Vol. 45 No. 4 November 1991

ISSN 1171-0195

NEW ZEALAND JOURNAL OF

MEDICAL LABORATORY SCIENCE

OFFICIAL PUBLICATION OF THE
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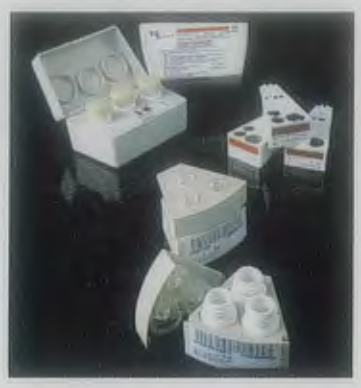

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MEDICAL LABORATORY SCIENCE

VOL. 45 NO. 4 NOVEMBER 1991 ISSN 1171-0195

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From Vol. 36 No. 1 all papers published will be in the form known as "Vancouver Style" or Uniform Requirements for Manuscripts submitted to Biomedical Journals. Full details may be found in the New Zealand Journal of Medical Laboratory Science, Vol. 45, No. 4, page 108 to 111 or from the Editor.

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Original Articles
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Refer to preparation of manuscript for further detailed information.

Preparation of Manuscript

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The title page should contain a concise title of the article. The title generally should not exceed three lines (40 characters per line), including punctuation and spacing. All authors must be identified with first name, middle initial and last name of each author, with highest academic degree(s). The title page should also include the name of the institution with which each author is affiliated and to which the work should be attributed.

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Wherever possible, observational or experimental articles should be divided into sections headed,

* Introduction
* Materials and Methods
* Results
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Long articles may need subheadings within some sections, especially the results and discussion section to clarify their content.

Introduction

Clearly state the purpose of the article. Summarise the rationale for the study or observation. Give only strictly pertinent references, and do not review the subject extensively.

Materials and methods

Describe the selection of the observational or experimental subjects (patients or experimental animals, including controls) clearly. Identify the methods, apparatus and procedures in sufficient detail to allow other workers to reproduce the results. Give references to established

Table 1 Commonly used approved abbreviations

Term	Abbreviation or symbol
Standard units of measurement	
ampere	A
ångström	Å
barn	b
candela	cd
coulomb	C
counts per minute	cpm
counts per second	cps
curie	ci
degree Celsius	°C
disintegrations per mintue	dpm
disintegrations per second	dps
electron volt	eV
equivalent	Eq
farad	F
gauss	G
gram	g
henry	H
hertz	Hz
hour	h
International unit	IU
joule	J
kelvin	K
kilogram	kg
litre	L
metre	m
minute	min
molar	*M*
mole	mol
newton	N
normal (concentration)	*N*
ohm	Ω
osmole	osmol
pascal	Pa
revolutions per minute	rpm
second	s
square centimetre	cm^2
volt	V
watt	W
week	wk
year	yr
Combining prefixes	
tera-	10^{12}) T
giga-	(10^9) G
mega-	(10^6) M
kilo-	(10^3) k
hecto-	(10^2) h
deca-	(10^1) da
deci-	(10^{-1}) d
centi-	(10^{-2}) c
milli-	(10^{-3}) m
micro-	(10^{-6}) μ
nano-	(10^{-9}) n
pico-	(10^{-12}) p
femto-	(10^{-15}) f
atto-	(10^{-18}) a

Term	Abbreviation or symbol
Statistical terms	
correlation coefficient	r
degrees of freedom	df
mean	$\bar{x}$
not significant	NS
number of observations	n
probability	p
standard deviation	SD
standard error of the mean	SEM
"Student's" *t*-test	*t*-test
variance ratio	F
Others	
adenosine diphosphatase	ADPase
adenosine 5'-diphosphate (adenosine diphosphate)	ADP
adenosine 5'-monophosphate (adenosine monophosphate, adenylic acid)	AMP
adenosine triphosphatase	ATPase
adenosine 5'-triphosphate (adenosine triphosphate)	ATP
adrenocorticotropic hormone (adrenocorticotropin)	ACTH
bacille Calmette-Guérin	BCG
basal metabolic rate	BMR
body temperature and pressure, and air saturated with water vapour	BTPS
central nervous system	CNS
coenzyme A	coA
deoxyribonucleic acid (deoxyribonucleate)	DNA
dihydroxyphenethylamine	dopamine
electrocardiogram	ECG
electronencephalogram	EEG
enteric cytopathogenic human orphan (virus)	ECHO
ethyl	Et
ethylenediaminetetraacetate	EDTA
gas-liquid chromatography	GLC
guanosine 5'-monophosphate (guanosine monophosphate, guanylic acid)	GMP
hemoglobin	Hb
logarithm (to base 10; common logarithm)	log
logarithm, natural	ln
methyl	Me
Michaelis constant	K_m
negative logarithm of hydrogen ion activity	pH
partial pressure of carbon dioxide	Pco_2
partial pressure of oxygen	Po_2
per	/
percent	%
radiation (ionizing, absorbed dose)	rad
respiratory quotient	RQ
specific gravity	sp gr
standard atmosphere	atm
standard temperature and pressure	STP
ultraviolet	uv
volume	vol
volume ratio (volume per volume)	vol/vol
weight	wt
weight per volume	wt/vol
weight ratio (weight per weight)	wt/wt

methods, including statistical methods. Adequately describe new or substantially modified methods.

Identify precisely all drugs and chemicals used, including generic name(s), dosage(s) and route(s) of administration. Do not identify patients or hospitals without consent.

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Present results in a logical sequence in the text, tables and illustrations. Do not repeat in the text all the data in the tables or illustrations. Emphasise or summarise only important observations.

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Indicate the new and important aspects of the study and emphasise the conclusions that follow. Do not repeat in detail data given in the Results section. Include in the Discussion the implications of the findings and their limitations and compare the observations to other relevant studies. Link the conclusions with the goals of the study but avoid unqualified statements and conclusions not completely supported by your data. Avoid claiming priority and alluding to work that has not been completed. State new hypotheses when warranted, but clearly label them as such. Recommendations may be included if appropriate.

Acknowledgements

Acknowledge the people who have made substantive contributions to the study. Authors are responsible for obtaining consent from everyone acknowledged by name as readers may infer their endorsement of the data and conclusions.

References

Throughout the body of the manuscript number references consecutively in the order in which they are first mentioned and identify references in text, tables, and legends by arabic numerals in parentheses (eg) (1), (2) or (3, 4 and 5). References cited only in tables or in legends to figures should be numbered in accordance with a sequence established by the first identification in the text of the particular table or illustration.

When citing authors in the text, where there are three or more authors acknowledge only the first author, (eg) Smith et al. (1981) ... Where there are less than three authors the following style should be used, (eg) Smith and Brown (1981).

Cite all references following the format established by the American National Standards Institute (ANSI) in 1977. The adoption of this style of presentation is now internationally accepted by book and journal publishers and by indexing and abstracting services. Authors unfamiliar with this citation format should refer to the list of abbreviations accepted by *Index Medicus* which adopted the ANSI standard in 1980. (A list of these abbreviations appears annually in the January issue of *Index Medicus*.) Authors should comply with the style of the examples given for references at the end of this section. List all authors in list of references.

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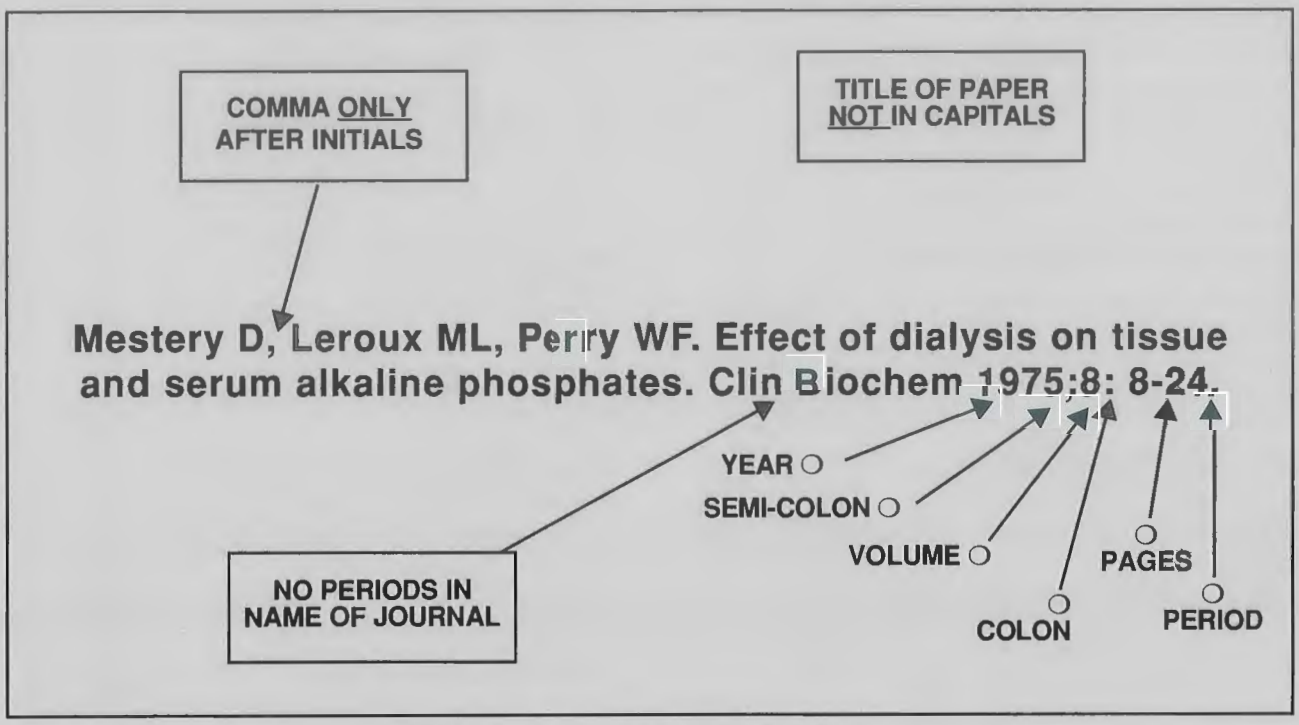

Figure 1 *- Vancouver style for citing references.*

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REVIEW ARTICLE

Effect of Race on Laboratory Parameters. A Review of the New Zealand Data.

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Abstract

A brief review of the literature pertaining to the effect of race upon laboratory parameters in New Zealand is presented. Various biochemical and haematologic test results show radical differences, some of which may be due to weight differences between the Maori and non Maori. Laboratory parameters known to demonstrate racial differences in New Zealand are cholesterol, urate, immunoglobulins, platelets, red cell count, eosinophil count and the ESR. These findings may have implications for laboratory reference ranges.

Key words:

Reference ranges, race, weight, laboratory parameters.

Introduction

When determining reference ranges, laboratories generally take into account factors known to influence test results such as age, sex, physical activity and the prandial state. Until recently little attention has been paid to the possible influence of racial differences thereon. New Zealand is a multiracial society, with the population predominantly made up of Caucasian, Maori and Pacific Island races. The known difference in disease prevalence amongst these races, such as the higher incidence of diabetes and hypertension in Maori, is well known [1], and this can affect various laboratory parameters such as urate [2,3]. The Maori are generally heavier than their Caucasian counterparts [1], and it has been shown that weight per se influences urate [3], and various haematologic parameters [4]. Racial differences in laboratory parameters may therefore exist which may have to be taken into consideration when determining laboratory reference ranges.

Biochemistry

At least 25% of Maori males exhibit hyperuricaemia with serum urate concentrations of >0.44 mmol/L [3,5]. The corresponding incidence is less than 5% in Caucasian males. Weight is positively correlated with serum urate [3] but even when the weight differences between Maori and Caucasian males are accounted for a significant difference in serum urate concentrations between the two races remains. There is a strong genetic component to hyperuricaemia in the Maori [2], predominantly due to increased renal tubular urate reabsorption resulting in decreased renal clearance of urate [5]. Tokelauan males resident in New Zealand also demonstrate higher serum urate levels which are similar to results in Maori males [6].

Prior, has shown that adult Europeans have higher serum cholesterol values than Maori [2], while a subsequent smaller study found no difference in serum cholesterol results between Maori and Caucasian males [3]. Differences in populations studied, body weights, sample size and different analytical methods may have contributed to the discordant results. Adolescent European boys and girls, aged 13-16 years, had higher total serum cholesterol but lower serum triglycerides than their Maori counterparts [7]. Higher serum urate concentrations, as previously noted in adult Maori subjects [2], together with increased weight, were also found in Maori adolescents in this study [7].

Stanhope et al have demonstrated higher levels of serum IgA, IgM and IgG in New Zealand-born Tokelauan children compared to New Zealand-born European children [8]. The increase in serum IgG was negatively correlated with breast feeding in the Tokelauan children, and the increase in immunoglobulins in this group is most likely due to a mix of genetic and environmental factors. In the USA lower serum total bilirubin concentrations were found in Blacks compared to Whites [9], this racial difference remained even in disease states which are known to affect bilirubin metabolism. One New Zealand study examined infants with jaundice but found no difference in the increase in serum bilirubin concentrations between European, Pacific Island and Maori infants [10]. No racial differences in plasma sodium, urea or creatinine were demonstrated between Maori and Caucasian men, but slightly higher plasma potassium concentrations in Maori men were found which just failed to reach statistical significance at the 95% confidence limits [3]. Overseas studies have demonstrated racial differences in serum enzyme activities, such as creatine kinase [11] and both alkaline phosphatase and aspartate aminotransferase [12]. No comparable New Zealand data is available which is worthy of further study.

Haematology

Racial differences in haematologic parameters in New Zealand have been described for the eosinophil count [13,16], ESR [13,14], platelet count [15,16], red cell count [15], and the white cell count [16]. Eosinophilia is higher in Cook Island males (38%) compared to European and Maori males, and to a lesser degree (21%) in Cook Island females [13]. The increased rate of eosinophilia in Cook Islanders was not considered to be due to microfilarial infestation as the number of subjects with eosinophilia was equally divided between those who had recently been to the Cook Islands and those who had not. Another study found no evidence for eosinophilia in pregnant Cook Island females, but eosinophilia was present in a third of pregnant Samoan females, the reason for this was not known [16].

The ESR is also higher in both Cook Island males and females, the next highest values were for Maori, while Caucasians demonstrated the lowest ESRs [13]. Raised ESR in Cook Islanders was not attributable to their eosiniphilia as the number of Cook Island subjects with a raised ESR was not significantly different between those with and those without eosinophilia. The ESR is raised in pregnancy, but Maori and Polynesian pregnant females have higher ESRs than pregnant European females during all three trimesters [14]. Only 8% of all pregnant females with an ESR outside the established reference ranges demonstrated clinical abnormalities (predominantly urinary tract infections) and it was concluded that the ESR is of limited value in the routine evaluation of pregnancy [14].

Both higher platelet and red cell counts have been demonstrated in Maori males compared to Caucasian males [15]. Additionally, an association between weight and both the platelet and red cell count respectively was noted. The platelet and red cell counts remained statistically significantly different between the two racial groups when weight as a confounding variable was accounted for. Subsequently an interrelationship between the circulating blood cells and weight was observed [4] indirectly supporting the concept of a single pluripotent stem cell under control of a haematopoeitic growth factor affecting all three circulating blood cell lines [18].

After 28 weeks of gestation Maori and Cook Islander females tend to have lower haemoglobin and MCV results compared to European [16]. This was hypothesised as most likely due to a higher prevalence of the alpha thalassaemia

trait previously demonstrated in Maori and Cook Islanders [17]. Statistically significant but clinically insignificant differences between races for the total white cell count in pregnant females after 26 weeks of gestation were also found [16]. No comparable information is available for racial differences in haematological parameters in non-pregnant females. Unpublished results from our group demonstrate that Maori females have slightly higher platelet counts ($x = 333 \times 10^9/L$, S.D. = $68 \times 10^9/L$, n = 42) than their Maori males [15] and are slightly higher compared to female Caucasians in New Zealand [19].

Table 1: *Documented racial differences in laboratory parameters*

Biochemistry	Haematology
Total cholesterol [2,3,7]	ESR [13,14]
HDL cholesterol [7]	White cell count [16]
Triglycerides [7]	Eosinophil count [13,16]
Urate [2,3,5,7]	Platelet count [15,16]
IgA, IgM and IgG [8]	Haemoglobin [16]
Potassium [3]	MCV [15,16]
	Pct [15]

Numbers in parentheses refer to references.

Table 2: *Documented differences in laboratory parameters in Maoris and Polynesians compared with Caucasians*

Maori	Polynesian
↓ Total cholesterol	↑ IgA, IgM and IgG (infants)
↓ HDL cholesterol	↑ Urate
↑ Triglycerides	↑ Eosinophils
↑ Urate	↑ ESR
↑ Potassium (?)	
↑ ESR	
↑ Platelet count	
↑ Red cell count	

↓ *decreased* ↑ *increased (compared with Caucasians)*

Conclusions

This brief review is presented to make laboratory scientists aware that racial differences in various laboratory parameters can exist in New Zealand. These differences may be due to genetic factors or they could reflect weight differences between races [1] given the association between weight and various laboratory parameters [3,4]. As intra-individual variability is small, but inter-individual variability can be great such as has been demonstrated for the platelet count [19,20], it could be argued that racial differences in laboratory parameters would be of limited importance when establishing laboratory reference ranges. Certainly for urate it is advocated to use the non-Maori reference range, as urate concentrations of >0.44 mmol/L should be further investigated. For the platelet count which demonstrates a much wider range of normal values, the reported increase in platelet counts of ±20% in Maori men [15], possibly linked with higher body weights in the Maori may well indicate a need for separate reference ranges for the platelet count for Maori and non-Maori subjects. Body weight is a factor which thus may need to be taken into account when determining laboratory reference ranges.

Acknowledgements

The author is supported by the National Heart Foundation of New Zealand, and thanks Mrs H. Bark and Ms M. Gordon for typing this manuscript.

References

1. Pomare EW, de Boer GM. Hauroa: Maori standards of health. Special report series 78; Medical Research Council of New Zealand, 1988.
2. Prior IAM. Cardiovascular epidemiology in New Zealand and the Pacific. *NZ Med J* 1974;**80**:245-52.
3. Siebers RWL, Murphy C, Chisnall W, Maling TJB. Effect of race and weight on plasma urate: implications for laboratory reference ranges. *NZ J Med Lab Technol* 1989;**43**:92-3.
4. Siebers RWL, Carter JM, Wakem PJ, Maling TJB. Interrelationship between platelet count, red cell count, white cell count and weight in men. *Clin Lab Haemat* 1990;**12**:257-62.
5. Gibson T, Waterworth R, Hatfield P, Robinson G, Bremner K. Hyperuricaemia, gout and kidney function in New Zealand Maori men. *Br J Rheum* 1984;**23**:276-82.
6. Prior IAM, Welby TJ, Ostbye T, Salmond CE, Stokes YM. Migration and gout: the Tokelau Island migration study. *B Med J* 1987;**295**:457-61.
7. Stanhope JM, Prior IAM, Malcolm JB. Coronary risk factors in New Zealand Maori and European adolescents: the Rotorua Lakes study 2. *NZ Med J* 1975;**82**:336-9.
8. Stanhope JM, Tonkin SL, Martin TB, Dixon-McIver DN. Serum levels of immunoglobulins A, G and M in New Zealand and Tokelauan children. *NZ Med J* 1977;**86**:126-30.
9. Carmel R, Wong ET, Weiner JM, Johnson CS. Racial differences in serum total bilirubin levels in health and disease. *JAMA* 1985;**253**:3416-8.
10. Dixon-McIver DNM, Sargon SG. Evaluation of the Minolta transcutaneous bilirubin meter as a screening device in a mixed race population. *NZ J Med Lab Technol* 1987;**41**:76-7.
11. Black HR, Quallich H, Gareleck CB. Racial differences in serum creatine kinase levels. *Am J Med* 1986;**81**:479-87.
12. Laskarzewski P, Kelly KA, Mellies MJ et al. Clinical chemistry determinations for a biracial cohort of 1,605 normal and hyperlipidaemic school children aged 6 to 17. *Am J Clin Path* 1980;**74**:371-80.
13. Caradoc-Davies TH, Daniels J. A survey of ESR and eosinophil count in a racially mixed population in New Zealand. *NZ Med J* 1984;**97**:232-4.
14. Brosnan EA, Eales MM. The erythrocyte sedimentation rate (ESR) in pregnancy. *NZ J Med Lab Technol* 1990;**43**:4-6.
15. Siebers RWL, Carter JM, Wakem PJ, Maling TJB. Racial differences in platelet counts in New Zealand men. *NZ Med J* 1989;**102**:588-9.
16. Bluck R, Dixon M, Ramage C, Blacklock H. A reference range for the haematological changes of pregnancy. *NZ J Med Lab Technol* 1990;**44**:103-6.
17. Mickleson KNP, Dixon MW, Hill PJ, et al. Influence of thalassaemia on haematological parameters in Polynesian Patients *NZ Med J* 1985;**98**: 1036-8.
18. Milner PC, Johl S, Martin JF. Platelet count is positively correlated with white cell count and red cell count. *Haemostasis* 1987;**17**:211-6.
19. Carter JM, Siebers RWL, Wakem PJ. Intra-individual variation of platelet indices in pre- and post-menopausal females. *Med Lab Sci* 1991 (in press).
20. Siebers RWL, Wakem PJ, Carter JM. Long-term intra-individual variation of platelet parameters. *Med Lab Sci* 1989;**46**:77-8.

NZMJ Med Lab Science. 1991; **45**(4): 114-116.

The A_1 phenotype and the incidence of the A-type-3/4 epitope in Polynesians

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Abstract

Monoclonal anti-A-type-3/4 was used to investigate whether the A-type-3/4 epitope was the probable basis of the A_1 phenotype seen in Polynesians. Although no biochemical analysis was undertaken, the presence of this epitope on all Polynesian samples reactive with *Dolichos biflorus* strongly suggests it is responsible for the Polynesian A_1 phenotype.

Introduction

Blood group A has been divided into the two major subgroups, A_1 and A_2 since they were first described by von Dungern and Hirszfeld in 1911 (1). For many years there was much debate as to the reason for the differences seen between the A_1 and A_2 phenotypes. Some investigators believed the difference was solely quantitative (2) whereas others believed it had a qualitative basis (3,4). Although the serology and genetics of A_1 and A_2 had been well established (5) and enzymatic studies in 1973 confirmed the likelihood of two distinct glycosyltransferases (6), the probable chemical basis for the distinction between A_1 and A_2 was not resolved until 1985 (7).

The blood group A determinant which consists of the trisaccharide GalNAcα1-3(Fucα1-2) Galß can be carried by one of four distinct types of carbohydrate chains, thus yielding A epitopes with different specificities (Figure 1).

Figure 1

A trisaccharides on four distinct carbohydrate chains

Common Name	Carbohydrate Structure
A-type-1	GalNAcα1-3Galß1-3GlcNAcß- 2 Fucα1
A-type-2	GalNAcα1-3Galß1-4GlcNAcß- 2 Fucα1
A-type-3	GalNAcα1-3Galß1-3GalNAcα- 2 Fucα1
A-type-4	GalNAcα1-3Galß1-3GalNAcß- 2 Fucα1

A-type-1 chain structures are present on erythrocytes in minor quantities, and their expression is dependent on the secretor and Lewis blood group status (8,9). A-type-2 chain structures in contrast, constitute the bulk of the blood group A antigens on erythrocytes with branched and unbranched structures (10,11). The A-type-3 chain structure which is especially abundant in subgroup A_1 erythrocytes is a repetitive structure with an A-type-2 chain extended with the A-type-3 determinant (7). The A-type-4 chain "globo-A" structure is also expressed, although in trace amounts, almost exclusively on A_1 erythrocytes (12).

Essentially the difference between the A_1 and A_2 phenotypes is both quantitative and qualitative. The quantitative difference is as a result of the A_1 transferase being more efficient than the A_2 transferase in transferring N-acetylgalactosamine residues onto H-type-1 and H-type-2 structures (8,13). The qualitative difference arises from the relative inability of the A_2 transferase to catalyse the addition of a N-acetylgalactosamine to the H-type-3 antigen which exists only as an elongation of the A-type-2 antigen (14). The A_1 phenotype is therefore characterised by the repetitive-A antigen, whereas the A_2 phenotype expresses the A-associated-H antigen (see Figure 2) as reviewed in Oriol *et al* (15) and Clausen *et al*, (16).

Figure 2

$A_1$and A_2 phenotype associated structures

Repetitive A-type-3 (A_1-associated structure)

GalNAcα1-3Galß1-3GalNAcα1-3Galß1-4GlcNAcß-
2 2
Fucα1 Fucα1

A-associated-H (A_2-associated structure)

Galß1-3GalNAcα1-3Galß1-4GlcNAcß-
2 2
Fucα1 Fucα1

In Maoris and other Polynesians the frequency of the A_1 phenotype is considerably higher than in Europeans (17,18). Also the serology of the A_1 phenotype in some Polynesians is somewhat atypical with samples having significantly greater *Ulex europaeus* (H antigen) reactivity than Caucasians (19). Furthermore, it has been suggested from serological studies that the molecular conformation of the Polynesian A and H epitopes may differ from that of Caucasians (19) and may also influence the expression of the Lewis phenotypes (20). As the reported incidence of the A_1 phenotype is serologically based on reactivity with lectins and in view of the somewhat atypical A_1 phenotypes of Polynesians, we used anti-A-type-3/4 to establish whether the reported high incidence of the A_1 phenotype was due to expression of the A-type-3/4 epitope.

Materials and Methods

Anticoagulated blood samples were collected from 96 group A or AB Polynesians (including Maori) blood donors during the course of a routine blood donation. All individuals were checked to ensure they had not previously participated in this survey. Samples were also obtained from 18 Polynesian and 47 Caucasian group A or AB adults identified in parentage cases performed at this centre. Group O and B samples from 65 Caucasians and 29 Polynesians were tested in parallel as negative controls, with no unexpected reactions being found.

Phenotyping was performed by observing for agglutination (10 x magnification eyepiece) of washed red cells which had been allowed to incubate and sediment with antibody/lectin for 1 hour at room temperature. Caucasian lectin defined A_1 and A_2 red cells were used as controls for all procedures.

Footnote

Gal = galactose, GalNAc = *N*-acetylgalactosamine, GlcNAc = *N*-acetylglucosamine, Fuc = fucose

Murine monoclonal anti-A, and anti-B reagents (Seraclone, Biotest, WG) were used to establish the ABO group of the samples. A_1 phenotyping was performed with the lectin *Dolichos biflorus* (Biological Laboratories Ltd, Auckland) and with murine monoclonal antibody KB-26.5, a generous gift from Drs Urdaniz and Vinas from Knickerbocker S.A.E. (Barcelona, Spain). KB26.5 reacts with blood group A antigens based on type-3 and type-4 chains (21).

Results

Results for the Polynesian and Caucasian group A and AB samples using KB26.5 and the lectin *D. biflorus* are shown in Table 1. Of the 114 Polynesian samples tested 111 reacted with both *D. biflorus* and KB26.5, giving a concordant frequency for the A_1 phenotype of 97%. However, of the 47 Caucasian samples tested, *D. biflorus* reacted with 38 (81%) and KB26.5 with 39 samples (83%). The one sample reactive only with KB26.5 was group A and strongly reactive. Titration studies (results not shown) indicated the expression of the A-type-3/4 antigen on these cells was approximately half that of control A_1 samples.

Table 1

A_1 phenotyping with monoclonal anti-A-type-3/4 and D. biflorus

	Total	Group		No. Positive		
	n	A	AB	K26.5	*D. biflorus*	$\%A_1$*
Polynesians	114	108	6	111	111	97
Caucasians	47	43	4	39	38	83

*KB26.5 = Monoclonal anti-A-type-3/4 defined.

Discussion

Traditionally the difference between A_1 and A_2 phenotypes have relied on reactivity with the crude lectin extract from *D. biflorus*. The ability of this lectin to discriminate the A_1 and A_2 phenotypes is based on suitable dilution, thereby discriminating the two phenotypes on the basis of their quantitative differences in terminal N-acetylgalactosamine residues. As lectins have broader specificities than antibodies they may also react equally well with other antigenic determinants than the one in which they are being used to define. Such is the case for the lectin *D. biflorus* which although considered to be blood group A specific (22), is found to react much more strongly with the terminal disaccharide unit (αGalNAc(1-3)ßDGalNAc) of the Forssman antigenic determinant (23).

More recently, monoclonal antibodies that recognise A_1 but not A_2 erythrocytes have been reported (7,21). These reagents discriminate between the two red cell phenotypes on the basis of their ability to recognise the A-type-3/4 determinants on A_1 erythrocytes.

We used monoclonal anti-A-type-3/4 (KB26.5) to A_1 phenotype Polynesians in order to establish if the Polynesian A_1 phenotype was due (at least in part) to the presence of the A-type-3/4 epitope. All 111 Polynesian samples reactive with the lectin *D. biflorus* also reacted equally well with KB26.5 and the incidence of the A_1 phenotype found was in agreement with previous reports (17,18).

Of the 47 group A Caucasian samples selected as controls, the incidence of the A_1 phenotype was also in agreement with previous reports (17,18). However, one of the nine samples that was unreactive with *D. biflorus* reacted with KB26.5. Such anomalous results found between *D. biflorus* and KB26.5 have been extensively studied by Prof. Samuelsson's group in Sweden.

They found that monoclonal antibody KB26.5 reacted in most cases strongly with *D. biflorus* reactive samples and only weakly or not at all with unreactive samples. However, the erythrocytes from some A_1 individuals reacted weakly or not at all with KB26.5 while other *D. biflorus* unreactive samples show moderately strong reactivity with KB26.5. These results were interpreted as demonstrating that the expression of type-3/4 chain based A antigens is not an all or nothing phenomenon in A_1 and A_2 individuals (L Rydberg, personal communication).

These discrepancies do however raise the question of what is defined as the "A_1 epitope" or the "A_1 phenotype"? Is it the presence of a particular epitope such as the repetitive A-type-3 structure (with or without A-type-4?) as suggested by Clausen and co-workers or is it merely an increased expression of A as has been traditionally defined by the lectin *D. biflorus*. The answer at this stage is not clear, but with the ever increasing introduction of monoclonal antibodies into routine serology it may become necessary to re-define the A_1/A_2 phenotypes.

Conclusion

We used anti-A-type-3/4 to investigate whether the A_1 phenotype seen in Polynesians had an A-type-3/4 basis or whether it was due to some other *D. biflorus* reactive epitope. Although no biochemical analysis was undertaken, the presence of the epitope on all samples reactive with *D. biflorus* strongly suggests the basis of the A_1 phenotype in Polynesians is due to expression of A-type-3/4.

Acknowledgements

Dr Lennart Rydberg from the Regional Blood Centre, Sahlgrens Hospital, Göteborg and Department of Biochemistry, University of Göteborg, Sweden, is gratefully acknowledged for allowing us to quote his unpublished findings on the reactivity of KB26.5 and *D. biflorus*. Drs Urdaniz and Vinas from Knickerbocker S.A.E. (Barcelona, Spain) are thanked for their generous gift of the monoclonal antibody KB26.5. Thanks are also due to Alison Dent, Katrena Keane and the donor nurses for their help.

References

1. von Dungern E, Hirszfeld L. Über gruppenspezifische Strukturen des Blutes. III. *Z Immunitäsforsch* 1911;**8**:526-562.
2. Mäkela O, Ruoslahti E, Ehnholm C. Subtypes of human ABO blood groups and subtype specific antibodies. *J Immun* 1969;**102**:763-771.
3. Economidou J, Hughes-Jones NC, Gardener B. Quantitative measurements concerning A and B antigen sites. *Vox Sang* 1967;**12**:321-328.
4. Mohn JF, Cunningham RK, Bates JF. In: Human Blood Groups, eds Mohn J, Plunkett R, Cunningham R, Lambert R. Karger, New York 1977, 316-325.
5. Race RR & Sanger R. In: Blood groups in Man, Blackwell Scientific, Oxford, 6th edition 1975.
6. Schachter H, Michaels MA, Tilley CA, Crookston MC, Crookston JH. Qualitative differences in the N-acetyl-*D*-galactosaminyltransferases produced by human A_1 and A_2 genes. *Proc Natl Acad Sci USA*. 1973;**70**:220-224.
7. Clausen H, Levery SB, Nudelman E, Tsuchiya S, Hakomori S. Repetitive A epitope (type 3 chain A) defined by blood group A_1-specific monoclonal antibody TH-1: Chemical basis of qualitative A_1 and A_2 distinction. *Procl Natl Acad Sci USA* 1985; **82**:1199-1203.
8. Watkins WM. Biochemistry and genetics of the ABO, Lewis and P blood group systems; In: Harris, Hirschhorn, Advances in Human Genetics. Plenum Press, New York 1980;**10**:1-136.
9. Clausen H, Levery SB, McKibbin JM, Hakomori S. Blood group A determinants with mono- and difucosyl type 1 chain in human erythrocyte membranes. *Biochemistry* 1985;**24**:3578-3586.

10. Fukuda MN, Hakomori S. Structures of branched blood group A-active glycosphingolipids in human erythrocytes and polymorphism of A- and H-glycolipids in A_1 and A_2 subgroups. *J Biol Chem* 1982;**257**:446-455.
11. Clausen H, Levery SB, Nudelman E, Baldwin M, Hakomori S. Further characterisation of type 2 and type 3 chain blood group A glycosphingolipids from human erythrocyte membranes. *Biochemistry* 1986;**25**:7075-7085.
12. Clausen H, Watanabe K, Kannagi R, Levery SB, Nudelman E, Arao-Tomono Y, Hakomori S. Blood group A glycolipid (A^x) with globo-series structure which is specific for blood group A_1 erythrocytes: One of the chemical bases for A_1 and A_2 distinction. *Biochem Biophys Res Commun* 1984;**124**:523-529.
13. Cartron JP, Badet J, Mulet C, Salmon C. Study of the α-N-acetylgalactosaminyltransferase in sera and red cell membranes of human A subgroups. *J Immunogenet* 1978;**5**:107-166.
14. Clausen H, Holmes E, Hakomori S. Novel blood group H glycolipid antigens exclusively expressed in blood group A and AB erythrocytes (type 3 chain H). II. Differential conversion of different H substrates by A_1 and A_2 enzymes, and type 3 chain H expression in relation to secretor status. *J Biol Chem* 1986;**261**:1388-1392.
15. Oriol R, Le Pendu J, Mollicone R. Genetics of ABO, H Lewis, X and related antigens. *Vox Sang* 1986;**51**:161-171.
16. Clausen H, Hakomori S. ABH and related histo-blood group antigens; Immunochemical differences in carrier isotypes and their distribution. *Vox Sang* 1989;**56**:1-20.
17. Woodfield DG, Simpson LA, Seber GAF, McInerney PJ. Blood groups and other genetic markers in New Zealand Europeans and Maoris. *Ann Hum Biol* 1987;**14**:29-37.
18. Mourant AE, Kopec AC, Domaniewska-Sobczak K. The distribution of the human blood groups and other polymorphisms. Oxford University Press, Oxford 1976.
19. Booth PB & Cartwright L. H, A and A_1 reactivity in two Polynesian groups. *Vox Sang* 1977;**32**:283-289.
20. Henry SM, Simpson LA, Woodfield DG. The Le(a+b+) phenotype in Polynesians. *Hum Hered* 1988;**38**:111-116.
21. Le Pendu J, Lambert F, Samuelsson BE, Breimer ME, Seitz RC, Urdaniz MP, Suesa N, Ratcliffe M, Francois A, Poschmann A, Vinas J, Oriol R. Monoclonal antibodies specific for type 3 and type 4 chain-based blood group determinants: Relationship to the A_1 and A_2 subgroups. *Glycoconj J* 1986;**3**:255-271.
22. Etzler ME, Kabat EA. Purification and characterisation of a lectin (plant hemagglutinin) with blood group A specificity from *Dolichos biflorus*. *Biochemistry* 1970;**9**:869-877.
23. Baker DA, Sugii S, Kabat EA, Ratcliffe RM, Hermentin P, Lemieux RU. Immunochemical studies on the combining sites of forssman hapten reactive hemagglutinins from *Dolichos biflorus, Helix pomatia*, and *Wistaria floribunda*. *Biochemistry* 1983;**22**:2741-2750.

New Zealand Institute of Medical Laboratory Science

Presidential Report August 1991

Paul McLeod

Before preparing this report, I spent some time reading over the presidential reports of recent years. Reference was often made to either impending changes, changes that are upon us or changes that have been made. We were advised to prepare for these changes and to meet the challenges head on. Well, here we are again, in 1991, with another president raising the issue of changes in the health sector, but at least I can take comfort in the fact that I am not breaking with recent tradition in doing so.

I cannot speak for all technologists, but I am certain that I speak for many when I say that we are becoming increasingly intolerant with the continual upheavals of changes occurring throughout the health sector which now is becoming a political football. I feel that the opposing political teams have between them, numerous more tactical moves in their game plans before the final whistle is blown. In the early 1970's we saw the general hospitals and psychiatric hospitals brought together under one management. Next we saw the integration of these hospitals into the primary health sector in defined regions by virtue of the Area Health Boards. There were varying degrees of success with the Area Health Board model but it was obvious that the metropolitan centres were in difficulty applying this principle to their regions. If I can quote from a well known television programme, another "cunning plan" has been presented, this time as a fait a compli to the country. We are again in a position of finding the health sector picked up, shaken around and flung out like a dice on a gaming table. The politicians are hoping for a pair of sixes but we in the industry suspect the outcome is more likely to be the old one-two, once the next cunning plan is conceived. I do not want to argue the pros and cons of the new health sector approach. It is difficult to do so anyway, as it is becoming clear that the new structure is exactly that ... the framework of a building with yet to be constructed walls and rooms. The architects are yet to consider their choice of outside cladding, furniture and colour schemes. Not only have these decisions not been made, but it appears that they have not yet even been considered. What I do wish to comment on is the continual changes in direction that we in the health industry have had to put up with over the last decade and will have to endure into the next.

The health service is essentially a people industry, and this is reflected in salaries and training which cost more than seventy percent of the health vote. The people involved in the numerous professions of health simply want to get on and do the job. Much of the change and turmoil is being instigated on a different level to us, the health care providers. The policy makers, administration and top level management sectors have been coming and going, toing and froing for years, at the whims of their political masters. However, I ask you to take a step back and try to find a space where there is not quite so much dust in amongst the mayhem of change, and look at what is actually going on. You will see nurses bustling about and tending to their patients, you will see doctors in huddled consultation in ward corridors, and the ECG technician pushing her trolley of the latest technology into the lift. The food trolleys rumble along, the physiotherapist is assisting a patient in a walking frame, the X-ray machines are still working and of course, the laboratory continues to be a scene of high activity. In other words, despite the constant change, the health industry is still

actually working and trying to do what they are trained to do — provide health care to the best of their abilities. But I fear that their tolerance of working in such conditions is now rapidly diminishing and we have seen recent evidence of frustrated professionals quitting the public health sector or indeed, the country.

Like politicians, top level management will come and go. However, I am of the firm opinion that regardless of the structural changes, new environments and all the other ways they describe change, at the end of the day the professional health services such as we provide will be wanted, required and demanded by the people of this country. I would be a fool if I thought that we had now reached one hundred percent productivity and efficiency in our laboratories. No doubt we still have room for improvement and this may well show itself in the future by fewer but larger laboratories, but in essence our professional skills are required now and will be demanded even more in the future. I ask you not to be too distracted by the changes constantly occurring and concentrate on your professional skills and service. There are many issues we can concern ourselves with. There is our training and education, our standards and quality and our responsibilities to the people of New Zealand and indeed out into the Pacific basin to provide a top quality service.

The education and training of our technologists is of the utmost importance. Equally, our ongoing education for technologists in the workplace is of high priority. The Council has over the last year focused very closely on our future education requirements and as you are all no doubt aware, has been pursuing with considerable vigour the establishment of the Bachelor in Medical Laboratory Science through the University of Otago.

Like several presidential reports before this one, I cannot announce with absolute certainty that the University of Otago is to accept its first intake of students into year two of the BMLSc next year. However, I can report that Otago have no intention of withdrawing from their plans to do just that. There are still a few hurdles to clear and it would be devastating to the profession if we fell at this late stage. Our efforts to secure the Otago degree have not been helped by an untimely intrusion into our affairs by the Central Institute of Technology with a questionnaire being circulated widely throughout the country. I submit it was untimely because it came after the Needs Analysis Enquiry had been held as to our training requirements. Submissions had been requested some months prior to this, and that was the time when such opinion seeking should have been carried out. The results of the CIT survey are questionable for several reasons which I need not go into in this report, but I view this intrusion into our professional affairs as most regrettable. Despite their apology at a later date, the fact remains that the CIT upset many of our members and indeed added to the confusion on the decision-making processes in regard to the Otago degree. I now regard this whole sorry saga closed and will continue to focus sharply on our goal of achieving the commencement of our degree in 1992.

Another issue I would like to raise is the registration of our profession. There has been comment in the past that the Medical Laboratory Technologists Board's registration, offers little in the way of protection to our profession. I would agree that the MLTB appears to have some difficulty in policing the regulations. However, I would suggest to you that this is not and should not be a major function of the board. Their role is to protect the patient and not necessarily the profession. Recently we have seen the teaching profession lose its registration by having it downgraded to a voluntary status. In effect, this means that anyone can now be employed by school boards of trustees, as teachers. As financial constraints continue to tighten, the trustees will no doubt be looking to contain salary costs or even reduce them. To attract teachers into the classrooms, the temptation to employ lower cost, unregistered teachers will increase. The battle to preserve standards and quality in the teaching profession will now shift to their professional body who will have to take a rear guard approach to maintaining their standards. I would like you to consider this situation being applied on us. The concept worries me very much but confirms my belief that we must be ready to fight for the retention of our registration status, not for our own protection but for the guarantees of standards and quality which registration engenders. It ensures that management are bound to provide quality staff and therefore maintain acceptable standards in patient care.

The Council has had an active and busy year. Our Executive Officer has now been with us for twelve months and the transition to this appointment and the establishment of our office has been very smooth considering the complexity of the change. I wish to thank Fran for her patience, her organisational skills and her capacity to grasp the complex issues involved in organising and running many of the tasks involved with our Institute. I also wish to thank the members of the Council for their help and assistance. There is a lot of work going on behind the scenes which the members are probably not aware of, and this is done without question or complaint. The members are indeed fortunate in having such a dedicated team on their council. But it is not only the Council members who do work for the Institute. Many members willingly give their time as examiners and involve themselves in the Special Interest Groups, arrange workshops, seminars and conferences. I would also like to thank Trish Reilly for all the work she does, because without her efforts I doubt that the Institute could financially support our journal. Thanks goes as well to the businesses who support our profession in so many ways. Your financial support in advertising, sponsorship and prizes is very much appreciated by everyone.

Soon after last year's Annual General Meeting, our Secretary, Barrie Edwards resigned from his position on the Council. A promotion in his career meant that he could not continue to dedicate the time required for the secretarial position. Obviously his departure left some fairly large holes in the administration of the Institute, but as I have already mentioned, the transition to our Executive Officer position has been successful.

We all wish Barrie well and it does not require me to tell you of the contribution he has made to our Institute and profession. However, it was his idea and enthusiasm which contributed much to the concept of the South Pacific Congress and was the driving force behind the inaugural congress in Christchurch nine years ago. We are to see and experience a continuation of this during the next few days.

At this meeting our Vice President, Dennis Dixon-McIver is standing down from office. I am personally sad to see Dennis depart from the executive and his input around the council table will be missed. Dennis has put a great deal of his time and effort into the Institute and I make particular reference to his role as the Editor of our journal. Prior to the formation of our union, Dennis was very involved with our Industrial Relations Committee. He has been very supportive as Vice President and of course, the last year has seen him heading the organising committee for the South Pacific Congress. Dennis, on behalf of the Council and members of the Institute, thank you to you and your family for all the contributions you have made to our Institute.

It is my guess, that the next year is going to be a watershed for our profession. It is going to be an exciting and challenging year. I am ready to meet it, I know the Council too is ready. With the support of the membership I know that we can make all of our plans and goals become a reality.

Minutes of the 47th Annual General Meeting of the New Zealand Institute of Medical Laboratory Science (Inc) Held in Auckland on 27 August 1991 Commencing at 2.00pm.

Present

The President (Mr P McLeod) presided over the attendance of approximately 70 members.

Apologies

It was resolved that apologies be accepted from C Hickton.

D Pees/W Wilson

Proxies

A list of 24 proxy holders representing 65 proxies was read by the Secretary.

Minutes

It was resolved that the Minutes of the 46th Annual General Meeting held on 29 August 1991 be taken as read and confirmed.

M McCarthy/D Pees

Annual Report

It was resolved that the Annual Report be received.

P McLeod/A Paterson

Speakers to the Annual Report were as follows:

M Gillies	Publications Committee
E Norman	Overseas Aid Committee
A Paterson	Education Committee

D Pees brought to the attention of the meeting, the mistake in the figures under 1990/91 in the Membership report. This is to be amended appropriately.

It was resolved that the Annual Report be adopted.

P McLeod/D Pees

Financial Report

It was resolved that the Financial Report be received.

D Reilly/W Wilson

D Reilly spoke to the report.

W Wilson enquired as to the estimated cost of running the Institute's office this year. It is anticipated this cost will be in the vicinity of $15 - 20,000.

R Austin pointed out a mistake in the Financial Report under the examination account. It should read "income over expenditure", not "expenditure over income".

It was resolved that the Financial Report be adopted.

D Reilly/D Dixon-McIver

Election of Officers

The following members of Council were elected unopposed:

President	P McLeod
Vice President	D Reilly
Treasurer	S Gainsford
Region 1 Representative	G Rimmer
Region 2 Representative	E Norman
Region 4 Representative	J Le Grice
Region 5 Representative	A Paterson

Elections were necessary for the position of Secretary and Region 3 Representative.

Election results are as follows:

Secretary	—	S Gainsford	163
	—	K McLoughlin	90

Ms S Gainsford was declared elected.

Region 3 Representative

—	C Kendrick	49
—	R Siebers	26

Mr C Kendrick was declared elected.

It was resolved that all voting papers be destroyed.

D Dixon-McIver/R Austin

Awards

The award winners were announced and the awards presented by the President:

CERTIFICATE EXAMINATION AWARDS

Clinical Biochemistry	Lisa Brennan
Cytology	Ann Gordon
Haematology	Steven Schischka
Histology	Frances Edwards
Immunohaematology	Rebecca Horder
Immunology	Peter Robson
Microbiology	Darren Welch
Nuclear Medicine	Elizabeth Keightley
Virology	Patricia Lush

SPECIALIST CERTIFICATE AWARDS

Cytogenetics	Judy Norrish
Cytology	Andrew McHutchison

QUALIFIED TECHNICAL ASSISTANTS AWARDS

Cytology		Karen Curd
		Erika Skarsholt
Haematology	Joint award	{Sonia Crowley
		{Susan McIntyre
Histology		Dorothy McKane
Immunohaematology		Fiona Ross
Immunology		Nicola Worboys

JOURNAL AWARDS

Hilder Memorial Prize	Jeremy Brett
Roche Diagnostic Microbiology Award	Michael McCarthy

LIFE MEMBERSHIP CERTIFICATE W Wilson

Honoraria

It was resolved that no honoraria be paid.

D Dixon-McIver/E Norman

Auditor

It was resolved that Deloitte, Ross, Tohmatsu be reappointed as the Institute's auditors.

Future Annual Scientific Meeting

The President confirmed that the 1992 Annual Scientific Meeting will be held in Wellington.

The President asked if any centre was interested in hosting the 1993 meeting. K McLoughlin, on behalf of C Hickton, offered Christchurch as the next venue. This was met with acclamation.

There being no further business the meeting closed at 2.40pm.

Minutes of the Special General Meeting of the New Zealand Institute of Medical Laboratory Science (Inc) held at Auckland on 27 August 1991 at 2.40pm.

Chairman

Mr P McLeod

Minutes

It was resolved that the Minutes of the Special General Meeting held on 29 August 1990 be taken as read.

D Dixon-McIver/G Rimmer

Business Arising

Direction to Council to investigate the establishment of National Quality Control programmes to be available within New Zealand had been considered and found not to be a function of the NZIMLS.

Remits

1. It was moved by D Dixon-McIver, seconded by B Edwards that Rule 13(a) be amended to read — "The Officers of the Institute shall consist of a President, a Vice-President, a Secretary/Treasurer and five (5) ordinary members. These shall constitute the Council. All members shall retire annually from office and shall be eligible for re-election".

   D Dixon-McIver stated that this rule change reduces the size of the Council by combining the position of Secretary and Treasurer.

   Carried

2. It was moved by D Dixon-McIver and seconded by W Wilson "that the new Policy Decision No 4 that the Code of Ethics as circulated to all members be adopted by the Institute."

   It was moved by J Le Grice, seconded by A Paterson that General Obligation No. 2 be amended to read "Medical Laboratory Technologists shall practise in accordance with the Law and with the New Zealand Code of Good Laboratory Practice as is currently defined by the National Authority for Quality Assurance Laboratory Testing and Industrial Design (TELARC).

   It was further moved by Allan Johns, seconded by D Pees that "the New Zealand Code of Good Laboratory Practice" be replaced with "the New Zealand Code of Laboratory Management Practice."

   It was moved by D Dixon-McIver, seconded by A Paterson that Policy Decision No. 4 that the Code of Ethics as circulated to all members be accepted subject to all amendments voted on.

   Carried

3. It was moved by S Gainsford, seconded by A Paterson that Policy Decision No. 6 (1979) be reaffirmed.

   Policy Decision No. 6 (1979): That the Council must be informed in advance of national workshops, seminars or similar gatherings which are being conducted under the aegis of NZIMLS branch organisations.

   It was moved by G Rimmer, seconded by J Le Grice that the word "branch organisations" be deleted from Policy Decision No. 6.

   Carried.

The Chairman then called for remits from the floor.

It was moved by J Wright, seconded by K Williams that future NZIMLS Annual and Special General Meetings be scheduled so as not to coincide with workshops or user-group seminars.

It was moved by D Dixon-McIver, seconded by A Paterson that the above motion be amended to "wherever possible".

Carried

It was moved by J Wright, seconded by K Williams that the NZIMLS make a formal response to the "Statement of Health Policy" that advocates the role of the NZIMLS in determining and monitoring standards of practice in all areas of medical laboratory science.

P McLeod told the meeting of the Council's decision to address this matter.

Carried

General Business

D Philip questioned the legality of the election of the positions of the Secretary and Treasurer under the Rules of the NZIMLS.

It was moved by B Edwards, seconded by W Wilson that the Council refer this matter to their Solicitor for interpretation.

Carried

C Watts spoke on the most recent developments regarding the Otago degree course and his promotion tour of the country.

K Williams stated that the NZIMLS has been singly supporting the Otago course and has not extended support to the CIT and ATI.

There being no further business, the Chairman closed the meeting at 3.15pm.

CHAIRMAN

3RD SOUTH PACIFIC CONGRESS ON MEDICAL LABORATORY SCIENCE

AUGUST 26 — 30, 1991
Auckland, New Zealand

ABSTRACTS

SOUTH PACIFIC FORUM

EVOLUTION OF MEDICAL LABORATORY TECHNOLOGY IN DEVELOPING COUNTRIES. SP1

Monica Cheesbrough FIMLS.
Director Tropical Health Technology, 14 Bevills Close, Doddington, March, Cambridgeshire, PE15 OTT, UK

Overview

National health services in developing countries face the difficult challenge of how to resource increasing health demands:

— low GNP
— man-made and natural disasters
— population migration
— malnutrition
— inadequate water supplies and sanitation
— low immunisation coverage
— poor infrastructure and lack of appropriately trained personnel
— unhelpful international policies,

these and other factors have been, and continue to be, powerful selection forces in the evolution of all aspects of health care in developing countries, including medical laboratory technology practice.

Limited resources call for greater care in their allocation and utilisation. Causes of illhealth and ways of promoting total health, need to be clearly defined, managed, and monitored in communities.

What of the past?

In the past, laboratory services in most developing countries have had only a limited impact on the health of most of the population but not just because of under-resourcing. Laboratories were established mainly in hospitals in the cities and in university training hospitals as part of national health policy. Furthermore, laboratory personnel were trained overseas, often inappropriately, or were trained internally following overseas training curricula. Few countries had their own national Association of Medical Laboratory Technology. But this is now changing.

What of the present?

The current pattern of evolution for medical laboratory practice in developing countries is characterised by INDIGENISATION with expectations of a more equitable distribution and accessibility of essential laboratory facilities. The laboratory is becoming important and increasingly used to provide accurate data on the prevalence and incidence of endemic and epidemic disease, emergence of drug resistance, and safety of water supplies. Community health laboratory training programmes are being developed to support district primary health care systems. Existing curricula are being reviewed and indigenised to meet national health policies with technologies that are matched to available resources and working environments. National Associations are being formed to support laboratory workers, set standards, issue certificates of practice to practising personnel, and liaise with Ministry of Health officials. Such Associations are also enabling links to be formed with international professional bodies.

What of the future?

The more indigenous medical laboratory medicine becomes in developing countries:

- The more successful will be the use of financial and human resources in achieving national health targets.
- The more effective laboratory services will become in community health care, rapid investigation of epidemics, control and surveillance of communicable disease and collection of reliable data for health planning.
- The greater will be the care with which laboratories select and apply new technologies.
- The more laboratory services will seek to become self-sufficient by creating the demand for local production and control of reagents and the design and field-testing of equipment that will "live" in developing countries.
- The greater will be the recognition and support of Ministry of Health departments for national training, continuing education, grading and appropriate salary structure for all laboratory personnel.

Summary: Medical laboratory practice is needed more than ever before in health care in developing countries. Every effort should be made to generate indigenous technology laboratories that are accessible, reliable, adequately resourced, and staffed by well trained and motivated personnel, licensed to practise and supported by a national Association of Medical Laboratory Technology. Only then will laboratory practice become integrated fully into national health programmes and provide the scientific basis for cost- and care-effective community health care. In developing countries, laboratory medicine is following a pattern of evolution which is bringing it closer to the patient and the achievement of its professional objectives.

NEW ZEALAND SOCIETY FOR HAEMATOLOGY

SH1

DETECTION AND ANALYSIS OF EBV AND HTLV-1 GENOMES IN HUMAN LYMPHOPROLIFERATIVE DISEASE.

MJ Inglis, LA Williams, DNJ Hart
Haematology Department, Christchurch Hospital, Private Bag, Christchurch, New Zealand.

The Epstein-Barr Virus (EBV) is associated with distinct forms of human lymphoid malignancies, including the endemic (eBL) and sporadic forms of Burkitt's lymphoma (sBL), acquired immunodeficiency syndrome — associated non-Hodgkin lymphoma (AIDS-NHL), Ki-I positive anaplastic large cell lymphoma (Ki-I+ALC) and Hodgkin's disease. Whether EBV or (or retroviruses) have a pathogenetic role in these cellular proliferations, or are passenger viruses, has not been conclusively demonstrated. One element to distinguish between these two possibilities is to determine whether EBV infection has preceded, and thus possibly contributed to, clonal expansion, or whether infection has occurred after clonal expansion and thus is unlikely to contribute to pathogenesis. With this in mind, we have screened 25 patients with large granular lymphoproliferative disease for evidence of EBV and HTLV-1 genomes by the polymerase chain reaction (PCR). Patients recording a positive PCR signal were further analysed by Southern blotting to determine the structure of the heterogeneous genomic termini of EBV as markers of clonal infection.

SH2

FLOW CYTOMETRIC CHARACTERISATION OF LEUKAEMIC CELLS.

Joy Nimmo
Haematology Department, Christchurch Hospital, Private Bag, Christchurch, New Zealand.

Flow cytometry is an automated and quantitative technique for examining cells in suspension. Cells are made to flow in 'single file' through a laser light beam and the interaction of each cell with the light beam is recorded. Cells may be characterised by this method in conjunction with a 'labelling' technique using monoclonal antibodies either directly coupled to a fluorochrome or in a two layer method. This allows detection of specific molecules (antigens) providing information relating to cell lineage, cell maturation and any aberrant features which may be present on malignant cells. This technique is sensitive, comparatively rapid and gives reproducible results. However when using flow cytometry, some crucial technical factors must be appreciated to ensure accurate results. These include (a) the age of the sample, (b) preparation of the cells, (c) the morphological features of the abnormal cells and any residual normal haemopoietic cells present. These factors must be assessed prior to analysis to enable the correct population to be identified on the flow cytometer.

Other important aspects are the selection of an appropriate panel of monoclonal antibodies, the use of controls and a flow cytometer with optimum sensitivity. Having observed all the above criteria the results obtained need to be critically assessed to produce the correct interpretation of the flow cytometric analysis.

SH3

Ph NEGATIVE, M-bcr REARRANGEMENT NEGATIVE, CHRONIC MYELOID LEUKAEMIA : A CASE REPORT.

IM Morison, CJ Newhook, PH Fitzgerald*
Pathology Services, Dunedin Hospital, Dunedin, *Cytogenetic and Molecular Oncology Unit, Christchurch Hospital, Christchurch.

We report a case of chronic myeloid leukaemia (CML) which showed neither the Philadelphia chromosome translocation nor rearrangement of M-bcr. The patient presented at age 77 with low grade fever symptoms, weight loss, malaise, mild splenomegaly and no hepatomegaly. Total leucocyte count was 51 x 10^9/L, the granulocyte morphology was typical for CML (blasts 3%, basophils 1%). Neutrophil alkaline phosphatase score was 25. Marrow biopsy showed hyperplastic and left shifted granulopoiesis (2% blasts, M:E ratio 14:1). A poor quality metaphase showed 47, XX, +8. Thirteen months later the patient was readmitted with shortness of breath, abdominal pain and marked and tender hepatosplenomegaly. Leucocyte count was 90 x 10^9/L (blasts 4%, bsasophils 3%). Over 18 days the leucocyte count rose to 205 x 10^9/L (blasts 5%) and the patient died two days later. Cytogenetic analysis of bone marrow obtained six weeks before death and peripheral blood obtained on the day of death showed 47, XX, +8. The Philadelphia chromosome was not detected. The patient's DNA did not show rearrangement of M-bcr using either the 5′ or 3′ probes.

This case resembles the cases of bcr rearrangement negative CML described by Kurzrock et al (Blood 1990; 75: 445) which were characterised by the absence of a blast crisis and their marked hepatosplenomegaly. It provides further evidence that CML, perhaps with a distinct clinical course, may occur without the associated bcr rearrangement.

SH4

TREATMENT OF ADULT ACUTE LYMPHOBLASTIC LEUKAEMIA: REVIEW OF EXPERIENCE WITH THE AUCKLAND PROTOCOL.

K Fay, AR Varcoe, PJ Browett
For the Auckland Haematology Group, Auckland Hospital, Auckland.

In 1981 the Auckland Group reported improved survival in adults with acute lymphoblastic leukaemia (ALL) treated with combination chemotherapy (vincristine, adriamycin, prednisone) to induce remission, followed by CNS prophylaxis, two drug maintenance and reinduction courses every 6 months. Treatment continued for 30 months. The remission rate was 89%, with predicted 5 year survival 61% [Cancer 48:1931-1935, 1981].

Since this initial report 87 adult patients have been referred between 1979 and 1990 with a diagnosis of ALL. Of these, 65 were treated on the standard Auckland protocol. The mean age of the protocol treated group was 37 (12-67). Immunophenotyping studies were performed in 47 patients of whom, 25 were common ALL, 16 null cell, 2 T cell and 4 mixed leukaemias.

Complete remission (CR) was obtained in 52 patients (80%) with the median time to CR 37 days (11-88). Six patients were refractory to initial induction therapy and 7 died prior to assessment of leukaemic status, 6 of sepsis and 1 post cardiac arrest. Twenty-one patients (32%) remain alive with a mean follow up time of 53 months (1-120). The median survival time was 16 months, with a predicted long term survival of 20%.

Thirty-one patients have relapsed with the median duration of remission 9 months. The majority of relapses occured within 12 months (74%) with only 3 relapses occurring off treatment.

These results fail to confirm the initial success obtained with the Auckland protocol, but are probably not significantly different from other more intensive protocols currently in use.

IS ELECTRON MICROSCOPY HELPFUL? SH5

R Thula, J Nelson
Department of Haematology, Auckland Hospital, Department of Molecular Medicine, University of Auckland School of Medicine.

Opinions vary as to the value of Electron Microscopy (EM) in establishing the cell lineage of an acute leukaemia in cases that are difficult to diagnose by light microscopy. In the last decade cell marker and molecular studies have complemented and possibly superceded cytochemistry and EM studies.

However EM still has a role in classifying difficult cases of acute leukaemias, particularly the application of ultra structural cytochemistry to the diagnosis of megakaryoblastic, mast cell, basophilic and erythroleukaemia, and some cases of granulocytic sarcoma.

Electron microscopy also has a role in the diagnosis and subtyping of the chronic lymphoproliferative disorders eg hairy cell leukaemia, splenic lymphoma with villous lymphocytes, sezary syndrome. Diagnostic features at the ultrastructural level and usefulness of cytochemistry will be discussed with appropriate case illustrations.

ONCOGENES. SH6

Graeme Finlay
Cancer Research Laboratory, Auckland University School of Medicine.

When genes responsible for the control of cell growth are damaged cell growth itself may become abnormal. When the damage to such key genes contributes to the development of cancer, the guilty genes are called oncogenes. They may be damaged in several ways.

1. Oncogenes were first studied as the genes of birds or mammals which had become damaged due to random recombination with retroviral genomes ("v-*oncs*").
2. In human disease, point mutation of the *ras* gene has been shown to be a frequent (and early) occurrence, particularly in adenocarcinoma of the pancreas, lung, and colon, follicular carcinoma of the thyroid and AML. The *fms* gene (which encodes the CSF-1 receptor) also frequently contains point mutations.
3. Gross structural damage can be effected by chromosomal rearrangements. The resulting altered key enzyymes have elevated protein tyrosine-specific kinase activity. This kind of lesion is observed in CML, glioblastomas, and papillary carcinoma of the thyroid.
4. Chromosomal rearrangements can also deregulate gene expression by disrupting their regulatory regions. The *c-myc, bcl-1 and bcl-2* genes suffer this fate when they become translocated into immunoglobulin genes in B cell tumours.
5. Excessive production of key proteins can occur by several mechanisms, of which amplification (an increase in gene copy number) is frequently observed.

SH7

CLONALITY IN HAEMATOLOGICAL MALIGNANCIES — APPLICATION OF X — CHROMOSOME LINKED PROBES.

Jan Nelson and **Peter Browett**
Department of Molecular Medicine, University of Auckland School of Medicine.

Neoplasia represents transformation of a single cell resulting in proliferation of a clone or population of cells with identical genetic features. Within the spectrum of haematological malignances, the study of clonality has given insight into the nature of these disorders and effects of therapy, as well as providing potential diagnostic information.

Clonality in lymphoid malignancies maybe detected by demonstration of light chain restriction or clonal rearrangement of the immunoglobulin or T cell receptor genes at the molecular level. Chromosomal abnormalities eg the Philadelphia chromosome in chronic myeloid leukaemia, provide cytogenetic or molecular markers of clonality in a limited number of cases of myeloid leukaemia but in the majority of these disorders disease specific markers are not present.

The recent availability of X chromosome linked markers potentially provides a more universal means of determining clonality in these disorders, at least in females. Their use is based on the principle of Lyonisation where the random inactivation of one X chromosome in females results in 50% of cells with an active maternal X and 50% an active paternal X chromosome. When a clone is present all the cells will have the same X chromosome active altering the ratio of 1:1 seen in a polyclonal population.

The active or inactive genes can be identified at a molecular level on the basis of differences in methylation patterns. For example in the HPRT system, a BamHI polymorphism can distinguish the paternal and maternal gene in heterozygote females. Digestion with a second methylation sensitive enzyme (eg HhaI or HpaII) determines whether an allele is active or not. Probes for the X-linked genes HPRT and PGK, in combination with the recently described M27β may be informative in up to 90% of females.

THE PHILADELPHIA CHROMOSOME - FROM CYTOGENETICS TO ONCOGENES. SH15

Peter Browett, Sanjay Tiwari and **Neil Van de Water,**
Department of Molecular Medicine, University of Auckland School of Medicine, Auckland.

The Ph chromosome, an acquired cytogenetic abnormality present in over 90% of cases of chronic myeloid leukaemia (CML), results from a reciprocal translocation between the long arms of chromosomes 9 and 22: t(9;22) (q34;q11). The translocation juxtaposes the c-abl gene from chromosome 9q with the breakpoint cluster region (M-bcr) on chromosome 22q, resulting in the formation of a hybrid gene, bcr-abl. The fusion gene bcr-abl encodes a novel protein of 210Kd (p210) which has potent tyrosine kinase activity, and recent evidence suggests is important in leukaemogenesis.

In CML the chromosome 22 breakpoints are localised to a small region of 5.8kb region and can be mapped using Southern blot hybridisation techniques. Patients with Ph negative CML, variant (x;22) or complex translocations (9;x;22) also have bcr rearrangement typical of that seen in Ph positive CML, with expression of the novel p210 protein.

At least 20% of cases of adult ALL have a Ph chromosome which is identical to that seen in CML. At a molecular level, approximately 50% of these cases have bcr rearrangement and express the p210 protein. In the remaining cases, the breakpoint is 5' to bcr on chromosome 22, with generation of a 190 Kd (p190) tyrosine kinase. The molecular alteration does not appear to distinguish de novo Ph positive ALL from CML in lymphoid blast crises.

Molecular analysis of the chromosome 22 breakpoints may be used diagnostically as an adjunct to cytogenetics, and to monitor the leukaemic clone following chemotherapy or bone marrow transplantation. Future directions include application of PCR amplification of bcr-abl in the tracking of minimal residual disease, and assessment of the prognostic significance of the breakpoint site on chromosome 22.

THE TOOLS OF THE NEW GENETICS. SH16

Neil Van de Water
Department of Haematology, Auckland Hospital and Department of Molecular Medicine, School of Medicine, Auckland.

Advances in molecular biology have revolutionised the study of leukaemia. The application of recombinant DNA technology has enabled us to begin to understand the multistep process of carcinogenesis. Basic tools and techniques developed over the last 20 or so years include: nucleic acid hybridisation, ligase enzymes, restriction enzymes, gene cloning, Southern blotting, and more recently, gene amplification using the polymerase chain reaction (PCR). These tools allow us to manipulate DNA in a variety of ways. We can now join, cut, isolate, insert, rearrange, copy, multiply and sequence pieces of DNA. Through the use of these tools it is not only possible to define malignancy as a series of discrete genetic events involving the alteration or rearrangement of specific genes, but also to identify malignant cells and to follow the disease from onset to remission.

SH17

THE IMMUNOGLOBULIN AND T CELL RECEPTOR GENES: APPLICATION TO THE DIAGNOSIS OF LEUKAEMIA.

Peter Browett
Department of Molecular Medicine, University of Auckland School of Medicine, Auckland.

During B lymphocyte development the immunoglobulin (Ig) genes undergo rearrangement as part of the generation of antibody diversity. Similar changes take place in the genes which encode for the chains of the T cell receptor (TCR).

Leukaemias represent clonal expansion of normal, albeit rate, haemopoietic precursors which are frozen at an early stage of either B or T cell development. As a consequence, within the lymphoid malignancies, all the cells will have the same pattern of rearrangement of either the Ig or TCR genes.

Clonal rearrangement of the Ig genes in B cell lymphoproliferative disorders, and of the TCR genes in T cell disorders, may be detected by Southern blotting techniques. In conjunction with morphology, cytochemistry and immunophenotype studies, demonstration of clonal rearrangement of these genes provides important data with respect to diagnosis, subclassification and lineage assignment in cases of acute and chronic lymphoproliferative disorders. Future application will include the use of these techniques to monitor response to therapy, and use of PCR gene amplification of unique rearrangements to detect minimal residual disease.

ADVANCES IN CHEMOTHERAPY OF NON-HODGKIN'S LYMPHOMA (NHL). SH18

M Wolf
Peter MacCallum Cancer Institute, 481 Little Lonsdale Street, Melbourne 3000, Australia.

Results of treatment of advanced-stage aggressive histologic subtypes of NHL have improved over the last 15 years with the development of effective combination chemotherapy regimens. The Southwest Oncology Group achieved complete remission (CR) rates of 53% and long term disease free survival in 30% using the CHOP regimen. Newer regimens such as MACOP-B, COP-BLAM and ProMACE-CytaBOM have produced CR rates of over 80% with long-term survival of 60-70% in single institution studies.

Over the past 16 years the ANZ Lymphoma Group has conducted a series of clinical trials. Since 1986 a prospective randomised study of MACOP-B versus CHOP has been conducted in patients with intermediate grade NHL. To date 199 patients have been randomised of which 142 (76%) have diffuse large cell (DLCL) or immunoblastic lymphoma (IBL). Median follow-up of patients still alive is 32 months. The CR rates were 43% for MACOP-B and 52% for CHOP for the whole patient population ($p = 0.3$). For DLCL and IBL, CR rates were 48% for MACOP-B and 53% for CHOP ($p=0.4$). There was no significant difference in progression free survival or overall survival between the two arms. Haemopoietic toxicity and stomatitis were more severe in the MACOP-B arm whereas nausea and vomiting worse in the CHOP arm. Further advances in the therapy of aggressive NHL may require new approaches such as the use of bone marrow or peripheral stem cell transplantation or colony stimulating factors to support high-dose chemotherapy programs.

LMW HEPARINOID IN THROMBOPROPHYLAXIS FOR CANCER SURGERY. SH19

J. Cade, A. Gallus, P. Ockelford
A.N.Z. Study Group.

Org 10172 is an antithrombotic low molecular weight heparinoid with a better safety margin than heparin in animal studies. In a double blinded randomised controlled trial Org 10172 (Lomoparan) has been compared with heparin in the prevention of venous thrombosis in general surgery for cancer. Org 10172 (750 antiXa units BD) and Na heparin (5,000 u sc BD) were the trial regimens, patients were 40 years and I ^{125}fibrinogen leg scanning was used for detection of operative thromboses. There were 514 patients randomised and analysis was both by 'intention to treat' and 'efficacy'. The DVT rate was 6.8% for Org 10172 and 10.7% for heparin (0.1 p 0.2) with bleeding 4.4% and 3.6% respectively. Transfusion rates were similar and mortality the same in both groups. Fatal bleeding was 0.4% for each of the treatments. The apparent trend towards improved prophylaxis with Org 10172 did not reach statistical significance. We conclude that Org 10172 has similar efficacy and safety in thromboprophylaxis in patients having elective surgery for suspected malignancy.

CURRENT CONCEPTS IN CHRONIC LYMPHATIC LEUKAEMIA. SH20

DNJ Hart
Haematology Department, Christchurch Hospital, Private Bag, Christchurch, New Zealand

Modern techniques for analysing cells have considerably improved our understanding of the well differentiated lymphoid malignancies. Chronic lymphatic leukaemia (CLL) in its typical form has a CD5 positive, FMC-7 negative, weak smIg phenotype. The relationship of these cells to the small normal population of CD5 positive B cells and autoimmune paraphenomena is uncertain. 'Atypical' forms of CLL which are often but not always morphologically more variable than 'typical' CLL can be identified by FMC-7, CD22 and strong smIg reactivity, suggesting a follicular origin to the disease. Lymphoid malignancies of late B cell, FMC-7 positive phenotype include prolymphocytic leukaemia, prolymphocytoid change in CLL and hairy cell leukaemia. The t (11;14) translocation involving the IgH locus on chromosome 14 and bcl-I on chromosome 11q13 is observed in many cases of CLL and t (14;18) (q32;21) involving the IgH locus and bcl-2 on chromosome 18 is often found in follicular neoplasms. Survival and or growth of these cells may be controlled by various gene products. Rare cases of CD4 positive T cell CLL are described but the other most common lymphoid proliferation involving mature lymphoid cells involves the large granular lymphocyte lineage. These cells often demonstrate NK and ADCC activity and express one or more of the CD16, CD56 and CD57 NK associated antigens. Approximately two thirds of the cases are CD3 positive and have clonally rearranged T cell receptor genes. Occasional cases appear to be associated with Epstein-Barr virus infections.

SH21

DIFFERENTIATION INDUCING AGENTS: MODULATION OF THE GROWTH AND DEVELOPMENT OF LEUKAEMIC CELLS.

Peter Browett
Department of Molecular Medicine, University of Auckland School of Medicine, Auckland.

Traditional therapy of acute myeloid leukaemia has focussed on elimination of the malignant clone with cytotoxic drugs. An alternative model is to induce further differentiation of leukaemic cells. These mature and functional cells lack proliferative capacity and are subsequently replaced by normal haemopoietic cells. A number of agents have some differentiation activity in vitro, including the retinoids, interferon-alpha, vitamin D_3 metabolites and cytosine arabinoside. Clinical use of these agents has to date been predominantly restricted to the myelodysplastic syndromes and results have generally failed to fulfil the early promise of in vitro studies. Retinoic acid (RA), the active metabolite of vitamin A, is a potent inducer of myeloid differentiation, both in the promyelocytic cell line HL60 and leukaemic cells from patients with acute promyelocytic leukaemia (APL). Both all-trans RA and 13-cis RA are effective in vitro and at concentrations obtainable in humans. All-trans RA, however, is effective at a significantly lower concentration than 13-cis RA. Early case reports documented the potential value of 13-cis RA in patients with relapsed or refractory APL. Recent studies have now reported high remission rates in de novo and relapsed cases of APL treated with all-trans RA. Response appears to be associated with morphological and molecular evidence of leukaemic cell differentiation. Many cytokines (IFN-γ, IL-1α. IL-1β, IL-2, IL-4, IFN-α, G-CSF) also have growth modulating activities. The addition of cytokines to RA substantially potentiates RA induced differentiation, although patterns vary with different combinations of cytokines and different cell lines. Future trials might address the question of which retinoid with which cytokine is optimal for a given disorder.

TUMOUR SUPPRESSOR GENES AND LEUKAEMIA. SH22

Gordon Royle
Department of Haematology, North Shore Hospital, Private Bag, Takapuna, Auckland, New Zealand.

Studies of chromosomal regions where loss of heterozygosity occurs in human malignancies have led to the localization of a number of genes the inactivation or deletion of which is crucial for the development of particular types of tumours.

These are termed tumour suppressor genes ('anti-oncogenes'), implying that the presence of the normal gene is necessary to prevent development of particular malignancies. Specific regions are associated with specific types of tumours, and it has been shown that a number of different chromosomal regions may be involved in the development of any one given tumour. In myeloid leukaemias, a putative tumour suppressor gene is the p53 gene on the short arm of chromosome 17. Rearrangements of one allele of the p53 gene has been reported in chronic-phase CML, and of both alleles in blast crisis. In vitro studies suggest that this gene suppresses the function of other oncogenes. This presentation will discuss the concepts involved, recent developments in this field and the implications for our understanding of the biology of malignancy in humans.

THE RE-EMERGENCE OF CYTOGENETICS. SH23

Gordon Royle
Department of Haematology, North Shore Hospital, Private Bag, Takapuna, Auckland, New Zealand.

There has recently been a resurgence of interest in cytogenetics with the discovery from cytogenetic studies, often in conjunction with molecular genetic studies, of new clues to tumorigenesis. Investigation of translocations in myeloid leukaemias and in T and B cell lymphomas have demonstrated the effect on proto-oncogenes of transposition within the genome, and have led to the discovery of new oncogenes, not accessible by other means. The observation of non-random chromosome deletions in various malignancies has aided in the elucidation of another class of oncogene, called tumour suppressor genes. Chromosomal studies have helped demonstrate the clonal nature of most neoplasms and the sequential somatic genetic changes within the neoplastic clone. Finally, cytogenetic studies have demonstrated residual malignant disease following chemotherapy or bone marrow transplantation.

MANIPULATING BLOOD CELL GROWTH. SH24

Kathryn E. Crosier
Department of Molecular Medicine, School of Medicine, University of Auckland, Park Road, Auckland.

The cloning of haemopoietic growth factors, their respective cell curface receptors, and DNA-binding proteins that activate the transcription of genes important in regulating haemopoietic development have provided important tools with which blood cells can be manipulated in vitro. In addition, laboratory cell culture techniques have defined conditions in which the growth of normal bone marrow progenitors is favoured over that of leukaemic cells. These developments have paved the way for new approaches to the management of leukaemia, and we are beginning to witness the results of novel therapies based on these findings in clinical trials.

Several recently identified haemopoietic regulators possess properties that are likely to be important therapeutically. The ligand for the receptor encoded by c-kit has activity on haemopoietic stem cells, and the cytokines interleukin 6, interleukin 11 and leukaemia inhibitory factor promote megakaryocyte development. Macrophage inflammatory protein 1α maintains stem cells in the quiescient phase of the cell cycle and offers the prospect of a therapeutic agent that will protect normal blood stem cells from the effects of chemotherapy and irradiation. These proteins are now being studied in vivo in animal models and initial results offer exciting prospects for clinical development.

Our ability to regulate the activity and number of blood cells at varying stages of maturation establishes unique therapeutic opportunities in the management of leukaemia.

THE FAB CLASSIFICATION. SH25

John Matthews
Department of Haematology, Auckland Hospital, Auckland, New Zealand.

The clinical entity of acute leukaemia was probably first described by Friedriech in 1857 and the actual term first used by Ebstein in 1889. It was, however, the work of Erlich and descriptions by Naegli that led to morphological classification.

Over the next 75 years attempts were made to correlate clinical behaviour with morphological appearance and more latterly with therapeutic response and prognosis. Various classificaitons were proposed and applied but it was really only in 1976 with the original proposal of the FAB classification that some degree of uniformity was established, allowing a better assessment of therapeutic modalities. Since this time classification has become more complex, utilising a combination of morphology, chromosomal abnormalities and surface marker patterns. The FAB classification, although pragmatic in origin, is still the standard for making initial therapeutic decisions in many units. Some morphological entities are now described which correlate with prognostically favourable and unfavourable chromosomal patterns (T8:21, inversion 16 aneuploidy, T15:17,-7, +8, del 5). The expansion of the original classification and fuller subdivision of the myelodysplastic syndromes (MDS) as described in 1985 means that only 2% of adults and 1% of children wll fail to be categorised by the FAB classification.

With recent advances in management additional information provided by surface marker, chromosomal and oncogene studies is now necessary to make appropriate therapeutic decisions in a significant percentage of patients.

MISMATCHED BONE MARROW TRANSPLANT AND MATCHED UNRELATED TRANSPLANT. SH26

Stephen J Palmer
Director, Bone Marrow Transplant Unit, Auckland Hospital, Auckland, New Zealand.

Bone Marrow Transplantation is an established therapy for a number of haematological disorders. However, only a minority of patients in the Western World have an HLA compatible sibling..

For the patient without an HLA compatible sibling donor the alternatives include a partially matched transplant, an autologous transplant and a matched unrelated transplant.

This presentation will review the results of partially matched sibling transplants and matched unrelated transplants.

SH27

CELLULAR PROTEIN PROFILES OF THE HODGKIN'S DISEASE CELL LINES SUGGEST A UNIQUE HAEMOPOIETIC (DENDRITIC CELL?) ORIGIN.

Barry D Hock, Derek NJ Hart
Immunology Department, Christchurch Hospital, Private Bag, Christchurch, New Zealand.

The origin of the mononuclear Hodgkin's cell and the Reed-Sternberg cell in Hodgkin's disease (HD) remains controversial. Immunohistological analysis of HD lymph nodes and phenotypic and functional analysis of the HD cell lines, L428, KHM2 and HDLM2 suggests that these cells may be related to dendritic cells. We therefore used SDS-PAGE to analyse the NP40 solubilised

proteins from these three HD cell lines and compared them with the other haemopoietic cell types. The HD cell lines were more readily distinguished from the myeloid and to a lesser extent the lymphoid cell lines by silver staining, but HD cell line specific proteins (13,19,36,60,150kD) were detected only on one line, L428. Iodination of cell membrane molecules, SDS-PAGE and subsequent autoradiography revealed three molecules, (118,22,12 kD) which were restricted to the HD cell lines and U937. Molecules unique to HDLM2 (211 kD and L428 (46 kD) were also detected by this method. Cell surface labelling with NaB^3H_4 identified a glycoprotein of 102kD limited to HDLM2 and L428, as well as a glycoprotein of 97 kD present on KMH2 alone and one of 63kD on L428 alone. Overall the HD cell line protein profiles showed little similarity to the patterns of the other cell types studied. This information provides further evidence to support the view that Hodgkin's cells are a unique cell type, possible of dendritic cell origin. The molecules identified as HD cell line restricted may have potential as markers for this cell type.

SH28

EFFECT OF ALPHA INTERFERON ON AUTOCRINE GROWTH FACTOR LOOPS IN B-LYMPHOPROLIFERATIVE DISORDERS.

HE Heslop, JE Reittie, ACM Bianchi, FT Cordingly, AV Hoffbrand, MK Brenner.
Division of Bone Marrow Transplantation, Department of Haematology/Oncology, St Jude Childrens Research Hospital, Memphis TN. and Department of Haematology, Royal Free Hospital School of Medicine, London UK.

Autocrine production of growth factors to which a malignant cell expresses receptors is a mechanism of tumor growth that may operate in several haematological malignancies. We and others have shown that the B lymphoproliferative disorders B-CLL and hairy cell leukaemia (HCL) produce a number of growth factors including TNF, IL6 and IL1, all of which may induce autocrine feedback loops. If such malignancies depend on these autocrine growth loops for survival interruption may be therapeutically valuable. Alpha interferon (αIFN) has been shown to block TNF or BCDF induced proliferation of HCL or B-CLL cells *in vitro* and has activity in these diseases *in vivo*. Incubation of purified CLL or HCL cells *in vitro* with αIFN inhibits the expression of TNF, IL1 and IL6 mRNA normally induced by culture with TNF protein. There is also a fall in serum TNF and IL6 levels in patients with HCL during αIFN therapy. Therefore αIFN may mediate its therapeutic effects in HCL and B-CCL by blocking autocrine growth factor loops. The antiproliferative effect of αIFN may potentially be augmented by the administration of other agents antagonising growth factor action such as antibodies to cytokine proteins or receptors. To investigate this possibility we have examined the effect of anti-TNF antibody in a Phase I-II trial in patients with HCL or B-CLL.

SH29

AUTOLOGOUS BMT FOR LYMPHOMA.

S.E. Kinsey and **A.H. Goldstone**
Department of Haematology, University College Hospital, London.

The role of marrow transplant in lymphoma is already changing. Undoubted cures occur following high dose therapy and autologous bone marrow transplantation for relapsed high and intermediate grade lymphoma still responding in some way to conventional salvage therapy, but ABMT for refractory or resistant NHL now appears contraindicated without a new approach. First remission ABMT for poor prognosis NHL is becoming more fashionable and appears safe but such cases are difficult to identify and it will require many additional transplants to produce few extra cures. Allogenic transplant for NHL does not appear superior to ABMT for the vast majority of patients but may have something to offer for the very young recipient in whom toxicity is low, or the very occasional patient with excellent performance status and minimal disease save modest marrow involvement. The low grade lymphoma data for ABMT still appear unclear but the ability to detect very minimal disease by PCR for the bcl-2 oncogene may be a significant advance. In Hodgkin's disease amongst relapsed patients, even refractory patients may sometimes be converted by ABMT to CR; Hodgkin's disease remains a prime indication for ABMT.

SH30

MANAGEMENT OF CHRONIC LYMPHATIC LEUKAEMIA (CLL).

M.E.J. Beard
Department of Haematology, Christchurch Hospital.

Treatment for CLL has not substantially improved survival although control of disease without excessive toxicity has improved the quality of life. Because many patients with CLL are elderly a conflict often exists between what can be done and what should be recommended.

Chlorambucil in a range of schedules remains the most commonly used therapy when such is required but has not proven helpful in low risk disease. More intensive treatment for progressive disease has usually involved the addition of an anthracycline as in one of the CHOP schedules, but again improved survival has not been consistently demonstrated.

The newer forms of treatment Epirubicin, fludarabine, 2-chlorodeoxyadenosine, 2 deoxycoformycin and interferon alone or in combination with established drugs are now under evaluation and the current results will be reported. Monoclonal antibodies reactive with leukaemic CLL B cells are also being used in CLL treatment.I The younger patient (under 50 or so) presents special problems in management and warrants consideration for potentially curative treatment and the role of bone marrow transplantation in CLL will be reviewed.

Reference 5th International Workshop in CLL : Sitges (Barcelona) April 26-28 1991.

SH31

BLEEDING TIMES AND D-DIMER LEVELS IN GPH.

Dr Gordon Royle and **Dr Christopher Barry**
Department of Haematology, North Shore Hospital, Private Bag, Takapuna, Auckland, New Zealand.

D-dimer levels and bleeding times have been prospectively studied in 35 pregnant women with gestational proteinuric hypertension (GPH). Forty-four women with GPH and 55 healthy pregnant controls were included in the study. Fourteen of the normal pregnant controls had mildly elevated D-dimers; none of these developed GPH. Eighteen (41%) cases of GPH were severe. D-dimer levels in the women with GPH did not differ significantly from those of the normal pregnant women. We found no correlation between D-dimer levels in GPH and any adverse outcome, nor between dimer levels and overall severity of GPH. Bleeding times were measured in 27 of the 44 women with GPH. All were within normal limits. These results suggest that D-dimers in normal pregnant women do not have a useful positive or negative predictive value for GPH *per se* nor do they appear to predict an adverse outcome when elevated in GPH, in this population.

BIOCHEMISTRY

CONTROL OF CELL GROWTH AND ITS DEREGULATION IN NEOPLASTIC DISEASE. BC1

GJ Finlay
Cancer Research Laboratory, Auckland University School of Medicine.

Cells become cancerous when growth-stimulating regulatory mechanisms are abnormally activated and when growth-restraining mechanisms are inactivated. Multiple genetic changes including both types of abnormality have been implicated in the pathogenesis of most human cancers.

Over the last decade, many oncogenes have been identified and shown to be damaged genes responsible for the generation of inappropriate signals which drive cell proliferation. The protein products of such oncogenes operate at different steps of the biochemical pathway(s) which transmit mitogenic signals from the cell surface, through the membrane across the cytoplasm and into the nucleus where they include DNA-binding proteins which control gene expression.

More recently, tumour-suppressor genes have been implicated in many human cancers. The functional inactivation of such genes (by point mutations, DNA rearrangements and deletions, and chromosome loss) allows cells to escape growth control. Several tumour-suppressor genes have been cloned including those implicated in retinoblastoma, nephroblastoma, neurofibromatosis and many types of carcinoma. Colon carcinomas show loss of at least one of the genes identified on chromosome 5q (the FAP locus), 17p (the p53 gene, mutated also in most lung, breast and bladder tumours) and 18q (which encodes a putative cell adhesion molecule). Like oncogenes, tumour suppressor genes encode proteins which control signal transduction, ultimately regulating expression of the genetic programme.

THE SCIENTIFIC BASIS OF CANCER CHEMOTHERAPY. BC2

Bruce C Baguley
Cancer Research Laboratory, University of Auckland Medical School, Auckland, New Zealand.

The majority of molecular events now identified as being associated with the development of cancer are concerned with the control of signalling pathways for cell proliferation. The process of DNA replication and cell division involves the cell-cycle specific induction of various enzymes and other proteins. A number of these are targets for exploitation by anticancer drugs. Examples of targets are DNA polymerase and topoisomerases, which are essential for chromosome duplication as well as enzymes which synthesise the component units of DNA and proteins involved in cell division. Susceptibility of cancer cells to these drugs generally (but not always) means that the cancer cells have more of a target enzyme than the host cells. Conversely, cancer cells losing a target enzyme or resisting drug entry into the cytoplasm will become resistant to treatment. Developing methods of testing cancer cells (particularly from patient tumour biopsy material) for drug sensitivity presents one of the most challenging problems for medical technology in the future.

THE MANAGEMENT OF PATIENTS WITH CANCER IN NEW ZEALAND. BC3

V Harvey
Department Clinical Oncology, Auckland Hospital

Although cancer is responsible for 25% of all deaths in New Zealand and other "Western" countries, it is probably the most treatable and certainly the most curable of chronic conditions. Treatment options include: surgery (responsible for 20 to 30% of all cures), radiotherapy (10 to 15% of cures) and cytotoxic chemotherapy (6 to 10% of cures). Hormonal manipulation is effective in disease palliation in hormonally responsive cancers and immunotherapy is a treatment of considerable potential actively being investigated.

Before therapy can be decided a firm diagnosis must be established (preferably pathologically) and the extent of dissemination (if any) discovered. Whether therapy is undertaken with curative or palliative intent will be determined by the precise diagnosis and disease extent. Whilst curative therapy is generally to be preferred where possible, a realistic approach when this is not possible may prevent excessive toxicity from treatment.

Surgery is the longest established and most widely used treatment modality. Its advantages include a single procedure for diagnosis and treatment in many cases and rapid results. The major disadvantage is the local nature of treatment and secondarily the need to remove the affected part. Radiation treatment, using either man made x-rays or natural radioactive sources, may often replace surgery when tissue removal would lead to excessive morbidity (e.g. head and neck cancer especially laryngeal cancer) but has the similar disadvantage of being a localized treatment. In addition it may require prolonged treatment courses extending daily for several weeks. Radiation is increasingly used in conjunction with surgery to limit the mutilation of surgery without compromising the results e.g. in breast cancer, bladder cancer and anal cancer.

Drug therapy (cytotoxic chemotherapy) is the youngest of the three established methods of cancer treatment. It was developed during and after the 2nd World War but only became widely applied in the last 25 years. The major advantage of chemotherapy is the systemic nature of the treatment but disadvantages include the greater exposure of normal tissues with consequent side effects, the prolonged treatment courses often over several months and the limited effectiveness of many of our drugs. Chemotherapy is indicated as primary treatment for generalised cancers, e.g. leukaemia, lymphoma, gestational choriocarcinoma and germ cell tumours but may also enhance the cure rates of diseases treated primarily with surgery, e.g. childhood tumours, ovarian cancer and the adjuvant therapy of breast cancer, osteosarcoma and possibly colorectal cancers.

When cure is not possible, anti-cancer therapy may still provide effective symptom control and prolong survival in many cancers. In palliative therapy the balance between symptoms of disease and side effects of treatment must be carefully considered before and during therapy to obtain optimal quality of life. The wishes of the informed patient are vital in determining the appropriate therapy for the individual.

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
N.Z.I.M.L.S. Membership Application Form

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:—

The Executive Assistant
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
Nuclear Medicine
Cytogenetics
Microbiology
Immunohaematology (Transfusion Science)
Medical Cytology
Immunology
Virology

PREREQUISITES

1. Candidates for the examination must have passed a Certificate Examination offered by the Medical Laboratory Technologists' Board or be granted an exemption by the Council of the NZIMLS.

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, P.O. Box 3270, Christchurch. A charge of $15 (GST incl) is made for each syllabus.

EXAMINATIONS

1. The examinations will be held annually 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.

NEW ZEALAND INSTITUTE OF MEDICAL LABORATORY SCIENCE

Application to sit Specialist Certificate Examination
11th and 12th November 1992

SECTION A — TO BE COMPLETED BY THE CANDIDATE

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

Laboratory .....

Laboratory Address .....

.....

Examination Subject .....

Medical Laboratory Technologist Board Certificate Examinations passed:

Subject ..... Year Sat .....

Subject ..... Year Sat .....

EXAMINATION FEE: $450 (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 29 MAY, 1992

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 $88.40 (GST incl.) ASSOCIATE $33.80 (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.

When effective therapy is exhausted, many patients are interested in experimental therapy. New cytotoxic drugs must be tested in such patients because their potential side effects preclude their testing in human volunteers (where most other new therapies are tested). Immunotherapy which uses various means of enhancing the body's own defence systems has long been an area of great promise but with little substance to date. The burgeoning basic knowledge in this area in the last several years and the ability to produce recombinant versions of natural substances in commercial quanitities will finally allow such therapy to be rationally tested.

Improved communication worldwide and the scientific focus of modern medicine have allowed the introduction of many revolutionary treatments and the dismissal of other worthless therapies. Public pressure to discard the scientific basis of medicine in favour of "greener" alternative methods must be vigorously resisted. However good scientific medicine should not replace but must be coupled with the practice of medicine as an art.

AMYLIN, INSULIN RESISTANCE AND DIABETES MELLITUS. BC4

Garth J.S. Cooper
Amylin Corporation, 9373 Towne Centre Drive, San Diego, CA 92121, U.S.A.

Recent studies have shown that the major protein present in the islet amyloid associated with type 2 diabetes mellitus is a hormone-like peptide, amylin[1], which is normally secreted from the islet B-cells synchronously with insulin. Amylin exerts regulatory effects on important pathways of carbohydrate metabolism consistent with it being a newly discovered hormonal regulator of intermediary metabolism. Amylin is also able to induce, in experimental systems, insulin resistance consistent with that seen in type 2 diabetes.

We have recently proposed a unifying hypothesis for the molecular basis of type 2 diabetes. According to this hypothesis, increased amylin secretion occurs in genetically predisposed individuals, in response to environmental factors, thus setting in train a process which gives rise to: (i) progressive deposition of islet amyloid, (ii) insulin resistance in liver and skeletal muscle, (iii) consequent hypersecretion of insulin, (iv) gradual loss of islet B-cells and progressive islet dysfunction, (v) ultimate restriction of insulin and amylin secretion, and (vi) deterioration in carbohydrate metabolism (consequent on (iii), (iv), and (v)), which progresses through impaired glucose tolerance (IGT) to type 2 diabetes.

[1] Cooper GJS, Day AJ, Willis AC, Roberts AN, Reid KBM, Leighton B. *Biochim Biophys Acta* 1989; 1014: 247-258.

AN EVALUATION OF THE FRUCTOSAMINE ASSAY ON THE TECHNICON CHEM 1 ANALYSER. BC5

Dennis Reilly
Diagnostic Laboratory, PO Box 5728, Auckland, N.Z.

The Technicon Chem 1 Analyser was evaluated for the routine assay of serum Fructosamine.

The Chem 1 using a two reagent system, where 7 ul of Reagent 1 (carbonate buffer) is mixed with 1 ul of sample and a blank reading is made at flow cell 1. After passing through the 'Vanish Zone' 7 ul of Reagent 2 (nitroblue tetrazolium) is mixed, then the absorbance difference is calculated between flow cell 7 and flow cell 9.

Within run and between run imprecision was less than 3%.

Regression Analysis was used to compare the assay with the Roche Fara Centrifugal Analyser methodology.

n	Slope	Intercept	r	r^2
206	0.91	21.1	0.987	0.974

The benefits have been the reagent reduction from 200 to 14 ul per test, samples run in Random Access rather than batch and the slightly improved precision values.

TSH - A GOOD MODEL FOR IMMUNOASSAY COMMUTABILITY. BC6

Frank Watson*, Peter Boyne+, Elizabeth Gregory+, John Beilby*
*Department of Clinical Biochemistry, Queen Elizabeth II Medical Centre, Nedlands, Western Australia 6009, Australia. +Western Diagnostic Pathology, Myaree, Western Australia 6154, Australia.

Commutability refers to the ability of a reference material to show inter-assay properties comparable to those demonstrated by authentic clinical specimens. Reference material differs from clinical specimens in a variety of ways which may include addition of preservative chemicals; matrix composition; species origin of the calibrator for the analyte. Significant non-commutability of calibration materials was demonstrated by Rej and Drake[1] in nine commercial immunoassays for TSH in a study performed in 1990 between 275 laboratories. In a smaller study by the Western Australian Biochemistry Quality Control subcommittee, eight (8) TSH calibrators (IRP 2nd 80/558) and two (2) serum pools were analaysed by ten (10) metropolitan laboratories using six (6) different assays for TSH, 4 of which were IRMA's and 2 were non-isotopic immunoassays.

The TSH calibrators showed considerable between laboratory imprecision especially for the top calibrator which had a target value of 50.6. The mean of the 10 laboratories for this calibrator was 58.3 (SD 12.6) and the values ranged between 41.7 and 77.9. Inter-laboratory imprecision was also high for the two serum pools: i) $\bar{x}$ =5.6 (SD 1.4) 4.1-8.5; ii) $\bar{x}$ = 20.3 (SD 5.6) 14.3-32.4. Recalculating the serum pool results by a) using a matched IRP target value and b) using one pool mean to calculate the other reduced the range and SD significantly for method b) $\bar{x}$ = 20.2 (SD 1.26) 18.4-21.8 but less significantly for method a) $\bar{x}$ = 19.1 (SD 4.2) 12.0 - 25.2. Results of a further study to be carried out using human serum as a common standard for the six TSH methods used by the ten laboratories will also be discussed.

[1] Robert Rej and Patricia Drake. The Nature of Calibrators in Immunoassays: Are they commutable with test samples? Must they be? International Federation of Clinical Chemistry, 3. Bergmeyer Conference Immunoassay Standardization, Lenggries, DE 1990, pages 47-54.

BC7

URINE TOTAL PROTEIN METHOD USING BENZETHONIUM CHLORIDE ADAPTED FOR USE ON THE HITACHI 717.

A Rees, B Andrew and R Pratt
Medlab Ltd., Box 4120, Auckland, New Zealand

An adaption of the benzethonium chloride turbidimetric urine protein method is described for the Hitachi 717.
Human serum albumin standards (Fraction V) were used to calibrate the assay.
Between run imprecision was 8.0% at a protein concentration of 0.20 g/L and 2.0% at 0.51 g/L.
Regression alaysis was used to compare this assay (x) with the Biorad Total Protein kit: Coomassie Brilliant Blue (y), for urine protein values below 1.0 g/L.

n	Slope	Intercept	r	r2
98	1.103	0.01	0.987	0.975

Linearity was up to 2.0 g/L on undiluted urine samples. Interference was minimised by a 1% sodium hypochlorite cell wash before analysis.
Response of gamma globulin was found to be approximately 80%.
The sensitivity of the assay was 0.03 g/L.

BC8

PROSTATIC ACID PHOSPHATASE: HITACHI 717 APPLICATION.

BC Andrew and R Pratt
Medlab Ltd., Box 4120 Auckland, New Zealand.

A stabilised substrate, 2-amino-2 methyl-1,3 propandiol-thymolphthalein monophosphate (AMPD-TMP), for measuring prostatic ACP activity was adapted to the Hitachi 717.
A sample or reagent blank channel to correct for non-ACP or serum turbidity in serum samples was performed.
Within run imprecision was 2.0% at 1.85 U/L, and 0.5% at 5.1 U/L (n = 40).
Between run imprecision over a period of 3 months was 8% at a level of 0.8 U/L and 4% at 3.5 U/L.
The adult male reference interval was determined to be 0.1-0.6 U/L.
Linearity of the assay was up to at least 100 U/L.
The substrate was stable for one month.
Interferences from the calcium reagent were minimised by a 1% sodium hypochlorite wash on a daily basis.

BC9

IMPACT OF DIABETES ON A NEW ZEALAND COMMUNITY AND STRATEGIES FOR INTERVENTION.

DJ Scott* and D Simmons†
*Department of Medicine, School of Medicine, University of Auckland and †Middlemore Hospital, South Auckland.

The prevalence of diabetes in a working population of adults in Auckland aged 40 to 65 is 2.3% for Europeans, 9.0% for Maori and 10.8% for Pacific Islanders. Only 42% are known diabetics. Of the three ethnic groups 75% of the Europeans and only 50% of the known Maori diabetics and 25% of the known Pacific Island diabetics are well controlled. Using these prevalence figures there are approximately 10,000 diabetics in the South Auckland health district.

Hospital inpatient costs continue to escalate despite the introduction of a comprehensive hospital based outpatient clinic and an innovative community diabetes centre staffed by lay health workers drawn from the ethnic groups in the multi-cultural society. A current survey of 750 patients drawn from the community diabetes centre, the hospital outpatient clinic and from general practice confirm tissue damage, poor glycaemic control and the large amount of chronic renal failure and foot problems which will make demands on the dialysis programme and hospital beds.

Money needs to be diverted from the care of end stage disease and invested in community education, earlier diagnosis especially case detection drives among vulnerable groups in general practice. A community intervention team has been formed to assist general practitioners and their practice nurses, improve the education, glycaemic control and detection and care of metabolic complications for the diabetic patients looked after in general practice.

BC10

PREDICTION AND PREVENTION OF TYPE 1 DIABETES (I.D.D.M.).

RB Elliott
Child Health Research, Dept of Paediatrics, School of Medicine, Auckland.

Islet cell antibodies (I.C.A.) develop in the blood of preschool children, who are subsequently fated to get I.D.D.M. The sensitivity of screening for potential diabetes at >4 years age is close to 100% at a screening level of 10 I.V. Although the likelihood of diabetes increases, the higher the level of I.C.A., the specificity of the test is probably less than 30% at all levels ≥10 I.U. Insulin autoantibodies may be concurrently found (in about 50% I.C.A. +ve individuals) and when present increase the specificity somewhat. First phase insulin release is an outcome measure of islet β cell damage, and as such may signal imminent diabetes.

The immune process leading to β cell death is mediated by activated macrophages and cytotoxic T-lymphocytes, which in turn generate directly, or indirectly via cytokines NO_2 or O°. The pancreatic β cells are unique in their inability to neutralize free radicals, except by the process of protein denaturation including heat shock protein, and intracellular enzymes, including GABA synthetase and respiratory enzymes. Nicotinamide is able to protect β cells against such disasters.

In genetically predisposed mice nicotinamide protects against diabetes, and it appears to do the same in humans, who have a similar genetic background, when tested in either first degree relatives of I.D.D.M., or a large group of normal school children.

STANDARDISATION OF APOLIPOPROTEIN ASSAYS. BC11

Charles W Small
Department of Biochemistry, Greenlane Hospital, Auckland, New Zealand.

In common with most methods for the estimation of specific proteins it has proven difficult to obtain adequate interlaboratory standardisation for assays of the apolipoproteins. Consistent results may be maintained in a particular laboratory by using a clearly defined stable set point. However large differences may be found when results are compared between laboratories.

To propose a reasonable solution to this problem the nature of these differences must be explored.

Interlaboratory differences arise from four sources; the standard or calibrator used, the analytical method, the antiserum and the patient (unknown) sample.

1) The apolipoprotein in the calibrator is often in a different physical form or environment to that of the unknown sample. Bias may also be introduced in assigning a value to the calibrator.
2) Although almost all methods involve antigen/antibody binding the different detecting systems used may modify the signal obtained from the reactants.
3) The specificity and avidity of the antibody may significantly affect the magnitude of the results obtained.
4) The sample matrix exerts several effects on the signal produced by the detection system. Sample turbidity plays a major role in these effects.

The nature and properties of the apolipoprotein particle itself are often responsible for these effects. In particular the conditions used to prepare and store the samples alters the stability and accessibility of the lipoproteins and therefore the way they behave in a particular assay system.

A knowledge of the relationship of these variables will enable us to formulate a satisfactory proposal for the interlaboratory standardisation of these analytes using a set of calibrated fresh frozen serum samples.

Q-PROBES IN THE HOSPITAL. BC12

SA McCulloch
Biochemistry, Southland Hospital.

Q-Probes is a term coined because of an increased need for Quality Assurance within Hospitals.

Q-Probes in the Hospital gives background on, explains the basic principles and gives examples of Quality Assurance throughout the hospital and within the laboratory.

INTRODUCING THE NZIMLS BIOCHEMISTRY SPECIAL INTEREST GROUP. BC30

Alison Buchanan
Clinical Biochemistry Department, Auckland Hospital, Auckland.

This group was formed 18 months ago, following a request to Council at the Annual General Meeting of the Institute, to facilitate Continuing Education in the discipline.

The group meets in Auckland, once a month, and Regional representatives have been nominated/volunteered for most areas in the country.

This poster is to advertise its existence, provide information, and give names of contacts for the specific regions.

IMMUNOLOGY

IM1

ATTITUDES AND CONCERNS REGARDING HIV SPECIMEN HANDLING. A SURVEY OF THE WELLINGTON AREA HEALTH BOARD LABORATORY STAFF.

RW Siebers, R Mackenzie, M Lynch and M Humble.
Departments of Medicine and Laboratory Services, Wellington School of Medicine and Wellington Hospital, Wellington, New Zealand.

An anymous questionnaire was distributed amongst all laboratory staff at Wellington, Hutt, Kenepuru and Masterton Hospitals to determine attitudes and concerns regarding handling of potentially HIV positive specimens. Total completed questionnaires numbered 133 giving a response rate of 56.4%. When handling biological specimens 20% of respondents were concerned about acquiring AIDS, 16% hepatitis, 50% both equally and 6% neither. Despite current precautions 54% believed they could become infected with HIV through handling biological specimens. Although 40% of respondents reported that family and/or friends expressed serious concern regarding their work in relation to AIDS only five were considering leaving and 10 would have chosen another career if they had prior knowledge that they could be handling HIV positive biological specimens. Needle-stick injuries were reported by 42% of respondents. Only 10% of laboratory staff always wear safety gloves, 65% sometimes and 25% never. All respondents, except one either strongly agreed (79%) or agreed (20%) that they had a right to be informed if HIV positive specimens were present in their laboratory work area. Provision of adequate safety measures by their employer, was agreed on by 65% while 23% disagreed. Provision of satisfactory education regarding AIDS by their employer was agreed on by 53%, while 38% disagreed.

From the results obtained from this survey, it seems that there are some genuine concerns and some misconceptions regarding handling of potentially HIV positive specimens in the laboratory. A significant number of respondents felt that their employer did not provide either adequate safety measures and/or education in regard to AIDS and biological specimens. A continuous universal education programme is warranted.

IM2

THE SKIN AS A MAJOR IMMUNE ORGAN.

J McKay
Immunology Department, Auckland Hospital.

The skin, in particular the epidermis functions as an initiation site of the immune response. It is now recognised as a highly complex and heterogeneous organ consisting of not only keratinocytes and melanocytes but also bone marrow derived cell populations which are responsible for immune surveillance.

The epidermis contains all elements necessary to mount an immune response — T-Cells, cytokins and accessory cells for antigen uptake and processing.

Autoimmune diseases such as Lichen Planus and Epidermolysis Bullosa are good examples of immunological breakdown at the skin site.

IM3

MUCOSAL IMMUNOLOGY AND THE DEVELOPMENT OF NEW GENERATION VACCINES.

Dr Randall Allardyce
Senior Lecturer in Surgical Science, Christchurch School of Medicine, Christchurch, New Zealand.

Mucosal surfaces are the major portals of contact between us and our environment. They contain 85% of the lymphoid cells in the body, and secretory IgA production is greater than all other immunoglobulins combined. Host protection from many disease-causing microorganisms is most closely associated with immunity at mucosal surfaces such as the gut, bronchus, genital tract and eye. In addition, breast milk antibodies may be the most important factor determining infant survival from infection in developing areas.

Antigenic challenge at one mucosal site may result in immunity that is shared between distant mucosal surfaces. This common mucosal immune system forms the basis for the development of new vaccines and delivery systems that may prevent infection and major diseases in animals and man.

IM4

ANTICARDIOLIPIN ANTIBODY AND LUPUS ANTICOAGULANT ARE SEPARATE ANTIBODIES.

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*Department of Obstetrics and Gynaecology, National Women's Hospital, Auckland. +Immunology Department, Auckland Hospital, ++Haematology Department, Auckland Hospital and **Rheumatology Department, Auckland Hospital.

Antiphospholipid antibodies (lupus anticoagulant and anticardiolipin antibodies) are markers of increased risk of thrombosis, recurrent fetal death, and thrombocytopenia. Failure to detect these antibodies may have important consequences for the patient. Lupus anticoagulant (LA) and anticardiolipin antibodies (aCL) were initially believed to be different manifestations of the same antibody. We screened 87 patients with SLE for both LA and aCL. 26 (30%) were found to have LA (abnormal APTT, KCT, DRVVT or DTTA). 27 (31%) of these patients had raised aCL. Despite the similar numbers of patients detected by each assay only 9 patients had both LA and aCL. Thus the anticardiolipin antibody ELISA showed a sensitivity of only 33% in detecting lupus anticoagulant. This sensitivity was unexpectedly low. Replacing cardiolipin with phosphatidyl serine in the ELISA did not improve the sensitivity for lupus anticoagulant greatly (37%). This result suggests that the anticardiolipin antibody ELISA detects a different antibody to that detected by lupus anticoagulant assays.

We confirmed that these are two different antibodies using simple chromatographic techniques to separate the LA from aCL in the serum of a patient with both antibodies.

IM5

ANTIPHOSPHOLIPID ANTIBODIES AND ADVERSE PREGNANCY OUTCOME. A TWO YEAR EXPERIENCE.

Dr MA Birdsall, L Chamley, Dr NS Pattison
Department Obstetrics & Gynaecology, National Women's Hospital, Auckland.

A strong association was observed between pregnancy complications including fetal loss and antiphospholipid antibodies. A retrospective analysis of 261 women who were tested for both anticardiolipin and lupus anticoagulant was performed at National Women's Hospital.

Four subgroups were analysed:

1. 81 women with recurrent spontaneous miscarriage (3 or more miscarriages).
2. 62 women following a stillbirth or intermediate fetal death.
3. 105 women with either a poor obstetric history or current pregnancy complication
4. 13 women with SLE in pregnancy.

The prevalence of antiphospholipid antibodies range from 40% in those with a history of recurrent miscarriage, 29% in those after a stillbirth, 19% in group 3 and 70% in pregnant patients with SLE.

In the group as a whole, 31% had antiphospholipid antibodies which contrasts markedly with the 3% prevalence in a normal obstetric population. Approximately 1/5th of patients with pregnancy complications such antepartum haemorrhage, preterm labour, pre-eclampsia or growth retardation were found to have these antibodies.

The results of this study suggest that women with these pregnancy complications should be tested for antiphospholipid antibodies.

IM6

DISCORDANT RESULTS IN ANTICARDIOLIPIN ANTIBODY ASSAYS CAUSED BY THE BLOCKING AGENT.

Chamley LW*, Pattison NS* and McKay EJ+
*Department of Obstetrics and Gynaecology, National Women's Hospital, Auckland and +Immunology Department, Auckland Hospital.

The anticardiolipin antibody ELISA is a relatively new assay which has been established independently in several laboratories around the world. Although these assays are basically the same, results from groups studying apparently similar populations can be startlingly different. The first attempt to standardise these assays lead to the finding that only those assays using bovine serum as a diluent gave reliable results.

We present two observations which explain the variability of apparently similar assays and the dependence upon bovine serum as the assay diluent.

The first is the requirement of a cofactor (B_2 Glycoprotein 1) to enable the binding of some anticardiolipin antibodies to cardiolipin. We have shown that for some but not all patients this cofactor is derived principly from bovine serum used as the assay blocking agent and serum diluent. Secondly, we have also demonstrated that the amount of cofactor present in different batches of bovine serum varies widely.

We believe that differences in the amount of cofactor present between batches of bovine serum may account for much of the variability seen with anticardiolipin antibody assays.

IM7

IMMUNOCROSSREACTIVITY OF ANTI PHOSPHOLIPID ANTIBODIES.

J McKay*, L Chamley+ and N Pattison+
*Immunology Department, Auckland Hospital and +Department of Obstetrics and Gynaecology, National Women's Hospital, Auckland.

Antiphospholipid antibodies (APL) are associated with many clinical symptoms that are characterised by an increased risk of vascular thrombosis, recurrent abortion and specified neurological conditions.

Earlier work has led to the premise that cross reactive APL's in patients with SLE are directed against a host of anionic phospholipid and nucleic acid molecules. Other negatively charged molecules such as heparin sulphate and hyaluronic acid are also shown to inhibit APL's in both SLE and non SLE patients. This indicates that "charge density" rather than common phosphodiester groups are the dominant feature in crossreactive antigen structure.

Anti phospholipid antibodies are also observed in certain infectious diseases but differ from autoimmune disease in that they do not require a "co factor" (B_2 glycoprotein 1) for reactivity.

These observations taken in concert, provide clues as to the mechanism of pathogenicity.

IM8

ANTIPHOSPHOLIPID ANTIBODIES AND FETAL LOSS. CURRENT THOUGHTS ON MECHANISM OF ACTION AND THERAPY.

NS Pattison*, L Chamley* and EJ McKay+
*Department Obstetrics and Gynaecology, National Women's Hospital, and +Immunology Department, Auckland Hospital.

Antiphospholipid antibodies are associated with fetal loss, DVT, pulmonary embolism and thrombocytopenia. The common factor in this diverse presentation is vascular thrombosis. The current hypothesis for the mechanism of action of antiphospholipid antibodies is that these antibodies directly effect either the platelet or the endothelium such that platelet aggregation within the uteroplacental circulation is enhanced i.e. thrombosis is initiated. The result is a reduction in placental blood supply, placental infarction, platelet consumption and pregnancy complications including fetal death.

Treatment is based on either a reduction of the antibody level or modulation of its effect. The first can be achieved with corticosteroids, high dose immunoglobulin or chemotherapeutic agents. The second, a modulation of antibody effect, can be achieved with heparin or low dose aspirin.

Aspirin remains the drug of choice for most patients with antiphospholipid antibodies.

HIV/AIDS: THE DISEASE. IM9

Ronald Penny
Clinical Immunology Department, Centre for Immunology, St Vincents Hospital, Sydney, Australia.

Efforts to classify the manifestations of HIV infection continue but currently the CDC criteria remain in use. These criteria are clinically based and extend from the primary or acute seroconverting illness through a long asymptomatic phase with or without lymphadenopathy to symptomatic HIV infection which includes constitutional disease, neurological manifestations, secondary infections and malignancies. These manifestations are closely linked to the relationship between the virus and the immune response. The mechanisms of immune deficiency, the nature of the virus and the immune responsiveness will be discussed in the light of clinical disease.

THE AGEN TEST TECHNOLOGY. IM10

A Baldassi
c/- John Knowles Scientific Ltd, P.O. Box 34306, Birkenhead, Auckland, New Zealand.

Scientists at Agen Biomedical Ltd have developed a novel, rapid, whole blood immunoassay system which has been generically named as the Agen Test technology.

The Agen Test technology allows the detection of circulating antigens, antibodies or drugs in whole blood without specialised personnel or equipment. This is achieved by the use of bispecific reagents, which comprise specific antibodies or antigens that are coupled to a non-agglutinating antierythrocyte antibody.

The Agen Test Technology has been commercialised into several products under the brand name SimpliRED for human applications and Vet RED for veterinary applications.

Clinical results obtained using Agen's SimpliRED HIV-1 Ab kit on blood donors, high risk groups and seroconversion panels indicate that the test performs at an equivalent sensitivity and specificity as the conventional laboratory based ELISA HIV tests.

Other applications of the technology include tests for the detection of fibrin degradation products and canine heartworm. Hepatitis B and a HIV 1/2 combination are currently in the Research and development phase.

SEROLOGY, IMMUNOLOGY QUALITY CONTROL PROGRAMME. IM30

Andrew R Thakurdas [1] and David Haines [2]
Telarc New Zealand, Private Bag, Remuera, Auckland 5, New Zealand [1], and Auckland Hospital Immunology Department, Park Rd, Auckland 1, New Zealand [2].

This external quality control programme is designed for diagnostic laboratories involved in a wide variety of serology and immunology tests.

The SINZ programme is operated by Telarc New Zealand in conjunction with the Auckland Hospital Immunology Department. In its first year of operation, there are about twenty-nine New Zealand laboratories involved in the programme.

The most striking feature of the first round was the diversity of methods and reagents used for such a small country. Varying methods, sensitivities and normal ranges produced a large variation of results.

IM31

MONITORING CIRCULATING B CELLS IN PATIENTS WITH MULTIPLE MYELOMA AT DIAGNOSIS OR IN PLATEAU PHASE. HOW PREVALENT IS LIGHT CHAIN ISOTYPE SUPPRESSION?

M. King, M. Radicchi
Kolling Institute of Medical Research, Royal North Shore Hospital, St. Leonards, NSW 2065, Australia.

It has been reported that the majority of multiple myeloma patients at diagnosis or in plateau phase manifest a phenomenon known as light chain isotype suppression (LCIS). According to this hypothesis some suppressor mechanism causes a drop in the number of ciculating B cells bearing the same immunoglobulin (Ig) light chain isotype as the myeloma paraprotein, producing an abnormal ratio of kappa to lambda B cells. Hypothetically, LCIS is indicative of stable disease and measurement of the blood B cell ratio thereby provides valuable prognostic information. We tested this hypothesis by dual parameter flow cytometry, using a combination of anti-CD19 and anti-light chain antibodies. Furthermore, we compared polyclonal to monoclonal anti-light chain antibodies, and lysed blood to mononuclear cell samples. We confirmed that dual parameter analysis was necessary to distinguish B cells (CD19+, Ig+) from non B cells bearing cytophilic paraprotein. We found good correlation between results obtained using polyclonal versus monoclonal anti-light chain antibodies, and lysed blood versus mononuclear cells. We examined samples from 14 patients but could not detect an abnormal kappa/lambda ratio in any patient. We conclude that LCIS is certainly not as frequent as has been reported, which raises doubts regarding its value as a prognostic indicator.

EDUCATION/MANAGEMENT

QUALITY ASSURANCE AND ACCREDITATION. EM1

Andrew R Thakurdas
Telarc New Zealand, Private Bag, Remuera, Auckland 5, New Zealand

Quality is today's primary competitve force. Quality (fitness for use or conformance to requirements) characteristics once defined and described must be satisfied so as to meet the needs of users or funders of service.

Accreditation is a process through which formal recognition is achieved. Telarc accreditation focuses on an organisation's quality management. It is a process through which disciplined improvement of quality can be effected. Furthermore, it is a mechanism through which quality standards can be demonstrably realised.

Both quality assurance and accreditation activity requires adequate and appropriate people training systems to be in place so that quality of service as well as standards of quality can be cost effectively and reliably achieved. The presentation will describe how training systems for laboratory staff must cover laboratory quality assurance and accreditation.

ACCREDITATION AND TRAINING. EM2

Andrew R Thakurdas
Telarc New Zealand, Private Bag, Remuera, Auckland 5, New Zealand.

Accreditation is a process through which formal recognition is achieved. Telarc accreditation focuses on an organisation's quality management. It is a process through which disciplined improvement of quality can be effected. Furthermore, it is a mechanism through which quality standards can be demonstrably realised.

Training is a process through which learning objectives can be met. Vocational training systems must have measurable objectives and providers of training services must be able to demonstrate their capability for ensuring that learners achieve expected performance levels.

Providers of on-the-job training services can be acredited in a similar fashion as providers of diagnostic or blood bank laboratory services.

RESPONSIBILITIES OF AUTHORS AND EDITORS. EM3

KL Harrison, GR Cannell, BA Walker
Aust.J.Med.Lab.Sci., PO Box 450, Toowong 4066, Australia.

It is the author's right to assume that, when a manuscript is submitted to a journal for publication, it will be reviewed fairly and expeditiously. Once accepted it should be published at the earliest possible opportunity. It is the aim of the Australian Journal of Medical Laboratory Science to have papers reviewed by a panel of experts and replied to within one month of receipt. Papers not requiring revision should then be published in the next edition of the Journal.

In return, editors require certain standards from authors. Journal style and layout should be adhered to meticulously. In addition a number of ethical standards should be observed. It has become the responsibility of the editorial team to not only judge manuscripts on the basis of their scientific content but also to attempt to identify and eliminate the numerous forms of scientific deception. To this end the peer review system employed by the Journal uses highly qualified reviewers selected for their expertise, integrity and fairness. This is critical to the continued development of the Institute's Journal and also our profession.

DOES LEADER BEHAVIOUR OF HOSPITAL LABORATORY MANAGERS EFFECT UNIT OUTCOMES? EM4

GR Douglass* JM Wood and PK Walsh****
* Division of Microbiology, Hunter Area Pathology Service, Newcastle Mater Hospital, Waratah, NSW 2298 Australia: **Graduate School of Management and Public Policy, University of Sydney, NSW 2006 Australia.

Recently, laboratories have been operating in difficult, volatile, unpredictable environments. Changes in legal-political, socio-cultural, economic and educational aspects of the environment and the differing contexts affecting laboratories have increased leadership difficulties. Using the Multiple Influence Model of Leadership as a framework, we investigated whether leadership effects unit outcomes in this turbulent environment. The study showed that managers prepared to exercise Discretionary Leadership do overcome difficulties in their workplace and environment to improve worker perceptions of job satisfaction, workplace environment, and performance. Through use of Discretionary Leadership to support their staff and to attend to job supervisory roles, they also promote job commitment, perceptions of equity, and an increased desire to retain positions amongst the staff. However, Required Leadership was not associated with either performance or employee maintenance in the study.

EM5

EXPLORING THE OPTIONS: FUTURE DIRECTIONS FOR THE EDUCATION AND TRAINING OF MEDICAL LABORATORY SCIENTISTS AND TECHNICIANS.

Mr Peter Bruhn (Manager, Curriculum Development-TAFE, RMIT) and **Mr Bruce Watson** (Head, Department of Health Sciences, School of Information and Health Sciences, RMIT).

This paper will explore the trends in education and training from selected countries which, if implemented, offer the greatest potential for knowledge acquisition and the development of technical/management skills by medical laboratory science trainees and graduates.

Initiatives which appear to offer the most benefits are:

1. Innovative curriculum development processes which incorporate one or more of the following features: the new teaching/learning technologies, competency-based training, workplace (on-the-job) assessment, research into student learning, alternative instructional strategies, course structures and course delivery modes.
2. Cooperative learning ventures between educational institutions and the employers.
3. A greater emphasis on post-graduate continuing professional education.

This paper will also address the apparent decline, observable in Australia and elsewhere, in the numbers of students undertaking science-based subjects at secondary and tertiary level. This trend has significant implications for educators in the way they select students and design courses.

A brief discussion on establishing mechanisms to compare educational programmes between countries is included.

EM6

DEVELOPING COMPETENCY IN THE WORKPLACE: ON-THE-JOB ASSESSMENT OF MEDICAL LABORATORY SCIENCE TRAINEES.

Mr Peter Bruhn (Manager, Curriculum Development-TAFE, RMIT) and **Mr Bruce Watson** (Head, Department of Health Sciences, School of Information and Health Sciences, RMIT).

High quality and reputable on-the-job assessment is crucial to the success of the workplace training component of formal educational programmes. Initiatives in competency-based training and assessment, currently being undertaken in Australia, provide some guidance to the development of reliable and valid assessment standards and methods. These can be adapted for workplace assessment of medical laboratory science trainees.

This paper will present the experiences of the authors and others in Australia in the development of workplace assessment schemes and the factors that are critical for their success.

EM7

A UNIVERSITY EDUCATION IN MEDICAL LABORATORY SCIENCE. THE OTAGO MODEL.

Associate Professor C Watts
Department of Pathology, Otago Medical School, University of Otago, PO Box 913, Dunedin.

The degree of B.Med.Lab.Sci at the University of Otago will be described in the context of the environment of the Division of Health Sciences at the University. Its structure and future development will be discussed with particular emphasis on the opportunities for advanced study at the postgraduate level.

EM8

COPING WITH CHANGE.

Mr Martien Kilderman

Laboratory Services have, like every structure depending on finance for existence, become the arena for application of new economic philosophies. These theories have never before been applied so directly to Health Services in New Zealand as they are today. "Contracting out", "User pays", "Market driven", "a better IRR on investments" are the catch-cries of the age we live in, and concepts we will learn to live with and understand. We have no choice given us.

These changes if unwelcome turn us into "victims", experiencing the anger and grief that is part of moving toward acceptance of new realities. Approval is not required in acceptance. It is in acceptance that we release the skills to deal with the related stresses and to even master the new climate and become a player in it. This occurs by allowing a new management theory to achieve its goals while you use it to achieve your own. To recapture some control of the process is desirable personally and professionally.

In sport it is the team that takes control and outplays the other team at its own game that wins and survives. Withdrawal is not a step toward overcoming. Overcoming is the process of accepting new realities, learning the new rules and out-playing the managers of change by superior management of our own change experience.

EM9

ETHICS IN CARDIAC TRANSPLANTATION — RECIPIENT AND DONOR ISSUES.

Carol Whitfield
Recipient Transplant Co-ordinator, Green Lane Hospital

Where no other treatment is available, cardiac transplantation for end-stage myocardial failure is now an accepted procedure with a one year survival of over 80%. In 1989 over 2000 cardiac transplants were performed world wide. Scarcity of donor organs is now the main factor limiting cardiac transplantion and up to 30% of patients in some centres die waiting. Since the development of organ transplantion 35 years ago, many ethical issues have evolved. Recipient issues facing us include our rights to all types of health care and medical treatment, ensuring equal access to this treatment and selection of suitable patients for transplantion. Now, in a climate of cost cutting, should expensive 'high-tech' medical treatment be considered at all? With respect to the organ donor, who consents to organ donation? Are there ways of increasing the donor pool? Currently New Zealanders use an 'opt-in' method to declare their willingness to donate organs, other countries use an 'opt-out' or 'required request' system. For the future, should expansion into other forms of transplantation such as lungs or liver be considered in New Zealand?

A PATHWAY TO THE FUTURE: "HE ARA KI TE AOMARAMA". EM10

Pauline Kumeroa Kingi
118 Grafton Road, Auckland.

Overview on Medical Ethics, the different schools of thought and preferred approach followed by an introduction to the Maori method of decision-making. The place of the "cultural imperative" in the world of western ethics, and the bicultural policy of the Auckland Area Health Board. Some comment on general issues considered by Ethics Committee and suggested strategies for responding to the racial, women's rights and legal implications.

LIFE AND DEATH IN THE ICU: ETHICAL CONSIDERATIONS. EM11

Sharon L. Kletchko
Medical Specialist Intensive Care Physician, Nephrologist, Middlemore Hospital, Auckland.

In this paper I will discuss the "raison d'etre" for the existence of the Intensive Care Units, concentrating on the care of those patients for whom the ICU is of no, or only marginal, benefit.

Five guiding principles helpful in facilitating ethical decision-making about the withdrawal or withholding of aggressive, life-sustaining technologies will be discussed in detail. They are (1) Potential for Salvageability; (2) Preservation of Life; (3) Nonmaleficence; (4) Autonomy; and (5) Justice.

The final principle of justice or fairness, especially when referring to distributing scarce resources equitably, will be extensively reviewed. I suggest that physicians and society should develop categorical standards setting limits to the actions of individual physicians in a decisive way.

In conclusion, I suggest that scarcity, by definition requires choice and that honest responses to these situations may yield long-term advantages. Scarcity forces both societies and institutions to establish priorities which may give rise to more efficient resource use such as devoting more resources to those medical circumstances where returns in terms of health outcomes are likely to be greatest. Doing everything in some patients should mean maximizing comfort measures and not aggressive efforts at unobtainable survival.

PROBLEMS ENCOUNTERED WITH THE INTRODUCTION OF ASSESSMENT LOGBOOKS. EM12

Graham Thorne
Senior Laboratory Training Officer, Auckland Hospital, Park Rd, Auckland.

The use of job analysis has been an assessment tool for many years especially in occupations such as the Armed Forces. The New Zealand Medical Technologists' Board introduced Practical Competence Assessment Logbooks in 1989 to replace the existing practical examination for qualification in 1990. The objectives for tasks to be met by the trainee and the assessment required by senior laboratory staff appeared a daunting encounter. The initial perception of the logbook was the great deal of effort that would be required by both participants. Also, the realization that some of the objectives apparently new to the subject had always been part of the Certificate Syllabus requirement.

Practical Competence Assessment is not an awesome experience if the author prepares very specific and rigid objectives. The trainee must be given the correct training with each step explained and practiced. It is important for the trainer and assessor to gain professional training if possible.

Practical Competence at mastery level is like obtaining a licence, however it is time (experience) that provides the ability of higher throughput and the greater awareness of the problems and their solutions.

MEDICAL LABORATORY SCIENCE EDUCATION IN THE UNITED KINGDOM. EM13

DM Taylor
Applied Science Department, Auckland Institute of Technology.

The proportion of graduates from both universities and polytechnics entering the profession of Medical Laboratory Science has increased steadily since the mid 1970's. Many courses have been specifically designed for the profession with the subjects designed with current laboratory practice in mind.

Inter institute moderation of examinations ensures a high standard throughout the country.

Fellowship courses offered by the IMLS are being replaced by Master courses — Post Graduate Diplomas.

EM14

THE NATIONAL DIPLOMA OF MEDICAL LABORATORY SCIENCE. CHANGING EDUCATIONAL PERSPECTIVES.

PR Lucas and DJ Stannard
The Auckland Institute of Technology

The development of the NDMLS course from the previous part-time NZCS (Medical Options) certificate course is discussed, and the essential changes in the course objectives, outline, and learning goals presented.

The National Diploma course has now had three intakes of students, and because the rigorous Auckland Area Health Board selection criteria for prospective candidates have not been changed from NZCS courses, it has been possible to compare student learning outcomes under different teaching regimes. Student results, from three intakes, for year one of the diploma course are compared with student results from the former NZCS course, and the implications discussed, particularly in terms of future developments.

Strong advantages of the current NDMLS course favouring successful student learning have been employer input at an early stage, collective student involvement, specialist course development, and ongoing lecturer-student liaison.

An identified disadvantage has been higher stress learning situations.

NDMLS HAEMATOLOGY: THE BASIS IS ON STUDENT LEARNING. EM15

Colin McGough
Senior Lecturer, Sports and Health Science Department, Auckland Institute of Technology.

This short paper discusses the concept of student learning being the ultimate aim of the Haematology program in the ATI National Diploma in Medical Laboratory Science.

Comment will be made about problems involved in setting up the program in this way. These included the large amount of initial preparation required as well as student acceptance of the style of course.

Broad objectives better suit this type of program, allowing students to read widely so that broad concepts are understood well. Hopefully memory of minute detail will follow as practical experience fills out a solid basic understanding.

HISTOPATHOLOGY

THE USE OF ULTRASOUND IN IMMUNOPEROXIDASE STAINING. HP1

Stewart H-G Chew.[1] Robert D Cook [2]
Curtin University of Technology, GPO Box U 1987, Perth, WA 6102 [1] and School of Veterinary Studies, Murdoch University, Perth WA 6150 [2].

Ultrasound has been used to reduce both the incubation time and the concentration of the primary antibody during immunoperoxidase staining. Paraffin sections of lymphoid and myeloid tissues were immunostained with commercially available mactophage markers using the Avidin-Biotin-Complex (ABC) technique. It was found that a 2-minute exposure to ultrasound followed by a 10 minute incubation of the primary antibody at the recommended dilutions produced a similar result to that seen in a control slide incubated with the same antibody overnight. Also, it was found that the same primary antibody could be effectively diluted up to 8 times the recommended strength. Ultrasound exposure for longer than 2 mintues did not in any way intensify the effectiveness of staining process. The morphologic qualities of the sections were unaffected by the use of ultrasound over the 2-minute period.

SKELETAL MUSCLE BIOPSY — A USEFUL TOOL FOR DIAGNOSIS OR A LAST RESORT? HP2

Silverstone M
Histopathology Department, North Shore Hospital, Auckland.

How often are skeltal muscle biopsies performed as a primary diagnostic tool or as a last resort to try to diagnose patients where all other investigations have failed to produce a satisfactory answer? If a diagnosis is made, to what use can this information be put?

The remaining abstracts will be published in the NZJ Med Lab Science Vol 46, No1, March 1992.

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Membership fees for the year beginning April 1, 1991 are:

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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 Sub-Committee Report — August 1991

Since the May meeting there have been the following changes:

	26.8.91	23.5.91	28.2.91	7.11.90
Membership	1297	1202	1277	1272
less resignations	37	33	10	3
less G.N.A.	8	7	11	5
less deletions	81	-	116	-
less deceased	-	2	-	-
less duplications	1	-	-	-
	1170	1160	1140	1264
plus applications	10	22	61	3
plus reinstatements	-	116	2	1
	1180	1298	1202	1268
Composition				
Life Member (Fellow)	12	12	12	12
Life Member (Member)	5	5	5	5
Fellow	21	21	22	22
Member	666	711	679	724
Associate	395	462	399	424
Non-practicing	60	57	56	60
Honorary	29	29	30	30
Total	1188	1297	1202	1277

Applications for Membership
J TAPP, National Womens, Cytology; K YOUNG, Diagnostic; C SUBRAMANIAN, MedLab; S BROCKIE, Wellington, Diagnostic; A MacDONALD, Auckland; C KING, Waikato; K STOCKMAN, Waikato; J O'SULLIVAN Waikato; S COCKER, Nelson, Diag.; L GASELEY, Dunedin, MedLab; F FAIGA, Wellington.

Resignations
D STEVENS, Dunedin; G LIBBY, Hastings; T BRADFIELD, Invercargill; D STOCKDILL, Tauranga; B GLASS, Dunedin; J EPPLETT, Royston; L HESLIN, North Shore; F McKINNON, Royston; S WESTAWAY, Dunedin MedLab; J MORGAN, Northland Path Lab; K CALDWELL, Waikato; T CORMACK, Christchurch; T LOGAN, Waikato; A FINNERTY; B GUILLIARD, Napier; S HILL; K JONES, Christchurch; T LANGFORD, Palmerston North; A MEE, Christchurch; B MONTGOMERY, North Shore; F PATERSON; D PATTERSON, Invercargill; L SEAWARD, Christchurch; A THOMPSON, Middlemore; S WILKINS, Auckland; S LANGFORD, Palmerston North; L McMILLAN, Christchurch; L PALMER, Middlemore; C WESTWOOD, Waikato; M SMITH, Lower Hutt; M COCKBURN, Stratford; G TUNBRIDGE, Wanganui; N EGERTON, Middlemore; M BUCHANAN; L SCHOLLUM; K SIMS; J FORTUNE.

Gone No Address
K POWELL, Hamilton Med Lab; J ANDERSON, Waikato; D NIXON, Greenlane; D OWEN, Greenlane; P RICE, Taranaki; M DIXON, Greenlane; G BEATTIE; N CRONIN.

LETTERS TO THE EDITOR

Dear Sir

Walker, Wilson and Till (NZJ Med Lab Science 1991; 45(2): 45-47 concluded from their nationwide survey of New Zealand laboratories that their "limited results offer no support to the hypothesis that water is the major route of transmission of Giardiasis in New Zealand." Regrettably however, even this cautious conclusion is an overstatement. Even if water is the major vehicle of transmission of cases of giardiasis in New Zealand, this would have been unlikely to have been detected in the research design used for several reasons.

Firstly, cases were classified according to the water supply of their dwelling (presumed to be the same type of supply received by the doctors "locality"). Yet many cases of water borne giardiasis would be expected to be acquired outside of the home; eg at work, or during recreational pursuits away from home on weekends, holidays etc (1). Such cases acquired from contaminated waters in rural areas, national parks etc, would subsequently be diagnosed on return from holiday by their urban general practitioner and incorrectly classified as a case from a "safe" water area.

Secondly, it appears that smaller towns and communities, who are more likely to have unsafe water supplies (2), were under-represented in the sample. Given the logistic and practical difficulties of arranging investigation of their patients, it is not surprising that general practitioners in these centres are less likely to seek a laboratory diagnosis, and rural cases would always be systematically under-represented in a national survey such as this. In these two respects Table 2 was always destined to say more about the existing patterns of water supply in this country and the relative propensity of urban and rural practitioners to request laboratory investigation, than about the contribution of water to the national case load.

Thirdly, the timing of the survey was unfortunate. For reasons that are still obscure, there is almost certainly a marked seasonal variation in the occurrence of giardiasis in New Zealand, with a summer/autumn peak. This was observed in our study of giardiasis in Dunedin (3), as well as in the study reported from the Bay of Plenty (4). This seasonal variation, is prima facie evidence for water or other factors especially associated with recreational activity, as a vehicle of transmission.

The relative importance of water in the transmission of giardiasis in this country remains unknown (5). Analytical rather than descriptive studies of the epidemiology of giardiasis in humans will be necessary to determine this, and should be a research priority. If our experience proves to be similar to that of the United States (and the circumstantial evidence so far suggests it could be so), about 25% of the total community caseload could be the result of transmission by water (6, 7).

Walker et al have overstated the conclusions that can be drawn from an otherwise useful national survey. The data presented add essentially no information to the assessment of the contribution of contaminated water to a significant public health problem in this country.

Yours sincerely

Graham Fraser MB, MCCMNZ
CLINICAL LECTURER

References

1. Meyer EA, Jarrol EL. Giardiasis. *Am J Epidemiol* 1980;**111**:1-12.
2. Craun GF. Surface water supplies and health. *J Am Water Works Assoc* 1988;**80**:40-52.
3. Fraser GG, Cooke KC. Endemic giardiasis and municipal water supply. *Am J Public Health* 1991;**81**:760-2.
4. Okell RS, Wright JM. *Giardia lamblia*; an assessment from the Eastern Bay of Plenty: September 1 1986 - September 30 1989. *NZ J Med Lab Technol* 1990;**44**:64-6.
5. Fraser GG. Giardia and water supply. *NZ Med J* 1991;**104**:203-4.
6. Craun GF. Water not sole source of disease transmission (letter). *J Am Water Works Assoc* 1986;**78**:4.
7. Chute CG, Smith RB, Baron JA. Risk factors for endemic giardiasis. *Am J Public Health* 1987;**77**:585-87.

Ed Note:
This letter was referred to the authors of "Giardiasis in New Zealand, Results of a Laboratory Based Survey" [NZJ Med Lab Science 1991; 45(2): 45-47]. Their reply is published below.

Dear Madam

The major purpose of our survey was to begin to accumulate information concerning the prevalence of giardiasis in New Zealand and to determine the different laboratory testing methodologies in use. In addition however, we attempted to see if there was any relationship between type of water supply and reports of positive tests for giardiasis. We acknowledged that the method used for examining this area had many important limitations and indeed stated that these results "require cautious interpretation". In his letter, Dr Fraser has added further discussion of these limitations with which we agree. We would differ with his view about overstating these findings however, and believe they were worthwhile presenting in the context of a discussion of their limitations as we attempted in the discussion section of our paper. The statement that the limited results did not support water as a major route of transmission is only good for those results and in no way suggests that water on any given occasion could not have the potential to be a major route of transmission.

Nevertheless we are also in agreement that further studies are necessary to obtain even a basic understanding of the epidemiology of giardiasis in this country.

Yours sincerely

N K Walker N A Wilson D G Till

NZIMLS CONTINUING EDUCATION
SPECIALIST INTEREST GROUP UPDATE

FROM THE CONVENOR OF THE NZIMLS SPECIAL INTEREST GROUPS

Dennis Reilly, NZIMLS Vice President

The concept of Special Interest Groups (SIGS) arose from Council's need to have a ready reference point for professional comment on issues relating to each discipline of Medical Laboratory Science. As the size of council has decreased, it was important to set up smaller management groups which could advise as well as promote their discipline, ideally through continuing education programmes.

Because staff need to develop their practical and theoretical skills as well as keep up with recent advances, another role of the SIGS is to provide a consistent and planned education programme.

SIG Objectives

* Advise Council on all matters pertaining to their respective discipline.
* Nominate suitable individuals to act as examiners and to act as a review group for syllabi and logbooks.
* Organise workshops relevant to the discipline within the resources of the NZIMLS.
* Liaise with the Annual Scientific Meeting Organising Committee on the components of workshops and forums.
* Target appropriate persons to publish papers of interest in the NZIMLS Journal.

The SIGS are still in an embryonic stage, but it is planned that in the March edition of the Journal, a programme be published outlining courses that are to be held during the year.

Already seminars have been held, with new and innovative ideas which will develop Technologists' knowledge and communication.

Council is thankful of the support already received from the SIGS. Now that the Industrial responsibilities have gone, education and communication are the major responsibilities of the NZIMLS. Together with the SIGS, Council believes Medical Technology will flourish.

FROM THE IMMUNOLOGY SPECIAL INTEREST GROUP (ISIG)

Convenor: Gillian McLeay

Contact address: Laboratory Training Centre, Building 18, Auckland Hospital, Park Road, Auckland 1.

Now we are One

ISIG has its first birthday on 10 October 1991. Currently, the ISIG Network has 48 members, and while a few are content to be on the mailing list only, the majority are keen to participate in the Group's activities.

ISIG's first seminar and AGM, took the form of a cruise on Auckland's Waitemata Harbour. The weather was typically wet and windy for the time of the year, but did not detract from a most enjoyable occasion. The "Floating Forum" was planned to run concurrently with the South Pacific Congress, and a number of ISIG members were attending Congress also. We were delighted to have Lisa Evans from Brisbane and David Gaunt from Canberra, join us for the day.

The AGM saw the formation of ISIG as a national body with the national committee comprising:

Convenor:	Gillian McLeay	Auckland
Secretary:	Mary Ann White	Auckland
Treasurer:	Angela Wheeler	Auckland
Regional representatives:		
Region 1.	Joy Odgers	Northland
Region 2.	Sherryn Cepulis	Waikato
Region 3.	Gerry Campbell	Wellington
Region 4.	Diane Phillips	Christchurch

(Region 5 (Otago/Southland) has combined with Region 4 due to small membership numbers at this stage.)

In addition to the Auckland Group, Gerry Campbell reports that a Wellington Group has been formed and has had one meeting already, with further get-togethers planned.

If you are interested in joining in ISIG activities or being on the mailing list, contact Gillian McLeay or your regional representative. New members are always welcome.

FROM THE HAEMATOLOGY SPECIAL INTEREST GROUP (ISIG)

Blood Line

A very successful inaugural meeting of HSIG was held in conjunction with the South Pacific Congress in August. Regional representatives will be circulating copies of the minutes. Two new members have been recruited as representatives to the HSIG network, they are Audrey Grimmer for the Central North Island — Hawkes Bay/Manawatu/Taranaki region and Cindy Lincoln for the Northland/Auckland region.

A brief profile of these representatives is published with other representatives' profiles pending!

Central North Island Representative: Audrey Grimmer

Present Position:	Staff Technologist, Haematology, Palmerston North Hospital
Past Positions:	Taumarunui Hospital Laboratory, National Women's Hospital Laboratory
Marital Status:	Contentedly married to Brad with three school age children, Murray, Sarah and Paul.
Other Interests:	Bull farming, Gardening, Hockey

I gained my NZCS in the 1970 era and left to raise my family. I returned to work in a part-time position in the 1980's and soon came to the conclusion that it would be wise for me to complete my training. So back to the books, and I completed my training in the late 1980's.

Having gained my qualifications recently I can appreciate the value of the continual education of our staff and so am willing to be the regional representative for HSIG for my area.

I trust we can all learn "heaps" together through the Special Interest Groups, especially at this time when there is so much changing in our New Zealand health scene.

Audrey Grimmer — Central North Island "HSIG" Representative

Auckland and Northland Representative: Lucinda (Lindy) Lincoln

Position:	Grade Officer - automation
Interests:	Cycling, jogging, swimming, reading and travel
Qualifications:	Certificate and Specialist Level Haematology, Certificate in Adult Education - Auckland Technical Institute

My position at Auckland Site Haematology Laboratory involves general first line management, automation maintenance, quality assurance and staff training.

I have a strong interest in on-going education in Haematology and have been involved in lecturing at most levels as well as being involved in the new Diploma course in Auckland. It is this interest which led me to accept the position of regional representative for the Auckland/Northland area for the Haematology Special Interest Group.

Cindy Lincoln — Northland/ Auckland "HSIG" Representative

Blood Line wish to include news of new appointments and changes in Haematology Laboratories throughout New Zealand. Please send in your contributions for publications to the HSIG Secretary - Ann Cooke, Laboratory Training Centre, Building 18, Auckland Hospital or your own representative.

News this time is of John Peters' appointment as Grade Laboratory Officer in Charge of Coagulation, Haematology Dept. Middlemore Hospital. Older news is Lynette Rudkins' appointment as Technologist in Charge of Special Haematology at Auckland Hospital.

Changes are to take place on the Auckland Hospital site in mid November, the Laboratory Services for Auckland and Princess Mary Children's Hospital will be integrated. Princess Mary Laboratory has been open since the early 1950's so the integration will see the end of a unique era — a dedicated paediatric laboratory.

The integration of services is a direct result of present health sector financial constraints. Permanent appointments have been made in Haematology, these are Allan Johns as Charge Technologist and Rennie Dix as Graded Technologist, Deputy Charge Technologist and Paediatric Liaison Technologist.

Monica Cheesbrough, recently in NZ as guest speaker at the South Pacific Congress, took home with her a copy of the Haematology Standardised Reporting and Nomenclature Slide set. Monica, who works as Director of Tropical Health Technology, is from Cambridgeshire in England and is the author of two laboratory manuals for developing countries covering Microbiology, Parasitology, Biochemistry and Immunology. Monica is about to begin working on the third volume in the series which will include Haematology and Immunohaematology. We may well see some of the slides and cell descriptions used in this edition, an exciting prospect!

Lastly but not least the beginnings of our own Trade and Exchange — if anyone is interested, the Haematology Dept., Middlemore Hospital has about 10,000 diluent reservoirs for use with a Clay Adams Model 800 — used for platelet counts.

FROM THE BIOCHEMISTRY SPECIAL INTEREST GROUP (BSIG)

Convenor:	Alison Buchanan
Contact address:	Clinical Chemistry Dept., Auckland Hospital, Park Road, Auckland.

It was rather disappointing to see how few papers there were in the Clinical Biochemistry forums at the South Pacific Congress.

This lack was noted by many. It would be sad to see the many years of effort spent building our profession, wasted by lack of effort and enthusiasm now.

Boehringer Mannheim have offered to help renew a bit of this enthusiasm by donating a medal and a very generous travel award, to the best Clinical Biochemistry paper presented at our Annual Conference.

Many thanks to Ross Hewitt and his organisation.

More details in this column as they are finalised.

A small group of Clinical Biochemists met briefly during the South Pacific forum - another attempt to find what the members want from the Special Interest Group.

The results:

- Robert Siebers, Dept. of Medicine, Wellington School of Medicine, has volunteered to act as the Regional Representative for the Wellington area.
- The Special Interest Group will help to provide three Seminars in 1992.

June:	Auckland
August:	Wellington (associated with the Annual Conference)
September:	Christchurch

WATCH THIS SPACE FOR FURTHER DETAILS.

MICROBIOLOGY SPECIAL INTEREST GROUP REPORT 0CTOBER 1991

Convenor: Shirley Gainsford

Planning is underway for workshops to be held at the NZIMLS annual conference in Wellington on August 27th and 28th, 1992. A workshop on the Identification of Yeasts is very likely, other ideas are still being developed. The Certificate level Microbiology syllabus is presently being revised for the MLTB. A revision of the Specialist level syllabus will be completed by the end of the year and anyone interested in commenting on the proposed changes can obtain a copy from me.

We intend producing a list of technologists who can act as examiners. We envisage a new examiner setting QTA examinations for two years than setting Certificate level for two years, while more experienced technologists set Certificate level examinations for two years then go on to Specialist level for two years. We will be contacting charge technologists soon asking for nominations, so please support your profession by agreeing to act as an examiner if asked.

The Journal club will continue next year but the subscription is probably going to increase to cover the cost of copying. New subscriptions will be due in January.

TRANSFUSION SCIENCE SPECIAL INTEREST GROUP

To date, the Transfusion Science Special Interest Group has been very quiet and there are doubts that many people involved in Transfusion Science even know who the members are. In an effort to remedy this situation, one of the presentations made at the N.I.C.E. Weekend in Nelson in April this year was information regarding this group.

For those of you who are interested and who were unable to attend the N.I.C.E. Weekend the following is a transcript of the presentation on the Transfusion Science Special Interest Group.

History

From late 1989 the Council of the NZIMLS sought to establish Special Interest Groups for each of the disciplines practised in Medical Laboratory Technology to provide advice and comment in respect of professional and educational matters as may pertain to each discipline.

The brief for the Transfusion Science SIG is as follows:

1. Organise Seminars and Workshops relevant to Transfusion Science within the resources of the NZIMLS.
2. Create new opportunities for learning.
3. Nominate suitable individuals to act as examiners for the Institute Examinations, including Fellowship, Specialist Certificate and Qualified Technical Assistant Certificate.
4. Be able to advise the Annual Scientific Meeting Organising Committee on the Transfusion Science component of Workshops and Forums.
5. Coordinate the syllabus review for the Specialist and Qualified Technical Assistant Certificate on an annual basis.
6. Advise the Council of the NZIMLS on matters pertaining to Transfusion Science.

The line of communication with Council is through the Chairman, NZIMLS Continuing Education Sub Committee to the Education Committee, except in relation to matters raised under Item no. 6 when communication is directly from Council to the Special Interest Group.

Continuing Education Programme Workshops, Seminars, etc organised by the Special Interest Group under the auspices of the NZIMLS must be approved by the Continuing Education Sub Committee based on an annual programme with expected costing etc prepared by the Special Interest Group.

The NZIMLS Council review the programme, approve and endorse or recommend alterations and forward the requested sum of money involved to a bank account nominated and operated by the Special Interest Group which remains under audit of the NZIMLS. Money is set aside annually for the activities of each Special Interest Group, to pay for any approved activity including meetings of the Special Interest Group.

Reports of Special Interest Groups are published in the May and November issues of the NZIMLS Journal.

Establishment

It seemed logical that a body that had been in existence for at least 18 years, ie the Technical Working Party of the Transfusion Advisory Committee with its well established system of communication, should form the initial Transfusion Science Special Interest Group with the ability to co-opt others as it saw fit. The Technical Working Party is comprised of the following members:

Walter Wilson Auckland
Grant Storey Hamilton
David Wilson Palmerston North
Stewart Dixon Wellington
Kevin McLoughlin Christchurch
Alan Knight Dunedin

This recommendation was made to the Council of the NZIMLS and was accepted. Two additional members were immediately co-opted. They were:

Roger Austin New Plymouth
Lindsey Browning Invercargill

Achievements

In the eight months since the Special Interest Group was formed, progress has been slow. There has been one previous report in the NZJ Med Lab Science and the Committee selected the Specialist Certificate Examiners for 1991.

It has been a disappointment that so little has been achieved. It may have been considered by some that the N.I.C.E. Weekend and South Pacific Congress were sufficient in these early days of the Special Interest Group, though it should be noted that the N.I.C.E. Weekend is not currently an event which is organised by the Special Interest Group. The Special Interest Group will meet within the next two or three months with the intention of forming a programme of Continuing Education for 1992.

Suggestions

A number of suggestions have been made to the Special Interest Group as to the type of activities that they could pursue. These include:

Mini Workshops or Seminars on particular topics to be presented at several venues.

Evaluations of new products, such as liquid Papain, monoclonal Anti-As capable of detecting A_x cells etc.

Self-examination quizzes — send out the quiz followed by the answers later.

Interesting Case Studies.

Make more use of currently available resources such as:

Publish Transcripts of conference papers.

Publish transcripts or abstracts of N.I.C.E. Weekend presentations.

Publish findings of in-house reagent evaluations.

Publish transcripts of in-house presentations such as journal clubs.

Get involved in a newsletter with the abstraction of short articles of general interest. This could go out with the NIPS mailings 2 to 4 times a year and could include information from journals, TAC, AABB "news briefs" and others.

It is evident that considerable effort will be required to organise these types of activities.

The following is a list of questions put to those attending the N.I.C.E. Weekend:

1. What do you feel the activities of the Special Interest group should encompass?
2. Are you interested in assisting with these activities?
3. Are you interested in becoming members of the Special Interest Group, bearing in mind that a high level of commitment will be required?
4. Are there any other points that you would like the Special Interest Group to note?

The answers to question 1 echoed the suggestions made earlier.

Everyone present expressed an interest in either becoming a member of the Special Interest Group or at least assisting in the organising of activities in their area.

Those people who expressed an interest were asked to express their interest in writing to the Convener.

Likewise those of you reading this article who are interested in answering any of these questions or assisting with the activities of the Special Interest Group in any way should write to:

David Wilson
Convener
Transfusion Science Special Interest Group
Department of Transfusion Medicine
Palmerston North Hospital
Private Bag
Palmerston North

FROM THE HISTOLOGY SPECIAL INTEREST GROUP

Convenor: Ken McGrath
National Womens Hospital Auckland

This is the most recently formed special interest group. We await their first report in the March 1992 issue of the Journal.

Wellcome Travel Award — Geneva 1990

Roger Austin
Taranaki Base Hospital, New Plymouth

August 1989 and the Annual Scientific meeting in New Plymouth had been going smoothly and we were all enjoying the Conference Banquet when the announcement of the winner of the Wellcome International Travel Award was made. I could not believe that it was my name being read out. The announcement and the way that it was received by my colleagues was a moment I will always treasure.

July 21st 1990. Thirteen months of planning and belt tightening had the entire Austin family on an Air New Zealand flight to Auckland. As a family, Glenys, Kelly (15) and Shannon (13) and I had already decided to make a trip to Europe well before I got the Award — that was a real bonus. We had seven weeks of travel ahead of us and were really excited at the prospect. First stop for winter weary Kiwis was hot, muggy Hong Kong where we did all the usual things like sightseeing, riding on the Star ferry and Peak Tram, partaking of the local cuisine (this was to become a favourite pastime in the weeks to come), and of course shopping. This place has to be seen to be believed, abject poverty in the form of a blind double amputee sitting on a skate board pushing a begging bowl along the crowded pavement. There were many other examples to offset the other end of the scale where the penthouse suite dwellers with $NZ300,000 income and a five star luxury lifestyle are envious of the lifestyle of the penthouse dwellers with greater incomes.

Flying from Hong Kong to London at night on the upper deck of a British Airways 747-400 enabled a visit to the cockpit where the pilot pointed out flashes of light over to the east — this was Kabul where the fighting continues about a year or so after the Russian withdrawal.

London, quick tour of sights, pick up campervan and provisions and head for the ferry at Dover by taking the wrong turning out of the camper suppliers and spend half an hour going in the completely wrong direction. We get to France via Calais and Kelly and Shannon get to exercise their linguistic ability on the locals. Travelling around France we can understand why New Zealand has trouble selling its butter here. As far as the eye can see there are sunflowers their heads following the sun throughout the day. They must be easier to graze than herds of dairy cattle. Pass through the picturesque Loire Valley with imposing chateaux and quaint villages. Following maps, signposts and guesswork with the added confusion of asking locals directions has got us confident that we can handle this lark without any problems. A great delight throughout the trip was supermarket shopping — mixing with the locals in supermarkets that handle everything from normal items through to motorbikes, lawnmowers, and high tech audio equipment. We spent a night in a camping ground in the Chateau de Chazeul near Vichy and met an English couple. The first people we had been able to converse with without lots of gestures and frowning and multiple "pardons?", since we left England. The couple turned out to be Dr Derek Tovey, the recently retired director of the Yorkshire Regional Blood Centre and his wife Kay. We spent a very pleasant evening under the stars of central France talking shop.

On through the French countryside climbing into the Alps before passing into Northern Italy and some frantic driving completely surrounded by huge articulated rigs on the Italian autostrada. Pass through little villages of red brick with slate roof shingles, lots of vineyards and nectarine orchards along with maize crops in the fields. It was at this stage that we manage to interpret the local newspaper headlines "Iraq invades Kuwait", of no consequence to us as we visited the Italian lakes, Venice and up through the Dolomites into Austria and some of the most spectacular scenery that we saw during the whole trip. Through Liechtenstein and into and across Switzerland to the World Congress of the International Association of Medical Laboratory Technology in Geneva.

Geneva on the shores of Lac Leman, the city of John Calvin, is typical of most old European cities with picturesque and historical buildings, narrow winding streets all but impossible to thread a large campervan through. The Noga Hilton was the venue for a rather grand Presidential reception where Des Philip welcomed the delegates and invited representatives from each country to present their country's flag to the congress. Awards were presented to various winners for scientific achievement. The congress coincided with the Fete de Geneve and on the opening night on the lake outside the Noga Hilton was the most magnificent fireworks display imaginable, lasting two hours and keeping the huge crowd who had lined the lake front throughout the day Oohing and Aahing as one spectacular set followed another. We had a great introduction to Geneva.

Social events continued throughout the week with a reception by the Authority of the City of Geneva in the elegant Palais Eynard, Dinner Dance cruise on the M.V. Helvetique on Lac Leman and walks around the old town of Geneva. Swiss folk singing, dancing, yodelling and Alpenhorn playing featured at the International Folk Evening where Swiss sausages and raclette (melted cheese on potatoes) were the menu highlights. Tom Lindsay the Marketing Communications Manager for Wellcome UK represented the parent company of the award of which I was recipient and made sure that Glenys, Kelly, Shannon and I wanted for nothing throughout the Congress. We very much appreciated his help and humour and enjoyed meeting him and his enchanting family once we returned to England a few weeks later.

The General Assembly of Delegates (GAD) is a major focus of the Congress, this is preceded by a pre GAD where every item that is to be raised at the GAD is discussed at the pre GAD. This is a curious state of affairs that does nothing to speed up the decision making process. The GAD saw the admission of Canada, Israel, Italy, Kenya, Malaysia, Tanzania and Uganda to the IAMLT, and the election of officers for the forthcoming term with Mr Thomas Yeung of Hong Kong being the representative in our region. A matter of concern was the state of the International Association's financial affairs which show a shortfall of 3000 Swiss Francs and a missing Treasurer, (I thought this only happened in comics). The South African delegates were invited to address the GAD for ten minutes and indicated the situation with Laboratory Technology in their country and how it meets all of the IAMLT membership requirements. Mr Du Plesse stated their intention to reapply for membership at the Dublin 1992 Congress. After this presentation the representatives from the other African nations and Nordic countries asked that it be recorded that they regretted that the proposal for South Africa to speak was on the agenda and they disassociated themselves from the proposal. The NZIMLS has every reason to be proud of Des Philip the retiring President of the IAMLT for the way he chaired a very difficult

Dennis Dixon-McIver, Chairman of the 1991 South Pacific Congress Organising Committee, presenting the prize for best industrial display exhibit to George Bongiovanni and Ray Sowden of Bayer Diagnostics at the Congress 'ice-breaker' held at the Aotea Centre, Auckland.

meeting in a potentially volatile situation.

The Japanese delegates proposed the following statement which was unanimously supported and adopted by the General Assembly of Delegates. "To ensure the patients life and health we strongly recommend clinical laboratory test services and the important diagnostic work both in private and public clinics be done by fully qualified Medical Laboratory Technologists".

The Scientific Programme as is normal for such a congress, was very wide ranging and of course I was unable to attend all of the concurrent fora. I concentrated on those within my area of interest and listened to papers on topics such as Autologous Blood Transfusion, Biochemistry of blood groups, recombinant erythropoietin, Quality Assurance, Computing in Medical Laboratories, education and an extensive symposium on AIDS, HIV and Hepatitis. Most of these came from Swiss speakers and in the main reinforced information already known, although being the city that contains the headquarters of the World Health Organisation, absolutely up to date statistics were available especially when papers on AIDS were presented. I was wanting to attend the New Techniques symposium but as it coincided with the pre GAD meeting, I took the opportunity to talk at length with the presenters in this symposium, at a later stage.

The most interesting was the demonstration of the Diamed Gel technique where a card of plastic tubes with gel and an appropriate antisera, sera or antihuman globulin has red cells added. After incubation and centrifugation agglutinated red cells sit on top of the gel or are suspended in the gel whilst unagglutinated cells fall to the bottom. The results are clearcut and the coombs test requires *no washing*.

Poster presentations covered a wide variety of topics and there were always groups of people debating points made in the posters. This contrasts with most poster presentations I have seen where the posters are read and then ignored. The Trades displays covered a large area of the conference centre and would be the equal of but no better than those of our own NZIMLS Scientific Meetings. However most companies were giving out Swiss chocolates from their stands. Waist lines were very stretched throughout.

We left Switzerland via Basle and entered Germany and the Black Forest staying in a camping ground billed as the best in West Germany at Munsterthall: — complete with a 25 piece Bavarian Omp-pa-pa orchestra which played for about three hours for all the campers. We saw the reception that followed a local wedding with smashed crockery, and baby clothes on the line. Throughout Germany there were high concentrations of military troops on the move, mainly United States and German. I assumed that these were preparations for what was to become the Gulf War. Passing through Belgium and Luxembourg we were back in France in the Champagne region of Epernay where we went underground to view some of the 250 million bottles of Champagne produced in a year and stored in 18 kilometres of tunnels dug into the surrounding hills. Visited Paris and Dunkirque before returning to England bemoaning the fact that throughout our travels to date only the English and French Customs wanted to look at our passports. At all other borders we were simply waved on through — the unification of Europe is well advanced.

England and Wales. Watching Hastings rock being made, going down a Welsh coal mine, visiting Stonehenge, Avebury and other historic sites and "chocolate box" villages. The sights and sites of London, seeing the musical Miss Saigon and travelling on British Rail were all surpassed by the week we spent navigating fifty foot of British steel along canals not much wider than the boat and over spectacular Aqueducts at Chirk and Ponteysyllte on the Llangollen canal.

We arrived back in New Plymouth on 8th September 1990 having stopped in on Disneyland, Universal Studios and some extensive shopping in Los Angeles. Our baggage which started out seven weeks before as 46kg was now 114kg along with four very heavy cabin bags.

This is being written six months after the congress and going through our trip diary and videos has brought back a flood of happy memories of a very special time. Once again I would like to thank both the NZIMLS Council and Wellcome NZ Limited, especially Liz Fox for the part they played in making this award possible.

BOOK REVIEW

"Laboratory Quality Management, QC <=> QA"

George S. Cembrowski and R. Neill Carey (1989).

ASCP Press

Reviewed by Andrew Thakurdas, TELARC New Zealand

As James Westgard announces in the Foreword to this book, George Cembrowski and Neill Carey provide the reader with both theory and practical applications of "state of the art" procedures in statistical quality control (SQC) in laboratories. Selected contributions from other authors broaden and extend the theory and practice of quality control (QC).

The first chapter of the book is compulsory reading for everyone involved with the management of laboratories. The conceptual framework for the management of quality (fitness for use on conformance to requirements) is very clearly and quickly explained. After describing quality as todays primary competitive force, the authors discuss the quality characteristics of laboratory tests; turnaround time, accuracy, cost and diagnostic effectiveness. Specifications or quality standards for these indicators of a test's quality once described, must be satisfied in order to meet the needs of users or requesters of laboratory services.

Laboratory services are shown to be a series of events including test ordering, sample collection, identification, transportation, preparation and analysis as well as result reporting. Each step has its own quality characteristics whose quality requirements must be achieved so that the final product, the result, is fit for use. The QC process consists of observing the laboratory's actual performance, comparing the performance with the standard for performance, and then taking action if the observed performance is significantly different from standard — Juran's feedback loop.

For me the key message of the book is in the section headed Error Prevention v Error Detection. In this section the authors lay the foundation for the statistical and clinical principles and techniques that they describe in the rest of the book. Error prevention involves developing a laboratory process inherently capable of meeting the quality specifications required for medical usefulness. Error detection involves the examination of the process for the presence of error. The monitoring activity of QC is error detection. The laboratory has two options, either improve the capability of the process or ensure that errors are detected and contained. A process which inherently produces many errors (relative to the quality specification) requires much more sensitive error detection to maintain an acceptable rate of errors than a process which produces few errors. Traditionally, clinical laboratory QC practices have focussed on error detection; atually a co-ordinated combination of error prevention and error detection is required.

The cost of quality is briefly discussed. The authors quote that on average 25% of consumable costs of clinical laboratories are for quality control. For some assays QC consumables can amount to 50% of the testing effort.

QC when measured in terms of time, consumables, and excess testing is expensive. However, when assessed from the perspective of overall patient care QC is found to be a cost cutting tool. QC reduces the frequency of erroneous test results, which include the costs of repeat testing, duplicate testing, as well as the difficult to document costs of prolonged patient hospital stays, poor patient outcomes and malpractice costs.

The rest of the chapters of the book can be categorised as:

I A SQC definitions, theory, rules, procedures and examples

Chapters 2, 3, 4, 7, 8 and 9.

These chapters cover basic as well as the elaborate calculations applicable in SCQ and trend analysis. The discussion includes definitions of analytical errors and the treatment of components of variation.

Single and multi-rule procedures applicable when using stable control material are explained as well as their performance characteristics in terms of rejecting runs with differing amounts of errors. The analysis is extended to include some applications of predictive value theory to the QC testing situations.

Chapter 8 contains practical examples of the application of all the principles and theory. The "nuts and bolts" of implementing QC procedures is described in detail and condenses all the theory to recommendations of what to do in specific situations.

The authors show how the principles and theory developed for reference sample QC procedures also can be applied to patient data QC procedures.

A chapter on "quality control in haematology" shows that the quantitative theory applies in areas other than clinical chemistry.

I B Complicating factors — Chapter 6

This chapter describes in quantitative terms the effects of between run components of variation on the performance of QC procedures. It documents the effects of data rounding. This chapter is very aptly named.

I C Interlaboratory Quality Control — Chapter 11

Here the quantitative theory is extended into the area of external QC or proficiency testing. It provides much needed guidance in the use and efficiency of proficiency testing schemes. Of particular interest is the discussion concerning statistical limit versus fixed limit schemes and the trends in the organisation of such schemes.

II Medical Usefulness Requirements — Chapter 5

Chapter 5 provides a thorough review of the medical requirements recommended in the technical literature. It is very useful for interpreting current guidelines when designing QC procedures.

III Computers in Quality Control — Chapter 10

This chapter reviews the capabilities that can be expected in micro-processor controlled instrumentation, microcomputer application programmes and laboratory information systems.

IV Accreditation: Benefits to quality management — Chapter 12

In this concluding chapter the authors review the requirements of specific American accreditation agencies and regulatory bodies. They return to the broader issues of quality management and emphasise the role of the laboratory director in leading the laboratory staff in the management of quality.

While providing a fleeting overview of laboratory quality management the authors restrict themselves to their primary subject of statistical quality control by dedicating 8 of the 12 Chapters to it. One hopes that readers do not take home the message that quality management is only about statistics and therefore not for them. In fact, laboratory quality management is a wide field and statistical quality control is a specialist area within it.

TELARC is happy to co-ordinate the purchase of this book from ASCP Press for anyone who is interested in buying it. Please contact Andrew at TELARC ph (09) 523-1045.

Vibrio vulnificus: Two Case Reports.

Jacqueline M. Wright FNZIMLS;
Laboratory, Whakatane Hospital, Bay of Plenty Area Health Board.

Abstract

Two cases of infection with the lactose positive halophile: *Vibrio vulnificus*, serve to highlight the rapid progression of this infection in the susceptible host. One patient required digit amputation because of tissue necrosis and the other succumbed to septic shock within 48 hours of hospital admission; both had alcoholic liver disease.

It is apparent that this potentially lethal organism is present in New Zealand coastal waters.

Introduction

The lactose positive halophile, *Vibrio vulnificus*, is a commensal of the marine environment [1]. The syndromes associated with infection with this organism were first described by Blake *et al*, in 1979 [1], however only one documented isolate has so far been recorded in New Zealand (Personal communication: D. Fraser, New Zealand Communicable Disease Centre).

I report here two cases of infection with *Vibrio vulnificus* detected at the Whakatane Hospital in 1990 and review clinical and laboratory findings for this organism.

Case 1.

In February 1990, a 36 year old man was seen at the accident and emergency department 12 hours after he had fallen on his outstretched hand into a tidally affected river. The patient had a previous history of alcoholic hepatitis and on this admission, his gamma glutamyl transferase level was 135 IU/l (normal range: 5-51 IU/l).

On examination, the right hand was acutely swollen and tender and a minor abrasion was noted on the hand. Possible diagnoses included: impacted fracture and compartment syndrome. An X-ray showed no fracture and the patient was commenced on Flucloxacillin.

The following day the right hand was grossly swollen and the index finger was beginning to discolour; a fasciotomy was performed and Augmentin commenced.

No improvement was noted and the finger was amputated on the third day, treatment was again altered this time to Cephradine. Post amputation, the right arm became grossly swollen and a swab taken from the amputation site showed numerous Gram negative bacilli on direct Gram stain. Gentamicin and Metronidazole were commenced on the basis of this report. Culture of the swab yielded a heavy pure growth of an organism which was identified as *Vibrio vulnificus* using the ATB 32E strip (API Systems France) and this identification was confirmed by the New Zealand Communicable Disease centre (NZCDC).

The patient was discharged after a total of 15 days hospitalisation.

Case 2

In April 1990, a 51 year old man presented at the accident and emergency department with a trauma ulcer on his leg. He had a previous history of alcoholic hepatitis and his gamma glutamyl transferase level on admission was 241 IU/l.

The initial injury occurred one week previously when he struck his leg on a trailer hitch.

On admission the ulcer was sore, swollen and weeping and poor foot pulses were noted. The initial diagnosis was superficial thrombophlebitis with deep vein thrombosis and prescribed treatment included: heparin, Augmentin and saline rinses of the ulcer.

The following day the patient exhibited signs of acute renal failure and septic shock. He suffered a cardiac arrest and died early on the third day. The only bacteriological specimen received was a swab taken from the ulcer at time of admission.

This yielded: *Vibrio vulnificus, Pseudomonas aeruginosa* and *Aeromonas sobria*. Unfortunately the Vibrio failed to survive stock culturing and the identity was not confirmed by the NZCDC.

It was later noted that the patient had been collecting kina (sea eggs) with his family early on the day of admission.

Discussion

Environmental studies [2-5] have shown that *Vibrio vulnificus* is part of the normal bacterial flora of the United States coastline. The organism is concentrated in filter feeders such as clams, mussels and oysters, and its presence in coastal waters is not related to faecal contamination. A three year study of the waters of Long Island sound [5] demonstrated that *Vibrio vulnificus* is only isolated when the water temperature exceeds 17°C.

Infection with *Vibrio vulnificus* can be initiated in three main ways: ingestion of uncooked, infected shellfish; contact of an existing wound with contaminated seawater; and a primary wound sustained in the marine environment [6].

Clinical syndromes include primary septicaemia; and wound infection, with or without secondary sepsis [1,6]. Primary septicaemia follows ingestion of raw or partially cooked infected bivalves. Sepsis is due to the organism's direct invasion of the bloodstream via the gastrointestinal tract. Initial features include fever, malaise and chills, and hypotension is a predictor of high mortality. Necrotising vasculitis and secondary skin lesions may develop — the later usually within two days of symptom onset. These lesions may be culture positive for *Vibrio vulnificus*. Primary septicaemia has an associated mortality of 50%.

Wound infection may be due to a wound being sustained in the marine environment, (case 1); or, contamination of a pre-existing wound (case 2). Incubation time between exposure and symptom onset is often less than 24 hours. In a reported study of 17 cases of *Vibrio vulnificus* wound infections [6], four patients died and the median time between exposure and death was 4.5 days.

A report from 1974 [7], describes an infection with a "halophilic non-cholera vibrio" which resembles Case 1. The patient cut his thumb on a shrimp shell and within hours, the thumb was swollen and painful. A fasciotomy was performed and although no pus was found, a swab was taken for culture. The isolate was cultured from this swab and the patient responded to Gentamicin therapy.

It is apparent that in vitro susceptibility results may not predict therapeutic outcome. The isolate from Case 1 exhibited large zones of inhibition to Ampicillin, Cephradine and Augmentin, however neither Cephradine nor Augmentin showed therapeutic effect. The duration of treatment with each of these agents may have been inadequate to promote a recognisable improvement, however, it has been demonstrated that regardless of in vitro results, *Vibrio cholera* may not respond to Ampicillin [8] and for this reason it is suggested that Penicillins not be considered as sole therapeutic agents for *Vibrio vulnificus* [9].

How the organism causes disease is not well understood, but it is evident that the presence of a polysaccharide capsule is strongly associated with virulence [10,11]. Exo-enzymes such as: cytotoxic haemolysin, protease, collagenase, and phospholipase are produced, but their exact contribution to virulence remains undetermined [12].

The organism's exquisite sensitivity to iron may account for disease severity in some patients. Animal studies have shown that the intraperitoneal 50% lethal dose for *Vibrio vulnificus* in mice drops from 10^5-10^6 to 10^2 if the mice are injected with iron prior to bacterial challenge [13,14]. In the human host, capsulated *Vibrio vulnificus* can use transferrin-bound iron if the transferrin is 100% iron saturated. However, in the human host with normal saturation of 30%, iron is not

Footnote:

Ed Note — This paper was published in the NZJ Med Lab Science 1991; 45(2): 55-56. Columns were inadvertently transposed during editing — it is now presented as originally intended by the author.

available for organism use. For this reason haemachromatosis patients are at risk of severe infection with this organism; other at risk groups include patients suffering from haematological disorders, renal failure, diabetes, other immunosuppresive disorders and those on immunosuppressant therapy [1,15,16].

Alcoholic liver disease is a risk factor for severe infection, but the reason for this is unclear. Historically, haemachromatosis has developed in some alcoholics as a result of high iron levels in some alcoholic beverages, however this is rarely seen nowadays [17].

In acute liver disease, iron release from liver ferritin stores may result in elevated plasma iron levels [17] which could predispose to infection with this organism. It has been demonstrated that people suffering from alcoholic cirrhosis exhibit immunosuppression in the form of impaired opsonisation and deficiencies in leucocyte function [18], this may be the more likely explanation.

One other possibility, as yet unexplored in the literature, is whether or not alcoholic liver damage impairs the liver's potential to detoxify *Vibrio vulnificus* exo-enzymes.

Laboratory findings:

After 24 hours incubation on 5% sheep blood agar, *Vibrio vulnificus* appears as a large, grey colony with a small zone of β haemolysis. On McConkey agar the colony size is reduced. The organism may initially be confused with *Streptococcus agalactiae* if an oxidase test is not promptly performed. *Vibrio vulnificus* is oxidase positive and is glucose fermentative (thus distinguishing it from the oxidative Pseudomonads). It grows well at 42°C, which may aid in distinguishing from Aeromonads which often grow poorly, if at all, at temperatures above 37°C.

The specific characteristics of *Vibrio vulnificus* include fermentation of lactose, but not sucrose; indole positive; Voges-Proskauer negative; lysine decarboxylase positive; ornithine decarboxylase variable; and growth in 6%, but not 8% sodium chloride [19]. Exceptions do occur — the isolate from Case 1 was negative for both lysine and ornithine.

In this laboratory, oxidase positive colonies isolated from wound or faecal samples are all tested for glucose reaction and ability to grow at 42°C; all glucose fermentative organisms are then inoculated in the ATB 32E strip (API Systems, France), a manual identification test, requiring 4-5 hours incubation time. The isolates from both cases reported here gave identification profiles with >99% certainty of *Vibrio vulnificus*, one of which was confirmed as such by the NZCDC. The isolate from Case 2 failed to survive stock culturing and the identification was not confirmed. The inability of *Vibrio vulnificus* to survive stock culturing has been previously described [19].

In environmental studies, thiosulphate citrate bile-salt sucrose (TCBS) agar has been shown to be an effective isolation medium [5].

In conclusion, this organism is capable of causing rapidly fatal infection after glancing exposure. Infection has followed: being bitten by insects in the marine environment; cuts sustained on fish fins and mollusc shells; and wading in estuaries [20].

In this country many potentially infected bivalves are collected and consumed during the summer months and beach swimming is a widely indulged pastime in the summer — thus many hundreds of people may be potentially exposed to the organism.

Why then are so few cases noted?

A likely explanation is that *Vibrio vulnificus* is a relatively avirulent organism which is only capable of causing disease in specific host groups, and it appears that people with no underlying disease or immune-suppression are not at risk.

The severity of infection in the susceptible host is such that the risks of consumption of raw shellfish and of contact with sea water must be publicised to at risk groups. Such publicity must be done carefully so as not to jeopardise coastal tourism. In the United States, families of victims of *Vibrio vulnificus* infection have instigated law-suits against Public Health authorities, citing failure to warn of a known health hazard [12].

Vibrio vulnificus is in our coastal waters so we must familiarise ourselves with the clinical and laboratory aspects of this organism.

References

1. Blake PA, Merson MH, Weaver RE, Hollis DG, Heublein PC. Disease caused by a marine *Vibrio:* Clinical characteristics and epidemiology. *N. Engl J Med* 1979; **300**: 1-5.
2. Oliver JD, Warner RA, Cleland DR. Distribution and ecology of *Vibrio vulnificus* and other lactose fermenting marine vibrios in coastal waters of the southeastern United States. *Appl Environ Microbiol.* 1982; **44**: 1404-14.
3. Tamplin M, Rodrick GE, Blake NJ, Cuba T. Isolation and characterisation of *Vibrio vulnificus* from 2 Florida estuaries. *Appl Environ Microbiol* 1982; **44**: 1466-70.
4. Kaysner CA, Abeyta C Jr, Wekell MM, De Paola A Jr, Stott RF, Leitch JM. Virulent strains of *Vibrio vulnificus* isolated from estuaries of the United States West Coast. *Appl Environ Microbiol.* 1987; **53**: 1349-51.
5. Tilton RC, Ryan RW. Clinical and ecological characteristics of *Vibrio vulnificus* in the Northeastern United States. *Diagn Microbiol Infect Dis* 1987: **6**: 109-117.
6. Klontz KC, Lieb S, Schreiber M, Janowski HT, Baldy LM, Runn RA. Syndromes of *Vibrio vulnificus* infections: Clinical and epidemiological features in Florida cases, 1981-1987. *Ann Intern Med* 1988; **109**: 318-23.
7. Thorsteinsson SB, Minuth JN, Musher DM. Clinical manifestations of halophilic non-cholera *Vibrio* infections. *Lancet* 1972; **2**: 1283-4.
8. Northrup RS. Antibiotics in cholera therapy. *J Pak Med Assn* 1969; **19**: 363-5.
9. Morris JG Jr, Tenney J. Antibiotic therapy for *Vibrio vulnificus* infection. Letter. *JAMA* 1985; **253**: 1121-1122.
10. Yoshida S, Ogawa M, Mizuguchi Y. Relation of capsular materials and colony opacity to virulence of *Vibrio vulnificus. Infect Immun* 1985; **47**: 446-51.
11. Simpson LM, White VK, Zane SF, Oliver JD. Correlation between virulence and colony morphology in *Vibrio vulnificus. Infect Immun* 1987; **55**: 269-72.
12. Morris JG Jr. *Vibrio vulnificus* — a new monster of the deep? Editorial. *Ann Intern Med* 1988; **109**: 261-3.
13. Wright AC, Simpson LM, Oliver JD. Role of iron in the pathogenesis of *Vibrio vulnificus* infections. *Infect Immun* 1981; **34**: 503-7.
14. Morris JG, Wright AC, Simpson LM, Wood PK, Johnson DE, Oliver JD. Virulence of *Vibrio vulnificus:* association with utilisation of transferrin-bound iron, and lack of correlation with levels of cytotoxin or protease. *FEMS Microbiol Lett* 1987; **40**: 55-9.
15. Tacket CO, Brenner F, Blake PA. Clinical features and an epidemiological study of *Vibrio vulnificus* infections. *J Infect Dis* 1984; **149**: 558-61.
16. Johnston JM, Becker SF, McFarland L.M. *Vibrio vulnificus:* man and the sea. *JAMA* 1985; **253**; 2050-3.
17. Zilva JF, Pannall PR. Iron metabolism. in *Clinical Chemistry in Diagnosis.* London: Lloyd-Luke (Medical Books) ed **4**, 1984. pp 413-430.
18. Rajkovic IA, Williams R. Abnormalities of neutrophil phagocytosis, intracellular killing and metabolic activity in alcoholic cirrhosis and hepatitis. *Hepatology* 1986; **6**: 252-62.
19. Farmer SJ III, Hickman-Brenner FW, Kelly MT. Vibrio. *In* Lennette EH editor-in-chief: *Manual of Clinical Microbiology.* Washington DC: American Society for Microbiology, ed **4** 1985. pp 282-301.
20. Howard RJ, Lieb S. Soft tissue infections caused by halophilic marine vibrios. *Arch Surg* 1988; **123**: 245-9.

N.I.C.E. Weekend 91

"NICE" is an abbreviation of "National Immunohaematology Continuing Education" and the NICE Weekend meeting comprises a weekend of presentations on many aspects of Transfusion Medicine. Everyone who attends must present a 1 to 5 minute paper or poster. It is this participation by all of the delegates which leads to the success of the meeting and provides a very supportive atmosphere for those people who are presenting for the first time. This is definitely a meeting not to be missed. You have only to ask someone who has attended one to have this confirmed.

A number of companies have assisted this meeting with cash donations and their help is greatly appreciated.

Abbott Laboratories present an annual award to the presenter of the best paper. The winner of this award receives an all expenses paid trip to the NZIMLS conference later in the year to present their paper again. The two recipients of the Abbott NICE Award so far have been Ailsa Signal who was presented the Award in 1990 for her paper on Burnout in Blood Bank Technologists, and Alison Dent from Auckland who received the Award in 1991 for her paper "Welcome to PEG". Thanks must go to Abbott Laboratories for this generous annual award.

Nice Weekend 92 will be held at the Wairakei Resort Hotel on 25 and 26 April 1992. Swimming togs are a must for this venue as they have a wonderful outdoor hot pool which is the perfect place for discussing Cold Haemagglutinin Disease on a frosty evening.

Registration forms will be distributed with the NIP Survey later in the year and as numbers will be limited, it will be essential to register early.

If you are not a NIPS participant and would like to receive a registration form you should write to:

David Wilson
Charge Technologist
Department of Transfusion Medicine
Palmerston North Hospital
Private Bag
Palmerston North

In April this year the second NICE Weekend was held in Nelson. This year the meeting was attended by 23 delegates who presented papers on a variety of topics. The following are the abstracts of those presentations:

Platelet Shimmering

Tirath Lakshman. The Blood Centre, Wellington Hospital. Wellington.

Some aspects of a non-invasive test for the quality assessment of stored platelet concentrates.

Should Anti-D Immunoglobin Be Given Routinely To Rh Negative Women Antenatally at 28 Weeks Gestation As Well As Postnatally?

Marie Willson, Blood Bank, Gisborne Hospital, Gisborne.

In Australia, Canada and the U.S.A., Anti-D Immunoglobulin is given at 28 weeks; in the U.K. injections are given at both 28 and 34 weeks. There has been controversy as to whether antenatal prophylaxis is cost effective. This talk looks into the cost effectiveness of this practice in a small city.

References:
Transfusion Vol 30 Nvs- 1990
International Society of Blood Transfusion
I.S.B.T. Technical Guide
Haemolytic Disease of the Newborn Prevention and Management - 1988

Stem Cell Transfusion. A New Alternative To Bone Marrow Transplantation

Lisa Bridson, Auckland Regional Blood Centre, Auckland.

Bone marrow transplants are used in various disease states to replace the haematopoietic stem cells in the marrow.

Umbilical cord blood is another rich source of stem cells and is usually discarded.

It was suggested that cord blood could be used as an alternative source for haematopoietic reconstitution. An international study has been done to evaluate cord blood for transplantation . At least three patients have been successfully transplanted.

These case studies will be discussed to evaluate the future of this technique.

Gel Test for Red Cell Antibody - Antigen Reactions

Roger Austin, Immunohaematology. Taranaki Base Hospital, New Plymouth.

A "new" method of blood grouping / antibody screening will be demonstrated and discussed.

Immunoassay Systems for Immunohaematology

Katya Dmitrieff, Hoechst (NZ) Ltd, Auckland.

In less than five years, PB Diagnostic Systems, Inc (PBDS) of Westwood, MA, has progressed from a promising research project to an exciting new corporation of almost 250 people. As a joint venture between Polaroid Corporation, the international innovator in instant film technology and Behringwerke AG, a pioneer in immunochemistry and the subsidiary of Hoechst AG of West Germany, PBDS boasts the energy levels of a start up company as well as the financial resources necessary to develop technologies that meet the demands of the '90s for more efficient clinical laboratory products.

Research led to breakthrough reagent technology that resulted in the OPUS. A dry film, multi-layer format, self contained in a $1^1/_2$" test module was also adapted by scientists to the fluorogenic ELISA technology so it too would be housed in the modular format with all the reagents necessary to run each assay.

The above integration allows "OPUS" to handle a broad range of assays including infectious diseases.

MAb -D 2 B

Lorraine Rimmer, Auckland Regional Blood Centre, Auckland.

A good quality reliable Anti-D is a vital reagent in any blood bank.

The evaluation and selection protocol used at the Auckland Regional Blood Centre is reviewed and the pros and cons of reagents currently available discussed with particular reference to a newcomer on the market - Monoclonal Polyclonal Anti-D.

The advantage of this reagent is the selection of a clone that produces very avid IgM antibodies. In addition to this a small amount of pooled human polyclonal IgG anti-D is added to detect the weak D+ (D^u) by the Anti-Human Globulin Test.

The Manufacture and Assessment of AHG Reagents

Will Perry, Salmond Smith Biolab, Auckland.

Anti-Human Globulin (A.H.G.) Reagents are very easy to manufacture. High quality A.H.G. Reagents which satisfy requirements of the F.D.A., I.S.B.T., current trends and, most importantly, end users are more challenging. Some aspects of A.H.G. production will be discussed, particularly the use of Monoclonal antibodies, as a replacement for the traditional rabbit serum.

Variations on a Theme

Sheryl Khull, The Blood Centre, Wellington Hospital, Wellington.

A "fussy" antibody demonstrates that not all LISS techniques are created equal.

Welcome to PEG

Alison Dent, Auckland Regional Blood Centre, Auckland.

Yet another method of antibody screening which gives us increased sensitivity by Indirect Coombs technique, with a reduction in incubation time.

Advantages are a reduction in serum: cell ratio, the elimination of the need for LISS Techniques, and thus a saving on cost, and the suitability of this method for all antibody screening and identification procedures.

A Major Foul-up

Dianne Griffiths, Immunohaematology, Napier Hospital, Napier.

An ABO incompatibility which could have been prevented.

The wrong patient was bled as a result of not checking the patient's wristband. Mrs S "S" was therefore bled and the tube labelled Mrs R "S" from the request form. A request for units of blood meant that A Negative units were crossmatched for Mrs R "S" who was O Positive. The end result was ...

CHD - A Case Study

Ailsa Signal, Department of Transfusion Medicine, Palmerston North Hospita,l Palmerston North.

Cold Haemagglutinin disease belongs to the Antibody Induced Haemolytic Anaemias.

Idiopathic CHD is usually a disease of the elderly. A case involving a 16 year old girl is presented and discussed.

Delayed Transfusion Reaction - A Case Study

Lisa Wardill, Immunohaematology, Southland Hospital, Invercargill.

An elderly man was admitted to hospital with chest pain and mild anaemia. The patient was transfused and some days later a falling haemoglobin and other symptoms led to a full investigation for a delayed transfusion reaction.

Significant Haemolysis

Greg Baker, The Blood Centre, Wellington Hospital, Wellington.

A case study elucidating the identity of a haemolysing antibody to high incidence red cell antigen.

HDN Due to Anti-S

Max Love, Immunohaematology, Hutt Hospital, Lower Hutt.

This antibody caused mild HDN which did not require exchange transfusion and was chiefly notable for its inability to react in an eluate prepared by the chloroform method although detectable in a saline heat eluate.

A Computerised Blood Management System In A Medium Sized Laboratory

Raewyn Clark, Blood Bank, Rotorua Hospital, Rotorua.

An ICS Blood Bank / Blood Donor programme was adapted for use in a medium sized laboratory where non blood bank people are involved in shift and call.

The system came into operation in October 1990.

Training schedules and disadvantages of the system are reviewed.

Computerisation - Pleasure or Pain

Lindsey Browning, Immunohaematology, Southland Hospital, Invercargill.

A brief look at the selection, installation and introduction of computers to a blood bank.

Who Got What

Geoff Herd, Immunohaematology, Northland Base Hospital, Whangarei.

An approach to the problems associated with the documentation and end use of blood products is presented. An IBM compatible PC with barcode reader and commercial software have been installed to address this section of day to day blood bank activities. The advantages of this system are its simplicity and low capital cost.

10 Months Later

Tony Mace, Laboratory, Waipukarau Hospital, Waipukarau.

It has been approximately 10 months since the last NICE Weekend and I have asked myself "what did I learn?" In this fast changing world - not only of technology but also of Health Ministers - did I eventually change anything?

One of the Most Important Aspects in the World ...

Yvonne Geeraedts, Immunohaematology, Taranaki Base Hospital, New Plymouth.

The relationship between quality and documentation and the application of a documentation control system in blood bank.

Success Lies in Blood and Bone

Warwick Henry, Blood Bank, Nelson Hospital, Nelson,

..... well so the farming press and the home gardener would lead us to believe. But what about an Immunohaematology lab where the accreditation of blood donations is similar to that of donated femoral heads.

Can We Do It Better?

We are consumers of a wide range of products and service. Good Quality Assurance requires that we should evaluate or receive proof of attainment of acceptable standards of products before we put them into use. Financial, staffing and time constraints and sometimes knowledge gaps mean that this is not done in a lot of cases and buying is based on the sales pitch. We also have products inflicted on us through a national tender for which the customers have not been consulted (or have been ignored).

Can these problems be overcome by the use of a national tender into which we can have some input? Should suppliers be required to provide independent evaluations of their products to assist the consumer? How do we differentiate between hype and fact.

The Transfusion Science Special Interest Group

David Wilson, Department of Transfusion Medicine, Palmerston North Hospital, Palmerston North.

The special interest groups (SIG) were established by the Institute in order to form an advisory body to council on topics pertaining to that discipline, this being of importance when a particular discipline was not represented on council.

This brief presentation is aimed at informing you of the purpose of the SIG and to give you an opportunity to let the SIG know what you want and expect from it.

The Pacific Way

South Pacific Congress

The 3rd South Pacific Congress was held in the Aotea Centre, Auckland, New Zealand on the 28th - 30th August, 1991. The Organising Committee was delighted to welcome approximately 15 Pacific Island delegates to the Congress from various islands in the Pacific.

Monica Cheesbrough from Tropical Health Technology, who was a keynote speaker at the Congress, chaired three sessions for the Pacific Island Delegates. Each delegate was asked to greet the audience in his/her own language, and outline teaching programmes in his/her own country. The participation of the Pacific Island Delegates in these sessions was most welcome and augers well for future South Pacific Congresses.

This was an important milestone for the South Pacific Congress as it was the first time such forums were included and run concurrently with Microbiology, Haematology, Biochemistry, Immunohaematology etc.

Since the Congress, ways and means of improving these sessions have been discussed and suggestions will be forwarded to the Australian organisers of the 4th South Pacific Congress to be held in Brisbane in 1995. Suggestions from any Pacific Island Delegates who were at the Congress would be most welcome.

To All Pacific Island Technologists

This page ("The Pacific Way") is to inform people about aspects of Medical Laboratory Technology in the Pacific region. The best people to do "the informing" are those living and working there. If you have any items of interest, news, or better still, an article or case history, please forward to The Editor, P.O. Box 9095, Newmarket, Auckland, New Zealand.

Tropical Health Technology

This Organisation run by Monica Cheesbrough is a non-profit organisation formed to assist developing countries in the field of medical laboratory sciences by providing low priced publications, learning aids and microscopes.

Tropical Medicine Microscope

Special Features

- to meet the high demands of tropical medicine work, the optics, illumination system, focusing and mechanical stage are of high quality and modern proven design with metal body for trouble-free long life.
- mains, electricity and battery powered with solar panel option for battery charging.
- exceptionally easy to use with enhancements not found on any other microscope, ensuring best use of the microscope even by those with limited technical experience. An easy to understand microscope manual is provided.
- 70 colour plates with text are provided to help laboratory staff and primary health care workers diagnose tropical diseases microscopically.
- low price available to developing countries.

The Tropical Medicine Microscope (TMM) has been developed by Gillett and Siebert with the assistance of Tropical Health Technology.

Availability

For ordering and enquiries re textbooks, learning aids and microscopes, contact:

Tropical Health Technology
14 Bevills Close, Doddington March,
Cambridgeshire, PE15 0TT, United Kingdom.

Item of Interest — ASPECT

(Australian South Pacific Eye Consultant Teams)

ASPECT was formed following visits since 1971 of Australian Opthalmic Surgeons to the Solomon Islands. In 1984, the Australian International Development Assistance Bureau (AIDAB) decided that it wanted to provide a multi-country medical aid programme, and so, ASPECT was born, largely with the existing team members who had been involved in the original Solomons project. The programme involved Vanuatu, Solomons, Kiribati and Tuvalu.

Since that time, the Solomons have taken on their own eye specialist, and the programme has been extended to cover Tonga and the Cook Islands.

Problems addressed by ASPECT include

- Cataracts, which have to be removed by surgery and the patient fitted with special glasses.
- Pterygium. This is common in the region and is directly related to sunlight — to high ultra-violet exposure areas. It leaves fleshy scars on the surface of the eye which irritate and sometimes block vision.
- Trachoma. This occurs in nearly 40% of school children in the Pacific in the 10-14 year age group. Trachoma is an eye infection caused by the organism "Chlamydia". Trachoma is prevalent where flies are numerous. It causes redness and irritation with a rather watery discharge. It only causes serious eye problems with repeated infection.

NEW PRODUCTS AND SERVICES

SAFECUT 7000 CRYOSTAT PASSES INDEPENDENT TEST

Antec International, one of the leading manufacturers of disinfecting agents for laboratory use, has released the results of its own tests on the self-cleaning Safecut 7000 cryostat, made by the Bright Instrument Company Limited. They confirm the claims of the cryostat's manufacturers that the decontaminating system is effective.

Chief Chemist at Antec, Mr Mark Squire, took one of the cryostats to his laboratory and carried out his own independent assessment using Antec's "Virkon", the disinfectant with the widest proven spectrum available. He artificially contaminated the cryostat with a culture of *Pseudomonas aeruginosa*, then operated the instrument's standard decontamination programme. Afterwards he swabbed various parts of the mechanism and tested both the disinfectant and rinse fluids.

The results were unequivocal. Neither the Virkon solution nor the rinse showed any bacterial presence and only one colony could be grown from the twenty-four cultures made from the swabs taken. This single colony was almost certainly due to airborne contamination of the cultures.

Mark Squire says "We had been advising Bright on the use of Virkon, but because this is such a new application we wanted to see for ourselves that it really worked. Of course, we knew Virkon would kill this organism but I wanted to be sure that the entire cryostat chamber was decontaminated. So we deliberately took swabs from the most inaccessible places. We are now convinced that the system is effective."

The Safecut 7000 cryostat is designed to be used in pathology laboratories to cut frozen sections of fresh tissues which may have come from infected patients. It is the only cryostat in the world which uses a safe and odourless liquid decontaminant. As concerns over the risks of handling unfixed human tissues grow, the results

of these independent tests will help reassure laboratory scientists that there is an instrument available which will minimise these risks.

For further information contact; Wilton Instruments, P. O. Box 31044, Lower Hutt, Phone (04) 697-099, Fax (04) 697-240.

SARTORIUS BALANCE AND MAGNETIC STIRRER ALL IN ONE UNIT

What has been considered wishful thinking in technology until now — combination of a balance and magnetic stirrer in a single unit — has just been transformed into reality by Sartorius design engineers. This two-in-one model will be premiering at the ACHEMA '91 in Frankfurt, Germany.

For the first time ever, users are offered the capability of stirring liquids and weighing accurately down to 0.01g, all at the same time. The new magnetic stirrer balance saves users the annoying "footwork" of having to move back and forth between a conventional balance and a magnetic stirrer. Moreover, it saves time and makes formulation and preparation of solutions considerably easier. In this way, the unit allows users to avoid hassles in preparing solutions from components that tend to clump if they are not stirred fast enough, because they now can be weighed-in precisely and stirred quickly before they have a chance to cause problems.

The magnetic stirrer is integrated into the weighing pan. The unit's effective shielding from magnetic fields and the powerful signal filtering by MC1 technology ensure that the weight readout is not affected by the magnetic field of the stirrer or by the motion of a liquid as it is being stirred. The stirrer is operated by the control panel on the balance.

The standard IAC - short for "Integrated Applications Computer" — supports users as they operate the magnetic stirrer balance. The IAC offers practically written and easy-to-run application programmes, such as statistics, formulation, and entering ID numbers for batches and operators.

Since the stirrer and balance functions can also be controlled via the standard, built-in RS232C/423 interface, this magnetic stirrer balance can be readily integrated into laboratory automation systems. As for additional features, both the stirrer and balance functions can be used independently.

For further information contact;

Wilton Instruments, P. O. Box 31044, Lower Hutt, Phone (04) 697-099, Fax (04) 697-240.

BENCH-TOP REFRIGERATED CENTRIFUGE

The Mistral 3000i, from MSE, is the bench-top refrigerated centrifuge designed to meet the challenges of the future.

With angle rotors, top speed for the Mistral 3000i is 6000rpm, with a maximum RCF of 6030g. Using the 4 x 750ml windshielded swing-out rotor, it can spin a full 3 litre load in a sealed condition to over 3000g.

Microprocessor controls ensure precise, concise measurements and simplicity of use. The well-designed control panel means it is easy to set up all parameters and the bright, easy-to-read LED display enables all parameters of a given run to be reproduced accurately. Up to 9 runs can be stored in battery protected memory, and quickly recalled through a very straight-forward routine.

The Mistral 3000i is driven by a brushless induction motor for long life, fast acceleration and accurate speed control. There are 10 programmable rates of braking, including brake-off.

Also incorporated is automatic rotor recognition which enables the operator to obtain a direct RCF reading at any time during the run. It means that it is impossible for the operator to set a speed beyond the safety limit for that particular rotor.

The timer can be set in 0.1 minute steps to 99.9 minutes, and thereafter in 1 minute stages to 999 minutes. A hold mode is available for even longer runs.

Safety features on the MSE Mistral 3000i include: a lid interlock, rotor imbalance detection and protection system, a heavy duty steel guard ring and a counter-balanced lid.

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For further information contact;

Labsupply Pierce (NZ) Ltd, P. 0. Box 34-234, Auckland, Phone: Auckland (09) 433-5867, Christchurch (03) 358-7410.

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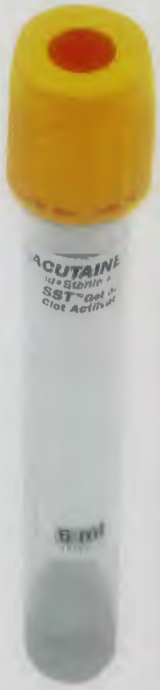

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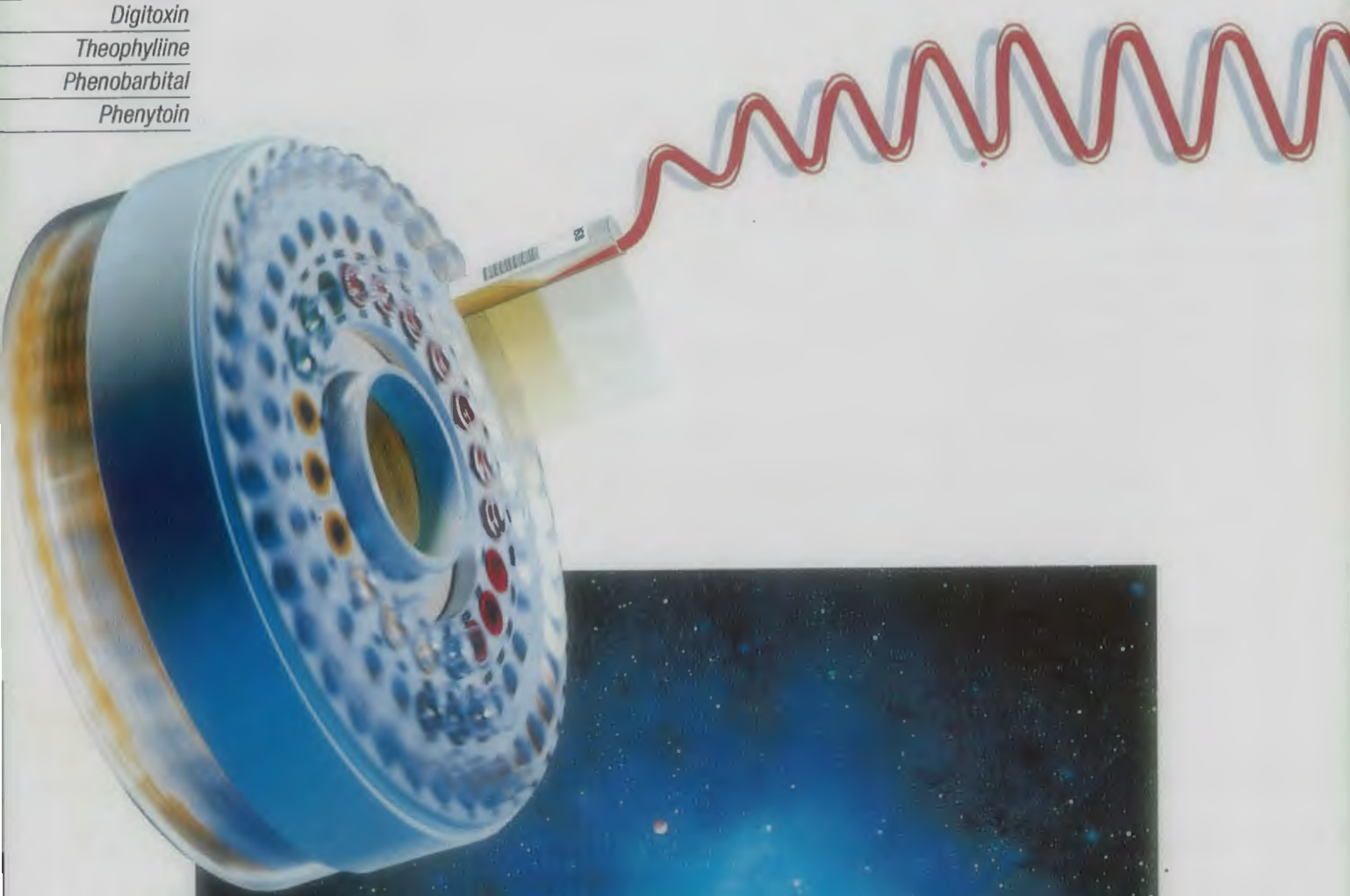

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