Minimising microbubble delivery in CPB

Jon Bering Kristensen, ECCP
Århus Universitetshospital, Skejby
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Pathophysiology of microbubbles

Etiology

Minimizing, practical guidelines
Neurological impairment in adult cardiac surgery:

50-70% one week postOp

30-40% three months postOp
Attention, concentration, short term memory and hand-eye coordination

(Pugsley et. al 1994)
Pathophysiology

Activation:

- Coagulation
- Complement
- Immune

- Platelets & leucocytes (Philp et.al. 1972)
- Platelets 1987 (Thorsen et.al.)
- Complement (Pekna et.al. 1993)
Ischemia

With no obstruction, still inflammation and oedema, decreased cerebral blood flow

Decreased cerebral blood flow progressively up to 90 min after exposure.

(Helps SC et.al. 1990)

Earlier studies suggested only microbubbles >35 $\mu$m was associated with increased morbidity.
Pathophysiology

- Size
- Volume, numbers
- Composition
Pathophysiology

Size:

<10 µm all passes the capillary bed.

10- 15 µm generally passes the capillary bed, 1/3 showed transient blockage.

>15 µm blockage of the capillary bed.

(Feinstein et.al.1984)
Pathophysiology

Numbers:
Direct correlation between GME numbers and change in accuracy of memory

(Stump, et. al. 1996, Fearn et.al. 2001)

<table>
<thead>
<tr>
<th>HITS count during CPB</th>
<th>No. of patients</th>
<th>No. with deficit</th>
<th>% with deficit</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;200</td>
<td>58</td>
<td>5</td>
<td>8.6</td>
</tr>
<tr>
<td>201-500</td>
<td>13</td>
<td>3</td>
<td>23.1</td>
</tr>
<tr>
<td>501-1000</td>
<td>16</td>
<td>5</td>
<td>31.3</td>
</tr>
<tr>
<td>&gt;1000</td>
<td>7</td>
<td>3</td>
<td>43</td>
</tr>
</tbody>
</table>

Many small bubbles as bad as a few large.
(Pugsley et.al.1994)
Pathophysiology

Composition:

Solubility:
CO₂, O₂, N

2-32 minutes for oxygen:nitrogen ratio 1:1

(Dexter et.al. 1998)

Foam like bubbles increases absorption time
Etiology

Venous air
Fluid addition
Drug administration
Blood sampling
Vent & suction
Residue from priming
Rollerpump cavitation
Temperature gradients
Low level venous reservoir
Oxygenator  bubble migration
Etc.....
Etiology

Venous air
Fluid addition
Drug administration
Blood sampling

Oxygenator bubble migration
Venous air:

Major source of GME in the arterial line

Not completely de-aired venous line, pre- bypass
incl. unprimed venous line to reduce volume

Air from around the purse string sutures.
From right atrium during surgery.

Augmented by VAVD. GME increases x 10 in the arterial line
(Willcox et. al. 2002)
Etiology

Arterial line counts post arterial filter  
(Willcox et al. 1999)
Figure 7. Time course of CABG procedure. Every peak is related with manipulation, which generates bubbles. Bubble activity can be observed in front and behind the oxygenator.
Etiology

Fluid addition
Drug administration
Blood sampling

Small bubbles of air is introduced through the sampling manifold along with drugs or purging of the sampling manifold during blood sampling.

(Borger et. al. 2002)
Etiology

• Perfusion interventions

Up to 75% of GME correlates with perfusionist intervention.

(Taylor et.al.1999, Borger et.al. 2002)
The more interventions, the higher degree of neuropsychological impairment.

More versus less: 10 interventions showed increased impairment.

(Borger et. al. 2002)
Practical guidelines

Venous air:
Level in venous hardshell reservoir

250 ml as stated by the manufacturer are safe.
Lowering level from 1000 to 250 ml, increased bubblevol.
by 12,4 % after reservoir and 40,2% after arterial filter.

(Nielsen et.al.2008)

Reduce vacuum to a minimum.

Reduce surgical source, make noise!!!
Practical guidelines

Fluid addition
Drugs administration
Blood sampling

De-air syringes prior to drug administration

Limit purging to a minimum, discard syringes.
Etiology

Oxygenator bubble migration:

Negative pressure in the oxygenator causes bubbles to migrate across the microporous hollowfiber membrane.

(Jegger et. al. 2003)
Hypothesis:

- The use of vacuum assisted venous drainage with a roller pump in between the venous reservoir and the oxygenator cannot lead to damaging micro bubbles being transported across the membrane of the oxygenator to the arterial line during CPB, following a no-flow situation.

- There is no difference in bubble migration for the two commercial available oxygenators after a period of no-flow with vacuum.
Master thesis
- 2 oxygenators
- High vacuum vs. no vacuum
- 2 or 10 minutes no-flow
- Soft occlusion of rollerpump
Compactflo EVO
QuadrOx-I

0mmHg 2min 0mmHg 10min 60mmHg Vac 2min 60mmHg Vac 10min
Master thesis
Mean air volume nL no-flow for 10 min. with vacuum

![Graph showing air volume comparison between Compactflo EVO and QuadrOx-I](image)
It is possible to migrate air across the membrane

Increased air with increased time (2 min vs. 10 min)

Measured on volume alone: significant difference Comp vs. Quadr (p<0.001)

Measured on ratio: no significant difference Comp vs. Quadr (p=0.129)

2 of 28 measurements higher than venous air, but for a shorter period of time and pre-arterial filter.
Summary

Avoid GME!!!