Mechanical Circulatory Support in Cardiogenic Shock – What every cardiologist needs to know The Surgeon's view

ACCA Masterclass 2017

R Trimlett (London, UK)

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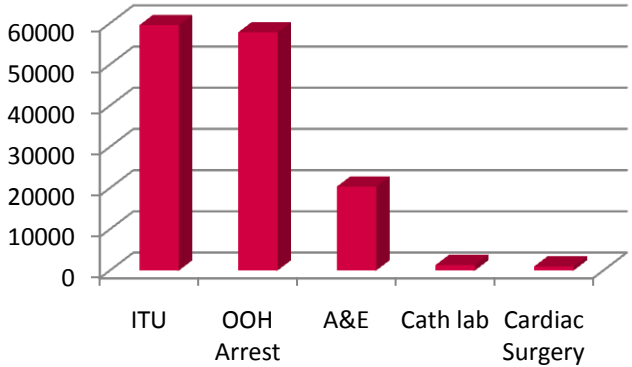
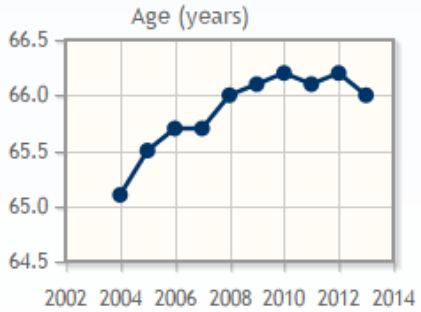
Potential for Cardiac Support

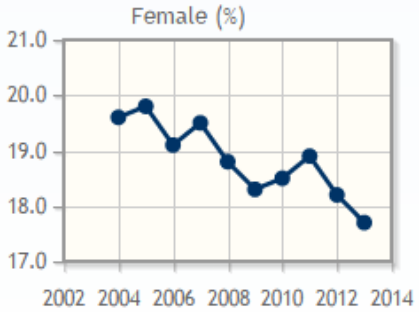
	TOTAL	*DIED*
Cath Lab	25,011	1,317
Cardiac Surgery	36,134	990
Intensive Care	238,248	59,562
Accident & Emergency	18,142,311	20,358
Ambulance OOH Arrests	60,000	57,800
Overall UK Mortality	**501,424**	

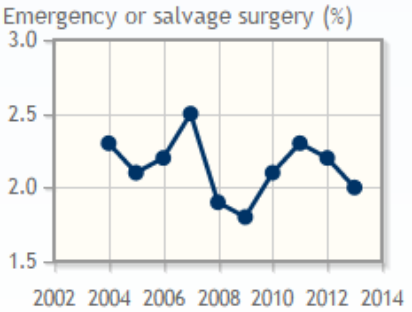


Isolated first-time CABG (overall cohort)

Year	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013
Age (years)	65.1	65.5	65.7	65.7	66.0	66.1	66.2	66.1	66.2	66.0
Female (%)	19.6	19.8	19.1	19.5	18.8	18.3	18.5	18.9	18.2	17.7
Emergency or salvage surgery (%)	2.3	2.1	2.2	2.5	1.9	1.8	2.1	2.3	2.2	2.0







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Cardiogenic shock (CS) is the leading cause of death for patients with acute myocardial infarction (MI) who reach the hospital alive. Its incidence has remained constant for 20 years.[1,2] Rapidly re-establishing infarct-related artery (IRA) blood flow is essential in the management of patients with shock due to right ventricular or left ventricular (LV) failure. A strategy of early revascularization is superior to initial aggressive medical therapy.[3–5] Despite the advantages of early percutaneous coronary intervention (PCI) or coronary artery bypass graft surgery (CABG), once shock is diagnosed, the mortality rate remains high (≈50%) despite intervention, and half of the deaths occur within the first 48 hours.

CLINICIAN UPDATE

Cardiogenic Shock Complicating Acute Myocardial Infarction

Expanding the Paradigm

Judith S. Hochman, MD

Classic Shock Paradigm

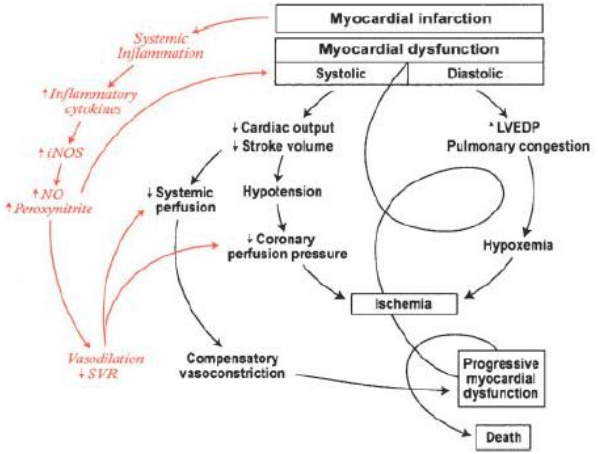
The underlying pathophysiology of CS is profound depression of myocardial contractility, resulting in a vicious spiral of reduced cardiac output (CO), low blood pressure, further coronary insufficiency, and further reduction in contractility and CO. The classic paradigm predicts that compensatory systemic vasoconstriction with high systemic vascular resistance (SVR) should occur in response to the depression of CO (Figure 1).[9]

Autopsy studies have shown that the pathological basis of CS is extensive MI. Varying pathological stages of infarction confirm the stuttering and progressive nature of the myocardial necrosis as a corollary of the vicious spiral. Combined new and old infarctions consistently involve at least 40% of the LV myocardium in these autopsy specimens.[10]

Observations That Challenge the Classic Paradigm

There are several observations derived from the SHOCK (SHould we emergently revascularize Occluded Coronaries in cardiogenic shocK?) trial and registry about patients with CS due to LV failure not easily explainable by our traditional concepts. These include the following:

- Average LV ejection fraction (EF) is only moderately severely depressed (30%), with a wide range of EFs and LV sizes noted.
- SVR on vasopressors is not elevated on average, with a very wide range of SVRs measured.
- A clinically evident systemic inflammatory response syndrome is often present in patients with CS.
- Most survivors have class I congestive heart failure (CHF) status.



European Heart Journal (2004) 25, 322–328

ELSEVIER

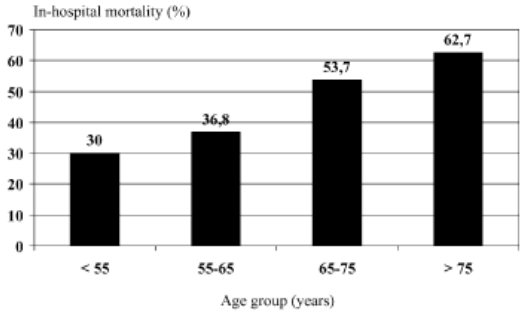
EUROPEAN SOCIETY OF CARDIOLOGY

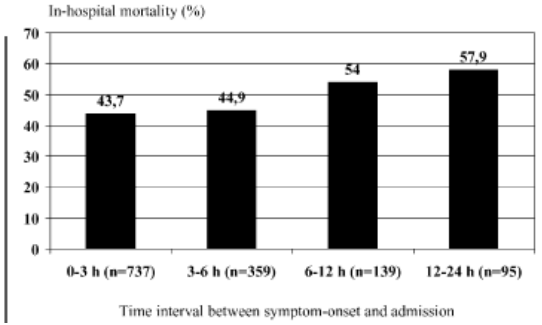
Clinical research

Predictors of in-hospital mortality in 1333 patients with acute myocardial infarction complicated by cardiogenic shock treated with primary percutaneous coronary intervention (PCI)

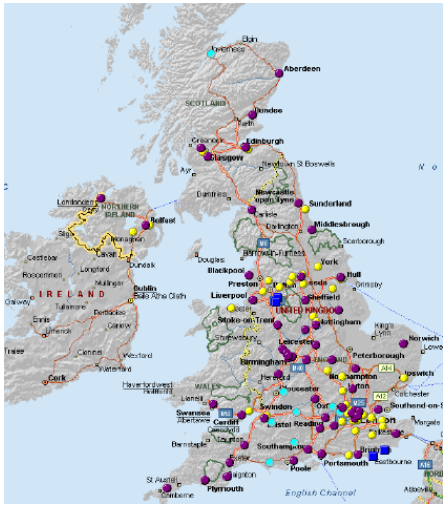
Results of the primary PCI registry of the Arbeitsgemeinschaft Leitende Kardiologische Krankenhausärzte (ALKK)[1]

Uwe Zeymer[a*], Albrecht Vogt[b], Ralf Zahn[a], Michael A. Weber[c], Ulrich Tebbe[d], Martin Gottwik[e], Tassilo Bonzel[f], Jochen Senges[a], Karl-Ludwig Neuhaus[b], for the Arbeitsgemeinschaft Leitende Kardiologische Krankenhausärzte (ALKK)



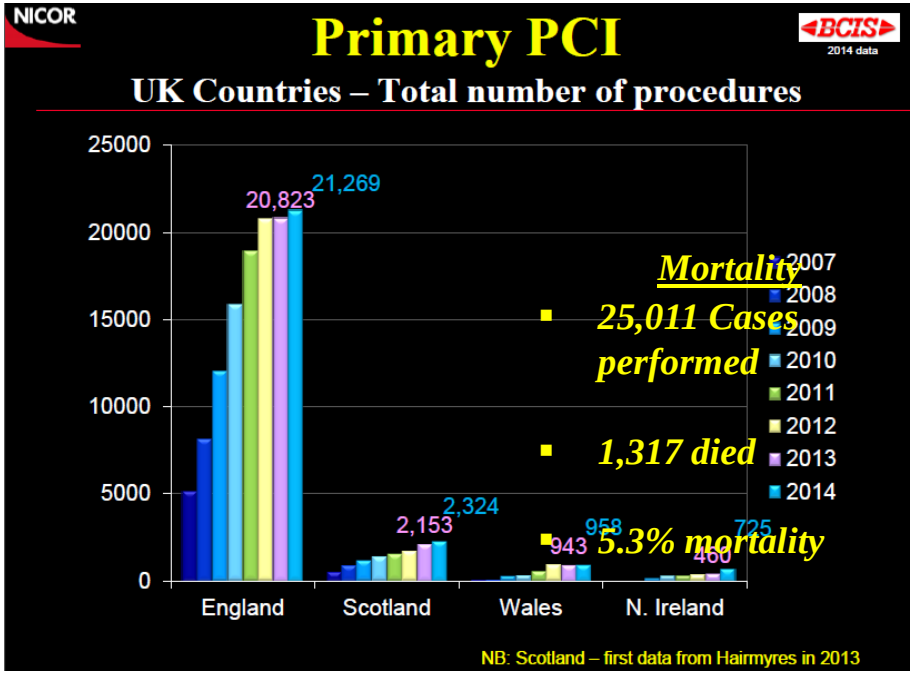


Catheter Labs



81 Centres providing at least a working-hours Service.

22 performing less than 400 cases per year.



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Out of Hospital Cardiac Arrest

	Ventilated before or during	n with data	No Ventilation	n with data
OOHA cases (n)	556		527	
Cooling (%)	40.9	479	2.6	421
pH (mean)	7.14	320	7.3	100
pH (min)	6.0	18.0	7.0	
pH (max)	7.52	9.1	7.6	
Lactate (Mean)	6.7	270	4.1	85
30 day mortality (%)	47.6	494	6.9	480

= 1,083

BCIS

IABP – SHOCK II Trial

The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 OCTOBER 4, 2012 VOL. 367 NO. 14

Intraaortic Balloon Support for Myocardial Infarction with Cardiogenic Shock

Holger Thiele, M.D., Uwe Zeymer, M.D., Franz-Josef Neumann, M.D., Miroslaw Ferenc, M.D., Hans-Georg Olbrich, M.D., Jörg Hausleiter, M.D., Gert Richardt, M.D., Marcus Hennersdorf, M.D., Klaus Empen, M.D., Georg Fuernau, M.D., Steffen Desch, M.D., Ingo Eitel, M.D., Rainer Hambrecht, M.D., Jörg Fuhrmann, M.D., Michael Böhm, M.D., Henning Ebelt, M.D., Steffen Schneider, Ph.D., Gerhard Schuler, M.D., and Karl Werdan, M.D., for the IABP-SHOCK II Trial Investigators*

RESULTS

A total of 300 patients in the IABP group and 298 in the control group were included in the analysis of the primary end point. At 30 days, 119 patients in the IABP group (39.7%) and 123 patients in the control group (41.3%) had died (relative risk with IABP, 0.96; 95% confidence interval, 0.79 to 1.17; P=0.69). There were no significant differences in secondary end points or in process-of-care measures, including the time to hemodynamic stabilization, the length of stay in the intensive care unit, serum lactate levels, the dose and duration of catecholamine therapy, and renal function. The IABP group and the control group did not differ significantly with respect to the rates of major bleeding (3.3% and 4.4%, respectively; P=0.51), peripheral ischemic complications (4.3% and 3.4%, P=0.53), sepsis (15.7% and 20.5%, P=0.15), and stroke (0.7% and 1.7%, P=0.28).

CONCLUSIONS

The use of intraaortic balloon counterpulsation did not significantly reduce 30-day mortality in patients with cardiogenic shock complicating acute myocardial infarction for whom an early revascularization strategy was planned. (Funded by the German Research Foundation and others; IABP-SHOCK II ClinicalTrials.gov number, NCT00491036.)

IABP – SHOCK II Trial

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Patients were not eligible for the study if they had undergone resuscitation for more than 30 minutes; had no intrinsic heart action; were in a coma with fixed dilatation of pupils that was not induced by drugs; had a mechanical cause of cardiogenic shock (e.g., ventricular septal defect or papillary muscle rupture); had onset of shock more than 12 hours before screening;

PATIENTS

Patients were eligible for the trial if they presented with an acute myocardial infarction (with or without ST-segment elevation) complicated by cardiogenic shock and if early revascularization (by means of PCI or CABG) was planned. A patient was considered to be in cardiogenic shock if he or she had a systolic blood pressure of less than 90 mm Hg for more than 30 minutes or needed infusion of catecholamines to maintain a systolic pressure above 90 mm Hg, had clinical signs of pulmonary congestion, and had impaired end-organ perfusion. The diagnosis of impaired end-organ perfusion required at least one of the following: altered mental status; cold, clammy skin and extremities; oliguria with urine output of less than 30 ml per hour; or serum lactate level higher than 2.0 mmol per liter.

123 patients in the control group (41.3%)

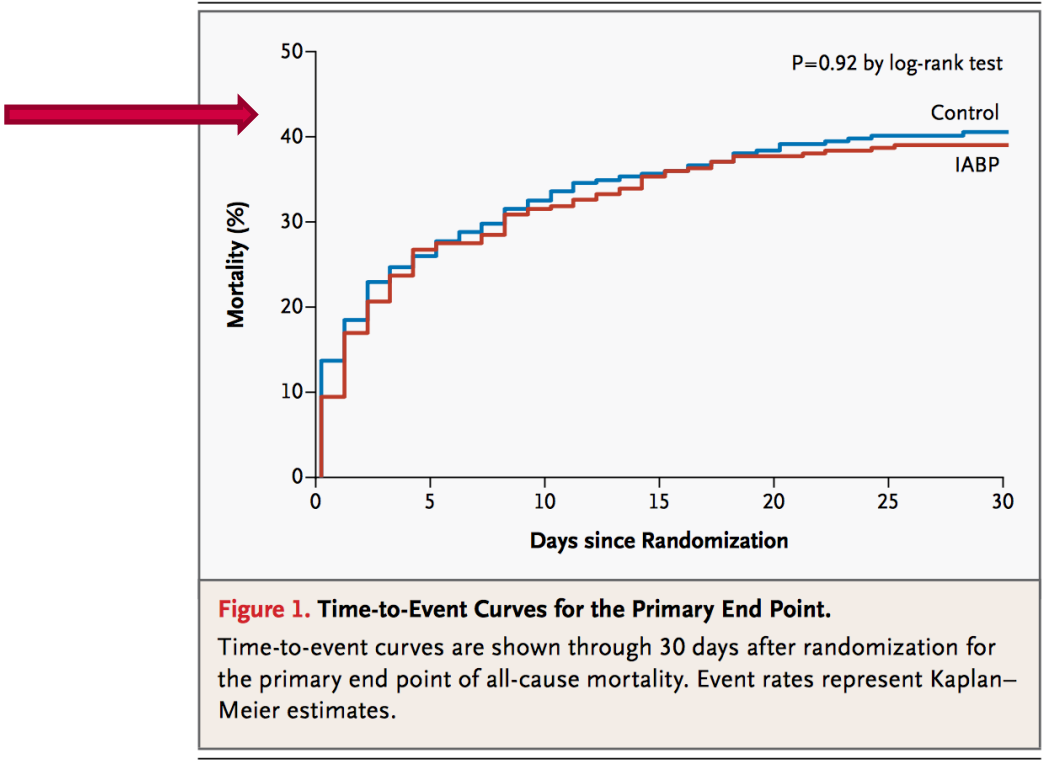

Figure 1. Time-to-Event Curves for the Primary End Point.

Time-to-event curves are shown through 30 days after randomization for the primary end point of all-cause mortality. Event rates represent Kaplan–Meier estimates.

IMPELLA DEVICE

Per-cutaneous / Surgical

2.5L / 5L+

Already anticoagulated.

May cause Haemolysis.

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WORKS IN PROGRESS

A Randomized Clinical Trial to Evaluate the Safety and Efficacy of a Percutaneous Left Ventricular Assist Device Versus Intra-Aortic Balloon Pumping for Treatment of Cardiogenic Shock Caused by Myocardial Infarction

Melchior Seyfarth, MD,*† Dirk Sibbing, MD,* Iris Bauer, MS,* Georg Fröhlich, MD,† Lorenz Bott-Flügel, MD,† Robert Byrne, MB, MRCPI,* Josef Dirschinger, MD,† Adnan Kastrati, MD,* Albert Schömig, MD*†

Munich, Germany

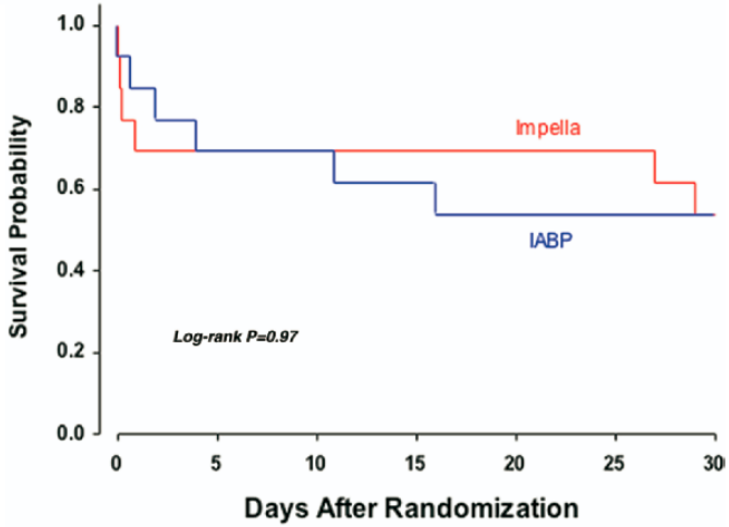
Objectives The aim of this study was to test whether the left ventricular assist device (LVAD) Impella LP2.5 (Abiomed Europe GmbH, Aachen, Germany) provides superior hemodynamic support compared with the intra-aortic balloon pump (IABP).

Background Cardiogenic shock caused by left ventricular failure is associated with high mortality in patients with acute myocardial infarction (AMI). An LVAD may help to bridge patients to recovery from left ventricular failure.

Methods In a prospective, randomized study, 26 patients with cardiogenic shock were studied. The primary end point was the change of the cardiac index (CI) from baseline to 30 min after implantation. Secondary end points included lactic acidosis, hemolysis, and mortality after 30 days.

Results In 25 patients the allocated device (n = 13 IABP, n = 12 Impella LP2.5) could be safely placed. One patient died before implantation. The CI after 30 min of support was significantly increased in patients with the Impella LP2.5 compared with patients with IABP (Impella: $\Delta CI = 0.49 \pm 0.46$ l/min/m^2; IABP: $\Delta CI = 0.11 \pm 0.31$ l/min/m^2; $p = 0.02$). Overall 30-day mortality was 46% in both groups.

Conclusions In patients presenting with cardiogenic shock caused by AMI, the use of a percutaneously placed LVAD (Impella LP 2.5) is feasible and safe, and provides superior hemodynamic support compared with standard treatment using an intra-aortic balloon pump. (Efficacy Study of LV Assist Device to Treat Patients With Cardiogenic Shock [ISAR-SHOCK]; NCT00417378) (J Am Coll Cardiol 2008;52:1584–8) © 2008 by the American College of Cardiology Foundation



Complex organ dysfunction scores (MODS and SOFA) were used to evaluate overall outcome. Reversal of the hemodynamic derangement resulted in better scores at 30 days in both groups without a significant difference between treatment arms. Explanation for the overall lack of a significant improvement in clinical outcome may be attributable to the protocol used, which left it to the discretion of the physician how long the mechanical device was used, after the primary end point was reached.

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Percutaneous Coronary Intervention for Cardiogenic Shock in the SHOCK Trial

John G. Webb, MD, FACC,* April M. Lowe, MS,† Timothy A. Sanborn, MD, FACC,‡ Harvey D. White, DSc,§ Lynn A. Sleeper, ScD,† Ronald G. Carere, MD, FACC,* Christopher E. Buller, MD, FACC,‖ S. Chiu Wong, MD, FACC,¶ Jean Boland, MD,# Vlad Dzavik, MD,** Mark Porway, MD, FACC,†† Gordon Pate, MB,* Geoffrey Bergman, MD, FACC,¶ Judith S. Hochman, MD, FACC,‡‡ for the SHOCK Investigators

Vancouver and Toronto, Canada; Watertown and Springfield, Massachusetts; Evanston, Illinois; Auckland, New Zealand; New York, New York; and Liege, Belgium

Table 4. Multivariate Cox Regression Results for One-Year Survival*

Parameter	Parameter Estimate	Standard Error	Hazard Ratio (95% CI)	p Value
Age (yrs)	0.077	0.020	2.17 (1.46, 3.22)†	< 0.001
Systolic blood pressure (mm Hg)‡	−0.025	0.010	0.78 (0.65, 0.94)†	0.009
Time from randomization to PCI (h)	0.253	0.108	1.29 (1.04, 1.59)	0.019
Final post-PCI TIMI flow (0/1 vs. 2/3)	2.385	0.614	10.86 (3.26, 36.20)	< 0.001
Multivessel PCI	1.012	0.494	2.75 (1.05, 7.25)	0.040

*Variables with significance $p < 0.05$ are shown (n = 76). †The hazard ratios and confidence intervals for age and systolic blood pressure are per 10-year or 10 mm Hg increase, respectively. ‡Measured while on support.
CI = confidence interval; PCI = percutaneous coronary intervention; TIMI = Thrombolysis In Myocardial Infarction.

Mitral insufficiency. The SHOCK study excluded enrollment of patients in whom the investigator determined that mitral valve replacement was clinically indicated. Among enrolled patients who had severe mitral regurgitation and underwent PCI alone, the one- year survival rate was a disappointing 33%. Mitral insufficiency is easily missed in shock patients and should be specifically sought with echocardiography or ventriculography before PCI. Although a reduction in mitral insufficiency may occur with PCI, this is unpredictable and infrequent. Severe mitral insufficiency may warrant early surgical correction.

Randomized Ischemic Mitral Evaluation (RIME) Trial

Conclusions

- Compared to CABG alone, addition of MV annuloplasty to CABG in patients with moderate functional ischemic MR improves:
  - Functional capacity and symptoms
  - LV reverse remodelling
  - Mitral regurgitation
  - BNP levels
- The impact of these benefits on longer term clinical outcomes remain to be defined.
- CABG plus MV annuloplasty required longer operation times, increased intubation and hospital stay duration, and blood transfusion.
- Concomitant CABG plus MV annuloplasty should be considered in patients with moderate functional ischemic MR.

Imperial College London

Compact CardioHelp VV / VA Portable Device

External Artificial Heart and Lungs

Uses of VA-ECMO

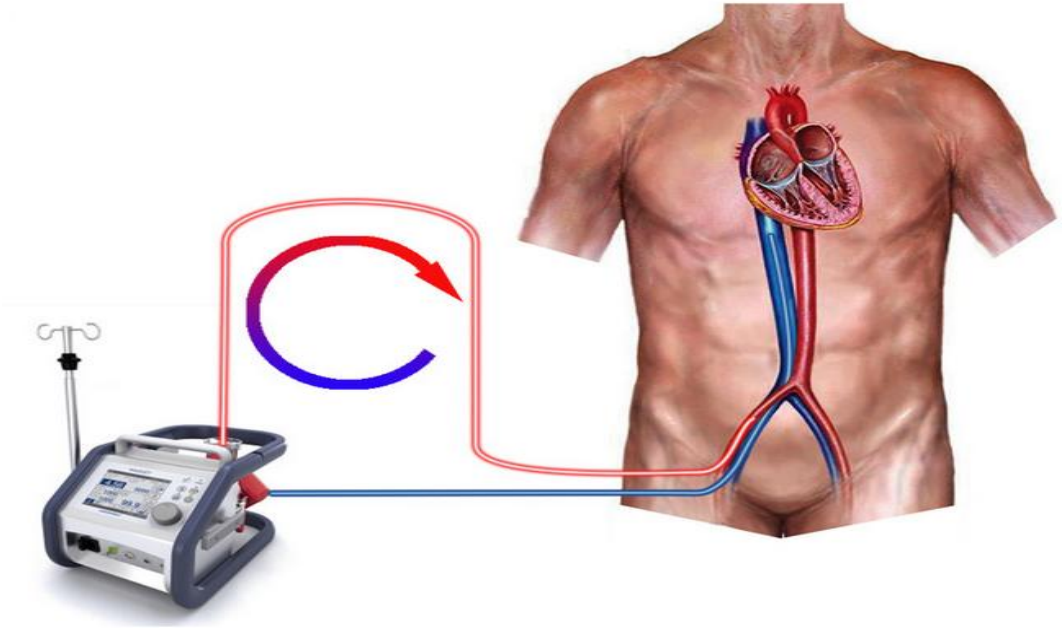
- Cardiogenic shock
- Large myocardial infarction (MI)
- Assistance with CPR using (E-CPR)
- Post-cardiotomy shock

- Bridge to more definitive treatment,
- Bridge to left ventricular assist device (LVAD)
- Bridge to decision

- Cardiomyopathic process
- Fulminant myocarditis
- Sepsis-associated cardiomyopathy

- Pulmonary hypertension
- Pulmonary embolism with right heart failure

- Class IV/stage D heart failure
- Post heart transplantation



CONSIDERATIONS FOR V-A ECMO CANNULATION

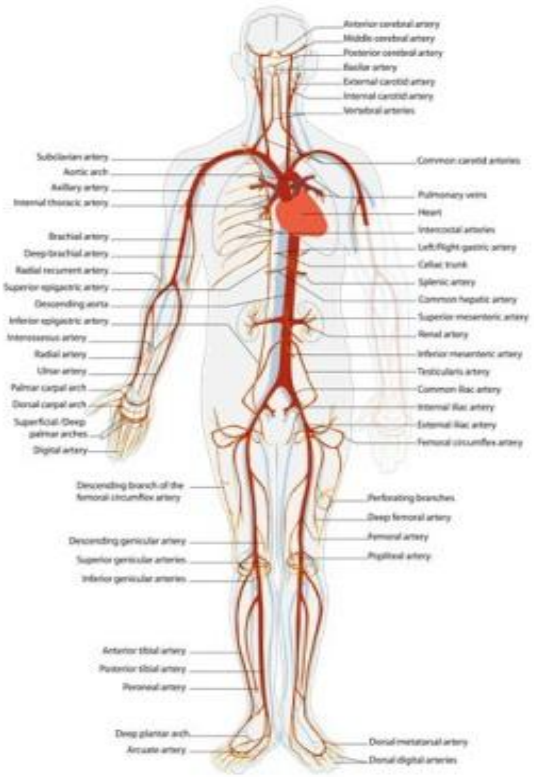
- Time / Urgency
- Facilities / Location
- Anatomical Considerations / Physical Size
- Previous or planned Surgery / Vascular Access

CONSIDERATIONS FOR V-A ECMO CANNULATION

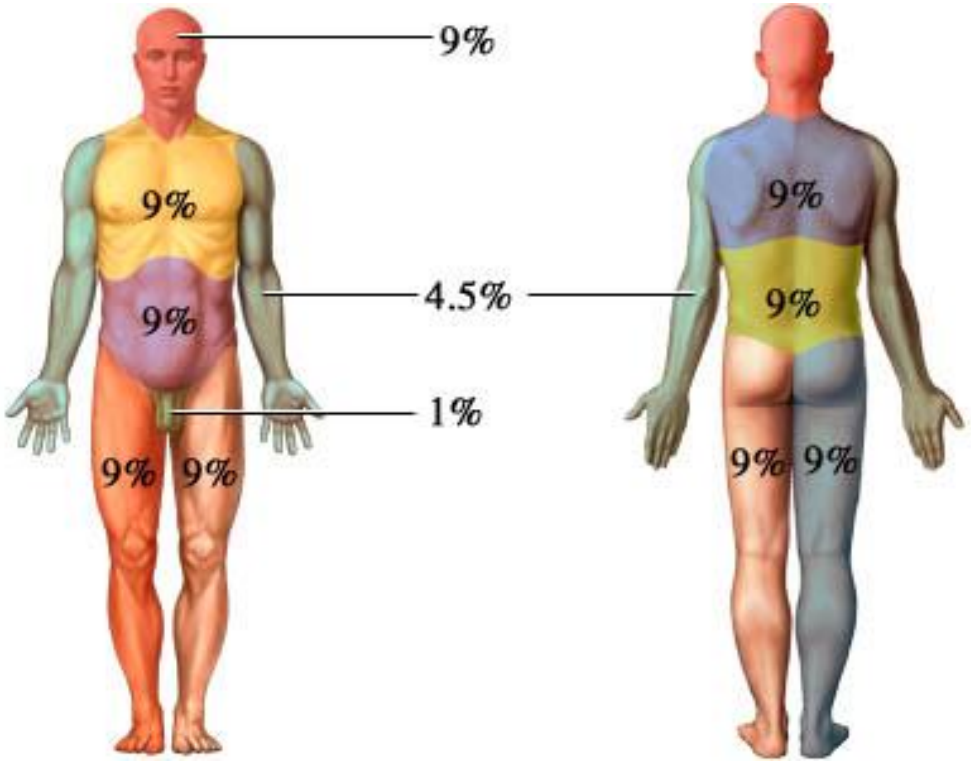
ARTERIAL ACCESS

Single Cannula or Multiple Cannulae

- Femoral
- Subclavian
- Aorta
- Left Ventricle
- Carotid



BODY SURFACE AREA – RULE OF NINES



PRESSURE OF TIME – TWO QUICKEST STRATEGIES (I)

1. FEMORAL CANNULATION

Ultrasound

Bilateral approach

Percutaneous vs. Open

Sterile Field

Small Cannulae

PRESSURE OF TIME – TWO QUICKEST STRATEGIES (II)

2. EMERGENCY STERNOTOMY

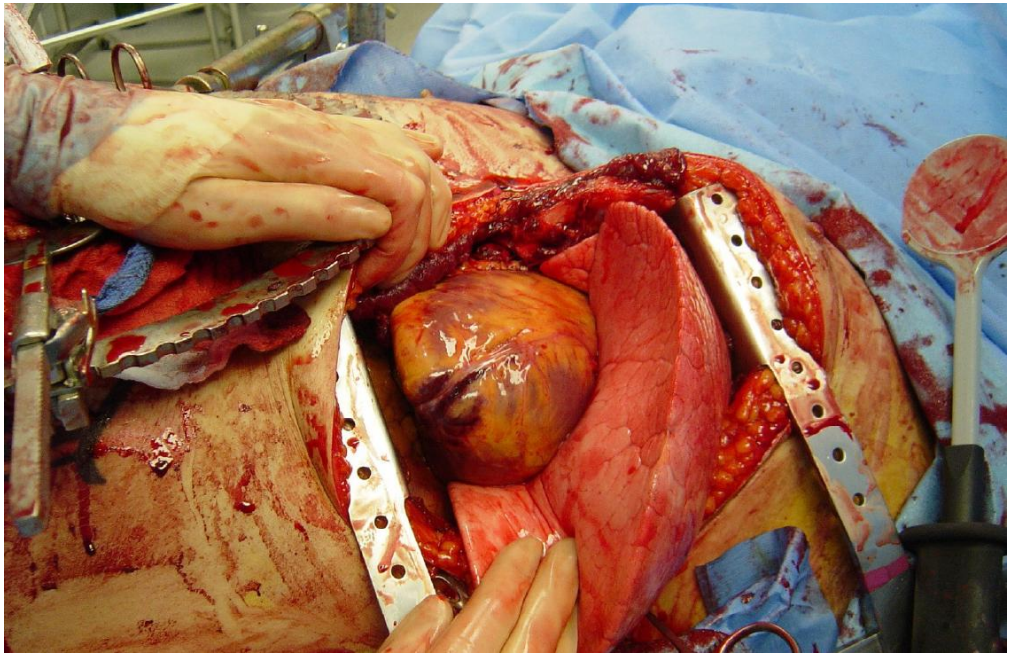
You will need a saw.

If you have a saw, this is very quick

Bleeding

Sterility

Transport

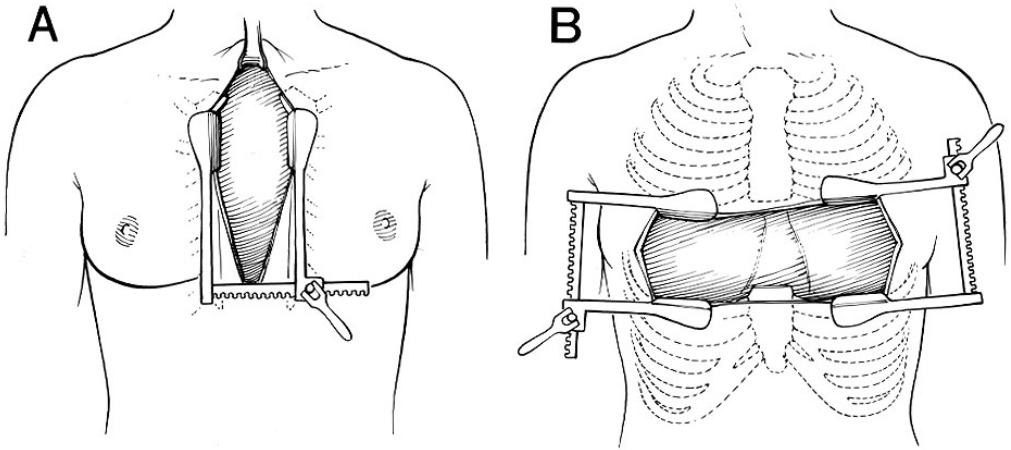


PRESSURE OF TIME – TWO QUICKEST STRATEGIES (II)

Sternotomy and ‘Clam shell’ incisions both give good emergency access to Heart and Great Vessels.

Clam shell can be done Without a saw.

Need two retractors for Best access.



FEMORAL CANNULATION

X-Ray Guided Approach

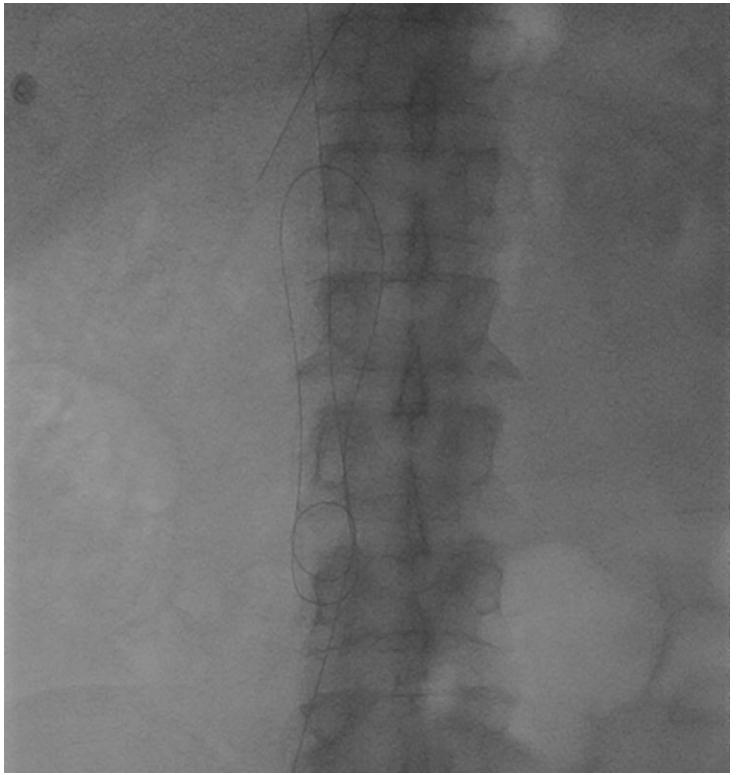
0.035” J-wire provided (soft)

Amplatz Super Stiff if prev. femoral op.

Dilate properly and incise skin

Wire can loop down opposite leg

Wire can enter Hepatic or renal veins



FEMORAL VENOUS CANNULATION

CANNULA CHOICE

Size

Multi-stage

Dual Drainage / Ascites

Cannula Positioning

FEMORAL ARTERIAL CANNULATION

CANNULA CHOICE

Size

Distal Perfusion

Side arm vent

Wire re-inforced

ALTERNATIVE FEMORAL ARTERIAL CANNULATION

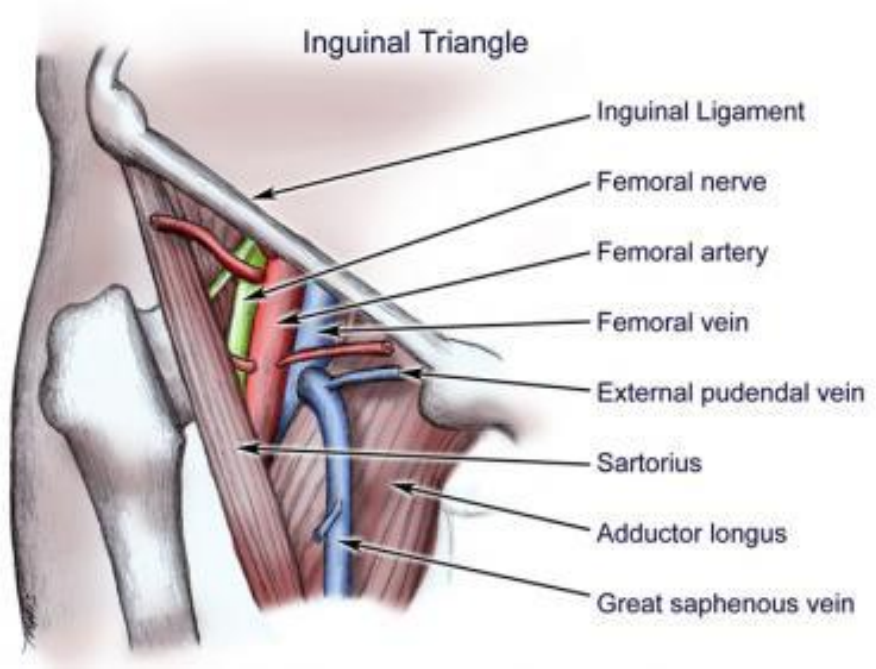
Division of Inguinal Ligament

10mm Side Graft to External Iliac A.

No Cannula Used

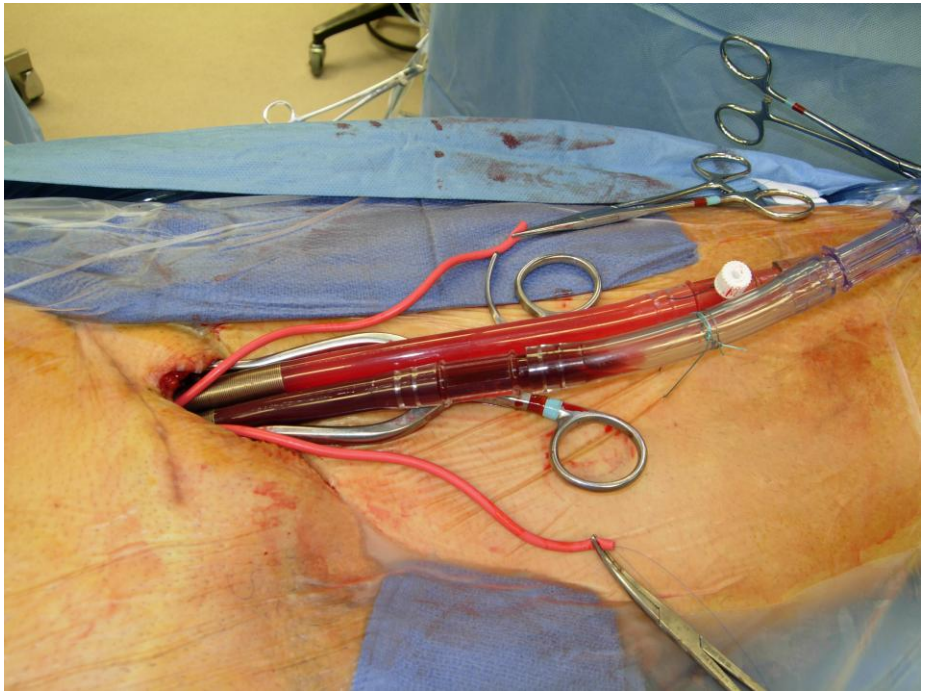
No Distal Perfusion Issues

Simple Decannulation



ALTERNATIVE FEMORAL ARTERIAL CANNULATION

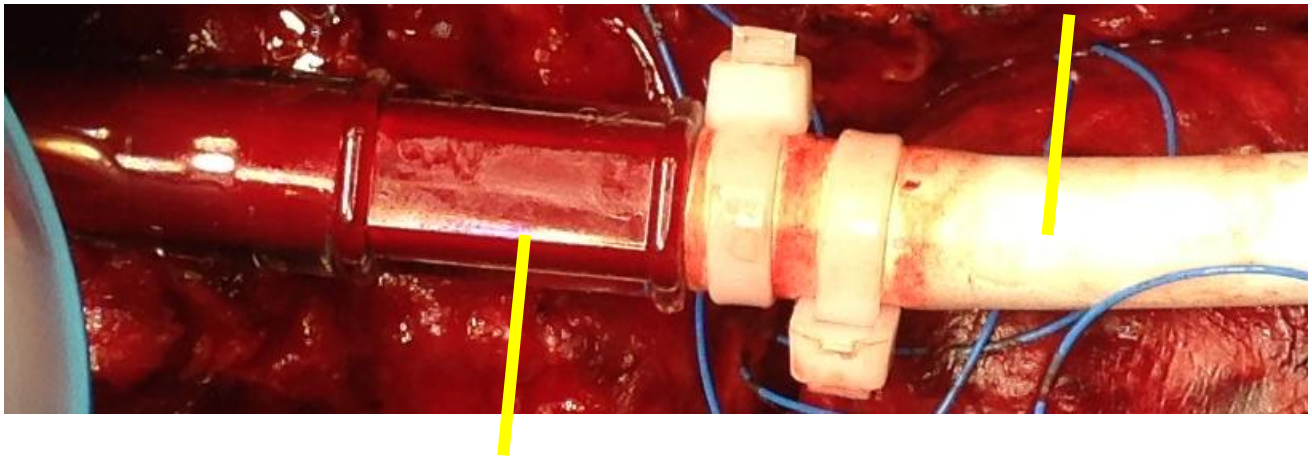
Conventional with Cannulae



Surgical Side Graft
10mm Gelseal

Technique for Cannulation

10mm Graft



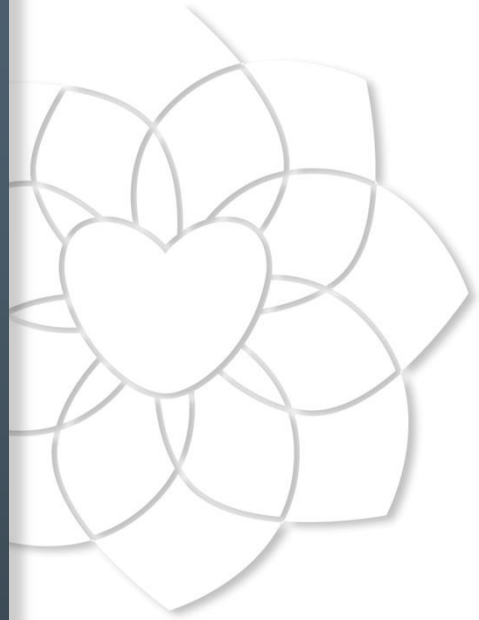
3/8” – 3/8” connector

3/8” = 9.56325mm

CONSIDERATIONS FOR V-A ECMO CANNULATION – FACILITIES

Accident and Emergency Resus.

Hybrid
Theatre Suite



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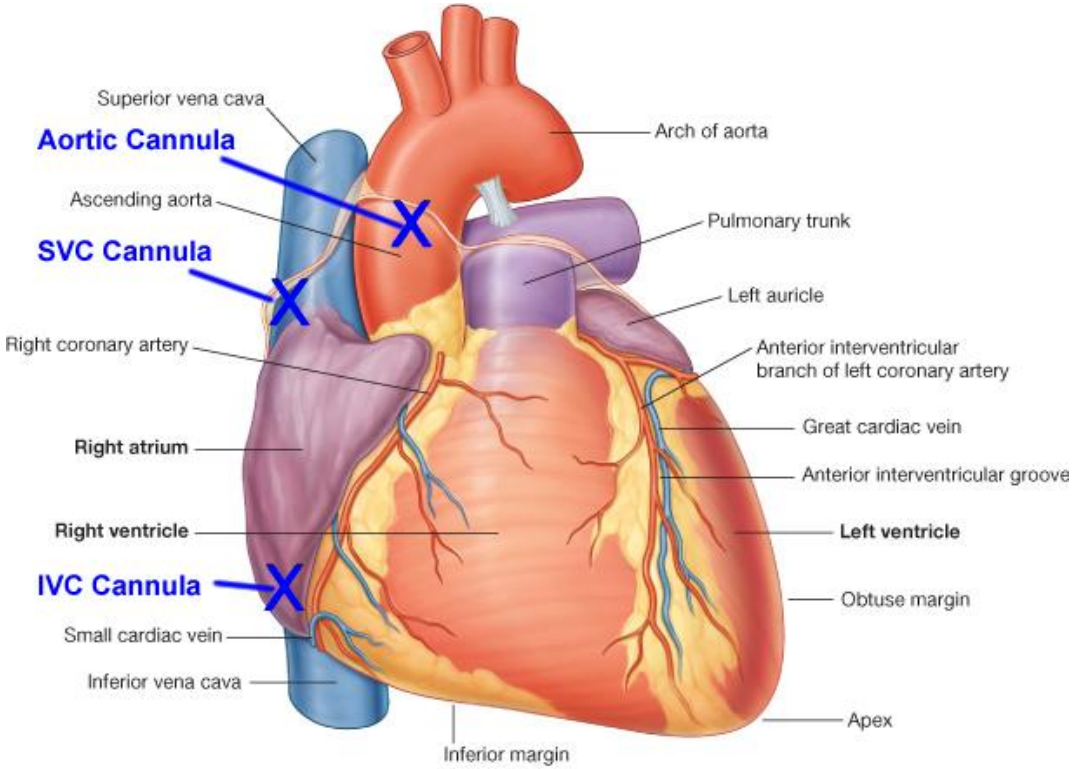
ALTERNATIVE FEMORAL ARTERIAL CANNULATION

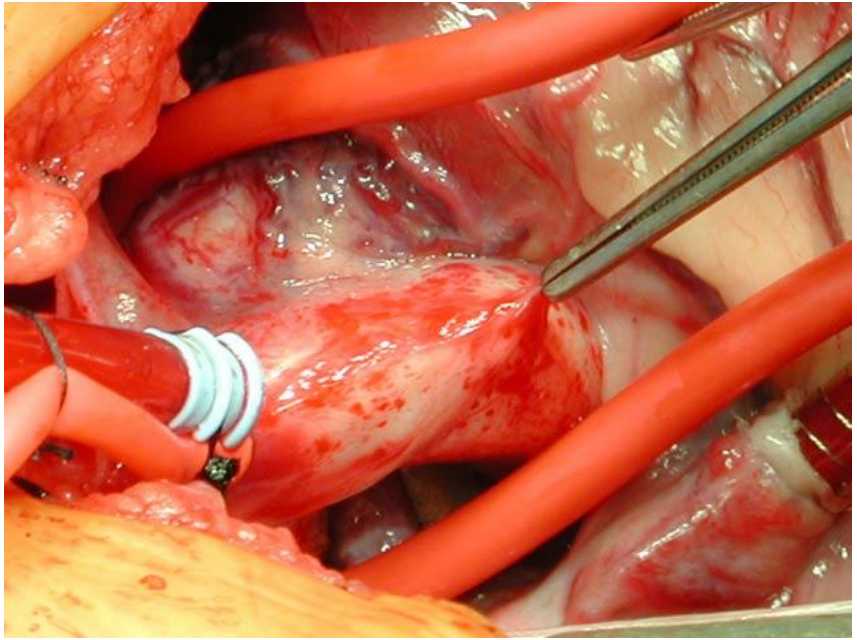
STANDARD CANNULATION SITES IN OPEN CHEST



OPEN-CHEST SITUATIONS

In an Emergency pipes can be held in place.

Minimizes retrograde Aortic flow.

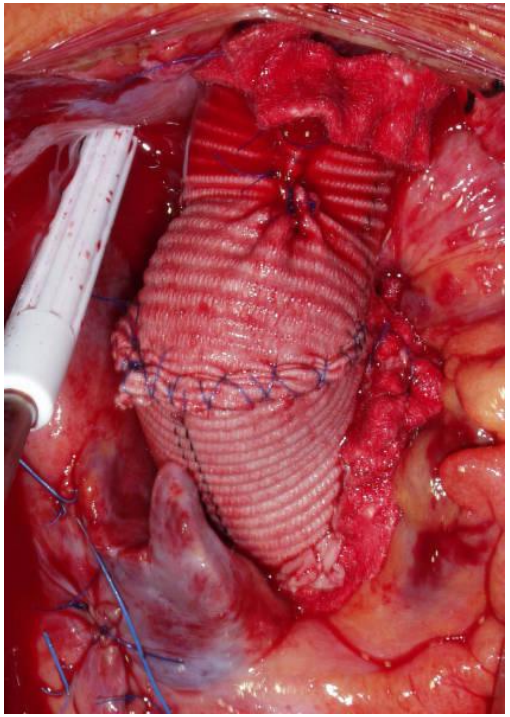


OPEN-CHEST SITUATIONS

Often, in complex Aortic cases, the whole Aortic is replaced by a woven Dacron tube.

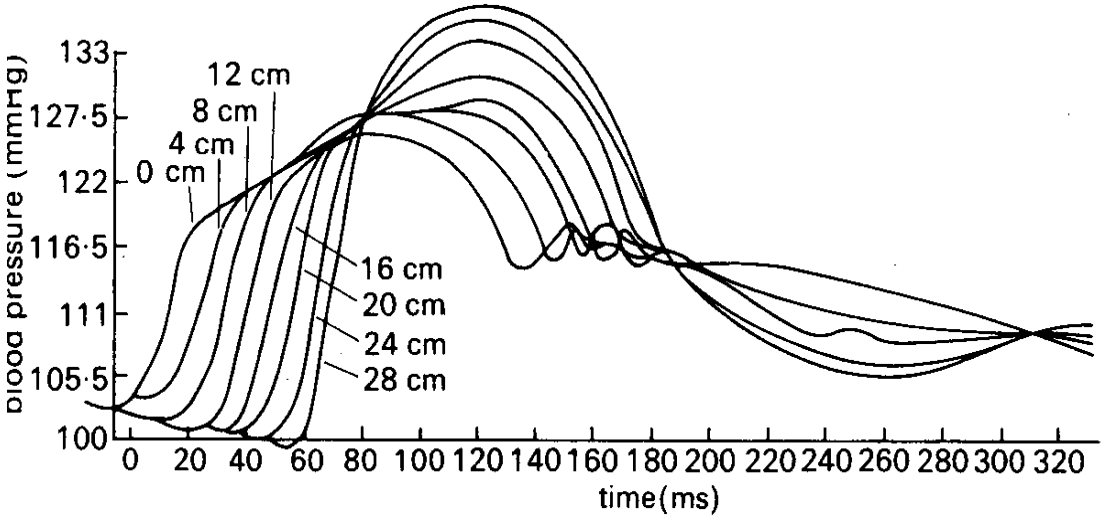
Haemostasis is a major challenge.

Kinking of grafts is an issue.



Aortic Pulse Amplification

As mean pressure falls along the aorta, the pressure wave is delayed and the pulse amplitude raised.



Aortic Compliance

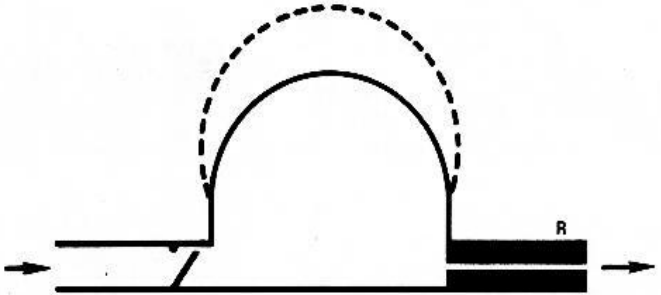


Figure 6.13. Standard *Windkessel* model of the aorta and major arteries. Flow enters chamber through one-way valve on the left faster than it can leave through the exit on the right, raising the pressure and distending the elastic wall to position shown by *dashed line*. When inflow stops, valve closes and fluid leaves the chamber through the narrow resistance (**R**) on the right.

$$\frac{1}{RC} = \frac{dP}{PdV} \cdot \frac{dV}{dt} \qquad (6.18)$$

Integration of equation 6.18 with respect to time shows that pressure in the chamber declines exponentially from its initial value, P_0, during the period of outflow:

$$P(t) = P_0 \exp\left[\frac{-t}{\mathbf{R}C}\right] \qquad (6.19)$$

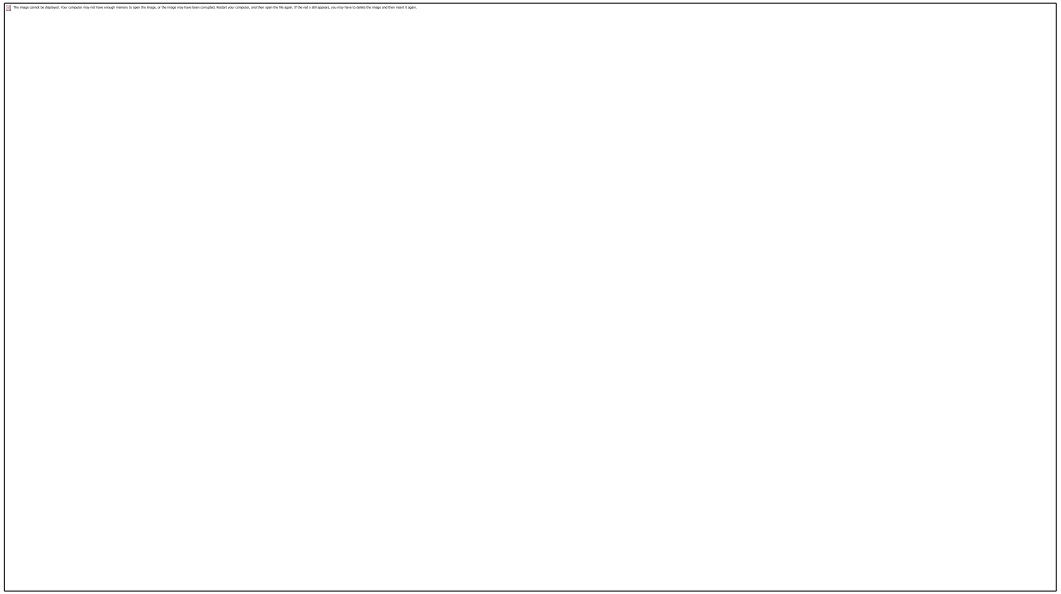
THE IMPORTANCE OF LOW SHEAR STRESSES

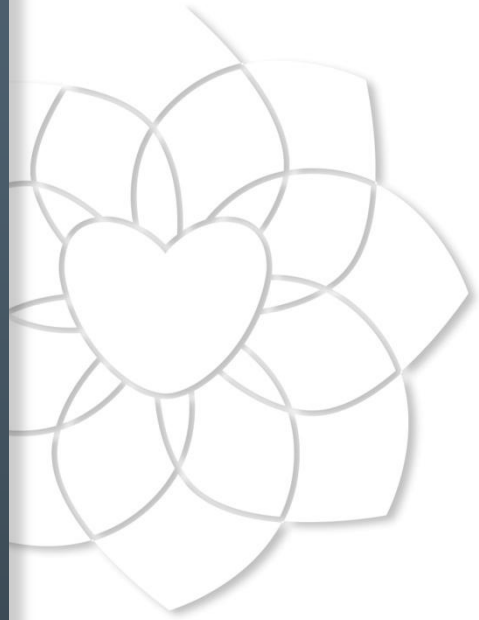
Haemolysis

? CVA

Lower Pump RPM

Less ‘Jet Wash’ of Aorta





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AVOIDING FLOW-LIMITING CANNULAE WITH GELSEAL GRAFTS

No need to remove when weaning off ECMO

Close with Stapler and leave a small stump.

VA-ECMO IN THE PRESENCE OF AORTIC REGURGITATION

Relative Contraindication

Ignore.

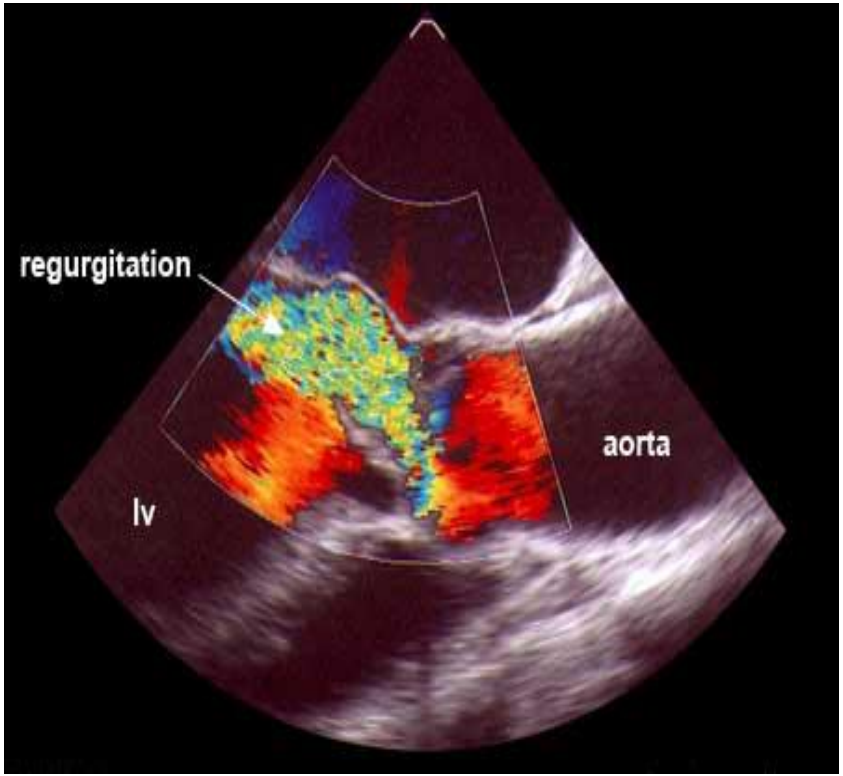
Balloon Pump.

LV Vent.

Change Valve (AVR).

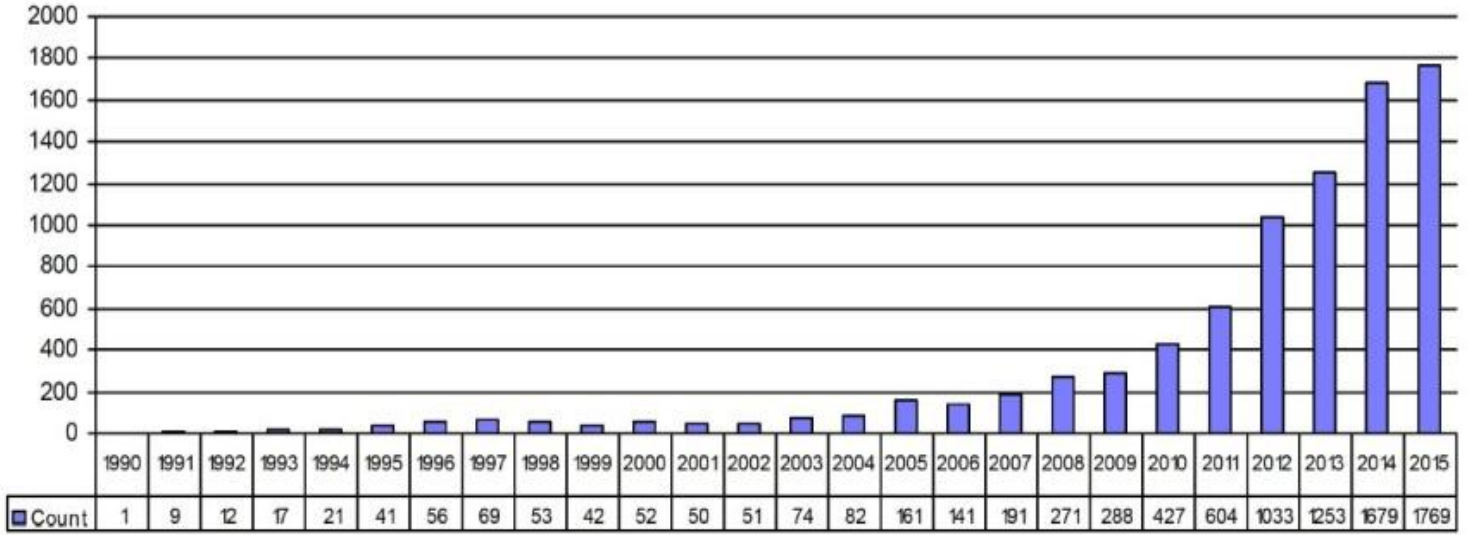
TAVI.

Impella Device.



IMPELLA DEVICE

Rapid Expansion in Cardiac ECMO (UK)



	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015
Count	1	9	12	17	21	41	56	69	53	42	52	50	51	74	82	161	141	191	271	288	427	604	1033	1253	1679	1769

Extracorporeal Life Support Organization

Overall Outcomes

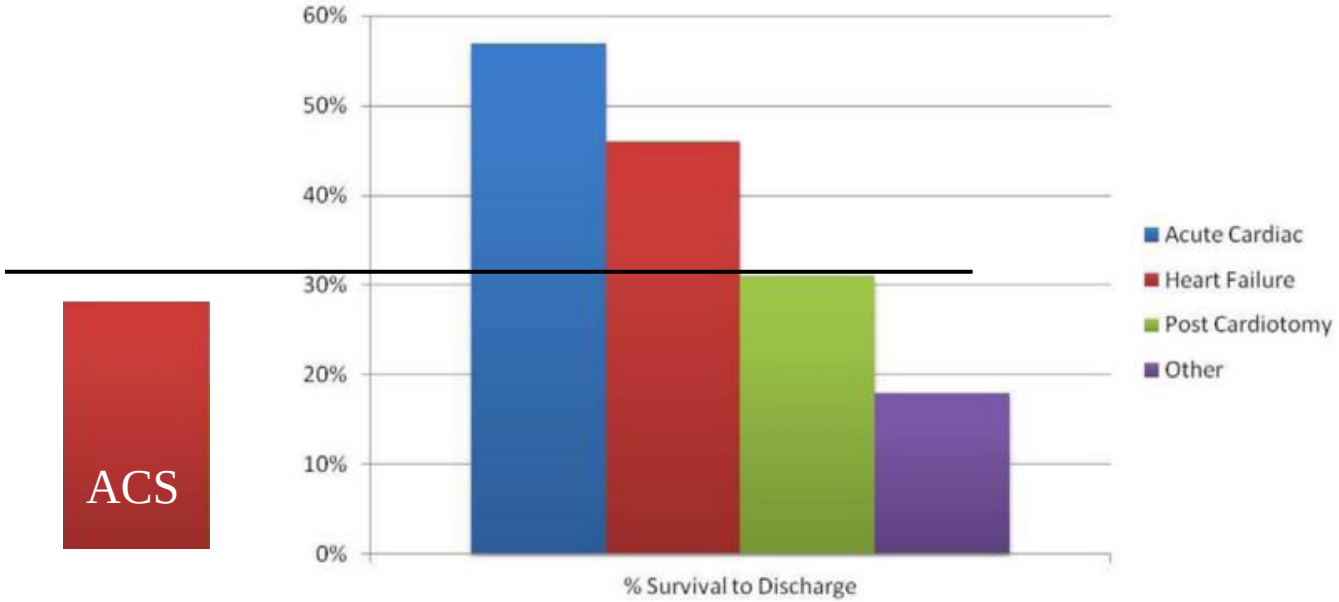
	Total Runs	*Survived ECLS*		*Survived to DC or Transfer*	
Adult					
Cardiac	10,982	6,251	56%	4,466	40%
ECPR	3,485	1,382	39%	993	28%

A Systematic Research and Meta-analysis

patients with a diagnosis of ACS treated with extracorporeal circulatory support

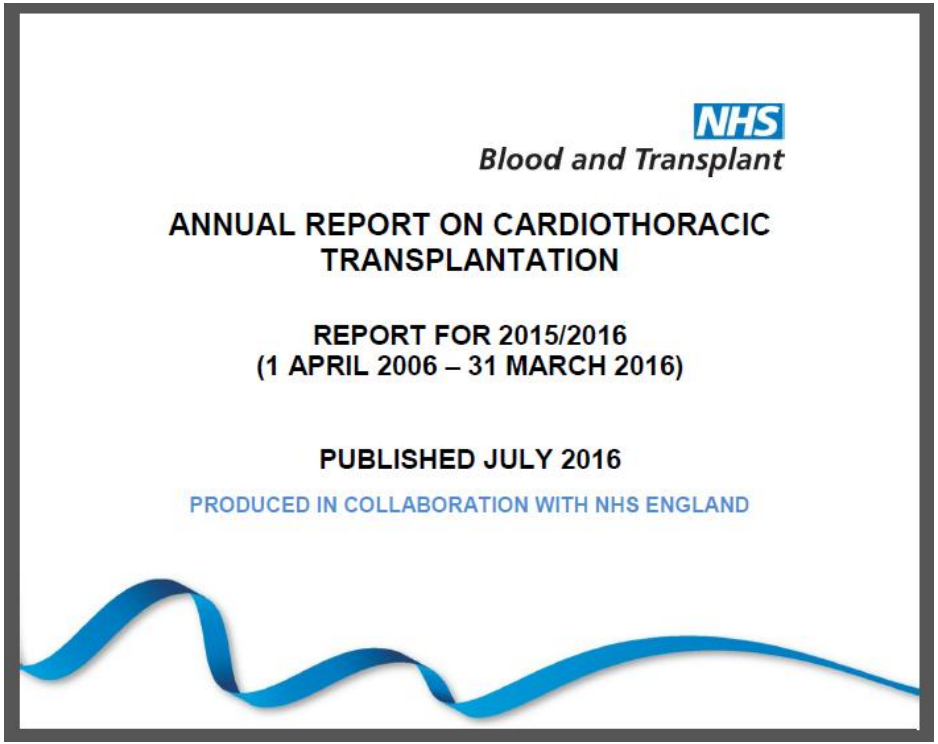
Results: A total of 913 patients were included in the meta-analysis [redacted]. The event rate of short-term mortality was 62% [redacted]. 6 months mortality was affecting 24% [redacted] of patients and 1-year mortality 17% [redacted]. The event rates of ECS-related complications were: acute renal failure [redacted], bleeding [redacted], neurologic damage in survivors [redacted], sepsis/infections [redacted] and leg ischemia [redacted]. Between causes of death, multi-organ failure and brain death affected respectively [redacted] and [redacted] of patients. Bridge to ventricular assistance device was offered to [redacted] of patients treated with ECS and [redacted] received a transplant.

913 Patients, short term mortality 62%
347 Patients, 6-month mortality 24%
264 Patients, 1-year mortality 17%
219 Patients alive at 1 year = 76% mortality

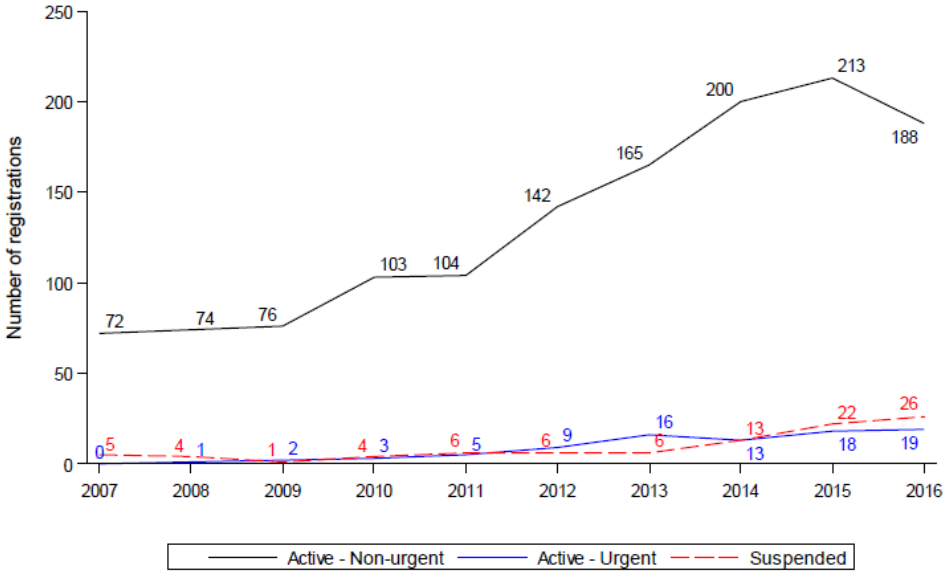


Percentage Survival to Discharge of Patients placed on. VA ECMO patients stratified by Indication.

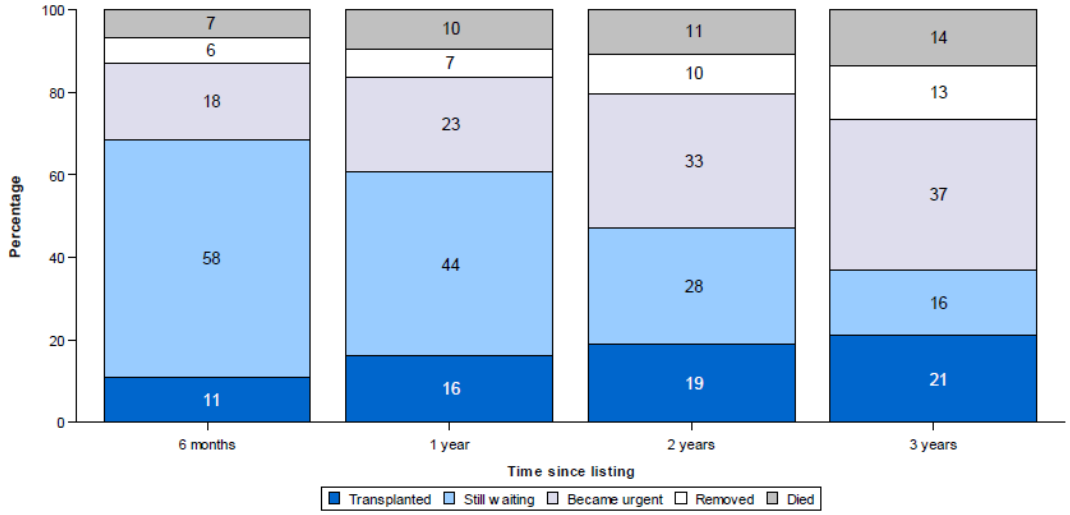
Acute Cardiac Diagnoses.
Post-cardiotomy.
Acute Decompensation of Chronic Heart Failure.
"Other"

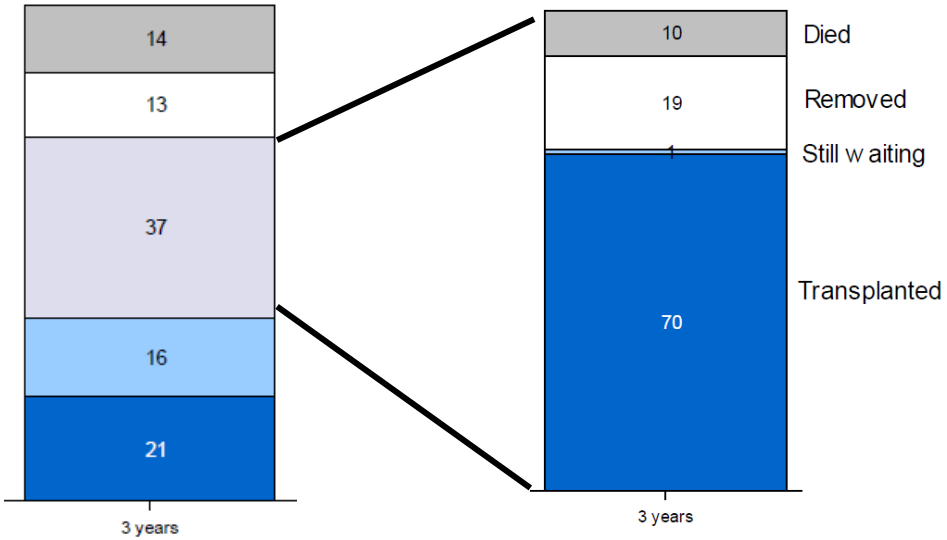

NHS
Blood and Transplant
ANNUAL REPORT ON CARDIOTHORACIC TRANSPLANTATION
REPORT FOR 2015/2016
(1 APRIL 2006 – 31 MARCH 2016)
PUBLISHED JULY 2016
PRODUCED IN COLLABORATION WITH NHS ENGLAND

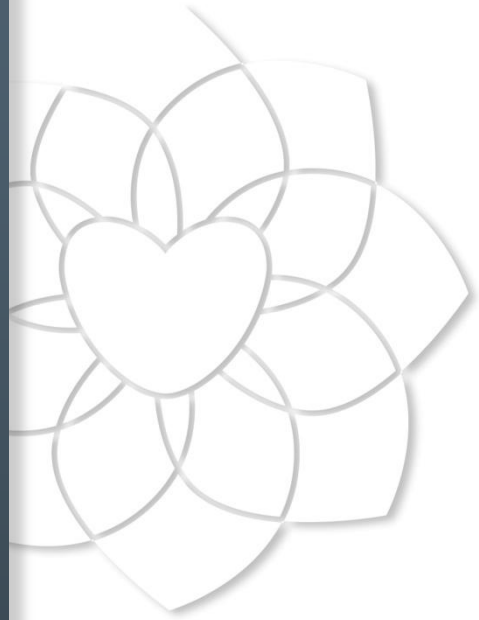
Adult patients on the heart transplant list at 31 March each year for the last 10 years, by year



Post-registration outcome for 147 first non-urgent heart only registrations made in the UK, 1 April 2012 to 31 March 2013




14
13
37
16
21
3 years
10
19
1
70
Died
Removed
Still w aiting
Transplanted
3 years



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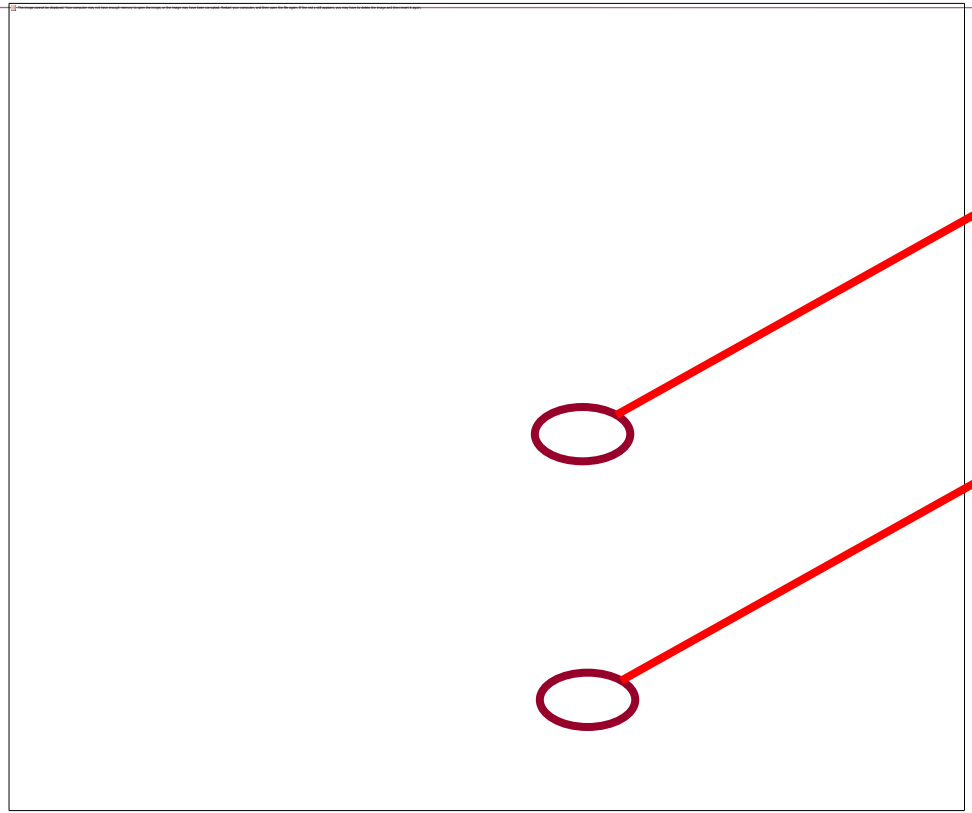
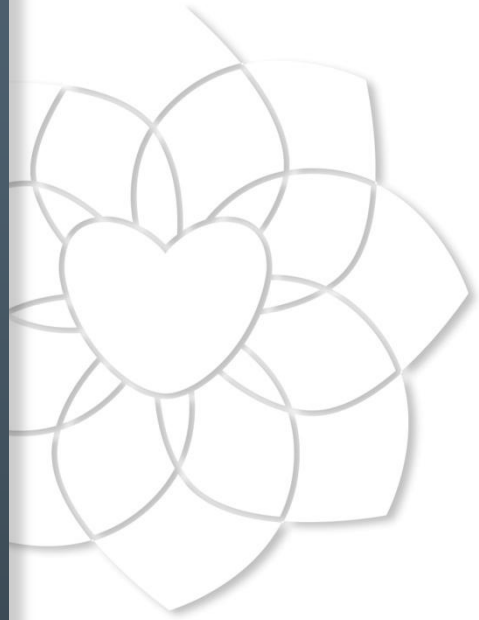

3 Months
3 years, 1 month



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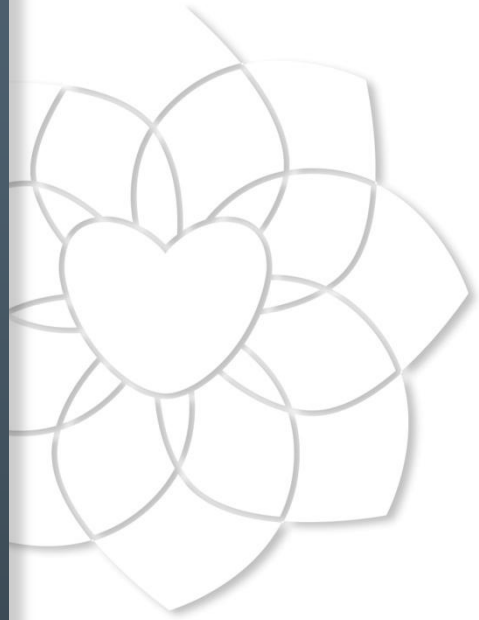

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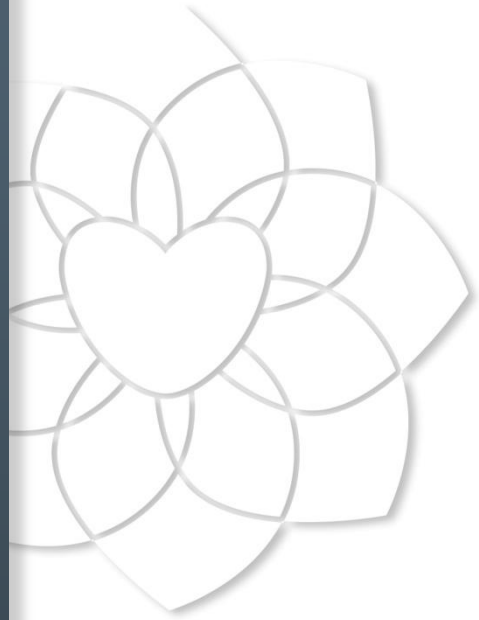

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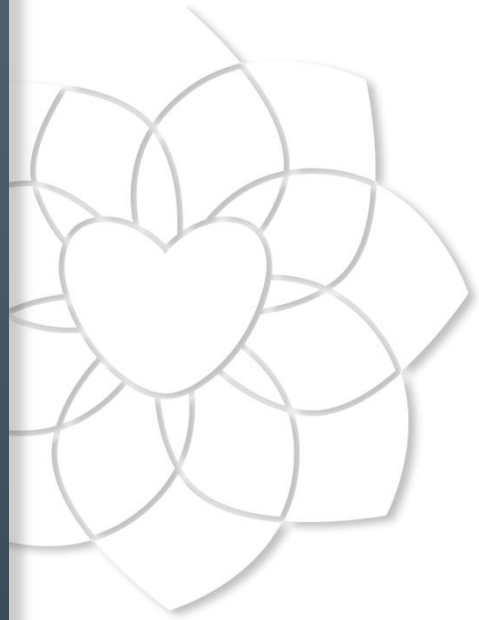

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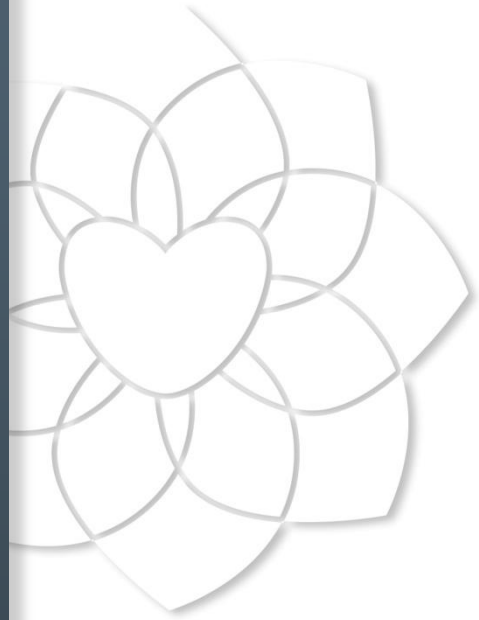

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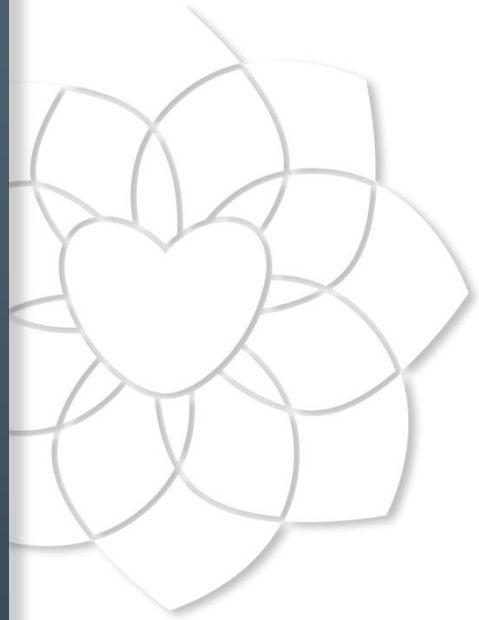

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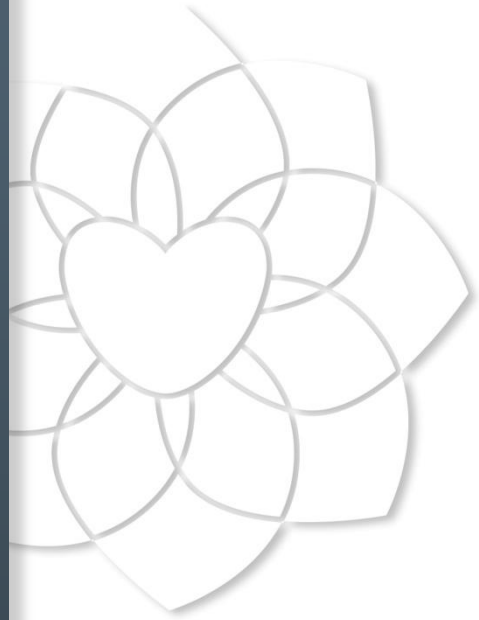

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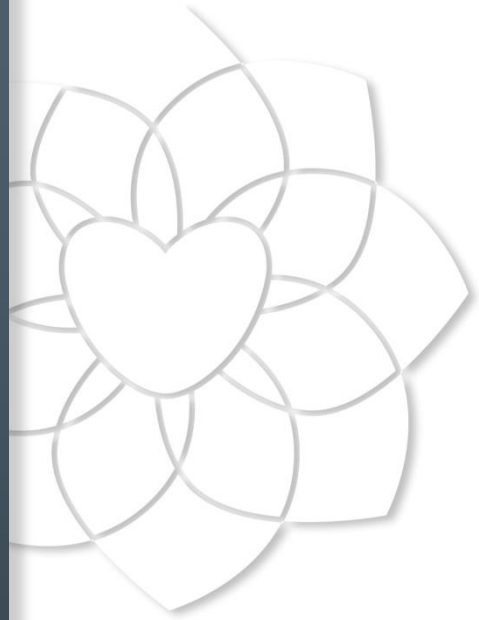

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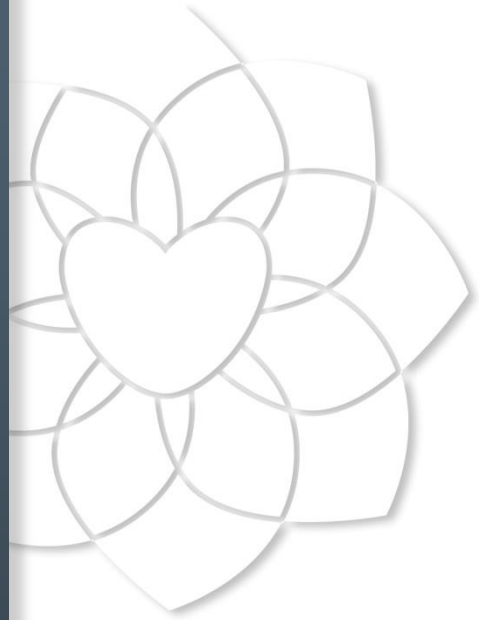

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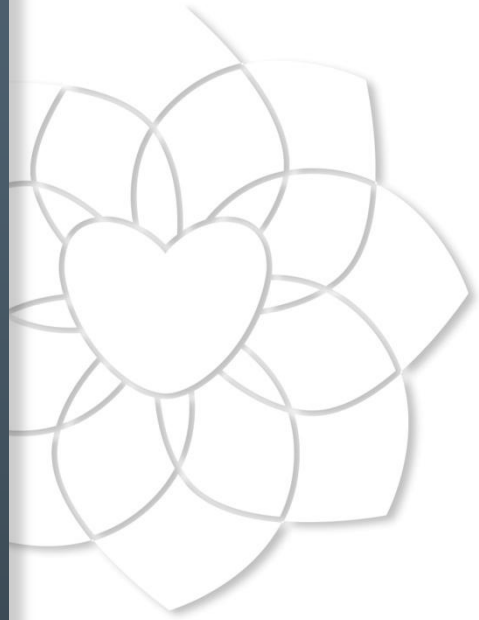

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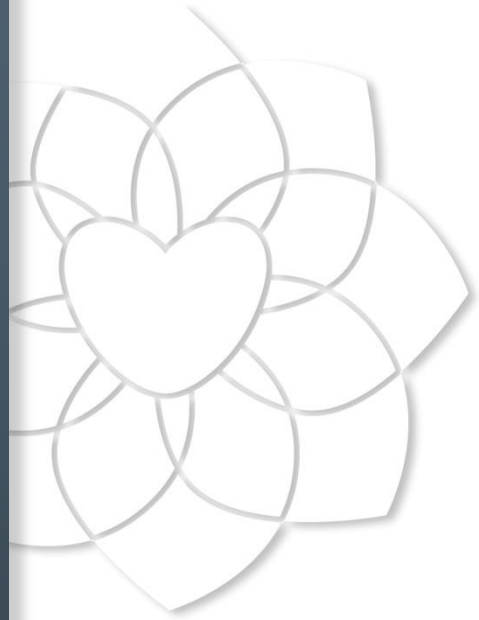

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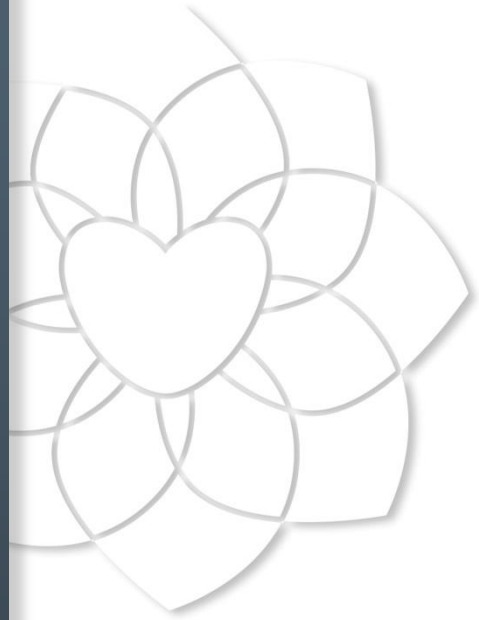

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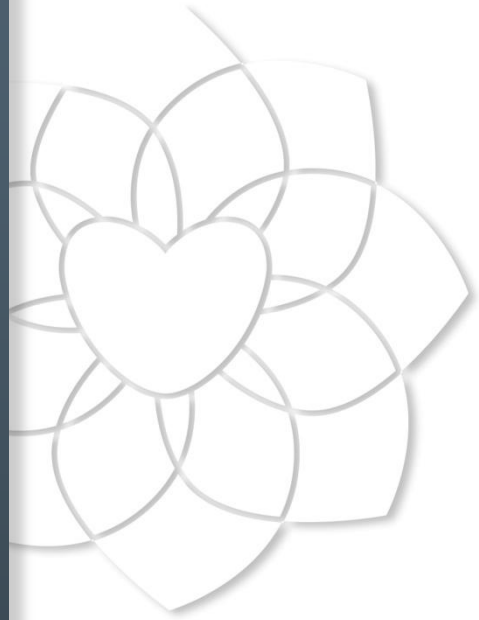

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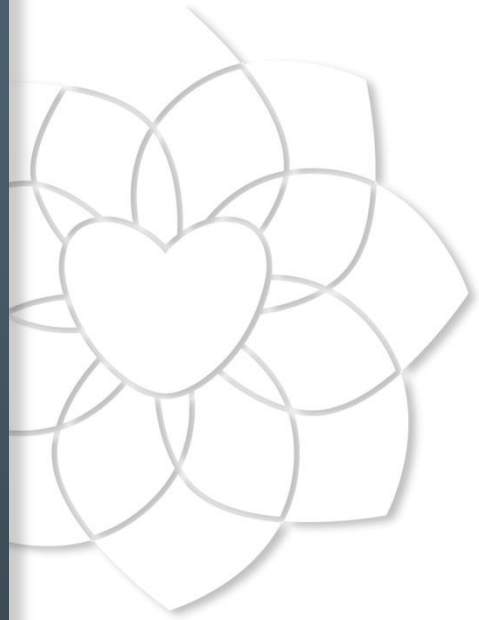

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