Mr GH: Pericardial Window

Anaesthetic Management of Cardiac Tamponade
Mr GH

- 56 yo M
  - HOPCx
    - Asbestosis, adenoCA R lung 8/52
    - 6/52 cisplatin/ taxol chemo
    - Weekly pleural taps for effusions
    - Sent from Bendigo bc of ↑SOBOE now SOBAR
    - Orthopnoea/ PND ++
  - PHx
    - Lumbar spinal fusion
    - L phrenic N palsy 2o CVC insertion
Mr GH

- HR 92, RR 20, O2sat 92% RA, BP 130/80
  - Chest R base PND/ quiet BS
  - Dual HS nil else
  - JVP +4 cm
  - Bilat SOA
Investigations

- TTE
  - Moderate-large circumferential pericardial effusion 1.8-2.3cm depth
  - N RA and RV size and systolic function
  - Marked RA and RV diastolic collapse
  - N LV size, systolic function
<table>
<thead>
<tr>
<th>Monitoring</th>
<th>Time</th>
<th>Event</th>
</tr>
</thead>
<tbody>
<tr>
<td>Machine CK</td>
<td>50</td>
<td></td>
</tr>
<tr>
<td>SpO2</td>
<td>100</td>
<td></td>
</tr>
<tr>
<td>Heart Rate</td>
<td>150</td>
<td></td>
</tr>
<tr>
<td></td>
<td>200</td>
<td></td>
</tr>
</tbody>
</table>

**Anaesthetic Technique (ICOM10)**

- GA: Inhalational alone
- GA: IV and Inhalational
- IV Sedation
- Epidural Infusion
- Spinal infusion
- Caudal infusion

**Infiltration**

- LA injection
- Block injection

**Monitoring Care**

- ECG Lead: 1, 2
- Blood pressure: 115/68
- Heart rate: 100
- SpO2: 98%
- Blood gases: pH 7.45, PaCO2 4.9, PaO2 100

**Performance**

- Time elapsed: 1 hour
- One anaesthetic agent used

**Postoperative**

- Patient monitored for 2 hours
- Blood pressure stable
- Heart rate stable
Most important features

- Reduced LV filling hence fixed stroke volume
  - SNS stim to ↑contractility/ EF and HR to ↑CO
  - Also ↑SVR to maintain MAP
  - CO is dependant on HR
Pathophysiology

- Atria and R side of heart affected first
  - Thinner walled, more easily compressed

- ↓L heart VR
  1. ↓R CO and hence ↓L heart VR
  2. Direct compression and limitation of LV distension and filling
Pathophysiology

- All chambers compressed by effusion
  - When transmural pressure negative, chamber collapses
  - Transmural pressure = $P_{in}(\text{chamber}) - P_{out}(\text{pericardial})$
Pathophysiology

- ↑LVED filling pressures
  - Does not actually mean ↑”preload” bc:
    - Volume of blood to eject next cycle is still low
    - The “real” ED pressure or preload is the transmural pressure (which is decreased)
Pathophysiology

○ “Pressure plateau”

• Pressures across all 4 chambers eventually equalise (lack of distensibility means that any chamber that would naturally distend first squashes all others around it)

• At this point LV filling directly dependant on pericardial compliance (unless LV was already stiffer than pericardium)
Pathophysiology

○ Subendocardial ischaemia

○ Usu only those with pre-existing CAD

○ ↑HR, ↑LVEDP, ↓MAP
PV loop

- ↓LVEDV
- ↓SV
- N or ↑contractility line
- ↑EF hence ↓LVESV
- ↑Afterload line
Factors affecting LV Filling

- **Cardiac cycle**

<table>
<thead>
<tr>
<th>Normal</th>
<th>Tamponade</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood enters atria throughout cardiac cycle</td>
<td>• Total intrapericardial volume (pericardial fluid + blood in heart) is fixed</td>
</tr>
<tr>
<td></td>
<td>• Blood can only enter when blood leaves</td>
</tr>
<tr>
<td></td>
<td>• ie. Atrial filling only occurs during ventricular contraction ($x$ descent – ventricle shortens, atria lengthen, more negative pressure)</td>
</tr>
</tbody>
</table>
Systole

Diastole

Aortic pressure
(at O, the aortic valve opens; at C, it closes)

Left ventricular pressure (---)

Left atrial pressure (---)
(right is similar)

Left ventricular volume
(at C\', the mitral valve closes; at O\', it opens)

Jugular venous pressure,
showing a, c, and v waves

Pressure (mm Hg)

Ventricular volume (mL)
Factors affecting LV Filling

- Cardiac cycle

<table>
<thead>
<tr>
<th>Normal</th>
<th>Tamponade</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diastole allows both ventricle and atria to fill with blood</td>
<td>“Redistribution” of blood in heart from atria to ventricle during diastole – no further atrial filling – means less ventricular filling</td>
</tr>
</tbody>
</table>
Factors affecting LV filling

- **Pulsus paradoxus**
  - Intrapleural pressure inc or dec pressure around pericardium
  - Inspiration: inc R heart VR from dec compression of IVC/SVC but inc PVR so dec VR to L heart
  - Expiration: reverse – L heart fills > R
  - Tamponade: fixed intrapericardial volume means when one side of the heart fills more, the other side must fill less (bulging of IV septum to L and R during insp and exp respectively)
CVP trace

<table>
<thead>
<tr>
<th>Normal X descent</th>
<th>X descent N or inc descent in tamponade</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ventricular contraction lengthens atrial chamber – dec pressure</td>
<td>impaired atrial filling – less blood to distend atrium</td>
</tr>
</tbody>
</table>
CVP trace

![CVP trace diagram](image)

- **Normal Y descent**
  - AV valve opens, allows atrium to empty and ventricle to fill

- **Absent Y descent in tamponade**
  - \(\downarrow\) Rate of atrial emptying (less blood in atria from impaired filling so less pressure grad bw atria and ventricle)
Clinical features

○ Beck’s triad
  ○ ↓BP, ↑JVP, small quiet heart

○ Symptoms and signs of CCF – initially RHF

○ Pulsus paradoxus
  ○ Only if pre-existing ↓LV compliance; if so, expiration phase will not assist LV filling as this is already impaired by poorly compliant LV – eg. LVH, AS, HOCM
Investigations

- **CXR**
  - "globular" heart

- **ECG**
  - Electrical alternans – phasic change in size/ axis of QRS/ p/ t waves
  - Universal low voltages

- **Echocardiography**
  - Effusion size, RA/ RV collapse in diastole, L displacement of IV septum, "septal swing" bw insp/ exp
Investigations

- **Swan-Ganz**
  - Demonstrate equalisation of pressures across all chambers
  - Exceptions:
    - Pre-existing poorly compliant LV (pressure will be higher)
    - Post CTS – open pericardium, isolated chamber compression with clot
Anaesthetic Management

- Severity based
  - Pt grossly decompensated, shocked
    - Subxiphisternal percutaneous drainage under LA
    - Cx haemorrhage, worsened tamponade, coronary artery laceration
  - Signs of SNS compensation but patient stable
    - Pericardial window under GA
    - Open, laparoscopic, thoracoscopic, transbronchial
Anaesthetic Management

○ “Full, Fast, Tight”

○ Fixed SV, so to maintain CO and BP:
  ○ Avoid bradycardia
  ○ Avoid vasodilators
  ○ Attempt optimum volume state to maximise LV filling
  ○ Attempt to maintain sympathetic tone in compensated pt
Anaesthetic Management

- Awake options/ ?Maintenance of SV
  - Avoid further ↓VR caused by IPPV

- Low TV, high RR to avoid extremes of pulsus paradoxus/ ↑PVR
  - Maintain low airway pressures

- "The treatment of myocardial ischaemia must wait on the treatment of tamponade"
Ketamine anesthesia for pericardial window in a patient with pericardial tamponade and severe COPD

[La kétamine utilisée pour l'anesthésie d'une ponction péricardique chez une patiente qui présente une tamponnade et une MPOC sévère]

Tim Aye MD CCFP FRCP, Brian Milne MD MSc FRCP

From the Department of Anesthesiology, Queen’s University, Kingston, Ontario, Canada.

Address correspondence to: Dr. Brian Milne, Department of Anesthesiology, Kingston General Hospital, 76 Stuart Street, Kingston, Ontario K7L 2V7, Canada. Phone: 613-548-7827; Fax: 613-548-1375; E-mail: milneb@post.queensu.ca

Accepted for publication October 31, 2001.
Revision accepted December 12, 2001.
Case report

A 73-yr-old, 72 kg woman with long-standing COPD and cor pulmonale was admitted with pericardial effusion and tamponade. Admission one month prior for drainage of a pericardial effusion was complicated by insertion of a pulmonary artery catheter introducer into the carotid artery and postpericardiocentesis intrapericardial hemorrhage. Her pulmonary artery pressure at that time was 54/25 mmHg. She was discharged home on nasal O₂ 4 L-min⁻¹. Prior to the present admission she experienced increasing dyspnea, orthopnea and peripheral edema. Other medical problems were obesity, stable angina, and hypothyroidism. Medications included enalapril, furosemide, isosorbide dinitrate, ranitidine, thyroxin, salbutamol and ipratropium bromide. She reported allergies to penicillin, codeine and morphine. Blood pressure on admission was 130/80 mmHg, heart rate 90 beats-min⁻¹, respiratory rate 24 breaths-min⁻¹ and a pulsus paradoxus of 10 mmHg. Breath sounds were decreased bilaterally with bibasilar crackles. Her SpO₂ was 98% on nasal O₂ 4 L-min⁻¹, and jugular venous pressure (JVP) was 3 cm H₂O.

The following day she was in distress with increasing shortness of breath. She was unable to lie down stating she had never been this bad before. There had been no improvement with salbutamol inhalation and she had been started on prednisone. Her blood pressure was 116/68 mmHg, heart rate 110 beats-min⁻¹, pulsus paradoxus 26 mmHg and SpO₂ 93% on nasal O₂ 4 L-min⁻¹. Examination revealed decreased air entry with faint wheezes, JVP 8 cm H₂O, with poorly palpable peripheral pulses. The patient refused to have another pericardiocentesis “awake” because of her previous experience and requested surgical drainage be performed under a general anesthetic.
function tests were normal, an arterial blood gas on 4 L·min⁻¹ nasal O₂ showed pH 7.38, PO₂ 78.9 mmHg, PCO₂ 71.8 mmHg, HCO₃⁻ 37.5 mmol·L⁻¹. Her elettricle with systolic and diastolic collapse. Under echo
guidance, in the intensive care unit (ICU) in the sit-
ting position, 400 mL of chocolate coloured pericar-
dial fluid was drained. Three boluses of midazolam 0.5
mg and propofol 20 mg iv were given during the pro-
cedure. Hemodynamics remained stable throughout
the procedure and at the termination, right ventricu-
lar collapse was still present on echocardiogram. In the
operating room with continuous intra-arterial moni-
toring, automated segment lead I, II, and V analysis,
nasal O₂ 4 L·min⁻¹, the patient was prepped and
draped in the sitting position. After administration of
glycopyrrolate 0.3 mg, and midazolam 1 mg, anesthe-
sia was induced with ketamine in 25 mg increments to
a total of 100 mg. Blood pressure was 120 mmHg sys-
tolic, and heart rate 100 beats·min⁻¹. The patient was
gradually returned to the supine position and lidoc-
caine 1% was infiltrated locally. Subxiphoid drainage of
1000 mL of effusion increased her blood pressure to
140 mmHg systolic. Heart rate was 100–115
beats·min⁻¹ and SpO₂ >93% throughout. An intraop-
erative blood gas postdrainage showed a pH 7.28,
PO₂ 102 mmHg, pCO₂ 96 mmHg, HCO₃⁻ 44
mmol·L⁻¹. A total of 450 mg of ketamine were used
during the procedure. The xiphisternum and a large
Ketamine

○ Phenylcyclclidine derivative
  ○ Dissociative anaesthetic
  ○ “relative” preservation of laryngeal reflexes
  ○ Displaces NAd from SNS ganglia
  ○ Direct negative inotrope in the NAd deplete
  ○ ↑HR ↑SVR ↑contractility ↑BP
  ○ Potentially ↑PVR

○ Mild ↓MV w ↑RR
○ Bronchodilator
Ketamine

- **Benefits**
  - Attractive CVS profile
  - Use for awake analgesia/induction/maintenance

- **Disadvantages**
  - More $\uparrow$PVR > $\uparrow$SVR ?overall $\downarrow$ LV output
  - Potential worsening of contractility
Ketamine

○ Reviews

○ *Spotoff et al* – anecdotal reports of severely ↑ R ventricular failure w use of ketamine induction - ↑PVR w normal PaO2/ CO2

○ *Rees et al* – No ↑in shunt fraction with ketamine vs enflurane in 1-lung anaesthesia

○ *Balfors et al* – No ↑PVR w ketamine anaesthesia during IPPV