Vascular Society of Great Britain and Ireland

The following abstracts are from papers presented at the 46th annual scientific meeting of the Vascular Society of Great Britain and Ireland, held in Edinburgh on 24–26 November 2011. The President of the Society, Mr Peter Lamont, was in the Chair. The BJS Prize was won by S Harrison of Centre for Cardiovascular Genetics, University College, London, and the Sol Cohen (Founder's) Prize was won by RE Clough of NIHR Comprehensive Biomedical Research Centre of Guy's and St Thomas' NHS Foundation Trust and King's College London, London.

Evaluation of the cathepsin gene and abdominal aortic aneurysms

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Objective: There is a large amount of evidence to suggest that abdominal aortic aneurysms (AAA) are part of a genetic disease. Whilst several genes have been implicated, no conclusive evidence of association has been found. We set out to investigate the relationship between the cathepsin gene and AAA.

Method: DNA samples from 932 individuals (466 AAA; 466 controls) were genotyped for 96 variant polymorphisms within cathepsin genes on an illumina Golden Gate assay. The most significant result was used for replication and was further genotyped using Taqman assays on four separate replication cohorts from Belfast, UK (211 AAA; 262 controls), Leeds, UK (214 AAA; 249 controls), Viborg, Denmark (473 AAA; 195 controls) and an independent local cohort (266 AAA; 143 controls).

Results: Sixty-six SNPs passed quality controls and were included in the analysis. Seven SNPs were associated with AAA with p values 0·05 or less in the primary study. The rs217120 SNP within the cathepsin C (CTSC) gene demonstrated the strongest association with AAA (OR 1·55, p = 0·005). This association was maintained after replication in the Belfast cohort (OR 1·93, p = 0·0001), but not in the cohorts from Leeds (OR 1·01, p = 0·742), Viborg (OR 0·77, p = 0·047) or the independent local group (OR 1·07, p = 0·658).

Conclusion: We have identified a biologically plausible candidate gene for AAA, which has shown association in two separate populations. The overall role of this association for all AAA cannot be confirmed since no association was seen in three other populations. This highlights the need for replication in genetic studies.

Normalization of the pro-thrombotic diathesis in patients with abdominal aortic aneurysm (AAA) following endovascular (EVAR) and open aneurysm repair (OAR)

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Objective: AAA is associated with a prothrombotic diathesis that may increase the risk of cardiovascular events. The effect of EVAR and OAR on this prothrombotic diathesis is not fully understood in the medium and long term. The aim of this study is to investigate the long-term effects of EVAR and OAR on this prothrombotic diathesis.

Method: Markers of coagulation (prothrombin fragment [PF] 1+2 and thrombin anti-thrombin [TAT] complex) and markers of fibrinolysis (plasminogen activator inhibitor [PAI] activity and tissue plasminogen activator [t-PA] antigen) were measured in eight age-matched controls (AMC), 29 patients with AAA pre-operatively and at 24 hours, 1, 6 and 12 months after EVAR. Comparison was made between AMC, pre-operative and 12-month postoperative results with 11 patients at 12 months following OAR.

Results: Pre-operatively, PF1+2 was significantly higher in AAA compared to AMC. PF1+2 did not change at 24 hours and 1 month, but decreased significantly at 6 months. At 12 months post-EVAR, PF1+2 was significantly lower than pre-operative values and similar to AMC. There was no significant difference in TAT, PAI and t-PA between AMC and AAA pre-operatively. They increased significantly at 24 hours after EVAR and returned to pre-operative levels at 1 month and remained unchanged over 12 months. Twelve months following OAR, PF1+2 was significantly lower than pre-operative values and similar to AMC. PAI activity was significantly higher than pre-operative levels.

Conclusion: Patients with AAA have a prothrombotic status. Both EVAR and OAR normalize this prothrombotic, hypofibrinolytic diathesis, although there is a tendency for increased fibrinolysis with OAR.

Whole-transcriptome modulation by endovascular aortic aneurysm repair: a novel microarray-based study

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Objective: Microarray technology has produced transcriptional signatures with prognostic value in inflammatory and malignant diseases. We determined transcriptomic profiles of individuals with abdominal aortic aneurysms (AAA) compared to screened controls and explored the effect of EVAR on gene expression.

Method: Blood samples were obtained from 12 males undergoing EVAR (AAA mean 6·25 cm) and 12 age-sex-matched controls (aorta < 2·5 cm). Twelve matched postoperative samples were obtained at a median interval of 9·5 months. High quality RNA was extracted (mean RIN 9·18) and samples hybridised to Illumina HT-12 arrays, each representing 37 846 genes. Results were analysed with GenomeStudio (v1·9).

Results: Gene expression analysis (t-test), inclusive of multiple testing correction, revealed 48 genes to be significantly differentially expressed in AAA against controls ($p < 0.05$). Eleven genes were upregulated, including CASP2 and CARD8, collectively involved in regulating caspase activity, Il-1/β secretion and apoptosis. Thirty-seven genes, including PSMB10 and NT5C, were downregulated and conferred roles in the electron transport chain, humoral response and proteolysis. Postoperatively, 8/11 previously upregulated genes, including CARD8, reversed expression to become downregulated compared to the pre-operative state ($p < 0.05$), whereas 8/37 previously downregulated genes, including PSMB10, became upregulated ($p < 0.05$).

Conclusion: We have demonstrated differential expression of previously undescribed transcripts in AAA with functions involving proteolysis, inflammation and apoptosis. Close to 2/3 (58%) of transcripts were further modulated by EVAR, suggesting the impact of surgery at the transcriptomic level and the latter biological processes. Extended replication of results is warranted to validate transcript roles and prognostic value in AAA.

Toll-like receptor 2 and 6 heterodimerisation contributes to skeletal muscle damage in critical limb ischaemia (CLI)

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Objective: The pathophysiology of skeletal muscle damage in CLI is poorly understood. Toll-like receptors (TLRs) have been implicated in ischaemia-induced tissue damage. TLR 2 in particular has been implicated in critically ischaemic muscle. TLR 2 heterodimerises with TLR 6 under certain stimuli. We hypothesize that TLR 6 expression is upregulated and its signalling pathway activated in ischaemic skeletal muscle, resulting in cytokine-mediated muscle damage.

Method: TLR 2 and 6 expression and distribution in ischaemic and control human muscle biopsies and in C2C12 myotubes cultured in ischaemic conditions were studied using Western blot and immunohistochemistry. Co-immunoprecipitation was used to confirm heterodimerisation of the two receptors. Functional effects of TLR 2 and 6 antagonism on ischaemia-induced IL-6 release and apoptosis were studied in myotubes incubated with neutralizing TLR 2 and 6 antibodies. IL-6 release was assayed by ELISA. Apoptosis was assessed using cleaved caspase-3 and bax/bcl-2 ratio measurements.

Results: TLR 2 and 6 protein expression was significantly upregulated in ischaemic muscle and ischaemic C2C12 myotubes ($p < 0{\cdot}05$). TLR 2 and 6 heterodimerise under ischaemic conditions with consequent activation of the signalling pathway. TLR 2 and 6 antagonism reduced ischaemia-induced IL-6 production and apoptosis.

Conclusion: Upregulation of TLR 2 and 6 expression occurs in ischaemic muscle. Heterodimerisation of TLR 2 and TLR 6 and the subsequent activation of the signalling pathway results in IL-6 release and apoptosis, which contributes to inflammation and muscle damage in CLI. Therefore, TLR 2 and 6 antagonists may be of potential benefit in reducing skeletal muscle damage in CLI.

Encapsulated angiogenic cells: a viable strategy for the treatment of critical limb ischaemia

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Objective: Angiogenic therapy for critical limb ischaemia (CLI) has been hampered by loss of cells from the injection site. Encapsulation of these cells in a polymeric semi-permeable membrane prior to delivery would improve cell retention and allow the outward diffusion of therapeutic factors into ischaemic tissue. We assessed the effect of encapsulation on the function of angiogenic monocytes, candidate cells for therapy in CLI.

Method: Monocytes were isolated using magnetic-assisted-cell-sorting and encapsulated in an alginate polymer (FMC Biopolymer) using a novel bio-electrospraying (BES) technique. The cell/alginate suspension was injected through a 21G needle across an electric field (10 kV, 15nAmp, flow rate $10^{-9}m^3/s$) to form a jet of cell-bearing encapsulated droplets. Cell viability, phenotype (CD14 and CD16 expression) and intracellular signalling of encapsulated and non-encapsulated monocytes were measured using flow cytometry. The angiogenic potential of cells (isolated from three volunteers) in each group was measured using the Matrigel tubule assay.

Results: Monocyte viability was preserved following BES compared with controls (95·1% [range 93·4–97·3] *versus* 95·2% [94·2–96·8], respectively), and there was no change in CD14 and CD16 expression following spraying. Intracellular signalling was also preserved following BES (fold changes, MAPK: 1·5 *versus* 1·6, Akt: 1·2 *versus* 1·1, Erk1/2: 9·6 *versus* 8·9, respectively). Conditioned media from encapsulated and control monocytes were equally angiogenic (tubule length: 342 ± 21 *versus* 321 ± 23; area: 22421 ± 3245 *versus* 21983 ± 2924).

Conclusion: The viability and function of angiogenic monocytes are preserved following encapsulation. This technique may increase the longevity and effectiveness of therapeutic cells when injected into the ischaemic limb.

Nitric oxide bioavailability decreases with severity of peripheral artery disease

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Objective: Platelet activation is increased in patients with peripheral arterial disease and correlates with the severity of disease. Nitric oxide plays an important role in regulating platelet function. We aimed to determine: 1) if endogenous inhibitors of nitric oxide synthase: dimethylarginines (DMA) (asymmetric and symmetric) and L-arginine levels varied with the severity of PAD; and 2) to assess their relationship, if any, with platelet activation.

Method: SDMA, ADMA and L-arginine levels were measured by hydrophilic-interaction liquid chromatography (HILIC)-electrospray tandem mass spectrometry. Platelet p-selectin expression and bound fibrinogen were measured by flow cytometry and platelet aggregation using the rapid platelet function assay with arachidonic acid (AA) and thrombin-related activation peptide (TRAP) as agonists.

Results: 226 patients who had intermittent claudication (IC, n = 148) or severe limb ischaemia (SLI, n = 78) were recruited. Patients with SLI *versus* IC had significantly higher levels of ADMA (median [inter-quartile range] 0·49 μmol/L [0·43–0·56] *versus* 0·42 μmol/L [0·39–0·46], $p < 0{\cdot}001$) and significantly lower levels of L-arginine (54·7 μmol/L [41·9–65·8] *versus* 65 μmol/L [48–79·35], $p = 0{\cdot}03$). SDMA levels were similar. ADMA correlated inversely with ABPI ($r = -0{\cdot}237$, $p = 0{\cdot}001$). Patients with SLI had significantly increased levels of ADP stimulated p-selectin and fibrinogen binding ($p < 0{\cdot}05$) and TRAP stimulated aggregation. TRAP stimulated aggregation directly correlated with ADMA level ($r = 0{\cdot}223$, $p = 0{\cdot}023$).

Conclusion: This is the first study that has shown that the severity of peripheral arterial disease is reflected by increased levels of endogenous nitric oxide synthase inhibitor and decreased L-arginine. This may be implicated in the increased platelet activation observed in these patients.

Macrophage subtypes and 18-fluorodeoxyglucose positron emission tomography (18F-FDG PET) imaging of symptomatic carotid and femoral artery plaques

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Objective: Atherosclerotic plaque rupture is associated with inflammatory cell activity and occurs in, or adjacent to the macrophage-rich area. FDG PET uptake has been shown to occur within macrophages. We aimed to compare FDG PET uptake in patients with symptomatic carotid disease to that in patients with symptomatic femoral disease and how this relates to macrophage subtypes.

Method: Patients presenting with either symptomatic significant carotid artery stenosis (n = 29) or femoral disease (n = 29) were recruited. A dynamic 18F-FDG PET and co-registered computed tomography angiography scan were performed prior to surgery. Dual staining for macrophages (CD68) and the M1 pro-inflammatory markers, iNOS, MHC class II and SOCS3, or the M2 anti-inflammatory markers, dectin-1, SOCS1 and CD163, was performed.

Results: Carotid artery plaques had greater numbers per plaque area of macrophages (median [IQR] 39·76 [34·2–49·96] *versus* 13·20 [7·8–21·13] counts per mm^2; Kruskal Wallis test $p < {\cdot}001$). The proportion displaying M1-macrophage activation markers, iNOS, MHC class II and SOCS3, was significantly increased in the carotid compared to femoral plaques ($p < {\cdot}001$). Femoral plaques displayed a greater proportion of M2-macrophage markers, dectin-1, SOCS1 and CD163 ($p < {\cdot}001$). The maximum metabolic rate calculated from the dynamic 18F-FDG PET uptake was similar for both

carotid and femoral plaques and did not correlate with maximal CD68 counts or macrophage subtypes.
Conclusion: Carotid plaques from recently symptomatic patients exhibit significantly more plaque destabilising, M1-macrophages, whereas femoral plaques have a predominance of anti-inflammatory, tissue reparative M2-cells. Despite this, there was no quantifiable increase in PET uptake in the carotid compared to femoral plaques. The ability of 18F-FDG PET uptake to identify the unstable plaque appears to be limited.

Identifying the unstable plaque in the clinic – a new model

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Objective: The aim of this study was to use pre-operative clinical data and B-mode duplex imaging to identify patients at high risk of having an unstable carotid atherosclerotic plaque, defined using well validated histological criteria.
Method: Patients undergoing carotid endarterectomy (CEA) in our unit over a 2-year period were recruited prospectively into the study after giving informed consent. Plaques harvested during CEA were histologically graded by two independent histopathologists using nine criteria based on a modified AHA scoring model to give a final 'stable' or 'unstable' classification. Independent clinical and imaging variables that significantly affected outcome were entered into a logistic regression analysis to create a model for predicting unstable plaques.
Results: 197 histological samples were analysed and graded as stable, n = 66 (33%), and unstable, n = 131 (67%). Features found to be significantly related to instability included symptoms within 2 weeks (OR 2·28; 95% CI, 1·01–5·142; p = 0·04), GSM < 25 (OR 2·942; 95% CI, 1·004–8·62; p = 0·02), plaque area > 80 mm^2 (OR 2·74; 95% CI, 1·16–6·46; p = 0·02). Logistic regression modelling determined that patients with all of the above features present had a 92% chance of having an unstable plaque, compared to only a 42% chance if none of these features were present.
Conclusion: This study identifies simple clinical and imaging criteria that can predict an unstable plaque in patients with carotid artery disease. This model once validated can be used by clinicians in managing patients with carotid artery stenosis, particularly in identifying patients for urgent intervention.

Critical limb ischaemia promotes an angiogenic drive in the circulation

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Objective: Circulating monocytes ($CD14^+$) consist of at least two distinct subsets: inflammatory/phagocytic ($CD14^+/CD16^-$); and patrolling/remodeling ($CD14^{low}/CD16^+$). In tumours, $CD14^{low}/CD16^+$ monocytes orchestrate blood vessel growth in the presence of vascular endothelial growth factor (VEGF) and angiopoietin-2 (Ang-2). We examined whether an angiogenic drive is associated with changes in these monocyte populations in patients with critical limb ischaemia (CLI).
Method: Circulating monocytes were phenotyped (CD14 and CD16 expression) using flow cytometry in patients with CLI (n = 30) and age-matched/young controls (n = 15/group) before and after surgical revascularisation/amputation. VEGF and Ang-2 levels were measured in the serum from CLI patients and controls (n = 10/group) by ELISA, while global angiogenic activity was assessed using the Matrigel assay (n = 5/group). Intracellular proangiogenic signalling (Erk1/2 and Akt phosphorylation) was measured in monocytes using flow cytometry.
Results: Patients with CLI had > 2-fold more $CD14^{low}/CD16^+$ monocytes compared with matched and young controls ($12 \pm 3\%$ *versus* $4 \pm 1\%$ *versus* $5 \pm 2\%$, respectively, $p < 0{\cdot}05$), but similar numbers of $CD14^+/CD16^-$ monocytes ($78 \pm 6\%$ *versus* $82 \pm 4\%$ *versus* $86 \pm 5\%$, respectively, $p > 0{\cdot}05$). Revascularisation/amputation reduced the numbers of $CD14^{low}/CD16^+$ monocytes ($8 \pm 2\%$) to levels not significantly different from controls. Circulating levels of VEGF and Ang-2 were higher in CLI patients than controls by 5- and 2-fold, respectively ($p < 0{\cdot}05$). CLI serum induced greater angiogenesis than controls ($p < 0{\cdot}01$). Stimulation of $CD14^{low}/CD16^+$ (but not $CD14^+/CD16^-$) monocytes induced phosphorylation of angiogenic intracellular signalling pathways, Erk1/2 and Akt.
Conclusion: CLI promotes an angiogenic drive in the circulation that is associated with a rise in $CD14^{low}/CD16^+$ monocytes. Manipulating this endogenous response may be a novel therapeutic strategy to revascularise critically ischaemic limbs.

Platelet, endothelial and coagulation factors and the patency of arteriovenous fistulae: prospective analysis

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Objective: Measurement of cardiovascular biomarkers is increasingly used in cardiovascular risk stratification to guide therapy. It is unknown if similar markers can help identify patients who are at increased risk of native arteriovenous fistula (AVF) occlusion. We prospectively assessed the relationship, if any, between platelet, endothelial and coagulation markers, and patency rates of AVF in haemodialysis (HD) patients.
Method: Blood samples were taken from the AVF immediately before HD. Platelet function was assessed by: 1) Ultegra rapid platelet function assay using the agonists thrombin receptor activating peptide (TRAP) and arachidonic acid (ASA); 2) flow cytometry P-selectin expression and fibrinogen binding; and 3) plasma soluble P-selectin, soluble CD40. Coagulation and fibrinolysis were assessed by ELISA determination of thrombin-antithrombin (TAT) and D-dimer. Correlation and Cox regression analyses were performed.
Results: Forty-three patients were studied with a median follow-up of 24 months (range 4–52). Primary patency was 77% at 1 year, with 35 at risk. Twenty patients had interventions to maintain primary patency during follow-up. Secondary patency was 59% at the end of follow-up, with 22 at risk (15 dead, 6 transplanted). Higher levels of TAT correlated to shorter primary patency (Spearman's correlation co-efficient $r = -{\cdot}564$; $p < 0{\cdot}001$). No other variables were significant.
Conclusion: TAT reflects activated coagulation and may be a sensitive biomarker of an impending AVF occlusion. Further studies are required to determine if active surveillance or anticoagulant therapy may be beneficial in patients with high TAT levels.

The effect of anticoagulation therapy on the incidence of endoleak or aneurysm sac size after endovascular aneurysm repair

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Objective: The effects of anticoagulation therapy on the incidence of endoleak and aneurysm sac size after endovascular aneurysm repair (EVAR) are unclear. This study determined whether postoperative anticoagulation affected the incidence of endoleaks or aneurysm sac size.
Method: Case notes were available for 373 patients (333 men; mean age 78 years) who underwent elective EVAR between September 1997 and July 2010. Postoperative follow-up was with ultrasound scans and abdominal X-rays at 3–6-monthly intervals.
Results: The mean follow-up was 27 months. There were 40 (10·7%) patients on warfarin (WA), 250 (67%) patients on single antiplatelet (SA) therapy

(aspirin, clopidogrel or dipyridamole), 11 (2·9%) patients on dual antiplatelet (DA) therapy and 72 (19·3%) patients on no anticoagulation (NA). During the study period, 69 (18·5%) endoleaks were documented. There was no significant difference in the incidence of all endoleaks (p = 0·49, X2 for trends; 10·0% WA, 19·6% SA, 18·2% DA, and 19·4% NA), type I endoleak (p = 0·89, X2 for trends; 2·5% WA, 3·2% SA, 0·0% DA, and 1·4% NA) or sac expansions (p = 0·29, X2 for trends; 7·5% WA, 6·8% SA, 9·1% DA, and 13·9% NA). There was also no significant difference in the period of time from surgery (in months) to endoleak (p = 0·78, unpaired ANOVA; WA 15·0 (1–24), SA 12·4 (1–48), DA 6·0 (6) and NA 15·0 (1–42).

Conclusion: Anticoagulation with warfarin or antiplatelet agents was not associated with an increase in the incidence of postoperative endoleaks or aneurysm sac expansion after EVAR. These data support the safe use of anticoagulant medications in patients undergoing EVAR.

Plasminogen activator receptor cleavage: an important mechanism in ulcer healing

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Objective: Plasminogen activation may play a critical role in venous leg ulcer healing. Urokinase plasminogen activator receptor (uPAR) has three domains (DI; DII; DIII) that can be cleaved from the cell membrane to yield a soluble fragment (suPAR I-III). Cleavage also occurs within the receptor to produce suPAR-I and II-III fragments. We have compared the levels of these chemotactic and mitogenic fragments in healing and non-healing ulcers to determine whether they are associated with healing.

Method: Patients with venous leg ulcers (treated with compression dressings) were recruited from a dedicated clinic and prospectively followed for healing, defined as re-epithelialisation of the ulcer within 6 months. Immunoassays were validated and used to quantify suPAR fragments in exudates obtained from under Opsite® dressings (levels expressed as fmol/mg soluble protein). The effect of exudates on human keratinocyte migration was measured by scratch assay.

Results: Exudates were collected from 30 patients (median age 68 years; 14 males; 9 healers). Healers had higher levels of suPAR I-III (19 ± 5 *versus* 6 ± 1; $p < 0{\cdot}005$), suPAR I (144 ± 70 *versus* 70 ± 12; $p < 0{\cdot}05$) and suPAR II-III (138 ± 19 *versus* 50 ± 6; $p < 0{\cdot}0001$) fragments. Scratch colonization was greater following treatment with healing ulcer exudates ($p < 0{\cdot}05$). SuPAR depletion in exudates from healers and non-healers resulted in cell death.

Conclusion: This is the first study to show the presence of suPAR in venous ulcer exudates. SuPAR fragments are associated with improved ulcer and *in vitro* wound healing. Maintenance of suPAR levels prevented cell death and is necessary for optimal wound healing. Differences in suPAR fragment levels may identify non-healers that would benefit from early skin grafting.

National Vascular Database analysis: the relationship between AAA repair volume and outcome

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Objective: Current evidence suggests that AAA repair mortality improves with increasing workload volume. However, data are lacking for the effect of case mix and repair type on this relationship and for the effect of case volume on complications. This analysis examines the effect of case volume on AAA repair outcomes after adjustment for confounders.

Method: Between January 2008 and December 2010, 13,068 elective AAA repairs were registered with the NVD. The number of open (OAR) and endovascular (EVAR) procedures per surgeon and per unit were extrapolated and categorised incrementally. Logistic regression analysis of risk of death or any complications was performed, adjusting for gender, ASA and screening status.

Results: For OAR mortality, surgeon volume of 11–20 cases/year and unit volume of 41–50 cases/year had a reduced risk (OR 0·70 [95% CI, 0·92–0·52]; p = 0·013) and (0·57 [0·87–0·38]; p = 0·008), respectively. For OAR complications, surgeon volume of > 30 cases/year was associated with a reduced risk (0·54 [0·32–0·89]; p = 0·014). Unit volume was not associated with a reduced complications risk at any level. For EVAR mortality, neither surgeon nor unit volume was associated with risk reduction at any level. For EVAR complications, surgeon volume of 21–30 cases/year showed a reduced risk (0·75 [0·93–0·60]; p = 0·009). Unit volume did not reduce the complications risk at any level. Unit volume of > 32 cases/year was not associated with risk reduction in any category regardless of repair type.

Conclusion: This analysis demonstrates that the relationship between AAA repair volume and outcome is not linear. Adjusting for case mix, AAA repair volume is more relevant to OAR than EVAR and to surgeon than unit outcomes.

Treatment of abdominal aortic aneurysm in nine countries 2005–2009 – a Vascunet report

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Objective: To study contemporary treatment and outcome of abdominal aortic aneurysm (AAA) repair in nine countries.

Method: Data on primary AAA repairs from 2005–2009 were amalgamated from national and regional vascular registries in Australia, Denmark, Finland, Hungary, Italy, Norway, Sweden, Switzerland and the UK. Primary outcome was in-hospital or 30-day mortality. Multivariate logistic regression was used to assess case-mix.

Results: 31,427 intact AAA repairs were identified, mean age 72·6 years (95% CI, 72·5–72·7). The rate of octogenarians and use of endovascular repair (EVAR) increased over time (p < 0·001). EVAR varied between countries from 14·7% (Finland) to 56·0% (Australia). Overall peri-operative mortality after intact AAA repair was 2·8% (2·6–3·0) and was stable over time. The peri-operative mortality rate varied from 1·6% (1·3–1·8) in Italy to 4·1% (2·4–7·0) in Finland. Increasing age, open repair and presence of comorbidities were associated with outcome. 7040 ruptured AAA repairs were identified, mean age 73·8 (73·6–74·0). The overall peri-operative mortality was 31·6% (30·6–32·8), and decreased over time (p = 0·004).

Conclusion: The rate of AAA repair in octogenarians as well as EVAR increased over time. Peri-operative outcome after intact AAA repair was stable over time, but improved after ruptured repair. Geographical differences in the treatment of AAA remain.

National Vascular Database analysis: independent pre-operative predictors of abdominal aortic aneurysm repair outcomes

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Objective: The National Vascular Database for AAA procedures has a large number of data fields. The aim of this analysis is to identify data fields that are most relevant to AAA repair outcomes.

Method: Between January 2008 and December 2010, data for 13,068 elective AAA repairs were entered in 379 NVD data fields. Of these fields, 96 were pre-operative for both open (OAR) and endovascular (EVAR) repair. Sixteen additional fields were EVAR-specific. Logistic regression analysis for the odds of death and for any complications was performed adjusting for gender, ASA and screening status.
Results: Of the combined data fields, 38 showed an independent association with operative mortality or any complications. For the EVAR-specific variables, an additional seven fields showed such association. Apart from conventional outcome predictors, some associations were novel. Examples include an increased risk of EVAR mortality for transferred patients (OR 4·12 [95% CI, 2·77–6·15]; p = 0·000), a reduced risk of mortality in patients on antiplatelets undergoing EVAR (0·57 [0·40–0·81]; p = 0·029), but an increased risk of complications in OAR (1·16 [1·01–1·35]; p = 0·049). History of cardiac disease was associated with an increased mortality risk for OAR (1·52 [1·15–2·02]; p = 0·002), but not for EVAR, whereas atrial fibrillation was associated with an increased mortality risk for EVAR (2·74 [1·06–7·05]; p = 0·029), but not OAR.
Conclusion: Of the current NVD pre-operative data fields, 40% correlate independently with AAA repair outcomes. Some of these correlations are novel and have the potential for improving pre-operative risk management and stratification for AAA surgery. Whilst it may benefit from refining its fields, the NVD remains unique and invaluable.

Long-term impact of the volume-outcome relationship in elective abdominal aortic aneurysm repair

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Objective: Robust, risk-adjusted analyses have demonstrated that a reduction in peri-operative mortality is associated with the repair of abdominal aortic aneurysms (AAA) in centres with high operative caseload (volume). However, the long-term impact of this volume-related effect on mortality remains unknown.
Method: Demographic and clinical data were extracted from UK Hospital Episodes Statistics for patients undergoing elective repair of infrarenal AAA from 1 April 2000 to 31 March 2005. The long-term mortality of this cohort of patients was investigated through linkage to the UK Office of National Statistics (ONS). Risk-adjusted survival was analysed using Cox's Proportional Hazards Modelling to identify the effect of hospital volume on long-term mortality. To isolate the effect of postoperative medical care, data were re-modelled after exclusion of 30-day mortality and after exclusion of in-hospital mortality.
Results: 14,396 patients with a mean age of 72 years, of whom 85·7% were male, underwent elective repair of infrarenal AAA in England and were linked to follow-up using ONS statistics. Risk-adjusted analysis of all-cause mortality by Cox's Proportional Hazards Modelling demonstrated a significant effect of hospital volume across all quintiles at up to 2 years (p = 0·013). Remodelling of the data after excluding 30-day mortality demonstrated a late significant effect of hospital volume.
Conclusion: The present study provided the first evidence that there is a long-term benefit to patients undergoing the elective repair of AAA in high-volume hospitals. Both a surgical and medical effect was observed.

Putting TIA/stroke in the FAST lane: but not if you present with leg weakness or visual loss

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Objective: The Department of Health's 'FAST' campaign stresses the importance of seeking immediate medical help should anyone suffer: Facial weakness, Arm weakness or Speech problems, and awareness about the need to report symptoms as soon as possible is fundamental if the campaign to offer carotid interventions in < 14 days (or 48 hours) is to be achieved. However, despite national media campaigns, just how aware are the public about stroke symptoms?
Method: A 'face to face' survey of 1300 members of the public was conducted across all age groups and ethnic backgrounds.
Results: 1248 (96%) had heard of stroke, but only 880 (68%) knew that stroke involved the brain. 907 (70%) had heard of the FAST campaign, but a larger proportion (1006 [80%]) recalled seeing the image of the 'burning head'. The vast majority were aware of the FAST symptoms (facial weakness 89%, arm weakness 83%, speech problems 91%). However, only 738 (57%) considered leg weakness to be a symptom of stroke and only 570 (44%) knew that visual loss was a stroke symptom. A similar proportion thought that headache (53%) and arm/leg pain (51%) were stroke symptoms. These findings were consistently replicated across all age groups and ethnic backgrounds.
Conclusion: While the public appear aware of FAST and its warning symptoms, the failure to include leg weakness and visual loss within the media campaign has meant that a significant proportion of the public may not recognise these as important symptoms which require urgent assessment.

Triaging TIA/minor stroke patients using the ABCD2 score does not predict those with significant carotid disease

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Objective: 'Rapid access' TIA clinics often use the ABCD2 score to triage patients. Those scoring 0–3 are seen < 7 days, while patients scoring 4–7 are seen as soon as possible (preferably < 24 hours). It was hypothesized that patients scoring 4–7 would have a higher yield of significant carotid disease.
Method: A prospective study of correlation was conducted between GP and stroke physician-measured ABCD2 score and prevalence of > 50% carotid stenosis.
Results: Between 1·10·2008 and 31·04·2011, 2452 patients were referred to the Rapid Access Service. After stroke physician review, 785 (32%) were thought to have suffered a carotid territory stroke/TIA, all had ABCD2 scores measured by both the referring GP and a stroke physician and all underwent duplex ultrasound imaging within the Rapid Access Clinic. A GP ABCD2 score of 0–3 was associated with a 16·3% prevalence of > 50% stenosis (46/283), compared with 14·3% for a physician ABCD2 score of 0–3 (35/245). A GP ABCD2 score of 4–7 was associated with an 11·4% prevalence of > 50% stenosis (57/502), compared with 12·6% for a physician ABCD2 score of 4–7 (68/540). Analyses of the area under the receiver operating characteristic curve (AUC) for referrer and stroke specialist ABCD2 scores, showed no prediction of carotid stenosis (GP: AUC 0·50 [95% CI, 0·44–0·55; p = 0·9]; Specialist: AUC 0·51 [95% CI, 0·45–0·57; p = 0·78]).
Conclusion: The ABCD2 score cannot identify TIA/minor stroke patients with a higher prevalence of clinically important carotid disease. In particular, a higher ABCD2 score was associated with a lower yield of significant carotid disease.

Can we measure carotid plaque volume and does it matter?

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Objective: Stroke is the third leading cause of death and the leading cause of disability with 150,000 sufferers/year in the UK. Carotid disease causes 30% of ischaemic strokes, probably due to embolism of atherosclerotic material. Severity of stenosis measured by duplex is currently the indication for surgery, but is a poor predictor of stroke: asymptomatic carotid stenosis > 70% predicts an annual stroke risk of only 2·5%. As stenoses may merely be a surrogate for carotid plaque volume (CPV), we investigated the relationship between CPV and symptoms in carotid disease.
Method: Plaque volume index on pre-operative duplex was compared with a precise measure of the operative specimen using an Archimedes suspension method in 30 patients undergoing carotid endarterectomy. A detailed history on risk factors and cerebral symptoms was recorded.
Results: Plaque volume index underestimated CPV by a mean (±sd) of $0{\cdot}41 \pm 0{\cdot}49\ cm^3$ with a poor correlation of r = 0·6 (p < 0·05). Mean CPV for stroke, TIA and asymptomatic patients were $1{\cdot}53 \pm 0{\cdot}80$, $1{\cdot}11 \pm 0{\cdot}42$ and $0{\cdot}60 \pm 0{\cdot}20\ cm^3$, respectively, with the 21 symptomatic patients having

significantly larger CPV at $1{\cdot}24 \pm 0{\cdot}61$ cm^3 than that of $0{\cdot}67 \pm 0{\cdot}18$ cm^3 in the nine asymptomatic patients ($p = 0{\cdot}011$).

Conclusion: CPV can be measured accurately using an Archimedes suspension method and was strongly associated with symptoms in patients undergoing carotid surgery. As all our patients had > 70% carotid stenoses, CPV clearly has greater potential to predict symptom status. If 3-D imaging measures CPV accurately, the indication for carotid surgery may need to be reconsidered.

Early carotid endarterectomy (CEA) for a symptomatic carotid stenosis (SCS) is associated with a higher adverse event rate. Data from symptomatic patients participating in the GALA Trial

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Objective: The Oxford Vascular Study showed high stroke rates within 7 and 28 days of the presenting neurological symptom (PNS) in SCS patients (TIA: 8·0% and 11·5%; minor stroke: 11·5% and 15·0%). Thus, current guidelines recommend early surgery. Although a recent systematic review allays concerns about the safety of early surgery, evidence is conflicting and this has been re-assessed in 2164 symptomatic patients from the GALA Trial (general [GA] *versus* loco-regional [LA] anaesthesia for CEA).

Method: Thirty-day outcomes (stroke, death, MI) were analysed for early (ES: < 14 days from PNS, n = 317) or later (LS: > 14 days, n = 1847) surgery. The influence of anaesthetic type, age, sex, PNS (ocular, TIA, stroke), and contralateral carotid occlusion were also examined.

Results: 18·4% of TIA patients and 16·5% with ocular symptoms had ES *versus* 10·1% after stroke ($p < 0{\cdot}005$, Chi-squared). ES had a higher risk of an adverse outcome (8·2% *versus* 5·1%; OR 1·69; 95% CI, 1·07–2·66; $p = 0{\cdot}025$) that was not influenced by other factors examined (logistic regression analysis). For ES the risk of an adverse outcome (GA *versus* LA) was: men: 11·1% *versus* 7·6% (OR 1·46; 95% CI, 0·94–2·25; $p = 0{\cdot}09$); women: 4·8% *versus* 4·7% (OR 0·64; 95% CI, 0·31–1·31; $p = 0{\cdot}22$).

Conclusion: These data suggest that whilst ES is probably beneficial, strategies to improve safety are required. This might include early ICA clamping or pharmacological stabilisation of carotid plaques. Further studies are required to confirm ES safety and reporting the time from PNS to surgery should be mandatory in all CEA studies.

The war against error: a 15-year experience of completion angioscopy following carotid endarterectomy

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Objective: In an earlier audit, a policy of intra-operative transcranial Doppler (TCD) and completion angioscopy was associated with virtual abolition of intra-operative stroke (apparent upon recovery from anaesthesia) following carotid endarterectomy (CEA). The aims of this study were to determine whether the prevalence of technical error diminished with experience and whether our monitoring/quality control policy was still associated with low rates of intra-operative stroke 20 years after its introduction.

Method: A retrospective review was conducted of four consecutive cohorts of 400 patients undergoing CEA between October 1995 and March 2010 (1600 CEAs in total).

Results: 104 patients (7%) had thrombus removed following angioscopy and prior to flow restoration, while 31 (2·1%) underwent repair of a distal intimal flap. The prevalence of intimal flaps diminished from 4·9% in the first 400 patients to 0·8% in the last 400 patients ($p = 0{\cdot}006$). By contrast, the prevalence of retained thrombus did not decline with experience (8·5%, 3·7%, 10·3% and 5·4% for the four consecutive periods). Intra-operative TCD and completion angioscopy was, however, associated with extremely low rates of intra-operative stroke (0·25%, 0·25%, 0·5% and 0·25% during the four study periods).

Conclusion: Most intra-operative strokes probably follow embolisation of thrombus following restoration of flow. This can be prevented by angioscopy which has the advantage of being performed prior to flow restoration. Increasing experience was associated with a decline in the detection of intimal flaps, but not in the prevalence of retained thrombus. Even the most experienced of surgeons can still be responsible for inadvertent technical error.

The cost utility of a multidisciplinary foot protection clinic (MDFPC) in an Irish university hospital setting

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Objective: Diabetes is a growing epidemic and diabetic foot complications place a significant social, psychological and economic strain on patients and the Health Service. The lifetime incidence of foot ulceration in diabetics has been estimated as high as 25%, and up to 85% of diabetic lower extremity amputations are preceded by ulceration and diabetic foot infection. Many studies have proposed dedicated diabetic foot teams as the mainstay of diabetic foot care. We aimed to quantify the cost benefit and sustainability of a MDFPC in an Irish university hospital setting.

Method: A dedicated bi-weekly consultant-led multidisciplinary foot protection clinic (MDFPC) involving vascular surgery, endocrinology, orthopaedic surgery, podiatry, orthotics and tissue viability was established in June 2008.

Results: Between 2006 and 2010, a total of 221 lower limb procedures (major/minor amputations and debridement) were carried out. The number of major amputations decreased from 12 during the control period (2 years before the clinic) to 7 in the study period (2 years after the clinic). After costing all activity associated with the clinic, there was an overall saving of € 114, 063 per year associated with the introduction of the MDFPC.

Conclusion: This is the first study in an Irish context, and one of few international studies to demonstrate that an aggressive coordinated approach to diabetic foot care is both cost effective and clinically efficient in reducing the burden of foot-related complications in a diabetic population.

An integrated foot team improves outcomes in diabetics

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Objective: Diabetic amputations account for two thirds of all major amputations in the UK with a two-fold variation in amputation rates nationally. Two-year mortality post- amputation is 50%. Foot complications are the single largest cause of diabetic hospital stay (average 22 days), costing £200 million in England. Based in a general hospital in an area of very high deprivation and diabetes prevalence (9·7%), we established a multidisciplinary specialist foot team providing a Monday-Friday rapid access service. By locating a primary care podiatry clinic in our diabetes centre, primary care patients can get immediate access to a diabetologist. Aggressive medical management of osteomyelitis is combined with integrated working with vascular surgery and radiology. A rapid response outreach team initiates and continues treatment across primary and secondary care according to the patient's needs. We evaluated the impact of the new service on outcomes.

Method: We audited activity of our specialist foot team between October 2009-September 2010. Amputation rates were obtained from the York & Humber Public Health Observatory.

Results: Prevented admissions: 81; bed days saved: 567; facilitated early hospital discharges: 20; bed days saved: 100. The total cost saving was (£350/ day): £198,450 +£35,000 = £233,450. The amputation rate was 1·1 per 1000, c.f average 1·7 for neighbouring PCTs and 2·5 nationally.

Conclusion: Our integrated foot team ensures rapid, coordinated access to vascular input with amputation rates half the national average. Most acute foot complications are safely managed in the community with home parenteral

antibiotics and community/outpatient review within 24 hours, reducing hospital admissions and the risk of hospital-acquired complications.

Distal bypass outcome in diabetic *versus* non-diabetic patients – the multidisciplinary team and the diabetic foot clinic impact

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Objective: To assess the impact of a multidisciplinary team (MDT) and diabetic foot clinic (DFC) on the outcome of distal bypass surgery in patients with critical leg ischaemia (CLI).
Method: Patients with CLI undergoing distal bypass were included. Diabetic patients were managed by dedicated MDT and DFC care. The non-diabetic patients were cared for by a vascular surgeon and vascular nurse specialist and followed up in a regular vascular clinic. Patency, major amputation, mortality rates and amputation-free survival were analyzed.
Results: Between 2004 and 2011, 210 consecutive patients underwent 231 distal bypasses (157 men; median age: 76 years, range: 20–96 years). 149/231 (65%) were diabetic *versus* 82/231 (35%) who were non-diabetic. Chronic renal failure was significantly higher in the diabetic group (p = 0·0062). At 1 year the primary and secondary patency rates in the diabetic group were 74·8% and 89·9% *versus* 61·8% and 85·9% in the non-diabetic group, respectively. The major amputation and mortality rates in the diabetic group were 6·5% and 11·9% and in the non-diabetic group were 14·8% and 6·6%, respectively (p = 0·0026 and p = 0·1491). The amputation-free survival rates at 12 and 48 months were 82·4% and 46·3% for the diabetic group *versus* 84·1% and 49·7% in the non-diabetic group (p = 0·4084).
Conclusion: Diabetic patients had a significantly better major amputation rate compared to non-diabetic patients in spite of increased morbidity. Non-diabetic patients should be integrated in the same MDT and DFC to improve their major amputation rate.

The impact of the angiosome principle on foot ulcer healing in distal bypass surgery

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Objective: To evaluate the time to healing of tissue loss following foot revascularisation in patients undergoing distal bypass surgery and the impact of the angiosome principle.
Method: Patients undergoing distal bypass for foot tissue loss (Rutherford 4 and 5) were divided into two groups where the angiosome principle of revascularisation was applied and not applied. The site of foot tissue loss, the perfused angiosome artery, the time to healing and patency rate were compared in the two groups.
Results: Ninety-nine consecutive patients (71 men, median age 76) underwent 108 distal bypasses. The incidence of diabetes mellitus, renal failure and ischaemic heart disease was 82%, 31% and 48%, respectively. The presenting symptom was ischaemic ulcers in 68 (63%) cases and gangrene in 40 (37%). The site of tissue loss was along the posterior tibial angiosome in 7 (6%), medial plantar in 44 (42%), lateral plantar in 30 (28%), anterior tibial in 12 (11%) and peroneal in 3 (3%); another 12 (11%) were in two angiosome territories. Out of 108 bypasses, 48 (44%), the perfused angiosome artery matched that of the foot tissue loss; in 60 (66%) cases it did not. In the group with direct angiosome perfusion, 85% of the ulcers healed, median healing time was 76 days (range 11–647) *versus* 83% in the non-direct group, median 108 days (range 24–582). At 1 year, the primary, secondary patency and the amputation-free survival rates were 60%, 91% and 81% in the direct group *versus* 68%, 93% and 83% in the non-direct group, respectively.
Conclusion: There was no difference in healing time or outcome between these two patient groups. The angiosome principle of revascularisation is not supported in this study.

A randomised controlled trial to evaluate different treatment regimes with topical wound oxygen (TWO2) on chronic wounds

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Objective: Chronic wounds of the lower leg and foot are frequent, difficult to treat and show high rates of complications. After positive results with a pressurized topical oxygen therapy (TWO2) in other studies we were interested in evaluating whether 4 weeks of TWO2 treatment would show similar results to those after 12 weeks of treatment.
Method: This randomised, controlled study was conducted at the National Wound Institute in Santiago de Chile. In an outpatient setting with patients with severe diabetic foot ulcers (DFU) (n = 20) and chronic venous ulcers (CVU) (n = 20), all patients received TWO2 for a period of 1 month. The groups were then randomised to continue with TWO2 (TWO-TWO group) or receive AMWT (advanced moist wound therapy) for 2 more months (TWO-AMWT group). TWO2 patients were treated daily for 2 hours five times a week. The device delivered humidified medical grade oxygen with pressure cycles between 5 and 50 mbar. Dressing changes in the control group were performed according to best practice at a minimum of twice a week. The primary endpoint was complete ulcer closure after 12 weeks.
Results: 90% of the DFU patients in the TWO-TWO group healed within 12 weeks *versus* 40% in the TWO-AMWT group. Patients with CVU had 50% healing *versus* 30%, respectively.
Conclusion: Patients with complicated ulcers benefit from the treatment of topical localized oxygen (TWO2). Continuous TWO treatments for 12 weeks showed significant better outcomes than a shorter TWO2 treatment regime of 4 weeks followed by AMWT.

Durability of a brief psychological intervention to increase walking in patients with intermittent claudication – 1-year follow-up of a randomised controlled trial

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Objective: Increased walking is often recommended as the primary treatment of intermittent claudication (IC), both for symptom reduction and to improve cardiovascular fitness. This study assessed the longer-term effect on walking and uptake of surgery/angioplasty of a brief psychological intervention designed to increase walking in patients with IC.
Method: Fifty-eight patients newly diagnosed with IC were randomised into two groups. The control group (n = 30) received usual care, and the treatment group (n = 28) received usual care and a brief (two session) psychological intervention to modify illness and walking beliefs and develop a personalised walking action plan. Participants were followed up after 4 months and 1 year. Daily steps were measured by pedometer at each time point. Analysis was by intention to treat.
Results: At 1 year, participants in the intervention group walked less steps per day than at 4-month follow-up, but still walked significantly more steps per day (1374, 95% CI, 528–2220) than participants in the control group. At 1 year, significantly more participants in the control group (20/30) had received angioplasty/surgery than participants in the intervention group (10/28), c; 2(1) = 5·56, p = ·018.
Conclusion: These results demonstrate that this brief psychological intervention for patients with IC leads to the maintenance of improved walking behaviour over a 1-year period, and to a reduced demand for revascularisation. A brief psychological intervention which improves walking and reduces surgical intervention rate, and which is less costly than current treatments could improve health and provide substantial savings in the management of IC.

Antiplatelet agents for intermittent claudication (IC): results of a meta-analysis (Cochrane review)

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Objective: Evidence for antiplatelet agents in intermittent claudication (IC) is sparse and is mainly inferred from meta-analysis of patients with coronary and cerebrovascular disease. This meta-analysis was performed specifically to address this deficiency.
Method: A Cochrane review was performed of all randomised controlled trials (RCTs) of antiplatelet agents in patients with stable IC (stage II Fontaine). Trials of patients undergoing planned or recent endovascular or surgical interventions were excluded.
Results: Fourteen RCTs of patients with IC (15,630 patients: 7802 antiplatelet, 7828 placebo) were reviewed. All-cause (RR 0·77; 95% CI, 0·61–0·98) and cardiovascular mortality (RR 0·61; 95% CI, 0·38–1·00) were significantly reduced with antiplatelet therapy compared to placebo with a relative risk reduction of 23% and 39%, respectively. Cardiovascular events (fatal and non-fatal MI or stroke) were also significantly reduced (RR 0·74; 95% CI, 0·59–0·93) with antiplatelet therapy. Three RCTs compared other antiplatelet agents (clopidogrel, clopidogrel plus aspirin and picotamide) against aspirin. Meta-analyses showed that other antiplatelet agents were significantly better at reducing all-cause mortality (RR 0·80; 95% CI, 0·67–0·96), cardiovascular events (RR 0·78; 95% CI, 0·66–0·92) and MI (fatal and non-fatal)(RR 0·65; 95% CI, 0·52–0·81) compared to aspirin. Adverse events including dyspepsia (RR 2·11; 95% CI, 1·23–3·61) and adverse events leading to early cessation (RR 1·66; 95% CI, 1·33–2·07) were significantly worse with antiplatelet therapy compared to placebo. Major bleeding was more pronounced with antiplatelet therapy but this did not reach statistical significance (RR 2·30; 95% CI, 0·77–6·88). When compared to aspirin, major bleeding with other antiplatelet agents was non-significantly reduced (RR 0·50; 95% CI, 0·08–3·17).
Conclusion: Antiplatelets significantly reduce mortality and cardiovascular events in IC patients. Evidence for the ongoing use of aspirin in IC is weak. Other antiplatelet agents appear to confer a more significant benefit.

National Clinical Audit can drive quality improvement; lessons from the Carotid Intervention Audit (CIA)

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Objective: The UK Carotid Intervention Audit (CIA) is a continuous national audit that has been running since December 1st 2005. It contains 17,263 cases for analysis to 30th September 2010. We report on the first three rounds with particular emphasis on performance against national standards (NICE CG68) for intervention following TIA or minor stroke. The standard sets a target of 14 days for the time between symptom and intervention.
Method: Data were collected in real time using a web-based data tool. The Vascular Society of Great Britain & Ireland and the Clinical Effectiveness Unit of the Royal College of Physicians conducted analysis jointly.
Results: During the three rounds of the audit, the number of surgeons contributing rose from 61% to 87%. Case ascertainment rose from 56 to 70, to 79% compared to HES. The interval from symptom to intervention fell from a median of 40, to 28 and then 21 days. There is evidence of geographical variation in service quality. Data completeness was greater in larger units. The time from referral to surgery was significantly quicker in larger stroke services, suggesting that volume of cases may be a factor in making progress along the pathway of care run smoothly.
Conclusion: The CIA provides evidence of improvement in the delivery of care to patients presenting with TIA or minor stroke. There is still unacceptable variation in practice, with significant delays in access to treatment nationally. Vascular services need to ensure that their TIA/minor stroke care pathways provide rapid access to intervention.

Conservative coil embolisation of the internal iliac artery prevents associated Type II endoleaks after endovascular aneurysm repair (EVAR)

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Objective: EVAR may require external iliac limb extension with internal iliac artery (IIA) embolisation to prevent a Type II endoleak. Packing the IIA with coils until antegrade flow has ceased is expensive, exposes patients to additional contrast and radiation, and risks embolisation. We adopted a policy of minimal coil utilisation without an emphasis on immediate flow cessation. This study investigated whether this technique prevented associated Type II endoleak following IIA embolisation during EVAR.
Method: Patients were identified through a retrospective review of an EVAR database from January 2008 to January 2011. Inclusion criteria were all patients undergoing EVAR and IIA coil embolisation with 1 month postoperative CTA/US duplex. Primary outcomes included IIA flow cessation upon completion of EVAR, and freedom from IIA associated Type II endoleak at 1 month.
Results: During this period 295 patients underwent EVAR, of whom 48 (16%) required IIA coil embolisation (median age 75 years, male 44). Median IIA diameter was 12 mm (7–74 mm) and median length was 32 mm (14–83 mm). Nester coils were used in all patients with 1–3 coils being deployed in most cases. Angiography following EVAR confirmed proximal IIA occlusion in 26 patients (54%). Forty-six patients fulfilled the inclusion criteria, of whom 45 (98%) had freedom from IIA associated Type II endoleak at 1 month. The one IIA associated Type II endoleak observed at 1 month had resolved on 1-year duplex.
Conclusion: Conservative coil utilisation for IIA embolisation is successful in preventing associated Type II endoleak and evidence of flow cessation is unnecessary as the majority will occlude at 1 month.

Training in vascular surgery following the separation from general surgery – current dilemmas in delivery

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Objective: Establishing a vascular specialty creates considerable concerns around delivery of training. This study examines current problems in training through trainees' experiences.
Method: Trainees with a particular interest in vascular surgery in a large deanery were identified at their RITA/ARCP. Trainees in ST3+ posts subsequently completed a deanery educational survey.
Results: Of 1426 surgical trainees, 11 of 215 core trainees (5%) and 50 of 371 general surgery higher trainees (13%) had a major interest in vascular surgery. 76% of HSTs agreed with the separation, but only 52% felt 2 years of general surgical exposure was adequate. HSTs requested additional exposure to dialysis access (86%), cardiothoracic surgery (76%) and lymphoedema (64%), 93% wanted experience in a major trauma centre and 93% considered a vascular/endovascular fellowship necessary to complete their training (the majority stating abroad). 69% of vascular trainees felt that 6 years of higher training was inadequate. ST6+ trainees were separately questioned on training they considered deficient; responses included branched/fenestrated EVAR (83%), open AAA repair (78%), open TAAA repair (56%), infrarenal EVAR (50%), peripheral angioplasty/stenting (61%), peripheral bypass surgery (33%) and CEA (28%).
Conclusion: Without substantial changes, the current proposed programme, based upon the training actually delivered, is inadequate for many vascular trainees. Exposure to open and endovascular surgery is grossly deficient. Major discussions are needed on the role of the future vascular surgeon in a major trauma centre if they have inadequate general surgical experience. Trainee numbers in vascular surgery are small and declining. Trainees' experiences demonstrate that many are requesting fellowships abroad to complete both open and endovascular training. This is likely to be a national problem.

Deficiencies in experience in UK vascular trainees persist: a survey of Rouleaux Club members

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The Rouleaux Club

Objective: To evaluate the training experience of current United Kingdom (UK) vascular trainees.

Method: A web-based questionnaire of the 217 members of the Rouleaux Club which represents UK vascular and endovascular trainees. Members were asked to complete the survey between May and June 2011.
Results: 153 trainees (71% response rate), representing all of the UK training deaneries, completed the survey. 52% are currently in posts that do not offer endovascular training. 88% reported having performed fewer than 10 peripheral angiograms in the last year. 67% of trainees had performed fewer than 10 EVARs in the past year, either in part or in whole. Half of endovascular fellowships are still being taken overseas. Fifteen members hold a formal qualification in ultrasound scanning but 49% have no access to formal ultrasound training. 85% of trainees believe that vascular access will play a role in their future practice. 33% of trainees currently have exposure to vascular access training and 49% have performed no vascular access procedures in the past 12 months. No experience of endovenous laser ablation, radiofrequency ablation, or foam sclerotherapy was reported by 33%, 49% and 46%, respectively. 27% reported having received some training within the private sector, largely as an assistant, but 11% have performed parts of operative cases under supervision.
Conclusion: Current training delivery in critical aspects of the vascular and endovascular curriculum is unable to equip existing trainees with the skills required for a future specialist vascular practice.

Final results from the MASS trial of AAA screening

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Objective: Whether benefit continues more than 10 years after a single ultrasound screen for abdominal aortic aneurysm (AAA) is uncertain. Here we report the final 13-year follow-up results from the Multicentre Aneurysm Screening Study (MASS) randomised trial.
Method: The MASS trial recruited a population-based sample of 67,770 men aged 65–74. Half were invited to screening for AAA, followed by surveillance for small aneurysms (3·0–5·4 cm) and surgical intervention, if appropriate, for large aneurysms (> 5·5 cm). The main outcome was AAA-related mortality, including both deaths from AAA rupture and all deaths within 30 days of AAA surgery.
Results: Overall, 213 AAA-related deaths occurred in the invited group, compared to 370 in the control group: hazard ratio 0·57 (95% CI, 0·48 to 0·58). Total mortality was also significantly lower: hazard ratio 0·97 (95% CI, 0·95 to 0·99). The overall incremental cost-effectiveness ratio of AAA screening at 13 years was estimated as £4700 per life-year gained, substantially lower than the estimate of £7600 at 10 years. During years 10–13, despite an increase in AAA ruptures in those originally screened as normal and continuing opportunistic detection of AAA in the control group, the number of AAA-related deaths remained considerably lower in the invited group: 62 *versus* 79, hazard ratio 0·78.
Conclusion: The MASS trial has provided the majority of the worldwide randomised evidence on the benefit of AAA screening. These final results indicate continued benefit in terms of AAA-related mortality and a statistically significant reduction in total mortality. Offering population-based screening to men is also extremely cost-effective in NHS terms. Despite a reported reduction in the prevalence of AAA, these results remain relevant to the current NHS AAA Screening Programme.

Should we follow-up men with screening-detected aortas 2·5–2·9 cm?

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Objective: Surveillance to detect late aneurysm development in patients with aortas (2·5 cm to 2·9 cm) detected on screening is not currently offered in the NHS AAA Screening Programme. The objective of this study was to gather individual patient data from centres with AAA surveillance programmes to determine how many patients progressed to AAA.
Method: Individual patient data from four studies were pooled and analysed by creating survival tables and performing Kaplan-Meier analysis. Any inner-to-inner measures were adjusted to outer-to-outer by adding 3 mm to the aortic diameter.
Results: 794 patients were included in the analysis with a mean follow-up of 4·7 years; 38% were followed to 5 years, and 11·3% to 10 years. A total of 469 patients (59·1%, 95% CI, 55·6 to 62·4) progressed to an aortic diameter over 3 cm at a mean time of 5·1 years (4·7 to 5·4). Of these patients, 81·9% (78·1 to 85·1) reached 3 cm by 5 years and 98·7% (97·2 to 99·4) by 10 years. A total of 47 patients (5·9%, 4·5 to 7·8) developed an AAA greater than 5·4 cm at a mean time of 13·7 years (12·9 to 14·6). Of those patients who developed AAA greater than 5·4 cm, 6·4% (2·2 to 17·2) did so by 5 years and 46·8% (33·3 to 60·7) by 10 years. AAA rupture was recorded in three studies (704 patients) and occurred four times (0·6%, 0·2 to 1·5) after 9·8, 10·8, 11·4 and 14·7 years.
Conclusion: These data demonstrate that a reasonable number of patients with sub-aneurysmal aortic dilatation will go on to develop true aneurysms.

Individual patient characteristics which influence small abdominal aortic aneurysm expansion and rupture: an analysis of > 15,000 persons' records

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Objective: To assess which individual patient characteristics, other than aneurysm diameter, may influence the aneurysm growth and rupture rates.
Method: Individual patient data have been collated from 15,475 persons followed-up for a small aneurysm (3·0–5·5 cm only) from 18 studies. The rates of aneurysm growth and rupture (analysed using longitudinal random-effects modelling and survival analysis) and the influence of covariates (including demographics, medical history and drug therapy) on these parameters have been summarised in an individual patient meta-analysis. All analyses were adjusted for aneurysm diameter.
Results: The average aneurysm growth rate of 2·18 mm/y was similar in men and women and independent of age. The growth rate was increased in smokers (by 0·35 mm/y) and decreased in patients with diabetes (by 0·51 mm/y). Anti-hypertensive medications had a very small and non-significant effect on aneurysm growth. Statins reduced aneurysm growth by 0·21 mm/y, but this was not significant ($p = 0·121$). Rupture rates were almost four-fold lower in men *versus* women ($p < 0·001$), doubled in current smokers ($p < 0·001$), increased with higher mean arterial pressure ($p < 0·001$) and decreased with calendar year ($p = 0·032$). There were no convincing effects of any drugs on aneurysm rupture rates.
Conclusion: Recommendations for surveillance frequency of individual patients may need to consider diabetes and smoking, in addition to aneurysm diameter. No single drug used for cardiovascular risk reduction was shown to have a major effect on the natural history of aneurysms, but reducing blood pressure may reduce the risk of rupture.

Outcomes for patients who do not undergo repair of their large aortic aneurysms

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Objective: Little information is available on the outcome for patients declined elective and emergency aneurysm repair. We present the turn-down rates and outcomes for patients with abdominal aortic aneurysms who did not undergo repair.
Method: A prospective observational study was conducted of all patients with AAA who did not undergo elective (group X) or emergency (group Y) repair between 1st January 2007 and 1st May 2011. Patient demographics, aneurysm size and reasons for non-intervention were recorded on a database. Mortality data and causes of death were obtained from case-notes and via the Office of National Statistics.
Results: 429 patients were assessed (elective 328: emergency 101). 241 underwent elective repair (open = 124, EVAR = 117, in-hospital-mortality =

4·1%). Eighty-seven patients (26%) were not treated (group X) (49 males, mean age 82 years). The reasons for non-intervention were: significant comorbidity (90), malignancy (17), age (28), patient-choice (37). In group X, median survival was 276 days (range 25–1881), 71 died; < 40% were aneurysm-related deaths. Sixty-six underwent emergency repair (in-hospital-mortality = 37%). Thirty-five patients (34·5%) were not treated emergently (group Y) (22 males, mean age 82 years). The reasons for non-intervention were: significant comorbidity (30), malignancy (5), age (12), patient-choice (7). In group Y, median survival was 2 days (range 0–17).

Conclusion: A significant percentage of patients did not undergo aneurysm repair. Despite being high risk for not surviving aneurysm repair, patients turned down for elective treatment have a reasonable life expectancy and are less likely to die from their aneurysm. In patients unlikely to survive emergency surgery, death is not necessarily immediate. Time is allowed for arrival of family and quality-closure in death, which is often denied with expeditious attempt at futile repair.

Explaining the reduction in mortality from ruptured abdominal aortic aneurysm in England and Wales 1996–2009

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Objective: Last century a steady rise in incidence of abdominal aortic aneurysm (AAA) was reported. Mortality from rupture peaked in 1996. We have investigated reasons for the decline in mortality from rupture since then.

Method: Routine statistics for mortality, hospital admissions and procedures in England and Wales were investigated. Data were age-standardised and mortality reported per 100,000 population. Trends in smoking, hypertension and hypercholesterolaemia, and statins, together with regression coefficients for mortality, were available from public sources for those aged 65+ years, with an effect on rupture deaths prevented in this age group, estimated from "deaths prevented = (deaths in index year) x (relative risk factor decline) x β-regression coefficient" (IMPACT equation).

Results: Since 1996, deaths from ruptured aneurysm have decreased sharply, almost two-fold in men. Hospital admissions for elective repair have increased modestly (from 40 to 45), attributable entirely to more repairs in those 75+ years, $p < 0.001$. Admissions for rupture have declined (from 18 to 13 across all ages), with the proportion offered and surviving emergency repair unchanged. Since 1996, mortality from ruptured aneurysm in those 65+ years has fallen from 66 to 44. An estimated 8–10 deaths were prevented by a reduced prevalence of smoking. Estimates for the effects of blood pressure and lipid control are uncertain but may have contributed to a decrease of 0–2 and 7–17 deaths, respectively.

Conclusion: The reduction in incidence of ruptured aneurysm since 1996 is probably due more to changes in smoking and perhaps lipid control (statin use) than to increases in elective aneurysm repairs, particularly in those < 75 years where elective repairs have not increased.

National Vascular Database analysis: independent operative predictors of abdominal aortic aneurysm repair outcomes

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Objective: To analyse the relationship between elective infrarenal abdominal aortic aneurysm (AAA) repair outcomes and each of the operative data fields collected by the National Vascular Database (NVD).

Method: Between January 2008 and December 2010, data for 13,068 elective AAA repairs were entered in 379 NVD data fields. Ninety-nine fields were operative. These included 44 combined endovascular (EVAR) and open (OAR), 44 EVAR-specific and 11 OAR-specific fields. Logistic regression analysis for the odds of death and for any complications was performed adjusting for gender, ASA and screening status.

Results: For the combined and OAR-specific fields, data were available for 87% of cases on average. For the EVAR-specific fields, less than 15% of cases had any data. Twenty combined, 5 EVAR-specific and 3 OAR-specific fields were independently associated with mortality or major complications. Examples included reduced mortality risk with tube OAR grafts (OR 0·74 [95% CI, 0·56–0·99]; p = 0·042), increased mortality risk with aorto-mono-iliac EVAR grafts (1·88 [95% CI, 1·08–3·26]; p = 0·022) and with percutaneous EVAR access (2·64 [95% CI, 1·05–6·64]; p = 0·031). Spinal/epidural EVAR anaesthesia was associated with reduced mortality (0·3 [95% CI, 0·18–0·51]; p = 0·000), whereas the opposite was true for local anaesthesia (2·85 [95% CI, 1·88–4·32]; p = 0·000). A 1 mmHg rise in lowest intra-operative systolic pressure reduced the odds of mortality by 3·3% (2·7%–3·8%; p = 0·000) and complications by 1·2% (95% CI, 1%–1·5%; p = 0·000) for both EVAR and OAR.

Conclusion: Approximately 40% of the non-EVAR-specific NVD operative fields correlate independently with outcome. Some of these correlations have the potential for improving current operative practices. For EVAR-specific fields, only 11% were associated with outcome. This may be due to poor compliance with these fields.

Angiotensin-converting enzyme inhibitors are associated with significant reduction in abdominal aortic aneurysm prevalence

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Objective: The aetiology and natural history of abdominal aortic aneurysm (AAA) disease remain largely unknown. This study examines the correlation between AAA prevalence and angiotensin-converting enzyme inhibitors (ACEI) intake in men aged 65.

Method: Between 2001 and 2008, 17,363 men reaching the age of 65 were invited for AAA screening. Those attending were given a health and current medications questionnaire. Mean blood pressure was measured prior to abdominal aortic ultrasonography.

Results: 13,982 (80·5%) men attended for screening and completed their questionnaires; 380 (2·7%) AAAs were identified. Over the study period, AAA prevalence declined from 3·2% to 2·1%. This was associated with an increase of those on ACEI from 11% to 30%. Correcting for history of cardiac disease, stroke, hypertension and smoking, those who were on ACEI (n = 1996) had a reduced risk of developing an AAA (OR 0·55 [95% CI, 0·37–0·83]; p = 0·003). For those who were on ACEI as the only antihypertensive medication (n = 676), the odds for having an AAA were further reduced at 0·1 (95% CI, 0·05–0·51), p = 0·0004. The odds for developing an AAA in those who were on antihypertensives other than ACEI (n = 2823) were 2·47 (95% CI, 1·70–3·57), p = 0·000. The odds for developing an AAA in those who were not on any antihypertensive medications (n = 8178) were 0·87 (95% CI, 0·53–1·42), p = 0·581.

Conclusion: The prevalence of AAA disease appears to be significantly lower in men aged 65 who are on ACEI. This observation was independent to major confounders. ACEI appear to play an important role in the pathophysiology of AAA disease. The potential value of this drug class in the prophylaxis of AAA disease warrants appraisal.

Motif-chemokine 19 (CCL19) and cathepsin G (CTSG) are upregulated in highly unstable carotid atherosclerotic plaques

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Objective: Carotid atherosclerotic plaque instability is a leading cause of morbidity and mortality in the western world. We aimed to identify markers of plaque instability using whole genome microarray analysis with qRT-PCR validation on a larger independent cohort of patients. Instability was defined on the basis of symptoms within 2 weeks, evidence of microembolisation and histological appearance.

Method: 120 patients undergoing carotid endarterectomy (CEA) were recruited prospectively. RNA from 24 plaques harvested during CEA were hybridised onto a whole-genome microarray. Genes were chosen for qRT-PCR validation

based on hierarchical significance and gene ontology processes on a separate cohort of 96 patients.

Results: Results of differentially expressed genes (> 1·3 fold, $p < 0·05$ after correction for Multiple Hypothesis Testing) were sought for: 1) recency of symptoms – 177 genes; 2) evidence of embolisation – 2294 genes; 3) histological grading – 134 genes. Fifty-four genes were identified in at least two of the three analyses. Eleven (85%) of 13 genes chosen for validation using qRT-PCR showed concordance in directionality with microarray findings. Motif-chemokine 19 (CCL19) (× 1·92 fold; $p = 0·02$) and cathepsin G (CTSG) (× 3·7 fold; $p = 0·002$) were significantly upregulated in both microarray and qRT-PCR analyses.

Conclusion: CCL19 and CTSG have been demonstrated in plaques that have been independently shown to be histologically and phenotypically unstable. These genes are involved in inflammation and connective tissue remodelling and now form targets for pharmacotherapy.

In vivo assessment of the POSS-PCU small-calibre graft in a sheep carotid artery interposition model

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Objective: There is a clinical need for a small-calibre (< 6 mm) synthetic cardiovascular bypass graft with function and durability equivalent to autologous conduits. The main objective of this study was to assess *in vivo* performance based on EN-14299 standards of a previously developed nanocomposite polymer graft with these characteristics.

Method: POSS-PCU grafts (silsesquioxane polycarbonate-urea urethane) with a 5 mm internal diameter were implanted in sheep (n = 10) as carotid interposition grafts for 9 months. Flow rate pre- and post-implantation were measured. Change in vessel wall diameter in each cardiac cycle was analysed at discrete sites along graft and carotid artery per-operatively to calculate compliance. Patency was monitored monthly with duplex ultrasound and vascular wall tracking. Similarly, six PTFE grafts were implanted as a control.

Results: Graft implants were between 5–8 cm long and stretched by 10%, hence allowing replacement of longer-length blood vessels. Flow rate before implantation was 480 ± 48 ml/minute and post-implantation was reduced to 460 ± 46 ml/minute (NS). Patency was 85% at 9 months. Patency of PTFE grafts was 0% at 1 month. Compliance of the POSS-PCU grafts was significantly greater at implantation and maintained at 9 months.

Conclusion: The 85% patency rates achieved at 9 months with POSS-PCU grafts are unparalleled in this sheep carotid test and not seen in any previous studies on any other synthetic materials. The *in vitro* properties previously reported have been confirmed *in vivo*. This compliant graft with significantly less thrombosis, and intimal hyperplasia, has potential for clinical use in coronary artery bypass, vascular access and infrainguinal revascularisation.

Genetic determinants of vascular diameter and the risk of abdominal aortic aneurysm

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Objective: There is evidence that abdominal aortic aneurysm (AAA) is a manifestation of a systemic dilating diathesis. Common carotid artery diameter (CCAD) is a quantitative trait that is correlated with the risk of AAA. We sought to determine the genetic determinants of CCAD and their association with AAA.

Method: We performed genetic association analyses of CCAD in 3408 individuals from the IMPROVE study using the Illumina 200k Cardiometabochip platform. Lead SNPs were taken forward for replication in the Whitehall II study (n = 2091), and tested for association with AAA in four case-control studies (4,317 cases and 36,776 controls), infrarenal aortic diameter in a prospective study (n = 8347) and expression of nearby genes in aortic tissue.

Results: In the IMPROVE study there was a strong association between variants on chromosome 1p24·3 and greater CCAD. Two SNPs (in weak LD) at this locus were prioritised for follow-up studies. Rs4916251 was associated with greater CCAD in the IMPROVE study ($p = 1·1 \times 10^{-7}$), but this did not replicate in the WHII study ($p = 0·39$, combined $p = 2·4 \times 10^{-6}$). This SNP was, however, associated with AAA (OR 1·1, $p = 6 \times 10^{-3}$), larger infrarenal aortic diameter ($p = 0·04$) and expression of PIGC in aortas ($p = 4 \times 10^{-3}$). Rs74126223 showed association with CCAD in IMPROVE ($p = 2·8 \times 10^{-6}$) and WHII ($p = 0·03$, combined p-value $3·9 \times 10^{-7}$), but no association with the other trait.

Conclusion: These data suggest that a locus on Chr1p24·3 is important in vascular remodelling and the risk of developing AAA. This provides genetic evidence of a systemic diathesis in AAA.

Tissue engineering small-diameter vascular grafts: decellularisation of porcine arteries

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Objective: The clinical performance of small-diameter synthetic grafts in arterial bypass has been poor. The aim of this study was to develop a biocompatible, biomechanically stable, small-diameter vascular graft suitable for cardiac and peripheral bypass using porcine arteries.

Method: A novel detergent-based protocol was developed to remove all cellular components from porcine common carotid arteries to render them non-immunogenic. Representative tissue sections (n = 6) were assessed histologically using native artery as controls. Fresh and acellular arteries (n = 6) were subject to biochemical analysis: DNA, collagen, denatured collagen and sulphated proteoglycans were quantified. PCR analysis was carried out to determine the coding potential of residual DNA fragments. *In vitro* biocompatibility was assessed using two distinct cell lines (murine 3T3 and baby hamster kidney cells). Burst pressure, suture retention, compliance and low rate failure testing were carried out to identify any biomechanical changes following decellularisation; fresh and acellular arteries were compared (n = 6).

Results: The decellularisation protocol resulted in arteries free from cells, > 95% of the total DNA had been removed and PCR identified the residual DNA to be non-coding. Acellular arteries were shown to be biocompatible *in vitro*; cells grew in contact with the tissues and there was no decrease in cell viability after incubation with soluble tissue extracts. Biochemical and biomechanical analysis indicated the properties of each acellular artery had not significantly altered following decellularisation, with compliance and maximum burst pressures remaining unchanged.

Conclusion: The study resulted in biocompatible, biomechanically competent acellular arteries, which may have utility in arterial bypass.

Video motion analysis for objective assessment in catheter-based endovascular intervention

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Objective: Wider uptake and increasing case complexity in endovascular intervention mandates proficiency in guidewire/catheter manipulation. Objective motion analysis has been comparatively unexplored in this field. This study assesses its feasibility and its role for evaluation of technical skill and technology benefit.

Method: A semi-automated motion tracking software was developed, utilizing CT-calibrated and noise-corrected cumulative translational motion between frame-by-frame fluoroscopic video co-ordinates, to calculate the 2D catheter-tip path length (PL) with visual representation in an AP projection. Motion analysis was performed for 64 simulated endovascular procedures involving 20 experienced operators. Subjects cannulated aortic arch and visceral branches within CT-reconstructed pulsatile silicon phantoms in the angiography suite, using robotic *versus* conventional catheter techniques.

Results: Median PL was significantly reduced using robotic catheterisation techniques: 2093 mm IQR (1471–3554) *versus* 1352 mm (1111–1668) in the arch ($p = 0{\cdot}001$); 2340 mm (1860–3754) *versus* 572 mm (474–767) in the visceral segment ($p = 0{\cdot}001$). PL was significantly shorter for the less angulated aortic branches with conventional cannulation techniques ($p < 0{\cdot}02$). Further analysis revealed statistically significant correlations between PL and total procedure times (Spearman's rho $= 0{\cdot}749$; $p < 0{\cdot}001$), catheter movements (rho $= 0{\cdot}7$; $p < 0{\cdot}001$) and vessel wall hits (rho $= 0{\cdot}468$; $p = 0{\cdot}005$). An inverse correlation between PL and qualitative procedure scores was also found to be statistically significant (rho $= -0{\cdot}662$; $p < 0{\cdot}001$).

Conclusion: Endovascular instrument video motion analysis is feasible, and may act as a useful tool to assess endovascular skill and technology benefit. With further refinement and extraction of other descriptive metrics in addition to PL, it may provide an attractive platform for objective evaluation of endovascular performance.

Trends in mortality and incidence of abdominal aortic aneurysms in England and Wales

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Objective: Recent studies from Australia and New Zealand reported declines in both abdominal aortic aneurysm (AAA) mortality and incidence. This has important implications for screening policies. This study examined trends in AAA mortality and incidence in England and Wales.

Method: The UK Office for National Statistics provided cause-specific death data for England and Wales; Hospital Episode Statistics supplied hospital admissions and procedures data for England from 2001 to 2009. Poisson regression models were constructed to estimate the relative change over time.

Results: Age-standardized rates for AAA mortality in England and Wales fell significantly by 35·7% from 2001 to 2009. Ruptured AAAs were a major contributor of AAA mortality (84·6%) and the sharp decline in AAA mortality was largely due to a 35·3% drop in age-standardised ruptured AAA deaths. During the same period, ruptured AAA admissions in England significantly declined by 25·8% (6·1/100,000 to 4·5/100,000) and emergency AAA repairs similarly fell by 29·6% (3·1/100,000 to 2·2/100,000). In contrast, non-ruptured AAA admissions increased by 6·4% (14·4/100,000 to 15·4/100,000) and non-emergency AAA repairs increased by 19·6% (7·1/100,000 to 8·8/100,000). Total AAA admissions remained the same (20·5/100,000 to 19·9/100,000) and total AAA repairs increased by 7·3% (10·2/100,000 to 11·0/100,000).

Conclusion: Unlike Australia and New Zealand, the falling AAA mortality in England and Wales did not mirror a decline in disease incidence, but appeared to be related to a lower incidence of ruptured AAA. The overall AAA case-load has not decreased in England. In contrast to Australia and New Zealand, these data provide support for a continued policy of AAA screening.

A new 4D aortic imaging technique to quantify vessel wall mechanics *in vivo*

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Objective: Vessel wall mechanics are important in the development, progression and treatment of cardiovascular disease. Current imaging techniques (e.g. dynamic CT) cannot quantify detailed aortic movement in three dimensions (3D) over time (4D). We aimed to develop a new 4D magnetic resonance (MR) imaging technique to accurately quantify aortic wall mechanics *in vivo*.

Method: Fifty different image acquisition parameters were individually tested in simulations and phantoms to design the MR sequences. The accuracy of different rigid (affine) and non-rigid image registration techniques was investigated. The optimised combined image acquisition and registration scheme was then prospectively applied to 12 human volunteers.

Results: New high-resolution MR sequences were successfully developed to characterise aortic movement caused by: 1) cardiac motion (4D-cine, ECG-gating, respiratory navigator [3 mm], 1·5 mm^3, 25 phases, SENSE = 2, half-scan-y = 0·625, typical scan-time 5–10 minutes); 2) respiratory motion (4D-respiratory-resolved data, 3 mm respiratory bins, 1·5 mm^3, 4–9 dynamics, radial-P-E, SENSE = 6, 4–8 minutes). Affine techniques accurately quantified rotation, translation, scaling and shear (Target Registration Error: $1{\cdot}4 \pm 0{\cdot}7$ mm) and non-rigid quantified whole body aortic motion (WBM) (foot-head, right-left, anterior-posterior)(TRE:$1{\cdot}2 \pm 0{\cdot}4$ mm). Significant inter-individual differences in aortic deformation and dynamic curvature were seen. Cardiac motion caused high levels of aortic rotation (mean: 25° [SD31·1]) and shear (11° [SD6·7]), whereas respiratory motion resulted predominantly in foot-head WBM (8 mm [SD5]). Overall, the motion of the ascending aorta (6·45 mm [SD1·57 mm]) was significantly greater than the aortic arch (3·50 mm [SD1·02]; $p = 0{\cdot}002$) and descending thoracic aorta (2·41 mm [SD0·98], $p = 0{\cdot}0001$).

Conclusion: We have successfully developed, validated and applied a new method to quantify aortic wall mechanics *in vivo*. For the first time we have seen significant inter-individual differences in vessel wall motion. This information will improve our understanding of cardiovascular disease and can be used to risk stratify and optimise treatment for individual patients.

Carotid plaque imaging: ready for prime time?

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Objective: Histopathological evidence indicates that carotid intraplaque haemorrhage (PH) is associated with plaque instability. PH in carotid disease can be detected by MRI. We assessed the predictive value of MRIPH for recurrent ipsilateral ischaemic events in symptomatic carotid stenosis (SCS).

Method: 176 patients with SCS (50–99%) from three prospective studies underwent 3D carotid MRI and were followed up until recurrence of ipsilateral ischaemic event, CEA or death. Multivariate Cox regression analysis was performed to determine hazard ratios for time to event.

Results: 112 patients with MRI hyperintense plaque were classified as PH+; 106 patients had 70–99% stenosis. 68% had CEA resulting in a follow-up range of 0–3128 days (median 531 days). During follow-up, 48 recurrent events were noted (19 stroke) in PH+ patients, compared with only four events (one stroke) in the PH-group. Backward conditional analysis showed that PH (HR 13·1; 95% CI, 4·6–37·2; $p < 0{\cdot}0001$) and degree of stenosis (HR 13·1; 95% CI, 4·6–37·2; $p < 0{\cdot}0001$) independently predicted recurrence of ischaemic events. Backward conditional analysis for stroke alone revealed that MRIPH was the only significant predictor (HR 22·3; 95% CI, 3–169; $p = 0{\cdot}003$) with a trend for higher degree of stenosis.

Conclusion: MRIPH independently predicts recurrent ipsilateral ischaemic events, and stroke alone, in > 50% SCS. For stable carotid plaques as indexed by absence of PH, the estimated event risk may invalidate RCT evidence for

efficacy of CEA. We conclude that PH imaging should become an integral part of diagnostic assessment for SCS with the potential to influence management in 50–69% of stenosis, and sufficient equipoise to warrant a RCT for all grades.

Risk-adjusted retrospective concurrent cohort study of fenestrated endovascular repair (f-EVAR) *versus* open surgery for juxtarenal aneurysms

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Objective: There is a need to compare f-EVAR against open repair for juxtarenal aneurysms, but there is little equipoise for an RCT. Our purpose was to conduct a risk-adjusted retrospective concurrent cohort comparison of the techniques.

Method: All patients who underwent repair of a juxtarenal aneurysm within one institution between January 06 and December 10 were included. Case notes and CT scans were retrieved for data, V-POSSUM score and aneurysm morphology.

Results: Open surgery cohort: n = 54 (median age 72, 36 men). The aortic cross-clamp was infrarenal in 20 patients, suprarenal or above in 21 and inter-renal in 8. Postoperatively, 63 major complications were noted in 30 patients, 9 of whom required 16 re-interventions. Total hospital stay was 1170 days, 234 in ITU. Peri-operative mortality was 9·2% (n = 5), exactly as estimated by V-POSSUM. f-EVAR cohort: n = 53 (median age 76, 47 men). Two fenestrations and one scallop was the most frequent configuration (n = 31). Postoperatively, 37 major complications were noted in 18 patients, 6 required re-intervention. Total hospital stay was 559 days, 31 in ITU. Two died (3·7%) peri-operatively, compared to the V-POSSUM estimate of 9·4% (n = 5). Crude absolute risk reduction was 5·5%. In the hypothetical event of a f-EVAR cohort undergoing open repair instead, V-POSSUM estimated 7 deaths (13·2%), with a risk-adjusted absolute risk reduction due to f-EVAR of 9·5%.

Conclusion: f-EVAR reduces mortality and morbidity substantially, while lowering hospital resource utilisation. When adjusted for differences in operative fitness, a switch from open surgery to f-EVAR is estimated to provide a substantial (9·5%) absolute risk reduction for peri-operative mortality.

Randomised trial of endovenous laser ablation *versus* surgery for small saphenous varicose veins

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Objective: No randomised clinical trial (RCT) comparing treatment options for small saphenous vein (SSV) incompetence exists. There is evidence that SSV may behave differently to great saphenous vein (GSV) incompetence following treatment, hence available evidence for GSV cannot be extrapolated to SSV management. This RCT aimed to compare the clinical efficacy and quality of life (QoL) outcomes for conventional surgery and endovenous laser ablation (EVLA) in the treatment of SSV incompetence.

Method: Patients with unilateral, primary saphenopopliteal junction (SPJ) incompetence with SSV reflux were randomised equally into parallel groups receiving either conventional surgery or EVLA. Patients were assessed at baseline and at 1, 6, 12 and 52 weeks. Outcome measures included: Visual Analogue Pain scores; Quality of life (generic – SF36, EuroQol 5D and disease-specific – AVVQ); Venous Clinical Severity Score (VCSS); time taken to return to work and normal function; and complication rates.

Results: 106 patients (74 women), median age 47 (IQR 39–57) years were recruited and randomised to surgery (n = 53) or EVLA (n = 53). Intragroup analysis: both groups demonstrated significant improvement in VCSS ($p < 0·001$), disease-specific AVVQ ($p < 0·001$), generic SF36 and EQ5D QoL ($p < 0·05$). Intergroup analysis: postoperative pain was significantly lower after EVLA ($p < 0·05$), allowing an earlier return to work and normal function ($p < 0·001$). Sural nerve sensory disturbance was significantly lower in the EVLA group 7·5% *versus* surgery 26·4% ($p = 0·009$).

Conclusion: EVLA was as effective as surgery in the treatment of SSV incompetence, but associated with less peri-procedural pain, faster recovery and fewer neural complications.

Management and outcome of prosthetic patch infection after carotid endarterectomy: a single-centre series and systematic review of the literature

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Objective: To determine outcomes following prosthetic patch infection after carotid endarterectomy in a single centre and within a systematic literature review.

Method: A retrospective audit and systematic review of the literature.

Results: Twenty-two patients with patch infection after carotid endarterectomy (CEA) were treated between January 1992 and June 2011. Five were referred from other centres, giving an infection rate of 0·8% (17/2136) in the host centre. The commonest infecting organism was Staphylococcus (n = 11). One patient was treated by antibiotic irrigation, one was stented, while 20 underwent debridement and patch excision plus carotid ligation (n = 3), vein patch (n = 3) or vein bypass (n = 14). There were no peri-operative deaths; one suffered a disabling stroke (30-day death/stroke 4·5%). Nine (41%) suffered cranial nerve injuries, four persisting at 30 days. There were no secondary re-infections. The systematic review identified 123 patch infections. Thirty-six (29%) presented < 2 months (usually after peri-operative wound complications) with wound infection/abscess and patch rupture predominating; 78 (63%) presented > 6 months (sinus, false aneurysm). Seventy-nine out of 87 (91%) with a positive culture yielded Staphylococci/Streptococci. Seventy-four patients were treated by patch excision and autologous reconstruction with a 30-day death/stroke rate of 8·1%. Four survivors undergoing autologous reconstruction developed re-infection < 60 days, none suffered later re-infection. Seven of nine patients (78%) undergoing prosthetic reconstruction died or suffered re-infection. Five patients have been treated with a covered stent, none developing re-infection.

Conclusion: Patch infection following CEA is rare but under-reported. Few patients have undergone stenting and long-term data are awaited. For now, patch excision and autologous reconstruction remain the gold standard.

Should we be using VO_2 peak or the anaerobic threshold to risk stratify patients prior to repair of an abdominal aortic aneurysm (AAA)?

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Objective: Cardiopulmonary exercise testing (CPX) is a measure of patient fitness and is used to risk-stratify patients undergoing endovascular (EVAR) or open repair (OR) of an abdominal aortic aneurysm (AAA). We examined which CPX data independently predict mortality.

Method: All patients undergoing AAA repair between 01/01/2007 and 31/12/2008 who underwent CPX assessment were included. Peak oxygen concentration (VO_2 peak) and anaerobic threshold (AT) were recorded. Intervention type, Lee cardiovascular risk score and mortality data were obtained from casenotes. Adjusted Cox-Regression analysis was used to determine which CPX outputs independently predicted mortality.

Results: 134 patients (115 men [85%]) had CPX assessment, with a mean age of 75·4 years (IQR 69·8–80·3). Seventy cases had open repair; 30-day and 1-year mortality, 2·9% and 8·6%. Sixty-four cases had EVAR; 30-day and 1-year mortality, 4·7% and 14%. Overall, the mean Lee score was 1·85, median VO_2 peak was 15·1 (IQR 12·6–17·3) and median AT was 10·6 (IQR 9·1–12·5). Irrespective of intervention, the hazard ratio (HR) for the relative risk of death was 0·88 for every 1 ml/kg/minute reduction of VO_2 peak ($p = 0·002$). This remained significant when adjusted for age (HR 0·88; $p = 0·003$), sex (HR 0·88; $p = 0·002$) and Lee score (HR 0·89; $p = 0·009$). AT was not a significant

predictor of relative risk of death, both unadjusted and adjusted for age, sex and Lee score.

Conclusion: VO_2 peak, but not AT, was an independent predictor of mortality in those undergoing EVAR or OR for AAA. These results require confirmation, but suggest risk stratification should be based on VO_2 peak in preference to AT.

Is cardiopulmonary exercise testing (CPET) useful for predicting survival following elective AAA repair?

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Objective: Elective abdominal aortic aneurysm (AAA) repair aims to improve survival by preventing AAA rupture. Our objective was to assess whether data obtained from pre-operative CPET can predict survival following elective AAA repair.

Method: Data were collected prospectively on all patients who had CPET prior to elective AAA repair from two University Hospitals between September 2005 and June 2011. Mortality data were obtained from the NHS Demographic Batch Service. Abnormal CPET values were defined as an anaerobic threshold of $< 10{\cdot}2$ ml/kg/minute, $VE/VCO_2 > 42$, peak $VO_2 < 15$ ml/kg/minute and inducible cardiac ischaemia. Univariate and multivariate analyses were used to identify variables associated with survival.

Results: Data were available for 375 consecutive patients. The mean age was 74 (range 23–90) with 85·3% being men. Endovascular aneurysm repair (EVAR) was performed in 247 (66%) patients and open repair in 128 patients. The 30-day mortality rates were 2% and 4·7% for EVAR and open repair, respectively (2·9% overall). Over a median follow-up of 19 months (range 0–68 months), 58 (15·5%) patients died. For patients with more than three abnormal CPET values, survival at 24 months was 62% compared to 89% for patients with less than three abnormal CPET values ($p < 0{\cdot}001$). On multivariate analysis, peak $VO_2 < 15$ ml/kg/minute (OR 2·6; 95% CI, 1·4–5·0; $p = 0{\cdot}003$), $VE/VCO_2 > 42$ (OR 2·8; 95% CI, 1·7–4·9; $p < 0{\cdot}001$) and more than three abnormal CPET values (OR 3·2; 95% CI, 1·9–5·6; $p < 0{\cdot}001$) were associated with reduced survival.

Conclusion: Despite good 30-day mortality results following elective AAA repair, CPET identifies patients more likely to die over the following 2 years; elective AAA repair in such patients may need to be reconsidered.

Availability of emergency endovascular aortic interventions: evidence from the IMPROVE trial

R. Hinchliffe, on behalf of the IMPROVE trial Investigators

Objective: Vascular emergencies may benefit from endovascular interventions. The potential benefits of endovascular *versus* open repair for ruptured abdominal aortic aneurysm (AAA) are being investigated in the IMPROVE trial. We sought to identify the reasons for failure to recruit to the trial and document the availability of endovascular surgery at trial centres.

Method: In 20 active UK trial centres, all cases of ruptured AAA are logged and reasons listed for non-randomisation of patients into the IMPROVE trial. CT scans of randomised patients are evaluated in a core laboratory.

Results: Early core laboratory data for aneurysm anatomy have demonstrated complex anatomy: median AAA diameter 7·8 cm (IQR 6·9–9·3), neck length 1·3 cm (IQR 0·5–3·3), neck diameter 2·3 cm (IQR 2·1–2·7) and neck angle 43° (IQR 27°–67°). Seven out of 26 (27%) had > 4 mm dilatation of the neck over the proximal centimetre. Now, with over 200 patients randomised, the commonest reason for non-randomisation of eligible patients is the inability to perform an endovascular procedure (52/148 patients). This reason is used in 8/20 trial centres, including four aspiring trauma centres, and many centres can only offer endovascular repair during the day Monday-Friday.

Conclusion: Ruptured aneurysms presenting to hospital have very large diameters and short, conical aneurysm necks, which are a challenge for endovascular repair. However, the NHS does not appear to provide adequate endovascular cover for this vascular emergency. Provision for out of hours endovascular interventions needs to be urgently addressed.

There is no consensus in the UK on the role of fenestrated endovascular aortic aneurysm repair (fEVAR): the case for a randomised trial

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Objective: In the UK, approximately 100 fEVAR procedures are carried out yearly, mostly in patients with complex aortic aneurysms who are deemed unfit for open repair. Based upon present rates of AAA detection and treatment, if fEVAR was to be extended to all technically suitable patients, irrespective of their 'fitness for open surgery', this number could increase up to 2000 per annum. At current prices this would equate to a cost to the NHS in the order of £30,000,000. The objective of this study was to establish whether consensus exists amongst UK vascular specialists on appropriate indications for fEVAR.

Method: A stepwise consensus process was undertaken using the RAND appropriateness methodology. All UK fEVAR centres with a total experience > 10 procedures were invited to participate.

Results: There was a consensus against fEVAR (> 90% agreement) in patients > 85 years with supra-SMA AAA, < 6 cm diameter and high risk for open surgery and a consensus for fEVAR in patients 65–74 years with supra-SMA AAA > 8 cm diameter and moderate risk for surgery. There was no consensus on the appropriate role for fEVAR in relatively fit patients with juxta or pararenal aneurysms, who represent the majority for whom this approach is technically feasible.

Conclusion: The absence of consensus regarding indications for fEVAR demonstrated by this study support the case for a national RCT to compare fEVAR with open repair in patients who are deemed fit for open surgery. Based upon case series published to date, an 80% power calculation indicates that 564 patients will be needed to give an 80% risk reduction with a 5% significance level. This is a feasible target given the rates of AAA detection and treatment in the UK today.

EVAR for ruptured AAA – does an endovascular first strategy reduce rupture mortality on a centre basis?

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Objective: Endovascular repair (rEVAR) as a primary strategy for ruptured AAA (rAAA) is often promoted. We analysed the results of rAAA repair in Sweden to assess if centres with high penetrance of rEVAR achieved better survival.

Method: All rAAA repairs registered in the Swedish Vascular Registry May 2008-May 2011 were analysed (n = 837). Centres with < 5 rAAA repairs annually were excluded. Outcome was compared for centres with > 50% rEVAR compared to centres with < 50% rEVAR.

Results: Three centres treated 62–82% (mean 72%) of rAAAs with rEVAR (n = 164 patients); 19 centres treated 0–35% (mean 16%) of rAAAs with rEVAR (n = 673). There was no difference in mean age (high rEVAR 74·5 years, low rEVAR 73·9; $p = 0{\cdot}416$), rate of diabetes (10·9 vs. 11·3%; $p = 1{\cdot}0$), cardiac disease (43·2 vs. 37·1%; $p = 0{\cdot}216$) or hypertension (70·1 vs. 68·7%; $p = 0{\cdot}827$). High rEVAR centres had a higher rate of lung disease (35·9 vs. 23·0%; $p = 0{\cdot}010$), higher mean creatinine level (128 vs. 114 µmol/L; $p = 0{\cdot}024$) and larger mean aneurysm size (83 vs. 75 mm; $p = 0{\cdot}022$). The mean pre-operative systolic blood pressure was higher in the high rEVAR centres (87 vs. 72 mmHg; $p < 0{\cdot}001$). There was no difference in 30-day mortality (high rEVAR 28·4 vs. low rEVAR 26·9%; $p = 0{\cdot}695$). In the high rEVAR centres, 30-day mortality was 38·6% after open repair and 24·3% after rEVAR vs. 32·5% and 21·5% in the low rEVAR centres.

Conclusion: Centres with high rEVAR penetrance treated patients with more comorbidities, who were more stable pre-operatively. The primary treatment strategy at centre level did not affect the peri-operative survival.

Statin therapy is associated with reduced risk of abdominal aortic aneurysm rupture

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Objective: The incidence of AAA rupture is declining and may be partly related to increased prescription of statins. This study investigated the association between statin therapy and rupture in patients with AAAs.
Method: We retrospectively analysed a prospectively maintained database of 1098 consecutive AAA patients. These patients were either admitted with ruptured AAAs or for repair of intact large AAAs in a single tertiary unit (2004–2010). Patients were assessed as to whether they were taking statins prior to diagnosis of ruptured or intact AAAs. Patients that have been on a surveillance programme (n = 109) were excluded.
Results: There were 315 ruptured (134 unoperated) and 674 intact large AAAs with no prior surveillance. Patients who received statin therapy prior to AAA diagnosis were significantly less likely to present with ruptured AAA (odds ratio [OR] = 0·33; 95% CI, 0·23–0·48; p = 0·0001). Adjustment for risk factors for rupture (size, female gender, smoking, age, hypertension) and comorbidities (ischaemic heart disease, diabetes mellitus, chronic renal failure and cerebrovascular disease) produced similar results (0·56; 0·35–0·90; p = 0·016). With the exception of smokers (0·79; 0·30–2·0; p = 0·62), statins consistently conferred protection in analyses of subgroups at risk of rupture: older patients > 75 years (0·45; 0·29–0·71; p = 0·001), females (0·32; 0·14–0·72; p = 0·006) and hypertensive patients (0·44; 0·23–0·83; p = 0·011). Uptake of statin therapy amongst patients on a surveillance programme was only 36·7%.
Conclusion: Statin therapy is associated with a reduced risk of AAA rupture in addition to its other known beneficial effects in AAAs. Measures to improve the uptake of statins in patients with AAAs should be instituted.

The practice of UK vascular surgeons with regard to informed consent

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Objective: In clinical negligence litigation, it is a defence to an allegation of failure to warn of a particular risk pre-operatively to show that the practice adopted would be supported by a responsible body of surgeons in the relevant field. This study aimed to find out what risks UK vascular surgeons do and do not warn patients about.
Method: Questionnaires were posted to all UK-based members and associate members on The Vascular Society's mailing list. Respondents were asked to complete the section of a standard NHS consent form beginning "Serious or frequently occurring risks" as they normally would when seeking consent from an averagely fit patient undergoing open AAA repair, EVAR, carotid endarterectomy, below-knee femoro-popliteal bypass and high tie and stripping for varicose veins.
Results: 516 questionnaires were distributed and 196 replies were received (38%). The proportion who gave a written warning of possible peri-operative death was 83% (OAAA), 65% (EVAR), 60% (CEA), 40% (fem-pop) and 0% (VVs). Warnings regarding all other complications of all five operations were given by less than 50% of surgeons, except for bleeding, infection, limb loss (fem-pop only) and venous thrombo-embolism (VVs only). In aneurysm patients, many complications which are common, or which are uncommon but serious, were warned about by less than 20% of surgeons, including renal ischaemia/failure (18%), incisional hernia (17%), paraplegia (9%) and late adhesion obstruction (0%).
Conclusion: The practice of UK vascular surgeons in seeking informed consent is unlikely to meet the requirements of the GMC and, perhaps, of the Courts. The legal and ethical requirements relating to consent require clarification.

Surgical nurse practitioners and core surgical trainees: a word of warning at a time of major service reconfiguration

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Objective: Surgical nurse practitioners (SNPs) can be trained to perform specific operative tasks such as vein harvesting or wound closure and are popular in cardiac surgery. They are reliable, without on-call commitments, whilst core surgical trainees (CSTs) are increasingly required for service duties. We report the effect of SNPs on CST surgical exposure and present to The Vascular Society a word of warning at a time of significant service reconfiguration.
Method: Operative experience for CSTs who rotated through one department that used SNPs and one that did not, in the same year of training was collected from their Intercollegiate Surgical Training Programme (ISCP) Logbook. The total number of operations, the number as assistant and the number performed (either supervised [STS/STU] or independently [P]) were compared based on the presence of SNPs (SNP− vs. SNP+). Results are presented as a median (IQR) with non-parametric statistics.
Results: Six CSTs (four CT1s, two CT2s) were included. The total operative experience was SNP−: 85 (84–89·75) cases vs. SNP+: 18 (12·5–19·75) cases; p = 0·004. The number of operations assisting was SNP−: 55·5 (47·5–60·5) cases vs. SNP+: 11·5 (10·25–18) cases; p = 0·02. The number of operations performed (either supervised or unsupervised) was SNP−: 27 (24–36) cases vs. SNP+: 3·5 (0·75–4·75) cases; p = 0·004.
Conclusion: SNPs occupy the traditional SHO role in the operating theatre but will never progress to provide a higher-level surgical service. They dramatically reduce the quantity and quality of operative training opportunities for CSTs. We urge The Vascular Society to consider this when planning service reconfiguration to ensure training of young surgeons remains high on the agenda.

Area of treatment independently predicts treatment and outcome for peripheral vascular disease in the UK population after controlling for demographic and disease risk factors. Analysis of English Hospital Data 2003–2009

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Objective: To investigate whether area of treatment independently predicted those having a lower limb amputation without revascularisation.
Method: Hospital Episode Statistics (2003–2009) were used to determine numbers of procedures as well as risk factors. Prevalence rates, per 100,000 (95% CI) of major lower limb amputation and revascularisation (endovascular and surgical) were calculated using census data in those aged 50–84. These procedural data were case-matched to define those having amputations without revascularisation within the 6-year period.
Results: Between 2003 and 2009 there were 21,056 amputations and 109,079 revascularisations. The prevalence rate, per 100 000, of amputations was over double in males (32; 31·2–32·2) compared with females (13; 12·8–13·5). Revascularisation rates showed a similar pattern (males 160; 158·6–160·9; females 72; 71·5–73·0). Logistic regression demonstrated that after controlling for demographic (age, sex, social class) and disease risk factors (diabetes, hypertension, hypercholesterolaemia, smoking, previous coronary heart disease or stroke), location independently predicted those having major lower limb amputation without prior revascularisation; areas of significance were the North East, South East, Midlands and East Midlands.
Conclusion: Area of treatment independently predicted those having amputation without revascularisation. Reasons for this geographical difference warrant further investigation.

Validation of five risk prediction models for mortality in 10,891 elective AAA repairs from the National Vascular Database

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Objective: There is no consensus on which risk prediction model for mortality following elective AAA repair should be used in the UK. Our objective was to

assess the performance of five risk prediction models for elective AAA repair using the National Vascular Database (NVD).

Method: Data held on the NVD (excluding the North West of England) between January-2008 and December-2010 were analysed. The Glasgow Aneurysm Score (GAS), physiological component of the Vascular Physiological and Operative Severity Score for enUmeration of Mortality (V-POSSUM), Vascular Biochemical and Haematological Outcome Model (VBHOM), Medicare and Vascular Governance North West (VGNW) models were tested. Model performance was assessed by area under the receiver operating characteristic (ROC) curve and ability to predict observed risk in low-, medium- and high-risk sub-groups.

Results: Data from 10,891 elective AAA repairs were analysed (mean age 74, 87·3% men). The in-hospital mortality rates following endovascular repair and open repair were 1·3 and 4·7%, respectively (2·9% overall). The Medicare and VGNW models both showed good discrimination (ROC = 0·71), while the GAS, VBHOM and V-POSSUM showed poor discrimination (ROC = 0·60, 0·61 and 0·62, respectively). The VGNW model was the only model to accurately predict risk in low- (1·7% predicted vs. 1·7% observed; p = 0·899) and medium-risk groups (4·7% predicted vs. 4·3% observed; p = 0·555), but over predicted risk in the high-risk group (10·6% predicted vs. 7·6% observed; p = 0·009).

Conclusion: The Medicare and VGNW models contain similar risk factors and showed good discrimination when applied to the NVD. Both models would be suitable for risk prediction in elective AAA repair in the UK.

Ethnicity independently predicts major lower limb amputation without revascularisation after controlling for demographic and disease risk factors. Analysis of English Hospital Data 2003–2009

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Objective: To document the prevalence of leg amputation and revascularisation by ethnic group aged 50–84 and determine whether ethnicity independently predicted amputation without revascularisation after controlling for known atherosclerotic disease risk factors.

Method: English hospital (HES) and census data were used to calculate age standardised prevalence and proportional rates (95% CI). Cases were matched to identify those having amputation without revascularisation between 2003 and 2009. Demographic (age, sex, social class, area of treatment), disease risk factors (diabetes, hypertension, high cholesterol, history of coronary heart disease or stroke and smoking) and ethnicity (White British, Asian, Black) as documented by HES were compared with the White British population (WB).

Results: Between 2003 and 2009 there were 21,056 amputations and 109,079 revascularisations. The prevalence rate per 100,000 in WB was: leg amputation (males = 26; females = 19), revascularisation (males = 128; females = 93). In men, the proportional rate (WB = 100) of amputation was one third higher in Blacks (136; 117–155) and half in Asians (55; 47–64). Asian women also experienced half the rate of amputation (49; 35–63) and a lower rate of revascularisation (74; 67–81). However, Black women had 2·6 times the rate of amputation (261; 215–306) with no significant difference in revascularisation. Ethnicity independently predicted those having amputation without revascularisation after controlling for demographic and disease risk factors.

Conclusion: Blacks and Asians experience significantly different rates of leg amputation and revascularisation, with ethnicity independently predicting amputation without revascularisation after controlling for known risk factors. The implication of this finding warrants further study.

A dedicated multidisciplinary amputee service achieves improved patient experience and reduces length of stay

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Objective: To assess the clinical and patient outcomes of a dedicated amputee service introduced to a vascular unit in January 2008.

Method: Retrospective appraisal of a prospective database (2008–2010) according to the guidelines of the Quality Improvement Framework for major amputation surgery.

Results: Ninety-seven major lower limb amputations were performed from January 2008 to December 2010: 66 men, 31 women, aged 43–87 years (mean 65·8). All pre-operative framework criteria were met. Peri-operative compliance was achieved with respect to timing of operation and appropriate level of surgeon and anaesthetist. All patients were assessed pre-operatively and immediately postoperatively by physiotherapists, specialist counsellors and occupational therapists, with twice daily access to the rehabilitation team. A weekly Amputee Support Group was instigated in 2008 for patients and relatives. Fifty-five below-knee amputations and 42 above-knee amputations were performed. This complies with the framework objective of below: above-knee ratio > 1. Patient satisfaction questionnaires demonstrated consistently high levels of satisfaction with the amputee service and support offered to relatives. Hospital length of stay was reduced from 126 days (in 2005) to 46 days in 2008 to 23 days in 2010. In-hospital mortality was 9% (2008–10). The dramatic reduction in bed days made the introduction of this service cost neutral.

Conclusion: The dedicated amputee service has achieved a significant reduction in length of stay and improvement in patient satisfaction. We need to continue to audit our practice to achieve the aims of improving the above- to below-knee amputation ratio and of reducing mortality to less than 5%.