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Background There has been a drive to change access site practice for PCI with the aim of reducing access site complications. This is increasingly important with the shift to more acute PCI with use of more potent antithrombotics. With the rapid uptake of transradial access in the UK we looked for changes in outcome using nationally collected data from the UK BCIS PCI dataset.

Methods Retrospective analysis of data submitted from all UK PCI centres between January 2006 and September 2011. Incomplete data and cases where multiple access sites were recorded were excluded.

Results Of 413 146 documented procedures a single access was recorded in 92.7%. The two cohorts, transradial and transfemoral were well matched for age. Transradial patients had less diabetes and fewer previous CABG but were more likely to be smokers, hypertensive, hyperlipidaemic and male. The use of transradial access increased from 17.2% in 2006 to 57% in 2011. Over this time PCI for ACS increased from 47% to 63% procedures, with the same increase in the proportion of transradial access. Primary PCI increased from 6% of all PCI to 25% in 2011. For femoral procedures mortality more than doubled (0.55% in 2006 to 1.36% in 2011) while for transradial procedures mortality only increased from 0.42% to 0.52%. When shock and IABP use are excluded the reduction in mortality for transradial PCI remains though the difference is reduced.

Conclusion The rapid change from femoral to radial access in the UK has been accompanied by a reduction in access site complications. As mortality and adverse outcomes have increased in keeping with patient and PCI complexity the rate of adverse outcomes in the patients treated radially has remained stable and lower than in the femoral group.

Abstract 047 Figure 1 In-patient outcomes following PCI January 2006 to September 2011.

Abstract 047 Table 1

	Radial	Femoral	p Value
Total number	14 6358	236 098	
Age (years)	64.04	64.85	
Diabetes	24.36%	25.25%	<0.0001
Hypertension	50.83%	47.65%	<0.0001
Hyperlipidaemia	54.98%	51.93%	<0.0001
Current smoker	24.61%	18.93	<0.0001
Never smoked	30.85%	29.34%	<0.0001
PVD	4.96%	3.94%	<0.0001
Previous MI	24.36%	25.25%	<0.0001
Previous CABG	4.83%	10%	<0.0001
Previous PCI	18.37%	20.67%	<0.0001
Previous stroke	3.93%	3.21	<0.0001

Abstract 047 Table 2

	Radial (%)	Femoral (%)	p Value
Death	0.47	0.86	<0.0001
MACCE	1.25	1.68	<0.0001
Blood Transfusion	0.05	0.13	<0.0001
Arterial Complication	0.20	0.49	<0.0001
Stroke	0.10	0.09	<0.0001

048

IS IT SAFE TO DISCHARGE PATIENTS 24 H AFTER UNCOMPLICATED SUCCESSFUL PRIMARY PERCUTANEOUS CORONARY INTERVENTION?

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Background Primary percutaneous coronary intervention (PPCI) has led to improved mortality, reduced rates of recurrent ischaemia and shorter hospital stays compared to thrombolysis. Data from our centre published previously show that in low-risk patients very early discharge at 48 h is feasible and safe. We investigated whether in a group of low risk patients stratified to 48-h discharge, 24-h discharge would be potentially feasible.

Methods We undertook an observational study at an interventional cardiology centre involving 2980 patients who underwent Primary PCI from January 2004 to July 2011. Patients with TIMI 3 flow, ST segment resolution, good or moderate left ventricular function, and no dysrhythmia post-PPCI were discharged at 2 days. Remaining higher risk patients were discharged when judged clinically fit. All patients were offered outpatient review by a multidisciplinary team. Follow-up was collected for a median of 2.8 years (IQR 1.3–4.4 years).

Results Of the 2980 patients, 1174 (39.4%) were judged suitable for 48-h discharge. Of these, 964 patients (82.1%) were discharged at 48 h, with 210 discharged after longer inpatient stays. Of these 210 patients discharged after 2 days, 150 were delayed due to timing issues (admission at unsociable hours, eg, 01:00). 60 (5.1%) patients fitting criteria had their planned 48-h discharge delayed due to a clinical complication, of which 53 occurred within the first 24 h (including six MACE events and seven arrhythmias, there were no deaths) (Abstract 048 table 1). Only seven patients (0.60%) developed complications after 24 h, of which only 1 (0.09%) suffered a MACE event (target vessel revascularisation), with the remaining complications being abnormal blood tests (renal/liver function) or drug reactions (eg, rash). There were no in-hospital deaths in the 48-h group.

Abstract 048 Table 1 Reason for prolonged admission in patients initially selected for 48-h discharge

Reason	First 24 h	After 24 h	Total
MACE*	5 (0.42%)	1 (0.09%)	6 (0.51%)
Thrombocytopenia/bleeding	15 (1.28%)	0 (0%)	15 (1.28%)
Haematoma	2 (0.17%)	0 (0%)	2 (0.17%)
Renal/liver dysfunction	2 (0.17%)	4 (0.36%)	6 (0.51%)
Infections and lung disease	7 (0.60%)	0 (0%)	7 (0.60%)
Further chest pain/STE (non-MACE)	5 (0.43%)	0 (0%)	5 (0.43%)
Arrhythmia	6 (0.51%)	1 (0.09%)	7 (0.60%)
CCF/Pulmonary oedema	2 (0.17%)	0 (0%)	2 (0.17%)
Other	9 (0.76%)	1 (0.09%)	10 (0.85%)
Total	53 (4.50%)	7 (0.60%)	60 (5.10%)

*MACE denotes major adverse cardiac events.

Conclusion Simple clinical criteria can be used to identify low-risk patients suitable for very early discharge 48 h following uncomplicated successful primary PPCI. With only a small percentage of complications occurring after the first 24 h, discharge after 24 h may be safe and warrants further study.

049 THE ROLE OF THE NEURAL NETWORKS IN IDENTIFICATION AND ABLATION OF PULMONARY VEIN ECTOPIC TRIGGERS

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Introduction The cardiac neural network comprises ganglionated plexi (GP) adjacent to each pulmonary vein (PV) which can trigger AV block and PV ectopy. We hypothesised that AV block in response to GP stimulation is mediated through the right lower GP (RLGP) and could be abolished by ablation of the RLGP, whereas a local PV ectopy response could be abolished by selective local GP ablation.

Methods and Results 10 patients undergoing left atrial (LA) ablation with autonomic modification were recruited. Continuous HFS was delivered endocardially at presumed GP sites using a Grass Stimulator (20 Hz, 10 ms pulse duration, 10 V). GP sites with connections to the AV node (producing AV block) were marked on LA geometry. RLGP group (N=5) had RF ablation of RLGP and 2 of 15 sites remained positive ($p<0.001$) demonstrating that ablation of the RLGP can disrupt the neural connections to the AV node. The other group (N=5) had RF ablation of non-RLGP sites followed by HFS retesting of all previously positive unablated sites. All 19 unablated GP sites were positive after ablation of target the GP site indicating that the neural connections to the AV node remained intact. A further 18 patients in sinus rhythm at the time of AF ablation were recruited. Short bursts of HFS (12 V, 50 Hz, 10 ms pulse width), synchronised to local refractory period, were delivered at presumed GP sites during fixed rate pacing. Positive sites initiating PV ectopy were recorded on the LA geometry and presumed to have PV connections. In a control group (N=3) to check reproducibility, 10 positive sites were identified and all 10 remained positive following catheter relocation and retesting. In the PVI group (N=8), 36 sites were positive, PVI was performed in all four veins without targeting GP sites during ablation. No sites were positive on retesting proving neural connections to the PV could be disrupted by ablation between the GP and PV. Finally in the GP ablation group (N=7), 29 sites were positive, two patients remained in AF, therefore further testing was not possible. RF ablation was performed around positive site and on retesting, five of 27 sites were positive. After additional RF ablation at these sites, no further ectopy could be initiated on retesting. This indicated that the GP connections into PVs could also be rendered ineffective by GP ablation.

Abstract 049 Figure 1 Effect of other GP site ablation on RR interval change following HFS at distant GP sites. Effect of RLGP ablation on RR interval change following HFS at distant GP sites.

Conclusions Our study shows that neural inputs from LA GPs act on the AV node via the RLGP "gateway" and if AV block is used to identify GP sites for autonomic modulation, intact connections via the RLGP are required. We propose using synchronised HFS to identify and ablate GP sites initiating ectopic firing from the PV, as it only requires intact local connections to the PV. PVI acutely prevents this ectopic activity, possibly by transecting neural inputs to the veins. However, reconnection of PVs is common and RF ablation of the GP site alone may prevent ectopic firing within PVs.

Abstract 049 Figure 2 Number of sites initiating PV ectopy before and after intervention in the GP ablation group.

050 AUTOMATED ANALYSIS OF ATRIAL DELAYED ENHANCEMENT CARDIAC MRI CORRELATES WITH VOLTAGE, AF RECURRENCE POST-ABLATION, AND HIGH STROKE RISK

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Introduction Visualisation of atrial scar using delayed-enhanced MRI (DE-MRI) may reveal causes for atrial fibrillation recurrence following ablation. To develop an objective method for delineating

Abstract 050 Figure 1 Voltage type. ** $p<0.001$, * $p<0.005$ compared to preceding intensity level.