTB).

- A professional voice - The Journal
- Degree status - BMLS
- A qualification for Laboratory Assistants
- Continuing Education
- Encouragement of Laboratory Accreditation Standards (Telarc)
- Encouragement to implement the Maintenance of Laboratory Standards (MOLS) programme.
- In earlier days guidelines for hours of work, remuneration, annual leave and sick leave.
- Registration of Medical Laboratory Technologists to protect patient care and professional interests.
- Support and encouragement to the successful Pacific Paramedical Training Centre in Wellington which began in 1981 and is now recognised as a World Health Organisation collaborating centre.
- Participation in the affairs of and Representation on the Council of the International Association of Medical Laboratory Technologists first with Desmond Philip and as from June of this year Dennis Reilly.

All of these things would not have been achieved without the input of dedicated people who care about the profession of Medical Laboratory Technology and its direction into the future.

Nothing can be achieved without people contribution and as with all major achievements it has been the dedication and quality of a handful of the membership that have made these achievements possible. The majority of members and an even greater number of non-members have taken a free ride on all these achievements over the years whilst saying "what has the Institute done for me?" There will be many in the audience today!!

There have been predictions that by the year 2000 our profession will cease to exist, that we are finished, that we have come to the end of the road. Our profession as we know it may cease to exist but we the people (those who are allowed to, or choose to survive) will still be there.

Our professional Organisation must survive to be the beacon, the guiding light of our aspirations our Olympic flame burning warmly to encourage us on. As we race headlong towards the year 2000 with automation, computers, paperless and as the facetious say "people-less" laboratories we will need more than ever before, that

beacon, that Olympic flame providing guidance and encouraging the maintenance of our professional standards.

This year for the first time in history we will have a female president and a female vice-president - two women at the helm. Are the men all scampering because the going is getting tough? or are enlightened men of this era awakening to the golden qualities of human understanding that women can contribute as we go for gold together!! The road will not be easy, they will have to be women of substance to keep alive and nourish our Organisation into the future.

Please if you care about patients, work quality, continuing education, professional standards and professional ethics give them and their committee your support.

As you go into the unknown seeking gold, take the gold standards of the past 50 years with you.

We enter the future facing backwards seeing only the road on which we have just travelled. For a few brief moments this morning we have reflected on the achievements of the past. The advent of the computer has had an enormous effect on the lives of human beings, far greater than the Industrial Revolution in the middle of last century. It has changed the way we live, the way we do things, and we must change to meet the challenges that this microchip brings in its wake.

For the experienced Technologist I have this message - "Experience is a quality you have, no one can buy it, it is invaluable, but you may have to change in the way in which you present your experience. Be pro-active rather than reactive. In the competitive world of private enterprise those who say "it cannot be done" will be swept away by those who work out a way to deliver the required improvements."

To the young graduates of today I say "Go for it, but let your employer know that you want to learn as well as work, demand that your bright intuitive minds do not get neglected. Encourage the Institute in its endeavours to provide Continuing Education. "

The day may well come when routine medical laboratories no longer exist because the ultimate testing device has been developed - a walk in chamber where at the flick of a switch the whole body is scanned and within a few minutes the laboratory results, x-ray films and electrocardiogram results are displayed on a screen in the Doctor's office next door, without any invasive techniques whatsoever!!

Should the Doctor not be in the room when the screen results come through, the automatic locator attached to the Doctor, complete with mini-screen highlighting the abnormal findings would not only beep but send mini-shock waves to the receiver until the screen had been cleared thus ensuring the results had reached their ultimate destination, the Medical Officer concerned.

The Technologist of today may be transformed into an Information Systems expert. We must now live for the future.

The Health Reforms are here to stay. Our 50 years of evolution are over. We face the dawn of the next 50 years in a state of revolution driven by fiscal considerations.

As we "Go for Gold" we must hang on tightly to our evolutionary Olympic torch and pass it safely on through the revolution to bum ever more brightly in the future.

If we drop the baton, all the golden endeavours of people like Dr Thos Pullar, Dr Stephen Williams, Dr Dennis Stewart (who died only last week), Douglas Whillans who began the Journal in 1947 and all the other Institute Office bearers over the last 50 years; will be lost forever.

Don't let them down.

New Products and Services

New Dako Catalogue for 1997

Many new products have been introduced to DAKO's expanding line of products. DAKO supplies a wide spectrum of high-quality antibodies, monoclonal and polyclonal, for both diagnosis and research. They also have a wide range of products for Clinical Immunochemistry, Clinical Microbiology and Flow Cytometry.

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DAKO IDEIA PCE Chlamydia

IDEIA PCE Chlamydia is a new dual amplification enhanced EIA for the detection of Chlamydia. It provides a combination of excellent clinical performance and ease-of-use.

The antigen is "actively" captured onto the solid phase by chlamydia-specific monoclonal antibody. Bound antigen then becomes labelled by an alkaline phosphatase conjugate.

The alkaline phosphatase molecules are covalently bound to a polymer backbone. Lots of alkaline phosphatase molecules can be bound on to this polymer backbone, which amplifies the rate of product (NADH) formation from substrate (NAPH). This amplification step significantly enhances the test sensitivity.

But it doesn't stop there! The NADH catalyses a cyclic, dual enzyme reaction, which is the secondary amplification. Molecules of product are repeatedly cycled in the enzyme reaction to produce the coloured signal (a distinct red), which can be read visually or in a spectrophotometer at 490nm. This signal amplification is DAKO patented.

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Bayer Builds Strong Internet Following With Central Lab "VIRTUAL" Symposium

Tarrytown, NY. January 30, 1997 – Bayer's Business Group Diagnostics invites the worldwide audience of health care professionals and their interested parties to log-on to its "virtual symposium" – Bayer Diagnostics Central Laboratory Internet Symposium: Series 1 featuring Anemia Management and Workflow.

While Bayer's Central Laboratory Symposium is being held in Rome, April 20-22, 1997, an interactive Internet site featuring the Anemia Management and Workflow portions of the Rome Symposium will be made available (beginning April 22) to expand participation. If you plan to attend on-line, you are invited to use Bayer's Internet Symposium website e-mail feature to dialogue with the Scientific Faculty. Presentations will cover topics such as a keynote on Anemia Management by Profs. Price and Newland; Workflow and its Economic Impact on the Laboratory; and others. The Scientific Faculty will respond to your questions and comments via Bayer's Internet Symposium website which is accessible at www.labfocus.com and www.bayerdiag.com.

The Bayer Diagnostics Central Laboratory Internet Symposium: Series 1 featuring Anemia Management and Workflow will continue for three weeks to give participants sufficient time to communicate with the Scientific Faculty.

David Davenport, Senior Marketing Manager from Bayer's Business Group Diagnostics commented:

"By offering this symposium on the Internet, we open up productive and rewarding dialogue with a worldwide audience.

Anyone with access to the Internet and an interest in the subjects can log on to Bayer's Internet Symposium. They will have a chance to have questions answered by a highly respected faculty which will include former AACC President Peter Wilding, workflow expert Dr. Craig Lehmann and keynote speakers Professors Adrian Newland and Chris Price from the United Kingdom."

Bayer Corporation, Diagnostics Division, headquartered in Tarrytown, NY, is part of Bayer's worldwide Business Group Diagnostics. One of the largest diagnostics businesses in the world, the group serves customers in 100 countries. Customer offerings include in-home blood glucose monitors such as the GLUCOMETER ELITE® and GLUCOMETER ENCORE Diabetes Care Systems: urinalysis systems such as the CLINITEK® 50 analyzer, and large automated laboratory analyzers such as the opeRA system, the BAYER IMMUNO™ 1 System and the TECHNICON H*3® Hematology System. The Group has some 4,800 employees. 1995 sales were over \$1 billion.

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Gen-Probe Announces the Release of New Amplified Chlamydia Test for Urine Testing

Gen-Probe, the leading provider of tests for *Chlamydia trachomatis* (CT) to reference and hospital laboratories worldwide, announced it has added an extremely sensitive, non-invasive CT test, the Gen-Probe Amplified CT assay, to its line of top selling diagnostic kits for sexually transmitted diseases. The test, which can detect as few as one Chlamydia organism present in a patient's urine sample, is now available world wide.

The Gen-Probe Amplified CT Assay complements Gen-Probe's existing line of non-amplified products, including PACE 2 and PACE 2C Systems, which can accurately and cost-effectively detect the presence of both *Chlamydia trachomatis* and *Neisseria gonorrhoeae* with one swab, one test. In instances that demand the highest possible level of sensitivity – cases where patients are asymptomatic or where low numbers of infecting organism may be present – the heightened sensitivity of Gen-Probe's amplified CT diagnostic is beneficial. The test has the ability to offer physicians a diagnosis through two sampling methods: collecting a patient sample with a urogenital swab or using urine as a specimen.

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Freefax 0800-688-883

Cardiac Troponins in routine clinical practice – a cardiologist's perspective

A lunchtime lecture series on the above topic will be given in major centres around the country in April.

The speaker, Dr Peter Stubbs, is a visiting United Kingdom cardiologist with extensive knowledge of the biochemical markers of myocardial damage and their use in clinical practice.

Among other topics the role of Troponin T as a diagnostic marker and in risk stratifying patients admitted with acute coronary syndromes will be explored. As well a protocol for managing the chest pain patient will be discussed, as will the associated cost/benefit analyses.

Dates for the lectures will be:

Thursday 17th April	Christchurch Hospital
Friday 18th April	Dunedin Hospital
Monday 21st April	Wellington Hospital
Tuesday 22nd April	Boehringer Mannheim Auckland

For more information contact your local Boehringer Mannheim office, or call 0800 652634 (Auckland 2764157)

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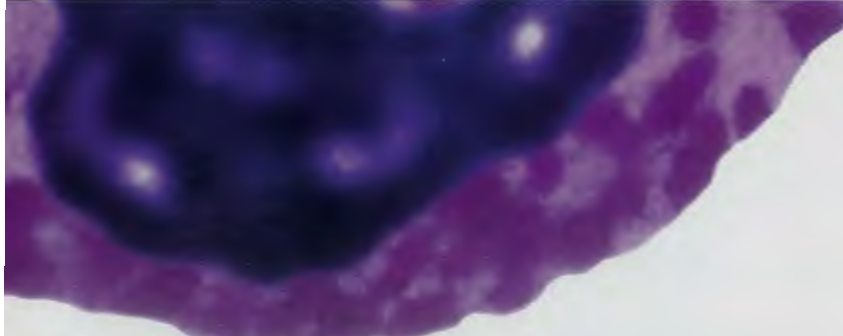


Workshop Announcement

Introduction to Molecular Genetics and Gene Manipulation

A one week non-credit introductory workshop will be held in the Microbiology and Genetics Department of Massey University during the mid-semester break 30 June - 4 July 1997. The aim of the course will be to provide a working introduction to the powers and limitations of molecular genetic techniques, for people with a professional interest in the subject. Material to be covered in lectures and discussions will include DNA and genome structure, DNA polymorphisms, gene regulation, the molecular genetics of plasmids and transposons and basic strategies of recombinant DNA research, including PCR and sequencing. Concurrent sessions will be held on the final day to allow for specialised interests in the areas of plant and medical molecular biology. Practical work will include plasmid isolation, transformation/electroporation, restriction enzyme mapping, DNA cloning, PCR and RFLP analysis. Background assumed will be the equivalent of Introductory Genetics and Introductory Biochemistry (200-level). Although Boehringer-Mannheim are continuing their generous sponsorship for this course in the form of biological materials, there will be a charge of \$500 (plus GST), in order to cover the cost of additional materials and facilities. A lectures-only option is also available (\$150 + GST). Accommodation will have to be arranged off campus as, unfortunately, extramural fully books the campus accommodations. The enrolment will be limited to 30 (the capacity of the teaching laboratory). For further information and an enrolment form, please contact:

Dr Rosie Bradshaw (Organiser)
or Ms Fiona Elrington (Departmental Secretary)
Department of Microbiology and Genetics
School of Biological Sciences
Phone: (06) 350 4025 (R. Bradshaw)
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Pacific Paramedical Training Centre (PPTC)

The PPTC has recently published a new brochure updating information about the PPTC as follows:

The Pacific Paramedical Training Centre is located at Wellington Hospital, Wellington, New Zealand. It is a non-profit Organisation supported by the New Zealand Ministry of Foreign Affairs and Trade, the New Zealand Ministry of Health, the New Zealand Red Cross, the New Zealand Institute of Medical Laboratory Science, the World Health Organisation, and others.

- The Centre offers courses and training programmes in medical laboratory technology and blood transfusion service development both in Wellington and in countries throughout the Pacific region and Southeast Asia.
- The Centre assists developing countries in the development of their own training programmes in medical laboratory technology.
- The Centre serves as the major advisory agency for the placement of overseas technicians who wish to come to New Zealand for specialised training in medical laboratory technology.
- The Centre runs an external quality control assessment programme specifically designed for medical laboratories in developing countries.
- The Centre has been designated by the World Health Organisation as a Collaborating Centre for external quality assessment and training in health laboratory services.
- Personnel from the Centre are available for short term consultancies.
- The centre collects and supplies used equipment to developing laboratories.
- The Centre will appraise new instruments for laboratories.
- Centre staff carry out short term consultancies for the WHO.
- Centre staff engage in research projects in the Pacific Islands and support local researchers.
- All course lecturers are Registered Medical Laboratory Scientists and have experience working in developing laboratories.
- Centre staff have worked in most Pacific Islands, Papua New Guinea, Vietnam, Cambodia, Lao, China and Philippines.

Courses offered include haematology, blood bank technology, medical microbiology, clinical chemistry, water and food testing, HIV/HBV serology, laboratory equipment



maintenance, laboratory management, the laboratory diagnosis of acute respiratory, diarrhoeal and sexually transmitted diseases and general updating courses.

The courses conducted at the Centre are usually of two months duration and include both theory and practical work. They are designed to meet the needs of each participant and his/her home laboratory. The emphasis on practical and appropriate training ensures that the trainee will be able to use effectively the newly acquired skills immediately upon returning home.

Sponsorship

Application for sponsorship to attend training courses at the Centre or for training by attachment to a hospital laboratory should be made by the Government or State Training Officers to the nearest World Health Organisation office or to the nearest New Zealand High Commission or Embassy.

Those not requiring sponsorship or just wanting further information should write to:

**Pacific Paramedical
Training Centre
PO Box 7013
Wellington
New Zealand
Tel/Fax 64 4 389 6295**

Solomon Islands

The Rehabilitation Workshop at the Central Hospital is manufacturing wheelchairs basically made from fibreglass and stainless steel with solid and wide tyres. The fibreglass components are produced by a local fibreglass manufacturer, Aruligo Fibreglass and the rest of it is assembled in the Rehabilitation workshop by John Alingbatu, a disabled person. The wheelchairs have minimal maintenance and whatever is required should be able to be done in remote villages. The aim is to provide wheelchairs to disabled people in the Solomon Islands with a design appropriate for local environmental conditions simple yet strong, rust-proof and puncture proof.

The material cost of each fibreglass wheelchair is roughly about \$1,000. The concept came from the New Zealand High Commission in Honiara. Donations from both the New Zealand and the British High Commissions in Honiara have enabled the first 35 wheelchairs to be manufactured.

The wheelchairs will be monitored for performance in the local conditions and

will be available free of charge to disabled patients. Until now the Solomon Islands have relied on receiving donated wheelchairs from Australia and New Zealand as the cost of importing new wheelchairs is extremely high. There will still be a demand for some imported wheelchairs as well as the locally made fibreglass ones. The type of wheelchair used will depend on the varying requirements of the disabled.

This project is an exciting one as it provides work for local business as well as providing economically viable wheelchairs for the disabled.

Papua New Guinea

Provincial governments are to shoulder the bulk of the Papua New Guinea's health services under health sector reforms approved by Parliament. Health Minister Philemon Embel said significant aspects of the reforms include a shift in emphasis from urban to rural and from curative to preventive health care. He said public hospitals were now national functions and all rural hospitals, health centres, clinics and aid posts were the responsibility of provincial governments. PNG has a total of 2,399 aid posts, 199 health centres, 303 health sub-centres, 45 urban clinics and 19 hospitals. Churches provide 45 per cent of the total services including 49 per cent of the rural health services.

\$40 million project to improve health

Australia is to fund a multi-million health project in Papua New Guinea, aimed at improving health standards of women and children. The project, expected to cost more than \$40 million over five years, will upgrade health centres and aid posts around the country. Aid posts and health centres will also be funded to establish regular outreach clinics in villages to ensure that women and children in even the remotest regions have regular access to health care. The project will also train nurses, community health workers and village-based health workers and birth attendants.

Leprosy - Further Decline in Prevalence

As a public health problem, leprosy is slowly declining throughout the Pacific. However, the problems caused by disability remain. Patients who may no longer have active leprosy must still live with its devastating effect on their lives. Without continuous care and rehabilitation people will suffer needlessly.

The number of registered leprosy cases in the world has fallen below one million for the first time since global statistics

on the disease began to be collected. This suggests convincingly that WHO's strategy for eliminating leprosy as a public health problem is well on track.

In fact, the overall prevalence of leprosy, which declined by 27% between 1994 and 1995, has fallen by a further 28% between 1995 and this year. Over the past ten years, the world's leprosy burden has been reduced by 83%.

Leprosy remains a public health problem in 60 countries or areas, but 16 countries contribute to about 90% of the leprosy problem in the world. India heads the list with 560,000 registered cases, far ahead of Brazil with 95,564.

Then follow Indonesia, Myanmar, Nigeria, Nepal, Bangladesh, Philippines, Mozambique, Ethiopia, Zaire, Madagascar, Sudan, Tanzania, Guinea and Cambodia.

Dengue Fever Outbreaks

Dengue fever first appeared in the 18th century but the World Health Organisation has only recently begun to count it as one of the most dangerous tropical diseases. The number of countries fatally hit by dengue fever has risen since 1970 from nine to more than 40, and the WHO estimates that more than 20,000 people now die from the disease each year. Deadly outbreaks have erupted this year in Singapore, Indonesia, Malaysia and the Philippines. None took anywhere near as many lives as a recent epidemic in India.

In this epidemic, over 7,000 people were stricken and 270 died. It was India's worst dengue fever outbreak since the 1960s. There is no vaccine and no cure for dengue fever.

Dengue fever has occurred recently in Western Samoa and sporadic cases have been reported from other Pacific Islands.

Tuberculosis

Tuberculosis (TB) is now the world's biggest disease. The disease is now the world's leading cause of death from infectious disease. Ten million people become infected and three million people die from TB every year and the figures are increasing. Haphazard use of drugs has led to the bacterium that causes the disease, *Mycobacterium tuberculosis* (MTB), becoming resistant to all the drugs used against it in some areas of the world.

British and United States scientists have developed a new kind of vaccine to protect against TB. Unlike conventional vaccines this is made not from dead or weakened bacilli (bacteria) but from pure DNA genetic materials.

Research has shown that this should make the vaccine cheaper to produce, more effective and easier to store and transport in

the tropics.

Pacific Paramedical Training Centre

Extracts from the Annual Report of the Chairman, Dr R McKenzie

Activities 1996

The official opening of the new PPTC premises in December last by Her Excellency Dame Catherine Tizard marked the beginning of another active year for the Centre.

The activities included training courses at the Centre, training placements in specialised laboratory disciplines and the maintenance and further development of the Pacific Regional Health Laboratory Quality Assessment Programme.

The work of the Centre has continued to broaden during 1996 with co-operative links being made with other NGO groups working in health areas in the Pacific and SE Asia.

Of special note is the joint venture established between VSA, PPTC and LABNZ. This has involved the placement of a laboratory scientist in the Binh Dinh Province of The Socialist Republic of Vietnam.

The assignment is for a two year period and will involve medical laboratory and blood transfusion service development in district hospitals.

The focus of the Centre's activities during 1996 has been and will continue to be in the Pacific Region. Some 28 Pacific Island Laboratory Technicians attended courses or had work attachments.

1996 was also year one of the third cycle of the three year Samoan Laboratory Technical Programme at the National Hospital Laboratory in Apia. This course continues to run well under the direction of the PPTC and technicians who have completed the course are making a significant contribution to the health laboratory schemes in Western Samoa.

During 1996 negotiations were conducted with MFAT and WHO and planning is under way for the running of in-country medical laboratory quality assurance courses in Papua New Guinea and Fiji.

Planning was also continued with the University of Papua New Guinea and MFAT to begin the second cycle of in-country courses for rural medical technicians. It is hoped this will take place in 1997.

During 1996 the Centre has continued to serve as a coordinating agency for the collection of appropriate surplus medical laboratory equipment and textbooks for donation overseas.

The PPTC is indebted to the Communicable Disease Centre and a number of New Zealand hospitals for surplus equipment, journals and other reference

materials for this purpose. The PPTC are also most grateful to Mr David Wiseman of Medical Aid Abroad/The Peace Council for packing and shipping arrangements.

Projected Activities 1997

The Wellington based training courses are planned for 1997. They are Blood Bank Technology in February, Laboratory Management in July and Blood Cell Morphology in September.

The Pacific Regional Quality Control Programme will continue to run monthly from March to November and the second year of the third cycle of the Western Samoan Laboratory Technology course will commence in February. There are 10 trainees sitting the end of Year I examinations in November 1996 and the successful trainees will proceed to Year II.

At the present time, there are two attachments at New Zealand laboratories being arranged for 1997.

Negotiations are under way with Papua New Guinea and MFAT to undertake an evaluation of the two training courses run by Gilbert Rose for Rural Laboratory Workers in 1995. The University of Papua New Guinea and the World Health Organisation office in PNG have asked the PPTC to participate in two workshops on Quality Assessment in early 1997.

The PPTC will also be involved in providing learning experience for a senior technician from the Provincial Hospital at Binh Dinh, Vietnam, and two blood bank technologists will be attending training in English language and blood bank technology.

The World Health Organisation Regional Headquarters in Manila have requested the services of Mr Lynch to act as consultant for a regional workshop on the Regional External QC Programme. This workshop will be held during the second half of 1997.

Acknowledgements

The Pacific Paramedical Training Centre is indebted to a number of organisations and individuals for ongoing support and encouragement.

To the following the PPTC extend sincere thanks for generous assistance:

The New Zealand Ministry of Foreign Affairs and Trade; the New Zealand Ministry of Health; Capital Coast Health, Department of Laboratory Services; New Zealand Red Cross, National Headquarters; New Zealand Red Cross, Central Region; New Zealand Institute of Medical Laboratory Science; Norman Kirk Memorial Trust; the Royal College of Pathologists Australasia; Australasian Association of Clinical Biochemists; CITEC Training Solutions.

Prize Giving Address – November 1996

Margaret Chamberlain, International Liaison officer, Ministry of Health presented the prizes and gave the following address at the Prize Giving ceremony for the Medical Laboratory Course and the Cytology Training students at Wellington Hospital on Friday, 1 November 1996.

Thank you for this opportunity today to address the prize giving ceremony for the trainees of the medical update course and the cytology trainees attached to Wellington Hospital. My congratulations to you and your tutors and management of this centre who together have made this event possible – well done.

I always enjoy the prizegivings at this centre and try to attend as often as I can for it gives me the opportunity to meet the students, who up until then have been names that have come across my desk. It is good to see so many countries represented here today. I note that we have students from Papua New Guinea, Solomon Islands, Kiribati, Vanuatu, Fiji, Palua, Marshall Islands and Tuvalu.

Back in 1979 I had the pleasure of attending an address given by Dr Alec Sinclair from the Department of Health on International Health. In those days he wore two Department hats one being the Director of Hospitals Division and the other as Principal Medical Officer, International Health. I was most impressed with the International Health work the Department was doing then in its advisory role to the Ministry of Foreign Affairs on development assistance being given to the South Pacific and the role that WHO was planning in the same area – this is for me, I thought! I'd like to work in the International health field.

After spending time in the Department's Public Health and the then Hospitals Divisions, in 1980, I was appointed to the International Health Team led by Alec Sinclair and soon found that I was involved in the planning and establishment stages of what is today the Pacific Paramedical Training Centre.

The establishment of this facility was almost entirely due to the drive and enthusiasm of Professor Ford the then Head of Department, Clinical Pathology at Wellington Hospital and Dr Ron McKenzie at that time an administrative technologist at the hospital, later Director of Laboratory Services and currently the Chairman of PPTC.

By the time I came on the scene, the ground work had been well laid:

- a no longer used laboratory at Wellington Hospital had been identified
- Wellington Hospital had been

persuaded to lease the space

- and the Professional Organisation for Medical Laboratory and Technologists had given its support.

For many years before 1981 New Zealand had been accepting ODA and WHO Fellowships for some 5 - 6 laboratory technologies every year for "on-the-job training." in general hospital laboratories. In 1981 however it became apparent that because of the widening differences in the practices and technology developing between the countries seeking Fellowships the training provided was not entirely appropriate to meet their needs. The Department of Health was therefore very supportive of the initiative to establish PPTC. The International Health section, through the Ministry of Foreign Affairs, was subsequently successful in persuading the New Zealand Government to provide some financial support towards the venture together with the Red Cross and WHO.

So I have had the privilege of being associated with the Pacific Paramedical Training centre, its students, tutors and management, in its planning stages and inauguration in 1981 with its first Food and Water course run in September of that year.

Since then laboratory trainees from abroad have been sent to this centre to take advantage of training programmes that are pitched to their appropriate level of practice and technology.

I have seen this centre expand with the development of the Pacific Regional Health Laboratory Quality Assessment Programme and the In Country Training Scheme in Western Samoa – and not least the moving of the PPTC Operation, last year to these premises which were opened by the Governor General.

This centre has captured the interests of previous Governors General, Ministers, Senior Health Officials, Red Cross, Health Authorities in the Western Pacific Region. The World Health Organisation which has designated PPTC as a WHO collaborating centre. Many other Organisation have also given support. The work done by PPTC is highly regarded in the Western Pacific Region. In fact I have attended two meetings in the region – one WHO Regional Committee meeting in Hong Kong and the WHO Fellowships officers meeting in Manila. At both these meetings the participants highly commended the work of PPTC and the benefit to their countries had got from the students who had attended PPTC.

You the students from the Medical update course and the Cytology Trainees will be an asset to your Health Services in your home countries just like the other 450 students that have trained at this Centre

before you.

Well done on your great achievement and knowledge that you will take back to your countries. You have had great tutors. Your country and families will be very proud of you today.

It has been a privilege to have been associated with this centre as I have recalled over the years the one thing that has always impressed me is the spirit and goodwill that is shared amongst the students, tutors and management on each course and Mr Lynch tells me that this group has been one of the best.

I hope you have enjoyed your stay in New Zealand. Have a safe journey home and I wish you well with your careers.

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Please address all correspondence to the Executive Officer, including Examination and Membership enquiries.

Editor

Rob Siebers
Dept. of Medicine, Wellington
School of Medicine, P.O. Box 7343
Wellington South.
E-Mail: rob@wnmeds.ac.nz

Membership Fees and Enquiries

Membership fees for the year beginning April 1, 1997 are:

For Fellows – \$101.40 GST inclusive

For Members – \$101.40 GST inclusive

For Associates – \$48.10 GST inclusive

For Non-practising members – \$44.20 GST inclusive

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

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

Membership Report – February, 1996

Membership	13.02.96	19.09.95	19.07.95	07.06.95
	1006	1079	1084	1174
Less resignations	11	68	6	28
Less G.N.A.	15	7	6	16
Less deletions	–	–	–	109
Less deceased	1	2	–	–
Less duplications	–	–	–	–
	979	1002	1072	1021
Plus applications	12	4	5	62
Plus reinstatements	3	–	2	1
Total	994	1006	1079	1084

Composition

Life Member (Fellow)	12	12	12	12
Life Member (Member)	9	9	9	9
Fellow	21	21	21	21
Member	618	621	645	644
Associate	258	266	311	317
Non Practising	49	50	54	54
Honorary	27	27	27	27
Total	994	1006	1077	1084

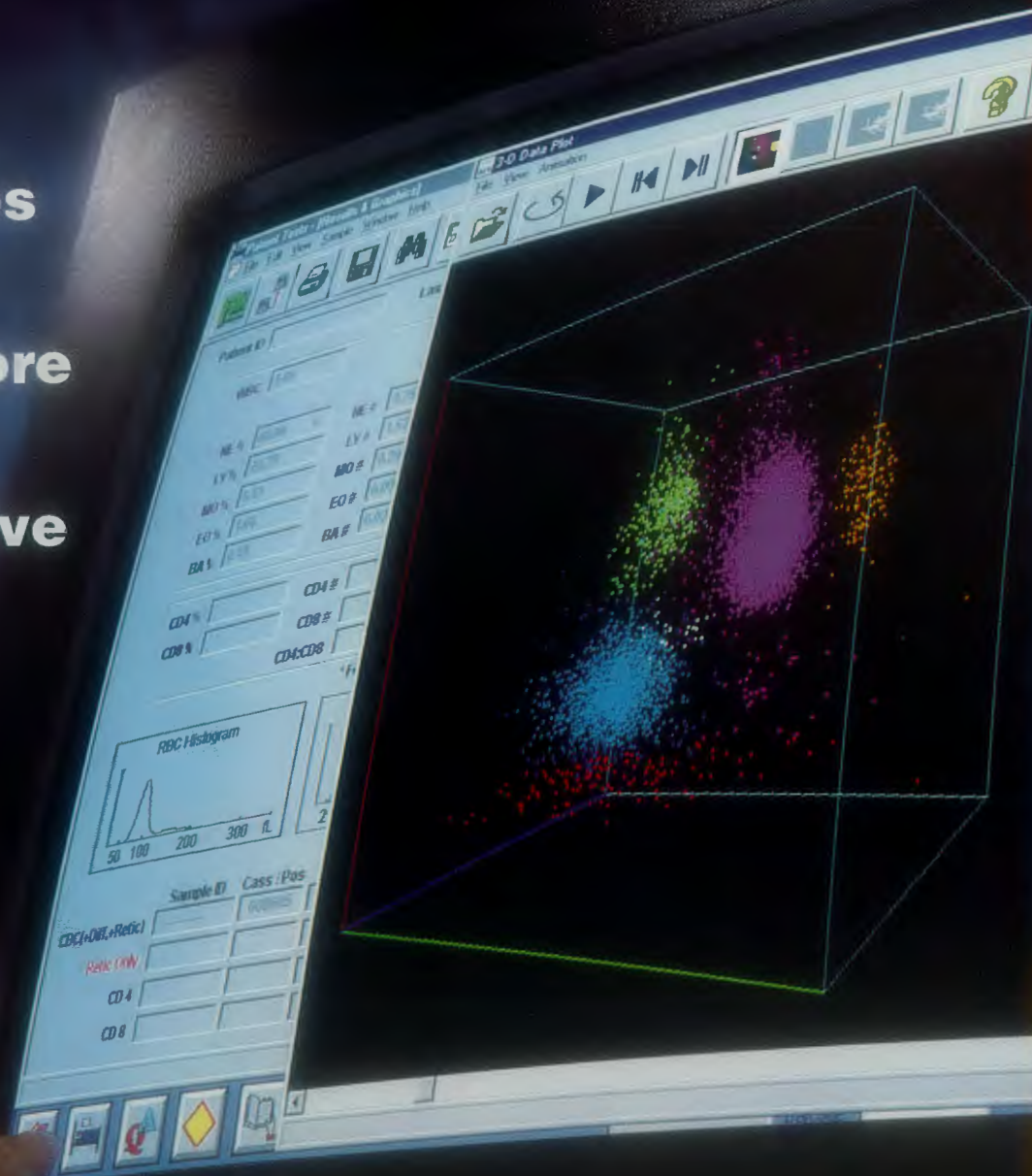
New Members

M. NULSEN, Massey University,
K. COOPER, Diagnostic
S. HACKSHAW, Green Lane

NEW ZEALAND INSTITUTE OF MEDICAL LABORATORY SCIENCE 1997 CALENDAR

March	South Island Seminar – Methven
March/April	Council Meeting – Wellington
30 April	Committee Annual Reports to be with the Executive Officer
30 April	All accounts to National Treasurer for auditing
30 April	Proposed rule changes and remits to be with the Executive Officer
23 May	Applications close for Specialist Certificate examinations
23 May	Applications close for QTA examinations
27 June	Nomination forms for the election of Officers and Remits to be with the Membership (60 days prior to AGM)
1 July	Annual Staffing Survey
8/9/10 July	Fellowship examinations
18 July	Nominations close for election of Officers (40 days prior to AGM)
6 August	Ballot papers to be with the membership (21 days prior to AGM)
13 August	Annual Report and Balance Sheet to be with the membership (14 days prior to AGM)
20 August	Ballot papers and proxies to be with Executive Officer (7 days prior to AGM)
25 August	Council Meeting – Wellington
27 August	AGM – Wellington
26-29 August	Annual Scientific Meeting – Wellington
5 November	QTA examinations
12/13 November	Specialist Certificate examinations
December	Council Meeting

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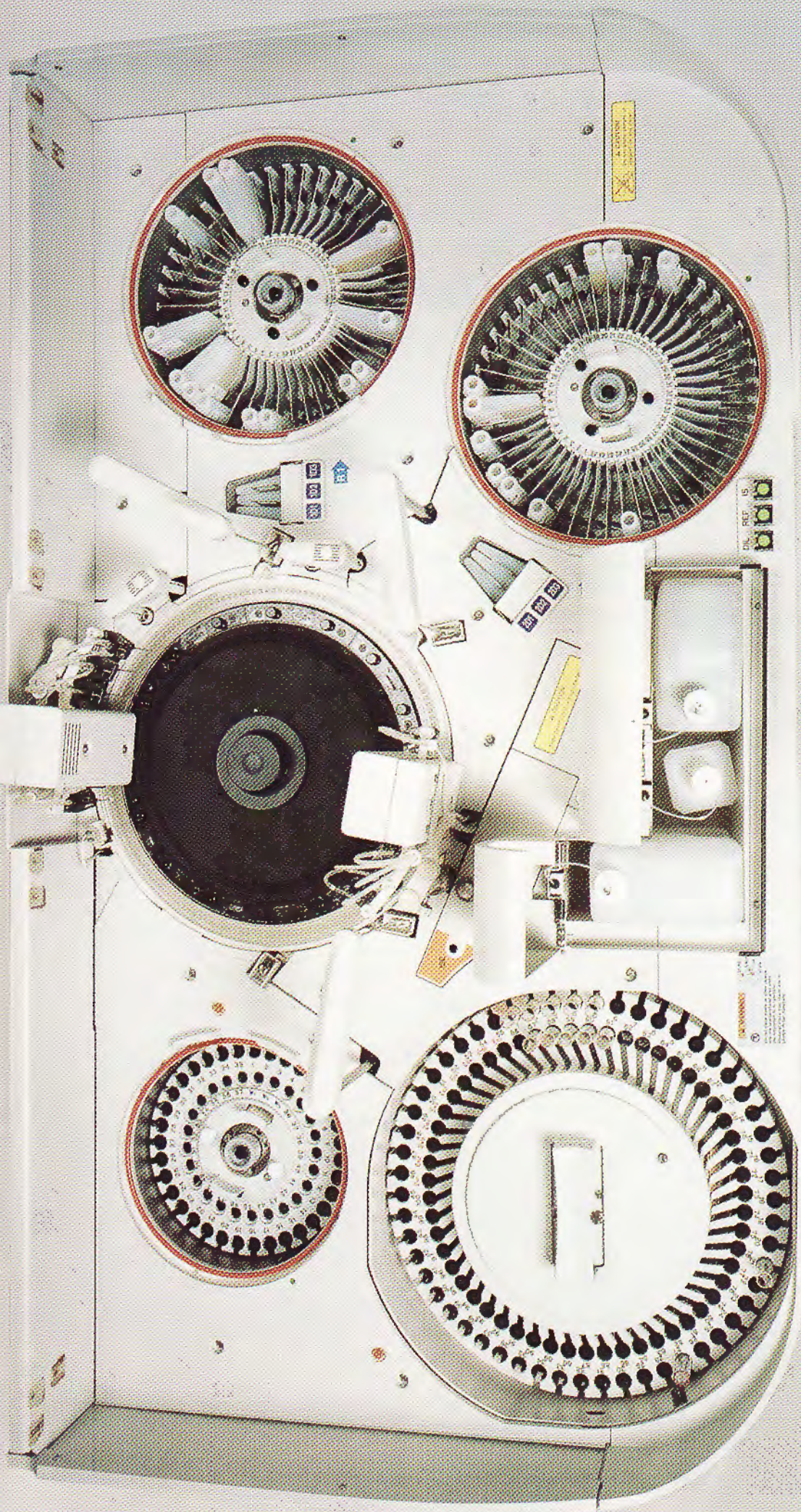
Tel: _____

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The kites of Elecsys® represent three fundamental principles of Boehringer Mannheim's new immunoassay systems.

Novel Chemistry

To overcome the limitations of conventional immunochemistry Elecsys® utilises a novel detection technology called electrochemiluminescence (ECL).

Now you don't have to trade-off sensitivity, measuring range and assay time. For example, while employing 9 or 18 minute incubations ECL delivers TSH sensitivity of <math><0.005</math> mIU/L with an upper measuring range of 100 mIU/L.

Unrivalled Efficiency

Two dimensional barcodes, called Portable Data Files, encode all necessary assay and calibration information to enable rapid and error-free scheduling of tests. Long re-calibration intervals, combined with up to 8 weeks on-board stability of reagents, give you improved equipment utilisation and better operator productivity.

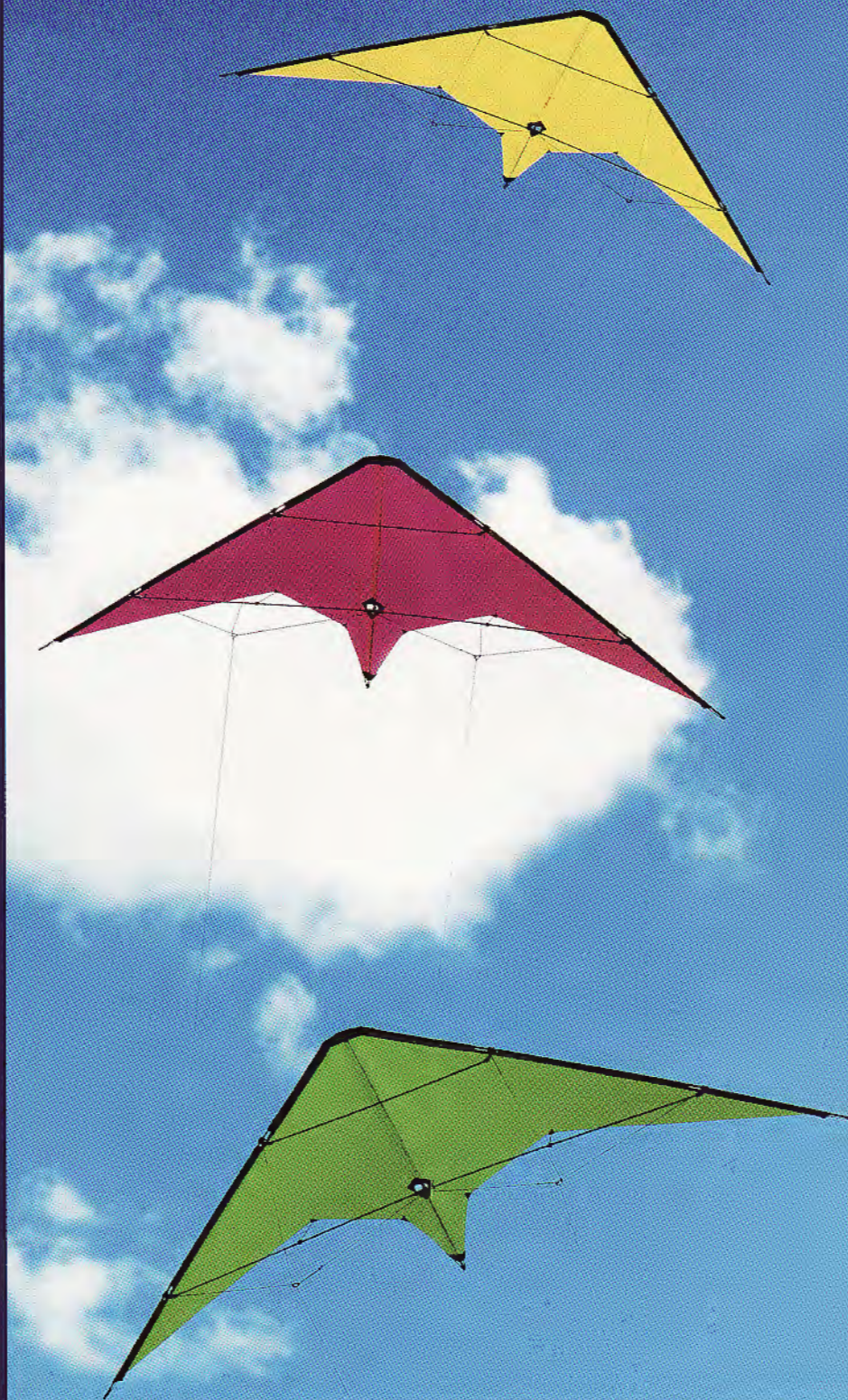
Flexible Organisation

Two Elecsys® analysers, the BM Hitachi Elecsys®2010 and BM Elecsys®1010, have been designed to accommodate widely varying work patterns. To increase work flow multiple units can be linked by sample rack conveyor in combination with a Laboratory System Manager for optimal sample distribution.

STAT samples can be introduced at any time on either analyser, retaining the ability to obtain urgent results when needed.

Boehringer Mannheim's Elecsys® analysers — reaching new heights in immunoassay testing.

Elecsys Reaching new heights



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