If It’s Not Tamponade . . .
Rarer Complications of Cardiac Surgery

I have no financial or other conflicts of interest to disclose.

Objectives

Review less common early and late complications of cardiac surgery
Add rare complications to the list of “possible” and enhance surveillance and ability to rescue
Identify own role in early recognition of less common complications

Cardiac surgery in the US

Continued evolution of discipline and technology
Declining/fluctuating volumes
All-cause 90 day mortality for primary isolated CABG is 1 - 4%

General predictors for complications

Extremes of age
Underlying cardiac function (NYHA FC IV)
History of prior heart surgery
Surgical procedure
Emergency procedure

“Common” complications

Perioperative myocardial infarct < 3-15%
Re operation for bleeding < 6%
Renal failure requiring hemodialysis < 2%
Stroke < 2%
Cardiac tamponade (early and late) < 1% to 5%
But if it’s *not* tamponade . . .

Vasoplegic syndrome
Neurological complications
Mesenteric ischemia
Non infectious sternal dehiscence

Vasoplegic syndrome (VS)

Catecholamine resistant hypotension in the presence of adequate volume administration
Intra operative or within 6 hours post op

Vasoplegic syndrome (VS)

Hemodynamic criteria*

- Hypotension (MAP < 60-65 mm Hg)
- Hyperdynamic (CI > 2.5 - 3.5 L/min/m²)
- Low filling pressures (CVP < 5 mm Hg)
- Low peripheral resistance (SVR < 800)
- Poor response to catecholamines despite “filling the tank”
  - > 0.3 mcg/kg/min norepinephrine
  - > 0.1 units/min vasopressin

Risk factors for VS

- Transfusion*
- Prolonged CPB time
- Reduced LV function
- Early intra-operative instability
- VAD placement
- Heart transplant

Pre operative meds
- ACE Inhibitors
- ARBs
- CCBs
- BBs
- Intravenous heparin
- New amiodarone
- Vasoactives infusions

Proposed pathophysiology

Loss of vascular smooth muscle tone resulting in severe and refractory vasodilation
- Systemic inflammatory response
- Increased nitric oxide (NO) activity
- Low endogenous arginine vasopressin (AVP)

Proposed to be related to CPB*, surgical trauma, blood product administration, hypothermia, acute hemodilution, use of calcium-binding citrate in cardioplegia solution
Treatment & prevention of VS

Guidance is limited
Limited evidence
No standard definition for VS
No standardization for treatment strategies
Three identified treatment strategies
Conceivably may be used in combination

Vasopressin

Based on suspected deficiency in endogenous AVP
Dosing strategies vary . . . .
Initial bolus if life-threatening hypotension
0.01 - 0.1 U/min (0.6 - 6U/hour) infusion
Keep stable and titrate the catecholamines
> 6 U/hour associated with peripheral ischemia (e.g., mesenteric)

Methylene blue

Interferes w/ the production of nitric oxide
Limited evidence
Controversial
First line or rescue therapy only?

Methylene blue

Dose: 1.5 mg/kg via central line over 20 min +/- maintenance infusion
Adverse events rare below 2.0 mg/kg
Contraindications
Severe renal insufficiency
Hypersensitivity to MB
Pre operative SSRI (serotonin syndrome)
G6PD deficiency (hemolytic anemia)

Side effects of MB

Self limiting angina & arrhythmias
Reduced CO/Ci
Reduced renal & mesenteric blood flow
Increased PVR
Methemoglobinemia
Pulse oximetry readings may be falsely depressed
Urine and skin transiently turn green

High dose vitamin B12

Hydroxocobalamin (cyanide antidote)
Dose: 5 grams IV over 10 minutes
**Preventative strategies**

All controversial and with limited evidence
- Stop ACE Is, ARBs and CCBs for 48 hours pre op
- Prophylactic administration of MB in high risk patients
- Prophylactic low-dose vasopressin infusion starting 20 minutes prior to CPB

**Summary of VS**

Overall mortality rate 11 - 27%
- Early recognition at the bedside in the ICU setting, combined with early treatment attempts is important

**Abdominal complications**

Relatively rare (incidence 0.3 - 3%)
- Independent risk factors: smoking, NYHA FC III and IV
- Often catastrophic ~ mortality 13- 67%
- In some series, GI bleeding remains the most common GI complication s/p cardiac surgery
- Most patients have additional complications
- Gut is uniquely susceptible to low flow states and

**Acute mesenteric ischemia**

Incidence: 0.6 - 0.8%
- Mortality approaches 100% with extensive ischemia
- Clinical challenge

**Etiologies for AMI**

Non occlusive mesenteric ischemia (NOMI)
- Related to splanchnic hypoperfusion, persistent vasoconstriction secondary to LCOS, prolonged CPB
- Less common: thromboembolism of the SMA (e.g. HITT, artheroembolization), mesenteric venous thrombosis

**Risk factors for AMI**

- Low cardiac output states
- Advanced age
- Prolonged CPB time
- Malpositioned IABP
- Other: hypertension, perioperative renal failure with requirement for HD or CRRT, emergent surgery, perioperative IACPT, high dose pressors and inotropes, dehydration
## Detecting AMI

**Clinical suspicion reigns**
- Abdominal pain and guarding
- Challenge is detection in an intubated and sedated patient
- Cyanosis secondary to ischemia

Diagnostics may include KUB (low sensitivity), CTA, mesenteric angiography (definitive), endoscopy, and laproscopy

Note: metabolic acidosis and elevated lactate levels may be absent!

## Three presentation modes

<table>
<thead>
<tr>
<th>Mode</th>
<th>Description</th>
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<tbody>
<tr>
<td>Early (immediate)</td>
<td>Widespread MI in the setting of LCOS and MSOFS -&gt; terminal event</td>
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<tr>
<td>Intermediate</td>
<td>Acute pain, tenderness, nausea with chance for recovery if local resection is completed within 6 hours</td>
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<tr>
<td>Late (up to POD #10)</td>
<td>Abdominal distention, pseudo-obstruction and ileus. May warrant ‘conservative’ treatment initially</td>
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## Treatment

- No clear therapeutic or diagnostic guidelines
- Early exploratory laproscopy may reduce mortality

## Ultimately . . . 70%+ die

- Early recognition is key
- Signs and symptoms may be absent - diagnostic challenge
- Pain may be a late sign
- In Hasan, all patients with "clear signs" died
- Once infarcted, death is almost certain
- In one review, > 50% of patients with mesenteric ischemic were dead within 3 days of surgery

## Neurological complications

Overall neurological complications in 1 - 2%
- Rarer complications
  - Brachial plexus injury
  - Phrenic nerve injury
  - Vision complications


## Brachial plexus

- **Superficial**
- Fixed proximally & distally
- Positioned between clavicle and 1st rib
- Lower roots (C8 - T1) most commonly stretched
Brachial plexus injury

Incidence 0.2 - 15%
Dependent largely on operative retraction techniques/requirements
Stretch, direct injury, and compression

Proposed risk factors for BPI

IMA dissection and retraction
Intra operative positioning techniques
Sternal retraction
Asymmetric, caudal, extensive
First rib fracture
Central venous catheter placement (IJ)
Advanced age
Prolonged CPB

Consequence of BPI: Upper extremity neuropathy

Occurs in 2 - 15% after open heart surgery
Variety of sensory and motor symptoms in ulnar distribution: numbness, weakness, pain, paresthesia, discoordination
Presence of pain suggests peripheral process

BPI - Prevention & Treatment

Intra operatively, careful positioning, retraction and sternal division (among other practices) may reduce incidence
Supportive treatment
Neurological evaluation for significant dysfunction or persistent discomfort

BPI - Prognosis

“iatrogenic but benign”
Most upper extremity neuropathies s/p cardiac surgery resolve over 3 weeks
In a prospective review
4.6% had symptoms at discharge
~ 1% with symptoms at 3 - 4 months
May last up to 1 year

Phrenic nerve injury (PNI)

Phrenic nerve innervates the diaphragm bilaterally
Arises from C3-C4-C5
Phrenic nerve injury (PNI)

Incidence 10 - 40% in prospective studies
Diaphragmatic dysfunction in approximately 2%
Majority of PNI is unilateral (75% left involvement)
New interest as complication of AF catheter ablation
Most consequential for patients with underlying pulmonary disease and pediatrics

Etiology of PNI

Cold injury related to topical hypothermia
Ice slush felt more injurious than cold saline
Inadvertent stretch
IMA harvest (independent risk factor)

Hemidiaphragm elevation

Diaphragmatic elevation does not equal paralysis
50 - 70% of patients have new diaphragmatic elevation after cardiac surgery
Causes: effusions, atelectasis, postoperative ileus
Paralyzed diaphragms don’t always elevate

Common: Unilateral PNI

50% are asymptomatic
Signs and symptoms may include
- Recruitment of accessory muscles
- Orthopnea, paroxysmal nocturnal dyspnea, mild dyspnea with exertion, symptoms when immersed
- Chest discomfort
No difference in ventilator hours, ICU stay or overall complication rate (in most patients)
Most regain function in 3 - 6 months

Unilateral PNI & COPD

Increased LOS
Increased vent hours
Reintubations
Readmissions
Lower overall survival rates

Bilateral PNI

Very rare
Can result in death or very prolonged ventilation
Diagnosis of suspicion if failure to wean
CXR & PNI

CXR often useful but not specific
PPV results in unreliable CXR (and fluoroscopy)
Upright CXR w/ high false negative rate
End-expiratory CXR in spontaneously ventilating patient in the RECUMBENT position (removes the influence of the abdominal muscles)

Additional diagnostics: PNI

Ultrasound
Nerve conduction studies
Spirometry (restrictive pattern)
Esophageal/gastric catheters to measure trans-diaphragmatic pressure
Fluoroscopy

PNI and pediatric patients

Incidence of diaphragmatic paralysis is high (~ 5%) in pediatric cardiac surgery
- Increased incidence with younger age
- Highest risk related to arterial switch, Fontan and Blalock-Taussig shunt procedures
Spontaneous recovery rare in children
More susceptible to consequences secondary to low ability to recruit accessory muscles and sternal mobility
Early diaphragmatic plication common

PNI prognosis & treatment

Unilateral and uncomplicated by COPD: Good
Unilateral + pre-existing COPD: Less good
Bilateral hemidiaphragm dysfunction: Can be fatal
Limited treatment modalities for adults
- VATS diaphragmatic plication is a emerging treatment

Complications of Vision

Optic neuropathies & cortical blindness
Incidence: 0.05 - 0.1%
Risk factors
- Anemia
- Hypotension
- Emboli
Unintended consequences of management changes?
“Normal” changes

I have no evidence to support this
Visual changes occur - include blurring, sensation that prescription is “off”, floaters, streaks
Attributed to CPB and/or edema
Ophthalmology usually non-informative

Optic neuropathies

Two distinct types of perioperative optic neuropathies
Anterior ischemic optic neuropathy (AION)
Posterior ischemic optic neuropathy (PION)
Occur after many types of surgery (e.g. spinal surgery)
Present hours - weeks after surgery
Linked to ischemia, inflammation, infarction, embolization
Various visual field complaints and moderate-severe vision loss, changes in color vision (PION)

AION after cardiac surgery

More common than PION, but still extremely rare
Proposed mechanisms:
Increased blood viscosity secondary to hypothermia and/or complement activation
Ischemia related to hemodilution, LCOS
Elevation of IOP

Prognosis

In general, the prognosis for AION and PION is poor
Patients may have persistent, bilateral vision loss
Some references indicate a role for transfusion and/or steroids, others indicate there is no adequate treatment strategy
Regardless, consult with specialist

Cortical blindness

Occipital lobe & optic radiation damage related to ischemia and infarct
Attributed to hypotension, anemia, embolization or clot, artheroma, air, or fat
Infarct may be confirmed on MRI or CT
Gradual improvement may occur

Necessary anatomy

Optic nerve with two distinct sections (anterior & posterior) and a vascular source for each
Anterior ischemic optic neuropathy and posterior ischemic optic neuropathy are two distinct complications
Occipital lobe is the primary visual area
Complications of vision

Very rare
Limited therapeutic options
Catastrophic
Increasing rate of some forms (e.g. PION) may be consequence of shifts in post operative management
Recommend early consultation with specialist if suspected

Non infectious sternal dehiscence (NISD)

0.2 - 5% incidence
Related to primary non union, poor wound healing, premature overexertion
Technical issues with bone, wire and surgical technique also implicated
Leads to pain, pulmonary dysfunction and infection

Risk Factors for NISD

DM
Obesity (BMI > 36)
COPD
Smoking
Chronic cough
NYHA FC IV
Osteoporosis
Immunosuppression
Previous sternotomy
Bilateral IMA harvest
“Excessive” transfusions
Prolonged CPB
Non midline division with prophylactic weave

NISD management

Surgical repair/fixation
Rewire, plates, wire removal etc
Flaps
Negative pressure wound therapy (NPWT)

Flaps

Latissimus dorsi
Rectus abdominus
Omentum
Pectoral major (bilat)

Negative pressure wound therapy

Introduced in 1997
Provides sternal & chest cage stability
Negative pressure (-125 mm Hg)
Retrospective reviews suggest equal or improved outcomes, reduced incidence of infection, reduced or similar LOS
RV rupture, graft dehiscence and bronchopleural fistulas reported
Prevention of NISD

Adequate pre operative screening and education
Some advocate prophylactic sternal weave or plating
Role for new therapies (e.g., external fixation garment)

NISD after revision

75% with no or mild limitations
27% with moderate or severe limitation

Better outcomes than those with infectious dehiscence but still > 20% with long term morbidity

Conclusion

Less common complications occur – and range from catastrophic to disabling to “benign”
Early recognition and awareness of the range of complications is important for nurses in a variety of roles
Early recognition can improve outcomes related to some serious complications