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Intending contributors should submit their material to the Editor, M. Gillies, Microbiology Laboratory, Auckland Hospital, Auckland, New Zealand. Acceptance is at the discretion of the Editor, and no undertaking is given that any article will be published in a particular issue. The copy deadline for each issue is the first of the month prior to the month of publication.

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DATES OF PUBLICATION

The months of publication for 1992 are March, May, August and November.

CURRENT COMMENT

A DEGREE IN MEDICAL LABORATORY SCIENCE — PASSPORT TO THE FUTURE

E. Norman
Rotorua Hospital

We must all by now be aware that our current Technical Institute based Training Courses have meant that we are growing increasingly remote from our colleagues overseas, most of whom now have degrees in Medical Laboratory Science. This loss of reciprocity is of real concern.

In New Zealand it is now necessary to have a basic science degree to be able to successfully apply for positions involving routine basic chemistry and microbiology in industries such as dairying, the freezing works and other food manufacturing. A BSc or BSc with honours may enable employment as a technician in a university molecular biology department and applicants who do not have Masters Degree are precluded from applying for a position to undertake routine microbiology on fish. An honours degree or PhD in chemistry or biochemistry is needed to undertake routine toxicology for the D.S.I.R. These and many other similar examples clearly indicate that the need to upgrade our education to degree status is now no longer desirable, it is an urgent necessity.

Despite this there are some in our profession who have difficulty adjusting to the fact that they should be able to think of themselves as Laboratory Scientists and also a number who consider that a degree would result in over qualification for what they consider to be an essentially practical job.

Unfortunately the professional inferiority complex which has pervaded our profession through the years is, for some, still a powerful force.

Our degree course will open the way to postgraduate education at Masters and PhD levels resulting in a school of Medical Laboratory Science which will ultimately be headed by a Medical Laboratory Scientist. Thus we will be able to produce our own academic leaders who will be in a position to help shape the future educational directions and needs of our profession. It has never been logical that Laboratory Scientist training should be remote from the Schools of Medicine and Pathology. It is a basic and integral part of medicine. Pharmacy and Physiotherapy are now integrated.

Pathology plays the role of bridging the gap between basic science and clinical practice. At a time when different frontiers of science are pushing out rapidly it is important that Medical Laboratory Scientists and Pathologists be actively involved as part of a team.

At present it appears to be increasingly evident that the gap between Pathologists and Laboratory Scientists is widening. This was noted by Professor Peter Herdson when he wrote in a recent bulletin of the Royal College of Pathologists of Australasia that, *"Another continuing problem for the College is the tendency for many Pathologists not to pursue their vital role as Consultants.*

The appropriate Consultant interface between the highly technical and sophisticated laboratory on the one hand and the needs of the patient and his or her attending doctor on the other. So often this vital role is omitted and consequently there is the ever present threat of takeover either by highly qualified technical staff or by other Physicians."

He also noted that "*It is a worry that during the 1980's the number of trainees has diminished and the ever present threat of Laboratory Technologists or of Clinicians encroaching on the pathology domain looms large*". This is happening already as, increasingly, laboratory staff are being consulted by junior and often senior medical staff, especially in medium and small-sized laboratories where there are no Specialist Pathologists.

It is unlikely that the number of Pathologists training will increase in the near future and it is known that a significant number of senior Pathologists will retire in the next few years. It is unlikely that those Pathologists who are still in practice will move back to fill the gap which Professor Herdson has so clearly identified.

The degree course will provide a much better background in subjects such as anatomy, physiology and cell biology and thus it will provide a platform from which we can play our part in helping to fill the consulting role. It is a tribute to those Laboratory Scientists who have gained the knowledge to enable them to fill this role as our various training schemes up to the present have not adequately fitted them for the role.

The opportunity is there if we are willing to take it. We will not be Pathologists, but a better educated Laboratory Scientist will surely play a significant part in patient care in the future. We must start now building toward the time when Laboratory Scientists and Pathologists have developed a relationship of partnership.

The degree course will produce people who are able to have some influence in the training of doctors. In the USA it has been noted that a laboratory course in the Medical Schools has had the ultimate effect of improving the laboratory utilisation habits of medical staff.

It is clear that in the future Medical Laboratory Scientists will be better equipped to play their part in the Health Care Team. They will need to be to confront the rapid technicological changes which will occur. Some of the many areas in which advances are being made include immunohistochemistry, flow cytometry immunopheno-typing, monoclonal antibody technology and nuclear DNA analysis.

Developments will parallel increases in medical knowledge and the new techniques will supplement rather than replace current laboratory techniques. In the future the Medical Laboratory Scientist will be increasingly required to move outside the laboratory in order to influence test ordering behaviour, to closely examine the clinical relevance of some laboratory procedures and practices, to set criteria for appropriate test use and to provide an input into the interpretive content of laboratory results. The role of providing an interface between the laboratory and other medical and para-medical disciplines, and between the laboratory and the patient will assume greater importance.

The financial restraints which are now starting to bite in most laboratories are not a passing phase, but will be with us well into the future and hence the days of forms with vast numbers of tick boxes providing work for large multi-channel analysers are fast disappearing. The Medical Laboratory Scientist of the future will need to look beyond the boundaries of quality control as many currently practice it and to become involved in quality assurance in the broad sense of Total Quality Management. A further change now starting which will almost certainly accelerate in the future is the trend for technology to move out from the laboratory and closer to the patient. Laboratory Scientists must ensure that they are in a position to move also, as failure to do so will result in a significant undermining of the profession.

The role of the Laboratory Scientist has changed significantly from what it was 20-30 years ago and given an ever increasing rate of change it will be very different by the turn of the century.

Our current training system is not able to prepare us adequately for an expanded future role.

If we fail to plan for the future by not fully supporting the degree course we are likely to consign the profession of Medical Laboratory Science to mediocrity for a long time to come.

47th ANNUAL SCIENTIFIC MEETING N.Z.I.M.L.S. PLAZA INTERNATIONAL HOTEL WELLINGTON 27TH 28TH AUGUST

PRELIMINARY PROGRAMME:

Wednesday 26th August:

WORKSHOPS:
Anti Nuclear Antibody (ANA)
Others to be confirmed.

SOCIAL:
Wine and Cheese.

Thursday 27th August

OPENING CEREMONY
General Forum.
Extra Laboratory Testing
Forum
NZIMLS Annual General Meeting

Friday 28th August

CONCURRENT FORUMS:
Biochemistry
Immunology
Haematology
Cytology
Microbiology
Histology
Transfusion Science
CLOSING CEREMONY.

CONCURRENT FORUMS.

Seminars are being organised to include:
HIV Testing - Guest speaker,Dr Elizabeth Dax,
HIV Reference Laboratory,
Fairfield Hospital,Melbourne
Respiratory Tract Infections - Guest speakers,
Dr Michael Humble and Dr Mark Jones, Wellington Hospital.

47th ANNUAL SCIENTIFIC MEETING N.Z.I.M.L.S.

ABSTRACTS FOR 1992 NZIMLS SCIENTIFIC MEETING

You are invited to submit 15-20 minute papers for the above meeting.
Abstracts must be typed in single space type in less than 250 words.
These must be free of grammatical and typographical errors.
The original and two photocopies should be submitted.

ABSTRACTS SHOULD INCLUDE:

Title (In Capital Letters)
Author's Names
Abstract Content

Also include presenting Author's name, address, telephone number and any visual aids required, e.g. 35mm Slide Projector, Overhead Projector.

Abstracts should be submitted before 30 June 1992.

THE CONFERENCE SECRETARIAT,
MICROBIOLOGY,
DEPARTMENT OF LABORATORY SERVICES,
WELLINGTON HOSPITAL

Poster presentations will also be accepted for the conference.
The submission date is 30 June 1992

New Zealand strains of *Giardia intestinalis* from humans: first isolations, culture and growth characteristics.

Tim J Brown, PhD; George Ionas, PhD; Sally J Miller, MSc; Michael C Tonks, MSc; Philip J Kelly, Dip. Hort.
Massey University / NZ Health Department *Giardia* Unit, Department of Microbiology and Genetics, Massey University, Palmerston North, New Zealand

Address for correspondence: Tim J Brown

Abstract

The first laboratory axenic cultures (eight strains) of *Giardia intestinalis* in New Zealand were established. They resulted from 129 attempts from 64 human faecal sources from seven New Zealand towns.

Using two New Zealand strains of *Giardia intestinalis* (Hast/87/MUGU/68 and Whan/87MUGU/48), the influence of pH, temperature, reducing conditions on axenic growth and trophozoite attachment was investigated. Variations in both temperature and pH were shown to affect the *in vitro* growth rate of the two strains examined. Optimal growth of both strains occurred at 37°C and growth continued at a reduced rate, approaching both 30°C and 40°C.

These strains exhibited marked variation in growth depending on pH, with growth optimal at pH 6.75-7.50 and a rapid decline in growth rates outside this range. A strong correlation was demonstrated between reducing conditions and the growth of these strains in culture. Enhanced growth of experimental cultures was directly related to increases in the concentration of L cysteine. Under elevated L-cysteine conditions (0.15-0.25% w/v) trophozoite attachment reached maximal levels (85-95%). In the absence L-cysteine, attachment of trophozoites in culture continued at a reduced rate (40-50%).

Trophozoites of the two strains of *G. intestinalis* displayed a similar oxygen sensitivity at 37°C. A slow decline in culture viability was recorded upon exposure of trophozoites to 4.0-6.0 ppm dissolved oxygen (DO). Rapid exponential killing of cultures occurred after exposure to 8.0ppm and 12.0ppm DO. Consumption of oxygen by Hast/87/MUGU/68 was demonstrated. Dissolved oxygen was removed by trophozoites from the culture medium at a rate of 3.2-5.3 x 10^{9} ppm/cell/hr.

Key Words

Giardia, New Zealand strains, Culture, Growth characteristics.

Introduction

G. intestinalis, the causative agent of giardiasis, parasitises a number of vertebrates including man; and has a worldwide distribution. Although giardiasis is now widely recognised as a public health concern problem, little is known of its aetiological agent.

Giardia intestinalis is not an easy organism to culture (1,2). *Giardia* has been reported in New Zealand in humans (3,4) from which source cultivations are rarely made. It is essential to have New Zealand isolates of *Giardia* in addition to imported strains to provide cysts and trophozoites for experimentation. This study sets out the methods used for the first isolations and culture of *Giardia* from human sources in New Zealand and the growth characteristics of selected isolates. A modification of the excystment medium to replace the bovine serum content component with a 50:50 foetal calf and bovine serum mix was found to be most successful for excystment. Eight positive cultures were established from 129 attempts from 64 human faecal samples taken from seven towns in New Zealand — Palmerston North, Hamilton, Hastings, Whangarei, New Plymouth, Tauranga and Blenheim.

Two New Zealand strains, one from Hastings (Hast/87/MUGU/68), and one from Whangarei (Whan/87/MUGU/48), were chosen for examination of their growth characteristics and *in vitro* requirements.

The correlation between pH and temperature in *in vitro* growth of the New Zealand isolates axenic cultures was investigated. The importance of L-cysteine as a component in the complex growth medium was also demonstrated.

Trophozoites of *G. intestinalis* are aerotolerant to a limited degree, although elevated dissolved oxygen (DO) levels in complex growth medium have been shown to be lethal to trophozoites (5). *G. intestinalis* may be described as an organism of limited aerotolerance which respires in the presence of oxygen (6,7,8). This study shows (i) the extent of trophozoite oxygen sensitivity of the two strains of *G. intestinalis*; (ii) the effect of elevated DO levels on trophozoite survival with respect to temperature; (iii) the extent of oxygen consumption by trophozoites, and (iv) the relationship of L-cysteine concentration to the attachment of trophozoites *in vitro*.

Materials and Methods

Isolation of New Zealand Strains of Giardia:

Human faecal samples from hospitals and medical laboratories were collected. A modified sucrose flotation technique was used to purify the cysts (9). The excystation methods of Rice and Schaefer (10) and Feely (11), were both used to excyst the cysts from the faeces. Initially, the excystation medium was complete TY1-S-33 as described by Caister (12) but this was soon modified by the addition of 25% cystein HCl (2). Because of an observable difference between excystation medium with bovine serum and that with foetal calf serum, a 50/50 bovine calf serum mix was used in the medium. The use of this medium encouraged not only excystment but growth and development *in vitro*.

The axenic growth medium TY1-S-33 was prepared according to Kasprzak and Majewska (2) and Giardia isolates were cultured in horizontal tubes at 37°C. 1-5x10^{5} cells were transferred axenically daily.

Preparation and enumeration of High Yield Trophozoite suspensions:

150 ml tissue culture bottles containing TY1 -S-33 growth medium were inoculated with 1.0-5.0 x 10^{5} trophozoites and grown to log phase (72-96 hours). Non-attached trophozoites were discarded and attached trophozoites were detached and suspended in chilled Hanks Balanced Salt Solution (HBSS), centrifuged at 400g for 10 minutes and resuspended in 1ml of chilled HBSS. Trophozoites were counted using a haemocytometer under phase contrast and the generations per hour were determined.

Effect of pH

TY1-S-33 medium was adjusted with 1.0 M HCl or 1.0 M NaOH to produce a range of pH levels (6.0- 7.0), 10ml aliquots were inoculated with 1.0 x 10^{5}- 5.0 x 10^{5} trophozoites and incubated at 37°C. Duplicate culture tubes were removed daily and the trophozoites were counted at each pH level.

Effect of Incubation Temperature

A series of 10ml cultures containing 1.0 - 5.0 x 10^{5} trophozoites/ml in TY1-S-33 medium were incubated over a temperature range of 25°C to 40°C over a period of seven days. Duplicate culture tubes at each temperature were removed daily and counted

The Effect of L-Cysteine Concentration and Redox Potential (E_h): on Culture Growth and Trophozoite Attachment.

Duplicate cultures of *Giardia* were propagated in 10ml aliquots of TY1-S-33 medium supplemented with varying concentrations of L-Cysteine. The cultures were inoculated with 1.5 x 10^{5} trophozoites and incubated at 37°C. The redox

Table 1: Excystment of *Giardia intestinalis* human sources in New Zealand.

Area	*Nº Samples*	*Nº of attempts*	*Culture +ve*
Palmerston North	3	10	1
Hamilton	42	55	2
Hastings	13	37	3
Whangarei	2	12	1
New Plymouth	2	6	1
Tauranga	1	1	–
Blenheim	1	8	–
TOTAL	64	129	8

Table 2: Generation time of 6 established cultures of *Giardia intestinalis.*

Strain	*Generation time in hours*
Ham/87/MUGU/84	10.4
Hast/87/MUGU/68	10.8
Hast/87/MUGU/76	10.8
Hast/87/MUGU/86	12.5
Whan/87/MUGU/48	12.6
N.Pl/87/MUGU/77	9.2

Table 3: The Relationship of L-cysteine concentration to E_h and the effect on *G. intestinalis* attachment *in vitro.*

L-cysteine conc. (%) w/v	E_h (mV)	Trophozoite Attachment (%)* at 120 minutes	
		Strain Hast/87/MUGU/68	Strain Whan/87/MUGU/48
0.00	-114.7	45.8	41.4
0.05	-191.5	59.1	67.1
0.10	-243.6	86.5	74.7
0.15	-272.4	92.4	89.6
0.20	-289.8	96.0	90.6
0.25	-302.1	95.5	92.9

**Trophozoite Attachment Percentages expressed as the mean of 2-3 determinations.*

Table 4: Killing of *G. intestinalis* trophozoites by exposure to elevated DO levels. LD50 killing = the time required for 50% of trophozoites to be non-viable. (A) Strain Hast/87/MUGU/68 (B) Strain Whan/87/MUGU/48.

DO Level (ppm)	(A) LD50 Killing (mins)	(B) LD50 Killing (mins)
4.0	360	324
6.0	236	192
8.0	202	186
12.0	74	72

Table 5: The effect of incubation temperature/time on *G. intestinalis* strain of Whan/87/MUGU/48 trophozoites by exposure to elevated DO.

DO Level	Incubation Temperature	Time for LD50
(ppm)	(°C)	(mins)
8.0	30	380
12.0	30	158
12.0	20	278

potential(E_h) was measured using a model 97-08 platinum Redox electrode with an expandable ion Analyser EA 920 (Orion Research). Culture tubes from each of the L-cysteine concentrations were counted daily. Free and adhered trophozoites were determined at 30 minute intervals during four hours incubation.

Sensitivity of Trophozoites to Elevated DO Levels:

A Corning 1000 ml 'Quickfit' Culture Vessel containing TY1-S-33 medium at 37°C and 1.0-5.0 x 10^5 trophozoites/ml was bubbled with pure oxygen to achieve the desired DO. A model 97-08 Oxygen Electrode with an expandable ion analyser EA 920 (Orion Research) was used to measure DO (ppm).

Oxygen Consumption by G. intestinalis

Changes in the DO Level in Phosphate Buffered Saline at 37°C were measured. Sterile PBS solution containing 1.0-5.0 x 10^5 trophozoites/ml was warmed to 37°C in a water bath, and transferred to a 250 ml Kimax flask. The DO was measured using a model 97-08 Oxygen Electrode (Orion Research).

Oxygen Consumption by Trophozoites in PBS:

The procedure as above was followed, with the addition of 6.8 x 10^7 and 2.6 x 10^6 trophozoites prior to sealing of the flask.

Results

Eight cultures were obtained from 129 culture attempts from 64 faecal samples from humans, cats and dogs, as shown in Table 1. The growth rates of six cultures are shown in Table 2.

Further examination showed that these strains have an optimum incubation temperature of 37°C. A slow decline in trophozoite numbers occurred at 25°C and reduced growth continued at 45°C.

A restricted pH range for culture growth and survival was observed for the two strains of *G. intestinalis.* Maximum growth was obtained at pH 7.0-7.5, however growth of both strains were significantly inhibited by acid pH.

Growth of the two *G. intestinalis* strains was strongly dependant upon L-cysteine concentration and E_h. A correlation existed between the increasing L-cysteine concentration/decreasing E_h of growth media. As the concentration of L-cysteine was elevated and the E_h decreased *in vitro* growth rates increased. The generation time was 9.2 hours at an L-cysteine concentration of 0.25% w/v(E_h=-302.1 mV). At L-cysteine concentrations <0.15%.w/v(E_h = -272.4mV) culture growth was supported, but at an increased generation time in both strains (12.5 hours).

G. intestinalis cultures failed to grow in the absence of L-cysteine and more than 50% of trophozoites remained unattached. With L-cysteine concentrations, up to about 0.1%, about 75-85% of the trophozoites were attached. Attachment was most rapid during the 60-120 minutes immediately following culture establishment. As L-cysteine levels were elevated above 0.15% w/v, the 'trophozoite attachment percentage' varied from 89.6-96.0% (Table 3).

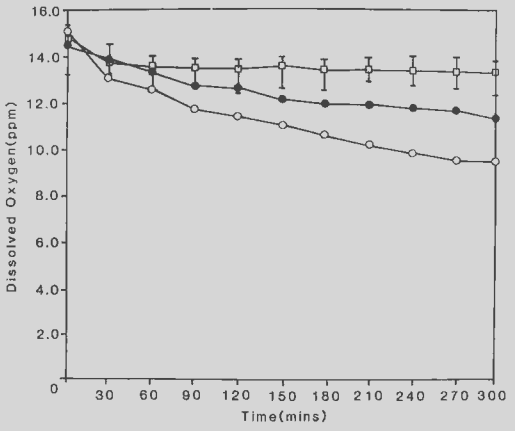
Under *in vitro* conditions elevation of DO at 37°C resulted in decreases in the LD_{50} of the two strains examined. The two strains (Hast/87/MUGU/68 and Whan/87/MUGU/48) exhibited similar aerotolerance limits. The LD_{50} of cultures exposed to 4.0 ppm DO ranged from 324-360 minutes. Under more extreme conditions (12.0 ppm DO) the LD_{50} killing decreased to 72-74 minutes. (Table 4).

Whan/87/MUGU/48 displayed a degree of temperature dependant oxygen sensitivity. At reduced temperatures (20°C-30°C) DO levels which had earlier caused rapid culture death at 37°C were less lethal to trophozoites i.e. the LD_{50} of cultures was extended. The sensitivity of trophozoites to 8.0-12.0 ppm DO was diminished with reduced temperatures. (Table 5).

Utilisation of oxygen by Hast/87/MUGU/68 suspensions of 6.8 x 10^7 and 2.6 x 10^8 cells was measured against time. Decrease in the DO level in excess of those observed in the control were due to consumption of oxygen by trophozoites. Decreases in the DO level of trophozoite suspensions were up to 4-5 fold greater than those in PBS controls. Oxygen was

consumed by trophozoites at a rate of 3.7-5.3 x 10^{-9} ppm/cell/hour. (Fig I).

Figure 1.
Oxygen Consumption by G. intestinalis strain Hast/87/MUGU/68 Trophozoites in PBS at 37°C



PBS control (❑) containing our trophozoites. Suspension A (○):- Inoculum = 6.8 x 10^7 trophozoites, Oxygen Consumption = 5.3 x 10^{-9} ppm/cell/hour. Suspension B (●):- Inoculum - 2.6 x 10^8 trophozoites, Oxygen Consumption = 3.7 x 10^{-9} ppm/cell/hour.

DISCUSSION

Using human sources *in vitro* excystment of *Giardia* cysts was successful in eight cases and the adjustment to the medium by the 50:50 mixing of foetal calf and bovine serum was effective. An important part of the isolation of *Giardia* was found to be the constant re-filling of the culture tube with media every three days. Other workers (2) also report the necessity of perserverance in this area and indicate that most cultures only become well established after several weeks.

Cultures were made from cyst sources that produced both very high and very low excystation percentages. The degree of excystation appeared not to be related to the likelihood of a culture being established.

Optimal growth of Hast/87/MUGU/68 and Whan/87/MUGU/48 was at pH 7.0. This is consistent with the successful growth of axenic cultures by Gillin and Reiner (14). These workers showed pH optima for trophozoite attachment to be pH 6.85-7.00. *In vitro* excystation of *G. intestinalis* has also been found to be pH-dependant occurring at a pH of 2.0 (10).

Reducing conditions are essential for optimal growth of *G. intestinalis* (1) however, it must be noted that even under ideal reducing conditions, the growth of *G. intestinalis* is impaired in the absence of L-cysteine. L-cysteine is known to be an essential component of TY1-S-33, though the functions of this thiol reducing agent in complex growth media have not been determined.

Cultures exposed to both elevated redox potentials resulting from reduced L-cysteine concentrations are markedly reduced. A reduction in the growth rate of cultures exposed to these conditions (0.00-0.10% w/v L-cysteine, E_h = -114.7 to -243.6 mV) was recorded in this study with rapid culture death occurring in the absence of L cysteine. L-cysteine concentrations above 0.15% w/v (E_h > -272.4 mV) exhibited a maximum rate of growth (generation time of 9.2- 12.6 hours). L-cysteine at 10% w/v supported maximum *in vitro* growth (13).

When axenic *G. intestinalis* trophozoites attach to the walls of a culture vessel to produce a confluent monolayer during the log phase of culture growth, trophozoites detach and reattach spontaneously. Attachment of *G. intestinalis* in culture is strongly dependant on the presence of reducing agents (5). While *G. intestinalis* exhibits a specific L-cysteine requirement for culture growth (13); the thiol requirement for trophozoite attachment is less specific. A number of thiol reducing agents support trophozoite attachment at 63-84% of that observed in L-cysteine-supplemented medium (14). A correlation exists between L-cysteine concentration (% w/v) in TY1-S-33 and attachment of New Zealand strains of *G. intestinalis* in culture. With increasing L-cysteine concentrations, and corresponding decreases in E_h Whan/87/MUGU/48 and Hast/87/MUGU/68 trophozoites exhibited 90-96% attachment.

It seems most likely that L-cysteine is utilised by G. *intestinalis* in its oxidised form. Oxidation of L-cysteine is rapid at physiological pH and temperature, and in the presence of iron (15). These conditions apply in the complex growth media used for axenic cultivation of *G. intestinalis*. Under the mild oxidising conditions of culture, the major products of L-cysteine oxidation are disulphides. It has been suggested that mixed disulphides formed between oxidised L-cysteine and peptides may be utilised by *G. intestinalis*. Once cysteine is oxidised in complex growth medium, such mixed disulphides would predominate over cysteine due to their increased solubility.

G. intestinalis is able to concentrate L-cysteine from growth medium but it is not known if trophozoites are able to synthesise it. The presence of a disulphide reductase in this organism has been proposed (5). Iron present in TY1-S-33 may form complexes with cysteinyl-SH groups within proteins to produce iron-sulphur proteins (16). Since Fe-S centres are an essential component of the electron-transport system of *G. intestinalis* (7,8); the specific requirement of *G. intestinalis* for L-cysteine to support growth and attachment in culture, may be attributable to this iron-cysteine complexing in growth medium.

Under the experimental culture conditions described, elevation of DO levels in TY1-S-33 at 37°C resulted in decreases in the LD_{50} killing of the two strains examined. This was not unexpected since an earlier report had indicated that under increased oxygen tensions in growth media, exponential killing of cultures occurs regardless of the presence of reducing agents; although, when present, L-cysteine and ascorbic acid delayed the onset of this killing by up to II hours under 'high PO_2' conditions. (136-144 mmHg) (5).

The reasons for the observed oxygen-sensitivity of *G. intestinalis* trophozoites are not completely understood. It is not known if the death of cultures upon exposure to elevated DO levels is a direct effect of oxygen itself, or is due to oxygen products (27). It is known that univalent reduction of oxygen produces a number of products which can be toxic to most forms of life (18).

Dissolved oxygen in growth medium may have a direct effect on the ultrastructure of trophozoites, e.g. oxygen produced through the 'excitation' of ground-state oxygen; is a powerful oxidant, which attacks polyunsaturated fatty acids (18). Such fatty acids are plentiful in cell and organelle membranes (19,20) and lipid peroxidation by oxygen would severely damage trophozoite cell membranes. Thus superoxide dismutase enzymes in *G. intestinalis* (7) may be essential protection against toxic species of oxygen (18).

Oxygen-sensitive *G. intestinalis* trophozoites have been shown to actively consume oxygen (6,7,8). Similar activities observed in *E. histolytica* have led to suggestions that this oxygen consumption represents some form of '0_2 scavenging' as a protective mechanism against toxic oxygen radicals which accumulate under reducing conditions.

The basis for the assumption that *G. intestinalis* is an anaerobe has been that its environment is essentially anaerobic (21), and that a low E_h is required for its optimal growth in culture. Furthermore, *G. intestinalis* trophozoites lack any structures identifiable as mitochondria (22). However, this study shows aerotolerance and oxygen consumption by trophozoites of New Zealand *G. intestinalis* isolates.

G. intestinalis is one of a small number of protozoa which consume oxygen while displaying a largely fermentative metabolism where carbohydrates are oxidised incompletely to produce ethanol, acetate and $C0_2$ (6,7,8). In *G. intestinalis* the consumption of oxygen is indicative of the unique carbohydrate metabolism of this protozoan. Carbohydrates

are utilised by *G. intestinalis* in the absence of a functional Krebs cycle and cytochrome mediated oxidative phosphorylation. Catabolism of carbohydrates to produce energy occurs via substrate level phosphorylation and a flavin-iron sulphur mediated electron transport system (6,7,8,22).

While the current study has demonstrated the high affinity of Hast/87/MUGU/68 and Whan/87/MUGU/48 for oxygen at low concentrations, the enzymes responsible are yet to be described. Gutteridge and Coombe (23) have suggested that the activity of pyruvate oxidase (CoA acetylating) in a physiologically similar aerotolerant anaerobe, *Entamoeba histolytica* may explain oxygen consumption by this organism. Considering the homogeneity which exists between the carbohydrate metabolism of *E. histolytica* and *G. intestinalis,* oxygen consumption by *G. intestinalis* may have a similar basis. Pyruvate oxidase in *E. histolytica* is equivalent in activity to Acetyl CoA synthetase in *G. intestinalis* therefore it seems reasonable to assume an analogous role for this enzyme in the mediation of oxygen consumption by *G. intestinalis.*

Acknowledgements

We wish to acknowledge the financial support of the New Zealand Health Department and the supply of faecal specimens from Dr A. Corbett, Pathology Laboratory, Palmerston North.

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THE NEW ZEALAND MEDICAL LABORATORY SCIENCE TRUST
ANNUAL REPORT
1991

N.Z. MEDICAL LABORATORY SCIENCE TRUST

TRUST OFFICE
P.O. BOX 12-260
WELLINGTON
Phone (04) 723-431
Fax (04) 727-181

The Trust has continued to operate over the last year within the confines of its financial situation. Like all bodies the Trust obviously feels the current economic situation in New Zealand and the level of giving is confined as shown in the financial statement. Nevertheless the Trust still feels that it is important to remain in existence and to operate at the level it does with the hope that economic times will brighten and allow the Trust to achieve more closely the original aims and aspirations.

1 NEW TRUSTEE

To meet the requirements of its own Deed the Trust needed to have another Trustee and this has resulted in the appointment of Barrie T. Edwards to the Trust.

2 APPROVAL OF TRUST BY INLAND REVENUE

Following approaches by our Solicitors to the Inland Revenue Department the Trust is now approved by that Department for the following purposes:

- Section 2 of the Income Tax Act 1976
- Section 56A of the Income Tax Act 1976
- Section 61(25) of the Income Tax Act 1976
- Section 61(27) of the Income Tax Act 1976
- Section 147 of the Income Tax Act 1976
- Section 5 of the Estate and Gift Duty Act 1968
- Section 73(1) of the Estate and Gift Duty Act 1968
- Section 18(b) of the Stamp and Cheque Duties Act 1971

This approval is conditional upon the Trust restricting its activities to those which have been established as being charitable at law. To gain this approval some slight amendments had to be made to the Deed. These amendments were perceived as minor by the Trustees and not affecting the original aims of the Trust and so were agreed to.

The changes required by the Inland Revenue Department were as follows:

Clause 3(f) allows the Foundation to co-operate with other bodies with similar objects. The word "charitable" should be added between the words "having" and "objects".

Clause 3(k) allows the Foundation to accept and carry out Trusts attached to gifts etc. A proviso is required that "any such Trusts are charitable in law".

Clause 3(m) requires the words "in the opinion of the Board" to be deleted.

Clause 5(d) allows the Foundation to carry on a business. The following additional clause should be added "Nothing in these presents shall be construed as to permit any Board or ordinary member or any associated person of any member deriving private, personal or pecuniary profit from the business or investments of the Foundation."

Clause 5(j) requires the words "proper and reasonable" to be inserted between the words "all" and "charges".

Clause 5(o) requires the words "the Board may deem" to be deleted and the clause to conclude "as are reasonable and proper".

Clause 11 provides for alterations to the Rules and requires the addition of a proviso to read "no alterations shall be made that would change the charitable aim or charitable objects of the Foundation". The appropriate changes were made and registered.

3 GRANTS

Three grants were made as shown in the financial statement. A report from Stephen Henry reporting his study trip to Sweden has been received. Copies of this report are available to N.Z.I.M.L.S. members. Applications for grants for the next financial year must be in the hands of the Executive Officer, Mr. J.C. Mann, by 31 May 1992. The address of the Executive Officer is:

Mr. J.C. Mann
Executive Officer
Medical laboratory Science Trust
C/-. Pathology Department
Palmerston North Hospital
Palmerston North

Some problems have arisen with people using the old Trust address in Wellington and this last year one application was received after allocations had been made although it was posted **in good time.**

4 DONATIONS

The Trust is willing to act as an intermediary in donor-directed gifts and grants. We understand that there is interest in this avenue of giving.

THE NEW ZEALAND MEDICAL LABORATORY SCIENCE TRUST (Inc.)

INCOME AND EXPENDITURE ACCOUNT

FOR THE YEAR ENDED 31 DECEMBER 1990

INCOME				
Donations Received			5,529.00	
Interest Received			650.42	
				$6,179.42
EXPENDITURE				
Printing & Stationery			711.00	
Grants:	S.J. Smith	583.00		
	M.J. Staveley	500.00		
	S. Henry	500.00	1,583.00	
Excess Income				2,294.00
				$3,885.42

THE NEW ZEALAND MEDICAL LABORATORY SCIENCE TRUST (Inc.)

BALANCE SHEET

AS AT 31 DECEMBER 1990

ACCUMULATED FUNDS:		
Balance as at 1 January 1990	8,444.29	
add Excess Income	3,885.42	
Total Funds	$12,329.71	
Represented by:		
Current Assets		
ANZ Banking Group Balance		$12,329.71

AUDITOR'S REPORT:

To the Trustees of the New Zealand Medical Science Trust (Inc.)

I have examined the financial records of the N.Z. Medical Laboratory Science Trust (Inc.) and have received such explanations and carried out such procedures as I considered necessary.
In my opinion, the above Income and Expenditure Account and Balance Sheet give a true and fair view of the financial position of the Trust's affairs as at 31 December 1990.

(Signed) **David R. Gordon Auditor**

Palmerston North
26 January 1991

The following does not form part of the financial statement but is an explanatory note to it.

DONATIONS

N.Z.I.M.L.S.	$5,000.00	28.05.90
N.Z.I.M.L.S., Q.T.A.Examiners Acct.	$250.00	10.12.90
Guy A. Hunt (Patient)	$200.00	23.03.90
Invercargill Conference Fund	$59.00	28.09.90
R.A. Peters (Patient)	$10.00	10.08.90
Pathology Social Club, Southland Hospital	$10.00	16.05.90
Bank Interest		
	$288.07	31.3.90
	$362.35	30.9.90

On behalf of the Trustees

Desmond J. Phillip
Chairman.

APPLICATION FOR GRANTS FROM THE MEDICAL LABORATORY SCIENCE TRUST (INC)

In 1987 the New Zealand Institute of Medical Laboratory Science (Inc) responding to a change in the direction of our society from "State funding" to "self support" and "user pays" and as there was no organisation with the specific responsibility for supporting and fostering the aims and ambitions of the New Zealand profession of Medical Laboratory Science, established the Medical Laboratory Science Trust, with the following principal objectives —

(a) To promote and assist research by members of the NZIMLS.

(b) To promote and assist the education of members of the NZIMLS by the provision of grants of money and the organisation of lectures, demonstrations and tutorials.

(c) To promote and assist in the provision of equipment, travel and accommodation for members of the NZIMLS to further their research and education.

(d) To promote and assist in the provision of course fees, enrolment fees, study bursaries and book purchases for members of the NZIMLS to further their education and research.

(e) To promote and assist in the publication of any research by members of the NZIMLS.

(f) To co-operate with other bodies or organisations, both within New Zealand and overseas, having objects in whole or in part similar to the objects of the Science Trust.

(g) To promote, obtain and achieve any of the objects of the Science Trust by or through the facilities available at any Hospital, University, or recognised medical, veterinary, scientific or research institute or other organisation and make grants of money. apparatus. equipment or otherwise, as the Trust Board may think fit.

The Trustees appointed by the Institute are Mr John S. Beattie of Wellington, Mr Colvin H. Campbell of Palmerston North, Mr Barrie T. Edwards of Christchurch, Mr Desmond J. Philip and Mr Walter J. Wilson, both of Auckland.

The Science Trust invites applications from financial members of the NZIMLS who wish support to enable them to attend the Annual Scientific Meeting of the NZIMLS in Wellington, 27-28 August, 1992.

All practising Fellows, Members and Associates of the NZIMLS are eligible to apply, applications will be considered on expected benefits from attendance at the Scientific Meeting, together with consideration for the members' participation in promoting Medical Laboratory Science.

Application forms are available from the following:

- The Executive Officer,
  NZIMLS
  P.O. Box 3270,
  CHRISTCHURCH.
- Executive Officer,
  Medical Laboratory Science Trust,
  C/- Pathology Department,
  Palmerston North Hospital,
  Private Bag,
  PALMERSTON NORTH.

Applications must be on the official Application Form and be received by the Executive Officer, NZMLST, no later than 5 pm on Friday, 29 May, 1992.

INSTITUTE BUSINESS

Office Bearers of the N.Z.I.M.L.S. 1991 - 1992

President
Paul McLeod
Microbiology Dept., Nelson Hospital

Vice President
Dennis Reilly
Diagnostic Laboratory, Auckland

Secretary/ Treasurer
Shirley Gainsford
Valley Diagnostic Laboratory, Lower Hutt

Council
Ted Norman, Anne Paterson, Jim Le Grice, Geoff Rimmer, Chris Kendrick

Executive Officer
Fran van Til
P.O. Box 3270, Christchurch
Phone/Fax (03) 313-4761.

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

Editor
Maree Gillies
Microbiology Dept., Auckland Hospital or The Editor, P.O. Box 9095, Newmarket, Auckland.

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

For Fellows — $88.40 GST inclusive

For Members — $88.40 GST inclusive

For Associates — $33.80 GST inclusive

For Non-practising members — $33.00 GST inclusive

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

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

Membership Sub-Committee Report — November 1991

Since the August meeting there have been the following changes:

	26.8.91	26.8.91	23.5.91	28.2.91
Membership	1188	1297	1202	1277
less resignations	6	37	33	10
less G.N.A.	3	8	7	11
less deletions	-	81	-	116
less deceased	1	-	2	-
less duplications	1	1	-	-
	1178	1170	1160	1140
plus applications	9	10	22	61
plus reinstatements		110	116	2
	1188	1180	1298	1202
Composition				
Life Member (Fellow)	12	12	12	12
Life Member (Member)	5	5	5	5
Fellow	21	21	21	22
Member	670	666	711	679
Associate	392	395	462	399
Non-practising	60	60	57	56
Honorary	28	29	29	30
Total	1188	1188	1297	1202

Applications for Membership
A THORNTON, Wellington; J HORNE, Whangarei; B ROWLANDS, Northland Path; N THOMAS, Middlemore; F FAIGA, Wellington; J ODGERS, Whangarei; S ANSELL, Whangarei; M PETRASICH, Auckland; C MURPHY, Wellington.

Resignations
K BONIFACE, National Womens; D ESCOTT, Medlab Sth; E CULLEN, Blenheim; L HOSKEN, ARBC;M THOMPSON; E THOMPSON.

Gone No Address
E MCCONNOCAIE, ARBC; N TAUAT, ARBC; P GOUNDER, ARBC.

Wellcome

Wellcome International Travel Award

WELLCOME INTERNATIONAL TRAVEL AWARD WINNER.

Liz Fox from Wellcome NZ Ltd presented Dennis Reilly with the Institute's most prestigious award, the "Wellcome International Travel Award", during the South Pacific Congress dinner. The award, presented biannually, provides a return airfare, Congress fees and a daily accommodation allowance to attend the World Congress of Medical Laboratory Technology.

Dennis will attend the 20th Congress of the IAMLT in Dublin, Ireland from July 26 - 31st 1992.

Dennis Reilly — 1991 Wellcome Travel Award Recipient

AN INVITATION TO THE 20th WORLD CONGRESS OF MEDICAL TECHNOLOGY

Dublin, Ireland 26th - 31st July 1992

Presented by:

THE INTERNATIONAL ASSOCIATION OF MEDICAL LABORATORY TECHNOLOGISTS

Hosted by:

THE ACADEMY OF MEDICAL LABORATORY SCIENCE AND THE MEDICAL LABORATORY TECHNOLOGISTS ASSOCIATION

BOOK REVIEW

"INTRODUCTION TO GENETIC ENGINEERING"

(1991) ISBN 0-7506-9114-X

William H. Sofer
Waksman Institute
Rutgers - The State University of New Jersey,
Piscataway, New Jersey, USA
Butterworth - Heinemann
Reviewed by Gillian McLeay
Laboratory Training Officer Auckland Hospital

In his book, "THE 2024 REPORT A Concise History of the Future 1974 - 2024" (published 1984), Norman Macrae wrote in the chapter "Genetic Engineering: Booms and Busts", that in the early 1980's, genetic engineering enthusiasts feared that one of the two factors that would delay progress in this developing field would be social caution. Science fiction writers have thrived on stories of experimental bacteria escaping from laboratories with horrific consequences. The other factor was problems with the development of the practical techniques beyond the experimental stage.

Even at the time of writing this most thought-provoking book, Norman Macrae foresaw that these fears would prove to be largely unfounded in the future.

Gene transfers are not confined to the DNA laboratory and take place in nature also. Commonsense safeguards are being put in place, and the applications of DNA technology, and the ethical considerations involved, are now, in the 1990's, a matter of debate at a universal level.

Since 1984 there have been great advances in DNA techniques, some of which have moved from the laboratory out into the commercial world, with a significant impact on certain aspects of medicine and agriculture.

It is for this reason that this article is being written to bring members' attention to the book actually being reviewed "Introduction to Genetic Engineering".

William Sofer, a lecturer and researcher in Molecular Biology, decided to collate his lectures of over "half a dozen years", using his beloved personal computer to build a book, doing the layout and the illustrations and thereby retaining considerable control over its appearance.

The result is a conveniently sized paperback edition with simple, clear diagrams, and each chapter written like a well presented lecture, complete with introduction, recap and a very brief summary at the end. Great technique. I should love to hear him give one of his lectures. (The book in a somewhat modified form is available as two computer tutorials designed for the Macintosh computers.)

To make recombinant DNA less intimidating, he has written a book which is not as comprehensive as other text books, but more accessible to people outside the fields of Biochemistry, Molecular Biology and allied disciplines. Those targeted were high school biology teachers, and middle and upper level management in biotechnology companies, who have to make decisions on technological matters without the benefit of practical expertise.

To this list of potential readers, I would add secondary school pupils, students in the different science courses at universities and polytechs, and in particular, our own folk in Medical Laboratory Science. We have the advantage of some back ground knowledge, but this very readable book is an excellent introduction for those who are commencing study of the subject, as well as for those who merely wish to "brush up" and "update". There is a bibliography with titles for further study.

The author makes no apology for oversimplification in some areas, (especially the first four chapters) but there is a glossary for the new technical words, and as you continue to read, it does become more complex.

The book is divided into four sections. The first four chapters deal with molecules, proteins and protein synthesis, and DNA and RNA at a very basic level. The author says most people can safely skip this if they have taken a biochemistry course in the last decade!

Chapters 5 - 10 deal directly with the various DNA recombinant techniques. For those who like to keep up with the current terminology, RFLPs (restriction fragment length polymorphisms) are known as "riff-lips"

Chapter 11 mainly deals with the applications of DNA technology, now and in the future in medicine, with some reference to agriculture also. A brief comment is made on the future impact of DNA technology on the chemical and pharmacological industries.

The final section of the book consists of two chapters. The one on the use of computers in molecular biology emphasises the increasing importance of the computer as a laboratory tool. The other is the author's personal views on ethical considerations and guidelines, (the acceptable and unacceptable uses), in the rapidly expanding field of genetic engineering.

I consider this to be a very useful, informative book for every medical laboratory book shelf, and for the science section of every secondary school, university, technical institute and general library. I am sure the pharmaceutical and chemical companies will have a copy on their shelves.

Copies may be obtained from:
Medical Books of New Zealand
Address:
8 Park Avenue, Grafton, AUCKLAND, 3
Postal Address:
PO Box 8565, Symonds Street, Auckland. 3.
Telephone: (9) 733 722 Facsimile: (9) 733 282

3RD SOUTH PACIFIC CONGRESS ON MEDICAL LABORATORY SCIENCE

AUGUST 26 — 30, 1991
Auckland, New Zealand

ABSTRACTS

MICROBIOLOGY

MB1

ONE YEAR'S EXPERIENCE WITH ROUTINE USE OF BACTEC TB SYSTEM IN A CLINICAL MICROBIOLOGY LABORATORY.

Angela Bricknell
Diagnostic Laboratory, PO Box 5728, Auckland

During May 1990 the Bactec radiometric TB detection system replaced the time honoured method of culture on Lowenstein-Jensen medium in our laboratory for the isolation of *Mycobacterium tuberculosis* and atypical Mycobacteria from clinical specimens received mostly from the patients of General Practitioners.

Following the introduction of the Bactec system the time taken to obtain a positive culture improved from an average of 28 days (range 11 days — 6 weeks) before Bactec to 7 days (range 1 day — 13 days) with Bactec. The isolation rate of *M. tuberculosis* from sputum specimens doubled. There were no positive *M. tuberculosis* from urine specimens in the year before Bactec but there were two patients with positive cultures in the following year.

Reagents for the Bactec system are more expensive than for the old method. Many more atypical Mycobacteria are isolated using Bactec and species identification of these adds to the cost.

The Bactec TB system provides a rapid and reliable procedure for the detection of Mycobacteria in the community.

MB2

DEVELOPMENT OF THE MCDONNELL DOUGLAS CLINICAL LABORATORY INFORMATION MANAGEMENT SYSTEM (CLIMS) WITHIN A DIAGNOSTIC MICROBIOLOGY LABORATORY.

AR Oakes
Department of Clinical Microbiology, The Queen Elizabeth Hospital, Woodville, South Australia 5011

The Queen Elizabeth Hospital is a 600-bed teaching hospital situated in western suburban Adelaide. Replacement of the existing 14-year old Pathology Computer System began in 1990 with the installation of the McDonnell Douglas M9200 series CLIMS system. The system supports all the main laboratory disciplines using PICK-based software, 8MB of RAM, 2GB Disk Capacity and up to 96 peripheral ports. The system accesses the hospital Patient Master Index (PMI) and Admissions, Transfers and Separations (ATS) data, to aid in registration of laboratory specimen details onto CLIMS.

Important facets of the Microbiology module are discussed with particular reference to table-driven software, test and profile definitions, management/workload statistics, epidemiological functions, design and use of OMR sheets for data entry, outstanding and unverified test reports, and interfacing with automated equipment (Bactec, Vitek).

MB3

NON-01 VIBRIO CHOLERAE IN THE EASTERN BAY OF PLENTY.

JM Wright
Laboratory, Whakatane Hospital, PO Box 241, Whakatane

Since December 1987, five cases of non-01 *Vibrio cholerae* gastroenteritis have been detected at the Whakatane Hospital laboratory. The organism has also been isolated from one Otitis patient.

In all cases, the primary isolation was made on 5% sheep blood agar and the organism was identified to species level at the above laboratory by using a combination of "home-made" and commercial biochemical tests. Confirmation of identity and organism serology were performed at the New Zealand Communicable Diseases Centre.

Ingestion of local shellfish was noted to have preceded symptoms in 3 cases indicating that non-01 *Vibrio cholerae* may be endemic in the coastal waters of the Eastern Bay of Plenty; however the small number of cases detected suggests many factors — both environmental and host — contribute to the pathogenic potential of this organism.

CRYPTOCOCCAL SEPTIC ARTHRITIS — A CASE REPORT. MB4

JM Wright
Laboratory, Whakatane Hospital, PO Box 241, Whakatane

An 80 year old man with a recent past history of pulmonary tuberculosis was admitted to Whakatane Hospital with suspected tuberculous arthritis of the knee.

An initial knee aspirate was unremarkable, however a second aspirate and a synovial biopsy both grew *Cryptococcus neoformans*. The organism's identity was confirmed at the New Zealand Communicable Diseases Centre and the Mycology Laboratory, Auckland Public Hospital performed antifungal susceptibility testing on the isolate. The patient responded to Fluconazole therapy. A review of the literature shows less than 20 cases of Cryptococcal arthritis have been documented and that the knee is the most common site of this infection.

UNUSUAL PSEUDOMONADS IN CLINICAL DISEASE: 2 CASE REPORTS. MB5

JM Wright
Laboratory, Whakatane Hospital, PO Box 241, Whakatane

Pseudomonas picketti meningitis. A 4 month old male presented as febrile, irritable and with neck stiffness. A lumbar puncture was performed and a bloody tap resulted. This was unremarkable on initial laboratory investigation; however, after 2 days incubation, the culture plates all showed scanty growth of an oxidase positive organism which was identified as *Pseudomonas picketti*. Reculture of all tubes confirmed the presence of the organism in the sample. The child was treated with Amoxicillin and Cefotaxime and defervesced.

MB6

DETERMINING THE PREVALENCE OF PARASITES IN VEGETABLES BY USING VARIOUS ISOLATION TECHNIQUES.

MJ Gharavi, A Kahnamouii
Iran University of Medical Sciences. P.O. Box 14155 - 6183, Tehran, Iran

In Iran vegetables may be considered as a potential source of parasitic infection since they are often used raw along with meals. A survey made on the vegetables obtained from various herb gardens in Tehran suburbs in a year, has led to noteworthy results. In this survey, isolation of protozoa and parasites ova and larva was made by means of the following technqiues:

1. Baermann's Technique: precipitation of parasites ova and larva present in the vegetable at the bottom of a funnel.
2. Centrifugation of the plant material: straining the precipitated ova through 1mm, 250μ, 100μ, and 37μ filter papers and using 1.18 percent sucrose to drive the ova to the surface.
3. Glycerol sedimentation: precipitation of the parasites present, and examining the solution by flotation or sedimentation technique overnight.

Parasites were demonstrated in 147 of 263 (55%) vegetable specimens obtained from 44 herb-gardens in Tehran; 86 (33%) of these were transmissible to man.

34 cases of Giardia cysts, 30 of Trichostrongylus larvae, and ova — 16 of Ascaris eggs, 2 of Trichuris trichiura and 4 cases of Entamoeba histolytica cysts were observed. There also existed 61 non pathogenic parasites of man including larvae and adult of soil nematodes, Strongyloides sp. and Ascaris and flagellates of animals. Discussion is continued.

ISOLATION OF TOXOPLASMA GONDII FROM DOMESTIC BIRDS. MB7

MJ Gharavi, A Kahnamouii
Department of Parasitology, Iran University of Medical Sciences, PO Box 14155 - 6183 Tehran, Iran.

Toxoplasma infections, diagnosed by means of parasitological and serological examinations, have been reported in mammals from Iran but have not been studied in birds yet.

In this study, 170 various birds from different parts of Iran were examined for detection of Toxoplasma antibodies by means of Indirect Haemagglutination Antibody Test (I.H.A.T.); sera of 47 of which were positive (27.5%). There was no evidence of the parasite in stained brain and blood smears of the birds. Therefore, a suspension of the brain was inoculated to mice intraperitoneally (Beverley method). The sera of the mice were examined by I.H.A.T. after 4-6 weeks. The brain was examined if the serum was positive.

It was realised that I.H.A.T. is a highly sensitive test for detecting toxoplasma antibodies in birds and mice.

In this study 12 strains of *Toxoplasma gondii* (7%) were isolated from hens, turkeys, roosters, geese, ducks and pigeons.

One of these strains from turkeys was highly virulent for mice and caused blindness 9-11 days after the inoculation. But there was no evidence of the organism in the histology sections of eyes. Further studies are required.

THE INCIDENCE OF HELICOBACTER PYLORI IN NON-ULCER DYSPEPSIA: A DUNEDIN STUDY. MB8

Anne Paterson, Ingrid Christiaans
Microbiology Laboratory, Dunedin Hospital, Dunedin

Over a 12 month period, a study was undertaken at Dunedin Hospital to determine the incidence of *Helicobacter pylori* in patients with Non Ulcer Dyspepsia (dyspeptic symptoms but normal gastroscopy).

Biopsies were taken from 294 patients, within 2 cm of the pylorus (in the stomach). These were analysed in 3 ways.

1. Urease Test.
2. Histological Analysis.
3. Microbiological Analysis.

These methods were evaluated for correlation. Overall results will be presented. The microbiological methods, findings and problems will be discussed more fully.

MB9

THE CARRIAGE OF GIARDIA BY SHEEP, CATTLE, HARES, POSSUMS AND DUCKS: A PRELIMINARY SURVEY.

LC Craighead[1], D Wallace[2], L Cochrane[3]
Microbiology Department, Dunedin Hospital, Private Bag, Dunedin[1]. Community Health, Dunedin Hospital[2] and Oamaru District Council, PO Box 72, Oamaru[3].

Because epidemiologic data from overseas, has implicated animals in the transmission of Giardia, a preliminary study was conducted to assess the carriage of Giardia by wild and domestic animals in the Oamaru and Dunedin areas. Faecal specimens were taken from sheep and calves, and duodenal contents from ducks in the catchment area of the Oamaru District. Duodenal contents of possums, hares and rabbits from the Dunedin City Council Forestry, were also examined for Giardia. Methods used were trichrome staining, and faecal concentrate. Giardia found in the animals was morphologically and antigenically similar to Giardia found in cases of human disease. Domestic ruminants may be a reservoir for water contamination, however further studies, including DNA fingerprinting, need to be carried out to confirm this. Problems involving epidemiology and identification are discussed.

MB10

LABORATORY DIAGNOSIS OF PNEUMOCYSTIS CARINII.

A Kahnamouii, MJ Gharavi
Department of Parasitology, Iran University of Medical Sciences, PO Box 14155 - 6183, Tehran, Iran

Pneumocystis carinii is an opportunistic pathogenic organism to the lungs of man and various hosts. This organism causes pneumonia in infants with congenital immunodeficiency, and in patients suffering from malnutrition, treatment with immunosuppressive drugs, and acquired immunodeficiency syndrome (AIDS).

In this study, 30 rats were injected with 1 ml corticosteroid (cortisone acetate 25%) twice a week for 8 weeks. Impression smears and histology sections were prepared from autopsied lung samples of the rats 6-8 weeks after the injections. Giemsa, Gram and toluidine blue 0 staining techniques were used for staining impression smear slides. Giemsa and Gram staining technqiues were the best for demonstrating the cysts and sporozoites, while in toluidine blue 0 staining technique, only cysts could be observed. Tissue sections of lung were stained with trichrome, haematoxylin schif reagent and papanicolaou's, stains; the organism was observed clearly in all the sections, but appeared as a cloudy mass in haematoxylin stained ones. No organism was found in impression slides and tissue sections of the autopsied lung samples obtained from 10 control rats.

The results of this study suggest that in appropriate cases biopsy material of lungs should be sent to the laboratory for identification of the parasite as infection is possible.

MB11

COMPARATIVE STUDY OF TWO TESTS: INDIRECT HAEMAGGLUTINATION TEST AND INDIRECT FLUORESCENT ANTIBODY TEST FOR DETECTION OF TOXOPLASMA GONDII ANTIBODIES.

A Kahnamouii, MJ Gharavi
Department of Parasitology, Iran University of Medical Sciences, PO Box 14155 - 6183, Tehran, Iran.

Toxoplasma gondii is a ubiquitous organism capable of infecting a wide range of hosts including mammals and birds.

Indirect Haemagglutination Test (IHAT) and Indirect Fluorescent Antibody test (IFAT) have been compared with the sera of 416 samples collected from clinically suspected patients and 45 control sera. 170 sera of domestic birds and about 600 sera from mice were also tested for detecting *Toxoplasma gondii* antibodies by the IHA method only. Qualitative agreement of 70% was observed between the two tests, and there was 80% quantitative agreement between them. From the sera of 170 domestic birds 47 samples (27.6%) were positive by IHA test and 14 groups of inoculated mice out of 170 groups were positive by this test.

All of the findings suggested that the IHA test generally appears to be of a higher titer than the IFA test and therefore that IHAT is more sensitive than the IFAT.

MB12

PAPERLESS MICROBIOLOGY.

Dave Aarons, Jennifer Mitchell
Microbiology Department, Diagnostic Laboratory, PO Box 5728, Auckland

Diagnostic Laboratory has been computerised since 1969. The Delphic software system was introduced on Burroughs (now UNISYS) hardware in 1979. Microbiology results were entered by key punch operators until an optical marked card (OMR) system was introduced in 1983. In 1987 we commenced conversion to a direct entry system based on Unysis B series microcomputers. The Microsmic system provides a data base for storage and collation of all documentation regarding specimens being processed. Patient demographics are downloaded from the mainframe, specimens are matched to patient records and appropriate test lists set up within the system. Lists are accessed by technicians for data entry when tests are performed. Work is collated and displayed for senior staff to authorize and report to the mainframe.

MB13

THE DIAGNOSTIC USE OF THE POLYMERASE CHAIN REACTION (PCR) FOR THE DETECTION OF MYCOBACTERIUM TUBERCULOSIS.

KM Weekes, MJ Pearse, AJF d'Apice
Department of Clinical Immunology, St. Vincent's Hospital, Melbourne

PCR is a powerful and sensitive technique with applications in many fields. Until recently, it has been used only in a research environment and this paper will describe the problems involved in the adaptation of the technique to a busy diagnostic laboratory.

M. tuberculosis is an organism that is presently difficult to detect in low numbers and cultures can take up to six weeks to grow. Using oligonucleotide probes specific for a repeated element in the *M. tuberculosis* genome (Crawford et al, Sem Respir Inf, 1989) we have been able to detect as few as ten bacteria using PCR amplification. This compares well with the Ziehl-Nielsson staining technique for acid-fast bacilli which requires about 10,000 per millilitre for a positive result.

The cell wall of Mycobacteria is resistant to lysis, so in order to maximise sensitivity a comparison of DNA extraction techniques has been undertaken.

A retrospective study of clinical specimens is being undertaken in conjunction with Fairfield Hospital and the results will be presented.

MB14

NOSOCOMIAL STRAINS OF ANTIBIOTIC-RESISTANT STAPHYLOCOCCUS EPIDERMIDIS IN A CARDIAC SURGERY UNIT.

R Menzies, D MacCulloch, B Cornere
Microbiology Department, Green Lane/National Women's Hospital, Greenlane Site, Greenlane West, Auckland 3, New Zealand

The origin of *Staphylococcus epidermidis* causing three cases of early prosthetic valve endocarditis was investigated by a detailed study of 13 patients from our cardiac surgery unit. *S. epidermidis* isolates recovered from patients' skin on admission to hospital were compared with isolates recovered after surgery. All isolates were typed by plasmid profile, antibiotic susceptibility and slime production. Nosocomial strains were recovered from eight patients, were present in the operating theatre environment and resembled each other and isolates from early prosthetic valve endocarditis. Common characteristics were slime production and resistance to oxacillin, gentamicin, kanamycin and tobramycin. A reservoir of related nosocomial *S. epidermidis* strains existed in our cardiac surgery unit and appeared to be the source of isolates responsible for sporadic cases of early prosthetic valve endocarditis.

MB15

TEICOPLANIN AND ENTEROCOCCAL ENDOCARDITIS.

J Koroivueta, D MacCulloch, B Cornere
Department of Clinical Microbiology, Green Lane/National Women's Hospital, Green Lane West, Auckland, New Zealand.

A patient with *Enterococcus faecalis* endocarditis involving the mitral valve was initially treated with the combination of penicillin and gentamicin. This strain lacked beta-lactamase activity and did not show high level resistance to gentamicin. She developed an embolus to the right kidney as well as a spiking temperature and cardiovascular instability during the first 2 weeks of antibiotics.

A semi-urgent mitral valve replacement was performed. Excised valve tissue showed numerous gram-positive cocci which eventually grew. At day 3 post-operative (day 18 of antibiotics) severe leukopenia developed. This prompted changing to vancomycin and gentamicin. By day 15 of this combination she developed renal impairment. Teicoplanin and netilmicin was commenced. She completed an additional 2 weeks of this combination, with a week's dose administered on an outpatient basis. At follow-up she was regarded cured. In vitro microbiological data will be discussed.

MB16

THE APPLICATION OF DNA TECHNOLOGY TO HAEMOPHILIA IN NEW ZEALAND.

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

The haemophilias are congenital coagulation disorders, due to deficiences of factors VIII or IX, which have an X-linked pattern of inheritance. Haemophilia A affects approximately 1 in 5000 male births whilst haemophilia B has an incidence approximately one fifth that of haemophilia A. In New Zealand 370 haemophilia A patients (FVIII $\leq$ 20%) and 72 haemophilia B patients (FIX $\leq$ 20%) are listed on the national register.

Essential components of haemophilia management are accurate carrier detection and early prenatal diagnosis. Both have been greatly improved by applying DNA technology. Conventional carrier detection based on pedigree analysis combined with the measurement of clotting activity and antigen levels has a significant error risk which may be as high as 15%. Early prenatal diagnosis (prior to 12 weeks gestation) is not possible using these phenotypic methods. When informative, DNA genotyping using intragenic RFLP analysis is highly accurate and may be used for prenatal diagnosis as early as eight weeks gestation by extracting foetal DNA from chorionic villi. Several RFLPs have been reported which are useful in linkage analysis within families at risk for haemophilia A and B. Genotypic analysis is not effected by lyonisation, variance in gene expression or gene product stability and is thus more accurate than phenotypic studies. DNA analysis is now part of the routine management of haemophilia families, for carrier detection and prenatal diagnosis, here in New Zealand.

MB17

MOLECULAR BIOLOGY OF AN IMMUNODOMINANT ANTIGEN FROM MYCOBACTERIUM LEPRAE.

Roger Booth*, Jeff McKee*, Diana Williams†
*Department of Molecular Medicine, Auckland University School of Medicine, Private Bag, Auckland. † Gillis W. Long Hansen's Disease Center, Carville, Louisiana, USA.

Studies using monoclonal antibodies against antigens of *M.leprae* identified, among a number of cross-reactive antigens, a highly-immunogenic 18kDa protein which appeared to be antigenically unique to *M.leprae*. Cloning and sequencing of the DNA which codes for this protein revealed a novel sequence with a small region of similarity to a family of low molecular weight heat-shock proteins. By using oligonuceotide primers derived from the 18kDa gene sequence, a polymerase chain reaction (PCR)-based test was developed which could specifically detect DNA from as few as 100 *M.leprae* bacilli in human tissue biopsies.

DNA hybridisation tests using the *M.leprae* 18kDa gene as a probe at high stringency did not detect homologous genes in any other Mycobacterial species. However at lower stringencies, hybridising DNA was detectable from *M.avium* and *M.intracellulare* but not from *M.tuberculosis*. When recombinant DNA libraries were probed with the *M.leprae* 18kDa gene at low stringency, two homologous genes were isolated from each of *M.avium* and *M.intracellulare*. The proteins from all five 18kDa genes displayed a high degree of similarity in all but the last 10-20 amino acids. Oligonucleotide probles have been constructed from the most highly-conserved regions of these genes and are being used to determine whether similar genes exist in all Mycobacteria.

A REVIEW OF DNA TECHNOLOGY IN FORENSIC SCIENCE. MB18

ME Lawton
DSIR Chemistry, PO Box 2224, Auckland.

In 1985 Professor Jeffreys of Leicester University published an article which changed the course of forensic biology. He adapted and extended molecular biology technology to provide a powerful means of identification. This had obvious applications in criminal matters and parentage disputes.

Since then forensic laboratories worldwide have implemented restriction fragment length polymorphism analysis or DNA profiling. The road has not always been easy and widespread debate has evolved in the laboratory and in the courtroom.

This is an area of rapid progress and while forensic scientists strive to employ current techniques new methods are evolving which may eventually optimise sensitivity and ease of use while retaining a high discriminating power.

APPLICATIONS OF DNA TESTING TO GENETIC DISEASE AND PATERNITY. MB19

Patricia Stapleton
DNA Diagnostics, Auckland.

Studies are presented showing the application of DNA technology to investigate a human genetic disease (cystic fibrosis) and paternity. These studies indicate the power of the new technology when applied to difficult problems of diagnostics and identification, not previously able to be resolved by more traditional means.

Prior to the location of the cystic fibrosis (CF) gene and identification of the predominant mutation called ΔF508, the CF mutation in a family could be followed using restriction fragment length polymorphism (RFLP) analysis in families with an affected child. With the identification ofΔF508, and now several other mutations, direct analysis is possible. Use of the polymerase chain reaction (PCR) enables rapid and sensitive analysis.

DNA profiling provides an extremely accurate method for establishing paternity. DNA probes are used to detect a DNA profile (somewhat analogous to a supermarket bar code) which is unique to an individual. The bars in the profile are inherited from one's biological parents and thus the child's paternal bars will be seen in the biological father and not in an unrelated individual. The analysis becomes more difficult when putative fathers are close relatives. Examples will be discussed.

AEROMONAS ISOLATION AND IDENTIFICATION. MB20

D. Riley
Diagnostic Laboratory, 43 Symonds St, Auckland.

Aeromonas species cause a wide spectrum of localised and systemic infections.
The isolation of Aeromonas species from specimens submitted to a Community Laboratory will be reviewed.
Identification methods and the use of Ryans selective medium for isolation from faecal samples will be described.

LEPROSY IN NEW ZEALAND. MB21

Mr Graham Cameron
Microbiology, Wallace Laboratory, Auckland Hospital, Auckland.

Leprosy in New Zealand is reviewed. All cases are imported from the Pacific Islands and Asia.
Methods used by the Microbiology Department, Auckland Hospital to identify and monitor cases are described.

ANOTHER OUTBREAK OF PERINATAL LISTERIOSIS IN AUCKLAND, MB22

Shanthi Ameratunga*, Diana Lennon, Keitha Farmer, David Becroft, Bruce Dove, Maree Gillies, Nigel Yeates, Patricia Short
Departments of Paediatrics, Pathology and Microbiology, Auckland Area Health Board Hospitals and NZCDC, Porirua, New Zealand. *Department of Paediatrics, School of Medicine, University of Auckland, Private Bag, Auckland.

Of all known food-borne pathogens, *Listeria monocytogenes* is associated with the highest case-fatality rate. Outbreaks of epidemic perinatal listeriosis have occurred in Auckland in 1969 and 1980, and we report the descriptive epidemiology of a further outbreak during 1989-1990.

During the 24-month period beginning in January 1989, 17 case pairs were identified. Four (24%) cases resulted in miscarriages, 3 (18%) were intra-uterine deaths, and 10 (58%) resulted in live born infants. Of the latter, 50% were born pre-term, and one was a case of late-onset disease. In all but 3 cases, the mother had a febrile illness during the peri-partum period. The case-fatality rate during this outbreak was 41% and none of the live born infants died. All but 3 isolates were classified as serotype 4b. No evidence of a common-source outbreak was found on the basis of demographic data or phage-typing of the isolates. The estimated annual attack rate for perinatal listeriosis in Auckland in 1989 and 1990, were 40 and 55 per 100,000 live births, respectively.

Conclusions: A marked increase in recognised cases of perinatal listeriosis was noted in 1989-90 in Auckland, which is likely to underestimate the true rate of disease in the population. We as yet have no explanation for the apparent 10-yearly cycles of perinatal listeriosis outbreaks in Auckland. Implications for intensified surveillance and further research will be discussed.

MB23

INTRACRANIAL INFECTION WITH CLOSTRIDIUM PERFRINGENS: CASE REPORT AND LITERATURE REVIEW.

Bruce Dove, Kerry Miller
Microbiology Department, Green Lane/National Women's Hospital, Green Lane West, Auckland 3, New Zealand

A mother presented at hospital for an unbooked delivery with a two week history of ruptured membranes. At birth the male baby weighed 1590g and had respiratory distress. Up until day 25 of age the baby steadily improved although he suffered from a variety of problems associated mostly with his prematurity. On day 26 the baby deteriorated rapidly, with clinical signs of severe sepsis so a lumbar puncture was performed. The CSF examination showed, 10,500 x 10^6/L white blood cells, 350 x 10^6/L red blood cells, numerous Gram positive bacilli, 1.9mmols/L Glucose and 36.8g/L Protein. The baby died shortly after. *Clostridium perfringens* was cultured both from the CSF and blood.

A review of the literature shows that intracranial infections with Clostridia are rare and the majority of cases involve head trauma or CNS surgery. Most clostridial infections of this type are caused by *Clostridium perfringens*. They have a short incubation time with a high mortality rate and classic signs of necrosis of the infected site with associated gas accumulation.

MB24

PNEUMOCOCCAL BACTERAEMIA IN SOUTH AUCKLAND.

T. Voolmann, S.D.R. Lang, K.P. Singh
Microbiology Dept, Middlemore Hospital, Private Bag, Otahuhu, Auckland, New Zealand.

Over 5 years, 1986-90, 149 cases of pneumococcal bacteraemia were identified at Middlemore, a general hospital serving a multiracial community. Records were available for 143. The incidence of pneumococcaemia was 12.1/10,000 admissions, the male to female ratio 1.2:1 and the mortality rate 8.4%. Almost half the patients were over 60 years of age. Over half attended hospital within 48 hours of becoming unwell and only 6% had had symptoms for more than one week. Pneumonia was seen in 82.5% and meningitis in 6%. Seventy six per cent had underlying medical conditions. Empirical treatment was with a single antibiotic in 94%. Following a microbiological diagnosis, more than half were treated with penicillin G, however in one quarter of cases amoxycillin-clavulanate was given as definitive treatment and this expensive practice has become increasingly common.

MB30

THE SENSITIVITY OF URINE SAMPLES COMPARED WITH URETHRAL SWABS FOR THE DETECTION OF CHLAMYDIA TRACHOMATIS INFECTION IN MALES BY ENZYME-IMMUNOASSAY (CHLAMYDIAZYME).

V Lal
Pathology Laboratory, CWM Hospital, Suva, Fiji

Chlamydia trachomatis genital infection has recently been shown to be present in 19% of ante-natal women in Suva, Fiji. In March 1991, a study looking at the prevalence of *Chlamydia trachomatis* infection in males attending the STD Clinic in Suva was done.

As part of this study, the sensitivity of first specimen urine was looked at and compared with urethral swabs for detecting *Chlamydia trachomatis* in males using Chlamydiazyme (Abbott Diagnostics).

Method: 15 to 20mL of first catch urine was collected in a sterile container, centrifuged for 20 minutes at 3000 g's and the supernatant discarded. The pellet was resuspended in 1mL of Chlamydiazyme specimen diluent buffer and Chlamydiazyme test performed. Urethral swabs were collected as per manufacturers guidelines.

Results: Specimens were collected from 82 males. Eighteen (21.9%) urethral swabs and twenty (24.4%) urine samples were positive. (3 specimens were urine positive only and 2 specimens were swab positive only).

Conclusion: First catch urine is a good alternative specimen to urethral swabs for the detection of *Chlamydia trachomatis* urethritis in males using Chlamydiazyme.

MB31

MALTASE ACTIVITY IN USTILAGO VIOLACEA.

F Ghazi
Iran University of Medical Science, PO Box 14155 - 6183 Tehran, Iran.

Specific activities of maltase were compared for strains of *Ustilago violacea* grown on maltose and glucose. The wild type strain produced a higher level of enzyme when grown on maltose than on glucose.

A maltase negative mutant showed poor growth on maltose and lowered specific activity when grown on glucose. This lowered maltase activity was recessive in diploid.

The presence of at least three structural maltases was indicated using ammonium sulphate fractionation gel filtration and a pH activity curve.

HAEMATOLOGY

IS MICROSCOPY ALIVE AND WELL TODAY? HM1

D Crowe, D Rosenfeld
The Liverpool Hospital, the South Western Sydney Area Health Service, Elizabeth Drive, Liverpool, NSW 2170

The place of a Morphologist in a Clinical Haematology Laboratory has been questioned over recent years.

The financial constraints faced by all laboratories has made us look critically at our work practices, to see how we can get best value for our operating budget dollar.

We, like many laboratories looked keenly towards the automated differential, toward a reduction in the number of hours experienced staff would spend in front of the microscope, possible reporting on up to 40% of our films as normal. The advent of some 10 years ago of machines giving multiples histograms and then 5 years ago the three cell differential and more recently five cell differential and scatterplot with Red Cell comments have increased our dependence on our automation.

I will present to you our evaluation and impressions in regard to reducing the number of blood films viewed. I will present to you examples of where dependence on automated differentials may require a change of attitude.

Results will be presented discussing where significant abnormalities were not indicated by the automated differential. The final decision made at our hospital was suitable for our situation. Each laboratory will be faced with similar decisions regarding rationalisation of workload practises while maintaining a quality service.

A CASE OF PYROPOIKILOCYTOSIS. HM2

Mr B Matthews
Haematology Department, the Adelaide Medical Centre for Women and Children, 72 King Williams Street, North Adelaide SA 5006.

A two week old infant presenting with anaemia for investigation was found to have a haemoglobin of 49 grams/litre with a red cell morphology showing greater than normal numbers of microspherocytes and poikilocytes.

Red cell enzymes, osmotic fragility, bone marrow examination and heat fragility led to a diagnosis of Pyropoikilocytosis.

The subsequent management of the anaemia by regular transfusions will also be presented.

INFLUENCE OF MICROCYTES ON PLATELET COUNTS FROM FOUR HAEMATOLOGY ANALYSERS. HM3

D McVeigh#, R Hallawell* and J Allen+
Haematology Departments of Alfred Hospital#, Commercial Rd, Prahran 3181, Royal Melbourne Hospital*, Grattan St, Carlton 3053 and St Vincent's Hospital+, Victoria Parade, Fitzroy 3065.

Modern haematology analysers either manipulate or tabulate data to reduce and/or alert for the presence of microcytic RBCs that can influence the PLT count.

PLT counts from a Technicon H*1, Ortho ELT-800, Coulter S Plus IV and a Sysmex NE8000 were compared with manual PLT counts. Four groups of data were examined. Group A (MCV 80-96, PLT 130-400, n = 31); Group B (MCV < 75, PLT 130-400, n = 36); Group C (MCV 80-96, PLT < 115, n = 35) and Group D (MCV < 75, PLT < 115, n = 9). In Groups A and C, PLT count confidence intervals (C.I.) showed similar results for all analysers and were not different from the manual PLT count C.I. In Group B, all analysers gave a higher C.I. for the PLT count when compared with the manual PLT count C.I., except the NE8000 which had similar results. For Group D all analysers had dissimilar C.I. compared with the manual PLT C.I., But due to small numbers studied little can be inferred from this group.

This study shows that manual PLT counts still need to be performed in patients with a low MCV.

HM4

A REVIEW OF THE ROYAL COLLEGE OF PATHOLOGISTS OF AUSTRALASIA QUALITY ASSURANCE PROGRAM IN HAEMATOLOGY.

JL Smith, WG Hughes
Haematology Quality Assurance Program of the Royal College of Pathologists of Australasia, Westmead Hospital, Westmead, NSW 2145

The objectives of the Haematology Quality Assurance Program are mutli-faceted and continually evolving relying on subscriber support to complete the feed back loop of service, performance, review, education, application and advice availability. In the past six months since accepting the position of Chief Scientist I have been viewing the Program from "the other side of the fence".

The aim of this presentation is to describe the origins, history and administration breakdown; subscriber support and changes in participation numbers over the years 1988 to 1991; precision variations in relation to new technology, reagents and acceptable reporting and to look briefly at the statistics format for results and grouping of performance reviews by method code. Finally I will be gazing into the future, our goals revealed for some additional procedures, education and conservation all of which rely on the continuing support of our subscribers.

ACUTE FATTY LIVER OF PREGNANCY. HM5

Deborah Peterson
Haemostasis Laboratory, Pathology Services, Christchurch Hospital, Private Bag, Christchurch

Acute fatty liver of pregnancy is a rare complication of the third trimester of pregnancy, more commonly affecting young primiparas. The aetiology is unknown but is presumed to be related to a disturbance in liver metabolism of fats.

Early studies reported high infant and maternal mortality rates. Patients may present with a range of clinical problems including malaise, epigastric pain, nausea and vomiting.

Biochemstry and Haemostasis tests show marked abnormalities consistent with liver disease. Haematology abnormalities may be mild, with only the occasional target cell noted in the blood film. Recently the Haemostasis Laboratory at Christchurch Hospital has investigated two patients with acute fatty liver of pregnancy.

These two cases, both with successful outcomes, wil be presented and laboratory findings discussed.

CD56 — CLINICAL RELEVANCE AS DEFINED BY FLOW CYTOMETRY. HM6

S Rockman, L Robb, S Houghton and K McGrath
Department of Diagnostic Haematology, Royal Melbourne Hospital, Grattan St, Parkville, Vic, 3050

CD56 is an antibody cluster that binds to NCAM, the neural cell adhesion molecule which mediates homeotypic interactions between neural and muscle cells. In the haemopoietic setting, this molecule is expresed by virtually all NK cells, the majority of these cells circulate in the peripheral blood (10-15% of normal lymphoid cells) and play a significant role in the host's defence against viral infections. CD56 has been suggested as being of value in the diagnosis of "chronic fatigue syndrome". To date we have tested 10 individuals referred to our laboratory and have failed to demonstrate a deficiency in NK cells compared to normals. The wide variation in this marker in normal individuals increase the false positive/negative result and, therefore, the test is not of any benefit. On the other hand, CD56 is a useful marker in the diagnosis of large granular lymphocytosis/leukaemia. Human NK cell malignancies are considered rare (3 cases of childhood ALL and some 100 described cases of chronic proliferative disorders), however, routine immunophenotyping of individuals with lymphocytosis over the past 3 years in our laboratory has revealed a significant number of cases (11/464). Each of these cases had distinct T cell phenotype patterns yet the CD56 positivity was consistent. Southern blotting analysis revealed that 5/6 of these cases had T cell receptor gene rearrangements.

IRON DEPLETION IN NON-ANAEMIC WOMEN. HM7

C Bowlen
Haematology Department, Gippsland Pathology Service, Traralgon, Victoria, 3844

Iron deficiency occurs as a result of a negative iron balance and usually develops over a long period of time in 3 stages. The first stage is Iron Depletion (or latent iron deficiency) in which iron stores are reduced but iron supply to the developing red cell is maintained and the haemoglobin, red cell parameters and film appearance are normal. In the 2nd stage, iron stores are low or exhausted, supply to the developing red cell is reduced and abnormal erythropoeisis ensues. At this stage the haemoglobin, indicies and film appearances are usually still normal. The final stage is iron deficiency anaemia where haemoglobin indicies and film appearances are all abnormal.

In a retrospective study of over 1000 women of child bearing age with reduced iron stores, over 30% were found to have normal haemoglobins, indicies and film appearance — fitting into stage 1 or 2.

The study suggests that women between the ages of 15 and 50 with haemoglobin levels approaching the lower limit of normal (120-125 g/l) and normal MCV's could well be in the first or second stage of iron deficiency and many benefit from iron supplementation.

(1) Cook JD. Clinical Evaluation of Iron Deficiency Seminars In Haematology 1982; 19.1: 6-17
(2) Charlton RW, Bothwell TH. Definition, Prevalence and Prevention of Iron Deficiency Clinics In Haematology 1982; 11-12: 309-325

HAEMATOLOGY REFERENCE RANGES IN PREGNANCY DERIVED FROM STORED PATIENT DATA. HM8

AJ Balloch and MN Cauchi
Department of Haematology, Royal Women's Hospital, Carlton 3053, Vic, Australia

The selection of individuals for the determination of reference ranges has, in the past, been difficult because health and normality are poorly defined conditions (1).

Several publications have suggested that reference ranges for Haematology parameters can be determined by the mathematical detection of Gaussian distributions (2) or Gamma distributions (3) of partitioned but unselected patient data.

These procedures were applied to our patient data with the aim of investigating the application of these methods and establishing reference ranges for our antenatal population.

The patient data was partitioned into four groups: 1) Non pregnant; 2) First Trimester (< 14 wks); 3) Second Trimester (14 - 26 wks); 4) Third Trimester (> 26 wks). The primary red cell parameters, in each group, were all shown to be well described by Gaussian distributions. Platelet and White Cell parameters were best described by Gamma distributions, with the exceptions of Eosinophils, Basophils and Band forms for which neither distribution was applicable.

These methods of detecting frequency distributions within patient data enabled us to determine refernece ranges in pregnancy specific to our antenatal population and methodolgies.

(1) IFCC The Theory of Reference Ranges (Part 2). J.Clin.Chem.Clin.Biochem. 1984; 22: 203-208
(2) Naus AJ, Borst A, Kuppens PS. J.Clin.Chem.Clin.Biochem. 1980; 18: 621-625.
(3) Swaanburg JCJM, Rutter WPF, Holdrinet ACJM, VanStrik R. A.J.C.P. 1987; 22: 182-191.

PROTHROMBIN FRAGMENT 1 + 2. HM9

MJ Elms
Haematology Division, Department of Pathology, Royal Brisbane Hospital, Brisbane, Australia

Prothrombin Fragment 1 + 2 (F1 + 2) is a prothrombin activation peptide useful for diagnosing the prethrombotic state and assessing thrombotic risk. It is a polypeptide released from prothrombin during its activation to thrombin by prothrombinase. F1 + 2 levels in plasma reflect the degree of prothrombin activation and correlate with the degree of thrombotic risk. Levels in normal

plasma range between 0.43-1.11 nm/L and increase with advancing age. Elevated levels have been demonstrated in individuals suffering a variety of thrombophilic situations including Deep Venous Thrombosis, Myocardial Infarct, presence of Lupus anticoagulant as well as Protein C/S deficiency, and Antithrombin III deficiency. F1 + 2 levels are reduced below normal upon introduction of oral anticoagulant therapy. F1 + 2 levels are lowest when the INR is highest. F1 + 2 is quantitated using Enzyme Linked Immunosorbent Assay (ELISA). Currently two ELISA's are commercially available in Australia (Behring and Organon Tecknika). This paper wil present initial experience with these two kits as well as present data of patient groups studied, and project potential uses for this important marker of thrombotic risk.

HM10

TWO RELIABLE METHODS FOR THE DETECTION OF TERMINAL DEOXYNUCLEOTIDAL TRANSFERASE (TdT).

Helen Hanlin and Robert Carter
Haematology Department, St Vincent's Hospital, Fitzroy, 3065. Australia

TdT is an important marker of primitive lymphoid cells, unfortunately dogged by technical difficulties associated with its assay. We have modified the functional biochemical assay of Bollum (Blood, **54**; 1203, 1979) to make it less laborious and more reproducible. This assay requires large numbers of cells and, although highly specific, does not give information about particular cells.

A flow cytometric method (Almasri, et al., Am J Clin Pathol, **95**; 376, 1991) using a mixture of monoclonal antibodies was compared with the functional assay. This method has the potential ability to analyze TdT in blast cells within a mixture of cells. Although the latter method is more expensive it has a high degree of sensitivity and specificity and requires far fewer cells.

HM11

A CASE OF AN INHIBITOR TO PROTEIN C.

CJ O'Malley, R Dauer, KM McGrath
Coagulation Laboratory, Department of Diagnostic Haematology, Royal Melbourne Hospital, c/- RMH Post Office, Parkville, 3050, Australia

Protein C is a vitamin K dependent protein which acts as a potent anticoagulant and profibrinolytic agent in vivo. A deficiency of protein C has been shown to predispose to an increased risk of thrombotic events. Methods for protein C estimation commonly used include chromogenic, antigenic, APTT based and Xa one-stage clotting assays. Reports (1) in the literature have described some Type II deficiencies of protein C with normal chromogenic levels of protein C but reduced levels in clot-based assays. A family is described here with normal levels of protein C by chromogenic, antigenic and two commercial APTT based assays with an abnormal (in-house) APTT assay. The investigation of these samples showed the presence of an inhibitor to activated protein C which appears to have different characteristics from previous inhibitors described in the literature.

(1) Vigana-D'Angelo S, Comp PC, Esmon CT, D'Angelo A. Relationship between protein C antigen and anticoagulant activity during oral anticoagulation and in selected disease states J Clin Invest 1986; 77: 416-25

HM12

THE SIMULTANEOUS QUANTITATION OF TOTAL AND FREE PROTEIN S BY ELISA.

RJ Dauer, SR Downer, CJ LO'Malley and KM McGrath
Coagulation Laboratory, Department of Diagnostic Haematology, Royal Melbourne Hospital, c/- RMH Post Office, Parkville, 3050. Australia

Deficiency of Protein S (PS), a Vitamin K dependent glycoprotein, has been associated with an increased risk of thrombosis. The measurement of PS is relatively simple using immunological assays. Electroimmunoassay is widely used, however, it is only sensitive to levels of 10% and is less suitable for the determination of free PS. We are currently performing PS assays using an ELISA method (1) which allows for the simultaneous measurement of both total and free PS following the precipitation of PS complexed to C4B binding protein using polyethylene glycol.

Using this system we have found the levels of free PS to be significantly reduced in liver disease patients ($n = 20$, $p = 0.0009$) and those treated with warfarin ($n = 13$, $p = 0.0001$) compared to healthy subjects ($n = 31$). Total PS was also significantly reduced in these two patient groups albeit to a lesser extent, $p = 0.0287$ and $p = 0.0279$ respectively.

This method for PS is a reproducible, reliable and inexpensive assay for the simultaneous quantitation of total and free portions of PS.

(1) Woodhams B. The Simultaneous Measurement of Total and Free Protein S by ELISA. Thrombosis Research, 1988, 50: 213-220.

HM13

NEW MUTATION IN CODON 22 of α SPECTRIN IS ASSOCIATED WITH STRUCTURALLY AND FUNCTIONALLY ABNORMAL α SPECTRIN (SP α I/74) IN HEREDITARY ELLIPTOCYTOSIS.

J Rutherford[1], L Glogoski[1], J Palek[2], H Blacklock[1]
Middlemore Hospital, Auckland[1] and St. Elizabeth Hospital, Tufts University, Boston, MA, USA[2].

Hereditary elliptocytosis (HE) is a red cell membrane disorder with not only heterogenous molecular mutations, but also variable clinical expression. Mutation of codons 22, 40 and 43 of α spectrin have been reported, resulting in defective spectrin heterodimer self assocation and abnormal tryptic cleavage of the 80kd α I domain with increased amounts of a 74kd peptide (Sp I/74).

In two apparently unrelated kindred of European extraction, a mutation at codon 22 in exon 2 of α spectrin has been identified, the mutation being CGT ---→ TGT; Arg ---→ Cys 22. Although each affected individual has the same molecular mutation in a ---→ heterozygous form, the clinical disease ranges from severe haemolysis in early childhood (with significant mortality) to mild haemolysis. Splenectomy in those severely affected has resulted in reduced haemolysis and normalisation of haemoglobin levels.

HM14

A COMPUTERISED ALGORITHM FOR HANDLING THE OUTPUT OF MODERN FULL BLOOD COUNT ANALYSERS.

G Kershaw, P Burgess, R Coleman, H Robin, C Coad, J Gibson
Haematology Department, Royal Prince Alfred Hospital, Missenden Road, Camperdown NSW 2050, Australia

This paper describes a computer system developed to handle the output of the Technicon H*1 (or H*2) and the Sysmex NE-8000. At the heart of the system is an algorithm which relieves the technologist of the complex task of reviewing each printout. The patient's current FBC (including morphology flags) and previous FBC with film comments are fed into the algorithm. The algorithm decides if a blood film is necessary and whether to accept, reject or delete the machine differential. The algorithm flows forward from step to step, with occasional forward jumping of steps if certain conditions are met. The majority of steps are simple delta checks, where the limits are the result of experience. Special routes are used for patients from special wards. A side printer listing for each specimen is produced immediately the specimen has been run, whether in automated or stat mode. This listing contains patient demographics, key results, phone alert messages, date and time run, and which additional tests to perform on the specimen. Abnormal samples requiring operator intervention are automatically placed into a hold file for later editing. The system operates 24 hours a day, 7 days a week and has greatly enhanced the efficiency of the laboratory.

HM15

INITIAL PERFORMANCE STUDIES OF A PLASMINOGEN ACTIVATOR INHIBITOR KIT.

K Kelly, J Madgwick
Haemostasis Laboratory, Department of Haematology, Auckland Hospital, Park Road, Auckland.

Elevated lavels of Plasminogen Activator Inhibitor (PAI) activity in plasma have been shown to correlate with a clinical thrombotic tendency. This study was performed to determine the suitability of the Behringwerke AG PAI kit for use in our laboratory. The assay is a simple amidolytic procedure. PAI levels were measured in 10 normal healthy controls and in 16 patients with recurrent thrombosis. Pre and post-venous occlusion samples were collected on each patient and Euglobulin Clot Lysis (ECL), Fibrin Plate Lysis (FPL) tests, and PAI levels were measured for each sample. Patients shown to have normal ECL and FPL results appeared to have PAI levels (1.5 + 1.2 IU/ml) similar to the normal controls (2.6 + 1.8 IU/ml) while the patients with abnormal ECL and FPL results had much higher PAI levels (4.6 + 0.8 IU/ml). Statistical correlations are not available due to the small size of sample groups. These results suggest that there is a correlation between fibrinolytic screening tests such as ECL and FPL and PAI levels as measured by this kit.

HM16

HAEMOSTASIS SCREENING IN PATIENTS PRESENTING WITH GASTRO-INTESTINAL BLEEDING.

J Madgwick, C Ellis, G Royle
Haemostasis Laboratory, Department of Haematology, Auckland Hospital, Park Road, Auckland and Haemostasis Laboratory, Department of Haematology, Middlemore Hospital, Middlemore Road, Auckland.

The results of coagulation screening tests carried out on 200 patients with gastro-intestinal bleeding seen at Auckland Area Health Board Hospitals were assessed in terms of their usefulness in patient management. Overall 24% of results were abnormal. 7.5% of patients had a prolonged activated partial thromboplastin time (APTT) and raised Prothrombin Ratio (PR), 8% had a prolonged APTT only and 8.5% had a raised PR only. 3% had reduced fibrinogen levels. However in no case was this the sole abnormality. Our findings indicate that although the APTT and PR are useful, fibrinogen assays contribute little information and we have modified coagulation screening accordingly. Such audits of local experience enable the laboratory to improve the quality of service it offers.

HM17

CLINICAL EVALUATION OF AN ERYTHROPOIETIN ASSAY (ELISA) AS AN ANCILLARY DIAGNOSTIC TEST FOR POLYCYTHAEMIC PATIENTS IN SOUTH AUCKLAND.

R Bluck, H Blacklock
Department of Haematology, Middlemore Hospital, Private Bag, Otahuhu, Auckland

An Elisa technique using the Amgen Clinogen™ Erythropoietin EIA kit has been used as an ancillary diagnostic aid in our patients with an absolute increase in red cell mass, particularly in those with idiopathic erythrocytosis or unclassified polycythaemia. Fifty patients were categorised using the criteria of the polycythaemia vera study group into 4 groups, A: polycythemia rubra vera, B: secondary polycythaemia, C: idiopathic erythrocytosis and D: those with criteria for both PRV and SP. The erythropoietin levels in the four groups were:

	RANGE*	No. TESTED
A median — 2 mU/ml	0.5 - 9	18
B median — 5.8 mU/ml	1.8 - 150	13
C median — 4.0 mU/ml	0.3 - 11	17
D	1.7 - 4.85	2

The results provide useful extra data in patients with polycythaemia rubra vera and secondary polycythaemia tested to date. Patients with idiopathic erythrocytosis or unclassified polycythaemia will be discussed in more detail.

*5 - 95%

HM18

CRYOHAEMOLYSIS: A NEW DIAGNOSTIC TEST FOR THE DETECTION OF HEREDITARY SPHEROCYTOSIS.

J Allen
Department of Haematology, St Vincent's Hospital, Victoria Parade, Fitzroy 3065, Victoria, Australia.

The characteristically spherical shape of red cells in hereditary spherocytosis may be quantitatively assessed in terms of osmotic fragility measurements. Due, not only to the complexity of the osmotic fragility test, but also to its lack of sensitivity even with blood preincubated at 37°C, more reliable screening tests have been sought.

The glycerol lysis test and its acidified modifications as well as other so called screening tests have appeared in the literature over the past 10 years, but these tests have not proved adequately specific.

The hypertonic cryohaemolysis test, which is based on the principle that red cells suspended in hypertonic solution are incubated at 37°C and then transferred to 0°C, has been reported to be highly specific and sensitive.

This paper provides the first independent report confirming the value of the cryohaemolysis test by providing a 100% detection rate of hereditary spherocytosis in over twenty patients, with no false positives detected due to spherocytes from acquired haemolytic anaemias. From the results obtained, this test is recommended as the diagnostic test of choice.

HM19

EVALUATION OF AN RIA ERYTHROPOIETIN ASSAY.

A Johnston, RJ Dauer, & K McGrath
Department of Diagnostic Haematology, Royal Melbourne Hospital, Grattan Street, Parkville, Victoria 3050, Australia.

Erythropoietin (EPO) is a glycoprotein hormone, primarily produced in the kidneys and released in response to anaemia or tissue hypoxia. It regulates the rate of proliferation and differentiation of erythroid precursors in the bone marrow via a complex feedback system.

The Incstar EPO-Trac ^{125}I RIA kit is a competitive binding, disequilibrium radio immunoassay which utilises recombinant human EPO for both tracer and standards. Using normal donors we derived a normal range of 4.4-24.6 mU/ml (n=85) with no statistical difference between males and females. There was a negative correlation between haemoglobin and the log EPO levels (r=0.64, n=27) in patients with simple anaemia not complicated by renal disease or inflammation. The renal group (n=14) had lower EPO levels compared with the simple anaemia group (n=27) which were significantly different (p=0.0011), suggesting that renal patients have a decreased EPO response to anaemia. We also found that the rheumatoid arthritis RA group when compared with the simple anaemia group with comparable haemoglobins. (80-110g/L), showed a significant difference (p=0.0089). Our results are consistent with Baer et al (1) and Takashina et al (2) who demonstrated a blunded EPO response in anaemic R.A. patients.

Our evaluation of this method proved it to be a robust and sensitive assay.

(1) Baer A.N. et al *British Journal of Haematology*, 1987, 66, 559-564.
(2) Takashina N. et al *J Reumatology* 1990; 17:885-7.

HM20

TRANSIENT MYELOPROLIFERATIVE SYNDROME IN A BABY WITH TRISOMY 21.

L Glogoski[1], E Thekston [1], J Nelson[3], N Stewart[1], D Newman[2], H Blacklock[1]
Departments of Haematology[1] and Paediatrics[2], Middlemore Hospital, South Auckland, and Haematology[3], Auckland Hospital.

A Tongan baby presented at birth with clinical trisomy 21, with hepatomegaly (4cms), myeloblasts in the cord blood (4.21 x 10^9/l) and thrombocytopenia (40 x 10^9/l). Subsequent problems included pulmonary hypoplasia with chronic oxygen requirement, and a VSD requiring medical management. Jaundice and melaena were also noted. PB cell surface marker studies showed a significant population of megakaryoblasts CD42b (GpIb) +ve 16%, CD61 (GpIIa) +ve 18%, but the majority of the blast cells were undifferentiated CD34 +ve 24%; td+ -ve; CD13 +ve 22% (weak), CD33 +ve 31% (weak). PB chromosomes confirmed standard trisomy 21 with no additional abnormalities.

The baby's blast count increased to 11.87 x10^9/l on day 11 and then slowly decreased over the next month. The increase in circulating blasts was associated in time with hepatomegaly (10cms), splenomegaly (4cms) and significant renal failure which all gradually improved. The presentation and clinical course were consistent with a transient myeloproliferative syndrome rather than acute leukaemia. Clinical and laboratory data to differentiate these two entities will be presented, with a review of the literature.

HM21

THALASSAEMIA AND OTHER HAEMOGLOBINOPATHIES IN SOUTH AUCKLAND — CLINICAL IMPLICATIONS OF RECENT IMMIGRATION PATTERNS.

H Blacklock[1], J Rutherford[1], J. Rea[2]
Department of Haematology[1], Middlemore Hospital, and Mangere Refugee Camp[2], South Auckland.

The change in New Zealand's immigration policy in recent years has increased the diversity of the population, particularly in South Auckland. This factor, as well as the immigration of refugees to our local area, has increased the number of people with clinically significant haemoglobinopathies (either directly or potentially) — the main contribution being from peoples of Indo-Chinese origin, but also from the Pacific Islands.

In the 12 months from May 1, 1988 to April 31, 1989, of 27,462 persons immigrating into New Zealand, 10,227 (37%) were from the South Pacific, 4,458 (16%) from South Asia and 3,689 (13%) from North Asia.

This immigration of peoples at risk, as well as the alpha thalassaemia that occurs in our own Maori and Polynesian population, has implications for the delivery of our future health services. These requirements, may include an increase in transfusion dependent patients, the cost of desferrisoxamine, increased demand for antenatal diagnosis, genetic counselling and DNA testing.

A CASE OF PRECIPITATING ANAEMIA. HM22

HF Stunzner
Medlab Limited. 125-129 Grafton Road, Auckland.

Two brothers aged eight and nine years have had haemolytic anaemia since birth. They have weekly blood counts to monitor the Hb level in order to determine the need for blood transfusion. Because transfusions were becoming more frequent, splenectomy was performed on both the patients.

Post splenectomy blood counts, particularly on the Technicon H*1 analyser showed very abnormal patterns in RBC volume and HB concentration. An increasing volume and decreasing concentration appears to give an indication of the amount of precipitated haemoglobin present. These patterns together with a falling Hb level and increase in nucleated red cells in the blood film, had some predictive value for a haemolytic crisis.

Previous tests to determine the cause of the haemolytic anaemia were inconclusive. However, following splenectomy, tests for haemoglobinopathy on one of the patients demonstrated the presence of an abnormal haemoglobin. This has been identified as a new unstable haemoglobin with a mutation in peptide beta 9 : beta 67 Valine → Glycine.

BLOOD GROUP TYPE IN VON WILLEBRAND'S DISEASE (vWD). HM23

K Belton and P Ockelford
Diagnostic Laboratory, 43 Symonds Street, Auckland.

Precise diagnosis of vWD is difficult because of the variable phenotypic expression both clinically and by in vitro laboratory tests. Diagnosis is further complicated by the confirmed reports of lower levels of von Willebrand factor (VIII:vWF), FVIII:C and VIII:RiCof (Ristocetin Cofactor) in patients with the blood group type O relative to other blood group types. A recent report suggested that patients with blood group O also have mildly but significantly longer bleeding times. The relationship between blood group type, factor VIII parameters and bleeding time has been evaluated in 143 consecutive patients presenting to Diagnostic Laboratory with a suspected minimal bleeding disorder.

	FVIII:C (%)	VIII:vWF (%)	VIII:RiCof (%)	BT
group O ($\bar{x}$±ISEM	72 ± 3	79 ± 6	73 ± 5	8.5 ± 0.5
non group O	105 ± 6	125 ± 5	125 ± 7	7.5 ± 0.5
p value	<0.001	<0.001	<0.001	n.s.

These results indicate significantly lower factor VIII parameters in patients with blood group type O and confirm the findings in normal subjects. A significant difference in bleeding times between the two patient groups was not observed although there is a trend towards longer bleeding times in the group O subjects. These findings suggest that the blood group type O should be considered before making a diagnosis of vWD, especially where factor VIII parameters are only marginally reduced.

HAEMATOLOGIC PARAMETERS IN BORDERLINE HYPERTENSIVE MEN. HM30

RW Siebers, TJ Maling and JM Carter
Departments of Medicine and Pathology, Wellington School of Medicine, P.O. Box 7343, Wellington South, New Zealand.

Alteration of haematocrit (Hct) and platelet mean volume (MPV) have been demonstrated in established hypertension. Increased Hct is attributable to reduced plasma volume[1] and may contribute to increased blood viscosity and thus mean arterial pressure (MAP), while the increased MPV is reported to reflect increased platelet reactivity[2]. The study objectives were to determine if the reported haematologic alterations in established hypertension are apparent in borderline hypertensive men (BHT).

Fourteen untreated BHT with MAP OF 113 mm hg (± 9.4) were age and weight matched with 14 normotensive men (NT) with MAP of 87 mm hg (± 6.1). All subjects were Caucasian because of reported racial differences in haematologic parameters[3]. Blood was sampled for determination of Hct, Hb, red cell count (RBC), platelet count and MPV. Statistical analysis was by unpaired student's t test, $p < 0.05$ was deemed statistically significant.

Hct, Hb and RBC were significantly increased in BHT (0.46 ± 0.032; 159g/L ± 11.8 and 5.19 x 10^{12}/L ± 0.42 respectively) compared to NT (0.43 I 0.014; 147 g/L ± 5.8 and 4.83 x 10^{12}/L ± 0.22). Platelet counts and MPV were not significantly different between BHT (258 x 10^9/L ± 48 and 7.8 fl ± 0.8 respectively) and NT (260 x 10^9/L ± 64 and 8.0 fl ± 1.2). MAP was significantly correlated with Hct (r = 0.67), Hb (r = 0.71) and RBC (0.70) in BHT, but not in NT. The results demonstrate that the previously reported alteration in Hct in established hypertension is also present in BHT, but MPV is not altered in BHT.

1 Kobrin I, et al. *J Lab Clin Med* 1984; 104:11-14.
2 Lande K, et al. *J Hypertens* 1987; 5:401-406.
3 Siebers R, et al. *NZ Med J* 1989; 102: 588-589.

HM31

DEVELOPMENT OF A SIMPLE ELISA BASED COLLAGEN BINDING ASSAY PERMITS SENSITIVE DISCRIMINATION BETWEEN TYPE I AND TYPE II VON WILLEBRAND'S DISEASE.

EJ Favaloro, L Grispo, T Exner and JH Koutts
Department of Haematology, Westmead Hospital, Westmead, 2145.

We have used modifications to the assay of Brown and Bosak[1] to develop a simple ELISA based collagen binding assay (CBA), that can be used by any laboratory currently performing a standard von Willebrand factor antigen (vWFAg) ELISA procedure, to permit simultaneous and sensitive discrimination between Type I and Type II von Willebrand's disease (vWD; see summary table

below, also ref.2.). Thus, while plasma samples from both Type I and Type II v WD patients show a reduction in vWFAg, CBA and Ristocetin cofactor (RCof) levels compared to normals, CBA values in Type II vWD are considerably lower than those observed in Type I vWD. Furthermore, whilst the 'ratio' of vWFAg:CBA in Type IvWD was not significantly different to those observed in normals (p=0.386), ratios observed in Type II vWD were invariably much higher, and have shown no overlap between patient groups in samples tested to date. The CBA therefore appears to constitute a novel functional assay for vWF, is far superior to the RCof assay in it's ability to distinguish between Type I and Type II vWD patients, and may allow for the future discovery of new dysfunctional forms of vWF in affected patients.

			PERCENT*			vWFAg:CBA
		n	vWFAg	CBA	Rcof	
a.	Type I vWD	37	47.3	60.7	31.1	0.94
b.	Type II vWD	16	34.9	1.6	11.9	48.0

* Meanvalues as percent of pooled normal plasma mean = 100%.

1 Brown JE, Bosak JO, Thromb Res, 1986; 43:303-311.
2 Favaloro EJ, Grispo L, Exner T, Koutts J. Blood Coag Fibrinol; 2:285-91.

ABNORMAL SCATTERPLOTS ON COULTER STKS: RELATIONSHIP TO CLINICAL ABNORMALITY. HM32

J Iles Mann
Department of Haematology, Westmead Hospital, Westmead, 2145.

Over the past 12 monthgs, 30 patients at this institution were noted to give grossly abnormal scatterplots on our Coulter STKS automated Haematology Analyser. Such scatterplots correlated very poorly with manually derived differential counts, and were seen to be consistent in their behaviour (that is, patterns were highly reproducible between this group of patients and appeared to by cyclic within patients). In order to assess the mechanism leading to abnormal scatterplots in these patients, biochemical data, other haematology data, and clinical parameters (including treatment protocols) were assessed and analysed. The majority of these patients (19/30; 63%) were found to have some manner of liver dysfunction whilst 10 patients (33%) suffered some form of renal disease. All samples giving abnormal scatterplot data were also separated into plasma and cell components; cells resuspended into normal AB serum gave normalised scatterplots, implicating the involvement of a plasma component in this phenomenon. In this "liver dysfunction" group, 18 patients (95%) were noted to have elevated bilirubin levels. 19 patients (100%) also had elevated Alkaline phosphotase (ALP) levels, 19 patients (100%) had elevated Gamma glutamyl transferase (∂ GT) levels,m and 9 patients (47%) had elevated urea. In the "renal disease" group, the majority of patients were also found to have elevated urea (7/10:70%) whilse a significant proportion also had elevated bilirubin (4/10:40%), elevated ALP (3/10:30%), or elevated ∂ GT (2/10:20%). Scatterplots also appeared to normalise in both patient groups, following treatment protocols to improve the symptoms of the prevailing disease (thus "biliary stent" treatment in the case of the liver dysfunction group resulted in normalised scatterplots as did plasmaphoresis/drug therapy for the "renal disease" group). Work is continuing in order to pinpoint the specific mechanism(s) involved in this interesting laboratory phenomenon.

EVALUATION AND COMPARISON OF COAGULATION QUALITY CONTROL PLASMAS. HM33

L Grispo, EJ Favaloro, T Exner
Department of Haematology, Westmead Hospital, Westmead, 2145.

Quality Control (QC) plasma samples from nine separate coagulation supply companies, comprising both normal and abnormal QC samples, have been evaluated by us in a comparative analysis study. Samples were tested for behaviour in both PT and APTT assays in regard to various parameters, including precision, stability following reconstitution, and for storage stability (using dry storage at 37°C to mimic longer term storage at more optimal conditions). Whilst most plasma samples performed adequately in our study, some gave unacceptable results. Inter-run precision for all QC 'normal' plasmas was such that CV's were in the vicinity of 2%, as were CV's for 'in-house' study control plasmas. CV's obtained for 'abnormal' QC plasma samples were generally higher (3-8%). Most (but not all) QC plasma samples showed notable signs of deterioration following storage of dry vials at 37°C for 1 month. Most (but not all) QC plasma samples (both 'normal' and 'abnormal') showed an acceptable level of stability following reconstitution. Interestingly, QC plasma stability (tested over a 24 hour period) appeared to be better for some QC plasmas (particularly 'normals') when held at room temperature rather than at 4°C. Whilst the general trend for most plasmas was an upward drift in PT/APTT with time, some plasma actually showed a downward trend. In an attempt to explain some of these findings, and to help provide reasons for discrepancies observed in the behaviour of different QC plasmas, measurements for pH, refractive index, estimates of turbidity and dry matter weights have also been undertaken and data compared.

HM34

AN EVALUATION OF THE SYSMEX R-3000 RETICULOCYTE COUNTER, AND ITS USEFULNESS IN PREDICTING BONE MARROW REGENERATION.

R Coleman, C Coad, H Robin, H Kronenberg
Haematology Department, Royal Prince Alfred Hospital, Missenden Road, Camperdown, New South Wales 2050, Australia.

The Sysmex R-3000 is a new instrument recently released by Toa Medical Electronics for counting reticulocytes. This instrument uses a flow cytometry system, incorporating an Argon laser light path which strikes a fine stream of red cells supravitaly stained with Auramine-0. A scattergram is produced which plots scatter versus fluorescence. Using this method, plus an Algorithm, the R-3000 provides RBC, Reticulocyte %, Reticulocyte absolute, and three new parameters which are an indication of reticulocyte maturation. Using these new parameters it is hoped to predict marrow regeneration after transplant.

FIXED WHITE CELLS FOR USE IN A SECONDARY STANDARD ON WHOLE BLOOD ANALYSERS. HM35

AP Dimos, R Coleman H Robin, I Pegler, H Kronenberg
Haematology Department, Royal Prince Alfred Hospital, Missenden Road, Camperdown, New South Wales 2050, Australia.

The introduction of new technology to whole blood analysers has rendered the previous method for preparation of white cells for in-house secondary standard control unsatisfactory. A simple, relatively quick method has been developed to produce pseudo white cells. Human white cells are obtained from suitalbe apheresis donors. Contaminating red cells and platelets are removed by lysis and centrifugation. The white cells are washed, fixed and stored in a stabilising solution at 4°C. These fixed white cells are then seeded into an in-house fixed red cell-platelet preparation. Testing for infectious contaminants is carried out. This preparation has been found to be suitable for use in Technicon H1, Sysmex NE8000 and Coulter S Plus technology giving satisfactory linear regression and coefficients of correlation over a period of two months in all machines. In addition it is supplied to the RCPA QAP for use in Sysmex NE8000 users' samples.

HM36

COMPARATIVE EVALUATION OF THREE HETEROPHILE ANTIBODY TESTS IN THE DIAGNOSIS OF INFECTIOUS MONONUCLEOSIS.

D Seely, JD Scarlett, K Stuart, D Herrmann.
Department of Haematology, The Geelong Hospital and Pathology Services, Geelong.

Infectious mononucleosis (IM) an acute Epstein Barr virus (EBV) infection, is associated with the presence of circulating heterophile antibody. Routine laboratory investigation includes a test for the heterophile antibody using a commercial kit. The majority of these kits are based on the agglutination of horse erythrocytes. The aim of this study was to evaluate the use of an alternative method employing latex particles in place of horse erythrocytes. The Oxoid latex agglutination kit (Oxoid Diagnostic Reagents) was compared to two red cell agglutination kits, Monospot (Ortho Diagnostic Systems) and CSL IM Differential kit (Commonwealth Serum Laboratories). These tests were performed on specimens obtained from 250 consecutive patients upon whom IM screening serology was requested. The results were correlated with the blood film comment and absolute lymphocyte count. Where a discrepancy existed between kit results EBV specific serology was performed. 46 were positive with all tests and most of these had absolute lymphocytosis and/or atypical lymphocytes reported on the blood film. Of the remaining 19 positive specimens, four were positive to Monospot and CSL, two to Monospot alone, two to CSL and Oxoid and 11 to Oxoid alone. EBV EgM antibody was assayed in 16 of the 19 "discrepant" positive specimens with negative results in all cases. Commercial heterophile antibody tests have a false positive rate and laboratories should not diagnose Infectious Mononucleosis on the basis of a positive test alone, without compatible blood findings. The latex test showed more false positive results in our study than the tests based on horse red cell agglutination.

EVALUATION OF THE COBAS ARGOS 5-DIFF HAEMATOLOGY ANALYSER. HM37

J Allen and B Sonza
Department of Haematology, St Vincent's Hospital, Victoria Parade, Fitzroy, 3065, Victoria, Australia.

The Cobas Argos 5-Diff Haematology Analyser is a fully automated cell counter providing full five part white cell differential counts and flagging, as well as the standard 12 part full blood count. The instrument combines electrical impedance measurements, incident light scatter and cytochemical staining to count and classify cells.

The instrument was evaluated according to the ICSH[1] protocol, the results proving satisfactory in all areas tested. The high throughput of 120 samples per hour combined with the very low number of false positive and false negative flags generated make the instrument not only extremely efficient but also extremely accurate.

This paper will present the results of the evaluation together with some illustrative examples of haematology cases analysed by the instrument.

[1] Protocol for the evaluation of automated blood cell counters. Clin. lab. Haemat. 1984, 6. 69-84.

vWF:Ag ESTIMATION BY LATEX AGGLUTINATION. HM38

P. Ockelford
Diagnostic Laboratory, Auckland.

A semiquantitative latex test for FVIII:vWF has been evaluated as a screening method for detecting a reduced level of vWF:Ag. One hundred and nine patients presenting to Diagnostic Laboratory for evaluation of a suspected minimal bleeding disorder had the vWF:Ag measured by the Dade Latex Aggflutination test (Baxter Healthcare Corp., USA) and by a reference EIA method. Three different lot numbers of the latex test were compared with the EIA method and the results expressed as positive and negative predictive values (PPV, NPV).

Kit		PPV	NPV
Lot 1	(n = 35)	95%	60%
Lots 2 & 3	(n = 74)	77%	63%
Overall	(n = 109)	83%	62%

The PPV, the likelihood of a latex agglutination result >70% being associated with an EIA result >70%, of the initial lot evaluated indicated that the latex test could be a suitable screening test for clinical use. Subsequent batches of the same kit did not confirm the same high predictive value. The variability between batches severely limits the usefulness of the Dade Latex vWF:Ag test as a screening method.

IMMUNOHAEMATOLOGY

H.D.N.B. ASSOCIATED WITH ANTI-ELO. IH1

DS Ford*, DA Stern*, DN Hawksworth*, A Lubenko+, JM Pope+, MS Chana+, PJ Better#
*National Blood Group Reference Laboratory, CSL, Parkville, Australia. +North London Blood Transfusion Service, UK. #Mercy Hospital for Women, Melbourne, Australia.

Antibodies to over 30 low frequency antigens (LFA) have been reported to have caused HDNB[1,2], but anti-ELO has never yet been implicated.

A 33 year old G5P3 woman of New Zealand Caucasian origin gave birth in August 1990. Tests on the child 24 hours after birth showed a raised bilirubin and positive direct antiglobulin test due to IgG coating. An antibody to an LFA was suspected when the maternal serum was non-reactive with all cells of red cell panels but reacted strongly with her husband's cells.

Investigations at NBGRL and North London BTS identified anti-ELO in the maternal serum. Family studies showed ELO to be inherited in a dominant fashion through three generations and to be independent of RH, Gc, ADA and PGM_1. Resistance to enzyme treatment supports independence from MNS.

References

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2 Mollison PL, Engelfriet CP, *Contreras M. Blood Transfusion in Clinical Medicine*, 8th ed. Blackwell Scientific, Oxford, 1987.

CHIMERISM — A TWIN DILEMMA. IH2

K McLoughlin, Sue Haines and Sue Robertson
Department of Transfusion Medicine, Christchurch Hospital.

The discovery of a weak agglutination reaction which, even on initial detection, was noted to be 'mixed field' agglutination, led to the investigation which is reported here.

In the 67 year old patient presented, two phenotypically distinct red cell populations were demonstrated by differential agglutination and sedimentation. These results were confirmed by flow cytometry using a Coulter EPICS Profiler. An attempt was made by HLA typing to show separate lymphocyte populations but no unexpected extra antigens were found.

Although artificial chimerism after transfusion and bone marrow transplantation has increased the apparent incidence of the phenomenon, natural chimeras are still relatively rare; or are they . . . . ?

General discussion will address this question.

CELL SEPARATORS: AN EVALUATION OF CURRENT NEW ZEALAND PRACTICE. IH3

EA Culverwell, A Inder, MEJ Beard
Department of Haematology, Christchurch Hospital, Christchurch, New Zealand.

We sent a questionnaire to all Haematologists and Immunohaematologists working in Hospitals with cell separators to evaluate the current usage of this equipment in New Zealand. All the Centres replied and this presentation analyses the results.

The machines in use are Fenwall Autopheresis C200 (7), Haemonetics 30 (3), Haemonetics V50 (3), Haemonetics V50+ (1), PCS (3), CS3000 (1).

Between January 1990 and January 1991 5,690 donor plasmapheresis procedures far outnumbered therapeutic plasmapheresis at 412 and plateletpheresis at 46. 28 out of 36 operators were registered nurses, some with up to 10 years experience in the use of cell separators. Data with regard to apheresis protocols, who takes the decision to undertake plasmapheresis and plateletpheresis, the cannulation of patient and donor for the procedure, and the current consent forms being used will be presented. As therapeutic apheresis is an expensive and time consuming technique a costing analysis will also be presented. If the Centres who have contributed to this analysis agree the information obtained will be included in the Australia and New Zealand Apheresis Association Machine Register.

IH4

EXPERIENCE WITH DIRECT FLOW CYTOMETRIC MEASUREMENT OF PLATELET ASSOCIATED IMMUNOGLOBULIN.

Robert Carter and **Helen Hanlin**
Department of Haematology, St Vincent's Hospital, Fitzroy, 3065, Australia.

Many methods have been published for the detection of Platelet Associated Immunoglobulin (PAIg), including some based on Flow Cytometry. We have developed a simple direct immunofluoresent technique for the detection of PAIg based on the Flow Cytometric method of Adult (Pathol Immunopath Res 1988,7,395) using two colour fluorescence. Platelets are reacted with FITC — conjugated antibodies to IgA, IgG, IgM and total immunoglobulin, and simultaneously with a Phycoerytherin labelled platelet specific marker.

Findings will be presented on normal donors and patients with non-immune thrombocytopenia, together with the behaviour of the PAIg in the presence of interferring substances such as Intravenous gamma globulin. The results indicate that the PAIg is a specific, sensitive test for platelet associated immunoglobulin. It requires only small volumes of blood, even when platelet counts are as low as $10x\ 10^9/L$. Positive PAIg results correlate well with clinical indications of Immune Thrombocytopenic Purpura.

QCING HEMOCUES. IH5

LJ Rimmer, RY Harding
Auckland Regional Blood Centre, C/- Auckland Hospital, Park Road, Auckland, New Zealand.

The Auckand Regional Blood Centre began using the Hemocue Blood Haemoglobin Meter in 1991 to measure donor haemoglobins.

This method uses disposable microcuvettes containing a powder that converts haemoglobin to azide methaemoglobin and this is read in a photometer. There was an increase in the numbers of donors rejected for low haemoglobins and this promoted the institution of a daily quality control for a trial period of six weeks to monitor the accuracy of the method.

Problem areas identified were calibration cuvettes, scratched cuvettes and poor technique. The method was found to be accurate and reproducible.

RESULTS — FIRST 15 DAYS

Day	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
% CV	3.8	4.0	3.1	3.9	1.7	1.9	1.9	1.4	2.2	2.8	1.4	2.4	1.9	1.7	1.4
% Concordance	41	45	37	70	80	94	81	94	76	88	95	95	100	82	94

THE USE OF ACID TREATMENT TO ELIMINATE HLA CLASS I ANTIGENS FROM NEUTROPHILS. IH6

RA Dunstan and GB Tan
School of Biomedical Sciences, Curtin University of Technology, GPO Box U1987, Perth, Western Australia 6001.

Kurata et al., (1989) described a new method for eliminating HLA Class I antigens from the surface of platelets. The reduction in antigenicity by acid treatment at pH 3.0 was quite marked. We have compared this technique to the currently popular chloroquine technique for neutrophil serology using flow cytometry. Patients sera which contained multispecific HLA antibodies reacted less strongly with acid-treated platelets than with untreated platelets. No changes were observed in the antigenicity of NA1 and NA2. The viability of acid-treated platelets is greater than that of chloroquine-treated platelets. The method appears to be superior to chloroquine treatment for the analysis of the specificity of anti-neutrophil antibodies.

Kurata et al., Vox Sang, 1989, 57, 199-204.

THE ESTIMATION OF FERRITIN LEVELS IN BRISBANE BLOOD DONORS. IH7

EB MacDonald, J Ullman, J Scott, J Collier and D Long
Queensland Red Cross Blood Transfusion Service, 480 Queen Street, Brisbane, Qld 4000, Australia.

Both the IMX (Abbott) and Stratus (Baxter) systems were used to determine the serum ferritin of Brisbane blood donors.

864 samples were tested by both machines and the data compared using a linear regression analysis. The analysis yielded co-efficient of determination (r^2) of 0.985, a slope of 0.984 and a constant of 1.96. This indicated that the Stratus read an average 2 ng/ml higher than the IMX but otherwise there was no significant difference in the results generated by either machine.

The precision and batch to batch variation of each machine was estimated.

	Coefficient of Variation	Batch/Batch Variation
STRATUS	4.5 — 8.8	3.3 — 5.1
IMX	3.5 — 6.8	2.8 — 4.2

These levels were comparable to the manufacturer's quoted ranges. Normal ranges: Ferritin levels of 780 female and 827 male donors were assayed using the Stratus system.

	Median Ferritin (ng/ml)	10th — 90th Percentile
Female new donors	62.1	20.0 — 156.5
Female 4th time donors	24.4	7.8 — 44.6
Male new donors	111.0	41.3 — 291.0
Male 4th time donors	95.0	36.0 — 254.0

The normal ranges obtained depended on the number of donations given but tended to level out after the fourth donation.

ANALYSIS OF POLYNESIAN LEWIS GLYCOLIPIDS. NEW EVIDENCE FOR THE *Se*^{W} GENE. IH8

Stephen Henry
Department of Transfusion Medicine, Auckland Regional Blood Centre, Auckland, New Zealand.

Monoclonal antibodies and thin layer chromatography have been used to show that the unusual erythrocyte Lewis phenotypes found in healthy Polynesians have a quantitative and qualitative basis. Glycolipid fractions prepared from the plasma and erythrocytes of selected Polynesian samples of the Le(a-b-), Le(a-b-) and Le(a+b+) phenotypes were found to have Le^{b} glycolipids. It is shown that the Le^{b} antigen in some individuals is so weakly expressed that it is undetectable by routine erythrocyte

phenotyping. Crypticity of the Le^b antigen appears to also contribute to the unusual phenotpyes found. The inability of most reagents to detect the Le^b epitope by routine methods is partially explained in terms of the presence of a weak secretor gene, Se^w, although this gene does not account for all the pecularities. Also found in some Polynesian samples were unusually large glycolipids bearing the Le^b epitope. Although the contribution of these novel glycolipids to phenotyping is unclear, their biological significance raises some intriguing questions.

BLOOD TRANSFUSION IN AUTOIMMUNE HAEMOLYTIC ANEMIA. IH9

Lawrence D. Petz, M.D.
University of California, Los Angeles, California, U.S.A.

Indications for transfusion in patients with autoimmune haemolytic anemia (AIHA) must be carefully considered because of unique risks. The autoantibody often complicates the compatibility test and may make it difficult to detect co-existing alloantibodies thereby increasing the risk of an alloantibody-induced haemolytic transfusion reaction. Secondly, the autoantibody itself may cause marked shortening of the survival of donor red cells.

The selection of donor units frequently requires the use of special serologic procedures for red cell typing and especially for alloantibody detection. In warm antibody AIHA, the warm autoabsorption and homologous absorption procedures are the methods of choice for detecting alloantibodies in the presence of autoantibodies. Clinically important autoantibody specificity may be determined using a small panel of phenotyped RBC. In the cold agglutinin syndrome, compatibility testing strictly at 37°C without using potentiating media usually suffices. An "in vivo compatibility test" has limited value in selection of donor units. The volume of blood transfused should be minimised so that one avoids increasing the amount of haemolysis due to an increased RBC mass.

PATHOPHYSIOLOGY AND LABORATORY DIAGNOSIS OF ACQUIRED HAEMOLYTIC ANEMIAS. IH10

Lawrence D. Petz, M.D.
University of California, Los Angeles, California, U.S.A.

Red blood cells can be destroyed by activation of the complement system leading to intravascular haemolysis, by cellular mechanisms leading to extravascular haemolysis, or by a combination of these mechanisms. There are two pathways by which the complement system may be activated, the classical and alternative pathways. Some antibodies and most immune complexes are capable of activating the classical complement cascade which leads to formation of a transmembrane channel resulting in disruption of the cell. Fundamental to cell-mediated destruction of RBC is immune adherence, the process by which the phagocytic or destructive cell is attached to the target cell. Consequences of immune adherence include phagocytosis and antibody-dependent cellular cytotoxicity.

Warm antibody AIHA, cold agglutinin syndrome and paroxysmal cold haemoglobinuria are distinguished by use of monospecific antiglobulin reagents and characterisation of the responsible antibody. Diagnosis of drug-induced immune haemolytic anemias involves recognition of "drug-dependent antibodies" and/or drug-induced autoantibodies and may require the use of drug metabolites.

CONCEPTUAL APPROACHES TO THE MANAGEMENT OF IMMUNE CYTOPENIAS. IH11

Lawrence D. Petz, M.D.
University of California, Los Angeles, California, U.S.A.

The two principle modes of therapy in immune cytopenias involve decreasing the amount of antibody available for reaction with the target cells or decreasing the rate of destruction of cells effected by the antibody. Several methods of doing each of these are available and successful therapy often results from the application of more than one such method. A decrease in antibody production can be effected either by decreasing the number of antibody-producing cells or by altering the control mechanisms which regulate antibody production. Corticosteroid drugs reduce IgG antibody production but have less effect on IgM production. Immunosuppressive chemotherapy may be effective but is associated with a tendency to increase the incidence of leukemia after chronic administration. Removal of antibody by plasmapheresis is only effective if antibody production does not immediately replace antibody that is removed. Methods for the diminution of destructive processes include splenectomy and down-regulation of immunoprotein receptor function. The latter may be accomplished by modification by drugs, modification of the receptor by alternative occupancy or immunologic down-regulation.

IMMUNE HAEMOLYSIS ASSOCIATED WITH TRANSPLANTATION. IH12

Lawrence D. Petz, M.D.
University of California, Los Angeles, California, U.S.A.

Immune haemolysis may occur following bone marrow transplantation (BMT) when there is blood group incompatibility between the donor and recipient, especially when it is within the ABO blood group system. With a major ABO blood group mismatch (e.g., group A donor and group O recipient), haemolysis may occur at the time of infusion of the donor marrow product or may occur as the newly engrafted marrow produces RBC of the incompatible type.

Haemolysis following minor ABO mismatched transplants (e.g., group O donor and group A recipient is particularly significant and occurs as a result of production of antibody by lymphocytes in the donor marrow ("passenger lymphocyte syndrome"), usually in patients receiving cyclosporine for post-BMT prophylaxis of graft-versus-host disease. Haemolysis typically begins near the end of the first week following BMT, is usually acute in onset, and may be severe with haemoglobinemia, haemoglobinuria and a marked fall in haematocrit. Haemolysis has been particularly severe in patients transplanted with marrow from unrelated donors. In addition, the haemolysis of transfused group O RBC has been observed in this setting, apparently as a result of "bystander haemolysis."

IH13

A SIX MONTH EXPERIENCE WITH THE POLYETHYLENE GLYCOL INDIRECT ANTIGLOBULIN TECHNIQUE.

AM Dent
Auckland Regional Blood Centre, C/- Auckland Hospital, Park Road, Auckland, New Zealand.

Polyethylene Glycol (PEG) is a potentiator which when used with the Indirect Coombs Test (ICT) results in a test which is both quick and very sensitive. The Polyethylene Glycol modification of the Indirect Coombs Test has been in routine use at the Auckland Regional Blood Centre since March 1991.

The PEG ICT was introduced in view of its increased sensitivity for antibody detection by comparison with conventional antiglobulin techniques. It also provides us with a single ICT which is suitable for both urgent and routine testing, due to the short incubation time. An additional advantage was the ease with which this new technique could be introduced to all staff.

The PEG ICT has proved itself as a sensitive technique for our routine bloodbank procedures. However, some problems associated mainly with patients with elevated serum proteins have been encountered.

References:
B Wenz, J Apuzzo: Transfusion 1989; Vol 29, No 2:218-20.
B Wenz, J. Apuzzo, DP Shah: Transfusion 1990; Vol 30 , 344-57.
AJM de Man, MAM Overbeeke: Vox Sang 1990; 58:207-210.

IH14

CHIMERA — A TWIN DILEMMA PART II.

LB Pinder
Department of Transfusion Medicine, Auckland Regional Blood Centre, New Zealand.

An ABO grouping anomaly in a 17 year old female in whom a mixed field agglutination pattern was present, revealed the presence of two separate populations of cells. These could be distinguished from each other in four genetic systems, namely the ABO, MNSs, Kidd and Phosphoglucomutase (PGM) types.

Analysis revealed that only 10% of the propositus' red cells were genetically her own, the remainder being derived from her twin brother. We were not able to demonstrate that red cells of the propositus were present in her twin brother.

Cytogenetic studies revealed that approximately 5% of the lymphocytes of the propositus were XX (female) and the remainder XY (male). A skin biopsy was performed and all cells were genetically XX. The lymphocytes of her brother were all XY. The HLA types of the twins appeared to be identical.

IH15

AUTO ID IN BLOOD MANAGEMENT SYSTEM.

WJ Wilson
Principal Technologist, Auckland Regional Blood Service, C/- Auckland Hospital, Park Road, Grafton, Auckland, New Zealand.

The use of electronic data processing systems to manage processes involving step-by-step methodology has proven to be an ideal solution to controlling critical procedures such as are performed in Medical Laboratories. Similarly, barcoded information has firmly established itself as an extremely reliable means of transferring data into an electronic data processing environment and can virtually eliminate transcription or identity errors.

This paper describes how barcoded identifiers are being used in a comprehensive computer controlled system to co-ordinate and manage the total functions of a Blood Transfusion Service. The barcodes provide the interface between the physical and electronic environment so that each procedure or process is fully controlled and monitored. This has resulted in an almost error-free system, producing safer blood products for transfusion.

IH16

THE SIGNIFICANCE OF IMMUNOGLOBULIN ON THE PLATELET SURFACE.

S. Gibbons
Director of Transfusion Medicine, Christchurch Hospital, Private Bag, Christchurch

The majority of platelet associated immunoglobulin (PAIg) is found in the alpha granules. Less than 1% of platelet associated IgG (PAIgG) is found on the platelet surface. An increase in surface and total PAIgG may occur with immune and apparent nonimmune causes of thrombocytopenia. Thus in autoimmune thrombocytopenia the measurement of surface PAIgG in isolation is of little diagnostic value.

In specific clinical circumstances for example drug induced immune thrombocytopenia, alloimmune neonatal thrombocytopenia, post transfusion purpura and platelet refractoriness to random donor platelets the ability to detect surface PAIgG or other PAIg is of value.

The difficulties of platelet serology and the significance of surface PAIgG in determining platelet survival will be discussed.

IH17

AUTO I D — THE POTENTIAL.

Gavin Hodder
Managing Director, SAITO New Zealand, Bath Street, Parnell, Auckland.

The use of barcoding and electronic systems goes beyond tracking in laboratories and into the whole patient management process.

This paper will take an overview of the application of Barcoding in the medical arena, providing some solution to effective resource management using real time data capture. An overview of the "components" that comprise a barcode system will be provided along with implementation guidelines.

THE FIRST AUSTRALIAN CASE OF AN UNPUBLISHED MILTENBERGER CLASS. IH30

PM Bradley*, DS Ford*, DA Stern*, J Poole#, JA Condon+.
*National Blood Group Reference Laboratory, CSL, Parkville, Australia. #International Blood Group Reference Laboratory, Bristol, UK. +Red Cross Blood Bank, South Melbourne, Australia.

The serum of a patient (AS) was found to react with the red cells of one donor (CH) but not with the cells from several panels. Further investigations revealed that AS serum contained antibodies to determinants within the Miltenberger (Mi) subsystem of the MNS system, namely anti-Vw and anti-Mur.Hut. The Mur.Hut specificity appeared monospecific as absorption with MiII or MiIII or CH cells removed reactivity against all three cell types. The cells of CH mimicked those of the MiIII phenotype being positive for Mur and Hil and negative for Vw, Hut, Hop and Nob. However unlike MiIII they also possessed a further "new" Miltenberger related determinant. This new determinant was identifiable by several Miltenberger related antisera including Anek. This phenotype had previously been detected on the cells of an individual of German origin (unpublished) and will extend the Miltenberger classes to at least 10.

Immunoblotting of CH red cell membranes was carried out with monoclonal antibodies to Glycophorins A and B (R18 — Glycophorin A and R1.3 — Glycophorins A and B). Two abnormal components (M, 38Kd and 58Kd) were detected with R1.3 but none were detected with R18. These results showed that CH red cells are indistinguishable from MiIV and MiVI by immunoblotting.

A COMPARISON OF COMMERCIAL MONOCLONAL ANTI-D REAGENTS. IH31

PM Bradley, DA Stern
National Blood Group Reference Laboratory, Commonwealth Serum Laboratories, Parkville, Victoria 3052, Australia.

Until recently anti-Rh(D) blood grouping reagents have been produced in Australia by blending sera from hyperimmune donors. Since the advent of monoclonal antibody technology, alternative antibody sources have become available. The earliest mention of monoclonal anti-Rh(D) was in 1980 [1,2], and thus an alternative to constant boosting of donors, with its inherent dangers had been found.

By its very nature, a monoclonal antibody reacts with a single epitope on an antigen. Thus it might be expected that different monoclonal anti-Rh(D) reagents may give varied results with cells of differing Rh(D) antigen expression. Manufacturers may seek to overcome this by blending. The blend may be of a single immunoglobulin class, a mix of classes or of polyclonal and monoclonal antibodies.

Over a period of eighteen months, twenty-five reagents have been investigated. Ten basic patterns of reactivity were found. These ten groups could be further divided on the basis of their composition: a) IgM only. b) Igm and IgG (including reagents with a polyclonal content and c) IgG only.

Some IgM reagents could detect $R_1{}^{U}r$ phenotype yet failed to react with $R_2{}^{U}r$ cells which might be expected to have a higher number of D antigen sites.[3].

References

1 Boyleston AW, Gardner B, Anderson RL, Hughes-Jones NC. *Scand J Immunol* 1980; 11:55-8.
2 Koskimies S. *Scand J Immunol* 1980; 11: 73-7.
3 Issitt PD. *Applied Blood Group serology* (3rd ed.). Miami: Montgomery Scientific, 1985.

AUSTRALASIAN HEPATITIS MARKER TESTING QUALITY CONTROL PROGRAMME. IH32

Andrew R Thakurdas[1] and Lorraine Rimmer[2]
Telarc New Zealand, Private Bag, Remuera, Auckland 5[1], and Auckland Blood Centre, Park Road, Auckland 1, New Zealand.[2].

This external quality control programme is designed for diagnostic and blood bank laboratories involved in testing for serological markers of viral hepatitis. The programme has been operated by Telarc New Zealand in conjunction with the Auckland Regional Blood Centre since 1986. There are now about seventy New Zealand and Australian laboratories involved in the programme.

Laboratories are invited to test for whichever markers they usually perform from the following:

*	HBsAg	Hepatitis B Virus surface antigen
*	Anti-Hbs	Antibody to Hepatitis B Virus surface antigen
*	Anti-HBc	Antibody to Hepatitis B Virus core antigen
*	HBeAg	e antigen; a component of the Hepatitis B virus core
*	Anti-HBe	Antibody to Hepatitis B Virus e antigen
*	Anti-HCV	Antibody to Hepatitis C Virus

COAGULATION TESTING QUALITY CONTROL PROGRAMME. IH33

Andrew R Thakurdas[1] and Lorraine Rimmer[2]
Telarc New Zealand, Private Bag, Remuera, Auckland 5[1], and Auckland Blood Centre, Park Road, Auckland 1, New Zealand.[2].

This external quality control programme is designed for diagnostic laboratories involved in coagulation testing. The programme has been operated by Telarc New Zealand in conjunction with the Auckland Regional Blood Centre since 1986. There are now about thirty-five New Zealand laboratories involved in the programme.

Laboratories are invited to carry out any of the following tests:

* Prothrombin Time (PT)
* Activated Partial Thromboplastin Time (APTT)
* Factor I Assay (Fibrinogen)
* D - Dimer (FDP)
* Factor VIII Assay (VIII:C)
* Factor IX Assay
* von Willebrand Factor Antigen Assay (vWF:Ag)

IH34

AN ASSESSMENT OF MONOCLONAL ANTI-A, ANTI-B AND ANTI-A, B/A+B AS REAGENTS FOR ROUTINE USE.

Ball S, Watt J. Pepper R.
Immunohaematology Department, Red Cross Blood Transfusion Service, 153 Clarence Street, Sydney, 2000, Australia.

A selected range of monoclonal Anti-A, Anti-B and Anti-A,B/A+B from different manufacturers were tested against normal donors and a referred patient population. Cells known to have weakened expressions of the 'A' and 'B' antigens were also included. Routine tube, tile and microplate methods were selected for the assessment.

The results varied with the type of population tested and the grouping method selected.

IH35

EVALUATION OF OLYMPUS PK7100 OVER TWELVE MONTHS.

A Phillips
Queensland Red Cross Blood Transfusion Service, 480 Queen Street, Brisbane, Queensland 4000, Australia.

In July 1990 the Red Cross Blood Transfusion Service, Brisbane took possession of an Olympus PK7100 blood group/viral microplate testing system. At present it is configured to perform ABO and Rhesus typing on up to 240 specimens per hour.

The Olympus system differs from other microplate systems in that the V shaped wells are of an amphitheatre design with terraced walls instead of smooth walls. Thus a positive reaction gives a carpet of red cells across the entire well, whilst a negative reaction settles to a single point in the bottom of the well.

The system is reliable. Some early mechanical problems caused slight disruption but only ten hours down time has been recorded in the past six months. This down time was due to operator error rather than machine malfunction. The PK7100 has performed in excess of 250,000 blood groups in the past twelve months of routine operation with only one ABO grouping result at variance to the previous ABO result.

The Olympus PK7100 has merged well with the blood grouping programme at the Blood Transfusion Service in Brisbane with considerable savings in reagents and operating time as it lives up to its reputation as a truly set and forget device.

IH36

AUTOMATED BLOOD GROUPING SYSTEMS — ONE BUYER'S PERSPECTIVE.

Stewart Dixon, Sheryl Khull, Denise Hoare
Transfusion laboratory, Wellington Regional Blood Service, Wellington Public Hospital, Private Bag, Wellington, New Zealand.

Prior to making the decision to purchase an automated blood grouping system, we tried three in our laboratory:

Inverness Blood Grouping System
Dynatech Micro Bank
Kontron Microgroupamatic

This poster presents a summary of our opinions, based on these experiences.

NZIMLS CONTINUING EDUCATION
SPECIALIST INTEREST GROUP UPDATE

FROM THE IMMUNOLOGY SPECIAL INTEREST GROUP (ISIG)

Convenor: Gillian McLeay
Contact address: Laboratory Training Centre,
Building 18,
Auckland Hospital,
Private Bag,
Auckland,

ISIG CALENDAR 1992

ISIG WAIKATO/BOP SEMINAR, SATURDAY 9 MAY 1992

The Waikato/Bay of Plenty Group is organising a one day seminar on Saturday, 9 May 1992, at the Disabled Living Centre, Palmerston Street, Hamilton. A warm welcome is extended to ISIG members and others who might be interested, from both within and outside the region.

Coffee will be served at 0930, and the Programme will commence at 1000 to give out-of-towners time to get to the venue. The seminar will finish at 1630. Lunch and afternoon tea will be provided.

A charge of $15 per head, which includes refreshments, has been set to cover some of the costs. ISIG will subsidise the rest.

Discussion topics suggested at the time of going to print are:

- Chlamydia Testing
- Hepatitis C
- HIV1 and HIV2
- ELISA Techniques
- Brucella ELISA
- Skin Allergy Testing
- Specimen Collection for DNA testing

It is planned to invite people with the necessary expertise to lead the discussion for each topic.

Immunohaematologists, Microbiologists and Virologists, as well as Immunologists, should find the programme of interest.

Requests for specific subjects to be included or offers of active participation would be appreciated. If folk from outside Hamilton wish to stay overnight and are interested in attending a post-seminar evening function, please make your suggestions known.

All enquiries should be directed to:

Sherryn Cepulis, Immunology Department, Waikato Hospital,HAMILTON. (Telephone 07-839-8637, Fax. 07-839-5759).

WELLINGTON ISIG ANA WORKSHOP, 26 AUGUST 1992

On Wednesday, 26 August (the day before the NZIMLS Conference) the Wellington group will hold an ANA workshop/ discussion with the emphasis being on standardisation of pattern recognition and comparison of various substrates.

It is planned to get as many commercial substrates as is practical and have them ready to read, with various known positives and negatives, for the group to compare and discuss.

Suggested starting time is 0900 with a break about 1300 for lunch. Further details will be published at a later date. The ISIG meeting will be an informal occasion in the afternoon.

All enquiries to

Gerry Campbell, Medlab Wellington, 89 Courtenay Place, WELLINGTON

IMMUNOLOGY FORUMS, NZIMLS CONFERENCE, 27/28 AUGUST 1992 .

It is hoped that the ANA workshop will provide an incentive for ISIG members to attend the NZIMLS Conference and Scientific Meeting, 27/28 August.

The Wellington group is planning an excellent programme for the Immunology Forums, including a very comprehensive symposium on HIV on the Friday morning.

The two papers organised, so far, for the General Immunology Forum on Thursday afternoon are on Chemilluminescent Immunoassays and Rheumatoid Factors.

Gerry Campbell says that two or three short papers from each of the other three regions will fill the rest of the programme and make this meeting a success, so please give him your support.

Regional reps are requested to canvass for papers in their regions.

MAINLAND GET-TOGETHER

The Deep South is saving its energies for organising the NZIMLS Conference next year. Plans are underway already.

However, some time this year, Diane Phillips (the regional rep), intends to hold an informal gathering of folk from the Canterbury/West Coast/Otago/Southland region to talk about matters of common interest, including methodology and what tests are provided by the laboratories in the region.

Anyone interested in participating should contact :

Diane at Medlab South, PO BOX 25-091, CHRISTCHURCH. (Telephone 03-650-624, Fax 03-650-920)

ISIG CALENDAR 1992
ISIG WAIKATO/BOP SEMINAR
SATURDAY 9 MAY 1992

VENUE: Disabled Living Centre
Palmerston Street
HAMILTON

COFFEE: 0930 - 1000

SEMINAR: 1000 - 1630
(includes lunch and afternoon tea breaks)

REFRESHMENTS:
Morning and afternoon tea and lunch will be provided.

PROPOSED TOPICS:

- Chlamydia Testing
- Hepatitis C
- HIV1and HIV2
- ELISA Techniques
- Brucella ELISA
- Skin Allergy Testing
- Specimen Collection for DNA testing

ENQUIRIES/REGISTRATION:
Contact Sherryn Cepulis
c/o Immunology Department,
Waikato Hospital, HAMILTON.
(Telephone 07-839-8637, Fax. 07-839-8759)

WELLINGTON ISIG ANA WORKSHOP
26 AUGUST 1992

TIME: 0900 - 1300
LUNCH 1300 - 1400

(Further details will be published at a later date).

ENQUIRIES: All enquiries to Gerry Campbell,
Medlab Wellington,
89 Courtenay Place,
WELLINGTON

FROM THE HAEMATOLOGY SPECIAL INTEREST GROUP (HSIG)

Otago/Southland

Profile of Steve Wilson HSIG Representative for the Otago Southland Region

Name:	Steve Wilson
D.O.B.	22.3.57
	Married to Kate with two children Anna (4) and Michael (2)
Present Position:	Deputy Charge Technologist Haematology Lab Dunedin Hospital
Interests:	Very committed armchair sportsman, but I do enjoy a regular game of squash.

As Otago/Southland representative for the H.S.I.G. it will be good to see involvement at a regional level specially for the smaller laboratories.

Steve Wilson - Southland/Otago "HISG" representative.

Auckland

The dates for the HSIG meetings in Auckland have been set for 1992 they are —

February 20th	August 20th
March 19th	September 17th
April 23rd	October 15th
May 21st	November 19th
June 11th	December 17th
July 16th	

Any matters for discussion at these meetings should be in the hands of the Secretary or Chairperson at least a week in advance of the meeting.

June Two Day Seminar

This has been set for Thursday 18th and Friday 19th of June at the Earnest and Marion Davies Postgraduate Medical Centre, Auckland Hospital.

The theme of this year's seminar is Exotic Haematology: the strange and wonderful world of haematological problems imported into New Zealand by travellers and immigrant groups. Topics will include haemoglobinopathies, malaria, filaria, case histories etc.

Details and registration forms will be distributed to laboratories in early March. Enquiries re the seminar should be directed to:

Rennie Dix, C/- Department Haematology, Auckland Hospital, Private Bag 92024, Auckland.

From Cindy Lincoln - representative for the Auckland Area.

Thank you for your prompt and informative replies to the circulated survey. The nomination list for possible future examiners/moderators for Certificate and Specialist examinations has been forwarded.

The results of the survey show that most areas of the region wish to:

1. Remain on the HSIG correspondence list
2. Participate in e interlaboratory Blood Film Survey.
3. Are not interested in being involved in a regional Journal Club.

It was felt by most areas that participation in a regional Journal Club was not possible due to time constraints and travelling difficulties. Perhaps a "Recommended Journal" publication would be more appropriate.

Thank you for your suggestions for future continuing education exercises, and all the best for 1992.

Waikato/Bay of Plenty

Syd Shepherd has been promoted to full time Principal Technologist at Hamilton Medlab. Syd is to retain an interest in the Haematology Department as a technical consultant in the areas of Bone Marrows, Abnormal films and Haemoglobinopathies. A new representative for the Waikato Bay of Plenty region is to be appointed.

News from Audrey Grimmer, Central North Island

A one day Education meeting is planned for Saturday the 28th March.The morning seminar session will be on Myelodysplastic Syndromes and the afternoon session will include presentations from Laboratory Staff and discussions with the local Institute representative and possibly a representative from the union.

A journal contents circular is to be started with photocopies of the articles available on request.

We were sorry to hear of the death of Bob Tracey, Charge Technologist of the Haematology Department, Napier Hospital. Jackie Wypych is Acting charge technologist.

SEMINAR

Biochemistry Special Interest Group
in association with
the AACB, NZ Branch

Friday/Saturday 19th/20th June 1992
Auckland Hospital

Topics for the Saturday Seminar

1. 10.00 - 12.00 hours
   Environmental health and safety - the Technologists role

2. 13.00 - 14.30 hours
   Therapeutic drug monitoring today.

3. 15.00 - 16.30 hours
   In Vitro Fertilisation - The practice, the professionals and the personal perspectives.

Friday is to be arranged by the NZ Branch of the AACB.
It will include a Guest speaker for a keynote address and presentation of proffered papers.

Registration fee $30 per day
$50 per 2 days

Morning, afternoon teas and lunches provided.

A final program and registration form will be circulated with the March Newsletter.

MICROBIOLOGY SPECIAL INTEREST GROUP REPORT FEBRUARY 1992.

Programme for 1992.
Seminars/workshops will be held in conjunction with the NZIMLS conference in Wellington on the 27th and 28th August.
A seminar on respiratory tract infections, on Friday 28th August, is nearly finalised. However if anyone would like to contribute to this seminar, contact Shirley Gainsford.

Journal club
Membership fee is $20.00 to receive indexes from about 15 different journals. It then costs 10 cents per page to copy any articles.
Contact Philippa Skellern, Microbiology Dept, MedLab, P.O. Box 4120, Auckland if you are in the top half of the North Island. For the rest of the country contact Pat Maddocks, Virology/Serology Dept, Wellington Hospital.

Specialist Level Study Guide
For those students sitting Specialist level Microbiology .Note that the syllabus has been revised. If interested, contact Shirley Gainsford.

Continuing Education:

SPECIALIST LEVEL MICROBIOLOGY STUDY GUIDE

Offered by Microbiology Special Interest Group to those doing Specialist Level Microbiology 1992.
This will:
- Suggest appropriate textbooks for the topics in the syllabus.
- Provide journal articles and material from textbooks to supplement the above.
- Provide references for additional
- information.

If interested, contact: **Shirley Gainsford**
Valley Diagnostic Laboratory
P O Box 30-044
Lower Hutt. Phone (09) 699-185

FROM THE TRANSFUSION SCIENCE SPECIAL INTEREST GROUP

The TSSIG had its inaugural meeting on 26 November 1991

Current membership is as follows:
David Wilson, Palmerston North - Convenor
Alison Dent, Auckland
Grant Storey, Waikato
Roger Austin, New Plymouth
Sheryl Khull, Wellington - Secretary
Kevin McLoughlin, Christchurch
Les Milligan, Dunedin
Lindsey Browning, Invercargill

We won't have a separate newsletter, but at this stage intend to use the NZIMLS publications (this Journal and perhaps the Newsletter) to keep you all informed. This means that it doesn't cost you anything extra, and anyone who is interested in any topic we are dealing with has an opportunity to find out about it even if Transfusion Science is not their primary field of employment.

We would like to have contributions from all of you for this TSSIG column.
Can you:
write an article?
tell us about an interesting case you have dealt with?
ask readers about a problem you are having?
review a new textbook?
summarise a Journal article which caught your attention?

It would be fun if we could have a logo for this column, a bit like the Pacific Way column. Is anybody talented enough to design something suitable? Maybe with a catchy name, like "The Blood Group"?

If you would like to contribute anything at all to this TSSIG column please send it to:
The Secretary,
TSSIG Red Cell Serology Laboratory
Wellington Regional Blood Service
Wellington Hospital
Private Bag
WELLINGTON

It is your participation that will add the spark of interest to everything that we do.

Programme for 1992:

We have two big events in 1992 - the NICE weekend in April and Conference in August. As well as these, we hope to bring you news, information and education throughout the year via these pages.

Nice Weekend

NICE Weekend 1992 will be held at the THC Wairakei Hotel on the weekend of 25 - 26 April 1992. This is a weekend for ALL people working in Transfusion Science in New Zealand.

Several companies have donated cash or given promises of financial support towards the NICE weekend (Nice companies?). Their names will be prominent in your NICE Weekend programme booklet. This sponsorship will be used to subsidise the cost to individual participants, decreasing registration for this event from the true cost of $250, to a fee of $200 per person. Your registration fee entitles you to the following:
Two nights (Friday 24 and Saturday 25 April) accommodation on a share twin basis
Breakfast Saturday and Sunday mornings
Lunch on Saturday and Sunday
Morning and afternoon teas on Saturday and Sunday
Dinner on Saturday night

The NICE Weekend (the name is derived from National Immunohaematology Continuing Education) is rather unusual in that all registrants are participants - everyone must present either a poster or a very short paper (1 to 5 minutes) on any topic, problem or area of their interest. Each presentation is followed by questions and discussion. This is a very supportive atmosphere for making a presentation since everyone must participate, and this is part of what makes the NICE weekend so successful.

At the inaugural NICE Weekend held in Wairakei in 1990, Abbott Laboratories initiated an annual "Abbott NICE Award". This award is presented to the person who delivers the best (most worthwhile) paper at the NICE Weekend. The recipient of the award receives an all expenses paid trip to the NZIMLS Conference the same year to present an expanded version of their paper there.

Registration application forms were distributed on 25 November with the November National Immunohaematology Proficiency Survey (N.I.P.S) preliminary results. Registrations closed on February 14 although late registrations may be accepted. If you have any queries about the NICE Weekend contact:
David Wilson
Manawatu Regional Blood Centre
Palmerston North Hospital
Private Bag
PALMERSTON NORTH

Conference

The Annual Scientific Meeting of the NZIMLS will be in Wellington in August 1992. We hope to hold a workshop in Transfusion Science in association with this event, as well, of course, as lots of fascinating papers from people like you. If you have something you wish to share at conference, please

get in touch with either the secretary of TSSIG, or the coordinator of the Transfusion Science conference programme:

Stewart Dixon
Wellington Regional Blood Service
Wellington Hospital
Private Bag
WELLINGTON

Planning is still at the embryonic stage, but we will keep you informed.

Annual Review of the Certificate Level Syllabus and Log Book

The Medical Laboratory Technologists' Board (M.L.T.B.) asked TSSIG to assist in the annual review of the Certificate level syllabus but requested that alterations be kept to a minimum since the Certificate level exam will soon be replaced by the degree and diploma courses. TSSIG recommended a small number of minor alterations to the syllabus for 1992.

The Log Book has inadequacies, but again is not likely to be in use for many more years. No funds have been allocated by the Department of Health specifically for updating the log book so it will remain unchanged for 1992. If you have specific queries about how to use the log book, feel free to contact any of the TSSIG members or the Charge Technologist of your Regional Blood Centre.

Continuing Education

TSSIG is investigating a couple of American programmes in Transfusion Science Continuing Education: "Audio Updates", which is a series of lectures on Blood Banking topics by expert speakers recorded on audio tapes; and "S.T.A.R." (Self Test And Review), which is a quiz designed for testing your own knowledge. We will keep you informed of our progress with these and any other continuing education programmes which become available. We believe that continuing education is a very important aspect of your working life, and that TSSIG has a role to play in helping you keep your knowledge and expertise up to date. If you have requests, suggestions, or offers of help, please contact any of the TSSIG members.

Abbott Laboratories Travel Fund

In December 1991 Abbott Laboratories established a travel fund of $ 5000 per annum for Transfusion Medicine staff to attend conferences and seminars etc. These funds are available to anyone involved in Transfusion Medicine and are intended for education in infectious diseases serology.

After considerable discussion it was decided that these funds should be administered by the Medical Laboratory Science Trust. Applications for these funds will close at the beginning of April each year. Your Charge Technologist will have received a letter outlining how access to this fund can be gained.

Report from Transfusion Advisory Committee (TAC) Meeting Held 28 - 29 November 1991

Compiled by Dr Humphrey Pullon
Regional Transfusion Director
Manawatu Regional Blood Centre

1. Blood Donor Registration Form

Again discussion took place on the format of the Blood Donor Registration Form. It was decided that when the form is next revised, a new question should be included as follows:

"Have you been overseas?" This will be specifically designed not only to alert Donor Attendants to those donors who have been in endemic HTLV-1 areas, but also alert Donor Attendants to persons who have been in high risk HIV areas such as San Francisco, New York or Africa. More specific guidelines will be issued when the new forms including this question come into use.

2. Malaria Policy

This issue was again raised. The guidelines are such that. if a person has visited a malarial zone and has been taking anti-malarial drugs, then their donation should be used for "plasma only" for three years from the time of their leaving the malarial zone. If however they visited a malarial zone and did not take anti-malarial drugs, their donation should be used for "plasma only" for the first six months after their return from the malarial zone. Following this time their donation can be used as a "normal donation". The guidelines regarding brief stop-overs in metropolitan areas that are within malarial areas were also discussed. If a donor stops over for less than 72 hours, no special precautions are required. If however they stop-over for longer than 72 hours, the guidelines as above apply.

3. HTLV-1 Policy

The TAC feels that the present guidelines should continue to apply, accepting that the guideline of a greater than one month residency in an endemic area does have some limitations. Nonetheless, the current guideline is that all potential donors who have resided in an endemic HTLV-1 zone for greater than one month should undergo an HTLV-1 test. If their antibody test is negative their donation can be used normally. If however they are found to be HTLV-1 antibody positive, then their donation should not be used and the Regional Transfusion Director should be notified immediately. As yet no HTLV-1 positive donors have been detected in either Australia or New Zealand despite this "target" testing. TAC felt that there was no indication to widen the spectrum of HTLV-1 testing, given the low prevalence of antibody positivity amongst our New Zealand donor population.

4. Hepatitis Policy

The current guideline is that any donor who has had contact with a person with hepatitis in the last six months should be deferred. If however that contact took place beyond six months then they can he accepted as a normal donor without any further testing required.

If the donor has had hepatitis then a one year deferral applies. When the donor presents following this one year period, full hepatitis testing is required. This includes HBsAg, anti-HBc, anti-HBs, a HCV antibody test and an ALT. If any of these tests reflect a viral carrier state or deranged liver function tests, then the donor should not be accepted. If you are in doubt as to how to interpret the results of these tests, please discuss them with your local pathologist before accepting the donor.

Note that this extra testing requirement applies only to donors who have clinical hepatitis themselves - not to contacts of hepatitis.

5. New Pamphlets

The AIDS pamphlet which every donor must read prior to signing the registration form has recently been revised by the Otago Blood Transfusion Service. The wording of the pamphlet remains the same, however the format and the colour layout has been altered such that donors will probably not recognise it as the old pamphlet. If you require copies of this pamphlet please contact the Donor Manager at your Regional Blood Centre who will arrange for supplies to be forwarded from Dunedin.

The Canterbury Transfusion Services are also revising a number of pamphlets designed for blood donors, in particular the pamphlet "What Happens to Your Blood" is being reworded and reprinted. The pamphlet "What Health Checks Are Done On My Blood" is also being reprinted. A new pamphlet entitled "Make a Heart Throb" is also being printed by the Canterbury region and will be available in early 1992. If you require supplies of these pamphlets, contact the Donor Manager at your Regional Blood Centre.

6. Donor Recruitment

A number of innovative strategies continue to attract good numbers of donors. In particular, sending out advertising notices with power bills has been used in the Waikato region to good effect. Palmerston North has recently seen an advertising campaign using the hospital payslips to attract donors. The Auckland region has explored putting blood donor advertisements in with K-Mart and Foodtown circulars. All donor attendants are encouraged to explore local initiatives to attract donors.

The TSSIG would be interested to hear of any that you have tried and found successful.

7. Iron Tablets

TAC currently recommends that ten days supply of iron tablets be given to all female donors of childbearing age following their donation. Iron deficiency is more common amongst this female population and therefore iron supplements should be targeted appropriately. Recent studies of ferritin levels in blood donors are interesting and show that depleted iron stores are quite common among our regular donors.

8. Change of Base Label on the Tuta Blood Packs

At present the base label on the standard Tuta pack is labelled "Whole Blood". During blood processing this label is covered with a further label: "Resuspended Red Cells". Since the majority of blood now used in New Zealand is in the form of resuspended red cells and such over labelling requires considerable manpower and is rather tedious, it has been recommended that the base label on the Tuta pack be changed from "Whole Blood" to "Resuspended Red Cells". This will mean that no overlabelling is required during blood processing and red cell resuspension. The Technical Working Party will arrange with Tuta Laboratories for this change to take place and no doubt packs arriving towards the end of 1992 will have the changed label. Circulars will be issued by Tuta Laboratories and by the Charge Technologist of your Regional Blood Centre to alert you to this change nearer the time when it occurs. We must be cautious that no confusion exists about labelling. In particular those centres who are still using small amounts of whole blood will need to acquire over labels so as the units of whole blood are properly labelled. The technical Working Party will arrange for these labels to be printed and ordering information will be made available to you by your Regional Blood Centre.

9. Drug Deferral List

All centres should be using the drug list revised by Professor McQueen in November 1990. It is recognised that this present drug list has considerable limitations and also that it does not include trade names — only generic names. This matter was discussed by TAC and Professor McQueen's retirement from the Otago Blood Transfusion Service was noted. The Auckland Regional Blood Transfusion Service has offered to revise the Drug Deferral List. This will take some time and the new list will probably not be available until late 1992. The new list will contain both generic and trade names.

I0. Hepatitis C Antibody Testing

The Minister of Health approved in principle that funds should be available for hepatitis C antibody testing of blood donations in New Zealand.

The exact amount of money for this activity has not yet been decided and therefore the exact format of testing has yet to be formulated. TAC have recommended that all blood donations be tested on every occasion and that HCV antibody testing - like HIV antibody testing — should be performed by the Regional Centres. It is hoped that Hepatitis C antibody testing will begin in June 1992. There is however some concern that there is no confirmatory test for hepatitis C antibody available in New Zealand at present. The CDC in Porirua are examining this issue and it is hoped that they will soon set up a confirmatory test.

The guidelines for handling HCV antibody positive donors are yet to be formulated. It is known from overseas studies that approximately 50% of blood donors that come up as HCV antibody positive will in fact be false positives and there is, therefore, some reservation about retiring all HCV antibody positive donors. This matter will need to be resolved before HCV antibody testing commences.

The Regional Transfusion Directors would like to be notified of all cases of post transfusion hepatitis C since the frequency of transfusion acquired hepatitis C does add weight to the argument for routine hepatitis C testing of all blood donations.

11. HIV Antibody Testing

Following the recent tender in June 1991, the majority of donor HIV testing laboratories in New Zealand changed to the Abbott HIV-1+2 kit with only Christchurch and New Plymouth using the Wellcome kit. Dr Gibbons commented that the number of repeat positive tests appears to be higher with the Abbott kit and in some kits the repeat positive rate was five to six times that of the comparative Wellcome kit. This is of some concern particularly since repeat positive tests mean an increased number of tests for the same number of blood donations and also necessitates an increased number of Western Blots to be performed. This matter will be discussed with Abbott representatives in the near future, particularly since there may be some concern about ongoing use of the Abbott HIV-1+2 kit.

No further confirmed HIV antibody positive blood donations have been found recently. Indeed the last HIV positive blood donation was detected in Auckland in January 1990. We hope that such encouraging results will continue.

12. The Use of Anti-A, B in Patient Grouping

After discussion with the Technical Working Party, it was felt that the use of anti-A,B in addition to anti- A and anti-B was probably not necessary if one was using monoclonal anti-A and anti-B sera capable of detecting the weak subgroups. Hence the use of Anti-A, B is no longer a mandatory requirement for patient grouping when monoclonal anti-A and anti-B capable of detecting weak subgroups are used. If you are uncertain about this guideline please do not hesitate to contact the charge technologist of your Regional Blood Centre.

13. Inventory of Rare Red Cell Types Stored in Liquid Nitrogen

As you are aware, the Auckland Regional Blood Centre has a liquid nitrogen storage facility for the storage of rare cell types so that patients with rare or complex antibodies can be readily transfused. It was recognised by some TAC members that a record of the exact red cell types currently stored by the Auckland Regional Blood Centre was not readily available. Dr Woodfield has therefore recently circulated an inventory of all rare red cell units that are currently in liquid nitrogen storage in Auckland. A copy of this inventory is available from your Regional Blood Centre. If you have a patient with a rare blood group antibody who requires transfusion, do not hesitate to contact your Regional Blood Centre who will tell you if Auckland are holding frozen cells appropriate to your patient's needs.

14. Policy for Post Anti-D Immunoglobulin Testing in Rh(D) Negative Mothers

Following the administration of anti-D immunoglobulin to Rh(D) negative mothers, many laboratories are performing a 48 hour antibody screen to ascertain if circulating anti-D is still present. It is clear from recently published literature that such testing is inappropriate and that the administration of further anti-D immunoglobulin on the basis of a negative anti-D screen Is pointless. It would seem that if any testing is appropriate then a Kleihaur test should be performed on the mother's blood immediately following delivery. Such testing

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would detect large feto-maternal bleeds and if the feto-maternal bleed is large then further anti-D immunoglobulin will be required. It is recognised that using Kleihaur tests immediately following delivery would overcome the problems of trying to follow up mothers who are discharged soon after delivery and may also result in savings in anti-D immunoglobulin.

It is suggested that Charge Technologists review their local policy for handling Rh(D) negative mothers following the administration of anti-D immunoglobulin. Should you require further technical guidance, do not hesitate to contact the Charge Technologist of your Regional Blood Centre.

15. NZBTS Standards

Further work continues on the NZBTS Standards. Sections 1 to 8 have now been completed and some work has also been undertaken on other sections. A number of appendices and in particular the large appendix describing technical methods remain to be completed. TAC members felt that the Standards which have been completed so far should be put in a loose leaf format and circulated to all Blood Transfusion Laboratories so that they can come into use as guidelines initially. Dr Faed is hoping to finalise the first 8 sections in the next few months and hopefully they will be issued to Blood Transfusion Laboratories by mid 1992. The delay in the publication of the Standards is appalling and due partly to a lack of financial support from the Dept of Health for their publication and also to secretarial pressures particularly in Dunedin. TAC apologises for the delay but hope that in time the high quality of the standards will make the delay worthwhile.

16. Revisions of the Accident Compensation Corporation Bill

A draft paper describing a number of modifications to the Accident Compensation Bill has been circulated recently and is available from the Government Printers. One area of concern is that there is no Accident Compensation cover to patients who happen to be "injured" following a blood transfusion. If this revised recommendation is adopted, it would mean that blood Transfusion Services and even individual blood donors may become legally liable should medical misadventure occur through blood transfusion. Indeed, this would mean that if a patient developed transfusion acquired HIV infection then he or she could, in fact, sue the particular donor that caused the infection. The Transfusion Advisory Committee are very concerned about this potential scenario and are therefore making submissions to the Parliamentary Select Committee currently considering the ACC Bill. It is hoped that the Bill will be amended so that Blood Transfusion Services and blood donors cannot be liable for medical misadventure occurring through blood transfusion.

17 Reactions to SPPS

Following considerable concern earlier this year, no further hypotensive reactions to SPPS solution have been documented. CSL have done considerable work looking into PKA-C1 esterase inhibitor levels in various batches. It would seem that those batches which caused hypotensive reactions did have higher levels of PKA-C1esterase inhibitor and that this may have caused the problem.

CSL are therefore still recommending that all stocks of SPPS are stored at 4°C. Small satellite stocks of SPPS can be stored at ambient temperature for up to three months.

Of note, CSL are hoping to develop a 5% albumin solution in 1993 which will replace SPPS solution. Hopefully this will obviate the problems of hypotensive reactions previously described.

18. Anti D Immunoglobulin

All centres should be aware that there is an ongoing national shortage of anti-D immunoglobulin and that over recent months supplies from Australia have been used in order to keep pace with the demand for this product. There has been a drive to recruit ladies with moderately high anti-D titres and to collect their plasma wherever possible. I would emphasise to all laboratories to ensure that anti-D immunoglobulin is being used appropriately and that no wastage is occurring.

19. Intravenous Immunoglobulin

Adequate supplies of this product are currently available although its usage is gradually increasing. There is currently a 200mL bottle available which is useful for those patients receiving high doses (immunosuppressive doses) of immunoglobulin. Apparently CSL are also considering manufacturing a 500ml bottle and this may become available in late 1992.

20. Factor VIII Concentrate

The current TAC policy is that Factor VIII concentrate will be allocated nationally on the basis of Factor VIII plasma input. The increasing usage of Factor VIII nationally is of some concern. TAC are writing to all "haemophilia treaters" and asking what their planned Factor VIII usage for the 1992/1993 year will be. There is also some concern that young severe haemophiliacs are being treated on a "prophylactic" basis which again may increase Factor VIII consumption. Once the Factor VIII requirements for the 1992/1993 year have been finalised, a review of national bleeding rates will be required in order to ensure that the collection of Factor VIII plasma is adequate.

21 Reactions to CSL fractionated products

TAC have asked that they be made aware of any patient who has a reaction, be this allergic or otherwise, to any CSL fractionated product. TAC are conscious that, in light of the recent SPPS reactions, they do not have any national system for collecting data about reactions to fractionated products. TAC therefore asks that laboratories forward information about patients who have such reactions to their Regional Transfusion Director. This information will then be sent to Dr Yvonne Harding at the Auckland Regional Blood Centre who will collect it nationally. This would mean that any particular batches of CSL products causing reactions will become evident fairly promptly and appropriate action can be taken to ameliorate the problems.

22. Contract with CSL

In the current environment of rising costs and increasing accountability, it is felt that a formal contract between the New Zealand Department of Health and Commonwealth Serum Laboratories would be desirable. Such a contract would define where liability exists should product failure occur. Hence, if say a batch of SPPS was unacceptable and caused a high rate of reactions, then the cost of this batch which would have to be destroyed would be met by the appropriate party as defined by the liability within the contract. The blood Transfusion Service would, of course, be liable for the quality of the collected plasma. This would mean that if a batch of plasma was contaminated by, for example, hepatitis B, then the total liability for such a contaminated batch would lie with the Blood Transfusion Service and not with CSL. It is hoped however that such a contract will result in both the improved quality of products and in the plasma forwarded to CSL alike. The Australian Red Cross Transfusion Service has just recently signed a contract with CSL. It is anticipated that the New Zealand contract will be formulated along similar lines.

23. Bone Marrow Donor Registry

The Blood Transfusion Service has agreed to recruit some blood donors for potential bone marrow donation to facilitate matched unrelated bone marrow transplantation. Dr Fang, Regional Transfusion Director in Wellington, is co-ordinating this registry although TAC is still awaiting appropriate computer hardware and software so as to make the registry a reality. Nonetheless, Bone Marrow Donor Consent Forms are now available from your Regional Blood Centre. If you have particularly dedicated donors who are keen to become bone marrow donors as well, a sample for tissue typing should be taken and sent to your Regional Blood Centre. The appropriate Consent Form should also be completed. At present only class 1 (HLA-A & B) tissue typing is required. If, however, a class 1 match is found with a potential bone marrow recipient then DR typing and mixed lymphocyte cultures must be undertaken.

It is hoped that New Zealand patients will also have access to the Australian Bone Marrow Registry and through the Sydney centre, to international registries. The cost of searching such registries is considerable. Any patient requiring a

matched unrelated donor transplant who wishes to search an international registry must pay A$ 5,000 to the Sydney centre before such a search is initiated.

24 . Bone Bank Operating Procedures

Following concerns raised by the Immunohaematology Department at Nelson Hospital, TAC have agreed to formulate some fairly standard procedures for bone banking. Dr Gibbons, Regional Transfusion Director in Christchurch is formulating these national guidelines and if you are involved in bone banking he would be interested in receiving a copy of your Standard Operating Procedures for this.

A recent guideline has been issued by the Department of Health requiring all patients who have donated bone samples to the bone bank to undergo a "90 day post donation" HIV antibody test and that this test be negative before the bone can be issued from the bone bank. I trust that all laboratories operating a bone bank are aware of this requirement and have altered their operating procedures accordingly.

TECHNICAL COMMUNICATIONS

Du Testing

Sheryl Khull,
Red Cell Serology Laboratory, Wellington Regional Blood Service, Wellington Hospital.

Ever since the Du phenotype was first described in 1946, (1) considerable time, effort and reagent has been used to detect it. We need to consider whether this practice is relevant, cost-effective, and makes a significant contribution to patient care. A study by Garetta et al in 1974 (2) found 0.56% of Caucasian samples to be Du. Ten years later a study by Knight, (3) found a frequency of only 0.23%, less than half that of the earlier report.

The frequency of the Du phenotype is not decreasing. Rather, anti-D typing reagents are becoming more potent. This, coupled with the use of monoclonal reagents which can detect most Du samples in direct agglutination tests, means that many samples which in the past have been called Du, are now grouped simply as D positive. Therefore the use of an antiglobulin test ("Du test") will detect fewer and fewer Du samples.

Is it worth doing?

Latest "Guidelines for Pre-transfusion Testing" (4) published by the Australasian Society of Blood Transfusion specifically state that "the Du test" (for potential blood recipients) "is not required." This policy is readily accepted.

Du testing of blood donors, and the resultant labelling and use of their donated blood is a much more controversial issue. As noted above, Du samples detectable only by the antiglobulin test are uncommon, and becoming even more so as reagents improve. Presumably, any Du bloods identified would be labelled and transfused as Rh Positive. However, the immunogenicity of Du transfused to D negative recipients is much lower than that of the normal D antigen.(5) In one study, (6) 68 D positive units were transfused to 45 D negative recipients, and not one produced anti-D, although one anti-K and one anti-E were stimulated.

What about the recipient who already has anti-D? In this case, a full crossmatch will be performed, and if a Du positive unit happened to be selected for crossmatching it should be incompatible. Even if a low-grade Du positive unit was transfused to a patient with anti-D, serious consequences are unlikely. In fact, Mollison suggests (7) there would probably be no accelerated red cell destruction.

So why should you want to do a test for Du? The phenotype is rare, not very immunogenic, detectable by crossmatch incompatibility, and even if transfused to a patient with pre-formed anti-D, red cell survival will probably be normal. For some years a number of blood transfusion workers (8) have recommended that the practice of special testing for Du be discontinued. Has the Du test outlived any usefulness it may once have had? Should it be relegated to the history books? In these days of potent anti-D reagents, when resources are stretched to their limits, we must ensure that every test we perform contributes significantly to better care of the patient.

References:

1. Stratton F. Nature 1946; 158:25.
2. Garretta M. Muller A and Gener J. "Determination du facteur Du sur Groupamatic. Etude comparee avec les techniques manuelles. Bilan sur 203,240 examens." Rev. Franc. Transfus. 1974; 17: 211
3. R. Knight, (personal communication) In: Blood Transfusion in Clinical Medicine, Mollison P L, Engelfriet C P, Contreras M, Eighth Edition, 1987: 335
4. Australasian Society of Blood Transfusion Incorporated "Guidelines for Pretransfusion testing", April 1988
5. Blood Transfusion in Clinical Medicine, Mollison F L, Engelfriet C P, Contreras M, Eighth Edition, 1987: 336 - 356
6. Schmidt P J, Morrison E G, Shohl J, "The antigenicity of the Rho (Du) blood factor." Blood; 20: 196
7. Blood Transfusion in Clinical Medicine, Mollison P L, Engelfriet C P, Contreras M, Eighth Edition, 1987: 336
8. Contreras M, Knight R C, "Testing for Du: Con". Transfusion, 1991: 31:3
9. Schmidt P J, "Do You Du?" Immunohaematology, 1987; 3:4
10. Walters M S, "More on Du" Immunohaematology, 1988; 4:1
11. Moore B P L, "Does knowledge of Du status serve a useful purpose?" Vox Sang 1984; 46: 95

* * * * * * *

A Detective Story

Act One

A blood sample is received in the laboratory with a request. for Group & Screen and investigation of haemolytic anaemia". Clinical details are: "A.R.F., jaundiced, raised LDH?, previous transfusion reaction". Routine batch group results are: blood group O Rh(D) Positive, antibody screen negative, direct antiglobulin test (DAT) weak microscopic positive. The serum is noticed to be a dark yellow-orange colour.

Act Two

The blood grouping tubes are observed more closely. Mixed field reactions are seen in the anti-A, anti-A,B and anti-D tubes.

Some More Clues

The blood film shows many microspherocytes, some macrocytes, shistocytes and polychromasia. The Schumm's test is positive. Haptoglobins are undetectable. Bilirubin (which had been normal before surgery) is now extremely elevated, with other liver functions essentially normal.

The Evidence

A telephone call reveals that five units of A Rh(D) Negative blood had been transfused to the patient a couple of days previously, prior to her renal failure.

The Solution

The case is fairly typical, and the indications clear:

dark yellow-orange serum
positive DAT but negative antibody screens
mixed field reactions in grouping tests
history of transfusion of group A blood (to a group 0 recipient)
acute renal failure shortly after transfusion.

Epilogue

After some weeks of renal dialysis, the patient made a full recovery.

ABO incompatible transfusions are mercifully rare, but the sooner they are recognised, the better for the patient. So keep your eyes and mind open.

* * * * * * *

A Horror Story

(This is a true story. Only the identifications have been obscured.)

In a medium-sized provincial hospital, Mrs N, a 62 year old lady, was scheduled for surgery for closure of a colostomy. A lab assistant with four years experience went to the ward, found "Mrs N", checked the family name on her wristband, collected samples for crossmatching, and labelled the tubes with full identification details copied from the request form.

Back at the Blood Bank, the sample was carefully checked against the request form, and blood grouped as a normal group A Rh(D) positive. No previous transfusion history for the patient was available to the Blood Bank. Although there was a record of a previous transfusion in the patient's medical records, this had not been recorded on the patient's request form. Since the Blood Bank was a bit short of A Rh(D) Positive units, but had plenty of A Rh(D) Negatives that were about to expire, A Rh(D) Negatives were chosen for crossmatching. The crossmatch was compatible.

Five units of blood were transfused to Mrs N. in the course of two visits to surgery. The patient's condition deteriorated, she went into renal failure, and was transferred to a regional hospital for dialysis.

The horror of this story arose because the "Mrs. N." from whom the A Rh(D) Positive blood sample was taken, is not the same Mrs. N. for whom the requisition was made out and who subsequently received the five units of A Rh(D) Negative blood. The real Mrs. N. is group O Rh(D) Positive. The ABO incompatible blood which she received certainly contributed to her renal failure. Just one moment's inattention on the part of an experienced lab worker during the process of checking identification was nearly fatal for Mrs. N.

Obviously the specimen should have been labelled by copying the identification details from the patient's wrist band. Clerical checking is an exacting, often laborious job, but it really is vital. Please do all your checks carefully — the patient's life may be at stake!

* * * * * * *

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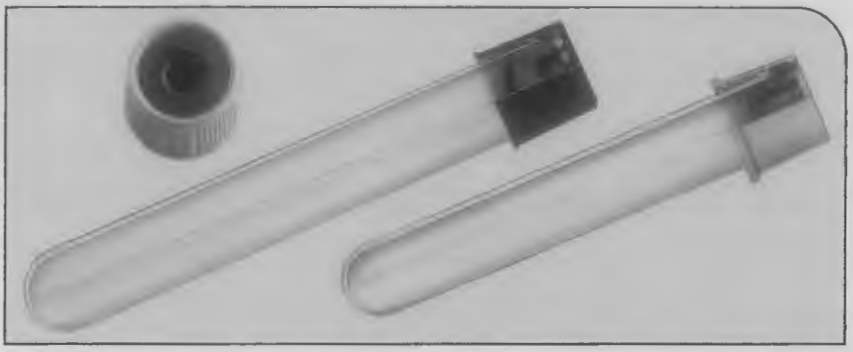
VACUETTE®

Vacuum blood collection system made of plastics



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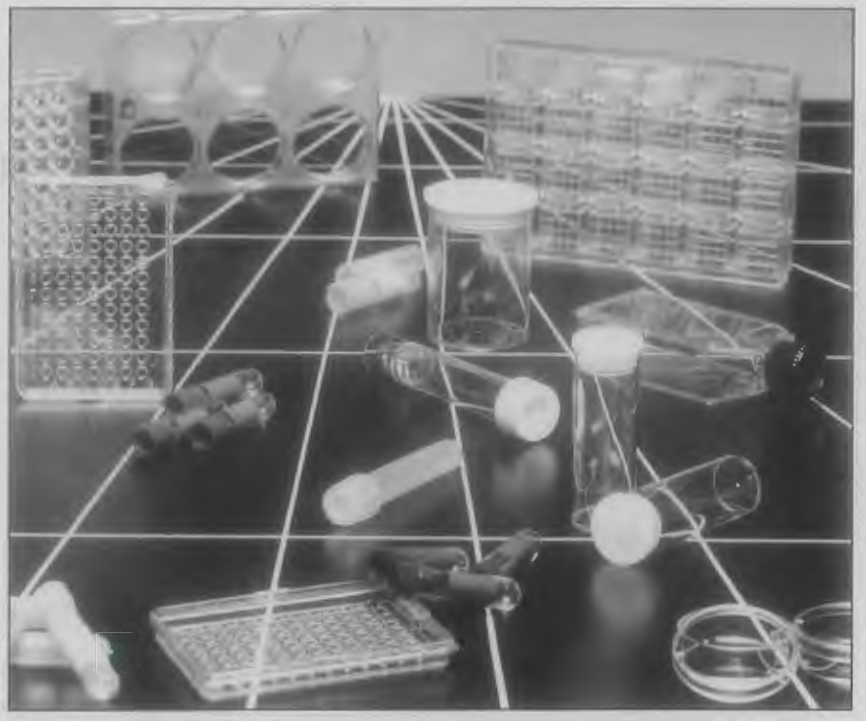
- Laboratory Instruments



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greiner
labortechnik

Medical Laboratory Technologists

Currently employed

	1984	1985	1986	1987	1988	1989	1990	1991
Clinical Biochemistry	174	187	186	187	187	175	208	182
Microbiology	164	168	172	176	186	189	204	183
Haematology	160	160	163	168	176	174	180	162
Immunohaematology	86	90	92	97	102	96	105	101
Histology	22	24	24	24	28	26	29	34
Cytology	6.0	5.2	7.2	5.7	7.8	9.5	22	26.6
Nuclear Medicine	6.2	8.5	8.0	5.8	9.0	7.0	8.4	9.2
Immunology	23	22	28	22	21	30	31	34
Cytogenetics	5.5	7.5	6.5	7.5	8.0	6.4	5.8	12.6
Virology	1.0	2.0	6.0	4.5	6.5	10	12	13.5
Administration (full time)	37	34	39	34	33	33	30	29
On rotation	46	41	55	41	44	40	31	31
Other	4.5	7.3	2.4	3.0	11	7.8	13	8.6
TOTAL	735.2	756.5	789.1	775.5	819.3	803.7	879.2	826.5

Current vacancies

	1984	1985	1986	1987	1988	1989	1990	1991
Clinical Biochemistry	9.0	8.5	15.3	11.5	14.0	15.0	10.6	
Microbiology	1.5	4.0	12.5	10.0	9.6	13.0	5.8	
Haematology	4.5	4.0	11.0	9.8	11.0	11.0	10.6	
Immunohaematology	6.0	4.0	6.5	7.3	6.3	3.0	5.0	
Histology	3.0	5.0	3.0	5.0	5.0	6.0	4.0	
Cytology	-	-	-	2.0	2.0	-	1.0	
Nuclear Medicine	-	-	1.0	1.0	1.0	-	1.0	
Immunology	1.0	-	2.0	2.0	5.0	1.0	1.0	
Cytogenetics	-	-	-	-	-	0.5	1.5	
Virology	-	-	-	1.5	0.5	-	-	
Administration (full time)	-	-	1.0	1.0	-	-	-	
On rotation	1.0	3.8	6.5	3.1	3.6	0.5	4.9	
Other	-	-	-	-	-	-	-	
TOTAL	26.0	29.3	58.2	54.2	58.0	50.0	45.4	

Medical Laboratory Assistants

Currently employed

	1984	1985	1986	1987	1988	1989	1990	1991
Clinical Biochemistry	188	193	183	169	174	177	154	133
Microbiology	165	186	168	152	188	176	185	156
Haematology	142	145	143	117	112	118	120	92
Immunohaematology	101	118	118	114	112	100	98	83
Histology	78	77	85	76	96	76	74	56
Cytology	40	32	36	40	35	56	59	49
Nuclear Medicine	16.0	12.5	16.8	11	13	9	4	3.2
Immunology	41	32	42	31	48	46	42	31
Cytogenetics	5.0	4.0	7.5	5.5	13	3.5	3.5	1.2
Virology	5.6	7.0	7.0	8.0	6.5	5.5	6.5	6.5
Blood collection	87	96	91	91	75	77	71	68
On rotation	56	44	51	56	67	64	28	40
Other	24	31	44	49	49	66	50	47
TOTAL	948.6	977.5	992.3	919.5	988.5	974.0	895	765.9

Current vacancies

	1984	1985	1986	1987	1988	1989	1990	1991
Clinical Biochemistry	5.5	5.5	7.0	11.0	5.3	2.0	8.0	
Microbiology	3.9	4.8	8.4	5.4	1.9	4.0	5.4	
Haematology	1.7	4.3	5.8	4.1	5.6	5.5	4.6	
Immunohaematology	2.1	1.0	2.5	4.6	10.9	5.5	10.3	
Histology	0.5	3.0	2.0	4.5	3.8	7.5	3.8	
Cytology	-	1.0	1.0	1.0	-	2.0	0.7	
Nuclear Medicine	-	1.0	-	-	1.0	1.0	1.0	
Immunology	-	1.0	-	2.4	-	1.0	2.6	
Cytogenetics	-	-	-	-	-	-	-	
Virology	-	-	-	-	2.0	2.0	-	
Blood collection	-	2.7	4.0	3.0	-	4.6	4.0	
On rotation	2.0	2.7	2.7	0.4	1.0	-	5.5	
Other	-	1.0	-	0.5	5.5	1.5	2.3	
TOTAL	15.7	26.3	33.4	36.9	37.0	36.6	48.2	

NZIMLS ANNUAL STAFFING SURVEY

(Annual Staffing Survey contd ...)

Medical Laboratory Trainees

	1991
Medical Laboratory Technologist	177
Science Graduates	18
Trainee vacancies	14
Diploma/NZCS Trainees	
First Year	33
Second Year	42
Third Year	37
Fourth Year	15
University	
First Year	1
Second Year	5
Third Year	–
Fourth Year	3
Certificate Level	
First Year	3
Second Year	4
Third Year	2
Fourth Year	64

COURSE ANNOUNCEMENT

INTRODUCTION TO MOLECULAR GENETICS AND GENE MANIPULATION

A one week non-credit introductory workshop will again be conducted in the Microbiology and Genetics Department of Massey University during the May Holidays 1992 namely 11-15 May. The aim of the course will be to provide for those people who may have a potential professional interest in the subject, a working introduction to the powers and limitations of the techniques. This year we shall endeavour to focus on topics in applied molecular genetics relevant to agriculture and medicine. Lecture material to be covered will include DNA and genome structure, the molecular genetics of plasmids and transposons, basic strategy of recombinant DNA research (both basic and applied) and the quasi-legal aspects of "genetic engineering". Practical work will include plasmid isolation, transformation/electroporation, restriction enzyme mapping DNA ligation, PCR and RFLP analysis. Background assumed will be the equivalent of Introductory Genetics and Introductory Biochemistry (200-level).

Although Boehringer-Mannheim are continuing their generous sponsorship for this course in the form of biological materials, there will be a charge of $400, in order to cover the cost of additional materials and facilities. Accommodation will have to be arranged off campus, as unfortunately, extramural fully books the campus accommodations. The enrolment will be limited to 24 (the capacity of the teaching laboratory).

For further information and an enrolment form, please contact:

Assoc. Prof. Eric Terzaghi
Department of Microbiology and Genetics
Massey University PALMERSTON NORTH

NEW ZEALAND INSTITUTE OF MEDICAL LABORATORY SCIENCE 1992 CALENDAR

12/13 March 1992	Council Meeting - Hamner Springs
30 April 1992	Committee Annual Reports to be with the Executive Officer
30 April 1992	Applications close for QTA examinations
30 April 1992	All accounts to National Treasurer for auditing
21/22 May 1992	Council Meeting
29 May 1992	Applications close for Specialist Certificate examinations
31 May 1992	Proposed rule changes and remits to be with the Executive Officer
27 June 1992	Nomination forms for the election of Officers and Remits to be with the membership (60 days prior to AGM)
I July 1992	Annual Staffing Survey
7/8/9 July 1992	Fellowship examinations
7/8 July 1992	QTA examinations
18 July 1992	Nominations close for election of Officers (40 days prior to AGM)
6 August 1992	Ballot papers to be with the membership (21 days prior to AGM)
13 August 1992	Annual Report and Balance Sheet to be with the membership (14 days prior to AGM)
20 August 1992	Ballot papers and proxies to be with Executive Officer (7 days prior to AGM)
25/26 August 1992	Council Meeting - Wellington
27 August 1992	AGM and SGM - Wellington
27 August 1992	Annual Scientific Meeting - Wellington
II/12 November 1992	Specialist Certificate examinations
12/13 November 1992	Council Meeting

The Pacific Way

PACIFIC PARAMEDICAL TRAINING CENTRE

Extracts from Dr. Ron McKenzie's Annual Report presented at the AGM on the 8th November, 1991

Activities of PPTC

"This year has seen further expansion of the Centre's activities. Two training courses involving 18 trainees were held, the Quality Assurance Programme for the Hospital Laboratories of the Pacific Islands was further developed and the three years technical Training Programme in the laboratory at the National Laboratory, Western Samoa, is at this time nearing the completion of its second year.

The impact on the Centre's activities since its designation as a WHO Collaborating Centre is difficult to measure as the Centre has had a high profile in Pacific Island Laboratory Training Schemes for the past 10 years Nevertheless, it has been noted that Pacific Island Laboratory staff tend to approach the Centre even more readily for help and advice since the Centre was thus designated.

It is also encouraging to observe that the activities undertaken by the Centre are reflected in the considerable improvement made by some of the Pacific Island Laboratories in the delivery of their service. WHO and Centre staff who have worked and travelled in the region during the past few years all report on this improvement. It is hoped that after the Quality Assessment Programme has been in operation for a further two years, that a more quantifiable measure of laboratory improvement can be made. It was pleasing that the trainees who attended the August WHO Regional Course in the laboratory Diagnosis of STD and HIV infection, were able to attend the third South Pacific Congress on Medical Laboratory Science held in Auckland. Two of the guest speakers at the Congress were Monica Cheesbrough and Peter Bruhn, both speakers have wide experience in training in Medical Laboratory Services for Developing Countries and conducted symposia in teaching methods and appropriate technology in which the trainees participated".

P.P.T.C. Trainees who attended WHO. Regional Course in the Laboratory Diagnosis of STD and H.I.V. Infection and also the 3rd South Pacific Congress on Medical Laboratory Sciences.

Back Row: (left to right) Augustine Bani (Vanuatu) Tonga Havili (Tonga), Rickie Eddie (Solomon Islands) Tebebeku Tela (Kiribati) Ron McKenzie (PPTC), Mike Lynch (PPTC), Viliame Tomaui (Fiji)

Front Row: Paul Lolita (Marshall Islands) Tautala Mouala (Western Samoa), Maria Marfel (Yap Micronesia), Vaevae Pare (Cook Islands).

Samoan Technical Training Programme

"This programme continued during 1991 and the second year of the three year course will finish in November, 1991 with the 8 trainees sitting year end examinations Eight trainees sat the Year 1 final examinations in November, 1990, with six gaining a complete pass and two a partial pass. The latter two successfully completed a special examination in January of this year and thus all 8 trainees were able to proceed to the second year of the study.

The Samoan Ministry of Health requested that the subject "Histological Techniques" be added to the training syllabus for 1991. This was agreed and Histology replaced the Laboratory Technology which is a Year 1 component only. The Centre is greatly indebted to Cheryl Goodyear of the Division of Anatomic Pathology for undertaking the task of preparing at short notice a full set of lecture notes on this subject. John Elliot carried out a WHO assignment in April of this year in Western Samoa to review the progress of the Training Programme, to conduct training sessions for the Tutors and to assess the laboratory Quality Control Programmes.

The success of students in the Year 1 final examinations of the Western Samoan Technical Training Project indicates the strengths of this programme and Tutors from the Centre will continue to take teaching assignments in Apia, to maintain the momentum of this. The first qualified technicians will graduate from this programme at the end of 1992"

Collaboration with WHO 1991

"The good working relationship that has existed between WHO and the Training Centre over the past decade was further strengthened in 1991, the Centre's first year as an official Collaborating Centre of WHO.

A visit was made to the Training Centre on the 23rd August, 1991, by WHO Regional Director, Dr. Sang Tae Han and WHO Representative for the South Pacific, Dr. Sung-Kyu-Ahn.

During 1991 Dr R. McKenzie, Mr. J. M. Lynch and Mr J E Elliot visited the WHO Regional Office at Manila while carrying out consultancy assignments for WHO.

Dr. R. McKenzie visited WHO Headquarters, Geneva, where he served as Chairman of a Consultancy Committee on the Assessment of Training Needs in Transfusion Medicine"

Students 1991

Students who completed courses at the Pacific Paramedical Training Centre during 1991 were as follows:

World Health Organisation—Regional Course in Laboratory Diagnosis of STD and HIV, 6th August - 23rd August, 1991.

Augustine Bani, Vanuatu; Rickie Eddie, Solomon Islands; Tonga Havili, Tonga; Maria Marfel, Yap - Federated States of Micronesia; Paul Lolita, Marshall Islands; Tautala Mouala, Western Samoa; Vaevae Pare, Cook Islands; Tebebeku Teia, Kiribati; Viliame Tamaui, Fiji.

Updated Course in Medical Laboratory Technology, 16th September - 18th November, 1991

Hendric Pangoa, Papua New Guinea; Arnold Tali, Papua New Guinea; Philp Tarbogani, Papua New Guinea; Nicole Kauri, Papua New Guinea; Ihave Aikelave, Papua New Guinea; Francis Termetect, Palua, Federated States of

Micronesia; Max Helkena, Marshall Islands; Orisi Matatolu, Fiji.

Cytology Training Course, February - December, 1991
Rebecca Pokou

Clinical Chemistry Training, February 1989 - Ongoing
Filipo Faiga

Kenneth Newell He Tangata Award

The first Kenneth Newell He Tangata Award winner was presented on the 8th November, 1991 to Arnold Tali Pali from Papua New Guinea by Jocelyn Keith, a Trustee of the Award. In her presentation she outlined the background to the Award as follows:

"Professor Kenneth Newell was the foundation Professor of Community Health at the Wellington School of Medicine. He had a long and distinguished career in Universities around the world. He had a long and distinguished career in the World Health Organisation. He had a long and distinguished career in the service of communities, working with them to achieve health by the people.

His premature death last year left many of us with a desperate feeling of loss of direction but also with a tremendous challenge to carry on the work he had set in train His advice was always challenging, direct and honest.

He was a New Zealander and an internationalist of the highest order. He was an Epidemiologist, a teacher, a writer and a storyteller. He believed that a community informed of and in control of its own development is a prerequisite for a truly healthy community. From the start of his career, he was devoted to primary health care which reflected the goals and values of the community it served rather than being opposed from above.

He has left behind his wisdom, a taonga here, and throughout the world. He has left behind a special family. His son, Jamie, is with us today as part of the living Newell presence. His first PhD student after return to New Zealand, Dr. Ron McKenzie is here. Dr. Neil Pearce, now an Epidemiologist on the world scene, is also a trustee.

This is the first Kenneth Newell He Tangata Award. It is our role to protect and nurture his knowledge, to support those who are to play a leadership role in our communities, to ensure that his knowledge and wisdom, continue to live and serve them.

The first recipient is Arnold Tali Pali. Arnold is from Papua New Guinea and is Officer in Charge of Laboratory Services for the Province of New Ireland and Senior Laboratory Technician at Kavieng General Hospital. Arnold has worked in many Provincial Hospital Laboratories in Papua New Guinea. He has just completed the Pacific Paramedical Training Centre Laboratory Update Course in Medical Laboratory Technology. The prize is awarded to Arnold for the best all-round theoretical and technical performance.

E taro nei nga kawai taura tangata
The human links extend like branches of a tree.
No reira tena koutou, tena koutou, tena koutou."

Arnold Tali Pali receiving the first Kenneth Hewell He Tangata Award from Jocelyn Keith, Trustee. In the background is Chris Hawley, Director of Development, Volunteer Service Abroad.

NEW PRODUCTS AND SERVICES

TABLETOP CRYOSTAT FROM BRIGHT INSTRUMENTS

The Starlet 2212 Cryostat from Bright Instruments is a fully function table top unit which takes up no more space than a large Microtome and is transportable enough to be taken from taken from site to site.

All professional facilities for surgical pathology are provided, including auto-defrost, freeze bar, digital displays for chamber and quickfreezer temperature, automatic advance and antiroll device.

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DOUGLAS SCIENTIFIC DIVISION INCORPORATING SCIMED

Douglas Scientific is a newly formed division of Douglas Pharmaceuticals Ltd. Douglas Pharmaceuticals is a New Zealand owned manufacturer and distributor of pharmaceutical products. The move reflects Douglas Pharmaceuticals desire to increase its presence in the analytical, healthcare and biotechnology markets.

The Division is characterised by the acquisition of SCI-MED and our appointment as exclusive distributors for SHIMADZU analytical products.

Douglas Scientific Division Head Office is located in Henderson, Auckland.

Agencies represented by Douglas Scientific incorporating SCI-MED include:

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POSITION WANTED

HAEMATOLOGY TRAINEE

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For further Information please write to:

Siran Lasong
General Hospital,
98000 Miri, Sarawak, Malaysia.

AACB NZ Branch Education Course 1992

Date: Saturday 23rd and Sunday 24th of May 1992.

Venue: Clarke Auditorium, Waikato Hospital.

Course Content:

- Clinical Biochemistry of Acute and Chronic Renal Failure - Dr M Wallace.
- Theory and Practise of Atomic Absorption Spectrophotometry - Ms E Windleborn.
- The Pathophysiology and Biochemistry of Blood Lipids - Dr C Small.
- Blood Gas Instrumentation - Mr D Mikkelsen.
- Carbohydrate Metabolism and Diabetes Mellitus - Dr D Jury.
- The Polymerase Chain Reaction - Dr R Cursons.
- Total Quality Management - Mr R Ward.
- Case Presentations - Various.

The course is open to all people with an interest in Clinical Biochemistry and is especially aimed at those who are preparing for examinations.

Cost: AACB Members - $35
Non Members - $45

Cost includes lunches and morning and afternoon teas.

Accommodation:

There is no on campus accommodation on offer but there are two motels within walking distance of the Hospital. Please make your own arrangements if you need accommodation.

Abbey Travel Lodge 12 Lorne Street, Hamilton Ph (07) 843 4368 $67.50 incl GST	Aloha Motel 52 Tawa Street, Hamilton Ph (07) 843 5284 $52.50 incl GST

Registration details:

Name .....

Address .....

.....

.....

AACB Member (Y/N)
Amount enclosed .....
Please make cheques payable to AACB Course.

Send to: D Mikkelsen
Biochemistry
Waikato Hospital
Private Bag 3200
Hamilton

Closing date for registrations 2nd May 1992.

NEW ZEALAND JOURNAL OF
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