

Dear Fellow Clotter

Welcome to the second newsletter for 2005. This issue is a little later than usual due to the timing of the ISTH. Many of you were able to attend the congress and the SSC meetings, but I am sure you didn't get to all the sessions you wanted to. This issue will feature reports on the recent meeting from five of our colleagues. I am indebted to them for their hard work in producing quality reports in such a short time. Thankyou to Emmanuel Favaloro, Mark Smith, Bronwyn Williams, Michael Ray and Murray Adams.

Please take time to check out the latest on the HAA to be held in Sydney mid-October from Chris Ward.

Also in this issue of the newsletter is a clinical trials group report from Tim Brighton, a message from the President, Hatem Salem and news on a potential new association between the ASTH and the British Society of Thrombosis and Haemostasis (BSTH) from Ross Baker

Thanks to all the contributors, I am very grateful for your efforts

Emma Perrin

COME TO THE ANNUAL SCIENTIFIC MEETING IN SYDNEY, OCTOBER 16-19

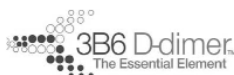
This year's combined haematology society meeting (HAA 2005) is the final chapter in an amazing twelve months for Sydney, following on from the successful international meetings on antiphospholipid antibodies, myeloma and the "mother" of all meetings, ISTH. The HAA meeting will be an ideal opportunity to review the clinical and research highlights of the haematology year, and will feature a strong programme in coagulation. We have several excellent international speakers to discuss the role of cell-derived microparticles in disease (Jean-Marie Freyssinet), new platelet agonists and inhibitors (Ken Clemetson) and management of coagulopathies (Lynn Boshkov). Agnes Lee from McMaster University will review the management of thrombosis in cancer and her masterclass on difficult coagulation cases is sure to be a highlight. This year's meeting will focus also on the excellent work from Australia's up and coming coagulation researchers and hospital scientists, to be presented in three free communications sessions, with the top abstracts featuring in the Presidential symposium. Come along and support your colleagues, enjoy the science and meeting friends old and new – and for anyone who missed out on ISTH, here's the ideal opportunity to catch up on what's new in clotting.

Chris Ward

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FROM THE PRESIDENT

Dear Colleagues,

Well doesn't time fly? It seems like yesterday when Sydney was announced as the 2005 host for the ISTH congress. Many meetings later and long hours of work by many committed members of our Society, the meeting came, and what a meeting it was. While now it is destined to history, the memories will live long. Everyone I spoke to was full of praise of the meeting. No one could fault any aspect of the event; especially the opening ceremony and the Congress party, both are etched in our minds eternally. As a society we are very grateful to Colin Chesterman for his astute and skilful stewardship and the very many long hours of work he put in organising the meeting. We also acknowledge the professionalism of all members of the Professional Conference Organiser; Event Planners, what a wonderful job they did.

During the course of the meeting, members of the Council had the opportunity to meet with Mike Greaves, President of the British Society for Haemostasis and Thrombosis, as well as members of the Council of the Asia Pacific Society for Hemostasis and Thrombosis. The discussions were very fruitful and conducted in high spirits. I am confident that some very useful and exciting outcomes will follow these discussions. I hope to report to you in more detail when we meet in Sydney.

As anticipated the scientific program for our annual meeting in Sydney, looks exciting. I am particularly looking forward to the Barry Firkin oration which will be delivered by Peter Castaldi. Peter is one of our senior members, and has had a long association with Barry. It is therefore very fitting that he was selected as the second Barry Firkin orator.

Prior to our Sydney meeting, the council of our society will be meeting with council members of the HSAZ and ANZSBT. The discussions will focus on ways by which these societies can work together to benefit the membership at a large. Central to our discussions will be the way we run our annual scientific meetings. If you have any suggestions, please do not hesitate to email me.

I look forward to seeing you all in Sydney. I hope that many of you will make a special effort by attending the Barry Firkin Oration and the Annual General Meeting.

Hatem Salem

CLINICAL TRIALS GROUP

The ASTH Clinical Trials Group (CTG) met at the ASPIRE Investigators meeting in May 2005 and also during the ISTH in Sydney during August 2005.

The ASPIRE study is building momentum. This study examines the benefits of low-dose aspirin as prophylaxis against recurrent venous thrombosis after initial warfarin therapy in patients with unprovoked DVT or pulmonary embolism. There are now 185 patients enrolled from 20 actively recruiting sites. A further 20 or so sites will be actively participating by the end of 2005. The Trial Management Committee is committed to find 50-60 sites across Australia and New Zealand so more sites are welcome to join the project. The recruitment period will be about 2 years so ending December 2007. The companion study in Italy, the WARFASA Study, is recruiting and preparation for the planned meta-analysis of both studies is well underway. Further international collaborations are being explored in the UK and other European and Nth American centres.

A major discussion topic in the management of patients with vein thrombosis is the prediction of recurrence. A sub-study of the ASPIRE study, the PREDICT study, will be examining the ability of residual thrombus, plasma D-dimer, and other clinical and laboratory parameters to predict late recurrence of vein thrombosis. I attended an interview with the National Heart Foundation and we are hopeful that funding for the PREDICT study will be forthcoming.

Primary Immune Thrombocytopenia remains an interest for the ASTH CTG. There are now at least 2 novel small molecular weight peptides with thrombopoietin agonist activity in clinical studies. Members of the CTG met with AMGEN during the ISTH meeting and hopefully will initiate an ITP registry in Australia and New Zealand as well as future collaborations with development of AMG531. The protocol for the randomised study of oral dexamethasone versus oral prednisone for acute initial therapy of adult ITP, the ASTH ITP1 study, is almost complete. There was a firm commitment by the group to proceed with this study.

An ASPIRE update/cocktails/dinner is being planned for the HAA meeting in Sydney during mid-October. An ASTH CTG meeting is also being planned for this time.

The ASTH CTG is always keen to receive new members and new ideas. Interested people or any enquiries may be directed to Tim Brighton (t.brighton@unsw.edu.au).

Tim Brighton

ASTH AND BSTH ASSOCIATION

A productive interchange of ideas was discussed recently between the President of the BSTH (Professor M Greaves) and the ASTH (represented by Professor H Salem, Dr T Brighton and A/Professor R Baker) during the ISTH August 2005 meeting. It was a well received interaction and the issues discussed include:

- Linking of ASTH/BSTH web pages and member discussion groups
- Sharing of newsletters/meeting announcements
- Position exchange in the UK/Australia/NZ for scientists/ registrars (6 to 12 months)
- Advertisement of locum and permanent positions of consultant, scientists and post doc positions (email notification for members)
- Participation in each Society's ASM
- Scholarships for young investigators
- Establishing Thrombosis registries of uncommon disorders (eg. Axillary vein and/or portal vein thrombosis)
- List and localities of clinicians in Australia, New Zealand and UK with an interest in thrombosis and haemostasis.

Stay tuned for the developments and if there are further ideas please let the ASTH council know.

Ross Baker

51st ANNUAL SCIENTIFIC SUBCOMMITTEE MEETING AND XXth CONGRESS OF THE INTERNATIONAL SOCIETY OF THROMBOSIS AND HAEMOSTASIS SYDNEY – 6th-12th AUGUST, 2005

BY MURRAY ADAMS

The 51st Annual Scientific Subcommittee Meeting and XXth Congress of the International Society of Thrombosis and Haemostasis (ISTH) were recently held in Sydney from the 6th to 12th August, 2005. The more than 4000 delegates attending the meeting at the Darling Harbour Convention Centre were greeted with magnificent weather and hospitality during the week. I am always amazed and somewhat daunted by the sheer volume of presentations and posters at the ISTH and with concurrent sessions throughout the meeting it paid to be well prepared at the start of each day. In this report I will summarise my observations of the meeting across a range of my own interests, with an emphasis on tissue factor (TF), tissue factor pathway inhibitor (TFPI) and the antiphospholipid syndrome (aPS).

It is always a pleasure to listen to the experts talk about their work and interests and their ability to summarise and identify the most significant aspects of complex areas of science is much admired (at least by me!) Some of the Plenary and State of the Art Sessions were highlights of the meeting. David Ginsburg from the University of Michigan (USA) spoke about the genetics of

various haemostatic disorders (“Identifying Novel Genetic Determinants of Hemostatic Balance”), providing a summary of the variability in single gene disorders of haemostasis and coagulation, the role of ADAMTS-13 in TTP and the role of genetic and environmental factors in combined FV and FVIII deficiency. From a personal point of view it was interesting to listen to the potential significance of TFPI polymorphisms in humans, particularly when an individual also has FVL. Ginsburg summarised experiments where mice homozygous for FVL or heterozygous for TFPI demonstrated normal survival, whereas a lethal phenotype was demonstrated in mice heterozygous for TFPI with a background of FVL.

Philip de Groot from the University Medical Centre Utrecht (Netherlands) started his State of the Art Lecture (“Pathophysiology of the Antiphospholipid Syndrome”) with reference to the new criteria for the diagnosis of aPS, which are currently being reviewed for publication. However, he changed track from a diagnostic theme to summarising the literature in relation to possible pathogenic mechanisms in aPS. The role of dimeric β_2 GPI stimulation of TF expression in endothelial cells has been described by numerous groups in the literature but it was still interesting to listen to this work and also on the roles

of receptors involved in β_2 GPI binding to cells e.g. toll-like receptor 4, annexin A2, LDLR members and Gp1b α . He later proposed that binding of antiphospholipid antibodies probably involves a variety of multi-ligand receptor families on a number of different cell types and that this results in responses through cell signalling that are specific for the cell type.

Graeme Hankey from Royal Perth Hospital (Australia) delivered an excellent overview of causal and treatable risk factors stroke ("Preventable Stroke and Stroke Prevention"). His ability to summarise large quantities of data from the literature made for easy understanding of a complex topic. As well as summarising risk factors he spent some time on treatment strategies and spoke of the probable increasing incidence of stroke in the near future due to the adaptation of Asian countries to western diets and lifestyle.

A couple of interesting talks during the Oral Communications investigated the effects of prolonged immobilisation on coagulation activation. I went along to this session interested to hear if there were any clearer developments from the late 1990's – early 2000's in this area. The WRIGHT Volunteers Study (Netherlands) reported activation of the coagulation system e.g. increased T-AT complexes, in some individuals after 8 hours flying, but not in individuals watching an 8 hour movie marathon or during daily life and suggested that there may be different mechanism(s) underlying air travel related thrombosis than immobilisation only. Conversely, Stricker *et al* (Switzerland) reported increases in FV and FVIII in individuals "subjected" to 6 hours immobilisation by sitting in a chair, but was offset by significant increases in TFPI and antithrombin with no change in ETP compared to controls. Not surprisingly there were more volunteers in the audience to participate in the former and not the latter study!

There were many other interesting talks throughout the Congress which I can't elaborate on due to space constraints, but it was clear that great variability exists in methods for the measurement of thrombin generation, TF associated with MPs, TF activity and ETP that require further investigation and standardisation. The poster sessions held at the end of each day were a great opportunity to seek out other scientists with similar

interests over a couple of drinks which made for a constructive but relaxed end to what was invariably a busy day

The week wasn't all work fortunately, with many highlights during the social programme. Sydney itself, for visitors anyway, is a real treat with Sydney Harbour, the Opera House and Harbour Bridge providing a magnificent backdrop for the Congress. Other highlights included the Welcome Reception, with James Morrison's sublime musical talents and other fabulous entertainers and later in the Week the Congress Party held at the International Passengers Boat Terminal – what an inspired night by the organisers! The only downside of the entire week was when the Aussies couldn't hold on for three more runs in the second test at Edgbaston, which made for a long night at dinner with my ex-colleagues from England!

Thank you to the organisers for a superb meeting and we wait in anticipation for Geneva 2007.

Murray Adams

BY BRONWYN WILLIAMS

VTE and paediatric malignancy

CVL thrombosis is associated with increased risk for bacterial line infection; pulmonary embolus (16%); postphlebotic syndrome (12%) and death (4%).

The true incidence of this complication is poorly defined. Its occurrence appears influenced by cancer type and by treatment protocol. The reported incidence is influenced by the diagnostic technique used; the site of thrombosis and by whether investigation is performed to detect silent or symptomatic thrombosis.

The highest rates of VTE are described in Wilms tumour (~43%) with lower rates in sarcomas (~20%); haematological malignancies (~20%) and brain tumours (~10%). Older age and advanced disease appear to confer a greater risk, particularly with sarcoma and ALL. The incidence of inherited thrombophilia is quite variable.

The occurrence of thrombosis is associated with reduced survival in brain tumour patients and a trend to worse outcome in sarcoma patients. In ALL the occurrence of VTE is associated with asparaginase treatment, but additional

factors relating to dosing schedule and the type and dose of corticosteroid agents used appear to affect incidence.

CVL thrombosis is not routinely looked for in most centres. It has been found in ~ 50% of cases where screening venograms were done. Studies have shown that upper system CVL thrombosis is more frequent with percutaneous lines (OR 3.1); subclavian (OR 3.1) and/or left sided placement (OR 2.5). Femoral lines are associated with a greater risk of thrombosis than subclavian lines. The occurrence of CVL dysfunction may be an indicator for risk of CVL thrombosis and should prompt screening.

Treatment with LMWH is recommended for a minimum duration of 3 months. Non functioning lines should be removed once the patient has been anticoagulated for 3–5 days. In patients where the lines are not removed, longer term therapy may be indicated. Reduction or cessation of LMWH is required during chemotherapy induced thrombocytopenia.

Fetomaternal Alloimmune Thrombocytopenia

Anti HPA – 1a: Management

Investigation of the utility of quantitation of antibody concentration using MAIPA technique in the management of high risk pregnancy failed to demonstrate a correlation with neonatal/fetal platelet counts. Studies measuring changes in antibody titre have also failed to demonstrate a correlation with severity. It is suggested that severity may be affected by placental, fetal and antibody affinity/function related factors. Further large prospective studies are needed to further elucidate the mechanisms of severe disease.

Treatment of the infants has historically included the use of HPA-1a negative platelet concentrates. This specific product is not always readily accessible. Several reports have shown efficacy of random donor platelets in producing platelet increments in affected infants. In some cases multiple platelet transfusions were required. There have been no reports of adverse events to date. Further study is warranted.

FMAIT diagnosis

In infants with severe thrombocytopenia, it is important to remember that negative screening for the common antibodies does not exclude the diagnosis of FMAIT.

Some HPA antibodies are difficult to detect. This applies to HPA 3-a antibodies. The antibody is best detected in whole platelet studies with homozygous platelets which are <7 days old. Other cases of FMAIT are related to antibody to low frequency antigens (HPA 6-14 and 16). These antibodies will not be detected by most screening laboratories. Cell lines have been developed but routine screening is not yet available. There is ongoing work aiming to provide appropriate screening and diagnostic tests for antibodies to these antigens. Genotyping techniques are also being developed.

Bronwyn Williams

BY MICHAEL RAY

One area of my research involves the prevention of thrombus formation during and after percutaneous coronary intervention (PCI). A plenary session of particular interest was Denise Wagner's presentation on the P-selectin, tissue factor coagulation triad.

P-selectin is an adhesion receptor found in the alpha granules of platelet and the Weibel-Palade bodies of endothelial cells. It also exists as soluble P-selectin in the plasma, levels being increased in coronary artery disease. P-selectin is expressed on activated platelets and endothelial cells and supports leucocyte rolling by attaching to a ligand on the leucocyte surface called P-selectin glycoprotein ligand 1 (PSGL-1). PSGL-1 is also present on platelets to a lesser extent and is thought to support P-selectin dependent platelet rolling.

Thrombi have been seen to contain microparticles (MP) expressing the tissue factor that is necessary for thrombus formation. Microparticles are vesicles released from endothelial cells, leucocytes or platelets after activation or during apoptosis. This presentation raised the possibility that during PCI, P-selectin is involved in thrombus formation by this recruitment of platelets, leucocytes, and tissue factor carrying MP to the coronary artery wall which has been injured not only by atherosclerosis and plaque rupture but also by inflation of the balloon catheter. Tissue factor pathway inhibitor (TFPI) balances the procoagulant effect of tissue factor.

Animal studies have shown P-selectin expression potentiates thrombus formation. P-selectin is involved in

the formation and recruitment of monocyte derived microparticles which contain tissue factor. P-selectin on activated platelets and endothelial cells, as well as the soluble P-selectin shed from these cells, bind with the PSGL-1 on monocytes (eg formation of platelet-monocyte aggregates) and shed tissue factor laden MP which bind via their PSGL-1 to the P-selectin positive platelets in the thrombus. This knowledge of tissue factor's role in thrombus propagation offers a target for new therapies to inhibit or promote clotting.

And so back to the flow cytometer with new ideas from the ISTH to explore this complex and fascinating area.

Michael Ray

BY MARK SMITH

In this review, I cover three areas: TF/VIIa, haemophilia inhibitor management and coffee.

The physiology and wide ranging relevance of tissue factor (TF) interaction with factor VIIa received substantial focus at ISTH 2005. Animal models were described that allow for dissection of this crucial interaction in a range of areas, including tumor metastasis. Dr Mackman from La Jolla presented to a full auditorium on the role of TF in haemostasis. He showed data on the effect of TF knockout on mice with or without additional TFPI knockout. He demonstrated an interesting haemorrhage-mediated sclerotic effect on cardiac muscle in the TF knockout model, "rescued" with TFPI deletion. He extrapolated this observation to the skeletal muscle bleeds seen in haemophilia. He then presented animal model data on the effect of membrane-tethered anticoagulants (hirudin and TFPI), and beneficial effect on thrombosis, metastasis, inflammation and angiogenesis. Dr Peppelenbosch from the Netherlands presented data on VIIa-induced intracellular signalling profile via PAR 1, 2 and 4, comparing with the profiles induced by Xa and IIa.

These themes surfaced again at the "Cancer and Thrombosis" oral communication session. TF / VIIa are activators of effector systems involved in inhibition of apoptosis. In this way, metastatic and angiogenic functions are promoted. Simvastatin reduced in vitro cancer cell line expression of TF mRNA and TF antigen, promoting apoptosis with potent influence on malignant cell

behaviour. ATRA reduces TF and VEGF expression and reduces the angiogenic effect of tumor cell lines on endothelial cells. Additionally, inhibition of the rennin-angiotensin system by ACE inhibitors reduces TF expression by breast tumor cell lines.

Kathy High presented on the effects of long term augmented FVIIa expression through gene transfer and transgenic mouse models. Both these models have functional VIIa secreted from the liver. "High secretor" mice (VIIa greater than 2ug/ml) suffered increased thrombotic mortality, while "low secretors" (1ug/ml VIIa) had normal lifespan. This work hints at a potential alternative approach for gene therapy of haemophilia that does not target the defective gene at all, but rather augments in vivo factor VIIa secretion.

R Montgomery gave an excellent presentation on a novel gene therapy approach to inhibitor management in a mouse model of haemophilia A. His team transferred the F8 gene with a platelet-specific promoter to induce host megakaryocyte expression of FVIII, which is then stored within the platelet alpha granule. He showed in a murine model that platelets activated at the site of vessel damage release this "internal" FVIII upon degranulation. Haemostasis in the setting of potentially lethal tail clip challenge was achieved even in the presence of high-titre anti-factor VIII antibodies.

While all the above was of interest, the show piece of the meeting for me was found in poster 0785, by Sumi et al. This was an elegant study on the "anticoagulant" effect of the coffee bean, both through tPA release and through AT-like activity within the bean, inversely proportional to roasting time. Armed with this informative gem, I quickly joined the queue to the espresso maker with new-found enthusiasm.

Mark Smith

BY EMMANUEL J. FAVALORO

The SSC meetings began on Saturday, 6th August. In my opinion, the previous standard SSC format has been changed for the worse. Instead of multiple SSC sessions over the two usual days, we had many multiple extended sessions over 1.5 days. Given my background, I was committed to attending the VWF/VWD SSC meeting. This session was basically a long 'Progress Reports' session,

where all the very many and various 'Working Parties' (WP) gave their updates. In the first part of the session there were WP reports on (i) VWF assays in VWD diagnosis (Anthony Hubbard, UK), (ii) different collagen reagents for use in VWF:CB (yours truly plus Dr De Marco, Italy), (iii) multimeric analysis (Ulrich Budde, Germany), (iv) VWD classification (Evan Sadler, US), (v) molecular biology & expression studies (David Lillicrap, Canada). Basically, all of these WP reports noted that studies are ongoing. The first WP (VWF assays in VWD diagnosis) should have a completed study within the next six months, ahead of the next SSC meeting in Oslo. The second WP (different collagen reagents for use in VWF:CB) should have a study initiated within the next 6 months. The VWD classification WP should have a revised classification document for submission to the Journal of Thrombosis and Haemostasis (JTH) within the next six or so months. The second part of the VWF/VWD SSC meeting focused on TTP and assays for ADAMTS13. We heard talks on a new fluorogenic substrate for ADAMTS13 (Dr Kokame), the action on ADAMTS13 on different VWF mutants (Dr Scheppenheim), the role of chloride ions on VWF-ADAMTS13 interactions (Dr de Cristoforo), and other delights. Although TTP is a rare disorder, it (with ADAMTS13) took up a third of the VWF/VWD SSC meeting! The last part of this session was a mixture of talks, including updates from the DDAVP and VWD WP, the long-term prophylaxis in VWD WP, the updated registry on acquired VWD, and non-immune thrombocytopenia and 2B VWD.

Later that day I attended the Lupus Anticoagulant/ Phospholipid-Dependent-Antibodies SSC (16:00 – 19:30). There were talks by Steve Krillis (Australia; Update on the criteria for the antiphospholipid syndrome), Phillip de Groot (the Netherlands; Prospectives in LA diagnosis), Guido Reber (Switzerland; Prospectives in anti-beta-2 GPI ELISA), Monica Galli (Italy; WAPS study: clinical correlations of anti-cardiolipin, anti-beta-2-glycoprotein I, ant-prothrombin, anti-annexin V and anti-protein S antibodies), and Jacob Rand (USA; Annexin A5 testing in APS). I listened to all but the last talk. Was it just me who sensed a case of *deja vu*? I was surprised by how little has really happened in this area. There are still major problems with the assays and with the clinical diagnosis of APS. Studies can show associations of various assays with thrombosis, but many gaps in our knowledge still prevail.

The ISTH meeting itself started on Monday 8th August. This was another major day for me, having to speak at a symposia and also having to co-chair a symposia, so I wore a suit! Even got a photo next to Ayer's Rock (sorry, Uluru). Today was the day of the two 'back to basic science' symposia that I had talked the ISTH organisers into letting me organise on their behalf. The first session ran from 09:30 to 11:00, and was entitled 'Laboratory Diagnosis of Bleeding Disorders'. The first 30min presentation was a three-part presentation on 'Diagnostic issues in haemophilia and VWD' from Steve Kitchen (UK, NEQAS), Piet Meijer (ECAT) and Sukesh Nair (India). Each of these highlighted some specific issues in diagnosis from a quality assurance (QA) perspective. Dr Kitchen gave a talk from the NEQAS perspective, Dr Meijer from the ECAT perspective, and Dr Nair from the perspective of a fledgling QA program being developed in a developing country. Sukesh Nair comes from the Christian Medical Centre in Vellore, and I have known him for some time. Sukesh gained his Australasian Fellowship in Pathology (FRCPA) when he trained here at Westmead a few years ago as a visiting haematology registrar. The last talk was from Marco Cattaneo (Italy), who spoke on Laboratory testing, diagnosis and management of platelet disorders. I estimated that we had over 500 people attending this session, so I consider this to be a great achievement for basic diagnostics, something that seems to have been moved aside at some recent ISTH meetings.

After a quick coffee came the second 'back to basic science' symposia session entitled 'Diagnostic Issues in Thrombophilia' (11:30 to 13:00). The first 30min presentation was another three part presentation on 'Diagnostic issues in thrombophilia' from Ian Jennings (UK, NEQAS), Piet Meijer (ECAT) and Roslyn Bonar (Australia, RCPA QAP). Each of these again highlighted some specific issues in diagnosis from a quality assurance (QA) perspective. I was particularly proud of Roslyn. The session was held in a very large Auditorium and I again estimated that we had over 500 people attend this session. As far as I am aware, this is the first time that Roslyn has spoken in such a forum, and she did splendidly. The next talk was another three part presentation (Testing for Thrombophilia: Benefits and hazards; uses and abuses, knowns and unknowns) given by yours truly (laboratory perspective), and Ross Baker and Alex Gallus (clinical cases perspective). We had planned for a smaller venue

and a more informal setting. That plan went out the window when we discovered the venue that we had been given on the day. Nevertheless, I think the presentation was well received (at least I hope so). The last presentation in the session was Monica Galli (Italy), who spoke on the utility of tests for anticardiolipin antibody and anti-beta-2-glycoprotein.

Again, I will state for the record that I believe these two basic diagnostic sessions to have been of considerable success, and I hope that this spurs future ISTH meeting organisers to retain such content at future meetings.

My Tuesday (now suit free and more incognito) was taken up with more posters, the trade display, and a smattering of talks. I tended to mostly follow the VWF/VWD trail, so I listened to Dr Lethagen (Sweden) talk of responsiveness of Type 1 VWD patients to DDAVP, Dr Haberichter (USA) talk on VWF propeptide assays to identify Type 1 VWD with reduced VWF survival, Dr Dong (USA) talk on ADAMTS13 cleavage of VWF under flow, and Dr Sadler's talk on VWF as two sides of a coin (bleeding risk vs thrombotic risk) in the Barry Firkin Memorial lecture.

My Wednesday and Thursday was more of the same: I particularly liked Prof Peake's presentation, where he updated findings from a European VWD Type 1 genetics study. The other major talk of note for me on Wednesday was 'high levels of FVIII and risk of thrombosis' (Doggen, Netherlands). Thursday included the Congress Party. What an occasion that was. I could be seen (if anyone cared to look) dancing the night away with colleagues, and

otherwise eating the various offerings and drinking fine Australian red. Thankfully for onlookers, I managed to avoid the karaoke machine.

By Friday, most would have stopped! The trade display had packed up, and the posters all pulled down. There were a few talks (including (i) genetic defects of VWF as a determinant of its survival in circulation (Denis, France); (ii) thrombospondin as a regulator of VWF multimer size (Hogg, Australia), and (iii) assays for ADAMTS13 (Miyata, Japan). I was particularly interested in Phillip Hogg's update talk. I had seen some data on thrombospondin and it's activity (reduction of VWF leading to loss of HMW multimers) from his group some time ago, but had lost track of the more recent work in the area. I had in particular always been a little confused on the relative roles of ADAMTS13 and thrombospondin in terms of VWF processing and in potentially adverse clinical events such as TTP or VWD subtypes displaying heightened clearance of plasma HMW VWF. I'm not sure that issue was resolved by Phillip, but he did take us on a journey through his more recent work, which related to analogous mechanisms of shear-related protein multimer processing in hair that leads to curling! What an interesting world we live in!

Well, from this conference junkie... apart from issuing a big congratulation to the conference organisers... that's all folks...

Emmanuel J. Favaloro

UPCOMING MEETINGS

MEETING	WHERE/DATES	CONTACT
XXX. World Congress of The International Society of Hematology. ISH2005	Istanbul 28th Sept-2nd Oct 2005	www.ish2005istanbul.org
13th National Haemophilia Conference – Integrating Knowledge and Practice	Melbourne 30th Sept-2nd Oct 2005	www.haemophilia.org.au
3rd International Conference on Thrombosis and Hemostasis Issues in Cancer (ICTHIC)	Bergamo, Italy 14-16 October, 2005	www.bergamoconference.com
HAA 2005 Annual Scientific Meeting	Sydney 16-19 October 2005	haa2005@fcconventions.com.au
American Society of Hematology	New Orleans 3-6 December, 2005	www.hematology.org