Histograph

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NEWSLETTER OF THE HISTOTECHNOLOGY GROUP of NSW

Contents

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Editorial

Nearly the end of the year! I suppose its time to ponder what we have done in 2004. Have we achieved our goals? Actually what were our goals? Oh well, I'll think of some new ones for 2005!! (And forget them by February!!).

I must apologise for the lateness of the last issue of Histograph. We are now using an external printer and we had some teething problems. Hopefully, all is fixed. Also what did you think of that issue? How can we improve Histograph for next year? (How about sacking the Editor??).

This issue has part one of an update on Amyloidosis as well as cryostat disinfection, and abstracts from the literature. An interesting story on Histotechnology in the Desert by Karen Appold, which was

originally published in "Advance for Medical Laboratory Professionals" (www.advanceweb.com) is also included.

We hope you had an enjoyable and restful Christmas and may the New Year bring you all the best and we look forward to hearing from you in 2005.

Tony Henwood
Editor
Histograph
anthonyh@chw.edu.au

Chairman's Message

Merry Christmas and a Happy New Year to all our members.
The past year has seen many things happen, particularly the Inaugural National Histotechnology Seminar at Olympic Park in Homebush, Sydney. The Victorian Group has preparations well under way for the next National Seminar being sponsored by the Victorian Group in October this year. Please remember to support the Victorians in this venture I am sure they will present an excellent Seminar. We will endeavour to keep you posted about happenings as we are advised of them.
The current committee is looking at holding some interesting workshops and meeting during this year and you will soon be getting notification of these meetings.

I apologise for the lateness of this newsletter and even though it is late it once again has some very interesting articles for your interest.

For those who did not attend the Christmas Lecture at North Ryde RSL all I can say is
You missed out on an extremely interesting talk by Stephen Doggett of the Entomology Department ICPMR talking about Horse Equine Virus, Maggots and their use in healing wounds and lastly bed bugs. The location was great, the food excellent and the company very friendly, although only 15 attended.
Please remember to support your committee and group by participating in as many functions as you can.

Yours in Histotechnology
Bill Sinai
Chairman.

Amyloid Update – Part 1

This is the first of a two part article. The rest you'll see in 2005.

The diagnosis of systemic amyloidosis is only occasionally suspected on clinical grounds alone and is more often considered when an associated condition, such as a chronic inflammatory disease or monoclonal gammopathy, is present. No blood test is diagnostic of amyloid although routine haematological and biochemical investigations have important roles in defining the underlying disease process in amyloidosis, and evaluating organ function. A number of non-invasive investigations including echocardiography, electrocardiography and soft tissue scintigraphy with bone-seeking tracers give characteristic results in some patients with amyloidosis, but are non-specific. The diagnosis can only be confirmed by demonstrating the presence of amyloid deposits in the tissues. Histology is the traditional method in routine clinical practice and is sensitive for revealing microscopic deposits and permits immunotyping of fibril proteins (Hawkins 1994).

The following review presents some of the recent findings in this fascinating disease.

Beta(2)-microglobulin amyloidosis (A beta(2)M)

Histology remains the gold standard to diagnose beta(2)-microglobulin amyloidosis (A beta(2)M), also known as Dialysis-related amyloidosis. Two diagnostic criteria are required: positive Congo red staining with typical birefringence under polarized light and immunostaining of amyloid deposits with a labelled anti-beta(2)M antibody. A beta(2)M is preferentially located in the joints. Small deposits are also found in various organs, mainly the heart and gastrointestinal tract (Jadoul et al 2001).

Pathologic studies have demonstrated a high prevalence of articular A beta(2)M early in the course of hemodialysis and peritoneal dialysis, antedating clinical manifestations by several years.

The stages of beta(2)M amyloid formation have been delineated:

1. Beta(2)M amyloid deposits first on the surface of the cartilage, in the absence of macrophages (stage 1),
2. Subsequently involves capsules and synovia (stage 2),
3. With eventual recruitment of macrophages around large beta(2)M amyloid deposits (stage 3).

Clinical manifestations are likely associated with the inflammation observed in stage three.

The factors triggering the fibrillar precipitation of beta(2)M remain unknown. Macrophages do not play a role: their presence is the consequence rather than the cause of beta(2)M amyloid deposits. Several substances coprecipitated with beta(2)M amyloid have been incriminated: highly sulfated glycosaminoglycans such as chondroitin or keratan sulfate, antiproteases such as alpha(2)-macroglobulin, and apolipoprotein E. As yet, no definitive conclusion has been reached (Jadoul et al 2001).

Cardiac amyloidosis

Cardiac amyloidosis is caused by amyloid deposits derived from different human plasma proteins. It can lead to cardiac conduction disturbances, restrictive cardiomyopathy, and low output heart failure. The heart is variably involved during the development of systemic amyloidosis and seems to be more frequently affected in immunoglobulin (primary) than in reactive (secondary) amyloidosis (Hesse et al 1993).
Amyloid is common in the elderly. Isolated atrial amyloid, for which a major subunit is the atrial natriuretic peptide, seems to be three times more frequent than senile cardiac amyloid, which is derived from normal prealbumin (transthyretin).

Like polyneuropathy, cardiac amyloidosis is a prominent clinical feature of hereditary amyloidosis, namely of the autosomal dominant transthyretin (TTR) type. All 28 cases of TTR amyloidoses reported so far were heterozygotes for a single nucleotide change in the gene for

TTR that resulted in amino acid substitutions in the mature protein.

A new TTR genetic variant was reported in a German family where the index patient presented at the age of 63 with anginal pain and arrhythmia. Electrocardiography was suggestive of a pseudoinfarction pattern, and echocardiography and cardiac catheterisation showed signs of hypertrophic nonobstructive cardiomyopathy with increased ventricular filling pressures and a prominent "a" wave.

Amyloid of the TTR type was identified by immunohistochemistry in the endomyocardial biopsy specimen. Hybrid isoelectric focusing established heterozygosity by showing normal TTR protein and an electrically neutral TTR variant differing from all known TTR variants so far. The patient died in an accident before investigations were complete. Electrophoretic analysis of the plasma from his first degree relatives (son, daughter, brother, and mother) identified the asymptomatic 22 year old son as an apparently heterozygous carrier of the mutant TTR protein. Comparative tryptic peptide mapping and sequencing showed that isoleucine at position 68 of the amino acid sequence was replaced by leucine (Hesse et al 1993).

New Methods For Staining Amyloid In Tissues

The traditional way of identifying amyloid in tissue sections has been staining with Congo red and demonstration of green birefringence under crossed polarisers. The original method of Congo red staining, described by Bennhold in 1922, has undergone several modifications to improve its sensitivity, specificity, and reliability. The most common modification is the alkaline Congo red method described by Puchtler and co-workers in 1962. Specificity is improved by using freshly prepared stain and a staining solution fully saturated with sodium chloride (Elghetany & Saleem 1988).

Amyloid proteins can be further distinguished by autoclaving or by treating the tissue with potassium permanganate or alkaline guanidine. Autoclaving the tissues at 120°C for 30 min causes protein AA to lose its affinity for Congo red. Prolongation of autoclaving to 120 min abolishes the Congophilia of protein AL, but prealbumin-related amyloid shows little or no change. Treatment of the tissue with potassium permanganate causes protein AA and B2-microglobulin amyloid to lose their affinity to Congo red. Protein AA fails to stain with Congo red after treatment with alkaline guanidine for 1 min and protein AL and systemic senile amyloid protein (SSA) after 2 hr. Familial amyloid protein (FAP), prealbumin type, can stand 2 hr of alkaline guanidine treatment without losing its ability to stain with Congo red (Elghetany & Saleem 1988).

Other methods of detection of amyloid include fluorescent stains, e.g., Thioflavin T or S, and metachromatic stains such as crystal violet.

Immunofluorescence and immunoperoxidase methods are used to identify and classify amyloid proteins in tissues. Antibodies against the P component, proteins AA and AL and FAP have been used with great precision. Due to cross-reactivity, these methods do not differentiate between some types of familial and senile systemic amyloidosis (Elghetany & Saleem 1988).

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Letter to the Editor

It was with regret that I read in the last newsletter of the death of Alan Smith.

Alan was involved in the gestation of the "Histo Group" in NSW and was on the committee during the formative years which produced a stable society.

He could always be called upon to give me the utmost support when needed and could be relied upon to carry any task through.

I am sorry, that in my retirement years, I didn't keep in touch. We each had our own burdens and I would see him occasionally for a chat usually in the shopping centre where he would be with his wife Beth.

To me, Alan was a true gentleman and a great master technician and he contributed greatly to our cause.

Bruce Munro
Founding Chairman
Histotechnology Group of NSW.

Histology Group of Victoria Inc.

The committee of the Histology Group of Victoria has commenced working already on the program and format.

Dates: Friday 21st – Sunday 23rd October, 2005

Venue: Carlton Crest Hotel
65 Queens Road
Melbourne
(4.5 km SE of the GPO, overlooking Albert Park Lake)

A range of accommodation is available at the Carlton Crest, at both 3 ½ and 4 ½ star rating. Room rates are as follows:

Standard Rooms	$134.00
Congress Suites	$149.00
Superior Rooms	$203.00
Carlton Club	$233.00

Car parking is available beneath the hotel.

The city centre, Spencer Street transport terminus and Airport bus terminals are a short tram ride away, as are Southbank and St Kilda.
Alternative accommodation is also available nearby.

Confirmed topics and/or speakers in the plenary sessions include:

- Mr Neil Hand — Queen's Medical Centre Nottingham UK
- Dr Cesar Wong — Chinese University of Hong Kong
- Lung morphometry — Women's and Childrens Hospital, Adelaide
- Non-clinical applications of histology — Anatomy Department University of Melbourne
- "Virgin Speakers"

HGV website: http://home.vicnet.net.au/~hgvinc/
Contact the HGV: hgv@netspace.net.au

Calling all Histologists!

The HGV is in the process of organising the program for the 2nd National Histology Meeting. We are calling for submissions from all interested parties, from all states and territories in this wide, brown land. The topics we have thought of so far are:

- *A day in the life of a regional Histology lab*
- *Histology of Hirschprung's disease*
- *Enzyme histochemistry*
- *Immunohistochemistry case studies*
- *Overview of further education available to Histologists*
- *"Virgin" speakers – (the first time is always special…)*
- *Liver special stain panels*
- *Histology of Food*
- *Sentinel node protocols*
- *Specimen collection*
- *"Silver" theme Case studies*
- *New use for old Ab's*
- *C4d renal rejection marker*
- *Rabbit monoclonals experience*
- *Forensic*
- *Special stains*
- *Mortuary practices*
- *Museum techniques*
- *Veterinary histology*
- *Marine histology*

The list is by no means exclusive, but is more a list of topics that we would like to include in the program so if there is anything you would like to present in a National forum, now is the time to collect your ideas, get them down in a Powerpoint presentation (or other format by arrangement) and impart your knowledge and experience to an eager audience.

Poster presenters will not be required to deliver an oral presentation, but will be expected to attend at their poster display at a specified time each day.

Yours sincerely

HGV Organising committee.

PROGRAM DETAILS

Workshops	Friday 21st October 2005	0900 – 1200 1300 - 1600

Workshop 1 **Basic Immunohistochemistry Techniques**
Presented by: *Dr Cesar Wong* Associate Professor,
Chinese University of Hong Kong

Workshop 2 **Solvent Recycling**
Presented by: *Mr Andrew Kennedy* Senior Science Officer
Mr Frank Quaranta Senior Technical Officer
Anatomical Pathology,
Concord Repatriation Hospital

(Workshops 1 and 2 will run concurrently)

Workshop 3 **Histology Mythology: Fact or Fiction?**
Presented by: *Mr Neil Hand* Chief Biomedical Scientist
Histopathology Department
Queen's Medical Centre Nottingham UK

Cocktail Party	Opening of the Trade Display	1800 – 1930
Plenary Sessions	Saturday 22nd October	0900 – 1030 Morning tea 1100 – 1230 Lunch 1330 – 1500 Afternoon tea 1530 – 1700
Conference Dinner	Saturday 22nd October	1900 – late

THE POINT Albert Park Lake

Plenary Sessions	Sunday 23rd October	0930 – 1030 Morning tea 1100 – 1230 Lunch 1330 - 1500 **CLOSE**

REGISTRATION DETAILS

Full registration (includes opening cocktail party, morning and afternoon teas and lunches) **$225.00**

1 Day registration (includes morning and afternoon tea and lunch) **$150.00**

Workshops (includes morning or afternoon tea) **$75.00**

Student registration (per day, includes morning and afternoon tea and lunch) **$50.00**
(Application for student registration must be accompanied by evidence of full-time enrolment in a recognized course)

Cocktail Party (extra tickets) **$45.00**

Conference Dinner **$85.00**

Late Registration Penalty (after August 19, 2005) **$75.00**

Abstracts from the Literature

Germicides, Liquid

From Health Devices Alerts - Abstracts Current Issue for Friday, June 18, 2004

Device: Luron Nonantiseptic Lotion Soap
Manufacturer: Dial Corp, Scottsdale AZ

Abstract:
The authors assessed the ability of nosocomial bacterial pathogens to persist on fingertips after hand washing with non-antiseptic lotion soap. The fingertips of 2 investigators were contaminated by handling a bacteria-contaminated gauze pad for 10 to 15 sec and sampled by impression plate or hand method. The investigators then washed their hands for approximately 30 sec, with constant rubbing under running water, using a non-antiseptic lotion soap. Hands were dried with a paper towel, and touch imprints were made. The procedure was performed for 5 consecutive hand washes. The gram-positive and negative nosocomial pathogens tested were able to survive and persist on the fingertips of both investigators despite five 30 sec hand washes. Most contaminating bacterial species were removed after the first hand washing, as evidenced by an approximately 106 decrease in colony-forming units. Between the second and fifth washes, the removal rate of persisting organisms slowed. Organisms could still be detected after the fifth washing. There was no difference in persistence between the strains of methicillin- susceptible and -resistant Staphylococcus aureus or between the strains of vancomycin-susceptible and -resistant Enterococcus faecium. For each nosocomial pathogen applied to the fingertips of 1 investigator, transmission by touch to the fingertips of the second investigator was also demonstrated. Transferred bacterium persisted on the recipient's fingertips following 3 additional hand washes. The authors conclude that a single 30 sec hand washing with non-antiseptic lotion soap will not reliably remove all pathogens between patient contacts, and they recommend using gloves and hand cleansing with an antiseptic soap containing chlorhexidine or alcohol.

Source: Bottone EJ, Cheng M, Hymes S. Ineffectiveness of handwashing with lotion soap to remove nosocomial bacterial pathogens persisting on fingertips: a major link in their intrahospital spread. Infect Control Hosp Epidemiol 2004 Mar;25(3):262-8.

Comment: While a specific product is identified in this report, ECRI believes that the intention of the article was not, necessarily, to implicate this particular product and that this problem and/or these results may occur with similar products of other manufacturers.

The Attachment to this email is a copy of ECRI's "Health Devices Alerts © Abstracts" for your information.

These abstracts represent a monthly summary of reported problems, hazards, recall, and evaluations associated with nonimplantable medical devices. Selections are derived from medical, technical, and legal literature; reporting networks; and international governmental sources.

Fixation Methods for the Study of Lipid Droplets by Immunofluorescence Microscopy

Deanna DiDonatoa and Dawn L. Brasaemlea

J Histochem Cytochem 51:773–780, 2003

The study of proteins associated with lipid droplets in adipocytes and many other cells is a rapidly developing area of inquiry. Although lipid droplets are easily visible by light microscopy, few standardized microscopy methods have been developed. Several methods of chemical fixation have recently been used to preserve cell structure before visualization of lipid droplets by light microscopy. We tested the most commonly used methods to compare the effects of the fixatives on cellular lipid content and lipid droplet structure. Cold methanol fixation has traditionally been used before visualization of cytoskeletal elements. We found this method unacceptable for study of lipid droplets because it extracted the majority of cellular phospholipids and promoted fusion of lipid droplets. Cold acetone fixation is similarly unacceptable because the total cellular lipids are extracted, causing collapse of the shell of lipid droplet-associated proteins. Fixation of cells with paraformaldehyde is the method of choice, because the cells retain their lipid content and lipid droplet structure is unaffected. As more lipid droplet-associated proteins are discovered and studied, it is critical to use appropriate methods to avoid studying artefacts.

Influence of slide aging on results of translational research studies using immunohistochemistry

Martina Mirlacher, Marlis Kasper, Martina Storz, Yvonne Knecht, Ursula Dürmüller, Ronald Simon, Michael J Mihatsch and Guido Sauter

Modern Pathology (2004) 17, 1414-1420

Several reports have shown that a long delay between cutting sections and immunohistochemical (IHC) staining can decrease the IHC reaction intensity. However, systematic large-scale studies to investigate to what extent this problem may influence the outcome of translational research studies are lacking. In this study, we used a tissue microarray (TMA) approach to investigate the influence of slide age on comparisons between the results of IHC analyses for estrogen receptor (ER), progesterone receptor (PR), cyclin D1, HER2 (HercepTest), and E-cadherin and clinical outcome in a series of 522 breast cancer patients. Old TMA sections stored for 6 months at 4°C and freshly cut sections were analyzed under exactly identical experimental conditions. As compared to results obtained on freshly cut sections, the frequency of positivity on old sections decreased from 65 to 46% for ER ($P<0.0001$), from 33 to 18.5% for PR ($P<0.0001$), from 16.3 to 9.6% for HER2 ($P=0.0047$), from 45.1 to 37.7% for cyclin D1 ($P=0.10$), and from 58.9 to 32.9% for E-cadherin ($P<0.0001$). Despite the lower fraction of positive cases, most associations between IHC data and tumor phenotype that were observed in fresh section analysis were also found when old section data were analyzed. The results confirm that slide aging has a great influence on the intensity of IHC staining in individual cases, but they also suggest that many clinicopathological associations can be detected if suboptimally processed sections are used for IHC.

Liver biopsies from human females contain male hepatocytes in the absence of transplantation

Anne M Stevens, W Michael McDonnell, Meghan E Mullarkey, Jennifer M Pang, Wendy Leisenring and J Lee Nelson

Laboratory Investigation (2004) 84, 1603-1609

Fetal cells derived from pregnancy can persist in a woman's blood and tissues for decades and have been implicated in the pathogenesis of autoimmune disease. Transplantation studies based on donor sex mismatch suggest that circulating stem cells can lead to liver regeneration with donor-derived hepatocytes. However, male cells in female liver could derive from pregnancy. We investigated male cells in liver biopsies from women with sons and asked whether they were hematopoietic cells or hepatocytes. Fluorescence in situ hybridization for X- and Y-chromosomes with concomitant immunohistochemistry was employed to study 28 female liver biopsies: 14 with the autoimmune disease primary biliary cirrhosis (PBC), eight with Hepatitis C, and six with other diseases. Total male cells and those expressing hematopoietic (CD45) or hepatocyte (CAM-5.2) markers were quantified. None of the male cells were hematopoietic in origin, as shown by lack of CD45 expression. Instead, male cells with hepatocyte morphology expressing the hepatocyte marker CAM 5.2 were found in 25% of all biopsies (36% of PBC and 14% of others). Overall, male cells were found in 36% of female liver biopsies. Of the PBC livers 43% had male cells compared to 25% of Hepatitis C biopsies and 33% of others. There was a trend toward increased numbers of male cells in PBC compared to others (mean 1 per 30 000 host cells vs 0.17 in Hepatitis C and 0.35 in others). Thus, male cells found in livers of women with sons include cells that express hepatocyte antigens. Therefore, transplantation and stem cell differentiation studies using sex difference to conclude that donor cells regenerate liver may be confounded by fetal microchimerism. Whether fetal cells play a role in autoimmune diseases like PBC merits further investigation.

Microwave exposure increases bone demineralization rate independent of temperature.

Tinling, S. P., Giberson, R. T., Kullar, R. S.

Journal of Microscopy. 215(3):230-235, September 2004.

There is a long-standing controversy regarding an effect of microwaves, independent of increasing temperature, on the rate of bone demineralization. In this study, we exposed standardized samples of gerbil femur to constant microwave exposure while maintaining the demineralizing solution (ethylenediamine tetraacetic acid, EDTA) at 20 [degrees]C. Random samples were selected at 3 h intervals, embedded in plastic and sectioned for histological evaluation to determine the extent of demineralization. The time to complete demineralization was significantly faster with microwave exposure (33 h) compared to non-exposure on a tissue rotator (45 h) in a limited amount (5 mL/24 h) of EDTA. The presence of bone marrow was a significant barrier to the rate of demineralization and resulted in an asymmetrical pattern of mineral extraction. Samples without bone marrow were completely demineralized after 21 h of exposure to microwaves and EDTA. Additional comparisons were made between samples exposed to an effectively infinite supply of demineralizing agent (bone marrow intact). There was a significant increase in rate with unlimited demineralizing agent with (24 h) or without (30 h) microwaves when compared to tissue demineralized on the rotator. Our results establish a positive effect of microwaves on the rate of bone demineralization which is independent of temperature.

Disinfecting the Cryostat- The American Response

Thomas A. Merrick (2004) http://www.cap.org/apps/docs/cap_today/q_and_a/qa_05_04.html

A cryostat is considered a high-risk instrument because it uses frozen, unfixed tissue that can contain viable infectious agents. Freezing the tissue does not inactivate infectious agents. In fact, question ANP.24250, which was added to the safety section of the anatomic pathology checklist to address these concerns, asks:

Is there a documented procedure for the routine decontamination of the cryostat at defined intervals and are decontamination records evident?

The explanatory commentary accompanying this question states that the interior of a cryostat should be decontaminated regularly with 70 percent ethanol. Trimmings and sections of tissue that accumulate inside the cryostat should be removed during decontamination. The cryostat should be defrosted and decontaminated with a tuberculocidal disinfectant at a time interval appropriate for the institution-once a week for instruments used daily. Freezing propellants under pressure is not advised because they may cause the splattering of droplets of infectious material.

The cryostat must be clearly marked as contaminated if a frozen section is performed on tissue from a patient known or suspected to be positive for HIV, hepatitis B or C, SARS-related coronavirus, prion disease such as Creutzfeldt-Jakob disease, or mycobacterial or systemic fungal disease. The cryostat must be decontaminated before further use. Laboratories, therefore, may need to have a backup instrument available.

Seventy percent alcohol has been shown to be effective against HIV and probably other viruses. To disinfect for tuberculosis or fungal disease, a lab should use a tuberculocidal or fungicidal disinfectant.

Bibliography

NCCLS. Protection of Laboratory Workers from Occupationally Acquired Infection; Approved Guideline—2nd Edition. NCCLS document M29-A2. Wayne, Pa.: NCCLS; 2001.

Old Farts Reminiscing [Histonet]

I thought I would include some history and thanks to Histonet – straight from the horse's mouth:

"My first encounter was with one Des Brady in Sheffield. He was great. If you asked for a special stain, he would say "do you want it positive or negative boss?" That's what I call skill."

- Dr Terry L Marshall

"….bring back a flood of memories ... the wax tea pot, Leukhart's embedding rings, sticking the blocks onto wooden blocks, taking them off again at the end of the day, etc. Safety precautions had not even been invented in those days. When I first started in Histology, there were five of us in the lab and every single one smoked a pipe. So, embedding, trimming and sticking on the blocks involved three of us ... all chuffing out smoke. The conversations that took place during these times were priceless. Sadly, this opportunity was lost with the advent of automated embedding centres.

All solvents went down the drain; old specimens were dumped into the sink to allow the formalin to drain away. There was no fume hood, so the formalin fumes were thick enough to cut with a knife. In retrospect, dumping solvents and fixatives down the drain was not the best idea! But at the time, that was standard practice. However, despite these horrendous practices, we are all still alive and well, and all went on to accept senior positions around the world.

There were no productivity units to count, no QC/QA demands (apart from self-imposed ones), no intrusions from misguided administrators. We had time to work on our own projects, investigate new procedures, and read journals looking for new methods. We made all of our own reagents from scratch (hematoxylin, Schiff's reagent, fixatives, etc). We sharpened our own knives. The camaraderie was wonderful, there was no bitching or whining, going for a beer at lunchtime was a routine practice. We did a great job, we went home happy, and provided great service

I would not give up the new developments in Histology (immunohistochemistry, monoclonal antibodies, disposable knives, or automated stainers, etc) they have produced quantum leaps in quality and diagnostic accuracy, but I sometimes I despair that the new generation of technologists have missed out on an invaluable learning experience.

- Paul Bradbury

"What about the blistered finger tips from sticking those wax blocks on wooden blocks (the smaller the block-the bigger the blister!), and all the hand processing, which mainly involved much frantic dashing about twirling multiple pots of tissue to provide agitation! Who can forget about mouth pipetting whilst eating sandwiches at the staining bench and brewing the tea over a bunsen during waiting times!"

- Bryan Hewlett

"OK - ! Let's go back into time about 35 years - - How about everyone smoking cigarettes and drinking coffee while cutting blocks - parties in the lab that included food, ethanol and orange juice (the pathologist's idea) - and a diener who could routinely be found sleeping in the walk-in morgue cooler? We had something on our processor between the acetone and xylene that was called "solvent 127". In 30 years, I've never been able to find out what that was - maybe Soylent Green? We occasionally used toluene and dioxane to 'rush process' tissues (no ventilation). How about making Bouin's solution with dry picric acid from a 5 pound jar? Sometimes if you spilled a little of the picric acid, and it was still there the next day, when you walked on it - it made little crackling noises."

- Ian Montgomery

"Ooohhh, I've got really bad memories of Sheffield. Leicester used to send techs on the advanced histology

course there. The Nurses home on the outskirts was where we stayed. After one very good night our with the local techs, who felt obliged to show us all the good pubs, I went for breakfast the next morning and was met by a pan full of tripe and onions, with a similar one next to it full of black pudding- and not a plastic bag in sight!! Some hospitality!

Did anyone else hand process and double embed eyes through celloidin in 1% methyl benzoate? The first time I was asked to do this procedure, I didn't realise you had to use polyproplylene pots and just a clear plastic one. I came in the next morning to find two eyeballs sitting on a shelf, in glistening goo, staring at me.

On very rare occasions, we had a leg amputation brought to the lab fresh from a case of osteosarcoma. We took great delight in scaring juniors to death, by asking for help and then running a scalpel blade over the nerves. No-one actually fainted when the toes curled up, but it was a wonder."

- Stephen Eyres

HISTOTECHNOLOGY GROUP of NSW

ABN: 63 128 868 343

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2004 - 2005

I wish to become a member of the Histotechnology Group of N.S.W. and enclose

PLEASE TICK

- ☐ $38.50 for annual subscription of $35.00 and $3.50 GST.
- ☐ $16.50 for student subscription of $15.00 and $1.50 GST (Full-time or working toward first qualification)
- ☐ $82.50 for company subscription of $75.00 and $7.50 GST (2 representatives, one of whom must be a NSW representative)
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