**MHIF FEATURED STUDY:**
**TAMBE**

**DESCRIPTION:**
Evaluation of the GORE® EXCLUDER® Thoracoabdominal Branch Endoprosthesis in the Treatment of Thoracoabdominal and Pararenal Aortic Aneurysms. This study will look at treating thoracoabdominal or pararenal aneurysm disease with a new stent-graft design.

**CONDITION:**
Para/Juxarenal AAA disease

**PI:**
Jesse Manunga, MD

**RESEARCH CONTACT:**
Jo Anne Goldman RT. CCRC
Joanne.goldman@allina.com | 612-863-3793

**SPONSOR:**
W.L. Gore

**OPEN AND ENROLLING!**
EPIC message: Research MHIF Patient Referral

**CRITERIA LIST/QUALIFICATIONS:**

<table>
<thead>
<tr>
<th>Inclusion</th>
<th>Exclusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Aortic aneurysm involving the visceral vessel(s) requiring treatment defined as at least one of the following:</td>
<td>1. Prior open, aortic surgery of the ascending aorta or aortic arch</td>
</tr>
<tr>
<td>• Fusiform aneurysm diameter ≥ 5 cm</td>
<td>2. Ruptured or leaking aortic aneurysm</td>
</tr>
<tr>
<td>• Saccular aneurysm (no diameter requirement)</td>
<td>3. Aneurysmal dilatation due to chronic aortic dissection</td>
</tr>
<tr>
<td>• Rapid aneurysm growth (≥ 5 mm in one year)</td>
<td>4. Infected aorta</td>
</tr>
<tr>
<td>2. Aortic aneurysm that involves the abdominal aorta, with:</td>
<td>5. Mycotic aneurysm</td>
</tr>
<tr>
<td>• Involvement of at least one visceral vessel and aneurysmal extension as far as 65 mm proximal to the celiac artery, and/or</td>
<td>6. Life expectancy &lt;2 years</td>
</tr>
<tr>
<td>• No normal aorta between the upper extent of aneurysm and renal artery(s)</td>
<td></td>
</tr>
</tbody>
</table>
ECMO Management of Amniotic Fluid Embolism

12/14/2020

Jonathan Urbach MD, Cardiology Fellow
Minneapolis Heart Institute
Disclosure

- None
Case

29-year-old healthy G1P1 presented at 39+0 for elective IOL

- Given misoprostol and went into active labor
- At 748 started feeling presyncopal and fetal bradycardia noted
- Arrive in OR at 754 for emergent Caesarean section
- During c-section, she had hemodynamic deterioration and ultimately PEA arrest
- No significant bleeding noted at this time, though the tissues were described as dusky in the op report
- CODE BLUE called at 0812
• LUCAS, ACLS

• 0829 VT
  ○ DCCV x3 0831, 0832, 0835

• Completion of the emergent c-section
  ○ Noted that she was seeping through the sterile dressing and from all line sites
  ○ Massive transfusion protocol
ECMO Activation

- ECMO activated in setting of refractory arrest
  - Working diagnosis was amniotic fluid embolism

- Cannulated for peripheral VA-ECMO via RFA and RFV and was on flow at 0900

- Continued to have uterine bleeding, but improving with blood product resuscitation

- Taken to cath lab for confirmation of cannula position as well as placement of a PA catheter and arterial line
Outline

- Indications / Contraindications for ECMO

- ECMO Complications
  - Thrombotic and hemorrhagic

- Anticoagulation in ECMO
  - AC of choice, monitoring strategies, institutional variation
  - ROTEM

- Amniotic fluid embolism
  - Pathophysiology, diagnosis, management
  - Role of ECMO
ECMO

● Indications
  ○ Inadequate perfusion (hypotension, low CO) despite adequate intravascular volume
  ○ Shock that persists despite volume, vasoactive meds, and IABP (if appropriate)
  ○ As a bridge
    ■ Recovery
    ■ Transplant
    ■ Durable MCS

● Contraindications
  ○ Absolute: unrecoverable condition / not a candidate for VAD or transplant, advanced age, chronic organ dysfunction (emphysema, cirrhosis, renal failure), compliance (financial, cognitive, psychiatric, or social limitations), or prolonged CPR without adequate tissue perfusion
  ○ Relative: unable to receive anticoagulation, advanced age, obesity
ECMO Stats

- Survival to hospital discharge for adults requiring ECMO 57% (respiratory illness) and 42% (cardiac disease)

- Bleeding / thrombotic complications are common with ECMO
  ○ In review of ELSO data from 2016:
    - Bleeding from surgical or cannula insertion sites 10-30%
    - CNS hemorrhage 2.2%-6%
    - Oxygentor thrombosis 7-13%
    - CNS infarction 2-4.4%
Anticoagulation in ECMO

- **Strategies for Anticoagulation**
  - Counterbalance exposure to non-endothelial surface of ECMO circuit
    - Bioactive coatings on surfaces limit coagulation response to circuit
  - Unfractionated heparin is the current international standard
  - Optimal anticoagulation (prevent thrombosis / limit bleeding risk) remains unknown

- **Monitoring Anticoagulation**
  - aPTT, ACT, anti-Xa, thromboelastography, absolute heparin dose
    - All used alone or in combination; no ideal strategy / consensus guidelines for monitoring

In the balance...
HELP-ECMO Pilot

- Randomized, controlled, un-blinded pilot study at 2 ICUs in Australia
  - VA and VV ECMO pts randomized to either “standard” (aPTT target 50-70s) or low-intensity heparin protocol (aPTT target <45 seconds)
    - 31 pts; 9/31 (29%) VA, 22/31 (71%) VV
    - 16 randomized to low-intensity protocol, 15 to therapeutic dose heparin

- Primary endpoint:
  - Difference between mean heparin dose and aPTT, anti-Xa levels

- Secondary endpoints:
  - Thromboembolic events, ECMO circuit thrombosis, bleeding events

<table>
<thead>
<tr>
<th></th>
<th>Low-intensity (n=16)</th>
<th>Standard (n=15)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>mean aPTT</td>
<td>48.1</td>
<td>56.2</td>
<td>0.03</td>
</tr>
<tr>
<td>mean anti-Xa</td>
<td>0.11</td>
<td>0.3</td>
<td>0.003</td>
</tr>
<tr>
<td>mean heparin dose</td>
<td>11784</td>
<td>22050</td>
<td>0.004</td>
</tr>
<tr>
<td>DVT</td>
<td>2</td>
<td>3</td>
<td>0.57</td>
</tr>
<tr>
<td>PE</td>
<td>1</td>
<td>0</td>
<td>0.33</td>
</tr>
<tr>
<td>CVA</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>intracardiac thrombus</td>
<td>1</td>
<td>2</td>
<td>0.51</td>
</tr>
<tr>
<td>distal perfusion</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>cannula thrombosis</td>
<td>2</td>
<td>0</td>
<td>0.16</td>
</tr>
<tr>
<td>acute pump</td>
<td>1</td>
<td>1</td>
<td>0.96</td>
</tr>
<tr>
<td>thrombosis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ICH</td>
<td>0</td>
<td>1</td>
<td>0.29</td>
</tr>
<tr>
<td>RPH</td>
<td>1</td>
<td>1</td>
<td>0.96</td>
</tr>
<tr>
<td>GI</td>
<td>0</td>
<td>2</td>
<td>0.13</td>
</tr>
<tr>
<td>haemoptysis</td>
<td>1</td>
<td>1</td>
<td>0.96</td>
</tr>
</tbody>
</table>
ECMO without AC

- Retrospective, single-center analysis
  - 203 adult patients treated with VA-ECMO
  - Primary endpoint:
    - Composite of hemorrhagic and thrombotic complications
  - Secondary outcomes:
    - Transfusion needs, HIT, hospital LOS, in-hospital mortality

- 35% (n = 75) were not anticoagulated
  - Lower complication rates in this group (57% vs 76%; p=0.007)
    - No difference in mortality, pump failure, or thrombotic complications
Meta-analysis of ECMO without AC

- 6 case series included (n=70)
  - 84% VA-ECMO

- Reason for no AC
  - High risk of bleeding after CV surgery (64%)
  - Active major bleeding (23%)
  - Severe traumatic injury (9%)

- Successful ECMO wean in 74% and survival to hospital dc 58%
  - There was significant variation in rates of circuit thrombosis
  - Patient complications inconsistently reported; small numbers
• Current ELSO guidelines recommend initial heparin infusion rate of 7.5-20.0 u/kg/h
  • No standardized method to achieve and monitor AC during ECMO
  • AC management / monitoring varies by institution
Institutional Variation

- Anticoagulant used
- Monitoring strategy
Institutional Variation

- Survey of transfusion and anticoagulation practices in adults at ECMO centers (54/166 surveyed)
  - 45/47 use heparin as primary AC
  - 1/47 uses heparin and bivalirudin equally
  - 1/47 uses bivalirudin only
<table>
<thead>
<tr>
<th>Drug</th>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unfractionated heparin</td>
<td>Well known</td>
<td>Non-linear, variable effect</td>
</tr>
<tr>
<td></td>
<td>Mechanism known</td>
<td>Possible HIT induction</td>
</tr>
<tr>
<td></td>
<td>Easy to antagonise (protamine)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Easy to monitor (aPTT/ACT)</td>
<td></td>
</tr>
<tr>
<td>Low-molecular-weight heparin</td>
<td>Easy to administer</td>
<td>Accumulation in renal impairment</td>
</tr>
<tr>
<td></td>
<td>Lower risk of HIT induction</td>
<td>Can only be partially antagonised</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Not easy to monitor (aXa levels)</td>
</tr>
<tr>
<td>Direct thrombin inhibitors</td>
<td>Independent of AT levels</td>
<td>No antagonist</td>
</tr>
<tr>
<td>- Bivalirudin</td>
<td>Good dose response</td>
<td>Lesser coagulation inhibition in areas of stasis</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Argatroban</td>
<td>Mainly renal clearance</td>
<td>Ceiling effect in aPTT</td>
</tr>
<tr>
<td></td>
<td>Mainly hepatic clearance</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Could interfere with INR measurement</td>
</tr>
<tr>
<td>Antiplatelet drugs</td>
<td>Inhibit coagulation at starting point</td>
<td>No sufficient anti-coagulation</td>
</tr>
<tr>
<td></td>
<td>Might reduce platelet consumption</td>
<td>No sufficient evidence</td>
</tr>
</tbody>
</table>

ACT = activated clotting time; aPTT = activated partial thromboplastin time; AT = anti-thrombin; HIT = heparin-induced thrombocytopenia; INR = international normalised ratio; VET = viscoelastic test.
Institutional Variation

- Anticoagulant used
- Monitoring strategy
Table 2: Monitoring coagulation

<table>
<thead>
<tr>
<th>Standard coagulation tests</th>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>aPTT (sec)</td>
<td>Well known Monitoring UFH Easy to interpret</td>
<td>Inter-laboratory variance (could be excluded by using ratio) Time consuming</td>
</tr>
<tr>
<td>ACT (sec)</td>
<td>Bedside method Easy to use Immediate results</td>
<td>Relatively insensitive to low doses of UFH Different devices with different reference ranges</td>
</tr>
<tr>
<td>Anti Xa assay (IU/ml)</td>
<td>Sensitive to UFH</td>
<td>Time consuming Needs calibration Free haemoglobin &amp; bilirubin could be underestimated</td>
</tr>
<tr>
<td>VETs (ROTEM/TEG)</td>
<td>Inhibit coagulation at starting point Might reduce platelet consumption</td>
<td>Poor specificity and sensitivity regarding therapy adjustment</td>
</tr>
<tr>
<td>Fibrinogen mg/L</td>
<td>Consumption marker</td>
<td>Increased in inflammatory situations Time consuming</td>
</tr>
<tr>
<td>D-dimer (mg/l)</td>
<td>Prognostic value for oxygenator failure</td>
<td>Time consuming Expensive</td>
</tr>
<tr>
<td>AT (%)</td>
<td>Heparin resistance (partial) Pro-coagulatory marker</td>
<td>Heparin resistance not completely relying on AT</td>
</tr>
<tr>
<td>Haemoglobin (g/dl)</td>
<td>Easy and fast</td>
<td>Not very relevant for coagulation</td>
</tr>
<tr>
<td>Platelet count 10^9/l</td>
<td>Easy and fast</td>
<td>No proven threshold Platelet count does not reflect platelet function</td>
</tr>
</tbody>
</table>

ACT = activated clotting time; aPTT = activated partial thromboplastin time; AT = antithrombin; ROTEM = rotational thromboelastometry; UFH = unfractionated heparin; TEG = thromboelastography; VET = viscoelastic test.
Rotational Thrombelastometry

- ROTEM is a method for measuring the quality of hemostasis
  - Uses viscoelastic properties of a blood clot
  - Measures clot formation, clot integrity, and the presence of fibrinolysis
<table>
<thead>
<tr>
<th>Test name</th>
<th>Reagent</th>
<th>Use</th>
</tr>
</thead>
<tbody>
<tr>
<td>INTEM</td>
<td>Ellagic acid</td>
<td>Intrinsic pathway defects of coagulation activation</td>
</tr>
<tr>
<td>EXTEM</td>
<td>Recombinant tissue factor</td>
<td>Extrinsic pathway defects of coagulation activation</td>
</tr>
<tr>
<td>FIBTEM</td>
<td>Recombinant tissue factor and Cytochalasin D (platelet inhibitor)</td>
<td>Assesses for fibrinogen deficiency by blocking platelet contribution to clot formation</td>
</tr>
<tr>
<td>APTEM</td>
<td>Recombinant tissue factor and Aprotinin (fibrinolysis inhibitor)</td>
<td>Assesses for hyperfibrinolysis</td>
</tr>
</tbody>
</table>
Back to our case...

**COAGULATION**

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>D-DIMER, QUANTITATIVE</td>
<td>&gt;4.00 *</td>
</tr>
<tr>
<td>PROTIME</td>
<td>26.6 *</td>
</tr>
<tr>
<td>INR</td>
<td>2.5 *</td>
</tr>
<tr>
<td>APTT</td>
<td>95 *</td>
</tr>
<tr>
<td>FIBRINOGEN, QUANTITATIVE</td>
<td>100</td>
</tr>
</tbody>
</table>

**INTEM** [demo]

```
CT: 512 s [127 - 208] ▲
CFT: 814 s [45 - 110] ▲
a: 22 % [70 - 81] ▼
A10: 16 mm [46 - 67] ▼
A20: 25 mm [51 - 72] ▼
MCF: 32 mm [51 - 72]
ML: 0 %
```

**EXTERN** [demo]

```
CT: 231 s [43 - 82] ▲
CFT: 707 s [48 - 127] ▲
a: 23 % [63 - 80] ▼
A10: 38 mm [46 - 67] ▼
A20: 26 mm [50 - 70] ▼
MCF: 33 mm [52 - 70]
ML: 0 %
```

**FIBTEM** [demo]

```
CT: 2712 s
CFT: s
a: *
A10: 1 mm [7 - 22] ▼
A20: 2 mm [7 - 24] ▼
MCF: 3 mm [7 - 24] ▼
ML: 73 %
```
Correction of Coagulopathy

- Massive transfusion protocol
  - RBC - 8u
  - PLT - 5u
  - FFP - 5u
  - Cryo - 5u
ROTEM 1341

---

**INTEM**

- CT: 217 s (122 - 208)
- CFT: 230 s (45 - 110)
- $\alpha$: 50° (70 - 81)
- A10: 37 mm (46 - 67)
- A20: 47 mm (51 - 72)
- MCF: 53 mm (51 - 72)
- ML: 0%

---

**EXTEM**

- CT: 59 s (43 - 82)
- CFT: 232 s (48 - 127)
- $\alpha$: 53° (65 - 80)
- A10: 38 mm (46 - 67)
- A20: 48 mm (50 - 70)
- MCF: 54 mm (52 - 70)
- ML: 0%

---

**FIBTEM**

- CT: 57 s
- CFT: 48 s
- $\alpha$: 48°
- A10: 8 mm (7 - 22)
- A20: 9 mm (7 - 24)
- MCF: 9 mm (7 - 24)
- ML: 0%
Amniotic Fluid Embolism

Amniotic Fluid Embolism

- Catastrophic complication of pregnancy
  - Cardiovascular collapse
  - Respiratory failure
  - Coagulopathy

- Incidence estimated to be 1/8000 - 1/80,000 deliveries
  - Inaccurate diagnosis/inconsistent reporting of cases

- Mortality rate
  - Prior estimates with mortality rate as high as 86%
  - More recent estimates 13-26%
    - Neurologically intact survival remains low, 15% of women

Disruption of the maternal-fetal interface

- Amniotic fluid enters maternal circulation
- Accompanied by procoagulants and fibrinolytic activators
Maternal hematologic effects

- Mediated by fetal prothrombotic substances, plasminogen activator, and plasminogen activator inhibitors
- Ultimately leads to DIC
Maternal Cardiovascular Effects

- Unbalanced activation of vasoactive substances
  \[\rightarrow\text{vasoconstriction of pulmonary vasculature with resultant hypoxemia and PH}\]
  \[\rightarrow\text{Cor pulmonale, cardiac arrest}\]
Disruption of the maternal-fetal interface allows amniotic fluid as well as procoagulants and fibrinolytic activators from injured blood vessels to gain access to the maternal circulation, contributing to the development of amniotic fluid embolism syndrome.

Maternal cardiovascular effects
Abnormal activation of humoral and immunologic processes leads to vasocstriction of the pulmonary vasculature, hypoxia, and pulmonary edema.

Maternal hematologic effects
Exposure:
- Prothrombotic substances
- Platelet activating factors
- Platelet activating factor inhibitors

Fetal effects
Maternal cardiovascular and hematologic changes
Placenta
Hypoxia
Abnormalities in the fetal heart tracing
Without prompt delivery, hypoxic-ischemic encephalopathy or death occurs

Disseminated intravascular coagulopathy
Hemorrhage

Phase 1
Pulmonary vascular changes cause right ventricular dilatation, which can result in increased preload constraint, profound hypoxia, and cardiac arrest.

Phase 2
Resolution of heart failure on the right side leads to heart failure on the left side and pulmonary edema.

If the patient survives, she will enter phase 2.

Normal heart
Compressed D-shaped LV
Right ventricular volume overload
Flattened septum

Lev. v.
Clinical Presentation

- **Symptoms often sudden**
  - Prodrome typically involves acute dyspnea or cough, AMS, sudden hypoxia
    - Up to $\frac{1}{3}$ of patients describe an aura of impending doom, chills, nausea/vomiting
  - Hypotension quickly follows
    - Postulated to be related to obstructive shock vs anaphylactoid reaction
  - Sudden cardiorespiratory failure
    - Typically related to sustained VT or VF, occasionally bradyarrhythmia

- If patient survives the initial cardiovascular collapse
  - DIC -> hemorrhage seen in >80% of pts with AFE
  - Seizures and/or stroke
    - Rarely reported as initial manifestations or complications of AFE
    - Case reports suggest related to presence of PFO

References:
AFE Diagnosis

- Society for Maternal-Fetal Medicine and the AFE Foundation proposed the following diagnostic criteria (all must be present)
  - Sudden cardiac arrest or hypotension (SBP<90) with evidence of respiratory compromise (e.g. dyspnea, hypoxia, cyanosis)
  - Documentation of DIC (using modified ISTH criteria)
    - PLT > 100,000 = 0; < 100,000 = 1; < 50,000 = 2
    - Prolonged PT or INR (<25% increase = 0, 25-50% = 1, >50% = 2)
    - Fibrinogen >200 mg/L = 0; < 200 = 1
      - Score of ≥3 compatible with overt DIC
  - Onset during labor or within 30 min of placental delivery
  - Absence of fever during labor

AFE Risk Factors

- Large, population-based studies evaluating multiple risk factors
  - Several identified, some discordance between studies
  - Most commonly cited risk factors
    - Cesarean delivery
    - Instrumented vaginal delivery
    - Placental abnormalities (previa, abruption, accreta)
    - Preeclampsia/eclampsia
  - No clinical or demographic risk factors consistently identified

Lancet. 2006;368(9545):1444.
Management of AFE

- SMFM Guidelines
  - Immediate high quality CPR (standard BCLS and ACLS) (Grade 1C)
  - Multidisciplinary team (Best Practice)
  - Vasopressors, antiarrhythmics, and defibrillation should be used with standard doses
  - Emergent perimortem cesarean performed simultaneously

**TABLE 1**

Components of high-quality cardiopulmonary resuscitation in pregnancy

<table>
<thead>
<tr>
<th>Components</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rapid chest compressions (100 x minute)</td>
</tr>
<tr>
<td>Perform hard compressions, achieving a depth of at least 2 inches</td>
</tr>
<tr>
<td>Assure adequate chest recoil between compressions</td>
</tr>
<tr>
<td>Minimize interruptions of chest compressions</td>
</tr>
<tr>
<td>Avoid prolonged pulse checks (no more than 5—10 seconds)</td>
</tr>
<tr>
<td>Resume chest compressions immediately after defibrillating</td>
</tr>
<tr>
<td>Switch provider of compressions every 2 minutes to avoid fatigue</td>
</tr>
<tr>
<td>Lateral displacement of uterus during resuscitation</td>
</tr>
</tbody>
</table>

AFE suspected

Immediate notification to neonatology, maternal-fetal medicine and/or obstetric care provider, anesthesiology, intensive care

Start immediate high quality CPR-ACLS and call for help.

Consider immediate delivery in viable pregnancies either by operative vaginal delivery or emergent cesarean section.

Early phase commonly characterized by right ventricular failure. May confirm with bedside transthoracic echocardiography.

Avoid excessive fluid resuscitation. Consider norepinephrine to maintain blood pressure. Right ventricular failure addressed with inotropes, such as dobutamine or milrinone. Decrease pulmonary afterload with inhaled nitric oxide or inhaled/intravenous prostacyclin if indicated.

Second phase characterized by left ventricular failure and cardiogenic pulmonary edema. Maintain hemodynamics with use of norepinephrine and inotropes, such as dobutamine or milrinone. Limit excessive fluid administration.

Coagulopathy may have immediate or delayed onset following cardiovascular collapse. Activate massive transfusion protocols where available. Aggressive treatment of uterine atony. Search for anatomic etiologies of bleeding such as pelvic lacerations.
Management of AFE

- Multidisciplinary, team-based approach recommended
  - CPR
  - Control hemorrhage / reverse coagulopathy
  - Deliver the fetus
  - Exclude alternative diagnoses
    - PE, sepsis, MI, OB hemorrhagic shock, air embolism, anesthetic complications, etc.

- Improved coordination of resuscitation postulated to account for improved mortality rate
  - What about ECMO?
Current Guidelines

• “The use of VA-ECMO has been described in cases of AFE refractory to conventional resuscitation maneuvers. However, the use of AC during ECMO may worsen bleeding in the profoundly coagulopathic patient with active hemorrhage. Because of these concerns, as well as lack of adequate evidence of benefit, ECMO is controversial and not routinely recommended in the management of AFE.”
Cases

Extracorporeal Therapies for Amniotic Fluid Embolism

Use of Extracorporeal Membrane Oxygenation for Amniotic Fluid Embolism Syndrome Immediate Temporal Extracorporeal Circuit Support for Catastrophic Amniotic Fluid Embolism

Author:

Sudheer N, Sun

Case report:

Michael S Firstenberg, Dimitrova, David Cohn, Philip Samuels

Records of the Massachusetts General Hospital

Case:

2012 — A 43-Year-Old Woman with Cardiopulmonary Bypass and Cesarean Section

Jeffrey L. Ecker, M.D., Ken Solt, M.D., Michael G. Fitzsimons, M.D., and Thomas E. MacGillivray, M.D.
Case

- 45 G2P0, h/o prior myectomy admitted 37.5 weeks for elective Cesarean
  - 1 minute after clamping cord --> acute SOB, desats, hypotension, and LOC
  - Intubation, CPR
  - TEE within 10 minutes of arrest
    - Dilated, akinetic RV
    - Underfilled, hyperdynamic LV
  - Central cardiopulmonary bypass 53 minutes following arrest
    - 30,000u heparin at start of run
    - Coagulopathy corrected with blood products
  - TEE showed normalization of RV function
    - Decannulated after 83 minutes of run time
  - Extubated POD1, DC POD7 with near complete recovery (memory loss surrounding event)
Case

- 34 G7P3A3 admitted at term in labor
  - Cervical dilation to 3cm, reassuring FHT
  - 4 hrs after admission, sudden onset of sharp thoracic pain, dyspnea, and cyanosis
    - Followed by maternal disorientation and fetal bradycardia
  - Emergency cesarean section under local anesthesia
  - During C-section:
    - Maternal tachycardia, loss of pulse, DIC
    - CPR, massive transfusion, DCCV
    - Bedside u/s with dilated RV, underfilled/hyperdynamic LV
  - Peripherally cannulated VA-ECMO along with an IABP
    - Heparin administered
    - MCS weaned at 40 hrs post-partum
    - Discharged after 24 days without complication
Case

- 36F primigravida with twins
  - Gestational DM and HTN
  - Admitted at 30x5 with uncontrolled progression of labor → C-section under general anesthesia
  - Shortly after extubation, became cyanotic and hypotensive → PEA arrest
  - CPR and “immediate” cannulation for VA-ECMO
    - 3,000u heparin administered
    - Weaned off of ECMO at ~72 hrs
  - Labs consistent with DIC
  - CTA without PE
  - Trach placed on day 7
  - Prolonged encephalopathy, discharged to nursing home on HD 40 with cognitive impairment
# TEE in AFE

<table>
<thead>
<tr>
<th>Year</th>
<th>Study</th>
<th>Findings</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>2010</td>
<td>Lee et al.</td>
<td>Severe right ventricular dysfunction&lt;br&gt;Free-floating clot in the right and left atria</td>
<td>Cardiopulmonary bypass and survival</td>
</tr>
<tr>
<td>2009</td>
<td>Vellayappan et al.</td>
<td>Enlarged right ventricle&lt;br&gt;Moderate right ventricular hypokinesis&lt;br&gt;Large mass in the right atrium through patent foramen ovale&lt;br&gt;Dilated tricuspid valve annulus&lt;br&gt;Trace-to-mild tricuspid regurgitation&lt;br&gt;Normal left ventricle</td>
<td>Cardiopulmonary resuscitation and survival (pathology report showed squamous-cell epithelium in the mass)</td>
</tr>
<tr>
<td>2004</td>
<td>James et al.</td>
<td>Normal left ventricular contractility&lt;br&gt;D-shaped left ventricle&lt;br&gt;Enlarged pulmonary artery and right ventricle&lt;br&gt;Sluggish flow in the pulmonary arteries</td>
<td>Cardiopulmonary resuscitation and death</td>
</tr>
<tr>
<td>2003</td>
<td>Stanton et al.</td>
<td>Massive right ventricular dilatation and akinesis&lt;br&gt;Vigorous, small left ventricle</td>
<td>Cardiopulmonary bypass and survival</td>
</tr>
<tr>
<td>1999</td>
<td>Shechtman et al.</td>
<td>Right ventricular failure&lt;br&gt;Bulging of interatrial septum and interventricular septum toward the left&lt;br&gt;Severe tricuspid regurgitation&lt;br&gt;Small and decompressed left ventricle</td>
<td>Cardiopulmonary resuscitation and death</td>
</tr>
</tbody>
</table>
The Case for ECMO in AFE

- Cardiovascular collapse is a hallmark of AFE

- Standard of care is supportive in nature
  - Case can be made that ECMO cannulation should be considered
  - Anticoagulation used in many of these cases, though may not be needed if deemed too risky
  - The importance of multidisciplinary teams in the rapid management of presumed AFE

- Guidelines lag behind
Back to our case...

- Initial hemodynamics upon arrival to ICU
  - 3-4 LPM at 3500 RPM
  - PA 38/28 (32), RA 20, MAP 94
  - Started on dobutamine, lasix gtt, and nitroprusside

- No heparin was administered with cannulation in setting of hemorrhage
  - Decision made to forgo AC during ECMO run pending correction of coagulopathy
  - In coordination with OB/GYN, intensivists, and heart failure teams → started low-intensity fixed rate heparin (500 u/hr on 3/24, ~32.5 hrs after starting run)

- Hemodynamics remained stable
  - Limited turndown echo 3/24 AM reassuring
  - Full turndown 3/25 with therapeutic AC followed by decannulation (51.3 hrs total run time)
3/24
Trouble waking with sedation reduction
- Neurology consulted
- Decreased movement R>L
- No movement of legs
- Not following commands

3/25
CT head
- Unremarkable
- Normal gray-white definition

3/26
MR brain
Weaned off of vasoactive medications

Ongoing acute encephalopathy
- Few signs of improvement
- Not following commands
- Efforts to wean sedation limited by agitation

Family meeting
- While neurologic prognosis unclear, proceed with restorative care
- Plan for trach/PEG and eventual LTACH
4/7  Trach placed

4/8  PEG placed
   - Gazes to left, able to track to the right
   - Not following commands consistently
   - Moves limbs spontaneously

4/9  Able to wean off of sedation completely
Continued improvement

- Now consistently following commands
- Mouths answers to questions
- Neurology notes “good prognosis” for first time

Discharged to CKRI

- Bilateral R>L weakness
- Ambulating with walker
- Impaired cognition

Discharged home

- Fully independent
- Return to normal cognition
- No appreciable deficits
Close to home

- A truly devastating turn of events in a moment supposed to be joyful
- An outcome worth celebrating
- Personal connection

Thank You!
Future Directions

• ELSO Registry
  • All cases of AFE managed with ECMO in the past 5 years
  • Will analyze clinical data and short-term outcomes
  • Collaboration with cardiology, ICU, OB/GYN
  • No prior case-series has been published