The Vascular Society of Great Britain & Ireland

Yearbook 2005

Contents

The Office

Address

The Vascular Society, The Royal College of Surgeons
35-43 Lincoln's Inn Fields, London, WC2A 3PE.

Company 5060866, registered in England, limited by guarantee and registered as charity no 1102769

Tel: 0207 973 0306
Fax: 0207 430 9235
E-mail: office@vascularsociety.org.uk
Website: www.vascularsociety.org.uk

Chief Executive
Miss Jeanette Robey

E-mail:
jeanette@vascularsociety.org.uk

Secretarial Assistant
Mr Audley Farrell

E-mail:
audley@vascularsociety.org.uk

Yearbook Editor
Mr Peter Lamont

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Tel: +44 (0)1952 510061
Fax: +44 (0)1952 510192
E-mail: nikki@tfmpublishing.com
Web site: www.tfmpublishing.com

Design and layout: Nikki Bramhill

The Council

Front row, left to right:
Mr S Ashley, Mr D C Berridge, Mr P M Lamont, Professor M Horrocks, Mr J Wolfe, Professor D J A Scott, Miss J Robey

Second row, left to right:
Mr A Davies, Mr M Adiseshiah, Mr R Vohra, Mr F C T Smith, Mr S MacSweeney, Mr T Lees

Back row, left to right:
Mr M Lewis, Mr R Fisher, Mr K Varty, Professor C Shearman

Message from the President

Expanding our horizons

Following the mandate at last year's extraordinary meeting of the Vascular Surgical Society of Great Britain and Ireland, the new Vascular Society was born. This coincided with the change in emphasis from treating vascular disease by conventional surgery to the increasing adoption of endovascular techniques with many exciting new treatment options. These changes have been embraced by the vascular community and herald a change in requirements for training and continuing professional development.

As a result of this endorsement of change by The Vascular Society your representatives have been working with the Royal College of Radiologists to develop a shared programme for training vascular specialists of the future, who will have both surgical and endovascular skills. As expected this has proved to be a challenging task, balancing the problems of general and vascular surgery on the one hand with different types of interventional radiology on the other. It is important that these proposed changes are appropriate to make vascular specialists of the future fit for purpose, but also are acceptable to all the existing services. It is particularly important that we have the support of our colleagues in interventional radiology as we continue to try and establish a common training pathway.

In addition to the changes in training it is clear that we need to work towards a change in the delivery of service for vascular disease, developing a disease-based approach working in multi-disciplinary teams. The prospect of a centrally funded screening programme for aortic aneurysms, hopefully to be announced at the end of this calendar year, should give us an opportunity to review the requirements for a vascular service, allowing reorganisation of existing services to improve the quality and range of patient care options. The recognition of vascular surgery as a mono-specialty in Europe and the recommendation that patients with vascular disease should be treated by recognised vascular specialists should all put pressure to allow change for the better. Whether vascular surgery will remain within the general surgical SAC is not a question that needs to be addressed at the present time, but with the reorganisation of postgraduate medical education it may be that change will come with time.

At the forthcoming Annual General Meeting there will be an opportunity to update you on progress, and for Council to hear views of the membership with regard to the future direction of the Society. Professor Julian Scott has worked hard throughout the year to refine the proposals for the common training pathway and this will be updated in Bournemouth.

This has been a busy year for the Society and our Secretary, Peter Lamont, has continued to take responsibility for the day-to-day management of the Society's affairs, admirably supported by our very efficient Chief Executive, Jeanette Robey, and Audley Farrell. The implementation of the new constitution of the Society is now complete as is the integration of the BVF. It is important for the Society to support the activities of the BVF and I hope you will all be able to support some of the many activities arranged for fundraising.

The Audit Committee under the Chairmanship of Simon Ashley has continued to evolve and develop and I am pleased to announce the support of the Healthcare Commission for a major project with regard to carotid endarterectomy. It is essential that the Society supports this venture which has been circulated in detail. A side effect of this is that it will allow the entry of data onto the National Database to be done on-line and I hope that the members of the Society will all continue to support this very important venture. I would formally like to thank Simon Ashley for his excellent Chairmanship of the Audit Committee and for his wise counsel whilst in office.

I would also wish to thank all members of the Council for their continued support and hard work during the past year. There are now many sub-committees of Council, ably supported by individual members, all of whom have contributed greatly to the efficient running and future progress of the Society. This has put a considerable burden of work on Council members who are already busy with their clinical work and I am grateful to them all for their time.

I am looking forward to welcoming you all to Bournemouth in November. The meeting should prove to be an interesting and exciting occasion with a mixture of scientific presentations, guest lectures and topical discussion. There is also an enjoyable social programme planned which I hope will be attractive to all.

With best wishes

Michael Horrocks
President

Office Bearers and Trustees of Council 2004-2005

President	Professor M Horrocks
President Elect	Mr J Wolfe
Honorary Secretary	Mr P M Lamont
Honorary Treasurer	Mr D C Berridge
Ordinary members	Mr M Adiseshiah Mr A Davies Mr T Lees Mr M Lewis Professor C Shearman Mr F C T Smith Mr K Varty Mr R Vohra
Training & Education Committee Chairman	Professor D J A Scott
Audit & Research Committee Chairman	Mr S Ashley
Affiliate member	Mr R Fisher
Vascular Tutor	Mr S MacSweeney
Observers	Ms P Morris-Vincent, The Society of Vascular Nurses Ms Y Sensier, The Society for Vascular Technology

Committees

Audit and Research Committee 2004-2005

Mr S Ashley (Chairman)
Mr C Gibbons
Mr T Lees
Dr D Prytherch
Mr D Wilson-Nunn
Mr S Parvin
Mr D Berridge
Mrs S Baker
Dr A Nicholson

Training and Education Committee 2004-2005

Professor D J A Scott (Chairman)
Professor M Horrocks
Dr D Kessel
Mr R Fisher
Mrs M Allen
Mr P M Lamont
Professor C Shearman
Mr F Smith
Mr S MacSweeney

Professional Standards Committee 2004-2005

Professor W B Campbell (Chairman)
Professor M Horrocks
Mr M I Aldoori
Mr I F Lane
Mr M J Gough

British Vascular Foundation Committee 2004-2005

Professor Sir P Bell (Chairman)
Mr J Wolfe
Mr A May
Professor K Burnand
Ms T Gatenby, Society of Vascular Nurses
Mrs C Flatman, Society for Vascular Technology
Professor M Horrocks
Mr D Berridge
Mr J Whitehead
Mr T Lees
Mr J Thompson

Vascular Advisory Committee 2004-2005

All Members of Council

Vascular Advisors:

Mr M Aldoori, Yorkshire
Vacancy, West Midlands
Mr J Clarke, East Anglia
Mr P Edwards, Mersey
Mr G Griffiths, Scotland (East)
Mr S Hardy, North Western
Mr R McFarland, South West Thames
Mr D Mehigan, Eire
Professor C Shearman, Wessex
Mr M Tyrrell, South East Thames
Mr J Beard, Trent
Mr B Braithwaite, East Midlands
Mr A Davies, North West Thames
Mr C Gibbons, Wales
Miss L Hands, Oxford
Mr R Holdsworth, Scotland (West)
Mr A May, North East Thames
Mr D Mitchell, South Western
Mr C Soong, Northern Ireland
Mr M Wyatt, Northern

Vascular Members of the SAC in General Surgery:

Mr R Grimley
Mr P M Lamont
Professor C Shearman
Mr M Wyatt
Mr B Gwynn
Professor D J A Scott
Mr S Silverman

Annual General Meetings

Year	Venue	President	Secretary	Treasurer
1966	Inaugural Meeting The Middlesex Hospital, London	Mr Sol Cohen	Mr JA Gillespie	Mr JA Gillespie
1967	Edinburgh	Mr Sol Cohen		
1968	Hammersmith Hospital, London	Mr PGC Martin		
1969	Royal Infirmary, Glasgow	Professor AW Mackay	Mr A Marston	Mr A Marston
1970	University College, Dublin	Professor FP Fitzgerald		
1971	St Mary's Hospital, London	Mr HHG Eastcott		
1972	The University, Dundee	Professor Sir D Douglas	Mr DGA Eadie	Mr DGA Eadie
1973	St Thomas's Hospital, London	Professor JB Kinmonth		
1974	Queen Elizabeth Hospital, B'ham	Professor G Slaney		
1975	St Bartholomew's Hospital, London	Professor GW Taylor	Mr CV Jamieson	Mr CV Jamieson
1976	Royal Infirmary, Bristol	Professor JH Peacock		
1977	Pfizer Foundation, Edinburgh	Mr AIS Macpherson		
1978	Liverpool	Mr CR Helsby	Professor AO Mansfield	Professor AO Mansfield
1979	John Radcliffe Hospital, Oxford	Mr D Tibbs		
1980	St Thomas's Hospital, London	Mr FB Cockett		
1981	University Hospital of Wales, Cardiff	Mr G Heard		
1982	University Hospital of South Manchester	Mr S Rose	Mr SG Darke	Mr SG Darke
1983	St Mary's Hospital, London	Mr JR Kenyon		
1984	Medical School, Birmingham	Professor F Ashton		
1985	The Middlesex Hospital, London	Mr A Marston		
1986	The Institute of Education, London	Mr M Birnstingl	Professor CV Ruckley	Professor CV Ruckley
1987	Civic Centre, Newcastle-upon-Tyne	Mr PH Dickinson		
1988	The University of Leeds	Mr J Shoesmith		
1989	Ninewells Hospital, Dundee	Professor W F Walker		
1990	Kensington Town Hall, London	Mr EJ Williams	Mr PL Harris	Mr PL Harris
1991	Royal College of Surgeons, Dublin	Mr WP Hederman		
1992	Metropole Hotel, London	Professor NL Browse		Mr MH Simms
1993	Royal Northern College of Music, Manchester	Mr D Charlesworth		
1994	Assembly Rooms, Edinburgh	Professor CV Ruckley	Mrs L de Cossart	
1995	Kensington Town Hall, London	Mr CW Jamieson		
1996	Bournemouth International Centre, Bournemouth	Mr SG Darke		Mr MJ Gough
1997	Royal Lancaster Hotel, London	Professor A O Mansfield		
1998	City Hall, Hull	Mr JMD Galloway	Professor WB Campbell	
1999	De Montfort Hall, Leicester	Professor PRF Bell		
2000	London Arena, Docklands, London	Professor RM Greenhalgh		Mr RB Galland
2001	Metropole Hotel, Brighton	Mr RN Baird		
2002	Waterfront Hall, Belfast	Professor AAB Barros D'Sa		
2003	Scottish Exhibition and Conference Centre	Professor KG Burnand	Mr PM Lamont	
2004	Harrogate International Centre, Harrogate	Mr PL Harris		Mr DC Berridge
2005	Bournemouth International Centre, Bournemouth	Professor M Horrocks		

Presidents

Professor M Horrocks
President 2005


Mr PL Harris **2004**


Professor KG Burnand **2003**


Professor AO Mansfield **1997**


Mr JMD Galloway **1998**


Professor PRF Bell **1999**


Professor RM Greenhalgh **2000**

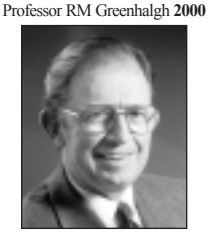

Mr R Baird **2001**


Professor AAB Barros D'Sa **2002**


Mr W Hederman **1991**


Professor NL Browse **1992**


Mr D Charlesworth **1993**


Professor CV Ruckley **1994**


Mr CW Jamieson **1995**


Mr SG Darke **1996**


Mr A Marston **1985**


Mr M Birnstingl **1986**


Mr PH Dickinson **1987**


Mr J Shoesmith **1988**


Professor WF Walker **1989**


Mr EJ Williams **1990**


Mr DJ Tibbs **1979**


Mr FB Cockett **1980**


Mr G Heard **1981**


Mr S Rose **1982**


Mr JR Kenyon **1983**


Professor F Ashton **1984**


Professor JB Kinmonth **1973**


Professor G Slaney **1974**


Professor GW Taylor **1975**


Professor JH Peacock **1976**


Mr AIS MacPherson **1977**


Mr CR Helsby **1978**


Mr S Cohen **1967**


Mr PGC Martin **1968**


Professor AW Mackay **1969**


Professor FP Fitzgerald **1970**


Mr HHG Eastcott **1971**


Professor Sir Donald Douglas **1972**

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 £500 payable to the Research Fund of the Department from which the paper was submitted.

The Venous Forum Prize is for the best paper in the *Venous Forum session*, organised by the Officers of the Venous Forum. The award is a cheque for £250.

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.

- 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 prize. 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 infra inguinal 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 Z A Ali, Addenbrooke's Hospital, Cambridge
Remote ischaemic preconditioning reduces myocardial injury after abdominal aortic aneurysm repair

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?

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 neovascularisation 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 IJ 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's 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

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 surgery 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)

2003 RJ Winterborn, Gloucestershire Royal Hospital, Gloucester
Late results of a randomised controlled trial of stripping the long saphenous vein

2004 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

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

1983 Professor Graham Douglas Tracy FRCS FRACS - *"Choosing a treatment plan for patients with leg ischaemia."*

1984 Mr Roger Neale Baird FRCS - *"Recognition of carotid artery disease."*

1985 Mr Adrian Marston FRCS - *"The Gut and its Blood-Supply."*

1986 Professor Sir Peter Morris FRCS - *"Whither carotid endarterectomy."*

1987 Dr J Connolly MD - *"Can paraplegia in aortic surgery be prevented?"*

1988 Dr Thomas F O'Donnell MD - *"Management of the high risk abdominal aortic aneurysm"*

1989 Professor Averil O Mansfield FRCS - *"An artery and a vein dancing - the management of arteriovenous malformation"*

1990 Mr CW Jamieson FRCS - *"Dilemmas in improving vascular surgical services"*

1991 Professor Norman Browse FRCS - *"The lymphatics"*

1992 Professor Alexander Clowes - *"Vascular biology - the new frontier"*

1993 Dr Ray Gosling - *"The mechanics of atherosclerosis"*

1994 Dr Hero van Urk - *"Future development in endoluminal vascular surgery"*

1995 Dr Timothy Chuter - *"Clinical experience of stenting aneurysms"*

1996 Dr Jerry Goldstone - *"Vascular surgery: training, certification and practice; observations from the USA"*

1997 Mr Alan Scott FRCS - *"Screening and the management of abdominal aortic aneurysms - the missing links"*

1998 Mr Peter Harris FRCS - *"Vascular surgery: the European perspective"*

1999 Mr Simon G Darke FRCS - *"Optimal management of venous ulceration: an enigma slowly unfolding"*

2000 Professor Janet Powell - *"The good, the bad and the ugly - a tale of aneurysms"*

2001 Mr Jonothan Earnshaw FRCS - *"Audit of Clinical Outcomes in Vascular Surgery: a Shield for our Profession"*

2002 Professor David Bergqvist - *"Management of Iatrogenic Vascular Injuries"*

2003 Professor Reginald Lord - *"Carotid Disease: the Burden of Proof"*

2004 Professor Roger Greenhalgh - *"The Impact of Vascular Clinical Trials on Clinical Practice"*

Programme

23rd-25th November 2005
Bournemouth International Centre

Wednesday 23rd November 2005

9.00am-12noon **EDUCATIONAL MASTERCLASS** **MEYRICK/BRANKSOME SUITES**

'Hot Topics'

Moderator: Professor Julian Scott

Breakout Sessions:

How I do an Eversion Carotid Endarterectomy
Mr Keith Poskitt, Cheltenham

How I do a Basilic Vein Transposition AV Fistula and the DRIL Procedure
Mr David Mitchell, Bristol

Flaps for the Vascular Surgeon
Mr Howard Peach, Leeds

ICU Updates: Adrenal Insufficiency and Tight Glucose Control in the Vascular Patient
Dr Andrew Cohen, Leeds

How I manage the Infected Carotid Patch
Professor Ross Naylor, Leicester

How I do Laser Varicose Vein Surgery
Mr Mike Gough, Leeds

Botox for Armpits and Feet
Mr Lasantha Wijesinghe, Bournemouth

How I do a Thoracoscopic Sympathectomy
Mr Shane MacSweeney, Nottingham

9.00am-12noon **VENOUS FORUM** **TREGONWELL HALL**

9.00-10.15am **Session 1**

Training in Varicose Vein Surgery - 'The best way to …'

Chairmen: Mr Richard Corbett, President, Venous Forum
Mr Jonothan Earnshaw, President Elect, Venous Forum

9.00-9.10am Welcome and Introduction
The numbers of cases done by trainees
Mr Richard Corbett, President, Venous Forum

9.10-9.18am The best way to treat varicose veins is by conventional surgery
Mr John Scurr, London

9.18-9.26am The best way to carry out pre-operative assessment
Mr Ashok Handa, Oxford

9.26-9.34am The best way to obtain pre-operative consent
Mr Haroun Gajraj, Yeovil

9.34-9.42am The best way to anaesthetise for varicose vein surgery - the options
Mr Frank Smith, Bristol

9.42-9.50am The best way to do phlebectomies
Mr Richard Corbett, Brighton

9.50-9.58am The best way to ligate and do inversion PIN stripping on the small saphenous vein (SSV)
Mr Barrie Price, Guildford (on behalf of Price BA, Holdstock JM, Smith C, Harrison C, McGuinness C, Whiteley M)

9.58-10.06am The best way to get the patient home as a day case and arrange analgesia
Dr Hans Klein (on behalf of Klein H, Scott DJA)

10.06-10.14am The best way to augment the teaching of varicose vein surgery
Mr Stephen Black, London (on behalf of Black SA, Pandey VA, Wolfe JHN)

10.15-10.45am **Coffee and Trade Exhibition** **PURBECK HALL**

10.45-12noon **Session 2**

The economics of varicose vein surgery - can it survive in the NHS?

Chairman: Mr Richard Corbett

10.45-10.55am Introduction: what it costs now and what the National Tariff will pay
Mr Richard Corbett, Brighton

10.55-11.15am Quantifying the costs and benefits of the treatment of varicose veins
Professor Jonathan Michaels, Sheffield

11.15-12noon **Debate**

This House regrets the introduction of rationing of varicose vein surgery in the NHS

For Professor Bruce Campbell, Exeter
Against Mr Martin Thomas, Chertsey

Debate in the House

For Mr Alun Davies, London
Against Mr Mark Whiteley, Guildford

The Vote

9.00am-12noon **SOCIETY OF ACADEMIC AND RESEARCH SURGERY TREGONWELL BAR**

Chairmen: Professor Michael Horrocks, President
The Vascular Society
Professor Kevin Burnand, President Elect
Society of Academic and Research Surgery

9.00-9.30am **KEYNOTE LECTURE**

Reperfusion syndrome: of molecules, mice and men

Professor Shervanthi Homer-Vanniasinkam, The General Infirmary at Leeds, Leeds

9.30-10.20am **SURGICAL RESEARCH PAPERS**

9.30-9.40am Differing levels of transcription factors and MMPs in different regions of Abdominal Aortic Aneurysm (AAA) wall help identify the sequence of signalling pathways in AAA development

Erdozain O, Bodamyali T, Stevens C, Horrocks M
Department of Surgery, School for Health, University of Bath, Bath

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9.40-9.50am Venous ulcer healing is associated with an increase in TGFβ1 in wound exudate

Gohel MS, Windhaber R, Taylor M, Tarlton JF, Whyman MR, Poskitt KR
Department of Vascular Surgery, Cheltenham General Hospital, Cheltenham

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9.50-10.00am Thrombogenicity and the unstable carotid plaque: gene expression profiling in symptomatic and asymptomatic patients

Sayed S, Cockerill G, Dawson J, Choke E, Loftus I, Thompson MM
Department of Vascular Surgery, St George's, University of London, London

page 32

10.00-10.10am **Cyclo-oxygenase-2 inhibition does not modify intimal hyperplasia but does increase graft thrombosis in a large animal model of carotid artery patch grafting**

McMonagle MP, Hawthorne WJ, Vicaretti M, Fletcher JP
Westmead Hospital and the University of Sydney, Australia

page 33

10.10-10.20am **Vascular endothelial growth inhibitor represents an important target for therapeutic angiogenesis in the lower limb**

Conway K, Harrison G, Price P, Harding KG, Jiang WG
Metastasis and Angiogenesis Research Group and the Wound Healing Research Unit, Wales College of Medicine, Cardiff University, Cardiff

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10.20-10.40am **Coffee**

10.40-11.10am **KEYNOTE LECTURE**

Reperfusion injury: host responses to hypoxia/reoxygenation of the limb
Mr Denis Harkin, Royal Victoria Hospital, Belfast

11.10-12noon **SURGICAL RESEARCH PAPERS**

11.10-11.20am **c-Jun N-terminal kinase-2 regulates skeletal muscle necrosis and remote acute lung injury in a mouse model of lower limb ischaemia-reperfusion injury**

Lewis A, Harkin DW, Degousee N, Stefanski E, Lindsay TF, Karin M, Rubin BB
Division of Vascular Surgery, The Toronto Hospital, Toronto, Ontario, Canada; Regional Vascular Surgery Unit, Royal Victoria Hospital, Belfast

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11.20-11.30am **A role for xanthine oxidoreductase in promoting the healing of chronic ulcers**

Bennett EJ, Winrow VR, Stevens CR, Horrocks M
Department of Surgery, School for Health, University of Bath, Bath

page 36

11.30-11.40am **The chemokine stromal-derived factor-1 (SDF-1/CXCL12) beta is the more potent variant in human microvascular cell survival and proliferation**

Ho TK [1], Xu S [2], Abraham DJ [2], Black CM [2], Baker DM [1]
[1] Vascular Unit, Department of Surgery and [2] Department of Rheumatology, The Royal Free Hospital, London

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11.40-11.50am

Plasma matrix metalloproteinases and IL 6 but not other acute phase cytokines are associated with embolic activity during carotid angioplasty: proteonomics and ELISA studies

Jindal R [1], Roberts G [2], Brightwell R [1], Peck D [2], Darzi A [2], Cheshire NJW [1,2]
[1] Regional Vascular Unit, St Mary's Hospital, London; [2] Division of Surgery, Imperial College School of Medicine, London

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11.50am-12noon

Interleukin-10 genotype: associated with AAA formation but not growth

Lloyd GM, Bown MJ, Thompson J, Sayers RD
Division of Vascular Surgery, Department of Cardiovascular Sciences, University of Leicester, Leicester

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9.00am-4.00pm **THE SOCIETY OF VASCULAR NURSES ANNUAL MEETING** **PURBECK LOUNGE**

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

THE VASCULAR SOCIETY MEETING

1.00-1.15pm Opening remarks and presentation of new Honorary Member
The President **TREGONWELL HALL**

1.15-2.15pm **Scientific session 1**
Chairmen: Mr Richard Corbett and Mr David Berridge

* Paper eligible for Venous Forum prize

1.15-1.25pm

Factors influencing the effectiveness of Endovenous Laser Treatment (EVLT) for varicose veins due to saphenofemoral (SF) and long saphenous (LSV) reflux*

Theivacumar N, Beale R, Mavor AID, Gough, MJ
The General Infirmary at Leeds, Leeds

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1.25-1.35pm

Superficial venous surgery for varicose veins affords the same improvement in generic health-related quality of life as elective laparoscopic cholecystectomy for symptomatic gall-stones*

Sam RC, Darvall KAL, Adam DJ, Silverman SH, Bradbury AW
University Department of Vascular Surgery, Heart of England NHS Trust, Birmingham

page 42

1.35-1.45pm **Prospective study of short saphenous varicose vein surgery: six weeks' results***

Vandenbroeck CP, Winterborn RJ, Hoult S, Campbell WB, Whitman B, Heather BP, Earnshaw JJ, on behalf of the Joint Vascular Research Group
Department of Vascular Surgery, Gloucestershire Royal Hospital, Gloucester

page 43

1.45-1.55pm **One-year results of a double-blinded randomised trial on the treatment of bilateral recurrent long saphenous varicose veins***

Hinchliffe R, Ubhi J, Beech A, Braithwaite BD
Queen's Medical Centre, Nottingham

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1.55-2.05pm **Initial experience in the treatment of varicose veins due to saphenopopliteal (SP) and short saphenous (SSV) reflux with Endovenous Laser Treatment (EVLT)***

Theivacumar N, Beale R, Mavor AID, Gough MJ
The General Infirmary at Leeds, Leeds

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2.05-2.15pm **Knee-length graduated compression stockings are as effective as thigh-length in thromboprophylaxis. A meta-analysis***

Sajid MS, Seifalian A, Hamilton G
Royal Free Hospital, London

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2.15-3.45pm **Scientific session 2 - BJS Prize**

Chairmen: Mr Peter Lamont and Mr Jonothan Earnshaw

2.15-2.30pm **Pre-operative ischaemia of the long saphenous vein predisposes to intimal hyperplasia in bypass grafts through enhanced smooth muscle cell migration**

Ruiz MC, Orr DJ, Teenan RP, Wadsworth RM
Peripheral Vascular Unit, Glasgow Royal Infirmary, Glasgow; Department of Physiology and Pharmacology, University of Strathclyde, Glasgow

page 47

2.30-2.45pm **Effect of low-dose (75mg) clopidogrel on platelet reactivity, ADP variability, and clopidogrel resistance when given before carotid surgery**

Payne DA, Jones CI, Hayes PD, Bell PRF, Goodal AH, Naylor AR
The Department of Cardiovascular Sciences, University of Leicester, Leicester

page 48

2.45-3.00pm **Mesenteric traction during open abdominal aortic aneurysm repair may lead to intestinal ischaemia**

Arya N, Lau LL, Lee B, Hannon RJ, Young IS, Soong CV
Vascular and Endovascular Unit, Belfast City Hospital, Belfast, Northern Ireland

page 49

3.00-3.15pm **Adenoviral delivery of the urokinase gene promotes venous thrombus resolution**

Gossage JA, Humphries J, Modarai B, Burnand KG, Smith A
Academic Department of Surgery, St Thomas' Hospital, King's College, London

page 50

3.15-3.30pm **Increased hypoxia inducible factor-1alpha and localisation of erythropoietin in human critical limb ischaemia**

Ho TK [1], Rajkumar V [2], Ponticos M [2], Garcia P [2], Khan K [2], Hart C [1], DiSalvo C [3], Walesby RK [3], Abraham DJ [2], Black C [2], Baker DM [1]
[1] Vascular Unit, Department of Surgery and [2] Department of Rheumatology, The Royal Free Hospital, London; [3] The Heart Hospital, London

page 52

3.30-3.45pm ***In vivo* attenuation of myointimal hyperplasia using transforming growth factor beta 3: an interposition graft model**

Murphy MO, Ghosh J, Khwaja N, Halka AT, Turner N, Ferguson MW, Kielty CM, Walker MG
Department of Vascular Surgery, Manchester Royal Infirmary, Manchester

page 53

3.45-4.15pm **Tea/Trade Exhibition** **PURBECK HALL**

4.15-5.15pm **Scientific session 3** **TREGONWELL HALL**

Chairmen: Mr John Wolfe and Mr Mo Adiseshiah

4.15-4.25pm **Biomechanical fatigue in aneurysmal abdominal aorta: a physical model of rupture**

Windhaber RAJ, Tarlton JF, Gohel MS, Poskitt KR, Earnshaw JJ, Mitchell DC
North Bristol NHS Trust, Bristol

page 54

4.25-4.35pm **Comparison of the fixation strength of fenestrated and non-fenestrated stent-grafts for endovascular abdominal aortic aneurysm repair (EVAR)**

Zhou SS, Brennan J, How TV, Gilling-Smith GL, Harris PL
Regional Vascular Unit, Royal Liverpool University Hospital, Liverpool

page 55

4.35-4.45pm

Aortic necks of ruptured abdominal aneurysms dilate more than asymptomatic aneurysms following endovascular repair

Badger SA, O'Donnell ME, Makar R, Boyd CS, Johnston LC, Loan W, Hannon RJ, Lau LL, Lee B, Soong CV
Vascular and Endovascular Unit, Belfast City Hospital, Belfast, Northern Ireland

page 56

4.45-4.55pm

Greater "oversizing" of aortic endografts is required for shorter aneurysm necks in endovascular aortic aneurysm repair (EVAR)

Zhou SS, Brennan J, How TV, Gilling-Smith GL, Harris PL
Regional Vascular Unit, Royal Liverpool University Hospital, Liverpool

page 57

4.55-5.05pm

Does acetylcysteine prevent contrast-induced nephropathy during endovascular AAA repair? A randomised controlled study

Moore NM, Lapsley M, Norden AG, Firth JD, Gaunt ME, Varty K, Boyle JR
Departments of Vascular Surgery, Clinical Biochemistry, and Renal Medicine, Cambridge University Hospitals NHS Trust, Cambridge; Clinical Biochemistry, Epsom and St Helier NHS Trust, Surrey

page 58

5.05-5.15pm

Impact of renal dysfunction on operative mortality following endovascular abdominal aortic aneurysm surgery

Statius van Eps RG, Leurs LJ, Buth J, Harris PL for the EUROSTAR Collaborators
Royal Liverpool University Hospital, Liverpool

page 59

5.15-6.15pm **SYMPOSIUM**

Renal Access for the Vascular Surgeon
Chairmen: Professor Michael Horrocks and Mr Chris Gibbons

Techniques and training
Mr David Mitchell, Bristol

Patient and operation selection
Professor Michael Nicholson, Leicester

Assessment of adequacy and maintenance
Professor Mitch Henry, Columbus, Ohio, USA

Complication, diagnosis and management
Dr Jan Tordoir, Maastricht, Holland

6.30-7.15pm **Welcome Civic Drinks Reception** **EXHIBITION AREA, PURBECK HALL**
Hosted by the Mayor of Bournemouth

Thursday 24th November 2005

7.00-8.00am **Breakfast Symposium on Medical Management** **PURBECK LOUNGE**

Future Model for Peripheral Arterial Disease (PAD) Management - What's best for patient, physician and politician?

Sponsored by sanofi-aventis/Bristol-Myers Squibb

Chairman: Professor Cliff Shearman

PAD and the GMS Contract
Dr George Kassianos, Berkshire

PAD and 'payment by results'
Professor Andrew Bradbury, Birmingham

A successful model for PAD
Professor Gerard Stansby, Newcastle

page 29

9.00am-4.30pm **SOCIETY FOR VASCULAR TECHNOLOGY ANNUAL MEETING** **PURBECK LOUNGE**

8.30-10.00am **Scientific session 4** **TREGONWELL HALL**

Chairmen: Professor Cliff Shearman and Mr Simon Ashley

+ Paper eligible for Richard Wood Prize

8.30-8.40am Expression of growth factors and growth factor receptor in non-healing and healing ischaemic ulceration

Murphy MO, Ghosh J, Khwaja N, Halka AT, Carter A, Turner N, Fulford P, Walker MG
Department of Vascular Surgery, Manchester Royal Infirmary, Manchester

page 60

8.40-8.50am The effects of major vascular surgery on platelet function

Rajagopalan S, Ford I, Bachoo P, Greaves M, Brittenden J
Departments of Vascular Surgery and Medicine and Therapeutics, University of Aberdeen, Aberdeen

page 62

8.50-9.00am The effects of acute exercise on haemostasis, inflammation and renal function in patients with intermittent claudication on statin and aspirin therapy

Collins P, Ford I, Croal B, Ball D, Greaves M, Macaulay E, Brittenden J
Departments of Vascular Surgery and Medicine and Therapeutics, University of Aberdeen, Aberdeen

page 63

9.00-9.10am

Abrogation of skeletal muscle reperfusion injury by simvastatin: the impact of nitric oxide synthase inhibition

Khanna A, Laws PE, Cowled PA, Fitridge RA
Department of Surgery, The University of Adelaide, The Queen Elizabeth Hospital, Adelaide, South Australia, Australia

page 64

9.10-9.20am

The anti-thrombogenic potential of a new nanocomposite polymer for the development of bypass grafts

Seifalian AM, Kannan R, Salacinski HJ, De Groot J, Bozec L, Horton M, Hamilton G
University College London, London

page 65

9.20-9.30am

Variability in responsiveness to clopidogrel in patients with intermittent claudication

Cassar K, Bachoo P, Ford I, Greaves M, Brittenden J
Departments of Vascular Surgery and Medicine and Therapeutics, University of Aberdeen, Aberdeen; Vascular Unit, Aberdeen Royal Infirmary, Aberdeen

page 66

9.30-9.40am

Transthoracic echocardiogram in the management of acute limb ischaemia

Lewis A, Kirk G, Kothurkar A, McKinley A, Blair PH, Hood JM, Harkin DW
Regional Vascular Surgery Unit, Royal Victoria Hospital Belfast, Belfast

page 67

9.40-9.50am

The effect of supervised exercise and cilostazol on coagulation and fibrinolysis in patients with intermittent claudication

Hobbs SD, Fegan C, Adam DJ, Bradbury AW
University Department of Vascular Surgery, Birmingham Heartlands Hospital, Birmingham

page 68

9.50-10.00am

Carotid endarterectomy under local anaesthetic - evaluating a high fidelity simulated environment for training and assessment+

Horrocks EJ [1,2], Black SA [1,2], Pandey VA[1,2], Harrison RH [1,2], Wetzel CM [2], Nestel D [2], Kneebone R [2], Wolfe JHN [1]
[1] Regional Vascular Unit, St Mary's Hospital, London; [2] Division of Surgery, Oncology, Reproductive Biology and Anaesthesia, Imperial College, London

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10.00-10.30am **Coffee/Trade Exhibition** **PURBECK HALL**

10.30-11.30am **Scientific session 5** **TREGONWELL HALL**

Chairmen: Mr Tim Lees and Mr Rajiv Vohra

+ Paper eligible for Richard Wood Prize

10.30-10.40am How cost-effective is screening for abdominal aortic aneurysms? A long-term perspective based on the MASS trial+

Kim LG, Thompson SG, Briggs AH, Buxton MJ, Campbell HE
MRC Biostatistics Unit, Cambridge

page 70

10.40-10.50am Abdominal Aortic Aneurysm (AAA) development following a "normal" aortic ultrasound scan

Hafez H, Druce S, Scott RAP, Ashton H
Scott Research Unit, St Richard's Hospital, Chichester

page 72

10.50-11.00am Statins are associated with reduced all-cause mortality after endovascular abdominal aortic aneurysm repair

Leurs LJ, Visser P, Laheij RJF, Buth J, Blankensteijn JD, Harris PL, on behalf of the EUROSTAR collaborators
Royal Liverpool University Hospital, Liverpool

page 73

11.00-11.10am Abdominal Aortic Aneurysms (AAA) and the metabolic syndrome

Al-Barjas HSA, Ariëns RAS, Grant PJ, Scott DJA
The Academic Unit of Molecular Vascular Medicine; The L.I.G.H.T Laboratories; University of Leeds, Leeds

page 74

11.10-11.20am The IL-10 -1082 gene polymorphism: a candidate gene for abdominal aortic aneurysms

Lloyd GM, Bown MJ, Sayers RD
Division of Vascular Surgery, Department of Cardiovascular Sciences, University of Leicester, Leicester

page 75

11.20-11.30am Increased angiogenesis and activation of the HIF-1α/VEGF pathway in abdominal aortic aneurysm rupture

Choke E, Cockerill G, Wilson WR, Dawson J, Sayed S, Loftus I, Thompson MM
St George's, University of London, London

page 76

11.30am-12.30pm **SYMPOSIUM**

Carotid Stenting - The Way Forward?

Chairmen: Professor Michael Horrocks and Mr John Wolfe

Comparisons of carotid practice in the UK and elsewhere

Professor Nick Cheshire, London

Patient device selection

Dr Marc Bosiers, Dendermonde, Belgium

How to organise a stenting service

Professor Piergiorgio Cao, Perugia, Italy

Summary of evidence to date

Dr Trevor Cleveland, Sheffield

Panel Discussion

12.30-1.00pm **GUEST LECTURE**

The new vascular training programme in Australasia: a model for the future?

Lecturer: Professor Rob Fitridge, Adelaide, Australia

1.00-2.00pm **Lunch and Trade Exhibition** **PURBECK HALL**

2.00-3.30pm **Scientific session 6 - Sol Cohen (Founder's) Prize** **TREGONWELL HALL**

Chairmen: Professor Michael Horrocks and Professor Julian Scott

2.00-2.15pm Endovenous Laser Treatment (EVLT) or surgery for varicose veins? A randomised controlled trial in patients with saphenofemoral and long saphenous incompetence

Beale R, Theivacumar N, Mavor AID, Gough MJ

The General Infirmary at Leeds, Leeds

page 77

2.15-2.30pm Topical bupivacaine in the long saphenous vein tract provides excellent analgesia: a prospective double-blind randomised study comparing bupivacaine with placebo following varicose vein surgery

Kibria SMG, Mavor AID

The General Infirmary at Leeds, Leeds

page 78

2.30-2.45pm **A hybrid screening programme for clinically significant abdominal aortic aneurysms**

Heng MST, Venkatasubramaniam A, Lee DLH, Bryce J, Tennison C, Berry B, Chetter I, McCollum PT
Academic Vascular Unit, Hull Royal Infirmary, Hull; University of Hull, Hull

page 79

2.45-3.00pm **Results of open Abdominal Aortic Aneurysm (AAA) repair via a left upper quadrant transverse transperitoneal minilaparotomy incision**

Subramanian A, Sutaria R, Witcomb M, Hafez H
St Richard's Hospital, Chichester

page 80

3.00-3.15pm **The constitutive procoagulant and hypofibrinolytic state in patients with intermittent claudication significantly improves with percutaneous transluminal balloon angioplasty**

Hobbs SD, Fegan C, Adam DJ, Bradbury AW
University Department of Vascular Surgery, Birmingham Heartlands Hospital, Birmingham

page 82

3.15-3.30pm **Acquisition of endovascular skills by consultant vascular surgeons: effect of repetition in a virtual reality training model**

Aggarwal R [1], Black SA [1,2], Hance JR [1], Darzi AW [1], Cheshire NJW [1,2]
[1] Department of Biosurgery and Surgical Technology, Imperial College, London; [2] Regional Vascular Unit, St Mary's Hospital, London

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3.30-4.00pm **Tea/Trade Exhibition** **PURBECK HALL**

4.00-5.00pm **FOR DISCUSSION: THE FUTURE DIRECTION OF VASCULAR SURGERY** **TREGONWELL HALL**

The President and Chairman of the Training and Education Committee

5.00-6.00pm **Annual Business Meeting** **TREGONWELL HALL**

5.00-6.00pm **Rouleaux Club AGM** **PURBECK LOUNGE**

7.30 for 8.00pm **Annual dinner with entertainment** **THE BOURNEMOUTH PAVILION**

Friday 25th November 2005

7.45-8.45am **SYMPOSIUM** **TREGONWELL HALL**

The management of limb-threatening infra-inguinal vascular disease: results of the Bypass versus Angioplasty in Severe Ischaemia of the Leg (BASIL) Trial

Introduction: Professor Vaughan Ruckley

7.45-7.55am Severe Limb Ischaemia: The need for level I evidence
Professor Andrew Bradbury

7.55-8.05am Current management of SLI in the UK: The BASIL Trial Audit
Professor Gerry Fowkes/Mr Donald Adam

8.05-8.25am First presentation of BASIL Trial results
Aims and Methodology
Dr Joceyln Bell/Helen Storkey
Results
Professor Gillian Raab/Dr John Forbes

8.25-8.45am Panel Discussion, involving all speakers
Chairman: Professor Vaughan Ruckley

9.00-10.30am **Scientific session 7** **TREGONWELL HALL**

Chairmen: Mr Kevin Varty and Mr Frank Smith

9.00-9.10am VEGF gene therapy enhances venous thrombus resolution

Modarai B, Humphries J, Gossage JA, Burnand KG, Afuwape A, Paleolog E, Smith A
St Thomas' Hospital, Cardiovascular Division, King's College, London

page 84

9.10-9.20am Early experience of endovenous laser ablation of the short saphenous vein

Watson AB, Bani-Hani M, Modaresi K, Greenstein D
Department of Vascular Surgery, Northwick Park Hospital, Harrow

page 85

9.20-9.30am A double-blinded, randomised study to determine the effect of omega-3-marine triglycerides on intermittent claudication

Conway K, Dillon M, Evans J, Howells-Jones R, Price P, Harding KG, Hill S
Cardiff Regional Vascular Unit, University Hospital of Wales, Cardiff

page 86

9.30-9.40am **Risk factors for the development and subsequent growth of small abdominal aortic aneurysms**

Wilmink ABM, Adam DJ, Hubbard CS, Bradbury AW, Quick CRG
University Department of Vascular Surgery, Birmingham Heartlands Hospital, Birmingham

page 87

9.40-9.50am **Self-assessment of technical skill: the need for expert feedback**

Pandey VA, Wolfe JHN, Black SA, Liapis CD, Bergqvist D
On behalf of the European Board of Vascular Surgery

page 88

9.50-10.00am **A change in isolation policy reduces MRSA colonisation ten-fold**

Thompson MM, on behalf of the St George's Vascular Department
Department of Vascular Surgery, St George's Hospital, London

page 89

10.00-10.10am **Carbon monoxide-releasing molecules (CO-RMs) modulate the neuro-inflammatory response in BV-2 microglia: a novel approach to stroke**

Bani-Hani MG, Greenstein D, Mann BE, Green C, Motterlini R
Northwick Park Institute for Medical Research, Harrow; North West London Hospitals NHS Trust, Northwick Park Hospital, Harrow

page 90

10.10-10.20am **Value of MRI in post-procedural evaluation of carotid angioplasty and stenting**

McDonnell CO [1], Fearn SJ [1], Baker SR [1], Price D [2], Goodman MA [1], Lawrence-Brown MMD [1]
[1] Departments of Vascular Surgery and [2] Radiology, Mount Medical Centre, Perth, Western Australia

page 91

10.20-10.30am **The current performance of carotid endarterectomy (CEA) in the UK: an interim analysis of 1001 patients randomised in the GALA trial**

Dellagrammaticas D, Gough MJ, on behalf of the GALA Trial participants
The General Infirmary at Leeds, Leeds

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10.30-11.00am **Coffee/Trade Exhibition** **PURBECK HALL**

11.00-11.30am **GUEST LECTURE** **TREGONWELL HALL**

The trials and tribulations of a surgical editor

Lecturer: Professor Torben Schroeder, Copenhagen, Denmark

11.30am-12.30pm **SYMPOSIUM**

The Future Development of Vascular Services

Chairman: Professor Michael Horrocks

11.30-12noon The Implications of Aortic Aneurysm Screening

Professor Sir Muir Gray, Programme Director, National Screening Committee

Open Discussion

12noon-12.30pm The Future Audit of Carotid Surgery and the National Vascular Database

Mr Simon Ashley, Chairman, Audit and Research Committee

Open Discussion

12.30-12.35pm **Inauguration of the new President**

12.35-1.15pm **THE KINMONTH LECTURE**

Operative vascular training and assessment: the last century, the present and the future

Chairman: Mr Tony Giddings, Council Member, RCS(Eng)

Lecturer: Mr John Wolfe, London

1.15-2.00pm **Lunch and Trade Exhibition** **PURBECK HALL**

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.

Breakfast Session

Future model for peripheral arterial disease (PAD) management What's best for patient, physician and politician?

A breakfast symposium chaired by Professor Cliff Shearman

Thursday 24 November, 2005 - 7.00am - 8.00am
Purbeck Lounge - Bournemouth International Centre, Bournemouth

I would like to invite you to a breakfast symposium, on the Thursday of the Vascular Society Annual Scientific Meeting. Topical issues affecting the medical management of vascular patients will be considered, including the following: how the GMS contract may impact upon patients with PAD; the adoption of 'payment by results' by vascular surgeons and how this may change our role in the treatment of PAD; and a look at how primary and secondary care can work together to improve the service provided for patients with PAD. Speakers, who are recognised as experts in their field, will provide a stimulating session, which promises to impact upon, inform, and influence our vascular practice.

PAD and the GMS contract - Dr George Kassianos, Berkshire

- Is the prognosis for patients with PAD now worse?
- What impact will this have on vascular surgeons?

PAD and 'payment by results' - Professor Andrew Bradbury, Birmingham

- What is payment by results (PbR)?
- How should the vascular surgeon adopt PbR?
- How will PbR change the role of the vascular surgeon in the treatment of PAD?

A successful model for PAD - Professor Gerard Stansby, Newcastle

- How can primary and secondary care work together to improve the service provided for patients with PAD?
- What is the role of the vascular surgeon in a successful model?

Speakers will address their questions in succinct 10-minute presentations, followed by audience discussion. This promises to provide lively debate, to inform vascular surgeons and nurses on practical issues for everyday practice in vascular disease.

Breakfast will be available at the Purbeck Lounge, Bournemouth International Centre, from 6.45am.

I look forward to seeing you at what promises to be a stimulating and informative event.

Cliff Shearman
Consultant in Vascular Surgery
Southampton University Hospitals

This symposium is sponsored by an educational grant from sanofi-aventis and Bristol-Myers Squibb PLA-05/249

Abstracts

23rd-25th November 2005
Bournemouth International Centre

Differing levels of transcription factors and MMPs in different regions of Abdominal Aortic Aneurysm (AAA) wall help identify the sequence of signalling pathways in AAA development

Erdozain O, Bodamyali T, Stevens C, Horrocks M
Department of Surgery, School for Health, University of Bath, Bath

Objective

Upregulation of MMP-2 and later MMP-9 are implicated as the major factors in AAA development. Aortic wall pO_2 is reduced in aneurysmal aorta. We have shown that localised hypoxia of vascular smooth muscle cells *in vitro* correlates with upregulation of hypoxia inducible factor-1α (HIF-1α), MMP-2 and MMP-9. mRNA expression profiles for MT-MMP-1, MMP-3, MMP-7, MMP-1, MMP-2, MMP-9 and TIMP-1 have been evaluated for regions of aneurysm sac and site of rupture.

Method

Proximal, mid and distal regions of AAA sac were sampled at operation (40 elective and 8 ruptured). Zymography and RT and multiplex PCR were carried out on all regions. Immunohistochemistry with monoclonal anti-human MMP-2, MMP-9 and HIF-1α was performed on 5μm serial wax sections. Secreted MMPs were quantified using gelatin zymography with densitometric analysis. Control vascular smooth muscle cells (VSMC) from normal human aortic tissue were exposed to various levels of hypoxia over 72 hours in a controlled environment and also evaluated.

Results

Analysis of the three aneurysmal regions reveal distinct differences for MMP mRNA and protein levels. In the proximal segment there is increased MMP-2 mRNA, in the mid region MMP-3, MT-MMP-1 and MMP-7 are increased, distally MMPs-1, -2 and -9 are increased. MMP-2 is localised to the tunica media distant from the vasa vasorum and the inflammatory infiltrate. Nuclear staining patterns show an increase in HIF-1α with expression increased in proximal and mid areas. Results of the localised hypoxia *in vitro* analysis reveal induction of MMP-2, MMP-1, TIMP-1 and MT-MMP-1 mRNA synthesis in addition to the elevated MMP-2 secretion.

Conclusion

The finding of MMP-2 at sites furthest from the source of oxygen suggests possible hypoxic activation. The results further support the role of HIF-1α in the complex signalling cascade that mediates collagen and elastin degradation and aneurysmal development. The difference in regions of aneurysm wall reflect the sequence of signalling events in aneurysm wall destruction.

Venous ulcer healing is associated with an increase in TGFβ1 in wound exudate

Gohel MS, Windhaber R, Taylor M, Tarlton JF, Whyman MR, Poskitt KR
Department of Vascular Surgery, Cheltenham General Hospital, Cheltenham

Objective

Protracted healing in chronic venous ulcers is thought to be due to a disorder of the normal function of wound cytokines, but remains poorly understood. The relationship between changes in cytokine levels in venous ulcer exudate and healing was evaluated in this study.

Method

Consecutive patients with chronic leg ulceration and ABPI >0.85 were prospectively investigated. All patients were treated with multilayer compression bandaging. Wound fluid samples were taken at recruitment and 6 weeks later by aspiration under a clear adhesive dressing. In the wound fluid, cytokines reflecting the processes of inflammation (IL1β, TNFα), proteolysis (MMP2, MMP9), angiogenesis (bFGF, VEGF) and fibrosis (TGFβ1) were measured. Ulcer healing was assessed using digital planimetry.

Results

Eighty patients (43 male, 37 female) were investigated. Median (range) ulcer size reduced from 4.4cm^2 (0.1-142.4) to 2.2cm^2 (0-135.5) after 6 weeks ($p<0.001$; Wilcoxon-Signed rank), although 17/80 ulcers increased in size. Wound fluid collection was successful in 52/80 (65%) patients initially and 32/80 (40%) at both assessments. Volume of wound fluid collected strongly correlated with ulcer size (Spearman Rank 0.801, $p<0.001$). In the patients where wound fluid was collected at both assessments, changes in TGFβ1 levels correlated with ulcer healing (Pearson coefficient -0.642, $p=0.033$). There were no significant correlations between changes in other cytokines and ulcer healing.

Conclusion

Wound fluid collection correlates with ulcer size. Ulcer healing correlated with increased concentrations of TGFβ1, possibly due to increased fibrosis in the proliferating wound. A relationship between other cytokines and ulcer healing was not proven in this study.

Thrombogenicity and the unstable carotid plaque: gene expression profiling in symptomatic and asymptomatic patients

Sayed S, Cockerill G, Dawson J, Choke E, Loftus I, Thompson MM
Department of Vascular Surgery, St George's, University of London, London

Objective

The aim of this study was to identify a role for thrombomodulation in the evolution of the unstable plaque. The thrombogenicity of carotid plaques was assessed by measuring the gene expression of thrombomodulatory factors.

Method

Patients were classified into asymptomatic (group 1, n=11), early symptomatic (group 2, n=9) and late symptomatics (group 3, n=10) depending on their symptom-free duration (≤1 month/≥1 month) prior to surgery. Plaques retrieved at carotid endarterectomy were processed using real time quantitative RT-PCR to measure levels of tissue factor (TF), tissue factor pathway inhibitor, plasminogen activator inhibitor-1 (PAI-1), urokinase plasminogen activator, tissue plasminogen activator (tPA), thrombomodulin (TM), VE-Cadherin and CD68. To determine the relative RNA levels each patient sample was standardised to endogenous control (18SrRNA). Results expressed as medians were compared using Kruskall-Wallis analysis of variance.

Results

There was a significant increase in expression of thrombomodulatory factors in group 2, the recently symptomatic group, with a 20-fold increase in PAI-1 and tPA (PAI-1: 134 versus 6 and 6 for groups 1 and 3, p=0.046; tPA: 60 versus 3 and 3, p=0.026). Similarily, there was a 17-fold increase in TF (17 versus 1.2 and 1, p=0.036), and a 10-fold increase in TM (24 versus 3 and 2, p=0.033).

Conclusion

Within 1 month of a clinical event, the plaque is most active and is characterised by an upregulation of thrombomodulatory factors. Subsequently, levels are similar to those in asymptomatic plaques. This reflects the dynamic nature of the thrombogenic process, which may contribute to the development of the unstable plaque.

Cyclo-oxygenase-2 inhibition does not modify intimal hyperplasia but does increase graft thrombosis in a large animal model of carotid artery patch grafting

McMonagle MP, Hawthorne WJ, Vicaretti M, Fletcher JP
Westmead Hospital and the University of Sydney, Australia

Objective

Intimal hyperplasia (IH) is a troublesome complication occurring after vascular surgery and is the leading cause of restenosis in both the medium and long term. Cyclo-oxygenase-2 enzyme (COX-2) inhibition is a potential pharmacological target that has shown some promise in reducing neointimal formation after vascular intervention. We wanted to assess the utility of the Cox-2 inhibitor SC-76309 as a potential modifier of intimal hyperplasia after carotid artery patch grafting in an ovine model.

Method

Fifteen sheep underwent patch grafting of the left common carotid artery. Treatment consisted of a once daily intramuscular dose of drug (low or high dose respectively) or 0.9% saline. The animals were treated for a continuous 28-day period after which they were euthanased and the carotid arteries with graft *in situ* were removed for further analysis. Results were analysed using statistical regression mixed effects models for continuing variables and significance was set at 5% throughout.

Results

The overall IH index, IH area / lumen area was not significantly reduced between the treated groups and the controls (p=0.34 and 0.72 respectively). However, the incidence of graft thrombosis was significantly higher for the treated groups compared to the controls.

Conclusion

The use of COX-2 inhibitors does not alter the formation of intimal hyperplasia after carotid artery patch grafting in an ovine model. However, vascular thrombosis has recently been recognised as a complication of COX-2 inhibition, which is reflected in the high incidence of graft thrombosis in our study. The role of COX-2 inhibition in vascular disease is now uncertain.

Vascular endothelial growth inhibitor represents an important target for therapeutic angiogenesis in the lower limb

Conway K, Harrison G, Price P, Harding KG, Jiang WG

Metastasis and Angiogenesis Research Group and the Wound Healing Research Unit, Wales College of Medicine, Cardiff University, Cardiff

Objective

Therapeutic angiogenesis aims to enhance collateral vessel formation in peripheral arterial disease. To obtain this therapeutic goal it is necessary to identify the molecules that inhibit angiogenesis in a normally quiescent adult vasculature. Vascular endothelial growth inhibitor (VEGI) is thought to be involved in suppressing the proliferation and differentiation of endothelial cells. The aim of this study is to assess the ability of VEGI to inhibit angiogenesis using an *in vitro* model.

Method

Ribozyme transgene targeting human VEGI and an expression cassette for human VEGI were constructed using a mammalian expression vector. A human endothelial cell line (HECV) was transfected with the VEGI hammerhead ribozyme transgene, or the VEGI expression vector, respectively. The *in vitro* angiogenic properties of these cells were analysed and compared with the wild-type HECV cells.

Results

Over-expression of VEGI in vascular endothelial cells significantly reduced their capability of forming microtubules *in vitro* (762.9 ± 155.6μm in wild-type, compared with 273.3 ± 150.5μm in VEGI over-expressing cells, $p<0.0001$). A similar reduction was also seen when these endothelial cells were treated with an angiogenic factor, hepatocyte growth factor (HGF). HGF significantly increased tubule forming in wild-type endothelial cells (1379.3 ± 297.1μm, $p<0.0001$ vs without HGF). However, the response was reduced in VEGI over-expressing cells (949.9 ± 94.1μm, $p=0.0019$ vs wild-type).

Conclusion

This study confirms that VEGI acts as a suppressor to the proliferation and microtubule formation of endothelial cells. VEGI is therefore a major regulator of the angiogenic process and represents an important target for therapeutic angiogenesis in the lower limb.

c-Jun N-terminal kinase-2 regulates skeletal muscle necrosis and remote acute lung injury in a mouse model of lower limb ischaemia-reperfusion injury

Lewis A, Harkin DW, Degousee N, Stefanski E, Lindsay TF, Karin M, Rubin BB

Division of Vascular Surgery, The Toronto Hospital, Toronto, Ontario, Canada; Regional Vascular Surgery Unit, Royal Victoria Hospital, Belfast

Objective

Prolonged acute limb ischaemia (ALI) causes skeletal muscle necrosis and on reperfusion may initiate an inflammatory response characterised by neutrophil activation and remote acute lung injury. This study aimed to define the role of the intracellular transcription factor, c-Jun N-terminal kinase (JNK) enzymes, implicated in the cellular response to stress in ALI.

Method

JNK1- and 2-knock-out (ko) mice or wild-type (wt) littermates were subjected to 180 minutes of unilateral hindlimb tourniquet ischaemia, followed by 24 hours reperfusion. Gastrocnemius muscle viability was measured by nitroblue tetrazolium staining and computerised planimetry. Myeloperoxidase (MPO) and wet-weight measured neutrophil sequestration and oedema, respectively.

Results

Skeletal muscle viability following ischaemia-reperfusion was significantly increased in JNK2-ko mice compared to JNK2-wt controls, (52.8 ± 6.9% versus 0.2 ± 0.1%, $p<0.004$). Muscle oedema was significantly reduced in JNK2-ko mice compared to JNK2-wt controls, (1.2 ± 0.1 versus 1.6 ± 0.1, $p<0.04$). Muscle MPO was significantly increased in JNK2-ko mice compared to JNK2-wt controls, (0.019 ± 0.004 versus 0.007 ± 0.004, $p<0.02$). Lung MPO was significantly reduced in JNK2-ko mice compared to JNK2-wt controls, (0.049 ± 0.009 versus 0.195 ± 0.06, $p<0.01$). JNK1-ko mice resembled controls. Data represent mean ± standard error mean (SEM), ANOVA and Scheffe's test.

Conclusion

This study clearly shows that JNK2 is an important regulator of events culminating in muscle necrosis and remote lung injury. Inhibition of JNK2 activity may decrease muscle necrosis in those patients with limb ischaemia, and protect against acute neutrophil-mediated pulmonary damage.

A role for xanthine oxidoreductase in promoting the healing of chronic ulcers

Bennett EJ, Winrow VR, Stevens CR, Horrocks M
Department of Surgery, School for Health, University of Bath, Bath

Objective

The enzyme xanthine oxidoreductase (XOR) is a complex molybdoflavoprotein with broad substrate specificity. In aerobic conditions XOR catalyses the production of the bactericidal agents superoxide (O_2.-) and hydrogen peroxide (H_2O_2) by the reduction of molecular oxygen (O_2). We have shown that, under hypoxic conditions, XOR can reduce inorganic nitrate and nitrite to nitric oxide (NO). Superoxide (O_2.-), and NO rapidly interact to generate peroxynitrite (ONOO-) a more potent antimicrobial species. Hypoxia is a feature of chronic ulcers and encourages the growth of facultative bacteria. We postulate that, in the hypoxic environment of the chronic ulcer, XOR can aid healing through the generation of reactive species.

Method

Adult human dermal fibroblasts were treated with increasing XOR concentrations (0-50mU ml^{-1}) and exposed to varying oxygen concentrations (0%-21% O_2 / 5% CO_2 / balance N_2). DNA synthesis, proliferation and cytotoxicity were assessed using bromodeoxyuridine incorporation or MTT reduction. Facultative bacterial strains relevant to the chronic wound: *Escherichia coli* MC 4100, *Proteus mirabilis* DV429, *Streptococcus faecalis* 775, *Staphylococcus aureus* NCTC (National Collection of Type Cultures) 6571 were grown in the same range of oxygen concentrations and their sensitivity to XOR generated species assessed using growth curves and colony counts.

Results

High levels of XOR were shown to be cytotoxic to adult fibroblasts whereas lower levels appeared to increase DNA synthesis and proliferation. Optimal proliferation occurred at XOR activities at 1mU ml^{-1}. Bacterial strains responded differentially to XOR but all showed growth inhibition at levels >10mU ml^{-1}.

Conclusion

This study suggests that XOR has therapeutic potential in chronic wounds through the generation of reactive species which, while toxic to bacteria, can induce fibroblast proliferation. XOR is now being developed as a wound dressing.

The chemokine stromal-derived factor-1 (SDF-1/CXCL12) beta is the more potent variant in human microvascular cell survival and proliferation

Ho TK [1], Xu S [2], Abraham DJ [2], Black CM [2], Baker DM [1]

[1] Vascular Unit, Department of Surgery and [2] Department of Rheumatology, The Royal Free Hospital, London

Objective

Stromal-derived factor-1 (SDF-1/CXCL12) is an angiogenic chemokine with two spliced variants, alpha and beta. We aim to investigate the effects of these variants on proliferation, apoptosis and signalling pathways in human microvascular cells.

Method

Human dermal microvascular cells (HMEC-1) were grown to confluence in media supplemented with 10% foetal calf serum (FCS). The cells were then incubated in media supplemented with 0.5% FCS with or without chemokines. We assessed cell proliferation using the cell count method on day 4. To induce apoptosis, the cells were serum starved for 24 hours and the percentage of apoptotic HMEC-1 was determined by counting the pyknotic nuclei after DAPI staining. The activity of signalling pathways over 24 hours was measured using Western blot analysis. All experiments were performed in triplicate. Statistical analysis used Student's t-test.

Results

Both SDF-1 variants attenuated HMEC-1 apoptosis (control versus SDF-1alpha, 28.2 ± 4.3 versus 12.9 ± 2.8%, $p<0.001$; control versus SDF-1beta, 28.2 ± 4.3 versus 9.7 ± 1.1%, $p<0.0001$). The percentage of apoptosis was significantly lower in SDF-1beta-treated cells compared to SDF-1alpha ($p<0.05$). Cell proliferation was also stimulated by both SDF-1 isoforms (control versus SDF-1alpha versus SDF-1beta [53.7 ± 2.5 versus 80.0 ± 9.0 versus 114.7 ± 4.5 x 10^3 cells, $p<0.02$]). SDF-1beta had a significantly higher proliferative effect compared to SDF-1alpha ($p<0.01$). SDF-1alpha and SDF-1beta induced Akt phosphorylation and activation of the Akt pathway.

Conclusion

Our results suggest that although both SDF-1 variants have anti-apoptotic and proliferative effects on HMEC-1, SDF-1beta is the more potent variant. The Akt pathway may play an important role in these effects. The use of the more potent SDF-1beta to stimulate angiogenesis may be of benefit to patients with peripheral vascular disease.

Plasma matrix metalloproteinases and IL 6 but not other acute phase cytokines are associated with embolic activity during carotid angioplasty: proteonomics and ELISA studies

Jindal R [1], Roberts G [2], Brightwell R [1], Peck D [2], Darzi A [2], Cheshire NJW [1,2]

[1] Regional Vascular Unit, St Mary's Hospital, London; [2] Division of Surgery, Imperial College School of Medicine, London

Objective

Endovascular treatment of carotid artery stenosis is potentially an attractive alternative to CEA in the prevention of stroke. Several parameters of inflammation have been related to unstable coronary plaques, but those associated with carotid plaque instability during endoluminal intervention are not understood. We hypothesised that measures of plaque inflammation may predict embolic potential during carotid endoluminal intervention.

Method

Fifty-five patients (23 asymptomatic and 32 symptomatic) who underwent carotid endarterectomy were included. Pre-operative plasma samples were collected. We used human cytokine protein arrays to detect 110 cytokines in a subgroup of patients to identify sets of cytokines involved in the pathogenesis. ELISA confirmed the protein array findings. Circumferentially intact carotid endarterectomy specimens underwent a standardised angioplasty procedure in a pulsatile *ex vivo* model. Emboli collected in distal filters were counted. Immunohistochemistry was carried out on the atherosclerotic plaques.

Results

Preliminary studies using protein arrays showed higher levels of plasma MMP 9 and IL 6 but failed to show presence of other cytokines in the plasma of a subgroup of these patients. Plasma CRP, IL 6 and MMP 9 levels (ELISA) showed correlation with distal emboli number ($p=0.02$, $p= 0.05$, $p= 0.04$). Both CRP and MMP 9 also correlated with intraplaque macrophage grading. Symptomatic patients had higher macrophage scores in the carotid plaque ($p=0.05$) and higher total number of distal emboli ($p=0.04$).

Conclusion

These findings suggest that inflammation within human carotid plaques is associated with an increased risk of embolisation during endoluminal treatment of carotid disease. Protein array detection supports the fact that the main inflammatory markers involved in the pathogenesis of carotid embolisation are CRP and MMPs.

Interleukin-10 genotype: associated with AAA formation but not growth

Lloyd GM, Bown MJ, Thompson J, Sayers RD

Division of Vascular Surgery, Department of Cardiovascular Sciences, University of Leicester, Leicester

Objective

It is probable that similar mechanisms are responsible for the formation and growth of abdominal aortic aneurysms (AAA). A functional single nucleotide polymorphism in the Interleukin-10 gene (IL-10 -1082 G/A) is associated with the presence of an AAA. The aim of this study was to determine whether this polymorphism also influences AAA growth.

Method

A prospective-retrospective study of 178 patients with AAA (3.0 to 5.5cm). Patient data and a blood sample were obtained. IL-10 -1082 genotype was determined by induced heteroduplex genotyping. Current and previous AAA sizes were recorded and further measurements were recorded over a further period of 1 year.

Results

Median patient age was 71 years (51-89). Total follow-up was 604 person-years (mean 3.4). Allele frequencies were in Hardy-Weinberg equilibrium with a frequency of: -1082 G:A 0.49:0.51. Mean growth rate was not significantly different between patients with an IL-10 -1082 A allele (associated with AAA) (mean 0.279cm/yr, SD 0.290) and those without (0.224cm/yr, SD 0.235, p=0.28 Student's t-test). Mean growth rate increased stepwise with each additional A allele present, although this finding was not statistically significant. Growth rates for each genotype were as follows: GG (mean 0.224cm/yr, SD 0.235), GA (0.270cm/yr, SD 0.232), AA (0.301cm/yr, SD 0.398), p=0.468, one-way ANOVA. Growth rate correlated positively with smoking (p=0.02, Student's t-test) and age and sex (p=0.025 and p<0.001 respectively, Pearson).

Conclusion

AAA growth rate is highly variable. Rapid growth is associated with smoking, age and sex but not IL-10 genotype, a risk factor for AAA formation.

Factors influencing the effectiveness of Endovenous Laser Treatment (EVLT) for varicose veins due to saphenofemoral (SF) and long saphenous (LSV) reflux

Theivacumar N, Beale R, Mavor AID, Gough MJ
The General Infirmary at Leeds, Leeds

Objective

EVLT, a minimally-invasive out-patient procedure, is an alternative to conventional surgery for treating varicose veins due to SF/LSV incompetence. This study assesses factors that might influence its effectiveness in abolishing reflux.

Method

Maximum LSV diameter, length of treated vein, total laser energy (TLE) administered, energy density (ED: pulses/cm), and body mass index (BMI) were collected prospectively in patients undergoing EVLT (810nm diode laser, 12 watts power, 1 sec pulses). Data were compared from limbs with complete LSV occlusion (group 1) and those where SF/LSV reflux persisted (group 2).

Results

The LSV was fully occluded/non-visible (duplex ultrasound) in 432/476 (91%) limbs completing 6-month follow-up (group 1). In 44 limbs (group 2) the LSV was partially occluded (18) or patent (26). Neither BMI (group 1: 25.2 [23.0-28.5]; group 2: 25.1 [24.3-26.2]), which might affect the efficacy of post-treatment compression, nor LSV diameter (7.3mm [5.7-9.2] versus 6.8mm [5.5-7.7]) influenced occlusion rates. However, the median (± iq range) TLE delivered to group 1 was 1878 J (998-2351) at an ED of 5.1 (4.2-5.7) pulses/cm. These were significantly greater ($p<0.01$) than corresponding data for group 2 (1190 J [1032-1406], 3.6 [3.0-4.6] pulses/cm). Although the TLE delivered is partly explained by the greater LSV length treated in group 1 (33cm v 29cm, $p<0.05$) this does not influence ED. Further, complete LSV occlusion occurred in all LSVs receiving ≥5 pulses/cm.

Conclusion

The ED (pulses/cm) of laser delivery is the main determinant of successful LSV ablation by EVLT. LSV diameter and BMI do not appear to influence its effectiveness.

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Superficial venous surgery for varicose veins affords the same improvement in generic health-related quality of life as elective laparoscopic cholecystectomy for symptomatic gall-stones

Sam RC, Darvall KAL, Adam DJ, Silverman SH, Bradbury AW
University Department of Vascular Surgery, Heart of England NHS Trust, Birmingham

Objective

To compare changes in generic health-related quality of life (HR-QoL) after superficial venous surgery (SVS) and elective laparoscopic cholecystectomy (ELC).

Method

The Short-Form 12 (SF-12) was sent by post to patients (SVS: n=146, ELC: n=73) pre-operatively and at 3m, 6m and 12m postoperatively. Physical (PCS) and mental component summary (MCS) scores were calculated at each time-point.

Results

Pre-operatively, 3m and 12m after surgery, patients in the ELC group had a significantly lower (worse) PCS than those in the SVS group (39.3 vs. 49.4, 49.0 vs. 53.1, 45.6 vs. 53.8, p=0.001, 0.021, 0.001 Mann-Whitney U test [MWU]). However, the change in PCS from pre-operative to 3m, 6m and 12m was not significantly different between the two groups. Patients in the ELC group had a significantly lower MCS than those in the SVS group pre-operatively only (46.9 vs. 50.9, p=0.002 MWU) and there was no difference in change in MCS between the two groups.

Conclusion

Although patients who undergo LC have a worse HR-QoL pre-operatively than those who are to undergo SVS, the improvement in HR-QoL gained following each operation is not significantly different. These data provide further evidence that SVS for varicose veins should not be 'rationed' in the NHS any more than we should 'ration' ELC.

Prospective study of short saphenous varicose vein surgery: six weeks' results

Vandenbroeck CP, Winterborn RJ, Hoult S, Campbell WB, Whitman B, Heather BP, Earnshaw JJ, on behalf of the Joint Vascular Research Group
Department of Vascular Surgery, Gloucestershire Royal Hospital, Gloucester

Objective

The aim of this study was to follow a cohort of patients undergoing surgery for short saphenous varicose veins.

Method

The present study recruited consecutive patients with short saphenous varicose veins undergoing surgery in nine different vascular centres in England from October 2002 till the present. Patients attended duplex scans and clinical evaluation pre-operatively and 6 weeks after the operation. Specific data regarding the operation and complications were filled in on a questionnaire by the surgeon.

Results

So far 195 legs of 180 patients have been evaluated. In 30% (58/195) of the legs, the short saphenous vein (SSV) was stripped. Postoperative complications were reported in 37 legs (34% in the stripping group versus 21% in the no stripping group). Numbness was mentioned in 48 legs (24% after stripping compared to 26% in the group without stripping). Eleven percent had visible or palpable residual varicose veins after stripping compared to 12% in the no stripping group. An incompetent residual distal SSV was reported on postoperative duplex scan in 72 legs (38% after stripping versus 38% when no stripping). In only two cases (4%) of the stripping group was the saphenopopliteal junction (SPJ) incompetent versus 16 cases (12%) when the SSV was not stripped and four of these cases clearly mention neovascularisation at this stage.

Conclusion

Preliminary results of this study show no evidence of an increased risk of nerve damage or postoperative complications in patients who had their short saphenous vein stripped. These results however do suggest that there might be an advantage in the stripping group with future follow-up.

One-year results of a double-blinded randomised trial on the treatment of bilateral recurrent long saphenous varicose veins

Hinchliffe R, Ubhi J, Beech A, Braithwaite BD
Queen's Medical Centre, Nottingham

Objective

A randomised double-blind study was designed to assess treatment of bilateral recurrent long saphenous varicose veins with either endoluminal thermal ablation (VNUS) or traditional redo groin surgery (RGS) and long saphenous vein (LSV) stripping.

Method

Sample size calculations required 16 patients. Their median age was 54 and 11 were women. The median CEAP class was 3. At operation, one leg, chosen at random, was treated with VNUS. The other leg was treated with traditional RGS. Patients completed visual analogue scales for pain and bruising. Digital image analysis objectively assessed bruising. Patients were reviewed with duplex examination at 6 weeks and at a median of 388 days after surgery.

Results

Time to perform VNUS was 26.5 min (18-32) compared with 39.5 min (24-45.5) for RGS ($p=0.04$). Pain score was lower for VNUS (1.38 [0-4.8] vs 4.5 [1.3-8], $p<0.001$). Bruise score was lower for VNUS (2 [0.5-4.8], vs 5.2 [2.8-8.5], $p<0.001$). At 6-week follow-up, 13 patients (81%) stated that they preferred the VNUS treatment. At 1 year, three patients were lost to follow-up. In the VNUS group eight of 13 LSVs remained occluded while five had segmental reflux. In the RGS group 12 legs had a completely stripped LSV, one was partially removed. There were recurrent veins in five VNUS legs and two in the RGS group.

Conclusion

Patients preferred VNUS, it caused less pain and bruising and was done more quickly than RGS. Recurrence may be more likely with VNUS.

Initial experience in the treatment of varicose veins due to saphenopopliteal (SP) and short saphenous (SSV) reflux with Endovenous Laser Treatment (EVLT)

Theivacumar N, Beale R, Mavor AID, Gough MJ
The General Infirmary at Leeds, Leeds

Objective

Conventional surgery for varicose veins due to SP/SSV reflux is associated with high recurrence rates (up to 50%), many resulting from inadequate surgery. This prospective audit examines the safety and efficacy of EVLT in the treatment of SP/SSV reflux.

Method

Twenty-seven patients (28 limbs) with varicosities due to SP/SSV reflux underwent out-patient EVLT (810nm diode laser, 12 watts power, 1 sec pulses). The SSV was ablated from mid-calf to the SP junction. Symptomatic improvement (Aberdeen Varicose Vein Scores [AVVS]), post-EVLT analgesic requirements, mobility and complications were all recorded.

Results

Duplex ultrasound follow-up (median 6 months) confirmed complete abolition of SP/SSV reflux in all limbs following a median total laser energy delivery of 1230J (936-1386) at an energy density of 5.4 pulses/cm (5.0-5.7). AVVS improved from 14.24 (IQR 10.26-21.2) to 6.01 (IQR 2.63-11.56) by 12 weeks ($p<0.001$). Median analgesia requirement was 2 days (26% patients required none) and the median time to normal activity 3 (1-14) days, 63% returning to normal daily activity immediately. There were no instances of skin burns, superficial phlebitis or DVT, but three patients (11%) developed transient cutaneous numbness (sural nerve). Ninety-six percent of patients would choose EVLT over surgery again.

Conclusion

EVLT abolished SP/SSV reflux in all limbs and this is likely to be more effective than conventional surgery. Further, there was a significant improvement in symptom scores and a rapid return to normal activity. This and the absence of procedure-related complications confirm that EVLT is a safe and effective alternative to surgery for the treatment of SP/SSV varicosities.

Knee-length graduated compression stockings are as effective as thigh-length in thromboprophylaxis. A meta-analysis

Sajid MS, Seifalian A, Hamilton G
Royal Free Hospital, London

Objective

Use of graduated compression stockings (GCS) has been proven highly effective for thromboprophylaxis in all types of hospitalised patients and in long haul flights. This meta-analysis reports a systematic review, the objective of which is to evaluate the effectiveness of knee-length GCS in thromboprophylaxis in hospitalised patients and in a low, moderate and high-risk population in long haul flights. Another aim is to prove that knee-length graduated compression stockings are very useful in reducing the development of post-thrombotic syndrome after DVT.

Method

Generic terms including stocking/s, sock/s or hosiery/hosieries were used to search a variety of electronic databases. Based on selection criteria, decisions regarding inclusion and exclusion of the primary studies were made. Using a meta-analysis software program, relative risk for incidence of DVT was calculated.

Results

A total of 14 randomised controlled trials (RCTs) were included. In the below-knee stockings group, 25 of 1469 (1.70%) participants developed DVT; in comparison 79 of 1501 (5.26%) in the thigh-length stockings group/control group developed DVT. The two-tailed p value for this difference in incidence of DVT in both groups is 0.0001, which is statistically extremely significant. The weighted relative risk for DVT was 0.10, with a fixed 95% CI 0.02-0.21.

Conclusion

This meta-analysis paves the pathway to confirm that knee-length graduated compression stockings are as effective as thigh-length in DVT prevention, both in hospitalised patients and in the population on long haul flights.

Pre-operative ischaemia of the long saphenous vein predisposes to intimal hyperplasia in bypass grafts through enhanced smooth muscle cell migration

Ruiz MC, Orr DJ, Teenan RP, Wadsworth RM

Peripheral Vascular Unit, Glasgow Royal Infirmary, Glasgow; Department of Physiology and Pharmacology, University of Strathclyde, Glasgow

Objective

A major limiting factor in the success of infrainguinal grafting is vein graft stenosis due to intimal hyperplasia (IH). We investigated the role of pre-operative ischaemia on the development of IH and the effect of simvastatin on this process.

Method

Long saphenous vein (LSV) was obtained from nine amputated legs (ischaemic LSV [ILSV]) and eight patients undergoing coronary bypass grafting (non-ischaemic LSV [NILSV]). Vein rings were maintained in organ culture for 14 days in culture medium alone or with simvastatin 5uM. Rings were processed for histology and the area of intima was measured. Smooth muscle cells (SMC) explanted from vein rings were used in cell proliferation and migration assays.

Results

Intimal area increased in vein rings from both groups but was significantly greater in ILSV compared with NILSV (mean 23.5% [SEM 3.4%] and 19.6% [SEM 2.3%], respectively, $p<0.05$). Simvastatin inhibited the development of IH in both groups, but was more effective in the ILSV. SMC proliferation was no different between the two groups and simvastatin inhibited SMC proliferation equally (mean 50% inhibitory concentration 1.16uM & 1.22uM) in ILSV and NILSV, respectively. SMC explanted from the ILSV showed an increased rate of migration compared with SMC from NILSV ($p<0.05$).

Conclusion

Ischaemia in the lower limb pre-programmes the LSV to be more susceptible to the development of IH when used as a bypass graft. This is possibly due to enhanced SMC migration. Simvastatin inhibits the growth of IH in organ culture and this effect is mediated through inhibition of SMC proliferation and migration.

Effect of low-dose (75mg) clopidogrel on platelet reactivity, ADP variability, and clopidogrel resistance when given before carotid surgery

Payne DA, Jones CI, Hayes PD, Bell PRF, Goodal AH, Naylor AR
The Department of Cardiovascular Sciences, University of Leicester, Leicester

Objective

A single 75mg tablet of clopidogrel taken before carotid endarterectomy (CEA) significantly reduces postoperative embolisation, a surrogate marker of stroke. Given the low dose of clopidogrel used and the variability in platelet response to both ADP and clopidogrel, this study examines the mechanisms underlying this important clinical finding.

Method

Fifty-six patients on routine aspirin therapy (150mg) were randomised to receive clopidogrel (75mg) or placebo the night before CEA. Blood samples were taken pre- and post-drug administration, and at the end of surgery for whole blood flow cytometry and Born aggregometry to assess platelet activation.

Results

Post-drug platelet fibrinogen binding in response to ADP was significantly lower in clopidogrel compared to placebo treated patients ($1x10^{-6}$ M ADP: 62.6 2.5% vs. 68.1 2.7%, p=0.006). This difference was further accentuated after surgery in those taking placebo due to increasing platelet ADP responsiveness ($1x10^{-6}$ M ADP: 62.5 3.1% vs. 76.5 2.2%, p=0.001). At the end of surgery, clopidogrel also reduced fibrinogen binding in response to thrombin (TRAP) and collagen ($p<0.05$) resulting in further amplification of the initial small inhibitory feedback stimulus. The level of pre-operative ADP-mediated platelet aggregation was also seen to correlate strongly with the reduction in platelet aggregation following clopidogrel (r=0.68, p=0.0003); a significant 'resistance' to clopidogrel was seen in patients with a low response to ADP prior to ingestion of clopidogrel. A negative correlation was also seen between the patients' pre-operative weight and the inhibitory effect of clopidogrel (r=0.57, p=0.002).

Conclusion

These results together explain how low-dose clopidogrel produces a significant clinical impact on emboli reduction without an associated increased bleeding risk.

Mesenteric traction during open abdominal aortic aneurysm repair may lead to intestinal ischaemia

Arya N, Lau LL, Lee B, Hannon RJ, Young IS, Soong CV
Vascular and Endovascular Unit, Belfast City Hospital, Belfast, Northern Ireland

Objective

Intestinal manipulation and mesenteric traction during open transperitoneal abdominal aortic aneurysm (AAA) repair may lead to intestinal hypo-perfusion which can cause development of the systemic inflammatory response syndrome. The aim of this study is to assess if mesenteric traction will result in intestinal ischaemia.

Method

Thirty-four patients undergoing AAA repair were randomised into three groups. Group I (n=11) had repair via the retroperitoneal approach while group II (n=12) and group III (n=11) were repaired via the transperitoneal approach with bowel packed within the peritoneal cavity or exteriorised in a bowel bag respectively. Tonometric measurement of gastric intramucosal pH (pHi) was performed to assess intestinal perfusion just prior to aortic clamping, during clamping, and at 0.5h, 1h, 2h, 4h, 6h and 12h after clamp release. Patients with persistent low pHi measurements (i.e. pHi <7.30 on at least 50% of the time points) were considered to have significant diminished gastric mucosal perfusion. Results are expressed as mean + SD.

Results

The pre-clamp pHi was similar between the three groups (gpI=7.36+0.04, gpII=7.33+0.06, gpIII=7.31+0.04). The number of patients with persistent low pHi measurements less than 7.30 was greater in gpIII (10/11) in comparison to gpI (5/11). The fall in gastric pHi was also significantly more sustained in gpIII compared to gpI ($p<0.01$). The operative time, aortic clamp time, amounts of blood lost and transfused were similar in all three groups.

Conclusion

These results suggest that the retroperitoneal approach for AAA can minimise intestinal ischaemia by avoiding mesenteric traction that is associated with the transperitoneal approach.

Adenoviral delivery of the urokinase gene promotes venous thrombus resolution

Gossage JA, Humphries J, Modarai B, Burnand KG, Smith A
Academic Department of Surgery, St Thomas' Hospital, King's College, London

Objective

Catheter-directed thrombolytic therapy for deep vein thrombosis is associated with haemorrhagic complications. This study aimed to use adenoviral gene transfer as a novel method of therapeutic delivery for thrombolytic agents, urokinase (uPA) and tissue plasminogen activator (tPA).

Method

Production of functional tPA and uPA was confirmed by a plasmin activity assay after transfecting human embryonic kidney cells with either the adenovirus carrying the gene cassette for urokinase (ad.uPA) or for tissue plasminogen activator (ad.tPA). Thrombus was formed in the inferior vena cava of 70 wild-type mice before being directly injected with 10(x8) plaque-forming units of adenovirus at 48 hours. The thrombus was then weighed at 7 days after treatment with either ad.uPA, ad.tPA or control virus. The transfection efficiency into the thrombus was confirmed using a reporter gene, ad.GFP. Monocyte content, MMP and VEGF levels were measured to establish a possible mechanism of enhanced thrombus resolution.

Results

Urokinase (ad.uPA) reduced thrombus weight by two-fold when compared with control virus (15.1mg ± 1.1 vs 7.4mg ± 1.3, p=0.004). Urokinase activity was detected in all treated thrombi, but there was no difference in MMP 9, MMP 2 and VEGF levels between the two groups. Urokinase overexpression did not affect monocyte recruitment. Tissue plasminogen activator (ad.tPA) did not influence thrombus size.

Conclusion

Increasing urokinase activity within the thrombus significantly enhances thrombus resolution. Therapeutic delivery of ad.uPA in man may provide a novel and safe treatment for deep venous thrombosis.

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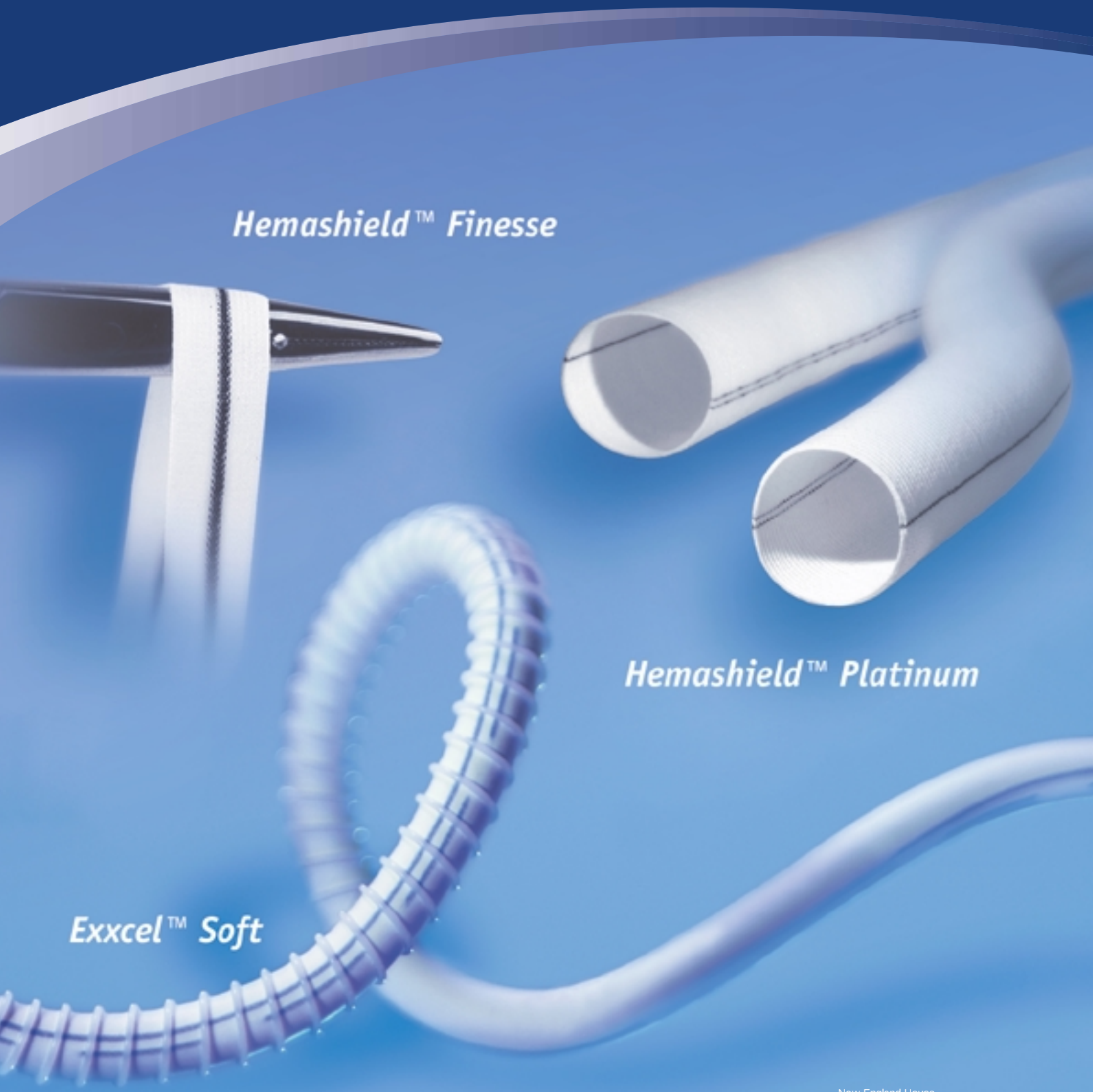

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Increased hypoxia inducible factor-1alpha and localisation of erythropoietin in human critical limb ischaemia

Ho TK [1], Rajkumar V [2], Ponticos M [2], Garcia P [2], Khan K [2], Hart C [1], DiSalvo C [3], Walesby RK [3], Abraham DJ [2], Black C [2], Baker DM [1]

[1] Vascular Unit, Department of Surgery and [2] Department of Rheumatology, The Royal Free Hospital, London; [3] The Heart Hospital, London

Objective

Physiological responses to ischaemia include angiogenesis and metabolic adaptation of end organ. Hypoxia inducible factor (HIF)-1alpha, a major transcription factor, promotes ischaemia-driven angiogenesis. Recently, increasing evidence demonstrates that erythropoietin (Epo) has a protective effect in ischaemic organs. We aim to investigate the extent of angiogenic response, expression of HIF-1alpha and localise Epo to skeletal muscle in critical limb ischaemia (CLI).

Method

Skeletal muscle biopsies were obtained from patients with CLI (n=12) undergoing major lower limb amputations and patients without limb ischaemia undergoing saphenous vein harvesting for coronary artery bypass graft as controls (n=12), with ethical committee approval. Microvessel density (MVD), capillary to muscle fibre (C:M) ratio, HIF-1alpha, Epo and muscle fibre type expressions were determined by immunohistochemistry. Western blotting and ELISA were used to quantify HIF-1alpha levels. Colocalisation between cell-specific antigens was investigated by double immunofluorescence labelling using confocal microscopy. Statistical analyses were performed using the Mann-Whitney U test.

Results

The CLI group had a significantly higher MVD and C:M ratio (three-fold and 1.7-fold higher than the control, $p<0.001$). HIF-1alpha expression was significantly higher by two-fold in CLI muscles ($p<0.001$) and was colocalised to endothelial cells. Epo was colocalised to muscle fibre type IIa and neonatal myosin heavy chain (MHC).

Conclusion

Our findings suggest that a physiological angiogenic response occurs in CLI, with increased HIF-1alpha expression and colocalisation to endothelial cells. Furthermore, this was the first study to localise Epo to muscle fibre type IIa and neonatal MHC in critically ischaemic human skeletal muscle. These suggest a potential physiological novel role of Epo in skeletal muscle metabolic adaptation to ischaemia and regeneration.

In vivo attenuation of myointimal hyperplasia using transforming growth factor beta 3: an interposition graft model

Murphy MO, Ghosh J, Khwaja N, Halka AT, Turner N, Ferguson MW, Kielty CM, Walker MG
Department of Vascular Surgery, Manchester Royal Infirmary, Manchester

Objective

The Transforming Growth Factor (TGF) beta family of cytokines exert pleiotropic actions on smooth muscle cell (SMC) phenotype, proliferation and extracellular matrix synthesis. This *in vivo* study assesses the use of TGFbeta 3 in attenuating the development of para-anastomotic myointimal hyperplasia in an animal model of small diameter vascular graft failure.

Method

Under general anaesthesia ten adult goats underwent bilateral polyurethane interposition graft insertion in the carotid position. Following completion of the anastamosis each artery received adventitial infiltration of 50ng of TGFbeta 3 around the anastamosis; the other side a placebo. Postoperatively, each animal received 150mg aspirin daily. The arteries were explanted, half at 6 weeks and the remaining five at 3 months for histological examination.

Results

Vessel wall thickness surrounding the anastamosis was reduced by 37% in TGFbeta 3-treated arteries compared to placebo at 6 weeks and 3 months, principally due to reduced SMC proliferation. Total collagen content was not significantly different between TGFbeta 3 and placebo sides. Further analysis for the subendothelial matrix component collagen type VIII showed decreased levels on the treated side. Total elastin content was reduced on the TGFbeta 3-treated side ($p=0.004$).

Conclusion

Direct, single-dose sub-adventitial infiltration of TGFbeta 3 following insertion of an interposition graft reduces SMC proliferation and elastin content. It would appear that TGFbeta 3 holds promise as a prophylaxis against the development of myointimal hyperplasia, the predominant cause of graft failure in peripheral bypass surgery.

Biomechanical fatigue in aneurysmal abdominal aorta: a physical model of rupture

Windhaber RAJ, Tarlton JF, Gohel MS, Poskitt KR, Earnshaw JJ, Mitchell DC
North Bristol NHS Trust, Bristol

Objective

Rupture in abdominal aortic aneurysms (AAA) is the result of mechanical failure. Understanding aortic biomechanics is important when interpreting the connective-tissue changes found in AAA. We investigated aortic wall biomechanics in aneurysmal disease.

Method

Site-matched aortic tissue was collected from 24 cadaveric renal donors without aortic disease and 30 patients undergoing elective open AAA repair. Transverse and longitudinal breaking strength, plastic strain and fatigue, at tensions equating to physiological blood pressure, were determined in adjacent 2mm aortic strips using a validated Instron-6022 mechanical test frame (100N load cell, jaw separation 6mm). Analysis was performed using student's unpaired t-test.

Results

Longitudinal breaking tension was increased in AAA vs. control (2.57 ± 0.29Nmm^{-1}, n=30; vs. 1.27 ± 0.12Nmm^{-1}, n=24; p<0.001); transverse breaking tension remained unchanged (1.92 ± 0.20Nmm^{-1}, n=30; vs. 2.10 ± 0.17Nmm^{-1}, n=24; p=0.489). The AAA wall was significantly stiffer (increased tissue modulus) than the control, in transverse (8.27 ± 1.28Nmm^{-2}, n=30; vs. 4.13 ± 0.42Nmm^{-2}, n=24; p=0.007) and longitudinal orientation (7.70 ± 1.47Nmm^{-2}, n=30; vs. 2.22 ± 0.35Nmm^{-2}, n=24, p=0.002). Plastic strain curves derived from the fatigue protocol, showed significant increase in plastic strain generated in AAA samples vs. control (p<0.01, n=12 both). Transverse breaking strength following fatigue testing was reduced in AAA compared with the control (1.575 ± 0.10Nmm^{-1}, n=12; vs. 2.27 ± 0.23Nmm^{-1}, n=12, p=0.03).

Conclusion

AAA exhibit increased longitudinal static breaking tension. This is a physiological adaptation to increased wall tension. Transverse static breaking tensions are similar in AAA and the control, but the aneurysm wall has a reduced capacity to withstand mechanical fatigue in a transverse orientation. This model demonstrates a mechanism of wall failure leading to rupture.

Comparison of the fixation strength of fenestrated and non-fenestrated stent-grafts for endovascular abdominal aortic aneurysm repair (EVAR)

Zhou SS, Brennan J, How TV, Gilling-Smith GL, Harris PL
Regional Vascular Unit, Royal Liverpool University Hospital, Liverpool

Objective

EVAR with a fenestrated stent-graft requires precise positioning reinforced by stenting one or more targeted vessels. The aim of this study was to determine whether a fenestrated stent-graft (FSG) provides better stability than a standard non-fenestrated stent-graft (SSG).

Method

Truncated FSG with single fenestration were deployed in bovine aortic segments with a side-branch. Balloon-expandable stents were then delivered into the branches. Similarly, SSG of the same dimensions were deployed for comparison. The aorta was pressurised and oversized by the stent-graft by 5%, 10% and 20%. The force required to cause distal migration (DF) was recorded by a digital force gauge attached to the stent-graft.

Results

Displacement of the stent-grafts occurred in two distinct phases: phase 1 was an initial "give" due to embedding of the hooks into the aortic wall; phase two was the migration with the device being completely dislodged. The DF that initiated each phase was dependent upon the degree of oversizing of the stent-graft relative to the aortic diameter. For 5%, 10% and 20% oversizing, the phase 1 DFs were 2.84 ± 0.31; 3.7 ± 0.21; 5.54 ± 0.44 newtons respectively for non-fenestrated grafts and 10.48 ± 1.23, 11.45 ± 1.48; 12.12 ± 1.42 newtons for fenestrated grafts. The phase 2 DFs were 8.1 ± 0.92, 12.03 ± 1.06, 17.33 ± 0.83 newtons for non-fenestrated and 22.56 ± 1.6, 28.24 ± 1.56, 33.01 ± 1.75 newtons for fenestrated stent-grafts. The differences in DF between fenestrated and non-fenestrated stent-grafts were highly significant for both phases ($p<0.001$ CI 95%).

Conclusion

Stent-graft migration occurs in two stages. The initial movement, which results from embedding of hooks into the aneurysm wall, could compromise the seal in short necks. FSG offers more secure fixation than SSG.

Aortic necks of ruptured abdominal aneurysms dilate more than asymptomatic aneurysms following endovascular repair

Badger SA, O'Donnell ME, Makar R, Boyd CS, Johnston LC, Loan W, Hannon RJ, Lau LL, Lee B, Soong CV

Vascular and Endovascular Unit, Belfast City Hospital, Belfast, Northern Ireland

Objective

Endovascular repair (EVR) of abdominal aortic aneurysm is increasingly used for both elective and emergency patients. We evaluated if a difference exists in the baseline and the rate of change of the aortic neck diameter between non-ruptured and ruptured AAA following EVR.

Method

Details of patients undergoing elective (group 1) and emergency (group 2) EVR between October 1998 and December 2004 were retrieved from the Endovascular Database of the Belfast City Hospital. Top neck diameters were recorded on the database prospectively from computerised tomographic scans. Measurements were taken pre-operatively and at 1, 3, 12, 24 months postoperatively. The rate of change of the aortic neck diameter (mm/month) was calculated for each group.

Results

One hundred and forty-six elective and 36 emergency patients had EVR. One hundred and twenty-one (92 male) elective and 26 (24 male) emergency patients were included in this analysis. Mean age was 75 years (± 6.5) in group 1, and 74 years (± 7.0) in group 2. Mean proximal aortic neck was larger pre-operatively in group 2 (25.0 ± 3.2mm) in comparison to group 1 (23.3 ± 2.9cm; p=0.011). The growth rate of the top neck diameter was significantly greater at 12 months (p=0.025) and 24 months (p=0.0044) in group 2 compared to group 1.

Conclusion

Aneurysm necks in patients with ruptured AAA are larger and dilate at a greater rate than those whose aneurysms are not ruptured. This increased expansion rate must be taken into consideration when oversizing the graft in emergency patients to allow adequate long-term exclusion of the aneurysm.

Greater “oversizing" of aortic endografts is required for shorter aneurysm necks in endovascular aortic aneurysm repair (EVAR)

Zhou SS, Brennan J, How TV, Gilling-Smith GL, Harris PL
Regional Vascular Unit, Royal Liverpool University Hospital, Liverpool

Objective

Successful EVAR requires an effective proximal seal to exclude the aneurysm from circulation. The aim of this study was to examine the effectiveness of the proximal seal in relation to aneurysm neck length and oversizing.

Method

An *in vitro* model of the proximal seal of an infrarenal aortic endograft was constructed with pulsatile flow. Aortic sections of bovine aorta with compliance matching that of the abdominal aorta of humans aged 60-70 years were selected. A truncated body of Zenith endograft was deployed into this model. The length of "neck" into which the graft was deployed was 10, 15, 20, 30 and 40mm. For each of the neck lengths, "oversizing" of a device was varied by increasing, decreasing pressure within the flow circuit in order to vary the distension of the "neck". Any leak of fluid from the seal zone was referred to as an "endoleak".

Results

A change of mean pressure from 60mmHg to 160mmHg results in an increase in the diameter of the "neck" from 26mm to 32mm. The minimal percentage of "oversizing" above which no "endoleak" decreased with increasing length of the "neck" is as follows: 10mm-20%, 15mm-16%, 20mm-10%, 30mm-7%, 40mm-4%.

Conclusion

In a healthy bovine aorta, the luminal diameter, the degree of oversizing of an endograft and security of seal are pressure-dependent. There is a reverse relationship between the length of the "neck" and the minimal degree of oversizing to preventing endoleak. Greater "oversizing" may be required for shorter aneurysm necks in EVAR.

Does acetylcysteine prevent contrast-induced nephropathy during endovascular AAA repair? A randomised controlled study

Moore NM, Lapsley M, Norden AG, Firth JD, Gaunt ME, Varty K, Boyle JR

Departments of Vascular Surgery, Clinical Biochemistry, and Renal Medicine, Cambridge University Hospitals NHS Trust, Cambridge; Clinical Biochemistry, Epsom and St Helier NHS Trust, Surrey

Objective

N-acetylcysteine (NAC) reduces the incidence of contrast nephropathy during coronary angiography. We hypothesised that NAC would reduce the incidence of contrast nephropathy during EVAR. The aim of the study was to investigate the effect of NAC on renal markers during EVAR.

Method

Twenty consecutive patients undergoing EVAR between February 2004 and January 2005 were randomised to receive standard intravenous fluid hydration or standard fluid hydration and NAC (600mg BD orally, four doses). Venous blood and urine were collected prior to the procedure and for 5 postoperative days and analysed blindly for serum urea and creatinine levels and the urinary retinol binding protein (RBP) and albumin creatinine ratio (ACR), sensitive markers of renal injury.

Results

There were no significant differences in baseline demographics, contrast volumes used and intravenous fluid administered between the groups. No patient developed acute renal failure; however, three patients in the treatment group had serum creatinine rises consistent with contrast-induced nephropathy (>25% above baseline). In both groups urinary RBP rose significantly from baseline $p<0.003$ (control, median 190µg/l to peak 6587µg/l: treatment 127µg/l to 7918µg/l). There were similar significant rises in ACR $p<0.02$ (control, median 1.9mg/Mmol to peak 5.0mg/Mmol; treatment 1.04mg/Mmol to 5.3mg/Mmol). There was however no significant difference in the postoperative RBP or ACR between the two groups at any time point.

Conclusion

EVAR causes significant acute renal injury in most patients. This was not attenuated by N-acetylcysteine in this study. The causes of renal injury are probably multifactorial, the long-term clinical significance of which are unclear.

Impact of renal dysfunction on operative mortality following endovascular abdominal aortic aneurysm surgery

Statius van Eps RG, Leurs LJ, Buth J, Harris PL for the EUROSTAR Collaborators
Royal Liverpool University Hospital, Liverpool

Objective

Pre-operative renal dysfunction is a significant risk factor for mortality following open surgery for AAA. This study examines the impact of renal dysfunction on postoperative mortality following EVAR.

Method

Five thousand one hundred and sixty-seven patients who had EVAR were divided into two groups according to their pre-operative renal status: group A (4198), serum creatinine <1.5mg/dl, creatinine clearance greater than 50ml/ml and group B (969) serum creatinine >1.5mg/dl, clearance <50ml/ml or on dialysis. Patient characteristics and postoperative complications in these groups were compared. Multivariate Cox models were used to determine whether baseline variables were independently associated with the adverse event.

Results

Patients in group B were significantly older than patients in group A (73.6 vs 71.7 years, p<0.0001) and less fit generally (ASA-classification >3, 67.7% vs 45.3%; p<0.0001). Renal dysfunction was associated with increased postoperative systemic complications (17.1% vs 10.5%, p<0.0001). Thirty-day mortality in group B was significantly higher than in group A (6.2% vs 2.0%; p<0.0001). A significant increase in mortality (5.5%) was also seen in patients with less severe renal dysfunction (creatinine 1.5-3mg/dl). Renal dysfunction was an independent risk factor for 30-day mortality (OR 2.3, CI 1.6-3.3, p<0.0001). Other independent risk factors were age at operation, pulmonary impairment and ASA >3.

Conclusion

Renal dysfunction is a significant and independent risk factor for operative mortality after EVAR. The mechanism of this effect is not entirely clear and needs further investigation to inform risk stratification and protective measures.

Expression of growth factors and growth factor receptor in non-healing and healing ischaemic ulceration

Murphy MO, Ghosh J, Khwaja N, Halka AT, Carter A, Turner N, Fulford P, Walker MG
Department of Vascular Surgery, Manchester Royal Infirmary, Manchester

Objective

To characterise the histological and cytokinetic characteristics of purely ischaemic ulcers and the processes that underpin healing following successful revascularisation.

Method

Biopsies were taken immediately pre- and 6 weeks post-revascularisation, incorporating surrounding skin, ulcer edge and base. They were evaluated for morphological changes by H&E and for Growth Factor (GF) and Growth Factor Receptor (GFR) expression using immunohistochemistry. Localisation and quantification of Platelet Derived Growth Factor Receptor (PDGFR), Epidermal Growth Factor Receptor (EGFR), TGFßreceptor3 (TGFßR3), Transforming Growth Factor Beta 1 and 3 (TGFß1 and TGFß3) and von Willebrand Factor (vWF) were examined systematically by three independent investigators who were blinded to the timing of biopsy.

Results

Pre-operatively there was small vessel vasculitis, necrosis and infection with a profuse neutrophil and macrophage infiltrate in all samples. Postoperative biopsies revealed a proliferation of new, small blood vessels in the surrounding skin, particularly in and around the ulcer edges and base. Overall, there was less infection and inflammation with minimal vasculitis. Accelerated epithelial proliferation was observed with detachment from the underlying dermis. These findings correlated with increased staining for PDGF receptor localised to fibroblasts and prominent staining for TGFß3, PDGF receptor and TGFßreceptor3 localised to areas of neovascularisation. vWF staining confirmed an endothelial lining within these new vessels.

Conclusion

Healing of purely ischaemic ulcers is by angiogenesis and this process is associated with increased activity of the pro-angiogenic cytokines PDGF and TGFß3. These findings show promise for the use of growth factor manipulation to aid healing in ischaemic ulcers.

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The effects of major vascular surgery on platelet function

Rajagopalan S, Ford I, Bachoo P, Greaves M, Brittenden J

Departments of Vascular Surgery and Medicine and Therapeutics, University of Aberdeen, Aberdeen

Objective

Abnormal platelet function has been implicated in the development and progression of atherosclerosis, as well as in the pathogenesis of acute ischaemic events. Little is known about the effect of vascular surgery on platelet function. We aimed to determine the effects of major vascular surgery on platelet aggregation and activation.

Method

Blood samples from 70 patients undergoing aortic aneurysm repair or lower limb revascularisation were taken: pre-operatively, immediately postoperatively and days 1, 2, 3 and 5. Platelet aggregation through COX-mediated and Thrombin Receptor Activator Peptide (TRAP)-stimulated GPIIb/IIIa pathways was measured by the Ultegra point of care system. Resting and ADP-stimulated platelet expression of P-selectin and fibrinogen were determined by whole blood-flow cytometry.

Results

TRAP-stimulated platelet aggregation increased in the immediate postoperative period and on day 1 ($p<0.001$, median increase of 18% [range -85 to +178]). COX-mediated aggregation significantly increased on day 1 ($p<0.001$, median increase of 7% [range-191 to +278]) and day 2. *Ex vivo* ADP-stimulated fibrinogen binding increased on postoperative day 1 ($p=0.03$, median rise of 12% [range -45.7 to +48.6]). P-selectin expression significantly increased on days 1, ($p=0.02$, median rise of 19% [range -3.10 to +5.49]) 2 and 3 as did *ex vivo* ADP-stimulated samples ($p<0.01$).

Conclusion

This study is the first to show that platelet aggregation and markers of platelet activation are increased in patients undergoing vascular surgery despite aspirin and statin therapy. Further work is required to determine if platelet function correlates with the occurrence of acute cardiac events observed in the peri-operative period in these patients.

The effects of acute exercise on haemostasis, inflammation and renal function in patients with intermittent claudication on statin and aspirin therapy

Collins P, Ford I, Croal B, Ball D, Greaves M, Macaulay E, Brittenden J

Departments of Vascular Surgery and Medicine and Therapeutics, University of Aberdeen, Aberdeen

Objective

Exercise in patients with intermittent claudication (IC) may induce a systemic thrombo-inflammatory response. The effect of secondary prevention therapy on this response is unknown. The aim was to investigate the effects of acute exercise on markers of coagulation activation, inflammation and renal function in patients with IC, receiving aspirin and statin therapy compared to healthy controls.

Method

Samples were taken before, immediately and 1 hour after exercising on a treadmill in 20 patients with IC and 20 healthy volunteers. Interleukin-6 (IL-6), thrombin-anti-thrombin complex (TAT) and fibrin D-dimer were measured by ELISA. High sensitivity CRP (HsCRP) and urinary albumin were measured via a nephelometric technique, urinary protein via a turbidometric assay and N-acetyl-ß-D-glucosaminidase (NAG) via a colorimetric assay.

Results

Elevated baseline levels of Hs-CRP, IL-6, white cell counts, D-dimer and urinary NAG occurred in patients with IC compared to volunteers ($p>0.05$). HsCRP or IL-6 did not increase following exercise. Both TAT and D-dimer levels significantly increased following exercise in both groups. A transient rise in the protein creatinine ratio also occurred in both groups ($p<0.05$). The albumin creatinine ratio increased following exercise in the patient group but there was no change in urinary NAG.

Conclusion

This is the first study to show that urinary NAG levels are elevated in patients with IC compared to healthy controls. Elevated markers of inflammation occurred in patients with IC despite statin and aspirin therapy. However, there is no evidence that acute exercise induces a pro-thrombotic state or renal tubular damage.

Abrogation of skeletal muscle reperfusion injury by simvastatin: the impact of nitric oxide synthase inhibition

Khanna A, Laws PE, Cowled PA, Fitridge RA

Department of Surgery, The University of Adelaide, The Queen Elizabeth Hospital, Adelaide, South Australia, Australia

Objective

Skeletal muscle ischaemia reperfusion injury (IRI) causes local and systemic injury. Statins attenuate cerebral, cardiac and renal IRI but their effects in skeletal muscle IRI are less well characterised. This study investigated whether simvastatin ameliorated skeletal muscle IRI and whether this occurred via nitric oxide (NO)-dependent mechanisms.

Method

Rats were administered simvastatin for 5 days before induction of 4h bilateral hindlimb ischaemia followed by 24h reperfusion. The nitric oxide synthase (NOS) inhibitor, L-Nio, was administered 20 minutes prior to induction of ischaemia. Skeletal muscle was examined for neutrophil infiltration using myeloperoxidase (MPO) assays. Tissue damage was assessed by collagen IV immunohistochemistry.

Results

IRI resulted in neutrophil infiltration in skeletal muscle, which was reduced by administration of either simvastatin or L-Nio. When administered together, simvastatin and L-Nio demonstrated a synergistic effect to prevent completely the IRI-mediated rise in neutrophil infiltration. Collagen IV immunohistochemistry demonstrated that simvastatin protected against IRI-mediated collagen breakdown, as did L-Nio. However, L-Nio plus simvastatin counteracted the protective effects of each agent on collagen breakdown.

Conclusion

Simvastatin protects against neutrophil infiltration and collagen degradation during IRI. Inhibition of NO reduces these events, suggesting that NO is cytotoxic in IRI. When administered together, these agents display paradoxical effects in reducing neutrophil infiltration without inhibiting collagen breakdown, suggesting collagen degradation is independent of proteases released from infiltrating neutrophils. This study indicates that simvastatin administration in patients undergoing elective or emergency surgery incorporating an IR injury may be of benefit in reducing the severity of skeletal muscle reperfusion injury.

The anti-thrombogenic potential of a new nanocomposite polymer for the development of bypass grafts

Seifalian AM, Kannan R, Salacinski HJ, De Groot J, Bozec L, Horton M, Hamilton G
University College London, London

Objective

We have developed a nanocomposite using a silica nanocomposite polyhedral oligomeric silsesquioxane (POSS) and poly(carbonate-urea) urethane (PCU) for potential use in vascular bypass grafts. In this study, we sought to compare its anti-thrombogenicity to conventional polymers used in vascular bypass grafts so as to improve upon current patency rates.

Method

Using atomic force microscopy (AFM) and transmission electron microscopy (TEM), surface topography and composition were studied respectively. The ability of the nanocomposite surface to repel both proteins and platelets *in vitro* was assessed using thromboelastography (TEG), fibrinogen ELISA assays, anti-factor Xa assays, scanning electron microscopy (SEM) and platelet adsorption tests.

Results

TEG analysis showed a significant decrease in clot strength (one-way ANOVA, $p<0.001$) and increase in clot lysis (one-way ANOVA, $p<0.0001$) on the nanocomposite when compared to both polytetrafluoroethylene (PTFE) and poly (carbonate-urea) urethane (PCU). ELISA assays indicate lower adsorption of fibrinogen to the nanocomposite compared to PTFE (one-way ANOVA, $p<0.01$). Interestingly, increasing the concentration of POSS nanocages within these polymers was shown to proportionately inhibit factor X activity. Platelet adsorption at 120 minutes was also lower compared to PTFE and PCU (two-way ANOVA, $p<0.05$). SEM images showed a 'speckled' morphologic pattern with Cooper Grades I and II platelet adsorption morphology compared to PTFE with Grade IV morphology.

Conclusion

Based on these results, we concluded that POSS-nanocomposites possess greater thromboresistance than PTFE and PCU making it an ideal material for the construction of bypass grafts.

Variability in responsiveness to clopidogrel in patients with intermittent claudication

Cassar K, Bachoo P, Ford I, Greaves M, Brittenden J

Departments of Vascular Surgery and Medicine and Therapeutics, University of Aberdeen, Aberdeen; Vascular Unit, Aberdeen Royal Infirmary, Aberdeen

Objective

The concept of clopidogrel resistance is frequently evoked in the cardiac literature. The variability of antiplatelet response in patients with intermittent claudication has not been investigated. The aim of this study was to describe the effect of the addition of clopidogrel to aspirin using *ex vivo* measures of platelet activation in patients with life-style limiting intermittent claudication.

Method

Data from 67 patients with intermittent claudication taking part in a randomised controlled trial and who received clopidogrel in addition to aspirin were analysed. Platelet activation was measured using whole blood-flow cytometric measurement of ADP-stimulated P-selectin expression at baseline and 12 hours after administration of a loading dose of 300mg clopidogrel. Patients continued to receive 75mg clopidogrel daily for 30 days. Compliance with treatment was assessed by counting returned tablets.

Results

Six patients were excluded from analysis because of incomplete compliance with treatment. Six out of 61 patients (9.8%) showed no reduction in platelet activation 12 hours after administration of the loading dose of clopidogrel. At 30 days these six patients still showed no response to clopidogrel. Amongst the remaining 55 patients, the mean reduction in P-selectin expression after clopidogrel administration was 51.5% (95% CI: 43.8-59.2). Amongst responders there was a wide variability in reduction of P-selectin expression in response to clopidogrel (range 8.11-97.7%). Four of these patients (7.3%) showed a reduction of more than 95% in P-selectin expression.

Conclusion

Patients with intermittent claudication show a wide variability in their response to clopidogrel. While a small proportion of these patients shows no response at all, another small group appears to respond excessively to clopidogrel. Clinical studies are required to identify whether hyper-responders are at increased risk of bleeding complications and whether hypo-responders are at a higher risk of thrombotic events.

Transthoracic echocardiogram in the management of acute limb ischaemia

Lewis A, Kirk G, Kothurkar A, McKinley A, Blair PH, Hood JM, Harkin DW
Regional Vascular Surgery Unit, Royal Victoria Hospital Belfast, Belfast

Objective

Acute limb ischaemia (ALI) is commonly a result of embolism from a cardiac source. Transthoracic echocardiography is often used to look for a residual cardiac embolic source. It is our impression that this investigation seldom influenced eventual treatment, and often led to delayed discharge. We therefore assessed the influence of echocardiography on treatment in 115 consecutive patients presenting with confirmed embolic ALI.

Method

We retrospectively analysed the case records of all patients requiring surgical embolectomy for ALI over a 4-year period (2000-2004). Information was retrieved from a prospective national vascular registry, theatre logbooks, and chart review. Patient details were cross-referenced with records from our inpatient echocardiography service over the same period. Data were analysed by Chi-squared test.

Results

We found 115 consecutive patients presenting to a single centre with ALI over the study period, with confirmed emboli at operation. With femoral embolectomy 49/79 patients (62%) had transthoracic echocardiography as an inpatient; 47/49 (96%) showed no embolic source. With brachial embolectomy patients 21/36 (58%) had echocardiography; 21/21 (100%) showed no embolic source. There was no significant difference with or without inpatient echocardiogram on rates of amputation, postoperative complications or death. Patients who had an inpatient echocardiogram had a significantly longer inpatient hospital stay ($p<0.05$).

Conclusion

Inpatient echocardiography delays discharge and does not influence patient management in embolic ALI. We suggest that routine echocardiography represents a poor use of resources and does not influence management in patients with ALI.

The effect of supervised exercise and cilostazol on coagulation and fibrinolysis in patients with intermittent claudication

Hobbs SD, Fegan C, Adam DJ, Bradbury AW
University Department of Vascular Surgery, Birmingham Heartlands Hospital, Birmingham

Objective

Intermittent claudication (IC) is associated with a prothrombotic, hypofibrinolytic state. Although supervised exercise (SE) and cilostazol provide symptomatic benefit in IC, neither have been shown to reduce the excessively high morbidity and mortality experienced by claudicants due to thrombotic events. This study assesses for the first time the effect of SE and cilostazol on the procoagulant diathesis in IC.

Method

Thirty-four patients (27 men and 7 women of median age 67, range 63-72 years) were randomised to receive SE (n=9), cilostazol (n=9), SE and cilostazol (n=7) or best medical therapy (BMT) alone (n=9) in a 2x2 factorial design. Patients were assessed at baseline, 3 and 6 months. Thrombin anti-thrombin complex (TAT) was measured as a marker of thrombin generation and plasminogen activator inhibitor (PAI) antigen as a marker of fibrinolysis.

Results

At 6 months, compared to the BMT-only group, SE and cilostazol both resulted in significant improvements in maximum walking distance (MWD) (40% and 64% respectively) and small increases in ABPI (18% and 13% respectively). The benefits of SE and cilostazol combined were additive. However, neither SE (mean [s.d.] TAT 2.01 [2.31] to 2.18 [2.84], p=0.929, PAI 22.2 [19.7] to 19.6 [18.1], p=0.533) nor cilostazol (mean [s.d.] TAT 1.39 [1.53] to 1.28 [1.34], p=0.65, PAI 22.6 [19.9] to 20.6 [16.9], p=0.55) had any effect on thrombin generation or fibrinolysis.

Conclusion

In contrast to balloon angioplasty, SE and cilostazol do not ameliorate the prothrombotic hypofibrinolytic diathesis observed in IC; as a result, patients are not protected from thrombotic morbidity and mortality over and above that afforded by BMT.

Carotid endarterectomy under local anaesthetic - evaluating a high fidelity simulated environment for training and assessment

Horrocks EJ [1,2], Black SA [1,2], Pandey VA [1,2], Harrison RH [1,2], Wetzel CM [2], Nestel D [2], Kneebone R [2], Wolfe JHN [1]

[1] Regional Vascular Unit, St Mary's Hospital, London; [2] Division of Surgery, Oncology, Reproductive Biology and Anaesthesia, Imperial College, London

Objective

To evaluate high fidelity simulation of carotid endarterectomy (CEA) under local anaesthesia as a tool for assessment of surgical competence.

Method

Each CEA was performed in our high fidelity simulated operating theatre. Three simulated patients (trained actors) linked to inanimate CEA models played the role of conscious patients during surgery. Sixteen vascular surgeons (group 1: SHOs n=8; 2: SPRs n=4; 3: consultants n=4) each performed a non-crisis scenario (NCS) followed by a crisis scenario (CS). Events within the CS included intra-operative bradycardia, stroke (requiring shunt insertion) and shunt dislodgement. All performances were assessed using general and procedure-specific rating scales for both technical and non-technical skills by four independent raters.

Results

A significant difference in technical skill with ascending grade was seen for both general (NCS: p=0.03; CS: p=0.03 Kruskall-Wallis) and procedure-specific scales (NCS: p=0.02; CS: p=0.03). Subset analysis showed a significant difference between groups 1 and 2 (p=0.008 Mann-Whitney U test) and groups 2 and 3 (p=0.03) for both scenarios. Inter-rater reliability was high (a=0.9). A significant difference in non-technical skill with ascending grade was seen for both scenarios (p=0.01 Kruskall-Wallis). Subset analysis showed a significant difference between groups 1 and 2 ($p<0.05$ Mann-Whitney U test) for the crisis scenario only. There was a significant difference between groups 2 and 3 for both scenarios (NCS: p=0.03; CS: p=0.02). Inter-rater reliability was high (a=0.85).

Conclusion

Significant differences between junior and senior trainees, and senior trainees and consultants, were shown for both technical and non-technical skills. Early results suggest that high fidelity simulation offers competency-based assessment of all grades and may provide a useful training environment for junior trainees as well as more experienced surgeons.

How cost-effective is screening for abdominal aortic aneurysms? A long-term perspective based on the MASS trial

Kim LG, Thompson SG, Briggs AH, Buxton MJ, Campbell HE
MRC Biostatistics Unit, Cambridge

Objective

Screening for abdominal aortic aneurysms (AAA) has been investigated in a number of randomised trials that have consistently reported an AAA-related mortality benefit in the group invited to screening. Reliable estimates of long-term cost-effectiveness are now needed to inform policy decisions for AAA screening programmes.

Method

A health economic decision model for screening is described in brief and extrapolated to 30 years. The strategy modelled involves a one-off scan at age 65, with annual and 3-monthly follow-up scans for small and medium aneurysms respectively. Referral for elective surgery occurs at an aortic diameter of 5.5cm. Model parameters are estimated from patient-level data from the UK Multi-centre Aneurysm Screening Study. At 4 years, the model structure results in similar outcomes and events as observed in MASS. Uncertainty in model inputs is addressed by probabilistic sensitivity analysis.

Results

The model confirms that cost-effectiveness improves dramatically when considered over longer timescales. Taking a 30-year perspective, screening for abdominal aortic aneurysms in men is highly cost-effective at £510 per life-year gained (95% uncertainty interval: £330 to £909). Adjusting life-years for the reduced health-related quality of life experienced in this population gave a figure of £676 (95% uncertainty interval: £437 to £1,203) per quality-adjusted life-year gained.

Conclusion

The long-term cost-effectiveness of screening for abdominal aortic aneurysms in men is highly attractive and this evidence provides further support for a national screening programme in the UK.

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Abdominal Aortic Aneurysm (AAA) development following a "normal" aortic ultrasound scan

Hafez H, Druce S, Scott RAP, Ashton H
Scott Research Unit, St Richard's Hospital, Chichester

Objective

To find what proportion of AAAs develop following a normal aortic screening scan.

Method

Over a 21-year period, men aged 65-80 participated in an AAA screening programme. Those with a maximum aortic diameter <30mm were classified as normal. A randomly selected sub-group was rescanned at 2 or 5-yearly intervals and data on aortic measurements, incidence of AAA events, and cause of death were collected. Data are presented as median (range).

Results

Twenty thousand five hundred and seventy-one men were scanned, of which 19,613 had an aortic diameter <30mm. Of these, a sub-group of 4,285 were rescanned and 122 (2.8%) were found to have developed an AAA (5.0 [0.3-11.1] years interval). Of the 122 patients, 92 (75%) had an initial aortic diameter of between 25-29mm. To date, 14 of these patients required surgery. Of the original 19,613 men classed as normal, 43 (0.2%) with an initial median scan of 25mm subsequently presented with an AAA event, which required surgery or caused death (interval from initial scan of 10.6 [3.8-16] years). Of these, 21 were treated electively with a 9.5% operative mortality, six had emergency surgery with a 50% operative mortality, and 16 died from rupture without surgery. In total, 21 (0.1%) patients with an initially normal scan died as a direct consequence of their AAA.

Conclusion

Of men aged 65-80 with an initial normal scan, 2.8% will eventually develop an AAA. Those with an initial aortic diameter of 25-29mm appear to be at an increased risk. More information is needed regarding the natural history of this subgroup particularly in view of their notably poor outcome.

Statins are associated with reduced all-cause mortality after endovascular abdominal aortic aneurysm repair

Leurs LJ, Visser P, Laheij RJF, Buth J, Blankensteijn JD, Harris PL, on behalf of the EUROSTAR collaborators

Royal Liverpool University Hospital, Liverpool

Objective

To investigate the influence of statins on early and late outcome following endovascular abdominal aortic aneurysm repair (EVAR).

Method

The study population, consisting of patients recruited into a registry during the period 1996-2005, was stratified in two groups according to whether or not they were taking a statin. Differences between the groups were assessed by Chi Square and t-tests for discrete variables and continuous variables. Outcomes during follow-up were assessed by life-table analysis and log-rank testing. A multivariate Cox proportional hazard model was used to identify independent risk factors and to correct for possible confounding factors.

Results

Of the 5892 patients enrolled in the registry, 731 (12.4%) patients were treated with statins for hyperlipidemia. Statin users were younger, more obese, and had a higher prevalence of diabetes, cardiovascular disease and hypertension. After 5 years of follow-up, the cumulative survival rate was 77% for non-users of statin vs. 81% for statin-users (p=0.005). After adjustment for age and other risk factors, statin use was still an independent predictor for improved survival (p=0.03).

Conclusion

Statins were prescribed more frequently for younger patients. After adjustment for age and medical risk factors the use of statins in patients who underwent EVAR was independently associated with reduced overall mortality. In the absence of specific contraindications statins should be prescribed for all patients who may be candidates for EVAR.

Abdominal Aortic Aneurysms (AAA) and the metabolic syndrome

Al-Barjas HSA, Ariëns RAS, Grant PJ, Scott DJA

The Academic Unit of Molecular Vascular Medicine; The L.I.G.H.T Laboratories; University of Leeds, Leeds

Objective

The metabolic syndrome (MS) is associated with increased risk of cardiovascular disease and thrombin generation. The aim of this study was to investigate the relationship between the metabolic syndrome, haemostatic proteins, and AAA.

Method

This cross-sectional survey consisted of 110 men with AAA, median age 78 years (IQR; 71-86), median max. AP diameter 4.5cm (IQR 3.2-5.2). The (MS) was diagnosed according to Adult Treatment Panel III criteria. All subjects had an ultrasound scan to determine the size of the aneurysm and the presence of intraluminal thrombus (ILT). Plasma fibrinogen, D-dimer, tissue-type plasminogen activator (t-PA), thrombin anti-thrombin (TAT), and prothrombin fragments 1+2 (PF1+2) were measured. The results were analysed using the Mann-Whitney U test and are expressed as a median below.

Results

The prevalence of the MS in the study group was 55%. AAA patients with MS had significantly higher fibrinogen concentration (2.84 vs 2.44g/L) $p<0.05$, D-dimers (496.50 vs 344ng/ml) $p<0.05$, t-PA (10.02 vs 8.50ng/ml) $p<0.05$, TAT (5.41 vs 4.17µg/l) $p<0.05$, and PF1+2 (1.08 vs 0.94µg/l) $p<0.05$, than AAA patients without MS respectively. Patients with MS had a larger AAA size than those without MS (4.30 vs 4.10cm) ($p<0.05$). AAA patients with MS have larger ILT within the lumen than those without MS (45% vs 35.0%)($p<0.05$).

Conclusion

In this study (AAA) patients with the metabolic syndrome had an elevated level of coagulation/fibrinolysis proteins and indirect evidence of increased ILT. Further longitudinal studies are required to quantify the volume of ILT and to explore the relationship between the MS and AAA expansion.

The IL-10 -1082 gene polymorphism: a candidate gene for abdominal aortic aneurysms

Lloyd GM, Bown MJ, Sayers RD

Division of Vascular Surgery, Department of Cardiovascular Sciences, University of Leicester, Leicester

Objective

Abdominal aortic aneurysms (AAAs) are characterised by loss of elastin, smooth muscle cell apoptosis and a chronic inflammatory infiltrate. Elastin loss is mediated by matrix metalloproteinases (MMPs) whose activity may be controlled by cytokines including interleukin-10 (IL-10). The aim of this study was to investigate the role of the IL-10 -1082 guanine (G) to adenine (A) polymorphism in the pathogenesis of AAAs.

Method

A prospective case-control study of 371 patients with AAAs and 346 screened control patients with normal aortic diameter was performed. IL-10 genotype at the -1082 position was determined by induced heteroduplex genotyping.

Results

The -1082 A:G allele frequency in AAA patients was 0.52:0.48 compared to 0.46:0.54 in controls (p=0.03, Fisher's exact test). The odds ratio for the A allele being a risk factor for AAA was 1.4 (95% confidence interval 1.02 to 2.0).

Conclusion

The presence of an A allele at the -1082 position in the IL-10 gene is associated with an AAA. This polymorphism appears important in the pathogenesis of AAAs.

Increased angiogenesis and activation of the HIF-1α/VEGF pathway in abdominal aortic aneurysm rupture

Choke E, Cockerill G, Wilson WR, Dawson J, Sayed S, Loftus I, Thompson MM
St George's, University of London, London

Objective

Angiogenesis, which involves the proteolytic degradation of extracellular matrix to facilitate endothelial cell migration, has been implicated in the aetiology of abdominal aortic aneurysms (AAAs). Clinical consequences of angiogenesis may include aortic wall weakening and rupture but its role in aneurysm rupture has never been investigated. We assessed the hypothesis that increased angiogenesis is involved in AAA rupture.

Method

Paired biopsies were obtained from the rupture site and anterior sac (control) in 12 ruptured AAAs. Further controls were obtained from the anterior sac of ten non-ruptured AAAs. Microvessel density was quantified using CD31 immunostaining. The mRNA of known angiogenic factors (relative to 18S rRNA) was quantified using qRT-PCR.

Results

Compared to the anterior sac, rupture site biopsies had significantly increased microvessel density (12.8+/-1.4 vs 4.1+/-0.2 vessels/HPF; $p<0.001$), smaller diameter microvessels (19.6+/-2.1 micrometer vs 45.6+/-3.5 micrometer; $p<0.001$) and increased mRNA levels of vascular endothelial growth factor (VEGF) (53.7+/-22.8 vs 22.6+/-10.5; $p<0.05$) and hypoxia-inducible factor (HIF)-1α (26.8+/-7.6 vs 11.6+/-4.3; $p<0.02$). VEGF mRNA expression correlated with microvessel density ($p<0.05$). There were no significant differences in mRNA levels of VEGF receptor-2 ($p=0.62$), VE-Cadherin ($p=0.17$), monocyte chemoattractant protein-1 ($p=0.12$) and Vimentin ($p=0.20$).

Conclusion

We have demonstrated increased angiogenesis at the aneurysm rupture site. Hypoxia-induced activation of VEGF transcription via the HIF pathway is an established key regulatory step in angiogenesis. The overexpression of both VEGF and HIF-1α at the aneurysm rupture site suggests that the increased angiogenesis is a homeostatic response to hypoxia. Further insights into the role of angiogenesis in AAA rupture may open novel therapeutic avenues to prevent AAA rupture.

Endovenous Laser Treatment (EVLT) or surgery for varicose veins? A randomised controlled trial in patients with saphenofemoral and long saphenous incompetence

Beale R, Theivacumar N, Mavor AID, Gough MJ
The General Infirmary at Leeds, Leeds

Objective

To compare EVLT, a minimally-invasive, local anaesthetic out-patient treatment, with conventional surgery, in the treatment of primary saphenofemoral and long saphenous incompetence (SF/LSVI).

Method

Sixty-one patients (75 limbs), 51% female, median age 49 (36-57) yrs, with CEAP class C2-C5 disease, were randomised to receive EVLT-1 (810nm diode laser, 12 watts pulsed laser), EVLT-2 (14 watts continuous laser) or surgery (SFJ ligation, LSV stripping, avulsions). Principal outcome measures at 3 months were abolition of SF/LSVI (duplex ultrasound) and symptomatic improvement (Aberdeen Varicose Vein Score - AVVS).

Results

Pre-treatment maximum LSV diameters were similar (EVLT-1: 8.1 [5.8-11.5] mm; EVLT-2: 8.1 [6.9-10] mm; surgery: 7.8 [6.4-9.7] mm). SF/LSVI was abolished in 73/75 limbs (EVLT-1: 29/30, EVLT-2: 20/21, surgery: 24/24). A significant improvement in AVVS ($p<0.01$, Wilcoxon Rank) occurred in all groups (median ± iq range) (EVLT-1: 13.17 [10.49-19.45] to 5.61 [0.60-7.52]; EVLT-2: 11.55 [8.45-19.19] to 3.94 [1.21-8.18]; surgery: 14.31 [9.41-20.23] to 5.80 [1.04-8.90]), although there were no inter-group differences (Kruskall-Wallis test, $p=0.842$). Analgesia use was similar in all groups, but return to normal activity (EVLT-1: 4 [1-7] days; EVLT-2: 4 [0-14] days) and work (EVLT-1: 4 [3-7] days; EVLT-2: 4 [3.25-14] days) were significantly quicker than after surgery (14 [2-28] days ($p=0.045$) and 17 [7-35] days respectively, [$p=0.011$] Kruskall-Wallis test). Thirty-five percent of EVLT patients required delayed sclerotherapy (1-2 treatments) to achieve satisfactory cosmesis.

Conclusion

EVLT is an effective alternative (resolution of symptoms, abolition of reflux) to conventional surgery for the treatment of primary varicose veins due to SF/LSVI. Further, it allows an earlier return to normal activity and work.

Topical bupivacaine in the long saphenous vein tract provides excellent analgesia: a prospective double-blind randomised study comparing bupivacaine with placebo following varicose vein surgery

Kibria SMG, Mavor AID

The General Infirmary at Leeds, Leeds

Objective

Early postoperative pain limits mobilisation after varicose vein surgery. This study investigated the efficacy of a new method of administering local anaesthesia and its effect on postoperative pain and mobility during the first 24 hours.

Method

The study group comprised 30 consecutive patients undergoing unilateral long saphenous vein stripping for varicose veins. Patients were randomised (15 in each arm) to receive either bupivacaine 0.5% or normal saline (supplied by pharmacy in coded syringes). Gauze soaked with 10ml of either saline or bupivacaine 0.5% was introduced into the tract of the long saphenous vein after stripping, left in the tract for the remainder of the operation and withdrawn just before completion. Postoperatively, a numerical rating and visual analogue scale assessed the pain levels. The analgesic requirements during the first postoperative day were recorded. A visual analogue scale also recorded the extent of limitation of movement during the same period.

Results

None out of 15 patients in the bupivacaine arm of the study required oral analgesia compared to 13 out of 15 patients in the saline arm needing between two and six (median three) oral doses of analgesia during the same postoperative period ($p<0.03$). There was a significant reduction in postoperative pain scores in the bupivacaine group ($p<0.0001$) and less restriction of movement ($p<0.0001$).

Conclusion

This method of delivering topical bupivacaine, to the long saphenous vein tract after stripping is easy, safe and provides excellent postoperative analgesia with resultant significant reduction in limitation of movement, making day-case varicose vein surgery much more acceptable.

A hybrid screening programme for clinically significant abdominal aortic aneurysms

Heng MST, Venkatasubramaniam A, Lee DLH, Bryce J, Tennison C, Berry B, Chetter I, McCollum PT

Academic Vascular Unit, Hull Royal Infirmary, Hull; University of Hull, Hull

Objective

Cost-effectiveness analysis of the Multicentre Aneurysm Screening Study showed the cost of a quality-adjusted life year (QALY) as £36,000 for the first year. We propose a hybrid screening programme with self-examination and abdominal ultrasound scan only in patients with a positive result, lowering costs to an acceptable level for a national screening programme. The aim therefore was: i) to assess the sensitivity and specificity of self-examination in the detection of clinically significant (>5cm) AAA in the community; and ii) to assess the psychological consequences of screening for AAA by self-examination.

Method

A thousand male patients, age 65 and above, were invited to participate in the screening programme by postal questionnaire using a novel technique of self-examination described in a previously validated study. All participants then had abdominal ultrasonography to determine aortic diameter. The psychological consequences of screening were assessed with the SF-36 (Short Form-36) health survey questionnaire, and HAD (Hospital Anxiety and Depression) scale, at the time of self-examination, after ultrasound scanning and 1 month after.

Results

Six hundred and ninety-one patients (69%), median age 72 (65-93) agreed to take part in the study. Twenty AAAs were detected by abdominal ultrasonography, of which six were >5cm. Sensitivity for self-examination in the detection of AAA >5cm was 83.3%. The specificity for self-examination in detecting AAA was 85.5%. Mental health was affected on introduction of screening but improved significantly ($p<0.021$) following the scan. Similar results were obtained with the HAD scale, with the patient's anxiety levels improving ($p<0.020$) following the scan.

Conclusion

Screening for AAA by self-examination is effective for clinically significant AAAs, and causes mild but transient psychological stress.

Results of open Abdominal Aortic Aneurysm (AAA) repair via a left upper quadrant transverse transperitoneal minilaparotomy incision

Subramanian A, Sutaria R, Witcomb M, Hafez H
St Richard's Hospital, Chichester

Objective

Traditional open AAA surgery involves full vertical or transverse incisions that are associated with extensive exposure of the abdominal cavity and its contents. Here we describe a versatile yet limited alternative exposure.

Method

Between June 2002 and May 2005, 65 consecutive patients undergoing elective (58) and urgent non-rupture (7) open AAA repairs were included. Aneurysm exposure was achieved via a horizontal muscle cutting and/or muscle-sparing transperitoneal left upper quadrant incision. The incisions were made within or up to, but not beyond, the midline medially and the anterior axillary line laterally. The bowel was retracted within the abdomen and standard open repair was carried out. Data are presented as the median (range).

Results

Age was 73 (60-85) years, BMI was 26 (21-35), ASA was 3 (2-4), and aneurysm size was 7 (5.2-10.2) cm. Eighteen percent had a bifurcated graft. Operation time was 145 (80-255) minutes. Blood loss was 1200 (350-4000) ml. Eighty-six percent of patients were able to consume free fluids by 6 hours and 56% were on a free diet by 18 hours. Respiratory complication rate was 7.5%. There were no wound infections. One patient in the elective group died from colonic ischaemia and one patient in the urgent group died from cord ischaemia (1.7% and 14% in-hospital mortality respectively). Postoperative hospital stay was 6 (3-68) days as compared to 9 (4-53) days for 160 patients with the traditional full exposure ($p<0.001$ Mann-Whitney).

Conclusion

Our described approach provides good access to the infrarenal abdominal aorta without unnecessary bowel exposure. It allows early oral intake and is associated with low morbidity and mortality and shorter hospital stay.

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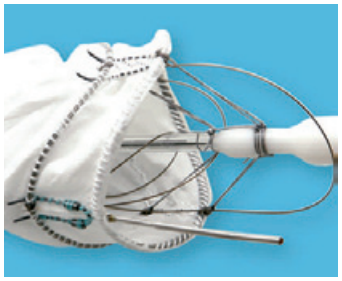

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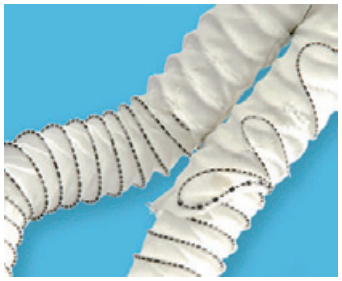

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The constitutive procoagulant and hypofibrinolytic state in patients with intermittent claudication significantly improves with percutaneous transluminal balloon angioplasty

Hobbs SD, Fegan C, Adam DJ, Bradbury AW

University Department of Vascular Surgery, Birmingham Heartlands Hospital, Birmingham

Objective

Patients with intermittent claudication (IC) exhibit a baseline prothrombotic diathesis that is acutely exacerbated by exercise. This may occur due to ischaemia-reperfusion injury during walking and contribute to the observed increased risk of thrombotic vascular events. This study compares the effect of angioplasty (PTA), supervised exercise (SE) and best medical therapy (BMT) alone on this prothrombotic state.

Method

Twenty-three patients (16 men and 7 women, median age 67, range 57-77, years) were randomised to receive PTA (n=9), SE (n=7) or BMT alone (n=7) as part of the EXercise versus Angioplasty in Claudication Trial (EXACT). Patients were assessed at baseline, 3 and 6 months. Thrombin anti-thrombin complex (TAT) was determined as a marker of thrombin generation and plasminogen activator inhibitor (PAI) antigen as a marker of fibrinolysis. Elevated TAT indicates a procoagulant state and elevated PAI a hypofibrinolytic state.

Results

At 6 months, subjects randomised to PTA demonstrated significant improvements in ankle:brachial pressure index (p=0.013) and maximal walking distance (p=0.008); a significant decline in resting thrombin generation (median [IQR] TAT 6.4 [2.7-13.5] to 1.5 [0.3-2.9] mg/l, p=0.038) and an improvement in resting fibrinolysis (median [IQR] PAI-1 10.0 [1.0-20.5] to 1.0 [1.0-14.8] ng/ml, p = 0.043). There was no significant change in any of these parameters in patients randomised to either SE or BMT alone.

Conclusion

Lower limb revascularisation by PTA results in an improvement in the resting procoagulant and hypofibrinolytic state in patients with IC. This may translate into a reduction in morbidity and mortality from thrombotic vascular events in this group of patients.

Acquisition of endovascular skills by consultant vascular surgeons: effect of repetition in a virtual reality training model

Aggarwal R [1], Black SA [1,2], Hance JR [1], Darzi AW [1], Cheshire NJW [1,2]

[1] Department of Biosurgery and Surgical Technology, Imperial College, London; [2] Regional Vascular Unit, St Mary's Hospital, London

Objective

It is no longer acceptable to learn to perform interventional procedures on real patients. Recent advances in virtual reality (VR) technology have enabled the use of simulators to teach skills for interventional vascular procedures. The aim of this study was to evaluate VR simulation for endovascular training of surgeons inexperienced in this technique.

Method

Twenty consultant vascular surgeons were divided into those who had performed >50 endovascular procedures (e.g. aortic and carotid stent) as primary operator (n=8), and those having performed <10 procedures (n=12). To test for endovascular skill rather than procedural knowledge, all subjects performed a renal artery balloon angioplasty and stent procedure. The simulator uses real tools with active force feedback, and provides a realistic image of the virtual patient. Surgeons with endovascular skills performed two repetitions and those without completed six repetitions of the same task. The simulator recorded performance for parameters such as time taken and amount of contrast fluid used.

Results

Surgeons with endovascular skills were significantly faster (median 571.5 vs. 900.0 seconds, p=0.039) and used less contrast fluid (19.1 vs. 42.9ml, p=0.047) than inexperienced operators. Over six sessions, the inexperienced group significantly improved their performances for time taken (p=0.007) and contrast fluid usage (p=0.021), achieving similar scores to experienced endovascular operators.

Conclusion

Surgeons with minimal endovascular experience can improve their performance during structured training on a VR endovascular task. Thus VR simulation may be useful for the early part of the learning curve for surgeons who wish to expand their endovascular interests.

VEGF gene therapy enhances venous thrombus resolution

Modarai B, Humphries J, Gossage JA, Burnand KG, Afuwape A, Paleolog E, Smith A
St Thomas' Hospital, Cardiovascular Division, King's College, London

Objective

Neovascularisation is associated with a rise in the expression of vascular endothelial growth factor (VEGF) in resolving venous thrombi. We investigated whether adenovirus-mediated transfection of the VEGF gene (ad.VEGF) enhances thrombus recanalisation and resolution.

Method

Thrombus was formed in the inferior vena cava of Wistar rats and SCID mice. Adenovirus gene constructs encoding green fluorescent protein (ad.GFP), ad.VEGF or empty control virus (ad.0) were injected into rat thrombus. Human monocytes, transfected *in vitro* with either ad.VEGF or ad.0, were injected into thrombi formed in SCID mice. GFP was localised at 3 and 7 days and VEGF concentration measured between 1-7 days. Thrombus size and recanalisation were measured after 7 days.

Results

At day 3, GFP expression was mainly seen in the vein wall and adventitia. By day 7, GFP expression was also located in cells within the body of thrombus. Expression of VEGF protein peaked at 4 days (660 pg/ml). Ad.VEGF caused >50% reduction in thrombus size (22.0 +/- 4.0mm^2 vs 47.7 +/- 5.1mm^2, p=0.0005, n=20/group) and over a three-fold increase in recanalisation (3.9 +/- 0.69% vs 13.6 +/- 4.1%, p=0.0003) compared with controls. Injection of ad.VEGF transfected monocytes almost halved thrombus size in mice compared with controls (6.1 +/- 1.4mm^2 vs 11.6 +/- 1.0mm^2, p=0.01, n=12/group), but had no effect on recanalisation.

Conclusion

The reduction in thrombus size and increase in recanalisation suggests that the ad.VEGF construct could be used as a novel treatment for venous thrombosis and may reduce post-thrombotic complications by prompt restoration of vein lumen patency.

Early experience of endovenous laser ablation of the short saphenous vein

Watson AB, Bani-Hani M, Modaresi K, Greenstein D
Department of Vascular Surgery, Northwick Park Hospital, Harrow

Objective

Endovenous laser ablation (EVLA) for the long saphenous vein has become an established alternative to surgical stripping. It can be performed under local anaesthesia in a minor operations room and is associated with early return to work and less postoperative pain, as no sutures are required. Data on EVLA for short saphenous vein (SSV) are lacking. We present our early experience of EVLA on the treatment of the SSV.

Method

Fifty consecutive patients with symptomatic varicose veins due to duplex proven SSV incompetence underwent EVLA using a 980nm diode laser (M:F 15:35, mean age 50 [30-93]). EVLA was applied from the saphenopopliteal junction (SPJ) to the lower third of the SSV. All procedures were performed in a sterile manner under a local anaesthetic (1% lignocaine/saline 0.9% solution) with no sedation in a minor operations room by a single surgeon. Follow-up was at 1 week, 2 months and 6 months.

Results

All 50 patients had duplex proven closure of the SPJ and at 6 months (100%). Mean energy was 1280 joules (530-2402) at a pull back rate of 40-50 joules/cm and power 8-12 watts. One patient had recanalisation of the SSV but the SPJ remained closed. One patient (2%) had a neuropraxia of the sural nerve which recovered by 6 months. No deep vein thrombosis (DVT) or skin burns occurred.

Conclusion

The early experience of EVLA shows this to be a safe and effective treatment for symptomatic SSV incompetence. Long-term studies are required before this can be fully recommended.

A double-blinded, randomised study to determine the effect of omega-3-marine triglycerides on intermittent claudication

Conway K, Dillon M, Evans J, Howells-Jones R, Price P, Harding KG, Hill S
Cardiff Regional Vascular Unit, University Hospital of Wales, Cardiff

Objective

Fish oils have been shown to be of benefit in patients with coronary artery disease. Their effect in peripheral arterial disease (PAD) is unclear.

Method

Fifty patients with lower limb intermittent claudication (IC) were recruited from consecutive presentations to a teaching hospital vascular unit and randomised to treatment or placebo groups. The treatment group were given 10g concentrated omega-3 fish oils (1.7g eicosapentaenoic acid, 1.15g docoshexaenoic acid) daily for 16 weeks. The placebo group received 10g mixed oils. Patients received best medical therapy and took part in a supervised exercise programme. Quality of life was assessed using the SF-36v2 questionnaire. Ankle Brachial Pressure Indices (ABPI), Initial and Absolute Claudication Distances (ICD, ACD) were measured before and after treatment. ICD and ACD were assessed by a graded-treadmill test.

Results

Thirty-five males and 15 females were recruited. The mean age was 66.1 years (49-82 years). Six patients in total withdrew; two patients suffered a myocardial infarction, 1 of which died. Four withdrew as a result of nausea. There was no difference between the treatment groups in the baseline characteristics of age, ABPI and ACD ($p>0.05$, t-tests). Post-intervention there was no significant difference in ABPI or ACD. Sub-stratification of patients by severity (fiftieth centile) found improvement in the ICD of 53.7% ($p=0.03$, Mann-Whitney U). There was no statistical difference in the quality of life outcomes.

Conclusion

Omega-3 fatty acid supplementation benefits patients with IC by increasing the distance walked before experiencing pain. Further studies are required to assess the long-term benefits in PAD.

Risk factors for the development and subsequent growth of small abdominal aortic aneurysms

Wilmink ABM, Adam DJ, Hubbard CS, Bradbury AW, Quick CRG
University Department of Vascular Surgery, Birmingham Heartlands Hospital, Birmingham

Objective

To examine risk factors associated with the development of new abdominal aortic aneurysms (AAA) and growth of small AAA.

Method

One thousand eight hundred and twenty men (mean age 60, range 50-86 years) had two or more ultrasound measurements of infrarenal aortic diameter at a median interval of 6 (range, 5-10) years apart. At first scan, 1726 men had a normal aorta (median diameter, 21 [range, 14-29] mm) and 94 (5.2%) had small AAAs (median diameter, 31 [range, 30-51] mm). Significant aortic growth was defined as diameter increase >5mm during follow-up. The following clinico-pathological data were collected prospectively: age, height, weight, blood pressure, current smoking status, smoking history, family history of AAA, occupational history, comorbidity and medication. Serum biochemistry, glucose and lipid profile were determined in a random sample.

Results

Significant aortic growth occurred in 95 of 1820 (5.2%) men: 47 (2.7%) with a previously normal aorta and 48 with small AAA. By multivariate analysis, history of ischaemic heart disease (RR 2.0, 95% CI 1.1-3.7; p=0.02), increased initial aortic diameter (1.32, 1.26-1.39; p<0.0001), current smoking (4.1, 1.8-9.5; p=0.001) and low HDL cholesterol (0.13, 0.05-0.34; p<0.0001) were associated with an increased risk of aortic growth. There was no relationship with serum total cholesterol, LDL cholesterol or triglyceride levels and aortic growth.

Conclusion

These novel data demonstrate, for the first time, a strong association between low levels of HDL cholesterol and new AAA development and AAA growth. Strategies to increase endogenous levels of HDL cholesterol may complement smoking cessation in the chemoprevention of AAA.

Self-assessment of technical skill: the need for expert feedback

Pandey VA, Wolfe JHN, Black SA, Liapis CD, Bergqvist D
On behalf of the European Board of Vascular Surgery

Objective

Technical skill has been assessed in the EBSQ-VASC examination since 2002. The purpose of this study was to examine the relationship between expert assessment and trainee self-assessment.

Method

Forty-two exam candidates performed a saphenofemoral junction ligation (SFJ) and an anterior tibial anastomosis on a synthetic simulation. They were rated by two independent examiners (using a validated rating scale of generic skill) for both procedures. Candidates then anonymously rated their own performance on the same scale. Parametric tests were used in the statistical analysis. $p<0.05$ was considered significant.

Results

The maximum mark in the assessment was 40. Twenty-four was considered competent. The inter-observer correlation for examiners' marks was high (SFJ ligation, alpha=0.68; distal anastomosis, alpha=0.76). Average examiners' marks for SFJ ligation ranged from 19-38.5 (mean 27.8), 36 candidates (85.8%) attaining a competent score. Self-assessment scores ranged from 24-40 (mean 30.7). Examiners' marks for the distal anastomosis ranged from 21-40 (mean 29.2). Thirty-nine candidates (92.8%) attained a competent score. Candidates' marks in this station ranged from 24-40 (mean 32.1). There was no correlation between examiner and self-assessment in either station (Pearsons Correlation Coefficient: SFJ, r=0.045, p=NS; distal anastomosis, r=0.089, p=NS). Bland and Altman plots were used to assess the agreement between examiner and self-assessment. These showed candidates marked themselves higher than examiners with a mean difference of 2.9 marks in each station.

Conclusion

Candidates' self-assessment and expert independent assessment correlate poorly. Trainees overestimate their abilities according to independent assessment; regular technical feedback during training is therefore essential.

A change in isolation policy reduces MRSA colonisation ten-fold

Thompson MM, on behalf of the St George's Vascular Department
Department of Vascular Surgery, St George's Hospital, London

Objective

In 2003, 18% of admissions to our vascular ward were colonised by MRSA, with an MRSA infection rate of 10.6%. Standard practice was to segregate patients with proven MRSA from the rest of the patient pool. After a prospective audit, regression analysis was used to identify factors that could stratify patients into high and low risk for MRSA colonisation. A change in isolation policy was introduced that segregated patients according to their risk of MRSA acquisition, and isolated all patients undergoing prosthetic vascular reconstruction. This study reports the impact of these changes on MRSA colonisation and infection rates.

Method

The MRSA status of patients during 777 in-patient episodes was prospectively recorded during three time spans; period 1 (11/02-4/03) before the change in isolation policy and, periods 2 (8/03-12/03) and 3 (10/04-1/05) after the change in policy.

Results

Hospital-acquired MRSA colonisation was reduced from 10.6% in period 1, to 1.1% and 1.4% in periods 2 and 3 respectively ($p<0.001$). Similarly, MRSA infection rates fell from 10.6% to 2.9% and 0.9% over the same time frame ($p<0.001$). The most dramatic changes in MRSA infection rates occurred in patients undergoing aneurysm repair (MRSA infection 30.1% in period 1 vs. 3.9% and 2.9% in periods 2 and 3) and lower limb revascularisation (31% vs 0% vs 4.2%). Stepwise regression analysis revealed that the system of isolation was a significant factor reducing MRSA infection and colonisation rates ($p<0.001$).

Conclusion

These data demonstrate that a change in infection control policy can significantly reduce MRSA infection in a vascular unit.

Carbon monoxide-releasing molecules (CO-RMs) modulate the neuro-inflammatory response in BV-2 microglia: a novel approach to stroke

Bani-Hani MG, Greenstein D, Mann BE, Green C, Motterlini R

Northwick Park Institute for Medical Research, Harrow; North West London Hospitals NHS Trust, Northwick Park Hospital, Harrow

Objective

Carbon monoxide-releasing molecules (CO-RMs) are emerging as a new class of pharmacological agents that modulate important cellular function by liberating CO in a biological system. In this study, we examined the role of CO-RMs in modulating neuro-inflammatory responses in BV-2 microglial cells, considering its practical application as a novel therapeutic alternative in the treatment of stroke.

Method

BV-2 microglial cells were cultured and grown in medium containing 10% foetal bovine serum. Sub-confluent cells were incubated for 16 h in normoxic conditions with thrombin alone or in combination with interferon-γ to simulate the inflammatory response. BV-2 microglia were also subjected to 12 h hypoxia and reoxygenated for 24 h in the presence of thrombin. In both sets of experiments, the anti-inflammatory action of CO-RMs was evaluated by assessing their effect on nitric oxide production (nitrite levels) and TNF-a release.

Results

CO-RMs (75μM) did not show any cytotoxicity and markedly attenuated the inflammatory response to thrombin both in normoxic and hypoxic conditions as evidenced by a significant reduction ($p<0.05$)(one-way ANOVA/Bonferroni) in nitrite levels and TNF-a production. CO-RMs also inhibited the pro-inflammatory effect of interferon-γ alone or in combination with thrombin. Inactive (CO-RMs), which do not liberate CO, failed to prevent the increase in inflammatory mediators suggesting that CO is responsible for the observed effects. CO-RMs appears to act at multiple levels, through interaction with several signal transduction pathways.

Conclusion

These results suggest that the anti-inflammatory activity of CO-RMs could be exploited to mitigate microglia activity in stroke and other neuro-inflammatory diseases.

Value of MRI in post-procedural evaluation of carotid angioplasty and stenting

McDonnell CO [1], Fearn SJ [1], Baker SR [1], Price D [2], Goodman MA [1], Lawrence-Brown MMD [1]

[1] Departments of Vascular Surgery and [2] Radiology, Mount Medical Centre, Perth, Western Australia

Objective

To assess diffusion-weighted MRI as a diagnostic tool in evaluating the incidence of neurological injury following carotid angioplasty and stenting (CAS).

Method

The first 110 cases of CAS in our unit were included in this series. The procedure was abandoned in three patients, the remaining 107 being divided into group A (n=12) who had no cerebral protection device (CPD), and group B (n=95) who had a cerebral protection device deployed during the procedure. Patients underwent diffusion-weighted MRI prior to, and following CAS and underwent a formal neurological assessment in the Intensive Care Unit after the procedure.

Results

One hundred and ten procedures were attempted in 98 patients. Twenty-eight percent were asymptomatic. Following CAS, 7.2% of patients had a positive neurological exam and 21% had positive DWI scans, equating to a sensitivity of 86% and a specificity of 85% for DWI in detecting cerebral infarction following CAS. The positive predictive value of the test was 0.3 and negative predictive value 0.99, with a likelihood ratio of 5.7. The major stroke and death rate was 1.8%. Use of a cerebral protection device significantly reduced the incidence of both clinical (5% in CPD vs. 25% in non-protected, $p<0.05$, Fisher's Exact Test), and DWI-detected subclinical cerebral infarction (18% CPD vs. 33% in non-protected, $p<0.05$, Fisher's Exact Test).

Conclusion

The incidence of subclinical DWI-detected neurological injury is significantly higher than clinical neurological deficit following CAS. More sensitive tests of cerebral function are required to establish whether these subclinical lesions are relevant.

The current performance of carotid endarterectomy (CEA) in the UK: an interim analysis of 1001 patients randomised in the GALA trial

Dellagrammaticas D, Gough MJ, on behalf of the GALA Trial participants
The General Infirmary at Leeds, Leeds

Objective

Data from the ECST, NASCET and Veterans' trials established the role of CEA for symptomatic carotid stenosis despite a stroke and death rate of 6.2%. An interim analysis of 1001 UK patients randomised within the GALA trial, blinded to anaesthetic allocation, has been performed to provide a contemporary comparison with these trials.

Method

Demographic data and 30-day stroke, death and MI rates (determined by an independent stroke physician) were collected prospectively in 1001 patients from 30 UK centres.

Results

The median time from symptoms to CEA was 80 days (37-142). Following surgery 54/1001 (5.4%) had a stroke (25/1001 [2.5%] minor [Rankin 0-2], 14/1001 [1.4%] disabling [Rankin 3-5], 15/1001 [1.5%] fatal within 6 months) and 5/1001 (0.5%) an MI. Thirty-day mortality was 19/1001 (1.9%) and the combined stroke/death rate 6.3%. Patients were older (median 72 years [65-78], 38% v 10% >75, $p<0.001$) and more likely to have a contralateral carotid occlusion (12% v 4%, $p<0.001$) than those in the landmark trials.

Conclusion

Current UK outcomes for CEA are identical to those of the landmark trials, despite potential risk factors (age, contralateral carotid occlusion) and compare favourably with those for carotid angioplasty and stenting (9.0% combined stroke and death rate, meta-analysis of randomised trials). Nevertheless, data from the landmark trials and the Oxford Vascular Study indicate that a significant proportion of patients will suffer an intervening stroke when CEA is delayed for >2-4 weeks after the initial event. Thus, primary care and emergency physicians must facilitate earlier referral to maximise stroke prevention by CEA.

Annual General Business Meeting Agenda

Thursday 24th November 2005 at 5.00pm

1. Apologies
2. Minutes of AGM 2004
3. President's Report: Professor Michael Horrocks
4. Honorary Secretary's Report: Mr Peter Lamont
5. Honorary Treasurer's Report: Mr David Berridge
6. Audit and Research Committee Report: Mr Simon Ashley
7. Training and Education Committee Report: Professor Julian Scott
8. Professional Standards Committee: Professor Bruce Campbell
9. Vascular Tutor: Mr Shane MacSweeney
10. British Vascular Foundation Subcommittee: Professor Sir Peter Bell
11. Election of Officers: result of ballot for Ordinary Members of Council
12. AGM 2006: Mr John Wolfe
13. Any other business

Honorary Secretary's Report

Peter Lamont

As we approach our 40th Annual Meeting in Bournemouth, The Vascular Society continues to stay at the forefront of the surgical specialist societies with progress on a number of initiatives designed to address the needs of our patients and improve the services we can offer them. Modernising Medical Careers has presented a real threat to recruitment into our specialty, but this threat has been recognised and turned into a major opportunity for us to lead the world in the development of the vascular specialist of the future. Initiatives with the Royal College of Radiology and PMETB, combined with continual lobbying of the General Surgery SAC, will hopefully ensure that the newly appointed vascular consultant of the future will be competent to treat elective and emergency vascular patients using the most appropriate open or endovascular interventional techniques, despite the limited training time available to them. The Society has also facilitated the dissemination of the highest quality vascular research to promote in particular the case for EVAR and aortic aneurysm screening. The National Screening Committee will make a final decision on their recommendations to the government regarding aneurysm screening the week after our AGM and their Programme Director, Sir Muir Gray, has been very impressed with the contribution of the Society to this initiative.

The Society continues to lobby the Department of Health and has made significant inroads politically, with increasing recognition of our role as a voice for vascular service development. Whether it be joint audits with the Healthcare Commission helping to fund the National Vascular Database, work on the development of appropriate HRGs and clinic types for vascular procedures under Payment by Results, pressure to include peripheral vascular disease as a target area under the new GP contract, collaboration with NICE over vascular guidelines or engagement with the DoH's Vascular Board overseeing National Service Frameworks for cardiac disease, diabetes and stroke, we continue to increase the profile of our Society with an increasing reputation for the quality and effectiveness of our initiatives. All of this success comes from the tremendous spirit of teamwork, expertise and collaboration which exists in your Council, all of whom deserve a huge vote of thanks for their unstinting contributions over the last year and whose support makes my job so rewarding.

Last year's Harrogate meeting was of the highest quality and continued the newly consistent trend of producing a steady income stream to support the Society's financial recovery. Over the past three years, the organisation, budgeting and administration of the annual meeting has been taken over by the Secretariat, which allows Jeanette Robey and I to carry over our experience from year to year and learn profitably from our successes and occasional mistakes. This frees the President to focus on the scientific and social programme,

which continues to expand and improve. We have also continued the initiative of holding a one-day educational event in the Spring, this year with a very successful meeting on the theme of lymphoedema, which resulted in an invitation to the British Lymphology Society to join our collaboration alongside other vascular interest societies. The Endovascular Forum represents just such a collaboration between The Vascular Society and the British Society of Interventional Radiology and their meeting at the Belfry this year provided a spectacular backdrop for the announcement of the one-year EVAR trial results to a packed hall.

The British Vascular Foundation has been incorporated into the Society for the past year now and has continued its programme of fundraising events throughout the year. Many thanks go to those members who have supported these events. The Secretariat has been making strenuous efforts to find an experienced professional fundraiser with a view to taking the BVF's income stream to higher levels by targeting trust funds and major donors. After much specialist advice, we now seem closer to an appointment and I hope will be able to report positively at the AGM in this area.

Finally, I should like to express my particular thanks to Julian Scott and Simon Ashley for their huge contributions to the Society over the past four years where they have both put in an immense amount of work and effort chairing their respective committees. They have both taken on significant challenges and have met them on your behalf with dedication, skill and determination. They have also been very effective members of the Society's Executive Committee. They will be replaced next year by Cliff Shearman and Tim Lees, both of whom have already begun to get to grips with their role and I am sure will build on Julian's and Simon's successes. I also look forward next year to working with Jonothan Earnshaw, who will shadow me for the year before taking over as Secretary after the 2006 AGM in Edinburgh.

Honorary Treasurer's Report

David Berridge

This year has been a year of consolidation. The 2004 AGM made a profit in line with our requirements for financial recovery. To reduce our liability on the stock market, whilst we are trying to stabilise the assets of the Society, all shares have been cashed and placed in a high interest account. It is intended to maintain this position until we have sufficient assets to allow the Society to function for a full year even in the event of a major loss at the AGM. A low risk investment is then likely to be undertaken.

Budgeting for the Bournemouth meeting is also on target to continue the financial recovery programme. Future AGM venues will inevitably be dictated by the need to generate sufficient profits to support the running of the Society, at the same time as maximising the quality and facilities of that meeting. The running costs have been scrutinised and are approximately £190,000. Major Sponsors and annual subscriptions contribute £110,000. It is inevitable that the running costs will continue to increase. I therefore estimate that an overall profit for future meetings needs to be a minimum of £70,000 in order to meet the running costs of the Society and to achieve financial stability. Annual subscriptions will, however, need to be increased to allow for the reduction in income from Major Sponsors. The charges to exhibitors will also need to be increased in an attempt to generate more income to further offset this loss of income. The first full year accounts under the new accounting year (July 2004-June 2005) are now available (pages 98-99). These show that your Society is in a stable position. However, we have still not achieved a more robust position to allow your Society to function for a full year in the event of any major loss at the Annual General Meeting. We continue to strive to achieve this level of security.

Financial arrangements, in the form of Memorandums of Understanding, have been agreed between the Society and the Venous Forum of the Royal Society of Medicine, The British Society of Interventional Radiology, the Society of Vascular Nurses, and the Society for Vascular Technology. A similar arrangement is currently being organised with the Joint Vascular Research Group. These will clarify arrangements to support each other's activities, to reimburse the Society for defined costs on an annual basis and to apportion profit or loss, in the event of an overall loss on the AGM. This is imperative to ensure that all Societies are very clear as to their individual liabilities.

The British Vascular Foundation's finances are managed separately from the main Society's account. The Owen Shaw legacy has contributed a significant amount of money to this account. Overall expenditure has been covered by continuing fund-raising activities including

charity dinners in London and Newcastle, organised by Andrew May and Tim Lees respectively, marathon runs, golf days and covenants. The uptake on Gift Aid has, to date, not been as widely taken up as the Committee had hoped.

The new website (www.vascularsociety.org.uk) continues to be developed with new patient information sheets designed by Simon Parvin. We now have web-based advertising for our Major Sponsors with direct links to their own websites. In addition, the new British Vascular Foundation website (www.bvf.org.uk) has been produced with the help of Kieren Hasler and Jeanette Robey. I encourage all members to email comments on either website to the office and we will then use these to further develop both sites.

I would like to thank our Major Sponsors Boston Scientific Limited, B Braun Medical Limited, WL Gore & Associates (UK) Limited, and Vascutek Limited. I would also like to thank Cryolife Europa Limited and Bard Limited for their past support of the Society, as they have decided not to continue as Major Sponsors.

Membership Categories		Subscription rate 1 January 2005	1 January 2006 *Proposed*
Ordinary	(n=451)	£135	£160
Affiliate, Overseas and Associate	(n=163)	£70	£90
Senior	(n=110)	NIL	
Honorary	(n=21)	NIL	
TOTAL	N=745		

VSSGBI Limited
Profit and loss account
Year ended 31st December 2004

	2004 £	2003 £
Turnover	319,859	254,043
Cost of sales	262,798	160,508
Gross Profit	57,061	93,535
Administrative expenses	(15,054)	(12,213)
Other operating income	22,303	13,250
Operating Profit	64,310	94,572
Interest receivable	292	-
Profit on ordinary activities before taxation	64,602	94,572
Tax on profit on ordinary activities	-	-
Profit on ordinary activities after taxation	64,602	94,572
Deed of covenant	64,602	67,578
Balance brought forward	1	(26,993)
Balance carried forward	1	1

The Vascular Society
Statement of financial activities
For the year ended 30 June 2005

	Year ended 30 June 2005			Period ended 30/06/04	Year ended 31/10/03
	Unrestricted Funds £	Restricted Funds £	Total Funds £	Unrestricted Funds £	Unrestricted Funds £
Incoming resources:					
Subscriptions	66,970	-	66,970	32,138	56,255
Deed of covenant	64,602	-	64,602	67,578	-
Sponsorship	44,000	-	44,000	24,000	48,000
Legacies	-	56,000	-	-	-
Donations, covenants, gift aid and other income	32,898	-	32,898	2,058	4,495
Activities to generate funds:					
Fundraising income	30,159	-	30,159	-	-
Bank interest	7,124	-	7,124	747	878
Investment income	2,755	-	2,755	4,531	6,917
Tax recoveries and interest	3,074	-	3,074	-	-
Total incoming resources	**251,582**	**56,000**	**307,582**	**131,052**	**116,545**
Resources expended					
Costs of generating funds:					
Fundraising expenditure	19,931	-	19,931	-	-
Direct charitable expenditure	5,500	-	5,500	5,250	7,750
Management and administration of the charity	222,306	-	222,306	134,402	180,424
Total resources expended	**247,737**	**-**	**247,737**	**139,652**	**188,174**
Net incoming/(outgoing) resources for the period	**3,845**	**56,000**	**59,845**	**(8,600)**	**(71,629)**
Other recognised gains:					
Realised profit on sale of investments	7,152	-	7,152	1,476	4,353
Net movement in funds	**10,997**	**56,000**	**66,997**	**(7,124)**	**(67,276)**
Funds bfwd	-	-	-	158,480	225,756
Funds transferred in from The Vascular Surgical Society of Great Britain and Ireland	151,356	-	151,356	-	-
Funds transferred in from the British Vascular Foundation	138,751	11,548	150,299	-	-
Fund balance cfwd	**301,104**	**67,548**	**368,652**	**151,356**	**158,480**

Note: There were no restricted funds in 2003 and 2004 for The Vascular Society

Audit and Research Committee Report

Chairman: Simon Ashley

This is my final year of chairmanship of the Audit and Research Committee and I wish to take this opportunity to thank members of the Committee, past and present, for their tremendous help and support during the last four years.

National Vascular Database (NVD)

The principal commitment of the Audit and Research Committee is development of the NVD. The aspiration is that the NVD will become the primary source of national audit data relating to index vascular procedures, as well as the foundation for clinical governance within vascular surgery. There has been continual expansion of the number of surgeons / centres contributing to the NVD. At the time of writing, 298 surgeons are coded and "data consented" (and therefore contributing or committed to contribute to the NVD) from 110 centres. The fourth NVD report was published earlier this year. In addition to a copy of the report, contributing surgeons received a personal summary of their own results showing risk-adjusted comparative analyses for each index procedure as well as Funnel Plots of crude mortality after surgery for unruptured-AAA (URAAA) by surgeon and by hospital.

This year has also seen the introduction of "minimal" datasets for each index procedure, as well as the addition of Major Lower Limb Amputation as the "4th" index procedure. The data collection forms have essentially been reduced to a single side of A4 for each procedure. These changes have been incorporated into a new version of the Microsoft Access Database, available for download from the Society's website. Please note that endovascular abdominal aortic aneurysm repairs should now be included within the NVD AAA dataset. Work has begun on establishing a means of web-based data entry available to any member connected to the Internet. This will enable surgeons to submit and update their NVD index procedure data online. The Vascular Anaesthesia Society of Great Britain and Ireland has successfully piloted a similar system of web-based data entry for vascular anaesthetic data.

Members are reminded to check the details of patient episodes recorded under their care, particularly deaths, so that the HES data submissions from their hospitals will be accurate. If you wish to start submitting your index cases (aortic aneurysm repair; carotid endarterectomy; infrainguinal bypass; major lower limb amputation), please contact Sara Baker (see below) as you will need to be data consented and coded. She will advise you on appropriate data collecting systems. Members are reminded that although it is one of the options, a Dendrite system is not essential for data collection and submission. The closing date for annual data submission is 31st March 2006.

Clinical governance of the NVD

Earlier this year the Audit and Research Committee proposed thresholds that could be used to trigger the clinical governance process formulated by the Professional Standards

Subcommittee. Council agreed that surgery for URAAA would be used as the "tracker" procedure. Surgeons with a crude mortality, aggregated over the preceding three financial years, within the upper 99% confidence limit will be deemed to meet the VSGBI standard. This threshold will only apply if a minimum of five URAAA cases has been submitted. Inevitably, some problems will arise due to data collection / processing errors. Therefore, it is vital that local checks are made regarding the completeness and accuracy of the data submitted to the NVD. Furthermore, it cannot be overemphasised that adverse outcomes do not necessarily imply poor surgical technique or clinical care. Surgeons are but one component of a complex multidisciplinary team and often changes to the organisation and process of care for these patients is required.

UK Carotid Endarterectomy Audit

The Society is embarking on a collaborative project in association with the Healthcare Commission and the Clinical Effectiveness and Evaluation Unit of the Royal College of Physicians, to conduct a national audit of carotid endarterectomy. In addition to the surgical details and outcomes already collected by the NVD, the UK CEA audit endeavours to obtain additional data regarding service provision and organisation, resource issues, delays to treatment and possible geographical variation in the delivery of carotid endarterectomy to patients. This audit is being funded by the Healthcare Commission and specifically dovetails with the NVD such that duplicate surgical data entry is avoided. Data collection for this audit will be online and it is hoped this will pave the way for the remaining index procedures to become web-based.

Registry Group

This group remains active in the follow-up of patients undergoing endovascular aneurysm repair (RETA), thoracic stent grafts and carotid angioplasty/stenting. All patients receiving an endovascular aneurysm stent should be entered into the UK Registry unless they are part of a randomised trial. With recruitment to the EVAR trials completed it is anticipated that submissions to the RETA Registry will increase substantially and funding to support this activity will need to be identified.

Website (www.vascularsociety.org.uk)

This site is under continual development and any comments from members would be helpful.

Patient information

We are gradually updating the information leaflets for patients which are downloadable from the website. It is hoped that these may facilitate the process of consenting patients for procedures.

Contact

Mrs S J A Baker, Vascular Surgical Assistant, Vascular Surgical Unit
Post Point D20-21, Royal Bournemouth Hospital, Castle Lane East, Bournemouth, BH7 7DW

Tel: 01202 303626 ext. 5939
Fax: 01202 704622
Email: sara.baker@rbch.nhs.uk

Training and Education Committee Report

Chairman: Julian Scott

Over the last year the Training and Education Committee has continued to work on a number of projects. Shane MacSweeney has revolutionised the training programme at the Royal College of Surgeons and has to be congratulated for his immense energy and enthusiasm. Frank Smith has been actively involved in the collation of data on training centres throughout the UK and provides an invaluable link with the UEMS Vascular Board. Jon Beard continues to develop his work on competency-based assessment and is now a member of the MMC. Marjorie Allen and Keith Jones have completed the Vascular Trainee Portfolio which will be used for the new training programmes and finally, Professor Cliff Shearman, Dr David Kessel and I have completed the vascular syllabus and the vascular curriculum, which is being incorporated into the web-based curriculum by Dr Ruth McKee (SAC Curriculum co-ordinator).

The Committee has worked with the Royal College of Radiology and the Society for Vascular Technology to develop training recommendations in vascular ultrasound. These can now be incorporated into vascular surgical training (available from the RCR website www.rcr.ac.uk; ISBN 1 905034 02 4). Further support has been received from the BMUS and a list of accredited training programmes will be available on the website.

Through the SAC in General Surgery, Peter Lamont, Cliff Shearman and myself have continued to make representations about the changing face of vascular surgery throughout Great Britain and Ireland. The rapidly changing face of surgery has and will continue to challenge all of us in vascular surgery. As a result a number of *ad hoc* combined surgery and vascular radiology training posts have been approved by the SAC.

In the future it is anticipated that, on completion of the F2 year, potential surgical trainees would enter STY1, comprising either two six-month posts in the generality of surgery or three four-month posts. On completion of these posts, trainees would undergo a series of assessments prior to entry into a six-year seamless training programme. The first two years would be spent in general surgery and we would expect that they gain exposure in abdominal and laparoscopic surgery. In the case of trainees wishing to pursue a vascular training programme, the following four years would be spent acquiring the competencies established in the SAC web-based curriculum, which would include modules in vascular surgery, ultrasound, basic radiology skills and vascular medicine.

During the last year Mike Horrocks, Peter Lamont and I have held joint meetings with the Royal College of Radiology (Dr P Dubbins, Professor A Dixon and Professor A Adam) and the British Society of Interventional Radiology (Professor D Martin and Professor T Watkinson). The aim of these meetings has been to develop a new training programme which addresses the needs of the Royal College of Radiology and the Vascular Society. After much discussion, agreement has been reached that for the future we should aim to develop a new, hybrid vascular specialist competent to undertake both endovascular and surgical treatment of patients with vascular disease. It is envisaged that doctors would be selected from the F2 year into a new post, equivalent to the STY1. This post would include exposure to surgery and basic radiology imaging and intervention, following which the trainee would have the option of entering a training programme in either general/vascular surgery, general/interventional radiology or one of the new hybrid surgery/radiology posts of which there might be up to 20 NTNs derived from the SAC in Surgery and Radiology. Two working groups have been established to produce reports on both the training and the service implications of such a development.

Finally, we come to the increasingly thorny issue of research in surgical training, which has yet to be resolved by the Royal Colleges and the MMC. The production of the Walport Report (see the website www.mmc.nhs.uk) has given some guidance about the future of academic surgery in the UK. The development of MB PhD and F2 academic programmes should produce better trained doctors for a career in academic medicine. It is up to the vascular academic community, the membership and the Council of the Society to get involved and support financially BSc, MB PhD and F2 programmes. Attracting junior doctors into these programmes and supporting them should produce a cohort of competent vascular surgical scientists who would be able to compete at a national level for NTNa posts.

The Masterclass project remains oversubscribed and this year we have redeveloped the programme to have more time in discussion with the "experts".

This year I demit office and wish my successor Professor Cliff Shearman all the very best for the future.

Professional Standards Committee Report

Chairman: Bruce Campbell

Governance of the National Vascular Database

The Society has been advised that it has a duty to take action if results of any contributor to the Database fall outside accepted thresholds. Proposals for action were discussed at the AGM last year and were subsequently ratified by Council after further amendment. The decision was made that the threshold would be the upper 99% confidence interval for mortality after any index operation (like the cardiac surgeons). It was clearly recognised that those who did not contribute to the Database would not be subject to this scrutiny, but it is the aspiration and advice of Council that all surgeons undertaking index procedures should contribute. To that end a letter has been sent to the Chief Executives of all acute Trusts informing them about the Database and about the surgeons in their Trust who are contributors: the letter asks about others who are not. It makes very clear the need for support and help with data entry, which the Society recognises to be problematic for many Members.

This year one surgeon's mortality for elective aortic aneurysm surgery transgressed the 99% confidence interval. The surgeon was informed by the President and a letter was then sent to the Medical Director of his Trust, emphasising that the figures needed to be verified and that there were many possible explanations. The Trust Chief Executive responded very positively to an offer of assistance from the Society, in the form of a visit by the President and myself to prepare a report for the Trust. We reviewed the notes of patients who had died and had an informal interview with the surgeon. A report was prepared for the Trust identifying areas of good practice and of practice which ought to be reviewed. The whole tone of the proceedings was constructive, co-operative and positive. Professor Horrocks and I both felt that this initial experience set an encouraging precedent.

Medico-legal claims

The Society remains interested in receiving information about any medico-legal claims in which Members become involved - either as a result of their own clinical practice or in the role of expert. Our hope to publish comprehensive information about successful (paid) claims relating to treatment of varicose veins has become derailed by a change in the regulations of the NHSLA, which has made it difficult to obtain the details we had originally requested. We intend to make available information gleaned from co-operation with the Medical Defence Union in due course.

Terms of Reference for the Professional Standards Committee

Council has advised that it may be appropriate for the Professional Standards Committee to become involved in advising on aspects of vascular services.

Vascular Tutor's Report

Shane MacSweeney

My aim as Vascular Tutor has been to provide a set of courses covering both conventional vascular techniques and to develop new courses that reflect the future development of the vascular specialist. I have also tried to tailor the courses closely to the level of experience of those attending to make them as relevant as possible. Your feedback has also indicated that you want a wider range of venues and that the cost of courses is an issue.

I am pleased to report that progress has continued on all these issues. Conventional surgical techniques are covered in "Core Skills" and "Advanced Skills 1" (aorta and lower limb) and "Advanced skills 2" (carotid and upper limb). We have courses on endoscopic thoracic sympathectomy, vascular ultrasound, and new courses this year on renal access surgery and an introduction to endovascular interventions (including endovascular AAA repair and peripheral angioplasty and stenting). I hope that there will be something of interest to all of you.

The range of venues has been expanded to include Bournemouth, Bristol, Nottingham, and Newcastle, in addition to London. I am also delighted that B Braun, our major sponsor, has made additional funding available so that we have been able to further reduce the cost of the courses.

I would like to acknowledge and thank the Faculty who give up their time to teach. Without them there would be no courses. I am also grateful to our sponsors including B Braun and Boston Scientific. I would welcome your ideas and feedback and will do my best to act on it. Please contact me by e-mail at shane.macsweeney@virgin.net or write to me c/o Anna Reichel at the Royal College of Surgeons, Lincoln's Inn Fields, London WC2A 3PE.

For further information on the courses available, see the Vascular Society website http://www.vascularsociety.org.uk/ or contact Anna Reichel at the Royal College of Surgeons, telephone 0207 869 6342, e-mail vascular@rcseng.ac.uk.

Chief Executive's Report

Jeanette Robey

The past year has seen the Society run as an incorporated company limited by guarantee and governed by Memorandum and Articles of Association. The Memorandum sets out the objects of the Charity, the powers exercisable by the Council members in pursuit of those objects and the provisions which apply in the event that the charity is dissolved. The Articles of Association contain certain procedural rules which govern the day-to-day operation of the Society. As Chief Executive, my role is to ensure that the Society is run in accordance with both Charity Commission guidelines and Company law and that the Council members (Trustees of the Society and Directors of the Company) are aware that they have a legal responsibility to the charity and should therefore act only in the charity's interests to ensure that the Society's affairs are managed prudently. It is essential that clinical governance is in place for the running of the Society so that it is seen to be working towards public benefit in accordance with its objectives.

As the charity has only two full-time members of staff, a lot of work is done behind the scenes by the Honorary Secretary and Honorary Treasurer on the day-to-day administration of the Society. The financial status of the Society is a key area for the secretariat and we have worked hard this year to establish the Society on a much sounder financial footing. We are continually looking for new ways to maintain expenditure and maximise income. Our increased experience in conference organisation has helped contribute to a steadier income stream than previous years as a result of strict budgetary management. Moreover, organisation of the exhibition in-house has enabled the secretariat to develop a better relationship with industry. This year's exhibition in Bournemouth is one of the biggest for the Society and I would urge members to visit company stands.

In addition to committee and financial administration, and conference organisation, the secretariat supports the work of our affiliated groups. Audley Farrell is responsible for administration of the Society for Vascular Technology and assists with managing its membership database, supporting their Executive meetings and undertaking regular mailings to members. We also liaise closely with both the SVT and SVN on their annual meeting, which is now an established part of the Society's AGM.

The secretariat has also become more involved with the work of the British Vascular Foundation following its merger last year, and we have supported Karen Lody, the BVF's administrator, in developing the fundraising potential of the BVF through various events and publicity. The employment of a full-time fundraiser based in the Society's office can only help to develop this further.

British Vascular Foundation

Keeping people in circulation

The British Vascular Foundation has now become part of the Vascular Society.

After the "marriage" £100,000 was carried forward from the accounts to be used for research purposes. This is clearly a small amount in relation to the needs of vascular research and we must work hard to increase it substantially. A new advisory board has been formed under my Chairmanship and we are currently in the process of inviting non-medical patrons to join the existing medical nucleus. We will try and choose people who will be able to assist us in our quest for funds. Hopefully I will be able to give you a list of names at the next meeting of the Society. If anyone has a contact who might be able to help, please let me know and we can approach them. We intend to appoint a fundraiser who will work with the Board and the patrons to try and raise significant amounts of money from individual donors, charities, industry and local vascular units. Any help you can give to this person when they are appointed would be greatly appreciated. If we are to succeed, it is important that members of the Vascular Society take ownership of the BVF, as this is their charity and will provide funds for their research. This is one of the reasons why the two organisations have been brought together. I have already written to members of the Society asking them to make regular donations and some have responded generously already; I hope many more will. It is only by quoting to non-medical donors the amount of support received from individual society members that one can gain their support. They often ask the question, how much have the doctors put into the research fund? At the moment the answer has to be, not very much. We also hope to encourage local vascular units to raise money by starting patient groups, rather like the diabetic and renal associations who have created very successful fundraising programmes in this way.

I would encourage anybody who has any ideas on how to raise money locally to do so and to advise your board appropriately. Please try and help as much as you can when asked to do so, as your assistance is vital if we are to succeed in raising significant amounts of money in the future. Those of you who are organising events already and those who have done things like run the marathon for the BVF, are already entering into the spirit of being involved and I would like to thank them on behalf of the Society.

I look forward to raising much more money in the future but we can't do so without your help.

Professor Sir Peter Bell
Chairman, BVF Sub-committee

Enquiries

The Vascular Society, 35-43 Lincoln's Inn Fields, London, WC2A 3PE
Tel: 0207 973 0306
Fax: 0207 430 9235
E-mail: office@vascularsociety.org.uk

Fides House, 10 Chertsey Road, Woking, Surrey GU21 5AB
Tel: 01483 726511
Fax: 01483 726522
Email: bvf@care4free.net

Website: www.bvf.org.uk

Society of Vascular Nurses

The Society is now in its second decade and provides a national network for nurses and allied professionals working in vascular care. Established to support nurses in a relatively new and small field of specialist nursing, the Society has become a respected organisation with close links to the Vascular Society, British Vascular Foundation, Venous Forum and other groups aiming to improve the provision of vascular services across Great Britain. The Society's primary role is to allow nurses to access information, education and resources to enable them to develop professionally and thus enhance patient care. The Society also aims to raise the public profile of peripheral vascular disease and to facilitate debate on all issues relating to the prevention and management of the disease. This year our membership totals over 250 and we now have the whole of the British Isles represented. We are also delighted to have members from as far afield as the USA, Canada, South Africa and Australia. The SVN produces a quarterly newsletter available to members - through this, and the website, we endeavour to keep vascular nurses in touch with each other and essential resources.

The Society supports vascular nurses in a number of ways, including the provision of bursaries, four of which are awarded annually to support individual or ward ventures. This year the Society will award the first educational scholarship to a nurse who demonstrates innovation in care through research or new practice. This follows the success two years ago of a travel scholarship which allowed one member to compare practice with our colleagues in Australia. The regional group networks provide a forum for nurses to share experiences beyond their own locality and to provide mutual support for each other. Many groups run half or full study days where members can network and support each other.

The focal point of the SVN calendar is the annual conference held in conjunction with the Vascular Society. The James Purdie prize, donated by the British Vascular Foundation, enables nurses undertaking research and audit to present their work and be formally recognised for their innovation and contribution to vascular care. Inevitably, the social aspect of this event has become almost as important as the formal learning component. This year the Nursing Standard Press, in association with Otsuka Pharmaceuticals, has approached us to work to develop a prestigious new award focusing on PVD and thereby adding to the awareness of the disease. This work will be published and the successful candidate will be presented with the award by the Secretary of State for Health.

Phyllida Morris-Vincent
President

President

Phyl Morris-Vincent
Vascular Nurse Specialist
The Royal Free Hospital, London
Tel: 0207 830 2451
E mail: p.morris-vincent@rfc.ucl.ac.uk

Vice President

Jo Gibson, Vascular Nurse Consultant
Southport District Hospital
Tel: 01704 547471
E mail: jo.gibson@southportandormskirk.nhs.uk

Past President

Jill Arthur, Vascular Nurse Specialist
Torbay Hospital, Torquay
Tel: 01803 655594
E mail: jill.arthur@nhs.net

Treasurer

Jill Bowman
E mail: bowmanjill@hotmail.com

Secretary

Nicky Fenwick, Ward Sister
Northern General Hospital, Sheffield
Tel: 0114 2714603
E mail: nikkifenwick@yahoo.com

Newsletter editors

Jane Gorman, Modern Matron
Mayday Hospital, Croydon
Tel: 0208 401 3283
E mail: jane.gorman@mayday.nhs.net

Sue Ward, Vascular Nurse Specialist
Royal Sussex County Hospital, Brighton
Tel: 01273 696955 ext 6437
E mail: susan.ward@bsuh.nhs.uk

Website: www.svn.org.uk

The Society for Vascular Technology (SVT) of Great Britain and Ireland

The Society for Vascular Technology is now in its 14th year. Over the last few years, and this year has been no exception, we have been working with the Department of Health to ensure the position of Vascular Technology within the Agenda for Change framework and are still actively pursuing our case for State Registration within Healthcare Science. This year has seen a move towards MSc as the registration level for a Clinical Vascular Scientist but this is only a first step in what may be a long process.

The Society serves 329 members and is administered by the Executive Committee assisted by two sub-committees. This year the publication of the final chapter of our Vascular Laboratory Practices completed the work of the Standards and Guidelines Committee which was formally disbanded. We now have six excellent volumes with anatomy and physiology, physics and instrumentation and step-by-step guides on how to perform vascular studies. The Education Committee continues to promote excellent standards of training for Clinical Vascular Scientists and this year we have formed a new sub-committee, the Professional Standards Committee, to keep our members abreast of current issues.

The Society produces a quarterly Newsletter and welcomes feedback from communication on our website. Two study days are held annually and a one day conference incorporating our Annual General Meeting is held alongside the Vascular Society's AGM.

This year the AGM will be taking place in Bournemouth. We have an interesting range of topics from guest speakers and the British Vascular Foundation will again be generously providing a prize for the best proffered paper.

Ann Donald
President

Study Days 2005

'Carotid Controversies' Salisbury, Friday 25th Feb 2005

'Basic Venous Study Day' Northampton, Thursday 15th Sept 2005

Annual General Meeting Bournemouth, Thursday 24th Nov 2005

Executive Committee

President
Ann Donald, ICH & Great Ormond St. Hospital, London
Tel: 0207 813 8223
E-mail: A.Donald@ich.ucl.ac.uk

Vice President
Paul Brannigan, Royal Victoria Hospital, Belfast

Past President
Yvonne Sensier, Leicester Royal Infirmary

Treasurer
Anne Wilcox, Wythenshaw Hospital

Membership Secretary
Rachel Walker, Royal United Hospital, Bath

Conference Secretary
Helena Bodill, Manchester Royal Infirmary

Newsletter Editor
Ian Cadle, Aberdeen Royal Infirmary

Member without portfolio
Claire Flatman, Queens Alexandra Hospital, Portsmouth

Education Committee

Chair
Hayley Handford, Sheffield

Study Days
Richard Pole, Manchester

CPD
Kate Harvey, Cheltenham

Exam registration
Lynne McCrae, Newport

Newsletter questions
Mark Young, London

Practical Exam
Andrew Beech, Nottingham
Sarah-Jane Johnston, Edinburgh
Clare Storry, London
Antonio Sassano, Bristol
Nuala McMahon, Dublin

Professional Standards Committee

Chair - Tim Hartshorne, Leicester
Crispian Oates, Newcastle
Theresa Fail, Portsmouth
Nicola Milburn, London
Pourandokht Khodabakhsh, Leicester

Website: www.svtgbi.org.uk
Email: svt@vascularsociety.org.uk

The Venous Forum of the Royal Society of Medicine

The Royal Society of Medicine

We would like to express our thanks to Linda de Cossart who completed her term of office at the November 2004 meeting and welcome to Richard Corbett, our new President. I would like to personally thank Ian Lane who has also demitted office. Ian did an excellent job as Secretary and leaves the Forum in a very strong position, currently with 190 members.

A very successful symposium on Training and Education in venous disease was held at last year's VSGBI meeting in Harrogate and we were pleased to have speaking, both our President, Linda de Cossart, and the President of the American Venous Forum, Bo Eklof. There was also an opportunity to cover other topics including the role of surgical assistants, training in sclerotherapy, and introducing new techniques into clinical venous practice.

The Spring Annual General Meeting in Brighton, organised by Richard Corbett, was oversubscribed this year. It was an excellent meeting and provided a well balanced combination of research presentations, lectures and an interactive workshop on sclerotherapy. A lively symposium was held on the short saphenous vein and Vaughan Ruckley provided a well researched and delivered review of venous thrombo-embolic disease.

We continue to improve our links with other societies and are pleased to announce a Tripartite meeting which will take place at the RSM on 29th June to 1st July 2006 between the Venous Forum of the RSM, The European Venous Forum, and the American Venous Forum.

Tim Lees
Secretary

Principal Officers

President

Mr Richard Corbett
Tel: 01444 484524
Email: crrcorbett@doctors.org.uk

Secretary

Mr Tim Lees
Tel: 0191 223 1269
Email: Tim.Lees@nuth.nhs.uk

Treasurer

Mr Michael Callam
Tel: 0207 290 2988
Email: mical@dsl.pipex.com

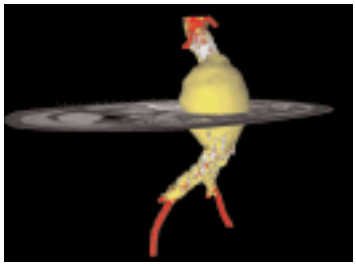
Editor, Phlebology

Mr Alun Davies
Tel: 0208 846 7320
Email: a.h.davies@ic.ac.uk

Academic Administrator

Mrs Christina Feind
Tel: 0207 290 3942
Email: venous@rsm.ac.uk

Endovascular Forum



An extraordinary meeting of the Endovascular Forum was held at the Belfry Hotel, Warwickshire on Thursday 16th and Friday 17th June 2005. The meeting was organised to co-incide with the presentation of the mid-term results of the EVAR 1 and EVAR 2 trials. In addition, there were sessions devoted to ruptures and new technologies, carotid intervention, and thoracic aneurysms, as well as the highly popular disasters sessions.

The meeting was exceptionally well attended and highly successful. The EVAR mid-term results were a surprise to most of the audience, especially EVAR 2. Question time was especially well received and included such panellists as Sir Ian Chalmers (James Lind Library), Dr Peter Littlejohn (NICE), Professor Martin Buxton (Health Economist), Dr Philip Poole Wilson (Chairman DMC), in addition to representatives from the BSIR and VS.

On the following day, the mid-term EVAR results were published in the *Lancet* and marked the end of randomisation to the EVAR trials. Efforts are now underway to secure funding for EVAR procedures to be performed within the NHS environment.

For the next meeting of The Endovascular Forum we will be returning to the Moat House Hotel, Stratford upon Avon. The dates for your diaries are 23rd and 24th June 2006, and due to the popularity of this meeting and limited number of places, early registration is recommended. We look forward to your participation.

Michael Wyatt
Co-chairman

Rob Morgan
Co-chairman

Organising Committee

Mr Mike Wyatt (Joint Chairman)
Mr Mo Adiseshiah
Professor Matt Thompson
Dr Rob Morgan (Joint Chairman)
Dr Nick Chalmers (Treasurer)
Dr David Kessel

Contact details

Miss Jeanette Robey, Chief Executive
The Vascular Society
The Royal College of Surgeons
35-43 Lincoln's Inn Fields
London WC2A 3PE

Tel: 0207 973 0306
Fax: 0207 430 9235
E-mail: office@vascularsociety.org.uk
Website: www.vascularsociety.org.uk

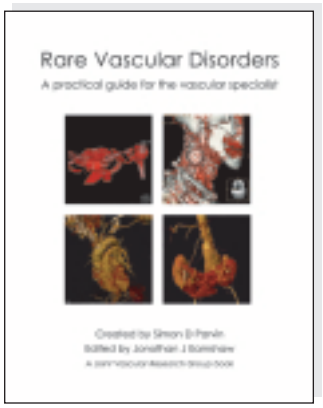
Ruth Moss, Conference Organiser
BSIR
PO Box 2769
Bearsden
Glasgow
G61 4WR

Tel: 0141 942 8104
Fax: 0141 942 8278
Email: ruth.moss@ntlworld.com

The Joint Vascular Research Group

The Joint Vascular Research Group is a collection of surgeons and radiologists who join together to undertake vascular research. Within the Group there has been much debate about our role but we have decided to concentrate on our main strength which is collaborative, mostly observational research. Ongoing projects on short saphenous veins and mesenteric ischaemia have been joined by new studies of acute arm ischaemia, carotid subocclusion and prevention of MRSA infection after major leg amputation.

Conducting research is an increasingly bureaucratic exercise due to difficulties in regulation and governance. We are very lucky to have Christine McGrath as our co-ordinator, since she is conversant with the regulations and paperwork. In the last year the Group has also welcomed a new Treasurer, Mike Clarke from Newcastle. We gratefully acknowledge the efforts of former Treasurer, John Thompson from Exeter, who occupied the role for over a decade.



The Group has also broadened its focus to take on an educational role. Following the success of the series of symposia on Pathways of Care in Vascular Surgery, we recently launched a new symposium on Rare Vascular Disorders at the Belfry, accompanied by a full colour textbook that is recommended to every vascular specialist.

Jonothan Earnshaw
Chairman

Contact

Christine McGrath - Group co-ordinator
29 Sydenham Road, Cotham
Bristol, BS6 5SJ

Tel: 0117 928 3473; Fax: 0117 928 3524
Email: christine@jvrg.freeserve.co.uk

Website: www.jvrg.org.uk

Rouleaux Club

The Rouleaux Club continues to actively represent the views of trainees in vascular surgery. We currently have over 140 members nationally, and as such are the only representative voice for vascular surgical training in Great Britain. We have seats on many committees including the Vascular Society (VS) Council and Association of Surgeons In Training Committee. Membership of the Rouleaux Club is open to Surgical SpRs who have declared an interest in Vascular Surgery.

We are very aware of the desire of trainees to gain training in both open and endovascular procedures and realise that endovascular training is a pre-requisite if vascular surgery is to split away from general surgery in total.

We meet twice a year, in November at the VS AGM, and in June after the Joint Vascular Research Group (JVRG) meeting. Both meetings are sponsored, informal and very enjoyable.

We have fostered very good links with the VS and JVRG, and as such the Rouleaux Club acts as a conduit for exchange of research projects, information and ideas between the most senior and most junior members of our specialty.

We urge all trainees to join us, and to contact us via rouleaux@btinternet.com. A new website will be launched before the Vascular Society AGM.

Martin Claridge
Secretary

Officers of the Club and representatives are elected at the AGM and in 2005 are:

Post	Holder
President	Rob Fisher
Vice-President	Toby Richards
Secretary	Martin Claridge
Webmaster	Martin Claridge
VSGBI Affiliate Rep	Rob Fisher
ASIT Rep	Colin Bicknell
EAVST Rep	Vikas Pandey

Website: www.rouleaux.co.uk

Exhibitors

23rd-25th November 2005
Bournemouth International Centre

Alphabetical list of confirmed exhibitors as at 14th October 2005; number = 55

Company	Stand
Actamed Ltd	55
Advancis Medical	42
Atrium Medical Int	19
B Braun Medical Ltd	6
Bard Ltd	57
Benefoot UK Ltd	28
Boston Scientific	2
Brownes GU	46
BVM Medical	34
Credenhill Ltd	16
Cryolife Europa Ltd	3
Dendrite Clinical Systems Ltd	51
Diomed	32
Edwards Lifesciences	21
Elsevier	27
Ergo	15
Griffiths and Nielsen	26
Huntleigh Healthcare	41
Johnson and Johnson Medical Ltd	67
Jotec GmbH	30
Juzo UK Ltd	47
Karl Storz Endoscopy (UK) Ltd	24
KCI Medical	48
Le Maitre	65
Limbs & Things	38
Lombard Medical	37
Mantis Surgical Ltd	29
Medi UK Ltd	66
Medtronic Vascular Ltd	59
MIUS	54
Otsuka Pharmaceuticals	64
Perimed UK Ltd	43
Pfizer	17
Promed Ltd	7
Pyramed Ltd	33
Sanofi-aventis	39
Siemens	20
Sigvaris Britain Ltd	13
Smith and Nephew Healthcare Ltd	8
Soering Ltd	53
Sonosite Ltd	35
STD Pharmaceuticals	50
TSL plc	36
Tyco Healthcare	18
Vascutek Ltd	1
Viasys Healthcare	12
Wisepress Online Bookshop	63
WL Gore & Associates	4
York Medical Technologies Ltd	45

Other exhibiting companies:

ACST

Cochrane Peripheral Vascular Diseases Group

Gala Trial

tfm Publishing Ltd

The Vascular Society/British Vascular Foundation

UK Carotid Endarterectomy Audit (UKCEAA)

The Society would also like to express their thanks to Edwards Lifesciences for their support of the Renal Access Symposium.

Acknowledgement

The Society would like to thank the following Major Sponsors for their support of this meeting and throughout the year:

B BRAUN SHARING EXPERTISE

Boston Scientific

CryoLife Europa, Ltd.

GORE Creative Technologies Worldwide

VASCUTEK TERUMO

FUTURE ANNUAL MEETINGS

22-24 November 2006	Edinburgh International Conference Centre, Edinburgh
28-30 November 2007	Manchester International Conference Centre, Manchester

B BRAUN
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Boston Scientific

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London
WC2A 3PE

Tel: 0207 973 0306
Fax: 0207 430 9235
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Website: www.vascularsociety.org.uk