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## NEW ZEALAND JOURNAL OF MEDICAL LABORATORY SCIENCE

ISSN 0028-8349

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# **3RD SOUTH PACIFIC CONGRESS ON** MEDICAL LABORATORY SCIENCE



## AUGUST 26 - 30, 1991 Auckland, New Zealand

## Invitation to attend

It is my very great pleasure, on behalf of the organising committee, to invite you to the 3rd South Pacific Congress in Auckland from 26th to 30th August 1991.

This meeting is the joint annual scientific meeting of NZIMLS and AIMLS and for this congress we have been joined by the NZ Society of Haematology. As you can see from the outlined programme, there is a wide variety of scientific and social activities. We have a world acclaimed venue, speakers of the highest calibre and a social programme to satisfy the most avid conference goer.

To make this Congress a success all that is needed is your participation. Registration forms were published in the March 1991 issue of the NZJ Med Lab Science or are available from:

South Pacific Congress 1991. Guthreys Pacific Ltd, P.O. Box 22-255, Christchurch. Telephone (03) 668-711 Fax (03) 790-175

**Dennis Dixon-Mclver** Chairman, Organising Committee **3rd South Pacific Congress** 

## **Tentative Programme**

Since last published, the Society of Haematology programme has been incorporated within the main Scientific programme.

## **Monday 26 August**

- Possible workshops and User Group meetings (held offsite).

## **Tuesday 27 August**

- Immunohaematology workshop on Diagnosis of Autoimmune Haemolytic Anaemia - Dr Lawrence Petz.
- Workshops and User Group meetings.
- NZIMLS Annual General Meeting (afternoon).
- Wine and Cheese Icebreaker (evening).

## Wednesday 28 August

- South Pacific Congress on Medical Laboratory Science Opening Ceremony and Address.
- Evolution of M.L.T. in Developing Countries (Monica Cheesbrough).
- General Forum on Medical Ethics.
- Concurrent Fora

Biochemistry	: Haematolog
Immunology	: Microbiology
Radioassay (Dr Jan Stockigt)	

- Evening Meal and entertainment - NZ Expo Centre.

## **Thursday 29 August**

- Plenary Session AIDS Prof. D. Sutherland (WHO), Prof. Ron Penny (Australia).
- Concurrent Fora
  - Haematology (Dr Ken Bradstock, Dr A.H. Goldstone)
  - : Immunology
  - : Immunohaematology (Dr Lawrence Petz)
  - : Biochemistry (Dr Garth Cooper)
  - : Education (Peter Bruhn)
  - : Microbiology
- Conference Dinner.

## Friday 30 August

- Plenary Session Recombinant DNA Technology -Dr Tom Gillis.
- South Pacific Forum examining the problems and progress of M.L.T. in the Pacific Islands.
- Concurrent Fora
  - : Microbiology
  - : Immunohaematology : Management
- : Biochemistry : Haematology
- : Immunology
- Closing Ceremony.

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PROGRAMME OVERVIEW WEDNESDAY 28 AUGUST 1991							
	0830-1015		1100-1230		1330-1500		1530-1700
ASB THEATRE	0830-0900 Registration 0900-0945 Maori Welcome 0945-1015 Opening Address		-		-		-
KUPE ROOM	-		1100-1145 Evolution of M.L.T. M. Cheesborough 1145-1230 Overview of Medical Ethics Pauline Kingi		GENERAL FORUM Medical Ethics Ray Naden Carol Whitfield		SYMPOSIUM Malignancy Bruce Baguley Vernon Harvey Graham Finlay (BIOCHEMISTRY)
HAURAKI ROOM	-	ING TEA	-	UNCH	SYMPOSIUM Transplantation Immunology (IMMUNOLOGY)	NOON TEA	SYMPOSIUM Antiphospholipid Antibody Syndrome (IMMUNOLOGY) (HAEMATOLOGY)
KAIKOURA ROOM	-	MORN	-		ORAL PRESENTATIONS Changing Patterns (MICROBIOLOGY)	AFTER	ORAL PRESENTATIONS Changing Patterns (MICROBIOLOGY)
GOODMAN- FIELDER- WATTIE ROOM	-		SYMPOSIUM New Therapies in the treatment of Leukaemia		SYMPOSIUM Chronic Lymphocytic Leukaemia in the Nineties		FREE COMMUNICATION
AEPB ROOM	-		-		SYMPOSIUM Dr J. Stockigt		ORAL
					(RADIOASSAY)		(RADIOASSAY)

PROGRAMME OVERVIEW - THURSDAY 29 AUGUST 1991								
	0830-1015		1100-1230		1330-1500	1	1530-1700	
ASB THEATRE	-		-		-		-	
KUPE ROOM	PLENARY SESSION AIDS D. Sutherland WHO Ron Penny AUS Mark Thomas NZ		CONTINUING EDUCATION Update on Diagnosis and Classification of Leukaemia (SOC. HAEM)		CONTINUING EDUCATION Update on Diagnosis and Classification of Leukaemia (SOC. HAEM)		CONTINUING EDUCATION Update on Diagnosis and Classification of Leukaemia (SOC. HAEM)	
HAURAKI ROOM	-	VING TEA	SYMPOSIUM Mucosal Immunity and Allergic Response (IMMUNOLOGY)	UNCH	ORAL PRESENTATIONS Pathogens Revisited (MICROBIOLOGY)	NOON TEA	ORAL PRESENTATIONS General Papers (IMMUNOHAEM.)	
KAIKOURA ROOM	CONTINUING EDUCATION Update on Diagnosis and Classification of Leukaemia (SOC. HAEM)	MORN	SYMPOSIUM Diabetes Garth Cooper Bob Elliott David Scott (BIOCHEMISTRY)		SYMPOSIUM On Job Assessment Peter Bruhn Andrew Thakurdas (EDUCATION)	AFTERI	SYMPOSIUM AIDS Dave Sutherland (IMMUNOLOGY)	
GOODMAN- FIELDER- WATTIE ROOM	SYMPOSIUM The Pacific Way of Teaching (SOUTH PACIFIC)		SYMPOSIUM Comparison of Training Systems Peter Bruhn Andrew Thakurdas (EDUCATION)		ORAL PRESENTATIONS Immune Haemolysis associated with Transplantation Lawrie Petz (IMMUNOHAEM.)		ORAL PRESENTATIONS	
AEPB ROOM	-		SYMPOSIUM Mechanics of Transfusion (IMMUNOHAEM.)		(HISTOPATHOLOGY)		ORAL PRESENTATIONS Extra Lab Testing (BIOCHEMISTRY)	

	PROGRAM	MEC	OVERVIEW — FF	RIDA	Y 30 AUGUST	1991	
	0830-1015		1100-1230		1330-1500		1530-1700
ASB THEATRE	-		-		-		-
KUPE ROOM	PLENARY SESSION Recombinant DNA Technology - An Overview Tom Gillis		SYMPOSIUM DNA Diagnostics - the wave of the future? (MICROBIOLOGY)		SYMPOSIUM DNA Diagnostics - the wave of the future? (MICROBIOLOGY)		CLOSING CEREMONY Details to be Advised
HAURAKI ROOM	-	VING TEA	ORAL PRESENTATIONS	UNCH	SYMPOSIUM Autoimmune Haemolytic Anaemias Lawrie Petz (IMMUNOHAEM.)	NOON TEA	-
KAIKOURA ROOM	SYMPOSIUM Current Issues in the Management of Lymphoma (SOC. HAEM)	MORN	ORAL PRESENTATIONS Management Issues in Bone Marrow Transplantation (HAEMATOLOGY)		SYMPOSIUM Coping With Change (MANAGEMENT)	AFTER	-
GOODMAN- FIELDER- WATTIE ROOM	-		SYMPOSIUM Teaching Skills (SOUTH PACIFIC)		SYMPOSIUM Update on Relevant Methods of Technology (SOUTH PACIFIC)		-
AEPB ROOM	FREE COMMUNICATION (HAEMATOLOGY)		(HAEMATOLOGY		SYMPOSIUM Floating Seminar Technical Aspects ?? Harbour Cruise 1030-1500 (IMMUNOLOGY)		-

N.Z.J. Med. Lab. Science, 1991



3RD SOUTH PACIFIC CONGRESS ON MEDICAL LABORATORY SCIENCE



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> Mrs Dinah Parr Mycology Unit Auckland Hospital AUCKLAND

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South Pacific Congress, Guthrey's Convention Planners, P.O. Box 22-255, Christchurch.

NZJ Med Lab Science. 1991; 45(2): 45-47.

## Giardiasis in New Zealand, Results of a Laboratory Based Survey. Natalie K Walker MSc<sup>1</sup>, Nicholas A Wilson MB ChB MCCM<sup>2</sup>, Desmond G. Till COPMLT MASM<sup>1</sup>.

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Address for correspondence: Natalie K Walker, Public Health Laboratory, NZCDC, PO Box 50-348, Porirua.

#### Abstract

A survey of medical laboratories throughout New Zealand was carried out in order to accumulate information concerning the prevalence of giardiasis throughout the country and laboratory testing methodology. Testing for *Giardia lamblia* in faecal specimens was found to be routinely performed in 46% of laboratories and performed only at the request of a practitioner in 54%. Specimen concentration was routinely performed in 67% of laboratories. The most common procedure for analysis of faecal specimens for *Giardia lamblia* was a direct wet film preparation with subsequent staining using iodine or trichrome (44%). Immunofluoresent Assays (IFAs) are not widely used for testing.

Of the faecal specimens tested over a period of three months 2.7% were found to be positive for *Giardia lamblia* (N = 839). This would suggest that there are at least 3,356 cases of giardiasis per year in New Zealand.

Data linking positive cases with the type of water supply offer no support to the hypothesis that water may be a major route of transmission of giardiasis in New Zealand.

#### Key Words

Giardiasis, laboratory survey, testing methodology, water transmission.

#### Introduction

Although *Giardia lamblia* is known to have been identified in New Zealand since the 1940s, there is virtually no information on the role of *Giardia lamblia* as a cause of disease in this country. Since it is not a notifiable disease in New Zealand, the only information available is limited to positive cases reported on an intermittent basis by some laboratories as published in the Communicable Disease New Zealand (CDNZ). This data alone suggests that probably over two thousand cases are tested positive by laboratories annually. A larger number of cases may be asymptomatic and therefore remain undetected in the population.

Concern over giardiasis is growing in the general population, along with increasing levels of media coverage. In April 1990, *Giardia lamblia* cysts were identified in several sites of the Kakanui water supply system (a small South Canterbury town). The water supply was examined because of human cases of giardiasis in this population. Another chlorinated (but unfiltered) water supply has been more recently documented as being involved in a probable waterborne outbreak of giardiasis in Whangarei (1).

The chlorination-only treatment used for these water supply systems is similar to many others around New Zealand. Very few water supplies in New Zealand have in fact been tested for *Giardia lamblia*. This is mainly due to the cost and time involved and the lack of laboratories that have the facilities to test water. Such a situation raises the possibility that contamination by this organism could exist in many of New Zealand's water supplies without our knowledge.

In view of these concerns a survey was conducted in an attempt to accumulate some general information concerning the prevalence of giardiasis in New Zealand and the testing methodology used by medical laboratories throughout the country for the detection of the parasite.

## METHODS

All of the seventy-one medical laboratories in New Zealand at this time were asked to complete a questionnaire relating to methodological aspects of testing for giardiasis. Also included were questions relating to the number of tests performed and number of positive results obtained in the months of May, June and July 1990. The names of the doctors involved with positive cases were requested so as to locate the type of water supply existing in the doctor's locality and therefore presumably the patient's locality. This was an attempt to determine if there might be any association between the type of water supply and the number of cases. A comparison group of doctors' addresses was randomly selected from telephone directories with the number selected being weighted according to the population size in the areas covered by the directories. No practitioners or patients were contacted in this study.

Information on the various types of water supplies and associated treatment was obtained by contacting city and regional councils and by referring to a number of booklets on water supply statistics (2,3,4). Water supplies were then graded on the following scale of one to five according to the decreasing theoretical likelihood of *Giardia lamblia* cysts being present.

- 0 = No water treatment
- 1 = Chlorination only
- 2 = Filtration and chlorination
- 3 = Filtration, flocculation and chlorination
- 4 = Flocculation, filtration, sedimentation, and chlorination
- 5 = Artesian water

#### Results

There was a 100% response rate to the questionnaire with eight (12%) of the responding laboratories reporting that they sent all microbiology specimens to larger or private laboratories for analysis. Another six (8%) laboratories were unable to supply data due to a variety of reasons, the main one relating to the capacities of their information systems. Four of these were from small towns and two were moderately large laboratories in two cities. Although there was a 100% response rate to the questionnaire, not all questions in the questionnaires were answered.

The fact that giardiasis is not a notifiable disease also meant that detailed records have not been kept in some laboratories and thus for several laboratories only approximations of the number of specimens tested for *Giardia lamblia* have been made.

#### Testing Methodology

Testing for *Giardia lamblia* on faecal specimens was found to be routinely performed in 25 (46%) of the laboratories and performed only at the request of a practitioner in 30 (54%) of the laboratories. Specimen concentration was routinely performed in 36 (67%) of the laboratories and not performed in 18 (33%). It was noted that if testing for *Giardia lamblia* was carried out at the request of a practitioner it was eight times more likely that specimen concentration would also be carried out.

The most common laboratory procedure used for analysis of faecal specimens for *Giardia lamblia* was a direct wet film preparation with subsequent staining, using iodine or, more commonly, trichrome (Table 1). The next most frequent procedure was staining alone (31%) followed by the use of only wet film preparations (19%). No laboratories used the IFA procedure only for *Giardia lamblia* testing although it was used in 4% of the laboratories in conjunction with wet film preparations. At one laboratory the IFA method was carried out on faecal specimens only if specifically asked for, and only one laboratory used all available procedures. There was no significant relationship found between the use of specimen concentration and testing methodology.

Table 1. Laboratory procedures used to diagnose Giardiasis

Laboratory	Concer	ntration ormed	Total		
Flocedule	No.	⁰⁄₀	No.	%	
Wet film + Stained	16	44	24	44	
Stained	14	39	17	31	
Wet film	5	14	10	19	
Wet film + IFA	0	0	2	4	
All	1	3	1	2	
Total	36	100	54	100	

#### Diagnoses of Giardiasis

The results obtained found that a total of 31,239 specimens were tested by the laboratories involved, of which 2.7% were positive for giardiasis (N = 839). In terms of individual laboratories, the average results were 253 negative specimens and 6 positive specimens per month per medical laboratory. The number of negative and positive specimens did not significantly differ over the three months surveyed.

#### Relationship Between the Diagnosis of Giardiasis and Water Treatment

The only large area for which virtually all laboratories provided data of numbers of specimens and the doctors' names attached to these, was the South Island (with the exception of one small town laboratory). The type of water supply associated with these positive cases was usually artesian, though this was also the major supply in the randomly selected areas as shown in Table 2.

**Table 2.** Water supply associated with estimated location of cases with positive stool specimens and for randomly selected areas (For the South Island only).

		Areas With Cases*		Areas With Randomly Cases* Selected Area			lomly I Areas†
Water Supply	Code	No.	%	No.	%		
Nil	0	5	4	11	8		
Chlorination	1	19	13	28	19		
C + F	2	15	10	28	19		
C + F + FI	3	2	1	3	2		
C + F + FI + S	4	8	6	1	1		
Artesian	5	95	66	73	51		
Total		144	100	144	100		

Notes:

\*As determined by the address of the doctor submitting the specimen.

† Based on randomly selected doctor's address (weighted by population).

- C = Chlorination
- F = Filtration
- FI = Flocculation
- S = Sedimentation.

When the mean value of the codes given to the types of water supply used were compared, it was found that the mean was higher for water supplies associated with the positive specimens, ie, 3.9 compared to 3.2 for the controls (significantly different using the Kruskal-Wallis test for two groups, p=0.003). This difference was no longer significant, however, when rural and urban areas (over 20,000 population) were examined separately.

It was found that 81.9% of positive specimens were from designated urban areas as compared with 66.7% of the randomly chosen areas being urban.

This amounted to a significantly higher rate of positive specimens from urban areas compared to rural areas by a factor of over two (Odds Ratio = 2.27, 95% Confidence Intervals = 1.26, 4.10).

#### Discussion

Giardiasis is normally diagnosed by faecal examination. In active diarrhoea a thin wet film preparation will identify trophozoites, while normal or soft stool is more likely to contain cysts only (5). Detection using a direct wet film preparation with iodine or methylene blue is the simplest and quickest method although the frequent inability of this test to identify cysts reduces its worth. Trichrome, although a longer procedure, is one of the better identification methods available especially if coupled with some form of concentration procedure (6). Unfortunately these results indicate that concentration and trichrome staining tend to be used only when specifically requested by a practitioner, if foreign travel is mentioned or if a test for Giardia lamblia is specifically asked for. The number of procedures used and their accuracy greatly increase the chance that cysts will be identified. The fact that the microscopy methods used for the detection of Giardia lamblia cysts do not detect all cases means that the number of positive specimens noted in this study are an underestimate of actual numbers.

Immunoflorescent assays (IFAs) are considered significantly more accurate than other microscopy procedures. Other methods of detection can be time consuming and fatiguing due to the poor visual contrast between cysts and contaminants, and the low number of cysts found amongst debris and microorganisms (7). The use of the fluorescein-labelled monoclonal antibody means that cysts are clearly visible and therefore identification is less tiresome. Due to the high cost of this relatively new test, few laboratories can invest in the materials and equipment required for the procedure, as reflected in the results. One laboratory that did use this test commented that an IFA was only carried out at the request of a practitioner.

Extrapolating the positive number of results obtained for the three month period to a year suggests a total of 3,356 cases per year in New Zealand. It is likely however that seasonal trends in giardiasis occur (8), which would suggest a higher total.

Also information concerning the number of positive and negative specimens is, unfortunately, somewhat inaccurate due the fact that multiple specimens may be included in the figures. Traditionally when patients are suffering from giardiasis three faecal cultures are requested, due to the intermittent excretion of the cysts. It is easier for some laboratories to access data on the number of specimens tested rather than the number of cases, so in this sense results may be overestimated by at most, a factor of three. A range of other factors however would tend to suggest that laboratory positive results represent only a small fraction of the total number of cases present in the community. These factors include the following:

- a) The high proportion of cases that are asymptomatic (9,10).
- b) Many patients may not seek medical advice if symptoms are mild.
- c) Some practitioners may treat patients on symptoms alone rather than requesting faecal specimens.
- d) Some patients may not supply faecal specimens when these are requested.
- e) Certain laboratory isolation techniques may be inadequate to identity *Giardia lamblia*.

Due to all these factors any efforts to predict the number of giardiasis cases in New Zealand is likely to be a large underestimate. Indeed it is conceivable that the total number of cases estimated here for a year (3,356) would need to be multiplied several times to produce the true number of symptomatic cases alone.

Water is a well documented source of giardiasis in many countries. An estimated 69% of waterborne *Giardia lamblia* outbreaks occurring in the United States from 1965-1984 were in community water supply systems. Of these community outbreaks, 74% involved inadequately treated or chlorinated water only (11). The effectiveness of chlorine in inactivating *Giardia lamblia* is dependent on pH and temperature, water turbidity and the contact time of the chlorine (11,12,13). With so many factors involved there is a high probability of treatment failure occurring. Chlorine levels used in routine disinfection of municipal drinking water are not effective against *Giardia lamblia* cysts. Jarroll (1988) considers a concentration of 3 ppm to be cystacidal. Standard chlorine levels in New Zealand drinking water are however approximately 0.5 ppm.

Current research indicates that *Giardia lamblia* cysts can be effectively removed and inactivated in water by using a combination of filtration and disinfection (14). Most forms of filtration such as diatomaceous earth filtration, slow sand filtration and coagulation filtration can remove 99% or more *Giardia lamblia* cysts if the processes are operated correctly and adequately maintained (11,14,15).

The results obtained in the part of this study that examined numbers of positive specimens diagnosed by type of water supply, require cautious interpretation due to the various methodological problems in the approach used. The major biases are probably the lower rate of symptomatic patients attending doctors and of doctors requesting laboratory specimens in rural areas. This could well explain the 2.3-fold higher rate associated with the positive specimens in urban areas. The apparent association between a higher grading of water supply (according to the arbitrary scale used) and number of positive specimens for giardiasis is also probably due to these same biases. Nevertheless these limited results offer no support to the hypothesis that water is the major route of transmission of giardiasis in New Zealand. Indeed the association with urban areas may provide some support to the view that person to person contact is more important. This is presuming that in urban centres there is more interpersonal contact, eg, a greater proportion of children attending childcare centres. Okell and Wright (1990) also considered this after an in-depth study in the eastern Bay of Plenty area of New Zealand which found high numbers of positive specimens despite fully treated or artesian water (8).

#### **Acknowledgments**

The authors acknowledge the staff of the medical laboratories throughout New Zealand for their assistance and support in conducting this investigation.

This paper is published with the authority of the Director General of Health; views expressed are not necessarily those of the Department of Health.

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## PACIFIC PARAMEDICAL TRAINING CENTRE ANNIVERSARY FUNCTION

The Pacific Paramedical Training Centre (PPTC) has recently celebrated its 10th year of operation. This occasion was marked by a luncheon held at the Beehive, Parliament Buildings, Wellington.

Formally established in 1980, the Pacific Paramedical Training Centre is based at Wellington Hospital and provides training in appropriate medical laboratory technology for lab technicians from the Pacific and Asian Regions.

First established in 1972 by a Red Cross initiative to assist with the development of blood transfusion services for Pacific Island countries, the Centre has grown from very modest beginnings to become an important contributor to the health services of the Pacific Islands, and as a significant part of New Zealand's Development Aid Programme in that area.

The Centre is an excellent example of co-operative effort with a partnership comprising the Wellington Area Health Board, New Zealand Ministry of External Relations and Trade, the New Zealand Red Cross Society, the World Health Organisation and the New Zealand Institute of Medical Laboratory Science.

Since its inception, over 200 trainees have attended courses and, in 1990, the Centre was recognised as a centre of excellence. The P.P.T.C. has been invited to become a Collaborating Centre of the World Health Organisation and is responsible for Medical Laboratory Teaching programmes and quality assurance programmes in hospital laboratories throughout the Pacific Region.

For more information on the PPTC turn to "The Pacific Way."



10th Anniversary Celebration of the P.P.T.C.

## Stability of Whole Blood for Plasma Sodium and Potassium Measured With Ion Specific Electrodes.

## Robert W.L. Siebers, FNZIMLS, MIBiol.

## Department of Medicine, Wellington School of Medicine. Address for Correspondence: P.O. Box 7343, Wellington South, Wellington.

## Abstract

Heparinised whole blood samples left standing at room temperature for up to three hours show an increase in plasma Na<sup>+</sup> and K<sup>+</sup> concentrations when measured by direct reading ion specific electrodes. Plasma Na+ concentrations measured at 0 h, 1 h, 2 h and 3 h were (x mmol/L±1 S.D., n = 14) 139.9  $\pm$  1.56, 140.6  $\pm$  0.78, 141.0  $\pm$  0.78 and 141.6  $\pm$ 0.84 respectively (p < 0.05 at 2 h and 3 h compared to 0 h). Plasma K<sup>+</sup> concentrations measured at the corresponding times were 3.63  $\pm$  0.53, 3.68  $\pm$  0.55, 3.79  $\pm$  0.52 and 3.93  $\pm$ 0.55 respectively (p < 0.05 at 1 h, 2 h and 3 h compared to 0 h). It is hypothesised that loss of bicarbonate upon standing results in the observed increase of measured plasma Na\* and K<sup>+</sup> concentrations as these cations can form complexes with bicarbonate which are not quantitated as only free ions are measured by ion specific electrodes. Ion specific electrode analysis of whole blood for plasma Na<sup>+</sup> and K<sup>+</sup> concentrations should be performed as soon as possible after blood sampling.

#### **Key Words**

Sodium, potassium, ion specific electrodes, bicarbonate.

#### Introduction

Instruments for the measurement of plasma sodium (Na+) and potassium (K+) concentrations with ion-selective electrodes (ISEs) are increasingly used in clinical laboratories and special-care units. These instruments are easy to use by non-laboratory personnel providing rapid results without the need for centrifugation of blood when using direct reading ISEs with whole blood. ISEs measure free ions compared to total ion concentration by flame photometry. Generally agreement between ISEs and flame photometry is good provided that there are no abnormal concentrations of lipids and/or proteins [1]. As Na\* and K\* can bind to bicarbonate [2], differences in plasma Na+ and K+ results between ISEs and flame photometry could theoretically arise, especially if there is CO2 loss from specimens which have been standing for a while prior to analysis. Thus whole blood, if kept standing for a period of time prior to analysis with ISEs, could show changes in plasma Na<sup>+</sup> and/or K<sup>+</sup> concentrations. This study was undertaken to determine the effects of storing blood specimens for various time periods at room temperature on plasma Na<sup>+</sup> and K<sup>+</sup> concentrations as measured by ISEs on whole blood.

#### Methods

Blood was obtained by venepuncture from 14 subjects and each subject's blood specimen was placed into four heparinised Vacutainer™ tubes. One tube from each subject was placed in a beaker of water for three minutes to allow them to reach room temperature prior to analysis for Na+ and K+ with an Ionetics™ electrolyte analyser as previously described [3]. The other blood samples were then left at room temperature and one sample from each subject was analysed at 1 h, 2 h and 3 h post-blood sampling respectively, the specimens being gently mixed by inversion before analysis. A primary standardisation and quality control check of the instrument was performed just prior to these measurements. The results obtained at 0 h, 1 h, 2 h and 3 h were compared by analysis of variance (ANOVA) with repeated measures taking all individual results into consideration. A p value of <0.05 was deemed statistically significant.

#### Results

The results shown in Table 1 demonstrate a progressive increase in plasma Na<sup>+</sup> and K<sup>+</sup> concentrations when whole

blood is left standing at room temperature over a 3 h period. Although the mean difference in plasma Na+ at 2 h and 3 h compared to 0 h was less than 2 mmol/L, this was statistically significantly different (p = 0.029 and 0.0042 respectively). A change of 2 mmol/L in plasma Na<sup>+</sup> would have little clinical significance but could be of importance in comparative studies with other methodologies. Plasma K\* progressively increased over time, the changes at 1 h, 2 h and 3 h compared to 0 h being statistically significantly different (p = 0.033, <0.0001 and <0.0001 respectively).

#### Table 1

Comparison of results for plasma Na<sup>+</sup> and K<sup>+</sup> of whole blood progressively left standing at room temperature

Time after venepuncture	Na+	К+	Mean difference compared to 0 h Na+ K+		
0 h	139.9 (1.56)	3.63 (0.53)			
1 h	140.6 (0.78)	3.68 (0.55)	0.71	0.05*	
2 h	141.0(0.78)	3.79 (0.52)	1.14*	0.16*	
3 h	141.6 (0.84)	3.93 (0.55)	1.79*	0.30*	

Results shown are mean values (± 1SD) in mmol/L

#### Discussion

There are various possibilities for the findings of this study, and one of these could be the bicarbonate factor. It has been previously demonstrated that bicarbonate forms complexes with Na<sup>+</sup> and K<sup>+</sup> under physiological conditions [2]. In plasma  $\pm$  3% of total Na<sup>+</sup> and K<sup>+</sup> is bound to bicarbonate (at a bicarbonate concentration of 25 mmol/L) which could explain some of the differences in results obtained when comparing ISEs with flame photometers. Upon standing blood may lose CO2 resulting in a lowering of plasma bicarbonate concentration. This loss of bicarbonate could release Na<sup>+</sup> and K<sup>+</sup> from their respective bicarbonate complexes, thus potentially increasing the amount of free Na+ and K+ in plasma. As ISEs measure free ions, this may explain why when blood is left for a period of time at room temperature, plasma Na<sup>+</sup> and K<sup>+</sup> results increase, as evidenced in the present study. Alternatively, a shift of water from the extracellular to intracellular space as a result of metabolic processes within the red cell may have contributed to the observed increase in plasma Na\* and K\* concentrations over time. The increase in plasma K\* over time could also be due to partial inactivation of Na+-K+ ATPase allowing K+ to passively diffuse down its concentration gradient as previously noted in heparinised whole blood specimens kept at 4°C [4].

Neither loss of CO2 nor cellular gain of water was measured in this study. Furthermore, similar studies need to be done on blood samples from patients with acid-base and electrolyte disturbances to see if measured plasma Na<sup>+</sup> and K<sup>+</sup> concentrations change in a similar manner as in this study. It is concluded that analysis of plasma Na+ and K+ concentrations by ISEs in whole blood specimens, should be performed shortly after venepuncture especially if comparison studies between ISEs and flame photometry are contemplated.

#### Acknowledgments

This study was supported by grants from the Lottery Board and National Heart Foundation of New Zealand. Mrs H. Bark kindly typed the manuscript.

Statistically significant at 95% by ANOVA (p <0.05).

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## **ERRATA: BOOK REVIEW**

## "Manual of Laboratory Immunology"

Linda E Miller, Harry R Rudke, Julia E Peacock, Russell H Tomar

Second Edition, 1991 Publishers — Lea and Febiger

This book was reviewed in the March Journal [NZJ Med Lab. Science. 1991, **45**:1 p29]. One very red-faced reviewer would like to apologise to the Editor for a lapse in proof reading which resulted in a mistake not only in the title, but in the name of the publishers also.

I stand by what I wrote for the rest of the review. This is a splendid little book and I hope it will be on the shelves of all labs using immunodiagnostic techniques.

Gillian McLeay Convenor, Immunology Special Interest Group

## NZIMLS CALENDAR 1991

NZIWILS CALENDAR 1991				
22 February	Applications close for QTA			
	examinations.			
27/28 February	Council Meeting - Auckland.			
1 April	Staffing Survey forms to be			
	completed			
25 April	Committee Annual Reports to be with			
	the Executive Officer.			
7/8 May	QTA examinations			
23/24 May	Council Meeting - Christchurch			
20 May	Proposed rule changes and remits to			
50 May	he with the Executive Officer			
Ot Mou	Applications along for Specialist			
31 May	Applications close for specialist			
	Certificate examinations			
31 May	All accounts to National Treasurer for			
	auditing			
27 June	Nomination forms for the election of			
	Officers and Remits to be with the			
	membership (60 days prior to AGM)			
3.5 July	Fellowship examinations			
10 hill	Nominations close for election of			
To July	Officere (40 days prior to ACM)			
	Officers (40 days prior to AGM)			
6 August	Ballot papers to be with the			
	membership (21 days prior to AGM)			
13 August	Annual Report and Balance Sheet to			
0	be with the membership (14 days			
	prior to AGM)			
20 August	Ballot papers and provies to be with			
20 August	the Executive Officer (7 days prior to			
	the Executive Officer (7 days prior to			
	AGM)			
26/27 August	Council Meeting — Auckland			
27 August	AGM and SGM - Auckland			
28-30 August	South Pacific Congress			
13/14 November	Specialist Certificate examinations.			

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## Membership Sub-Committee Report — February 1991

Since the November meeting there has been the following changes:

	28.2.91	7.11.90	27.8.90	29.5.90
Membership				
	1277	1272	1315	1702
less resignations	10	3	19	16
less G.N.A.	11	5	35	51
less deletions	116	-	-	328
less deceased	40	-	-	+
less duplications	-	-	9	-
	1140	1264	1252	1307
plus applications	71	3	0	8
plus reinstatements	2	1	11	-
	1213	1268	1272	1315
Composition				
Life Member (Fellow)	12	12	12	12
Life Member				
(Associate)	5	5	5	5
Fellow	22	22	22	23
Member	679	724	721	688
Associate	399	424	425	503
Non-practicing	56	60	56	53
Honorary	30	30	31	31
Total	1213	1277	1272	1315

## **Applications for Membership**

Peter MARSHALL, Diagnostic; Sonya KENNEDY, Invercargili; Alison GROOBY, Greymouth; Fiona BLOOR, Gisborne Medical Lab; Rowena KILROY, Med Lab South; Susan McINTYRE, Med Lab South; Christine ROTHERY, Med Lab South; Donna NASH, Med Lab; Terrence PRIDDLE, Auck Med School; Dawn FOSTER, Waikato, Immunohaem; Melanie DUTHIE, Royston; Davine WIMSETT, New Plymouth Med Lab; Nicola PREBBLE, New Plymouth Med Lab; Sandra DAVIDSON, Med Lab South; Wendy TAYLOR-HAYHURST, Med Lab South; Margaret NISBET, Middlemore, Haem; Sonia Membership Convenor Geoff Rimmer P.O. Box 9095, Newmarket, Auckland.

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For Non-practising Members — \$33.00 GST inclusive

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

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

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#### Resignations

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(POSITIONS I	HELD)	
PAPERS PUBLISH	HED OR PARTICIPATION IN F	RESEARCH:
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DO YOU INTEND	TO SUBMIT A PAPER FOR I	PRESENTATION AT THE CONGRESS?
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NZJ Med Lab Science. 1991; 45(2): 55-56.

## Vibrio vulnificus: Two Case Reports.

## Jacqueline M. Wright FNZIMLS;

## Laboratory, Whakatane Hospital, Bay of Plenty Area Health Board. Abstract

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

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

#### Introduction

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

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

#### Case 1.

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

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

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

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

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

#### Case 2

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

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

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

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

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

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

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

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

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

Laboratory findings:

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

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

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

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

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

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

Why then are so few cases noted?

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

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

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

#### Discussion

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

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

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

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

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

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

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

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

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

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## NZIMLS CONTINUING EDUCATION SPECIALIST INTEREST GROUP UPDATE

## FROM THE MICROBIOLOGY SPECIAL INTEREST **GROUP (MSIG)**

#### **Convenor:** Shirley Gainsford

Contact Address: Valley Diagnostic Laboratory, P.O. Box 30044, Lower Hutt.

The main event for the MSIG this year is the Symposium on Nosocomially Acquired Infections being held at Victoria University Wellington on Thursday May 16th. We have a varied programme which has been sent to all laboratories. The MSIG is also holding a Mycology workshop in Auckland the day before the South Pacific Congress. Allan Woodgyer of NZCDC and Dinah Parr of Auckland Hospital will co-host this workshop entitled "Identification of Medically Important Saprophytic Fungi." This follows on from the Dermatophyte workshop held in Invercargill last year. Membership of the Microbiology Journal Club is now available to any NZIMLS member in New Zealand. The librarian for Hawkes Bay, Taranaki, Manawatu, Wellington and the South Island is Pat Maddocks, Virology/Serology Lab., Department Laboratory Services, Wellington Hospital. For the rest of the North Island it is Philippa Skellern, Med Lab, P.O. Box 4120, Auckland.

We are offering to help Specialist Level Microbiology candidates again this year with study guides. We do not have the resources to do the same for Certificate level candidates but any Institute member who is having problems finding material for this exam is welcome to write for advice.

#### FROM THE BIOCHEMISTRY SPECIAL INTEREST **GROUP (BSIG)**

#### Convenor: Alison Buchanan

Contact Address: Clinical Chemistry Dept., Auckland Hospital, Park Road, Auckland.

Thank you to all the Charge Technologists who returned the Questionnaire we sent in November. It was somewhat disappointing to get only a 29% return.

We are attempting to set up regional representation to make dissemination of information more efficient and to date have received acceptance from:-

Richard Ward for Waikato/Bay of Plenty

Alistair Kerr for Manawatu/Wanganui

Rob McKenzie for Nelson/Marlborough/Greymouth

The guestionnaire replies received indicated a need for information to/from the smaller laboratories with the suggestions of Regional seminars and the circulation, monthly, of the index pages of Clinical Biochemistry Journals on a user pays basis.

The Journals suggested are:

1. Annals of Clinical Biochemistry

- 2. British Medical Journal
- 3. Clinical Biochemist Reviews
- 4. Clinical Biochemistry
- 5. Clinical Chemistry
- 6. Clinics in Laboratory Medicine
- 7. Critical Reviews in clinical laboratory sciences

8. Journal of Clinical Chemistry and Clinical Biochemistry. The cost will be \$30 per year.

If you wish further information, or would like to join the please contact: Linda Kilminster, Clinical mailing Biochemistry Dept. Middlemore Hospital. AUCKLAND

In July the Waikato Regional Biochemistry Q.A. Group and the Auckland Biochemistry Regional Q.A. Group join for a meeting.

Further information is available from: Richard Ward, Clinical Dept. Waikato Hospital, Private Biochemistry Bag, HAMILTON.

## FROM THE IMMUNOLOGY SPECIAL INTEREST **GROUP (ISIG)**

## Convenor: Gillian McLeay

Contact address: Laboratory Training Centre, Building 18, Auckland Hospital, Park Road, Auckland 1. (Please note change of address).

Thanks to all those who responded to the questionnaire. We have adopted the 5 regional centres proposed by the HSIG and currently ISIG has 27 members from every centre except Otago/Southland. Anyone who would like to join us would be most welcome. Just write to me at the above address.

Feedback indicates the group's requirements fall into two broad categories - communication and education. The first edition of the "ISIG Network News" was sent out early March to start the ball rolling. However, I need material for further editions, so "keep those cards and letters coming". On the educational side, I should like to hear from our members of any seminars which they would like to hold in their region with support from ISIG.

The Auckland group is investigating the possibility of holding a Floating Forum (ie. on the Waitemata Harbour) round about the time of the South Pacific Congress in August. If you plan to be in Auckland for the Congress or the August holidays, please let me know if you would like to take part. We will organise the forum so that it does not clash with any of the Congress programmes. It is planned to make the occasion informative and fun, and to show off our beautiful harbour.

## FROM THE HAEMATOLOGY SPECIAL **INTEREST GROUP (HSIG)**

Convenor: Ross Anderson Contact Address: C/- Miss M Eales, Department of Haematology, Middlemore Hospital, Private Bag, Otahuhu

H.S.I.G. Regional representatives have been appointed for the following regions

Auckland/Northland Mr Ross Anderson C/- Miss M Eales Department Haematology Middlemore Hospital **Private Bag** Otahuhu Waikato/Bay of Plenty Mr S Shepherd C/- Medlab Waikato P.O. Box 52 Hamilton Hawkes Bay/Manawatu/Taranaki To be Confirmed Wellington/Wairarapa/Nelson/Marlborough Mr Errol Crutch Haematology Laboratory Department of Laboratory Services Wellington Hospital Wellington Canterbury/West Coast Miss Christine Hickton C/- Department of Haematology Christchurch Hospital **Private Bag** Christchurch Otago/Southland Mr Steve Wilson Department Haematology

Dunedin Hospital P.O. Box 946 Dunedin

The regional groupings are not intended to be hard and fast and obviously changes will be made to suit the wishes of particular districts. The initial criteria was that there should be no more than about two hours car travel to a central location in the region. It could well be that in many cases people will wish to attend seminars in adjacent areas.

Please contact your regional representative if you feel you can help or have ideas to contribute.

The H.S.I.G. group will be sending a regular newsletter to Laboratories every two months and we would welcome any contributions for inclusion, these might be reports of regional meetings or seminars, interesting case histories or technical problems etc. These contributions should be addressed to Mr Ross Anderson, Chairperson, H.S.I.G., Auckland.

## NEW PRODUCTS AND SERVICES

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Contact: JOJO V TAYAG

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A position is required for a Canadian Registered Technologist seeking work in New Zealand. She has worked at the Hospital for Sick Children in Toronto for six years. Trained in Biochemistry, Haematology, Immunohaematology, Histology and Microbiology she would prefer to work in Microbiology or Biochemistry.

References and resume available by contacting:

Janice E Gravett 2 Bryant St Palmerston North Tel: (063) 81-606



## SPECIALIST LEVEL EXAMINATIONS 1990. EXAMINERS' REPORTS.

## MICROBIOLOGY.

## PAPER 1.

SECTION A:

Range of marks: 9.5 - 19.0 Average: 14

As indicated by the average mark most candidates answered this section adequately.

In some questions e.g. Q2(b) some candidates went into far too greater detail for the marks available while in Q2(a) a number of candidates had either little or no knowledge of P.C.R.

#### SECTION B:

Range of marks: 3.5 - 17.5 Average: 13.1

Only two candidates failed to pass this section. One of these two failed to answer question 1. The answers to the mycology and true/false questions were in general quite satisfactory. In contrast, nearly all the candidates gave poor answers to question 3 (definitions).

SECTION C:

Range of marks: 9 - 16 Average: 11.5

Question 1: Matching organisms to the most appropriate statement. Adequately answered by all candidates.

Question 2: Part (b) poorly answered by all candidates. 6 received 0 marks in reasons for choice.

Question 3: All candidates answered this question satisfactorily.

SECTION D:

Range of marks: 2 - 10.5 Average 6.5

This section was poorly answered by all candidates. The impression gained is that this section was the last answered and candidates were pressed to finish in time. Several omitted to answer parts of this section which is reflected in the low marks attained.

In Question 1: Many candidates gave answers which were too detailed. Examples were not required.

## PAPER 2:

SECTION A:

Range of marks: 4.5 - 17.5 Average: 10

Question 1: 9 candidates attempted this question on community acquired atypical pneumonia.

As this average mark shows this question was not well answered by most candidates in fact only 2 candidates received more than a pass mark of 12.5.

A large number of candidates lacked a knowledge of the most likely aetiological agents but went into depth on unlikely agents such as pneumocystis or cryptococcus. It was specifically mentioned that the patient was not immunocompromised and so although marks were given for these agents they were not heavily weighted.

Very few candidates gave information as to why they would prefer a particular type of specimen over another in helping to isolate the causative agent. Little regard was given to serology and blood cultures as part of the laboratory "workup'

Some candidates I assume must have left this question till last as they gave very brief answers as shown by their marks.

Question 2: One candidate attempted this question and achieved a satisfactory mark of 14.

SECTION B:

Question 1: Range of marks: 13 - 17 Average: 15

Nine candidates attempted this question. The answers on laboratory diagnosis of Giardia was good, but in general the answers to the other sections on epidemiology, pathogenesis, symptoms and chemotherapy revealed some lack of knowledge.

Question 2:

Only 1 candidate attempted this question attaining over half marks. Part (e) on chemotherapy was the only part inadequately answered.

SECTION C:

Question 1:

Range of marks: 16 - 21 Average: 18

Four candidates answered this question. All the candidates gave good answers in general. However, some lack of knowledge on antibiotic susceptibility of MRSA isolates was demonstrated by most candidates. SECTION D:

Question 1: Candidates 6

Range of marks: 2.5 - 16.5 Average 10

More candidates answered this question than question 2. Candidates scored best under the section on laboratory diagnosis, but generally failed to get good marks because they could not discuss the relative merits of the different available methods. Their knowledge of the virulence factors was virtually nil but most showed some understanding of the clinical disease. Overall, this question was only modestly attempted.

Question 2: Candidates 6

Range of marks: 7 - 14 Average: 10

This question was poorly attempted and most answers were brief. Candidates knew least about the principles of antimicrobial susceptibility testing and the various therapeutic regimens that are available. The candidates knew more about the isolation and identification of these two mycobacteria but didn't appear to know why the methods they had mentioned should be chosen. The epidemiology section was generally well attempted.

## CYTOLOGY.

## **GENERAL COMMENTS.**

The standard of legibility and layout was good in all papers. Use of diagrams was limited but appropriate.

The examiners considered that the papers set were difficult, even for the specialist level. Interpretation in gynaecological cytology was given minor emphasis, allowing extensive coverage of other aspects, some of which may have been available in practice to the candidates only to a limited extent.

## SPECIFIC COMMENTS.

- 1. Questions of an interpretive nature were dealt with well overall. There were notable areas of weakness in two candidates in non-neoplastic pulmonary cytology, cerebrospinal fluid and gastric cytology.
- 2. The questions on quality assurance and laboratory safety were dealt with uniformly well.
- 3. Questions of a technical nature showed considerable variation in responses, with particular weakness in one candidate in immunocytochemistry.
- 4. The question on requirements for a national cervical cancer screening registry was well answered by all candidates.
- 5. Questions dealing with general pathology such as amyloidosis and apoptosis were done rather poorly.
- 6. The question on thyroid cytology was answered well.

## HAEMATOLOGY.

Ten candidates sat this examination, 7 passed and 3 failed. The mark range was 37 - 61%, with a mean mark of 50%.

Paper One consisted of 5 questions. Four questions presented laboratory data for interpretation and 1 question was an instrument evaluation. This paper included questions of a similar format to previous practical papers. The mark range was 30 - 67%, mean 50%. Four candidates failed to gain a pass mark in this paper. This included the 3 who subsequently failed the examination.

Paper Two contained 6 essay questions with the candidate required to answer 4 of them. The mark range was 34 — 58%, mean 50%. Four candidates failed to gain a pass mark in this paper and this included 2 who subsequently failed.

Overall only 4 candidates gained more than 50% in both papers. Those who failed the examination, failed convincingly with the highest overall mark by a failed candidate being 43%. The mean overall mark of those who passed was only 54%.

Apart from a few isolated flashes of brilliance, the standard overall was at best average. The interpretation of laboratory data was particulary disappointing, most candidates detecting the obvious, but ignoring results they were unable to explain.

Examination technique must again be questioned. This has been a recurring theme in the last few years Examiner's Reports.

## CLINICAL BIOCHEMISTRY PAPER 1.

#### Question 1.

Six candidates answered this question, one answer was excellent. Simple arithmetic errors still plagued the answers to the calculation question. Others were unable to use simultaneous equations to solve the answer.

Generally the short answer part of the question was poorly done.

Question 2.

Six candidates attempted; 2 passed.

- (a) i. Some excellent answers with good marks. Nobody gave as an advantage or disadvantage the linearity limits of the glucose methods discussed.
  - ii. Most candidates could give the principles but the advantages and disadvantages were not well done.
- (b) Five out of 6 gave the correct answer.
- (c) Three out of six gave the correct answer. Disappointing to see two candidates missing parts of the question out and as a consequence, failing this question.

Question 3

Six candidates answered this question, 5 of them gained 60% or more. The diseases of the adrenal gland and their influence on biochemical analytes were well answered while the difference between homogeneous and heterogeneous immunoassays was less well understood.

Question 4.

Five candidates attempted; 3 passed.

Cholinesterase and a -antitrypsin question was well answered in 4 out of 5 papers. The section on Lactate was generally not well done.

Question 5.

Six candidates answered this question, 5 gaining over 50%. Generally the standard of answer was good with a sound knowledge of the methodologies but less certainty of all the clinical implications.

Question 6.

Seven candidates attempted; 4 passed.

The question was well answered by the 4 that passed. The major problem with the failed candidates was not answering the question as asked, i.e. poor examination technique.

Question 7.

Six candidates answered this question and all gained over 50%. Most knew the principle of fluorometry but the advantages and disadvantages were less well answered. Question 8.

Five candidates attempted; 1 passed.

- (a) Excellent answers by three candidates. One candidate did not answer the question and failed.
- (b) Only one candidate passed. Very poor answers given, especially on the standardisation of neonatal bilirubin assays. Only 1 candidate mentioned the National Women's neonatal bilirubin survey and standardisation programme.

Question 9.

Surprisingly only 4 candidates tackled this topical question, 3 gained over half marks. Once again, the technical aspect of the question was well answered while the interpretive aspect not so.

Question 10.

Two candidates answered this question. Very average — not enough detail supplied for the detection of the bands. The section on clinical significance was satisfactory.

## PAPER 2.

Question 1

Overall, the standard was very disappointing, considering the fact that creatinine is probably the most common test done in Biochemistry, and that there has been so much written in the literature.

Four attempted; 1 passed.

Only one candidate gave a reasonable resume of the various methods available while some answers given were only a few lines. The question said discuss, not make short notes.

Question 2

Seven attempted; 4 passed.

The standard of answers was satisfactory. Those areas that were not well done included staffing. Most candidates seemed to forget that a Stat service is required 24 hours/day. Result reporting was also less than satisfactory; many candidates did not mention modern communication tools such as computers and fax machines.

#### Question 3.

Of the 6 candidates that answered this question, most had plenty to write about the metabolism of bilirubin and in doing so failed to answer the question.

Question 4

Two candidates attempted this question. They tended not to directly answer the question but got involved in the diagnosis of renal failure.

Question 5

Six attempted; 4 passed.

This question was generally well done but two candidates did not mention purchasing arrangements for the consumables and spares and had little idea on a structured preventative maintenance programme.

Question 6

Three candidates attempted this question, one of whom properly understood the Technical Article satisfactorily.

## **OVERVIEW.**

All candidates answered some questions very well but were unable to maintain a high enough standard throughout their paper. This meant good answers were offset by the poor ones. One candidate only answered 5 out of the 8 required in Paper 1.

Paper 1 was changed this year; it contained no sections and the selection was reduced from answering 9 questions out of 12 to 8 out of 10 with no compulsory questions. Paper 2 remained the same with a choice of 3 out of 6 questions. The LYPHOCHEK family of TDM Controls makes Bio-Rad the single source for complete therapeutic drug monitoring. Each control provides accurate and precise validation for assays of drug families most often encountered in the laboratory. Designed for both manual and automated methodologies, the LYPHOCHEK controls are versatile and economical.

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# ANTIMICROBIAL SUSCEPTIBILITY TESTING A system for standardization

BBL from Becton Dickinson, offering a complete line of products for susceptibility testing, including dispensers and the most complete listing of antibiotic and antimicrobial discs.

BBL Sensi-Discso serve as a convenient method for addition of antimicrobial agents to culture media, especially for qualitative susceptibility testing of mycobacterla and related organisms. The discs are added to measured amounts of liquified medium in quadrant plates to prepare exact concentrations of the antimicrobial agent in solidified agar. Following innoculation with the test organism, growth or no growth will occur, depending on whether the organism is resistant or susceptible to the agent and concentration used in the quadrant.





The self-tamping 6 and 8 place dispensers are designed for 100mm style plates. Either dispenser can be used for susceptibility testing or for the presumptive identification of anerobic bacilli using Sensi-Disc antimicrobial discs. They dispense and tamp discs onto the agar surface, eliminating the need for tamping with forceps. The units are less than 20cm high, are provided with an interlocking base, cover and reusable indicator dessicant container. The unit fits readily between shelves in the refrigerator

**Zone Size Interpretation** 

Measure zone sizes with a ruler, calipers or template provided to the nearest whole millimetre. End-point is the limit of the area showing no obvious growth. Report susceptible, intermediate or resistant using standard tables provided. (6-disc test shown)



# **ENRICHMENTS**

Isovitalex• Enrichment, is a chemically defined supplement used as an additive to media for isolation and cultivation of nutritionally fastidious microorganisms.

Cat.No. BBL 11876 5 x 10ml

Cefinase Discs are intended for use in rapid testing of isolated colonies of Neisseria gonorrhoeae, Staphylococcus spp., Haemophilus influenzae and anaerobic bacteria for the production of B-lactamase. Cat.No. BBL 31650 Vial of 50 discs

Penicillinase is a purified, nontoxic, clear enzyme preparation used to neutralize penicillin and permit growth of organisms ordinarily inhibited by the antibiotic.

Cat.No. BBL 11897 20mls

Coagulase Rabbit Plasma is used in qualitative procedures to determine the pathogenicity of staphylococci. Plasma with EDTA is used in the same way.

Cat.No. BBL 40658 Cat.No. BBL 40659 Cat.No. BBL 40661 Cat.No. BBL 40826 Cat.No. BBL 40827

(Sod.citrate) 10 x 1ml reconstitutes to 3mal (Sod.citrate) 10 x 2.5ml reconstitutes to 7.5ml (Sod.citrate) 10 x 5ml reconstitutes to 15ml (EDTA) 10 x 5ml reconstitutes to 15ml (EDTA) 10 x 1ml reconstitutes to 3ml

CVNT Inhibitor is an antibiotic mixture of vancomycin, colistin, nystatin and trimethoprim which is incorporated into culture media to permit the selective isolation of Neisseria gonorrhoeae and N.menIngitidis by increasing the selectivity of the Isolation media.

Cat.No. BBL12408 10 x 10ml



AUCKLAND Private Bag Northcote Ph: (09) 418-3039 Fax (09) 418-0729

WELLINGTON P.O. Box 31-044 Ph: 0800 807 809 Fax: (04) 697-240

CHRISTCHURCH P.O. Box 1813 Ph: 0800 806 974 Fax: (03) 663-647

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## **BM-Test Colon Albumin:** quick, easy, specific and clean!



BM-Test Colon Albumin is the f immunochemical test strip for colon diagnosis combining sim plicity of use with exact results Due to its high specificity dieta interference (e.g. with vitamin meat etc.) is eliminated. That is why no dietary restrictions for patients are necessary. No han ling of feces in the laboratory of additional apparatus required.

Simple two-step test. Insert test strip in stool application slide and add developer solution. No further attention required.



The result can be read any between 5 and 15 minutes will be a definite yes or no. and



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