New Zealand Journal of Medical Laboratory Science

Official Publication of the New Zealand Institute of Medical Laboratory Science Incorporated

Basics and Beyond
Christchurch 2005
14-18 August
STOP PRESS
THE NUMBERS ARE OUT!!

Record Numbers of ARCHITECT systems placed in 2004!

☑ 375 c8000 chemistry analysers
☑ 198 ci8200 integrated IA-CC systems
☑ 512 i2000SR immunoassay analysers

Over 550 c8000 chemistry analysers have been sold internationally since launch less than 18 months ago. Talk to us to find out why our ARCHITECT systems are outselling all others internationally and in NZ!

ABBOTT Diagnostics Division 0800 656 233
www.abbottdiagnostics.com
Editor
Rob Siebers, MBiol, FNZIC, FNZIMLS; Wellington School of Medicine & Health Sciences

Deputy Editor
Ann Thornton, FNZIMLS; Wellington School of Medicine & Health Sciences

Editorial Board
Jenny Bennett, FNZIMLS; ESR, Porirua
Gloria Evans, MMLSc, FNZIMLS; Canterbury Health Laboratories
Graeme Paltridge, FNZIMLS; Canterbury Health Laboratories
Vanessa Thornson, FNZIMLS, Hawkes Bay Hospital Laboratory
Jackie Wright, FNZIMLS, Nelson Hospital
John Stirling; Adelaide
Tony Woods, PhD; Adelaide

Statistical Adviser
Gordon Purdie, BSc; Wellington School of Medicine & Health Sciences

All editorial matter including submitted papers, press releases and books for review should be sent to the Editor: Rob Siebers, Dept. of Medicine, Wellington School of Medicine, PO Box 7343, Wellington South. Phone: (04) 385 5999 Ext. 6838. FAX: (04) 389 5725. E-mail: rob@wnmeds.ac.nz. Comprehensive instructions for authors can be found in the NZ J Med Lab Science 2000; 54(3): 108-10 or on the website (www.nzimls.org.nz). Submit two copies of the manuscript (and on disk if possible) together with a covering letter from the corresponding author stating that the work is original, is not under consideration for publication elsewhere, that all listed authors have contributed directly to the planning, execution, analysis or to the writing of the paper, and that all cited references have been checked against the original article or appropriate data bases. Contributors and advertisers are responsible for the scientific content and views. The opinions expressed in the Journal are not necessarily those of the Editor or Council of the NZIMLS

The New Zealand Journal of Medical Laboratory Science is the official publication of the New Zealand Institute of Medical Laboratory Science (NZIMLS) who owns the copyright. No parts of this publication may be reproduced in anyway without the written permission of the NZIMLS. The Journal is published three times per year in April, August and November, printing by Centurion Print, PO Box 6344, Wellesley St., Auckland.

The Journal is abstracted by the Cumulative Index to Nursing and Allied Health Literature (CINAHL), Index Copernicus, Excerpta Medica/EMBASE, Chemical Abstracts, and the Australian Medical Index. The Journal, through its Editor, is a member of the World Association of Medical Editors (WAME).

Advertisement bookings and enquiries should be addressed to the Advertising Manager: Trish Reilly, 4B Towai St., St Heliers, Auckland. Phone: (09) 575 5057, Fax: (09) 575 0698. Enquiries regarding subscription and address changes should be addressed to the Executive Officer of the NZIMLS: Fran van Til, PO Box 505, Rangiora. Phone: (03) 313 4761. E-mail: fran@eenz.com.
Editorial

CPD programme update
Jillian Broadbent, FNZIMLS
Canterbury Health Laboratories, Christchurch

CPD coordinator
The beginning of February saw me taking up my appointment as the Coordinator for the NZIMLS CPD Programme. Many of you may already know me, but for those who don’t, I shall give you a brief overview of my career to date.

Jillian Broadbent (nee Wilson) - I trained at Auckland Hospital Laboratory and completed a Part 2 and Part 3 in Clinical Biochemistry. Having trained the “old fashioned” way means that I have spent time in most disciplines of Medical Laboratory Science. I spent some time working at Greenlane Hospital laboratory where I was Technologist in Charge of the Protein and Lipid Laboratory before moving to Christchurch to work as the Diagnostic Sales Representative for Boehringer Mannheim (now Roche Diagnostics). My portfolio included Medical Laboratory Diagnostic products for the Clinical Biochemistry department including reagents and major instrumentation, Research Biochemicals especially those for the Molecular Biology sector, Point of Care instruments, Food Analysis kits and Customer Training.

Family considerations kept me temporarily out of the work force for a while, but I now work part-time in the Steroid and Immunobiochemistry laboratory at Canterbury Health Laboratories. In 2003 I was awarded my Fellowship of the NZIMLS for my treatise on Salivary Testosterone.

My experience on the bench, my part-time position in the laboratory and my sales and marketing skills will hopefully make me a capable and understanding person to coordinate and facilitate the implementation of the CPD Programme into participating NZ Medical Laboratories. (Yes, I also have to earn CPD points!)

CPD enrolments
These are continuing to be processed with many laboratories having taken advantage of the discount offered for group membership. Individual email addresses are necessary for access to CPD records and all CPD activities for each participant can be logged through the NZIMLS website.

Regulations now require all Medical Laboratory Scientists to participate in a competency programme and Annual Practising Certificates will not be issued by the Medical Laboratory Science Board (MLSB) this year unless you are enrolled in a CPD programme.

Activities
The NZIMLS council will meet with the MLSB in Wellington, areas of interest to include MLS registration and CPD programme review.

Negotiations are under way for the audit process of the CPD programme. This will be an annual requirement and approximately 10% of participants will be audited. It is important to keep records of all activities you have claimed points for.

SIG Convenors are currently working on programmes for the Structured Reading section of the CPD programme. As soon as these are available, they will be electronically mailed to all participants and also posted on the NZIMLS website.

A section for Frequently Asked Questions (FAQ) is currently under development on the NZIMLS website and all suggestions and feedback are welcome from participants.

A web-based process for application for approval of meetings as CPD approved activities and guidelines for submission of scientific

programmes for CPD points approval will be developed.

Web based learning programmes are being researched so that staff working part-time or permanent shifts will not be disadvantaged and Continuing Education will not be inaccessible to them.

I hope to visit as many Medical Laboratories as possible during this year to discuss the programme and to understand any issues staff may have with managing and accruing their points. I also hope to recruit CPD officers in most of the laboratories so there is someone on-site to answer any questions or clarify any matters you may have difficulty with.

I look forward to any communication you may have on the programme and all questions and comments are to be encouraged and appreciated. The introduction of this programme will hopefully strengthen our profession, enhance our continuing education and ultimately benefit and protect the public of New Zealand.

Contact details: Jillian Broadbent
Email: cpd@nzimls.org.nz
Phone: +64 3 343 5177
Mobile: 027 242 4712

NZ J Med Lab Science 2005
2
Abstract
Toxigenic strains of Vibrio cholerae, although not endemic in New Zealand, are isolated occasionally from recent overseas travellers. Isolates should be confirmed biochemically and serologically and the presence of cholera toxin genes established by a reference laboratory, as non-toxigenic strains are found here in the summer months. Serotypes O1 and O139 are the causative agents of epidemic cholera, but the potential exists for other serotypes to become of public health significance because the production of cholera toxin is phage-encoded. It is also likely that the receptor enabling uptake of the cholera toxin phage and attachment of the organisms to the gut wall is phage-encoded, and thus both virulence factors are potentially transferrable to previously non-pathogenic vibrios. Toxigenic strains of Vibrio cholerae are notifiable in New Zealand.

Key words: Vibrio cholerae, toxigenic strains, serotype, New Zealand

Introduction
Modern air travel has changed our expectations of likely gastrointestinal pathogens. An organism that has been ingested on one side of the world can reappear in a faecal specimen taken on the other side of the world 12 hours later, which can be less than the incubation period of some diseases. A history of recent overseas travel is hugely relevant to the microbiologist trying to decide on which pathogens are likely to be isolated. Sadly, these details are often not provided and workers have to either guess (patient name likely to indicate travel...? patient age group likely to indicate travel...? medical practice/doctor/clinic referring specimen specialises in travel medicine...? sample looks like it contains 'something really nasty'...?) or culture the sample on every available medium to ensure nothing is missed.

This is not a new situation. In November 1972, a 65-year-old man died in Lower Hutt from cholera, acquired following consumption of an aircraft-meal taken on board at Bahrain (1,2). Three cases of cholera were not seriously ill, but this scenario illustrates the need to be aware that a faecal sample may contain a pathogen not endemic in New Zealand.

The Enteric Reference Laboratory at ESR generally confirms one or two cases of toxigenic Vibrio cholerae a year, all acquired from overseas. Non-toxigenic strains are endemic in the warmer months and are usually associated with bathing, especially in the Eastern Bay of Plenty. These non-toxigenic strains are reported as V cholerae non-O1, non-O139 and were previously known as non-agglutinating vibrios or NAGs. In June 2004, two unconnected cases of toxigenic V cholerae infection were confirmed in one week, prompting this technical report.

Microbiology
V cholerae is a gram negative, oxidase positive, fermentative bacillus that exhibits characteristic darting motility. It is readily grown on thiosulphate citrate bile sucrose agar (TCBS) agar, on which colonies are luxuriant and yellow in colour, due to the fermentation of sucrose. Initial pre-enrichment in alkaline peptone water (pH 8) can be used with subculture to TCBS after 5-8 hours. For convenience, 25ml aliquots of TCBS agar can be stored, melted and poured as required. Isolation media are incubated at 30°C but biochemical testing is undertaken at 37°C. V cholerae can be distinguished from Vibrio alginolyticus, which it resembles on TCBS, by the latter's requirement for NaCl. V alginolyticus is a strict halophile, which will grow in 10% but not 0% NaCl. Suspected isolates of V cholerae should be referred to a reference laboratory for confirmation, serotyping and toxin testing.

Biochemical confirmation
The Enteric Reference Laboratory at ESR confirms isolates using conventional biochemical tests, including indole, MRVP, lysine and ornithine decarboxylase, arginine dihydrolase, and the following carbohydrates: glucose (acid and gas production) L-arabinose, arabitol, cellobiose, lactose, maltose, D-mannitol, salicin and sucrose (Table 1). Salt tolerance is tested using broths with various NaCl concentrations ranging from 0% to 10%. Slide agglutination is performed with O1 and O139 antisera and isolates reactive with anti-O1 are further sub typed using antisera specific for Ogawa, Inaba and Hikojima strains.

All isolates are tested for sensitivity to the vibriostatic agent O129 (2,4-diamino-6,7-dipropylpteridine). Sensitivity to this agent was once regarded as a diagnostic feature of Vibrio cholerae serotype O1, and resistance indicative of Vibrio cholerae serotype O139, but isolates of both serotypes may be sensitive or resistant. Resistance is associated with chromosomal insertion of a transposon (3). A 1992 study in India of O129 resistance in 98 consecutively isolated strains of V cholerae O1 biotype El Tor (49 strains each of subtypes Ogawa and Inaba) found that 90% were resistant to 10g and 82.7% were also resistant to 150g of O129 (4).

Table 1. Biochemical characterisation of Vibrio cholerae

<table>
<thead>
<tr>
<th>Substrate</th>
<th>% Positive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Indole</td>
<td>99</td>
</tr>
<tr>
<td>VP</td>
<td>75</td>
</tr>
<tr>
<td>Lysine dcarboxylase</td>
<td>99</td>
</tr>
<tr>
<td>Arginine dihydrolase</td>
<td>0</td>
</tr>
<tr>
<td>Ornithine dcarboxylase</td>
<td>99</td>
</tr>
<tr>
<td>Glucose acid</td>
<td>100</td>
</tr>
<tr>
<td>Glucose gas</td>
<td>0</td>
</tr>
<tr>
<td>L-arabinose</td>
<td>0</td>
</tr>
<tr>
<td>Arabitol</td>
<td>0</td>
</tr>
<tr>
<td>Cellobiose</td>
<td>8</td>
</tr>
<tr>
<td>Lactose</td>
<td>7</td>
</tr>
<tr>
<td>Maltose</td>
<td>99</td>
</tr>
</tbody>
</table>
D-mannitol 99
Salicin 1
Sucrose 100
Salt tolerance 0% 100
1% 100
6% 53
8% 1
10% 0

Subtyping:

There are over 155 (O) antigenic types of V cholerae, but prior to 1992, epidemic cholera was restricted to serotype O1. There are two biotypes of O1, classical and El Tor, which are differentiated by the VP test, sensitivity to Polymyxin B SOU, haemolysis of sheep erythrocytes and agglutination of chicken erythrocytes (Table 2). Bacteriophage typing can also be used to differentiate the biotypes.

Table 2. Differentiation of classical and El Tor biotypes of V cholerae O1.

<table>
<thead>
<tr>
<th>Test</th>
<th>Classical</th>
<th>El Tor</th>
</tr>
</thead>
<tbody>
<tr>
<td>VP</td>
<td></td>
<td>+</td>
</tr>
<tr>
<td>Polymyxin B SOU</td>
<td>Sensitive</td>
<td>Resistant</td>
</tr>
<tr>
<td>Haemolysis sheep rbc</td>
<td>Non-haemolytic</td>
<td>Haemolytic</td>
</tr>
<tr>
<td>Agglutination chicken rbc</td>
<td></td>
<td>+</td>
</tr>
</tbody>
</table>

In addition to serotyping and biotyping, O1 strains can be further characterised by subtyping the minor O antigens or O factors (Table 3). In late 1992, a new serotype causing epidemic cholera emerged in India and Southern Bangladesh (5,6). Serotype O139 is thought to have evolved from a strain of O1 biotype El Tor, by substitution of rfb genes that code for the O1 lipopolysaccharide (LPS) in V cholerae O1 with genes that code for the capsular antigen of V cholerae O139 (7). This serotype, also referred to as the Bengal strain, was responsible for disease in the Indian subcontinent, neighbouring countries in Asia, and imported cases in developed countries. It was the dominant strain of V cholerae for a time but now the El Tor biotype of serotype O1 again predominates.

Table 3. Subtypes of V cholerae O1

<table>
<thead>
<tr>
<th>Subtype of O1</th>
<th>Minor antigens/O factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ogawa</td>
<td>A and B</td>
</tr>
<tr>
<td>Inaba</td>
<td>A and C</td>
</tr>
<tr>
<td>Hikojima</td>
<td>A, B and C (this subtype is rare and unstable)</td>
</tr>
</tbody>
</table>

Toxin testing

Cholera toxin (CT) causes the disease of cholera. Toxinogenic strains are notifiable in New Zealand and our Ministry of Health is obliged to report them to the World Health Organisation (WHO). CT production is associated with serotypes O1 and O139, both of which cause epidemic cholera. Rarely, CT-negative strains of these serotypes are also found. In addition, CT-production has been reported in serotypes other than O1 and O139, although in considerably smaller amounts than is usual with the epidemic strains (8). Non-O1, non-O139 strains have been associated with sporadic cases of gastroenteritis, including cholera-like diarrhoea, mainly in tropical areas (9).

However, the toxin is encoded by a temperate bacteriophage, CTX (10), which requires the presence of a specific receptor, the toxin coregulated pilus (TCP). TCP is one of the colonisation factors produced by vibrios, enabling attachment to the gut wall (11). The gene cluster encoding TCP is found on the V cholerae pathogenicity island (VPI). Karolis and colleagues (1999) reported that the VPI is also phage encoded (12), although this has not been confirmed by other workers (13). The possibility remains that any serotype of V cholerae has the potential to acquire the genes for both the receptor and the cholera toxin and therefore strains of V cholerae should be examined for toxigenicity (9). This is done by PCR in the Enteric Reference Laboratory, using primers for the O1 and O139 serogroups of V cholerae, and CT-specific sequences (14), as shown in Figure 1. The toxin itself is very similar to the heat-labile toxin produced by toxigenic strains of E coli and can be demonstrated in tissue culture.

Figure 1. PCR gel showing O1, O139 and CTX-specific bands. New Zealand isolates of V cholerae

Toxigenic strains of V cholerae are rarely isolated in New Zealand and infections are invariably acquired overseas. The situation is different for non-toxigenic strains however. Table 4 gives the incidence of both toxigenic and non-toxigenic strains isolated over the last five years. There have been two Vibrio cholerae isolates in 2004 to date (October). Both were toxigenic strains of O1 biotype El Tor, subtype Ogawa and were resistant to O129 150U. One strain was isolated from a patient in Rotorua and one was isolated from an Auckland patient. Although unconnected, both patients had recently travelled to India.

Conclusions

Strains of Vibrio cholerae capable of causing epidemic cholera are isolated rarely in New Zealand, but a fatal case of cholera did occur in the 1970s. The potential for non-O1, non-O139 strains to cause epidemic cholera exists as the toxin gene is phage encoded and therefore mobile, and the genes encoding the toxin receptor may also be acquired. Recently serotypes other than O1 and O139 have been shown to produce small amounts of cholera toxin. It is important that isolates of Vibrio cholerae are examined for the potential to produce cholera toxin in addition to being serotyped.

Non-O1, non-O139 strains are endemic in New Zealand and may be isolated from faecal samples or infected ears, especially in the summer months. These strains do not produce cholera toxin but they do produce other toxins and may cause diarrhoea. Susceptibility to the vibriostatic agent O129 can no longer be used as an indication of Vibrio species as many strains are now resistant. The serotype V cholerae O1 biotype El Tor, subtype Ogawa is currently the most frequently isolated toxigenic type in New Zealand.
### Table 4. *Vibrio cholerae* isolates 1999-2003

<table>
<thead>
<tr>
<th>Date</th>
<th>Organism</th>
<th>Number</th>
<th>District</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>1999</td>
<td><em>V. cholerae</em> non-01,</td>
<td>4</td>
<td>Waikato: 3</td>
<td>3 faecal isolates, 1</td>
</tr>
<tr>
<td></td>
<td>non-0139</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Otago: 1</td>
<td>isolate from otitis media</td>
</tr>
<tr>
<td>1999</td>
<td><em>V. cholerae</em> O1 biotype</td>
<td>1</td>
<td>Central</td>
<td>Recent overseas</td>
</tr>
<tr>
<td></td>
<td>El Tor, subtype Ogawa</td>
<td></td>
<td>Auckland: 1</td>
<td>travel to Fiji</td>
</tr>
<tr>
<td>2000</td>
<td><em>V. cholerae</em> non-01,</td>
<td>6</td>
<td>Central</td>
<td>All faecal isolates</td>
</tr>
<tr>
<td></td>
<td>non-0139</td>
<td></td>
<td>Auckland: 1</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Wellington: 1</td>
<td>1 ear isolate</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Canterbury: 1</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Tauranga: 1</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Hastings: 1</td>
<td></td>
</tr>
<tr>
<td>2001</td>
<td><em>V. cholerae</em> O1 biotype</td>
<td>2</td>
<td>Central Auckland</td>
<td>1 ROT Bali</td>
</tr>
<tr>
<td></td>
<td>El Tor, subtype Ogawa</td>
<td></td>
<td></td>
<td>1 ROT India</td>
</tr>
<tr>
<td>2001</td>
<td><em>V. cholerae</em> O1 biotype</td>
<td>1</td>
<td>Central Auckland</td>
<td>1 ROT China</td>
</tr>
<tr>
<td></td>
<td>El Tor, subtype</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hikojima</td>
<td></td>
<td>Auckland: 1</td>
<td></td>
</tr>
<tr>
<td>2002</td>
<td><em>V. cholerae</em> non-01,</td>
<td>4</td>
<td>Central Auckland</td>
<td>3 faecal isolates</td>
</tr>
<tr>
<td></td>
<td>non-0139</td>
<td></td>
<td></td>
<td>1 ear isolate</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Waikato</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Wellington</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Blenheim</td>
<td></td>
</tr>
<tr>
<td>2002</td>
<td><em>V. cholerae</em> O1 biotype</td>
<td>1</td>
<td>Central Auckland</td>
<td>ROT India</td>
</tr>
<tr>
<td></td>
<td>El Tor, subtype Ogawa</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2003</td>
<td><em>V. cholerae</em> non-01,</td>
<td>13</td>
<td>Northland: 1</td>
<td>11 faecal isolates</td>
</tr>
<tr>
<td></td>
<td>non-0139</td>
<td></td>
<td></td>
<td>2 ear isolates</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Central Auckland</td>
<td>1 ROT Cambodia</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Tauranga: 1</td>
<td>1 ROT not</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Gisborne: 1</td>
<td>specified</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Manawatu: 1</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Canterbury: 1</td>
<td></td>
</tr>
<tr>
<td>2003</td>
<td><em>V. cholerae</em> O1 biotype</td>
<td>1</td>
<td>Central Auckland</td>
<td>ROT Thailand</td>
</tr>
<tr>
<td></td>
<td>El Tor, subtype Inaba</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### References


**Address for correspondence:** Jenny Bennett, Enteric Reference Laboratory, ESR Kenepuru Science Centre, Porirua. Email: Jenny.Bennett@esr.cri.nz

*NE J Med Lab Science* 2005
Foetal maternal haemorrhage detection with the Kleihauer technique for postnatal immunoglobulin dose evaluation in Sudan

Mohamed Siddig M. Ali, PhD, Assistant Professor and Head of the Department of Haematology; Ahmed Younis M El Amin, MBBS, Assistant Professor of Haematology; Maysoon Gamal, BS, Teaching Assistant; Nasrin Abdulla, BS, Teaching Assistant; Amal Mohamed, BS, Teaching Assistant.

Haematology Department, Faculty of Medical Laboratory Sciences, El Neelain University, Khartoum, Sudan

Abstract

Objective: The intent of this study was to evaluate the standard routine dose (500 IU) of Rh immune globulin (RHIG) therapy, which is offered routinely to all RhD-negative mothers delivering RhD-positive babies in Sudan.

Methods: Blood samples from 140 pregnant women who were admitted for delivery to various Khartoum State hospitals were tested by the Kleihauer technique to determine the amount of fetomaternal haemorrhage (FMH) in the maternal circulation.

Results: The results of the study demonstrated that the circulation of 10 out of 140 mothers (7.1%), tested by the Kleihauer method, contained more than 4ml of foetal blood. In addition, the association between foetal haemorrhage and mothers' age, duration of pregnancy, baby weight, circumcision of the mother (cutting of the clitoris), type of delivery, and mothers' gravidity was statistically insignificant.

Conclusions: The study concluded that 7.1% of mothers had a possibility of greater than 4ml of FMH. For these mothers, the standard RHIG dose of 500IU would be inadequate and they would need additional RHIG to prevent sensitization and potential harm to future babies. It is recommended to test all RhD-negative women delivering RhD-positive babies routinely with the Kleihauer method for detection and quantification of FMH in order to determine the correct dose of RHIG to be administered.

Key words: Rh immune globulin, Kleihauer technique, foetal maternal haemorrhage, Rh sensitization, Sudan

Introduction

Red cell sensitization is a reversible binding between an antibody and its corresponding red cell antigen. It can occur as a result of the passage of foetal red cells into the maternal circulation transplacentally. Moreover, miscarriage, ectopic pregnancy, blood transfusion and episodes during pregnancy that causes transplacental bleeding, such as amnioncentesis and chorionic villus sampling, can also precipitate sensitization (1). Asymptomatic transplacental passage of foetal red cells occurs in 75% of pregnant women either during pregnancy or during labour and delivery. The volume of foetal red cells that enter the maternal circulation also increases as the pregnancy progresses (2). Potential sensitization of mother's red blood cells (RBCs) is determined by the existence of a maternal-foetal blood group incompatibility and by the extent of foetal maternal haemorrhage (FMH). However, the primary immune response, which usually results in the production of IgM antibodies, is often weak. IgM antibodies do not cross the placenta. Therefore, ABO haemolytic episodes, most often caused by IgM antibodies, are usually mild and rarely responsible for foetal death (2-4).

However, RhD antibodies are primarily IgG and are able to cross the placenta once the primary response has developed. Subsequent exposure to RhD-positive RBCs produces a rapid increase in anti-D antibodies, which are primarily IgG, resulting in haemolytic disease of the newborn (2,4). The disease occurs when RhD-negative mothers, immunized to RhD-positive RBCs during a previous pregnancy become pregnant again with an RhD-positive foetus (5). Haemolytic disease of the newborn born due to RhD incompatibility has become much less common following the introduction of anti-D prophylaxis (1), the dose for which depends upon the size of the foetal maternal haemorrhage as determined by the Kleihauer technique (6). Since primary immunization during pregnancy can precipitate sensitization, underestimation of the volume of transplacental haemorrhage is the major cause of treatment failure due to inadequate dose of Rh immune globulin (RHIG) (5).

Severity of the disease depends upon the nature of the individual's immune response, which may range from mild haemolysis to severe anaemia with compensatory hyperplasia of erythropoietic tissue leading to massive enlargement of the liver and spleen (4). The standard dose of 500 IU of RHIG given in Sudan to RhD-negative mothers antenatally is lower than that used in Europe, the United States and Canada, where 1000-1500 IU is the standard dose (7).

This dose is given to all RhD-negative mothers without measuring the volume of transplacental haemorrhage. However, adjustment of this dose according to the amount of foetal maternal haemorrhage of mothers is mandatory. Since this treatment is expensive, many of these mothers may not have the money for this treatment and accept an inadequate dose, which may result in sensitization with its unfavourable consequences.

The Kleihauer technique, based on acid elution of maternal red cells, is the most widely used technique for screening and estimating the volume of FMH and for determining the need for additional doses of RHIG to prevent maternal allo-immunization (8). In Sudan, this test is mainly used for the diagnosis of haemoglobinopathies. The test is simple and inexpensive and can be routinely performed by any hospital laboratory in Sudan.

The present study was intended to evaluate the effectiveness of the standard dose, which is given to all RhD-negative mothers with RhD-positive babies since, according to our knowledge, such studies have not been previously conducted in Sudan.

Materials and methods

The design of the study is descriptive, cross-sectional and facility based. It was conducted in the El Neelain University laboratories using a total of 140 maternal blood samples collected from the Bahri Hospital, the Maternity Hospital, and the Ibrahim Malik Hospital. Samples were collected between March and September 2004. All RhD-positive mothers (n=30) and RhD-positive mothers (n=110) who were admitted for delivery to these hospitals during the study period and who delivered an RhD-positive baby were included in the study. Two ml of blood were collected from each subject (RhD-negative mothers who...
delivered RhO-positive babies in EDTA blood collection tubes.

This blood films were prepared from each blood sample, and then stained using an acid-elution cytochemical method, which was introduced by Kleihauer and colleagues (6), and examined for foetal red cells. The technique was modified by increasing the reaction time and incorporation of new methylene blue in the buffer solution used for washing the films. A positive control (cord blood) and negative control (adult male blood) were processed in parallel with the mother's blood samples.

Elution solution A was prepared by adding 7.5 µl haematoxylin to 90 % ethanol while solution B was prepared by dissolving 2.4 g of FeCl₃ in 20 ml of 2.5 mol/l HCl and bringing to one litre with distilled water. Five volumes of solution A were mixed to one volume of solution B for the working elution solution. The counter stain was prepared by dissolving 2.5 g of the aqueous eosin in one litre of distilled water.

The fresh air-dried thin blood films were fixed for 5 minutes in 80 % ethanol in a Coplin jar. Next, the slides were rinsed rapidly in distilled water and left vertically on blotting paper for about 10 minutes to dryness. The slides were then placed for 20 seconds in a Coplin jar containing the working elution solution and then rinsed in tap water before being allowed to air-dry. Finally, the slides were stained in a Coplin jar containing eosin for two minutes and washed with tap water.

The stained slides were then examined microscopically using a high dry objective lens (40X) and foetal cells were counted against ghost cells. The foetal haemorrhage was calculated as follows:

Uncorrected volume of bleed: 1800 ml / foetal cells counted (F) / adult cells counted (A). Where 1800 is the presumed maternal cell volume.

Correction for foetal volume: (F) / (1800 ml / F/A) x 1.22 (foetal cells are 22% larger than maternal cells)

Correction for staining efficiency: FMH = F x 1.09 (only 92% of the foetal cells can be detected).

Results

The Kleihauer technique was performed on a total of 140 maternal blood samples collected within 72 hours after delivery. Blood samples were categorized into two groups. The major group included the Rh-D positive mothers, which accounted for 110 of the total number. The second group included 30 RhD-negative mothers. The ages of 135 mothers were between 18 and 45, while 5 mothers were less than 17 years old. Thirty of the mothers were not circumcised and were RhD-negative, while 110 were circumcised and were RH-positive. Forty of the mothers were primigravida and 60 were delivered by caesarean section versus 100 multigravida and 80 mothers delivered normally. Ten mothers had a foetal maternal bleed of greater than 4 ml while the other 130 had foetal maternal bleeds of less than 4 ml.

<table>
<thead>
<tr>
<th>Mother's age</th>
<th>Foetal haemorrhage Total Average</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0 - 4 ml</td>
</tr>
<tr>
<td>0 - 17 Years</td>
<td>Number</td>
</tr>
<tr>
<td></td>
<td>Mean</td>
</tr>
<tr>
<td>18 - 45 Years</td>
<td>Number</td>
</tr>
<tr>
<td></td>
<td>Mean</td>
</tr>
<tr>
<td>Total</td>
<td>Number</td>
</tr>
</tbody>
</table>

Four mothers less than 17 years old had a foetal maternal haemorrhage of less than 4 ml, while only one mother in this group had more than a 4 ml foetal maternal haemorrhage. Nineteen mothers in the 18-45 year old group (6.7%) had a foetal maternal haemorrhage of more than 4 ml, versus 126 (93.3%) with less than a 4 ml bleed. However, the correlation between mother's age and amount of foetal maternal haemorrhage was not statistically significant (P=0.256).

| Table 2. Correlation between type of delivery and foetal maternal haemorrhage |
|---------------------------------|-------------------|------|
| Type of delivery | Foetal haemorrhage | Total | Average |
|--------------------|-------------------|------|
| Normal             | 0 - 4 ml          | 6    | 82    |
|                    | > 4 ml            | 58   | 8.62  |
| Caesarean          | 0 - 4 ml          | 4    | 58    |
|                    | > 4 ml            | 12.28| 6.37  |
| Total              | 0 - 4 ml          | 130  | 140   |
|                    | > 4 ml            | 14.47| 7.50  |

Six mothers (7.5%) who delivered normally were found to have more than 4 ml of the foetal maternal haemorrhage. Four mothers (6.7%) delivered by caesarean section. However, the association between the type of delivery and foetal maternal haemorrhage was not statistically significant (P=0.85).

| Table 3. Correlation between mother’s Rh blood group and foetal maternal haemorrhage |
|---------------------------------|-------------------|------|
| Mother’s Rh blood group | Foetal haemorrhage | Total | Average |
|--------------------------|-------------------|------|
| Negative                 | 0 - 4 ml          | 30   | 30    |
|                          | > 4 ml            | 0    | 0.37  |
| Positive                 | 0 - 4 ml          | 100  | 110   |
|                          | > 4 ml            | 14.9 | 7.71  |
| Total                    | 0 - 4 ml          | 130  | 140   |
|                          | > 4 ml            | 7.45 | 4.04  |

The association between the mother’s Rh blood group and foetal maternal haemorrhage failed to reach statistical significance (P=0.087). All mothers who were RhD-negative had less than 4 ml of foetal blood, while 10 (10%) of the RhD-positive mothers had more than 4 ml of foetal blood.

| Table 4. Correlation between circumcision of mothers and foetal maternal haemorrhage |
|---------------------------------|-------------------|------|
| Circumcision of mothers | Foetal haemorrhage | Total | Average |
|--------------------------|-------------------|------|
| Circumcised              | 0 - 4 ml          | 28   | 30    |
|                          | > 4 ml            | 2    | 17.2  |
| Mean                     | 0.48              | 17.2 | 3.20  |

NZ J Med Lab Science 2005
was found between baby’s weight and the amount of them (7.2%) were found to have more than
weighing 3-4 Kg. No foetal haemorrhage was detected in mothers who delivered 4-5 Kg weight babies. No statistically significant correlation was found between baby’s weight and the amount of foetal maternal haemorrhage (P=0.655).

Table 5. Correlation between mother’s gravida and foetal maternal haemorrhage

<table>
<thead>
<tr>
<th>Mother’s gravida</th>
<th>Foetal haemorrhage</th>
<th>Total</th>
<th>Average</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primigravida</td>
<td>0 - 4 ml</td>
<td>&gt; 4 ml</td>
<td></td>
</tr>
<tr>
<td>Number</td>
<td>37</td>
<td>3</td>
<td>40</td>
</tr>
<tr>
<td>Mean</td>
<td>0.42</td>
<td>25.5</td>
<td>12.96</td>
</tr>
<tr>
<td>Multigravida</td>
<td>93</td>
<td>7</td>
<td>100</td>
</tr>
<tr>
<td>Mean</td>
<td>0.56</td>
<td>10.36</td>
<td>5.46</td>
</tr>
<tr>
<td>Total</td>
<td>130</td>
<td>10</td>
<td>140</td>
</tr>
<tr>
<td>Mean</td>
<td>0.49</td>
<td>17.93</td>
<td>9.21</td>
</tr>
</tbody>
</table>

Three of 40 primigravida (7.5%) mothers were found to have more than 4ml of foetal maternal haemorrhage, compared to seven of 100 (7%) of multigravida mothers. No statistically significant correlation was found between the mother’s gravida and the amount of foetal maternal haemorrhage (P=0.917).

Table 6. Correlation between baby’s weight and foetal maternal haemorrhage

<table>
<thead>
<tr>
<th>Baby’s weight</th>
<th>Foetal haemorrhage</th>
<th>Total</th>
<th>Average</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 - 4 ml</td>
<td>&gt; 4 ml</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2-3 Kg</td>
<td>Number</td>
<td>77</td>
<td>6</td>
</tr>
<tr>
<td>Mean</td>
<td>0.48</td>
<td>16.65</td>
<td>8.57</td>
</tr>
<tr>
<td>3-4 Kg</td>
<td>Number</td>
<td>50</td>
<td>4</td>
</tr>
<tr>
<td>Mean</td>
<td>29.7</td>
<td>12.28</td>
<td>20.99</td>
</tr>
<tr>
<td>4-5 Kg</td>
<td>Number</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Mean</td>
<td>0.47</td>
<td>0</td>
<td>0.24</td>
</tr>
<tr>
<td>Total</td>
<td>Number</td>
<td>130.1</td>
<td>0</td>
</tr>
<tr>
<td>Mean</td>
<td>10.22</td>
<td>9.64</td>
<td>9.93</td>
</tr>
</tbody>
</table>

Only two of the circumcised mothers (6.7%) were found to have more than 4ml of foetal maternal haemorrhage post delivery, versus eight non-circumcised mothers (7.3%). However, no statistically significant association was found between circumcision of mothers and foetal maternal haemorrhage (P=0.909).

Table 7. Correlation between duration of pregnancy and foetal maternal haemorrhage

<table>
<thead>
<tr>
<th>Duration of pregnancy</th>
<th>Foetal haemorrhage</th>
<th>Total</th>
<th>Average</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 - 4 ml</td>
<td>&gt; 4 ml</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7 months</td>
<td>Number</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Mean</td>
<td>1</td>
<td>0</td>
<td>0.5</td>
</tr>
<tr>
<td>8 months</td>
<td>Number</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Mean</td>
<td>0</td>
<td>5.2</td>
<td>2.6</td>
</tr>
<tr>
<td>9 months or more</td>
<td>Number</td>
<td>127</td>
<td>9</td>
</tr>
<tr>
<td>Mean</td>
<td>0.52</td>
<td>15.98</td>
<td>8.25</td>
</tr>
<tr>
<td>Total</td>
<td>Number</td>
<td>130</td>
<td>10</td>
</tr>
<tr>
<td>Mean</td>
<td>0.51</td>
<td>7.06</td>
<td>3.78</td>
</tr>
</tbody>
</table>

One mother of the three who carried their babies for 8 months was found to have more than 4ml foetal haemorrhage as well as nine mothers (6.6%) carrying their babies for 9 months. All mothers who carried their babies for 7 months had less than 4ml of foetal maternal haemorrhage. However, no significant correlation was detected between the duration of pregnancy and the amount of foetal maternal haemorrhage (P=0.198).

Discussion

Sensitization of women of childbearing age has serious consequences on subsequent pregnancies. Therefore, the administration of a suitable dose of RHIG is recommended within 72 hr after delivery for the prevention of sensitization to the RhD antigen and development of RhD antibodies. In Sudan, a constant dose of 500 IU is given routinely to all RhD-negative mothers who deliver RhD-positive babies regardless of the severity of sensitization, which is closely correlated with the number of foetal cells that enter the mother’s circulation. The number of foetal cells in the maternal circulation is used for the determination of the foetal maternal haemorrhage, which in turn determines the correct dose of RhD antibodies. The routine dose of 500 IU of RHIG can only be justified when foetal maternal haemorrhage is less than 4ml. However, this dose is unsatisfactory when mothers have a foetal maternal haemorrhage of more than 4ml. As a result, mothers become sensitized and produce antibodies that cross the placenta to the foetal circulation during subsequent pregnancies and cause spontaneous abortion or hydrops fetalis. This not only results in an increase in morbidity and mortality, but also an increase in costs to the healthcare system.

Although most of the mothers who participated in this study were found to have less than 4ml foetal maternal haemorrhage (93%), the smaller portion (7%) that was found to have more than 4ml haemorrhage is considerable. This implies that the routine dose of RHIG is unsatisfactory for 7% of Sudanese mothers. Therefore, they are at a higher risk for complications. Thus, the performance of Kleihauer test within 72 hours after delivery will undoubtedly help to determine the appropriate RHIG dose to be offered to these mothers.

Moreover, the application of this technique is easy, rapid and has a low cost. It can be utilized in all hospitals in the Sudan, including regional and outlying hospitals. This conclusion is in agreement with the comments of Ghosh and Murphy (9) who determined that failure to give adequate postnatal RHIG is the most identifiable cause of

NZ J Med Lab Science 2005 8
sensitization in four of 70 cases in Scotland between 1985 and 1990.

In the present study, the correlation between the foetal maternal haemorrhage and mother's age, type of delivery and baby weight was found to be insignificant, which illustrates that mothers with variable age ranges are equally susceptible to the passage of foetal cells into the maternal circulation. Furthermore, the number of foetal cells entering the mother’s circulation does not depend on the type of delivery or the weight of the baby.

Although circumcision of mothers is known to increase the difficulty of delivery, its association with foetal maternal haemorrhage was not significant, circumcision does not contribute to the number of foetal cells in the maternal circulation.

This study also showed that the mother’s RhD blood group is not associated with the amount of foetal maternal haemorrhage. This is seen in the observed insignificant statistical difference between the mother’s RhD blood group and the number of detected foetal cells. Furthermore, duration of pregnancy was not significant for foetal maternal haemorrhage. Similarly, foetal maternal haemorrhage was not related to the mother’s gravidity; multi- or primigravida mothers had equal foetal maternal haemorrhages.

In conclusion, the number of RhD-negative mothers in Sudan with more than 4ml foetal maternal haemorrhage (7%) is considerable. Determination of foetal maternal haemorrhage after delivery is mandatory in order to determine the appropriate dose of RHIG. Foetal red cells entering the mother’s circulation was not associated with the mother’s age, duration of pregnancy, circumcision, baby’s weight, mother’s gravidity, RhD blood group, nor type of delivery.

It is recommended to perform a Kleihauer technique as a routine test to determine the amount of foetal cells in the mother’s circulation within 72 hours after delivery on all RhD-negative mothers delivering RhD-positive babies. The RHIG dose should be based on the amount of foetal maternal haemorrhage.

References

Correspondence: Mohamed Siddig Mohamed Ali. Faculty of Medical Laboratory Sciences, AI Neelain University, P.O. Box 12702, Khartoum, Sudan. E-mail: mohdaru@hotmail.com
HSIG questionnaire

Journal - Blood Reviews - Volume 17, Number 4, December 2003, Pages 209-213 Congenital Neutropenia

Questions

Q.1 Define congenital neutropenia
Q.2 Name 2 underlying clinical features seen in congenital neutropenic children
Q.3 State 3 causes of leukaemic transformation
Q.4 What is the most common characteristic of cyclical neutropenia?
Q.5 G-CSF treatment increases the amplitude of monocyte, lymphocyte and neutrophil oscillation, shortens both the cycle length and duration of monocytopenia - True/False
Q.6 Shwachman-Diamond Syndrome (SDS) is a sex linked disease characterized by skeletal abnormality - True/False
Q.7 Name 4 clinical features associated with SDS
Q.8 Glycogen storage disease type results from deficiency of the ................. enzyme (GSD16)
Q.9 GSD16 transports glucose-6-phosphate into the endoplasmic reticulum for conversion to glucose and phosphate - True/False
Q.10 State 4 rare causes that contribute to congenital neutropenia
Q.11 Myelokathexis is an autosomal dominant moderate to severe neutropenia with an unusual morphology - True/False
Q.12 ................. is characterized by unfortunate combination of neutropenia and cardiomyopathy
Q.13 Pearson's syndrome manifests as ................. with vacuolization of bone marrow precursors, ................. and variable degrees of neutropenia and thrombocytopenia

Answers on page...20
Microbiology SIG Meeting 2005

Wellington
Friday 27th - Saturday 28th May
For Provisional Programme Details and Accommodation Options, visit the NZIMLS website www.nzimls.org.nz
Registration forms available on-line soon, and from your Microbiology Dept.

Please Submit Papers
We are looking for 10min proffered papers to be presented on Saturday 28th May. Deadline for offering papers is April 30th.
Please send your expression of interest in giving a paper, title and general topic description to Jenny Bennett at jenny.bennett@esr.cri.nz or Joan Byrne at joan.byrne@ccdhb.org.nz
Inheriting the beauty of performance

The latest member of the SYSMEX X-family haematology analyzers is the new XT-2000i. It inherits the successes of its big brother, the XE-2100, so you can really believe in its performance.

**The new XT-2000i haematology analyser**

- Employs highly advanced Fluorescence Flow Cytometry
- Features most of the parameters and channels of the XE-2100
- Conforms to today's hospital budgets with its compact size and economical approach
- Offers high-quality results with clinically significant information and excellent efficiency for the separation of normal and pathological samples
- Features the famous system stability and ease of operation – characteristics of all SYSMEX instruments

**SYSMEX XT-2000i**
Inherited Performance
# Index to Volume 58, 2004

**Fellowship treatises**

Human parvovirus B19: a review of the disease and diagnostic tools  
*Sheryl Young* .......................................................... 2-8

Method evaluation for methylmalonic acid: use for assessing vitamin B12 deficiency  
*Christine C Lever* .................................................. 27-39

Monoclonal gammopathies: a laboratory perspective  
*Rachael L Findlater* ................................................ 41-48

The implications of methicillin resistant *Staphylococcus aureus* and why we should keep it out of hospitals  
*Ann Thompson* ....................................................... 49-56

Neonatal alloimmune thrombocytopenia. A review  
*Susan Evans* .......................................................... 74-82

**Articles**

Drugs of abuse testing  
*Ross Hewett* .......................................................... 9-12

Faecal occult blood testing: guaiac vs immunochemistry: which method should we use?  
*Christian Sies, Christopher Florkowski, Peter George* ............ 58-60

Psuedothrombocytosis caused by white blood cell fragments  
*Kevin A Taylor* .......................................................... 83-84

The diagnosis and treatment of prostate cancer: will commonsense prevail?  
*Brett Delahunt, David S Lamb, John N Nacey* ........................ 86-89

**Scientific letter**

Factor VIII binding assay - one year on  
*David M Patterson, Campbell R Sheen* ................................ 90

**TH Pullar Memorial Address**

Outside, looking in  
*R Siebers* .......................................................... 71-73

**Editorials**

Geopolitical intrusion on editorial decisions  
*Rob Siebers, Ann Thornton* ........................................... 26

Prostate specific antigen: to screen or not to screen  
*R Siebers* .......................................................... 70

**Book review**

The Urinary Sediment - An Integrated View (2nd edn) by Giovanni Fogazzi  
Reviewed by *Julie Vincent* ............................................. 20

**Reports**

Abstracts by New Zealand presenters at the South Pacific Congress, Brisbane, 2003 ........................................ 14-16

President's report  
*Chris Kendrick* ............................................................. 91-92

Conference report  
*Robin Allen* ............................................................. 93-94

Abstracts from the NZIMLS ASM, Hamilton, August 2004 ........................................ 98-115

**Author index**

Allen R.......................................................... 93

Delahunt B......................................................... 86

Evans S.......................................................... 74

Findlater RL......................................................... 41

Florkowski C........................................................ 58

George P.......................................................... 58

Hewett R.......................................................... 9

Kendrick C......................................................... 91

Lamb DS.......................................................... 86

Leaver CC......................................................... 27

Nacey JN.......................................................... 86

Patterson DM....................................................... 90

Sheen CR.......................................................... 90

Siebers R.......................................................... 26

Siebers R.......................................................... 70

Siebers R.......................................................... 71

Sies C.......................................................... 58

Taylor KA......................................................... 83

Thompson A....................................................... 49

Thornton A......................................................... 26

Vincent J.......................................................... 20

Young S.......................................................... 2
Introducing UniCel® DxC Synchro® Clinical Systems.

Capacity, performance, simplicity. As never before, they all come together in Beckman Coulter’s new generation of analysers: the UniCel® DxC 600 and 800 systems.

The capacity of UniCel systems takes your lab to a higher level, with 65 or 70 on-board, random-access reagents, covering a broad menu of more than 100 tests. You also get exceptional throughput of 1440 tests per hour on the DxC 800 and 990 tests per hour on the DxC 600—ample capacity to meet your needs.

When it comes to performance, the DxC 800 delivers 11 critical care tests in a blazing 42 seconds, giving real meaning to the word ‘stat.’ What’s more, UniCel systems use next-generation technology to maximize reliability—delivering performance that, for practical purposes, is almost non-stop.

And few systems are simpler to operate. UniCel systems have automated procedures and on-board action logs to take maintenance demands to a new low. The enhanced touch screen interface provides built-in trouble-shooting. Plus, only Beckman Coulter offers closed-tube sampling on its chemistry systems—making it easier than ever to improve your lab’s productivity and safety.

The growing UniCel family offers a system for every laboratory. To find out which one blends best with your chemistry, call Beckman Coulter today, or visit beckmancoulter.com.

For more information:

Australia: P 1800 060 880 New Zealand: P 0800 442 346
E sales_aust_nz@beckman.com W www.beckmancoulter.com

© Copyright 2005 Beckman Coulter, Inc.
Because *rapid* diagnosis leaves no margin for compromise...

**Troponin I**

**CK-MB**

**hCG**

**Myoglobin**

**D-Dimer** NEW

Phone 0800 284 825
Fax 0800 284 835
Web www.biomerieux.co.nz

---

**WORK IN THE UK.**

- UK Welcome Service
- Holiday Pay
- Travel Benefit Scheme
- Discount Car Hire

**A JOB FOR YOUR LIFE.**

BMG Associates is the New Zealand division of Reed Health, one of the leading recruitment agencies for skilled health professionals in the UK. We have temporary and permanent positions available for qualified medical scientists across England, Wales and Scotland.

For more information contact BMG Associates today or register online.

Freephone: 0800 803 854
Email: info@bmgassociates.co.nz
www.bmgassociates.co.nz

United Kingdom • Ireland • Australia • New Zealand
Where: Rydges Rotorua, 272 Fenton Street, Rotorua
When: Saturday 21 May 2005
Registration: 8.45 - 9.30am, Morning tea and coffee on arrival.

Proffered papers for seminar: 9.30am to 5.00pm

Costs:
Registration:
Members NZIMLS: $75.00,
Non members NZIMLS: $112.50
Student registration available on request.
Includes morning and afternoon tea, lunch and access to papers at seminar.
Seminar Dinner: $50.00 inclusive of GST

Accommodation: Rydges, 272 Fenton Street, Rotorua
Single/Twin/Double Room: $130 + GST
For accommodation bookings, please contact Rydges Rotorua direct on 07 349 0099

For alternative accommodation venues, please contact
Tourism Rotorua
Debbie Woolton
Phone: 07 343 1732
Fax: 07 343 1740
BSIG Seminar
Registration Form

Rydges Rotorua, 272 Fenton Street, Rotorua
Saturday 21 May 2005

Registration Fees

$75 NZIMLS Member  
$112.50 NZIMLS Non-member  
$50.00 Seminar Dinner  

Total: $..............

On-line Registration available: www.nszimls.org.nz

Please make cheques payable to NZIMLS-BSIG Seminar

Any special dietary requirements:


Please return this form and payments by 1 May 2005 to:

Fran van Til
Executive Officer
NZIMLS
P.O. Box 505
Rangiora
BSIG Seminar
Proffered Papers
Rydges Rotorua
Saturday 21 May 2005

<table>
<thead>
<tr>
<th>Presenter:</th>
</tr>
</thead>
<tbody>
<tr>
<td>First Name: ________________________________</td>
</tr>
<tr>
<td>Surname: ________________________________</td>
</tr>
<tr>
<td>Laboratory / Organisation: ________________________________</td>
</tr>
<tr>
<td>Address: ________________________________</td>
</tr>
<tr>
<td>Contact Phone Number: __________________ Fax: ___________</td>
</tr>
<tr>
<td>Mobile: __________________ Email Address: __________________</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Title of Presentation:</th>
</tr>
</thead>
<tbody>
<tr>
<td>________________________________</td>
</tr>
<tr>
<td>________________________________</td>
</tr>
<tr>
<td>________________________________</td>
</tr>
</tbody>
</table>

Abstract Provided Yes ☐ No ☐ Oral presentation ☐ Poster ☐

<table>
<thead>
<tr>
<th>Equipment Required:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overhead projector ☐</td>
</tr>
<tr>
<td>Slide Projector ☐</td>
</tr>
<tr>
<td>Data show (Powerpoint) laptop provided ☐</td>
</tr>
<tr>
<td>Other: ________________________________</td>
</tr>
</tbody>
</table>

Please return this form to: Martin Hampson, Diagnostic Rotorua, P.O.Box 481 Rotorua
Tel: 07 348 2106, Mob: 027 4832 969, Fax: 07 348 2277. Email: martinh@diagroto.co.nz
Pathologists,  
Medical Lab. Scientists,
We have permanent and temporary employment options in New Zealand and Australia. Contact us and talk to qualified staff. We provide a professional, confidential, no obligation and free service to you, the candidate.

For further information contact us: info@healthstaffspecialists.com

For further employment opportunities visit: www.healthstaffspecialists.com

Health Staff Specialists Ltd, is a member of the RCSA, the Recruitment Consulting Society for Australia & New Zealand.

Advertisers in this issue

Abbott Diagnostics .............................................................. IFC
Beckman Coulter ................................................................. 15
Biomerieux ................................................................. 16
BMG Associates ................................................................. 16
Health Staff Specialists ............................................................... 20
MedBio ............................................................... BCB
Medica Pacifica ................................................................. 9
Roche Diagnostics ............................................................... OBC & 13

Med-Bio Journal Award

Med-Bio offers an award for the best article in each issue of the New Zealand Journal of Medical Laboratory Science. All financial members of the NZIMLS are eligible. The article can be an original, review or technical article, a case study or a scientific letter. Excluded are editorials, reports, or fellowship treatises. No application is necessary. The Editor and Deputy Editor will decide which article in each issue is deemed worthy of the award. If, in their opinion, no article is worthy, then no award will be made. Their decision is final and no correspondence will be entered into.

Winner of the November 2004 issue was Kevin Taylor, Canterbury Health Laboratories, for his article "Pseudothrombocytosis caused by white blood cell fragments"
Finding H. pylori Has Never Been This Easy!

Monoclonal Antibody Rapid Stool Antigen Test

Sensitivity = 96%*
Specificity = 97%*
ppv = 96%*
npv = 97%

* Helicobacter Vol. 9, No. 4, 2004, pp347 - 367

- A non-invasive faecal antigen testing approach
- Unique immunoassay amplification technology resulting in clear-cut positive and negative results
- Recommended for routine screening
- Recommended method for monitoring treatment and eradication
- Suitable for pediatric diagnosis
- Cost-effective alternative to invasive methods
- Ultra-sensitive, rapid, and easy to perform
- Improved performance compared to conventional fecal antigen tests
- Automated protocols available

* Reference: European Helicobacter Study Group Guidelines, September 2000

For more information or an evaluation, contact Med-Bio Limited. Phone: 0800 633 246 E-mail: custserv@medbio.co.nz
Welcome to the cobas family

Roche Diagnostics customers have always appreciated the quality of Roche’s products and services. Roche is now introducing the cobas family of Clinical Diagnostics products to make this service even better.

New generation cobas inherits the very best from its previous product generations. Roche seamlessly combines this quality with knowledge and understanding of customer needs. cobas will be the single umbrella brand that unifies all our systems and reagents into a single, easy to understand product family, with a standardised “look and feel”.

cobas is designed to grow with you - a family of product and service offerings for Clinical Chemistry, Immunoassay, Near Patient testing, Molecular diagnostics and Haematology.

Welcome to the world of cobas

Diagnostics

Over 50 years of innovation