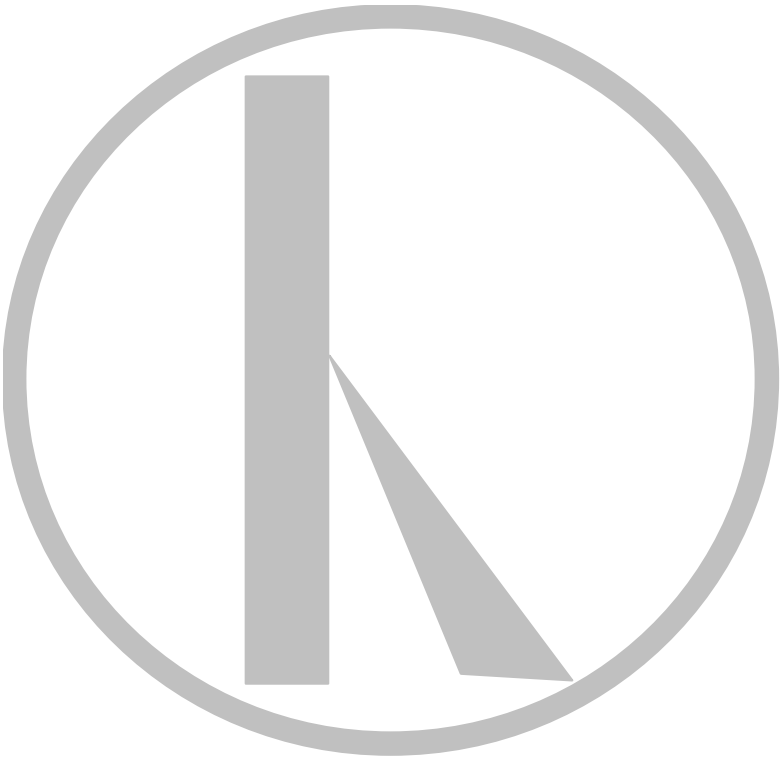
Histograph

ISSUE 1
March 2007



Newsletter of the Histotechnology Group of NSW

Contents

Editorial

We hope you had an enjoyable Holiday break and look forward to a year of Professional Growth. There are many events and meetings planned for the year and if anyone has some ideas for meetings please email me or the Histotechnology Group (nswhistogroup@bigpond.com).

This issue has an excellent review of Rasmussen's Encephalitis by Linda Gomes where the best treatment is removal of half of the brain! Our yearly Update on Amyloid is included (give the editor a break) as well as a reminder on the toxicity of xylene.

We hope you enjoy this issue and have a prosperous year.

Tony Henwood
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Chairman's Report

We are now rapidly approaching our next NSW Conference in Canberra. The dates are the 21st, 22nd and 23rd of September and the venue is Rydges Capital Hill.

There are two Rydges hotels in Canberra so make sure you book into "Capital Hill" and NOT the "Lakeside".

As "Floriade" is being held in Canberra at the same time it will be important to book your accommodation early. Make sure you mention you are with the "Histotechnology Group of NSW" as we have special rates organised with the Hotel.

We have had to make some changes to the planned "Cut Up Workshops". Several issues have arisen in organising a "wet" workshop. Tony Woods and Grant Taggart have gone to considerable trouble to reconfigure the day to ensure attendees will gain a lot of useful information on "Cut up for Technicians".

Our Canberra organisers have done a wonderful job with the remainder of the programme which covers a wide range of interesting and informative topics.

In the meantime we will have a number of informative night time meetings which we invite you to attend.

For further information on our activities, please visit our website www.nswhistogroup.org

Trevor Hinwood, Chairperson, Histology Group of NSW.

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Rasmussen's Encephalitis

Linda Gomes, Science Officer, Histopathology, The Children's Hospital at Westmead.

The human immune system is an amazing system that is constantly on alert 24/7 protecting us against disease. Lots of white blood cells continuously fighting foreign invasion. Now imagine a condition where this awesome system turns against the most powerful and complex organ in the body, the brain. This condition is known as Rasmussen's Encephalitis (RE).

RE is a progressive inflammation of the part of the brain called the cerebral cortex, which is made up of the right and left hemisphere. The disease starts at one part of the hemisphere and spreads to adjoining sides on the same side. Fortunately it does not spread to the other hemisphere. This disease usually develops in childhood, most commonly between the ages of 1 and 10 years. The inflammation usually causes scaring, loss of nerve cells and severe disability (1).

Clinical features

Rasmussen's original description of chronic localized encephalitis reported an inflammatory process, with perivascular cuffing by round cells in both the cortex and white matter (see figure). Diffuse patchy inflammatory changes were seen in the cortex and white matter with prominent microglia in addition to small round cells and occasional polymorphonuclear cells. There was loss of nerve cells, some spongy degeneration, and hypertrophy of the astrocytes, which were also increased in number (2).

Causes

The first meaningful research on RE was begun (unintentionally) by Scott Rogers and Lorise Gahring. They were two neurologists who at the time were measuring glutamate receptor distribution in the brain. Knowledge on RE was very unclear at the time of their research. All that was known was that it was a degenerative disease of the brain that caused seizures, hemiparesis, and dementia in the first 10 years of life. These seizures were not stopped using normal anti-seizures drugs.

The first clue was discovered when Rogers and Gahring were trying to map the distribution of the glutamate receptors (GluR) using antibodies that label the receptor itself. The proteins that make up the GluR are only found behind the blood brain barrier (BBB). Glutamate and a few related amino acids are excitatory neurotransmitters in the central nervous system of mammals. If one of these GluRs happens to wander into the bloodstream that is outside the BBB the body would destroy it immediately thinking it is an outsider. So if these GluRs were put into the bloodstream then the immune system would produce antibodies which could then be used in the searching for the glutamate receptors.

In order to test this theory, the researchers injected the GluRs into the blood stream of normal healthy rabbits hoping to produce useful results. After receiving a few doses of the protein two of the four rabbits started to twitch, as though they were suffering the pain of an epileptic seizure. Now the help of McNamara and Andrews was enlisted.

When McNamara and Andrews examined the brain tissue of the rabbits, they saw what seemed to be a familiar inflammatory pattern. Microscopic examination of the rabbit brains

demonstrated chronic inflammatory changes consisting of microglial nodules and perivascular lymphocytic infiltration mainly in the cerebral cortex as well as lymphocytic infiltration of the meninges, and clumps of immune cells around the blood vessels. RE sufferers had the same microscopic appearance. This appearance is not found in a healthy brain. A healthy brain has its blood capillaries enclosed by the BBB membrane.

The protective BBB can sometimes be breached by a head injury. What then happens is antibodies which are directed against the GluR proteins now have access to the brain. They then commence an attack on all the GluR proteins in the brain.

After more study, McNamara and Rogers decided that these attacks were the cause of the seizures that were often experienced by RE sufferers. If RE was caused by the presence of these antibodies in the bloodstream, then healthy people shouldn't have these antibodies. When this was tested the results showed that Rasmussen's sufferers did have these antibodies in their bloodstream and healthy people did not. Thus when these antibodies were removed by plasma exchange it caused a temporary relief from the seizures but soon the body started producing more antibodies and the seizures returned.

The two main questions that arise are, why does the body mount an immune response against one of its own brain proteins, and how do these antibodies get through the BBB. People produce these antibodies when they are infected by a microorganism that is similar in structure to the GluR. When this happens the body mounts an immune response. If, that at this stage, a person has a head injury, then this will open the BBB to the antibodies and they will attack the friendly GluRs in the brain, causing seizures which further opens the BBB to more antibodies.

Because of this, a rhythmic cycle begins. Antibodies break through the BBB, inflammation results due to the break in, seizures then occur, thus opening up the BBB further, causing more seizures. The inflammation is caused by the autoimmune process against the GluR. All the seizures result when the initial break in the BBB happens due to the head injury. This explains why the seizures are confined to just one area.

The recurrent plasma exchange is a temporary relief. This only stops the seizures for a while, and they will start again when the body makes more antibodies. After a few courses of plasma exchange, the RE sufferers will deteriorate to a point where hemispherectomy (surgical removal of part of the brain) has to be performed. This will render the person prone to mental disability and paralyses (3, 4).

Diagnosis

A serious disease needs intensive investigation. The tests are designed to confirm RE and to exclude other conditions. Diseases that can mimic RE include viral and toxoplasma encephalitis, autoimmune disorders such as vasculitis, and tumours. The most useful investigations are (1):

- Brain scans: MR, SPECT, and if available PET scans are useful.
- Blood tests: These include assays for a range of antibodies and tests to exclude infection.
- Lumbar Puncture: Spinal fluid is examined for evidence for inflammation and infection.
- Brain biopsy. This is needed to confirm the diagnoses.
- Electroencephalogram (EEG). This records the electrical activity of the brain and is used in characterizing the type of seizures the patient has.

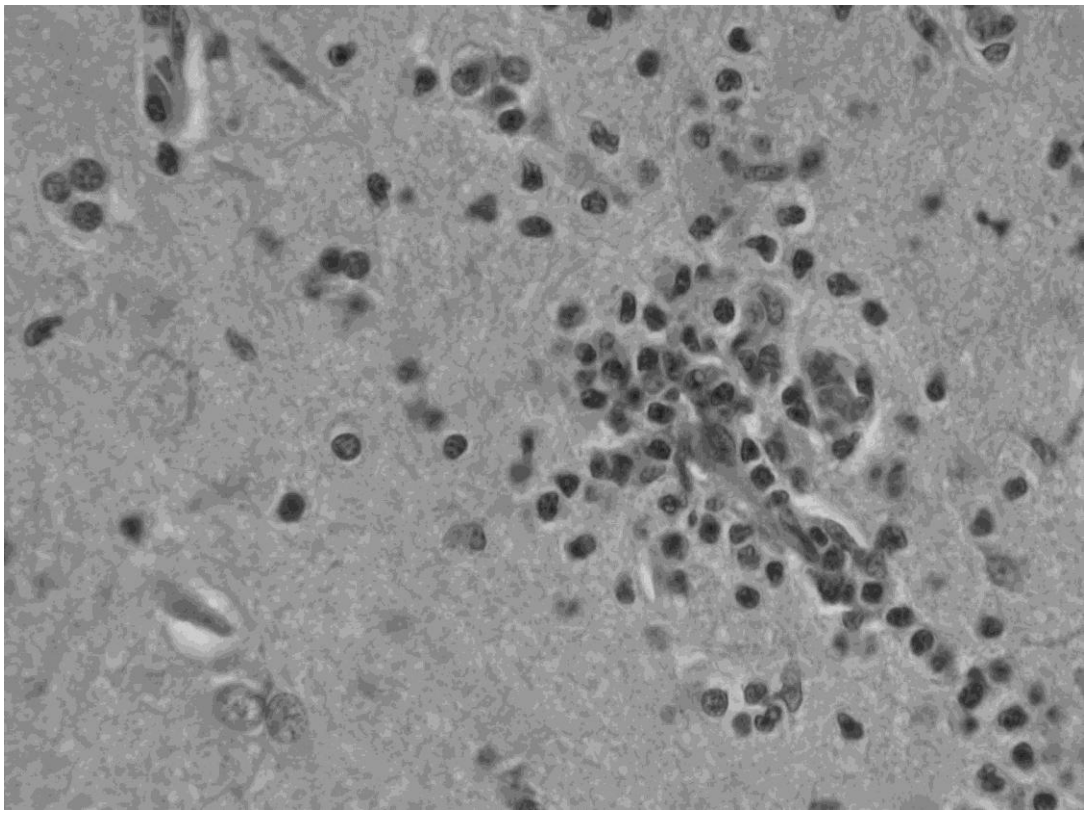
Treatment

Since the seizures do not improve in RE with anti-epilepsy drugs and the disease only ends with the destruction of the affected cerebral hemisphere, surgical removal of large areas, sometimes the entire hemisphere becomes the standard treatment. Trials of various combinations of powerful drugs that suppress the immune system (prednisolone, azathioprine, methotrexate and cyclophosphamide) and therapies that modulate the function of the immune system (PEX and intravenous immunoglobulin) have been tried. However these trials have been done on individual patients and there is no convincing data on the optimum combination, dosing or duration of these treatments. As epilepsy in RE is usually difficult to control, patients often try many anti-epileptic drugs before the best combination is found. These drugs have no effect on progression of the underlying encephalitis.

Currently there are no FDA approved drugs for RE sufferers. Researchers are working on a drug that will block the activity of this particular antibody, but this could lead to further problems. If this drug is being administered and a bacteria or virus with a similar structure as GluR is present, the body would disregard the micro-organism and this could cause other health problems (1).

References

1. Encephalitis-Types of Encephalitis-Rasmussen's Encephalitis by Dr Ian Hart Senior Lecturer in Neurology Neuroimmunology Group, University department of Neurological Science. http://www.encephalitis.info/TheIllness/TypesEncephalitis/Rasmussen.html
2. Robitaille Y. Neuropathological aspects of chronic encephalitis. In: Andermann F, editor. Chronic encephalitis and epilepsy: Rasmussen's syndrome. Boston, Butterworth-Heinemann, 1991:79-110
3. Rasmussens Encephalitis. Anti Essays. Retrieved March 6, 2007, from the World Wide Web: http://www.antiessays.com/free-essays/1324.html
4. Whisenand, "Autoantibodies to glutamate receptor GluR3 in Rasmussen's encephalitis," Science. July 29 1994



Xylene: Its toxicity, measurement of exposure levels, absorption, metabolism and clearance

Editor's Note: This is an abstract from an article published in Pathology, Volume 26, Issue 3 in July 1994 (pages 301 – 309) by Jenifer Langman from the Division of Tissue Pathology, Institute of Medical and Veterinary Science in Adelaide. I believe that it is still quite current and is well worth the read.

Xylene is an aromatic hydrocarbon widely used in industry and medical technology as a solvent. Health and safety authorities in most countries, including Australia, recommend a threshold limit value (TLV) of 100 ppm in the working environment. Recently, the amount of the major metabolite of xylene, methylhippuric acid (MHA), in urine has been recommended as a better indicator of exposure. The American Conference of Governmental Industrial Hygienists has recommended an upper limit for this indicator, called a biological exposure index (BEI), of 2.0 g MHA/L urine (SG 1.016).

Xylene vapour is absorbed rapidly from the lungs, and xylene liquid and vapour are absorbed slowly through the skin. Of the xylene absorbed, about 95% is metabolised in the liver to MHA and 70 to 80% of metabolites are excreted in the urine within 24 hours. However, the many variables which affect the absorption, metabolism and clearance of xylene include exercise, alcohol intake, cigarette smoking, co-exposure to other solvents, gender, and gastrointestinal, hepatic and renal pathology.

Xylene in high concentrations acts as a narcotic, inducing neuropsychological and neurophysiological dysfunction. Respiratory tract symptoms are also frequent. More chronic, occupational exposure has been associated with anemia, thrombocytopenia, leukopenia, chest pain with ECG abnormalities, dyspnea and cyanosis, in addition to CNS symptoms. Concomitant exposure to xylene and other solvents, including toluene, affected hematological parameters, liver size, liver enzymes, auditory memory, visual abstraction, and vibration threshold in the toes. Normal metabolic pathways were altered and significant increases in some serum bile acids may reflect early liver damage. Skin contact has caused burning, erythema and dermatitis. In experimental studies, xylene at about 100 ppm had a deleterious effect on equilibrium, reaction time and manual coordination in non-adapted subjects. At higher concentrations some neurophysiological parameters were altered, particularly when xylene concentration fluctuated. Exercise and alcohol consumption increased blood xylene levels, but altered the neurophysiological effects of xylene in an inconsistent manner. Two reports associating exposure to solvents, including xylene, and increased risk of carcinoma were inconclusive, as were studies on reproduction.

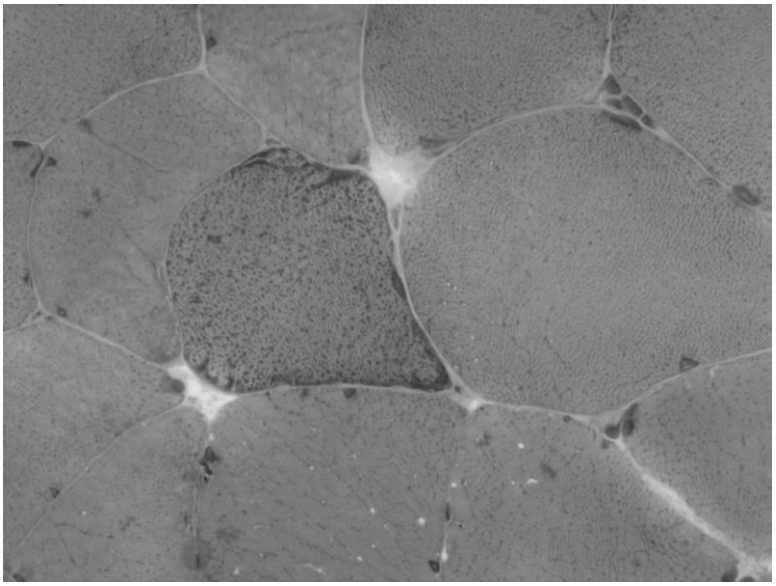
While animal studies fail to provide convincing evidence that xylene is carcinogenic or has significant genetic or reproductive effects, they do confirm that xylene has effects on many organ systems, including the

CNS, liver, kidney, hemopoietic tissues and respiratory tract. However, there are factors which require animal studies to be interpreted cautiously.

Differences are suspected between animal species, and between animals and humans, in the metabolism of, and sensitivity to, xylene. Conditions of exposure to xylene in animal experiments and human studies, both occupational and experimental, are also usually very different.

Muscle Biopsies - An Informative Talk about Muscle Enzymehistochemistry

By Dr. Janice Brewer



Date: Wednesday 16th May 2007
Where: Douglass Hanly Moir,
95 Epping Road
Macquarie Park

Time: **6:30pm**
RSVP: 9th May 2007.
For catering purposes.

Contact:
Di Reader
042397884
eeppleigh1@optusnet.com.au
or Liam O'Donnell
Liam@sydneyskinpathology.com.au

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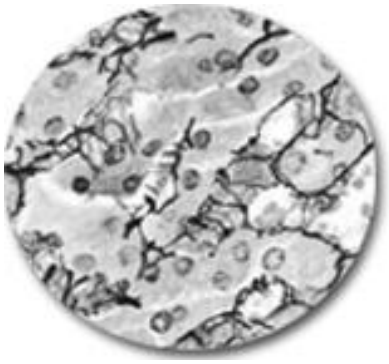
Talk to Lawrence Young after the muscle talk if you are interested.



HD Scientific Advert here

Histotechnology Group of NSW Annual Scientific Meeting

Canberra 21 – 23 September 2007

Provisional Programme

DAY	TIME	TITLE	SPEAKER
Friday	9:00-10:30	Scientific cut up for Technicians. Reviewing the UK system the South Australian course	Tony Woods
	10:30-10:45	Morning Tea	
	10:45-12:30	Scientific cut up for Technicians. Protocols. How, When, Where and Why.	Grant Taggart
	12:30-13:30	Lunch	
	13:30-15:00	Scientific cut up for Technicians. Review of morning sessions including questions.	Tony Woods/ Grant Taggart
	15:00-15:15	Afternoon Tea	
	18:00	Cocktail Party	Trade Display Opening

Note: You will need to register for the whole day. ie. If you do theory in the morning you will do practical in the afternoon and vice versa.

Saturday	8:30- 9:00	Registration	
	9:00- 9:15	Opening	Sat Morning Chair
	9:15- 9:45	Abstracts of Posters	Sat Morning Chair
	9:45-10:00	CSI - Canberra	TV stars
	10:00-10:20	Proffered Papers – 2 x 10 mins	Sat Morning Chair
	10:20-10:45	Trade Talks	
	10:45-11:10	Morning Tea	
	11:10-11:30	Forensic Dentistry	Dr David Griffiths
	11:30-11:50	Difficult Stains – bring your problem stains to us!	Panel Discussion
	11:50-12:10	Histology Education	Panel Discussion
	12:10-13:20	Lunch	
	13:20-13:50	IPX and Flow on Bone Marrow Trephines	Dr Dipti Talaukar
	13:50-14:10	Botanical Histology	Andrew Thornhill
	14:10-14:30	Sarcomas	Dr Chris Hemmings
	14:30-14:50	Proffered Papers. 2 x 10 Mins	Sat Afternoon Chair
	14:50-15:20	Trade Talks	
	15:20-15:45	Afternoon Tea	
	15:45-16:05	Entomological Histology	Eric Hines CSIRO
	16:05-16:40	Drug Development and Oncology links with Histology	Professor Chris Parish with Dr Yip
	17:30	Happy Hour	
	19:00	Dinner "What's at the bottom of your garden"	
Sunday	9:00- 9:30	Registration	
	9:30- 9:35	Opening	Sun Morning Chair
	9:35- 9:55	Tough Love – Preparing bone and other tough tissues	Penny Whippy
	9:55-10:15	Trade Talks	
	10:15-10:35	Debutant Speakers - 2 x 10 mins	Sun Morning Chair
	10:35-11:00	Morning Tea	
	11:00-11:30	Quiz	Sun Morning Chair
	11:30-12:00	Trade Talks	
	12:00-12:20	Why we need re cuts ,etc	Dr Genevive Bennett
	12:20-12:40	Forensic Histology	Elizabeth Brooks
	12:40-13:00	Quiz and Poster results. Close and parting words	Trevor Hinwood

The 2007 Amyloid Update

The last update on Amyloid was given in the April 2005 issue of Histograph. Several interesting developments have occurred since then.

Kebbel & Rocken (1) reassessed the suitability of immunohistochemical classification of amyloid in surgical pathology. One hundred sixty-nine biopsies from 121 patients diagnosed with amyloid during the period from 1994 to 2004 were included. Amyloid was classified immunohistochemically, using antibodies directed against amyloid P-component, AA amyloid, apolipoprotein AI, fibrinogen, keratoepithelin, lactoferrin, lysozyme, [beta]2-microglobulin ([beta]2M), immunoglobulin-derived [lambda]-light and [kappa]-light chains, and transthyretin. Amyloid was most commonly present in biopsies from the hepatogastrointestinal tract.

The deposits were classified immunohistochemically in 156 (92%) biopsies. In 13 biopsies of 12 patients, amyloid remained unclassified. AL amyloidosis was diagnosed in 76 (45%) biopsies and was further categorized into AL amyloid of kappa-light chain origin [32 (42%) biopsies] or lambda-light chain origin [20 (26%)]. In 24 (32%) biopsies, the amyloid deposits did not show unequivocal staining for lambda-light or kappa-light chain. However, these cases were categorized as "probably AL amyloid, not otherwise specified", because no other antibody showed unequivocal staining of the amyloid deposits. AA amyloidosis was diagnosed in 32, ATTR amyloidosis in 21, and AApoAI amyloidosis in 3 biopsies. Other types of amyloid included AKer and ALac amyloids each in 1, and ALys and ACal amyloids each in 2 biopsies. A[beta]2M amyloid was not diagnosed in any case.

Immunohistochemical classification of amyloid still poses problems. Although classification of AA, AApoAI, ALys, ALac, and ATTR amyloids is relatively straightforward, classification of AL amyloid and rare hereditary amyloidoses is a serious obstacle and sometimes even impossible when conclusive clinical information or additional protein biochemical or molecular biologic studies are not available (1).

Linkea et al (2) state that classification of every individual case of amyloid disease is necessary in order to recognize its origin and its possible pathogenesis for therapeutic consideration. Classification of the amyloids can be performed in different ways. One method primarily exploits serum proteins-but these are risk factors only, and therefore render only ancillary information. In principle, one cannot establish the diagnosis alone through their use. Another approach analyses the origin of the deposited amyloids, either by extracting the amyloid proteins followed by immunochemical or chemical analysis, or by using immunohistochemistry.

Based on chemical analysis of prototypes of amyloid fibril proteins, Linkea et al (2) have developed a profile of antibodies over the years that specifically identify amyloid in tissue sections. These antibodies have been used for years as a routine service for clinicians and pathologists in immunohistochemically classifying amyloid found in formalin-fixed tissue sections. The typing is always controlled by established amyloid classes. In several cases, they have been asked for a second opinion on a diagnosed amyloid class. Their own immunohistochemical data were then compared with those submitted. These

submitted immunohistochemical results represented misdiagnoses of amyloid classes in most patients, since the technique performed was usually incomplete. Some of their findings were:

- Amyloid in tissues is not a single, pure substance that can be identified by a single all-or-nothing signal.
- The reaction with a single antibody on a structure that does not present a single protein can never be decisive, since even a single antibody has two different kinds of reactivities towards various amyloids on tissue sections; that is, the strong consistent diagnostic reaction that identifies the amyloid class and the various weaker, inconsistent, non-specific reactions. Without a comparison using a panel of appropriate antibodies, there is no way to differentiate between the two possibilities.
- Multiple reactions seen in amyloids require either micro-extraction or a comparative immunohistochemical approach.

Bély & Makovitzky (3) have found that when using a less sensitive staining method, some true positive cases of amyloidosis remain undetected. A more highly specific method potentially detects more cases and reveals amyloidosis in an earlier stage of deposition.

In their paper, the Congo red staining method according to Romhányi is discussed in comparison with Puchtler's and Bennhold's methods. Using Romhányi's technique, there is no alcoholic differentiation, and thus no dye molecules are washed off the amyloid filaments. The binding of the oriented dye molecules is optimal for polarization microscopy. With this method, the polar hydrophilic mounting medium, gum Arabic is used. Mounted in this carbohydrate-containing, hydrophilic medium, the Congo red molecules are oriented parallel to the surface of the amyloid filaments and the sign is linear positive, corresponding to an additive character of topo-optical staining reactions. Otherwise, the Congo red molecules are oriented perpendicular to the surface of collagen, reducing the intensity of birefringence and even inducing an inversion of the original sign of the collagen birefringence.

With alcoholic differentiation, Congo red dye molecules are extracted and this decreases the birefringence of amyloid deposits, i.e. minimal amyloid deposits may be missed. Using the apolar hydrophobic mounting medium, Canada balsam, an axis-parallel arrangement of Congo red dye molecules on the surface of collagen fibres and amyloid will occur, resulting in an additive topo-optical reaction with a green polarization colour and a false positive diagnosis of amyloidosis (“phantom amyloidosis”).

References:

1. Kebbel & Rocken (2006) Am J Surg Pathol 30(6):673-683.
2. Linkea et al (2006) Acta Histochemica 108(3): 197-208.
3. Bély & Makovitzky (2006) Acta Histochemica 108(3): 175-180.

The Deadly Broomstick and Pink Teeth – Forensic Abstracts

It is often educational to peruse the Forensic Journals to keep up with what our colleagues are up to.

Pink teeth have most often been observed in victims of drowning but have also been reported in subjects who died suddenly and unnaturally. There is general agreement that there is no obvious connection between the occurrence of pink teeth and the cause of death, but the condition of the surroundings (especially humidity) must certainly play an important role in the development of the pink-tooth phenomenon. Campobasso et al (1) studied the frequency and distribution of post-mortem pink coloration of the teeth among a representative sample of 52 cadavers. All the bodies were victims of a single shipwreck that occurred on March 13, 1997, in the middle of the Otranto Canal (Mediterranean Sea). The bodies were recovered from the seawater after approximately 7 months. A distinct pink coloration of the teeth was found in only 18 cadavers (13 females and 5 males) of ages ranging between 13 and 60 years. The phenomenon was more pronounced in younger individuals due to age-related changes of the root canal, less penetrable by the pigment responsible for the post-mortem pink staining. Using histochemical methods and autofluorescence, Campobasso et al (1) identified haemoglobin and its derivatives as the most likely pigments responsible for this post-mortem process that can be considered analogous to post-mortem lividity. This data is consistent with previous reports on pink teeth, indicating that the diffusion of the blood in the pulp into the dentinal tubules causes the red discoloration of the teeth. Based on the results, the pigmentation is more prominent on the teeth with single roots rather than in the posterior teeth with multiple roots.

Byard & Tsokos (2) reported the autopsy findings of a pilot and his passenger who were killed on impact with the ground when their light aircraft crashed. Both deaths were caused by extensive injuries involving severe craniocerebral, skeletal, soft tissue, and organ trauma. In both victims, the legs were shortened, with stripping of muscle and soft tissues from the shafts of the lower limb long bones. In addition, fragments of distal tibial shaft had been forced through the soles of the victims' shoes. This sign indicated a fall from height and showed that the direction of the decelerative forces had been along the axis of the legs and that the force of impact was severe enough to cause fracturing of the lower limb bones, with subsequent extrusion of bone fragments downwards through the shoes. When present, Byard & Tsokos (2) believe that this observation represents another feature at autopsy that can add to the understanding of the circumstances of a fatal air crash and the position of the victims immediately prior to impact.

Heat stroke is the most serious and potentially life-threatening condition of the heat-related illnesses. Heat stroke deaths caused by electric blanket are rarely reported (3). Zhou et al (3) reported two cases of fatal heat stroke caused by overheating from electric blankets in winter. One was a 41-year-old man who was found unresponsive in bed on an electric blanket. His wife shared the same bed with him and was found unconscious. The wife's axillary temperature was 40^{o}C when she was admitted to the hospital. She fully recovered after medical treatment. The husband was pronounced dead at the scene, with a rectal temperature of 41.2^{o}C. The other case was a 13-year-old

girl who was found dead in bed on an electric blanket, with rectal temperature at 41^{o}C. Zhou et al (3) stress the importance of scene investigations and post-mortem examination.

Bolliger et al (4) described a forensic case where a 51-year-old man was struck by the tip of a broomstick weighing 1000 g at the left side of the neck, upon which he collapsed. Intense but delayed cardiopulmonary resuscitation restored the circulation roughly 30 minutes after the incident. Upon admittance to a nearby hospital, an extensive hypoxic cerebral damage was diagnosed. Death due to the severe cerebral damage occurred 5 hours after the incident.

An autopsy demonstrated a severe subcutaneous traumatization of the left side of the neck, with a haemorrhage compressing the carotid bifurcation. A prolonged excitation due to this ongoing compression of the baroreceptors in the carotid sinus was assumed to have led to a cardiac arrest.

In this case report, Bollinger et al (4) discussed the underlying pathophysiology of this potentially lethal and rare reflexogenic incident also known as the Hering reflex and discussed possible therapeutic measures.

References:

1. Campobasso et al (2006) Am J Forensic Med & Path. 27(4):313-316.
2. Byard & Tsokos (2006) J Forensic Med & Path. 27(4): 337-339.
3. Zhou et al (2006) J Forensic Med & Path. 27(4): 324-327.
4. Bolliger et al (2006) J Forensic Med & Path. 27(4): 304-306.

What is happening this Year

16 May	Muscle biopsies
13 June	Nerve biopsies
11 July	Prostate antibodies; AGM
21-23 September	Canberra conference
November	Christmas dinner and trivia night
8-9 March 2008	Newcastle Meeting

Histotechnology Group of NSW Membership Application

2006 - 2007

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

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