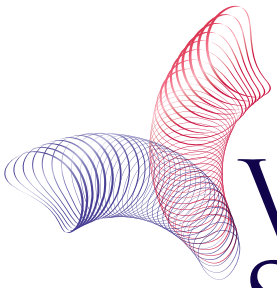

VASCULAR SOCIETY

YEARBOOK 12

OF GREAT BRITAIN AND IRELAND

CONTENTS

The Vascular Society AGM: 28 - 30 November 2012, Manchester

MESSAGE FROM THE PRESIDENT

Professor Ross Naylor

The 2012 AGM is the 25th Anniversary of my first attendance at the (then) Vascular Surgical Society of Great Britain and Ireland in Newcastle in 1987. To be attending this year as Society President is an immense honour.

2012 has been a momentous year for our Society. On March 16th, Vascular Surgery became a Specialty independent of General Surgery through an Act of Parliament. This was no mean feat and represented the culmination of many years of determined effort. It is impossible to name and thank everyone who contributed towards achieving specialty status, but it would be inappropriate not to acknowledge the endeavours of certain key individuals. These include a number of recent Presidents who were personally determined to see the project through, despite the many curve-balls, challenges and pitfalls en route (Mike Gough, Peter Taylor, George Hamilton, Cliff Shearman and Peter Lamont); Honorary Secretaries (Jonothan Earnshaw, Mike Wyatt) and the two Chairmen of the Education and Training Committee who developed the new Curriculum (Cliff Shearman, Jon Beard). I would also like to pay particular tribute to vital support from John Black (as President of the Royal College of Surgeons of England) in driving forward the specialty application at 'higher administrative levels' and to our colleagues in the Royal College of Radiologists (RCR) and the British Society of Interventional Radiology (BSIR) for supporting the bid.

So what does specialty status achieve? First and foremost, the prospect of developing a high-class training structure dedicated towards the needs of vascular trainees, rather than being dictated by 'service requirements' and the constraints imposed by emergency general surgery. In addition, vascular surgeons will now have a better chance of 'being heard' when key issues are being discussed. As we are now a specialty in our own right, the Vascular Society President becomes a member of the Council of the Royal College of Surgeons of England; he/she also becomes a member of the Forum of the four Royal Colleges of Surgeons and the Specialty Associations of Great Britain and Ireland, as well as joining the Federation of Surgical Specialty Association Presidents. These changes in the way vascular surgery has an independent voice comes at a time of increasing change in the way vascular surgeons (and units) will work in the future. Reconfigurations are well underway, centralised specialty commissioning awaits us in April 2013, and, at the 2012 Lister Meeting, Sir Bruce Keogh made clear the Government's intention to ensure Consultant delivered care and seven day working. For these reasons (and many more), it is vitally important that when decisions are being made, our views are represented by vascular surgeons and not by general surgical colleagues (however well intentioned they may be). I also predict that, rather than fuelling a schism between vascular surgeons and interventional radiologists, the advantages of co-operation, mutual professional interest and the potential for flexible working practices enshrined in the principles of the Specialty bid will encourage a much closer working relationship for the future. In some centres, this has already started to happen as far-sighted vascular surgeons and interventional radiologists combine to work within single business units. The future is a partnership and this will be of singular importance when applications for training centre status are being considered later this year.

'Outcomes after elective infra-renal abdominal aortic aneurysm' which showed that 30-day mortality rates were <3%; ie exceeding the target set by the AAA Quality Improvement Programme

2012 also saw the Society publish the latest iteration of the 'Provision of Vascular Services' document, which has assumed considerable importance in many of the regional configurations (many thanks to Mike Wyatt). We have also demonstrated our willingness to put even more outcome data into the public domain. In March, the Vascular Society published 'Outcomes after elective infra-renal abdominal aortic aneurysm' which showed that 30-day mortality rates were <3%; ie exceeding the target set by the AAA Quality Improvement Programme and well before the scheduled 2014 timeline. In August, the Vascular Society (in conjunction with HQIP and the Royal College of Physicians) published the 4th Round of the UK Carotid Endarterectomy Audit (many thanks to David Mitchell). The headline news was that the median delay from index symptom to surgery was now only 15 days. Few countries in the world can match this and our Members should be very proud of this initiative.

2012 has been a momentous year for our Society. On March 16th, Vascular Surgery became a Specialty independent of General Surgery through an Act of Parliament. This was no mean feat and represented the culmination of many years of determined effort.

I particularly look forward to welcoming you to this year's AGM, where the programme has been designed to attract and sustain your interest. Five symposia, guest lectures, prize sessions, scientific sessions, Masterclasses, workshops and many controversies await you (but sadly only one carotid symposium). We will award Manish Mehta and Henrik Sillesen with Honorary Society membership; we will recognise the work and endeavours of our Lifetime Achievers (Janet Powell, Chris Gibbons) and we will publicly thank George Davies for yet more financial support for funding research via the Circulation Foundation. For the first time, the Vascular Society has offered the Rouleaux Club an opportunity to run a dedicated symposium on training in the new Specialty. In the late 1980s, I succeeded Simon Hardy as Rouleaux Club Secretary (then viewed by Vascular Society grandees as a rebel group to be ignored, perhaps even suppressed) and I take great pleasure in being the first Rouleaux Club member to have been elected President. On Friday, the AGM will conclude with my friend and ex-Edinburgh research colleague, Peter Rothwell, delivering the Kinmonth Lecture.

In concluding, I would like to thank everyone who has supported me during my six years on Council and during my year as President. I thank the Chairmen of the various Executive Committees (Jon Beard, John Brennan, David Mitchell, Shervanthi Homer-Vanniasinkam, Ian Franklin, Cliff Shearman) for their sterling efforts. Jeanette Oliver has been a fantastic CEO and, in conjunction with Mike Wyatt, the Society has been run with marvellous efficiency. Thank you both. I would also like to pay particular tribute to Simon Parvin. He finishes his term of office as Treasurer and will be replaced by Tim Lees. Simon has been a superb Executive colleague and has thrived on dazzlingly complex spreadsheets. The bottom line, however, is that he has ensured that the Society's finances remain healthy. At the Business Meeting, we will announce newly elected members of Council and say goodbye to those (like me) who are demitting office. I have greatly enjoyed being part of an organisation dedicated to raising the standards of vascular surgery. So why don't you put your name forward for election next year? Who knows what a difference you could make.

As I step down from office, I am delighted to welcome Julian Scott as incoming President. Julian has already done a huge amount of work in getting the new FRCSvasc examination ready for 2016 and I have every confidence that he will be very successful in leading the Society into an exciting future.

PROGRAMME

WEDNESDAY 28th NOVEMBER

9.00am - 12noon	VENOUS FORUM | EXCHANGE 9
9.00am - 12noon	SOCIETY OF ACADEMIC AND RESEARCH SURGERY | EXCHANGE 6 & 7
9.00am - 12noon	EDUCATIONAL MASTERCLASS | CHARTER 4
9.00am - 12noon	ENDOVASCULAR WORKSHOP | EXCHANGE 8 & 10
9.00am - 4.00pm	SVN ANNUAL MEETING | EXCHANGE 11
10.20am-11am	COFFEE | EXHIBITION HALL
12noon - 1.00pm	LUNCH | EXHIBITION HALL
1.00pm - 1.15pm	SCIENTIFIC MEETING: OPENING CEREMONY AND PRESIDENT'S WELCOME | EXCHANGE AUDITORIUM
1.15pm - 3.00pm	SYMPOSIUM | NEW SPECIALTY, BIG CHALLENGES
3.00pm - 3.30pm	TEA | EXHIBITION HALL
3.30pm - 5.00pm	SCIENTIFIC SESSION 1: THE FOUNDER'S PRIZE
5.00pm - 6.15pm	SYMPOSIUM: VASCULAR TRAUMA UPDATE
6.15pm - 7.00pm	DRINKS RECEPTION | EXHIBITION HALL

9.00am - 12noon	EDUCATIONAL MASTERCLASS | CHARTER 4 **Complications in vascular and endovascular surgery: how to avoid them and how to get out of trouble** Facilitator: Professor Jonathan Beard, Chairman, Education Committee
9.00am - 12noon	INTERACTIVE TEACHING SESSIONS: **Complications related to diabetes in vascular patients** | Professor Bruce Campbell, Exeter **Associated renal complications and how to prevent them** | Professor George Hamilton, London **The management of haemorrhage in vascular surgery** | Mr Rod Chalmers, Edinburgh **Drugs and medicines associated with complications in vascular patients** Professor Cliff Shearman, Southampton **Management of complications relating to the acutely ischaemic limb** | Mr David Ratliff, Northampton **Complications due to peripheral angiography, angioplasty and stenting** | Mr Rob Fisher, Liverpool
9.00am - 12noon	ENDOVASCULAR WORKSHOP: INTRODUCTION TO SIMULATORS | EXCHANGE 8 AND 10 Facilitator: Mr John Brennan, Chairman Elect, Education Committee
9.00am - 12noon	SOCIETY OF ACADEMIC AND RESEARCH SURGERY | EXCHANGE 6 AND 7 Co-Chairmen: Professor Rob Sayers, Leicester; Mr Bijan Modarai, London
9.00am - 9.10am	**Micro-embolic signals and carotid plaque haemorrhage predict recurrence in patients with symptomatic carotid artery disease** N Altaf[1,2], N Kandiyl[2], AA Hosseini[2], ST MacSweeney[1], DP Auer[2] [1]Department of Vascular and Endovascular Surgery, Queen's Medical Centre, Nottingham [2]Division of Radiological and Imaging Sciences, University of Nottingham
9.10am - 9.20am	**Aortic masks generated by 3D rapid prototyping show 18F-FDG PET-CT uptake correlates with inflammation and ECM degradation in aneurysm wall** R Attia, A Patel, A Smith, P Taylor, C Young, B Modarai, M Waltham King's College London BHF Centre of Clinical Excellence/NIHR BRC King's Health Partners, Academic and Cardiovascular Surgery, St Thomas' Hospital, London
9.20am - 9.30am	**18F-FDG PET-CT uptake correlates with inflammation, ECM degradation and aneurysm expansion in the ApoE-/-/Angiotensin II infusion model of aortic aneurysm** R Attia, A Patel, A Smith, B Modarai, P Taylor, M Waltham King's College London BHF Centre of Clinical Excellence/NIHR BRC King's Health Partners, Academic and Cardiovascular Surgery, St Thomas' Hospital, London
9.30am - 9.40am	**Low haemoglobin concentration is associated with poor outcome after peripheral arterial surgery** OA Oshin, F Torella Liverpool Vascular and Endovascular Service
9.40am - 9.50am	**Apolipoprotein A-1 and Kininogen 1 are biomarkers for abdominal aortic aneurysm (AAA)** S Ehsan, K E Herbert, R D Sayers, M J Bown University of Leicester

WEDNESDAY 28TH NOVEMBER

9.50am - 10.00am	**Increased mortality in subjects with small abdominal aortic aneurysm (AAA) without previous history of cardiovascular disease compared to controls is associated with increased levels of hs-CRP and H-FABP** S Sohrabi[1,2], S Wheatcroft[1,2], JH Barth[3], M Bailey[1,2], K Griffin[1,2], A Johnson[1,2], PD Baxter[4], DJA Scott[1,2,5] [1]LIGHT, Division of Cardiovascular and Diabetes Research, University of Leeds [2]University of Leeds [3]Leeds General Infirmary Biochemistry Department [4]Leeds Centre for Epidemiology & Biostatistics, University of Leeds [5]Leeds General Infirmary
10.00am - 10.10am	**Fibrin-targeted molecular MRI predicts successful venous thrombolysis** P Saha[1], M Andia[2], AS Patel[1], O Lyons[1], S Grover[1], J Humpheries[1], B Modarai[1], R Botnar[2], AS Smith[1], M Waltham[1] [1]Academic Department of Surgery, Cardiovascular Division, BHF Centre of Excellence, King's College London and NIHR Biomedical Research Centre at Guy's and St. Thomas' NHS Foundation Trust, London [2]Division of Imaging Sciences, Cardiovascular Division, BHF Centre of Excellence, King's College London and NIHR Biomedical Research Centre at Guy's and St Thomas' NHS Foundation Trust, London
10.10am - 10.20am	**Molecular regulators of venous valves in development and disease** OTA Lyons[1], A Sabine[2], S Grover[1], E Bazigou[3], T Petrova[2], T Makinen[3], A Smith[1] [1]Academic Department of Surgery, King's College London, BHF Centre of Research Excellence & NIHR Biomedical Research Centre at King's Health Partners, London [2]CePO, CHUV, Universite de Lausanne [3]Lymphatic Development Laboratory, London Research Institute, CRUK
10.20am - 10.50am	COFFEE AND VIEWING OF TRADE EXHIBITION | EXCHANGE HALL
10.50am - 11.00am	**Functional consequences of ischaemia-induced Toll-like Receptor 4 (TLR4) activation in skeletal muscle** A Navi[1], H Patel[1], D Abraham[2], X Shi-Wen[2], D Baker[1], J Tsui[1] [1]Division of Surgery and Interventional Science, University College London, Royal Free Campus, London [2]Centre for Rheumatology and Connective Tissue Disease, University College London, Royal Free Campus, London
11.00am -11.10am	**Tissue engineering a small diameter vascular graft-cell seeding of a decellularised porcine arterial scaffold** M Tatterton[1], S-P Wilshaw[2], E Ingham[2], S Homer-Vanniasinkam[1] [1]Leeds Vascular Institute, Leeds General Infirmary, Leeds [2]Institute of Medical and Biological Engineering, University of Leeds, Leeds
11.10am - 11.20am	**Regional changes in aortic stiffness measured by phase contrast magnetic resonance imaging in the presence of small abdominal aortic aneurysm** A Abbas[1], A Smith[1], M Cecelja[2], M Hussain[3], G Greil[3], P Chowienczyk[2], M Waltham[1] [1]King's College London British Heart Foundation Centre of Research Excellence, Academic Department of Surgery, Cardiovascular Division, NIHR Biomedical Research Centre at Guy's and St Thomas' NHS Foundation Trust and King's College Hospital, London [2]King's College London BHF Centre of Research Excellence, Clinical Pharmacology Department, Cardiovascular Division, NIHR Biomedical Research Centre at Guy's and St Thomas' NHS Foundation Trust and King's College Hospital, London [3]King's College London BHF Centre of Research Excellence, Division of Imaging Sciences, NIHR Biomedical Research Centre at Guy's and St Thomas' NHS Foundation Trust and King's College Hospital, London
11.20am - 11.30am	**MicroRNAs implicated in peripheral vascular disease** PW Stather[1], N Sylvius[2], JB Wild[1], E Choke[1], RD Sayers[1], MJ Bown[1] [1]Department of Cardiovascular Sciences, University of Leicester [2]University of Leicester
11.30am - 12noon	KEYNOTE LECTURE **Publish well or be cast aside** | Professor Janet Powell, London

9.00am – 12noon	VENOUS FORUM | EXCHANGE 9 **Welcome and Introduction** | Professor Andrew Bradbury, President of the Venous Forum
9.00am - 9.20am	KEYNOTE LECTURE **Progress towards a commercially available foam for the treatment of varicose veins** Mr David Wright, Vice President, Medical Affairs, BTG International Ltd
9.20am - 10.40am	SYMPOSIUM 1 **Modern treatment options for varicose veins – an up-date** Chairs: Professor Gerard Stansby and Mr Isaac Nyamekye
9.20am - 9.30am	**Which LASER for which patient, and what sort of results can we expect in the long term?** Professor Michael Gough, Consultant Vascular Surgeon, Leeds
9.30am - 9.40am	**Radiofrequency ablation: a 5 year experience of delivering a totally endovenous service** Mr Kenneth Woodburn, Consultant Vascular Surgeon, Royal Cornwall Hospital, Truro
9.40am - 9.50am	**Foam sclerotherapy: is it possible to achieve a European Consensus?** Mr Philip Coleridge-Smith, Consultant Vascular Surgeon, British Vein Institute, Amersham
9.50am - 10.00am	**How should we define an incompetent perforator, when are they relevant, when should we treat them, and how?** | Mr Mark Whiteley, Consultant Surgeon, The Whiteley Clinic, Guildford
10.00am - 10.10am	**Steam to treat varicose veins: worth further consideration in the UK?** Dr René Milleret, Vascular Surgeon. Pasteur Clinic, Pézenas, France
10.10am - 10.40am	**Panel Discussion to involve all speakers**
10.40am - 11.00am	COFFEE AND VIEWING OF TRADE EXHIBITION | EXCHANGE HALL
11.00am - 11.30am	DEBATE **"This House believes that it is not currently possible to determine which treatment for varicose veins is the most cost-effective"** | Chairs: Mr David Berridge and Professor Andrew Bradbury
	VOTE
11.00am - 11.10am	**For the motion** | Professor Alun Davies, Professor of Vascular Surgery, Charing Cross Hospital, London
11.10am - 11.20am	**Against the motion** Professor Jonathan Michaels, Honorary Professor of Clinical Decision Science, University of Sheffield
11.20am - 11.25am	REBUTTAL Professor Alun Davies
11.25am - 11.30am	REBUTTAL Professor Jonathan Michaels
	VOTE
11.30am - 12noon	SYMPOSIUM 2 **How can we train future vascular specialists in the management of varicose veins at a time when the NHS has disinvested from such treatments?**
11.30am - 11.40am	**View from the Trainers** | Professor Ian Chetter, Chair of Surgery, Hull York Medical School/University of Hull
11.40am - 11.50am	**View from the Trainees** | Mr James Scurr, Trainee Representative, Council, Venous Forum
11.50am - 12.00noon	DISCUSSION TO INVOLVE ALL SPEAKERS

WEDNESDAY 28TH NOVEMBER

12noon - 1.00pm	LUNCH AND VIEWING OF TRADE EXHIBITION | EXCHANGE HALL
1.00pm	THE VASCULAR SOCIETY MEETING | EXCHANGE AUDITORIUM Opening Ceremony Welcome by Professor Ross Naylor, President Presentation of Lifetime Achievement Awards to Mr Chris Gibbons and Professor Janet Powell Presentation of Honorary Membership to Professor Henrik Sillesen and Dr Manish Mehta
1.15pm - 3.00pm	SYMPOSIUM: NEW SPECIALTY, BIG CHALLENGES | EXCHANGE AUDITORIUM Co-Chairmen: Professor Ross Naylor, President; Mr Ian Loftus, London
1.15pm - 1.35pm	**Elective and emergency vascular interventions in children** | Professor George Hamilton, London
1.35pm - 1.55pm	**Endovascular treatment of mycotic aneurysms and infected aortic grafts: A bridge to success or a bridge too far?** | Mr Mike Jenkins, London
1.55pm - 2.15pm	**EVAR: Live now and pay later, or live now and still live later?** | Professor Rob Sayers, Leicester
2.15pm - 2.35pm	**CCSVI is a real entity that may become more surgical than interventional** Dr Manish Mehta, Albany, New York
2.35pm - 3.00pm	DISCUSSION
3.00pm - 3.30pm	TEA AND VIEWING OF TRADE EXHIBITION | EXCHANGE HALL
3.30pm - 5.00pm	SCIENTIFIC SESSION 1: THE SOL COHEN (FOUNDER'S) PRIZE | EXCHANGE AUDITORIUM Co-Chairmen:Professor Julian Scott, Leeds; Mr Mike Wyatt, Newcastle
3.30pm - 3.45pm	**Predicting aortic complications after endovascular aneurysm repair: development and external validation of a morphological risk score** A Karthikesalingam[1], PJE Holt[1], EC Choke[2], BO Patterson[1], A Vidal-Diez[3], JD Poloniecki[3], LJ Thompson[1], MJ Bown[2], RJ Hinchliffe[1], RD Sayers[2], MM Thompson[1] [1]Department of Outcomes Research, St George's Vascular Institute, London [2]Vascular Surgery Group, University of Leicester, Leicester Royal Infirmary, Leicester [3]Department of Community Health Sciences, St George's University of London, London
3.45pm - 4.00pm	**Development of a patient-specific risk stratification system for patients undergoing thoracic aortic aneurysm repair** BO Patterson[1], A Karthikesalingam[1], PJE Holt[1], C Nienaber[2], R Fairman[3], MM Thompson[1] [1]St George's Vascular Institute, London [2]University of Rostock, Rostock, Germany [3]Hospital of the University of Pennsylvania, Philadelphia, USA
4.00pm - 4.15pm	**Development of a decision tree to streamline infra-inguinal vein graft surveillance** O McBride[1], R Mofidi[2], SA Suttie[3], RTA Chalmers[1], PA Stonebridge[3], ARW Dawson[1] [1]Department of Vascular Surgery, Royal Infirmary of Edinburgh [2]Department of Vascular Surgery, James Cook University Hospital, Middlesbrough [3]Department of Vascular Surgery, Ninewells Hospital, Dundee

4.15pm - 4.30pm	**A comparison of the effectiveness of treating those with and without the complications of superficial venous insufficiency** D Carradice, T Wallace, N Samuel, R Gohil, I Chetter Academic Vascular Surgical Unit, Hull York Medical School
4.30pm - 4.45pm	**Associations between histological features of symptomatic carotid plaque and predicted stroke risk: implications for carotid plaque imaging** DPJ Howard[1], GW van Lammeren[2], JN Redgrave[1], FL Moll[2], DPV de Kleijn[3], G Jan de Borst[2], G Pasterkamp[3], PM Rothwell[1] [1]Oxford University Hospitals NHS Trust [2]Department of Vascular Surgery, University Medical Centre Utrecht, The Netherlands [3]Experimental Cardiology Laboratory, University Medical Centre Utrecht, The Netherlands
4.45pm - 5.00pm	**Poorly controlled BP and impaired baroreceptor function (but not impaired autoregulation) are associated with a significantly higher prevalence of post-carotid endarterectomy hypertension** JE Newman[1], MJ Bown[1], RD Sayers[1], JP Thompson[2], TG Robinson[3], B Williams[4], RB Panerai[5], PS Lacy[4], AR Naylor[1] [1]Department of Vascular Surgery, Leicester Royal Infirmary, Leicester [2]Department of Anaesthetics, Leicester Royal Infirmary, Leicester [3]Department of Stroke Medicine, Leicester Royal Infirmary, Leicester [4]Department of Hypertensive Medicine, Leicester Royal Infirmary, Leicester [5]Department of Medical Physics, Leicester Royal Infirmary, Leicester
5.00pm - 6.15pm	SYMPOSIUM: VASCULAR TRAUMA UPDATE | EXCHANGE AUDITORIUM Chairman: Professor Karim Brohi, London
5.00pm - 5.15pm	**Vascular injuries of the limbs: Lessons for the inexperienced** | Mr Paul Blair, Belfast
5.15pm - 5.30pm	**Abdominal vascular injuries: Lessons for the inexperienced** Professor Ken Boffard, Johannesburg, South Africa
5.30pm - 5.45pm	**Endovascular treatment for aortic transection** | Professor Peter Taylor, London
5.45pm - 6.15pm	DISCUSSION
6.15pm - 7.00pm	WELCOME RECEPTION | EXHIBITION HALL

PROGRAMME

THURSDAY 29th NOVEMBER

7.00am - 8.00am	BREAKFAST SYMPOSIUM: BRITISH SOCIETY OF ENDOVASCULAR THERAPY | EXCHANGE 6 & 7
9.00am - 5.30pm	SVT ANNUAL MEETING | EXCHANGE 9
8.00am - 9.30am	SCIENTIFIC SESSION 2 | EXCHANGE AUDITORIUM
9.30am - 10.00am	COOK FELLOWSHIP REPORTS
10.00am - 10.30am	KEYNOTE LECTURE | PROFESSOR HANS PRIEBE
10.30am - 11.00am	COFFEE | EXHIBITION HALL
11.00am - 12.30pm	SCIENTIFIC SESSION 3: THE BJS PRIZE
12.30pm - 1.30pm	LUNCH | EXHIBITION HALL
1.30pm - 3.00pm	SCIENTIFIC SESSION 4
3.00pm - 3.30pm	CIRCULATION FOUNDATION: 2012 – AN OUTSTANDING YEAR
3.30pm - 4.00pm	TEA | EXHIBITION HALL
4.00pm - 5.30pm	SYMPOSIUM: FUTURE OF VASCULAR TRAINING
5.30pm - 6.30pm	ANNUAL BUSINESS MEETING
5.30pm - 6.30pm	ROULEAUX CLUB AGM | EXCHANGE 6 & 7
7.30pm for 8.00pm	ANNUAL SOCIETY DINNER AND PRIZE PRESENTATIONS MANCHESTER CENTRAL CONVENTION CENTRE

7.00am - 8.00am	SYMPOSIUM: BRITISH SOCIETY OF ENDOVASCULAR THERAPY | EXCHANGE 6 AND 7 **Who is responsible when an aortic stent graft fails?** Co-Chairmen: Mr Jon Boyle, Cambridge; Mr Ferdinand Serracino-Inglott, Manchester
7.00am	INTRODUCTION Mr Jon Boyle, Consultant Vascular Surgeon, Cambridge University Hospitals
7.10am	**'Hips and PiPs: The lessons learned about implantable medical devices'** Miss N Lennard, MD, FRCS, Medical Director MHRA
7.35am	**"Medico-legal aspects of medical device failure and how the surgeon protects against it"** Dr N Hayes, BSc(Hons) MB ChB(Hons) MD GDL MFFLM LPC, Senior Medical Advisor, Medical Defence Union
9.00am - 5.30pm	SOCIETY FOR VASCULAR TECHNOLOGY ANNUAL MEETING | EXCHANGE 9
8.00am - 9.30am	SCIENTIFIC SESSION 2 | EXCHANGE AUDITORIUM Co-Chairmen: Mr Lasantha Wijesinghe, Bournemouth; Professor Ian Chetter, Hull
8.00am - 8.10am	**Variable Life Adjusted Display methodology for continuous performance monitoring of carotid endarterectomy** G Kuhan[1], DP McCollum[1], IC Chetter[2], B Akomolafe[2], BF Johnson[2], PM Renwick[2], PT McCollum[2] [1]Central Manchester University Hospitals NHS Trust [2]Academic Vascular Unit, Hull Royal Infirmary, Hull
8.10am - 8.20am	**Performance of risk prediction scores for carotid endarterectomy** D Doig, RL Featherstone, T Richards, MM Brown | University College London
8.20am - 8.30am	**The potential effect on vascular services of the implementation of recent NICE guidance for the management of deep vein thrombosis – VF** JH Saunders, P Arya, Y Yong, S Abisi, ST Macsweeney, BD Braithwaite, N Altaf Department of Vascular and Endovascular Surgery, Queen's Medical Centre, Nottingham
8.30am - 8.40am	**Long term functional outcome of treatment for Paget-Schroetter and venous thoracic outlet syndrome – VF** JM Taylor, RJ Telford, DC Kinsella, AF Watkinson, JF Thompson Royal Devon and Exeter Hospital NHS Foundation Trust, Exeter
8.40am - 8.50am	**Early results for the use of NHS blood and transplant derived Decellular Dermis in the management of treatment resistant leg ulcers** N Greaves[1], M Baguneid[1], A Bayat[2] [1]University Hospital of South Manchester [2]Plastic & Reconstructive Research, Manchester Interdisciplinary Biocentre, University of Manchester
8.50am - 9.00am	**Effect of the angiotensin converting enzyme inhibitor, ramipril, on walking distance, arterial stiffness and quality of life in patients with intermittent claudication (ACEIIC): randomised, double blind, placebo-controlled trial** Y Shahin, I Chetter | Academic Vascular Surgery Unit, University of Hull, Hull

VF = Paper to be considered for the Venous Forum prize

THURSDAY 29TH NOVEMBER

9.00am - 9.10am	**The development of a national risk prediction model for elective abdominal aortic aneurysm (AAA) repair** SW Grant[1], GL Hickey[1], AD Grayson[2], DC Mitchell[3], CN McCollum[1] [1]University of Manchester, Manchester [2]Liverpool Heart and Chest Hospital NHS Foundation Trust, Liverpool [3]Vascular Society Audit Committee, The Royal College of Surgeons of England, London
9.10am - 9.20am	**A systematic review and meta-analysis of Duplex ultrasound, contrast-enhanced ultrasound or computed tomography for surveillance after endovascular aneurysm repair** A Karthikesalingam[1], W Al-Jundi[2], D Jackson[3], JR Boyle[4], JD Beard[2], PJE Holt[1], MM Thompson[1] [1]Department of Outcomes Research, St George's Vascular Institute, London [2]Sheffield Vascular Institute, Northern General Hospital, Sheffield [3]MRC Biostatistics Unit, Institute of Public Health, University of Cambridge, Cambridge [4]Department of Vascular Surgery, Cambridge University Hospitals NHS Foundation Trust, Cambridge
9.20am - 9.30am	**Magnetic resonance imaging using a novel elastin peptide (MRI-EP) tracer demonstrates inflammation precedes matrix degradation and expansion in aortic aneurysms** R Attia, A Patel, A Smith, R Botnar, B Modarai, M Waltham King's College London BHF Centre of Clinical Excellence/NIHR BRC King's Health Partners, Academic and Cardiovascular Surgery, St Thomas' Hospital, London
9.30am - 10.00am	COOK/VASCULAR SOCIETY ENDOVASCULAR FELLOWSHIP REPORTS Chairman: Professor Jonathan Beard, Chair, Education Committee Presenters: Mr Manjit Gohel, London, Mr Nick Matharu, Coventry, Mr Matt Metcalfe, Hertfordshire
10.00am - 10.30am	KEYNOTE LECTURE **Benefits of peri-operative B-Blocker therapy: Myth or reality?** Professor Hans Priebe, Freiburg, Germany. Chairman: Professor Rob Sayers, Leicester
10.30am - 11.00am	COFFEE AND VIEWING OF TRADE EXHIBITION | EXCHANGE HALL
11.00am - 12.30pm	SCIENTIFIC SESSION 3: BJS PRIZE | EXCHANGE AUDITORIUM Co-Chairmen: Professor Shervanthi Homer-Vanniasinkam, Leeds; Professor Matt Thompson, London
11.00am - 11.15am	**Refining the association between low density lipoprotein receptor related protein 1 and abdominal aortic aneurysm** JB Wild[1], HZ Butt[1], N Sylvius[1], PW Stather[1], GT Jones[2], DJA Scott[3], J Lindholt[4], E Choke[1], RD Sayers[1], MJ Bown[1,5] [1]University of Leicester [2]University of Otago [3]University of Leeds [4]Viborg Hospital, Denmark [5]The Aneurysm Consortium
11.15am - 11.30am	**Transcriptomic profiles in abdominal aortic aneurysm - a validated microarray based study** HZ Butt, MK Salem, JB Wild, N Dattani, N Sylvius, S Ehsan, E Choke, RD Sayers, MJ Bown University of Leicester
11.30am - 11.45am	**18F-FDG PET-CT uptake is a feature of both normal diameter and aneurysmal aortic wall and is not related to aneurysm size** OTA Lyons[1], T Barwick[2], M Waltham[1], M O'Doherty[2] [1]Academic Department of Surgery, King's College London, BHF Centre of Research Excellence and NIHR Biomedical Research Centre at King's Health Partners, London [2]Clinical PET Centre, St Thomas' Hospital, King's Health Partners, London

11.45am - 12noon	**Reduced nitric oxide bio-availability post-major vascular surgery pre-disposes to myocardial injury and predicts All Cause Mortality in medium term** S Rajagopalan[1], A Al-Shaheen[2], P Bachoo[1], J Brittenden[1,2] [1]Vascular Unit, Aberdeen Royal Infirmary [2]Division of Applied Medicine, University of Aberdeen
12noon - 12.15pm	**Atorvastatin reduces vein wall inflammation and enhances recanalisation following venous thrombosis** SP Premaratne, S Grover, K Nuthall, P Saha, A Patel, B Modarai, A Smith, M Waltham Academic Department of Surgery, King's College London, BHF Centre of Research Excellence and NIHR Biomedical Research Centre at Kings Health Partners, London
12.15pm - 12.30pm	**Apoptotic cell death in ischaemic skeletal muscle is mediated via the Toll-like receptor (TLR) adapter protein MyD88** H Patel[1], X Shi-wen[2], D Abraham[2], D Baker1, S Shaw[3], J Tsui[1] [1]Royal Free Vascular Unit, University College, London [2] Centre for Rheumatology and Connective Tissue Disease, University College, London [3]Department of Clinical Research, University of Bern, Switzerland
12.30pm - 1.30pm	LUNCH AND VIEWING OF TRADE EXHIBITION | EXCHANGE HALL
1.30pm - 3.00pm	SCIENTIFIC SESSION 4 | EXCHANGE AUDITORIUM Co-Chairmen: Mr David Mitchell, Bristol; Mr Isaac Nyamekye, Worcester
1.30pm - 1.40pm	**The effect of acute kidney injury (AKI) on outcomes following repair of abdominal aortic aneurysm (AAA) – RW** H Hindley[1], J McCleary[2], A Lewington[3], CRV Tomson[4], ARW Weale[5], DC Mitchell[5] [1]AKI Data Manager, North Bristol NHS Trust [2]Research Administrator, North Bristol NHS Trust [3]Consultant Nephrologist, Leeds General Infirmary [4]Consultant Nephrologist, North Bristol NHS Trust [5]Associate Director, Carotid Interventions Unit
1.40pm - 1.50pm	**Long term results of endovascular aortic aneurysm repairs in the young** N Altaf, S Abisi, Y Yong, JH Saunders, ST MacSweeney, BD Braithwaite Department of Vascular and Endovascular Surgery, Queen's Medical Centre, Nottingham
1.50pm - 2.00pm	**Superficial Femoral Artery (SFA) stenting. Does it offer any advantage?** MG Bani-Hani, A Odurny, D Thompson, S Baxter, CP Shearman | University Hospital Southampton
2.00pm - 2.10pm	**Airborne contamination during graft insertion in vascular surgery – B** M Salji, S Hassan, M Abbas, A Ahmed, H Mills, T Elston, C Backhouse, A Howard, S Choksy Colchester Hospital University Foundation Trust
2.10pm - 2.20pm	**Patients with symptomatic carotid stenosis, turned down for endarterectomy, have high re-stroke and mortality rates** A Malkawi[1], T Martin[1], D Mason[1], P Leopold[1], P Chong[1], D Gerrard[1], S Sonnenberg[1], B Clarke[2], WR Wilson[1] [1]Department of Vascular Surgery, Frimley Park Hospital NHS Foundation Trust [2]Acute Stroke Unit, Frimley Park Hospital NHS Foundation Trust
2.20pm - 2.30pm	**Endovascular recanalization of deep veins to treat C6 leg ulcers - going beyond standard therapies of compression and varicose veins surgery – VF** RK George, H Verma, S Rajesh, RK Tripathi | Narayana Hrudayalaya Hospital, Bangalore, India
2.30pm - 2.40pm	**The prevalence and impact of antiplatelet resistance in carotid artery disease – RW** R Taylor, K Kanesalingam, A Schiro, C McCollum | The University Hospital of South Manchester

THURSDAY 29TH NOVEMBER

2.40pm - 2.50pm	**Histological features of carotid plaque in patients with ocular ischaemia versus cerebral events** DPJ Howard[1], GW van Lammeren[2], JN Redgrave[1], FL Moll[2], JP de Vries[3], DPV de Kleijn[4], GJ de Borst[2], G Pasterkamp[4], PM Rothwell[1] [1]Oxford University Hospitals NHS Trust [2]Department of Vascular Surgery, University Medical Centre Utrecht, The Netherlands [3]Department of Vascular Surgery, St Antonius Hospital, Nieuwegein, The Netherlands [4]Experimental Cardiology Laboratory, University Medical Centre Utrecht, The Netherlands
2.50pm - 3.00pm	**Is there a role for C-Reactive protein, Myeloperoxidase or Beta-2 Microglobulin as a biomarker of carotid plaque instability? – RW** RK Birk[1], MK Salem[1], K West[2], D Moore[2], A Nicolaides[3], RD Sayers[1], AR Naylor[1], MJ Bown[1] [1]Vascular Surgery Group, Department of Cardiovascular Sciences, University of Leicester, Leicester [2]Department of Histopathology, University Hospitals Leicester, Leicester [3]Department of Vascular Surgery, Imperial College, London
3.00pm - 3.30pm	CIRCULATION FOUNDATION: 2012 – AN OUTSTANDING YEAR Presentation by Mr Ian Franklin, Chairman Presentation of Awards – President's Early Career Award George Davies Award for Visionary Vascular Research Surgeon Scientist Awards Presentation of Ann Donald Memorial Bike Ride funds Guest of Honour: Mrs Fiona Davies
3.30pm - 4.00pm	TEA AND VIEWING OF TRADE EXHIBITION | EXCHANGE HALL
4.00pm - 5.30pm	SYMPOSIUM: ROULEAUX CLUB | EXCHANGE AUDITORIUM Co-Chairmen: Professor Ross Naylor, President, Vascular Society; Mr Femi Oshin, President, Rouleaux Club **Quality of vascular training in the UK: A survey of Rouleaux Club members** Mr Femi Oshin, Rouleaux Club President **The future of vascular training: ask not what can be done for us- ask what we can do for ourselves** Mr James McCaslin, Rouleaux Club Vice-President and Craig Nesbitt, Vascular Trainee, Northern Deanery **The new specialty: are we in danger of creating a lost tribe?** Mr Gary Lambert, Rouleaux Club ASiT Representative **Out of Hours activity: implications for a consultant delivered service** Mr Alan Karthikesalingam, Rouleaux Club Secretary **Training - who is in charge?** | Professor Cliff Shearman, Chairman, Vascular SAC **Discussion** **Presentation of Golden Graft award**
5.30pm - 6.30pm	ANNUAL BUSINESS MEETING | EXCHANGE AUDITORIUM
5.30pm - 6.30pm	ROULEAUX CLUB AGM | EXCHANGE 6 AND 7
7.30pm for 8.00pm	GALA DINNER AND PRESENTATION OF PRIZES CHARTER 1 - 3 | MANCHESTER CENTRAL CONVENTION CENTRE

RW = Paper to be considered for the Richard Wood prize

B = Paper to be considered for the Brighton prize

VF = Paper to be considered for the Venous Forum prize

PROGRAMME

FRIDAY 30th NOVEMBER

8.00am - 9.00am	SCIENTIFIC SESSION 5 | EXCHANGE AUDITORIUM
9.00am - 10.30am	SCIENTIFIC SESSION 6
10.30am - 11.00am	BRUNCH | EXHIBITION HALL
11.00am - 12.20pm	SYMPOSIUM: CEREBROVASCULAR DISEASE UPDATE
12.20pm - 12.30pm	INAUGURATION OF THE NEW PRESIDENT
12.30pm - 1.10pm	KINMONTH LECTURE | PROFESSOR PETER ROTHWELL

8.00am - 9.00am	**SCIENTIFIC SESSION 5 | EXCHANGE AUDITORIUM** Co-Chairmen: Mr John Brennan, Liverpool; Mr Tim Lees, Newcastle
8.00am - 8.10am	**The shortfall in long-term survival of patients with repaired thoracic or abdominal aortic aneurysms** A Karthikesalingam[1], BO Patterson[1], G Peach[1], A Vidal-Diez[2], JD Poloniecki[2], RJ Hinchliffe[1], PJE Holt[1], MM Thompson[1] [1]Department of Outcomes Research, St George's Vascular Institute, London [2]Department of Community Health Sciences, St George's University of London
8.10am - 8.20am	**A comparative study of abdominal aortic aneurysm (AAA) in men and women** S Sohrabi[1,2], S Wheatcroft[1,2], M Bailey[1,2], K Griffin[1,2], A Johnson[1,2], PD Baxter[3], DJA Scott[1,2,4] [1]LIGHT, Division of Cardiovascular and Diabetes Research, University of Leeds [2]University of Leeds [3]Leeds Centre for Epidemiology and Biostatistics, University of Leeds [4]Leeds General Infirmary
8.20am - 8.30am	**Crural vessel assessment in critical limb ischaemia - Dependent Doppler versus Magnetic Resonance Angiography** J E Coulston, R Forsythe, F C T Smith, M J Brooks, P M Lamont Department of Vascular Surgery, University Hospitals Bristol, Bristol Royal Infirmary, Bristol
8.30am - 8.40am	**The value of surveillance of Arterio-Venous-Fistulas that are not in use at 24 weeks following formation** J Todd, M Savage, LA Williams, R Chandrasekar | Wirral University Hospital Trust

FRIDAY 30TH NOVEMBER

8.40am - 8.50am	**Interim results on abolishing reflux from a randomised controlled trial on laser ablation with phlebectomies versus foam sclerotherapy – VF** CR Lattimer, E Kalodiki, M Azzam, GC Makris, S Somiayajulu, G Geroulakos Josef Pflug Vascular Unit, Ealing Hospital and Imperial College
8.50am - 9.00am	**Does pre-operative dual antiplatelet therapy reduce microembolisation rates after a carotid endarterectomy?** V Robba[1], P Vitish-Sharma[1], N Hussain[1], D Nix[2], K Makhdoomi[1] [1]Department of Vascular Surgery, King's Mill Hospital, Mansfield, [2]Vascular Studies Unit, King's Mill Hospital, Mansfield
9.00am - 10.30am	SCIENTIFIC SESSION 6 Co-Chairmen: Mr Simon Parvin, Bournemouth; Mr John Thompson, Exeter
9.00am - 9.10am	**High-density lipoprotein cholesterol and abdominal aortic aneurysm - a Mendelian randomisation study** SC Harrison[1], MV Holmes[2], MJ Bown[3], GT Jones[4], S Grettarsdottir[5], O Agu[1], A Van Rij[4], FW Asselbergs[6], AF Baas[7], SE Humphries[1] [1]Centre for Cardiovascular Genetics, University College London [2]Genetic Epidemiology Group, UCL [3]Department of Vascular Surgery, Leicester University [4]University of Otago, Dunedin, New Zealand [5]Decode Genetics, Iceland [6]University Medical Centre Utrecht, the Netherlands, On behalf of the SMART study [7]University Medical Centre Utrecht, the Netherlands
9.10am - 9.20am	**A 10-year prospective population-based study of the incidence and outcome of acute abdominal aortic aneurysms: implications for screening** DPJ Howard, A Banerjee, J Fairhead, A Handa, PM Rothwell | Oxford University Hospitals NHS Trust
9.20am - 9.30am	**Metabolic profiling of human atherosclerosis tissue reveals mechanisms of atherosclerosis progression and differences between carotid and femoral plaques** J Shalhoub[1], PA Vorkas[2], G Isaac[3], EJ Want[2], S McDonald[3], J Langridge[3], A Millar[3], JP Shockcor[3], JK Nicholson[2], AH Davies[1] [1]Academic Section of Vascular Surgery, Department of Surgery & Cancer, Imperial College London [2]Biomolecular Medicine, Department of Surgery & Cancer, Imperial College London [3]Waters Corporation, Milford, MA, USA
9.30am - 9.40am	**Endarterectomy of the common femoral bifurcation: a decade of experience from two UK centres in the endovascular era** M Desai[1], Z Ahmed[2], K Hussey[2], W Stuart[2], G Hamilton[1] [1]Department of Vascular Surgery, Royal Free London NHS Foundation Trust, London [2]Department of Vascular Surgery, Western Infirmary, Glasgow
9.40am - 9.50am	**A UK perspective on post-operative mobility in patients undergoing lower limb bypass surgery (LLBS) for critical limb ischaemia (CLI)** | N Al-Zuhir, J Boyle, K Varty, P Hayes, PA Coughlin Vascular Surgical Unit, Addenbrooke's Hospital, Cambridge
9.50am - 10.00am	**Microemboli present in the right heart during thermoablation of varicose veins** V Sounderajah[1], HM Moore[1], A Thapar[1], TRA Lane[1], K Fox[2], IJ Franklin[1], AH Davies[1] [1]Academic Section of Vascular Surgery, Imperial College School of Medicine [2]Department of Cardiology, Imperial College Healthcare NHS Trust
10.00am - 10.10am	**Have we reduced the prevalence of leg ulcers? A repeat audit after 20 years in an urban health district – VF** | A Sala Tenna[1], H Stevens[2], T Lees[1] [1]Northern Vascular Centre, Freeman Hospital, Newcastle-upon-Tyne [2]Newcastle Hospitals Community Health, Newcastle-upon-Tyne

10.10am - 10.20am	**Lipid management after carotid endarterectomy: comparing the effectiveness of primary and secondary care** R Durairajan[1], A Sivaramakrishnan[1], K Lund[1], A Kundu[1], T Kalra[1], T Loganathan[1], D Sinha[1], P Guyler[1], L Coward[1], A O'Brien[1], JRI Brown[2], MS Jakeways[2], S Patel[2] [1]Acute Stroke Unit, Southend University Hospital [2]Department of Vascular Surgery, Southend University Hospital
10.20am - 10.30am	**Safety of carotid endarterectomy following thrombolysis for acute ischaemic stroke: single centre experience and systematic review** Y Yong[1], J Saunders[1], S Abisi[1], N Sprigg[2], ST MacSweeney[1], N Altaf[1] [1]Department of Vascular and Endovascular Surgery, Queen's Medical Centre, Nottingham [2]Stroke Medicine, Nottingham University Hospitals
10.30am - 11.00am	BRUNCH AND COFFEE | EXCHANGE HALL
11.00am - 12.20pm	SYMPOSIUM: CEREBROVASCULAR DISEASE UPDATE | EXCHANGE AUDITORIUM Co-Chairmen: Professor Ross Naylor, President; Professor Henrik Sillesen, Copenhagen
11.00am - 11.15am	**How rapid should 'rapid access' CEA/CAS be in symptomatic patients?** Professor Peter Rothwell, Oxford
11.15am - 11.30am	**Prognosis and management of symptomatic vertebral artery stenoses** Professor Hugh Markus, London
11.30am - 11.45am	**Prognosis and management of asymptomatic carotid disease** Professor Henrik Sillesen, Copenhagen
11.45am - 11.55am	**What do we still need to learn? A surgeon's perspective** | Professor Michael Gough, Leeds
11.55am - 12.05pm	**What do we still need to learn? A stenter's perspective** | Dr Trevor Cleveland, Sheffield
12.05pm - 12.20pm	DISCUSSION
12.20pm - 12.30pm	PRESENTATION OF VENOUS FORUM PRIZE INAUGURATION OF PRESIDENT FOR 2012-2013
12.30pm - 1.10pm	KINMONTH LECTURE | Professor Peter Rothwell, Oxford CAROTID INTERVENTIONS: PAST, PRESENT AND FUTURE Chairman: Professor Mike Horrocks, Vice-President, RCS(Eng).

VF = Paper to be considered for the Venous Forum prize

POSTERS

The Vascular Society AGM: 28 - 30 November 2012, Manchester

1. Centralisation of vascular surgery in the UK: transition in caseload and early outcomes at a newly-formed regional unit

   D Gill, J Pigott, J Shalhoub, T Hussain
   Vascular Surgery Department, Northwick Park Hospital, North West London Hospitals NHS Trust

2. Concomitant ambulatory phlebectomy versus foam sclerotherapy with Endovenous Laser Ablation: A prospective cohort study

   T Wallace, N Samuel, D Carradice, I Chetter
   Academic Vascular Surgical Unit, University of Hull/ Hull York Medical School

3. Incidentally detected AAA: are we missing a vital source of patients?

   P K Jha, A Brunswicker, M Kibiro, F J Meyer, Y G Wilson, M P Armon, J F M Clarke, D Morrow
   Norfolk & Norwich University Hospital, Norwich

4. Diabetes Mellitus: Does it protect aortic aneurysm rupture?

   NS Theivacumar, MA Stephenson, H Mistry, D Valenti
   King's Health Partners, King's College Hospital, London

5. Dynamic contrast enhanced ultrasound for assessment of perfusion and ulceration in carotid plaque

   A Thapar[1],Y Zheng[2], M Averkiou[3], B Dharmarajah[1], A Davies[1], E Leen[1]
   [1]Imperial College London [2]Sun Yat Sen University, Guangzhou China [3]University of Cyprus

6. Comparison of spiral laminar flow and conventional PTFE infrainguinal grafts

   K Lummis, M Treavis, N Shaper
   Bradford Royal Infirmary

7. Statin therapy enhances aneurysm sac regression following EVAR

   SA Badger[1], C Gray[1, 2], P Goodman[2], MK O'Malley[1], MK O'Donohoe[1], CO McDonnell[1]
   [1]Dept Vascular Surgery, Mater Misericordiae University Hospital, Dublin [2]School of Physics, Dublin Institute of Technology

8. Reducing the delay for carotid endarterectomy in South East Scotland

   KA Gaba, MBJ Syed, Z Raza
   Edinburgh Vascular Surgery Service, Royal Infirmary of Edinburgh, Scotland

9. The influence of Clopidogrel on endoleak, intervention, and sac expansion following endovascular aortic aneurysm repair

   PW Stather, JB Wild, N Dattani, E Choke, RD Sayers, MJ Bown
   Department of Cardiovascular Sciences, University of Leicester

10. ClariVein - Results using a novel treatment for varicose veins

    K Stenson
    University Hospital Lewisham

11. Temporary vessel occlusion using reverse thermo-sensitive polymer in infra-popliteal bypasses for severe leg ischaemia

    M Sallam, K Mani, R Clough, B Moderai, H Rashid, H Zayed
    Vascular Unit, King's Health Partners

12. Aortic rupture and sac expansion after endovascular repair of abdominal aortic aneurysms are not as common as previously reported

    A Karthikesalingam, PJE Holt, BO Patterson, LJ Thompson, RJ Hinchliffe, IM Loftus, MM Thompson
    Department of Outcomes Research, St George's Vascular Institute, London

13. Closure technique following CEA influences local haemodynamics

    GJ Harrison[1, 2], TV How [2], RJ Poole[2], JA Brennan[1], JB Naik[1], SR Vallabhaneni[1], RK Fisher[1, 2]
    [1]Royal Liverpool and Broadgreen Hospitals NHS Trust
    [2]University of Liverpool

14. Intra-operative cerebral oximetry could predict 1-year mortality in patients with critical leg ischaemia undergoing infra-inguinal bypass

    H Badri, H Bidd, H Slim, D Green, M Edmonds, H Rashid

15. Validating endovascular simulation as a training tool in the vascular curriculum

    L Green[1], R Lakshminarayan[2], I Chetter[1]
    [1]Academic Vascular Surgical Unit, Hull Royal Infirmary, Hull York Medical School, University of Hull [2]Department of Interventional Radiology, Hull Royal Infirmary

POSTERS

The Vascular Society AGM: 28 - 30 November 2012, Manchester

16. Management of the left subclavian artery and neurological complications following thoracic endovascular repair - lessons from the MOTHER database

BO Patterson[1], PJE Holt[1], IM Loftus[1], R Cambria[2], R Fairman[3], MM Thompson[1]
[1]St Georges Vascular Institute, London [2]Massachusetts General Hospital, Boston, USA [3]Hospital of the University of Pennsylvania, Philadelphia, USA

17. High incidence of true vein graft aneurysms following popliteal aneurysm repair

AJ Sharples, M Kay, C Merriman, A Fox, T Sykes, A Houghton
Shrewsbury and Telford NHS Trust

18. Protecting the spinal cord during Thoraco-Abdominal Aortic intervention

C Ceresa, DJ Ablett, C Moores, AF Nimmo, RTA Chalmers
Department of Vascular Surgery, Royal Infirmary of Edinburgh, Edinburgh

19. Supervised exercise programme improves aerobic fitness in patients awaiting abdominal aortic aneurysm repair

HM Barakat, Y Shahin, J Khan, P McCollum, I Chetter
Academic Vascular Surgical Unit, University of Hull & Hull York Medical School, Hull

20. Delays to expedited carotid endarterectomy following referral: a prospective audit of practice

M A Ali[1, 2], J A Stephenson[1], A R Naylor[1, 2]
[1]University Hospitals of Leicester [2]University of Leicester

21. One-stage repair of the aortic arch and descending thoracic aorta with a hybrid stent-graft

M Iafrancesco[1], A Ranasinghe[1], I McCafferty[1], MW Claridge[1, 2], DJ Adam[1, 2], J Mascaro[1], RS Bonser[1]
[1]University Hospital Birmingham NHS Foundation Trust [2]University Department of Vascular Surgery, Heart of England NHS Foundation Trust

22. Could increasing the half-life of sodium tetra-decyl sulphate (STD) foam increase the efficacy of great saphenous vein (GSV) foam sclerotherapy (FS)?

B McAree[1], A Ikponmwosa[1], K Brockbank[2], C Abbott[1], S Homer-Vanniasinkam[1], M Gough[1]
[1]Leeds Vascular Institute [2]Institute of Pharmaceutical Innovation, University of Bradford

23. It is not just the size but flow which matters in autogenous arteriovenous fistula for renal access

RPS Gambhir, P Pedgaonkar, CP Singh, M Prabhu, A Dabbas, S Singh, VS Bedi
Army Hospital (Research and Referral), Delhi, India

24. Can we measure carotid plaque volume?

K Kanesalingam[1], R Taylor[1], A Schiro[2], F Serracino-Inglott[2], C McCollum[1]
[1]Department of Vascular Surgery, University Hospital of South Manchester [2]Department of Vascular Surgery, Central Manchester University Hospital

25. Does carotid artery plaque volume predict stroke symptoms?

K Kanesalingam[1], R Taylor[1], A Schiro[2], F Serracino-Inglott[2], C McCollum[1]
[1]Department of Vascular Surgery, University Hospital of South Manchester [2]Department of Vascular Surgery, Central Manchester University Hospital

26. Survival of octogenarians in the Rapid Access Carotid Endarterectomy (RACE) era

M Abdelhamid, N Abo-Sufian, TE Rix, R Insall, J Senaratne
East Kent Hospitals University NHS Trust, Kent and Canterbury Hospital

27. Population-based study of the incidence and outcome of acute aortic dissection: implications for pre-morbid risk factor control and the limitations of hospital-based registries

DPJ Howard, A Banerjee, J Fairhead, J Perkins, PM Rothwell
Oxford University Hospitals NHS Trust

28. Risk factors for microemboli in patients with symptomatic carotid artery stenosis

M Saedon[1], DRJ Singer[1], C Hutchinson[1], CHE Imray[2]
[1]Warwick Medical School, University of Warwick [2]University Hospital Coventry and Warwickshire NHS Trust

29. Regional variation in delay to treatment for symptomatic carotid disease within the UK

DCM Mitchell[1], ARN Naylor[2], A Rudd[3], A Halliday[4], G Cloud[5], S Waton[6]
[1]Associate Director, Carotid Interventions Unit [2]Professor of Vascular Surgery, Leicester Royal Infirmary [3]Professor of Stroke Medicine, Guy's & St Thomas' Medical School [4]Professor of Vascular Surgery, John Radcliffe Hospital, Oxford [5]Stroke Physician, St George's Hospital, London [6]Project Co-ordinator, Carotid Interventions Project

ABSTRACTS

SARS

Micro-embolic signals and carotid plaque haemorrhage predict recurrence in patients with symptomatic carotid artery disease

N Altaf[1], [2] N Kandiyl[2] AA Hosseini[2] ST MacSweeney[1] DP Auer[2]

[1]Department of Vascular and Endovascular Surgery, Queen's Medical Centre, Nottingham [2]Division of Radiological and Imaging Sciences, University of Nottingham

Objectives

Carotid plaque haemorrhage as detected by MRI (PH) and microembolic signals (MES) detected by transcranial Doppler (TCD) are known risk factors for recurrent cerebrovascular events in patients with carotid disease. We previously found an association between them, but it is unclear whether they allow independent risk prediction. This study assessed whether MES and PH independently predict recurrent cerebrovascular events in symptomatic carotid artery disease.

Methods

134 prospectively recruited patients (mean age 72 +/- 9.8years, 33% female) with symptomatic severe (60-99%) carotid stenosis underwent preoperative TCD and MRI of the carotid arteries to assess PH. Patients were followed up until carotid endarterectomy, recurrent cerebral event, death or study end. Event-free survival analysis was done using backward conditional Cox regression analysis.

Results

Eleven patients had no acoustic window. Of the remaining 123 patients, 82 (66%) demonstrated PH and 46 (37%) had MES. 37 cerebrovascular events were observed over 22 weeks: 34 (92%) patients had PH and 21 (57%) had MES. Carotid PH (HR=10.2; 95% CI: 3.1-33.1) and to a lesser degree MES (HR= 3.3; 95% CI: 1.7-6.4) predicted recurrence, while degree of stenosis and time from symptom did not.

Plaque haemorrhage predicted recurrence independent of age, sex, time from symptom, stenosis and MES presence (HR=8.6; 95% CI 2.6-29.0; P=0.001)

Conclusion

The presence of carotid plaque haemorrhage is an independent and more powerful predictor of recurrent cerebrovascular events in patients with symptomatic severe carotid stenosis than the presence of microembolic signals.

ABSTRACTS

SARS

Aortic masks generated by 3D rapid prototyping show 18F-FDG PET-CT uptake correlates with inflammation and ECM degradation in aneurysm wall

R Attia A Patel A Smith P Taylor C Young B Modarai M Waltham

King's College London BHF Centre of Clinical Excellence/NIHR BRC King's Health Partners, Academic and Cardiovascular Surgery, St Thomas' Hospital, London

Objectives

Imaging with 18F-FDG PET-CT in aortic aneurysms has been associated with aneurysm growth and risk of rupture. We aimed to investigate the biological correlates of 18F-FDG uptake.

Methods

Fifteen patients underwent aortic PET-CT. The images were segmented to generate high affinity 3-dimensional aortic reconstructions. 3D rapid prototyping using laser scintigraphy generated patient-specific aortic masks to match aneurysm morphology. These allowed accurately targeted aortic biopsies from sites of varying 18F-FDG uptake at surgery. 152 biopsies were treated enzymatically and analysed using flow cytometry and immunohistochemistry for inflammatory cell content.

Results

Ascending aortic (TAA n=10), aortic arch (n=3) and abdominal aortic aneurysms (AAA n=2) were studied. Maximal TAA diameter was 6.5cm and AAA 8.5cm. The mean patient age was 65.5(35-7 7years). There was a significant positive correlation between 18F-FDG uptake and total leucocyte (CD45) content of the diseased vessel wall ($R2=0.75$, $P<0.0001$). Leucocyte sub-populations were analyzed: only B cell (CD3, $R2=0.79$, $P<0.0001$), T cell (CD19, $R2=0.73$, $P<0.0001$) and natural killer cells (CD56, $R2=0.79$, $P<0.001$) positively correlated with 18F-FDG uptake. There was no correlation between macrophage numbers (CD68, HLA-DR expressing cells) and 18F-FDG uptake ($R2=0.25$, $P=0.283$). Elastin and collagen content was reduced at sites with chronic inflammatory cell infiltrate.

Conclusion

By using rapid prototyping technology, this study demonstrates for the first time focal 18F-FDG uptake in aneurysms accurately localises to inflammatory infiltrate and matrix degradation.

ABSTRACTS

SARS

18F-FDG PET-CT uptake correlates with inflammation, ECM degradation and aneurysm expansion in the ApoE-/-/Angiotensin II infusion model of aortic aneurysm

R Attia A Patel A Smith B Modarai P Taylor M Waltham

King's College London BHF Centre of Clinical Excellence/NIHR BRC King's Health Partners, Academic and Cardiovascular Surgery, St Thomas' Hospital, London

Objectives

Functional imaging aims to detect physiological and biological activity in tissues to determine disease progression. We aimed to investigate the role of functional imaging with 18F-FDG PET-CT in aortic aneurysms and assess the inflammatory processes that precede the increase in vessel diameter, are involved in aneurysm growth and rupture.

Methods

Murine ApoE-/-/Angiotensin II infusion model was used to generate aortic aneurysms. These were serially imaged and with aortic wall biopsies analysed for cellular composition and activity from sites of varying SUVmax.

Results

Studies in the murine aneurysm model showed that there was heterogeneous 18F-FDG uptake in the suprarenal aortic aneurysm and that this varied with time course of aneurysm development. Uptake was associated with aneurysm expansion. Sites with the highest SUVmax of >3.1 had increased number of B cells (550±51, $p<0.001$, 95%CI 0.41) and T cells (330±70, $p<0.001$, 95%CI 0.52) vs. sites of moderate SUVmax 2.1-3.0 B cell (258±85, $p<0.001$, 95%CI 0.50) T cell (170±75, $p<0.001$, 95%CI 0.48) and low SUVmax >2.1 B cell (77±42, $p<0.001$, 95%CI 0.23) T cell (35±20, $p<0.001$, 95%CI 0.28). The number of NK cells was also significantly increased from aortic biopsy sites at varying SUVmax. At high SUVmax NK cells were (74±22 $p<0.01$, 95%CI 0.52), at moderate SUVmax (23±14, $p<0.001$, 95%CI 0.64) and at low SUVmax (10±8, $p<0.001$, 95%CI 0.21).

Conclusion

18F-FDG uptake is highly heterogeneous and varies with time over the course of aneurysm development. It is associated with chronic inflammatory cell activity in the aortic wall and elastin degradation.

ABSTRACTS

SARS

Low haemoglobin concentration is associated with poor outcome after peripheral arterial surgery

OA Oshin F Torella

Liverpool Vascular and Endovascular Service

Objectives

To assess the influence of low haemoglobin (Hb) concentration on major adverse cardiac events (MACE) and mortality in patients undergoing peripheral arterial surgery.

Methods

This was a retrospective cohort study of patients undergoing reconstructive arterial surgery for peripheral arterial disease at a tertiary vascular centre. Preoperative factors linked with the occurrence of MACE and death on univariate analysis ($P < 0.1$), were included in a multivariate model to confirm independent association with the outcome variables.

Results

Three hundred and sixty consecutive patients (238 men) with a mean (SD) age of 69 (10.7) years and Hb of 13.0 (2.12) g/dl treated under the care of a single specialist between January 2004 and December 2011 were included in the analysis. Of these, 193 (53.6%) were anaemic. Twenty-six (7%) suffered a postoperative MACE and 18 (5%) died. On multivariate analysis, age >80 years (OR = 3; 95% CI [1.2-7.5]; P = 0.025), renal impairment (OR = 3.2; 95% CI [.99-10.2]; P = 0.053), coronary disease (OR = 3.6; 95% CI [1.5-8.7]; P = 0.005) and low Hb (OR for each 1g/dl drop below the mean = 1.4 [1.13-1.7]; P = 0.002) were independent risk factors for MACE. Unplanned surgery (OR = 4.5; 95% CI [1.2-16.9]; P = 0.025), and low Hb (OR for each 1g/dl drop below the mean = 1.5; 95% CI [1.14-1.86]; P = 0.002), were independent risk factors for death.

Conclusion

In peripheral arterial surgery, preoperative low Hb is associated with MACE and death. Further investigation is necessary to elucidate whether this relationship is causal. Meanwhile, consideration should be given to treating preoperative anaemia as a significant risk factor for adverse outcome in this setting.

ABSTRACTS

SARS

Apolipoprotein A-1 and Kininogen 1 are biomarkers for abdominal aortic aneurysm (AAA)

S Ehsan K E Herbert R D Sayers M J Bown

University of Leicester

Objectives

The aim of this study was to determine if mass-spectrometry could identify clinically relevant biomarkers of AAA.

Methods

We performed a MALDI-ToF-Mass Spectrometry study where the plasma proteome of 13 patients with AAA were compared to 13 screened controls. Following bioinformatic analysis of the output from this study two protein markers were selected for verification and their levels measured in an independent sample of 20 AAA and 16 controls using Enzyme-Linked-Immunoassays.

Results

The mass spectrometry analysis identified Apolipoprotein A-1 (APOA1) and Kininogen 1 (KNG1) as upregulated in patients with AAA. Laboratory analysis of these proteins in the independent cohort of plasma samples confirmed that these proteins were both associated with AAA. Median plasma levels were as follows: APOA1, 114 mg/dl (range 60.4 to164mg/dl) in AAA and 90.4 mg/dl (26.6 to 151 mg/dl) in controls (P=0.03); KNG1 0.37 ng/ml (0.22 to 1.10 ng/ml) in AAA and 0.24 ng/ml (0.13 to 0.39 ng/ml) in controls (P=0.003). ROC analysis revealed an area under the curve (AUC) of 0.69 for APOA1 and 0.86 for KNG1. Levels of APOA1 and KNG1 did not correlate with AAA size.

Conclusion

This study demonstrates the principle that mass spectrometry is a reproducible method of choice for the detection of AAA associated biomarkers and that APOA1 and KNG1 are associated with AAA.

ABSTRACTS

SARS

Increased mortality in subjects with small abdominal aortic aneurysm (AAA) without previous history of cardiovascular disease compared to controls is associated with increased levels of hs-CRP and H-FABP

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Objectives

AAA and Cardiovascular Disease (CVD) are strongly related and current evidence shows that CVD is the major cause of mortality in small AAA (3-5.4 cm). There is no evidence that AAA screening programmes reduce all cause mortality.

We hypothesized that patients with small AAA, without previous history of CVD, there is higher all cause mortality with raised inflammatory and cardiac biomarkers (hs-CRP and H-FABP), compared to controls without previous history of CVD.

Methods

In a prospective study, out of 476 AAA and 364 controls, there were 86 small AAA and 183 controls, all without any previous history of CVD. hs-CRP and H-FABP were measured at the time of recruitment. All-cause mortality and long term survival was recorded.

AAA group was older than the control group (73(68-80) vs. 70(65-75) years, $p<0.001$). There was no difference in gender ratio and diabetes prevalence. The AAA group had significantly higher usage of cardioprotective medications (aspirin, statin, ACEInh) ($p<0.001$).

Results

Mean survival was 6.3(5.6-7.9) and 8.0(7.7-8.2) years for the AAA and control respectively (Logrank <0.001). Age adjusted hazard ratio for all-cause mortality in the AAA compared to the control was 3.51(1.77 - 6.96) ($p<0.001$). AAA group showed significantly higher levels of H-FABP (4.62(3.56-6.11) vs. 3.97(3.18-4.85) umol/L, $p=0.001$) and hs-CRP (2.76(1.21-6.00) vs. 1.27(0.47-3.51) mg/L, $p<0.001$) compared to the controls.

Conclusion

Despite higher usage of cardioprotective medications in the AAA group, all-cause mortality was significantly higher in the AAA group without CVD compared to controls without CVD. Higher hs-CRP and FABP in the AAA group suggests higher incidence of subclinical CVD in this group, explaining lower survival compared to the controls.

ABSTRACTS

SARS

Fibrin-targeted molecular MRI predicts successful venous thrombolysis

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Objectives

Venous thrombolysis is associated with significant morbidity and should be used selectively. This study aims to investigate a fibrin-specific MRI contrast agent (EP-2104R) to stage venous thrombus organisation in order to better identify thrombi susceptible to lysis.

Methods

Venous thrombi were induced in mouse vena cava (n=72) and imaged by MRI (3T Philips Achieva) at days 2,4,7,10,14,21 after thrombus induction (n=12/gp). Each group was scanned pre- and 2hrs post-injection of EP-2104R (EPIX Pharmaceuticals, 8.0μmol/kg). An inversion recovery gradient echo (TFE) sequence was performed and T1 maps of the thrombus calculated using custom-made software. Fibrin contrast uptake in the thrombus was correlated with fibrin assessed by histology and Western Blot (n=6/gp). A separate group underwent systemic venous thrombolysis (10 mg/kg of tissue plasminogen activator (Actilyse, Boehringer Ingelheim, Germany) at each time point (n=6/gp). 24 hours after thrombolytic treatment, mice were re-scanned to examine restoration of caval blood flow.

Results

After injection of EP-2104R, large areas with high signal intensity and short T1 relaxation times were observed. A larger visualised thrombus enhancement volume demonstrated younger thrombi. Contrast uptake positively correlated with the fibrin content of the thrombus ($R2=0.97$, $P<0.01$). ROC curve analysis showed that a mean thrombus T1 relaxation time less than 630 ms on post contrast images had sensitivity of 94% and specificity of 99% to predict successful thrombolysis (AUC 0.993 [CI95%: 0.98-1.00]).

Conclusion

Fibrin-targeted MRI can be used to stage venous thrombus organisation and allow accurate stratification of thrombi amenable to lysis.

ABSTRACTS

SARS

Molecular regulators of venous valves in development and disease

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Objectives

Lymphoedema and venous reflux are associated in rare single-gene disorders but the overall molecular regulators of venous valve (VV) development and maintenance are poorly understood. Recently we compared the expression profile of murine and human VV, characterised normal VV formation in mice and used knockout lines to show that genes required for regulating lymphatic valve development are required for VV development and maintenance (J Clin Invest doi:10.1172/JCI58050). More recent developments will be presented and the genetic patterning of venous valves with respect to the genetics of human venous disease will be discussed.

Methods

Murine valves were examined by light microscopy, wholemount confocal immunofluorescence and scanning electron microscopy in wildtype mice and genetic reporter lines. Human valves were examined by immunohistochemistry, scanning electron microscopy and transmission electron microscopy. Tissue-specific conditional knockout lines were used to identify roles of genes in valve formation/maintenance.

Results

Murine and human venous valves exhibit a similar structural and expression pattern. Novel regulatory genes were found to be required for valve formation/maintenance.

Conclusion

We have established the use of murine knockout lines in the study of venous valve disease. Venous and lymphatic valves share a common gene-expression profile and some developmental pathways, which explains the shared phenotype of lymphoedema and venous reflux seen in the clinic. Further work should be aimed at defining other genetic and environmental factors required for the development and maintenance of these complex structures, and their role in disease.

ABSTRACTS

SARS

Functional consequences of ischaemia-induced Toll-like Receptor 4 (TLR4) activation in skeletal muscle

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Objectives

Inflammation contributes to ischaemia-induced myopathy in patients with peripheral arterial disease (PAD). TLR4 is a well-recognized pattern recognition receptor that has been implicated in ischaemia-induced tissue injury. We hypothesize that TLR4 is upregulated and its signalling pathway activated in cultured human myotubes exposed to simulated ischaemia.

Methods

Human gastrocnemius muscle biopsies were taken from patients with critical limb ischaemia undergoing major lower limb amputation (Group 1) and from patients with no PAD (Group 2). Human myoblasts were isolated, cultured to myotubes and then pre-treated with TLR4 neutralizing antibody prior to exposure to simulated ischaemia. Western blot analysis of TLR4, P-NFkB (signal-related kinase) and cleaved caspase III were carried out on the cell lysates to assess TLR4 expression, downstream signalling activity and apoptosis respectively.

Results

Upregulation of TLR4, P-NFkB and cleaved caspase III was observed after simulated ischaemia in both groups ($P<0.05$). Further, pre-treatment with TLR4 neutralizing antibody prior to simulated ischaemia reduced the expression of P-NFkB and cleaved caspase III in ischaemic cell lysates as compared to ischaemic cell lysates with no pre-treatment ($P<0.05$).

Conclusion

TLR4 is upregulated and its downstream signalling pathway is activated in the ischaemic human myotubes in vitro. Further, inhibition of TLR4 prior to ischaemia was associated with inhibition of the signalling pathway and reduced ischaemia-induced apoptosis. Further studies are required to understand the downstream signaling, which may lead to the development of novel therapies aimed at reducing muscle damage in patients with CLI.

ABSTRACTS

SARS

Tissue engineering a small diameter vascular graft-cell seeding of a decellularised porcine arterial scaffold

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Objectives

The patency rate of small diameter synthetic grafts for arterial bypass is poor. The use of decellularised xenogeneic tissue as a bypass conduit may provide a suitable alternative to synthetic graft material. The objective of this study was to seed ovine endothelial and smooth muscle cells onto the surface of decellularised porcine carotid arteries in vitro to determine their ability to support attachment and growth of xenogeneic cells.

Methods

Porcine carotid artery arteries were decellularised using an established protocol developed within the Institute of Medical and Biological Engineering at the University of Leeds. Ovine endothelial and smooth muscle cells were harvested and characterised using antibodies to vascular cell markers (VWF, CD31, myosin, α-actin,) by indirect immunofluorescence.

Two-dimensional static seeding of arteries was performed using seeding rings and ovine vascular cells through a range of concentrations (10^3-10^5 cells.ml^{-1}) to determine optimal seeding concentration. Cell adherence was determined using scanning electron microscopy. Three-dimensional dynamic seeding of the arteries (n=6) was then performed using a seeding concentration of 10^4 cells.ml^{-1} for 48 hours. Viability of seeded cells was determined using an ATPLite assay and Live/Dead staining.

Results

Ovine vascular cells were successfully seeded onto the surfaces of decellularised arteries in both two and three-dimensional experiments. Immunohistochemical and molecular analysis confirmed the vascular phenotype and viability of seeded cells.

Conclusion

The surface of acellular porcine carotid arteries provides an ideal matrix for ovine vascular cell incorporation without the need for additional surface-binding techniques. Such a property is ideal for the development of a tissue-engineered graft.

ABSTRACTS

SARS

Regional changes in aortic stiffness measured by phase contrast magnetic resonance imaging in the presence of small abdominal aortic aneurysm

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Objectives

Arterial stiffness is an independent predictor of cardiovascular risk. Regional changes in aortic stiffness have not previously been measured in the presence of abdominal aortic aneurysm (AAA). We used phase contrast cardiovascular magnetic resonance (PC-CMR) to quantify pulse wave velocity (PWV), a measure of stiffness, along aortic segments. Stiffness was related to calcification.

Methods

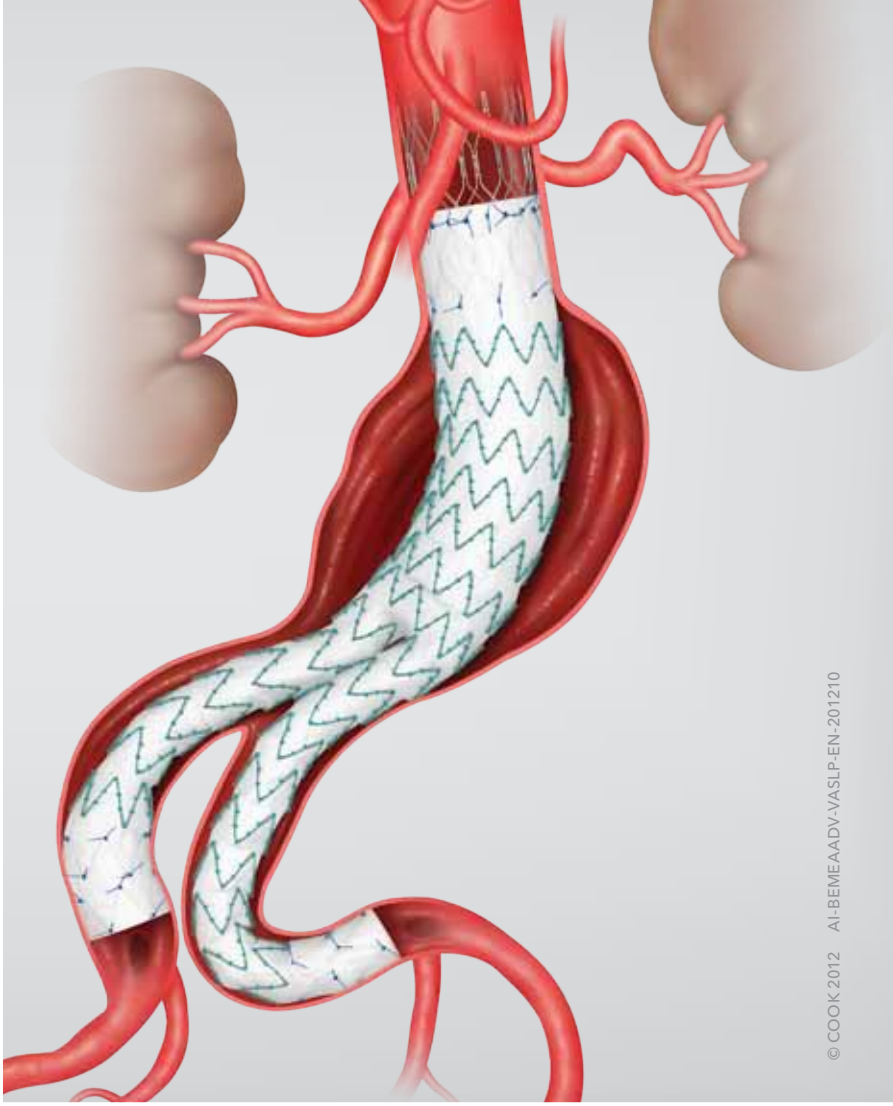
Regional aortic stiffness was measured in 77 (39 small AAA, 38 control) subjects using PC-CMR along ascending, descending and abdominal aortic segments. Calcification was measured using non-contrast CT.

Results

Mean AAA diameter was 3.9cm (range 3.0-5.6cm). PWV was significantly increased in subjects with AAA (10.1±0.34 mean±SEM m/s) vs controls (8.4±0.25; $P < 0.0001$). PWV was greater in thoracic segments of AAA patients (9.8±0.46) than control subjects (8.1±0.40; $P = 0.006$). There was no difference in PWV of aneurysmal vs abdominal segments ($P=0.137$). PWV did not change along the aortic segments in AAA subjects ($P=0.132$) but was significantly greater in abdominal segment vs thoracic segment in control subjects (9.8±0.52 vs 8.1±0.40; $P=0.012$). AAA subjects had greater total (6.68±0.88 vs 1.38±0.31; $P < 0.0001$) and segmental aortic calcium ($P < 0.0001$). CMR-PWV correlated with aortic calcium($r=0.45$, $P < 0.0001$). Linear regression showed PWV was independently correlated with AAA ($P = 0.036$). Logistic regression showed an association between AAA and PWV, mean arterial pressure and age ($P=0.047$, 0.032 and 0.016 respectively).

Conclusion

This is first study to show how segmental stiffness is modified in the presence of AAA. Aneurysm formation may be an adaptive remodeling response to hypertension.



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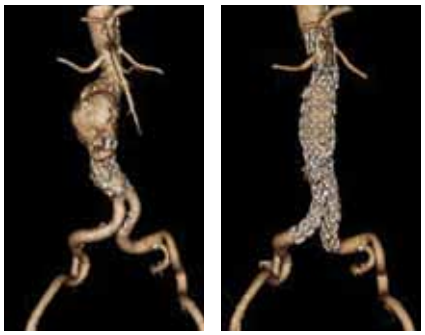

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ABSTRACTS

SARS

MicroRNAs implicated in peripheral vascular disease

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Objectives

This study aims to determine whether circulating miRNAs are differentially expressed in patients with Peripheral Vascular Disease (PVD).

Methods

Peripheral blood samples were collected from 8 controls and 5 patients with symptomatic and radiological evidence of PVD. Total RNA was extracted, converted to cDNA and pre-amplified. Quantitative PCR was performed using TaqMan Human MicroRNA cards allowing quantification of 754 microRNAs. Raw qPCR data were normalised to Mammalian U6. Students t-test was used for statistical analysis with a fold change >2, and $p<0.05$ as selection criteria. Bioinformatic analysis of predicted target genes was performed using MirWalk and Arraytrack.

Results

Background comparison revealed no significant difference in smoking, myocardial infarction, blood pressure, cholesterol, stroke or COPD history, however there was a significantly lower age in the control group (control mean age 67.3, PVD mean age 77.0, $p=0.02$).

Analysis of all 754 microRNAs revealed 9 which were differentially expressed in patients with PVD compared to controls. Two were up-regulated (miR-511 and miR-22-5p) and 7 were down-regulated (miR-672, miR-1274A, miR-1274B, miR-328, miR-1291, miR-720, and let-7g-3p).

Pathway enrichment analysis of the genes targeted by the differentially expressed microRNAs revealed targets associated with platelet aggregation, coagulation, angiogenesis, endothelial cell proliferation, and blood vessel remodelling

Conclusion

This is the first microRNA transcriptome research into PVD, identifying 9 microRNAs which are differentially expressed in patients with PVD. Predicted target analysis identified plausible biological pathways involved in the pathogenesis of PVD, bringing new insight into vascular function, and potential therapeutic pathways.

ABSTRACTS

Founders Prize Session

Predicting aortic complications after endovascular aneurysm repair: development and external validation of a morphological risk score

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Objectives

Lifelong surveillance is mandatory after endovascular repair of abdominal aortic aneurysms (EVAR), but remains costly, heterogeneous, and poorly-calibrated. The ability to predict aortic complications would allow cost-effective, risk-stratified surveillance. This study aimed to develop and validate a scoring system for aortic complications after EVAR.

Methods

Patients undergoing EVAR at two centres were studied from 2004-2010. Pre-operative morphology was quantified using three-dimensional computed tomography according to a validated protocol, by investigators blinded to outcomes. Proportional Hazards modelling was used to identify factors predicting aortic complications at the first centre, and thereby derive the risk score. Sidak tests between risk quartiles dichotomised patients to low or high-risk groups. Aortic complications were reported by Kaplan-Meier analysis and risk groups were compared by log-rank test. External validation was by comparison of aortic complications between risk groups at the second centre.

Results

761 patients aged 75 +/- 7 years underwent EVAR (AAA 66 +/- 12mm). Mean follow-up was 36+/- 20 months. Physiological variables were not associated with aortic complications. A morphological risk score incorporating maximum aneurysm diameter ($p<0.0001$) and largest common iliac diameter (measured 10mm from internal iliac origin, $p=0.0037$) allocated 75% of patients to a low-risk group, with excellent discrimination between 5-year rates of aortic complication in low-risk and high-risk groups at both centres (centre 1,12% vs. 39%, $p<0.001$ log-rank test; centre 2, 12% vs. 45%, $p=0.002$ log-rank test).

Conclusion

Our validated score uses commonly available morphology to stratify the risk of complications after EVAR. This will inform rationalised surveillance and improve cost-effectiveness.

ABSTRACTS

Founders Prize Session

Development of a patient-specific risk stratification system for patients undergoing thoracic aortic aneurysm repair

BO Patterson[1] A Karthikesalingam[1] PJE Holt1 C Nienaber[2] R Fairman[3] MM Thompson[1]

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Objectives

Endovascular repair of thoracic aneurysms (TEVAR) is associated with less short-term mortality and morbidity than open surgical repair. Despite this, serious adverse events can occur in 10-15% of patients. The objective of this study was to develop a method predicting patients most at risk of short and long-term complications.

Methods

A database was built from five prospective studies and a single institutional series. Logistic regression modelling was used to construct models to predict adverse outcomes using pre-operative patient data, which included physiological and morphological variables. Cox's regression analysis was performed to determine factors influencing mid-term re-intervention. ROC curve analysis and the Hosmer-Lemeshow test were used to determine discrimination and goodness of fit of the models.

Results

Of 670 patients that underwent TEVAR, 5% died, 5% had a stroke and 3% developed spinal cord injury (SCI) post-operatively. At 6-year follow-up, 16% of patients had underdone aortic re-intervention. The model predicting 30-day death contained age, ASA grade and emergency admission (area under receiver operator characteristic curve (ROC)=0.76). Stroke was predicted by female gender, renal insufficiency, previous CVA, coverage of the left subclavian artery and length of overage (ROC= 0.77). SCI was predicted by female gender, smoking, previous CVA, emergency admission and coverage (ROC = 0.72). Aortic re-intervention was predicted by aneurysm length, maximum diameter and iliac tortuosity (ROC=0.74)

Conclusion

Models developed using logistic regression can accurately predict important outcomes following TEVAR. External validation of these risk stratification systems is required before they can be introduced into clinical practice.

ABSTRACTS

Founders Prize Session

Development of a decision tree to streamline infra-inguinal vein graft surveillance

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Objectives

Duplex ultrasound (DU) remains the gold standard for identification and grading of infrainguinal vein graft stenosis. However, DU based graft surveillance remains controversial. The aim of this study was to develop a decision tree in order to identify high risk grafts which would benefit from DU based surveillance.

Methods

Consecutive patients undergoing infrainguinal vein graft bypass were enrolled in a duplex surveillance program. An early post operative DU was performed at a median of 6 weeks (range: 4-9). Based on the findings of this scan and four established risk factors for graft failure (diabetes, smoking, infragenicular distal anastomosis, revision bypass surgery), a classification and regression tree (CRT) was created in order to stratify grafts into grafts which are either at high or low risk of developing severe stenosis or occlusion. The accuracy of the CRT model was evaluated using area under receiver operator characteristic (ROC) curve.

Results

Of 796 vein graft bypasses performed (760 patients), 64 grafts had occluded by the first surveillance visit; 732 vein grafts were entered into surveillance programme. 126 (17.2%) developed critical vein graft stenosis. Overall 30-month primary patency, primary assisted and secondary patency rates were 76.2%, 83.6% and 85.3%, respectively. The area under ROC curve for the CRT model was 0.84 (95% CI: 0.78-0.91).

Conclusion

A prediction model based on commonly recorded clinical variables and early postoperative DU scan is accurate at identifying grafts which are at high risk of failure. These high risk grafts may benefit from DU based surveillance.

ABSTRACTS

Founders Prize Session

A comparison of the effectiveness of treating those with and without the complications of superficial venous insufficiency

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Objectives

A commonly held view is that superficial venous insufficiency (SVI) is only of cosmetic concern, yet many suffer physical symptoms impacting upon quality of life (QoL). Treatment of SVI is a key issue in the commissioning debate and commonly subjected to rationing, with reimbursement often reserved for those with complications. The aim of this study is to test this hypothesis: that patients suffering more advanced disease have greater benefit from treatment.

Methods

280 participants with SVI were randomised in an RCT to receive EVLA or conventional surgery. This secondary analysis divides patients into those with simple varicose veins (C2) and those with early complications (C3-4). Multivariable analysis was used to compare the groups over a comprehensive range of outcomes.

Results

Both C2 and C3-4 groups saw significant improvements in quality of life (QoL). These were equal between the two groups apart from Bodily Pain (SF36); where C2 saw an improvement of 12.8(95%CI 4.8-20.8) points over C3-4($p=0.002$). The C3-4 group also suffered more recurrence (OR(95%CI) 2.7(1.2-6.1)$p=0.022$) and required more secondary procedures (OR(95%CI) 4.4(1.2-16.3)$p=0.028$).

Conclusion

Patients with C3-4 disease do not see any additional QoL benefits following treatment over those with uncomplicated disease; indeed the associated pain does not fully reverse. Furthermore, whilst there is no difference in the technical success of the procedure, C3-4 patients are more likely to suffer early recurrence and require further treatment. This study demonstrates that rationing by clinical severity is misguided; waiting for the development of skin damage leads to irreversible morbidity.

ABSTRACTS

Founders Prize Session

Associations between histological features of symptomatic carotid plaque and predicted stroke risk: implications for carotid plaque imaging

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Objectives

For symptomatic patients with moderate carotid artery stenosis the risk-benefit for intervention is often marginal and is dependent on timing of surgery, presenting event, gender, age, and co-morbidity. Various modalities of plaque imaging have been promoted as potential tools for additional risk stratification, particularly in this patient group. However, it remains uncertain to what extent those carotid plaque components that can be imaged predict risk of future ipsilateral ischaemic stroke.

Methods

We related carotid plaque histology (using validated semi-quantitative scales) to predicted individual 5-year stroke risk in two large consecutive series of patients (n=1640) undergoing endarterectomy using a validated risk prediction model.

Results

Predicted 5-year stroke risk (top versus bottom quartile) was related to plaque thrombus (OR=1.42, 95%CI 1.11-1.89, p=0.02), fibrous content (0.65, 0.49-0.87, p=0.004), macrophage infiltration (1.41, 1.05-1.90, p=0.02), high micro-vessel density (1.49, 1.05-2.11, p=0.03), and overall plaque instability (1.40, 1.05-1.87,p=0.02), but not to cap thickness (1.17, 0.68-2.02, p=0.57), calcification (0.88, 0.67-1.16, p=0.37), intra-plaque haemorrhage (1.15, 0.84-1.59, p=0.38), lymphocyte infiltration (1.21, 0.74-1.99, p=0.44), or plaque rupture (1.39, 0.85-2.27, p=0.19). No significant heterogeneity between the studies was found. Plaques removed <30-days of last symptomatic event were most strongly correlated with predicted stroke risk whereas those removed >30-days revealed no significant relation.

Conclusion

Some carotid plaque features correlate with predicted stroke risk, but other features targeted by current imaging modalities (cap thickness, intra-plaque haemorrhage, calcification, and plaque rupture) are not correlated with predicted stroke risk. It remains uncertain whether any plaque features predict stroke independently of age, sex and more easily measured traditional vascular risk factors.

ABSTRACTS

Founders Prize Session

Poorly controlled BP and impaired baroreceptor function (but not impaired autoregulation) are associated with a significantly higher prevalence of post-carotid endarterectomy hypertension

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Objectives

Post-endarterectomy hypertension (PEH) is associated with intracranial haemorrhage (ICH), hyperperfusion syndrome stroke and cardiac complications. Whilst well recognised, its pathophysiology is poorly understood.

Methods

106 patients undergoing carotid endarterectomy (CEA) underwent investigations to evaluate the pathophysiology of PEH including; 24-hour ambulatory BP, central aortic BP, baroreceptor sensitivity (BRS), cerebral autoregulation and transcranial Doppler (TCD) measurement of middle cerebral artery blood flow velocity (MCAV); Clinical details, BP readings from the ward, induction of anaesthesia and during surgery, mode of anaesthesia, vasoactive medications and MCAV changes following flow restoration. Patients with PEH (defined as systolic pressure (SBP)>170mmHg + no symptoms or >160mmHg with headache/seizure/deficit) were treated according to Unit guidelines.

Results

40/106 required treatment for PEH (26 in recovery, 27 on the ward), while 7 had surges in SBP>200mmHg on the ward. PEH (recovery/ward) was not associated with pre-operative patient characteristics or TCD variables and was not associated with impaired autoregulation (autoregulation was better preserved in PEH patients (ARI 4.3 +/-1.4 vs. ARI 3.5 +/-1.6 ($p=0.03$)). PEH was significantly associated with; (i) higher pre-operative BP (peak SBP>170mmHg = 59% prevalence); (ii) peak SBP>170mmHg before induction of anaesthesia (61% prevalence) and (iii) impaired BRS (3.4 +/- 1.7ms/mmHg vs. 5.3 +/-2.8ms/mmHg, $p=0.001$). Length of stay was significantly increased in PEH patients ($p<0.001$), while three patients with temporary headache/seizure/deficit and one with delayed ICH required treatment for PEH ($p=0.02$).

Conclusion

PEH was associated with poorly controlled BP and impaired baroreceptor sensitivity, but not impaired autoregulation. Units benefit from guidelines for managing PEH, but BP optimisation should begin pre-operatively.

ABSTRACTS

Session 2 - Thursday 8.00am - 9.30am

Variable Life Adjusted Display methodology for continuous performance monitoring of carotid endarterectomy

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[1]Central Manchester University Hospitals NHS Trust [2]Academic Vascular Unit, Hull Royal Infirmary, Hull

Objectives

The aim of this study was to use Variable Life Adjusted Display (VLAD) methodology to monitor performance of six vascular surgeons undertaking Carotid Endarterectomy (CEA) in a single institution.

Methods

This was a prospective study with continuous analysis. A risk score model to predict 30 day stroke or death for individual patients was developed from data collected from 839 patients from 1992 to 1999. The model was used to monitor performance of 6 surgeons from 2000 onwards. Individual risk factors and 30 day outcomes were analysed and VLAD plots were created for the whole unit and for each surgeon.

Results

Among the 941 CEAs in the performance analysis, 28 adverse events were recorded, giving an overall stroke or death rate of 3.06%. The risk model predicted there would be 33 adverse events. There were no statistical difference between the predicted and the observed adverse events ($p>0.2$, $\chi 2$ value 1.25, 4 degrees of freedom). The VLAD plot for the whole unit shows an overall net gain in operative performance; however, this could have equally been to chance variation. The individual VLAD plot showed surgeons 1, 2, 3 & 6 to have an overall net gain in the number of successful operations. The changes observed between the surgeons was not significant ($P>0.05$) suggesting chance variation only.

Conclusion

Performance of CEA can be continuously assessed using VLAD methodology for units and individual surgeons. Early identification and correction of performance variation could facilitate improved quality of care.

ABSTRACTS

Session 2 - Thursday 8.00am - 9.30am

Performance of risk prediction scores for carotid endarterectomy

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University College London

Objectives

Risk scores for CEA may give patients an individualised estimate of their risk of suffering stroke or death within 30 days of the procedure. We examined the performance of three existing risk scores in the International Carotid Stenting Study (ICSS).

Methods

Baseline patient demographic data was collected by ICSS investigators. One risk point was allocated for female sex, peripheral vascular disease or systolic BP>180mmHg (Rothwell et al, 1999); one point for TIA or stroke as the index event, atrial fibrillation, heart failure, diabetes or contralateral carotid occlusion (Tu et al, 2003); or one point for heart disease, diabetes or stroke as the index event (Kuhan et al, 2001).

Results

The observed rate of stroke or death within 30 days of CEA was 2.5% for patients with a Rothwell score of 0, 4.4% for score of 1, and 5.4% for score 2 (Chi-squared test for trend p=0.10). The event rate was 2.6% in patients with a Tu score of 0, 3.4% for score 1 and 4.7% for score 2 (Chi-squared test for trend p=0.49). The event rate was 2.2% for patients with a Kuhan score of 0, 3.7% for a score of 1, 4.2% for a score of 2 and 5.9% for a score of 3 (Chi-squared test for trend p=0.15). More than 50% of events occurred in lower-risk groups in all three risk models.

Conclusion

Existing risk scoring systems for CEA do not adequately predict individual risk. The observed rate of stroke or death within 30 days of CEA in ICSS is low.

ABSTRACTS

Session 2 - Thursday 8.00am - 9.30am

The potential effect on vascular services of the implementation of recent NICE guidance for the management of deep vein thrombosis

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Objectives

Recent NICE guidance recommends the use of catheter-directed thrombolytic therapy (CDT) in selected patients with symptomatic ilio-femoral deep vein thrombosis. The aim of this study was to assess the new case load that this recommendation would cause and its effect upon service provision.

Methods

Using our radiology database, a retrospective review was done of all the DVTs diagnosed at a large teaching hospital between August 2010 and Feb 2012. NICE guidance was applied retrospectively to this cohort, using case note review and an independent dual clinician analysis to determine those patients that would have been suitable for CDT.

Results

563 patients with radiological confirmed DVTs were identified over the 18 month period. 53 of the 128 patients with iliofemoral DVT would have been eligible for intervention with CDT using the NICE guidelines. Only 8 of the 53 (15%) were referred to vascular services. All 8 patients had successful CDT, which involved stays in critical care for monitoring (median number of sessions = 2, range 1-3).

Conclusion

Vascular units should be prepared for a 7 fold increase in requests for CDT for iliofemoral DVT. This increase will affect in-patient bed, interventional suite and critical care usage.

ABSTRACTS

Session 2 - Thursday 8.00am - 9.30am

Long term functional outcome of treatment for Paget-Schroetter and Venous Thoracic Outlet Syndrome

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Objectives

Consensus on the management of Paget-Schroetter (PSS) and venous thoracic outlet syndrome (VTOS) is lacking. Some advocate anticoagulation only; others lysis and decompression.

Methods

Analysis of clinical and functional outcome of patients treated for PSS and VTOS over 16 years was performed with follow up to 10 years. Patients were divided into five groups depending on presentation. Outcomes were analysed using the Disabilities of Arm, Shoulder and Hand (DASH) questionnaire.

Results

132 patients (M:59, F:73, mean age 32 years) were included. DASH scores in surgically treated groups improved significantly ($p=<0.0001$). Early surgery had better outcomes than delayed surgery ($p=0.04$). Conservatively managed patients failed to improve ($p=0.12$). Venoplasty was successful in 22 (66%) patients. Delayed surgery in smokers and short duration (<24 h) lysis increased pulmonary embolism (PE) ($p=0.014$) and re-thrombosis ($p=0.02$) rates. Choice of post-operative anticoagulation did not influence re-thrombosis. There were three haemothoraces, one PE and no neurovascular injuries.

Conclusion

Surgery for PSS is safe and has better outcomes than conservative treatment. Prompt thrombolysis and surgery is superior to delayed management regarding re-thrombosis and functional outcome. Smoking increased the risk of pulmonary embolism. The benefit of post-operative anticoagulation is questioned and requires further elucidation.

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ABSTRACTS

Session 2 - Thursday 8.00am - 9.30am

Early results for the use of NHS blood and transplant derived Decellular Dermis in the management of treatment resistant leg ulcers

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Objectives

We present early findings of an original clinical trial, investigating efficacy of a novel skin substitute called "Decellular Dermis" (DCD) produced by NHS Blood and Transplant (NHSBT), as part of a unique one stage therapeutic strategy for recalcitrant ulcers of the lower limb.

Methods

Twenty patients were recruited from hospital and community leg ulcer services. On day 0, ulcers were hydrosurgically debrided before DCD was applied, secured with tissue glue and covered with a compact, mobile and disposable negative pressure dressing for one week. Patients were followed up on 7 occasions over 6 months. Imaging methods including clinical photography, full field laser perfusion imaging and spectrophotometric intracutaneous analysis were used alongside full thickness skin biopsies from the wound periphery before and after therapy, to assess the rate and nature of wound healing.

Results

All patients had chronic treatment resistant ulcers of variable duration. 70% were venous in origin, 15% diabetic and the remainder of mixed aetiology. 6 weeks after grafting all ulcers had reduced in size and laser perfusion imaging showed significantly increased haemoglobin flux in the wound bed. Histological analysis confirmed the DCD was progressively integrated into the wound bed with increasing cellularity, vasculogenesis and granulation tissue formation.

Conclusion

Application of DCD safely produced substantial macroscopic and microscopic improvement in treatment-resistant lower limb ulcers. As there is no requirement for hospital admission, anaesthetic, or autogenic skin grafting, this novel application method could be widely adapted to hospital and community settings. DCD offers potentially significant clinical and financial advantages in chronic wound management.

ABSTRACTS

Session 2 - Thursday 8.00am - 9.30am

Effect of the angiotensin converting enzyme inhibitor, ramipril, on walking distance, arterial stiffness and quality of life in patients with intermittent claudication (ACEIIC): randomised, double blind, placebo-controlled trial

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Objectives

To investigate ACE inhibitors effect on walking distance, ankle brachial pressure index (ABPI), arterial stiffness and quality of life (QoL) in patients with intermittent claudication (IC).

Methods

33 patients (25 males, mean age: 65+/-7.8) with IC (Fontain II or higher) were randomised to receive ramipril (5 mg od for 2 weeks increased to 10 mg od for 22 weeks, n=14) or placebo (n=19) for 24 weeks in a double blind study. Walking distance was assessed using a standard laboratory treadmill test (1.6 mph at 10 degree incline). ABPI was assessed pre (ABPI-r) and post exercise (ABPI-t). Arterial stiffness indices were measured using the SphygmoCor device.

Results

After 24 weeks, ramipril improved maximum treadmill walking distance (ramipril 153.6+/-96.7 metres vs placebo 23.5+/- 79.9 metres, p<0.001), improved treadmill intermittent claudication distance (ramipril 141.6 +/- 112 metres vs placebo 24.8 +/- 55.8 metres, p=0.005) and improved patient reported walking distance (ramipril 180+/-195.6 metres vs placebo 35+/-162.5 metres, p=0.03). Ramipril reduced carotid femoral pulse wave velocity (a measure of arterial stiffness) by -0.89+/-1.13 m.s-1 compared to an increase by placebo of 0.56+/- 1.16 m.s-1, p=0.003. However, ABPI-r and ABPI-t minimally changed in both groups (ramipril -0.01+/-0.16 vs placebo 0.01+/-0.18, p=0.64) and (ramipril 0.01+/-0.18 vs placebo 0.01+/-0.14, p=0.87), respectively. Physical domains of short form 36 and EQ5D scores insignificantly improved after ramipril treatment compared to placebo.

Conclusion

Ramipril improves walking distance in patients with intermittent claudication; however, this improvement is not related to improvement in ABPI but might be due to ramipril ability to reduce arterial stiffness. ACE inhibitors effect on QoL needs to be validated in a larger RCT.

ABSTRACTS

Session 2 - Thursday 8.00am - 9.30am

The development of a national risk prediction model for elective abdominal aortic aneurysm (AAA) repair

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Objectives

Mortality results for elective abdominal aortic aneurysm (AAA) repair are published by the Vascular Society of Great Britain and Ireland. These mortality results are not currently risk-adjusted. The objective of this study was to develop a national risk prediction model for elective AAA repair.

Methods

Data for 11,423 consecutive patients undergoing elective AAA repairs from the National Vascular Database (NVD) between April-2008 and March-2011 were analysed. Multiple logistic regression and backwards model selection were used for model development. The primary outcome measure was in-hospital mortality. Model calibration and discrimination were assessed for all AAA repairs and in open AAA repair and EVAR sub-groups separately.

Results

There were 312 in-hospital deaths (2.7%; 95%CI 2.4%-3.0%). Mortality following open AAA repair was 4.5% (95%CI 4.0%-5.1%) and following endovascular AAA repair was 1.3% (95%CI 1.1%-1.6%). Variables associated with in-hospital mortality included in the final model were: open AAA repair, increasing age, female gender, creatinine > 120μmol/l, cardiac disease, abnormal ECG, previous aortic surgery or stent, abnormal white cell count, abnormal serum sodium, AAA diameter and ASA grade. The area under the receiver-operating characteristic (ROC) curve was 0.774 (95% CI: 0.747-0.799) with a bias-corrected value of 0.766 respectively. Model calibration was good based on the Hosmer-Lemeshow test (P = 0.933), [bias-corrected] calibration curves and recalibration regression.

Conclusion

This multivariable model, developed to predict in-hospital mortality following elective AAA repair, can be used to risk-adjust current clinical governance analyses and can also be used to calculate patient specific risk.

ABSTRACTS

Session 2 - Thursday 8.00am - 9.30am

A systematic review and meta-analysis of Duplex ultrasound, contrast-enhanced ultrasound or computed tomography for surveillance after endovascular aneurysm repair

A Karthikesalingam[1] W Al-Jundi[2] D Jackson[3] JR Boyle[4] JD Beard[2] PJE Holt[1] MM Thompson[1]

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Objectives

Previous analyses suggested Duplex ultrasound (DUS) detected endoleaks with insufficient sensitivity, but have not specifically examined type 1 and 3 endoleak, which, if untreated, leads to aneurysm-related mortality. In light of changes to clinical practice, the diagnostic accuracy of DUS and contrast-enhanced ultrasound (CEUS) for type 1 and 3 endoleak requires focused appraisal.

Methods

Studies comparing DUS or CEUS with CT for endoleak detection were identified. CT was taken as the 'gold-standard' in bivariate meta-analysis.

Results

25 studies (3985 scan pairs) compared DUS with CT for all endoleaks. The pooled sensitivity was 0.74 (95% CI 0.62 - 0.83), pooled specificity was 0.94 (95% CI 0.90 - 0.97). 13 studies (2650 scan pairs) reported detection of type 1 and 3 endoleak by DUS; its pooled sensitivity was 0.83 (95% CI 0.40 - 0.97) and its pooled specificity was 1.00 (95% CI 0.97-1.00). 11 studies (961 scan pairs) compared CEUS to CT for all endoleaks. The pooled sensitivity of CEUS was 0.96 (95% CI 0.85-0.99), pooled specificity was 0.85 (95% CI 0.76-0.92). 8 studies (887 scan pairs) reported detection of type 1 and type 3 endoleak by CEUS. The pooled sensitivity of CEUS was 0.99 (95% CI 0.25-1.00) and its pooled specificity was 1.00 (95% CI 0.98-1.00).

Conclusion

Both CEUS and DUS were specific for type 1 and type 3 endoleak. Estimates of their sensitivity were uncertain but there was no evidence of clinically important difference. DUS detects type 1 and 3 endoleak with sufficient accuracy for surveillance after EVAR.

ABSTRACTS

Session 2 - Thursday 8.00am - 9.30am

Magnetic resonance imaging using a novel elastin peptide (MRI-EP) tracer demonstrates inflammation precedes matrix degradation and expansion in aortic aneurysms

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Objectives

Elastin degradation is an important pathological process that occurs in aortic aneurysms. For the first time in-vivo MR imaging was used to quantify elastin degradation with a novel elastin peptide.

Methods

We used the ApoE-/-/Angiotensin II murine model to generate aortic aneurysms and serially imaged with MRI-EP at six time points over five weeks. We determined the aortic diameter, aortic wall elastin uptake and T1-relaxation times for aneurysm vs. normal aortic wall. Aortic wall from sites of varying elastin tracer uptake underwent flow cytometric, immunohistochemical and histological analysis.

Results

At regional aortic wall sites preceding elastin tracer uptake had increased number of B cells (63.8±13.0, $p<0.001$, 95%CI0.54) and T cells (82±, 9.8 $p<0.001$, 95%CI63.4) vs. sites of no elastin peptide binding B cell (19.4±14.1, $p<0.001$, 95%CI0.54) T cell (57.8±8.9, $p<0.001$, 95%CI0.64). The number of NK cells, cells from monocyte/ macrophage lineage was not significantly altered. EP tracer uptake correlated with high anatomic specificity to elastin and tropoelastin in the tunica media on immunohistochemistry.

Conclusion

For first time in-vivo imaging with a novel MR elastin tracer has shown the relationship between inflammation and aneurysm wall matrix degradation. Functional imaging with matrix-targeted tracers will increase our understanding of aneurysm pathology.

ABSTRACTS

BJS Prize Session

Refining the association between low density lipoprotein receptor related protein 1 and abdominal aortic aneurysm

JB Wild[1] HZ Butt[1] N Sylvius[1] PW Stather[1] GT Jones[2] DJA Scott[3] J Lindholt[4] E Choke[1] RD Sayers[1] MJ Bown[1, 5]

[1]University of Leicester [2]University of Otago [3]University of Leeds [4]Viborg Hospital, Denmark [5]The Aneurysm Consortium

Objectives

A recent genome wide association study (GWAS) has identified a significant association between LRP1 and abdominal aortic aneurysms (AAA). We aimed to refine regional associations at this locus and determine if any genotypes influenced the expression of LRP1 in peripheral blood samples.

Methods

Imputation of GWAS data (5435 controls and 1866 AAA) and bioinformatic analyses of the LRP1 gene were undertaken to determine the targets for laboratory genotyping studies of 589 controls and 925 patients with AAA. Peripheral blood gene expression of LRP1 was determined in a sub-group of these samples (83 controls and 75 AAA).

Results

Imputation and bioinformatic analyses identified six single nucleotide polymorphisms (SNPs) of interest within the LRP1 gene. Combined imputed GWAS data and laboratory genotyping revealed that one previously untyped SNP (rs11172114) had a stronger association with AAA (P=3.87x10-8, OR 1.22 (95% CI 1.14 to 1.31)) than the lead SNP from the GWAS (rs1466535, P=7.42x10-7 OR. 1.16, 95% CI 1.09 to 1.22). There was a significant difference in LRP1 haplotype frequencies between the patients with AAA and the control group (P = 0.02). Peripheral blood LRP1 mRNA levels were unaffected by any of the SNPs or haplotypes tested.

Conclusion

This study demonstrates that the strongest regional association at the 12q13.3 locus lies within the first intron of the LRP1 gene and that there is a significant variation in LRP1 haplotypes between AAA and controls.

ABSTRACTS

BJS Prize Session

Transcriptomic profiles in abdominal aortic aneurysm - a validated microarray based study

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University of Leicester

Objectives

We previously conducted microarray-based transcriptomic profiling comparing large abdominal aortic aneurysms (AAA) to screened controls and identified a panel of differentially expressed genes. This study aimed to validate these findings in an extended cohort of patients.

Methods

RTPCR was used to quantify the expression of 11 genes in peripheral blood samples from patients with large AAA (>5.5cm) (n=61), controls (n=93), post EVAR (n=24) and post open repair (n=15). We also measured gene expression in biopsies from patients undergoing open AAA repair (n=18) and in vascular smooth muscle explants cultured from these biopsies (n=7).

Results

Amongst all 11 genes quantified, MSN (moesin) expression was significantly lower in large AAA against matched healthy control subjects (fold change (FC) 0.89, p=0.024). MSN expression however remained unchanged after AAA repair (p>0.2).

There was reduced CASP2 (caspase 2) expression after both open AAA repair (FC 0.83, p=0.04) and EVAR (FC 0.91, p=0.02). In addition, EIF3G, SIVA, PUF60, CARD8 and FIBP expression was lower post EVAR compared to their preoperative expression levels (FC 0.78-0.91, p<0.03).

Expression of all above genes was detected in matched full thickness aortic biopsies obtained at open AAA repair, and evident in cultured smooth muscle derived from the same aortic biopsies.

Conclusion

We demonstrate validated differential expression of previously undescribed transcripts in AAA with functional roles in proteolysis, inflammatory and apoptotic processes. These differentiated AAA from screened controls, were modulated by surgery, and also expressed in matched aortic wall biopsies and vascular smooth muscle.

ABSTRACTS

BJS Prize Session

18F-FDG PET-CT uptake is a feature of both normal diameter and aneurysmal aortic wall and is not related to aneurysm size

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Objectives

Aneurysm wall 18F-FDG uptake on PET-CT has been used as a measure of metabolic activity and inflammation, but in fact the relationship between uptake and aortic size is poorly understood. We studied 18F-FDG uptake in a large population of infra-renal AAA and matched controls.

Methods

The PET-CT database of 5000 sequential body 18F-FDG PET-CT studies from 2005-2008 performed for routine indications was searched for a diagnosis of infrarenal AAA. Exclusion criteria were prior repair, vasculitis, and saccular/mycotic thoracic or thoracoabdominal aneurysms. Matching of non-aneurysmal (<3cm) sequential controls from the same population was assessed using t- and chi-squared tests. Standardised uptake value (SUV)-max of the infrarenal-aorta and background SUV and maximum infrarenal aortic diameter were extracted by an experienced nuclear medicine radiologist. Follow-up questionnaires were sent to referring clinicians. Statistical analysis was performed in SPSS 20.

Results

Aneurysms (n=151) and controls (n=159) were matched ($p>0.05$) for age, sex, diabetes, hypertension, smoking status, statin use and indication for PET-CT. Median aortic diameter was 5.0cm (range 3.2-10.4) for aneurysms and 2.1cm (1.4-2.9) for controls. During a median 8 (IQR 2-21) months follow-up 22 were repaired and 4 were confirmed ruptured. Aneurysm diameter correlated with age ($r=0.18$, $p=0.001$). SUVmax was slightly lower in the aneurysm group versus controls (1.75 vs 1.84, $p=0.02$). No other associations with SUVmax were identified.

Conclusion

Although previous studies have shown 18F-FDG uptake may be related to aneurysm dilation we have shown it also occurs in non-aneurysmal aortic wall and in AAA is not related to diameter.

ABSTRACTS

BJS Prize Session

Reduced nitric oxide bio-availability post-major vascular surgery pre-disposes to myocardial injury and predicts All Cause Mortality in medium term

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Objectives

Recent interest has focused on the role of the endogenous inhibitors of nitric oxide synthesis (NOS) asymmetrical dimethylarginine (ADMA) and symmetrical dimethylarginine (SDMA) in patients with cardiovascular disease and as prognostic indicators. We aimed to determine the effect of major vascular surgery on dimethylarginines, L-arginine (NO precursor) and the relationship with post-operative myocardial injury and long term outcomes.

Methods

128 patients undergoing lower limb revascularisation or open repair of AAA L-arginine, ADMA and SDMA levels were measured pre- and post-operatively (day 1 cardiac events were documented in the early post-operative period and a 2 year follow up was done.

Results

L-arginine levels fell significantly following surgery [Median(IQR) 56.4μmol/L(44.1-68.9] pre-op versus 53.2μmol/L(43.2-62.8) post-operatively, $p=0.03$). SDMA levels increased post-operatively [0.491μmol/L(0.409-0.612) versus 0.559μmol/L(0.476-0.701),$p<0.001$] while ADMA level was unchanged.

27(21%) patients had a post-operative myocardial injury (rise in troponin-I> 0.1ng/ml). Baseline L-arginine levels were similar in the troponin-I positive and troponin-I negative patients. Post-operative levels of L-arginine were lower in the troponin positive group [45.8μmol/L(37.1-56.7) versus 54.4μmol/L(43.9-65.9);$p=0.04$]. SDMA levels were significantly higher in the troponin positive group compared to the troponin negative group both pre-operatively [0.599μmol/L(0.483-0.825) versus 0.479μmol/L(0.395-0.593);$p<0.001$] and post-operatively. SDMA remained an independent predictor of post-operative cardiac injury and All Cause Mortality (ACM) in two year follow up.

Conclusion

This study shows for the first time that nitric oxide availability is reduced in patients undergoing major vascular surgery, particularly in those who sustained a post-operative myocardial injury. This is also the first one to show that SDMA predicts adverse outcome in immediate post-operative phase and long term.

ABSTRACTS

BJS Prize Session

Atorvastatin reduces vein wall inflammation and enhances recanalisation following venous thrombosis

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Academic Department of Surgery, King's College London, BHF Centre of Research Excellence & NIHR Biomedical Research Centre at Kings Health Partners, London

Objectives

Statins (HMG-CoA reductase inhibitors) have anticoagulant and anti-inflammatory actions, and promote angiogenesis and fibrinolysis. We evaluated the effects of atorvastatin in a model of venous thrombus formation, organisation and recanalisation.

Methods

The effect on thrombus formation was studied by treating BalbC mice with Atorvastatin 1,3,10,30 mg/kg or vehicle for 14 days (n=14/group) prior to vena cava thrombus induction. Vena cava containing thrombi were harvested one day later.

The effect on thrombus resolution was studied by inducing thrombi in mice. One day later mice were randomised to treatment with Atorvastatin (30mg/kg/day or 3mg/kg/day) or vehicle (n=7/group). On day-7 thrombi were harvested. Vein recanalisation, thrombus volume and inflammatory cell content [macrophages (MAC-2) and neutrophils NIMP-R14)] in thrombus and vein wall were measured.

Results

Statin treatment did not affect venous thrombus formation (1mg, 11/14 formed thrombus; 3mg, 11/14; 10mg, 12/14; 30mg, 12/14; vehicle, 11/14; $P > 0.05$).

Vein recanalisation was greater following treatment with high dose Atorvastatin ($0.50 \pm 0.13 mm^3$) compared with low dose or vehicle ($0.29 \pm 0.11 mm^3$, $0.27 \pm 0.13 mm^3$ respectively, $P=0.002$ ANOVA). Significantly more neovascular channels developed in thrombus in both high and low dose treatment groups (5.0 ± 0.4 and 5.1 ± 0.5 respectively) compared with vehicle (3.1 ± 0.4; $P=0.009$). Areas of macrophage staining ($0.41\% \pm 0.04$, $0.45\% \pm 0.04$) and neutrophil staining ($3.92\% \pm 0.38$, $3.76\% \pm 0.48$) were significantly lower in the vein wall of statin treated groups compared with vehicle ($0.97\% \pm 0.05$, $P<0.001$; $7.33\% \pm 0.36$, $P<0.001$ respectively).

Conclusion

Atorvastatin treatment had no effect on thrombus formation, but significantly enhanced vein recanalisation and inhibited vein wall inflammation. Statin treatment may be beneficial following deep vein thrombosis.

ABSTRACTS

BJS Prize Session

Apoptotic cell death in ischaemic skeletal muscle is mediated via the Toll-like receptor (TLR) adapter protein MyD88

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Objectives

Critical limb ischaemia (CLI) is a cause of significant morbidity and mortality and is associated with skeletal muscle damage and death in the form of apoptosis. TLRs have been implicated in ischaemia-induced tissue damage. Two TLR signalling transduction pathways exist: the MyD88-dependent and TRIF-dependent pathways. We aim to investigate which of these are involved in ischaemia-induced muscle damage and apoptosis. We hypothesize that skeletal muscle ischaemia induces apoptotic cell death which is mediated via activation of the MyD88-dependent pathway.

Methods

TLR 2/6 expression and apoptosis was studied in ischaemic and control human muscle biopsies and in ischaemic C2C12 myotubes using western blot and immunofluorescence. Activation of the signaling pathway was assessed using phosphorylated inhibitor of kappa B (**IκBα**). Functional effects of MyD88 and TRIF inhibition on ischaemia-induced IL-6 release and apoptosis were studied in myotubes incubated with MyD88 and TRIF inhibitors. IL-6 release was assayed by ELISA. Apoptosis was assessed using cleaved caspase-3.

Results

TLR 2/6 and vcleaved caspase-3 protein expression was upregulated in ischaemic human muscle and ischaemic C2C12 myotubes ($p<0.05$). TLR 2/6 signaling pathway was activated in ischaemic conditions as shown by upregulation of phosphorylated **IκBα**. Ischaemia-induced IL-6 release and apoptosis was reduced by MyD88 but not TRIF inhibition ($p<0.05$).

Conclusion

Upregulation and activation of TLR 2/6 occurs in CLI resulting in IL-6 release and apoptosis which contributes to inflammation and muscle damage in ischaemia. The MyD88-dependent pathway is important in TLR mediated muscle damage and death and thus may have potential therapeutic implications.

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ABSTRACTS

Session 4 - Thursday 1.30pm - 3.00pm

The effect of acute kidney injury (AKI) on outcomes following repair of abdominal aortic aneurysm (AAA)

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Objectives

Data were collected, using the AKI network definitions, to determine the rate and effect of AKI on outcomes following repair of AAA.

Methods

Data were collected using AKI network definitions, through the National Vascular Database. Missing data were sought by follow up questionnaire. AKI was defined as a rise in creatinine (from pre- to 48h post-operative) of > 1.5.

Results

There were 8088 patients. AKI data were entered in 45% of cases. Urine output was only recorded in 18.8% for 24 hour and 13.7% for lowest 6 hourly totals. In those with creatinine available, 3083 patients did not have AKI, and 576 developed AKI (19%). Elective patients with missing AKI data were 2.3x more likely to die (CI 1.4-3.8) than patients whose AKI data were recorded (OR (955) 2.3% with, 5.4% without AKI data ($p=0.001$), EVAR(1759) 1.1% with, 1.4% without AKI data ($p=0.324$) chi square).

Conclusion

Using binary logistic regression, risk factors for death for elective patients with 48 hours of AKI data were: age above median OR 1.05(ci(1.009-1.01) $p<0.05$), cardiac history OR 2.4(ci(1.2-4.5)$p<0.05$), AKI OR 11(ci(5.9-20.9)<0.001).

The risk of death was 2-3 fold higher in open repair and in patients with cardiac disease. Patients who develop AKI are 11 times more likely to die following AAA repair than those who do not. Failure to supply AKI data may reflect a complicated post-operative course.

ABSTRACTS

Session 4 - Thursday 1.30pm - 3.00pm

Long term results of endovascular aortic aneurysm repairs in the young

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Objectives

To determine the long term outcome and the re-intervention rate after elective endovascular aortic aneurysm repairs in patients under the age of 65 years.

Methods

A retrospective analysis of prospectively collected data was performed of all patients under the age of 65 years who had elective EVAR between 1994 and 2012. Mortality and re-intervention rates were determined.

Results

Of 1100 EVARs performed over this period 97 were in patients under the age of 65 years (93 male, mean age 61 years +/- 4). The 30 day mortality rate was 2% whilst the long term mortality rate was 34% at a median follow up of 6 years (IQR 2-12 years). Cause of death was non-AAA related in 74%, AAA related in 18%, information was unavailable in 8%. The mean life expectancy of this group of young patients with aneurysm disease was 6 years (+/- 4 years).

Data on re-intervention rates was available in 83/97 patients. There were 9 (11%) re-interventions; of which 8 re-interventions occurred in 46 patients operated pre-2000 (mean 1 intervention/year) and 1 re-intervention occurred in 51 patients operated post 2000 (mean 0.2 intervention/year).

Conclusion

The present study identified that despite the good 30 day mortality rate for EVARs, over a third of all patients died over a six year follow up, mainly from non-aneurysm related deaths. Young patients with AAA do not necessary have long lifespans due to the significant comorbidities and EVAR should be considered as a treatment option in these patients.

ABSTRACTS

Session 4 - Thursday 1.30pm - 3.00pm

Superficial Femoral artery (SFA) stenting. Does it offer any advantage?

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Objectives

Meta-analysis suggests routine SFA stenting is not associated with a significant reduction in the rate of restenosis or target vessel revascularisation. (TASC I), (TASCII) and NICE guidelines recommend only an adjunctive role for femoropopliteal stents following suboptimal angioplasty.

Despite the lack of evidence, SFA stenting seems to be a common practice across UK.

Methods

Retrospective review of all patients who underwent SFA stenting between Apr 04-Apr 12 (N=235). Mean follow up 5y [range 2-8].

Except for the earliest 2 years, all patients were enrolled in surveillance programme for 18-24m. Symptomatic patients were followed up for longer. Recurrent symptoms triggered further surveillance.

Results

Indications for stenting:
53; sub-optimal angioplasty (22.5%)
146; critical ischaemia (62%)
18; acute ischaemia (7.7%)
20 cases incomplete data (8.5%)
29 stents failed within 30 days (12.3%), 2 in claudicants, 23 in critical ischaemia and 4 in acute ischaemia.

There were 90 late failures (38.3%) resulting in surgical bypass (14.7%), amputation (7.3%), or further angioplasty (11%).

Overall primary patency (PP) was 81.7%, secondary patency (SP) was 86%. Average time to secondary intervention was 7 months. TASC A/B (N=85) PP (82%), SP (90%). TASC C/D (N=129) PP (73.6%), SP (89%).

Conclusion

SFA stenting appears to improve patient outcome. However, in our hands the rate of restenosis is quite high. A randomised controlled trial is essential if the role of SFA stents is to be identified.

ABSTRACTS

Session 4 - Thursday 1.30pm - 3.00pm

Airborne contamination during graft insertion in vascular surgery

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Objectives

To compare bacterial fallout during vascular prosthesis insertion and orthopaedic major joint replacement performed in conventional and laminar flow ventilation respectively.

Methods

Potential microbial contamination was measured in 21 patients undergoing vascular surgery (infra inguinal bypass n=8, aortic surgery n=2 and carotid endarterectomy with prosthetic patch, n=11) performed in conventional theatre ventilation compared with 24 patients undergoing major joint replacements (hip replacement n=16, knee replacement n=7 and elbow replacement n=1) performed in laminar flow ventilation.

Bacterial fallout was measured using agar plates, 4 plates were exposed throughout instrument preparation, patient transfer and procedure, 4 plates were covered during patient transfer and 2 control plates remained covered though out. Duration of exposure was recorded. After 24 hours incubation bacterial colonies were counted by an independent assessor.

Results

Bacterial fallout in the vascular group was 15 fold greater than in the orthopaedic one (15, (IQR) 15 vs 1, (IQR) 3 respectively, $p<0.0001$, Wilcoxon). Although vascular procedures took twice as long, no correlation between bacterial fallout and duration was detected in either group (vascular R-squared=.001, $p<0.30$, orthopaedic R-squared=.001, $p=0.28$). There was no difference in plates which were exposed during patient transfer compared to plates that were covered ((median (IQR) vascular covered 13.5(8-23) vs exposed 15(9-24) and orthopaedic covered 1(0-3) vs exposed 1(0-3)) indicating there was no significant bacterial fallout during patient transfer.

Conclusion

Orthopaedic surgery is routinely performed in laminar flow ventilation which dramatically reduces airborne bacterial contamination of prosthetic joint replacements. However this practice has not been widely adopted for vascular surgery in which prosthetic infection may also result in significant mortality and morbidity.

ABSTRACTS

Session 4 - Thursday 1.30pm - 3.00pm

Patients with symptomatic carotid stenosis, turned down for endarterectomy, have high re-stroke and mortality rates

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Objectives

Early carotid endarterectomy (CEA) incurs significant reduction of further ischaemic events in patients with symptomatic carotid artery stenosis. There is limited current information about patients with symptomatic carotid artery disease turned down for CEA. Data on patients with symptomatic stenosis presenting to a regional vascular unit is presented including patients turned down for CEA.

Methods

Departmental databases were interrogated between January 2010 and April 2012. The demographics and outcomes of symptomatic patients who underwent CEA (n=114) and CEA turndowns (n=39) were evaluated. Patient management was determined jointly by the stroke physicians and vascular surgeons within the multidisciplinary team.

Median time to CEA from presentation was 7 days (interquartile range 2-12 days). Thirty-day CEA outcomes were; stroke 2.6%, stroke/death 3.5%, stroke/death/myocardial infarction 5.2%. Seventy-four percent of CEAs had post-operative stroke physician follow up.

Results

CEA turndowns were predominantly for physical (46%) and cognitive co-morbidity (15%), 30-day re-event rate was 28%. Median follow up of 11 months demonstrated mortality was 21%. Mortality was higher for patients with re-events versus those with no further ischaemic symptoms (37% versus 7%, p=0.04).

Conclusion

Early CEA for symptomatic carotid artery stenosis was associated with low mortality and morbidity in this series. The outcome for CEA turndowns with a further ischaemic event is poor. There is a need to examine this subgroup in order to reconsider best treatment options.

ABSTRACTS

Session 4 - Thursday 1.30pm - 3.00pm

Endovascular recanalization of deep veins to treat C6 leg ulcers - going beyond standard therapies of compression and varicose veins surgery

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Objectives

To review our experience with endovascular recanalization of the deep venous system of the leg in the management of active venous ulceration.

Methods

Patients with active venous ulceration (CEAP class C6) who had undergone any form of endovascular recanalization between February 2011 and July 2012 were identified from a prospectively maintained database. Demographics, history, presentation, procedural and post intervention outcomes were evaluated.

Results

Twenty-six patients (32 legs) (M:F 20:6) with mean age 45 years were identified. Six patients had bilateral leg ulcers; 20 unilateral (L:R 11:9). The median duration of ulceration was 36 months prior to intervention. Only 2 patients had a documented history of DVT, but radiological findings were consistent with post thrombotic sequelae in 23. The iliac system was involved in 23/26 patients and 8/26 had additional IVC disease, with 1 patient having only IVC involvement. Three patients needed treatment of the common femoral vein or femoral vein. All stenotic or occluded veins were treated with angioplasty and stenting using a combination of ELuminexxTM and NitiTM Nitinol stents.

At median follow up of 30 weeks (2-70), complete and sustained ulcer healing was achieved in 65% of limbs with 50% healed by 6 weeks. Ulcers had improved in 26%. There was one recurrence and one failure to improve. One stent occluded within the first week.

Conclusion

Disease in the deep system of patients with venous ulceration can be satisfactorily treated to achieve rapid wound healing in patients with long standing ulcers due to such disease.

ABSTRACTS

Session 4 - Thursday 1.30pm - 3.00pm

The prevalence and impact of antiplatelet resistance in carotid artery disease

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Objectives

Stroke risk in carotid artery disease (CAD) stems from embolic material arising from carotid plaques. As platelet activation plays a key role in plaque initiation and growth, platelet inhibition through Aspirin and/or Clopidogrel is the cornerstone of clinical management in CAD providing an annual reduction in stroke risk of 9%. Research has indicated that resistance to antiplatelet agents may be significantly prevalent in the population, although antiplatelet resistance could render the current pharmacological management of CAD ineffective the prevalence and impact of antiplatelet resistance in CAD is yet to be determined.

Methods

Response to antiplatelet therapy in 30 CAD patients taking Aspirin and/or Clopidogrel was assessed via Multiplate Impedance Aggregometry. Factors known to be associated with increased stroke risk in CAD were measured in these patients in order to investigate the impact of resistance on stroke risk.

Results

37% of patients did not experience effective platelet inhibition despite intervention with an antiplatelet agent. Resistant patients were shown to have large carotid plaque volumes, increased frequency of microembolic signals in the middle cerebral artery and had more commonly suffered from cerebrovascular events such as stroke, transient ischaemic attack and amaurosis fugax than antiplatelet responders, therefore indicating increased stroke risk and clinical priority.

Conclusion

Results from this study suggest that antiplatelet resistance is a significant issue impacting the level of safe treatment currently available in CAD. Therefore, the implementation of antiplatelet resistance screening should be considered as a realistic option to improve clinical management of the disease.

ABSTRACTS

Session 4 - Thursday 1.30pm - 3.00pm

Histological features of carotid plaque in patients with ocular ischaemia versus cerebral events

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Objectives

Patients with carotid artery stenosis and ocular ischaemic events have a much lower risk of future ipsilateral ischaemic stroke on medical treatment and lower procedural risks for endarterectomy and stenting than patients with cerebral ischaemic events and are closer in risk to patients with asymptomatic stenosis. The reasons for this difference in prognosis are not fully understood, but may reflect differences in carotid plaque pathology.

Methods

In consecutive patients undergoing carotid endarterectomy for recently symptomatic stenosis (n=1640), we compared carotid plaque histology (using validated semi-quantitative scales) in those who had cerebral events within the last six months (n=1317) versus those with ocular events only (n=323).

Results

Compared with plaques from patients with ocular events only, those from patients with cerebral events had significantly more large lipid core (OR 1.38, 95%CI 1.05-1.82, p=0.02), inflammation (1.32, 1.02-1.72, p=0.04) and overall plaque instability (1.37, 1.05-1.80, p=0.02), and less fibrous content (0.71, 0.54-0.92, p=0.01) and calcification (0.70, 0.54-0.91, p=0.008). The overall number of histological features known to be associated with vulnerable plaque was greater in patients with cerebral events than in those with ocular events (p=0.002).

Conclusion

Carotid plaques from patients undergoing endarterectomy for recent ocular ischaemic events only have fewer vulnerable plaque features than those from patients with recent cerebral ischaemic events, possibly explaining some of the differences in risk of stroke between these groups.

ABSTRACTS

Session 4 - Thursday 1.30pm - 3.00pm

Is there a role for C-Reactive Protein, Myeloperoxidase or Beta-2 Microglobulin as a biomarker of carotid plaque instability?

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Objectives

To evaluate the roles of C-Reactive Protein (CRP), Myeloperoxidase (MPO) and Beta-2-Microglobulin (B2M) as potential biomarkers of carotid plaque instability.

Methods

Plasma samples from symptomatic (n=122) and asymptomatic (n=27) patients undergoing carotid endarterectomy were analysed for CRP, MPO and B2M levels using enzyme-linked immunosorbent assays (ELISAs). Plasma protein levels were then correlated with defined clinical, Duplex ultrasound and validated histological criteria of plaque stability using non-parametric statistical tests.

Results

Plasma CRP levels were significantly elevated in symptomatic patients (median=2.80mg/L) compared with asymptomatic patients (median=1.53mg/L, P=0.02). Plasma CRP levels were not, however, associated with any specific histological or imaging features of plaque instability and did not correlate with recent symptoms in symptomatic patients. Plasma MPO levels were not related to any clinical, imaging or histological criteria of plaque instability. Plasma B2M was significantly elevated in asymptomatic patients (median=8.21mg/L) compared with symptomatic patients (median=5.60mg/L, P=0.002), and there was no association between B2M and the recency of symptoms. Plasma B2M levels correlated significantly with histological evidence of intraplaque haemorrhage (P=0.013).

Conclusion

Plasma biomarkers offer the potential to influence management, but two of the three biomarkers tested in this project failed to demonstrate significant correlation with established histological, clinical or imaging criteria of unstable plaques, especially in symptomatic patients. However, the novel finding that elevated plasma B2M was increased in the presence of intraplaque haemorrhage suggests that it could represent another method for identifying 'high risk for stroke' asymptomatic patients in the future. This finding requires validation in an independent cohort.

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ABSTRACTS

Session 5 - Friday 8.00am - 9.00am

The shortfall in long-term survival of patients with repaired thoracic or abdominal aortic aneurysms

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Objectives

Previous data have shown long-term survival after aneurysm repair to be poorer than survival in comparable individuals without an aneurysm, largely due to excess cardiovascular mortality. This discrepancy may have been due to suboptimal management of atherosclerotic risk, and it is not known whether the shortfall persists in contemporary practice. This population study aimed to quantify the current disparity in life expectancy between survivors of aneurysm repair and matched controls.

Methods

Patients undergoing repair of abdominal (AAA) or thoracic aortic aneurysm (TAA) from April 2006 to March 2011 were identified from UK Hospital Episodes Statistics. Control subjects were identified who underwent elective inguinal hernia repair, total knee, or total hip arthroplasty with the same date of operation, hospital, age, gender, date of birth, and social deprivation as each AAA/TAA. The primary outcomes were all-cause mortality and adverse cardiovascular events (myocardial infarction, stroke, emergency amputation or limb revascularisation), reported with Kaplan-Meier analysis and compared by log-rank test.

Results

26,976 AAA and 1,404 TAA repairs were identified, with 102,174 and 5,458 controls respectively. Five-year survival was 61% for AAA vs 77% for controls, and 58% for TAA vs 83% for controls ($p<0.001$). Freedom from adverse cardiovascular events was 67% for AAA vs 83% for controls and 75% for TAA vs 87% for controls ($p<0.001$).

Conclusion

Long-term survival after aneurysm repair remains poor and adverse cardiovascular events are common, relative to comparable patients without an aneurysm. A randomised trial of intensive, goal-directed cardiovascular therapy is urgently required to bridge this deficit.

ABSTRACTS

Session 5 - Friday 8.00am - 9.00am

A comparative study of abdominal aortic aneurysm (AAA) in men and women

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Objectives

Despite lower incidence of AAA in women compared to men, studies have shown inconsistencies in comparison of risk factors and AAA growth rate between the groups. Our aim was to compare AAA risk factors and growth rate between the two genders.

Methods

In a prospective study, subjects with AAA (confirmed with US/CT, aorta >3cm) were recruited. All-cause mortality was recorded. Mixed - effects linear regression analysis was used to calculate AAA growth. Kaplan-Meier and Cox regression analysis was used to analyse survival.

Results

There were a total of 471 men and 99 women with AAA. There was no difference in age, BMI, blood pressure, diabetes and family history of AAA between the groups. Women had higher smoking pack-year, higher cholesterol and lower HDL ($p<0.05$). The overall incidence of cardiovascular-disease was similar (56% vs. 66% in women and men, $p=0.067$) but there were higher CABG in men (15% vs. 4%, $p=0.004$). There was no difference in aspirin and statin usage.

There was no difference in AAA growth between men and women (1.8 ± 0.1 vs. 1.9 ± 0.3mm/year respectively, $p=0.726$).

Long term survival was compared between 252 men and 69 women who did not have AAA repair. Women had significantly lower survival compared to men (4.5(3.8-5.2) vs. 6.1(5.6-6.5) years, Logrank=0.012). After adjustment for confounding variables, women's survival was still significantly lower than men's (HR 1.98, 95%CI (1.17-3.34, $p=0.01$).

Conclusion

Contrary to most studies, we did not show any difference in AAA growth between men and women. Women compared to men had significantly lower survival (higher all-cause mortality) even after adjusting for confounding variables.

ABSTRACTS

Session 5 - Friday 8.00am - 9.00am

Crural vessel assessment in critical limb ischaemia: Dependent Doppler versus Magnetic Resonance Angiography

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Objectives

Femoro-distal graft patency is known to correlate better with Doppler ultrasound than with digital subtraction angiography. Magnetic Resonance Angiography (MRA) has, in many centres, replaced digital subtraction angiography as the principle modality used to assess peripheral arterial status. This study compares crural vessel patency in patients with critical limb ischaemia using both Dependent Doppler (DD) and MRA.

Methods

Fifty two patients with unilateral critical ischaemia were identified who had been assessed by both MRA and Dependent Doppler (DD). The number of vessels identified, the dominant vessel feeding the foot arch and surgical outcome data were analysed.

Results

Dependent Doppler identified 128 patent crural vessels at the ankle compared to 84 vessels identified with MRA (mean 2.5 to 1.6 : P=0.0001). In 42 (81%) patients, DD identified a crural vessel feeding the foot arch compared to only 14 (27%) patients using MRA (P=0.0001). There was low overall agreement between the two modalities as to crural vessel patency (kappa=0.115). Twenty two patients went on to have reconstructive surgery with a median follow up of 12 (1-96) months and a graft patency rate of 77% and a limb salvage rate of 91%.

Conclusion

A greater number of patent crural vessels were identified by Dependent Doppler than by MRA. We recommend that all patients being considered for distal revascularisation be assessed with Dependent Doppler and that reconstructive surgery should not be denied on the basis of MRA alone.

ABSTRACTS

Session 5 - Friday 8.00am - 9.00am

The value of surveillance of Arterio-Venous-Fistulas that are not in use at 24 weeks following formation

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Objectives

It is accepted practice to create an Arterio-Venous-Fistula (AVF) in advance of the anticipated date of haemodialysis. However the deterioration of renal function is not always progressive or predictable. Unfortunately such AVFs do thrombose, even before they are ever used.

There is no evidence based guidance for surveillance of predialysis AVFs, who may be at risk of thrombosis.

Early identification and appropriate intervention of AVFs, not used 24 weeks following creation, could preserve the patency, providing access for haemodialysis, when required.

This is a retrospective study of patients who had AVFs from January 2007 to December 2011, not in use for haemodialysis at 24 weeks. The AVFs underwent 6-monthly Doppler surveillance whilst not in use.

Methods

55 AVFs met the criteria - 20 brachiocephalic, 32 radiocephalic and 3 brachiobasilic transposition fistulas. 33 AVFs were used at 24 weeks or more following formation, average time to first dialysis was 38 weeks (range 24-79 weeks). Prior to starting dialysis, 10 patients in this group required intervention; 7 angioplasty, 1 transposition and 2 ligation.

Results

22 of the fistulas were still not in use at time of study (40.0%), average time elapsed since creation 116.7 weeks (range 29-214 weeks). 15 of these AVFs required intervention; 11 angioplasty, 2 ligation and 1 patch. One fistula in this group predictably failed to mature and alternative access was considered.

The primary and primary assisted patency rates were 55% & 98%.

Conclusion

A significant number of predialysis AVF can be salvaged with surveillance and appropriate intervention.

ABSTRACTS

Session 5 - Friday 8.00am - 9.00am

Interim results on abolishing reflux from a randomised controlled trial on laser ablation with phlebectomies versus foam sclerotherapy

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Objectives

The early results of a randomised clinical trial comparing local anaesthesia Endovenous Laser Ablation (EVLA) with concurrent phlebectomies versus Ultrasound-guided Foam Sclerotherapy (UGFS) into the Great Saphenous Vein (GSV) revealed that laser was more expensive but the results on abolition of reflux were similar. The interim results at 15 months follow-up are reported.

Methods

Evaluations included ultrasound, the venous clinical severity score (VCSS), the Aberdeen varicose vein questionnaire (AVVQ) and the saphenous treatment score (STS). The global absence of reflux defined technical success. Adjuvant sclerotherapy to areas of reflux was administered on patient choice.

Results

Occlusion of the GSV was more effective with EVLA at 42/44(95.5%) versus 31/46(67.4%) for UGFS ($P=0.0008$). However, both techniques were equally effective at abolishing global venous reflux. The number of legs ($n=100$) with total reflux abolition, above-knee, below-knee or combined reflux and loss to follow-up was 18(41%),6,12,8,6 with EVLA and 20(43%),8,11,7,4 with UGFS, respectively. The VCSS, AVVQ and STS reduced compared to baseline ($P<0.0005$), but there was no statistical difference between the groups. The AVVQ remained unchanged between 3-15 months ($P=0.601$). Also during this time, 19/46(41%) UGFS versus 9/44(20%) EVLA legs received adjuvant treatment (2.1 times increase). However, overall, adjuvant foam was given 4.7 times more frequently in the UGFS patients.

Conclusion

EVLA and UGFS are equally effective at abolishing global venous reflux with overall success of 41% and 43%, respectively. The high reflux rate was not related to deterioration in quality of life indicating that this reflux was largely asymptomatic.

ABSTRACTS

Session 5 - Friday 8.00am - 9.00am

Does pre-operative dual antiplatelet therapy reduce microembolisation rates after a carotid endarterectomy?

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Objectives

Postoperative thromboembolic stroke following carotid endarterectomy (CEA) is often preceded by higher microembolic signals (MES) rates. This study aimed to evaluate whether dual anti-platelet therapy (clopidogrel and aspirin) reduces MES rates without increasing the risk of bleeding following CEA.

Methods

82 consecutive patients undergoing CEA from January 2008 to December 2011 were separated into Group 1 (aspirin or clopidogrel) and Group 2 (aspirin and clopidogrel). The ipsilateral middle cerebral artery was insonated using TCD, intra- and post-operatively. The MES rates were assessed for the initial 30 minutes in the post-operative period.

Results

Of the 82 patients, TCD monitoring was unavailable for 10 patients and another 7 patients did not have a suitable temporal window and therefore were not included. In Group 1 (n=33), MES were detected in 17 (52%) patients, but were only significant (>60 signals/hour) in 3 (9%) patients. One patient from Group 1 died from contra-lateral haemorrhagic stroke 11 days after CEA, giving a stroke and death rate of 1.2%. In Group 2 (n=32), MES were detected in 6 (19%) patients but none were significant. No patient returned to theatre for bleeding. A Fisher's exact two-tailed test was used showing a statistically significant reduction in MES in the first 30 minutes post carotid endarterectomy (p=0.009) in Group 2.

Conclusion

Dual antiplatelet therapy did not increase the risk of post-operative bleeding, but did significantly reduce post-operative MES rates.

ABSTRACTS

Session 6 - Friday 9.00am - 10.30am

High-density lipoprotein cholesterol and abdominal aortic aneurysm - a Mendelian randomisation study

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Objectives

Lower circulating high-density lipoprotein cholesterol (HDL-C) has been associated with increased risk of AAA, but it is not clear if this is a causal relationship. Genotypes are randomly assigned at conception and can be used as instruments to investigate causality.

Methods

Initially, a systematic review and meta-analysis of the published literature was performed to define the association between HDL-C and AAA. Two mendelian randomization analyses were then carried out. First, a genetic risk score composed of 35 single nucleotide polymorphisms (SNPs) associated with higher HDL-C levels was tested for association with AAA. Second, a SNP in the promoter of CETP, with a range of effects similar to CETP-inhibition was tested for an association with AAA.

Results

Six studies (1,056 cases, 21,450 controls) reported an odds ratio for AAA per unit change in HDL-C concentration, adjusted for other traits. A one SD increase in HDL-C was associated with lower risk of AAA (OR 0.69, 95% CI 0.63 - 0.76, I2=0%, P =4.1 x 10-16). In analysis of 2,367 cases and 44,437 controls from two studies, one SD increase in weighted genetic risk was associated with a lower risk of AAA (OR=0.89, 95%CI 0.86-0.93, P = 2.4x10-7). rs3764261 in CETP was also associated with a lower risk of AAA in meta-analysis of data from 5 cohorts (4,889 cases and 52,482 controls, OR 0.91, 95% CI 0.86 - 0.95, I2 = 21%, P=1.3x10-4). Instrumental variable regression showed consistent results from observational and genetic data.

Conclusion

This study suggests that HDL-C mediated pathways play a causal role in AAA pathogenesis. Trials of CETP inhibition in AAA may be warranted.

ABSTRACTS

Session 6 - Friday 9.00am - 10.30am

A 10-year prospective population-based study of the incidence and outcome of acute abdominal aortic aneurysms: implications for screening

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Objectives

Current screening programmes are confined to men aged 65 years. The prevalence of AAAs maybe declining in this age group and estimates of the screening effectiveness are based on studies that excluded older age groups. We aimed to determine event rates at older ages.

Methods

In the first prospective population-based study of all acute aortic events irrespective of age (92,478 population, Oxfordshire, UK, 2002-2012), we report age and sex-specific rates per 100,000 population per year.

Results

Of 169 acute aortic events in 150 patients, 113 (66.9%) were acute aneurysms, of which 102 (90.3%) were acute abdominal aortic aneurysms (aAAAs) (incidence = 11/100,000; 95%CI 9-13, male 69%, mean age 78.3yrs). Of these, 77 (75.5%) were ruptured/leaking (RAAA) (incidence= 8/100,000; 6-10) and 25 (24.5%) were symptomatic (SAAA; 3/100,000; 2-4). The incidence of aAAAs increased steeply with age, with rates in men >75-years (184/100,000) being far greater than in those aged 65-74 (55/100,000). Rates in women >75-years (59/100,000) were similar to men aged 65-74yrs. aAAA 30-day fatality was 54.9% (38.7% (29/75) for patients reaching hospital); 68.8% (53/77) for RAAAs; 12.0% (3/25) for SAAAs. Only 22 (21.6%) of aAAA events and 6 (10.7%) of all related deaths occurred in men aged 65-74. Pre-morbid hypertension was diagnosed in 96.8% (30/31) of females versus 59.2% (42/71) of males ($p<0.001$).

Conclusion

Our findings raise concerns regarding current screening programmes. Screening women with hypertension at age >75 years could identify >70% of women who would have aAAA events (24.5% of the total aAAA event burden). As the majority of events and related deaths occurred in men aged >75 screening should target older men (>70 years) to improve cost-effectiveness.

ABSTRACTS

Session 6 - Friday 9.00am - 10.30am

Metabolic profiling of human atherosclerosis tissue reveals mechanisms of atherosclerosis progression and differences between carotid and femoral plaques

J Shalhoub[1] PA Vorkas[2] G Isaac[3] EJ Want[2] S McDonald[3] J Langridge[3] A Millar[3] JP Shockcor[3] JK Nicholson[2] AH Davies[1]

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Objectives

There is a clinical need to develop understanding of triggers underlying immune activation underlying atherosclerosis progression and biological differences between atherosclerosis occurring at anatomically disparate sites within the arterial tree.

Methods

75 consenting patients undergoing endarterectomy surgery had 89 tissue samples collected: carotid plaque (CP) n=48; carotid intimal thickening (IT) n=6; femoral plaque (FP) n=27; femoral IT n=14. Samples had consecutive metabolite extractions to yield polar aqueous (AE) and organic (OE) extracts. Ultra-performance liquid chromatography mass spectrometry (UPLC-MS) and metabolite features were extracted from UPLC-MS data using the MarkerLynx package. Multivariate statistical analyses were performed using SIMCA-P+ 12.

Results

There was no difference in metabolite features between IT from different anatomical locations, so were handled as a single group for subsequent analyses. Principal Component Analysis (PCA) and Orthogonal Projections to Latent Structures (OPLS) multivariate statistics of OE and AE was able to discriminate between the three groups (IT, CP and FP). Free cholesterol was elevated in both CP and FP compared to IT. Several lipid classes, including sphingomyelins (SM), phosphocholines (PC) and phosphoethanolamines (PE) appeared reduced in CP and FP, compared to IT. d18:2 sphingomyelins were elevated in CP compared with FP, with SM(d18:2/24:1) and SM(d18:2/16:0) being the most discriminant. Global elevation of triacylglycerides (TG) was observed in the FP compared with CP, particularly TG(16:0/18:1/18:1), TG(16:0/18:2/18:1) and TG(16:1/18:1/18:1).

Conclusion

Metabolic profiling has generated disease biomarker ‘signatures’ and can contribute to elucidation of disease mechanisms underlining atherosclerosis at different sites, and those responsible for or associated with disease progression.

ABSTRACTS

Session 6 - Friday 9.00am - 10.30am

Endarterectomy of the common femoral bifurcation: a decade of experience from two UK centres in the endovascular era

M Desai[1] Z Ahmed[2] K Hussey[2] W Stuart[2] G Hamilton[1]

[1]Department of Vascular Surgery, Royal Free London NHS Foundation Trust, London [2]Department of Vascular Surgery, Western Infirmary, Glasgow

Objectives

To analyse long-term outcomes following femoral endarterectomy (FE) and profundaplasty from two large vascular units in the current endovascular era.

Methods

Between 1999 and 2007, data of all patients undergoing FE at two centres (London and Glasgow) were retrospectively analysed. Patients were stratified based on Fontaine classification and endpoints of thirty-day mortality and morbidity; re-intervention and amputation in the ipsilateral limb, overall survival and amputation free survival were evaluated using Kaplan Meier plots. In addition factors associated with re-intervention and amputation were assessed with logistic regression analysis.

Results

FE was performed in 158 cases (mean age 71 SD9.4; male 84%) and combined with profundaplasty in 60% of cases. 87(55%) were performed for incapacitating claudication and 71(45%) for critical limb ischaemia. All hybrid procedures requiring further distal outflow correction were excluded. Mean follow-up was 44 months (range 1-144 months). There were 7(4%) major and 15(9%) minor complications with 2(1%) perioperative deaths. Freedom from the need to undertake further revascularisation was 91% at 5 years. Limb salvage at 5 years in the critical limb population was 75% at 5 years. Overall amputation free survival at 5 years of the cohort was 78%. Predictors of the need for revascularisation included smoking status and age. The only predictor for the need for amputation was Fontaine classification 4.

Conclusion

Our present series encompasses a wide UK based patient demographic. It confirms safety, low morbidity and excellent long-term durability of femoral endarterectomy and profundaplasty with low re-intervention and high limb salvage rates.

ABSTRACTS

Session 6 - Friday 9.00am - 10.30am

A UK perspective on post-operative mobility in patients undergoing lower limb bypass surgery (LLBS) for critical limb ischaemia (CLI)

N Al-Zuhir J Boyle K Varty P Hayes PA Coughlin

Vascular Surgical Unit, Addenbrooke's Hospital, Cambridge

Objectives

Evidence, largely from the USA, suggests that LLBS does not improve mobility in patients with CLI. This is an important outcome measure for patients. We determined mobility rates at discharge in patients undergoing LLBS.

Methods

All LLBS procedures performed for CLI (Jan 2009 - Dec 2010) were analysed. Mode of presentation, co-morbidity, procedure, length of stay (LOS) and ambulatory status on discharge was determined. Risk scoring measures of outcome (FINNASC and PREVENT III) and cardiac risk (Revised Cardiac Risk Index - RCRI) were calculated.

Results

Sixty-three patients were analysed (45 men, median age 70 yrs). Thirty two were admitted urgently and 32 had tissue loss. Median scores for the FINNVASC, PREVENT III and RCRI were 1, 3 and 0 respectively. Forty-seven patients received an autologous conduit, 5 composite and 11 prosthetic. Two patients underwent a major amputation on the same admission. There were no in-hospital deaths. Overall median LOS was 10 days (IQR: 6-16 days). Adverse risk scoring (FINNVASC) resulted in a longer median LOS (7.5 vs. 12 days; $p<0.01$) but did not predict mobility. Twenty seven patients required mobility aid on discharge (stick 4, frame 9 and wheelchair 14). Older patients and patients with tissue loss were more likely to requiring some form of walking aid ($p<0.01$). Patients requiring a walking aid had a longer LOS (7.5 vs. 14 days; $p<0.01$).

Conclusion

A significant proportion of patients require aid for mobility LLBS. In depth assessment of corrective factors that predict poor mobility need to be identified to improve patient outcome and potentially reduce LOS.

ABSTRACTS

Session 6 - Friday 9.00am - 10.30am

Microemboli present in the right heart during thermoablation of varicose veins

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Objectives

Cerebrovascular events have been noted after foam sclerotherapy for varicose veins. One hypothesis is migration of microemboli to the brain through a cardiac septal defect. The aim of this study was to identify whether microemboli are found in the right side of the heart during endothermal ablation of varicose veins, as neurological events are not reported during these procedures.

Methods

Transthoracic echocardiography was performed during local anaesthetic radiofrequency ablation (VNUS - Closure Fast) of the great saphenous vein in 14 patients. An apical view was captured at the start of the procedure, during each cycle of heating and at one minute post-treatment. Patients were monitored for one hour.

Video loops were read by an independent cardiologist and the sonographer. The presence of microemboli was classified as: 0=absent, 1=occasional microemboli, 2=stream of microemboli, 3=complete opacification.

Results

Loops were of diagnostic quality in 11/14 (79%) patients. After the second cycle of heating, microemboli moving through the right heart were seen in 5/11 (45%) patients. These were classified as grade 1 in 4 patients and grade 2 in 1 patient. No microemboli were seen in the left heart. Inter-reader κ was 0.5 (95% CI 0.2-0.79). No neurological symptoms were reported.

Conclusion

Microemboli in the right heart are a common finding during radiofrequency ablation of varicose veins. Considering the prevalence of cardiac septal defects (26%), more neurological events would be expected if these particles were responsible for these events. Further work is required to elicit the mechanisms underlying neurological complications following sclerotherapy.

ABSTRACTS

Session 6 - Friday 9.00am - 10.30am

Have we reduced the prevalence of leg ulcers? A repeat audit after 20 years in an urban health district

A Sala Tenna[1] H Stevens[2] T Lees[1]

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Objectives

To study the prevalence and management of leg ulcers in the Newcastle Health District 20 years after a previous study, to assess whether leg ulcer care has improved healing.

Methods

A citywide audit using a questionnaire based on that used in 1992 was sent to each District and Practice Nurse at 37 General Practices and nursing homes with an active caseload in March 2012.

Results

(1992 data in brackets).

In a population of 101900 > 45yrs of age, 211 (206) lower limb ulcers were identified. The incidence was 4.0 - 6.1 (3.5) per 1000. The prevalence was 2.1 (1.9) per 1000. 86% of these were cared for either at home or via the GP practice. 25% (30%) of ulcers were present for < 3 months, 19% (20%) for 3 - 6 months, 15% (15%) for 6 - 12 months, 22% (26%) for 1 - 5 years and 5% (9%) > 5 years. Recurrent ulcers represented 48% (47%) of the total with 42% (50%) of ulcers being present for > 6 months. 90% of ulcers were deemed to have a vascular aetiology. Only 55% (35%) were referred to a hospital specialist and 38% (7%) were referred to a vascular surgeon. 35 (35) different types of dressings were identified in the treatment of leg ulcers. Compression bandaging was deemed the best treatment with 76% (14%) of patients being treated in this way.

Conclusion

Despite increased referral to vascular specialists, improved compression rates and established community leg ulcer care, the prevalence, incidence and chronicity of leg ulceration essentially remains unchanged over 20 years.

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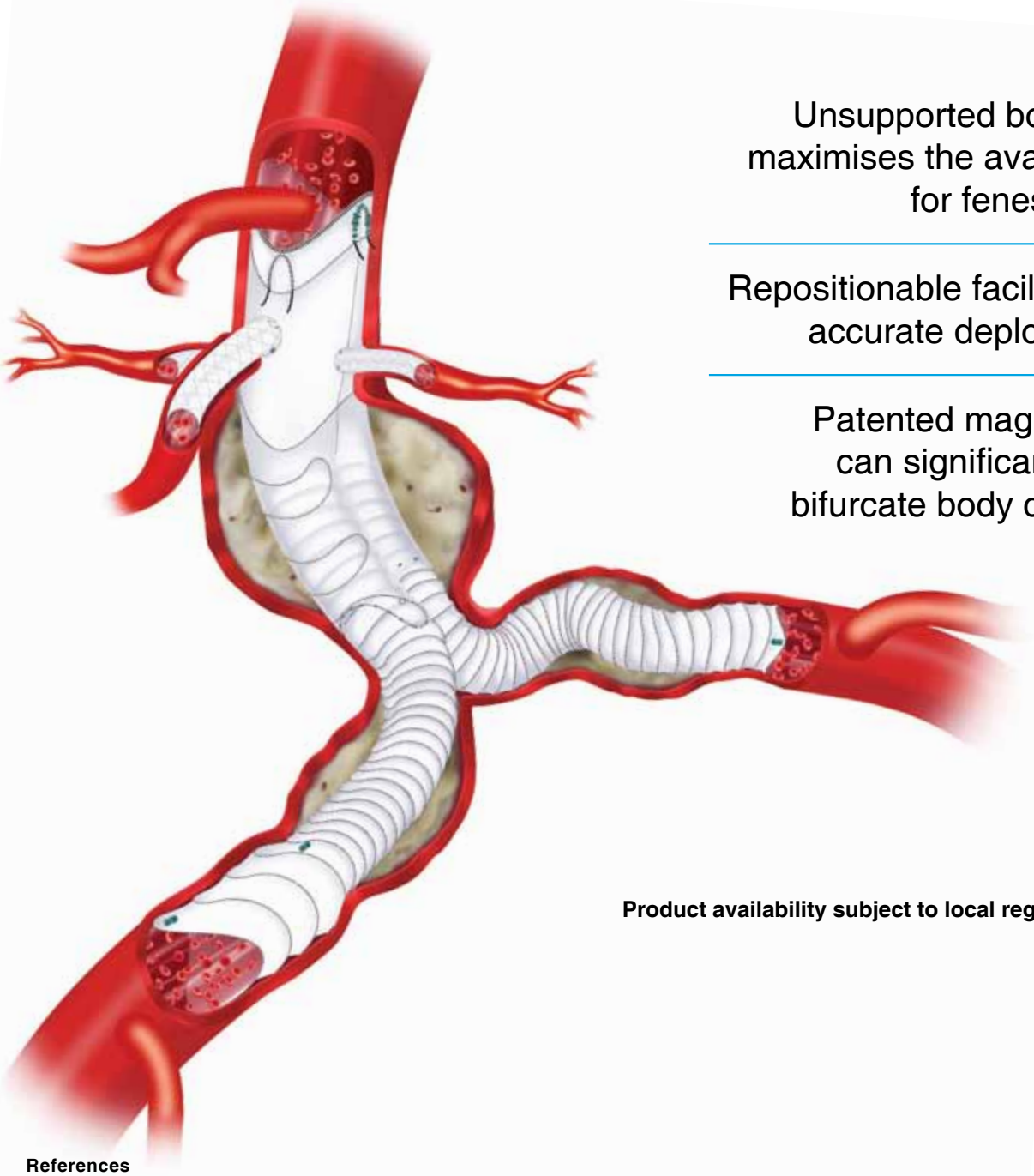

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ABSTRACTS

Session 6 - Friday 9.00am - 10.30am

Lipid management after carotid endarterectomy: comparing the effectiveness of primary and secondary care

R Durairajan[1] A Sivaramakrishnan[1] K Lund[1] A Kundu[1] T Kalra[1] T Loganathan[1] D Sinha[1] P Guyler[1] L Coward[1] A O'Brien[1] JRI Brown[2] MS Jakeways[2] S Patel[2]

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Objectives

Patients undergoing carotid surgery following ischaemic stroke need risk factor modification to optimise secondary prevention. We aimed to analyse the difference between primary and secondary care in lipid monitoring and achieving target levels.

Methods

All patients who underwent carotid endarterectomy (CEA) between August 2009 and July 2011 were included. Follow-up was one year.

109 patients underwent CEA of which 91 (83.5%) were symptomatic. 74 (67.8%) were male. 33 (52.3%) were current or ex-smokers. 100 (95.2%) were discharged on statins. 16 (15.2%) had their statin changed due to intolerance. 25 (22.9%) had type 2 diabetes and 35 (32.1%) had IHD. 7 (6.4%) had PVD. 47 (43.1%) had no contralateral carotid artery stenosis.

Results

104 (95.4%) were on lipid-lowering therapy at the time of surgery. 97 (89%) had fasting lipid profile prior to CEA.

77 patients (71%) had their lipid profile checked in primary care following surgery. Of these only 43 patients (55.8%) achieved target levels in the 3-6 month timeframe.

One patient had myocardial infarction, two had TIAs and three had strokes during follow-up.

Conclusion

There is a significant difference between primary and secondary care in monitoring and achievement of lipid control. From this study we conclude that patients are not aggressively followed-up in primary care. It is important to achieve target levels in this high risk population for optimal secondary prevention. There is an ongoing discussion between primary and secondary care to improve this in line with local and national guidelines.

ABSTRACTS

Session 6 - Friday 9.00am - 10.30am

Safety of carotid endarterectomy following thrombolysis for acute ischaemic stroke: single centre experience and systematic review

Y Yong[1] J Saunders[1] S Abisi[1] N Sprigg[2] ST MacSweeney[1] N Altaf[1]

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Objectives

Carotid endarterectomy (CEA) following thrombolysis for acute ischaemic stroke constitutes a subgroup whereby the timing and benefit of surgery remain controversial. The aim of this study was to determine safety of CEA post thrombolysis in our unit and to perform a systematic review.

Methods

A retrospective analysis of prospectively collected data of patients who underwent CEA following thrombolysis from 2010 to 2012 was performed. The primary outcome measure was post-operative stroke and death. A systematic literature review was performed using Pubmed, Embase, Ovid and Cochrane database.

Results

Of the 213 patients who underwent intravenous thrombolysis for stroke, 7 patients (4 men, mean age 69 years +/- 5) underwent CEA post thrombolysis. CEA was performed at a median of 10 days (IQR 3-12 days) from symptom onset. There was 1 patient who experienced haemorrhagic transformation of ischaemic stroke post-operatively. There were no deaths.

Seven studies with 42 patients were included in the pooled data synthesis. Timing of CEA from symptom onset ranged from 6 hours to 54 days. The pooled 30-day stroke and death rate was 4.2% (2 strokes) and 0% respectively.

Conclusion

CEA appears safe following thrombolysis for acute ischaemic stroke, with stroke rates comparable to randomised controlled trials.

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MAQUET
Major Sponsor
Stand number: 51

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MEDICAL DEFENCE UNION
Stand number: 19

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www.the-mdu.com

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EXHIBITORS

The Vascular Society AGM, Manchester: 28 - 30 November 2012

MEDI UK LTD
Stand number: 25

Plough Lane, Hereford, Herefordshire, HR4 0EL
Contact: medi Customer Services
T. 01432 373500
E. enquiries@mediuk.co.uk
www.mediuk.co.uk

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Building 9, Croxley Green Business Centre,
Watford WD18 8WW
T. 01923 212213 F. 01923 241004
www.medtronic.co.uk

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E: sales@evrf.co.uk
www.evrf.co.uk

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M3 MEDICAL LTD
Stand number: 2

Unit 3, Leopardstown Office Park, Sandyford, Dublin 18
T: +353-(0)1-2885000

NHS AAA SCREENING PROGRAMME/ NORTHGATE INFORMATION SOLUTIONS
Stand number: 31

NHS Abdominal Aortic Aneurysm Screening Programme
Victoria Warehouse, The Docks, Gloucester GL1 2EL
T. 01452 318844 F. 01452 318837
http://aaa.screening.nhs.uk

The NHS AAA Screening Programme aims to reduce aneurysm-related deaths through early detection, monitoring and treatment. The Programme now covers approximately 80% of England and full implementation should be complete by March 2013. The Programme's national roll-out has been a significant driver of the reconfiguration and improvement of

EXHIBITORS

The Vascular Society AGM, Manchester: 28 - 30 November 2012

vascular services in the UK.

During the Programme's first three years (April 2009 to March 2012), more than 165,000 men were screened, more than 2,600 aneurysms detected and more than 300 aneurysms successfully repaired. The national SMaRT IT solution, developed with Northgate Information Solutions, helps ensure men are tracked from screening invitation to clinical outcome.

NUROS LTD
Stand number: 38

6 Abbey Lane Court, Evesham,
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NATIONAL VASCULAR DATABASE
Stand number: 30A

35-43 Lincoln's Inn Fields, London. WC2A 3PE
T: 0207 973 0306
E: office@vascularsociety.org.uk
www.vascularsociety.org.uk

PERIMED UK LTD
Stand number: 29

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For further information see our conference bag insert, visit the Perimed stand in the exhibition hall or www.perimed-instruments.com

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The Vascular Society AGM, Manchester: 28 - 30 November 2012

PHILIPS HEALTHCARE
Stand number: 30

The Philips Centre, Guildford Business Park,
Guildford, Surrey GU2 8XH
T. 01483 792004 F. 01483 298831
E. katie.odriscoll@philips.com
www.medical.philips.com/uk

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PIERSON SURGICAL LTD.
Stand number: 41

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Stand number: 35

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E. kathy@promedltd.com
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Plough Lane, Hereford, HR4 0EL

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E. enquiries@stdpharm.co.uk
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Stand number: 21

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West Sussex. RH10 9AX
T. 01293 653700 F. 01293 653770
E. tmsuksalessupport@tmse.nl
www.toshiba-medical.co.uk

Toshiba Medical Systems (TMS) is a leading worldwide provider of medical diagnostic imaging systems, such as CT, X-Ray and vascular, ultrasound and MRI. Toshiba is a leading supplier to the NHS and independent healthcare sector. An independent UK study of customers with responsibility for clinical services rated Toshiba's imaging products for reliability, ease of use and image quality at over 90%. Furthermore, 94% were either satisfied or very satisfied with the people at Toshiba.

TRIVASCULAR
Stand number: 48 and 49

Lake Geneva Business Park,
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www.trivascular.com

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VASCULAR FLOW TECHNOLOGIES LTD
Stand number: 58

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www.vascular-flow.com

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VASCUTEK
Major Sponsor
Stand number: 53

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www.vascutek.com

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Stand number: 36

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www.yorkmedicaltechnologies.com

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THE COUNCIL

Front row, left to right:

Ms J Oliver; Mr D C Mitchell; Professor D J A Scott; Professor A R Naylor; Mr M G Wyatt; Professor J D Beard; Mr S D Parvin

Second row, left to right

Ms K Tinkler (SVT); Professor A Halliday; Mr I Franklin; Mr T Lees; Mr J Brennan; Mr J Thompson; Mr I Nyamekye; Professor S Homer-Vanniasinkam; Professor C Shearman (Chair, Vascular SAC); Ms E Bond (SVN)

Back row, left to right

Professor D Ettles (BSIR): Mr F Oshin; Professor M Thompson; Professor R Sayers; Mr I Loftus; Mr L Wijesinghe

OFFICE BEARERS AND MEMBERS OF COUNCIL 2011 - 2012

President	Professor A R Naylor
President Elect	Professor D J A Scott
Vice-President Elect	Professor J D Beard
Honorary Secretary	Mr M G Wyatt
Honorary Treasurer	Mr S D Parvin
Honorary Treasurer Elect	Mr T Lees
Ordinary members	Mr J Brennan Mr I Franklin Professor A Halliday Mr I Loftus Mr I Nyamekye Professor R D Sayers Mr J Thompson Professor M Thompson Mr L Wijesinghe
Education Committee Chair	Professor J D Beard
Education Committee Chair Elect	Mr J Brennan
Audit and Quality Improvement Committee Chair	Mr D C Mitchell
Research Committee Chair	Professor S Homer-Vanniasinkam
Research Committee Chair Elect	Professor R D Sayers
Circulation Foundation Committee Chair	Mr I J Franklin
Affiliate member	Mr F Oshin
Vascular Tutor	Professor I Chetter
Observers	Professor D Ettles (BSIR) Ms K Tinkler (SVT) Ms E Bond (SVN)

VASCULAR SOCIETY COMMITTEES 2011-2012

The Vascular Society AGM, Manchester: 28 - 30 November 2012

AUDIT AND QUALITY IMPROVEMENT COMMITTEE

Mr D C Mitchell (Chair)
Mr I Loftus
Mr S D Parvin
Mrs S Baker
Ms H Hindley
Mr J V Smyth (Co-opted)
Dr A Pichel (VASGBI)
Professor D J A Scott
Mr P Barker
Ms R Potgieter
Mr J J Earnshaw
Dr D Thomas (BSIR)
Dr C Snowden (VASGBI)

EDUCATION COMMITTEE

Professor J D Beard (Chair)
Professor I Chetter
Mr L Wijesinghe
Dr S Chakraverty (BSIR rep)
Mr J Brennan
Mr J Thompson
Mr I Nyamekye
Mr F Oshin (Affiliate rep)

RESEARCH COMMITTEE

Professor S Homer-Vanniasinkam (Chair)
Mr T Lees
Professor J Brittenden
Professor A Smith
Professor R Sayers
Professor A Halliday

PROFESSIONAL STANDARDS COMMITTEE

Professor M J Gough (Chair)
Mr D C Mitchell (as Chair of the Audit Committee)
Mr P M Lamont
Professor D J A Scott
Professor A R Naylor
Mr P Blair
Mr J J Earnshaw

CIRCULATION FOUNDATION COMMITTEE

Mr I Franklin
Mr S D Parvin
Professor G Stansby
Mr J McCaslin
Ms K Tinkler
Professor S Homer-Vanniasinkam
Mr T Lees
Mr M Baroni
Ms L Allen

ANNUAL GENERAL BUSINESS MEETING AGENDA

Thursday 29th November 2012 at 5.30-6.30pm, Manchester Central Convention Centre

1	Apologies
2	Minutes of AGM 2011
3	Any other business
4	**President's Report:** Professor Ross Naylor
5	**Honorary Secretary's Report:** Mr Mike Wyatt
6	**Honorary Treasurer's Report:** Mr Simon Parvin
7	**Audit and Quality Improvement Committee Report:** Mr David Mitchell
8	**Education Committee Report:** Professor Jonathan Beard
9	**Research Committee Report:** Professor Shervanthi Homer-Vanniasinkam
10	**Circulation Foundation Committee:** Mr Ian Franklin
11	**Professional Standards Committee Report:** Professor Michael Gough
12	**Vascular Tutor:** Professor Ian Chetter
13	**President Elect's Report:** Professor Julian Scott
14	**Election of Officers:** Result of ballot for Ordinary Members of Council
15	**Date of next meeting:** Thursday 28th November, Manchester Central Convention Centre

HONORARY SECRETARY'S REPORT

Mike Wyatt

This has been a monumental year in the history of the Vascular Society and has seen several major changes to the way in which we deliver care and service to patients with Vascular Disease.

At last year's AGM in Edinburgh, your Council was given a mandate to proceed with the specialty status application and to publish the "Provision of Services to Patients with Vascular Disease 2012", to assist clinicians and managers in the reconfiguration of vascular services for the arrival of the new vascular specialty.

The New Vascular Specialty

After much work by numerous Presidents and Officers of the Society, specialty status was granted on March 16th 2012. This essentially means that, from 2013, vascular trainees will be appointed at national selection to be trained as vascular surgeons and will gain a CCT in vascular surgery (not general surgery). Initially the numbers will be 20 per year, but this can rise in the future if the service requires.

We have been working closely with both the Royal College of Radiologists (RCR) and the British Society of Radiology (BSIR) to agree joint training programmes, which will equip these new trainees with the specific skills required to effectively practice vascular and endovascular surgery when they are eventually appointed to Consultant posts after CCT. A new vascular curriculum has been accepted by the GMC and is fully supported by each of the Royal Colleges, the RCR and the BSIR.

Recently it has become clear that old style trainees will continue with the current general surgery curriculum, and (as happens presently) will be accredited in general surgery with an interest in vascular surgery at the end of their training. This will not in any way disadvantage them in the job market as they will be "through the system" before the new trainees are finished.

To accompany the new training programme, a new Vascular FRCS (vFRCS) has been created and was approved by the GMC in October 2012 along with some minor changes to the submitted curriculum. This will be piloted and ready for the new 2013 vascular trainees when they complete their programmes.

I would like to extend a big thanks to Jonathan Beard and Julian Scott who have led on the Curriculum and the vFRCS respectively and to all of those individuals who have assisted with these enormous tasks.

a new Vascular FRCS (vFRCS) has been created and was approved by the GMC in October 2012

Provision of Vascular Services 2012

Your Council has been working hard on the production of the "Provision of Services to Patients with Vascular Disease 2012" document, which was published in February 2012. This document was approved by the AGM last year and provides guidance based on best medical practice to inform the various regional reviews of service that are occurring across the country. We know that with national commissioning of vascular services, there will be far fewer hospitals providing service on the future and are delighted that the Vascular Clinical Reference Group, chaired by Matt Thompson has used this document as a template for providing service requirement recommendations to the National Commissioning Board.

The Society is very aware that not all of our members support rationalization of service and throughout this last year we have been asked to provide named experts to help with many of these reviews. We know that change is always difficult but we are clear in our mission statement that "all patients with vascular disease

should have 24/7 access to a specialist vascular team in all parts of the country".

As the National Commissioning Board is formed from 2013 onwards, it will become clear if the new modern vascular networks have been accepted for the provision of vascular services to local populations within the United Kingdom of Great Britain and Northern Ireland. At the time of writing, this document does not apply to Ireland, where vascular surgery is still a sub-specialty of general surgery.

Executive and Council News

Each of your committees has been working extremely hard for the Society, and a record of their achievements is included in the individual Chairman's reports. Nevertheless, we are indebted to Jonathan Beard, David Mitchell, Shervanthi Homer-Vanniasinkam and Ian Franklin for steering the Society so expertly in their elected roles. We lose Jonathan as a Chair this year, but welcome him back as President Elect. John Brennan is the new Chair of the Education Committee and I'm sure will continue Jon's excellent work. We also lose Mike Gough as Chair of the Professional Standards Committee. Mike has been an excellent ambassador for the Society and will be much missed. His replacement is my predecessor Jonothan Earnshaw, who we welcome back to the Committee.

Our Treasurer, Simon Parvin is also leaving this year. Simon has been instrumental in guiding the Society through the difficult financial waters afforded by the recent double dip recession, and has ensured that the Society remains financially sound. We are most grateful for his expertise in managing our assets, our Council and our Executive. Simon has left us in good financial shape as we welcome his successor Tim Lees.

I am most grateful to Ross, Jeanette, Neelam and Rebecca, without whom this Society could not function. Ross has been a superb President and as he 'returns to the backbenches" (his words not mine) will be remembered for his sound leadership in establishing the new specialty. We welcome Julian Scott as our new President and are delighted that Paul Blair has been elected as President for 2014/15. The Society has strong leadership and I'm sure will continue to grow as we embed ourselves as a new Surgical Specialty in the UK.

Our CEO, Jeanette, Business Manager, Neelam, and Fundraising and Events Manager, Rebecca are all invaluable to Society. They have each worked tirelessly on your behalf to ensure that the Society continues to thrive and that the Executive and Council members have the resources required to enable them to work effectively. We are indebted to each of them and I offer my personal thanks to each, for without them, none of our achievements would be possible.

you will notice that we have rebranded the Vascular Society to mark the birth of our new Specialty

Your Council members are superb. They are involved in all of the decisions of the Society and are the unsung heroes of the achievements we have enjoyed this year. Three members step down at this AGM. Mr. John Brennan (continues as Chair of the Education Committee), Mr. Ian Franklin (continues as Chair of the Circulation Foundation) and Isaac Nyamekye. A big thank you to all of them and a big welcome to those of you who are elected to replace them at the 2012 AGM.

Finally, you will notice that we have rebranded the Vascular Society to mark the birth of our new Specialty. This is a new dawn, a new Specialty and a new Society. We hope you enjoy being part of this historic occasion and we look forward to a most exciting meeting, expertly prepared by your President, Ross Naylor.

We are back in Manchester again next year and until then, farewell.

HONORARY TREASURER'S REPORT

Simon Parvin

This is my last report as Treasurer. It has been a privilege to serve as the 13th Treasurer for 4 years since 2008

2012 has seen our reserves drop by approximately 25% as a consequence of a much reduced profit from the Annual Meeting in Edinburgh. Despite this, the finances of the Vascular Society remain in good shape, with reserves of approximately £150,000. We have already taken steps to reduce the cost of the annual meeting by committing to less expensive venues and by agreeing multiple visits to those venues over the coming years.

Membership

Our membership remains the same as in 2011, at approximately 740, with a slightly increased income from memberships as a result of the inevitable annual increase in membership fees. Once again the number of Affiliates has increased from 121 to 139, and this is promising for the future as a new specialty. We will need to work hard to maintain this number with the expected changes to the number of trainees required to maintain the separate specialty.

Events

As predicted, the AGM in Edinburgh, though scientifically successful, made a reduced profit. This was due to the expense of the venue and as a result of a reduction in income from the exhibition. We anticipate an improved position this and next year in Manchester.

During this year, the Spring Meeting in Belfast "Vascular Emergencies" made a small surplus, and the Marathon and Golf Day events raised income for the CF.

Future expenditure

Cost pressures for the Society are principally from the ever increasing cost of staging the annual meeting coupled with the gradually reducing income from registrations and the exhibition. Office costs are contained and barely changing due to the vigilance of Jeanette and Neelam.

The Circulation Foundation

During 2012, the Circulation Foundation has received further legacies of approximately £100,000, and its underlying income is slowly increasing. George Davies has agreed to invest £100,000 per year to the "George Davies Visionary Award", an extremely generous move. The CF has a well established ambitious programme of grant awards worth £200,000 per year, which it should be able to sustain going forward.

Major Sponsors

I would like to thank our Major Sponsors, Covidien, Cook, Maquet, Gore, and Vascutek for their ongoing support.

Personal note

This is my last report as Treasurer. It has been a privilege to serve as the 13th Treasurer for 4 years since 2008. It is a daunting task trying to ensure the financial stability of the organisation, and I would like to acknowledge the essential role of Jeanette Oliver. Without her breadth of knowledge, skills and commitment to detail it would not have been possible.

THE VASCULAR SOCIETY (A Company limited by guarantee)

DETAILED PROFIT AND LOSS ACCOUNT – TOTAL FOR THE YEAR ENDED 30 JUNE 2011

TOTAL – VS, CF AND GRANT

Incoming resources	2011 Unrestricted Funds	2011 Restricted funds	2011 Total	2010 Total
Voluntary income:	£	£	£	£
Subscriptions	101,034	-	101,034	95,145
Deed of covenant	39,432	-	39,432	52,801
Sponsorship	58,000	-	58,000	57,500
Legacies	61,102	-	61,102	412,801
Grants	-	457,049	457,049	302,970
General and Trust donations and other income	53,082	-	53,082	78,231
Activities for generating funds:				
Fundraising income:				
- Golf day	-	-	-	18,192
- Marathon	5,374	-	5,374	15,783
- Annual dinner	528	-	528	15,862
- Other	-	-	-	-
Investment income:				
Bank interest	4,627	-	4,627	8,617
Total incoming resources:	**323,179**	**457,049**	**780,228**	**1,057,902**
Resources expended				
Fundraising expenditure:				
- Golf day	500	-	500	12,519
- Marathon	4,523	-	4,523	8,100
- Annual dinner	-	-	-	9,365
- Merchandise	4,633	-	4,633	0
- Other	46,683	-	46,683	13,105
	56,339	**-**	**56,339**	**43,089**
Costs of charitable activities:				
Research costs and awards	162,175	308,472	470,647	48,000
Database and software expenditure	-	107,740	107,740	-
Donations	29,093	-	29,093	6,000
	191,268	**416,212**	**607,480**	**54,000**
Costs of generating voluntary income:				
Travel and subsistence	54,261	-	54,261	34,511
Office costs	16,603	-	16,603	27,134
Salaries and wages	135,970	-	135,970	132,217
Research costs	27,642	-	27,642	27,408
Tutor costs	7,500	-	7,500	7,500
Printing	13,212	-	13,212	14,833
Computer support costs	10,054	-	10,054	4,200
Stationery, postage and photocopying	7,913	-	7,913	5,871
General expenses	7,725	-	7,725	2,718
Prizes	1,500	-	1,500	2,350
Depreciation	26,805	-	26,805	22,162
	309,185	**-**	**309,185**	**280,904**
Governance costs:				
Audit and accountancy	11,065	-	11,065	9,348
Insurance	751	-	751	1,075
Legal and professional	782	-	782	20,483
Management and administration of the charity	321,783	-	£321,783	311,810
Total resources expended	569,390	416,212	985,602	408,899
Net incoming resources for the year	(246,211)	40,837	(205,374)	649,003

VSGBI LTD

DETAILED PROFIT AND LOSS ACCOUNT – AGM FOR THE YEAR ENDED 30 JUNE 2011

		2011		2010
	£		£	
TURNOVER				
Exhibition fees	131,577		123,071	
Registration fees (including course and dinner fees)	173,553		137,617	
	305,130		260,688	
		305,130		260,688
COST OF SALES				
Venue	150,807		133,328	
Travel and accommodation expenses	25,033		22,085	
Annual dinner	28,487		19,646	
Presidents dinner	9,568		7,353	
Book and programme printing	14,309		16,180	
Exhibitions	11,674		9,419	
Staffing	2,621		2,952	
Entertainment	145		255	
		(242,644)		(211,218)
OVERHEADS				
Insurance	4,150		4,150	
Office expenses	6,501		4,845	
Printing, postage and stationery	690		6,082	
Sundry expenses	-		174	
Accountancy fees	5,250		4,595	
Bank charges	6,324		886	
		(22,915)		(20,732)
OTHER OPERATING INCOME				
Donations and sponsorship		14,082		24,056
INTEREST RECEIVABLE				
Bank interest receivable		1		7
PROFIT ON ORDINARY ACTIVITIES		**53,654**		**52,801**

AUDIT AND QUALITY IMPROVEMENT COMITTEE REPORT

Chairman: David Mitchell

The last year has been marked by steady progress in reporting improved service quality for our patients. Following the endorsement of the Vascular Society membership, we published our mortality rates by unit for elective infra-renal abdominal aortic aneurysm for the first time in March of this year.

The data speak for themselves; mortality is down to 2.4%. The data from 2008 to 2010 show that the Society is able to absorb criticism and respond both positively and quickly. Part of the improvement is due to increasing use of EVAR (now more than 50% of elective AAA), with mortality from open repair higher at 4.3%, so there is still room to improve outcomes further.

The carotid audit has reported for the fourth time. We are treating more patients, more rapidly than ever before. These measures, coupled with improving data contribution, demonstrate our on-going commitment to improving the quality of vascular surgery in the UK.

The next step is for us to build on our success. The Society has obtained funding for a National Vascular Registry ((NVR) please see the article on the NVR in the yearbook). I talked about reporting against national standards last year. This year the society is engaged in the national commissioning process through a clinical reference group chaired by Professor Matt Thompson. This group is providing a framework and guidance with which we can build clear national standards. These will guide the development of the NVR. The registry will allow the society to report on outcomes for our index procedures and to develop our quality improvement work to include patients with PAD needing both revascularisation and amputation.

As units re-configure, we are hearing of more examples of larger centres being able to attract support for audit and quality improvement. It is important for us all to engage in gathering accurate audit data, not only to publicise the quality of service we provide, but also to meet national targets for revalidation. We will continue to report outcomes for AAA and carotid surgery, and will add in PAD and amputation outcomes once the registry is up and running. Ensuring good quality data both within the registry and in HES (or your national equivalent) through local coding reviews will become increasingly important in the future. We will be getting additional information on amputation from NCEPOD, which has set up an enquiry into amputation outcomes. This will report in 2014 and provide much food for thought. Vascunet is also undertaking external validation of registries with the support of the ESVS. They wish to validate UK data and I believe that we should support an external review of our data (from 2012 probably) as a benchmarking process against which we can assess ourselves in the future.

Another part of the changing landscape is the continuing change to data protection legislation. The new registry will operate under increasingly stringent controls on data. For this reason, we will be reviewing how data may be used by the membership for studies. It is likely that we will develop a much more formal and centralised process, requiring contributing teams to work with the registry to develop their ideas. Patient consent will be required and it is likely that we will need to obtain consent to hold data from all our patients in the future.

As the year ends, the personnel are changing. We are bidding farewell to Sara Baker. Sara has been the lynch pin of the NVD and has served the Society as trouble shooter and unit liaison for the NVD for many years. We owe her a very large debt of gratitude and I would like to wish her well in the future. Emily Diment was instrumental in helping us to obtain essential data handling approvals for our work to allow us to link our data to ONS datasets. Roxanne Potgieter, Julia McCleary and Helen Hindley have all moved on from their work on the AAA QIP. You will have received a report from the programme in the autumn (if not

you can get one from our stand). This details all the hard work that has gone on to improve team working, patient assessment and communication. We will also be reporting on our AKI audit at the AGM.

The Society owes them its thanks for providing enthusiastic support for our members as we modernise our audit practices. I am sure that you will join with me in wishing them well in their future careers.

Peter Barker, our national patient representative has worked with the Society for the last six years. He has proved a tireless critic and supporter of our work. He is moving on to other roles within the NHS and I would like to thank him for both his national work and his personal support of the AAA QIP team.

I would personally like to thank the many individuals, surgical, anaesthetic, radiological and nursing who helped with our work over the last years. You are too numerous to mention, but I hope that you will find some that your efforts rewarded in your clinical practices. We have endeavoured to mention you all in our AAA QIP report, any omissions are ours.

Finally, we are nothing without our patients and I want to record my thanks to the many individuals who gave up time to help us in the last year. We are beginning to engage more widely and to seek their views more reliably. This is very important for us in the future as we look to report outcomes not just from our professional perspective, but from the perspective of our patients. I would encourage all of you to develop focus groups to which you can turn to help shape your local services. The patient view can be both challenging and an important motivator for change.

Best wishes for an enjoyable meeting and the challenges of the year ahead.

Why are we changing from the NVD to the NVR?

The National Vascular Database (NVD) evolved from the Vascular Society's original voluntary audit database. It built upon the Society's ability to report outcomes following major index procedures (AAA, carotid, bypass and amputation), and was entirely funded by subscriptions from the Society membership. This changed in 2005 when the VSGBI obtained funding from HQIP (an arm of the Department of Health) to audit outcomes following carotid endarterectomy in partnership with the Royal College of Physicians. This relationship allowed us to develop the audit to report improvements of delivery of care to patients requiring carotid endarterectomy or stenting. We have now completed four rounds of reporting and have used this audit to drive quality improvement. We currently treat significantly more patients within the two week standard set by NICE than we did at the outset. Our partnership with the RCP, coupled with additional funding, has demonstrated how increased resources have helped us deliver improved care to our patients. These resources have also enabled us to report these improvements both publicly and to individual units.

At the same time as the carotid audit was delivering change, other agencies reported how poor our outcomes were for Abdominal Aortic Aneurysm (AAA) surgery. As a Society we recognised that things had to improve, but we lacked resources to do this. The QIP grant from the Health Foundation has proved liberating, allowing us to work to improve data entry into audit and subsequently in March this year to publish our mortality report, demonstrating significantly improved outcomes for patients undergoing elective AAA repair.

The message is clear; we need high quality audit to allow us to report how we as vascular surgeons deliver care to our patients. In addition we have to meet ever louder political demands for better audit and outcomes. High quality audit requires continual data analysis and reporting and this means money to employ personnel and analyse data. As a Society, we cannot afford to do this through subscriptions.

The Department of Health has also recognised that audit requires funding. In 2011, it invited bids to HQIP to fund national clinical audit. We were successful with our bid, with the contract being awarded in September 2012. One condition was that we had to demonstrate that we had a plan to improve, not just to conduct "business as usual". The National Vascular Registry (NVR) is the outcome of this process. The plan is to develop our datasets, to make them directly relevant to national standards set by NICE, the National Commissioning Board, the national AAA screening programme and ourselves through our quality improvement programmes. In addition, it will provide personnel to provide both support to members and casemix adjusted reporting of outcomes.

With secure funding, these activities will cease to be on an 'ad hoc' basis, dependent on the energy of busy clinicians, but run by a dedicated team within the Clinical Effectiveness Unit at the Royal College of Surgeons of England, who hold the contract on our behalf and will also hold the NVD data. The team will also seek to link data in the NVR to other relevant datasets to provide increasing richness of data about patient care for peripheral arterial disease and diabetes. This will take time, but we hope will begin to yield useful information within a few years. We will need to build links with our interventional radiology colleagues and anaesthetists to provide a more complete picture of how we deliver care.

There will be issues to address as we develop the NVR. One is the increasing demands of data protection. We will be obliged to seek our patients' consent to hold identifiable data about them. Whilst this may seem an imposition, the legal framework requires it. We have discussed this within our patient focus groups around the UK, and they are overwhelmingly in favour of us gathering robust audit data and linking datasets. Another consequence is that we will no longer "own" the data. This means that we will need to develop policies to enable us to use data for patient benefit beyond the immediate requirements of our audit contract. We will need to work in partnership with other healthcare agencies while meeting demands for patient confidentiality.

Whilst this may seem intimidating, it will not affect local units' abilities to download their data and manage it in house. What it will impact upon is our ability to share national data. We anticipate that non routine data analyses will need to be conducted within the CEU at the Royal College of Surgeons of England. To this end, the audit committee is preparing a formal process that will allow any clinical group to bid to undertake studies using the national dataset. Once in place, we will publicise this to the membership.

In addition to providing outcome reporting, we plan to maintain the current online real time reporting with funnel plots that we are used to. We hope to improve this by providing benchmarking against regional and national comparator data. This data will be useful both for local audit and to support revalidation. Whilst outcomes are an important part of audit, they can only be regarded as reliable if validated as complete. We hope that the NVR will be able to report data entry quality (in much the same way as the AAA mortality report) to provide a quality marker for outcomes.

> The message is clear; we need high quality audit to allow us to report how we as vascular surgeons deliver care to our patients.

When will this all happen? The contract was awarded in late September. We need to clarify details and hope to be able to start work in December this year. Building the datasets, delivering and testing them and moving from the NVD to the NVR will take some months, but we anticipate the NVR being live in midsummer 2013.

We will be keeping you updated about our progress throughout the year and plan to run workshops at the AGM in 2013 to help teams with queries about how to get the most out of the NVR. This is a tool that the society is developing to support both specialists and patients, please get involved and let us know your views.

EDUCATION COMMITTEE REPORT

Chairman: Jonathan Beard

I am sure you will know by now that the new Vascular Curriculum has been approved by the GMC. There were a few inconsistencies within the submitted endovascular competencies, but these have been corrected and were accepted by the GMC in October.

When accepted, the finalised curriculum will be available on both the VS and BSIR websites. I would like to thank the RCR and BSIR for their active engagement in this process through the RCR/VS/BSIR Liaison Group. I remain confident that high-quality imaging and endovascular training will be provided for both Vascular and IR trainees with the move to Specialty status and the instigation of the new Vascular Training Programme.

Cliff Shearman has been appointed Chair of the new Vascular SAC (vSAC). The vSAC has now selected its members and is working on the implementation of the new training programme. As 2013 will be the first time our new specialty has recruited, it is likely that a large number of trainees who apply for Vascular Surgery will also apply for General Surgery. In order to reduce the number of interviews trainees would have to attend and the need for interview panels a National Selection process will be held for trainees eligible to enter training at ST3 in March 2013 in conjunction with the General Surgery. These will be trainees who have completed core training or who have equivalent competencies. Six months of vascular surgery is desirable but not essential. There will be 20 posts available in the UK and trainees will receive a CCT in Vascular Surgery on successful completion of training.

Trainees currently in ST3 of General Surgery may be eligible to apply to transfer into the vascular training programme. However, this would have to be by local agreement from the relevant Deanery and General Surgery Programme. There would also have to be a limit to the numbers allowed to transfer to ST4 in Vascular Surgery (certainly no more than 20). The Deans are currently discussing this matter and as soon as we have clear guidance we will circulate to all eligible trainees.

While Deaneries must apply for approval of training posts to the GMC, the vSAC wish to adopt the model used by the Cardiothoracic SAC. Bids have been invited from Deaneries or consortia of Deaneries for 6 year training programmes. Together with the normal GMC application forms, the SAC will ask for additional information as recommended by the VS document on Standards of Training. This will include specific details about the clinical and educational opportunities of the posts and in particular include the names of the Vascular Surgery and Radiology trainers who have agreed to deliver the training. As there are more units offering vascular training than there will be trainees, this seems a fair way to allocate the numbers, rather than basing it on historical grounds. The vSAC has asked Schools of Surgery if they can identify a local lead or the Programme Director for Vascular Surgery (if appointed), who would progress the application process. They would be required to identify training units to ensure trainees receive high quality training over 6 years. In particular, they would need to negotiate with radiology trainers locally to ensure they are willing to be involved. We hope that this process will start in October with a view to getting GMC approval of the placements by January 2013.

Other requirements for a new specialty include a vascular logbook and a vascular FRCS exam. John Brennan and his working group have done a great job in writing a new Vascular Logbook, which is hosted by the e-Logbook facility at the RCSEd. This logbook enables separate attribution of each part of a major procedure. The logbook has also been designed so that it can be used by consultants, as it will also provide codes for HES and the NVR.

Julian Scott has made a fantastic start towards creating the new Vascular FRCS exam. As the current Chair of the Vascular Surgery Exam Board, he has begun appointing

a question writing panel. There are about 200 questions in the existing General Surgery bank and we will need about 1000 for a separate exam. The outline of the exam has already been agreed and a detailed submission was sent to the GMC in September. It seems likely that the first diet of the exam will be in 2015 at the earliest, and before then examiners will also have to be appointed – fortunately many existing 'General Surgery' examiners are actually vascular surgeons!

Two Cook Fellows were appointed to 6-month Endovascular Fellowships at interviews held during the spring meeting of the Society in Belfast. The successful candidates were James Coulston and Said Abisi, who are going to Liverpool and St Thomas' Hospital, London respectively. I am pleased to announce that these fellowships have been accredited by the RCS(Eng), which means that they are now part of the National Surgical Fellowship Scheme. Two more Cook Fellowships have been advertised for 2013 and the shortlisted applicants will be interviewed shortly. We are indebted to Cook for their continued support.

I am delighted to report that John Brennan has been appointed as the new Chair of the renamed Education Committee (training is now the responsibility of the vSAC). He will take office after this meeting and will work closely with both vSAC and radiology to ensure the development of the world-class education required to support all aspects of vascular training. It only remains for me to thank the Committee members for all the hard work and support they have given to me over the last four years.

PROFESSIONAL STANDARDS COMMITTEE REPORT

Chairman: Michael Gough

It has been the quietest year yet for the Professional Standards Committee.

Thus far there have been no outliers identified from the National Vascular Database and no requests from individual Trusts for help in assessing outcomes in individual units. It would be wonderful if this state of calm were to become permanent.

My term as Chair of this Committee ends at the AGM this year, and with it a long association with the Vascular Society in the various posts that I have been honoured to hold. Thank you.

Jonothan Earnshaw has been elected by Council as the next Chair of this Committee and I hope he is as untroubled as I have been!

RESEARCH COMMITTEE REPORT

Chair: Shervanthi Homer-Vanniasinkam

I am delighted to report on the activities of the Research Committee (RC), which was established in 2009 as the engine within the Vascular Society to drive excellence in vascular research.

We are all aware how difficult it is for busy clinicians to find the time to pursue their research interests – yet we are also aware how very important it is, both for the profession and for our patients, that they should. During the past three years the RC has established grants for surgeons, to be awarded at key stages of their careers, which we believe will make a crucial difference in turning the almost impossible into a challenging but achievable goal.

Working in close partnership with the Circulation Foundation, we now fund three annual awards: the President's Early Career Award (PECA), now in its third year; the Surgeon Scientist Award (SSA), now in its second year; and a new award this year, the George Davies Visionary Award (GDVA).

The PECA is for consultants within 5 years of their first substantive appointment: they are supported by funding of £100,000 over 2 years, while they develop an independent research career.

The SSA funds vascular surgical trainees while they engage in a period of formal research. Trainees receive a total grant of £55,000 for 12 months, to cover their salary and up to £10,000 towards laboratory expenses, for project work that would lead to a higher degree (MD or PhD).

The GDVA aims to bridge the gap between the SSA and the PECA and to support creative research talent in individuals committed to innovation and excellence in vascular research. This award, which provides £80,000 over 2 years, is available to individuals who have completed their work for, or are already in possession of, a higher degree (MD or PhD), and are committed to developing their clinician-scientist careers.

The 2012 awards will be presented at the AGM in Manchester in November.

> 'Man's mind, once stretched by a new idea, never regains its original dimensions.'
>
> **Oliver Wendell Holmes (1809-1894)**

Further details of these awards can be found on the Circulation Foundation website http://www.circulationfoundation.org.uk/

Also on this website is the 'Research Strategy', which was drawn up by the Committee in the past 12 months, in conjunction with the Circulation Foundation. This, along with the Peer Review Panel which is currently being established for all the major grants to be awarded in 2013, means that we now are compliant with the Association of Research Medical Charities who govern good practice in research funds awarded by charities.

Looking ahead, the RC will be engaged in establishing External Peer Review Panels for all the major grants to be awarded in 2013. We will also be discussing and debating topical issues such as open access publishing of publicly funded scientific research, as recommended in the Finch Report, and the potential implications for future grants awarded by the Society. This document, entitled 'Accessibility, sustainability, excellence: how to expand access to research publications', was produced by the 'Working Group on Expanding Access to Published Research Findings', chaired by Dame Janet Finch, CBE, and published in June this year. I feel it is very important that the RC keeps abreast of such national developments and initiatives, and that we are ready to respond with our own clear views.

The RC is charged with stimulating and encouraging young surgeons to actively pursue the quest for new knowledge; I believe we have made significant progress in this by establishing a pipeline of grants to fund vascular research.

As I conclude this report, I would like to take the opportunity to thank my colleagues on the RC for their hard work over the past year. I would also like to acknowledge the support given to the RC by Council and the CF Committee, and I look forward to working with them in all our future endeavours.

CIRCULATION FOUNDATION REPORT

Chairman: Ian Franklin

2012 has been the most productive and effective year ever for the Circulation Foundation. We have granted more research awards than ever before. Grass-roots support from vascular surgeons and trainees has increased more this year than in any previous year.

Monthly income from regular donors has more than doubled. We have been fortunate in attracting major individual donations from generous benefactors and a substantial legacy has swelled coffers still further.

The Circulation Foundation now makes three major awards each year. These are designed to support vascular surgeons at sequential and important stages of their careers and produce the finest research possible for the money spent.

The Surgeon Scientist Award (SSA) is aimed at vascular surgeons in training pursuing a higher degree. Three SSA recipients are currently funded by the Circulation Foundation: Alan Karthikesalingam (SSA 2011) at St George's, Hospital in London is working on optimising post-operative surveillance protocols following EVAR. Nikesh Dattani (SSA 2012) in Leicester is studying the significance of glucose transporters in aortic aneurysm pathogenesis. This year we were able to fund an extra SSA thanks to our major benefactor George Davies and the George Davies SSA was given to Nadeem Mughal in Leeds researching the basic mechanisms of atherosclerotic disease.

A new award, the George Davies Visionary Award has been developed and will be awarded for the first time in autumn 2012 and presented to the winner at the annual meeting in Manchester. This is designed to support surgeons who have completed a higher degree but seeking to continue research before taking up a consultant post.

The flagship award is the President's Early Career Award, our most prestigious and highest value grant. Two PECA recipients are currently funded by the Circulation Foundation, Matthew Bown in Leicester and Colin Bicknell at Imperial in London. The winner of the 2012 PECA will be announced at the annual meeting.

Vascular Disease Awareness Week 2012 on the theme "It can happen to anyone at any time" was the most successful CF awareness week ever with over 80 events taking place over the country, numerous press and radio interviews, sponsored events and money raised. Throughout this week and the rest of the year races were run, roads were cycled, pools were swum, mountains climbed, golf balls hit and seas sailed to name just a few of the events organised to raise funds for vascular research in the UK. We are profoundly grateful to everybody who gave time, effort and money to bring in the money and raise awareness of the work of the Circulation Foundation.

Our 2013 Awareness Week will take place from the 11th – 17th March and we will be focusing on Peripheral Arterial Disease. Visit the Circulation Foundation stand for more information, please do get involved.

We are pleased with progress so far but there is much work still to do to realise our vision of transforming the Circulation Foundation into a major research charity and foster a renaissance of vascular research in the UK. Please help us by becoming a regular donor. Any amount, big or small, on a regular basis is hugely appreciated and we are grateful for every penny.

CIRCULATION FOUNDATION
The Vascular Research Charity

Our special thanks to the following Vascular Society members making a regular donation to the Circulation Foundation from July 2011-June 2012:

Mr Matthew Armon
Mr Marco Baroni
Professor Jonathan Beard
Mr David Berridge
Mr Jonathan Boyle
Mr Bruce Braithwaite
Mr John Brennan
Mr Marcus Brookes
Mr Richard Corbett
Professor Alun Davies
Mrs Linda de Cossart
Mr Richard Downing
Mr Ian Franklin
Mr Simon Fraser
Mr Andrew Garnham
Mr David Gerrard
Mr Chris Gibbons
Professor Michael Gough
Mr Gareth Griffiths
Professor George Hamilton
Mr Simon Hardy
Professor Shervanthi Homer-Vanniasinkam
Professor Michael Horrocks
Mr Craig Irvine
Mr Michael Jenkins
Mr Tim Lees
Mr Ian Loftus
Mr Shane MacSweeney
Mr Adrian Marston
Mr Andrew May
Mr James McCaslin
Mr David Mitchell
Mr Ian Muir
Professor A Ross Naylor
Mr Isaac Nyamekye
Mr Simon Parvin
Miss Sophie Renton
Mr Paul Renwick
Professor Rob Sayers
Professor Julian Scott
Mr Michael Sharp
Professor Cliff Shearman
Mr Malcolm Simms
Professor Gerard Stansby
Mr Peter J Taylor
Mr Martin Thomas
Professor Matt Thompson
Mr Kevin Varty
Miss Lucy Wales
Mr David Williams
Mr John Wolfe
Mr Kenneth Woodburn
Mr Mike Wyatt

Affiliate

Mr Manj Gohel
Mr Alan Karthikesaligam
Mr James McCaslin
Miss Hayley Moore
Mr Vikas Pandy

Allied Health Professionals

Mr David Carser
Mrs Mary Ellis
Mrs Emma Bond

Vascular Disease Awareness Week: 11th-17th March 2013

VASCULAR TUTOR'S REPORT

Ian Chetter

I have now completed almost 3 years as the Vascular Tutor and this time has flown.

My original brief was to evaluate and improve current courses; to match the course portfolio to the vascular curriculum; to introduce assessment into the courses; to improve/standardise course material (eg manuals) in order to promote transfer to e-learning; and finally to facilitate the regionalisation of courses. I have managed to complete the majority of these aims and this together with being appointed Training Programme Director (Vascular) for the Yorkshire & Humber Deanery led me to take the decision to demit the post as Vascular Tutor 4 months early. I hope I will be quickly replaced, but there are discussions within the college to move from specialty based to project based tutors – so watch this space! Either way, the position of RCS / VS tutor has been a fantastic experience and one I would recommend to anyone with an Educational interest.

Since my last report, we have been busy fine tuning courses, developing and piloting courses in evolution and designing future courses to mirror the new vascular surgical curriculum. Another busy 12 months! The vast majority of courses now have manuals and include some assessment of knowledge, motor skills and decision making. In addition all non – cadaveric courses are in a format which should be straightforward to regionalise. Indeed we are running 3 courses (essential vascular ultrasound, modern management of varicose veins and EVAR planning) back to back in Hull as a pilot "regionalised" vascular week.

Established courses. The ever popular cadaveric Amputation, Specialty Skills and Advanced Skills courses were fully subscribed this year and were well received according to feedback. Unfortunately, the EVAR planning course was cancelled due to circumstances beyond my control; however it is due to return to the portfolio on 14 and 15th December 2012.

Courses in evolution. The fresh frozen cadaveric Vascular Access for Dialysis course ran very successfully for the first time in September 2011, receiving excellent feedback. Following a few very minor alterations it was repeated on 24 and 25 September 2012, to similar acclaim. Fresh frozen material certainly seems to be the gold standard for this practical simulation! The Modern Management of Varicose Veins course ran in October 2011, and was a great learning experience. I aim to correct clearly identified deficiencies in the course before running a revamped version on 12-13th December 2012.

New courses. We are planning to introduce 2 new courses over the next 12 -24 months. The development of the Basic Vascular Ultrasound is progressing well. I visited the ESVS Basic Vascular Ultrasound course, hosted by Jonas Eiberg in Copenhagen. This is an excellent, established course and will function as a template for the new RCS course, which will pilot on 10-11th December 2012. Also in development is an Essential Vascular Interventional Radiology course.

Ansell and Maquet have very generously agreed to continue their sponsorship of the vascular portfolio this year by funding bursaries to support participant attendance at RCS(Eng) courses, and W L Gore have agreed to sponsor the Vascular Access course. Several potential new sponsors are being chased.

Potential participants and faculty, please note in your diaries the provisional dates of the vascular courses for 2013

- Amputation: 15th and 16th January 2013
- Specialty Skills in Vascular Surgery: 24th and 25th June 2013
- Advanced Skills in Vascular Surgery: 26th-28th June 2013
- Vascular Access: 2nd and 3rd September 2013
- EVAR planning: 5th and 6th December 2013
- Essential Vascular Ultrasound: 10th and 11th December 2013
- Modern Management of Varicose Veins: 12th and 13th December 2013

I would like to take this opportunity to say a huge thank you to the faculty who teach on the courses. My job as Vascular Tutor would be impossible without their support and enthusiasm – Thank you! Finally I would like to wish my successor all the very best and reassure him/her I will be available for consultation if required!

ROULEAUX CLUB

The Rouleaux Club has played a key role in the development of the UK Endovascular Trainees (UKETs) organisation, which was successfully launched in August.

Specifically designed to improve IR training opportunities through the use of high fidelity simulators, it has the added benefit of fostering multidisciplinary (surgery, cardiology, radiology) collaboration early in a trainee's career. Other training initiatives in development include a national vascular training day in 2013 and a greater focus on the development and dissemination of web-based distance learning resources.

The structure of the Rouleaux Club Executive Committee has also been augmented with the creation of new roles. These include representation on the Vascular SAC (Mr James Scurr), and the Council of the ESVS in Training (Mr Femi Oshin). Furthermore, the Rouleaux Club has also embraced social media by creating a Twitter account, in addition to its Facebook page, and introduced local representation at Deanery level.

We are grateful to Professor Naylor for timetabling a Rouleaux Symposium at this year's AGM. This will address the potential training implications of the new specialty and we will provide an opportunity to build on last year's success by announcing the winner of the 2013 Golden Graft award.

Rouleaux Club Executive Committee

Committee

President
Mr Femi Oshin

Vice President
Mr James McCaslin

Secretary
Mr Alan Karthikesalingam

Treasurer
Mr Michael Wall

Vascular Society Affiliate representative
Mr Femi Oshin

ASIT representative
Mr Gary Lambert

BSET and EVST representative
Mr Femi Oshin

SAC representative
Mr James Scurr

www.rouleauxclub.com

SOCIETY OF VASCULAR NURSES

The Society of Vascular Nurses is a society run by nurses for nurses and allied healthcare professionals. Our main aim is to provide a national network for healthcare professionals working within vascular care.

This network provides a forum for education and debate on vascular nursing issues.

The SVN was founded in 1993 and now has over 200 members from many different professional facets. These include tissue viability nurses, theatre practitioners, podiatrists, physiotherapists, staff nurses, health care support workers, nurse specialists, nurse practitioners, nurse consultants and research nurses.

2012 has been an interesting year for the SVN. The Vascular Matters Journal launched in 2011 by the SVN has sadly had a short lifespan. Although it was received very favourably by its readership, the publishing company folded due to the current economic climate and difficulty in securing advertising. The SVN has therefore returned to the SVN Newsletter, which under the guidance of the new editors, Wendy Hayes and Leanne Cook, has become a bigger and bolder quarterly edition.

As will be announced at this year's SVN Annual Conference, much work has been started on the roles of Vascular Nurses throughout the UK. With the changes within Vascular Surgery in the NHS, it is important to understand how nurses' roles have evolved. Once this data has been collated, a review of the Vascular Nurse Competencies will be performed.

The SVN continues to liaise with other National Vascular Societies and Charities and is proud to have the Annual SVN Conference under the supportive umbrella of the Vascular Society's Annual General Meeting. The SVN are also delighted to be included in the Venous Forum's Spring Meeting 2013 and has ongoing involvement in NICE policy formulation. The SVN continue to support the Circulation Foundation, who are holding an evening meeting for SVN members on the 27th of November 2012.

Membership to the SVN begins at £20 for one year and offers many benefits, including the opportunity to apply for a bursary up to the value of £500.

The SVN would like to take this opportunity to thank the Vascular Society President, Professor Ross Naylor, Honorary Secretary, Mike Wyatt, and Chief Executive Officer, Jeanette Oliver, for their continued time and support.

Emma Bond
President, SVN

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THE SOCIETY FOR VASCULAR TECHNOLOGY OF GREAT BRITAIN AND IRELAND

2012, our 21st year of the Society, has been a period of developments for the SVT. The SVT have been invited to sit on the new Academy for Healthcare Science Council.

This is a great opportunity to bring specialities together, raise our profile, and will provide a united and strong voice in the wider healthcare environment. We hope to pursue state registration for all healthcare scientists.

The DH Modernising Scientific Careers programme is well underway and is popular with vascular trainees. The SVT are currently working on the Higher Specialist Scientific Training routes as well as the Assistant level modules.

The Improving Quality In Physiological diagnostic Services (IQIPS), the new Service Accreditation programme is due to 'go-live' for Vascular Labs at the time of writing this. This will define the quality of service delivery and will help signpost commissioners.

The Circulation Foundation is kindly awarding 2 research awards this year to SVT members who are undertaking research as part of a Vascular MSc.

Our full-colour newsletter has proved very popular. The updated membership database and website has been a great success and we have now enabled on line payments for membership renewals, study days and exam registrations.

The SVT maintain strong links with the Vascular Society, NHS vascular screening programmes, the Circulation Foundation and BMUS.

We look forward to the AGM in Manchester this year.

Jo Walker
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THE VENOUS FORUM OF THE ROYAL SOCIETY OF MEDICINE

The Venous Forum (VF) held a highly successful 2-day meeting at the Royal Society of Medicine in London (http://www.rsm.ac.uk/academ/vec02.php) on 25 and 26 April 2012.

The meeting was well-attended with around 200 registrants from a wide range of different professional backgrounds, and was generously supported by 19 sponsors who provided an interesting and educational trade exhibition.

The first day focused on the epidemiology, diagnosis and management of venous thrombo-embolism (VTE) with a 'who's who' of UK experts, many drawn from the NICE Guideline Development Group on VTE (http://guidance.nice.org.uk/CG144), speaking to different aspects of this important and complex area of medicine.

As well as the plenary sessions, there was ample opportunity for young researchers to present their original work both orally and as posters. The VF awarded three Research Prizes, a Travelling Fellowship, and a Pump-priming Grant to support young venous specialists in their clinical and research training.

The VF continues to foster close links with other societies such as the American Venous Forum, the American College of Phlebology and the European Venous Forum (EVF). The Venous Forum was once again pleased to support the highly successful EVF Hands-On Workshop, held in Cyprus 31 October - 3 November 2012, and looks forward to working closely with the EVF in 2013.

The Venous Forum continues with its Venous INtervention (VEIN) Project with the aim of helping to define the 'standard of care' for patients in the UK and beyond presenting with venous disease.

VEIN-1 dealt with varicose veins; VEIN-2 with chronic venous insufficiency; and VEIN-3, which was also published as a separate supplement in Phlebology in April 2012 (to coincide with the Spring Meeting) (http://www.rsm.ac.uk/academ/forvenou.php) focused on the important topic of VTE. VTE remains an important cause of preventable death and has been made a priority for the NHS. The nine chapters in VEIN 3 are aimed at the multidisciplinary teams involved in the day-to-day care of patients with VTE as well as at allied health professionals and health-care commissioners.

The VF has begun work on VEIN-4 which will comprehensively review the wide range of new technologies being used to treat superficial venous insufficiency.

After two very enjoyable and rewarding years in post, Andrew Bradbury will be stepping down as President after the November 2012 meeting in Manchester, happy in the knowledge that he will be leaving a strong Venous Forum in the very capable hands of David Berridge.

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THE JOINT VASCULAR RESEARCH GROUP

Chairman: Frank CT Smith

The Joint Vascular Research Group is a collaborative network of vascular surgeons, interventional radiologists, research nurses and technologists, who share an interest in clinical research and educational aspects of vascular intervention.

Membership is by centre and if you are interested in becoming involved please contact our co-ordinator, Jude Day, who will be able to provide you with further details. We welcome interested contributors.

This year's AGM and Winter Meeting will take place at the Manchester Convention Centre, on Tuesday 27th November 2012.

Despite a shifting emphasis of the Group to evidence-based vascular education and publication, original clinical research studies undertaken recently includes investigations into acute arm ischaemia; mesenteric ischaemia; outcome of internal carotid artery sub-occlusion; iliac artery compression syndrome in cyclists and; use of Nordic walking poles in claudication.

The Group has published a number of evidence-based volumes in recent years including:

- The Evidence for Vascular Surgery (Editions One and Two)
- Pathways of Care in Vascular Surgery
- The lavishly illustrated volume, Rare Vascular Disorders.
- Complications in Vascular and Endovascular Surgery:
  How to Avoid Them and How to Get Out of Trouble (published in 2011).

All these publications can be obtained for Nikki Bramhill at tfm Publishing (nikki@tfmpublishing.com).

The JVRG is grateful to John Howard and Nuros for their continued support.

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