Histograph

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Newsletter of the Histotechnology Group of NSW

Contents

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Editorial

This year is definitely proceeding too quickly. Our Annual General Meeting is fast approaching (and so is my birthday). Unfortunately I will be unable to attend since the 24th is my birthday. My children have promised a mega-hangover for me on the 25th – on second thoughts I better attend the AGM!

As you may have heard on the grapevine, it has been proposed that we progress from a group to a Society. 30 years on and I think we have matured enough to become a Society, but then respect for the Groups rich history dictates no change. Please consider the resolution and have your say at the AGM.

This issue of Histograph looks at Controversial Artefacts in BCCs, Endogenous Peroxidase and Xylene Safety issues. The Oil Red O Stain is also critiqued and you are asked to consider solutions to problems with these stains. Please email me with your responses.

Tony Henwood,
Editor
anthonyh@chw.edu.au

Histotechnology Group of NSW

Annual General Meeting

FRIDAY, 24 AUGUST, 2012
at North Ryde RSL function room.

6.30pm Start

Chairman's Report

We are now heading into our Annual General Meeting. Most of our current committee is renominating for their positions. We are always looking for new committee members to join us, this is important as a good committee needs "new blood" every election to ensure we have new ideas coming forward. There are still committee positions available so please consider being part of our team.

As part of our process in becoming an incorporated body we need to submit the name we will use. As part of our Annual General Meeting our committee is putting forward a constitutional proposal that our name change from the "Histotechnology Group of NSW" to the "Histotechnology Society of NSW". We feel that we have now gone past a group of people who met and discussed and promoted Histotechnology. We have grown over the years to a large professional body of members where a "Society" better describes us. Each member will have one vote at the AGM; we will need over 75% of the votes for this proposal to be accepted. The committee asks for your support to enable us to implement this change.

Our guest speaker at our Annual General Meeting will be DR Geoffrey Hall, Pathologist at Douglas Hanley Moir Pathology. The topic will be "Coeliac Disease and Inflammatory Bowel Disease". One not to be missed.

Since our last Histograph we have held one workshop and one night time meeting. In May, Tony Henwood presented another one of his popular workshops on "Histo Hypotheticals" at the Children's Hospital at Westmead. This Saturday meeting was well attended.

In July Professors James Lawson and Noel Whitaker [University of NSW], presented their latest findings on "Viruses and breast cancer". This enthralling insight into the possible causes of breast cancer brought people out of the woodwork to hear updates on two previous presentations. Some fifty eight people attended.

Planned for the next six months are more interesting night time presentations and a workshop. Information will be posted on our website as it comes to hand.

A reminder that membership fees are now due.

Cheers,

Trevor Hinwood.
Chairperson.
Histotechnology Group of
NSW.

HISTOTECHNOLOGY GROUP OF NSW
NEW POSTAL ADDRESS

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BCC Clefts – Processing Artefact?

Basel Cell Carcinoma is one of the most common malignancies diagnosed in Australian histopathology Laboratories. Histologically, BCCs are characterized by a proliferation of basaloid cells, arranged in nodules of varying size, often appended to the under-surface of the epidermis and invading the underlying dermis. Tumour nodules show peripheral palisading of cells and nuclei, are surrounded by specialized connective tissue stroma and are often separated from it by optically empty clefts. These are usually regarded as an artefact, resulting from tumour retraction occurring during routine tissue processing (fixation/dehydration/embedding), and their formation has been tentatively explained by the reduced amount of basal membrane protein synthesis by BCC cells, rendering them less adhesive to the surrounding dermis. However, optically empty spaces around tumour islands of BCC are also observed in cryostat sections, suggesting that additional mechanisms might be responsible for this finding (1).

It has been suggested that that these clefts might be of two types (2). The first, a true retraction space, is an artefact produced during fixation and dehydration due to the softness of the epithelial tissues, and the second is a focus of stromal change associated with the synthesis and accumulation of mucinous ground substance, particularly hyaluronic acid and dermatan sulphate. McCardle et al (2) in Melbourne showed that the "retraction spaces" or clefts were of two distinct types. In processing artefacts, ie true tissue retraction spaces, the split occurs between the epithelial cells and the basal lamina. In areas of mucinous change, the accumulation of mucinous material in the dermis immediately adjacent to tumour nests gradually separates the normal stroma from the overlying basal lamina (2).
McCardle et al (2) presented criteria to differentiate tissue retraction spaces from stromal mucinous change (see table).

Laminins are basement membrane glycoproteins consisting of three polypeptide chains α, β and γ. The functional properties of laminins include cell adhesion, proliferation, differentiation, growth and migration. Laminins 5 and 1 are distributed mainly in the skin especially in basement membranes, where their biological functions involve anchorage and locomotion of cells (3). More recently, Bahadoran et al. (4) showed that in most examples of BCC, laminin-5 expression was absent or markedly reduced at the basement membrane zone surrounding epithelial nests. As laminin-5 is the main component of anchoring filaments that connect the hemidesmosomes of basal epithelial cells to the lamina densa, they suggested that the hemidesmosome-anchoring filament complex is not properly synthesized or assembled in BCC. Mostafa et al (3), though, did not find absence of laminin expression on the stromal side of the peritumoral lacunae, thus suggesting that laminin does not play a major role in lacunae formation.

Ríos-Martín et al (5), using Ber-EP4 immunohistochemistry, found Ep-CAM expression on both sides of the clefts in some BCCs. Ber-EP4 (Ep-CAM) stains two glycoproteins located on membrane surfaces and in the cytoplasm of many epithelial cells. In the skin, Ber-EP4 labels normal adnexal epithelium, BCC, Merkel cell carcinoma, trichoepithelioma and various other adnexal tumours. This finding supports the idea that peritumoral retraction (clefts) is a consequence of epithelial membrane breakdown instead of true stromal retraction. This breakdown may be related to an artefact occurring in the weakest zone of the basal epithelium during fixation and dehydration.

Ulrich et al (1), using reflectance confocal microscopy (RCM), showed that hypo-refractile cleft-like spaces observed on RCM imaging correlate to mucin-rich areas seen on histological sections. Mucin deposition around BCC nodules explains why cleft-like spaces may also be observed on cryostat sections. However, additional, larger clefts can be observed on histopathological examination, which are located between the surrounding collagen and the mucin; these could be because of mucin shrinkage and retraction from the surrounding dermis, and seem to represent an artificial phenomenon as has long been suggested.

References

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2. McCardle JP, Roff BT, Muller HK. (1984) "Characterization of retraction spaces in basal cell carcinoma using an antibody to type IV collagen" Histopathology 8: 447.
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Stromal Mucinous Change	**Tissue Retraction Space**
Space lies between stroma and overlying basal lamina	*Space lies between epithelial cells and basal lamina*
Space contains Alcian Blue (pH2.5) positive mucinous material	*Alcian Blue negative*
Space filled with floccular pale pink material on H&E	*Optically empty space*
Circumferential stromal change about tumour cell nests	*Clefting generally restricted to one side of tumour nests*
Peripheral basal cells normal	*Peripheral basal cells adjacent to cleft appear compressed and nuclei stain more intensely*

NATIONAL HISTOLOGY CONFERENCE 2013

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PRELIMINARY ANNOUNCEMENT

NATIONAL HISTOLOGY CONFERENCE APRIL 26-28, 2013

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Start planning your attendance at the National Conference in the April 2013 in Melbourne.

We are calling for proffered papers and posters.

NOTIFICATION OF THE 2012 ANNUAL GENERAL MEETING AND PROPOSED CHANGE TO THE CONSTITUTION

The 2012 Annual General Meeting of the Histotechnology Group of NSW will be held on Friday the 24th of August at the "North Ryde RSL Community Club" commencing 6.30PM.

Members and guests are cordially invited. Note: only current financial members will be able to vote. Membership forms and Committee nomination forms are on our website. Committee nominations need to be forwarded to the Secretary prior to the AGM. Where Committee positions have received no nominations or insufficient nominations, nominations will be taken from the floor.

Following the Annual General Meeting our guest speaker **Dr Geoff Hall** will speak on **Gastric Cancer**. At the conclusion of the presentation finger food will be served.

Constitution Change

The Committee of the Histotechnology Group of NSW proposes the following change to the Constitution.

Change of name from the "Histotechnology Group of NSW" to the "Histotechnology Society of NSW"

Reasoning

We have started the process of becoming incorporated with the NSW Department of fair Trading. As part of this process we need to register the name we will use. Once we register this name it will not be easy to change it in the future.

Many of our committee feel we have now gone past the original formation of a group of people who gathered together to discuss and promote Histotechnology. We have now become a more professional body and recognised as such by many other bodies. A Society [an organisation of persons associated together for religious, benevolent, literary, scientific, political, patriotic or other purposes], indicates a more organised and professional body of people.

Our Committee feels this is an ideal opportunity to make a name change and asks for your endorsement for this change at our Annual General Meeting. Each current member will have one vote on this proposal and must be present at our Annual General Meeting.

Secretary.
Bharathi Cheerala

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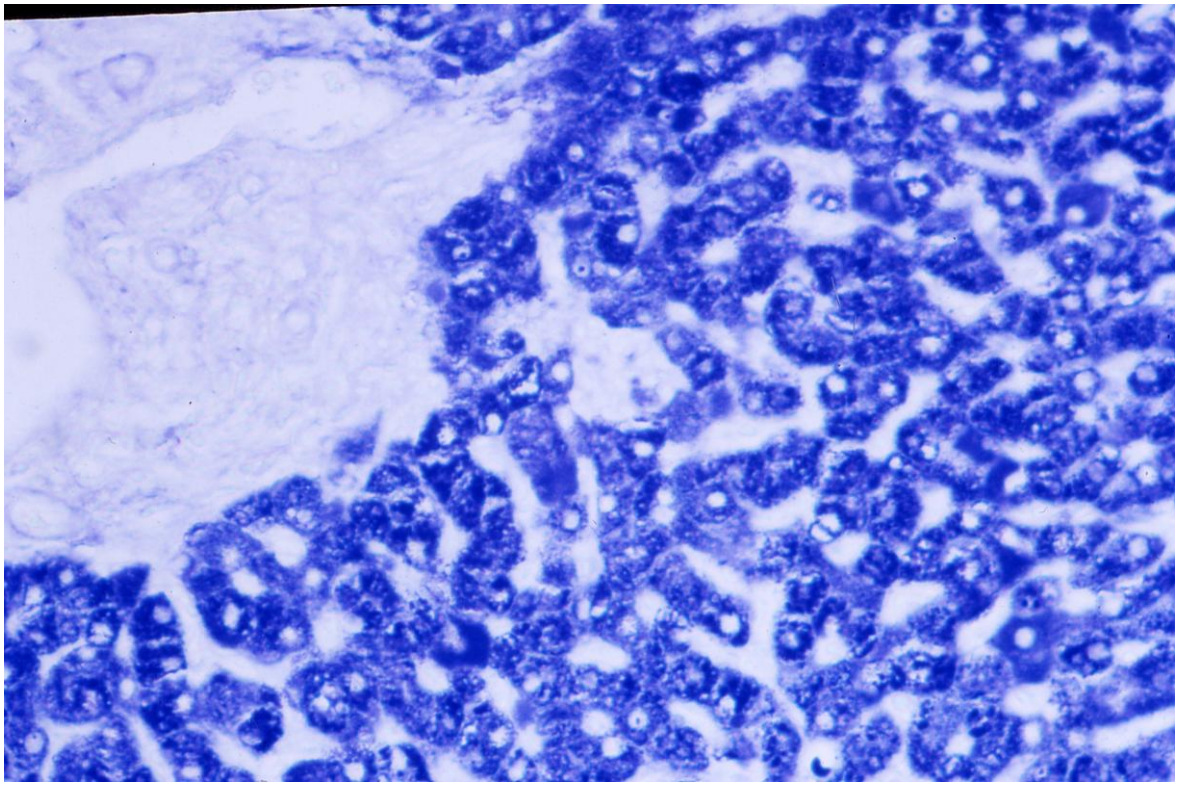
What Stain is this?

What is the tissue?

What is being demonstrated?



Answers next Issue

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Reference: Colley & Stead 2011, Optimized Immunohistochemistry Workflow Facilitated by New Dako Autostainer Link 48 Software
http://www.dako.com/au/index/knowledgecenter/kc_product_info_sites/pathology_instrumentation/dako_autostainerlink_48_prod_info_site/dako_autostainerlink_48_publications.htm

Safety Corner – Raynaud's Phenomenon in Histopathology Workers Who Work with Solvents

Gordon Purdie and his co-researchers at the University of Otago, in Wellington, New Zealand asked all the histology laboratories and cytology laboratories in New Zealand to participate in a study. Every laboratory department in the nation agreed and provided data and access to laboratory workers.

A total of 341 medical laboratory technicians participated in the study, most of whom were women (79%). They found that of those who came into contact with toluene and xylene on a regular basis were twice as likely to develop Raynaud's phenomenon. However, those who worked with acetone or chlorinated solvents combined with toluene and xylene were nine times more likely to develop severe Raynaud's phenomenon.

Traditionally, Raynaud's phenomenon results from exposure to cold or emotional stress that causes blood vessels in the hands, feet or other extremities to spasm. The alternating constricting and dilating of the vessels are called vasospastic attacks. They decrease the blood flow to those areas resulting in discoloration, and in extreme forms, can cause the skin to atrophy and lead to gangrene and necrosis.

This is the first study to demonstrate a link between medical laboratory worker solvent exposure and symptoms of auto-immune connective tissue disease, and has important implications for workplace health and safety.

Purdie et al (2011) "Raynaud's Phenomenon in Medical Laboratory Workers Who Work with Solvents" J Rheumatol 38(9):1940-1946

http://www.darkdaily.com/health-of-pathology-laboratory-technicians-at-risk-from-common-solvents-like-xylene-and-toluene-070511#axzz23DhKGCZZ

Solve a Problem – Fat Staining with Oil Red O

One of the most widely used dyes for staining fat is oil red 0. This dye stains fat more intensely than Sudan III and Sudan IV, which are similar in chemical structure. Sudan III contains no methyl groups, Sudan IV contains 2 methyl groups, and oil red 0 contains 4 methyl groups, all of them in the diazo group of the classification of dyes. These dyes are insoluble in aqueous solutions but soluble in ethanol, isopropanol, propylene glycol, and other organic solvents. Lillie's supersaturated isopropanol method using oil red 0 appears to be the most popular method for staining neutral fats (1).

The demonstration of intracytoplasmic fat at intraoperative diagnosis of parathyroid lesions is valuable in the diagnosis of parathyroid adenoma with oil red O positive fat being seen in normal or atrophic parathyroid but negative in parathyroid adenoma and hyperplasia (2). The presence of fat in the fetal zone of the adrenal cortex in the stillborn macerated fetus is indicative of the mode of intrauterine death. When there is only a scant amount of fat in the adrenal gland, then this indicates an acute mode of death, where as a massive fatty change of almost all cells throughout the fetal cortical zone indicates a chronic mode of death (3).

What suggestions can you make to solve the following problems?

1. Fat in skin sections fails to stain with Oil Red O.
2. The Oil Red O stained sections have been inadvertently over stained with Haematoxylin.

Email your solutions: tony.henwood@health.nsw.gov.au

Answers next issue.

References

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2. Clarke et al (1996) “Atypical fat staining patterns in hyperparathydism” Int J Surg Pathol 3:163-8.
3. Becker MJ, Becker AE. (1976) “Fat distribution in the adrenal cortex as an indication of the mode of intrauterine death” Hum Pathol 7(5):495-504.

Endogenous Peroxidase – Block It

Endogenous peroxidase activity (EP), which is physiologically present in many cells, such as erythrocytes, granulocytes, and neurons, can catalyse the breakdown of hydrogen peroxide and thereby the polymerisation of DAB, thus producing a staining identical to specific immunoperoxidase.

Fixation and embedding processes reduce endogenous enzymatic activity, but the residual tissue activity must be completely blocked to prevent false positivity. For instance, tissues with high blood content (eg, site of heavy haemorrhage), thus rich in haemoglobin or with intense granulocytic inflammatory infiltrate, need a stronger suppression of EP activity (1).

Commonly, the blocking procedures are based on oxidative agents, mainly hydrogen peroxide, but alternative and stronger oxidants such as periodic acid have also been proposed. Treatment with diluted Hydrogen Peroxide is the most recommended and widely practiced procedure for blocking EPs in formalin fixed paraffin-embedded tissue sections (1).

Straus (3) described a complete inhibition of peroxidase with absolute methanol containing 1% nitroferricyanide and 1% acetic acid; but with this treatment the antibody reaction was also weakened.

Streefkerk (4) showed that pretreatment with absolute methanol followed by hydrogen peroxide at a concentration of 0.006% (for unfixed tissue) or 0.0125% (for formaldehyde-fixed tissue) in PBS resulted in a strong inhibition of pseudoperoxidase activity in the erythrocytes. Peroxidase activity of the granubocytes was completely abolished.

Following on from this, a common procedure for blocking EP is to combine hydrogen peroxide and methanol in the same solution (eg 3%) for 5 minutes.

Vacca et al (5) found that reactions using diaminobenzidine (DAB) to localize the enzyme peroxidase in neutrophils and peroxidase-antiperoxidase (PAP) complex during immunological staining are usually performed in Tris-HCI or phosphate buffer at pH 7.2-7.6. However, DAB solutions at pH 7.2-7.6 often demonstrate erythocyte pseudoperoxidase as well. By lowering the pH of the DAB solutions, it was possible to selectively suppress the reactivity of pseudoperoxidase while maintaining optimal reactions in neutrophils and PAP complex. For this purpose they recommend that ammonium acetate/citric acid buffer at pH 5.5 (pH 5.0-6.0) containing 44 mg DAB per 100 ml buffer and 0.003-0.03% with respect to hydrogen peroxide.

It should be recognized that the antigenic reactivity of some cell surface antigens such as CD4 and Pax5 are sensitive to hydrogen peroxide thus requiring blocking to be done after localisation antibody incubation (1).

Cyclopropanone Hydrate

Conventional methods for inhibiting EP depend on complete denaturation or destruction of the heme-containing protein moiety, which not surprisingly has been found to be deleterious to many of the

delicate target antigens, such as lymphocyte surface markers. Therefore, the use of more sensitive reagents for inactivation of the heme group has been attempted for achieving non-deleterious yet broad-spectrum inhibition of EP. Phenylhydrazine, originally recommended by Strauss (2), has proved relatively useful for this purpose (2). It appears to be more efficient when combined with hydrogen peroxide (2). Andrew and Jasani (2) have further enhanced the blocking potential of phenylhydrazine and an analogous compound, sodium azide, by applying them with minute amounts of hydrogen peroxide supplied by the enzymatic action of glucose oxidase on glucose. This system is not only more efficient but is less deleterious to gross tissue morphology and antigenicity (2).

Schmid et al (2) showed that low concentrations of cyclopropanone hydrate in the presence of optimal amounts of nascent hydrogen peroxide was able to substantially reduce the staining intensity owing to EP associated with a wide variety of cell sources. It appears that in the presence of hydrogen peroxide, compound I analogues (a 2-electron oxidized form of peroxidase-bound heme moiety) and primary free radicals of propionic acid (generated from oxidative cleavage of cyclopropanone hydrate) were formed, leading subsequently to alkylation and consequent destabilization of the activated heme moiety.

Microwave Antigen Retrieval

Gao et al (6) have shown that microwave antigen retrieval using citric acid buffer as the retrieval buffer could completely block EP in FFPE tissue sections. They postulate that denaturation of endogenous peroxidase may be the reason that microwave antigen retrieval obviated the necessity of the hydrogen peroxide blocking step.

Jylling et al (7) used frozen sections of lymph nodes that were subsequently formalin fixed, used the cryojane tape system and then blocked endogenous peroxidase with a short microwave heating in TEG buffer. This resulted in morphologic quality almost comparable with paraffin sections. The endogenous peroxidase was effectively blocked, and the true reaction was easy to detect.

References:

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Neuro Histology Review – Glial Cells

I was reading a few recent articles on Neurohistochemistry and came across and article by Rivera-Zengotita and Yachnis (1). Apart from being an excellent review on Gliosis and Glioma, I also found it to have a gem of a review on normal glial histology. I have summarised it below for your educational use and strongly recommend the reading of the whole article from Advances in Anatomical Pathology.

"Astrocytes are the major supportive cells of the Central Nervous System (CNS) and provide diverse functions including glial-guided neuronal migration during development, interstitial fluid and electrolyte homeostasis, and trophic effects on the cerebral microvasculature. Fibrous astrocytes, which reside primarily in the white matter, express glial fibrillary acidic protein (GFAP) in the perinuclear cytoplasm and in markedly attenuated cell processes. The latter extend to the endothelial basal laminae of capillaries and other small cerebral blood vessels. Of note, the water channel protein aquaporin-4 is localized to astrocytic foot processes that are intimately associated with brain microvessels and is believed to function in fluid and electrolyte homeostasis. Antibodies to aquaporin-4 have been identified in association with the neuromyelitis optica (Devic disease) spectrum of demyelinating disorders.

In contrast to astrocytes, the 2 other types of neuroglia undergo more limited reactions to injury. Oligodendrocytes generate and maintain the myelin sheath of larger axons, which mediates rapid neurotransmission. Their degeneration results in local interruption in formation and maintenance of myelin (demyelination), resulting in impaired or blocked impulse conduction along axons. Specific oligodendrocyte injury because of a papovavirus occurs in progressive multifocal leukoencephalopathy (PML). Ependyma and choroid plexus line the ventricles and are responsible for cerebrospinal fluid production, respectively. Ependymal cells undergo necrosis and are not replaced, leaving a subependymal glial nodule (scar).

Microglia are not really "neuroglia" at all. They are resident histocytes of the CNS that interface with the immune system. A specialized type of perivascular microglial cell functions as the antigen-presenting cell of the brain. Activation results in morphologic transformation to so-called "pleomorphic" microglia (or "rod-cell" microglia), which respond to low grade, incomplete necrosis, or chronic infections and have already become capable of ingesting destroyed nerve cell fragments (neuronophagia) or myelin.

Activation to fully developed macrophages occurs with acute, severe tissue destruction, and with active demyelination. Reactive macrophages in brain, however, arise not only from tissue microglia, but also from circulating monocytes".

1. Rivera-Zengotita M, Yachnis AT (2012) "Gliosis Versus Glioma?: Don't Grade Until You Know" Adv Anat Pathol 19:239–249

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