Acute coronary syndrome and acute left /right heart failure in the ICU

Krisztina Madách

Department of Anesthesiology and Intensive Therapy

Mosaics of the lecture

- ACS
  - PCI guidelines
  - Periop. management of PCI, BMS, DES patients
- Heart failure
  - Pathophysiology
  - Pharmacological management
  - Mechanical management

Acute Coronary Syndrome

- Endemic disease
- Invasive investigation
  - majority of ACS patients
- Anticipated life expectancy at birth increased with 5,5 years
- Death of MI has halved!
- Indications – new guidelines
  - Revascularisation guideline (2010)
  - ESC NSTE-ACS guideline (2011)
  - ESC STEMI guideline (2008, 2012)

Diagnosis

Symptoms	→	ACUTE CORONARY SYNDROME		
		STEMI	NSTE-ACS	
ECG	→	Persisting ST elevation	ST-T deviation	No/ aspecific ECG change
Biomarker	→		pos	neg
Diagnosis	→	STEMI	NSTEMI	Unstable angina
			NSTE-ACS	

ESC STEMI guideline 2008
Early diagnosis

- On-site diagnosis
  - **Typical chest pain**
  - **ST elevation or newly developed LBBB**

    IS ENOUGH FOR PRIMER DIAGNOSIS
  - Elevated necroenzymes – not necessary to wait for the result
  - Echocardiography: to disclose other origin of acute chest pain

5

PCI indication-STEMI

- Within 12 hours: STEMI or chest pain and unknown LBBB (IA)
- After 12 hours: persisting chest pain and ST elevation (IIa)
- 12 - 24 hours: patient with no complaints (IIb)

- After 3 days: routine opening of occluded coronary arteries responsible for MI may be harmful if there is no viability!

ESC Revasc. Guideline 2010 6

6

PCI indication-NSTEMI

Risk factors

- troponin elevation
- dynamic ST-T changes
- diabetes mellitus
- renal insufficiency
- decreased left ventricular function (EF<40%)
- early post-MI angina
- PCI within 6 month
- previous CABG
- medium or high (>140) GRACE risk

7

PCI indication-NSTEMI

- <2 hours: very high ischemic risk
- <24 hours: GRACE score >140, multiple risk factors
- 72 hours <: GRACE score <140, but relapsing symptomes, positive stress test.

8

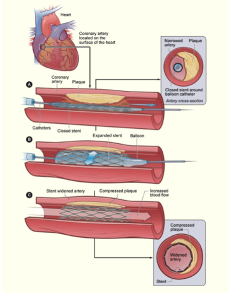
The problem

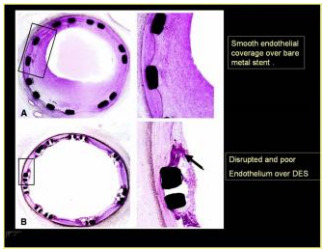
- 1990': increasing periop. morbidity/mortality in patients with PCI going for non-cardiac surgery
- 90% of patients going for PCI get one or several stent/s
- 5% will have non-cardiosurgical intervention within **1 year!**
- Periop. dilemma: bleeding or coagulation?
- **63% of anesthesiologists do not know the guideline!**

Vicenzi MN et al. Br J Anaesth 2006;96:686-93

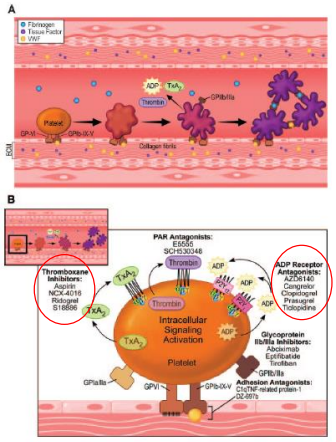
Patterson L et al. Can J Anesthesiol 2005;52:440-1

PCI, BMS, DES
The traps: restenosis or incomplete endothelisation and thrombosis?





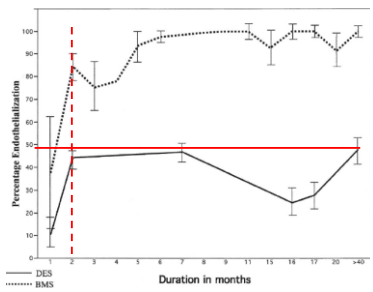
Venkatesan S. Why should a stent elute drug ? http//:drsvenkatesan.files.wordpress.com 2008



Platelet formation inhibiting therapies: What? For how long?

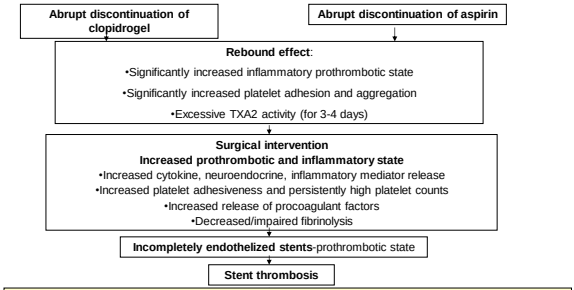
Mardows TA et al. Circ Res 2007;100:1261

Change in endothelisation of BMS and DES stents
(intravascular US, autopsy)



Joner M et al. J Am Coll Cardiol 2006;48:193-202

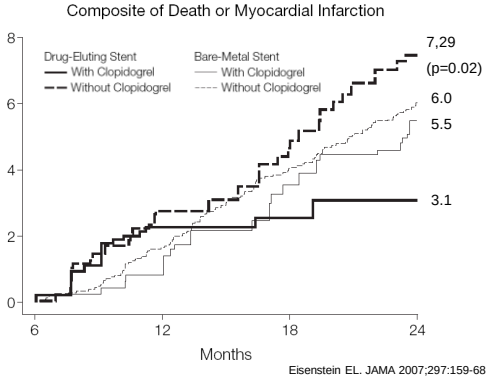
Consequences of abrupt discontinuation of antiplatelet drugs in the perioperative period



Discontinuation of antiplatelet drugs: 5-10x increased perioperative myocardial death?

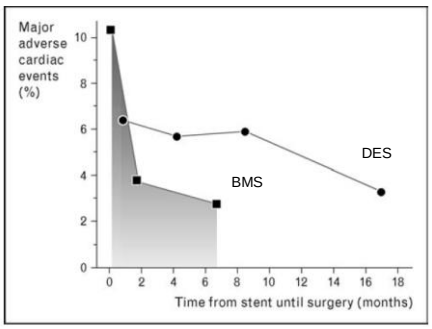
Chassot PG et al.. Br J Anaesth 2007;99:316-28

Clopidrogel use and long-term outcomes with BMS and DES



Eisenstein EL. JAMA 2007;297:159-68

Risk of major adverse cardiac events after stent implantation in non-cardiac surgery



Popescu WM. Current Opinion in Anaesthesiology 2010; 23:109-115

Perioperative bleeding

- ASA
  - 1,5x increased bleeding risk
  - BUT! Except intracranial operation and TUR it did not increase perioperative morbidity/mortality
- Clopidrogel+ASA (mainly cardiac surgery)
  - Bleeding↑, reexplorations, transfusions, increased ICU/hospital stay, no difference in mortality (?)
  - BUT! Long-term (10 years) mortality was 80% in patients transfused with 1 U rbc vs. 63% in not transfused patients!

Burger W et al. J Intern Med 2005;257:399-414

Koch CG et al. Ann Thorac Surg 2006;81:1650-7

Assessment of the risk of bleeding, prevention, treatment

- TEG/ROTEM
- Renal function
- Bridge therapy?
- Antidote: platelet transfusion

17

Required length of APT
TIMING of anticipated surgery

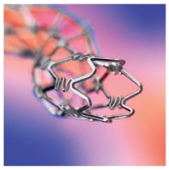
- Dilatation without stenting: 2-4 weeks dual APT
  - Delay for noncardiac surgery: 2-4 weeks
- PCI and BMS: min 4-6 weeks dual APT
  - Delay for noncardiac surgery : ≥ 6 weeks
- PCI and DES: 12 months dual APT
  - Delay for noncardiac surgery : ≥ 12 months
- Aspirin lifelong

2007 AHA/ACC Science Advisory and Society of Cardiovascular Angiography DES Task Force Recommendations for Timing of Noncardiac Surgery after PCI

18

Overview: prevention of periop.stent thrombosis

- Avoid preop. coronary stenting
- Stent selection (BMS-DES)
- Delay surgery
- There is no bridge therapy for PCI patients, **timing is crucial**
- Education, interdisciplinary teamwork
- Surgery only where PCI is available



Brilakis et al. JACC 2007;49,22:2145-50

19

HEART FAILURE

- > 65 age: most frequent cause of hospital admission
- The incidence of hospitalization and the incidence of mortality due to heart failure keeps increasing since 1968
  - Despite the decrease in overall mortality of cardiac diseases
- 5 year mortality ~ 50%
- Patient going under emergency laparotomy with untreated heart failure has a mortality of 20-30 %

Telford R. Oxford handbook of anaesthesia 2007;3:48-51

20

Definition

- Heart failure is a complex clinical syndrome, characterized by
  - Insufficient ventricular performance
  - Intolerance of workload
  - Frequent ventricular arrhythmias
  - Reduced anticipated lifetime.
- Classification:
  - Diastolic dysfunction (relaxation problem of the left ventricule)
  - Systolic dysfunction (can involve either ventricules)

21

Etiology

- Heart failure is the final common pathway of primer cardiovascular diseases
  - Acut/chronic
- Causes:
  - Most frequent: coronary disease, hypertension
  - Other: diabetes, valve disease, non-ischemic myopathies
- Indipendent risk factors:
  - Man, hypertension, coronary disease, diabetes, age

22

Pathophysiology

„The heart has no wrinkles."

Madame de Sévigné

But nevertheless it ages:

- Size of the ventricle, wall thickness, stiffness, myocyte loss, fibrosis, collagen bridges
- Max. frequency and CO decreases, SVR, systolic BP increases

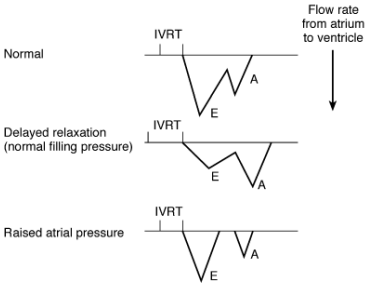
23

Pathophysiology

- Left ventricular relaxation disorder :
  - isovolaemic relaxation time increases,
  - sarcoplasmatic reticulum Ca^{++} excretion decreases
- Left ventricular diastolic filling becomes more dependent on atrial contractions

24

Changes in left atrial-ventricular heart failure ouflow in heart failure



E: passive flow in early diastole

A: active flow created by atrial contraction

IVRT: isovolaemic relaxation time

Magner J, Royston D. Br J Anaesth 2004;93:74-85

Underlying causes of acute heart failure

- Frequent: arrhythmia
- Prevalence of atrial fibrillation[1]:
  - Age 60: 5%
  - Age 90: 22%
- Short diastolic filling time, loss of „A wave”
- Other: ↓ compliance, warm weather, hypertension, anemia, infection, alcohol, thromboembolism, renal failure, thyroid dysfunction

[1]Aronow WS et al. J Am Geriatr Soc 1996;44:521-3

Compensatory and at the same time harmful reactions

- Neurohumoral:
  - RAAS activation
  - Sympathetic activation:
    - vasoconstriction, Na retention
    - plasma norepinephrine level correlates with prognosis
- Secondary answer: modification of receptors
  - endotheline, natriuretic peptide, tumour necrosis factor

Pre/afterload-, changes in wall tension, direct toxic effects, remodelling

[1]Aronow W. Heart Dis.2003;5: 279-94

[2]Cohn JN. Engl J Med 1984;311:819-23

Diagnosis

- TOE (TTE), Doppler flow studies
- BNP, NT-proBNP
- The cause pericardial, myocardial, valvular?
- If myocardial:
  - Systolic dysfunction (EF<40%) ?
    - Fractional Area Change= EDA-ESA/EDA (~ventriculography)
  - Diastolic dysfunction ?
    - reduced „E”, elongated „E” deceleration)

Therapy of heart failure

- Most cases can not be cured, but can be improved symptomatically, physical activity and quality of life / duration can be increased.
- End-stage heart failure: heart transplantation is currently the only therapy

- And until then?
  - Pharmacological
  - Non-pharmacological possibilities

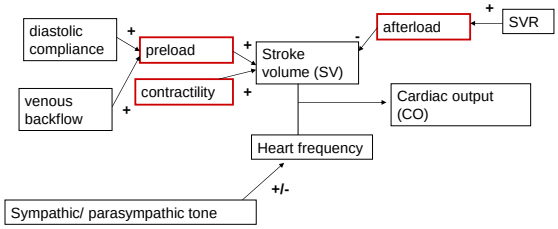
29

Therapy of heart failure

1. Treat the underlying cause
2. Elimination of associated factors further impairing heart failure (infection, hyperthyreosis, stb)
3. Treat heart failure itself

30

Factors determining cardiac output



31

Restrictive cardiomyopathy

- Difficult to manage
- Wall stiffness: decreased ventricular diastolic filling
- Right heart failure is common
- Cardiac arrest can be provoked by:
  - Myocardium depression
  - Vasodilatation
  - Decreased venous back-flow
  - Increased intrathoracal pressure (IPPV)
- Recommended: low intrathoracal pressure, elevated right ventricular pressure: **fluid therapy**

Bovill J. Semin Cardiothorac Vasc Anaesth 2003;7:49-54 32

Dialatative cardiomyopathy

- Low contractility, stroke volume is maintained by increased LV ebddiastolic volume
- Functional MI, TI
- Frequent arrhythmia → amiodarone
- Avoid myocardium depressive drugs
- **Inotrops** might be needed

Bovill J. Semin Cardiothorac Vasc Anaesth 2003;7:49-54 33

Hypertrophic cardiomyopathy

- Outflow obstruction (septal hypertrophy)
- Diastolic dysfunction
- Diastolic filling is atrial systole dependent: sinus rhythm!
  - Might be even 75% of end-diastolic volume!
- Tachycardia reduces diastolic filling time

- Inotrops can deteriorate outflow obstruction and oxygen demand of the myocardium, and increase wall tension
- Vasodilators not inducing tachycardia are recommended

Bovill J. Semin Cardiothorac Vasc Anaesth 2003;7:49-54 34

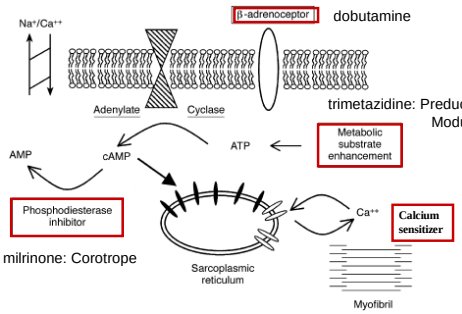
Basic pharmacological treatment

- ACE inhibitors: pre- and afterload↓, neurohormonal blockade, improve survival
- Beta blockers: myocardium oxygen demand↓
- Diuretics: spironolacton improves mortality when given with ACE inhibitor in severe heart failure (EF<25%)
- Cardiac glycosides: digoxin (frequency control, sympathetic hyperactivity↓)
- Avoid: NSAID, Ca channel blockers (except. amlodipine)

Lonn E. Drug treatment in heart failure. BMJ 2000;320:1188-92

35

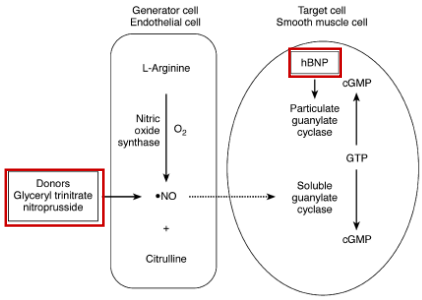
Inotrops



Magner J, Royston D. Br J Anaesth 2004;93:74-85

36

Decrease preload



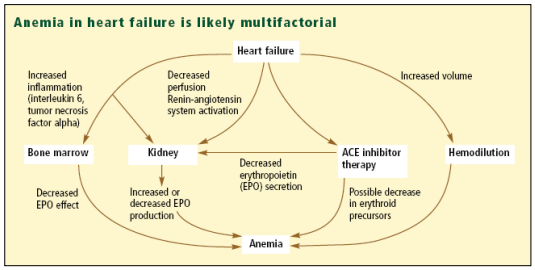
Nesiritide:

- •Dose dependent PCWP, MAP↓
- •Longer hypotonsion
- •No effect on contractility, electophysiology (animals)
- •Vascular permeability?

Magner J, Royston D. Br J Anaesth 2004;93:74-85

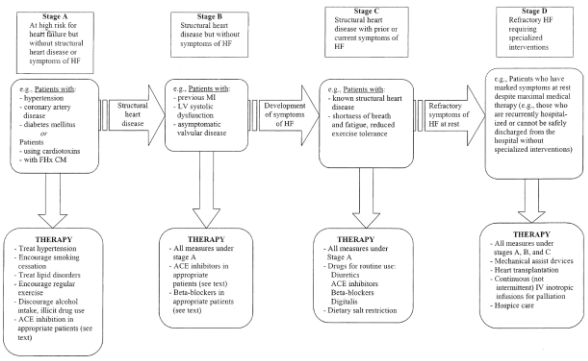
Mills RM et al. Drugs Today (BARC) 2003;39:767-74

Hemoglobin level: anaemia is common



Iyengar S Abraham WT. Anemia in chronic heart failure: can EPO reduce deaths? Cleve Clin J Med. 2005 Nov;72(11):1027-32.

Stages of heart failure



Hunt SA et al. ACC/AHA guidelines for evaluation and mamagement of chronic heart failure in the adult. Circulation 2001;104:2996-3007

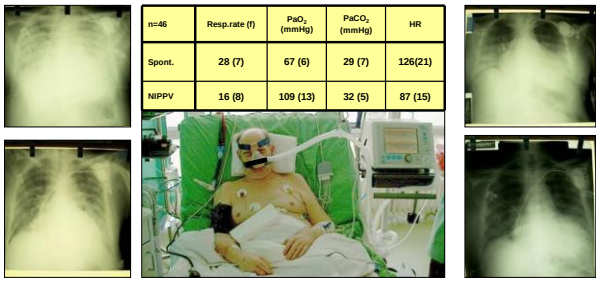
NON-PHARMACOLOGICAL TREATMENT

- Noninvasive ventilation
- Cardiac resynchronization therapy
- Surgical ventricular reconstruction
- Mechanical support

Is there a role for noninvasive positive pressure ventilation (NIV) in acute cardiogenic pulmonary oedema (ACPE)?

Effectivity of NIV in ACPE

- Predictors of success: ABG (pH, pCO_2), vital signs
- Patients like it



n=46	Resp.rate (f)	PaO_2 (mmHg)	$PaCO_2$ (mmHg)	HR
Spont.	28 (7)	67 (6)	29 (7)	126(21)
NIPPV	16 (8)	109 (13)	32 (5)	87 (15)

Cardiac resynchronization therapy

- Sudden death due to arrhythmia is common
- Sinus rhythm is essential
- Delay in interventricular conduct : non-synchronized ventricular contraction deteriorates LV systolic dysfunction
  - 30% of heart failure patients!

Abraham WT et al. N Engl J Med 2002;346:1845-53

43

Cardiac resynchronization therapy

- Atrium synchronized biventricular pacing
- EF↑, PCWP↓, diastolic ventricular filling time↑, mitral regurgitation↓, quality of life ↑, workload↑
- Mortality was reduced by 20%
- If ICD: mortality was reduced by 40%

Chow AW et al. BMJ 2003;326:1073-7

MUSTIC study. Linde C.et al. J Am Coll Cardiol 2002;40:111-18

COMPANION study. Bristow MR et al. J Card FAil. 2000;6:276-85

44

Cardiac resynchronization therapy: indications

- Systolis HF
- Non-reversible causes
- Significant symptoms despite optimized pharmacological treatment
- Sinus rhythm is essential
- Significant mitral regurgitation
- Ventricular dyssinchronity (LBBB and apical right ventricular pacing)

Chow AW et al. BMJ 2003;326:1073-7

45

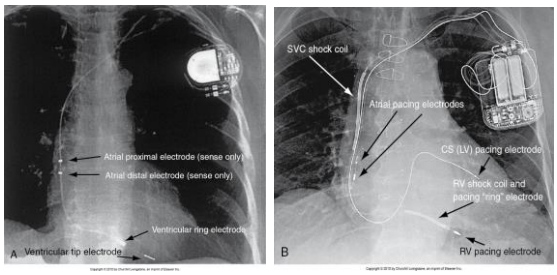
Implantable **C**ardioverter **D**efibrillator

- Malignant ventricular arrhythmia is frequent cause of death
- Indication:
  - High risk heart failure
- MADIT-II trial: 31% relative risk reduction in severe left ventricular insufficiency of ischemic origin

Moss AJ et al. N Engl J Med 2002;346:877-83

46

What is in the patient? Documentation?



In Miller ED et al. Miller's Anesthesia 7th Ed. 2010

47

Surgical ventricular remodelling

- Anterior myocardial infarction: change in ventricular volume an muscle structure (dyskinesis-akinesis)
- Anterior ventricular endocardial restoration

Athanasuleas CL et al.Am Coll Cardiol 2001;37:1199-209

48

Mechanical assist devices: MAD

- Reduce mechanical work of the heart, ameliorate coronary perfusion
- Difficult decision:
  - Can the heart improve? MODS?
  - Can this be influenced by MAD?
- Wide range of devices

49

Mechanical assist devices: MAD

- IABP
  - Short term support
  - Ambulatory, longer term application? [1]
  - Overall morbidity (limb ischemia, bleeding) 3-5%
- VAD-Ventricular Assist Device
  - End stage (lack of donor organs)
  - *FDA: Bridge* to transplant
  - REMATCH trial: patients inappropriate for transplantation[2]
    - 1 year survival 52% contra 25%
    - Morbidity 2,35x of pharmacologically treated group: bleeding, infection, malfunction

[1]Cochran RP et al. Ann Thorac Surg 2002;74:746-51

[2] Rose EA et al. N Engl J Med 2001;345:1435-43

50

Ventricular Assist Device: VAD

- Indication: postcardiotomy shock, AMI, myocarditis, ACS-cardiogen shock
- „Bridge to recovery”
- „Bridge to transplant”
  - MODS can be improved
- „Bridge to destination”
- Timing is crucial

Williams M et al. Cardiac assist devices for end-stage heart failure. Heart Dis 2001;3:109-15

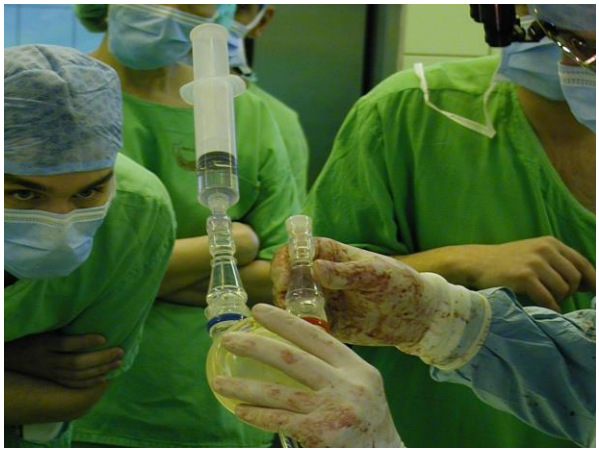
51

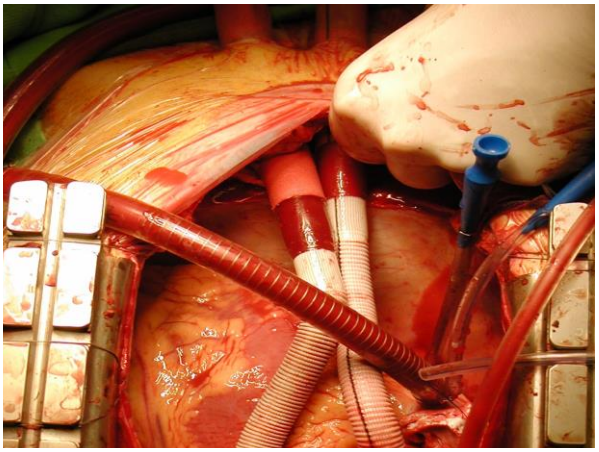
Ventricular Assist Device: VAD

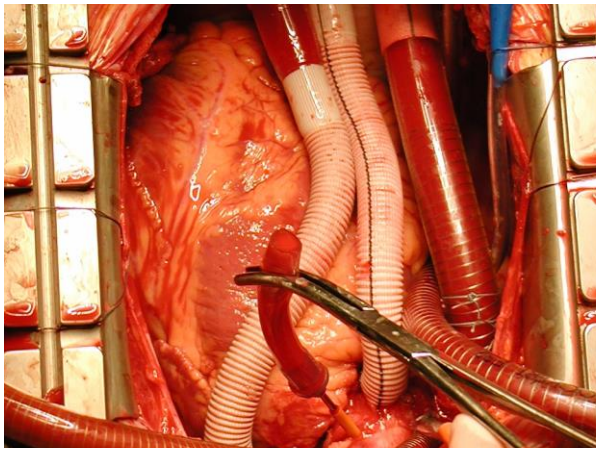
- Choice of equipment: duration, body surface, uni/biventricular support
- Extra-, intracorporal
- According to flow generator: centrifugal, axial, diaphragmatic
- Considerations: anticoagulation, ICU/outpatient, mobilization

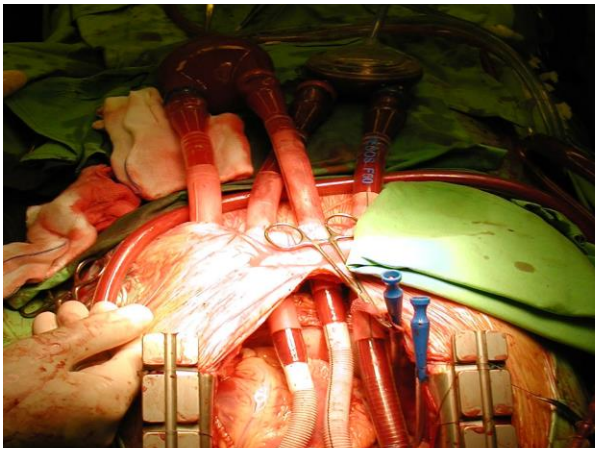
Williams M et al. Cardiac assist devices for end-stage heart failure. Heart Dis 2001;3:109-15

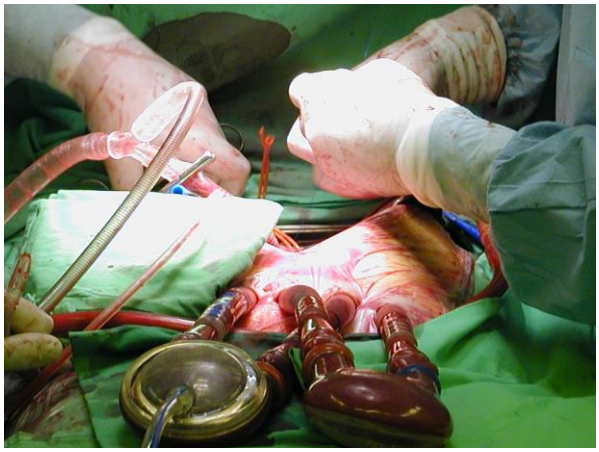
52











Summary

- Death of myocardial infarction has halved due to invasive investigations/interventions (ESC guidelines)
- PCI (BMS, DES stents): restenosis, incomplete endothelization, thrombosis is a problem
- Platelet aggregation inhibitors and bleeding risk: there is no bridge therapy, timing is crucial
- Heart failure is the final common pathway of primer cardiovascular diseases with increasing incidence
- Pharmacological and novel non-pharmacological therapies can improve life quality and expectancy of heart failure.

58