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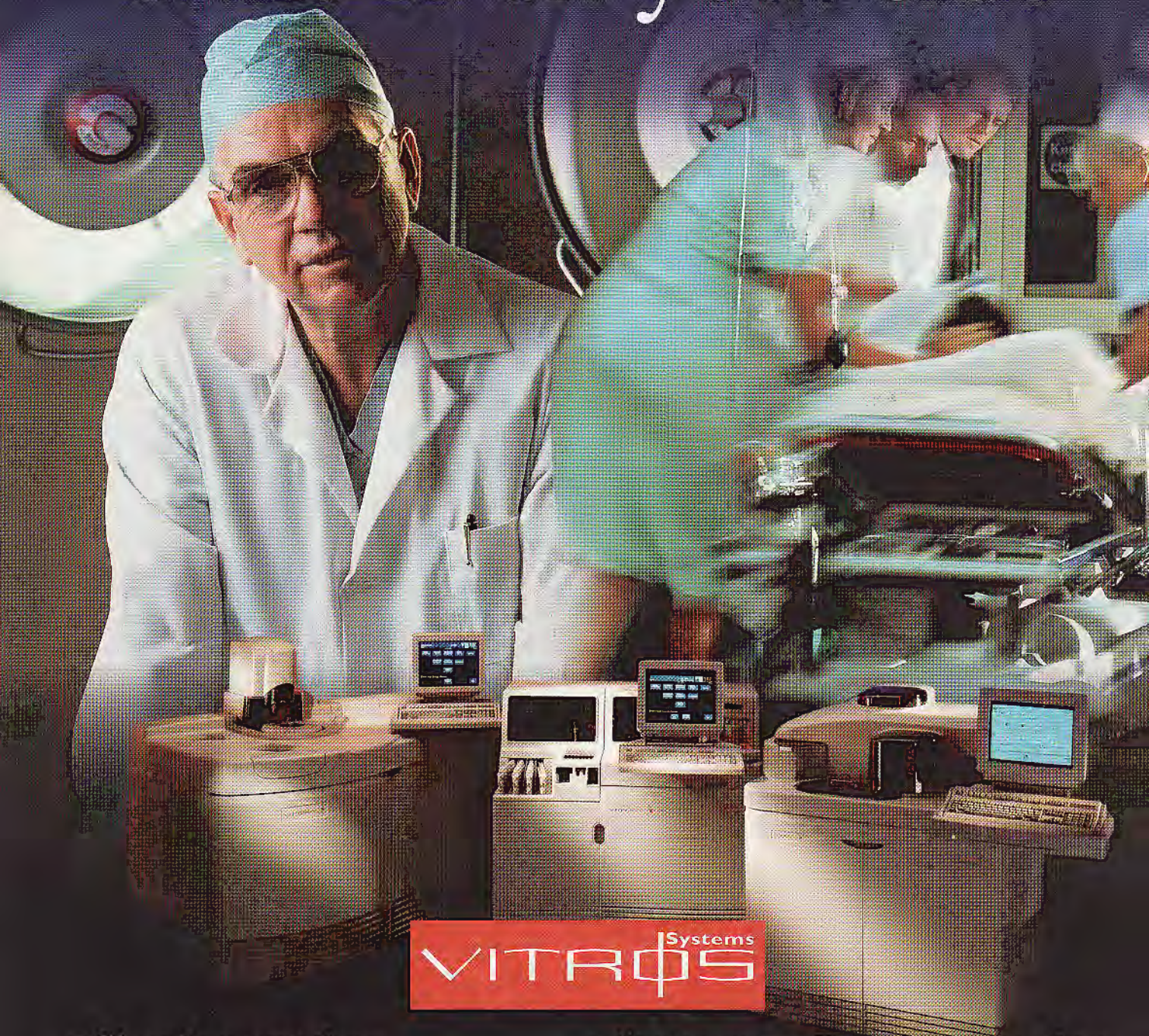

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Papers submitted to the Journal are refereed and acceptance is at the discretion of the Editor. Papers with substantive statistical analysis and data will be reviewed for appropriateness by the Statistical Adviser. No undertaking is given that any article will be published in a particular issue of the Journal. The copy deadline for each issue is the first of the month prior to the month of publication.

Manuscripts:
Submitted papers (**in duplicate**) should be typewritten, in double spacing throughout on one side of A4 paper. Generally each component of the manuscript should begin on a new page in the following sequence.

* **Title of paper**, authors (including first name and qualifications), and institution(s) where the work was carried out. Address for the corresponding author should also be given.

* **Abstract and keywords**. Abstracts should be structured and contain concise and precise information regarding the study's **Objective(s), Method(s), Result(s)** and **Conclusion(s)**. List up to 4 keywords using *Index Medicus* medical subject headings.

* **Text**, in the order of **Introduction, Materials and Methods, Results, Discussion and Conclusion**.

* **References** should follow the style adopted by the US National Library of Medicine as used in *Index Medicus*. Refer to papers in recent issues of the Journal for guidance (or see *NZ J Med Lab Science* 1991; 45 (4): 108-11). Authors are responsible for accuracy of all references.

* **Illustrations** must be provided with a suitable legend typed on a separate sheet. Graphs should be 2-3 times larger than they would appear in the journal and contain a minimum of lettering. Legends for these should also be typed on a separate sheet. Photographs should be original sharp, glossy black & white prints. Authors wishing to submit colour photographs must contact the Editor in the first instance.

* **Tables** should be typed on a separate page complete with a title at the top and footnotes at the bottom. The tables should be numbered as they appear in the text and must *not* contain vertical lines.

* **Acknowledgements** should be made to people and/or organisations who have made substantial contributions to the study. Authors are responsible for obtaining consent from those acknowledged. Financial contributions towards the study from granting bodies or commercial organisations must be stated.

Two copies of the manuscript are to be addressed to the Editor NZ J Med Lab Science, c/- Department of Medicine, Wellington School of Medicine, PO Box 7343, Wellington South, together with a letter from the corresponding author stating that the work is original, is not under consideration for publication elsewhere, and in the case of multi-authorship that all authors have contributed directly to the planning, execution, analysis or to the writing of the paper.

Salmonella – not your average enteric pathogen

Annmarie Clarkin, Senior Technologist
Microbiology Department, Waikato Hospital, Hamilton.

Case History

A 64-year-old woman with a history of angina and non-insulin dependant diabetes mellitus (NIDDM) presented with abdominal pain and dehydration. Routine blood, urine and faeces cultures were obtained.

The urine showed a white cell count of 450 x 10^6/L with bacteria present, and an initial diagnosis of pyelonephritis was made. A heavy pure growth of *Klebsiella pneumoniae* was isolated.

The blood cultures became positive on Day 1 with a gram negative bacillus in two bottles. This isolate was identified through the VITEK system as a presumptive Salmonella species, and typed as Group C using the Wellcolex Colour Salmonella latex. The faeces specimen also grew a Group C Salmonella, biochemically confirmed through the VITEK, and both of these isolates were typed at ESR CDC as *Salmonella Infantis.*

The patient was treated with cefuroxime and amoxycillin and discharged.

Five weeks later, the same patient presented again with abdominal pain. Urine and blood cultures were obtained. The urine white cell count was >1000 x 10^6/L and a preliminary diagnosis of pyelonephritis was made. Culture once again yielded a heavy pure growth of *Klebsiella pneumoniae.* The patient was commenced on 12-hourly ciprofloxacin.

Her blood results on admission showed a Hb of 55 g/L (normal range 115-165 g/L) and a WBC count of 37.3 x 10^9/L (normal range 4.0-11.0 x 10^6/L).

The patient complained of terrible pain in the left abdominal region and a CT scan showed a large area of blood loss in the abdomen. She received 10 units of blood and her results 6 hours after admission showed a Hb of 73 g/L and WBC count of 31.8 x 10^6/L.

The patient was taken to theatre where she had a cardiac arrest and died less than 12 hours after admission.

The patient's blood cultures became positive on Day 4 with a gram negative bacillus, later confirmed as *Salmonella Infantis.*

The post mortem report noted "...the left iliac artery shows a ruptured aneurysm...(which)...measures approximately 2 cm across and has ruptured into the retroperitoneal tissue on the left side causing some large collections of clot to form. The size of the haemorrhage is sufficient to have caused death."

Discussion

The unusual presentation of a repeated isolation of *Salmonella Infantis* from the blood cultures 5 weeks after the original episode, and the cause of death being attributed to haemorrhage from a ruptured iliac artery aneurysm is consistent with previously published data describing Salmonella bacteraemia associated with a mycotic aneurysm.[1]

The term 'mycotic aneurysm' was originally used in 1885 to describe a mushroom-shaped aneurysm (ie: local dilatation or stretching of an artery) that developed in a patient with SBE. At the time, 'mycotic' was used to refer to all micro-organisms. Presently, the use of 'mycotic' has been restricted specifically to fungal infections, but the term 'mycotic aneurysm' is still used for all intra-(or extra-) cardiac aneurysms of infectious etiology except for syphilitic aortitis.

Focal intravascular processes involving the wall of major arteries are uncommon. Haematogenous seeding of a previously damaged atherosclerotic vessel constitutes the most common mechanism of infection. When bacteria 'seed' a preexisting atherosclerotic vessel, gram negative bacilli (chiefly Salmonella) are isolated in 35 percent of cases. The presumed portal of entry is the gastrointestinal tract.

Salmonella infections of aortic aneurysms were first reported in 1948. The predilection for involvement by this organism is not understood but Salmonella organisms tend to seed abnormal tissues during bacteraemia eg: haematomas, malignant tumours, cysts, gall stones, bone infarcts and altered endothelium.[2]

Salmonella gastroenteritis may result in positive blood cultures in 1-4 percent of patients, and it has been estimated that 25 percent of patients over the age of 50 with Salmonella bacteraemia have an intravascular focus of infection because of the high likelihood of atherosclerotic lesions in this age group. Salmonella can produce a syndrome of sustained bacteraemia and fever, and it is important to determine whether the bacteraemia is of high grade (> 50 percent of blood cultures positive) because this suggests intravascular infection.[3]

Therapy

For uncomplicated bacteraemia due to Salmonella species, 7-14 days of antibiotic therapy with amoxicillin, cotrimoxazole or ciprofloxacin is adequate.[2,3] For infections showing evidence of intravascular complications, 6-8 weeks of intravenous antibiotic therapy is essential. (β-Lactam antibiotics are the preferred antimicrobial agents for this type of infection.) Surgical intervention with resection of the infected aneurysm or structure will also be required.[3]

Conclusion

The mortality for patients with infected atherosclerotic aneurysms is very high. A high index of suspicion is necessary for early surgical intervention before rupture occurs. Rupture of the aneurysm is uniformly fatal and occurs in about 80 percent of cases.

There are no characteristic laboratory abnormalities in cases of infected aneurysms. Bacteraemia is found in up to 90 percent of cases, is continuous and usually does not clear with antibiotic therapy alone. Evidence for a primary source of bacteraemia (eg: pneumonia, osteomyelitis) may be evident but is absent in up to 46 percent of cases.[2] A high index of suspicion is needed and factors such as the patient's age, likelihood of atherosclerotic lesions and the degree of bacteremia (ie: whether it is sustained and of high grade) should be taken into account when assessing whether an infected aneurysm is clinically likely.

Helpful diagnostic tools include computed tomography (CT), magnetic resonance imaging (MRI), x-ray, ultrasonography and echocardiography.

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Editorial

Write me a (research) letter

Rob Siebers
Wellington School of Medicine

Our Journal has a proud history of publishing continuously for the last 52 years. The original aims for producing the Journal were "(1) a mean of keeping all members of the Association acquainted with the progress of their fellow members and (2) the dissemination of all knowledge thought to be of interest and use". These aims are still relevant today, especially the latter.

Over the years Editors of the Journal have many times had great difficulty in attracting suitable papers for the Journal from members of our profession. This is despite the many excellent presentations made by them at conferences, seminars and Special Interest Group meetings, and the personal contact made by the Editors to those members presenting there.

Many reasons for not submitting their presentation to the Journal has been put forward, the main one seems to be that it takes much hard work (often in their own time due to heavy work commitments) to construct a paper suitable for publication in the Journal.

We owe it to ourselves, our colleagues who have assisted in the study and to the members of our profession to disseminate more widely the results and findings of the case studies, and method and instrument evaluations and improvements that we have presented orally at scientific meetings.

To make it easier for you to present your findings in the Journal a new category of publication will be introduced, namely the Research Letter. For this type of communication the following guidelines will apply.

1. In no more than 500 to 600 words give a brief background to why the study was done, briefly how it was done (methods), what the main findings were (where appropriate with statistical results), and the conclusion (based on the results).
2. Give the name(s) of the author(s), the institution where the work was done, and a brief title at the start of the Research Letter.
3. No more than one Figure or Table (with an appropriate title) will be allowed.
4. No more than 5 key references can accompany the Research Letter.
5. Acknowledge financial and other support (that does not justify authorship) at the end.
6. The submitted Research Letter will be peer-reviewed as happens at present for submitted papers. Thus if your Research Letter is accepted for publication (at the Editor's discretion) in the Journal it will count as a full publication for MOLS points.

This type of publication has recently been instituted by the Lancet where it has been very popular in relaying scientific study results in a brief but concise manner. It is hoped that the introduction of the Research Letter category to our Journal will result in members of our profession to disseminate their research findings to a wider audience. So if you have in the last couple of years presented at the NZIMLS Annual Scientific Meeting or Special Interest Group meetings and have not previously published it in any scientific or medical journal, please make that little extra effort and write it up and send it as a Research Letter to the Journal.

A Simplified 1gG Anti-D Quantitation Method

Jandhe Carter, Robert Coleman, Graeme Woodfield, Steve Henry.
Department of Transfusion Medicine, Auckland Regional Blood Centre, Auckland, and Auckland Institute of Technology, Auckland
Author for correspondence: Dr. S. Henry, Department of Transfusion Medicine, Auckland Regional Blood Centre, Auckland.

Abstract
Plasma containing anti-D for the production of anti-D immunoglobulin requires quantitation. A simple enzyme linked immunoassay procedure was established which was compatible with the routine procedures of a serology lab. The method developed is albumin free, performed in serological test tubes, and utilised routine serology cell washers. Unlike earlier methods no transferring of the tube contents is required other than to facilitate reading the final colour development with a microplate reader. The technique can reproducibly quantitate a single anti-D plasma dilution over the range of 10-70 IU of anti-D per ml.
Keywords: anti-D quantitation, plasmaphersis

Introduction
The classical and most widely used method for measuring anti-D levels is with a single channel autoanalyser [1,2]. However, other methods can also quantitate antibodies and these include enzyme immuno assays (EIA) of both intact [3] and solubilised red cells [4,5], and more recently by flow cytometry [6,7]. Although flow cytometry is suitable for the quantitation of red cell antibodies this instrumentation is generally not readily available to most laboratories. Serological titration, the most widely available technique, is at best only able to semi-quantitate antibody levels but is subjective in endpoint, and as a consequence has a large range of error.

In 1980 Leikola and Perkins [3] reported a simple enzyme-linked antiglobulin test for the quantitation of red cell antibodies. This test was sensitive and reproducible but encumbered by a requirement to transfer the reactants into new tubes during the procedure. We modified this method to produce one that was compatible with the technology used in a routine serology department that has access to a microplate reader. We could accurately measure anti-D in donor plasma, at levels that were suitable for fractionation into anti-D immunoglobulin.

Materials and Methods
Enzyme immunoasay
A 1:41 dilution of donor plasma (containing anti-D), controls, and standards were prepared by adding 25µl of sample to 1000µl of 0.1% PVP-PBS (0.1% polyvinylpyrrolidone 360,000, (Sigma P-5288, St Louis) dissolved in phosphate buffered saline, pH 7.0) and vortex mixed. Stored plasma samples were clarified by centrifugation in an Eppendorf centrifuge prior to dilution. From the 1:41 predilution, duplicate 100µl samples were added to 10 x 75 mm plain glass tubes followed by 100µl of a 15% washed red cell suspension. The red cells used were of the Rhesus R_or (ccDee) phenotype, recovered from liquid nitrogen, and diluted in 0.1%PVP-PBS. The reactants were incubated at 37°C, resuspended at 30 mins, then incubated for a further 30 minutes before being washed 4 times with PBS in a cell washer (Baxter Serocent, Heraeus Sepatech, WG). 100µl of anti-IgG-alkaline phosphatase conjugate (Dako Corp., D0336, Denmark) diluted 1:250 in 0.1%PVP-PBS was added. Reactants were mixed and allowed to incubate at RT for 30 minutes after which they were again washed 4 times in the cell washer. 200µl of the chromogenic substrate 1 mg/ml *p*-nitrophenylphosphate (Sigma N-9389, St Louis) in 0.1 M carbonate-bicarbonate buffer, pH 9.8 with 0.001 M M_gC1_2 was added. The reactants were allowed to incubate at 37°C for 10-15 mins. The time of incubation was dependent on the age of the conjugate, with longer times being required towards the end of the product's life. The reaction was stopped by the addition of 50µl of 0.2M EDTA. The reactants were then mixed by shaking and the red cells sedimented by centrifugation for 15 seconds in an Immufuge II centrifuge (American Dade, Miami). 100µl of supernatant was transferred to a microplate, 50µl of 1 M NaOH was added to further develop the reaction and the microplate was read at 405 nm. The microplate is used only to facilitate microplate reading and may be washed and reused. Quantitation results were read from the standard curve and reported to the nearest 5 IU.

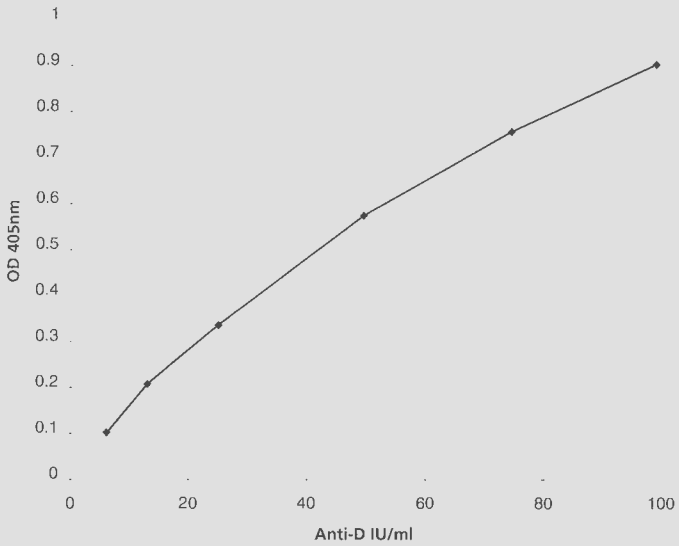
References, Standards and Controls
The primary external standard was the WHO reference prep 68/419 reconstituted to 50 IU/ml. Internal standards were prepared by diluting anti-D positive plasma in anti-D negative plasma to give standards with a range of 100, 75, 50, 25, 12 and 6 IU/ml. In-batch controls consisted of a negative plasma and positive plasma with a nominal value of 45 IU/ml.

Results and Discussion
The method reported here is a refinement of the original method of Leikola and Perkins [3], and incorporating steps which make the method compatible with a routine serology lab. The non-specific binding of immunoglobulins to glass tubes, as encountered by Leikola and Perkins, was eliminated by using 0.1%PVP in the diluents. The use of PVP removed the requirement to transfer the reactants into new tubes prior to addition of substrate. PVP has previously been reported by us to have superior qualities to albumin in microplate assays [8] and is frequently used as a blocking agent in commercial reagents. We chose 15µl of red cells (100µl of a 15% cell suspension) because this was the amount which, when sensitised with anti-D plasma diluted 1:41, poorly supported agglutination when the conujugate was added (ie. antigen in excess) and was compatible with insignificant loss during automated cell washing procedures. Liquid nitrogen recovered R_or cells were used not only to prevent quantitation of anti-C but also to have a constant cell between assays.

A standard curve was prepared with dilutions of a plasma containing anti-D, and were used to sensitise R_or red cells. The relationship

between the amount of anti-D and the final colour development is shown in Figure 1 (standard curve), which typically represents a quantifiable range of 10-70 IU/ml. Often, when using a 1:41 dilution, the curve reaches a plateau when the level of anti-D is in excess of 70-80 IU/ml. Donor samples with levels of anti-D in excess of 70 IU/ml require predilution in antibody negative plasma before testing.

Figure 1. Example of a typical anti-D quantitation standard curve.



Analysis of 26 replicate tests of the internal control (45 IU/ml) revealed that the average variation between duplicates within the same batch was 12% while the average difference in anti-D value between batches was ± 10% (data not shown). In order to validate our method we tested samples from the U.K. Blood Transfusion Service anti-D quality control survey. As this programme is intended for the quantitation of antenatal anti-D samples we selected only those with anti-D within our quantitation range (table 1). Most samples were quantitated within the reported range and were close to the median value found for the U.K. samples. All samples with values less than 10 IU/ml were below the quantitation range of our assay (not shown). As it is known that most autoanalyser users use pooled R_1R_1 (CDe/CDe) cells (personal communication) we exchanged 6 pure anti-D samples with two Australian laboratories (lab1 and lab2) and compared the results (table 2). There appeared to be a large inter-laboratory variation between values obtained for the individual samples. The range was not too dissimilar to that seen in the U.K. survey (table 1).

The U.K. and the two Australian labs used autoanalyser methods to determine anti-D levels. Autoanalyser technology is based on the automated detection of agglutination after significant dilution, while the EIA method relies on the detection of cell bound IgG. The IgM component of an anti-D plasma can make a considerable contribution to the overall value obtained by automated haemagglutination [9]. Ashford and coworkers report that in more than a third of anti-D plasmapheresis samples, after DTT treatment to destroy IgM, the level of anti-D dropped by more than 15%. In some instances much larger drops were observed. It is therefore not surprising that discordant results are seen with some samples when compared by different methods. An anti-D quantitation method using flow cytometry also reports finding several samples with significant variance when compared to an autoanalyser [7]. Like previous investigators [10,11] we also find that reproducibility is dependent upon the serum in use. Some samples had consistently high accuracy whilst others were low.

With the introduction of this technique into routine use for the quantitation of anti-D plasma we have found it to be compatible with routine practice, and of adequate sensitivity and accuracy [12].

Table 1. Comparisons of Anti-D Quality Assurance Data between Auckland and U.K. labs.

	UK range (IU/ml) min	max	U.K. median	Auckland result	ratio A/UK
UKNBS01/96-01 B	3	17	8	10	1.3
UKNBS07/95-03 D	3	22	9	10	1.1
UKBTS07/96-03 C	9	19	12	10	0.8
UKBTS04/94-02 A	6	21	12	10	0.8
UKBTS04/96-02 C	6	23	14	10	0.7
UKNBS01/96-01 D	12	25	18	45	2.5
UKBTS04/93-02 A	10	26	18	10	0.6
UKBTS10/95-04 D	14	35	20	20	1.0
UKBTS04/94-02 D	45	88	56	90	1.6
UKBTS04/93-02 D	33	99	59	70	1.2
UKNBS07/95-03 B	34	89	65	65	1.0

Table 2. Comparisons of Auckland's Anti-D EIA results with autoanalyser results from two independent labs.

	(IU/ml) Anti-D		
Sample	Auckland	Lab1	Lab2
D1	15	14	15
D2	40	31	17
D3	60	31	26
D3.1*	35	14	13
D4	35	71	27

*Sample D3.1 is a half dilution in negative plasma of sample D3.

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South Island Seminar Report

A proven successful format was retained by the organisers of this years' South Island Seminar held at Methven Sovereign Resort Hotel on Saturday 21 March 1998.

Delegates came from Invercargill in the south to Nelson in the north including two delegates from North Island laboratories. These, as well as a significant presence of representatives from a variety of scientific companies, made up the 150 delegates who assembled on a fine autumn Saturday morning in this rural mid-Canterbury setting.

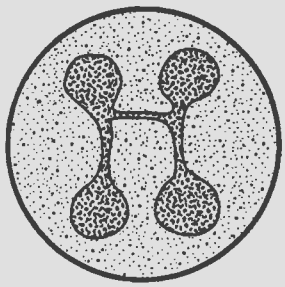
The delegates were welcomed by the Organising Convenor who challenged the audience to be unified in addressing the opportunities and challenges which exist in a continually evolving and changing healthcare environment. Four sessions of diversely interesting and topical papers of a highly commendable quality followed and were presented by a mixture of first time presenters and experienced campaigners who came from the large centralised metropolitan Canterbury Health Laboratories to the smallest laboratory in the country at Balclutha hospital where near patient testing plays a significant part in patient management. The Med-Bio award went to Judith Cooney for her case study in Haemostasis. This lucidly clear and most interesting presentation was of exceptional quality and should not be missed at this year's IMLS conference. The dual presentation of a successful near patient testing venture was very well received, particularly by those who come from the smaller centres. The presence and participation by a clinician intimately involved in this process added insight into the value and practical issues raised in this venture.

A discussion on the role of the South Island Seminar in continuing education resulted in a clear mandate to preserve the current format of the general forums in the South Island. The ball is clearly in our hands.

A panel interview of four scientific company representatives added interest and diversity to the day and was well received. Respect, honesty and openness are the fuel for successful dialogue between supplier and customer. Half hour tea breaks, lunch and a 5:00pm bar session allowed ample time for socialising and that exchange of personal and scientific information which is such a vital component of such a gathering. The evening dinner session which followed, allowed a further opportunity for fraternisation with colleagues and was a fitting conclusion to a most enjoyable informative and stimulating event.

Next year's seminar will be organised by Canterbury Health Laboratories and we look forward to this in anticipation.

J B Kitto
Convenor



Haematology Self-Assessment Journal Reading for MOLS
Questionnaire prepared by Soakimi Tai Po'uhila using true/false format
Reference article: Acute Promyelocytic Leukemia: Biology and Treatment

The following are the correct answers for this self-assessment exercise:

1. APL is characterised by the morphology of blast cells according to the FAB classification of AML — True
2. Cytogenetically APL involves translocation between chromosome 15 and 17 in most cases — True
3. APL cells are usually CD35-, CD11a, and CD15- — False
4. The discovery of the differentiation of APL blasts by all-transretinoic acid (ATRA) has changed the therapeutic approach of APL — True
5. Each APL patient is characterised by a specific fusion transcript bcr1, bcr2, or bcr3 — True
6. PML protein in normal cells is not specifically bound to a nuclear body — False
7. All transcripts bcr1, bcr2 and bcr3 are easily detectable on northern blot analyses — False
8. The APL specific fusion transcripts can all be observed in all cases by reverse transcriptase polymerase chain reaction (RT-PCR) — True
9. Morphological variants of APL are never observed — False
10. Other reported cases of APL showed translocations involving chromosomes other than 15 and 17 — True
11. APL is distinguished cytologically by an arrest at the promyelocytic stage of myeloid differentiation — True
12. Retinoids are successfully used to induce the differentiation in vitro of APL cells to polymorphonuclear cells — True
13. Differentiation of APL cell by retinoids is reduced with the addition of cytokines — False
14. Disseminated intravascular coagulation (DIC) is one of the complications associated with APL — True
15. Intensive platelet support during chemotherapy is a major factor in reduction of incidence of haemorrhagic deaths in APL — True
16. ATRA therapy has no major side effect — False
17. ATRA treatment and intensive chemotherapy have been proven to prolonged remission in APL patients
18. Addition of Low-dose chemotherapy and leucophereses are used to reverse ATRA syndrome — False
19. The ATRA syndrome is due to leukostasis and/or thrombosis — False
20. Cells that harbour translocations resulting in rearrangements of the retinoic acid receptor alpha gene (RARα) but involving chromosomes other than 15, do not differentiate in the presence of ATRA — True
21. Early reports showed that failure to achieve complete remission was due to central nervous system bleeding — True
22. Presence of tumour necrosis factor alpha (TNFα) significantly reduced the efficacy of ATRA to differentiate APL cells — False
23. Significant coagulopathy, present at diagnosis of 80% cases of APL is worsened by the onset of chemotherapy — True
24. Relapse is often associated with blasts acquiring resistance to chemotherapy and to ATRA — True
25. G-CSF reduces the differentiating effect of ATRA on APL cells — False

Journal Based Learning – Questionnaire

Pathogenesis of polycythaemia vera
S Hinshelwood, A J Bench, A.R. Green - Blood reviews (1997) 11:224-232

Please circle your choice of correct answer:

1. PV is divided into three phases — TRUE/FALSE
2. PV results from the transformation of a multipotent stem cell. — TRUE/FALSE
3. Progenitors from PV patients display abnormal responses to several growth factors. — TRUE/FALSE
4. PV with a normal karyotype will not progress to acute leukaemia. — TRUE/FALSE
5. PV is probably a clonal malignancy resulting from acquired genetic events. — TRUE/FALSE
6. X-inactivation occurs in human males and females. — TRUE/FALSE
7. Platelets, red blood cells, granulocytes and erythroid progenitors have all been shown to form part of the malignant clone. — TRUE/FALSE
8. The finding of skewed X-inactivation probably indicates a clonal disorder. — TRUE/FALSE
9. There is a suggestion that x-inactivation patterns could be used in diagnosis of PV. — TRUE/FALSE
10. Endogenous erythroid colonies yielded in PV BM culture are dependent upon erythropoitetin. — TRUE/FALSE
11. Normal progenitor cells do not exist in PV. — TRUE/FALSE
12. Erythropoietin independent BFU-E are found in 97% of PV patients and less frequently in ET. — TRUE/FALSE
13. Erythroid progenitors are hypersensitive to SCF, IL3, 6M-CSF and 16F-1. — TRUE/FALSE
14. Non erythroid progenitors are hyposensitive to IL-3 and GMCSF. — TRUE/FALSE
15. PV progenitors are hypersensitive to cytokines which results in the over production of characteristic blood cells. — TRUE/FALSE
16. EPO-R is activated indirectly by EPO. — TRUE/FALSE
17. 20q- is the most frequently observed chromosomal abnormality in PV patients, (and is observed in other myeloproliferative disorders, MDS and AML). — TRUE/FALSE
18. A raised red cell mass (>25% above normal), absence of cause of secondary polycythemia, thrombocytosis and splenomegaly constitute a diagnosis of PV. — TRUE/FALSE
19. PV is an acquired (somatic) chromosome mutation in a multipotential progenitor that results in dysregulated growth of a clone of abnormal cells. — TRUE/FALSE

Please contact Lynda Kape at Haematology Lab, Diagnostic Lab, P.O. Box 5728, Auckland or phone 357 4113, fax 357-4128, email lynda@diagnostic.co.nz; if you are unable to obtain a copy of this journal article.

Fellowship of the New Zealand Medical Laboratory Science

So what's it all about?

Who can sit and how do you find out more about it?

Application forms and an information booklet (which contains all the regulations for Fellowship) are available through the Executive Officer, NZS, P.O. Box 3270, Christchurch.

Here are some of the requirements for Fellowship of the NZIMLS that may be of interest especially if you already hold a Specialist Certificate.

Firstly the Specialist level examination no longer exist, it has been effectively replaced by the *Part 1* of Fellowship by examination.

General notes for applicants of Fellowship,

1. Applicants must be financial member of the NZIMLS at the time of application and examination. Must be a member of the NZIMLS for not less than 2 years, or are exempt as approved by the Fellowship Committee.
2. The NZIMLS will not accept material that has been submitted or accepted for any other qualification.
3. Any further applications will not be accepted from candidates who have previously made 3 unsuccessful attempts.

Fellowship of the NZIMLS may be gained;

a) by examination, or

b) by submission of a thesis, or

c) by publications

If gaining the Fellowship by a) examination. It consists of two parts.

Part 1: Two written papers each of three hours duration.

Application form and fee must be in by March 31st.

Examinations will be held in the first week of November.

Examinations are offered in all the major disciplines of Medical Laboratory Science, although the NZIMLS reserves the right not to offer al subjects in any one year.

Due to Medical Laboratory Science rapidly changing there will not be a syllabi available. However a list of textbooks and recommended Journals will be provided.

Costs $650 (incl. GST).

Part 2: Upon successful completion of Part 1 a dissertation of 3000-5000 words which directly related to the Part 1 examination.

The dissertation may take the form of;

I. a review

II. development of an hypothesis

III. any other presentation which meets with the approval of the Fellowship Committee

Part 2 must be submitted within three years of completing Part 1. (Extensions may be granted in special circumstances.)

The title and synopsis should be forwarded to the Fellowship Committee by March 31st in the year following successful completion of Part 1. Then submission of the dissertation by September 30th.

In the event of failing the Part 2, the candidate may resubmit their dissertation by March 31st of the following year. (This is at the discretion of the Fellowship Committee.)

Costs $250 (incl. GST).

The NZIMLS reserves the right to publish in the NZIMLS Journal and dissertation submitted.

****Medical Laboratory Scientists who hold a Specialist Certificate are exempt from sitting the Part 1 examination. This well be effective for a maximum period of 3 years, and the final date for applications and synopsis is March 31st 2000.**

If gaining the Fellowship by **b) thesis.**

The thesis should be submitted no later than three years following acceptance of the synopsis. (Extensions may be granted in special circumstances.)

The thesis must be original work and should not exceed 20,000 words.

The thesis must be based on the style of Master of Science by thesis requirements of Universities in New Zealand.

If gaining the Fellowship by **c) publications.**

A minimum of five peer reviewed articles published in international or discipline acknowledged scientific journals may be submitted for consideration. A review of the submitted articles of 3000-5000 words must also be submitted.

Histology

Special Interest Group

Convenor: Elaine Mullins
Contract Address: C/o Pathology, Taranaki Base Hospital, Private Bag, New Plymouth
Phone: 06 7536139 Ext 7874
Fax: 06 7532956

The Histology SIG will once again hold its Annual Seminar concurrently with the annual Conference of the New Zealand Society of Cytology. This year it will be held in Dunedin on 15, 16 and 17 October at Pacific Park.

The tentative programme has been distributed to laboratories, and the committee will be requesting abstracts within the next few months. Your participation in these Annual Seminars is important. No topic is too small or too trivial to be discussed.

Otago is celebrating the 150th anniversary of the first landing of the European settlers, so there will be numerous attractions. For further information or queries, contact Elaine Coory, Anatomy Department, Medical School, Otago University – phone (03) 479-7376, or email elaine.coory@stonebow.otago.ac.nz.

Book Review

Standardised Reporting of Haematology Laboratory Results Third Edition 1997

Ten years ago the Haematology Charge Technologists Group drew up recommendations for the reporting of blood films. These have been refined by the Auckland Haematology Special Interest Group and are presented in a Third Edition of this booklet.

The aims of this document are laudable. They provide a standard nomenclature for reporting results from the haematology laboratory especially the reporting of blood films. As well as standardising the authors have made every attempt to simplify the language used in haematology reporting. This is long overdue with the reporting of blood cell morphology riddled with synonyms which do nothing but confuse the laboratory scientists aspiring to report films and, equally importantly, the clinician trying to interpret the laboratory report. One of the interesting features of the booklet is the appendix section which lists a recommended name for a particular cell type and then next to it the synonyms that the authors have found in the literature. It is interesting to find for simple red cell fragments no fewer than 15 substitute names are listed, ranging from the dangerous sounding 'pincher cell' to the long winded 'keratoschistocyte'.

The format of the book is to present red cell morphology followed by white cell and then platelet morphology. A small colour photomicrograph is presented on the left of the page illustrating the abnormality under discussion. On the right of the page the correct name to describe it and any defining characteristics are presented. This format works well being easy to understand and access. The book provides a comprehensive listing of the common abnormalities seen in the routine haematology laboratory. The quality of the photomicrographs is adequate. The colour reproduction is of variable quality but in all of the picture the abnormality illustrated is clearly discernible.

One of the difficulties in reporting blood films is determining whether an occasional abnormal cell seen carries any pathological significance. A useful table is provided listing various abnormalities and the threshold (expressed as a percentage of cells) above which such an abnormality is significant. It is useful that these statistics are provided both for adult and cord blood samples.

This booklet sets out to present a standardised reporting system for blood films. It achieves this admirably. As such it should be available in all laboratories and will provide an excellent adjunct for teaching programmes for those students learning to interpret and report blood films. The booklet is not a substitute for detailed haematological atlases and, appropriately, no attempt is made to delve into the minutae of microscopy, and in particular the complex classification of leukaemia and myelo or lymphoproliferative disorders.

J. Carter
Wellington

New Products and Services

New configurations for biology and medicine using the more-than-routine microscope Axioskop from Carl Zeiss

Carl Zeiss' Axioskop was launched more than 10 years ago, closing the then existing gap between simple laboratory microscopes and expensive research microscopes. It was the first and only routine microscope which could be equipped for all imaging and contrasting techniques and featured Infinity-Corrected-System (ICS) optics.

The Axioskop is equipped to handle the extreme of situations. For example, it is currently ploughing Antarctic waters on the German polar and marine research vessel MS 'Polarstern' and the Australian research vessel MS 'Aurora Australis'. The Axioskop will be used even further away on board the ISS International Space Station, and has already been part of several Space Shuttle missions.

Carl Zeiss also offers special Axioskop configuration packages for the solution of 'terrestrial' problems, e.g. for histology, genetic engineering and immunology. In addition, special equipment is available for image processing and analysis.

The technology of the Axioskop more-than-routine microscope has proven successful in practical use, ensuring that it has maintained its strong position on the market to this very day.

For more information please contact:

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Fax: (09) 520 5619

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AVL & Med-Bio

Med-Bio Enterprises Ltd is pleased to announce that from the 23 April 1998, they became the exclusive New Zealand distributors of AVL Medical Instruments.

AVL Medical Instruments is a leading global supplier of blood gas and electrolyte analysers. They have a wide range of analysers, to meet any need a laboratory or critical care area may have.

AVL have done very well in the Australian market during the last couple of years and now have approximately 60 instruments place throughout Australia.

For your current and future blood gas and electrolyte analyser requirements contact Med-Bio Enterprises Ltd.
Med-Bio Enterprises Ltd
Rose-Marie Daniel - Technical Representative, North Island
Nicola Egerton - Technical Representative, Wellington South
Ph: 0800 733 599
Fax: 0800 101 441
email: jvincent@medbio.co.nz

Astrovirus detection

Astroviruses are now recognised as a common cause of viral gastroenteritis in young children worldwide. Recent reports indicate that the incidence of astrovirus infection may have been greatly underestimated and that after rotavirus, astrovirus may be the second most common cause of infantile viral gastroenteritis. Astroviruses have been associated with outbreaks of gastroenteritis in hospitals, families, community and adult institutions.

It is therefore important for the clinician to define the cause of infection to ensure correct patient care and to manage and control any outbreaks.

Recently, astrovirus specific monoclonal antibodies have been produced, which have been shown to react with al astrovirus types. This has lead to the development of an enzyme immunoassay for the detection of astroviruses.

DAKO IDEIA™ Astrovirus is a qualitative enzyme immunoassay for the rapid detection of astroviruses in human fecal specimens. The test utilises group reactive monoclonal and polyclonal antibodies.

For more information, please contact us.
Med-Bio Enterprises Ltd
Ph: 0800 733 599
Fax: 0800 101 441
email: jvincent@medbio.co.nz

Roche Boehringer Mannheim Diagnostics
Chlamydia trachomatis detection with COBAS AMPLICOR

Roche Boehringer Mannheim Diagnostics is pleased to announce the launch of COBAS AMPLICOR for diagnostic PCR in New Zealand.

The COBAS AMPLICOR system integrates five instruments into one compact unit, thereby fully automating all steps in the amplification and detection process.

The instrument is a two-segment thermal cycler capable of accommodating a total of 48 specimens, 24 in each segment. Each thermal cycler segment can be controlled independently. Two 12-well stations in the instrument amplify specimens and two stations detect the amplified specimens. An automated pipetting system is incorporated to standardise the aliquoting of reagents and specimens. Reagent dispensing is conducted in a closed-vial system to minimise contamination, and since the procedure is performed automatically, it reduces the risk of manual errors.

An incubation station holds the specimens at 37°C while the on-board washing system washes the magnetic beads which are coated with specific oligonucleotide probes and utilised in the capture of the hybridised nucleic acid complex. Finally a photometer is integrated into COBAS AMPLICOR to perform the colourimetric readings, and an on-line printer prints the results automatically.

With COBAS AMPLICOR up to six assays can be performed in one amplification and up to 100 detections can be run without operator intervention. Automation reduces the amount of labour and 'hands on' time and allows:

- Parallel amplification and detection
- Unattended overnight operation
- Self-monitoring software
- Automatic monitoring of reagent and consumables inventory
- Option for multiple diagnostic tests on one system

Moreover, standardisation of the assay and elimination of manual errors is assured with the following automated features:

- Quality control checks
- Internal control option
- AmpErase® contamination control
- Closed, contained amplification
- Standardised, automated protocols
- Precision pipetting of specimens and reagents
- Bar-coded data entry

COBAS AMPLICOR has been developed to allow both qualitative and quantitative detection. The quantitative systems (COBAS AMPLICOR MONITOR) for viral load measurement provide the additional automated features of calculation of titre and availability of raw data, and a data archive to allow future comparisons of MONITOR results.

For more information contact your Roche Boehringer Mannheim Diagnostics representative, or call David Beins at 09-276 4157, 025-270 5111 or beinsd@bmnz.co.nz.

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Membership Fees and Enquiries
Membership fees for the year beginning April 1, 1998 are:

For Fellows – $101.40 GST inclusive

For Members – $101.40 GST inclusive

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For Non-practising members – $42.20 GST inclusive

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

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

JIM LE GRICE AWARD

This is an award which covers the cost of travelling to and from the NZIMLS Annual Scientific Meeting, accommodation and registration fee.

Any student (in full time tertiary education), qualified technical assistant or staff technologist (with less than 5 years total work experience) is eligible for this award, provided they are a member of the NZIMLS.

The only criteria being presentation of a paper or poster at the Annual Scientific Meeting.

Application forms and any more details pertaining to this award are available from:

The Executive Officer
NZIMLS
P O Box 3270
Christchurch

The Pacific Way

More Fishing on the Reef by Bullumakau

Just received from Rarotonga was Volume 1, Issue 1 of the Rarotonga Hospital Labnews. It seems that all members of the laboratory staff have organised this newsletter – perhaps the first such newsletter in the Pacific. Great work. It contains information on new request forms, laboratory handbooks and updates about the tests available in the various sections of the laboratory. The final section of the newsletter is headed 'From The Bosses Desk'. The hospital in Raro is in the process of developing total quality systems and the lab is in the forefront of this movement. Well done.

It has been rumoured that Tirath Lakshman the PPTC tutor of the Blood Bank Technology course is running special courses in fence building for trainees on weekends. I am told that a young lady from Rabi Island is most useful with a spanner. The PPTC is thinking of running a course in the near future called 'Redesign and Rebuild your Laboratory in One Hectic Weekend'. Any applicants??

Who would have thought that the Highlands of Papua New Guinea would ever have been troubled by lack of rain. Well it happened in 1997 with the effects of El Nino and it seems that some 500 lives have been lost as a result of starvation and drought related diseases. Food aid has been entering the country but it is not easy to distribute it in a terrain where there are few roads.

In 1994 American scientists claimed they had discovered a cure for malaria. For security reasons (remember World War II was still on) the nature of drug was kept hush hush. Don't you think that it is about time the world was told about this new cure?

Bullumakau received good feedback about a presentation at the NZIMLS South Island Seminar from Liz Stephenson about her time living and working at the laboratory in Vanuatu. People at the Seminar were most interested in aspects of laboratory work in the tropics. Liz and her family are now off to Fiji for the next three years so we will keep in touch and I am sure that we will hear more from her in the future.

A bad start to the year in Fiji when in the first two weeks six people died of dengue fever. Bullumakau was told that the lab in Suva was performing over 100 dengue tests per week and that hospital blood banks were working hard to provide the needed supplies of platelet concentrate. The last dengue epidemic in Fiji was in 1991 when 30 people died. The answer to the problem is to tighten up the environmental conditions that allow the mosquito to breed. It seems that the Aedes mosquito bites during the day and lays its eggs in still water. Perhaps other Pacific countries should investigate the types of lab tests that are available for the diagnosis of dengue.

A group of lab techs and pathologists were in Fiji last November attending a WHO workshop at the Fiji Postgraduate Medical Centre. I am told that the workshop was very useful and all had the opportunity to visit the laboratory at CWM Hospital. For some at the workshop it was the first time that they had been back to the laboratory since graduating from the Fiji Laboratory Technology School away back in..., well a long time ago.

Pacific Profile

Name: Vasemaca Dolo Sigabalavu (Dolo)

Present Position: Laboratory Superintendent
CWM Hospital, Suva
Fiji.

Training and Qualifications: Started Medical Laboratory Course, Fiji School of Medicine 1962.
Graduated with Certificate in Medical Laboratory Technology, 1964.
Joined CWM Hospital Laboratory 1965.
Promoted to Technical Officer 1 in 1974.
Trained medical cytology at National Women's Hospital 1975.
QTA Medical Cytology 1977.
Promoted to Laboratory Superintendent 1987.

Main interests in laboratory work: Medical cytology.

Highlight of my career: Reaching the top most position in the laboratory structure and being the first woman in Fiji to do so.
Upgrading the Divisional and Sub-Divisional Hospital Laboratories in Fiji and introducing internal and external QC programmes.

What are the main issues facing medical laboratory work in Fiji: Maintaining adequate and skilled manpower in urban and rural hospital laboratories.
Keeping up with the latest technology and equipment.
Lack of continuing education opportunities (eg journals)
Lack of Registration of qualified staff and accreditation of laboratories.

Blood Morphology Course: 1-26 September 1997
Back row: Andrew Pala (PNG), Peter Kelinga (PNG), Marilyn Eales, Mike Lynch (PPTC), Paula Finau (Tonga), Aikun Naus (PNG)
Front row: Keshni Sharma (Fiji), Naomi Sameulu (Samoa), Ana Mosese (Solomons), Regina Flood (Kiribati)

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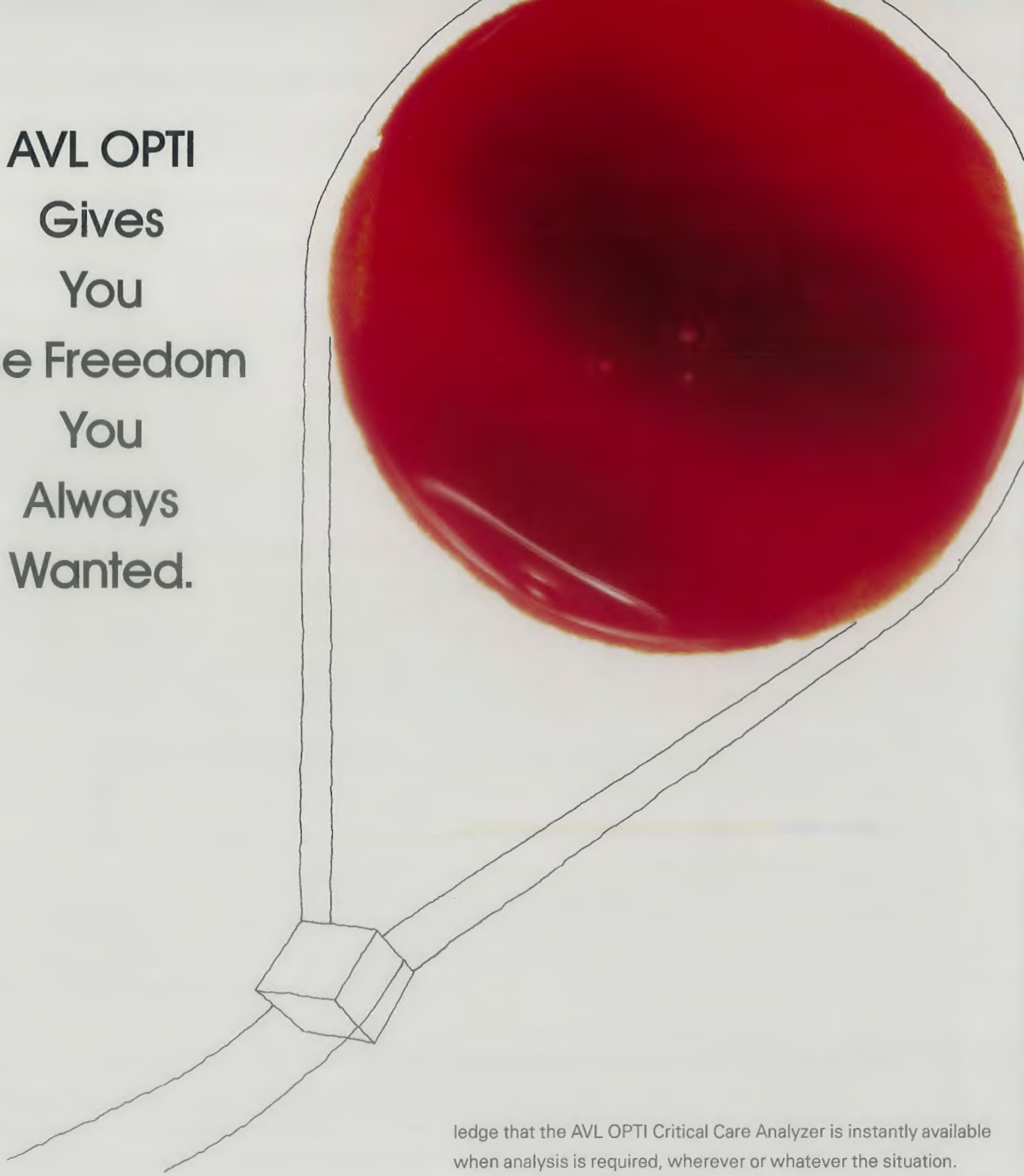

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