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The Office

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The Council

Front row, left to right:
Professor J D Beard; Mr P M Lamont; Mr J J Earnshaw; Professor C Shearman; Ms J Robey;
Mr S D Parvin; Mr D C Mitchell

Second row, left to right
Mrs S Ward (SVN); Professor A R Naylor; Professor G Stansby; Mr S MacSweeney; Mr R Holdsworth; Mr D Baker; Mr R Hinchliffe; Mr J Brennan; Ms E Young (SVT)

Back row, left to right
Professor D Ettles; Professor S Homer-Vanniasinkam; Mr I Nyamekye; Mr I Franklin; Mr I Chetter; Mr M G Wyatt

Not pictured:  Mr P Blair
As I write this report it seems unbelievable to think that I am nearing my term of office as your President. Prior to this year I have spent 3 years on Council and 4 years as Chair of the Training and Education Committee and I will miss the energy and enthusiasm of The Vascular Society Council very much. During this time I have worked with a number of very able and clear minded colleagues who have given a lot of time to The Vascular Society. In particular, Jonothan Earnshaw as Secretary and Jeanette Robey as Chief Executive together with her team have worked phenomenally hard to steer the Society through some challenging times. Their experience and organisation has also made my life much easier for which I am very grateful.

This last year has been both exciting and challenging for vascular surgery and vascular surgeons. A number of factors have come together to create a perfect storm; the ideal opportunity to bring about change. Several issues have been brewing for some time. The need to improve our training programmes to embrace endovascular surgery has necessitated seeking approval to become a new specialty, separate from general surgery. After numerous attempts to find other solutions, this approach turned out to be the only way to avoid our future vascular surgeons having to take time out of their training programme to gain endovascular training on fellowships, often abroad. With the strong support of John Black, President of the RCS, we have been successful at Stage 1 of the Department of Health process (justifying the need for a new specialty and gaining the support of all the related bodies). We now have to demonstrate we have the syllabus and structure to deliver this, and we are confident that we have. The aim is to recruit our first trainees in 2012 and I hope I will be able to give you more news on this in November.

Previously some Members of The Vascular Society complained that vascular surgery did not have a high profile in the public and political domain. I think this changed very dramatically this year! Concerns about poor outcome results for aortic surgery in the UK reported by Vascunet were picked up by the media, and the Guardian ran a series of high profile articles about this. Occurring at the time when the NHS Aneurysm Screening Programme is being rolled out, this was potentially very damaging. I believe the response of The Vascular Society was important in channeling the initial concerns into mechanisms to improve vascular surgical services. The development of the Quality Improvement Programme, led by David Mitchell with the aim to halve mortality from aortic surgery by 2013, is well underway. The importance of
high quality data was vividly highlighted, and the value of the National Vascular Database in obtaining this was clearly demonstrated.

In collaboration with the Circulation Foundation, The Vascular Society has also been to lobbying sessions in the Houses of Parliament to raise the profile of vascular disease and the need to improve vascular services with members of both houses. This led to an early day motion and debate in the House of Commons just before the end of the last Parliament, and we have been asked to go again in October to give evidence about the need to improve vascular services. In September, The Vascular Society supported by the Public Relations Department in the Royal College of Surgeons ran a high profile media campaign to announce the results of the carotid surgery audit and the organisation survey to which Members of The Vascular Society had contributed. This increased awareness of vascular disease has also been reflected by NICE who had just initiated a clinical guideline group for peripheral arterial disease chaired by a vascular surgeon, Professor Jonathan Michaels from Sheffield.

The demonstration that volume is linked closely with outcome and concerns with the provision of emergency services has stimulated many Strategic Health Authorities to review their provision of vascular services, moving towards larger units or networks. Coming at a time of financial austerity this is obviously going to be difficult for some Members, but it is probably the best opportunity we will all have to ensure that vascular surgery is fit for purpose over the next decade.

In the year since the Research Committee was established, chaired by Professor Shervanthi Homer-Vanniasinkam, the impact has been dramatic. Working with the Circulation Foundation, research grants of nearly £100,000 have been awarded and this year for the first time a President’s Early Career Award has been established and will be presented at the AGM to help a newly appointed consultant establish a research programme. The winner of the award worth £100,000 over 2 years, Mr Matt Bown, will be presenting his project at the SARS meeting on Wednesday morning.

I hope you will be able to attend the AGM in Brighton. The meeting is designed to offer you the opportunity to hear about how vascular surgery is shaping up to face the next decade as a new specialty, and for you to debate and share your ideas about issues that you feel are affecting you. I look forward to seeing you there.
Members of Council 2009-2010

President
Professor C Shearman

President Elect
Mr P M Lamont

Vice President Elect
Professor A R Naylor

Honorary Secretary
Mr J J Earnshaw

Honorary Secretary Elect
Mr M G Wyatt

Honorary Treasurer
Mr S D Parvin

Ordinary members
Mr D Baker
Mr P Blair
Mr J Brennan
Mr I Franklin
Mr R Holdsworth
Professor S Homer-Vanniasinkam
Mr S MacSweeney
Mr I Nyamekye
Professor G Stansby

Training & Education Committee Chair
Professor J D Beard

Audit & Quality Improvement Committee Chair
Mr D C Mitchell

Research Committee Chair
Professor S Homer-Vanniasinkam

Affiliate member
Mr R Hinchliffe

Vascular Tutor
Mr I Chetter

Observers
Professor D Ettles (BSIR)
Ms E Young (SVT)
Ms W Hayes (SVN)
Committees 2009-2010

AUDIT AND QUALITY IMPROVEMENT COMMITTEE
Mr D C Mitchell (Chair)  Mr J J Earnshaw
Mr D Baker  Mr S D Parvin
Mr I Franklin  Professor G Stansby
Mr P Barker  Mrs S Baker
Ms R Potgieter  Mr J V Smyth (Co-opted)
Mr R Chalmers (Co-opted)  Ms C Marshall (Co-opted)
Dr S Thomas (BSIR)  Dr I Robertson (BSIR)
Dr M Price (VASGBI)  Dr C Snowden (VASGBI)

TRAINING AND EDUCATION COMMITTEE
Professor J D Beard (Chair)  Mr I Chetter
Mr R Holdsworth  Mr S MacSweeney
Mr P Blair  Mr I Nyamekye
Mr J Brennan  Dr I McCafferty (BSIR)
Mr R Hinchliffe

RESEARCH COMMITTEE
Professor S Homer-Vanniasinkam (Chair)  Professor R Sayers
Mr T Lees
Ms J Brittenden

PROFESSIONAL STANDARDS COMMITTEE
Professor M J Gough (Chair)  Mr S D Parvin
Mr D C Mitchell  Mr D C Berridge
Mr P M Lamont  Mr J Thompson
Mr P R Taylor  Professor M J Gough

CIRCULATION FOUNDATION COMMITTEE
Mr A May (Chair)  Mr I Franklin
Professor G Hamilton  Mr M Bartlett (SVT)
Mr J Wolfe  Ms L Allen (SVN)
Mr T Lees
Mr R N Baird
Professor G Stansby

Membership of Vascular Advisory Committee
All Members of Council, plus Vascular Advisors:
Mr M Armon, East Anglia  Mr P Dunlop, Northern
Mr S Fraser, Scotland (East)  Mr A Garnham, West Midlands
Mr A Guy, Mersey  Mr R Hannon, Northern Ireland
Ms S Hill, Wales  Mr C Irvine, Yorkshire
Mr T Loosemore, South West Thames  Mr T Magee, Oxford
Mr M McCarthy, East Midlands  Mr C McDonnell, Republic of Ireland
Mr G Morris, Wessex  Mr J Mosley, North Western
Mr D Orr, Scotland (West)  Miss S Renton, North West Thames
Mr M Salter, North East Thames  Mr Singh, South Yorkshire and
Mr J Thompson, South Western  North Derbyshire
Mr S W Yusuf, South East Thames

Vascular Members of the SAC in General Surgery:
Professor M J Gough  Mr G Griffiths
Ms A Howd  Mr R Vohra
Wing Commander T Whitbread
### Annual General Meetings

<table>
<thead>
<tr>
<th>Year</th>
<th>Venue</th>
<th>President</th>
<th>Secretary</th>
<th>Treasurer</th>
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<tr>
<td>1966</td>
<td>Inaugural Meeting, The Middlesex Hospital, London</td>
<td>Mr Sol Cohen</td>
<td>Mr JA Gillespie</td>
<td>Mr JA Gillespie</td>
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<tr>
<td>1967</td>
<td>Edinburgh</td>
<td>Mr Sol Cohen</td>
<td>Mr PGC Martin</td>
<td>Mr A Marston</td>
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<td>1968</td>
<td>Hammersmith Hospital, London</td>
<td>Professor AW Mackay</td>
<td>Mr A Marston</td>
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<td>1969</td>
<td>Royal Infirmary, Glasgow</td>
<td>Mr HHG Eastcott</td>
<td>Mr DGA Eadie</td>
<td>Mr DGA Eadie</td>
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<tr>
<td>1970</td>
<td>University College, Dublin</td>
<td>Professor Sir D Douglas</td>
<td>Professor CV Jamieson</td>
<td>Professor CV Jamieson</td>
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<td>1971</td>
<td>St Mary’s Hospital, London</td>
<td>Professor JB Kinmonth</td>
<td>Professor AO Mansfield</td>
<td>Professor AO Mansfield</td>
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<td>1972</td>
<td>The University, Dundee</td>
<td>Mr CR Helsby</td>
<td>Mr PL Harris</td>
<td>Mr PL Harris</td>
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<td>1973</td>
<td>St Thomas’s Hospital, London</td>
<td>Mr S Rose</td>
<td>Mr SG Darke</td>
<td>Mr SG Darke</td>
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<tr>
<td>1974</td>
<td>Queen Elizabeth Hospital, B’ham</td>
<td>Professor F Ashton</td>
<td>Professor CV Ruckley</td>
<td>Professor CV Ruckley</td>
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<tr>
<td>1975</td>
<td>St Bartholomew’s Hospital, London</td>
<td>Professor GW Taylor</td>
<td>Professor AO Mansfield</td>
<td>Professor AO Mansfield</td>
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<tr>
<td>1976</td>
<td>Royal Infirmary, Bristol</td>
<td>Professor JH Peacock</td>
<td>Professor CV Ruckley</td>
<td>Professor CV Ruckley</td>
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<td>1977</td>
<td>Pfizer Foundation, Edinburgh</td>
<td>Professor RB Galloway</td>
<td>Professor WB Campbell</td>
<td>Professor WB Campbell</td>
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<td>1978</td>
<td>Liverpool</td>
<td>Professor PH Dickinson</td>
<td>Mr RJ Kenyon</td>
<td>Mr RJ Kenyon</td>
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<tr>
<td>1979</td>
<td>John Radcliffe Hospital, Oxford</td>
<td>Mr DP Tibbs</td>
<td>Professor JH Peacock</td>
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<td>1980</td>
<td>St Thomas’s Hospital, London</td>
<td>Mr FB Cockett</td>
<td>Mr PL Harris</td>
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<tr>
<td>1981</td>
<td>University Hospital of Wales, Cardiff</td>
<td>Mr G Heard</td>
<td>Mr MH Simms</td>
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<td>1982</td>
<td>University Hospital of South Manchester</td>
<td>Mr S Rose</td>
<td>Mr SG Darke</td>
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<td>1983</td>
<td>St Mary’s Hospital, London</td>
<td>Mr JR Kenyon</td>
<td>Mr PL Harris</td>
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<td>1984</td>
<td>Medical School, Birmingham</td>
<td>Professor F Ashton</td>
<td>Professor CV Ruckley</td>
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<td>1985</td>
<td>The Middlesex Hospital, London</td>
<td>Professor RB Galloway</td>
<td>Professor WB Campbell</td>
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<td>1986</td>
<td>The Institute of Education, London</td>
<td>Professor RB Galloway</td>
<td>Professor WB Campbell</td>
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<td>1987</td>
<td>Civic Centre, Newcastle-upon-Tyne</td>
<td>Professor RB Galloway</td>
<td>Professor WB Campbell</td>
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<td>The University of Leeds</td>
<td>Professor RB Galloway</td>
<td>Professor WB Campbell</td>
<td>Professor WB Campbell</td>
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<td>1989</td>
<td>Ninewells Hospital, Dundee</td>
<td>Professor RB Galloway</td>
<td>Professor WB Campbell</td>
<td>Professor WB Campbell</td>
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<tr>
<td>1990</td>
<td>Kensington Town Hall, London</td>
<td>Professor RB Galloway</td>
<td>Professor WB Campbell</td>
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<tr>
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<td>Royal College of Surgeons, Dublin</td>
<td>Professor RB Galloway</td>
<td>Professor WB Campbell</td>
<td>Professor WB Campbell</td>
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<td>Metropole Hotel, London</td>
<td>Professor RB Galloway</td>
<td>Professor WB Campbell</td>
<td>Professor WB Campbell</td>
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<td>1993</td>
<td>Royal Northern College of Music, Manchester</td>
<td>Professor RB Galloway</td>
<td>Professor WB Campbell</td>
<td>Professor WB Campbell</td>
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<tr>
<td>1994</td>
<td>Assembly Rooms, Edinburgh</td>
<td>Professor CV Ruckley</td>
<td>Professor AO Mansfield</td>
<td>Professor AO Mansfield</td>
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<td>1995</td>
<td>Kensington Town Hall, London</td>
<td>Mr CW Jamieson</td>
<td>Mr RJ Kenyon</td>
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<tr>
<td>1996</td>
<td>Bournemouth International Centre, Bournemouth</td>
<td>Mr SG Darke</td>
<td>Mr MJ Gough</td>
<td>Mr MJ Gough</td>
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<td>1997</td>
<td>Royal Lancaster Hotel, London</td>
<td>Professor A O Mansfield</td>
<td>Mr PL Harris</td>
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<td>1998</td>
<td>City Hall, Hull</td>
<td>Mr JMD Galloway</td>
<td>Mr RJ Kenyon</td>
<td>Mr RJ Kenyon</td>
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<tr>
<td>1999</td>
<td>De Montfort Hall, Leicester</td>
<td>Professor PRF Bell</td>
<td>Mr RB Galloway</td>
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<tr>
<td>2000</td>
<td>London Arena, Docklands, London</td>
<td>Professor RM Greenhalgh</td>
<td>Mr RB Galloway</td>
<td>Mr RB Galloway</td>
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<tr>
<td>2001</td>
<td>Hilton Brighton Metropole, Brighton</td>
<td>Mr RN Baird</td>
<td>Mr RB Galloway</td>
<td>Mr RB Galloway</td>
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<tr>
<td>2002</td>
<td>Waterfront Hall, Belfast</td>
<td>Professor AAB Barros D’Sa</td>
<td>Mr PM Lamont</td>
<td>Mr PM Lamont</td>
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<tr>
<td>2003</td>
<td>Scottish Exhibition and Conference Centre, Glasgow</td>
<td>Professor KG Burnand</td>
<td>Mr DC Berridge</td>
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<tr>
<td>2004</td>
<td>Harrogate International Centre, Harrogate</td>
<td>Mr PL Harris</td>
<td>Mr RB Galloway</td>
<td>Mr RB Galloway</td>
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<tr>
<td>2005</td>
<td>Bournemouth International Centre, Bournemouth</td>
<td>Professor M Horrocks</td>
<td>Mr RJ Kenyon</td>
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<tr>
<td>2006</td>
<td>Edinburgh International Centre, Edinburgh</td>
<td>Mr JHN Wolfe</td>
<td>Mr JJ Earnshaw</td>
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<tr>
<td>2007</td>
<td>Manchester Central Convention Complex</td>
<td>Professor G Hamilton</td>
<td>Mr RB Galloway</td>
<td>Mr RB Galloway</td>
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<tr>
<td>2008</td>
<td>Bournemouth International Centre, Bournemouth</td>
<td>Mr MJ Gough</td>
<td>Mr SD Parvin</td>
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<td>2009</td>
<td>BT Convention Centre, Liverpool</td>
<td>Mr PR Taylor</td>
<td>Mr RB Galloway</td>
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<td>2010</td>
<td>Hilton Brighton Metropole, Brighton</td>
<td>Professor C Shearman</td>
<td>Mr RB Galloway</td>
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</table>
Presidents

Professor C Shearman
President 2010
Prizes

The Sol Cohen (Founder’s) Prize is for the best clinical paper. The award is a silver salver engraved with the Society’s logo and the year, plus a personal cheque for £500.

The British Journal of Surgery Prize is for the best scientific paper. The award is a cheque for £600 payable to the Research Fund of the Department from which the paper was submitted.

The Venous Forum Prize is presented for the best research paper on venous disease presented at the AGM, and is adjudicated by the Officers of the Venous Forum. The prize is a £250 cheque and a certificate.

The Richard Wood Memorial Prize will be awarded for the best paper presented by a non-doctor in the scientific meeting. The award is an engraved medal, and a cheque for £250.

The Brighton Prize will be awarded for the best paper on the topic of vascular infections. The award is a cheque for £250 and a certificate.

- Vascular trainees are eligible for the Sol Cohen (Founder’s) Prize and the BJS Prize. Both vascular trainees and non-medics are eligible for the Venous Forum and Brighton prizes. The Richard Wood prize is for non-medics only.

- Applicants must be the first author of the abstract, must have made a substantial personal contribution to the work and must deliver the paper in person.

- Vascular trainees must be in a training post on the closing date for submission of abstracts.
List of prize winners

The Sol Cohen (Founder’s) Prize
1992 P Chan, St Mary’s Hospital Medical School, London Abnormal growth regulation of vascular smooth muscle in patients with restenosis
1993 PA Stonebridge, Edinburgh Royal Infirmary Angioscopically identified features related to infrainguinal bypass graft failure
1994 PJ Kent, Mater Misericordiae Hospital, Dublin Prognosis of vibration induced white finger after cessation of occupational vibration exposure
1995 BD Braithwaite, on behalf of the Thrombolysis Study Group Accelerated thrombolysis with high dose bolus t-PA is as safe and effective as low dose infusions - results of a randomised trial
1996 MM Thompson, Leicester Royal Infirmary A comparison of CT and duplex scanning in assessing aortic morphology following endovascular aneurysm repair
1997 IM Loftus, Leicester Royal Infirmary Vein graft aneurysms - conclusive proof of a systemic process
1998 P Renwick, Hull Royal Infirmary Limb outcome following failed femoro-popliteal PTFE bypass for intermittent claudication
1999 ME Gaunt, Leicester Royal Infirmary Intraoperative change in baroreceptor function during carotid endarterectomy
2000 FJ Meyer, St Thomas’s Hospital, London More venous leg ulcers are healed by three-layer paste than by four-layer bandages: a randomised, controlled prospective study
2001 N Lennard, Walsgrave Hospital, Coventry Crescendo TIAs: the use of pre-operative TCD directed IV Dextran therapy to control symptoms and emboli prior to elective carotid endarterectomy
2002 J Barwell, Cheltenham General Hospital, Cheltenham The Eschar Venous Ulcer Study: A randomised controlled trial assessing venous surgery in 500 leg ulcers
2003 R Wilson, St George’s Hospital Medical School, London The suitability of ruptured AAA for endovascular repair
2004 ZA Ali, Addenbrooke’s Hospital, Cambridge Remote ischaemic preconditioning reduces myocardial injury after abdominal aortic aneurysm repair
2005 R Aggarwal, Department of Biosurgery and Surgical Technology, Imperial College London and Regional Vascular Unit, St Mary’s Hospital, London Acquisition of endovascular skills by consultant vascular surgeons: effect of repetition in a virtual reality training model
2006 GS McMahon, University of Leicester, Leicester Low-molecular-weight heparin significantly reduces embolisation after carotid endarterectomy: a randomised controlled trial
2008 Joint winning paper PJE Holt, St George’s Regional Vascular Institute, London Endovascular aneurysm repair independently demonstrates a volume-outcome effect & Regionalisation of vascular surgery improves outcome: a model of service provision
2009 G McMahon, University of Leicester Heparin activates platelet 12-los - transient aspirin resistance explained?

Richard Wood Memorial Prize
2003 EA Nelson, Department of Health Sciences, University of York, York A randomised controlled trial of 4-Layer and short-stretch compression bandages for venous leg ulcers (VenUS I)
2004 S Maxwell, Regional Vascular Unit and the Department of Medical Bacteriology, St Mary’s Hospital, London Methicillin-resistant Staphylococcus aureus (MRSA): are we winning the war against infrainguinal bypass graft infection?
2005 E Horrocks, St Mary’s Hospital, London Carotid endarterectomy under local anaesthetic - evaluating a high fidelity simulated environment for training and assessment
2006 LC Brown, for the EVAR Trial Investigators, Imperial College, London Endovascular, not open repair, should be used in the fittest patients: the application of fitness scoring to EVAR trial patients
2007 P Bourke, Regional Vascular Unit, St Mary’s Hospital, London The proposed 18-week target - is there time for investigations?
2008 C Oakley, Sheffield Hallam University and Vascular Institute, Sheffield Nordic poles immediately improve walking distance in claudicants
2009 R Sharpe, Leicester Royal Infirmary, Leicester Dual antiplatelet therapy prior to carotid endarterectomy reduces postoperative embolisation and thromboembolic events: postoperative transcranial Doppler monitoring is now unnecessary

Brighton Prize
2006 AHR Stewart, Gloucestershire Royal Hospital and Musgrove Park Hospital, Taunton Systemic antibiotics prevent graft and wound infection in peripheral bypass surgery: a systematic review and meta-analysis
2008 RE Clough, Guy’s and St Thomas’ NHS Foundation Trust, London Endovascular management of mycotic aortic aneurysms

SARS Prize
2006 WRW Wilson, University of Leicester, Leicester and St George’s Hospital Medical School, London Decreased cellular telomere content is observed locally and systematically in abdominal aortic aneurysms
2007 TK Ho, Department of Surgery, The Royal Free and University College Medical School, The Royal Free Hospital, London Increased SDF-1 alpha and CXCR4 but not SDF-1 beta expression in human critical limb ischaemia
2009 C Allen, DJA Scott, P James, Leeds Vascular Institute Lymphangiogenesis: novel involvement in abdominal aortic aneurysm

Continued overleaf:
List of prize winners

The British Journal of Surgery Prize
1993 D Higman, Charing Cross Hospital, London Nitric oxide production is impaired in the saphenous vein of smokers
1994 GT Stavri, King’s College School of Medicine and Dentistry, London The role of hypoxia in neo-vascularisation of atherosclerotic plaque
1995 AD Fox, Royal United Hospital, Bath A new modular approach to endoluminal grafting for abdominal aortic aneurysms
1996 C Marshall, University of Newcastle upon Tyne Intravascular adhesion: a new assay to assess neutrophil adhesiveness in whole blood
1997 IM Loftus, Leicester Royal Infirmary Increased proteolytic activity in acute carotid plaques - therapeutic avenues to prevent stroke
1998 J Franklin, Charing Cross Hospital, London Non-steroidal anti-inflammatory drugs to treat abdominal aortic aneurysms
1999 DW Harkin, Royal Victoria Hospital, Belfast In major limb vessel trauma reperfusion injury is increased by delayed venous reflow and prevented by anti-oxidant pretreatment
2000 DW Harkin, Royal Victoria Hospital, Belfast Ischaemic preconditioning (IPC) prior to lower limb ischaemia reperfusion protects against acute lung injury
2001 SL Drinkwater, St Thomas’ Hospital, London Venous ulcer exudates inhibit in vitro angiogenesis
2002 M Griffiths, Royal Free Hospital, London Nicotine abolishes the hypoxic induction of VEGF in human microvascular endothelial cells
2003 DR Lewis, The Royal North Shore Hospital, University of Sydney, New South Wales, Australia Point of care testing of aspirin resistance in patients with vascular disease
2004 V Vijayan, Bristol Royal Infirmary The early and long term reduction of porcine saphenous vein graft thickening using a biodegradable polyglactin external sheath
2005 C Ruiz, Peripheral Vascular Unit, Glasgow Royal Infirmary Pre-operative ischaemia of the long saphenous vein predisposes to intimal hyperplasia in bypass grafts through enhanced smooth muscle cell migration
2006 MJ Bown, University of Leicester, Leicester The IL-10-1082 ‘A’ allele and abdominal aortic aneurysm
2007 A Thompson, Cardiovascular Genetics Departments, University College London, and the Vascular Department, Royal West Sussex NHS Trust, Chichester TGf3 and LTBP4 are associated with altered AAA growth: a candidate gene study
2009 R Aggarwal, Division of Cardiovascular and Diabetes Research, Leeds Aspirin and clot structure in patients with Abdominal Aortic Aneurysm (AAA): A mechanism for reduced AAA expansion?

Venous Forum Prize
2001 I Singh, St Thomas’s Hospital, London Inhibition of experimental venous thrombosis with a human anti-factor VIII monoclonal antibody
2002 J Barwell, Cheltenham General Hospital, Cheltenham The Eschar Venous Ulcer Study: A randomised controlled trial assessing venous ulcers in 500 leg ulcers
2003 EA Nelson, Department of Health Sciences, University of York, York A randomised controlled trial of 4-Layer and short-stretch compression bandages for venous leg ulcers (VenUS I)
2005 RJ Winterborn, Gloucestershire Royal Hospital, Gloucester Late results of a randomised controlled trial of stripping the long saphenous vein
2006 B Kianifard, Royal Surrey County Hospital, Guildford Perforator veins do not remain closed following long saphenous vein stripping - results of a randomised trial with a one year follow up
2007 RJ Winterborn, Department of Vascular Surgery, Gloucestershire Royal Hospital Prospective study of short saphenous varicose vein surgery: six weeks’ results
2008 R Eftek, Department of Surgery, Queen Elizabeth Hospital, Gateshead and Northern Vascular Centre, Freeman Hospital, Newcastle upon Tyne Quantitative measurement of superficial venous surgery using continuous ambulatory venous pressure measurement (CAVPM)
2008 R Winterborn, Gloucestershire Royal Hospital No advantage in performing flush saphenofemoral ligation: results of a randomised trial
2009 D Carradice, Academic Vascular Surgical Unit, Hull A randomised trial of EVLT vs. surgery for varicose veins
2009 A Shepherd, Imperial Vascular Unit, Imperial College, Charing Cross Hospital, London Early results of a randomised clinical trial (RCT) comparing VNUS ClosureFast Ablation and Laser for Varicose Veins (VALVV)

Best Video
2007 R Bulbulia, M Whyman, L Emerson, L Visser, F Slim and K Poskitt, Cheltenham General Hospital Laparoscopic aortic aneurysm repair

Best Educational/Training Video
2007 J Tsui, R De Souza, G Hamilton, Royal Free Hospital, London Carotid endarterectomy: retro-jugular approach and eversion technique

Best Poster
2007 G Atturu, S Brouillet, M Bown, NJ Samani, NJM London, R Sayers, University of Leicester, Leicester Leucocyte telomere length is reduced in patients with abdominal aortic aneurysm

Yearbook 2010
## John Kinmonth Memorial Lectureship

*Founded* in 1983 utilising a gift made in his lifetime by Professor John Bernard Kinmonth FRCS (Council 1977-82), and donations made in his memory. A bronze medal bearing the arms of the College on one side and a portrait head of John Kinmonth on the other, and engraved with the Lecturer’s name and the year in which the lecture is delivered, is presented on each occasion.

### Conditions
An annual lecture on a vascular topic. A nomination is solicited from the President of The Vascular Society and goes before Council for approval. The lecture is usually delivered at the annual meeting of the Society.

### Previous Lecturers

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<tr>
<th>Year</th>
<th>Lecturer</th>
<th>Title of Lecture</th>
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<tbody>
<tr>
<td>1983</td>
<td>Professor Graham Douglas Tracy</td>
<td>“Choosing a treatment plan for patients with leg ischaemia.”</td>
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<tr>
<td>1984</td>
<td>Mr Roger Neale Baird</td>
<td>“Recognition of carotid artery disease.”</td>
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<td>1985</td>
<td>Mr Adrian Marston</td>
<td>“The gut and its blood-supply.”</td>
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<td>1986</td>
<td>Professor Sir Peter Morris</td>
<td>“Whither carotid endarterectomy.”</td>
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<td>1987</td>
<td>Professor John E Connolly</td>
<td>“Can paraplegia in aortic surgery be prevented?”</td>
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<td>1988</td>
<td>Dr Thomas F O’Donnell</td>
<td>“Management of the high risk abdominal aortic aneurysm”</td>
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<tr>
<td>1989</td>
<td>Professor Averil O Mansfield</td>
<td>“An artery and a vein dancing - the management of arteriovenous malformation”</td>
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<td>1990</td>
<td>Mr CW Jamieson</td>
<td>“Dilemmas in improving vascular surgical services”</td>
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<td>1991</td>
<td>Professor Norman Browse</td>
<td>“The lymphatics”</td>
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<td>1992</td>
<td>Professor Alexander Clowes</td>
<td>“Vascular biology - the new frontier”</td>
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<td>1993</td>
<td>Dr Ray Gosling</td>
<td>“The mechanics of atherosclerosis”</td>
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<td>1994</td>
<td>Dr Hero van Urk</td>
<td>“Future development in endoluminal vascular surgery”</td>
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<td>1995</td>
<td>Dr Timothy Chuter</td>
<td>“Clinical experience of stenting aneurysms”</td>
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<td>1996</td>
<td>Dr Jerry Goldstone</td>
<td>“Vascular surgery: training, certification and practice; observations from the USA”</td>
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<td>1997</td>
<td>Mr Alan Scott</td>
<td>“Screening and the management of abdominal aortic aneurysms - the missing links”</td>
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<td>1998</td>
<td>Mr Peter Harris</td>
<td>“Vascular surgery: the European perspective”</td>
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<tr>
<td>1999</td>
<td>Mr Simon G Darke</td>
<td>“Optimal management of venous ulceration: an enigma slowly unfolding”</td>
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<tr>
<td>2000</td>
<td>Professor Janet Powell</td>
<td>“The good, the bad and the ugly - a tale of aneurysms”</td>
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<td>2001</td>
<td>Mr Jonothan Earnshaw</td>
<td>“Audit of clinical outcomes in vascular surgery: a shield for our profession”</td>
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<tr>
<td>2002</td>
<td>Professor David Bergqvist</td>
<td>“Management of iatrogenic vascular injuries”</td>
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<tr>
<td>2003</td>
<td>Professor Reginald Lord</td>
<td>“Carotid disease: the burden of proof”</td>
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<td>2004</td>
<td>Professor Roger Greenhalgh</td>
<td>“The impact of vascular clinical trials on clinical practice”</td>
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<td>2005</td>
<td>Mr John Wolfe</td>
<td>“Operative vascular training and assessment: the last century, the present and the future”</td>
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<tr>
<td>2006</td>
<td>Mr Peter Taylor</td>
<td>“Achieving the Impossible”</td>
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<td>2007</td>
<td>Professor Kevin Burnand</td>
<td>“Research in vascular diseases: achievements and unsolved problems”</td>
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<td>2008</td>
<td>Professor Shervanthi Homer-Vanniasinkam</td>
<td>“Translational vascular research: the road less travelled”</td>
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<tr>
<td>2009</td>
<td>Professor Roy Greenberg</td>
<td>“Perspectives on the future of vascular surgery and aortic interventions”</td>
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# Programme

**24-26 November 2010 Hilton Brighton Metropole, Brighton**

**WEDNESDAY 24TH NOVEMBER**

<table>
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<tr>
<th>Time</th>
<th>Session</th>
<th>Speaker(s)</th>
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| 9.00am-12noon | **VENOUS FORUM**  
**Oxford Suite**  
Chair: Professor Alun Davies, President, Venous Forum |  
9.00-9.10am | Epidemiology and aetiology of C4, C5, C6 disease | Professor Andrew Bradbury, Birmingham  
9.10-9.20am | What is PTS and its management? | Professor Gerard Stansby, Newcastle  
9.20-9.30am | Management of DVT to reduce the incidence of PTS | Professor Gordon Lowe, Glasgow  
9.30-9.40am | Investigation of the patient with a venous ulcer | Mr David Berridge, Leeds  
9.40-9.50am | Compression and dressings in C4, C5, C6 disease | Ms Andrea Nelson, Leeds  
9.50-10.00am | Pharmacological treatment in C4, C5, C6 disease | Mr Manj Gohel, London  
10.00-10.10am | Leg ulceration: the importance of treating the underlying pathophysiology | Mr Isaac Nyamekye, Worcester  
10.10-10.20am | Lymphoedema | Dr Hakan Brorson, Malmo, Sweden  
10.20-10.55am | **COFFEE** |  
10.55-11.05am | Societal costs and C4, C5, C6 disease | Professor Eberhard Rabe, Bonn, Germany  
11.05-11.15am | The need for a national strategic framework for leg ulceration | Mr Richard Bulbulia, Cheltenham  
11.15-11.25am | **PANEL DISCUSSION** |  
11.25-11.40am | **DEBATE: Should acute DVT be managed by vascular surgeons?**  
Chair: Mr David Berridge, Honorary Secretary, Venous Forum |  
Pre-vote | (Show of hands) |  
For: Mr Jonathan Earnshaw, Gloucester  
Against: Professor Mike Laffan, London |  
Post-vote | (Show of hands) |  
11.40-11.50am | Who should get VV treatment on the NHS? | Ms Sophie Renton, London  
11.50-12noon | **DISCUSSION** |
9.00am-12noon
EDUCATIONAL MASTERCLASS: RARE VASCULAR DISORDERS
BALMORAL SUITE

Interactive workstations on:

- **Carotid body tumours, aneurysms and trauma**
  - Professor Ross Naylor, Leicester

- **Thoracic outlet compression syndrome**
  - Mr John Thompson, Exeter

- **Vasospastic disorders and vasculitis**
  - Professor Andre van Rij, Dunedin, New Zealand

- **Visceral ischaemia and aneurysms**
  - Mr Frank Smith, Bristol

- **Iliac artery endofibrosis and fibromuscular dysplasia**
  - Mr Rob Hinchliffe, London

- **Popliteal artery aneurysms, entrapment and CAD**
  - Mr Shane MacSweeney, Nottingham

- **Iliofemoral thrombosis, phlegmasia and caval filters**
  - Professor Anthony Watkinson, Exeter

- **Pelvic congestion syndrome and related varices**
  - Professor Charles McCollum, Manchester

9.00am-12noon
SOCIETY OF ACADEMIC AND RESEARCH SURGERY
BUCKINGHAM SUITE

**Chairs:** Professor Cliff Shearman, President, The Vascular Society
Professor Rob Sayers, Leicester

9.00-9.10am
Restenosis post-angioplasty is associated with increased smooth muscle cell proliferation in patients with intermittent claudication
AM Wilson 1, 2, P Bachoo 2, I Ford 1, GF Nixon 1, J Brittenden 1, 2
1 University of Aberdeen, Aberdeen; 2 Department of Vascular Surgery, Aberdeen Royal Infirmary, Aberdeen

9.10-9.20am
Activation of hypoxia-inducible factor (HIF) pathway in varicose veins
CS Lim 1, 2, S Kiriakidis 2, A Sandison 3, EM Paleolog 2, AH Davies 1
1 Imperial Vascular Unit, Department of Surgery and Cancer, Faculty of Medicine, Imperial College London, London; 2 Cytokine Biology of Vessels, Kennedy Institute of Rheumatology and Department of Surgery and Cancer, Imperial College London, London; 3 Department of Histopathology, Charing Cross Hospital, Imperial College Healthcare NHS Trust, London

9.20-9.30am
Down-regulation of hypoxia-inducible factor 1α reduces venous thrombus resolution
CE Evans, J Humphries, K Mattock, M Waltham, A Wadoodi, P Saha, B Modarai, A Smith
Academic Department of Surgery, King’s College London, London

9.30-9.40am
The angiogenic potential of Tie2-expressing monocytes is impaired in patients with critical limb ischaemia
AS Patel, A Smith, P Saha, K Mattock, J Humphries, R Siow, M Waltham, B Modarai
King’s College London BHF Centre of Excellence, Academic Department of Surgery, Cardiovascular Division; The NIHR Biomedical Research Centre at Guy’s and St Thomas’ NHS Foundation Trust and King’s College London, London
9.40-9.50am  Advancing what we know about genetics and varicose veins  
J Krysa, G Jones, A van Rij  
Department of Surgery, Dunedin School of Medicine, University of Otago, Dunedin, New Zealand

9.50-10.00am  The effect of novel texture features of homogeneity and echolucency on carotid plaque characterization; results from the ACSRS study  
GC Makris 1, M Griffin 2, G Geroulakos 1, AN Nicolaides 3  
1 Imperial College of Science and Technology, London, Department of Vascular Surgery, Ealing Hospital NHS Trust, London; 2 Cardiovascular Disease Education and Research (CDER) Trust; 3 Imperial College of Science and Technology, London

10.00-10.10am  Helix-B peptide of erythropoietin could be used as pharmacotherapy in critical limb ischaemia  
D Joshi 1, J Tsui 1, X Shiwen 2, H Patel 1, S Selvakumar 1, D Lawrence 3, D Abraham 2, D Baker 1  
1 Vascular Unit, UCL Department of Surgery, Royal Free Hospital, London; 2 Centre for Rheumatology & Connective Tissue Disease, UCL, London; 3 Heart Hospital, University College London Hospitals, London

10.10-10.20am  Peak oxygen consumption is a useful biomarker in assessing survival after abdominal aortic aneurysm repair  
A Kordowicz 1, S Sohrabi 1, 2, M Bailey 1, K Griffin 2, T Rashid 2, K Foster 2, S Howell 3, DJA Scott 1, 2  
1 Leeds Vascular Institute, The General Infirmary at Leeds, Leeds; 2 Division of Cardiovascular and Diabetes Research, LIGHT Laboratories, University of Leeds, Leeds; 3 Academic Unit of Anaesthesia, The General Infirmary at Leeds, Leeds

10.20-10.50am  COFFEE

10.50-11.00am  Integrin-α9-fibronectin interaction is required for normal murine venous valve morphogenesis  
OTA Lyons 1, 2, E Bazigou 3, S Jeffery 4, A Smith 2, E Makinen 3, NA Brown 1  
1 Basic Medical Sciences at St George’s, University of London, London; 2 Department of Vascular Surgery, National Institute for Health Research Comprehensive Biomedical Research Centre at Guy’s and St Thomas’ NHS Foundation Trust and King’s College London, London; 3 Cancer Research UK’s London Research Institute; 4 Human Genetics at St George’s, University of London, London

11.00-11.10am  Intra-plaque production of M1-type cytokines and matrix metalloproteinases differentiate stable from unstable carotid atherosclerosis  
J Shalhoub 1, A Cross 2, DM Allin 1, IJ Franklin 1, C Monaco 2, AH Davies 1  
1 Imperial Vascular Unit, Imperial College London, Charing Cross Hospital, London; 2 Cytokine Biology of Atherosclerosis Group, Kennedy Institute of Rheumatology, Imperial College London, London
11.10-11.20am The potential therapeutic role of toll-like receptor 2 in critical limb ischaemia
H Patel 1, D Joshi 1, D Lawrence 2, X Shi-Wen 3, D Baker 1, S Shaw 4, J Tsui 1
1 Royal Free Vascular Unit, University College London, London; 2 Heart Hospital, University College London, London; 3 Centre for Rheumatology & Connective Tissue Disease, University College London, London; 4 Department of Clinical Research, University of Bern, Switzerland

11.20-11.30am Clinical cell tracking of mononuclear cells using magnetic resonance imaging and superparamagnetic particles of iron oxide
JMJ Richards 1, 2, KA Shaw 1, NN Lang 1, SK Semple 3, JA Crawford 4, M Williams 1, A Atkinson 5, E Forrest 5, NL Mills 1, A Burbess 1, 2, K Dhaliwal 6, AJ Simpson 6, H Roodie 5, G McKillop 7, TM Connolly 8, GZ Feuerstein 8, RH Barclay 4, 5, M Turner 4, 5, DE Newby 1, 3
1 Centre for Cardiovascular Science, University of Edinburgh, Edinburgh; 2 Centre of Clinical and Surgical Sciences (Surgery), University of Edinburgh, Edinburgh; 3 Clinical Research Imaging Centre, University of Edinburgh, Edinburgh; 4 Centre for Regenerative Medicine, University of Edinburgh, Edinburgh; 5 Scottish National Blood Transfusion Service; 6 Centre for Inflammation Research, University of Edinburgh, Edinburgh; 7 Department of Radiology, Royal Infirmary of Edinburgh, Edinburgh; 8 Wyeth Research

11.30-12noon LECTURE - THE GENETIC BASIS OF AAA GROWTH AND DEVELOPMENT
Mr Matt Bown, Leicester

9.00am-4.00pm
SOCIETY OF VASCULAR NURSES ANNUAL MEETING
CLARENCE SUITE

12noon-1.00pm Lunch and viewing of trade exhibition
EXHIBITION HALL

THE VASCULAR SOCIETY MEETING
OXFORD SUITE

1.00pm President’s welcome
Presentation of President’s Early Career Award

1.05-2.30pm The next decade - the future for the new specialty
Chair: Professor Cliff Shearman, President

Opening address
By Professor Sir Bruce Keogh, NHS Medical Director

The role of consultants in the NHS of the next decade
Professor Hugo Mascie Taylor, Medical Director, NHS Confederation

The future of surgical training
Professor Sir John Temple, Independent Chair of ‘Time for Training’

Vascular services of the future
Professor Nick Cheshire, Consultant Vascular Surgeon, London

Vascular surgeons of the future: who will they work for?
Mr Mark Goldman, Formerly Chief Executive of Heart of England NHS Trust and Consultant Vascular Surgeon
2.30-3.00pm  Tea

3.00-4.30pm  SCIENTIFIC MEETING: BJS PRIZE SESSION

Chairs:  Professor Ross Naylor, Vice-President Elect
         Professor Julian Scott, Leeds

3.00-3.15pm  Magnetic resonance T1 mapping predicts successful venous thrombolysis

P Saha 1, M Andia 2, U Blume 2, A Wiethoff 2, T Schaeffter 2, C Evans 1, AS Patel 1, A Ahmad 1, B Modarai 1, A 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

3.15-3.30pm  Uptake of ultrasmall superparamagnetic particles of iron oxide predicts growth in abdominal aortic aneurysms

JMJ Richards 1, 2, SL Semple 3, TJ MacGillivray 3, C Gray 3, JP Langrish 1, M Williams 1, M Dweck 1, W Wallace 4, G McKillop 5, RTA Chalmers 2, OJ Garden 2, DE Newby 1, 3

1 Centre for Cardiovascular Science, University of Edinburgh; 2 Centre of Clinical and Surgical Sciences (Surgery), University of Edinburgh; 3 Clinical Research Imaging Centre, University of Edinburgh, Edinburgh; 4 Department of Pathology, Royal Infirmary of Edinburgh, Edinburgh; 5 Department of Radiology, Royal Infirmary of Edinburgh, Edinburgh

3.30-3.45pm  The effect of n-3 long chain polyunsaturated fatty acid (n-3LCPUFA) supplementation on platelet and endothelial function in patients with peripheral arterial disease

I Mckay 1, F Thies 2, I Ford 2, S Fielding 2, P Bachoo 1, J Brittenden 1, 2

1 Aberdeen Royal Infirmary, Aberdeen; 2 University of Aberdeen, Aberdeen

3.45-4.00pm  Engineering a novel Ang1 mimetic for regenerative medicine applications

E Issa, AJM Moss, NJM London, NPJ Brindle

Vascular Surgery Group, University of Leicester, Leicester

4.00-4.15pm  Proteomic evidence of impaired resistance to oxidative stress and inflammation in the vasculature of patients with abdominal aortic aneurysms

I Nordon, R Hinchcliffe, G Pirianov, E Torsney, I Loftus, G Cockerill, M Thompson

St George’s Vascular Institute, London
4.15-4.30pm  Imaging of the vulnerable carotid plaque: biological targeting of inflammation using ultrasmall superparamagnetic particles of iron oxide (USPIO) and MRI
J Chan 1, C Monaco 2, K Bhakoo 3, RGJ Gibbs 1
1 Vascular Surgery Unit, St Mary's Hospital, Imperial College Healthcare NHS Trust, London; 2 Cytokine Biology of Atherosclerosis, Kennedy Institute of Rheumatology, Imperial College, London; 3 Translational Molecular Imaging Group, Singapore Bioimaging Consortium, Agency for Science, Technology and Research (A*STAR), Singapore

4.30-5.30pm  THE VASCULAR SOCIETY AND THE ROULEAUX CLUB: JOINT SYMPOSIUM
HOW TO TRAIN VASCULAR SPECIALISTS OF THE FUTURE
Chairs:  Professor Jonathan Beard, Chair, Training and Education Committee
Mr Jeremy Crane, President, Rouleaux Club

Training vascular specialists for the future  Dr David Kessel, President, BSIR
Simulators in surgical training  Professor Nick Cheshire, London
The invisibles  Mrs Linda de Cossart, Gloucester
How long does it take to train a surgeon?  Professor John Collins, Melbourne, Australia, Visiting Professor, University of Oxford

5.30-6.30pm  SCIENTIFIC MEETING: SESSION 2
Chairs:  Mr Peter Lamont, VS President Elect
Mr John Brennan, BSET President

5.30-5.40pm  ‘Mini-sternotomy’: a novel access for endografting of descending thoracic aortic aneurysms
I Ahmed 1, A Gamal 2, A Refaat 2, K El Sakka 1, M El Dessoki 2
1 Brighton & Sussex University Hospitals, Brighton; 2 Cairo University Hospitals, Egypt

5.40-5.50pm  Medium-term outcomes of emergency EVAR for ruptured AAA
A Noorani, SR Walsh, U Sadat, A Page, K Varty, PD Hayes, JR Boyle
Cambridge Vascular Unit, Addenbrookes Hospital, Cambridge

5.50-6.00pm  The effect of mismatch between native anatomy of visceral aorta and design of fenestrated stent-grafts
OA Oshin 1, TV How 2, JA Brennan 1, RK Fisher 1, RG McWilliams 3, SR Vallabhpaneni 1
1 Regional Vascular Unit, Royal Liverpool University Hospital, Liverpool; 2 Division of Clinical Engineering, University of Liverpool, Liverpool; 3 Department of Radiology, Royal Liverpool University Hospital, Liverpool

6.00-6.10pm  Reducing the risk of spinal cord ischaemia following endovascular repair of thoracoabdominal aneurysms - the ‘sac perfusion branch’
SC Harrison, J Raja, J Hague, O Agu, T Richards, K Ivancev, PL Harris
Multidisciplinary Endovascular Team, University College Hospital, London
6.10-6.20pm  Stent-graft limbs deployed into the external iliac artery are at increased risk of occlusion following EVAR
B Modarai, P Taylor, R Clough, A Patel, P Saha, S Thomas, M Waltham, T Carrell, R Salter, H Zayed, R Bell
Vascular Unit, King’s Health Partners, London

6.20-6.30pm  Endovascular repair of abdominal aortic aneurysms (AAA) with short and/or angulated necks: infra-renal sealing is not a safe option
J Cross, D Simring, J Raja, J Hague, O Agu, K Ivancev, P Harris, T Richards
Multidisciplinary Endovascular Team, University College Hospital, London

6.30-7.15pm  WELCOME DRINKS RECEPTION  EXHIBITION HALL: UPPER GALLERY

6.30-7.30pm  SYMPOSIA  CLARENCE SUITE
THE MANAGEMENT OF BLEEDING AND ITS IMPACT ON OUTCOMES IN VASCULAR SURGERY - SPONSORED BY ETHICON SURGICAL
Mr Rod Chalmers, Consultant Vascular Surgeon, Edinburgh Royal Infirmary

THURSDAY 25TH NOVEMBER

7.00-8.00am  BREAKFAST SYMPOSIA  CURRENT MEDICAL TREATMENT FOR PAD  CLARENCE SUITE
Chair: Ms Julie Brittenden, Aberdeen

Which PAD patients should receive aspirin?  Professor Gerry Fowkes, Glasgow
LDL-cholesterol: how low should you go?  Mr Richard Bulbulia, Cheltenham
Drug treatments for managing PAD symptoms  Professor Gerry Stansby, Newcastle

9.00am-5.00pm  SOCIETY FOR VASCULAR TECHNOLOGY ANNUAL MEETING  CLARENCE SUITE

8.30-9.30am  SCIENTIFIC MEETING: SESSION 3  OXFORD SUITE
RW = Eligible for Richard Wood Prize

Chairs:  Mr Paul Blair, Belfast
         Mr Ian Loftus, London

8.30-8.40am  Improving standards of care in AAA surgery: the patients’ perspective RW
R Potgieter 1, JV Smyth 2, DJA Scott 3, P Barker 4, G Stansby 5, S Hill 6, P Bachoo 7, D Mitchell 1
1 AAA QIP Team, North Bristol NHS Trust, Bristol; 2 Manchester Royal Infirmary, Manchester; 3 Leeds Vascular Institute, Leeds; 4 Patient Representative, VSGBI; 5 Freeman Hospital, Newcastle-upon-Tyne; 6 University Hospital of Wales, Cardiff; 7 Aberdeen Royal Infirmary, Aberdeen
8.40-8.50am Use of baseline factors to predict serious complications and re-interventions after endovascular aneurysm repair (EVAR) in patients with a large abdominal aortic aneurysm: results from the UK EVAR trials RW
LC Brown 1, RM Greenhalgh 1, JT Powell 1, SG Thompson 2, on behalf of the EVAR trial participants
1 Imperial College London, London; 2 Medical Research Council Biostatistics Unit

8.50-9.00am Acute aortic syndrome (AAS) treated by thoracic endovascular aortic repair (TEVAR)
RE Clough, B Modarai, OT Lyons, S Key, S Thomas, RE Bell, TW Carrell, M Waltham, HA Zayed, PR Taylor
NIHR Comprehensive Biomedical Research Centre of Guy’s and St Thomas’ NHS Foundation Trust and King’s College London, London

9.00-9.10am Use of CO₂ angiography for fenestrated endovascular aneurysm repair
J Cross, D Simring, O Agu, J Raja, J Hague, K Ivancev, T Richards, P Harris
Multidisciplinary Endovascular Team, University College Hospital, London

9.10-9.20am Radiation exposure during endovascular treatment of the aorta: increased risk with complex repairs
P Howells 1, R Eaton 2, R Dourado 1, S Black 1, H Zayed 1, R Bell 1, M Waltham 1, T Carrell 1, P Taylor 1, B Modarai 1
1 Vascular Unit, Guy’s and St Thomas’ Hospitals, King’s Health Partners, London; 2 Medical Physics Department, Guy’s & St Thomas’ NHS Foundation Trust, London

9.20-9.30am Assessment of National Vascular Database (NVD) quality RW
PD Baxter 1, TJ Fleming 1, RM West 1, MS Gilthorpe 1, TA Lees 2, DC Mitchell 3, DJA Scott 4
1 Division of Biostatistics, University of Leeds, Leeds; 2 Freeman Hospital, Newcastle-upon-Tyne; 3 Southmead Hospital, Bristol; 4 Division of Cardiovascular and Diabetes Research, University of Leeds, Leeds

9.30-10.30am SCIENTIFIC MEETING: SESSION 4
RW = Eligible for Richard Wood Prize; VF = Eligible for Venous Forum Prize

Chairs: Mr Ian Franklin, London
Mr Isaac Nyamekye, Worcestershire

9.30-9.40am Modifying the illness and treatment beliefs of patients with intermittent claudication increases daily walking and reduces demand for vascular intervention - results from a randomised controlled trial RW
M Cunningham 1, V Swanson 1, R O’Carroll 1, R Holdsworth 2
1 University of Stirling, Stirling; 2 NHS Forth Valley, Stirling

9.40-9.50am Modelling the effect of venous disease upon quality of life VF
D Carradice, FAK Mazari, N Samuel, AI Mekako, J Hatfield, IC Chetter
Academic Vascular Surgical Unit, University of Hull / Hull York Medical School, Hull
9.50-10.00am  A prospective double-blind randomised controlled trial of radiofrequency versus laser treatment of great saphenous varicose veins  
I Nordon, R Hinchcliffe, R Brar, P Moxey, S Black, M Thompson, I Loftus  
St George's Vascular Institute, London  
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10.00-10.10am  Combined medical therapy and carotid endarterectomy for asymptomatic stenosis: 10-year stroke prevention in ACST-1  
A Halliday, on behalf of the ACST collaborators  
Nuffield Department of Surgery, John Radcliffe Hospital, Oxford  
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10.10-10.20am  Clinical assessment pre-CABG identifies patients at risk for postoperative stroke  
PM Bevis 1, GD Nicholls 2, A Watson 1, V Vijayan 1, PM Lamont 1, FCT Smith 1, MJ Brooks 1  
1 Vascular Unit, University Hospitals Bristol NHS Foundation Trust, Bristol; 2 University of Bristol, Bristol  
page 68

10.20-10.30am  The burden of carotid endarterectomy (CE) complications - underneath stroke and death  
A Jayasekera 1, D Hargroves 2, H Baht 1, G Gunaratnam 3, I Burger 1, H Thambawita 1, R Insall 1,2, J Senaratne 1,3  
1 Kent & Canterbury Hospital, Canterbury; 2 William Harvey Hospital, Ashford; 3 QEQM Hospital, Margate  
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10.30-11.00am  COFFEE  
EXHIBITION HALL

11.00am-12.30pm  DIABETES - THE COMING STORM OF DIABETIC VASCULAR DISEASE  
Chairs: Dr George Andros, Los Angeles  
Professor Gerry Stansby, Newcastle

The extent of the problem  
Professor Andrew Boulton, Manchester

Charcot neuroarthropathy  
Professor William Jeffcoate, Nottingham

How to provide a vascular service for people with diabetes  
Dr Gerry Rayman, Ipswich

The foot - whose problem is it?  
Ms Louise Stuart, Manchester

Surgical revascularisation of the limb  
Dr David Campbell, Harvard, USA

The role of endovascular revascularisation in diabetes  
Professor Jim Reekers, Holland

12.30-1.30pm  Lunch  
EXHIBITION HALL
1.30-3.00pm  SCIENTIFIC MEETING: SOL COHEN (FOUNDER’S) PRIZE SESSION
OXFORD SUITE

Chairs:  Professor Cliff Shearman, President
         Professor Shervanthi Homer-Vanniasinkam, Chair, VS Research Committee

1.30-1.45pm  Rate and predictability of graft rupture after endovascular and open abdominal aortic aneurysm repair: data from the EVAR trials
TR Wyss, LC Brown, JT Powell, RM Greenhalgh, on behalf of the EVAR trial participants
Vascular Surgery Research Group, Imperial College London, London

1.45-2.00pm  A prospective study of the natural history of deep vein thrombosis: early predictors of poor late outcomes
J Krysa, G Hill, R Dickson, A van Rij
Department of Surgery, Dunedin School of Medicine, University of Otago, Dunedin, New Zealand

2.00-2.15pm  Flow-sensitised dynamic magnetic resonance imaging (MRI) can identify dominant false lumen flow and secondary entry tears in type B aortic dissection: implications for endovascular treatment
RE Clough, TW Carrell, S Uribe, M Waltham, T Schaeffter, PR Taylor
NIHR Comprehensive Biomedical Research Centre of Guy’s and St Thomas’ NHS Foundation Trust and King’s College London, London

2.15-2.30pm  Further evidence for the role of patch angioplasty (PA) over primary closure (PC) during carotid endarterectomy (CEA)
MW Twigg 1, R Maiti 1, S Lewis 2, MJ Gough 1, on behalf of the GALA trial collaborators
1 Leeds Vascular Institute, The General Infirmary at Leeds, Leeds; 2 University of Edinburgh, Edinburgh

2.30-2.45pm  Distal bypass grafts in patients with critical leg ischaemia with poor pedal arch
H Slim 1, A Tiwari 1, A Ali 1, JC Ritter 1, M Edmonds 2, H Zayed 3, H Rashid 1
1 Vascular Surgery Department, King’s College Hospital, London; 2 General Medicine Department, King’s College Hospital, London; 3 Vascular Surgery Department, St Thomas’ Hospital, London

2.45-3.00pm  The risk of occlusion and associated events in the Asymptomatic Carotid Surgery Trial: a 10-year prospective study
AG den Hartog 1, L Hirt 2, E Hayter 2, FL Moll 1, A Halliday 3, GJ de Borst 1, on behalf of the ACST-1 collaborators
1 University Medical Centre Utrecht, Utrecht, The Netherlands; 2 St. George’s Hospital Medical School, London; 3 NDS John Radcliffe Hospital, Oxford
3.00-3.30pm  Tea  EXHIBITION HALL

3.30-4.30pm  THE FUTURE - IMPROVING OUTCOMES IN VASCULAR SURGERY  
             OXFORD SUITE

Chairs:  Mr Jonathan Earnshaw, Honorary Secretary  
         Professor Rob Sayers, Leicester

Volume-related outcome  Professor Matt Thompson, London

Do quality improvement programmes improve results?  Mr David Mitchell, Bristol

Can guidelines improve outcomes?  Professor Bruce Campbell, Exeter

Patient selection  Professor Anthony Cunningham, Dublin

4.30-5.30pm  ANNUAL BUSINESS MEETING

5.00-6.00pm  ROULEAUX CLUB AGM  CLARENCE SUITE

7.30 for 8.00pm  ANNUAL SOCIETY DINNER  OXFORD SUITE  
                 to include Prize Presentations

FRIDAY 26TH NOVEMBER

8.00-9.30am  SCIENTIFIC MEETING: SESSION 5  OXFORD SUITE

Chairs:  Mr Daryll Baker, London  
         Mr Shane MacSweeney, Nottingham

8.00-8.10am  Laparoscopic aortic aneurysm surgery: early experience from three UK vascular centres  
             AQ Howard 1, CM Backhouse 1, AC Gordon 2, L Visser 3, RA Bulbulia 3, MR Whyman 3, KR Poskitt 3  
             1 Colchester General Hospital, Colchester; 2 Wexham Park Hospital, Slough; 3 Cheltenham General Hospital, Cheltenham

8.10-8.20am  Renal function in patients following open repair of Type IV thoracoabdominal aneurysms: long-term results  
             UI De Silva, S Thwaites, AL Tambyraja, AF Nimmo, C Moores, PJ Burns, RTA Chalmers  
             Vascular Surgery Unit, Royal Infirmary of Edinburgh, Edinburgh

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8.20-8.30am  Assessment of scoring for high-risk patients undergoing endovascular aneurysm repair
O Ehsan, AN Hopper, L Price, R Thomas, I Williams
University Hospital of Wales, Cardiff

8.30-8.40am  A pre-operative model for predicting mortality risk in elective AAA surgery
SW Grant 1, AD Grayson 2, D Purkayastha 1, CN McCollum 1, on behalf of the VGNW participants
1 Department of Academic Surgery, University Hospital of South Manchester, Manchester; 2 Southport & Ormskirk NHS Hospitals, Southport

8.40-8.50am  Aortic aneurysm repair in octogenarians
SW Grant 1, S Brookes-Fazakerley 2, AD Grayson 3, CN McCollum 1, on behalf of the VGNW participants
1 Department of Academic Surgery, University Hospital of South Manchester, Manchester; 2 University Hospital of South Manchester, Manchester; 3 Southport & Ormskirk NHS Hospitals, Southport

8.50-9.00am  Growth rate of very small aneurysms
SD Parvin
Royal Bournemouth Hospital, Bournemouth

9.00-9.10am  Early experience of the UK aneurysm screening programme
AM Conway, AH Malkawi, RJ Hinchliffe, D Rikhi, MM Thompson, IM Loftus
St George’s Vascular Institute, St George’s Healthcare NHS Trust, London

9.10-9.20am  Aneurysm screening results in North London - a world away from the MASS trial
C Forman, H Sales, O Trainor, G Hamilton, M Davis
Royal Free Hospital, London

9.20-9.30am  Prevalence of screen-detected AAAs in men aged 65 is decreasing; however, the prevalence of cardiac and respiratory diseases remains significantly higher in this group
S Penkar, S Druce, H Ashton, H Hafez
St Richard’s Hospital, Chichester
9.30am-10.30am
SCIENTIFIC MEETING: SESSION 6

Chairs: Mr David Mitchell, Bristol
        Mr Richard Holdsworth, Stirling

9.30-9.40am Establishing a volume-outcome relationship in lower limb bypass surgery using multi-level logistic regression modelling
        P Moxey 1, D Hofman 2, R Hinchliffe 3, K Jones 3, I Loftus 3, M Thompson 3, P Holt 1
        1 Department of Outcomes Research, St George's Vascular Institute, London; 2 Department of Outcomes Research, St George's University of London, London; 3 St George's Vascular Institute, London

9.40-9.50am Outcome in the management of acute diabetic limb emergencies - a revolving door?
        YMT Hui 1, T Ali 2, DJ Gerrard 2, PW Leopold 2, A Wee 1, EM Bingham 1, PFS Chong 2
        1 Multidisciplinary Diabetic Limb Service, Frimley Park Hospital NHS Foundation Trust, Frimley; 2 Department of Vascular Surgery, Frimley Park Hospital NHS Foundation Trust, Frimley

9.50-10.00am Training of future vascular surgeons in the UK in endovascular techniques is inadequate to train vascular specialists of the future. Results of a survey of UK vascular trainees
        CD Marron, RK George, SA Badger, B Lee, L Lau, RJ Hannon, JA Reid
        Vascular Surgery Unit, Belfast City Hospital, Belfast

10.00-10.10am Secondary medical prevention among Danish patients hospitalized to primary vascular surgery
        A Høgh 1, 2, SP Johnsen 2, JS Lindholt 1
        1 Department of Vascular Surgery, Viborg Hospital, Denmark; 2 Department of Clinical Epidemiology, Aarhus University, Denmark

10.10-10.20am The impact of standard treatment on balance and physical function among claudicants
        KA Mockford, FAK Mazari, J Khan, N Vanicek, IC Chetter, PA Coughlin
        Academic Vascular Surgical Unit, Hull and East Yorkshire Hospitals NHS Trust & Department of Sport, Health and Exercise Science, University of Hull, Hull

10.20-10.30am Incidence and survival outcome following femoral artery reconstruction during endovascular abdominal aortic aneurysm repair
        CP Twine, A Wood, A Gordon, S Hill, R Whiston, IM Williams
        University Hospital of Wales, Cardiff
10.30-11.00am COFFEE

11.00am-12noon
HORIZON SCANNING
OXFORD SUITE

Chairs: Professor Cliff Shearman, President
Mr Michael Wyatt, Honorary Secretary Elect

Aortic aneurysms
Professor Rob Sayers, Leicester

Treatment of carotid disease
Professor Ross Naylor, Leicester

The future of venous therapies
Professor Alun Davies, London

Vascular access
Dr Richard Fluck, Derby

PAD and diabetes
Professor Gerry Stansby, Newcastle

Trauma
Professor Karim Brohi, London

12noon-12.15pm
PRESENTATION OF LIFETIME ACHIEVEMENT AWARD AND HONORARY MEMBERSHIP

12.15-12.20pm
INAUGURATION OF PRESIDENT FOR 2010-2011

12.20-1.00pm KINMONTH LECTURE
Chair: Mr Peter Lamont, RCS(Eng) Council Member

CHANGING MANAGEMENT OF AORTIC ANEURYSMS - LESSONS FROM THE LIFE AND DEATH OF ALBERT EINSTEIN
Professor Matt Thompson, London

Continuing Medical Education
Delegates will be provided with a Certificate of Attendance which they can add to their appraisal folder as evidence in their appraisal that they have attended a CPD meeting.
Posters

24-26 November 2010 Hilton Brighton Metropole, Brighton

Posters will be displayed in the Upper Gallery at the conference centre during the meeting.

1 Performing amputations during normal working hours reduces mortality
BR Green 1, T Gatenby 1, G Harris 1, A Batterham 2, H Melsom 1, G Danjoux 1, A Parry 1
1 Department of Vascular Surgery, James Cook University Hospital, Middlesbrough; 2 Health and Social Care Institute, School of Health & Social Care, West Teesside University, Middlesbrough

2 Abdominal aortic aneurysm: end of the epidemic?
R Darwood, G Turton, JJ Eamshaw, on behalf of the Gloucestershire Aneurysm Screening Programme
Gloucestershire Royal Hospital, Gloucester

3 Ruptured abdominal aortic aneurysms: a network perspective
A Karthikesalingam 1, AI Awopetu 1, PJ Holt 1, RJ Hinchliffe 1, R Morgan 2, IM Loftus 1, MM Thompson 1
1 Department of Outcomes Research, St George's Vascular Institute, London; 2 Department of Radiology, St George's Hospital, London

4 Population differences in abdominal aortic aneurysm morphology
AL Tambyraja 1, JA Rodriguez-Lopez 2, V Ramaiah 2, EB Diethrich 2, JA Murie 1, RTA Chalmers 1
1 Royal Infirmary of Edinburgh, Edinburgh; 2 Arizona Heart Institute, Arizona

5 Fascial closure following percutaneous endovascular aneurysm repair
GJ Harrison, D Thavarajan, JA Brennan, SR Vallabhaneni, RG McWilliams, RK Fisher
Royal Liverpool and Broadgreen Hospitals NHS Trust, Liverpool

6 Multi-centre Data Quality Study (MDQS)
P Holt, J Poloniecki, U Khalid, M Thompson
Department of Outcomes Research, St George's Vascular Institute, London

7 Patient preference in the management of asymptomatic carotid stenosis
GS Jayasooriya, J Shalhoub, A Thapar, AH Davies
Imperial Vascular Unit, Imperial College London, Charing Cross Hospital, London

8 Can EVAR be performed safely, abandoning a policy of routine cross-matching of blood products?
Frimley Park Hospital NHS Foundation Trust, Frimley

9 Prevalence of abdominal aortic aneurysm in patients undergoing inguinal hernia repair
GA Antoniou 1, AD Giannoukas 2, GS Georgiadis 1, SA Antoniou 3, C Simopoulos 3, P Prassopoulos 4, MK Lazarides 1
1 Department of Vascular Surgery, University Hospital of Alexandroupolis, Demokritos University of Thrace, Greece; 2 Department of Vascular Surgery, University Hospital of Larissa, University of Thessaly Medical School, Larissa, Greece; 3 Second Department of Surgery, University Hospital of Alexandroupolis, Demokritos University of Thrace, Greece; 4 Department of Radiology, University Hospital of Alexandroupolis, Demokritos University of Thrace, Greece

10 Don't forget the medical referral! - Inpatient demand for vascular input post centralisation
SM Jones 1, S Kumar 2, J Joseph 3, RG Ward 3, AJ Guy 1
1 Mid Cheshire Hospitals NHS Foundation Trust, Leighton Hospital, Crewe; 2 Royal Liverpool and Broadgreen University Hospitals NHS Trust, Liverpool; 3 Aintree University Hospitals NHS Foundation Trust, Liverpool

11 Crossover bypass after aorto-uni-iliac stenting: ilio-iliac grafting is superior to femoro-femoral approach
M Kang 1, G McMahon 1, W Adair 2, A Nasim 1
1 Department of Vascular Surgery, University Hospitals of Leicester NHS Trust, Leicester; 2 Department of Interventional Radiology, University Hospitals of Leicester NHS Trust, Leicester
12 Total endovascular repair of thoracoabdominal aortic aneurysms
B Modarai, R Clough, R Salter, R Bell, M Waltham, T Sabharwal, H Zayed, P Taylor, T Carrell
Department of Vascular Surgery, Guy’s and St Thomas’ NHS Foundation Trust, King’s Health Partners, London

13 Repair of complex aneurysms using the visceral hybrid technique
SA Black, C Schneider, R Morgan, T Loosemore, IM Loftus, MM Thompson
St George’s Hospital Vascular Institute, London

14 Repair of thoracoabdominal aortic aneurysms and descending thoracic aneurysms can be performed with low rates of spinal cord ischaemia
S Thwaites, J Richards, A Nimmo, C Moores, P Burns, R Chalmers
Royal Infirmary of Edinburgh, Edinburgh

15 Chimneys, snorkels and periscopes - options for endovascular repair of complex abdominal aortic aneurysms
S Jacob, S Richardson, K Popuri, S Trevelyan, F Farquharson, F Serracino-Inglott
Manchester Royal Infirmary, Manchester

16 Short stay EVAR: selection criteria and outcomes
J Wong, K Varty
Cambridge Vascular Unit, Cambridge

17 Elastic stockings for DVT prophylaxis: what pressure is required
MJ Sultan 1, A Wijeratne 2, CN McCollum 1, 3
1 University Hospital of South Manchester, Academic Surgery Unit, Manchester; 2 University of Manchester, Manchester; 3 University of Manchester, Academic Surgery Department, Manchester

18 The risk of stroke in carotid endarterectomy patients with contralateral carotid artery stenosis
S Sriskandarajah, H Al-Khaffaf
Vascular Unit, East Lancashire Hospitals NHS Trust, Royal Blackburn Hospital, Blackburn

19 Role of salvage angioplasty in patients with critical leg ischaemia undergoing distal bypass graft
A Ahmad 1, H Slim 2, A Tiwari 2, J Wilkins 3, T Sabharwal 4, D Huang 3, R Salter 4, D Evans 3, H Zayed 1, H Rashid 2
1 Vascular Surgery Department, St Thomas’ Hospital, London; 2 Vascular Surgery Department, King’s College Hospital, London; 3 Interventional Radiology, King’s College Hospital, London; 4 Interventional Radiology, St Thomas’ Hospital, London

20 Two week duplex - an efficient approach to graft surveillance.
A Patel, MA Bailey, KJ Griffin, M Weston, DC Berridge, DJA Scott
Leeds Vascular Institute, Leeds

21 The Glasgow Aneurysm Score does not predict mortality following open abdominal aortic aneurysm repair in the era of EVAR
B Patterson, A Kathikesalingam, P Holt, R Hinchcliffe, I Loftus, M Thompson
St George’s Vascular Institute, London

22 Senior vascular trainee experience of assessment and management of patients undergoing endovascular repair of abdominal aortic aneurysm (EVAR). Results of a survey of UK vascular trainees
CD Marron, RK George, SA Badger, L Lau, RJ Hannon, B Lee, JA Reid
Vascular Surgery Unit, Belfast City Hospital, Belfast

23 Spontaneous embolisation in acutely symptomatic patients with TIA/minor stroke
MK Salem, APWW Watts, RD Sayers, MJ Bown, AR Naylor
Vascular Surgery Group, Department of Cardiovascular Sciences, University of Leicester, Leicester

24 Prediction of the impact of AAA screening programme in reducing ruptured aneurysm events after a decade
A Jibawi, SW Yusuf
Brighton & Sussex University Hospitals, Brighton

25 The AAA-Quality Improvement Program (AAA-QUIP) and preoperative cardiopulmonary exercise (CPX) testing: time for a national standard?
AP Navarro 1, Y Ahrned 1, G Atturu 1, H Buglass 2, C Irvine 1, P Curley 1
1 Department of Vascular Surgery, Pinderfields Hospital, Mid-Yorks Hospitals NHS Trust, Wakefield; 2 Department of Anaesthetics, Mid-Yorks Hospitals NHS Trust, Wakefield
Restenosis post-angioplasty is associated with increased smooth muscle cell proliferation in patients with intermittent claudication

AM Wilson 1,2, P Bachoo 2, I Ford 1, GF Nixon 1, J Brittenden 1,2
1 University of Aberdeen, Aberdeen; 2 Department of Vascular Surgery, Aberdeen Royal Infirmary, Aberdeen

Objective
A limiting factor in the success of percutaneous transluminal angioplasty (PTA) is restenosis, presumed secondary to vascular smooth muscle cell (SMC) proliferation. We aimed to determine if patients who developed symptomatic restenosis 2 years post-PTA, expressed higher levels of SMC proliferation.

Method
Fifty claudicants undergoing PTA were randomised to receive blinded clopidogrel or placebo for 30 days in an ongoing trial. The relative ability of their plasma to stimulate extracellular regulated kinase (ERK)1/2 activation in a vascular SMC line in culture was measured at baseline, 1-hour pre-PTA, and 1-hour, 24-hours and 30-days post-PTA. Patients were followed for 2 years post-PTA, via clinics and retrospective case note review, to determine symptomatic restenosis.

Results
ERK1/2 activation was significantly increased 1-hour post-PTA irrespective of treatment with clopidogrel (p=.001) or placebo (p=.013). Three patients were excluded (technical failure, n=1, abnormal baseline activation, n=2). Nine patients required later re-intervention at the site of PTA for symptomatic restenosis. The plasma from these patients at 1-hour post-PTA produced a significantly (p<0.05) higher level of ERK1/2 activation in cultured SMCs (median 300%, range 246.5-537.5) compared to plasma from the 38 who did not require re-intervention (200%, 150-300). At later time-points there was no statistically significant difference.

Conclusion
SMC proliferation (represented by plasma ability to induce ERK1/2 activation in cultured SMCs) was significantly increased in patients developing a symptomatic restenosis post-PTA. This suggests a direct relationship between restenosis and the ‘proliferative potential’ of plasma, and likely reflects in vivo SMC proliferation. Further work is required to evaluate potential therapeutic treatments which may reduce peripheral PTA-induced SMC activation.
Activation of hypoxia-inducible factor (HIF) pathway in varicose veins

CS Lim 1,2, S Kiriakidis 2, A Sandison 3, EM Paleolog 2, AH Davies 1

1 Imperial Vascular Unit, Department of Surgery and Cancer, Faculty of Medicine, Imperial College London, London; 2 Cytokine Biology of Vessels, Kennedy Institute of Rheumatology and Department of Surgery and Cancer, Imperial College London, London; 3 Department of Histopathology, Charing Cross Hospital, Imperial College Healthcare NHS Trust, London

Objective
Hypoxia has been postulated to contribute to varicose vein wall changes. Hypoxia-inducible factors (HIFs) are transcriptional factors that regulate the expression of genes of oxygen homeostasis. The study aimed to compare HIF-1α, HIF-2α, and their target genes expression in varicose with non-varicose veins.

Method
Varicose and non-varicose veins were surgically retrieved from patients with and without varicosity, respectively. Protein and mRNA expression of HIF-1α, HIF-2α, and their target genes in varicose and non-varicose veins were analysed with immunoblot and real-time polymerase chain reaction. Data were presented as mean±SEM, and analysed with an unpaired t-test and Mann-Whitney U test.

Results
HIF-1α and HIF-2α mRNA was up-regulated in varicose compared to non-varicose veins (89.8±18.6, n=11 versus 10.4±7.2, n=5; p=0.012) and (384.9±209.4, n=11 versus 8.1±4.2, n=5; p=0.008), respectively. Increased HIF-1α and HIF-2α protein expression was also observed in varicosities. The mRNA expression of HIF target genes was elevated in varicose compared to non-varicose veins; glucose transporter-1 (8.7±2.1, n=20 versus 1.0±0.3, n=10; p<0.001), carbonic anhydrase-9 (8.5±2.1, n=20 versus 2.8±1.2, n=10; p=0.006), vascular endothelial growth factor (7.5±2.1, n=20 versus 0.9±0.2, n=10; p=0.001), BNIP-3 (4.5±0.7, n=20 versus 1.4±0.3, n=10; p=0.004), enolase-1 (11.2±2.1, n=11 versus 3.1±1.9, n=5; p=0.019), prolyl-hydroxylase domain (PHD)-2 (5.6±1.1, n=11 versus 1.7±0.7, n=5; p=0.034), and PHD-3 (9.9±2.2, n=11 versus 2.4±1.2, n=5; p=0.047). HIF target genes up-regulation in varicosities was also reflected at protein.

Conclusion
HIF-1α, HIF-2α, and target genes were up-regulated in varicose compared to non-varicose veins. Our data suggest the HIF pathway may be an important contributor to various structural and biochemical changes in varicosities.
Down-regulation of hypoxia-inducible factor 1α reduces venous thrombus resolution
CE Evans, J Humphries, K Mattock, M Waltham, A Wadoodi, P Saha, B Modarai, A Smith
Academic Department of Surgery, King’s College London, London

Objective
Hypoxia-inducible factor 1 (HIF-1)-mediated angiogenic factors are induced within venous thrombus during its resolution, but the primary stimulus for their production and thrombus resolution is unknown. Our aim was to determine whether down-regulating HIF-1α in the thrombus and vein wall reduces angiogenic factor expression, inflammatory cell infiltration, and thrombus resolution.

Method
Thrombus was induced in the inferior vena cava (IVC) of 40 mice. The mice were treated with the HIF-1α inhibitor, 2-methoxyestradiol (2ME, i/p, 150mg/kg/day) or vehicle control (n=20/group). HIF-1α, VEGF, and PLGF expression in the thrombus and IVC were measured at days 1 and 10 (n=7/group) by enzyme-linked immunosorbent assay (ELISA). Thrombus size, neovascularisation, recanalisation, and macrophage and neutrophil infiltration were also measured at day 10 by image analysis (n=6/group).

Results
The levels of HIF-1α (p<0.001), VEGF (p<0.001), and PLGF (p<0.001), and macrophage (p<0.05) and neutrophil (p<0.005) numbers were decreased in the thrombus of mice treated with 2ME compared with vehicle control. The levels of HIF-1α (p<0.005), VEGF (p<0.005), and PLGF (p<0.001), and macrophage (p<0.005) and neutrophil (P<0.01) infiltration were also decreased in the IVC wall surrounding the thrombus of 2ME-treated mice compared with controls. Thrombus neovascularisation (p<0.005) and vein recanalisation (p<0.005) were decreased, while thrombus size (p<0.02) and weight (p<0.001) were increased in 2ME-treated mice compared with controls.

Conclusion
Reducing HIF-1α expression in the thrombus and vein wall reduces angiogenic growth factor expression, inflammatory cell infiltration, and thrombus resolution. These data suggest that HIF-1α activity is an important regulatory mechanism in thrombus resolution.
The angiogenic potential of Tie2-expressing monocytes is impaired in patients with critical limb ischaemia
AS Patel, A Smith, P Saha, K Mattock, J Humphries, R Siow, M Waltham, B Modarai
King's College London BHF Centre of Excellence, Academic Department of Surgery, Cardiovascular Division; The NIHR Biomedical Research Centre at Guy’s and St Thomas’ NHS Foundation Trust and King’s College London, London

Objective
Our pilot studies have shown that angiogenic monocytes (CD14+ve cells) expressing the angiopoietin receptor, Tie2, are mobilised in patients with critical limb ischaemia (CLI). We aimed to further investigate this angiogenic drive.

Method
The proportion of Tie2+ve monocytes was measured in blood from 40 patients with CLI, 20 age/sex-matched and 20 young controls by flow cytometry. A panel of 12 circulating angiogenic/inflammatory factors was measured by multiplex ELISA in CLI patients and controls (n=10/group). An in vitro angiogenesis assay was used to compare lysates of Tie2+ve and Tie2-ve monocytes from patients and controls.

Results
Patients with CLI had 10-fold higher CD14+ve/Tie2+ve cells compared with age/sex-matched and young controls (3.52%±0.28 vs 0.39%±0.09 and 0.23%±0.04, respectively p<0.0001). Tie2 expression was confirmed by RT-PCR. Circulating levels of angiopoietin-2 (799±128 vs 384±62pg/ml), IL-6 (79±29 vs 17±6pg/ml) and MCSF (39±11 vs 14±1pg/ml) were significantly higher in CLI patients compared with controls, respectively (p<0.05). Overall, Tie2+ve monocytes were more angiogenic compared with Tie2-ve monocytes (n=18/group), inducing greater microtubule length (p=0.03), tubule area (p=0.004) and number of nodes (p=0.04). However, the angiogenic potential of Tie2+ve monocytes from patients with CLI was lower than monocytes from healthy controls (n=9/group), with reduced microtubule length (p=0.04), tubule area (p=0.03) and number of nodes (p=0.04).

Conclusion
CLI is associated with a rise in Tie2+ve monocytes and the Tie2 receptor ligand, angiopoietin-2. This angiogenic drive may, however, be hampered by raised circulating levels of IL-6 and MCSF, which are systemic signals known to impair the angiogenic properties of monocytes.
Advancing what we know about genetics and varicose veins
J Krysa, G Jones, A van Rij
Department of Surgery, Dunedin School of Medicine, University of Otago, Dunedin, New Zealand

Objective
There is good circumstantial evidence which implicates genetics in the aetiology of varicose veins. The exact nature of the genetic basis remains unclear. The aim of this study was to consider the current reported genetic associations with varicose veins and to carry out a case control analysis to validate these using data from a genome wide association study (GWAS).

Method
An indirect, in silico, genome wide study of varicose veins was undertaken. This was based on our abdominal aortic aneurysm GWAS in which the frequency of varicose veins was similar in cases and controls. Genetic polymorphisms associations with venous disease to date were identified through a literature search. All known single nucleotide polymorphisms (SNPs), with >5% allele frequency, in the genes previously implicated were analysed. Genotyping was carried out using Affymetrix Genome-Wide Human SNP Array 6.0.

Results
349 patients with varicose veins and 857 controls were included. Genes which have been implicated in venous disease so far include FOXC2, HFE C282Y, Factor XIII V34L, oestrogen receptor B, TNF-A, MTHFR and thrombomodulin. None of the SNPs in these genes have shown significant association in this study. However, there were a number of other SNPs which were found to be associated with varicose veins and these are being validated in another cohort.

Conclusion
Previous candidate genes implicated in common venous disease have not been confirmed. A GWAS approach has been shown to be useful in validation and discovery of novel genes in venous disease but larger cohorts are required to confirm these.
INDICATION FOR USE: AngioDynamics® NeverTouch® – FRS Procedure Kit is indicated for endovascular coagulation of the great saphenous vein (GSV) in patients with superficial vein reflux, for the treatment of varicose veins and varicosities associated with superficial reflux of the GSV, and for the treatment of incompetence and reflux of superficial veins of the lower extremity. The VenaCure EVLT Procedure Kit is indicated for use with 810 nm and 980 nm Diode Lasers with SMA 905 connectors. CAUTION: Federal (USA) law restricts the sale of this device by or on the order of a physician.

CONTRAINDICATIONS: Patients with thrombus in the vein segment to be treated, patients with an aneurysmal section in the vein segment to be treated or patients with peripheral artery disease as determined by the Ankle Brachial Pressure Index with a value of <0.9 should not have their varicosities ablated.

WARNINGS AND PRECAUTIONS: Treatment of a vein located close to the skin surface may result in skin burn. Tissue not targeted for treatment must be protected from injury by direct and reflected laser energy. All persons in the treatment room MUST wear protective glasses with the proper rating for the wavelength being used.

CAUTION: This device is ethylene oxide sterilized and intended for single patient use only. Do not reuse or resterilize the fibers. Contents sterile in unopened, undamaged package. Do not use if opened or any sign of product damage is visible. Carefully read all directions and observe all Warnings and Precautions prior to performing the procedure.

POTENTIAL COMPLICATIONS: Adverse reactions may include, but are not limited to: vessel perforation, thrombosis, pulmonary embolism, phlebitis, hematoma, infection, skin pigmentation alteration, neovascularization, paresthesia due to thermal damage of adjacent sensory nerves, anesthetic tumescence, non-target irradiation, vasospasm, hemorrhage, necrosis, skin burns and pain.

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IMPORTANT RISK INFORMATION
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For more information about the science behind NeverTouch laser fibers visit www.VenaCure-EVLT.co.uk

Visit AngioDynamics at the Vascular Society AGM in Brighton
The effect of novel texture features of homogeneity and echolucency on carotid plaque characterization; results from the ACSRS study
GC Makris 1, M Griffin 2, G Geroulakos 1, AN Nicolaides 3
1 Imperial College of Science and Technology, London, Department of Vascular Surgery, Ealing Hospital NHS Trust, London; 2 Cardiovascular Disease Education and Research (CDER) Trust; 3 Imperial College of Science and Technology, London

Objective
The effectiveness of novel ultrasound texture features - of both echolucency and homogeneity - on carotid plaque characterization, based on their risk for cerebral or retinal ischaemic (CORI) events was evaluated.

Method
An observational study was conducted in patients with 50-99% asymptomatic internal carotid artery stenosis. Baseline images from 1,121 patients from the Asymptomatic Internal Carotid Artery Stenosis and Risk of Stroke study were evaluated. Dedicated software provided us with 51 histogram/texture features of the plaque image. Factor analysis was used to identify redundant features. Hazard ratio analysis for CORI events was performed for the resulting features after adjustment for stenosis and after being controlled for established plaque factors (grey scale median, plaque area). Receiver operator curves (ROC) were used for model evaluation.

Results
A total of 130 ipsilateral CORI events occurred after a mean follow-up of 48 (range: 6-98) months. On survival analysis only the texture features Angular Second Moment of the Spatial Gray Level Dependence Matrices (SGLD_ASM) and the Run length distribution of the Gray level run length statistic (RUN_RLD) remained significant (HR: 3.3, 95% CI: 2.02 to 5.58, p<0.0001 and 1.0, 95% CI: 1.01 to 1.04, p<0.0001, respectively). Using ROC area under the curve, the new features improved the values of the established ones in distinguishing between the occurrence or not of CORI events (0.845 SE: 0.018, p<0.0001 vs. 0.80, SE: 0.019, p<0.0001, respectively).

Conclusion
Carotid plaque echolucency and homogeneity can be simultaneously assessed by novel texture features. This may further improve the risk stratification of patients with asymptomatic carotid plaque disease.
Helix-B peptide of erythropoietin could be used as pharmacotherapy in critical limb ischaemia

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Objective
Pharmacotherapy has a limited role in the management of critical limb ischaemia (CLI). Erythropoietin (EPO), acting through its tissue-protective heteroreceptor complex (EPOR-CD131), is protective in many tissues and we have previously shown the expression and upregulation of EPOR-CD131 in ischaemic human skeletal muscle. However, EPO causes profound haemopoiesis, with increased risk of thromboembolism. ARA-290, a peptide that is derived from the helix-B of the EPO molecule, is tissue-protective but not haemopoietic. This makes it feasible for use in CLI. The aims of this study are to demonstrate the tissue-protective properties of EPO and ARA-290 in an in vitro model of skeletal muscle ischaemia and to assess the angiogenic potential of EPO and ARA-290 in vitro.

Method
An in vitro simulated model of skeletal muscle ischaemia was developed using skeletal myotubes cultured in hypoxic chambers. Myotubes were subjected to simulated ischaemia after pre-treatment with EPO or ARA-290. Apoptosis was measured by nuclear staining, cleaved caspase-3 assay and LDH release. Angiogenic potential of EPO and ARA-290 was assessed in human microvascular endothelial cells by proliferation, migration and capillary-like tube formation assays.

Results
ARA-290 and EPO significantly decreased the number of apoptotic nuclei, cleaved caspase-3 and LDH release in skeletal myotubes exposed to simulated ischaemia (p<0.01). However, only EPO was found to significantly increase proliferation, migration and capillary-like tube formation of microvascular endothelial cells (p<0.05).

Conclusion
ARA-290 may potentially reduce ischaemia-induced tissue damage in CLI whilst avoiding the side effects of EPO. The results have provided us with a basis to conduct in vivo experiments.
Peak oxygen consumption is a useful biomarker in assessing survival after abdominal aortic aneurysm repair

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Objective

Abdominal aortic aneurysm (AAA) repair should be reserved for patients with a reasonable postoperative life expectancy. Exercise capacity is a more powerful predictor of mortality than established cardiovascular risk factors. The objective of this study was to examine the association between performance on cardiopulmonary exercise testing (CPX) and 1-year survival in patients undergoing AAA repair.

Method

Between 2007 and 2009, 134 patients undergoing open or endovascular (EVAR) AAA repair underwent pre-operative CPX testing. Anaerobic threshold (AT) and peak oxygen consumption (VO$_2$ peak), expressed in ml kg$^{-1}$ min$^{-1}$, were determined. Cardiovascular risk factors, 30-day and 12-month mortality were obtained from our database. The data were analysed for two groups, survivors and non-survivors; data are expressed as median (interquartile range).

Results

134 patients (115 men) were studied; 70 open AAA repairs and 64 EVARs. Five patients died within 30 days (3.7%) and five within a year of surgery (1-year mortality of 7.5%). Groups were well matched for age and cardiovascular risk factors. Pre-operative VO$_2$ peak was significantly lower in non-survivors (15.3 [13.2 - 17.8] and 12.4 [9.3 -16.5] in survivors and non-survivors, respectively, p=0.031). AT was not different between the groups (10.7 [9.2 - 12.8] and 10.6 [8.4 - 12.5] in survivors and non-survivors, respectively, p=0.621).

Conclusion

Median VO$_2$ peak was significantly less in patients who died within a year of AAA repair. This may provide a means of identifying patients who will not benefit from surgery. In contrast, AT is not an independent predictor of 1-year mortality, although this may reflect marked inter-observer variability in its derivation.
Integrin-α9-fibronectin interaction is required for normal murine venous valve morphogenesis

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Objective
Treatment of the complications of varicose veins and venous hypertension in the leg consumes >2% of the NHS budget. It remains unproven whether a primary defect in the venous wall or the venous valve (VV) initiates VV failure. We have shown that integrin-α9, encoded by Itga9 (identified in humans with congenital chylothorax) is expressed throughout the forming lymphatic valve (LV) and that binding to the EIIIA splice variant of fibronectin is required in LV formation. There are no data describing murine VV development. This study set out to describe VV morphogenesis, and examine VV phenotypes in mutant (Itga9 and EIIIA knockout) mice.

Method
Tie2-LacZ endothelial reporter mice combined with scanning electron microscopy (SEM) were used to characterise stages in normal murine VV development. Fluorescent-labelled antibodies to integrin-α9 and EIIIA were used with laser scanning confocal microscopy to examine expression patterns in developing VV in wild-type mice. SEM was used to phenotype VV structure in Itga9 and EIIIA heterozygous and homozygous knockout mice.

Results
Both Itga9 and EIIIA are expressed in VV leaflets, and homozygous knockout mice (for either gene; n=6, n=2 valves, respectively) display gross VV malformation or aplasia. Heterozygous knockouts had structurally normal VV.

Conclusion
This is the first description of murine venous valve morphogenesis and shows that there are genetic similarities (requirement for integrin-α9, fibronectin-EIIIA) in the patterning of lymphatic and venous valves, which may reflect similar function. Characterisation of cell-extracellular matrix interactions in mice may provide insight into human VV disease.
Intra-plaque production of M1-type cytokines and matrix metalloproteinases differentiate stable from unstable carotid atherosclerosis

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Objective

Molecular and cellular characterisation of vulnerable atherosclerosis would help target functional imaging and plaque-stabilising therapeutics. We quantified cytokine and matrix metalloproteinase (MMP) protein production in symptomatic human carotid plaques to map the pro-inflammatory milieu responsible for plaque instability.

Method

Carotid endarterectomies from symptomatic (n=35) and asymptomatic (n=32) patients were enzymatically dissociated producing mixed macrophage-rich, atheroma cell suspensions which were cultured for 24 hours. Supernatants were interrogated with a 45-analyte panel on a Luminex 100 platform. Analyte inter-relationships were described statistically via Spearman correlation. Resulting sets were analyzed via Ingenuity Pathways Analysis v7.6. A 1.5-fold cut-off was set to identify proteins whose expression was significantly increased or decreased. Right-tailed Fisher's exact test determined significance (p-value) of protein over-representation compared to the result expected by a random protein set.

Results

Tumour necrosis factor (TNF)-alpha, interleukin (IL)-1alpha, IL-1beta, IL-6, IL-10, granulocyte-macrophage colony-stimulating factor (GM-CSF), macrophage colony-stimulating factor (M-CSF), CCL2, CCL5, CCL20, CXCL9, CXCL10, MMP1, MMP3, MMP8 and MMP9 levels were significantly higher in symptomatic than asymptomatic plaques (p<0.05). A number of inter-related analyte ‘clusters’ were identified. Top-ranked biological pathways associated with differences between symptomatic and asymptomatic data sets (identified via Ingenuity) focused on the interplay between innate and adaptive immunity, nuclear factor-kappaB and MAPK signalling, hypercytokinaemia in inducing pathology and matrix degradation, and IL17 signalling.

Conclusion

The inflammatory milieu within human unstable plaque is in keeping with a predominance of M1-type macrophages with signatures of interferon-gamma and IL17 signalling. The novel finding in atherosclerosis of elevated CCL20 is further supportive of this Th17/IL17 signalling.
The potential therapeutic role of toll-like receptor 2 in critical limb ischaemia

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Objective

Inflammation and cell damage contribute to the pathophysiology of critical limb ischaemia. Toll-like receptors (TLRs) play an important role in inflammation and tissue damage possibly in response to endogenous ligands such as high mobility group box 1 (HMGB1). Functional TLR2 is expressed in skeletal muscle. We hypothesize that TLR2 signalling is upregulated resulting in an increase in the release of inflammatory cytokines such as interleukin-6 (IL-6) in muscle ischaemia.

Method

TLR2 expression was studied in ischaemic and control human muscle biopsies and in vitro using C2C12 myotubes cultured in simulated ischaemic conditions using Western blot. The functional effects of TLR2 antagonism on ischaemia-induced IL-6 release and cell death were studied by incubating myotubes with neutralizing TLR2 antibody. IL-6 release was assayed by ELISA. Apoptosis was assessed using cleaved caspase-3 and bax/bcl-2 ratio measurements. HMGB1 levels were measured using Western blot.

Results

TLR2 protein expression was significantly upregulated in critically ischaemic muscle and in C2C12 myotubes cultured in ischaemic conditions (p<0.05). IL-6 production increased in C2C12 myotubes cultured in simulated ischaemia. TLR2 antagonism reduced ischaemia-induced IL-6 production and apoptosis. Raised levels of HMGB1 were also demonstrated in the ischaemic C2C12 myotubes.

Conclusion

These results show that TLR2 signalling is activated in ischaemic muscle leading to the release of cytokines such as IL-6 and contributing to inflammation and muscle damage. This could be activated by the endogenous ligand HMGB1. Further delineation of the TLR pathways may lead to strategies to reduce inflammatory damage in the treatment of critical limb ischaemia.
Clinical cell tracking of mononuclear cells using magnetic resonance imaging and superparamagnetic particles of iron oxide

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Objective
Cell therapy has emerged as a possible novel treatment option for critical limb ischaemia and following myocardial infarction. There is considerable interest in methods for in vivo cell tracking to determine whether cells reach and remain in the target site. We have developed a protocol for labelling monocytes with superparamagnetic particles of iron oxide (SPIO), and evaluated its potential for use in human cell tracking studies.

Method
Up to 10⁹ human mononuclear cells were labelled with SPIO. Labelling efficiency, viability and migration were assayed in vitro. Six healthy volunteers received intramuscular thigh injections of labelled cells, unlabelled cells and SPIO alone, and underwent T2-weighted (T2W) MRI. A phased-dosing protocol was used to assess the safety of intravenous infusion of labelled cells. Six further volunteers receiving ~10⁹ SPIO-labelled cells underwent multiecho T2*W imaging of the liver and spleen before and after (2 hours-7 days) administration of cells.

Results
Efficient SPIO cell labelling was achieved without affecting in vitro viability or migratory capacity. SPIO-labelled cells were visualised on T2W imaging following IM administration. Intravenous administration of approximately 10⁹ labelled cells was well tolerated and a significant reduction in T2* value was observed in the liver and spleen (p<0.001) reflecting accumulation of SPIO-labelled cells.

Conclusion
We have demonstrated for the first time in humans that SPIO-labelled mononuclear cells can be detected at a target site in a clinical MRI scanner following both local and systemic administration. This technique holds major promise as an important tool for the further development and monitoring of novel cell-based therapies.
Magnetic resonance T1 mapping predicts successful venous thrombolysis
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Objective
Novel thrombolytic delivery systems are changing the treatment paradigm for deep vein thrombosis (DVT), but older, well-organised thrombi remain unsuitable for intervention. A technique that identifies thrombi that are amenable to lysis is needed.

Method
An MRI 3D T1-mapping protocol was developed for imaging venous thrombi induced in mice. T1-relaxation times were quantified between 4 and 28 days after induction (n=33). The thrombus was sectioned along its entire length. Collagen content was measured histologically as a marker of organisation and compared with T1-relaxation times in corresponding MR slices. Results were validated by three blinded observers. Tissue plasminogen activator (Actilyse) was injected (10mg/kg) between 4 and 16 days after thrombus induction (n=16). T1-mapping was performed before and 24 hours after Actilyse administration. Successful thrombolysis (vein recanalisation) was measured as an increase in the velocity of flow across the IVC greater than 0.3cm/s (confirmed by histology).

Results
T1-relaxation time increases with thrombus age and organisation (763±22ms, 4d; 617±36ms, 7d; 673±51ms, 10d; 728±58ms, 14d; 945±66ms, 21d; 1194±59ms, 28d). Collagen content is proportional to T1-relaxation time during thrombus resolution (R2=0.80, p<0.0001, n=15). T1-relaxation times were significantly shorter in the group successfully treated with thrombolysis (p=0.002, n=16). ROC curve analysis shows an optimal cut-off point of ~700ms. The sensitivity and specificity for predicting successful thrombolysis was 100% and 88%.

Conclusion
This is the first study to show that T1-mapping quantifies organisation of experimental venous thrombi and predicts response to thrombolysis. This technique enables the objective selection of thrombi that could be successfully treated with lysis in patients presenting with DVT.
Uptake of ultrasmall superparamagnetic particles of iron oxide predicts growth in abdominal aortic aneurysms

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Objective

In abdominal aortic aneurysm (AAA) disease focal hotspots of neovascularisation, inflammation and proteolysis represent areas at risk of expansion and rupture. Prediction of disease progression is challenging and currently relies on the simple measure of aneurysm diameter. We aimed to assess whether areas of cellular inflammation correlated with the rate of aneurysm expansion.

Method

Patients (n=29; 27 male; aged 70±5 years) with intact, asymptomatic AAA (4.0-6.6cm) were recruited from a surveillance programme and underwent 3T MRI scanning before and 24-36 hours after administration of ultrasmall superparamagnetic particles of iron oxide (USPIO). The change in T2* value on T2*-weighted imaging was used to detect accumulation of USPIO within the aneurysm. Aneurysm growth rate was determined using ultrasound. In patients undergoing open surgery, aortic wall tissue was obtained and stained for CD68 (macrophages) and iron (Prussian blue).

Results

Histological examination of aneurysm tissue confirmed uptake of USPIOs in areas of macrophage infiltration. Patients with distinct hotspots of USPIO uptake in the aneurysm wall (n=13) had a three-fold higher growth rate (0.66cm/yr; p=0.020) than those with no (n=7; 0.22cm/yr) or non-specific USPIO uptake (n=9; 0.24cm/yr), despite having similar aneurysm diameters (5.4±0.6, 5.1±0.5 and 5.0±0.5cm, respectively; p>0.05). In one patient with an inflammatory aneurysm, widespread USPIO uptake extended beyond the aortic wall.

Conclusion

Uptake of USPIO in the aortic wall identifies cellular inflammation and appears to distinguish those patients with more rapidly progressive aneurysm expansion. This technique holds major promise as a new method of risk-stratifying patients with AAA.
INTRODUCING THE LOWEST-PROFILE AAA ENDOVASCULAR GRAFT SYSTEM ON THE MARKET
The effect of n-3 long chain polyunsaturated fatty acid (n-3LCPUFA) supplementation on platelet and endothelial function in patients with peripheral arterial disease

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Objective
n-3 LCPUFA supplementation reduces platelet and endothelial activation in patients with or at risk of cardiac disease. We aimed to determine if n-3 LCPUFA supplementation in addition to best medical therapy can reduce the increased platelet and endothelial activity that is present in patients with intermittent claudication.

Method
150 patients were recruited in a randomised cross-over double-blind study involving 6-week supplementation with OMACOR fish oil (850-882mg eicosapentaenoic and docosahexaenoic acid) versus placebo. A 6-week washout period occurred between treatments. Patients with diabetes were excluded. For each outcome a mixed model analysis in which treatment, period and baseline values were fixed and patients were randomised was performed.

Results
Supplementation with omega-3 significantly reduced unstimulated p-selectin expression (-0.33 % expression [95% CI -0.64 to -0.03], p=0.03). However, no effect was observed on other flow cytometry markers of platelet activation (stimulated p-selectin expression, stimulated/unstimulated fibrinogen binding) or platelet aggregation (ultegra point of care). Similarly, the markers of endothelial activation, pulse-wave velocity, S-ICAM and von-Willebrand Factor, along with the inflammatory markers C-reactive protein and IL-6, were also unchanged.

Conclusion
Supplementation with n-3 LCPUFA reduced unstimulated platelet p-selectin expression which has previously been shown to be elevated in patients with peripheral arterial disease and to increase with disease severity. It had no effect on markers of endothelial function.
Engineering a novel Ang1 mimetic for regenerative medicine applications

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Objective
In tissue engineering, improved vascularisation of the regenerated tissue is essential to overcome initial tissue mass loss. Angiopoietin 1 (Ang1) is an angiogenic ligand essential for formation of functional blood vessels and with great potential for promoting tissue vascularisation. However, recombinant Ang1 is a large glycoprotein with variable solubility and biological activity and is difficult to produce. This study aims to develop small, stable Ang1 mimetic proteins for use as potential therapeutic lead molecules.

Method
Based on the mechanism by which the native ligand activates its receptor, a small synthetic ligand was designed. DNA sequences were constructed and expressed in E Coli. The synthetic ligand was isolated and purified and its ability to bind the angiopoietin receptor analysed by in vitro ELISA. Cell surface binding was examined by immunofluorescence staining and the ability of the ligand to activate cellular signalling was tested by phospho-specific immunoblotting. Functionally, the influence of the ligand on endothelial cell migration was studied using Boyden chamber chemotactic assay.

Results
A small synthetic ligand was produced. The ligand binds and activates angiopoietin receptors. In addition, it stimulates downstream signalling pathways including the phosphatidylinositol 3-kinase/Akt and Erk1/2 pathways. The ligand activates endothelial cell migration.

Conclusion
The novel synthetic ligands are easy to produce, highly soluble and stable, and activate the angiopoietin receptor. The properties of these synthetic ligands suggest they may be lead molecules for generating potential therapeutic Ang1 mimetics. In addition, the synthetic ligands can be immobilized on a tissue engineering scaffold to improve vascularisation of the engineered tissue.
Proteomic evidence of impaired resistance to oxidative stress and inflammation in the vasculature of patients with abdominal aortic aneurysms
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Objective
Proteomics may provide important insights into abdominal aortic aneurysm (AAA) pathogenesis. AAAs represent a systemic disease of the vasculature. AAA tissue demonstrates the end-stage of disease, limiting its utility in identification of factors initiating aneurysm development. Comparable morphological and molecular changes have been demonstrated in the vasculature of AAA patients distant from the aneurysm. Using a controlled comparative technique on vascular tissue from AAA patients, we sought to identify modified protein expression in the vasculature.

Method
Thirty-two patients, 16 with large AAAs and 16 matched controls were prospectively recruited. Inferior mesenteric vein (IMV) was harvested, homogenised and mined for differential protein expression. Difference in gel electrophoresis (DiGE), using a 2-D platform, identified protein spots with significantly altered intensity. MS/MS liquid chromatography characterized proteins of interest. DiGE findings were validated by Western Blotting (WB) and protein expression was localised within the IMV by immunohistochemistry (IHC).

Results
1223 spots were demonstrated; 10 spots indicating clinically significant fold changes were explored further. 3/10 spots were identified: prohibitin (fold change [FC] x 1.9, p=.002) and annexin A1 (FC x 1.6, p=.002) were down-regulated in AAA patients; caspase-cleaved vimentin (FC x 1.5, p=.04) demonstrated increased expression. All fold changes were confirmed by WB and localized to vascular smooth muscle cells (VSMCs) by IHC.

Conclusion
Proteins important in combating oxidative stress (prohibitin) and modulating inflammation (annexin A1) are reduced in the vasculature of AAA patients. There is evidence of increased apoptosis in VSMCs in these patients (cleaved vimentin). The proteomic changes demonstrated in this model of early aneurysmal disease may provide future therapeutic targets to combat AAA development.
Imaging of the vulnerable carotid plaque: biological targeting of inflammation using ultrasmall superparamagnetic particles of iron oxide (USPIO) and MRI

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Objective

Inflammation drives atherosclerotic plaque instability and acute thromboembolism, such as stroke. There is currently no clinical imaging technique available to assess the degree of inflammation associated with plaques. This study aims at visualising and characterising atherosclerosis using targeted USPIO as an MRI probe for detecting inflamed plaque disease.

Method

The initial in vitro feasibility study involved MRI detection of activated endothelial cells using anti-E-selectin antibody conjugated USPIO with confirmatory immunocytochemistry. In the ex vivo stage we have detected inflammatory markers on human atherosclerotic plaques harvested during carotid endarterectomy by anti-E-selectin antibody and anti-VCAM-1 antibody conjugated USPIO using MRI. In the in vivo stage we have detected atherosclerotic lesions in ApoE-/- mice using dual-targeted USPIO against VCAM-1 and E-selectin.

Results

We have established an in vitro model of endothelial cell inflammation, confirmed with both MRI and immunocytochemistry. We can now image inflammation of human atherosclerotic plaques by ex vivo MRI. The preliminary results showed that we are able to detect atherosclerotic lesions in ApoE-/- mice using dual-targeted USPIO against VCAM-1 and E-selectin.

Conclusion

We have successfully developed an in vitro model to detect and characterise inflamed endothelial cells by immunocytochemistry and MRI. We are able to image the degree of inflammation associated with atherosclerotic plaques by ex vivo MRI, and able to detect atherosclerosis in ApoE-/- mouse mice by in vivo MRI. This provides a new biologically-based imaging modality to identify the ‘at risk’ group with carotid plaque disease and aid decision making for appropriate intervention.
‘Mini-sternotomy’: a novel access for endografting of descending thoracic aortic aneurysms
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Objective
Endovascular thoracic aortic aneurysm repair is an alternative to the traditional open surgical repair. The endovascular approach is attractive by its potential to avoid the high morbidity and mortality associated with standard open surgical repair. The complexity of endovascular repair of thoracic aneurysms is increased by extensive atherosclerosis involving the aortoiliac segment. This study demonstrates the feasibility of endovascular repair of thoracic aortic aneurysms via a mini-sternotomy.

Method
From January 2002 through to October 2003, 7 patients underwent transluminal endovascular stent-graft placement for repair of thoracic aortic disease (one patient for thoracic aortic dissection while the remainder had descending thoracic aneurysms). Patients included in the study were those with extensive atherosclerosis and kinking involving the aortoiliac segment rendering the femoral access or iliac conduit not applicable or even hazardous. The follow-up protocol included early chest physiotherapy, a daily chest X-ray, U&Es, arterial blood gases and regular checks of distal pulsation till discharge. Patients underwent follow-up CT at 1, 6 and 12 months or at any occasion of suspected complications.

Results
No operative or postoperative complications were encountered during the in-hospital course of management of these patients, while during the 1-year follow-up we had one mortality 8 months following the procedure due to myocardial infarction.

Conclusion
The use of mini-sternotomy (manubriotomy) allowed excellent exposure of the aortic arch with visualization of the exact morphology of the proximal landing zone, so it can be used in patients with extensive atherosclerotic changes involving the aortoiliac segment where femoral access or iliac conduit is not feasible.
Abstracts

Medium-term outcomes of emergency EVAR for ruptured AAA
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Objective
Emergency endovascular aortic aneurysm repair (eEVAR) is rapidly becoming the treatment of choice for ruptured abdominal aortic aneurysms (rAAA) where facilities and expertise are available. Although studies have demonstrated that eEVAR is associated with reduced peri-operative mortality, relatively little is known about longer-term outcome following eEVAR. In particular, whether eEVAR confers a long-term survival benefit compared to open rAAA repair remains unknown. The aim of this study was to evaluate the medium-term outcomes of eEVAR for rAAA at our institution.

Method
A prospective database of all patients undergoing eEVAR from January 2006 to April 2010 for rAAA at our institution was analyzed.

Results
Fifty-two patients (45 male), median age 78 years (range 62-92 years), have undergone eEVAR for rAAA. The median length of stay was 11 days (IQR 7-25 days). There have been three (6%) late re-interventions (2 endovascular, 1 surgical). At a median follow-up of 25 months (range 2-52 months) there have been 15 deaths (29%), of which 6 were inpatient deaths (12%). Overall survival was 82% at 1 year and 73% at 2 years.

Conclusion
Emergency EVAR is associated with excellent medium-term survival in this cohort that included patients deemed unfit for open repair. We would recommend eEVAR to be the management of choice for rAAA in anatomically suitable patients where the local facilities and expertise exist.
The effect of mismatch between native anatomy of visceral aorta and design of fenestrated stent-grafts

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Objective

Accurate measurement of native anatomy is required to plan devices for fenestrated endovascular aneurysm repair (fEVAR). Measurements are subject to observer variability and potentially mismatch between the native anatomy and the stent-graft configuration. The aim of this study was to examine the effect of mismatch between fenestrated stent-grafts and native anatomy on proximal seal.

Method

A 36mm proximal main-body incorporating two fenestrations and a scallop was deployed within a series of phantoms depicting visceral aorta. One phantom was produced with perfect alignment between the visceral vessels and fenestrations. Six additional phantoms were created with incremental mismatch in both the circumferential (n=3) and longitudinal position of the renal vessels (n=3). Qualitative assessment of apposition between the seal zone of the phantom and the stent-graft fabric was made by visual inspection and radiography. The degree of stent-graft distortion and misalignment of the scallop in relation to the superior mesenteric artery (SMA) as a result of stent-graft/phantom mismatch was also assessed.

Results

Fabric to lumen apposition (seal) was maintained in all phantoms. A circumferential discrepancy of 30° did not result in scallop misalignment. Partial SMA shuttering was observed at 45° and a discrepancy of 60° resulted in complete shuttering with partial shuttering of the renal arteries. Attempts to correct shuttering of the SMA with a balloon expandable stent resulted in partial crushing of the stent. In the longitudinal direction, discrepancies in vessel separation between -5 and 8mm were tolerated without compromising target vessel patency.

Conclusion

Fenestrated stent-grafts appear to tolerate considerable mismatch with aortic anatomy without compromising seal. Additional factors such as the effect of mismatch upon deployment and durability of target-vessel stents merit further study.
Reducing the risk of spinal cord ischaemia following endovascular repair of thoracoabdominal aneurysms - the ‘sac perfusion branch’
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Objective
Spinal cord ischaemia (SCI) is one of the most feared complications following repair of thoracoabdominal aneurysms (TAAA). The greatest risk is in the early postoperative period during episodes of cardiovascular instability. The aim of this study is to describe our early experience of a technique for maintaining perfusion of the intercostals and lumbar vessels in the early postoperative period following endovascular repair of Type II TAAA with branched stent-grafts.

Method
Patients underwent staged endovascular repair of TAAA. Perfusion of the intercostal vessels was maintained in the early postoperative period by a temporary controlled endoleak achieved with ‘sac perfusion branches’ added to custom-made stent-grafts. The repair was then completed 7-10 days later by percutaneous closure of the branches.

Results
There were five males and two females, mean age 74, with a mean aneurysm diameter of 6.8cm. Standard precautions for the prevention of SCI were taken, including spinal drains. One patient developed monoparesis of the right leg during a period of hypotension secondary to a cardiac event and succumbed within 30 days. Of the remaining patients, there was no permanent paraplegia. One developed lower limb weakness following closure of the perfusion branches, with recovery following CSF drainage and revascularisation of the left subclavian. There have been no further complications or re-interventions.

Conclusion
Controlled perfusion of the intercostal vessels with a temporary controlled endoleak is feasible. Sac perfusion branches may be a useful adjunct to prevent SCI, providing protection to spinal cord perfusion during the immediate postoperative period, when the risk of cardiovascular instability is greatest.
Stent-graft limbs deployed into the external iliac artery are at increased risk of occlusion following EVAR  
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Objective  
Stent-graft limb occlusion is a common reason for re-intervention following endovascular aneurysm repair (EVAR). We assessed whether deployment of the endograft limb in the external iliac artery (EIA) increased the rate of limb occlusion.  

Method  
A prospectively maintained database of infra-renal EVAR procedures carried out in a single centre was analysed for stent-graft limb occlusions.  

Results  
A total of 661 EVAR procedures were carried out between 1996-2010. In 567 patients (56 female), both endograft limbs were deployed in the common iliac artery (CIA) leaving 94 patients (9 female) with at least one limb in the EIA. An adjunctive bare metal stent was used in 8 (9%) limbs deployed in the EIA. Seventeen (3%) limbs occluded in the CIA group compared with 14 (15%, p<0.0001) in the EIA group. The time to occlusion was 3 months (0-60) and 1 month (0-36) in the CIA and EIA groups, respectively. Limb occlusions were treated by femoro-femoral bypass (CIA group n=16, EIA group n=9), axillo-femoral bypass (CIA group n=1, EIA group n=2) and limb thrombectomy (EIA group only n=3). No legs were amputated following occlusion of a limb placed in the CIA but there were three amputations in the EIA group (p=0.045). One patient died in each group.  

Conclusion  
Deployment of endograft limbs into the EIA led to a higher rate of occlusion and leg amputation. Increased tortuosity of the EIA, a smaller calibre vessel and the loss of internal iliac artery run-off are likely to account for the increased risk.
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Endovascular repair of abdominal aortic aneurysms (AAA) with short and/or angulated necks: infra-renal sealing is not a safe option
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Objective
The options for endovascular repair of aortic aneurysms with a neck length of <15mm and angulation of >60° (Cook Medical Inc. guidelines, 2003) are: 1) off-label use of an infra-renal device; and 2) use of a fenestrated endograft to extend the sealing zone proximal to the level of the renal arteries. In this paper we report the results of treatment of aneurysms with adverse necks using infra-renal grafts.

Method
A large international EVAR registry database was interrogated to identify a population of patients that met the above definitions of 'adverse neck'. Outcomes were assessed specifically with respect to early Type 1 endoleak.

Results
Data from a total of 11,208 patients having endovascular repair of abdominal aortic aneurysms were reviewed. We identified 672 patients with a neck length of 14mm or less and 2356 patients with angulation of >60°. The total mean aneurysm diameter was 57.8mm; mean diameter for short neck aneurysms was 59.9mm and 62.6mm for angulated necks. Overall, 2.8% of patients developed a proximal type 1 endoleak. The incidence of endoleak rate was 5.3% in angulated necks, 7.6% in short necks and 12.7% in aneurysms with both short and angulated necks.

Conclusion
Endovascular repair of abdominal aortic aneurysms with adverse necks using standard infra-renal endografts is associated with an unacceptable risk of proximal type 1 endoleak. The use of fenestrated endografts to extend the sealing zone proximal to the renal arteries is likely to be a better option for these patients.
Improving standards of care in AAA surgery: the patients’ perspective
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Objective
The national QIP seeks to drive up the standard of care provided to patients with AAA. An important component of this work is a better understanding of patients’ views.

Method
Patient groups were convened in five regions of the UK. A semi-structured focus group methodology was used to define important areas for improvement.

Results
There were clear concerns across all patient groups about the lack of national assessment and treatment protocols (i.e. a national care pathway). Patient groups wish to see a consistent approach nationally to assessing the risks and benefits of surgery for individual patients. Communication from medical teams was felt to be inadequate particularly around postoperative care. There was perceived to be a lack of information, and that provided was over optimistic with regard to recovery from open surgery. Uncorrected unrealistic expectations led to anxiousness and discouragement in a slower recovery. There were conflicting views about centralization that mirror the current debate about configuration of vascular services. Patient groups were supportive of the development of networks as a means for managing complex cases by pooling expertise. There was interest in surgeons using simulators to practice difficult cases pre-operatively.

Conclusion
Vascular surgeons have much to gain from wider patient involvement in planning care. We believe that patient groups have a central role in defining national standards of care.
Use of baseline factors to predict serious complications and re-interventions after endovascular aneurysm repair (EVAR) in patients with a large abdominal aortic aneurysm: results from the UK EVAR trials

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Objective

Data from the UK EndoVascular Aneurysm Repair (EVAR) trials were used to identify which factors are associated with the rate of graft-related complications and re-interventions after EVAR in patients with a large abdominal aortic aneurysm.

Method

Patients randomised to EVAR in either trial 1 or 2 were included providing they had undergone elective EVAR within 6 months of randomisation. Patients were followed by CT scans inspected by trial radiologists for graft-related complications and re-interventions. Analyses were timed from EVAR deployment and Cox regression (stratified by trial 1 or 2) was used to investigate whether any pre-specified anatomical or demographic factors were associated with time to first serious complication or re-intervention (type 2 endoleaks excluded).

Results

A total of 756 patients (588 EVAR trial 1) were followed for an average of 3.7 years, during which time there were 179 serious graft complications (rate 6.5 per 100 person years), and 114 re-interventions (rate 3.8 per 100 person years). The highest rate was during the first 6 months with an apparent increase again after 2 years. Multivariate analysis indicated that graft-related events increased significantly with larger aneurysm diameter (p<0.001) and older age (p=0.04). There was weaker evidence that patients with larger common iliac diameters experienced elevated complication rates.

Conclusion

Graft-related complication and re-intervention rates are high after EVAR in patients with a large aneurysm but younger patients and those with aneurysms closer to the 5.5cm threshold for intervention experience lower rates. This bodes well for patients whose aneurysms are monitored in screening programmes until referral for repair at 5.5cm.
Acute aortic syndrome (AAS) treated by thoracic endovascular aortic repair (TEVAR)
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Objective
Acute aortic syndrome (AAS) is a life-threatening condition and heralds imminent aortic rupture. Stent-graft repair offers a minimally invasive treatment solution and is becoming established as the treatment of choice for these patients. The objective of this study was to evaluate the safety and mid- to long-term efficacy of thoracic endovascular repair for AAS.

Method
Consecutive patients presenting with AAS and treated with TEVAR were prospectively enrolled in a database and followed up at 3 months, then annually thereafter. Death, stroke, paraplegia and secondary intervention were documented.

Results
110 TEVAR were performed for AAS between 1997-2010. Seventy-five were men and 35 were women. The median age was 68 (18-90). Fourteen patients died (12.7%). The pathologies treated included acute complicated dissection (34 patients; 3 deaths); symptomatic aneurysm (29;5); infected aneurysm (16;3); transection (14;1); chronic dissection (8;1); others (9;1). The causes of death were aortic rupture (5), myocardial infarction (4), stroke (3) and sepsis (2). Seven (6.4%) patients became paraplegic with one death; 8 (7.3%) patients had a stroke with three deaths. The median follow-up was 44 months (1-153). Eleven patients (10%) had secondary procedures: 8 for Type I endoleak (3 conversion to open repair, 1 further stent-graft, 3 extension cuffs, 1 aortic banding), 2 for ongoing aortic infection (2 further stent-grafts) and 1 for aortic rupture (further stent-graft).

Conclusion
Acute aortic syndrome can be treated with thoracic endovascular repair with acceptable mid- to long-term results. The secondary procedure rate demands vigilant postoperative clinical and imaging surveillance.
Use of CO\textsubscript{2} angiography for fenestrated endovascular aneurysm repair

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Objective
The development of fenestrated and branched EVAR is associated with increased usage of contrast and a significant incidence of postoperative renal dysfunction and renal failure has been reported. We describe the use of CO\textsubscript{2} as the primary contrast agent in patients undergoing complex EVAR.

Method
Two cohorts of patients undergoing fenestrated and branched EVAR were compared at a regional vascular unit. Sixty-one complex endografts were implanted between 2008 and 2010; 38 procedures were completed with only iodinated contrast media (group 1) and 23 utilised CO\textsubscript{2} as the primary contrast agent (group 2). The endpoint assessed was renal impairment, defined as an increase in creatinine of >25%.

Results
Baseline creatinine was similar between group 1 (mean 109, range 44-282) and group 2 (mean 117, range 74-310). There was no significant difference in the incidence of postoperative renal dysfunction; however, 7 (18%) in group 1 required temporary dialysis, compared to just 2 (9%) in group 2 (p=0.056). No patients required permanent dialysis. Further analysis between the groups demonstrated a reduction in mean volume of contrast used from 234ml to 138ml.

Conclusion
Renal impairment is a common postoperative complication amongst patients undergoing complex EVAR. The incidence of dialysis-dependent renal impairment is reduced by the use of CO\textsubscript{2} as a contrast medium.
Radiation exposure during endovascular treatment of the aorta: increased risk with complex repairs

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Objective
Exposure of the skin to radiation doses above 2 Gray (Gy) can cause burns. There is also an estimated excess lifetime cancer risk of 6% for every Sievert (Sv) of radiation absorbed. We measured patient radiation exposure during endovascular aortic procedures.

Method
Consecutive thoracic (TEVAR), infra-renal (IEVAR) and branched/fenestrated (BEVAR/FEVAR) aortic repairs carried out between 2003-2010 in a single centre were assessed. Indirect measurements of radiation dose (dose area product [DAP] and fluoroscopy time) were prospectively recorded with the C-arm dosimeter. Direct measurements of exposure at skin level [Gy] were obtained using Gafchromic dosimetry film. A whole-body effective dose (Sv) was calculated from DAP using PCXMC software.

Results
The TEVAR cohort (n=232, age 71[15-89]), which included patients treated for aortic transection and dissections, were younger (p<0.0001) than BEVAR/FEVAR (n=53, age 76[58-85]) and IEVAR (n=630, age 76[37-93]). Median DAP was higher (p=0.004) in BEVAR/FEVAR compared with IEVAR and TEVAR: 32,060cGycm² [17,207-213,322] vs 17,300cGycm² [10,940-334,340] vs 19,440cGycm² [11,284-35,101], respectively. The equivalent skin doses were BEVAR/FEVAR: 1.3Gy (0.7-8.7); IEVAR: 0.7Gy (0.44-13.7); TEVAR: 0.8Gy (0.46-1.4). The effective whole-body doses were BEVAR/FEVAR: 0.096Sv (0.052-0.64); IEVAR: 0.053Sv (0.033-1.00); TEVAR: 0.058Sv (0.034-0.11). The skin dose exceeded 2Gy more often during BEVAR/FEVAR (31% of patients) compared with TEVAR (11%, p=0.005) and IEVAR (11%, p=0.001). The whole-body dose was >1Sv in 4 patients (IEVAR=3, TEVAR=1).

Conclusion
Endovascular aortic procedures, in particular complex repairs, carry a substantial risk of skin damage. The excess malignancy risk is relatively small. Intra-operative image registration techniques should help to reduce the radiation dose.
Assessment of National Vascular Database (NVD) quality
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Objective
The NVD collects information on demographics and patient outcomes. Use for research is dependent on the ability to adjust for case-mix, which in turn depends on the completeness of data collected. We present data imputation techniques that allow missing data to be imputed (predicted) using the patient’s known characteristics.

Method
AAA patients were selected from the NVD and were analysed for data completeness for the preferred and required fields. We have developed a model to predict the missing data by multivariate imputation by chained equations (MICE). The vascular biochemistry and haematology outcome model (VBHOM) was calculated on: (1) only patients with a complete data set; and (2) all patients using MICE imputation.

Results
14,010 patients in the NVD-AAA data show that there is a range of missing data across variables (median 22%, interquartile range 10-64%). In particular, volume of cell salvage transfused, postoperative infections, and cardiovascular and renal events had missing data in excess of 50%. Geographical variation in missing data shows little variation amongst required fields (7%, 7-8%), but marked variation across fields not described as required or preferred (40%, 37-48%) and even greater variation amongst preferred fields (11%, 6-22%).

Conclusion
This analysis has identified hospitals with good data entry practices, and provides reassurance on the data quality in required fields; however, it also allows: (1) review of database design; and (2) the targeting of incomplete variables as a focus for the AAA QIP. Work is in progress to assess the effect of missing data on outcome models such as VBHOM.
Modifying the illness and treatment beliefs of patients with intermittent claudication increases daily walking and reduces demand for vascular intervention - results from a randomised controlled trial

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Objective
Supervised exercise has been shown to increase pain-free walking distance and reduce symptoms of claudication. However, supervised exercise programmes are not widely available, require patient commitment to attend, and may not lead to lasting behaviour change beyond attendance at the programme. This trial (ISRCTN28051878) studied whether a brief psychological intervention to modify patients’ beliefs about peripheral arterial disease and beliefs about walking would lead to increased walking and a reduction in the demand for vascular intervention.

Method
Sixty patients newly diagnosed with claudication were randomised into two conditions. The control condition received usual care, and the treatment condition received usual care and a brief psychological intervention to modify illness and walking beliefs, and develop a walking action plan. Participants were followed up after 4 months. Daily steps were measured by pedometer.

Results
Participants in the intervention group had significantly increased (p<0.01) mean daily steps, while participants in the control group had decreased mean daily steps from baseline to follow-up. Participants in the control group were four times more likely (p=0.009) to opt for vascular intervention than participants in the psychological intervention group.

Conclusion
This trial demonstrates that a brief psychological intervention for patients with claudication can increase daily walking, and reduce the demand for surgery at this stage of the disease. This has implications for the design of services to treat patients with intermittent claudication.
Modelling the effect of venous disease upon quality of life
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Objective
A clear understanding of the relationship of venous reflux, clinical venous disease and the effects upon quality of life (QoL) has eluded researchers. This study aims to illustrate the impact of venous disease and observe the incremental direct effect upon QoL. Commissioning bodies are progressively withdrawing funding for interventions; evidence of the impact of venous disease must be sought.

Method
Consecutive patients were assessed and those with isolated, unilateral, single superficial axial incompetence were included. Clinical grading was performed with the CEAP and Venous Clinical Severity scores (VCSS). Patients completed generic (SF36 and EQ5D) and disease-specific (Aberdeen Varicose Vein Questionnaire - AVVQ) QoL instruments. Multivariate regression modelling was performed, controlling for demographic and anatomical factors, to elucidate the association of clinical severity upon QoL impairment.

Results
456 patients with C2-6 disease were included along with control data for 105 people with C0-1 disease. Increasing clinical grading corresponds strongly with deterioration in disease-specific QoL (p<0.001). This is stratified into C0-1, C2-4 and C5-6 disease (p<0.001-0.006). Increasing clinical grading also corresponds with deterioration in the physical domains of SF36 (p<0.001-0.002), along with index utility scores (SF6D and EQ5D p<0.001). This was apparent from C2 disease, where the reported impact of pain was comparable with reference cases suffering with angina, whereas the physical impairment seen with ulceration was comparable with that seen in congestive cardiac failure and COPD.

Conclusion
Significant demonstrable morbidity is seen even with uncomplicated venous disease. This provides strong support for the continued funding of interventions to address this QoL impairment.
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A prospective double-blind randomised controlled trial of radiofrequency versus laser treatment of great saphenous varicose veins
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Objective
Endovenous ablation of varicose veins using radiofrequency (RFA) and laser fibres (EVLT) has reported advantages over traditional open surgical treatment. There is little evidence comparing the efficacy and patient-reported outcomes between the two endovenous solutions. This study compares the RFA and EVLT strategies in a prospective double-blind clinical trial.

Method
Consecutive patients with primary unilateral great saphenous vein (GSV) reflux undergoing endovenous treatment were randomised to RFA or EVLT. The primary outcome measure was GSV occlusion at 3 months following treatment. Secondary outcome measures were occlusion at 7 days, postoperative pain, analgesic requirement and bruising, assessed at day 7 following surgery. Quality of life (QoL) was assessed pre-operatively and 3 months following surgery using the Aberdeen Varicose Vein Questionnaire (AVVQ) and EQ-5D.

Results
159 patients were randomised to RFA (79 patients) or EVLT (80 patients). Groups were well matched for demographics, disease extent, severity and pre-operative QoL. Duplex scanning confirmed 100% vein occlusion at 1 week in both groups. At 3 months, occlusion was 97% for RFA and 96% for EVLT; p=0.67. Median (IQR) percentage above-knee bruise area was greater following EVLT 3.85% (6.1) compared to RFA 0.6% (2); p=0.0001. Postoperative pain assessed at each of the first 7 postoperative days was less after RFA (p=0.001). Changes in the AVVQ (p=0.12) and EQ-5D (p=0.66) at 3 months were similar in both groups.

Conclusion
RFA and EVLT offer equivalent venous occlusion rates at 3 months following treatment of primary GSV varices. RFA is associated with less peri-procedural pain, analgesic requirement and bruising.
**Abstracts**

**Combined medical therapy and carotid endarterectomy for asymptomatic stenosis: 10-year stroke prevention in ACST-1**  
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**Objective**
In a randomised trial (ACST-1) of 3120 asymptomatic patients with severe carotid narrowing comparing early with deferred carotid endarterectomy (CEA), CEA significantly reduced 10-year stroke risk. All other aspects of patient treatment were left to the discretion of the clinician. Throughout the trial antithrombotic and antihypertensive drugs were widely used but use of lipid-lowering therapy increased substantially. We examined the influence of different medical treatments on the overall stroke prevention effect of the trial.

**Method**
Use of lipid-lowering, antithrombotic and blood-pressure-lowering medical therapies was recorded at randomisation, 4 months and at yearly intervals for 10 years. Time-dependent analysis of event rates was compared between trial groups.

**Results**
From 1993 to 2003, antiplatelet drugs were taken by around 90% patients (with another 8-9% taking anticoagulant treatment instead), and most (~90%) patients were taking blood-pressure-lowering agents by the end of follow-up. Lipid-lowering therapy usage increased rapidly (to >80%). Stroke rate ratio (immediate CEA vs deferred) appeared similar for those on all three therapies and those not, but because absolute stroke rates were lower among those on lipid-lowering therapy, absolute difference in annual stroke rate for those allocated immediate CEA was not as great. Overall, stroke risk was reduced by about a third and, as with other analyses, there was additional (~6% ARR) stroke prevention benefit in those who had early CEA.

**Conclusion**
Event rates among those currently on lipid-lowering therapy suggest somewhat lower peri-operative risks and lower absolute benefits, but still with a significant reduction in net risk at year 10 (9.6% vs 14.5%, absolute difference 5.0%).
Clinical assessment pre-CABG identifies patients at risk for postoperative stroke
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Objective
The management of incidental carotid stenosis in patients undergoing coronary artery bypass grafting (CABG) remains controversial. Our cardiac unit refers patients for carotid duplex if they have one or more of: bruit, known cerebrovascular disease, known stenosis or amaurosis fugax. The aim of this study was to investigate the benefit of such a policy.

Method
Patients undergoing CABG without valve surgery over a 3-year period (2007-9) were identified. Patients undergoing carotid duplex scanning, carotid interventions, and patients who suffered a peri-operative stroke (not TIA) were identified from vascular and stroke databases.

Results
3076 patients (2368 male; median 68 years; range 35-88) underwent CABG. 105 patients (3.4%) were referred for carotid duplex (89 male; median 70 years; range 49-85); of these, 8% were normal carotid arteries, 70% had <75% carotid stenosis, 17% had >75% carotid stenosis and 6% had carotid occlusion. Seven patients with >75% carotid stenosis (7/18 identified) underwent endarterectomy, one pre-CABG and six combined procedures. Thirty-nine patients (1.27%) suffered a stroke within 30 days of surgery. The stroke rate was 8/105 (7.6%) in scanned versus 31/2971 (1.04%) unscanned patients (Chi-squared p<0.0001). There were no strokes in CEA patients. Nor did any patient with non-operated >75% stenosis suffer a stroke.

Conclusion
Simple clinical assessment identified a patient group at increased risk of CABG-related stroke. As no stroke patient in this group had a >75% carotid stenosis, strategies other than CEA are required to reduce this stroke rate.
The burden of carotid endarterectomy (CE) complications - underneath stroke and death

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Objective
CE is increasingly undertaken urgently and has potential significant complications. Our aim was to analyse the complication profile of CE especially in relation to undertaking the procedure urgently - rapid access carotid endarterectomy (RACE).

Method
Data were collected prospectively on consecutive CEs recording age, sex, presentation, anaesthetic, operation, surgeon, and outcome. Patients were evaluated by stroke physicians routinely pre and post-op at 30 days, 6 months and 1 year. All complications were meticulously recorded and classified.

Results
There were 255 CEs (166 elective; 89 RACE) from August 2007 to August 2010 and 84% were uncomplicated. Complications included death 3 (1.2%; 1 within 30 days from a bleeding DU [RACE], 2 after 30 days from an MI [RACE] and chest infection after fits from hyperperfusion syndrome-elective), peri-op stroke 3 (1.2%) and TIA 2 (0.8%), hyperperfusion syndrome 3 (1.2%), haematoma 9 (3.6%; 5 drained, 4 managed conservatively), nerve injury to the hypoglossal nerve 6 (2.3%) and recurrent laryngeal nerve 5 (1.9%), intra-operative complications 4 (1.6% which were prolonged bleeding, fits and conversion to GA) and other 6 (2.4% such as hyper/hypotension, arrhythmia, urinary retention, etc). There was no significant difference between elective CE and RACE for death or individual complications but there was significant difference (p=0.04; Fisher’s Exact Test) with relatively more minor local/general complications in elective 21 (Vs RACE 9) and more significant complications/death in RACE 8 (Vs Elective 2).

Conclusion
These findings have important implications for consent, considering that even with a 30-day death rate of 0.4% and stroke rate of 1.2%, still 16% of patients experienced some complication. Not unexpectedly, RACE had more significant complications but rates were low making it still worth undertaking.
Rate and predictability of graft rupture after endovascular and open abdominal aortic aneurysm repair: data from the EVAR trials
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Objective
Graft rupture after EVAR has been reported, often preceded by graft-related complications. Graft rupture also has been reported after open repair. The aim was to assess rate and factors associated with rupture after endovascular (EVAR) or open repair (OR) of abdominal aortic aneurysm.

Method
By July 2009, a total of 848 elective EVARs and 594 elective ORs had been performed in the United Kingdom EVAR trials 1 and 2. Patients have been followed for complications, re-interventions and rupture. The incidence of rupture was explored in relation to baseline anatomy and subsequent complications in a Cox regression analysis.

Results
There were no ruptures in the OR patients. A total of 27 ruptures occurred after EVAR during a mean follow-up of 4.8 years: crude rate = 0.7 [95% CI 0.5-1.0] ruptures per 100 person-years. Eighteen patients (67%) died within 30 days of rupture. Five ruptures occurred in the first 30 postoperative days and 22 after that: crude rates of 7.2 [95% CI 3.0-17.4] and 0.6 [95% CI 0.4-0.9] per 100 person-years, respectively. Previous complications (endoleak types 1, 2 with sac expansion, 3, migration or kinking) increased the risk of rupture, adjusted hazard ratio 8.83 [95% CI 3.76-20.76], p<0.0001.

Conclusion
There were no ruptures after OR and a low rate after EVAR. Mortality after graft rupture is high and previous serious complications are significantly associated with the risk of rupture. Few ruptures after EVAR appear to be spontaneous without complications identified during optimal surveillance.
A prospective study of the natural history of deep vein thrombosis: early predictors of poor late outcomes
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Objective
A proportion of patients with deep vein thrombosis (DVT) will develop post-thrombotic syndrome (PTS). Currently, the only clearly identified risk factors for developing PTS are recurrent ipsilateral DVT and extensive proximal disease. It is difficult to predict early on which patients will run into trouble. The aim of the study was to find better predictors of poor clinical outcome following a DVT.

Method
Patients with suspected acute DVT in the lower limb were assessed prospectively. All patients with a confirmed DVT were asked to participate in this study. Within 7-10 days following diagnosis of DVT, patients underwent a further review, involving clinical, ultrasound and air plethysmography assessment of both lower limbs. Patients were re-assessed at regular intervals for 5 years.

Results
122 limbs in 114 patients were enrolled. Thrombus regression occurred in two phases, with a rapid regression between 10 days and 3 months, and a more gradual regression thereafter. Four risk factors for PTS were identified as best predictors: clot load on presentation (>10), at 6 months <50% lysis, venous filling index >2.5mL/sec and abnormal outflow rate (<0.6). Patients with 3 or more of these risk factors had a significant risk of developing PTS with sensitivity 100%, specificity 80% and positive predictive value 55%. Patients scoring 2 or less were normal.

Conclusion
This is the first study which has shown that venous assessment at 6 months post-DVT can predict PTS at 5 years. Those who will not develop PTS can be reassured of this at 6 months.
Flow-sensitised dynamic magnetic resonance imaging (MRI) can identify dominant false lumen flow and secondary entry tears in type B aortic dissection: implications for endovascular treatment

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**Objective**

Endovascular repair is established as the treatment of choice for complicated type B dissection. Current imaging can identify primary but not secondary entry tears making device length and accurate placement difficult. This may result in continued false lumen perfusion requiring multiple re-interventions to prevent aortic dilation and rupture. The objective was to visualise and quantify blood flow in the true and false lumens to determine the location and contribution of entry tears in type B aortic dissection.

**Method**

Twenty patients with type B aortic dissection underwent magnetic resonance imaging at 3T. High resolution 3D anatomy images (inversion-recovery-3D-SSFP [resolution=1.0mm³, FA=20°, TI=350ms, TR/TE=4.0/1.3ms, TFE-factor=22]) were obtained using gadofosveset trisodium. Multi-directional blood flow information was acquired using time-resolved 3-directional phase contrast MRI (FA=10°, TR/TE=5.0/2.7ms, 25 cardiac-phases, VENC[FH/RL/AP]=150cm/s). Flow-velocity time curves were derived using semi-automatic segmentation algorithms.

**Results**

The primary entry tear was identified in all patients. The velocity of blood flow was greater in the true lumen compared to the false lumen (mean difference 16.58cm/s, p<0.0001 [95% CI 13.58-19.57]). In six patients (30%) the false lumen was the dominant flow system. In all patients false lumen flow was complex with high levels of backward and regurgitant flow (mean regurgitant fraction [%][SD]=29.42[27.13]). Changes in false lumen blood volume and regurgitant fraction indicated the position and contribution of secondary entry tears.

**Conclusion**

Flow-sensitised dynamic MRI is able to identify dominant false lumen flow and the location and contribution of entry tears in type B aortic dissection. This information will improve endovascular device sizing and placement, and result in improved clinical outcome.
Further evidence for the role of patch angioplasty (PA) over primary closure (PC) during carotid endarterectomy (CEA)
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Objective
Despite concerns about data quality/poor trial design the 2009 Cochrane review concluded that PA (n=1076) compared to PC (n=932) reduces ipsilateral peri-operative stroke (IS) risk with trends towards lower ‘any stroke’ (AS) and all-cause mortality (ACM) during CEA. This has been investigated further in an unrelated RCT of 3438 CEA patients with symptomatic or asymptomatic carotid stenosis.

Method
A 30-day independent neurological review assessed primary outcome events. Risk ratios were calculated comparing PA (n=1588) with PC (n=1850: 985 conventional [PCC], 712 eversion [PCE], 153 unspecified [excluded]). PA and PC data were then combined with Cochrane data.

Results
There were no differences in demographic data, ASA status, carotid stenosis, comorbidities, and anti-thrombotic use between the groups. Operative time was less for PC (81 v 107 min, p=0.001). Odds ratios (±95% confidence intervals) for PA versus all PC suggested no benefit for PA (IS: 0.98, 0.66-1.45; AS: 0.94, 0.66-1.33; ACM: 1.76, 0.97-3.17 [trend favouring PC]). However, when PCC (IS: 0.82, 0.52-1.28; AS: 0.82, 0.52-1.23) and PCE (IS: 1.19, 0.70-2.03; AS: 1.26, 0.78-2.03) are considered separately, trends favour PA over PCC and PCE over PA. Combining PA and PCC with Cochrane data strengthens the role for PA (IS: 0.62, 0.42-0.90, p=0.016; AS: 0.73, 0.52-1.02) in stroke reduction although the trend for PCC reducing ACM (1.09, 0.6-1.95) persists.

Conclusion
When data from a much larger study (with independent neurological review) is combined with the Cochrane data, the role for PA over PCC is stronger. However, PCE may offer similar benefits to PA with shorter operating times.
Distal bypass grafts in patients with critical leg ischaemia with poor pedal arch

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Objective
To evaluate the amputation-free survival and patency rates of distal bypass grafts in critical leg ischaemia (CLI) in the presence of a complete or incomplete pedal arch.

Method
A retrospective analysis of all patients with CLI undergoing distal bypass between January 2004 and April 2010 was conducted. Kaplan-Meier analysis was used to assess and compare amputation-free survival and patency rates at 12 months.

Results
129 consecutive patients (98 men, median age 76, range 19-96) underwent 144 distal bypasses. The incidence of diabetes mellitus and renal failure was 69% and 29%, respectively. 92% had a vein conduit and 8% a PTFE + Miller cuff. Out of 144 bypasses, 24 (17%) had a complete pedal arch (CPA), 40 (28%) had a dorsal pedal arch (DPA) only, 39 (27%) had a plantar pedal arch (PPA) only, 27 (19%) had no pedal arch (NPA), and 14 (10%) of cases could not be assessed due to inadequate images. In-hospital mortality was 2.1%. The amputation-free survival (AFS), primary, assisted primary and secondary patency rates for all four pedal arch groups at 1 year were: CPA: 84%, 63%, 95% and 95%; DPA: 73%, 61%, 88% and 88%; PPA: 84%, 52%, 84% and 90%; NPA: 78%, 52%, 87% and 87%.

Conclusion
Amputation-free survival and patency rates are comparable in all groups. The authors would therefore advocate distal bypass grafts in patients with CLI in the presence of a complete or incomplete pedal arch.
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*All products and indication are subject to local regulatory approval.
The risk of occlusion and associated events in the Asymptomatic Carotid Surgery Trial: a 10-year prospective study
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Objective
This study analyzes the risk of occlusion and associated neurological events in patients with severe asymptomatic carotid artery stenosis included in the ACST-1 trial.

Method
In ACST-1, 3120 patients were randomised between immediate surgery or deferral of CEA. During the study 198 patients developed occlusion of the internal carotid artery and we evaluated the associated neurological events. Patients with contralateral occlusion at baseline were excluded from analysis. Kaplan-Meier analysis was performed to estimate freedom from occlusion and occlusion-related stroke-free survival.

Results
Mean follow-up was 80.8 months (range 0-165 months); 144 ipsilateral and 54 contralateral occlusions (immediate: 38 vs. 33, p<0.01) occurred in 198 patients. Occlusion-free survival rates at 1, 5 and 10 years were 98%, 95% and 94% in the immediate CEA group, and 97%, 91% and 88% in the deferred CEA patients. The likelihood of occlusion was significantly greater (p<0.01) in the deferred group. Risk of symptomatic occlusion after 5 and 10 years was lower (0.5% and 0.7% versus 1.7% and 2.5%, respectively) in the immediate versus deferred groups. Nineteen patients developed an occlusion-related stroke. The overall stroke-free survival rate in patients with occlusion at 1, 5 and 10 years was 99.5%, 96.7% and 86%, respectively.

Conclusion
This long-term follow-up analysis shows that occlusion occurred more often in patients in whom CEA was deferred. Overall stroke-free survival did not differ between patients with or without occlusion.
Laparoscopic aortic aneurysm surgery: early experience from three UK vascular centres
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Objective
Laparoscopic aortic aneurysm surgery is an alternative or adjunct to open or endovascular repair and may confer certain advantages. We report our early experience of laparoscopic aortic aneurysm surgery.

Method
Peri-operative and postoperative data and outcomes were recorded prospectively on consecutive patients undergoing elective laparoscopic-assisted or total laparoscopic aortic surgery at three vascular centres.

Results
Following a period of formal mentorship, 64 patients with infra-renal aneurysmal disease including 43 aortic and 21 aortoiliac (6 juxtarenal) aneurysms, underwent surgery. Sixty were male with a median age of 72 years (range 58-88). Median aortic diameter was 6.0cm (IQR 5.7-6.5). Thirty-eight patients had laparoscopic-assisted surgery (2 required conversion to an open procedure) and 26 had total laparoscopic surgery. Median aortic clamp time was 95 minutes (IQR 75-126) with a median operative time of 355 minutes (IQR 315-395). Median postoperative epidural requirement was 1 day (range 0-3); median time to return to solid diet was 1 day (range 1-6) and median time to mobilisation was 1 day (range 1-4). Median postoperative hospital stay was 6 days (range 2-98). One patient died within 30 days (1.6%) and 13 (20%) developed early complications. Following a median follow-up of 24 months, 4 developed late complications (4 incisional herniae) with no late graft-related complications seen.

Conclusion
Laparoscopic aortic aneurysm surgery may be performed with a low peri-operative mortality and good early durability. By comparison with open surgery early operative times were long, epidural usage was limited, recovery of gut function and mobility was early and length of stay was short.
Renal function in patients following open repair of Type IV thoracoabdominal aneurysms: long-term results
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Objective
Limited longitudinal data exist on the effect of open repair of suprarenal aortic aneurysms on renal function.

Method
Consecutive patients undergoing repair of Crawford Type IV thoracoabdominal aneurysms over a 10-year period were included in a retrospective cohort study. Pre-operative, discharge and most recent estimated glomerular filtration rate (eGFR) were examined alongside survival.

Results
Eighty-two patients of median (range) age 69 (21–77) years underwent aneurysm repair. Five (6%) patients died in hospital and a further 19 (23%) died over a median (range) follow-up of 25 (1-125) months. Pre-operatively, 45 (56%) patients had an eGFR >60ml/min, 36 (43%) patients had an eGFR 15-60ml/min and one (1%) patient an eGFR of <15ml/min. On discharge, 42 (54%) patients had an eGFR >60ml/min, 33 (42%) had an eGFR 15-60ml/min and three (4%) had an eGFR <15ml/min. During follow-up, 11 (26%) of the 42 patients with an eGFR >60ml/min on discharge died, 24 (57%) were still alive with no change in renal function, 6 (15%) had deteriorated to an eGFR 15-60ml/min, and 1 (2%) had an eGFR <15ml/min. Of 32 patients with an eGFR 15-60ml/min on discharge, 6 (19%) died, 7 (22%) returned to an eGFR >60ml/min, and the remaining 19 (59%) patients had an eGFR 15-60ml/min. Two (67%) of the three patients discharged with an eGFR <15ml/min died, and the other recovered to an eGFR 15-60ml/min.

Conclusion
Open suprarenal aneurysm repair can be performed safely with minimal adverse effect on long-term renal outcome.
Assessment of scoring for high-risk patients undergoing endovascular aneurysm repair
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Objective
The 10-year follow-up results of the EVAR 1 and EVAR 2 trials have shown no difference in all-cause mortality when the patients are followed up over a longer period. Similar results were seen in the DREAM trial. These results highlight that there are some high-risk patients who may not be benefiting from endovascular intervention. A group from New York has suggested scoring to define this high-risk group based on comorbidities (age, sex, renal failure, peripheral vascular disease, pulmonary, cardiac and neurological problems) and experience. We have applied this scoring to our patients who underwent EVAR to assess the validity of this scoring system.

Method
We retrospectively analysed all the patients who had an EVAR for an infra-renal aneurysm and scored them on the proposed scoring.

Results
202 patients had EVAR between December 1997 and March 2010. Overall 30-day mortality was 3.96%. 96% of the patients had a score of 9 or less. 30-day mortality for patients with scores of 0-4 was 1.4%, for a score of 5-8 was 7.7%, for a score of 9-10 was 11.1% and for a score of 11 or more was 33.3%.

Conclusion
Although our numbers are small the results are very similar to the results from the Medicare data on which this scoring has been proposed. We therefore propose that this is a valid scoring system and should be considered to define high-risk patients undergoing EVAR.
A pre-operative model for predicting mortality risk in elective AAA surgery
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Objective
A reliable pre-operative prediction of risk for elective AAA repair would be valuable to surgeons and patients. A multivariate risk prediction model for 30-day mortality following elective open and endovascular abdominal aortic aneurysm (AAA) repair has been developed using the Vascular Governance North West (VGNW) database.

Method
Prospective data on 2765 consecutive patients undergoing elective open or endovascular AAA repair from Sept 1999-Oct 2009 was randomly split into development (n=1936) and validation datasets (n=829). Logistic regression analysis was undertaken using a forward-stepwise technique to identify risk factors for 30-day mortality.

Results
Variables associated with 30-day mortality (n=98, 5.1%) in the development dataset included: anti-platelet medication (p<0.001), female gender (p=0.002), open surgery (p=0.002), age (p=0.005), creatinine (p=0.006), diabetes (p=0.029) and respiratory disease (p=0.031). The receiver operating characteristic (ROC) curves for predicted probability of 30-day mortality in the development and validation datasets were 0.73 and 0.70, respectively. The model showed good calibration in both datasets. Observed versus expected 30-day mortality in the validation dataset (50 [6%] deaths) for low, medium and high-risk groups was 3.2% v 2.0%, (p=0.27), 6.1% v 5.1%, (p=0.67) and 11.1% v 10.7%, (p=0.88), respectively, with no significant difference between observed and expected mortalities.

Conclusion
This multivariate model predicted 30-day mortality following elective AAA repair across all risk groups particularly when mortality risk is high. Surgeons may find it useful to calculate patient-specific risk for case mix adjustment of their results and in the consent process. Validation against the National Vascular Database is now necessary.
Aortic aneurysm repair in octogenarians
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Objective
We report the results of abdominal aortic aneurysm (AAA) repair, postoperative complications and 30-day mortality, in octogenarians in the North West of England.

Method
Data were collected prospectively on all patients undergoing AAA repair at 22 hospitals between September 1999 and May 2010.

Results
AAA repair was performed in 575 patients aged >80 and 3314 patients aged <80. The mean AAA diameter was 7.0cm in octogenarians and 6.7cm in patients aged <80 (p<0.001). Elective repair was performed in 390 (68%) octogenarians, urgent repair in 51 (9%) and emergency repair in 134 (23%); mortality rates were 11.3%, 17.6% and 53.0%, respectively. These were significantly higher than the equivalent rates in patients aged <80 of 4.7%, 8.7% and 28.9% (p<0.001). Octogenarians were more likely to be female (25% v 16%) or have emergency surgery (23% v 18%), dyspnoea (35% v 28%), an abnormal ECG (39% v 29%), haemoglobin <13g/dL (42% v 29%), or creatinine levels >120µmol/L (31% v 19%) (p<0.05). They were less likely to have a history of ischaemic heart disease (34% v 38%) or to be taking a statin (30.8% v 41.4%) (p<0.05). Risk factors associated with increased 30-day mortality in octogenarians by multivariate analysis were: non-elective repair, supra-renal AAA and creatinine levels ≥150µmol/L. Compared to patients aged <80, octogenarians were more likely to suffer postoperative myocardial infarction (8.0%), respiratory failure (16.9%) and renal failure (13.9%) (p<0.05).

Conclusion
The risk of mortality and morbidity following AAA repair is higher in octogenarians. Careful patient selection is essential. The standard threshold of 5.5cm for elective AAA repair is inappropriate at this age.
Growth rate of very small aneurysms
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Objective
The UK national screening programme for aortic aneurysms has recommended no follow-up for aneurysms less than 30mm in AP diameter. The aim of this study was to examine the growth rate of aortic aneurysms up to 30mm diameter.

Method
A personal database, collected over 16 years, of 693 patients with a small aortic aneurysm <55mm was searched for those presenting with an aneurysm <31mm. The growth rate of this group was examined.

Results
Sixty-six patients had an AAA <31mm in AP diameter at first scan. All patients had at least two further measurements. Twelve aneurysms were <25mm, 35 were 25-27mm and 19 were 28-30mm at first scan. The growth rate for the whole group was 1.65mm/y (0-6.5mm) and for the three groups 1.42 (0-3.3), 1.85 (0.3-5.0) and 1.44mm/y (0-6.5), respectively. In only 2 patients did the aorta not grow at all. The rate of growth was not related to the diameter at first measurement being on average 6.32%, 7.08%, and 5.04%, respectively, for the three groups.

Conclusion
Small aortic aneurysms less than 31mm grow at much the same rate as those between 30-55mm. The UK aneurysm screening programme should reconsider its decision not to follow up this group of patients.
Early experience of the UK aneurysm screening programme
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Objective
The UK Multicentre Aneurysm Screening Study (MASS) provided evidence of the benefit from screening for abdominal aortic aneurysms (AAA). In view of these findings, the national AAA screening programme was recently introduced. We report our findings from the initial screening period in Southwest London, an early implementation screening site.

Method
A retrospective analysis was performed on data collected from all patients since the onset of the screening programme. Men aged 65 were invited to attend an ultrasound assessment as per the national protocol. Self-referrals (men aged >65) were also scanned.

Results
The total target population was 10,593. 6,091 males were invited between April 2009 and June 2010. 4,216 were screened, including 162 self-referrals (mean age 72.5 yrs). 2,037 (33.4%) failed to attend. Of those scanned, 4,136 (98.1%) had aortic diameters less than 3.0cm, including 24 (0.6%) with diameters 2.6-2.9cm. Seventy-five (1.8%) had aneurysms 3.0-5.4cm, and 5 (0.1%) had aneurysms 5.5cm and above. Aneurysms were identified in 1.7% of those invited, and 6.2% of the self-referral group. Of all 80 aneurysms, 79 were found in white males, while 1 (3.0cm) was found in a black male of Caribbean origin.

Conclusion
The prevalence of AAA is lower in this cohort than expected. Limiting invitations to 65-year-olds, a diverse patient population and the high rate of non-attendance are likely causative factors. Current aneurysm identification rates may have an impact on the cost-effectiveness of the screening programme, and further research may be justified to identify those 65-year-olds most likely to have an AAA.
Aneurysm screening results in North London - a world away from the MASS trial
C Forman, H Sales, O Trainor, G Hamilton, M Davis
Royal Free Hospital, London

Objective
In January 2009, the Royal Free Hospital began screening for AAA in advance of the national programme. The case for national screening was based, in large part, on the results of the 2002 MASS trial. Here we compare our data with that reported in the MASS trial.

Method
Men aged 65 in North London were identified by their GP practice and invited for an abdominal aortic ultrasound. In addition, men or women who fell outside the criteria or catchment area were allowed to self-refer for a scan.

Results
2346 patients were invited for scanning (1/5 self-referred). The mean age was 66.5 vs 69.2 in MASS. 93% were men over 65, 6% were men 60-64 (with a large skew to age 64), and <0.2% were women or men under 60. Ethnicity (where given) was 1337 white, 328 non-white (not given in MASS). The social deprivation categories included the more deprived than average vs the less deprived than average in MASS. A total of 1665 of 2346 were scanned (71%) vs 27,147 of 33,839 (80%) in MASS. Total aneurysms detected were 13 vs 1333 in MASS. The prevalence of AAA was 0.78% of scans vs 4.9% of scans in MASS (p<<0.01). The 99% CI of AAA prevalence was 0.13-1.43% vs 4.5-5.3% in MASS. The breakdown of positive scans by aneurysm size was: 3cm to 4.4cm: 7; 4.5cm to 5.4cm: 2; >5.5cm: 4 (prevalence 0.25%).

Conclusion
The prevalence of AAA is six-fold lower in North London than in the MASS trial population. Excluding men under 65 and women makes no meaningful difference to the analysis. The reasons for this are unclear. However, the difference certainly impacts on the cost-effectiveness of screening, and deserves further study.
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Prevalence of screen-detected AAAs in men aged 65 is decreasing; however, the prevalence of cardiac and respiratory diseases remains significantly higher in this group

S Penkar, S Druce, H Ashton, H Hafez
St Richard’s Hospital, Chichester

Objective
To study the prevalence of AAAs and major cardiac and respiratory diseases in men aged 65 attending a local AAA screening programme.

Method
Between 2001 and 2008, all 65-year-old men attending a local screening programme covering a population of 450,000 were given a questionnaire to complete prior to their aortic scan. Information regarding history of ischaemic heart disease, cardiac failure, hypertension, stroke, diabetes and chronic respiratory disease were collected. Data were analysed using cumulative moving averages analysis and Pearson's x² test where appropriate.

Results
Over the study period, 17,362 men were invited. Of these, 13,982 (80.5%) attended for a scan and completed their questionnaires. 389 men were found to have an AAA. A gradual decline in AAA average prevalence from 3.2% to 2.65% (17% reduction) was observed. This was associated with a decline in the prevalence of each of cardiac disease (6%), stroke (21%) and chronic respiratory disease (28%). The prevalence of pre-diagnosed hypertension increased by 16% and that for diabetes by 21%. When compared to men with normal aortic diameter, men with AAAs had a higher prevalence of each of cardiac disease (31.99% vs 16.80%, p<0.000), chronic respiratory disease (16.71% vs. 11.22%, p<0.022), hypertension (52.35% vs 38.06%, p<0.000) and stroke (7.89% vs. 3.55%, p<0.067).

Conclusion
In the population studied, a noticeable decline in AAA screening yield was observed. Whilst a reduction in the prevalence of major cardiovascular and respiratory diseases was also observed, the prevalence of these diseases remains significantly higher in AAA patients. These findings suggest that a combined strategy of earlier risk factor modification and later invitation for AAA screening may improve overall AAA disease management.
Establishing a volume-outcome relationship in lower limb bypass surgery using multi-level logistic regression modelling

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Objective

A volume-outcome relationship is known to exist for abdominal aortic aneurysm and carotid endarterectomy surgery. We sought to investigate if such a relationship exists for lower limb arterial bypass surgery in the UK.

Method

All femoro-popliteal bypass operations performed in England between 2002-2006 were identified from Hospital Episode Statistics data. A Charlson type risk profile, including operating hospital annual case volume, was identified for each patient. Outcome measures of revision bypass, amputation, death and a composite measure were established during the index admission and at 1 year. Multivariate modelling allows adjustment of results for significant determinants of outcome. Multi-level modelling adjusts for hospital trust level variations, including hospital volume, and therefore highlights significant variations in outcome between Trusts.

Results

25,133 popliteal bypass operations were identified. There were significant differences in outcome between NHS Hospital Trusts for repeat bypass (p=0.005), major amputation (p<0.001), in-hospital mortality (p=0.001) and the composite measure (p<0.001) with multi-level modelling. An increase in hospital volume by 50 bypass procedures per year reduced the odds of major amputation (OR 0.969, 95% CI 0.939-0.999, p=0.043), death (OR 0.972, 95% CI 0.949-0.996, p=0.024) and the composite outcome (OR 0.978, 95% CI 0.956-0.999, p=0.042) during the index admission. At 1 year, an increase in volume decreased the chances of death or amputation (OR 0.980, 95% CI 0.965-0.995, p=0.0110).

Conclusion

Variation in outcomes after lower limb bypass surgery exist between hospital Trusts. Hospital annual case volume is a significant factor in this variation, although the benefits are modest.
Outcome in the management of acute diabetic limb emergencies - a revolving door?
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Objective
An ‘ideal’ outcome in acute diabetic limb salvage may be defined as a patient who is discharged without a major amputation and has complete wound healing within a year without any unplanned readmissions. We examined the effect of a multidisciplinary approach in the pursuit of this composite outcome.

Method
A retrospective study of consecutive emergency admissions for acute diabetic foot complications was performed for the period between 1st Aug 2007 to 1st Aug 2009. Outcomes analysed included in-hospital mortality, limb salvage, wound healing and re-admission rates at 12 months.

Results
Ninety-four patients (male 66% with 95% Type 2 diabetes) were identified with a median age of 75 years (46-96). Median LOS was 24 days (1-233). Risk factors were smoking (54%), hypertension (62%), peripheral arterial disease (70%), ischaemic heart disease (30%) and chronic kidney disease (30%). Baseline median HbA1c levels were 7.7% (5.4-15.5). 98% presented with tissue loss (55% Wagner classification between 2 to 5). Medical therapy on admission included statins (86%), antiplatelets (77%), ACE inhibitors (53%), beta-blockers (20%) and antibiotics (94%). Thirty-seven limbs were revascularised - endoluminal (57%) and bypass surgery (43%). Initial amputation rates were minor amputations (30%) and major amputations (8.5%). In-hospital mortality was 8.5%. At 12 months, amputation-free survival was 70%. Only 49% of patients were alive without amputation and ulcer free at 12 months. When re-admissions were included the composite ‘ideal’ outcome was achieved in only 22% of all patients.

Conclusion
Although acceptable limb salvage rates are achievable, a significant proportion of patients presenting with acute diabetic limb emergencies suffer the ‘revolving door’ experience of incomplete wound healing and high re-admission rates despite a multidisciplinary team approach.
Training of future vascular surgeons in the UK in endovascular techniques is inadequate to train vascular specialists of the future. Results of a survey of UK vascular trainees

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Objective
Vascular surgery training for the future will require acquisition of skills in endovascular techniques and vascular ultrasound. This study aims to determine the current exposure of vascular trainees in the UK to this training.

Method
An email survey of 169 trainees, identified from the Association of Surgeons in Training database, with a declared vascular surgery interest was performed. Data were collected on experience in EVAR, peripheral endovascular interventions (PE) and vascular ultrasound (US).

Results
The response rate was 49% (83/169) from 89% of training regions in the UK. Trainees performed EVAR top-graft deployment (41.5%), contralateral limb cannulation (44.6%), and limb deployment (63.1%) in their current unit. 30.8%, 32.3% and 20%, respectively, had never performed these procedures. Vascular trainees gained experience of PE for iliac (25.8%), SFA (22.7%), and infrapopliteal (13.6%) intervention. 63.6%, 71.2%, and 86.2% have never performed PE. Trainees performed some arterial duplex in 29% of units, of which 70.8% are unvalidated. Venous duplex at outpatient clinics is performed by surgical consultants or trainees scan in 42% of units. Trainees feel they are not being trained in EVAR planning (30.1%), EVAR techniques (24.7%), PE (58.9%), and US (69.9%). Endovascular simulator training is available to 6.8%. 83.6% feel vascular surgery is unable to meet training expectations. The largest obstacle was felt to be lack of engagement of radiologists (60.6%).

Conclusion
Current training of vascular surgeons in the UK in endovascular techniques is insufficient to provide skills required to deliver specialist care, and to allow training of vascular specialists of the future.
Secondary medical prevention among Danish patients hospitalized to primary vascular surgery

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Objective

To analyse the use of secondary medical prevention among Danish patients hospitalized for primary vascular reconstruction between 1996 to 2006, with special attention to age differences.

Method

The nationwide Danish Vascular Registry was the main source of data, facilitated by other nationwide databases via the unique civil registration number. We assessed the proportion of prescriptions for secondary medical prevention in pre-defined time periods after surgery. To describe age differences we used logistic regression analysis, (age sub-groups 40-59, 60-79, and >80), adjusted for gender, socioeconomic variables, marital status and comorbidity.

Results

17,943 were included; 54.5% were males with a mean age of 68.8 years (range 40-99). Drugs prescribed during the entire period were as follows: lipid-lowering drugs 59.7%, anti-thrombotic drugs 84.3%, ACE/ATII antagonists 55.8%, and beta-blockers 45%. The proportion declined for all examined drugs throughout all pre-defined time periods; the exceptions were lipid-lowering drugs and anti-thrombotic therapy. This tendency was clearest in patients >80 years. Patients >80 years had an increased chance of receiving a prescription for beta-blockers (OR 1.31 [1.14;1.51]), anti-thrombotic therapy (OR 1.19 [0.99;1.44]), ACE/ATII (OR 1.48 [1.29;170]) and a decreased chance for lipid-lowering drugs (OR 0.62 [0.54;0.72]) compared to the age group of 40-59 years.

Conclusion

Prescription rates of examined secondary medical prevention were low in our study compared to national and international guidelines. A general decline of repeat prescriptions was observed over time, largest in the oldest part of the population, who must be expected to have the biggest comorbidity. Ongoing efforts to implement cardiovascular prophylactics are crucial, especially in elderly patients with symptomatic peripheral arterial disease.
The impact of standard treatment on balance and physical function among claudicants
KA Mockford, FAK Mazari, J Khan, N Vanicek, IC Chetter, PA Coughlin
Academic Vascular Surgical Unit, Hull and East Yorkshire Hospitals NHS Trust &
Department of Sport, Health and Exercise Science, University of Hull, Hull

Objective
Intermittent claudication is associated with deficiencies in physical function that may contribute to impaired balance and a higher risk of falls. Hence the aim of this study was to assess balance in claudicants undergoing treatment with exercise or angioplasty.

Method
A concurrent prospective case series study was carried out with two treatment arms of angioplasty (PTA) or a 12-week supervised exercise programme (SEP). Ninety-eight patients were recruited, 51 underwent SEP and 47 underwent PTA. All were assessed for severity of ischaemia and balance using the Sensory Organisation Test (SOT, NeuroCom).

Results
Both groups were comparable in terms of age, gender and comorbidities. The severity of ischaemia was significantly worse in the SEP group with lower ABPI and shorter initial treadmill walking distances (p<0.05, Mann Whitney U). At baseline, abnormal balance was seen in 46 patients (47%), comprising 28/51 SEP and 18/47 PTA (55% and 38%, respectively). At 3 months there was a significant improvement in the number of patients with normal balance in the SEP group (23/51 improved to 34/51; p<0.05, Chi squared); however, the PTA group showed no such improvement (29/47 at baseline versus 30/47 at 3 months; p>0.05).

Conclusion
Balance impairment is prevalent among older claudicants. Treatment of claudication with angioplasty makes no impact on this impairment, but supervised exercise leads to significant gains in balance despite their increased severity of ischaemic disease. Greater consideration to exercise programmes for claudicants should be addressed particularly in view of the potential to improve possible fall risk.
Incidence and survival outcome following femoral artery reconstruction during endovascular abdominal aortic aneurysm repair
CP Twine, A Wood, A Gordon, S Hill, R Whiston, IM Williams
University Hospital of Wales, Cardiff

Objective
Planned or unplanned reconstruction of the common femoral artery (femoro-femoral crossover and/or patch closure) may be required following endovascular abdominal aortic aneurysm (EVAR) stent-graft deployment for arterial closure or maintenance of lower limb perfusion. However, the incidence and impact on postoperative survival of such procedures is unknown. The aim of this study was therefore to determine the incidence of common femoral artery reconstruction (FAR) following EVAR and examine the effect of such procedures on patient outcome.

Method
178 patients undergoing EVAR were studied retrospectively.

Results
Thirty-one patients (17.4%) underwent FAR; 16 (51.6%) femoro-femoral crossover and 15 (48.4%) endarterectomy and patch closure. There was no significant difference in postoperative complications (p=0.057) or 30-day survival (p=0.454) between patients undergoing FAR and direct closure of the femoral arteries. However, long-term all-cause survival in patients undergoing femoral artery reconstruction was significantly poorer than those undergoing direct closure (Log rank Chi²=6.588, DF=1, p=0.010). On forward conditional regression analysis three factors - the need for FAR (HR=0.435, p=0.006), COPD (HR=0.424, p=0.002) and AAA size (HR=1.414, p=0.005) - were significantly and independently associated with survival.

Conclusion
FAR was performed in almost one in five patients undergoing EVAR and was significantly and independently associated with decreased survival. This effect did not seem related to the procedure itself, and may be a reflection of increased atherosclerotic disease in this group of patients. Multidisciplinary teams should be aware of these findings when planning EVAR, especially in borderline candidates.
Thursday 25th November 2010 at 4.30-5.30pm
Hilton Brighton Metropole, Brighton

1. Apologies
2. Minutes of AGM 2009
3. Any other business
4. President’s Report: Professor Cliff Shearman
5. Honorary Secretary’s Report: Mr Jonathan Earnshaw
6. Honorary Treasurer’s Report: Mr Simon Parvin
7. Audit and Quality Improvement Committee Report: Mr David Mitchell
8. Training and Education Committee Report: Professor Jonathan Beard
9. Research Committee Report: Professor Shervanthi Homer-Vanniasinkam
11. Vascular Tutor: Mr Ian Chetter
12. Circulation Foundation Committee: Mr Andrew May
13. President Elect’s Report: Mr Peter Lamont
14. Election of Officers: Result of ballot for Ordinary Members of Council
15. Date of next meeting: Thursday 24th November, Edinburgh International Conference Centre
Honorary Secretary’s Report

Jonothan Earnshaw

This is my fourth and final report as your Secretary, and it is impossible to resist sneaking a glance backwards. The Society has come a long way in the last four years. Although there is some unease about what the future holds, I sense a renewed professionalism and purpose amongst many Members, particularly the younger ones. The dream to become a separate specialty seems likely to be realised, affording the promise of a more secure future for the young vascular specialist looking forward to a long career.

In my term, I have been fortunate to have worked with four exceptional Presidents: George Hamilton, Mike Gough, Peter Taylor and Cliff Shearman. All have brought tremendous energy and enthusiasm to the post. It has been interesting watching them trying to stamp their mark on the Society in the short twelve months of their tenure. The focus they bring, together with their feeling of panic that the clock is ticking on their Presidency, is a very strong argument against raising the duration of the post to two years, as has been suggested. This year Cliff Shearman has worked tirelessly to promote the Society, and his reward has been the positive response of other Societies and professional groups towards our application for separate specialty status.

Other highlights of this year so far include the two excellent clinical meetings, the first in Nottingham about saving legs, and celebrating vascular disease awareness week. Co-ordinated locally by Bruce Braithwaite, the pinnacle of the meeting was the inclusion of patients who were prepared to talk publicly about their experiences. The second, Endovascular Fusion, was I hope the start of a fruitful collaboration between the VSGBI, BSIR and BSET. This opportunity to focus on all things endovascular is very popular amongst junior colleagues and trainees. I am sure that similar collaboration is important for the future.

The emerging confidence of David Mitchell’s Quality Improvement team is pleasing to see. Their growing role will ensure that the Society can have confidence in its outcome results, which are showing signs of improvement. The burgeoning documentation concerning quality aspects of our interventions is increasing our influence among commissioners of vascular services, and even at the Department of Health. Well supported Quality Improvement Frameworks on aortic aneurysm surgery, and this year on amputation surgery, are seen as excellent signs that the Society Members are conscious of issues within the profession, and taking steps to optimise performance.
AAA screening

The NHS AAA Screening Programme is entering an interesting phase. The excitement of the dream is being replaced with the reality of delivery. The Programme in England is on target to cover all men aged 65 in the country by 2013. Other home countries have established similar programmes with similar standards and similar schedules. Implementation does not come without a price. Some of you will have been involved in remodelling of your local vascular services to ensure that the delivery of interventions in your local programme meets NAAASP standards (which are based on the VSGBI AAA QIF). Others will be irritated by the increased time needed to enter data for AAA on the NVD. As previously stated, this increase in personal data is a necessary part of the governance of a national programme where men are invited to attend for the screening process that carries definite risks (every ten thousandth man will die after an elective AAA repair as a direct result of attending for screening). Finally, the data transparency that comes with the NAAASP will enable newspapers like the Guardian to use the Freedom of Information Act to see individual outcome results from the NVD. This may well cause concern among VSGBI Members, but I believe strongly that the Society should have no fear from being open about our results. The credibility that the cardiac surgeons now enjoy as a result of the annual publication of their individual outcome results should act as an encouragement to us all. I have mentioned before how important it is that the Society leads this process, rather than wait for the press to do it for us 1. We will all share in the success of AAA screening programmes, so it is important that vascular surgeons continue to commit their energy and enthusiasm towards successful implementation.

Secretariat

I leave this post with the Secretariat secure, stable and confident, each excellent at their job, but functioning well as a team, providing fine service to the VSGBI membership. I am grateful for their support and commitment for the past few years. I reserve my last comments, however, for Jeanette Robey, your Chief Executive. The Secretary post would not be possible for a busy vascular surgeon without her. She gets through an enormous amount of work, and keeps the VSGBI on the straight and narrow. She has excellent leadership skills within the office, and enormous common sense, combined with the ability to deal with difficult vascular surgeons (both within and outwith the Executive). She is a treasure, and the VSGBI is very lucky to have her. Next time you see her, or any of the Secretariat team, please remember to say thank you for their efforts.

Being VSGBI Secretary has been an enormous privilege, but also hugely enjoyable. I wish my successor, Mike Wyatt, the best of luck; he will need to hit the ground running, since the Society has many challenges ahead. Finally, I would like to thank all of you who have communicated with me (both positively and negatively). I thank you for the great support you have shown, and I encourage you to continue to communicate with us: you should keep the Executive on its toes at all times.

2010 has seen another successful year financially for The Vascular Society.

**Vascular Society and the AGM**

Because we have changed our year end for accounting purposes, the results for the year ending 30 June 2010 are not yet available. However, the results for the 18 months ending 30 June 2009 are detailed at the end of this report. They demonstrate that our finances are strong despite the difficult trading conditions.

The AGM in Liverpool last year is likely to show a healthy profit for the Society. However, the profit is less than the previous year in Bournemouth, and is one of the reasons why the cost of registration for the meeting this year has had to be increased by approximately 5% across the board.

The Society and Circulation Foundation have held successful and profitable events during the year, including the Spring Meeting in Nottingham, the golf day at Rudding Park, and the London Marathon.

The new combined meeting with the BSIR and BSET in June [Endovascular Fusion] was very successful from a scientific point of view, but the Society sustained a loss of £7000. This was mainly due to difficulties in the sponsorship arrangements. The Society plans to repeat the venture in 2012, with improved opportunities for our sponsors which should guarantee a profit.

For the AGM in Brighton we have introduced new opportunities for registration. There are discounts for consultants bringing their trainees, for trainees simultaneously joining the Society for the first time and for trainees presenting a paper.

Our office costs were substantially lower in 2009 compared with previous years. The last time costs were as low was in 2005. This is a significant credit to Jeanette Robey who has developed a new set of spreadsheets to help keep control of expenditure.

I would once again like to thank our Major Sponsors, AngioDynamics, Cook Medical, Le Maitre Vascular, Maquet, WL Gore and Vascutek, for their continued support this year.

**Circulation Foundation**

The Circulation Foundation has received a large legacy for the fourth year running. In 2010, a total of £121,492 was received from 2 legacies. The annual average income from legacies has been £157,000 since 2007. As a result of this dramatic increase in funds, grants totalling £170,500 will be allocated in 2010. This compares with a more typical £65,000 in previous years.
Membership

The Society currently has 697 members, a significant increase compared with previous years (644 in 2009 and 655 in 2008). The increase is amongst both Ordinary and Affiliate Members. I am hoping that our discounts for the AGM will encourage a further rise in the number of Affiliate Members. After a year when membership fees were pegged at the 2009 level, membership fees for 2011 will rise slightly.

VSGBI Ltd.

Profit and loss account

Year ended 30th June 2009

<table>
<thead>
<tr>
<th>Turnover</th>
<th>18 months to 30/06/09</th>
<th>12 months to 31/12/07</th>
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<td>(239,407)</td>
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<td>73,753</td>
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The Vascular Society
Income and expenditure accounts
Year ended 30th June 2009

<table>
<thead>
<tr>
<th>The Vascular Society</th>
<th>2009 Unrestricted Funds</th>
<th>2009 Restricted Funds</th>
<th>2009 Total</th>
<th>2008 Total</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Incoming resources</strong></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Voluntary income:</td>
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<tr>
<td>Costs of charitable activities:</td>
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<tr>
<td>Research awards</td>
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<td>-</td>
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The Vascular Society
Yearbook 2010

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<th>The Vascular Society</th>
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<th>2009 Restricted Funds</th>
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<th>2008 Total</th>
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<td><strong>Incoming resources</strong></td>
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<tr>
<td>Voluntary income:</td>
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<td>97,644</td>
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<td><strong>Total incoming resources</strong></td>
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<td><strong>Resources expended</strong></td>
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<tr>
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<tr>
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<td><strong>Net incoming resources for the year</strong></td>
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<td>58,919</td>
<td>28,081</td>
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The Vascular Society  
Income and expenditure accounts  
Year ended 30th June 2009

Circulation Foundation  

<table>
<thead>
<tr>
<th></th>
<th>2009 Unrestricted Funds £</th>
<th>2009 Restricted Funds £</th>
<th>2009 Total £</th>
<th>2008 Total £</th>
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<td><strong>Incoming resources</strong></td>
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<tr>
<td>Voluntary income:</td>
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<td>Research costs</td>
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<td>Tutor costs</td>
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<td>Computer support costs</td>
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<td>Stationery, postage and photocopying</td>
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<td>General expenses</td>
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<td>Recruitment fee</td>
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<td>Governance costs:</td>
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<tr>
<td>Audit and accountancy</td>
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<tr>
<td>Insurance</td>
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<td>-</td>
</tr>
<tr>
<td>Legal and professional</td>
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<td>-</td>
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<tr>
<td></td>
<td>1,831</td>
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<td>-</td>
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<td></td>
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<td>Management and administration of the charity</td>
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<td></td>
<td>54,871</td>
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<tr>
<td>Total resources expended</td>
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</tr>
<tr>
<td>Net incoming resources for the year</td>
<td>(29,758)</td>
<td>(1,161)</td>
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<tr>
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<td>(30,919)</td>
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Audit and Quality Improvement Committee Report

Chairman: David Mitchell

This has been a year of significant change and challenges. The award of a Health Foundation grant and a grant from Kidney Care UK allowed us to appoint a team to deliver our Quality Improvement Programme (QIP) for abdominal aortic aneurysm surgery. Part of this work involves gathering data on Acute Kidney Injury after aortic aneurysm surgery. Roxanne Potgieter was appointed Project Manager and is supported by Helen Hindley and Julia McCleary. The demands of audit and quality improvement meant that the Committee could no longer continue to manage a research role and a separate Research Committee was formed this year.

The Abdominal Aortic Aneurysm Quality Improvement Programme

The QIP began with a stakeholder meeting and the formation of a national implementation team. I would like to thank all the Members of the Society who have provided a lot of help getting us up and running. Information on the QIP can be found on our website www.aaaqip.com along with copies of the project plan. The first year of the QIP is focusing on encouraging better data entry rates into the NVD and developing national standards for care, as this was identified as an early priority by our patient groups. In addition to the support of the Members of the Society, we have received considerable help from the BSIR, VASGBI and SVN. We have established patient groups in five regions of the UK and we would like to see every region having a patient group within a year. The patient groups have provided us with valuable information on information and risk assessment. They expressed concern that there were no agreed national standards for delivery of care.

The key to delivery of the QIP and driving down mortality is consistent patient assessment and treatment based on nationally agreed documentation. We have been developing evidence-based assessment tools. In addition, we have begun work on regional action plans with input from surgeons, radiologists, anaesthetists, nurses and managers. These are intended to provide a care pathway tailored to patient and local needs, but incorporating consistent standards. The first meeting was held in the North East in July and we are looking to build on this with meetings in other regions throughout 2011. Please get in touch with the QIP office if you would like to help with developing an action plan in your region. We are working to produce care bundles that can be used as markers of good practice in AAA surgery. The delivery of high quality care in partnership with our patients is our goal.
You will all have seen the report in the Guardian earlier this year. There are significant concerns about the accuracy of the data that they received, but this showed that there is an improvement in outcomes compared to the data in the Vascunet report of 2008. Only by capturing all our cases can we speak confidently about outcomes following AAA surgery in the UK. Please enter all your cases into the NVD. If you need help with accessing the NVD, please do not hesitate to contact the team in Bristol (0117 32321620) or Bournemouth (01202 303626 ext 5939).

**NHS Abdominal Aortic Aneurysm Screening Programme (NAAASP) and the National Vascular Database**

The NVD has become the tool for managing data collection for the NHS AAA Screening Programme. This has necessitated a number of additional fields to collect demographic data. As patient identifiable information was already being held, we were advised that we would be in breach of patient confidentiality unless we obtained consent to hold data on any patient with an aneurysm. This has implications for retrospective data entry, so I would encourage everyone to move to real-time data entry. The NVD is now hosted on the secure N3 NHS server, so cannot be accessed remotely from NHS institutions, unless using a secure remote access device. The NAAASP brings in some monies to support the NVD. David Lockwood has been appointed to support the delivery of the programme; he is working with Sara Baker in Bournemouth.

**Acute Kidney Injury Audit**

The audit is gathering data on pre-operative risk factors, and peri-operative care in patients undergoing AAA repair. We need to obtain complete data on at least 500 cases. I would like to thank all those surgeons who have contributed. We need to build on the current data entry rates and would encourage all of you to support this national audit by spending a few minutes providing the extra data required. Once complete it will give the Society significant information on peri-operative care and improved follow-up data out to one year. The more cases we get, the more readily the results will be generally applicable to our patients. We want data from both elective and emergency open and EVAR cases. Helen Hindley runs the AKI audit and can be contacted on 0117 3232162 if assistance is required.

**National Carotid Intervention Audit**

The year also saw the publication of the second round of the carotid intervention audit and the national organisational survey. You will all have received copies of these and individual surgeon reports. The Society Members made a very significant contribution, with 93% of vascular surgeons capturing 70% of cases undertaken (compared to the Hospital Episode Statistics). The carotid intervention report attracted a lot of media interest and has highlighted the need for urgent intervention in patients with TIA and tight carotid stenosis. Much work remains to be done to meet national stroke targets, and success will depend on both increased public awareness and smoother referral pathways. As surgeons we need to provide
rapid access to carotid surgery and record all our cases in the NVD. Round 3 is underway and will have closed by the time you read this, but we will not be analysing the data until late December 2010, so please make sure that all your submissions are up to date by mid December. We need to increase our data capture rates to improve the quality of data generated by the audit.

Finally, you will all have noticed the changes to the NVD allowing our radiological and anaesthetic colleagues to input data. These important changes were managed expertly on behalf of the BSIR and VASGBI by Sara Baker. I would like to express my thanks to Sara for her continuing hard work on behalf of the NVD. Without her help, we would not have managed to update it to meet the changing demands of our specialty.
This is my first report as Chair of the Training and Education Committee. I would like to thank the outgoing Chair, Cliff Shearman, for his sterling work. I have enjoyed working with him this year in his role as President. I hope that my experience of Surgical Education as a trainer and researcher, combined with my links to other training organisations, including ISCP, PMETB/GMC, and the Academies of Medical Royal Colleges and Educators (AoMRC and AoME), will benefit our Society.

Our application for vascular surgery to become a new Medical Specialty, which had to be approved by the Minister for Health, unfortunately coincided with the General Election! However, we have now received a response from the Department of Health. Those consulted, including the Medical Royal Colleges, Societies and Associations, the GMC, CoPMED, and NHS Employers, have been generally supportive. There were a number of concerns and queries regarding the impact on other specialties and service provision, and we have responded to these. In the meantime, interventional radiology has been awarded sub-specialty recognition and our regular meetings with the RCR and the BSIR continue to ensure good communication and collaboration.

Cliff Shearman and the other Members of the Committee did a great job in writing the draft curriculum for our new Specialty and we are now working to ensure that there is reciprocity where there is overlap with other specialties such as interventional radiology and transplantation. For example, trainees in transplantation will need a placement in vascular surgery and all vascular trainees will need training in access surgery. We are also liaising closely with the ISCP to ensure that our new curriculum fits into its template. With this in mind, we have redrafted all the vascular Procedure Based Assessments (PBAs), and written some new endovascular ones, with the help of the interventional radiologists. These are available on the Society website. Please download and try them out, as we need your feedback to improve them before they are placed on the ISCP website.

After several rounds of interviews, largely caused by the limitations on eligibility, we were able to appoint all 7 of the post-CCT Endovascular Fellowships. These will not be reappointed as there is no further funding from the DoH. The 3 pre-CCT Endovascular Fellowships,
generously supported by Cook, were appointed at the beginning of the year; we have presently applied to Cook asking them to continue their support next year for a further 3 posts. These Fellowships were hotly contested and although they will have a relatively small impact on training opportunities, with help from the VAC and Rouleaux Club, we have also identified more than 30 units who are now offering radiological training to their own trainees, including duplex ultrasound and cross-sectional imaging interpretation/manipulation. Rob Hinchcliffe has kindly agreed to collate the information on the training opportunities provided by each unit. The initial feedback from trainees is that two different training opportunities are required. The first is good all-round exposure to duplex ultrasound, interpretation and manipulation of axial imaging and standard endovascular procedures. The second is for those who have already acquired these skills and who wish to master advanced endovascular skills.

Ian Chetter has taken up his post as Vascular Tutor in conjunction with the Raven Education Department of the Royal College of Surgeons. Ian is developing two new courses: the endovenous treatment of varicose veins; and complex vascular access, to add to the ones developed by our previous Tutor, Waquar Yusuf. One problem that we have identified with the current courses is that trainees often attend with insufficient knowledge, and have difficulty in practising their newly learned skills after a course. We aim to address these knowledge gaps by introducing e-learning modules, which must be passed before attending a course, and encouraging our network of Vascular Advisors to develop local/regional skills facilities for subsequent practice.

As Cliff Shearman wrote last year, so much of what we need and want to do in vascular training is dependent on us achieving specialty status. At present we have problems in attracting new trainees into vascular surgery. Once we achieve our goal, then my hope is that vascular training will take off, as it has in the United States.
This is my first report to the Society as Chair of the newly formed Research Committee.

As Members of the Society will know, for several years this aspect of the Society’s work was conducted by the Audit and Research Committee. However, it was increasingly felt that, whilst the audit work had progressed apace, the research component had lagged behind and it was time to look at establishing a separate Committee dedicated to research activities.

Following a discussion paper presented by me to Council in September 2009 (‘Re-energizing the national vascular research programme: the role of The Vascular Society’) in which the importance of the Society in advancing research was highlighted, a new Research Committee (RC) was set up with the specific remit of working to advance the role of the Society in shaping and supporting vascular research in this country. I was honoured to be invited to Chair this Committee, and joining me as Members are Miss Julie Brittenden, Mr Tim Lees and Professor Rob Sayers.

I will not go into too much detail in this report as to why it is crucial that the Society engages in actively shaping the landscape of national vascular research (the discussion paper may be read on the Society’s webpage). Suffice to say that, as The Vascular Society is the pre-eminent organization in this country promoting vascular health by supporting excellence in education, training and research, it is vitally important it plays a central role in furthering high quality scientific inquiry. Some Members, rightly, questioned this role of the Society; my riposte was to quote Jonathan Epstein, President of the American Society for Clinical Investigation: ‘Innovative scientific inquiry has been, and will continue to be, a potent spark that ignites economic growth while at the same time enhancing the quality of life’.

The first task of the RC was to look at new funding initiatives, especially targeted at supporting talented young vascular surgical colleagues as they seek to set up their own research careers. For some years now, the Circulation Foundation (CF) has been funding research projects through different awards. However, I felt it was time to look at specific, larger grants, which would make a fundamental difference to newly appointed consultants who aspired to excel in vascular research. I am delighted to report that at this year’s AGM, we will be awarding a novel grant, the ‘President’s Early Career Award’ (PECA). The PECA has a funding value of £100K (£50K per year for 2 years) and is aimed at supporting outstanding
young clinician-researchers. It is hoped that ‘the award would recognize, nurture and facilitate surgeons during the early years following their substantive appointment, as they set out to develop their independent research careers’.

The RC will also be working on other initiatives, and our next task – funds permitting – is to develop an award for trainee vascular surgeons who wish to commit time to pursue a higher degree by research. As we are all aware, funding (especially for the salary component, but also for bench expenses) for dedicated research-fellow posts is at an all-time low, and it is to address this that our efforts will be directed in the immediate future.

As I conclude this report, I wish to thank my colleagues on the RC who have worked hard to accomplish our tasks. 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 our future endeavours.
Congratulations! It has been a quiet year for the Professional Standards Committee. There are only two issues upon which to report.

The first was the accusation of discriminatory comments at last year’s AGM. A detailed enquiry did not find any evidence to support this and indeed none of those questioned could recall the incident.

Clearly it is important that the Society maintains the highest ethical standards and it will issue guidance to speakers in the future. In addition, all sessions at future AGMs will be recorded to facilitate the investigation of any future complaints. This also has a potential educational function, particularly for key-note addresses.

The second area of interest was the publication of an article by the Guardian newspaper which seemingly exposed centres with unsatisfactory results for aortic aneurysm repair. However, the single outlier that they identified had been picked up by the National Vascular Database (NVD) audit some 2 years earlier and had already stopped undertaking this type of surgery. Such reports can be misleading given the lack of risk stratification and inclusion of open and endovascular repair as a single entity. This highlights the importance of participation in the NVD where such information is collected. This has potential benefits for a surgeon faced with an enquiry into his/her outcomes and also to our patients through identification of global problems and the development of targeted initiatives like the Quality Improvement Programme for AAA surgery.
Since the AGM last November we have made considerable progress and have raised our profile in a number of ways.

The main event this year has been the first annual Vascular Disease Awareness Week which was held in March, and I must thank Rebecca Wilkinson for all her hard work in establishing this initiative. The aim of the week was to increase awareness of the dangers of Peripheral Arterial Disease (PAD) amongst the general public and to highlight the need for early diagnosis and referral. We also launched a patient information booklet on PAD which covers disease-specific information, along with a patient record of their risk factors and lifestyle advice. We are delighted that Sir Roger Moore has given his support and endorsed this booklet. Copies can be ordered from the CF office. The week was a great success. We held a Parliamentary Screening event at Portcullis House, which was hosted by Dr Brian Iddon MP and organised with the help of Mr Paul Bristow. Vascular Nurse Specialist, Louise Allen and I performed ABPI measurements on willing MPs to help raise awareness of PAD. Thankfully none of the MPs had worrying results, but all signed our Early Day Motion which was launched at our parliamentary reception in November 2009. An adjournment debate was also held by Dr Iddon to focus on the effect of PAD on the NHS. He paid tribute to the CF for the work they are doing to highlight the lack of awareness both within the medical community and the general public, and urged the Department of Health to look into diagnosis and referral.

Sponsored events were held during the Awareness Week to raise money for the Foundation, as well as awareness raising events in centres around the country. I would like specifically to thank consultant surgeon Marco Baroni and vascular nurse Nikki Barker who led a team of 80 people on a 2.5 mile sponsored walk around the walls of York, and who also held awareness raising events at York General Hospital. In addition, vascular nurse Neil Mitchard arranged a 13 mile sponsored walk along the Water of Leith and held an awareness event at Edinburgh Royal Infirmary. Vascular nurse specialist Lisa Smith, who runs a leg circulation service in North Manchester with her vascular podiatrist colleague, visited several public venues and health centres in her local area to help promote the Awareness Week and PAD. They had a stand outside their local ASDA store and a stall at the busy local market, handing out flyers and talking to people about PAD and risk factors.

After some late withdrawals due to injury, we had 7 runners in the London Marathon and I am most grateful to them for their efforts. Special thanks go to Consultant Surgeon Denis Harkin who ran a brilliant race and raised an incredible £4,200. If you are interested in running the London Marathon for the CF, please contact us.

Other fundraising events held during the year included the successful golf day at Rudding Park, near Harrogate, organised by Messrs Berridge, Gough and Scott, and we are extremely grateful to them. There were 40 golfers playing and about 140 people at the dinner. Another golf event is planned later this year. Can I suggest that all golfers approach their Club Captains and ask that the Circulation Foundation is considered as one of the Captain’s Charities?
The Vascular Society’s Spring Meeting was held in conjunction with the Awareness Week; thanks to Bruce Braithwaite who helped organise this event. Entitled “Saving Legs”, the meeting was a great success with talks from vascular surgeons, orthopaedic surgeons, vascular nurses and vascular nurse counsellors. We also had a panel of local amputees participate in a question and answer session. Over 140 delegates and 15 exhibiting companies attended. The Spring Meeting in 2011 will be held on March 10th at the Kings Fund in London, again as part of the Vascular Disease Awareness Week and will focus on the management of diabetes in peripheral vascular disease.

I am most grateful to Ian Franklin and Gerry Stansby who have joined the Committee this year. Both of them have been extremely generous donors through their work. I would also like to welcome Shervanthi Homer-Vanniasinkam as Chair of the Society’s Research Committee and thank her for introducing the new President’s Early Career Award, which will be given out at the AGM in November.

We have received some very generous legacy bequests over the last year. I would ask you all to promote the CF’s work by displaying information in areas such as vascular laboratories and exercise clinics, hospital wards, outpatient clinics and consulting rooms, in order to raise awareness of the CF’s work.

I am confident that we can continue to raise awareness of vascular disease and expand and grow our funding programme for research into vascular disease.

I look forward to seeing you at the AGM in Brighton.

Our special thanks to the following for their involvement in fundraising and awareness events during the year: Mr Denis Harkin (London Marathon); Mr Frank Smith (Marathon des Sables); Mr Andrew McIrvine, Professor Jonathan Beard, Mr James Brown, Mr Ray Dawson, Mr Martin Thomas, Mr Michael Wyatt, Mr John Thompson (NHS Regatta); Mr David Berridge, Professor Julian Scott, Professor Michael Gough, Mrs Moira Gough, Ms Nikki Dewhirst, Mrs Anne Johnson, Ms Sally Bucktrout, Professor Shervanthi Homer Vanniasinkam (Rudding Park Golf Day); Mr Marco Baroni, Ms Nikki Barker, Mr Steven Cavanagh, Mr Neil Mitchard and team at Edinburgh Royal Infirmary, Mr Andrew Parry (Vascular Disease Awareness Week); Mr Bruce Braithwaite (Spring meeting); Mr Ian Franklin, Professor Gerard Stansby and Professor Ross Naylor (Donation of speaker’s honoraria)

And to the following regular donors to the Circulation Foundation: Mr Munther Aldoori, Mr Roger Baird, Professor Jonathan Beard, Mr David Berridge, Mr Bruce Braithwaite, Mr Rod Chalmers, Mr Richard Corbett, Professor Alun Davies, Mrs Linda de Cossart, Mr Richard Downing, Mr Jonathan Earnshaw, 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 Michael Horrocks, Mr Michael Jenkins, Mr Tim Lees, Mr Shane MacSweeney, Mr Adrian Marston, Mr Andrew May, Mr David Mitchell, Professor Ross Naylor, Mr Simon Parvin, Mr David Reilly, Miss Sophie Renton, Professor Julian Scott, Professor Cliff Shearman, Mr Malcolm Simms, Professor Gerard Stansby, Mr Peter J Taylor, Mr Martin Thomas, Mr Kevin Varty, Miss Lucy Wales, Mr David Williams, Mr John Wolfe, Mr Kenneth Woodburn

Vascular Disease Awareness Week: 7th-13th March 2011

Contact details

Fundraising and Events Manager Ms Rebecca Wilkinson, Circulation Foundation
35-43 Lincoln’s Inn Fields, London, WC2A 3PE; Tel: 0207 304 4779; Fax: 0207 430 9235
E-mail: info@circulationfoundation.org.uk; Website: www.circulationfoundation.org.uk
I took over as RCS Eng / VS Vascular Tutor from Waquar Yusuf in February 2010, and would like to take this opportunity to thank all the staff at the College, the Vascular Society Training and Education Committee, the course Faculty members and particularly Waquar, Jonathan Beard (Chairman, Training & Education Committee) and Mike Larvin (RCS Director of Education) for their support. The course timetable and curriculum over the last 6 months was established prior to my appointment, thus I was in the fortunate position to be able to spend time assessing the content of the current course portfolio. I also took the opportunity to canvass the opinion of various other interested parties (Venous Forum, Rouleaux Club, British Society of Endovascular Therapy, Vascular Access Society of Great Britain & Ireland, Society for Vascular Technology). I have identified two future challenges:

- ongoing continued appraisal and progression of current courses;
- development and provision of new courses and learning materials / environments to encompass the complete specialist vascular curriculum.

**Current courses**

The current vascular portfolio contains four courses: EVAR Planning, Amputation, Vascular Ultrasound, and Specialist and Advanced Skills in Vascular Surgery. These courses provide excellent coverage of the practical (psychomotor) skills required by the specialist vascular curriculum. Participant feedback consistently scores this aspect of the course highly. This reflects the high quality of the course content, the College facilities and the skilled Faculty. The current courses, however, cover little cognitive (knowledge) or affective (decision-making) learning, and whilst it is beyond the scope of each course to provide exhaustive text on each topic, the aim is to develop an e-manual to accompany each course. This will contain short lecture notes, suggestions for further reading, direction to pivotal papers, and videos demonstrating surgical procedures. Affective learning will be facilitated by the inclusion of case-based discussion within the course.

Assessment drives learning and thus represents a fundamental part of any course. Currently, the only course within the vascular portfolio to contain assessment is Vascular Ultrasound. We aim to investigate the potential to include assessment within the other vascular courses, e.g. pre-course MCQs for cognitive assessment and in-course assessment of psychomotor skills using modified DOPS / PBAs.
New courses

Whilst the current course portfolio encompasses the majority of the specialist vascular curriculum, there are significant omissions including minimally invasive management of varicose veins and complex vascular access. These omissions will be rectified with the development of two new courses in the near future. A condensed pilot of the minimally invasive management of varicose veins course will run as an Endovascular Workshop at this year’s Vascular Society AGM on Tuesday 23rd November 2010.

Conclusion

The current RCS vascular courses are very popular which reflects their high quality. Planned future developments will provide complete and in-depth coverage of the specialist vascular curriculum (Figure 1) and perhaps with the inclusion of assessment will provide a ladder for the assessment of progression.

Finally, the dates for the vascular courses in 2011 are:

- Amputations - 26-27 January 2011;
- EVAR planning - 02-03 March 2011;
- Vascular Week - 13-17 June 2011 (Specialist skills 13-14 June; Advanced skills 15-17 June).

![Figure 1. Curriculum map.](https://example.com/curriculum-map.png)

Orange = current course

Green = course in development
The Rouleaux Club continues to provide an important voice for vascular trainees in Great Britain and Ireland, and 2010 has been another busy and successful year.

In March the Rouleaux Club was at the ASIT meeting in Hull. A Rouleaux Club prize was awarded to the best vascular abstract and was awarded to Ankur Thapar with his presentation on ‘Which measurement for abdominal aortic aneurysm screening?’. Selected conference abstracts were published in the *International Journal of Surgery*.

The annual summer meeting, held in conjunction with the junior BSIR, at the Endovascular Fusion Meeting was a great success. For the first time in its history, the Rouleaux Club had an integrated role within such a national meeting. There was a Rouleaux Club session focusing on service delivery and training and a lunchtime symposium with three excellent speakers, including Frank Veith who spoke about his thoughts on modern day training. It was extremely well attended with trainees both from vascular surgery and interventional vascular radiology. Attendees heard lectures from a wide range of UK and overseas specialists on the future of vascular surgery, training and education. The meeting was generously supported by Medtronic and we are also very grateful to the meeting organisers for including us in this way. Continuing in this vein, in conjunction with The Vascular Society, we hope to have a dedicated Rouleaux Club session at the VS AGM and the VS President will be addressing the Rouleaux Club at our AGM.

In the summer, Members of the Committee undertook review of vascular papers in the *British Journal of Surgery* and these were published as podcasts on the journal website (www.bjs.co.uk).

Our Members have consistently supported the move of vascular surgery to separate specialty status and were encouraged to hear of the progress the VSGBI has made in its application. The endovascular fellowships supported by the VSGBI, BSET and Cook have been well received and provide excellent experience for current trainees.

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**Rouleaux Club Committee**

**Committee**

**President**  
Mr Jeremy Crane  
Email: j.crane@imperial.ac.uk

**Vice President**  
Mr James Scurr  
Email: scurrj@hotmail.com

**Secretary**  
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**Treasurer**  
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**Vascular Society Affiliate Representative**  
Mr Robert Hinchcliffe  
Email: robhinchcliffe@gmail.com

**ASIT Representative**  
Mr Robert Davies  
Email: rsmdavies@mac.com

**BSET Representative**  
Mr Jeremy Crane  
Email: j.crane@imperial.ac.uk
The SVN is a professional society for nurses working with vascular patients in all care settings. Our membership stands at around 100, including a number of ward memberships (each with up to 20 members from one organisation); we also have several international members.

The SVN Committee, which directs the business of the Society, has 12 members including officers and two ‘Staff Nurse’ secondments. The AGM is held at the VS annual conference. We have developed a very diverse programme for this year’s conference, including the James Purdie Prize award presentation. We are planning future events such as the Spring Conference 2011 in collaboration with the Circulation Foundation and Vascular Society. We also award up to six bursaries a year, to a maximum value of £500 each, for which members of the SVN may apply to help their professional development.

This year we have launched a more professional website with detailed sections of information and contacts. New members are able to join electronically through this site. We aim to keep our members well informed of current and future developments in order to enhance the care of vascular patients. The website, together with the Newsletter, provides excellent opportunities to share ideas on clinical practice.

Wendy Hayes, President SVN
2010 has been a very exciting year for our Society.

We held our first joint meeting with the British Medical Ultrasound Society and our second joint meeting with the Venous Forum.

The pilot programme ‘Training for Vascular Surgeons’ has progressed and discussions for the coming year are underway.

Work continues within the Department of Health (DH) Modernising Scientific Careers programme, shaping the new career model and curriculum for future training of vascular scientists. Our accreditation programme has been updated along with the CPD scheme to deliver a comprehensive robust programme. The SVT, along with sonographers, continue to strive to have practice regulated by the Health Professions Council.

The SVT is leading the way in the DH Physiological Sciences Service Accreditation Programme, ensuring the quality of vascular science diagnostic services, as well as collaborating on Service Specifications for Commissioning Vascular Science services.

The Circulation Foundation research grant has been renewed and we are building further links with the CF for the coming year.

This year we have invested in a new membership database and website to support our Members. We are also working with the NHS AAA Screening Programme and have begun development of a ‘special interests’ SVT membership category.

The Care Quality Commission (CQC) has introduced new legislation affecting those providing independent ultrasound services. The SVT has provided advice to Members regarding the process for application or exemption under private practice privileges.

The SVT continues to be represented on the Federation of Healthcare Scientists, and The Institute of Physiological Sciences.

In its 19th year, the Society continues to grow and the profile of Clinical Vascular Scientists is being raised across the Department of Health and within the healthcare science community.

We look forward to the AGM in Brighton this year.

Kerry Tinkler, President
The Venous Forum of the Royal Society of Medicine

The Spring Meeting of the Venous Forum encompassed the British Association of Sclerotherapists and the Society for Vascular Technology. Lectures, symposia, and hands-on practical workshops were delivered over the two days. I would like to thank Phillip Coleridge-Smith, President of the BAS, and Ms Elaine Young, President of the Society for Vascular Technology, for helping to construct the programme and contributing to an excellent and very successful meeting. Dr Peter Gloviczki delivered the Phlebology RSM Press Lecture entitled ‘Surgical Management of Chronic Venous Insufficiency’. The winner of the Venous Forum Prize (£250) was Amanda Shepherd, second prize (£150) was S Harrison, and third prize (£100) Daniel Carradice. The winner also receives sponsorship to present at next year’s American Venous Forum (Travel Grant sponsored by The Venous Times). All three prize winners’ abstracts have been submitted to the American College of Phlebology. One will be chosen to present (fully funded) at their next meeting in Orlando, Florida, November 2010 – this is a reciprocal agreement with the Venous Forum of the RSM.

We are extremely grateful to our major sponsors (Angiodynamics, Biolitec, Medi, Olympus, and VNUS) who have enabled the Venous Forum to proceed with publishing of the VEIN 2 project, to award a travel fellowship of £1,000 to Amanda Shepherd and a pump priming grant of £10,000 to Peter Holt, and to undertake the reciprocal agreement with the American College of Phlebology. Publication of the VEIN 2 project is to coincide with the joint meeting with the VSGBI.

The Venous Forum continues in a healthy financial status, and wishes to continue the mutually beneficial relationship with The Vascular Society of Great Britain and Ireland. We have a new academic administrator, Louisa Mason, who takes over from Becky Hamer. I would like to thank Becky for all of her hard work, and wish her well in her new employment. Many thanks to Mr Frank Smith for his contributions as Honorary Treasurer. Mr Isaac Nyamekye has been appointed as the new Honorary Treasurer. Professor Alun Davies demits office at the end of the November meeting to be replaced as President by Professor Andrew Bradbury. Alun has been pivotal in obtaining significant extra funding to allow new initiatives, such as the travel grants, pump priming grants, and the two reciprocal agreements reached with the American Venous Forum and the American College of Phlebology. He has also been instrumental in collaborating with the BAS, SVN and SVT to provide joint meetings. On behalf of the Venous Forum Council, and its members, I would like to thank Alun for a very successful Presidency. Professor Gerry Stansby will take over as Honorary Secretary at the end of the Spring 2011 meeting.

I hope you enjoy the Venous Forum meeting at The Vascular Society in Brighton, and I look forward to welcoming you to the RSM on the 27/28th April 2011. This will be a joint meeting with the BAS, SVN and SVT. We anticipate another successful meeting with a combination of invited free papers, symposia, audience interaction sessions and hands-on demonstrations.

David Berridge, Honorary Secretary

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www.rsm.ac.uk/venous forum
The Joint Vascular Research Group

The Joint Vascular Research Group has had another excellent year and I would like to thank all Members for continued support. We are a collaborative network of vascular surgeons, research nurses and technologists, who share an interest in clinical research. Membership is by centre and if you are interested in becoming involved, please contact our co-ordinator, Lesley Wilson, who will be able to send you further details.

I am delighted that Chris Gibbon’s paper entitled ‘Surgical or endovascular treatment for chronic mesenteric ischaemia: a multicentre study’ has been accepted for publication in the Annals of Vascular Surgery. In addition, Jonathan Beard is organising a Masterclass on the morning of Wednesday 24th November 2010 at the VS Annual Meeting, based on the JVRG’s Rare Vascular Disorders book.

The JVRG books continue to sell well. If you would like a copy of The Evidence for Vascular Surgery (2nd Edition), Rare Vascular Disorders or Pathways of Care in Vascular Surgery, please contact Nikki Bramhill at nikki@tftmpublishing.com.

A new JVRG book entitled Complications in Vascular and Endovascular Surgery: How to Avoid Them and How to Get Out of Trouble is now in production and will be published in the Summer of 2011; again, Nikki can reserve a copy for you on request.

Finally, I would like to thank all of the JVRG members for electing me as their Chair. I step down in November and my successor is Daryll Baker. My thanks to Nuros and Sanofi Avensis for their continued support and I look forward to seeing you all at the AGM in Brighton.

Mike Wyatt, Chairman
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Radiometer Ltd - non-invasive assessment of chronic wounds.

Radiometer introduces a new, cost-effective, multi-channel transcutaneous oxygen monitor. This enables accurate mapping of tissue oxygenation and perfusion in patients with non-healing limb wounds or other types of peripheral vascular disease. Transcutaneous oxygen measurements are an objective and quantitative method to rapidly assess tissue oxygenation. Furthermore, since they are non-invasive, such measurements are easy to perform and provide important diagnostic and prognostic information about peripheral vascular disease and the likelihood of wound healing.

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Smith & Nephew Wound Management is a world leader in advanced wound care, providing a range of treatments for wounds such as pressure ulcers, leg ulcers, diabetic foot ulcers, surgical dehiscence and burns. It develops innovative new solutions to chronic and acute wound management problems, delivering the best and most cost effective outcomes available supported by comprehensive training and education platforms. Smith & Nephew have a wide portfolio of products including RENASYS negative pressure wound therapy.

We look forward to welcoming you onto our stand and discussing our products with you. Enjoy your conference.

STD Pharmaceutical Products Ltd
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STD Pharmaceutical is a family run business which started in 1967. We have products to support sclerotherapy and iontophoresis.

We make Fibro-Vein which is the only licensed sclerosant in the UK; it is effective on all sizes of veins from truncal veins to telangiectasia. Other products include micro-needles, syringes, bandages, etc., plus books and videos.

We also promote tap water iontophoresis, a simple but very effective treatment for patients suffering from hyperhidrosis of the hands and/or feet and axillae. The treatment is effective for over 85% of sufferers and being non-invasive is an ideal first-line treatment. There are machines for hospitals/clinics as well as smaller units for home use.
From the inventor of the Laptop PC and with decades of experience in ultrasound, Toshiba will be showcasing their latest addition to their ultrasound range with the new standard in portable ultrasound, the Viamo. A stunning piece of imaging equipment, small yet powerful, lightweight yet uncompromising to image quality, easy to use yet a fully fledged ultrasound system and shares specialty transducers with your Toshiba premium cart-based system.

Ultrasound diagnostics at a new level - that’s Toshiba’s Precision Imaging technology - a multi-resolution signal processing technique that analyses ultrasonic images on the fly and simultaneously at various orders of spatial resolution to separate structure from clutter or noise. Seeing is believing.

VASCUTEK, a TERUMO Company, is an established world leader in developing vascular grafts.

The Anaconda™ AAA Stent Graft System is the only repositionable device. Anaconda™ now features BluGlide™, a low-friction sheath featuring hydrophilic coating technology that significantly smoothes the passage of the delivery system through the arteries. The integral, graduated kink-resistant braided sheath provides controlled delivery with excellent trackability and maneuverability in varying patient anatomies while offering exceptional flexibility. The leg devices of the Anaconda™ AAA Stent Graft System are now approved for isolated iliac aneurysm repair. Innovative, patented magnet wire technology aids rapid cannulation of the contralateral limb.

York Medical Technologies Ltd (YMT) is the UK distributor for top surgical instrument manufacturers such as Stille, Medicon, Heinz Waldrich, Dufner and Thompson.

YMT also supplies British pattern instruments from B & H, Dixons, Murrays and others.

A wide range of associated disposable items, including Stille arthroscopy cannulae, Kirschner wires and skin staplers, are available along with the award-winning range of theatre fluid management products from Colby.
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ZONARE Medical Systems, Inc. designs, develops, and manufactures premium compact performance ultrasound solutions, which combine revolutionary technology with an innovative physical design.

Zone Sonography Technology™, ZONARE’s unique patented approach to ultrasound imaging, is focused on bringing the highest performance to all clinical settings, leading to advanced diagnostic capabilities, more cost effective operation and increased value to providers.

This technology enables ZONARE to deliver advanced software features such as Auto Optimisation™ and ZSTM™, which compensates for differing speed of sound in different body masses, IQ Scan™, which allows full retrospective imaging and compound tissue harmonics ensuring that ZONARE keeps the user at the leading edge of ultrasound technology.

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ZooBiotic Ltd manufactures and markets larvae products for the treatment of sloughy and necrotic wounds.

BioFOAM® Dressings, BioFOAM® Maintenance dressings and ‘Free Range’ LarvE® are Zoobiotic Ltd’s core products.

BioFOAM® dressings and BioFOAM® Maintenance dressings consist of larvae that are enclosed in net pouches which contain pieces of hydrophilic polyurethane foam.

The ‘free range’ LarvE® are applied directly to the wound and seek out areas of slough or necrotic tissue.

ZooBiotic has recently acquired a leading competitor, BioMonde GmbH, in a multi-million pound deal, securing our position as a dominant larval therapy player in the European wound healing market.
Other Exhibitors

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- AngioDynamics
- Cook Medical
- Maquet
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- Vascutek Terumo
- Gore

Future annual meetings

23-25 November 2011  Edinburgh International Conference Centre
28-30 November 2012  Manchester Central
Yearbook 2010