FUTURE MEETINGS & COURSES

2010
3 March 2010  Cases and Controversies in Obstetric Anaesthesia, London
19 - 21 May 2010  Obstetric Anaesthesia 2010 (Reception 19 May, Annual Meeting 20 & 21 May), The Sage, NewcastleGateshead
13 October 2010  Refresher Day on Obstetric Anaesthesia and Analgesia, London
8 - 10 November 2010  Three-day Course on Obstetric Anaesthesia and Analgesia, London
10 December 2010  Joint meeting with Royal Society of Medicine (Section of Anaesthesia and Section of Obstetrics and Gynaecology), London

2011
25 - 27 May 2011  Obstetric Anaesthesia 2011 (Reception 25 May, Annual Meeting 26 & 27 May), Edinburgh

2012
23 - 25 May 2012  Obstetric Anaesthesia 2012 (Reception 23 May, Annual Meeting 24 & 25 May), Liverpool

2013
22 - 24 May 2013  Obstetric Anaesthesia 2013 (Reception 22 May, Annual Meeting 23 & 24 May), Bournemouth
Dr Maggie Blott  
*Consultant Obstetrician*  
University College London Hospitals

**Professor Rob Dyer**  
*Professor & Second Chair, Department of Anaesthesia*  
Groote Schuur Hospital, University of Cape Town, South Africa

Dr Chris Elton  
*Consultant Anaesthetist*  
University Hospitals of Leicester

Dr Roshan Fernando  
*Consultant Anaesthetist*  
University College London Hospitals

Dr Wiebke Gogarten  
*Consultant Anaesthetist & Head*  
Department of Anaesthesiology, Intensive Care and Pain Medicine, Harlaching Hospital, Municipal Hospitals of Munich, Germany

Dr David Hill  
*Consultant Anaesthetist*  
Ulster Hospital, Belfast

Dr Paul Howell  
*Consultant Anaesthetist*  
St Bartholomew's & Homerton Hospitals, London

**Professor Beverley Hunt**  
*Professor of Thrombosis & Haemostasis*  
Guy's & St Thomas' Hospitals, London

Dr Ian Laing  
*Consultant Neonatologist*  
Edinburgh Royal Infirmary

Dr David Levy  
*Consultant Anaesthetist*  
Queen's Medical Centre, Nottingham

Dr Gordon Lyons  
*Consultant Anaesthetist*  
St James' University Hospital, Leeds

Dr Lucy Mackillop  
*Locum Consultant Obstetric Physician,*  
John Radcliffe Hospital, Oxford

Dr Geraldine O'Sullivan  
*Consultant Anaesthetist*  
Guy's & St Thomas' Hospitals, London

**Dr Peter Pan**  
*Professor & Director of Anesthesia Clinical Research*  
Wake Forest University School of Medicine, Winston-Salem, USA

Dr Anil Patel  
*Consultant Anaesthetist*  
Royal National Throat, Nose & Ear Hospital, London

Dr Felicity Plaat  
*Consultant Anaesthetist*  
Queen Charlotte's & Hammersmith Hospitals, London

Dr Mansukh Popat  
*Consultant Anaesthetist*  
John Radcliffe Hospital, Oxford

**Professor Felicity Reynolds**  
*Emeritus Professor of Obstetric Anaesthesia*  
Consulting Editor, International Journal of Obstetric Anesthesia

**Professor Steve Robson**  
*Professor of Fetal Medicine*  
Royal Victoria Infirmary, Newcastle-upon-Tyne

Dr Ian Russell  
*Consultant Anaesthetist*  
Hull Royal Infirmary

Dr Robin Russell  
*Consultant Anaesthetist*  
John Radcliffe Hospital, Oxford

Dr Mark Scrutton  
*Consultant Anaesthetist*  
St Michael's Hospital, Bristol

**Professor Andrew Shennan**  
*Professor of Obstetrics*  
Guy's & St Thomas' Hospitals, London

Prof Dr Marc Van de Velde  
*Professor of Anesthesiology*  
University Hospitals Gasthuisberg, Leuven, Belgium

Dr Cynthia Wong  
*Associate Professor, Chief of Obstetrical Anesthesia*  
Northwestern University Feinberg School of Medicine, USA
MONDAY 9 NOVEMBER

08.30  REGISTRATION, COFFEE & EXHIBITION
09.45  Opening remarks  Dr Roshan Fernando

**Session 1: Topical Issues 1**  ·  Chairman: Dr Roshan Fernando
09.50  New epidural drugs & adjuvants  Dr Mark Scrutton  4
10.10  What's new in obstetric anaesthesia - a snapshot of the literature  Professor Cynthia Wong  11
10.50  Discussion
11.00  COFFEE & EXHIBITION

**Session 2: Topical Issues 2**  ·  Chairman: Dr Gordon Lyons
11.25  Oxytocic drugs during caesarean delivery - essential, but toxic  Professor Rob Dyer  15
11.55  Neurological injury associated with childbirth & regional anaesthesia  Professor Cynthia Wong  19
12.20  Remifentanil for labour analgesia  Dr David Hill  24
12.50  Discussion
13.00  LUNCH & EXHIBITION

**Session 3: Labour Analgesia**  ·  Chairman: Dr David Hill
14.00  CSE or epidural for labour analgesia?  Professor Peter Pan  29
14.25  Maintaining labour epidurals - an update  Professor Cynthia Wong  35
14.50  Optimizing your labour epidural from insertion to delivery / clinical pearls  Professor Peter Pan  41
15.15  Discussion
15.30  TEA

**Session 4: New Developments**  ·  Chairman: Dr Roshan Fernando
16.00  Tracheal intubation - what's new?  Dr Anil Patel  47
16.30  Acute to chronic pain after delivery: from predictors to spinal oxytocin  Professor Peter Pan  48
17.00  Discussion
17.15  DRINKS (close 18.15)

TUESDAY 10 NOVEMBER

08.15  REGISTRATION, COFFEE & EXHIBITION

**Session 5: Blood Issues**  ·  Chairman: Dr David Levy
09.00  Obstetric Haemorrhage - rFVIIa, interventional radiology, cell salvage and beyond  Dr Felicity Plaat  55
09.25  Thromboprophylaxis, heparins and regional techniques  Dr Beverley Hunt  58
09.50  Use of the coagulation laboratory and point of patient testing in thrombosis & bleeding  Dr Wiebke Gogarten  59
10.15  Discussion
10.30  COFFEE & EXHIBITION

**Session 6: Caesarean Section**  ·  Chairman: Professor Marc Van de Velde
11.00  General anaesthesia for caesarean section - what's new?  Dr David Levy  63
11.25 Managing the difficult airway in obstetrics  Dr Mansukh Popat 71
11.55 Cardiac output monitoring during caesarean section - what have we learnt Professor Rob Dyer 82
12.20 Discussion
12.30 LUNCH & EXHIBITION

Session 6 (cont): Caesarean Section · Chairman: Dr Felicity Plaat
13.30 Spinal anaesthesia for caesarean section - dose, position & baricity Professor Marc Van de Velde 85
13.55 The obese parturient Dr Wiebke Gogarten 89
14.15 Managing pain & distress during caesarean section under regional anaesthesia Dr Geraldine O'Sullivan 93
14.35 Discussion
14.45 TEA & EXHIBITION

Session 7: The Baby 1 · Chairman: Dr Geraldine O'Sullivan
15.10 The decision to deliver - the obstetrician's viewpoint Professor Andrew Shennan 95
15.40 Emergency caesarean section following the decision to deliver - the anaesthetist's viewpoint Dr Chris Elton 96
16.00 Intrapartum fetal assessment and resuscitation - what the anaesthetist needs to know Professor Steve Robson 101
16.30 Discussion

WEDNESDAY 11 NOVEMBER

08.15 REGISTRATION, COFFEE & EXHIBITION

Session 8: Challenges for the Obstetric Anaesthetist · Chairman: Dr Chris Elton
09.00 Major trauma in pregnancy Dr Paul Howell 106
09.20 Pulmonary oedema in the third trimester - a perspective from South Africa Professor Rob Dyer 112
09.40 Prevention and treatment of postdural puncture headache Professor Cynthia Wong 115
10.00 Discussion
10.15 COFFEE & EXHIBITION

Session 9: The Baby 2 · Chairman: Dr Robin Russell
10.45 Current issues in neonatal resuscitation Dr Ian Laing 125
11.10 Effect of analgesia and anaesthesia on the baby Professor Felicity Reynolds 126
11.40 Discussion
11.55 LUNCH & EXHIBITION

Session 10: Pre-eclampsia Revisited · Chairman: Dr Roshan Fernando
12.45 Obstetric issues Mr Maggie Blott 133
13.10 Medical management of pre-eclampsia Dr Lucy Mackillop 138
13.35 Anaesthetic issues Dr Robin Russell 144
14.00 Discussion

The OAA Gold Medal Lecture · Chairman: Dr Paul Howell
14.10 Lateral thinking Dr Ian Russell
14.35 Discussion & Closing remarks Dr Roshan Fernando
14.45 TEA
15.15 Close
# Session 1: Topical issues 1

## New epidural drugs and adjuvants

**Dr Mark Scrutton**  
*Consultant Anaesthetist, St Michael's Hospital, Bristol*

## New LAs: Key issues

| 1. Sensory/motor differential  
2. Toxicity |

## New LAs

| 1. Sensory/motor differential |

Ropivacaine is a ‘weaker’ drug than bupivacaine  
Levobupivacaine is about the same as bupivacaine

## Ropivacaine

**Efficacy & duration of action**

| 0.1% Rop 20ml v 0.0625% Bup 20ml (+ sufentanil) |

Similar sensory & motor block  
Longer duration with Rop (119 v 89 min)*  

*Significant at p<0.0003  
Farpaglioni et al, IJOA 2000*

## Ropivacaine

**Motor block: human studies**

Some reduction in motor block with ropivacaine:  
Campbell, A&A 2000: 0.08% v 0.08% (fent)  
Fischer, Anesth 2000: 0.1% v 0.1% (suf)  
Meister, A&A 2000: 0.125% v 0.125% (fent)  
All PCEA studies
New LAs: Key issues

1. Sensory/motor differential

2. Toxicity

Bupivacaine toxicity

'Between the years 1973 and 1983, at least 24 maternal deaths were reported in the USA following accidental intravascular administration of bupivacaine....after intended epidural injection of bupivacaine in parturients scheduled for **cesarean section**

Marx 1997

UK experience

- 8 deaths assoc 0.25% bup in IVRA
- 1 obstetric death – caudal epidural

TOXICITY

...the evidence

Ropivacaine toxicity

animal studies

**Rats:**
- ↓ arrhythmias with ropivacaine

**Dogs:**
- CNS toxicity ratio (R:B): 1:1
- Deaths @ 2x CNS toxic dose (R:B): 1:5
- Bupivacaine dogs hard to resuscitate

**Pregnant sheep:**
- CNS toxic dose (R:B): 1.5:1
- CVS toxic dose (R:B): 1.5:1

Ropivacaine toxicity

human studies

- Infusion 10mg/m up to total 150mg
- Stopped when symptoms occurred
- **124mg ropivacaine v 99mg bupivacaine**


- Infusion 10mg/m
- Stopped when symptoms occurred
- **115mg ropivacaine v 103mg bupivacaine**

Knudsen et al, BJA 1997

BUT:

What about relative potencies?

The MLAC studies...........

**MLAC**

- Ropivacaine: 0.111%  *Polley, 1999*
- Bupivacaine: 0.067%  *Polley, 1999*
Predicted therapeutic index

<table>
<thead>
<tr>
<th></th>
<th>Rop</th>
<th>Bup</th>
<th>Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>ED$_{50}$ (mg)</strong></td>
<td>22.2</td>
<td>13.4</td>
<td>0.6</td>
</tr>
<tr>
<td><strong>TI$_{Scott}$ (Toxic dose) (mg)</strong></td>
<td>5.5</td>
<td>7.4</td>
<td>0.75</td>
</tr>
<tr>
<td><strong>TI$_{Knudsen}$ (Toxic dose) (mg)</strong></td>
<td>5.2</td>
<td>7.7</td>
<td>0.67</td>
</tr>
</tbody>
</table>

Therapeutic index (TI) = Toxic dose$_{50}$/Effective dose$_{50}$

_Columb & Polley, SOAP 1999_

Is MLAC relevant?

Only relates to FIRST dose for labour analgesia: 13-25mg

When does toxicity occur?

Big, intravenous doses

eg LSCS: 20ml 0.5% bupivacaine or 0.5% ropivacaine

ie: **100mg**

Ropivacaine toxicity

Caesarean section:

0.5% Ropivacaine 20ml is as effective as 0.5% Bupivacaine 20ml

Datta 1995
Griffin & Reynolds 1995
Alahuhta 1995
Crosby 1998

......but safer

Levobupivacaine toxicity

Isolated hearts: pig, rabbit, guinea pig

Intact animals: rat, sheep, human

...levobupivacaine safer than bupivacaine

Toxicity: human studies


Infusion 10mg/m up to total 150mg
Stopped when symptoms occurred

**56.1mg levobupivacaine v 49.6mg bupivacaine**

Ropivacaine toxicity human studies

Infusion 10mg/m up to total 150mg
Stopped when symptoms occurred

**124mg ropivacaine v 99mg bupivacaine**

Infusion 10mg/m
Stopped when symptoms occurred

**115mg ropivacaine v 103mg bupivacaine**
Knudsen et al, BJA 1997
**Toxicity: human studies**

Infusion 10mg/m up to total 150mg
Stopped when symptoms occurred

56.1mg *levobupivacaine* v 49.6mg *bupivacaine*

CVS changes occurred at lower plasma concentrations of bupivacaine

---

**MLAC**

<table>
<thead>
<tr>
<th>Levobupivacaine:</th>
<th>0.083%</th>
<th>Lyons et al</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bupivacaine:</td>
<td>0.081%</td>
<td>Lyons et al</td>
</tr>
</tbody>
</table>

---

**Are they worth it?**

1. No: only one maternal death in many years of UK use...

---

**Intravenous misplacement**

Accidental intravenous misplacement of the epidural catheter can occur in up to 15% of pregnant patients

- Negative aspiration tests may be unreliable
- Test doses are inconclusive
- Catheters may ‘migrate’ at any time

1. No: only one maternal death in years of UK use
2. Yes: targetted use

---

“Now don’t you fret Mrs. Firkin, my eyes are glued to your ECG”
Levobupivacaine (or Ropivacaine) are better options when……… large doses are needed, for example: epidural top-up for emergency LSCS when accidental i.v. injection is likely or may not be noticed

**Lipid and LA toxicity**

Pretreatment or resuscitation with a lipid infusion shifts the dose-response to bupivacaine-induced asystole in rats


Lipid emulsion infusion rescues dogs from bupivacaine-induced cardiac toxicity

Weinberg et al. Regional Anesthesia and Pain Medicine, 2003; 28(3): 198-202

Successful resuscitation of a patient with ropivacaine induced asystole after axillary plexus block using lipid infusion

RJ Litz et al. Anaesthesia 2006;61:800-1

Successful use of a 20% lipid emulsion to resuscitate a patient after presumed bupivacaine related cardiac arrest

M Rosenblatt et al. Anesthesiology 2006;105:217-8

• 1ml/kg over 1 minute with chest compressions
• Repeat every 3-5 minutes up to 3ml/kg
• Continuous infusion at 0.25ml/kg/min, until haemodynamic recovery
• Total dose above 8ml/kg is unlikely to be required in responders and of no benefit in non-responders

Weinberg. Regional Anesthesia and Pain Medicine, 2004; 29(1): 74

**Lipid regimen**

500ml 20% Intralipid £15
Shelf life of 2 years
Possibility of recycling it before it expires

**Lipid: practicalities**

Fentanyl: labour analgesia

Dose: Load 50mcg, Infuse 2mcg/ml
Pros: ↓ MLAC of local anaesthetics  
↓ motor block
Cons: ↑ side-effects for neonate  
↓ gastric emptying  
↑ Itching, nausea, vomiting

**2-chloroprocaine**

Dose: LSCS: 20ml 3% solution
Pros: Fast onset
Cons: not available in UK

Gaiser, IJOA 1994;3:208-10  
Taniguchi, Anesthesiology 2004;100:85-91

**Topics**

| 1. Local anaesthetics | Ropivacaine  
Levobupivacaine  
2-Chloroprocaine  |
|-----------------------|----------------|
| 2. Epidural adjuncts: | Opioids  
Adrenaline  
Bicarbonate  
Clonidine  
Neostigmine |

**Fentanyl: labour analgesia**

Dose: Load 50mcg, Infuse 2mcg/ml
Pros: ↓ MLAC of local anaesthetics  
↓ motor block
Cons: ↑ side-effects for neonate  
↓ gastric emptying  
↑ Itching, nausea, vomiting
### Adrenaline: labour analgesia

<table>
<thead>
<tr>
<th>Dose:</th>
<th>1:200,000-300,000</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pros:</td>
<td>↓ MLAC of local anaesthetics</td>
</tr>
<tr>
<td></td>
<td>Polley, Anesthesiol 2002:1123-8</td>
</tr>
<tr>
<td></td>
<td>Shue, Anesthesiol 2006;104(Sup):A-46</td>
</tr>
<tr>
<td></td>
<td>? Value as test dose</td>
</tr>
<tr>
<td>Cons:</td>
<td>? Motor block</td>
</tr>
<tr>
<td></td>
<td>Soetens, Anesth Analg 2006;103:182-6</td>
</tr>
</tbody>
</table>

### Clonidine: labour analgesia

<table>
<thead>
<tr>
<th>Dose:</th>
<th>50-75mcg (25-150mcg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pros:</td>
<td>↓ MLAC of local anaesthetics</td>
</tr>
<tr>
<td></td>
<td>↓ motor block</td>
</tr>
<tr>
<td></td>
<td>↓ shivering</td>
</tr>
<tr>
<td>Cons:</td>
<td>sedation (&gt;75mcg)</td>
</tr>
<tr>
<td></td>
<td>hypotension (&gt;75mcg)</td>
</tr>
</tbody>
</table>

### Neostigmine: labour analgesia

<table>
<thead>
<tr>
<th>Dose:</th>
<th>500mcg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pros:</td>
<td>↑ quality of block</td>
</tr>
<tr>
<td></td>
<td>Roelants, Anesthesiol 2004;101:439-44</td>
</tr>
<tr>
<td>Cons:</td>
<td>sedation</td>
</tr>
<tr>
<td></td>
<td>nausea and vomiting (spinal)</td>
</tr>
</tbody>
</table>

### Fentanyl: Epid LSCS

<table>
<thead>
<tr>
<th>Dose:</th>
<th>50-100mcg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pros:</td>
<td>↑ quality of the block</td>
</tr>
<tr>
<td></td>
<td>King, Anesthesia 1990;45:285-288</td>
</tr>
<tr>
<td></td>
<td>? speed of onset</td>
</tr>
<tr>
<td></td>
<td>Goring-Morris, IJOA 2006;15:109-14</td>
</tr>
<tr>
<td></td>
<td>Malhotra, IJOA 2006;15(Sup):S6</td>
</tr>
<tr>
<td>Cons:</td>
<td>↑ side-effects for neonate</td>
</tr>
<tr>
<td></td>
<td>↑ itching, nausea, vomiting</td>
</tr>
</tbody>
</table>

### Adrenaline: Epid LSCS

<table>
<thead>
<tr>
<th>Dose:</th>
<th>1:200,000 (100mcg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pros:</td>
<td>↑ quality of the block</td>
</tr>
<tr>
<td></td>
<td>Leaitshley, Anesthesia 1988;43:100-3</td>
</tr>
<tr>
<td></td>
<td>? speed of onset</td>
</tr>
<tr>
<td></td>
<td>? value as a test dose</td>
</tr>
<tr>
<td>Cons:</td>
<td>Hypertension</td>
</tr>
</tbody>
</table>

### Bicarbonate: Epid LSCS

<table>
<thead>
<tr>
<th>Dose:</th>
<th>1ml 8.4%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pros:</td>
<td>? speed of onset</td>
</tr>
<tr>
<td>Cons:</td>
<td>Precipitation with bupivacaine</td>
</tr>
</tbody>
</table>

### Bicarbonate: Speed of onset

- Lam, Anaesthesia 2001;56:790-4
- Gaggero, Can J Anaesth 1995;42:1080-4
- Fernando, Br J Anaesth 1991;67:699-703
- Berhamou, Reg Anesth 1989;14:240-3
- Allam, Anaesthesia 2008;63:243-9

### Category 1 LSCS

- Multiple drug combinations:
  1. Take time
  2. Introduce errors
- Lucas, Br J Anaesth 2000;84:494-6
- Goring-Morris, IJOA 2006;15:109-14

⇒⇒ ⇒⇒ Keep it simple, be prepared
### Future developments

Extended release epidural morphine (EREM):
- Single dose, prolonged effect *(Depodur)*

Extended release local anaesthetics:
- Phase II clinical trials

### Summary

**Epidural analgesia in labour:**
- Bupivacaine is cheap, safe and effective
- Opioids, adrenaline, clonidine will ↓MLAC and therefore may reduce motor block if LA dose is appropriately reduced
- Technique (infus/top-ups/PCEA) may be more important

**Epidural anaesthesia for LSCS:**
- Levobupivacaine or lidocaine
- Consider opioid, adrenaline and bicarbonate
- Preparation & technique
Session 1: Topical Issues 1
What’s new in obstetric anaesthesia - a snapshot of the literature

Professor Cynthia Wong
Associate Professor, Chief of Obstetrical Anesthesia,
Northwestern University Feinberg School of Medicine, USA

Antepartum


   Double-blind, randomized controlled trial of maintenance therapy for preterm labor.
   Groups: Nifedipine vs. placebo (N = 71).
   Outcome: Attainment of 37 weeks' gestation.
   Results: No difference between groups.
   Conclusions: no evidence that maintenance tocolysis with any agent prolongs pregnancy.


   Multi-center, double-blind, randomized controlled trial of magnesium therapy for women in preterm labor.
   Groups: Magnesium vs. placebo (N = 2241)
   Outcome: Primary: Composite of stillbirth or infant death by 1 year; secondary: moderate or severe cerebral palsy at or beyond 2 years of age.
   Results: Primary outcome, no difference between groups. Secondary outcome, less moderate or severe CP in magnesium group (1.9% vs. 3.5%, RR 0.55; 95% CI 0.32 to 0.95).
   Conclusions: Fetal exposure to magnesium may reduce risk of cerebral palsy in survivors.

Intrapartum Obstetric Complications


   Multi-center, 4-year observational study of vaginal birth after cesarean
   Groups: 0, 1, 2, 3, or ≥ 4 prior VBACs
   Outcomes: Successful VBACs, frequency of uterine rupture, neonatal morbidity
   Results: Increasing VBAC success in with increasing number of prior VBACs, rate of uterine rupture decreased after first successful VBAC, no difference in neonatal morbidity or mortality.
   Comments: Women with prior successful VBACs at low risk for maternal or neonatal complications.


   Case-controlled, 1-year study using the United Kingdom Obstetric Surveillance System of peripartum hysterectomy
   Groups: women who has peripartum hysterectomy (N = 318) and matched controls (N = 614)
   Outcomes: incidence, mortality, risk factors of hysterectomy
   Results: Incidence 4.1/10,000 births; maternal mortality 0.6%. Risk factors: previous cesarean delivery, age > 35 years, parity ≥ 3, previous manual removal of placenta, previous myomectomy, twin pregnancy, cesarean delivery.
   Conclusions: Peripartum hysterectomy strongly associated with previous cesarean delivery, age > 35 y, and parity ≥ 3.

Neuraxial Labor Analgesia


   Prospective observational study of second year anesthesia residents (N = 35) videotaped initiating epidural anesthesia and evaluated by blinded investigators.
   Outcomes: Scores of manual skills checklists, global rating scale, and an aseptic technique checklist.
   Results: Strong positive correlation between clinical experience and better scores on the manual skills checklist and global rating score, but nonsignificant or weak correlation with experience and aseptic technique score.
   Conclusions: There may be major gaps in how we teach trainees about aseptic technique.

Multi-center randomized controlled trial of spinal analgesia (administered via spinal microcatheters vs. epidural labor analgesia.
Groups: Intrathecal sufentanil analgesia (N = 329) with 28-g catheter vs. epidural bupivacaine analgesia (N = 100).
Outcome: Primary: incidence of neurologic complications at 24-, 48-h, 7-10 and 30-days.
Results: No patient had a permanent neurologic change. Intrathecal analgesia resulted in better early analgesia, more pruritus, less motor block and higher patient satisfaction, but more technical failures. There was no difference in the postdural puncture headache rate (9% vs. 4%).
Conclusions: The study was underpowered for the PDPH outcome.


Randomized controlled trial (n=77) of laboring women who received neuraxial labor analgesia.
Groups: Epidural vs. combined-spinal epidural analgesia.
Primary outcome: uterine tone and FHR abnormalities within 15 min of initiation of analgesia.
Results: Greater incidence of uterine hypertonus in CSE group (41% vs. 17%; P = 0.02) and FHR abnormalities (32% vs. 0.06%; P < 0.01).
Comments: Only evaluated FHR tracing for 15 min, assessed blood pressure every 5 min after analgesia.


Randomized controlled trial of 12,793 nulliparous Chinese women in spontaneous labor who requested labor analgesia greater than 1 cm and less than 4 cm cervical dilation.
Groups: Early vs. late (≥ 4 cm cervical dilation) epidural analgesia
Primary outcome: rate of cesarean delivery.
Results: No difference in cesarean delivery rate between groups (Early 23.2%, Late 22.8%; P = 0.51).


Prospective double-blind observational study of ED50 of intrathecal fentanyl for labor analgesia in women with single nucleotide polymorphism at position 304 for the mu-opioid receptor gene (OPRM1).
Groups: Nulliparous women (N = 224), homozygous 304AA vs. heterozygous or homozygous for the mutant 304A/G or GG.
Results: Using both a up-down sequential allocation method and random-dose allocation method, the ED50 was lower in women with the mutant a mutant allele.
Conclusions: Polymorphism at the mu-opioid receptor may affect response to opioids administered during labor.


Prospective observational study of post-cesarean morphine consumption in women with single nucleotide polymorphism at position 304 for the mu-opioid receptor gene (OPRM) who received spinal anesthesia with intrathecal morphine.
Groups: Nulliparous women (N = 588), OPRM1 homozygous 304AA vs. heterozygous 304A/G and homozygous 304GG.
Outcome: Self-administered intravenous morphine for 24 h after cesarean delivery.
Results: Morphine consumption and pain scores were lower in the AA (5.9 mg) compared to AG (8.0 mg) and GG 9.4 mg groups (P = 0.001).
Conclusions: The study results agree with other studies of postoperative pain: patients with OPRM1 304GG require more analgesia. The results are difficulty to reconcile with those of Landau, et al.

Drugs and Drug Toxicity


Randomized controlled trial in a porcine model (N = 10) of vasopressin/epinephrine vs. lipid emulsion for
success of resuscitation after IV bupivacaine cardiac arrest.
Groups: vasopressin/epinephrine vs. lipid emulsion
Outcome: Rate of survival.
Results: 5/5 vasopressin/epinephrine pigs survived and 0/0 lipid emulsion pigs survived.
Conclusions: Vasopressin/epinephrine resulted in higher coronary perfusion pressures and survival rates during CPR initiation for bupivacaine cardiac arrest.


Randomized trial comparing oxytocin to methylergometrine during elective cesarean delivery for signs of myocardial ischemia.
Groups: Term gestation: oxytocin (10 U IV bolus) vs. methylergometrine (0.2 mg) (N = 40) vs. non-pregnant control (oxytocin, N = 10).
Outcome: EKG changes (ST segment depression).
Results: Oxytocin produced greater ST depression and decreases in MAP than methylergometrine.
Conclusions: Oxytocin results in significant decrease in MAP and signs and symptoms (chest pain) of myocardial ischemia.


Randomized double-blind trial comparing placental transfer of ephedrine and phenylephrine used for the treatment of spinal anesthesia-induced hypotension for cesarean delivery.
Groups: Phenylephrine vs. ephedrine (N = 104)
Outcome: Placental transfer of phenylephrine and ephedrine, markers of fetal metabolism
Results: Placental transfer of ephedrine is greater than phenylephrine, as were umbilical artery concentrations of lactate, glucose, epinephrine, and norepinephrine.
Conclusions: Results support hypothesis that lower umbilical artery pH associated with ephedrine due to direct fetal metabolic effect.

General Anesthesia for Cesarean Delivery


Two observational studies (N = 61 and N = 21) assessing airway changes (modified Mallampati classification and acoustic reflectometry) in laboring women.
Groups: Women in labor.
Outcome: Modified Mallampati classification and upper airway volume
Results: Airway classification increased by one grade in 33% and two grades in 5%. There was a decrease in oral volume and pharyngeal airway as labor progressed.
Conclusions: Airway examinations worsen during labor, and presumably, airway management becomes more difficult. Airway examinations performed at the start of labor may not reflect actual airway conditions after hours of labor.


Multi-center, 2-year prospective observational study in 13 Australian and New Zealand hospitals in women receiving general anaesthesia for cesarean delivery.
Groups: Data from 1095 women (classes 1 to 4).
Outcome: Incidence of difficult and failed intubation, and aspiration.
Results: 3.3% considered difficult intubation, 4 failed intubations (0.4%; 95% CI 0.01-0.9%), 8 cases of aspiration (0.7%), no cases of serious airway morbidity.
Conclusions: Incidence of failed intubation consistent with other studies.


Multi-center, 2-year prospective observational study in 13 Australian and New Zealand hospitals in women receiving general anaesthesia for cesarean delivery.
Groups: Data from 1095 women (classes 1 to 4).
Outcome: Incidence of intraoperative awareness.
Results: 2 cases consistent with awareness (0.26%; 95% CI 0.03-0.9%); in 32% of cases a depth-of-anesthesia monitor was used.
Conclusions: Awareness remains a significant complication of general anesthesia for cesarean delivery.

comparison between women with and without prior labor. Anesth Analg 2008; 106: 1827-32

Observational study of BIS values in women (N = 40) undergoing cesarean delivery with sevoflurane/nitrous oxide anesthesia.

Groups: Laboring patients who required cesarean delivery, and non-laboring scheduled cesarean delivery patients.

Outcome: BIS values.

Results: BIS values between induction and delivery were lower in the labor group than non-labor group even though end-tidal sevoflurane concentrations were comparable.

Conclusions: Further study is required to elucidate the mechanism of higher BIS scores in non-laboring women. Does this mean they are at higher risk for awareness?


Secondary analysis of prospective, population-based, cohort study in 9 regions of France in 1977 in infants born between 27 and 32 weeks’ gestation, influence of maternal cesarean anesthesia on neonatal mortality.

Group: Etude épidémiologique sur les Petits Ages Gestationnels cohort, general vs. epidural vs. spinal anesthesia.

Outcome: Neonatal mortality.

Results: Neonatal mortality was 10.1% for general anesthesia, 12.2% for spinal anesthesia, and 7.7% for epidural anesthesia. There was a higher risk of neonatal death with spinal than general anesthesia (adjusted odds ratio 1.7; 95% CI 1.1-2.6).

Conclusions: Multivariate analysis does not prove causation, but it could exist. Further study is required.

Post-Cesarean Delivery Analgesia


Randomized controlled trial of transversus abdominis plane (TAP) block with ropivacaine after cesarean delivery (N = 50).

Groups: TAP block vs. saline.

Outcome: 48 h morphine consumption

Results: Mean 48-h morphine requirements were lower in the TAP group (18 vs. ± 14 mg vs. 66 ± 26 mg; P < 0.001).

Conclusions: The TAP block is an excellent component of multimodal postoperative analgesia that can be performed blindly or with ultrasound guidance.
Uterotonic drugs are essential pharmacological interventions during caesarean section, in order to diminish the risk of postpartum haemorrhage. They therefore contribute significantly to maternal safety. At the same time, these agents have a narrow therapeutic range in terms of maternal morbidity. The exact dose, route and rate of administration are thus important, as well as a detailed knowledge of their pharmacology.

Central to the mechanism of the contraction of uterine smooth muscle during labour, which is enhanced by the action of oxytocin, is the enzyme myosin light chain kinase (MLCK). Intracellular calcium, the levels of which are controlled by voltage and receptor operated channels and by release from the sarcoplasmic reticulum, is bound to calmodulin and stimulates conversion of MLCK-P to MLCK, which in turn phosphorylates myosin and initiates smooth muscle contraction.

The nonapeptide oxytocin was discovered by Sir Henry Dale and was the first polypeptide hormone synthesised, by Du Vigneaud, in 1953. The peptide binds to a G-Protein on the surface of the uterine myocyte, resulting in the generation of diacylglycerol (DAG) and inositol tri-phosphate (IP3) via the action of phospholipase – C on phosphatidyl inositol bisphosphate. DAG stimulates prostaglandin synthesis, and IP3 stimulates the release of calcium from the sarcoplasmic reticulum. Oxytocin also activates COX-2 via a further G-Protein interaction, and in so doing stimulates prostaglandin synthesis. The concentration of myometrial receptors as well as myometrial gap junctions increase as gestation advances, thus increasing sensitivity to oxytocin.

Oxytocin has numerous physiological effects. Most importantly, it causes contraction, followed by relaxation of the uterus, and, at pharmacological doses, can cause an increased frequency of contractions and incomplete relaxation of the uterine musculature. It also has a role in sexual and maternal behaviour, and in memory and the regulation of food and drink intake. This agent remains the first line uterotonic during caesarean section.

The cardiovascular effects are complex. Hypotension is predominantly caused by transient relaxation of vascular smooth muscle cells, probably via calcium dependent stimulation of the nitric oxide pathway. It also causes the release of atrial and brain natriuretic peptide, and oxytocin or its preservative chlorbutanol may have negative inotropic effects on atrial myocytes. Oxytocin causes selective vasoconstriction of coronary, renal, splanchnic and skeletal muscle arteries, as well as umbilical vessels. Several papers have suggested the possibility of positive inotropic effects. Due to structural similarities with vasopressin, overdose of oxytocin may cause water retention, hyponatraemia, seizures and coma.

Several papers have described the haemodynamic effects in the non-pregnant population and in the pregnant population during caesarean section. Early studies used intermittent indicator dilution or thermodilution technology, and more recently, beat by beat pulse wave form monitors, as well as monitors of changes in transthoracic bioimpedance, have elucidated a clinical picture of peripheral vasodilatation, hypotension, and increased cardiac output mediated by an increase in heart rate and stroke volume. Pulmonary artery pressures are markedly increased after a bolus of 10 IU. One observational study has demonstrated similar effects in patients with severe preeclampsia. These effects would be poorly tolerated if ventricular function were abnormal, and in the presence of mitral or aortic stenosis, or hypovolaemia. One fatality was recorded in the Confidential Enquiry into Maternal Deaths of the United Kingdom in the triennium 2002-2004, when oxytocin was administered during the resuscitation of a hypovolaemic patient during spinal anaesthesia for caesarean section.

Co-administration of phenylephrine with oxytocin has been shown to obtund the peripheral vascular effects. Also, administration of 5 IU of oxytocin by slow infusion has been shown to produce less cardiovascular instability than a bolus of 5 IU. The efficacy of such infusions in terms of uterine contraction is difficult to assess other than by subjective means, but these probably suffice in most cases. The use of oxytocin in incremental doses of 0.1 IU in parturients with cardiac disease has recently
been shown to be associated with haemodynamic stability.\textsuperscript{12}

In view of the multiple side effects of oxytocin, including nausea, vomiting, flushing, pruritus, headache, and possible chest pain,\textsuperscript{13,14} it is desirable to administer the lowest possible effective dose in the most haemodynamically stable manner. The dose and rate of intravenous infusion of oxytocin after delivery during caesarean section remain controversial. There have been only two dose response studies, one in healthy uncomplicated pregnancies at low risk for uterine atony,\textsuperscript{15} and another in a case series of patients with labour arrest.\textsuperscript{16} Both studies employed the up-down sequential method of establishing the median effective dose. The ED90 for low risk patients was found to be 0.35IU, and in patients with labour arrest the value was 3.0IU.

In keeping with the mechanism of action, involving G-Protein receptor interactions, the phenomenon of receptor desensitisation may influence the effectiveness of the dose given by the anaesthetist at delivery. A recent publication in which a second dose of oxytocin was administered in the same patient, suggested that the cardiovascular response to the second dose was diminished,\textsuperscript{17} although at least one previous investigator did not demonstrate this effect. A diminished cardiovascular response would be in keeping with down-regulation. More definitive laboratory work has shown that loss of oxytocin receptors does occur during oxytocin-induced and oxytocin-augmented labour.\textsuperscript{18} Prior exposure of rat myometrium to oxytocin suggests a concentration-, but not time-dependent, reduced efficacy of oxytocin.\textsuperscript{19} On balance, then, it appears that repeated doses of oxytocin may become progressively less effective, and that pharmacological alternatives are still required.

The newly developed synthetic analogue of oxytocin, carbetocin, has a half-life 4-10 times the duration of oxytocin. A tetanic uterine contraction is produced 2 minutes after an IV or IM injection of 100μg; this persists for 60 and 120 minutes after IV and IM injection respectively. Randomised trials suggest a diminished subsequent requirement for uterotonic when compared with oxytocin administered at caesarean section, and a lower risk for >500mL bloodloss following caesarean section.\textsuperscript{20} The side effect profile is similar to that of oxytocin. Preeclampsia remains a contraindication to its use, for reasons which are unclear.\textsuperscript{21} The issue of receptor desensitisation is as yet unstudied.

Currently, the ergot alkaloid ergometrine is the second line agent if uterine atony persists after oxytocin administration during caesarean delivery. Ergot alkaloids are all derivatives of 6-methyl ergolergine. Ergometrine is a naturally occurring alkaloid, first isolated in 1932 by Dudley and Moir. Ergometrine maleate or methylergometrine may be administered IV or IM after delivery. It causes a rapid and sustained contraction of the pregnant and non-pregnant uterus. Little is known about the mechanism of action, which may be via a calcium channel. Ergometrine is also a partial agonist at alpha-adrenergic, SHT-1, and dopamine receptors\textsuperscript{22} There are no published dose response curves. The recommended IV dose is 0.1mg IV slowly, to be followed by a further 0.1mg if required. The half-life of ergometrine is 120 minutes. Although ergometrine and methylergometrine have the least vasoconstrictor effects of all the ergot alkaloids, there is a significant incidence of hypertension, and myocardial infarction has been reported.\textsuperscript{23,24} In addition, troublesome nausea and vomiting can make caesarean delivery under spinal anaesthesia unpleasant. The incidence of nausea has been reported as 29%, and vomiting 9% after a bolus of 5 IU of oxytocin,\textsuperscript{25} and 10% after 250μg 15-methyl prostaglandin F2α\textsuperscript{26} during elective caesarean delivery under spinal anaesthesia. A high incidence of 46% of nausea or vomiting has been reported after 0.5mg ergometrine IV.\textsuperscript{27}

Prostaglandins also increase intramyometrial calcium concentrations and enhance uterine contraction. Side effects include fever, diarrhoea, nausea and vomiting. The use of intramyometrial prostaglandin F2α for atomic postpartum haemorrhage was first described by Takagi in 1976.\textsuperscript{28} Subsequently, 15-methyl prostaglandin F2α was shown to have an extended half-life, fewer gastrointestinal and vasopressor side-effects, and good uterotonie activity.\textsuperscript{29,30} Since 15-methyl prostaglandin F2α may be associated with bronchospasm,\textsuperscript{31} ventilation perfusion mismatch and hypoxaemia,\textsuperscript{32} this agent is best used as a last resort therapy and not as prophylaxis.\textsuperscript{33} There is very limited experience with intravenous administration.\textsuperscript{34} Infusion at 100µg/minute during early pregnancy has been shown to cause systemic and pulmonary hypertension, in contrast with prostaglandin E2, which is associated with a marked decrease in systemic vascular resistance and hypotension.\textsuperscript{35}

Prostaglandin E1 is a safe, cheap and widely available oxytocic which may be a useful adjunct administered rectally during caesarean section in a dose of 800-1000µg in cases of uterine atony.\textsuperscript{36}
In summary, uterotonic drugs remain an important adjunct to the surgical management of haemorrhage during caesarean section. Current evidence is that oxytocin remains the uterotonic of first choice. There are few definitive studies upon which to base a protocol for recommendations for dosing of oxytocin or second-line uterotonic. A bolus dose of 2.5 IU IV in combination with phenylephrine, or a 3-5 minute infusion of 2.5-5 IU is a reasonable approach, followed by a continuous infusion at less than 20 milliunits per minute. Down-regulation of oxytocin receptors following prior exposure to oxytocin, requires further study. This phenomenon results in the requirement for uterotonics with a different mechanism of action, such as ergometrine and prostaglandin F2α. Currently, ergometrine 0.1mg IV slowly, followed by a repeat dose of 0.1mg, is a logical second line agent in the absence of contraindications such as preeclampsia. 15-methyl prostaglandin F2α, 250µg intramyometrially, repeated every 15 minutes to a maximum of 8 doses, is the last resort. Dose response curves for the latter two agents would provide useful guidelines for the use of drugs with a very narrow therapeutic index.

References


Session 2: Topical Issues 2

Neurological injury associated with childbirth & regional anaesthesia

Professor Cynthia Wong
Associate Professor, Chief of Obstetrical Anesthesia,
Northwestern University Feinberg School of Medicine, USA

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**Neurologic Injury Associated with Childbirth and Regional Anaesthesia**

*Cynthia A. Wong, M.D.*

November 9, 2009

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**Direct Complications of Neuraxial Anaesthesia**

<table>
<thead>
<tr>
<th>Study</th>
<th>N</th>
<th>Technique</th>
<th>Incidence/10,000 Patients</th>
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<td>EP, SAB</td>
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Prospective studies

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**Multi-center French Study**

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<td>Central neurologic event</td>
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<tr>
<td>Meningitis</td>
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</table>

Events/10,000 obstetric patients

Auroy Y. Anesthesiology 2002

---

**Direct Complications of Neuraxial Anaesthesia**

- Direct trauma
- Injection into unintended places
- Injection of unintended substances
- Transient neurologic syndrome
- Spinal epidural hematoma
- Spinal infection
- Vascular catastrophes

---

**Ability to Determine Interspace**

<table>
<thead>
<tr>
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<th>Estimated Interspace</th>
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<td>S1-2</td>
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</table>

Broadbent CR. Anaesthesia 2000
Position of Conus Medullaris

Broadbent CR. Anaesthesia 2000

Damage to the Conus Medullaris

Reynolds F. Anaesthesia 2001

Preventing Direct Damage to the Spinal Cord/Nerve Roots

- Lumbar puncture < L3 vertebral body
- Halting needle advancement if patient reports pain
- Inject only if pain completely resolves

Direct Complications of Neuraxial Anesthesia

- Direct trauma
- Injection into unintended places
- Injection of unintended substances
- Transient neurologic syndrome
- Spinal epidural hematoma
- Spinal infection
- Vascular catastrophes

ASA Closed Claims Database

Lee LA. Anesthesiology 2004;101:143


Sources of Infection

- Abscess: skin flora
  - Staph aureus
- Meningitis: Streptococcus viridans species

Skin Preparation: PI vs. Duraprep

Positive cultures

<table>
<thead>
<tr>
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<th>DuraPrep N (%)</th>
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<td>9 (30)*</td>
</tr>
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<td>Catheter removal</td>
<td>15 (50)</td>
<td>29 (97)*</td>
</tr>
<tr>
<td>Catheter tip</td>
<td>2 (7)</td>
<td>6 (20)*</td>
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</table>

*P < 0.01

Birnbach DJ. Anesthesiology 2003

Skin Preparation: PI vs. Duraprep

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Birnbach DJ. Anesthesiology 2003

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<td>Catheter tip</td>
<td>2 (7)</td>
<td>6 (20)*</td>
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</table>

*P < 0.01

Birnbach DJ. Anesthesiology 2003
**Sources of Infection**
- Abscess: skin flora
- Meningitis: Streptococcus viridans

**Meningitis Reports**

<table>
<thead>
<tr>
<th>Clinical Scenario</th>
<th>Number</th>
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<tr>
<td>Spinal analgesia in labor</td>
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<tr>
<td>Spinal anesthesia for CS</td>
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<tr>
<td>Spinal anesthesia for retained placenta</td>
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<tr>
<td>CSE in labor</td>
<td>7</td>
</tr>
<tr>
<td>Epidural dural puncture in labor</td>
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<tr>
<td>“Uncomplicated” epidural in labor</td>
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</tr>
<tr>
<td>TOTAL</td>
<td>38</td>
</tr>
</tbody>
</table>

Reynolds F. Anesthesiol Clin 2008;26:23

Viridans streptococci: vagina and oral cavity

**Meningitis Case Clusters**
- 4 cases of viridans streptococci meningitis after spinal anesthesia in 15 months
- One anesthesiologist
  - Recurrent pharyngitis
  - No handwashing
  - Did not remove jewelry
  - Did not wear a face mask
  - Wore sterile gloves

Schneeberger PM. Infection 1996;24:29

**ASRA Consensus Conference 2004**

8. Alcohol-based/chlorhexidine antiseptic solutions significantly reduce the likelihood of catheter and site colonization and maximize the rapidity and potency of hemorrhelial activity when compared to other solutions. Therefore, alcohol-based/chlorhexidine solutions should be considered the antiseptic of choice before regional anesthetic techniques (Grade A).

Grades of Recommendations
- A Requires at least one prospective, randomized, controlled trial as part of a body of literature of overall good quality and consistency addressing the specific recommendation.
- B Requires at least one prospective, randomized, controlled trial and a good quality of safety and effectiveness of the specific recommendation.

Hebl JR. Reg Anesth Pain Med 2006;31:311-23

**AAGBI**

Maximal barrier precautions involve full hand washing, the wearing of sterile gloves and gown, a cap, mask and the use of a large sterile drape [42]. The skin entry site should be cleaned with an alcoholic chlorhexidine gluconate solution or alcoholic povidone-iodine solution [43]. The antiseptic should be allowed to dry before proceeding.

Certain invasive anesthetic procedures require this optimum aseptic technique:
- Insertion of central venous catheters.
- Spinal, epidural and caudal procedures.

Anaesthesia 2008;63:1027

**Direct Complications of Neuraxial Anesthesia: Lessons Learned**
- Aim low (< L3)
- Spinal lidocaine: OK in OB
- Skin prep with EtOH w/ PI or chlorhexidine
- Wear a mask and don’t be too chatty

**Direct complications of neuraxial anesthesia**
- Intrinsic obstetric palsies
Beatty 1838

Ann Kierman, aged 21 delivered of her first child, November 26th, 1836, after a labour of 7 hours; infant alive. Nothing remarkable occurred during labour, or afterwards, until she complained on the second day, that she could not move her right leg, and that it felt benumbed and dead….

...She continued to improve until the month of February, at which time she was walking about nearly well, and preparing to leave the hospital, when puerperal fever made its appearance in our wards.

She died of pericarditis 1 week later.

Intrinsic Obstetric Palsies

- Incidence

6157 Live births
6148 Interviewed
454 CS w/o labor ⇒ 1 Nerve injury (0.22%)
5594 Labored ⇒ 80 Nerve injuries ⇒ 72 examined by physiatrist
9 Pre-existing injuries
55 new injuries verified by physiatrist (0.92%)

Wong CA. Obstet Gynecol 2003

Postpartum Nerve Injury

<table>
<thead>
<tr>
<th>Study/year</th>
<th>N</th>
<th>Incidence/10,000 (%)</th>
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<tr>
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<td>Dar, 2002</td>
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<td>58 (0.58)</td>
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<td>Wong, 2003</td>
<td>6,157</td>
<td>92 (0.92)</td>
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Multivariate analysis

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<tr>
<td>Nulliparity</td>
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<tr>
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<td>1.11 – 4.84</td>
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<tr>
<td>Neuraxial analgesia</td>
<td>2.00</td>
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<td>0.057</td>
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</tbody>
</table>

Wong CA. Obstet Gynecol 2003

Intrinsic Obstetric Palsies and Neuraxial Analgesia

- Possible indirect relationship:
  - Neuroblockade
    - Prevents recognition of impending nerve injury
    - Prolongs 2nd stage of labor
Bibliography


7. Reynolds F. Damage to the conus medullaris following spinal anaesthesia. Anaesthesia 2001; 56: 238-47
12. Schneeberger PM, Janssen M, Voss A. Alpha-hemolytic streptococci: a major pathogen of iatrogenic meningitis following lumbar puncture. Case reports and a review of the literature. Infection 1996; 24: 29-33
Session 2: Topical Issues 2
Remifentanil for labour analgesia

Dr David Hill
Consultant Anaesthetist, Ulster Hospital, Belfast

Remifentanil for labour analgesia

50% don’t have an epidural
- Patient preference
- No “epidural service”
- Thrombocytopenia
- Anticoagulated
- “Back problems”
- “Neurological”
- “Sepsis”

50% don’t have a good alternative

Pethidine
- “more sedation than analgesia”
- Gastric stasis & hypoventilation
- Fetal effects after 40 mins
- Dose-delivery 2-3 hrs
- Modifies CTG & ECG
- Fetal acidosis
- Active metabolites for days

Effect site concentration after opioid bolus

Suitability of Remifentanil as a labour analgesic
- Theory
  - Placental transfer
  - Pharmacokinetics
- Practice
  - Efficacy
  - Maternal effects
  - Neonatal effects

Intravenous remifentanil:
placental transfer
- UV:MV ratio 0.88
- UA:UV ratio 0.29
- Clearance 93 ml/min/kg

“rapidly metabolised, redistributed or both”

Kan et al, Anesthesiology 1996, 88:1467-74
Remifentanil pharmacokinetics in neonates

- Infants under 2 months
- Pharmacokinetics similar to older children and adults

Davis, Ross Henson et al. Remifentanil pharmacokinetics in neonates. Anesthesiology 1997; 87: A1054

Pharmacokinetics

- Faster clearance in pregnancy
- Plasma levels 50% lower in pregnancy
- Effect site peak 1.3-1.6 min
- Analgesia peak 30 sec - 2.5 min
- Typical contraction 70 sec

can a single bolus = a single contraction?


Published data on efficacy

<table>
<thead>
<tr>
<th>Bolus Dose mcg/kg</th>
<th>Lockout time (min)</th>
<th>Median or reduction in pain scores (mm)</th>
<th>Conversion rate to regional analgesia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Remifentanil 0.2</td>
<td>2</td>
<td>15mm</td>
<td>Not reported</td>
</tr>
<tr>
<td>Remifentanil 0.4</td>
<td>2</td>
<td>5mm</td>
<td>Not reported</td>
</tr>
<tr>
<td>Remifentanil 0.6</td>
<td>2</td>
<td>15mm</td>
<td>Not reported</td>
</tr>
<tr>
<td>Remifentanil 0.8</td>
<td>2</td>
<td>42mm</td>
<td>Not reported</td>
</tr>
<tr>
<td>Thurlow 2002</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Volmanen 2002</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blair 2001</td>
<td></td>
<td></td>
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<tr>
<td>Blair 2005</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Evron 2005</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Volikas 2005</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Balki 2007</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Published data on efficacy: comparison with entonox

- Crossover study 15 women
- Entonox or 0.4mcg/kg bolus remifentanil
- Reduction in pain scores
  - 15mm remifentanil
  - 5mm entonox
- 14/15 preferred remifentanil

Volmanen et al 2005

Published data on efficacy: comparison with pethidine

- PCA pethidine
  - Analgesia similar
  - CTG benefit
  - Neonatal benefit
- IM pethidine
  - Analgesia benefit
  - More desaturation
- IVI pethidine
  - Analgesia benefit
  - CTG benefit
  - Desaturation benefit


Data on 603 labouring women using PCA remifentanil

- 85% satisfied or very satisfied
- 77% no pain or bearable pain
- 84% used Entonox
- 10% conversion to regional

Hodgkinson et al 2008

What do women want?

Systematic review of 137 reports of the views of 14,000 women in 9 countries

- Satisfaction
  - Expectations
  - Support
  - Quality of relationship with midwife
  - Involvement in decision making
- Dissatisfaction
  - Failure of timing & lack of availability of analgesia

Complete analgesia did not rate highly


Maternal adverse effects

Maternal adverse effects (603 labouring women with remifentanil)
Maternal desaturation: (603 labouring women with remifentanil)

- SpO2 <90% in 12%
  - Independent of sedation score
  - 0.66% (4 women) had PCA stopped
- Rx
  - Continuous portable pulse oximeter
  - Nasal oxygen 2l/min
  - Midwife

Hodgkinson et al 2008

Reasons for discontinuing (603 labouring women with remifentanil)

<table>
<thead>
<tr>
<th>Reason</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inadequate analgesia</td>
<td>3.98%</td>
</tr>
<tr>
<td>Sedation/pushing</td>
<td>2.65%</td>
</tr>
<tr>
<td>Equipment failure</td>
<td>0.83%</td>
</tr>
<tr>
<td>Desaturation</td>
<td>0.66%</td>
</tr>
<tr>
<td>Nausea</td>
<td>0.5%</td>
</tr>
<tr>
<td>Itch</td>
<td>0.17%</td>
</tr>
</tbody>
</table>

Hodgkinson et al 2008

Remifentanil PCA launched for routine use in August 2005

- Bolus 40 mcg
- Delivered over 10 sec
- Lockout 2 min

Remifentanil PCA: practical safeguards

- Prescribed by anaesthetist
- Drug made up by 2 midwives
- Dedicated pumps
- Pumps locked
- Dedicated labelled cannula

Remifentanil PCA: monitoring

1 to 1 Midwifery care
  - Continuous SpO2
  - Regular observations
  - CTG monitoring as normal
  - Anaesthetist present for 1st 4 doses

Remifentanil PCA: Midwife training

- Iv cannulation course
- Iv drug course
- Drug preparation "5-5-10"
- Analgesia management "5-5"
- Extended training folder
- 6 month mentoring

Remifentanil PCA: Governance

- Continuous audit
  - outcomes & user satisfaction
- Policies & guidelines review
- Risk assessment review
- Critical incident reporting
- Work force planning
- Staff feedback

Remifentanil PCA: change in practice

- 2nd stage
  - Pushing-sedation conflict
  - Pudendal nerve block "remi-pendal"

- More spinals
  - For trials
  - For caesarean
Impact of remifentanil PCA Northern Ireland Maternity System (NIMATS)

- Data retrieved from 5400 women
- Analgesia uptake
- Need for neonatal resuscitation
- Obstetric outcome

Analgesia uptake in 2yr period

Neonatal effects: APGAR scores from 5400 neonates

Neonatal resuscitation: data from 5400 births

Analgesia choice by parity in 5400 women

Mode of delivery by analgesia choice in 5400 women

Effect of analgesia or midwife influence?

NIMATS data on obstetric outcome:
- 2yr before remi (4942 women)
- 2 yr after remi (5874 women)
Obstetric outcomes 2 yr before (4942 deliveries) and 2 yr after (5874 deliveries)

Remifentanil PCA

- Epidural rate 33 vs 17%
- Overall Caesarean 23.8 vs 23.6%
- Elective caesarean 9.4% vs 9.5%
- Emergency caesarean 14.4 vs 14.2%
- Vacuum 9.8 vs 12.3%
- Forceps 6.2 vs 9.2%

Obstetric outcome within analgesia groups: Remi vs epidural

- Forceps 4.6 vs 21.4%
- Vacuum 12.4 vs 18.3%
- Emergency caesarean 2.4 vs 21.7%

Remifentanil PCA offers......

- Modest analgesia
- Few neonatal effects

But requires.......

- 1-1 midwifery
- Training
- Appropriate monitoring

In our unit.......

The introduction of Remifentanil PCA has

- Reduced the epidural rate
- Reduced anaesthetic workload
- Changed working practices
- Empowered our midwives

"midwive managed" analgesia:

Parturients using Remifentanil PCA should on average expect:

- 71% chance of a normal delivery
- 9.6% chance of caesarean delivery
- 2.4% emergency caesarean
- 6% neonates will require bag & mask

"midwive managed" analgesia

Parturients using Epidural analgesia should on average expect:

- 42% chance of a normal delivery
- 23% chance of caesarean delivery
- 21.1% emergency caesarean
- 11% neonates will require bag & mask
**Session 3: Labour Analgesia**

**CSE or epidural for labour analgesia?**

**Professor Peter Pan**

*Professor & Director of Anesthesia Clinical Research,*

Wake Forest University School of Medicine, Winston-Salem, USA

---

**CSE or Epidural for Labour Analgesia?**

"Old and New Thoughts on Combined Spinal Epidural (CSE): Can CSE be used for all Laboring Patients?"

Peter H. Pan, MSE, MD

Department of Anesthesiology

Wake Forest University School of Medicine

Winston-Salem, North Carolina

---

**Goals in Comparing CSE vs Epid**

- Efficacy
- Safety and Complications
- Maternal and Neonatal Effects
- Effects on Labor Progress and Delivery
- Efficacy of Subsequent Epidural Catheter
- Who should we use or not use CSE?

---

**Direct Comparison CSE vs Epid**

- Abouleish 1991
- Breen 1999
- Caldwell 1994
- Comet 2001
- Dunn 1998
- Gomez 2001
- Hepner 2000
- Kartawiadi 1996
- Medina 1994
- Parry 1998
- Patel 2003
- Price 1998
- Roux 1999
- Thomas 2005
- Cappiello 2008
- Grondin 2009

- Comparing:
  - Traditional Epidural
  - Low Dose Epid LA +/- opioid
  - Versus
  - CSE opioid +/- hupiv
  - CSE nil IT

---

**26 Outcomes Compared (CSE vs Epid)**

- **Efficacy:**
  - Time to effective analg
  - % pt effective @ 10 m
  - Need for rescue analg
  - % satisfy with analg
  - % Mobilise

- **Safety/Complications:**
  - PDPH
  - Dural Tap, EPB
  - Urinary Retent
  - Preripus
  - Nausea/Vomit
  - Hypotension
  - Resp Depress
  - HA
  - Sedation

- **Labor Progress:**
  - Augment: Post-Analg
  - Labor Augment
  - Normal Delivery
  - Instrument Delivery
  - Cesarean Delivery

- **Neonatal Effect:**
  - UC, UA pH, UV pH
  - Apgar c7, c8 @ 5 m
  - NICU admission

---

**Traditional Epid vs CSE**

- Only 3/26 outcomes showed differences**:

  **Need for Rescue Analgesia (0.31) [0.14, 0.70]**
Traditional Epid vs CSE – Need for Rescue

**Need for Rescue Analgesia**
- 0.31 [0.14, 0.70]

**Pruritus**
- 1.74 [1.40, 2.15]

**Urinary Retention**
- 0.87 [0.80, 0.95]

Traditional Epid vs CSE

Only 3/26 outcomes showed differences**: 
- **Need for Rescue Analgesia** (0.31) [0.14, 0.70]
- **Pruritus** (1.74) [1.40, 2.15]
- **Urinary Retention** (0.87) [0.80, 0.95]

---

Traditional Epid vs CSE – Pruritus

**Pruritus**
- 1.74 [1.40, 2.15]

Traditional Epid vs CSE

Only 3/26 outcomes showed differences**: 
- **Pruritus** (1.74) [1.40, 2.15]

---

Traditional Epid vs CSE – Urinary Retention

**Urinary Retention**
- 0.87 [0.80, 0.95]

Traditional Epid vs CSE

Only 3/26 outcomes showed differences**: 
- **Urinary Retention** (0.87) [0.80, 0.95]

---

Traditional Epid vs CSE – Onset Time

**Time to Effective Analgesia**
- -4.00min [-8.08, 0.08]

**Need for Rescue Analgesia**
- 0.96 [0.77, 1.20]

**Pruritus**
- 1.62 [1.34, 1.97]

**Umbilical Artery pH**
- -0.02 [-0.02, -0.02]

**Instrument Delivery**
- 0.82 [0.67, 1.00]

---

Low Dose Epid vs CSE

**Time to Effective Analgesia**
- -5.59 min [-6.59, -4.58]

**Analgesia Effective @10min**
- 1.96 [1.50, 2.57]

**Need for Rescue Analgesia**
- 0.96 [0.77, 1.20]

**Pruritus**
- 1.62 [1.34, 1.97]

**Umbilical Artery pH**
- -0.02 [-0.02, -0.02]

**Instrument Delivery**
- 0.82 [0.67, 1.00]

**Cesarean Delivery**
- 0.99 [0.82, 1.20]
Low Dose Epid vs CSE – Onset Time

Only 4/26 outcomes showed differences**: 

- Time to Effective Analgesia (-5.59 min) [-6.59, -4.58]
- Pruritus (1.62) [1.34, 1.97]
- Umbilical Artery pH (-0.02) [-0.02, -0.02]
- Need for Rescue Analgesia (0.96) [0.77, 1.20]
- Cesarean Delivery (0.99) [0.82, 1.20]

Low Dose Epid vs CSE – Effective @ 10min

- Time to Effective Analgesia (-5.59 min) [-6.59, -4.58]
- Analgesia Effective @10min (1.96) [1.50, 2.57]
- Pruritus (1.62) [1.34, 1.97]
- Umbilical Artery pH (-0.02) [-0.02, -0.02]
- Need for Rescue Analgesia (0.96) [0.77, 1.20]
- Cesarean Delivery (0.99) [0.82, 1.20]
CSE: A cause of fetal bradycardia?

**PROPOSED MECHANISM:**
- Labor → pain → ↑ epinephrine
- CSE → ↓ pain → ↓ epinephrine more than NE → ↓ β agonist (relaxant) effects on uterus → ↑ uterine tone

**Labor Progress and Process**

- Effects on C/S Rate
- Effects on Progress of Labor

**Meta-analysis of EA on OB outcomes**

1. Halpern (JAMA 1998)
   - 10 Randomized Clinical Trials, N=2369
   - Epid gp N=1183, IV/IM gp N=1186.
   - C/S Rate: No Difference between gps
     - 8.2% C/S (Epid gp)
     - 5.5% C/S (IV/IM gp)
   - 1st Stage Labor: Longer in Epidural gp
   - 2nd Stage Labor: Longer in Epidural gp
   - Instrument Delivery: Higher in Epidural gp
   - (Especially vs. high LA Conc Epid)

2. Leighton (AJOBG 2002)
   - 14 Randomized Clinical Trials Updated
   - Almost 4300 pts, Epid vs IV/EA group
   - 17.8% vs 20.7% (95% CI 1.04-3.1)

   - 4 RCTs, 4 observational studies.

All 3 series of meta-analysis concluded:
EA NOT affecting C/S rate.

**Effects of CSE on Labor Progress and C/S Rate**

  - 15% FHR changes with IT suf 10ug

  - IT fentanyl: 9 fetal brady, 5/9 with uterine tetany

- Pan et al. (SOAP 96,97) reported a shorter duration of 1st stage labor, and less instrument delivery with CSE fentanyl versus EA.

**Fetal Brady – CSE vs EA**

- **Cervical Dilation**
  - (Tou J. Anesthesiology 1990;72:5)
  - CSE sufentanil 10 ug + bup 2.5mg (n=60)
  - Epid 12ml 0.25% bupivacaine (n=60)
  - Nulliparous Spontaneous laboring parturients
  - Faster dilation (p<0.01) → 2.3 cm/hr CSE vs 1.3 cm/hr EA.

  - 24% (IT 7.5 ug suf) → Fetal Brady/Alan Dextil
  - 11% (EA) & 12% (IT 1.5ug suf/bup/epi)

  - 65 IT opioid vs 69EA sequentially
  - NO Diff in abn FHR changes (17%)

  - Review 24 studies
  - 2020 IT opioid, 1493 control
  - 1.8 X (95% CI 1.04-3.1) for IT opioid incr fetal brady
OAA: Three-day Course on Obstetric Anaesthesia and Analgesia, London, 9 – 11 November 2009

Fetal Brady – CSE vs Epid
- Abrao KC et al. (Obstetrics and Gynecology 2009; 113:41-7)
- RCT, mixed parity, < 7 cm, Intrauterine pressure
  - CSE (2.5mg bup + 2.5ug suf) (n=41)
  - EPID (10mL .125% bup + 10ug suf) (n=36)
- Primary Outcome measure: (monitor 15m post analg)
  - Prolong decel = baseline HR - 15 for >2-10min
  - Bradycardia = baseline <100 bpm
  - Incr Uterine tone = >10mmHg incr from basal

Abrao KC et al. (Obstetrics and Gynecology 2009; 113:41-7)

<table>
<thead>
<tr>
<th>Outcome Variables</th>
<th>O.R.</th>
<th>95% CI</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incr Uterine Tone After Analg</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CSE vs Epidural</td>
<td>0.926</td>
<td>0.71-10.36</td>
<td>0.922</td>
</tr>
<tr>
<td>Oxytocin dose @ Analg Initiation</td>
<td>0.89</td>
<td>0.89-1.09</td>
<td>0.719</td>
</tr>
<tr>
<td>FHR Abnormalities</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Incr Uterine Tone After Analg</td>
<td>1.8</td>
<td>4.48-77.2</td>
<td>0.001</td>
</tr>
<tr>
<td>Postblock Hypotension</td>
<td>0.55</td>
<td>0.09-3.57</td>
<td>0.541</td>
</tr>
<tr>
<td>FHR Abnormal + Incr Uterine Tone</td>
<td>0.772</td>
<td>0.59-0.99</td>
<td>0.049</td>
</tr>
<tr>
<td>Decr pain scores 5 m after Analg</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Impact of Bradycardia on OB Outcomes
- Gambling (98) showed no diff in spon del or C/S between CSE and IV meperidine.
- Albright et al series showed no difference in emergent C/S in CSE vs Epid.(1997)
- Abrao et al. No cases need tocolysis or C/S for suspected fetal distress. No neonatal outcome differences.

Attempts to answer the controversial question
- 3 yrs (2000-2)
- Retrospective Analysis
- 19 259 deliveries,
- 7849 (47%) EA
- 4741 (28%) CSE for Labor Analg
- Limitation – Retrospective, not controlled

Complications – FMC Experiences

<table>
<thead>
<tr>
<th>Labor Analgesia</th>
<th>Epidural (n=7849)</th>
<th>CSE (n=4741)</th>
<th>Total (n=12590)</th>
<th>( P )-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall Failure</td>
<td>14%</td>
<td>10%</td>
<td>12%</td>
<td>&lt;.000001</td>
</tr>
<tr>
<td>Initial IV Cath</td>
<td>7%</td>
<td>5%</td>
<td>6%</td>
<td>&lt;.00003</td>
</tr>
<tr>
<td>Subseq Migrated IV</td>
<td>24%</td>
<td>25%</td>
<td>25%</td>
<td>NS</td>
</tr>
<tr>
<td>Inadeq Epid Analg</td>
<td>8.4%</td>
<td>4.2%</td>
<td>6.8%</td>
<td>&lt;.000001</td>
</tr>
<tr>
<td>Epid Cath Replaced</td>
<td>7.1%</td>
<td>3.2%</td>
<td>5.6%</td>
<td>&lt;.000001</td>
</tr>
<tr>
<td>Multiple Replaced</td>
<td>1.9%</td>
<td>6.5%</td>
<td>1.5%</td>
<td>&lt;.000001</td>
</tr>
<tr>
<td>Wet Tap/ERP needed</td>
<td>1.4% (31%)</td>
<td>8% (28%)</td>
<td>1.2% (30%)</td>
<td>&lt;.002</td>
</tr>
</tbody>
</table>

How about Labor Epid Cath-in-situ needed for C/S?

<table>
<thead>
<tr>
<th>Labor Epid Cath</th>
<th>Labor Epid-in-situ for C/S</th>
<th>Labor CSE-in-situ for C/S</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pan</td>
<td>7.1%</td>
<td></td>
</tr>
<tr>
<td>Eappen</td>
<td>18%</td>
<td></td>
</tr>
<tr>
<td>Norris</td>
<td>6%</td>
<td>4%</td>
</tr>
<tr>
<td>Garry</td>
<td>10.5%</td>
<td></td>
</tr>
</tbody>
</table>
Is CSE catheter more likely to fail for C/S?
- Evidences do support higher CSE catheter failure.
- How often do you replace a catheter immediately after initial dosing (i.e. excluding spinal, iv and obvious misplaced indications)?
- Indications – IV/spinal cath, poor LOR, difficult catheter insertion, or (in CSE – no spinal CSF)
- Answer also depends on experience and aggressiveness of testing the catheter.

Labor Epidural vs CSE for C/S (2007-8)
- 2 year prospective data collection ongoing study
- All pts with Labor Epid/CSE presenting for C/S
- Incidence of Failed labor epidural catheters when needed for C/S
- Identify when catheters Failed or Replaced and for what reasons
- Normalize between groups for:
  - BMI
  - Provider experience
  - Labor duration.

Labor Epidural vs CSE for C/S (2007-8)

<table>
<thead>
<tr>
<th>Outcomes - (%) (Prelim Results)</th>
<th>CSE (n=310)</th>
<th>Epid (n=700)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. Catheters Replaced during Labor – (%)</td>
<td>5.8%</td>
<td>9.4%</td>
</tr>
<tr>
<td>B. Catheters Replaced just for C/S</td>
<td>2.3%</td>
<td>2.6%</td>
</tr>
<tr>
<td>C. Known Non-functioning cath + Failed cath for C/S despite adequate dosing – (%) (not B)</td>
<td>4.5%</td>
<td>5.4%</td>
</tr>
<tr>
<td>D. Catheters Failed and/or replaced just for C/S – (sum of B and C)</td>
<td>6.8%</td>
<td>8.0%</td>
</tr>
<tr>
<td>E. All Failed + Replaced @ labor or C/S – (%)</td>
<td>12.6%</td>
<td>17.4%</td>
</tr>
<tr>
<td>F. Unknown cath function @ C/S – (%) (not dosed or inadequate time) – (%)</td>
<td>0.5%</td>
<td>1.2%</td>
</tr>
</tbody>
</table>

Conclusion
- CSE faster onset labor analgesia, but more pruritus
- Motor block depends upon dose of LA used in EA or CSE.
- No significant diff in C/S rate
- Similar instrument delivery if low cone LA with opioid is used in EA.
- CSE “may” offer faster cervical dilation.
- Epid cath failure seems same or less with CSE with similar complications rate. ROTs to confirm or refute this finding.
- DP itself in CSE does not alter the epid cath success/failure, but may offer additional information on success/failure of epidural cath.

Who would I personally use or not use CSE labor analgesia
- Consider USE CSE
- Consider NOT USE CSE
- Early labor
  - Pending C/S (<1-2h or so)
- Late Labor
  - Underlying fetal heart rate abnormalities/concerns
- Severe Labor Pain
  - Underlying fetal stress/distress concerns
- ?back surgery pt

Old and New Thoughts on Combined Spinal Epidural (CSE): Can CSE be used for all Laboring Patients?

Peter H. Pan, MSEE, MD
Department of Anesthesiology
Wake Forest University School of Medicine
Winston-Salem, North Carolina
**Session 3: Labour Analgesia**
Maintaining labour epidurals - an update

Professor Cynthia Wong  
Associate Professor, Chief of Obstetrical Anesthesia,  
Northwestern University Feinberg School of Medicine, USA

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**Maintaining Labour Epidurals: An Update**

*Cynthia A. Wong, M.D.*

November 9, 2009

---

Why is method of maintaining epidural labor analgesia important?

---

**Epidural vs. Systemic Analgesia: Instrumental Vaginal Delivery**

Neuraxial labor analgesia is not all alike

---

**COMET Study (N = 1054)**

<table>
<thead>
<tr>
<th>Group</th>
<th>Initiation</th>
<th>Maintenance</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Epidural bupivacaine, 0.25%</td>
<td>Intermittent boluses, bupivacaine 0.25%</td>
</tr>
<tr>
<td>2</td>
<td>Epidural bupivacaine, 0.1% w/ fentanyl</td>
<td>Continuous infusion, bupivacaine 0.1% w/ fentanyl</td>
</tr>
<tr>
<td>3</td>
<td>CSE, bupivacaine/fentanyl</td>
<td>Intermittent boluses, bupivacaine 0.1% w/ fentanyl</td>
</tr>
</tbody>
</table>

COMET Group. Lancet 2001

---

**Risk of Instrumental Vaginal Delivery**

*P < 0.05*  
COMET Group, JAMA 2001
CSE vs. Epidural

Table 3. Characteristics of Vaginal Deliveries in the Study Groups.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Epidural Analgesia (N=210)</th>
<th>Spinal-Epidural Analgesia, Ambulation Discouraged (N=209)</th>
<th>Spinal-Epidural Analgesia, Ambulation Encouraged (N=212)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spontaneous delivery</td>
<td>130 (62)</td>
<td>350 (72)</td>
<td>432 (67)</td>
<td>0.03</td>
</tr>
<tr>
<td>— no. (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Instrumental delivery</td>
<td>36 (40)</td>
<td>59 (28)</td>
<td>70 (34)</td>
<td>0.65</td>
</tr>
<tr>
<td>— no. (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Birth weight of infant, g</td>
<td>3415±427</td>
<td>3407±434</td>
<td>3444±374</td>
<td>0.65</td>
</tr>
</tbody>
</table>

Nageotte MP. N Eng J Med 1997

Conclusions:
- Dense second stage analgesia may increase the rate of instrumental vaginal delivery

Methods of Maintaining Epidural Analgesia
- Manual bolus
- Continuous infusion
- PCEA with and without background infusion
- Programmed intermittent bolus

Epidural Maintenance
- Continuous infusion vs. bolus
  - Fewer manual boluses
  - Greater patient satisfaction
  - Greater anesthesiologist satisfaction
  - Greater bupivacaine consumption

Epidural Maintenance
- Continuous infusion vs. bolus
  - Fewer manual boluses
  - Greater patient satisfaction
  - Greater anesthesiologist satisfaction
  - Greater bupivacaine consumption
  - Greater motor block
  - Increased incidence of instrumental vaginal delivery

Intermittent Epidural Boluses vs. Continuous Infusion

<table>
<thead>
<tr>
<th>Study</th>
<th>Bupivacaine Dose (mg)</th>
<th>Motor Block</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Bolus</td>
<td>Infusion</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bogod 1987</td>
<td>130 ± 70</td>
<td>178 ± 83†</td>
</tr>
<tr>
<td>Smedstad 1988</td>
<td>87 ± 54</td>
<td>161 ± 71†</td>
</tr>
<tr>
<td>Boutros 1999</td>
<td>10.6 ± 2.4 (h)</td>
<td>14.6 ± 3.8† (h)</td>
</tr>
</tbody>
</table>

*P < 0.05

Conclusions:
- Dense second stage analgesia may increase the rate of instrumental vaginal delivery
- Continuous infusions result in more dense neuroblockade than bolus injections
  - Use dilute (< bupivacaine 0.125%) infusion solutions
Methods of Maintaining Epidural Analgesia

- Manual bolus
- Continuous infusion
- PCEA with and without background infusion
- Programmed intermittent bolus

PCEA (no BI) vs. CEI

<table>
<thead>
<tr>
<th>Outcome</th>
<th>PCEA</th>
<th>CEI</th>
<th>RD/WMD (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No interventions</td>
<td>187/272</td>
<td>100/243</td>
<td>27% (18 to 36%)*</td>
</tr>
<tr>
<td>LA consumption (mg/h)</td>
<td>257</td>
<td>221</td>
<td>-3.9 (-5.4 to -2.4)*</td>
</tr>
<tr>
<td>No motor block</td>
<td>131/167</td>
<td>103/129</td>
<td>18% (6 to 31%)#</td>
</tr>
<tr>
<td>Satisfied w/ analgesia</td>
<td>65/75</td>
<td>73/84</td>
<td>0% (-11 to 10%)</td>
</tr>
</tbody>
</table>

*P < 0.001, #P < 0.003

van der Vyver M. Br J Anaesth 2002:89:459

Epidural Maintenance

- Bolus vs. continuous infusion
- Continuous infusion vs. PCEA
- PCEA with and without background infusion
- Programmed intermittent bolus
- Maintenance solutions/protocols

PCEA: w/ and w/o Background Infusion

<table>
<thead>
<tr>
<th>N</th>
<th>BI (mL/h)</th>
<th>Bolus (mL)</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ferrante 1994</td>
<td>60</td>
<td>0, 3, 6, 12 (CEI)</td>
<td>3 Inc suppl at 0, 3 Inc dose at 12</td>
</tr>
<tr>
<td>Paech 1992</td>
<td>52</td>
<td>4</td>
<td>4 No differences</td>
</tr>
<tr>
<td>Petry 2000</td>
<td>80</td>
<td>3</td>
<td>3 No differences</td>
</tr>
</tbody>
</table>

Bupivacaine 0.125% / fentanyl or sufentanil

PCEA: w/ and w/o Background Infusion

<table>
<thead>
<tr>
<th>Supplemental bolus (1-2) (%)</th>
<th>0 mL/h N = 34</th>
<th>3 mL/h N = 34</th>
<th>6 mL/h N = 32</th>
<th>9 mL/h N = 33</th>
</tr>
</thead>
<tbody>
<tr>
<td>NSVD (%)</td>
<td>79</td>
<td>64</td>
<td>84</td>
<td>82</td>
</tr>
<tr>
<td>Satisfaction (0-10)</td>
<td>9</td>
<td>9</td>
<td>9</td>
<td>9</td>
</tr>
</tbody>
</table>

Boselli E. Anesthesiology 2004;100:968

OAA: Three-day Course on Obstetric Anaesthesia and Analgesia, London, 9 – 11 November 2009
PCEA: w/ and w/o Background Infusion

- Ropivacaine 0.16% / sufentanil 0.5 \( \mu \)g/mL / BI: 4 mL/h

- PCEA with and without background infusion
  - Low volume background infusions make little difference
  - Higher volume (> 5 mL/h) helpful with longer labors, patients wanting to sleep
  - ? Dependent of LA concentration
  - ? Increase in LA consumption
  - Little clinical significance with low concentration infusions
  - 30-50% total volume via infusion


Methods of Maintaining Epidural Analgesia

- Manual bolus
- Continuous infusion
- PCEA with and without background infusion
- Programmed intermittent bolus

Bolus injection through an epidural catheter may result in better drug distribution than continuous infusion

Kaynar AM. Anesth Analg 1999

0.4 mL 4 mL 10 mL

Tip epidural catheter

Hogan Q. Anesthesiology 1999:90:4

Bupivacaine 0.0625% / fentanyl 2 \( \mu \)g/mL

RCT, N = 126

12 mL/h vs. 6 mL q30 min

Labor Analgesia

<table>
<thead>
<tr>
<th>Group</th>
<th>PIEB (n=63)</th>
<th>CEI (n=63)</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline VAS (mm)</td>
<td>59 ± 18</td>
<td>57 ± 16</td>
<td>0.59</td>
</tr>
<tr>
<td>Labor pain( ^a )</td>
<td>6.7 (4.2, 3.3)</td>
<td>10.3 (6.6, 11.6)</td>
<td>0.25</td>
</tr>
<tr>
<td>Epidural dose (mg h(^{-1}))</td>
<td>6.7 (5.1, 7.7)</td>
<td>7.4 (5.7, 7.4)</td>
<td>0.001</td>
</tr>
<tr>
<td>PCEA requests (n h(^{-1}))</td>
<td>1.1 (0.5, 5.3)</td>
<td>1.3 (0.6, 7.3)</td>
<td>0.60</td>
</tr>
<tr>
<td>PCEA dose (mg h(^{-1}))</td>
<td>2.5 (1.6, 6.5)</td>
<td>2.5 (1.8, 7.4)</td>
<td>0.87</td>
</tr>
<tr>
<td>Manual dose (n)</td>
<td>20</td>
<td>34</td>
<td>-</td>
</tr>
<tr>
<td>Manual dose (mg h(^{-1}))</td>
<td>0 (0, 10)</td>
<td>1.6 (0, 17.4)</td>
<td>0.02</td>
</tr>
</tbody>
</table>

\( a = \text{(Area under the VAS * time curve)/duration of neuraxial analgesia.} \)

Wong CA. Anesth Analg 2006:102-904
**PIEB vs. Continuous Infusion**

<table>
<thead>
<tr>
<th></th>
<th>PIEB (n=30)</th>
<th>CEI (n=30)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breakthrough pain n(%)</td>
<td>3 (10)</td>
<td>11 (37)</td>
<td>0.04</td>
</tr>
<tr>
<td>Satisfaction score (0 – 100 mm)</td>
<td>97 ± 8</td>
<td>89 ± 7</td>
<td>0.003</td>
</tr>
</tbody>
</table>

L-bupivacaine 0.1% / fentanyl 2 µg/mL
5 mL q30 min vs. 10 mL/h

Lim Y. Int J Obstet Anesth 2005;14:305

- Programmed intermittent bolus
  - Less drug
  - Less breakthrough pain
  - Improved satisfaction
  - Current pump technology needs to catch up

**Conclusions**

- Dense second stage analgesia may increase the rate of instrumental vaginal delivery
- Continuous infusions result in more dense neuroblockade than bolus injections
  - Use dilute (< bupivacaine 0.125%) infusion solutions
- PCEA and programmed intermittent bolus techniques are methods of continuous analgesia that rely on bolus dosing
References


4. Bogod DG, Rosen M, Rees GA. Extradural infusion of 0.125% bupivacaine at 10 mL/hr to women during labor. Br J Anaesth 1987; 59: 325-30


11. Boselli E, Debon R, Cimino Y, Rimmele T, Allaouchiche B, Chassard D. Background infusion is not beneficial during labor patient-controlled analgesia with 0.1% ropivacaine plus 0.5 microg/ml sufentanyl. Anesthesiology 2004; 100: 968-72


15. Hogan Q. Epidural catheter tip position and distribution of injectate evaluated by computed tomography. Anesthesiology 1999; 90: 964-70


Session 3: Labour Analgesia

Optimizing your labour epidural - from insertion to delivery / clinical pearls

Professor Peter Pan
Professor & Director of Anesthesia Clinical Research,
Wake Forest University School of Medicine, Winston-Salem, USA

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**Optimizing your labour epidural – from insertion to delivery (clinical pearls)**

“From Literature to Daily Practice: Is Your Epidural Optimized?”

Peter H. Pan, MSEE, MD
Department of Anesthesiology
Wake Forest University School of Medicine
Winston-Salem, North Carolina

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**Introduction**

- 1885 - Epidural first use
- 1940 - Limited OB pain relief, mostly caudal
- Late 1940’s – lumbar epid gained popularity
- 1960 onward – research & clinical demonstration of reliability, flexibility & safety
- Then Refinement of Technique, Safety & Drug enhance optimal Labor Neuraxial Analgesia

---

**Is your Epidural Optimized?**

*From Literature to Clinical Practice*

- Preparation
  - Airway Examinations
  - Thromoprophylaxis and Thrombocytopenia
  - Aseptic Technique
- Procedure
- Optimize Analgesia
- Post Neuraxial Analgesia/Anesthesia

---

**Is your Epidural Optimized?**

*From Literature to Clinical Practice*

- Prospective AW study
  - Onset vs End of labor
- Part I
  - Samsoons mod Mallampati AW evaluation
- Part II
  - Acoustic reflectometry – oral/pharyngeal vol
- Sample size n = 61

---

**Samsoon modified Mallampati Airway Evaluation**

<table>
<thead>
<tr>
<th>Onset vs End Labor</th>
<th>Pre-labor Class I (n=9)</th>
<th>Pre-labor Class II (n=35)</th>
<th>Pre-labor Class III (n=17)</th>
<th>All grade classes (n = 61)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increase one or more grade</td>
<td>5/9 (56%)</td>
<td>12/35 (34%)</td>
<td>6/17 (35%)</td>
<td>23/61 (38%)</td>
</tr>
<tr>
<td>Increase two or more grade</td>
<td>1/9 (11%)</td>
<td>2/35 (6%)</td>
<td>-----</td>
<td>3/44 (7%)</td>
</tr>
</tbody>
</table>

---
Preparation - Parturients with LMWH thromboprophylaxis


- ASA concensus guidelines
- Heparin SQ
- Systemic therapeutic Heparin
- LMWH – QD, BID, high dose
- Antiplatelet agents:
  - ASA, NSAID
  - Thienopyridine derivatives (ticlopidine(Ticlid), clopidogrel(Plavix))
  - Platelet GP IIb/IIIa antagonists (abciximab, epifibatide, tirofiban)

Airway Lessons to Clinical Practice

- Airways can change significantly during L/D.
- Airway incr 1 or more grades in 38% of parturients during labor
- Co-inside with sig. Oral/Pharyngeal volumes decreases.
- A careful re-evaluation of airway prior to administration of analgesia & anesthesia during labor course is essential rather than relying on preop/prelabor exam.

Preparation - Parturients with SQ Heparin thromboprophylaxis


- Heparin SQ small dose 5000U SQ
- Typically not prolong aPTT or monitored
- 15% subset with measurable aPTT changes
- 2-4% subset assoc decr PLT with > 5 days Hep
- Non-linear (disproportionately) Dose dependent increase in effect
- Wide spread use and paucity of complications suggest safety with low dose SQ and neuraxial anesthesia
- Clinical judgement or monitoring may be required in higher doses or unique clincial situations

Preparation - Parturients with LMWH thromboprophylaxis


- LMWH – QD, BID, (high dose)
- 40mg QD; 30 mg q12h; (enoxaparin 1mg/kg q12h, dalteparin 120U/kg q12h; tinzaparin 175U/kg/day)
- Recommendation:
  - Wait 12h after last QD dose prior to needle placement
  - QD dose initiation 6-8 hr postop after needle placement
- Recommendation:
  - Wait 24h after BID/higher dose prior to needle placement
  - QD dose 6-8 hr postop after needle placement
- Recommendation:
  - Wait 12h(QDdose) or 24h(BID) after last dose prior to catheter removal
  - First dose administer Wait 2+ hours after needle placement and 6-8h postop(QDdose) and 24h post (if BID or high dose)

Preparation - Parturients with antiplatelet thromboprophylaxis


- ASA affects platelet lifetime
- NSAID affects platelet 3-4 days
  - (Collaborative Low Dose Aspirin Study in pregnancy – Lasoet 1996; 54:619-26 – 3422 high risk Obstetris ASA 300 mg daily complex)
- CLASP study & paucity of case reports given prevalence of ASA and NSAID use suggest safety of neuraxial analgesia in presence of low dose ASA/NSAID.

Preparation - Parturients with antiplatelet thromboprophylaxis


- Thienopyridine derivatives: (plavix, Ticlid)

RECOMMENDATION

Discontinue Time from last dose prior to neuraxial block

- Clopidogrel(Plavix) – Discontinue 7 days prior
- Ticlopidine(Ticlid) - Discontinue 14 days prior

<table>
<thead>
<tr>
<th>Oral Volume (mL)</th>
<th>Oral Area (cm²)</th>
<th>Pharyngeal Volume (mL)</th>
<th>Pharyngeal Area (cm²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre Labor</td>
<td>49.10</td>
<td>5.84</td>
<td>26.87</td>
</tr>
<tr>
<td>Post Labor</td>
<td>44.40 P&lt;0.05</td>
<td>6.06</td>
<td>21.80 P&lt;0.001</td>
</tr>
</tbody>
</table>
Is your Epidural Optimized? 
(From Literature to Clinical Practice) 
• Preparation  
  • Platelet GP IIb/IIIa antagonists (abciximab, epifibatide, tirofiban)  
  • RECOMMENDATION  
  • Discontinue Time from last dose prior to neuraxial block  
  • abciximab – Discontinue 24-48 hrs prior  
  • Epifibatide, tirofiban – Discontinue 8hrs prior  

procedure – needle bevel orientation  
• Bevel cephalad vs lateral and caudal when cath inserted  
• Single orifice catheter inserted 3 cm  
• Dose with total 13 mL 0.25% bupivacaine over 10mins (test on left, turn right, then turn left)
Is your Epidural Optimized?

(From Literature to Clinical Practice)

- Procedure
  - Needle Bevel Orientation
  - Patient Positioning
  - Air vs Saline
  - Effect of Epidural Saline
- Optimize Analgesia
  - Initial Dosing after placement
  - Incomplete Analgesia Management
  - Post Neuraxial Analgesia

Procedure – Lateral vs Sitting Positioning

From Literature to Clinical Practice

- Patient Comfort/Preference
- Vagal or Orthostatic Response
- Aortocaval Compression / CO
- Epidural Venous Puncture
- Distance/Movement to Epidural Space

Procedure – Amount of Saline

From Literature to Clinical Practice

<table>
<thead>
<tr>
<th>2mL air vs NS</th>
<th>Lateral</th>
<th>Sitting</th>
</tr>
</thead>
<tbody>
<tr>
<td>No Diff in IV cath, paresthesia &amp; DP; Air gp more</td>
<td>No Diff in IV cath, paresthesia &amp; DP; Air gp more</td>
<td></td>
</tr>
<tr>
<td>initial incomplete block, improve with suppl dose</td>
<td>initial incomplete block, improve with suppl dose</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>1, 5 &amp; 10mL NS</th>
<th>Lateral</th>
<th>Sitting</th>
</tr>
</thead>
<tbody>
<tr>
<td>No diff in block to cold; LESS pinprick block -16 dermatomes(10mL) vs 22(1 mL); suggest diluted LA</td>
<td>No diff in block to cold; LESS pinprick block -16 dermatomes(10mL) vs 22(1 mL); suggest diluted LA</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>1, 5 &amp; 10mL NS</th>
<th>Lateral</th>
<th>Sitting</th>
</tr>
</thead>
<tbody>
<tr>
<td>15 (10mL) vs 12 (1mL); spinal segments block to cold in 1% mepiv gp. No diff in all others</td>
<td>15 (10mL) vs 12 (1mL); spinal segments block to cold in 1% mepiv gp. No diff in all others</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>2 vs 10mL NS</th>
<th>Lateral</th>
<th>Sitting</th>
</tr>
</thead>
<tbody>
<tr>
<td>15-19(10mL) vs 12-15(2mL); dermatomes block to pinprick &amp; cold; No diff in IV cath</td>
<td>15-19(10mL) vs 12-15(2mL); dermatomes block to pinprick &amp; cold; No diff in IV cath</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>0 vs 10mL NS</th>
<th>Lateral</th>
<th>Sitting</th>
</tr>
</thead>
<tbody>
<tr>
<td>2% (10mL) vs 20% (2mL) in IV cath</td>
<td>2% (10mL) vs 20% (2mL) in IV cath</td>
<td></td>
</tr>
</tbody>
</table>

Procedure – Optimize Initial Dosing

(From Literature to Clinical Practice)

- 58 primiparae, active labor, < 5cm dilated
- RTG, 3 groups
- After test dose 3mL
- 2% lido with 200g epi
- Randomize to receive 20mg epidural bupiv using 1 of 3 solutions

<table>
<thead>
<tr>
<th>Complete Pain Relief</th>
<th>50% Pain Relief</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.5% - 4mL (n=19)</td>
<td>0.5% - 4mL (n=19)</td>
</tr>
<tr>
<td>Op 0.5% n = 19</td>
<td>Op 0.5% n = 19</td>
</tr>
<tr>
<td>4mL 0.5% bupiv</td>
<td>4mL 0.5% bupiv</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>0.2% - 10mL (n=20)</th>
<th>0.2% - 10mL (n=20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Op 0.2% n = 20</td>
<td>Op 0.2% n = 20</td>
</tr>
<tr>
<td>10mL 0.2% bupiv</td>
<td>10mL 0.2% bupiv</td>
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</tbody>
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<td>20mL 0.1% bupiv</td>
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</tbody>
</table>

Procedure – Air vs Saline LORT

From Literature to Clinical Practice

- Incomplete Analgesia
- PDPH
- Nerve Root Compression
- CSE – Air vs Saline LORT

Procedure – Optimize Initial Dosing

(From Literature to Clinical Practice)

- Procedure
  - Needle Bevel Orientation
  - Patient Positioning
  - Air vs Saline
  - Effect of Epidural Saline
- Optimize Analgesia
  - Initial Dosing after placement
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  - Post Neuraxial Analgesia

Procedure – Lateral vs Sitting Positioning

From Literature to Clinical Practice

<table>
<thead>
<tr>
<th>Lateral</th>
<th>Sitting</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI &lt; 25</td>
<td>BMI &gt; 30</td>
</tr>
<tr>
<td>0%</td>
<td>20%</td>
</tr>
<tr>
<td>Greater (-30%)</td>
<td>Less (-10%)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Epidural Venous Puncture</th>
<th>Lateral</th>
<th>Sitting</th>
</tr>
</thead>
<tbody>
<tr>
<td>6% (lax), 2% (fat Thaw)</td>
<td>7.8% &gt; 1 attempt</td>
<td>15.3% &lt; 5 attempt</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Distance to Epid Space (cm)</th>
<th>Lateral</th>
<th>Sitting</th>
</tr>
</thead>
<tbody>
<tr>
<td>5.0+/-1.1* cm</td>
<td>4.4+/-0.8*cm</td>
<td></td>
</tr>
</tbody>
</table>

Cath move (cm) flex-sitting to lat

<table>
<thead>
<tr>
<th>0.67+/-0.42</th>
<th>0.75+/-0.42</th>
</tr>
</thead>
<tbody>
<tr>
<td>(n=19)</td>
<td>(n=19)</td>
</tr>
<tr>
<td>1.04+/-0.68</td>
<td>1.09+/-0.68</td>
</tr>
<tr>
<td>(Range: 15 to 1+20)</td>
<td>(Range: 15 to 1+20)</td>
</tr>
</tbody>
</table>

Procedure – Optimize Initial Dosing

(From Literature to Clinical Practice)

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</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>0.2% - 10mL (n=20)</th>
<th>0.2% - 10mL (n=20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Op 0.2% n = 20</td>
<td>Op 0.2% n = 20</td>
</tr>
<tr>
<td>10mL 0.2% bupiv</td>
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</table>
Procedure – Optimize Initial Dosing

From Literature to Clinical Practice – RAPM 1998;23: 134-41

<table>
<thead>
<tr>
<th>Dermatome Blocked</th>
<th>Analgesic Duration (min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.5% - 4mL (n=19)</td>
<td>9 43+/-21</td>
</tr>
<tr>
<td>0.2% - 10mL (n=19)</td>
<td>17 100+/-26</td>
</tr>
<tr>
<td>0.1% - 20mL (n=20)</td>
<td>18 120+/-21</td>
</tr>
</tbody>
</table>

• Suggest # dermatomes blocked and analgesia is associated with epidural volume first and then drug conc second with same amount of drug(mg)

Is your Epidural Optimized?

(From Literature to Clinical Practice)

• Optimize Analgesia
  – Dosing Initial Dosing
  – Incomplete Analgesia Management
• Post Neuraxial Analgesia

Procedure – Optimize Incomplete Analgesia

From Literature to Clinical Practice – Anesthesiology 1998; 88:1502-6

• Incomplete Analgesia (78 pts) randomized to:
  • 1st Intervention:
    – (1) Additional .25% bupivacaine 5 mL
    – (2) Pull catheter back 1 cm, then admin same LA
  • If 1st intervention not successful, 2nd intervention is reversed
  • All dosed on lateral decubitus position with hurting side down (dependent)

Procedure – Optimize Incomplete Analgesia

From Literature to Clinical Practice – Anesthesiology 1998; 88:1502-6

<table>
<thead>
<tr>
<th>Local Anesth Only n = 39</th>
<th>Pull Cath + LA n = 39</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incomplete Analgesia after initial 13mL dose</td>
<td></td>
</tr>
<tr>
<td>R side 33</td>
<td>R side 30</td>
</tr>
<tr>
<td>L side 6</td>
<td>L side 9</td>
</tr>
</tbody>
</table>

Success after 1st Intervention

(74%) (77%)

OAA: Three-day Course on Obstetric Anaesthesia and Analgesia, London, 9 – 11 November 2009
Procedure – Optimize Incomplete Analgesia

<table>
<thead>
<tr>
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<th>Local Anesth Only n = 39</th>
<th>Pull Cath + LA n = 39</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incomplete Analgesia after initial dose</td>
<td>R side 33 L side 6</td>
<td>R side 30 L side 9</td>
</tr>
<tr>
<td>Success after 1st intervention</td>
<td>(74%)</td>
<td>(77%)</td>
</tr>
<tr>
<td>Success after 2nd (reverse) intervention</td>
<td>(100%)</td>
<td>(100%)</td>
</tr>
</tbody>
</table>

Procedure – Optimize Post Analg/Anesth

- Pregnancy Associated Deaths 1985-2003 in Michigan
  - Using MMIS and matched MDOH data with electronic linked matched death certificates
  - Of 855 pregnancy-associated death, 25 cases associated with perioperative arrest/anesthesia complication reviewed
  - 8 were anesthesia-related and 7 were anesthesiarelated contributing. Of the 8 anesthesia-related deaths, 4 had neuraxial blockade

- A Series of Anesthesia-related Maternal Deaths in Michigan, 1985-2003
  - Three Key Lessons:
    - All anesth-related deaths from airway obstruction/hypovent during emergence or recovery period and not induction
    - Lapses in postoperative monitoring and inadequate supervision contributed to 5/8 of anesth-related death
    - Obesity and African-American race are important risk factors.

Procedure – Optimize Post Analg/Anesth

- Preparation
  - Airway can change over labor course
  - Thromboprophylaxis & Thrombocytopenia

- Procedure
  - Sterile Technique – update with ASRA guidelines
  - Cephalad Needle Bevel Orientation
  - Positioning – sitting vs lateral
  - Saline may be more preferred

- Dosing
  - Volume over Concentration
  - Volume first, adjust cath next

- Post analgesia
  - Airway Obstruction, vigilance, monitor, risk factors

46
The standard Macintosh laryngoscope and direct laryngoscopy remains the main technique for visualisation of the laryngeal inlet and the passage of a tracheal tube for the majority of anaesthetists in the UK. When problems with direct laryngoscopy are anticipated or encountered different types of laryngoscope have been tried to improve the view of the laryngeal inlet. These have included laryngoscopes utilising a paraglossal straight laryngoscopy technique (Miller, Belscope, Choi, and Henderson).

More recently devices which allow indirect visualisation of the laryngeal inlet on a screen or eyepiece have been introduced in an attempt to improve the rate and speed of tracheal intubation, whilst minimising trauma to laryngo-pharyngeal tissues. Most of these devices are designed for patients in which conventional standard Macintosh laryngoscopy has proven to be or is expected to be difficult.

These new devices can be grouped into those that employ a rigid stylet onto which a tracheal tube is loaded. The tip of the stylet contains a camera or fiberoptic technology that allows visualisation of the tip through an eyepiece or screen. These devices include the Bonfils Intubation Fibrescope, Shikani Optical Stylet and Levitan Stylet. Some of these rigid ‘visualised stylets’ allow the tip to be bent to optimise laryngeal entry whilst others are fixed.

The second group include the Glidescope and McGrath laryngoscopes. These allow visualisation of ‘anterior’ structures in which conventional laryngoscopy would reveal a view of the tip of the epiglottis at best. These devices convert a difficult direct view to a better indirect view on a screen. Tracheal intubation can now be seen although the increased angle that the tube has to pass often requires the presence of a stylet or bougie to angle the tube in the correct direction.

The third group include the Airtrac and Pentax AWS airway scope. These devices have channels along their lateral border that allow a tube to pass along and guide the tube to the distal end of the device. This results in the tube emerging near the laryngeal inlet without the need for stylets or bougies. Both of these devices look, handle and are inserted in the mouth in a different manner to a standard laryngoscope.

The fourth group (Storz CMAC) handle and function in an identical manner to standard Macintosh laryngoscopes but have cameras incorporated into the laryngoscope blade. This allows visualisation of the laryngeal inlet and may aid or confirm tracheal intubation. These devices are also useful in teaching standard laryngoscopy.
Session 4: New Developments
Acute to chronic pain after delivery: from predictors to spinal oxytocin

Professor Peter Pan
Professor & Director of Anesthesia Clinical Research,
Wake Forest University School of Medicine, Winston-Salem, USA

Acute to Chronic Pain after Delivery: From Predictors to Spinal Oxytocin
Peter H. Pan, MSEE, MD
Section on OB/GYN Anesthesiology
Wake Forest University School of Medicine
Winston-Salem, North Carolina

Predictor to Oxytocin
• Multifactorial Preoperative Predictors for Postcesarean Section Pain and Analgesic Requirement.
  (Anesthesiology 2008;104:417–25)
• Severity of acute pain after childbirth, but not type of delivery predicts persistent pain and postpartum depression.
  (Pain 2008; 140:87–94)
• Prevention of chronic pain from childbirth by peripartum release of spinal oxytocin.

The Results – Pain Variability
Post Cesarean Section VAS Pain Scores

Results – Analgesic Variability
Post Cesarean Section Analgesic Consumption

Acknowledgement: Some of the slides & data are courtesy of Dr. Eisenach & lab.
Previous Studies for Predictors

- **Granot**: preop thermal suprathreshold pain scores (not threshold) on the arm correlated with postop pain. ($r = 0.4-0.5$)
- **Hsu**: preop pressure pain tolerance on the arm correlated with postop pain ($r = -0.5$) and pressure pain tolerance after fentanyl predict analgesic consumed ($r = -0.4$)

**Preop Assessment**

- **STAI** – State Trait and Anxiety Inventory
- **Questionnaire**:  
  - Preexisting Pain and Unpleasantness
  - Anxiety Level
  - Expectation of Pain and Analgesia Required
- A series Thermal Sensitivity Tests
- A series of Audio Sensitivity Tests

**Multifactorial Predictors**

- Previous studies focused on individual predictive factor have resulted in weak but significant correlation to postop pain.
- None in a comprehensive way to explore a multi-factorial model to predict postop pain and analgesic need.
- Goal: Assess thermal pain threshold and supra-threshold; audio sensitivity, as well as a series of psychological tests for anxiety, expectation and pre-existing pain.

**Factor analyses -> 7 Independent Predictive Factor Groups**

- Thermal Pain and Unpleasantness – arm & back
- Thermal Pain Threshold – arm and back
- Blood Pressure – Preop BP
- Preexisting Pain – Pregnancy Pain & Unpleasantness
- Expectation – Postop Pain & analgesia expected
- Intraoperative Factor – Surg dur & sensory block at incision
- **STAI**

**PostOperative Pain**

- **Multiple Factors**: Physiological, Psychological, Sensory, Affective, cognitive, socio-cultural, age and gender.
- High interpatient variability in postop pain and analgesic need
- High % hosp pts mod to severe postop pain
- JCAHO ->3/10 rest and/or activity pain
- Severe postop pain may lead to Chronic pain

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- Expectation – Postop Pain & analgesia expected
- Intraoperative Factor – Surg dur & sensory block at incision
- **STAI**
Results – Multifactorial Pain Predictors

• Resting Pain: (R²=.26, p<.01)
  – Thermal Pain & Unpleasantness
  – Expectation

• Evoked Pain: (R²=.20, p<.009)
  – Back Thermal Pain Threshold

• Composite Pain Score: (R²=.28, p<.008)
  – Thermal Pain & Unpleasantness
  – PreOp BP

Simplified Pain Predictors Model

- Audio Tone
- Anxiety
- Pain Expectation

Prelim Results – Simplified Pain Predictors
using Anxiety, Anticipated Pain and Audio Sensitivity

- Resting Pain: (R=.33, p<.005)
- Evoked Pain: (R=.41, p<.001)
- PACU Narcotic: (R=.39, p<.001)
- 24hr PostOp Narcotic: (R=.41, p<.001)

Prelim Results – Pain Predictors
using Anxiety, Anticipated Pain and Audio Sensitivity

- Simple, readily obtainable preoperative indices can predict narcotic usage and pain scores
- Cut-off scores can identify patients at risk for inadequate pain control.
- Patients may benefit from individually tailored, multimodal pain management strategies
- Future step:
  – Test model with cut-off scores;
  – Evaluate intervention strategies based on cut-off scores;
  – Application of models to other surgery

Does acute pain has long term implications?

Trauma and pain

Level of pain
**Pain After Delivery**

- **Goal:** Prevalence of acute & chronic PAD; Predictive factors; Long term implications.
- **Prospective, descriptive cohort longitudinal multi-center study** (4 international centers, 2350 patients)
- Interviewed in hospital, F/U 2, 8, 12 months
- In Hospital: maternal/neonatal demographics and medical hx, pre-existing pain, somatization, pain level
- F/U: postpartum pain (freq, loc, intensity, tx, daily activity impact), depression (Edinburgh Postnatal Scale); health changes.

**Chronic Pain Prevalence**

<table>
<thead>
<tr>
<th></th>
<th>Vaginal Delivery</th>
<th>Cesarean Delivery</th>
<th>Combined VD + C/S</th>
</tr>
</thead>
<tbody>
<tr>
<td>Percent of patients with pain @ 8 week</td>
<td>10.1%</td>
<td>9.2%</td>
<td>9.8%</td>
</tr>
<tr>
<td>Percent of patients with pain @ 6 month</td>
<td>1.8%</td>
<td>1.9%</td>
<td>1.9%</td>
</tr>
<tr>
<td>Percent of patients with pain @ 12 month</td>
<td>0.45%</td>
<td>0.0%</td>
<td>0.3%</td>
</tr>
</tbody>
</table>

**Does acute pain has long term implications?**

Severity of acute pain after childbirth, but not type of delivery predicts persistent pain and postpartum depression.

After controlling for propensity to deliver via C/S, every point increase in acute pain after delivery was associated with 8.3% increase in 8-wk Depression score and a 12.7% increase in odds of experiencing persistent pain at 8 wk. However pain at 1 yr remained very low.

**What is the incidence of chronic pain after delivery?**

- 0.3% @ 12 month
- 1.9% @ 6 month
- 9.8% @ 8 week

**What is the incidence of chronic pain after delivery?**

- 0.3% @ 12 month
- 1.9% @ 6 month
- 9.8% @ 8 week

**Persisten pain is amazingly rare**

- 0.3% @ 12 month
- 1.9% @ 6 month
- 9.8% @ 8 week

**Cumulative enrollment**

- 2004: 0
- 2005: 2400

**Odds of Persist Pain**

- Cesarean Delivery: 32.5% (4.0)*
- Acute Pain Score: 20.1% (7.5)*
- Depression Score: 4.4% (7.5)*
- Odds of Persist Pain: 9.3% (5.2)*

**Severity of acute pain after childbirth, but not type of delivery predicts persistent pain and postpartum depression.**

**Persistent pain is amazingly rare**

- 0% @ 1 Year
- 1.9% @ 6 month
- 9.8% @ 8 week

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- Cesarean Delivery: 32.5% (4.0)*
- Acute Pain Score: 20.1% (7.5)*
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But isn’t this strange since the system is setup for more pain?

- IL1β
- TNFα
- NO
- PGE2

Term pregnant

Why???

- Why delivery and/or immediate post delivery seem to provide protection from development of pain hypersensitivity?

Previous Laboratory Findings

This study shows the effect of delivery on Pre-Existing injury
Protection from neuropathic pain immediately after delivery is permanent!

Surrogate Mom (No Preg/Delivery) + Pup/Mom interact

MCN

Magnocellular Neurons

Parvocellular Neurons

(PVN)

Posterior Pituitary

Peripheral Oxytocin

Central/spinal Oxytocin

Atosiban – oxytocin antagonist result in decrease withdrawal threshold → loss of protection of pain hypersensitivity.

And oxytocin is protective in males

Protective Effect from Pain Hypersensitivity Development

• Seems to associate with an increase in CSF oxytocin (synthesized in parvocellular neurons) but not peripheral oxytocin (from magnocellular neurons).

• Central protective effect was inhibited by intrathecal oxytocin antagonist

OAA: Three-day Course on Obstetric Anaesthesia and Analgesia, London, 9 – 11 November 2009
Predictor to Oxytocin

Peter H. Pan, MSEE, MD

Section on OB/GYN Anesthesiology
Wake Forest University School of Medicine
Winston-Salem, North Carolina

Thank You!!

Peter H. Pan, MSEE, MD
Session 5: Blood Issues  
Obstetric Haemorrhage- rFV11a, interventional radiology, cell salvage and beyond  
Felicity Plaat  
Consultant Anaesthetist, Queen Charlotte’s & Hammersmith Hospitals, London

The need for effective therapies is indisputable. The World Health Organization estimates that >130 000 maternal deaths occur globally each year due to obstetric haemorrhage. A 1% case fatality rate suggests a yearly incidence of > 13 million cases. Thus obstetric haemorrhage represents the most common cause of severe maternal morbidity and long term sequelae world-wide [1, 2]. In the UK, the mortality rate per 100 000 maternities due to haemorrhage has not changed significantly over the past 15 years. Furthermore in the most recently published report ~60% cases were associated with substandard care, [3].

A review of the risk factors for haemorrhage suggest that both in Developed and Developing countries, the incidence is set to rise, with obesity and rise in cases of multiple section being of particular concern, [4]

This lecture will review those aspects of management most relevant to anaesthesia, where there have been recent changes and where there is controversy.

**Intravenous resuscitation, transfusion and medical management of coagulopathy**

The crystalloid – colloid debate in immediate resuscitation continues. Crystalloids have the advantage of being cheap, readily available and associated with a lower incidence of anaphylactoid reactions. In vitro and animal studies show that colloids impair coagulation by interfering with fibrin polymerisation [5] Newer colloids such as hydroxyethylstarch HES130/0.4, (Voluven®) may have less effect [6] However all clear fluids lead to dilutional coagulopathy and large volumes of 0.9% saline should be avoided as they cause hyperchlaemiac acidosis. The use of balanced solutions is advocated, [7] Many management protocols suggest the administration of 1500 ml clear fluid prior to transfusion. Given the problem of impaired coagulation, unless the situation appears to be rapidly self limiting, blood should be administered as soon as it is available.

In animal models of uncontrolled haemorrhage, and in non-pregnant trauma patients, aggressive fluid administration seems to be associated with decreased survival. [8] Because of the reliance of the placental circulation on maternal venous return however, hypotensive resuscitation may only be appropriate in the management of post-partum haemorrhage when definitive management is delayed.

Transfusion of red blood cells is required when 30% or more of the circulation blood volume is lost to maintain oxygen delivery to the tissues. Current guidelines state that transfusion is rarely necessary when the haemoglobin concentration is > 10g/dL but almost always required at concentration at or below 6g/dL. [9] Due to the torrential nature of obstetric blood loss and increased oxygen requirements during pregnancy and labour, it would seem prudent to aim towards the upper end of this spectrum.

Traditionally the use of blood products was dependant on the result of laboratory tests and if these were unavailable, empiric regimes such FFP to be given after a fixed number packed red cells, were widely advocated. Such formula based approaches have not been shown to improve outcome. [9] Recently such management has been challenged and experience gleaned from the management of military casualties, suggest that red cells and clotting factors should be given in roughly similar proportions especially early in resuscitation [10] In practice this is next to impossible to achieve as FFP has to be stored at -30°C and thawed to 37°C – a process that takes at least 30 minutes. Nevertheless in the UK, many units are now recommending such policies, (Personal communication). A possible adverse consequence may be an increase in transfusion related complications, such transfusion related acute lung injury (TRALI), which is 5-6 times more common after blood products than red cell transfusion. The latest report on transfusion related complications in the UK, includes 1 probable and one possible case of TRALI in obstetric patients [11]

FFP contains variable amounts of fibrinogen, and is too dilute to increase levels enough to be effective without risking fluid overload. Cryoprecipitate is more concentrated – containing 150-250 mg fibrinogen per 10 ml in addition to the other vitamin K dependant clotting factors. However cryoprecipitate is unavailable in the pathogen inactivated form. Recent studies suggest that fibrinogen concentrate and prothrombin complex concentrates may be used to reduce the need or as substitutes. [12, 13]
Cut-down methods have been advocated for patients with difficult intravenous access. Recently there has been a renewed interest in utilising the interosseous route and several new systems have been developed. The use of bedside investigations including thromboelastography will be discussed.

**Use of rFV11a in obstetrics**

Although rFV11a is still only licensed for use in haemophilia patients and for some platelet dysfunction disorders, its use in obstetrics is becoming widespread. Although often used as ‘last ditch’ therapy, there is growing evidence that used earlier, it may reduce the need for subsequent transfusion and or intervention.[15] There is currently a multicentre randomised controlled study underway to elucidate the role of rFV11a.[16]

The safety of rFV11a has not been established. In placebo controlled studies in non-pregnant patients, the risk of thrombosis is not increased, although thrombotic complications in an obstetric patient has recently been described.[17] Hypothermia, acidosis and depletion of fibrinogen and platelets should be corrected to maximize the efficacy of rFV11a.[18]

**Cell Salvage in obstetrics**

In the UK, the use of cell salvage in obstetrics was pioneered by Dr Catling in Swansea. To date there have been over 200 cases across Wales, in which cell salvage has been used. [S. Catling – Personal Communication] However blood was only re-transfused in less than ¼ of these cases, in contrast to the 80% re-transfusion rate for other specialties. The difficulty in predicting when haemorrhage will occur, lack of familiarity with equipment have been cited as impediments to its use.[19,20]

A intensive period of training seems to overcome some of these problems.[21] Serious consequences of amniotic fluid contamination and rhesus immunisation have not been reported. The use of a leucocyte depletion filter has been shown reduce the presence of squame cells and AFP to close to zero, even when a separate suction system for amniotic fluid was not used.[22]

In the current report on transfusion related injuries, there was one case of air embolism and 4 cases of hypotension associated with cell salvage.[11]

**Interventional radiology**

There have been several studies on the use of prophylactic interventional radiology for delivery when placenta accrete had been antenatally diagnosed. 3 studies showed a benefit but later ones showed little or no benefit.[23]

Although there is more evidence for the use of emergency procedures, practical considerations, including the need to transport haemodynamically compromised patients between maternity and radiology suites, restricts its use to a few patients in a few centres in the UK.

**References**

11. www.shot.org


Despite the increasing use of thromboprophylaxis, venous thromboembolism remains the number one medical cause of maternal death.

In response to this, the RCOG have revised their Green top guidelines again and the new version will increase the use of thromboprophylaxis further. One of the concerns is the increasing rate of obesity, which is epidemiologically a significant risk factor for VTE inside and outside pregnancy. In response the RCOG guidelines are advising routine thromboprophylaxis for women with a BMI >40 during antenatal admission and post delivery. Therefore there will be increasing numbers of patients on thromboprophylaxis prior to delivery: a challenge to the obstetric anaesthetists.

Despite a large number of new oral anticoagulants in clinical trial and some already licensed for use for thromboprophylaxis after hip and knee replacement, sadly these exciting new drugs will not impact on pregnancy due to concerns about teratogenecity. Thus low molecular weight heparin (LMWH) will remain the main thromboprophylactic and treatment agent in pregnancy for the foreseeable future. Occasional use of fondaparinux and warfarin will continue in those with heparin allergy.

The insertion of epidural and spinal anaesthesia in mothers receiving LMWH is a subject that has not been formally studied. Observational studies suggest that having a thromboprophylactic dose LMWH 8-12 hours pre insertion, along with a normal coagulation screen and adequate platelets is acceptable practice. Treatment dose LMWH required 24 hours cessation before insertion.

Fondaparinux has a longer half-life than LMWH and the American College of Chest Physicians recommend it is not used with regional anaesthesia til more data is acquired.
Session 5: Blood Issues
Use of the coagulation laboratory and point of patient testing in thrombosis & bleeding

Dr Wiebke Gogarten
Consultant Anaesthetist & Head,
Department of Anaesthesiology, Intensive Care and Pain Medicine, Harlaching Hospital,
Municipal Hospitals of Munich, Germany

Point of Care Laboratory Monitoring

Weibke Gogarten
Department of Anaesthesiology, Intensive Care and Pain Therapy
Harlaching Hospital
Municipal Hospitals of Munich, Germany

Bleeding History

- easy bruising
- epistaxis requiring cauterezation, lasting > 30 minutes
- menorrhagia
- oral cavity bleeding
- bleeding following dental extractions
- bleeding following invasive procedures
- bleeding during childbirth
- recent medication history
- bleeding requiring medical interventions, transfusions
- long standing history

Platelet Function Analyzer (PFA-100)

- Significantly abnormal in:
  - Glanzmann thrombasthenia
  - Bernard-Soulier syndrome
  - von Willebrand syndrome
- False negative results in several other platelet defects
- Affected by
  - platelet count
  - haematocrit
  - diet
  - aspirin
  - VWF levels
- Insensitive to ADP antagonists
- Correlation between closure time and perioperative bleeding?

Platelet Function in Patients treated with Aspirin undergoing CABG Surgery


Platelet Function and Preeclampsia (PFA 100)

Vincet et al., Br J Anaesth 2001; 87: 890-893
Multiplate Electrode Aggregometry

Abnormal platelet aggregation required more platelet transfusions
1.1 ± 1.2 U versus 0.3 ± 0.8 units*

Rahe-Meyer et al., Anesth Analg 2008; 107: 1791-1797

Aspirin Resistance: Comparison of Different Methods

In patients who are receiving antiplatelet drugs, we suggest against the routine use of platelet function assays to monitor the antithrombotic effect of aspirin or clopidogrel.

Rationale: The clinical significance of the assay result is uncertain as they have not been shown to identify patients at increased risk for perioperative bleeding.

Thrombelastography/Thrombelastometry

Hemostatic Function in Healthy and Preeclamptic Parurients

Fibrinogen, Platelet Numbers and Clot Strength

Fibrinogen Levels and Clot Strength in Thrombocytopenia

Lang et al., Anesth Analg 2009; 108: 751-758
Fibrinogen Levels and Major Postpartum Hemorrhage

Severe bleeding
Mild bleeding
Charbit et al., J Thromb Haemost 2006; 5: 266-273

Fibrinogen Levels and Chest Tube Drainage After CABG

Low drainage
High drainage
Blome et al., Thromb Haemost 2005; 93: 1101-1107

Preoperative Fibrinogen Substitution and Hemoglobin Level

20 patients, 2 g fibrinogen, CABG, no antifibrinolytics
Blood loss 565 ± 150 ml/12 h vs. 830 ± 268 ml/12 h*
Karlsson et al., Thromb Haemost 2009; 102: 137-144

Fibrinogen in Thoracic Aortic Aneurysm Repair

Retrospective vs. prospective fibrinogen 7.8 ± 2.7 g
Timing of blood loss assessment 5 minutes in group F, no definition in group A
Rahe-Meyer et al., J Thorac Cardiovasc Surg 2009; 138: 694-702

TEG-Guided High Fibrinogen Levels and Bleeding after Aortic Surgery

5.7 g fibrinogen
Rahne-Meyer et al., Br J Anaesth 2009; 102: 785-792

Standard Laboratory or Point of Care?

CABG surgery
Avidan et al., British J Anaesth 2004; 92: 178-186

Survival of Trauma Patients and Ratio of Plasma to RBC

246 trauma patients with massive transfusion
Knight et al., BJOG 2007; 130:1380-1387
At the present time there is probably insufficient evidence to indicate that the technology (thrombelastography) should be routinely adopted for the prediction or management of bleeding...

Summary

- Standard laboratory testing
  - Insensitive
  - Long turnaround time

- Point of care monitoring
  - Easy to use
  - Results rapidly available (20-30 minutes) (if not moved to a central laboratory)
  - Not recognized as valid and accurate by hematologists
  - Standardization, maintenance?
  - No consistent correlation with clinically significant bleeding
  - Advantage over conventional laboratory monitoring not proven
Session 6: Caesarean Section
General anaesthesia for Caesarean section: what’s new?

Dr David Levy
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A concise overview appeared in Anaesthesia and Intensive Care Medicine in 2007; an update on rapid sequence induction in obstetrics was published earlier this year in Current Opinion in Anesthesiology.

General anaesthesia: incidence and mortality
The Obstetric Anaesthetists’ Association’s National Obstetric Anaesthesia Database www.oaa-anaes.ac.uk revealed a rate of general anaesthesia (GA) for Caesarean section (CS) of 11% in 2005. There is an increasing likelihood that a trainee’s first experience of CS under GA will be an emergency case - a strong case for simulation-based team training.

GA is indicated for a majority of Category 1 CS, and other cases in which a regional block is absolutely contra-indicated, or has failed. The decision-making process for GA versus regional block is complex, and depends not only on clinical urgency, but the experience, attitudes and skill levels of anaesthetist and obstetrician - the quality of communication and teamwork is pivotal.

In the Report on Confidential Enquiries into Maternal Deaths (CEMD) in the United Kingdom for 1994-6, there were no deaths from GA: the solitary anaesthetic death followed combined spinal-epidural anaesthesia. However, a Confidential Enquiry into Stillbirths and Deaths in Infancy (CESDI) highlighted four cases from 1994-5 in which GA was associated with life-threatening hazards (two cases of anaphylaxis, two airway crises). Two deaths in the 1997-9 triennium were attributable to complications of GA - one case of aspiration in a postpartum woman re-anaesthetised for removal of abdominal packs, and a case of airway obstruction during change of tracheostomy tube in a woman who had sustained major haemorrhage and multi-organ failure. The 2000-2 Report described 6 deaths, and featured the unwelcome reappearance of oesophageal intubation as a cause of mortality. Although the Report for 2003-5 cited 6 anaesthetic deaths, none was attributable to airway management at CS.

1947: introduction of neuromuscular blocking agents
The introduction of neuromuscular block to inhalational obstetric anaesthesia allowed a lighter plane of anaesthesia, with respiration assisted to avoid any ‘suboxygensation’. In 1947, Gray described a technique of induction with nemithal (a thiobarbiturate), maintenance with cyclopropane, and the addition of d-tubocurarine 15 mg for neuromuscular block. The purported benefits were:

- Increased uterine contractility;
- Less sleepy neonates;
- Swifter maternal recovery.

Only one patient in the series required tracheal intubation, when the ‘ill-advised’ insertion of a pharyngeal airway provoked vomiting. Occasional signs of slight residual curarisation at the end of an unusually quick operation were treated with neostigmine and atropine.

1959: succinylcholine
12 years later, Hamer Hodges and colleagues published the results of a comparison of a thiopental, succinylcholine, nitrous oxide/oxygen technique with three anaesthetic regimens in which mothers received trichloroethylene, cyclopropane or diethyl ether. Babies born to mothers who had minimal anaesthesia facilitated by neuromuscular block had shorter times to regular breathing and active crying. Succinylcholine was the ‘relaxant of choice’ on account of its freedom from adverse neonatal effects: there were concerns about the extent of placental transfer of the only two alternatives, d-tubocurarine and gallamine. Resumption of maternal spontaneous respiration reassured the investigators that the baby would not be affected.

This landmark study led to the widespread replacement of mask and ether by intravenous induction, succinylcholine and tracheal intubation. The induction regimen has remained essentially unchanged for half a century, although certain aspects differed from contemporary practice:

- Women received pre-operative atropine, but no antacid;
- Anaesthesia was induced with steep head-up tilt;
- There was no attempt to relieve aortocaval compression;
- Pre-oxygenation was not undertaken;
- The drug doses were smaller: thiopental 200-250 mg, succinylcholine 50 mg;
- No application of cricoid pressure (it wasn’t described until 1961);
- The lungs were ventilated with oxygen by mask/Magill breathing system before intubation;
• There was ‘absolutely no justification’ for the administration of any agent other than N₂O between induction and delivery;
• 100% O₂ was given just before delivery. Although babies born to mothers anaesthetised with ether by facemask were undoubtedly sleepier than those whose mothers who had light anaesthesia and neuromuscular block, they were not necessarily compromised. It is open to debate whether in the hands of non-specialist anaesthetists the new technique was any safer. In the CEMD for 1964-6 technical difficulties with tracheal intubation were first mentioned, and the number of maternal deaths from anaesthesia rose steeply.¹³ Scott wrote of the introduction of neuromuscular block to obstetric practice ‘…if we had been honest, the attraction of the new method was the speed and ease with which we could present the intubated paralysed mother to the obstetrician. We did not advertise the increased incidence of death due to Mendelson’s syndrome and failed intubation resulting from suxamethonium and crush induction’.¹⁴ Cricoid pressure is used unquestioningly in the UK. The evidence base for the technique has been called into question by Priebe, who has argued that possibly more patients are endangered than protected, by interference with optimal airway management.¹⁵ In 1962, Crawford stipulated criteria to be satisfied in order that anaesthetic regimens for elective CS might be compared.¹⁶
• Women should be at or beyond the 36th week of pregnancy;
• Membranes must be intact; labour must not have been established;
• Neonatal weight must be greater than 2500 g.

He proposed categorising elective cases according to whether or not placental function and fetal health were considered satisfactory. Crawford also used thiopental and succinylcholine, N₂O and O₂ and regarded uterine relaxation as the only indication for halothane.¹⁷ He accepted that 4.5% of women having elective CS were awake at some point, and since ‘lightness of anaesthesia was the aim’, did not consider it justifiable to alter the technique. Some 25 years later, he wrote that uterine relaxation conferred by a volatile agent might actually improve fetal compromise to which uterine hyperstimulation had been contributory. It was by then recognized that a maternal stress response to excessively light GA is to the detriment of uteroplacental blood flow.¹⁸

1970: halothane
Moir demonstrated improved Apgar scores with N₂O/O₂ 50:50 + halothane 0.5% (inspired) compared to N₂O/O₂ 70:30 without halothane.¹⁹ He surmised that it was the higher FI₅ₐ₆ permitted by the use of halothane that was responsible for the better neonatal condition. Whereas two of 50 women who had unsupplemented N₂O/O₂ had clear recall, halothane assured lack of awareness and did not cause increased blood loss. This paper led the move away from maintenance with only N₂O/O₂. Halothane 0.8% (inspired) was dismissed because systolic arterial pressure decreased to <90 mmHg in a quarter of patients, although blood loss was no greater.

Moir’s paper was reproduced as a BJA 'citation classic' in 1998. In her commentary, Reynolds described GA as 'innocuous and reversible' for the baby.²⁰

In the absence of asphyxia, the Apgar score at one minute does not have prognostic significance.²¹ Provided the following are ensured, GA per se is not harmful to the baby:
• Maintenance of maternal oxygenation;
• Maintenance of maternal normocapnia for pregnancy;
• Strict avoidance of aortocaval compression;
• Uterine incision-delivery interval is minimised;
• A paediatrician is present to support neonatal ventilation.

GA for incidental surgery in pregnancy for which local anaesthesia is not feasible (e.g. drainage of a breast abscess) should not be withheld for fear of adverse effects on the baby or uterus. Lack of fetal heart beat-to-beat variability with normal baseline rate should not be interpreted as evidence of fetal compromise.²²

2000: opioids

In an investigation of the stress response to tracheal intubation and surgery, the supplementation of thiopental 4 mg kg⁻¹ at induction by alfentanil 10 μg kg⁻¹ was compared with saline.²³ Women in the alfentanil group had smaller increases in arterial pressure and heart rate. Mean plasma norepinephrine concentration was lower in the alfentanil group after tracheal intubation but at time of delivery was similar to that of the saline group. Apgar scores were lower at 1 minute in neonates whose mothers had received alfentanil, although not at 5 minutes, and neonatal adaptive capacity scores (NACS) were similar at 15 and 120 min. There was no difference in mean umbilical artery (UA) pH between groups. The slightly greater UA PO₂ in the alfentanil group was attributed to decreased oxygen consumption. Mean cord UA norepinephrine concentration in the alfentanil group was approximately half that in the control group. It is not know whether attenuating the neonatal stress response is good or bad: high sympathoadrenal activity may promote neonatal adaptation to extra-uterine life.
Remifentanil has been studied as an adjunct to GA regimens, and numerous case reports attest to haemodynamic stability in women with cardiac disease.

100% O$_2$

Both isoflurane and enflurane in oxygen without N$_2$O have been shown to provide satisfactory anaesthesia for CS. 50% N$_2$O/O$_2$ + 0.6% isoflurane (inspired) has been compared with 100% O$_2$ + 1.2% isoflurane for emergency CS. 100% O$_2$ resulted in increased mean umbilical venous (UV) PO$_2$, but no significant differences in UA pH. There was less need for neonatal oxygen and IPPV although a greater need for suction in the 100% O$_2$ group. The authors’ widely quoted claim that this amounted to ‘less resuscitation’ is open to question. Any improvement in fetal oxygenation from 100% O$_2$ might be limited by the prevailing pathophysiology (e.g. placental abruption). If N$_2$O is omitted, it is important that overpressure is used to accelerate attainment of the desired end-tidal vapour tension.

Administration of 100% O$_2$ to the mother is associated with increased fetal oxygen saturation and UV oxygen content. However, maternal hyperoxia during regional anaesthesia causes increased free radical activity. Free radicals cause depletion of intrinsic antioxidant systems and are potentially detrimental to a neonate’s ability to withstand subsequent insults. The relationship between free radical formation and neonatal outcome is uncertain.

The significance for neonatal well-being of minor differences in UA pH values between anaesthetic regimens is uncertain. Severe acidaemia (umbilical artery pH <7.0) with 1-minute Apgar ≤3 and 5-minute Apgar <7 is predictive of serious neonatal morbidity. It has been argued that UA standard base excess (SBE) is a better measure of fetal well-being than pH (which reflects both metabolic and respiratory acidosis). Respiratory acidosis can occur rapidly without hypoxic injury, whilst a large negative SBE might be indicative of prolonged oxygen debt and anaerobic metabolism. The validity of the Neonatal Neurologic and Adaptive Capacity Score (NACS) has been questioned. In short, there is insufficient evidence upon which to recommend an optimal $F_{I/O_2}$.

**Which i.v. agent?**

There is little evidence that any agent is better than thiopental. According to the respective data sheets, the dose of thiopental should be limited to 250 mg: propofol is explicitly contra-indicated for use in obstetrics. It has been shown in sheep that peak brain concentrations are reached earlier with thiopental compared to propofol. At CS in women, propofol 2 mg kg$^{-1}$ attenuated maternal haemodynamic and catecholamine responses to intubation more effectively than thiopental 4 mg kg$^{-1}$, although neonatal Apgar scores, NACS, cord blood gas tensions and catecholamine concentrations were similar. An OAA Controversies motion in 2003 was ‘Propofol should be the induction agent of choice for caesarean section under general anaesthesia’. There was overwhelming opposition to the motion amongst delegates before and after the debate.

There were no instances of recall, unpleasant dreams, or dysphoria when ketamine induction was followed by N$_2$O/O$_2$. It seems that emergence phenomena are avoided when awakening occurs some time after an induction dose of ketamine has been distributed. Investigations of midazolam were prompted by perceived potential benefits of its cardiovascular stability and amnesic effect. 1 minute Apgar scores gave cause for concern in one study but not another (in which a larger dose was studied). Small doses of midazolam are certainly not contra-indicated for sedation during awake fibre-optic intubation. Neonatal times to sustained respiration were shorter with etomidate 0.3 mg kg$^{-1}$ compared to a rather small dose of thiopental (3.5 mg kg$^{-1}$).

**Inhalation induction?**

Both sevoflurane and desflurane have been evaluated for maintenance of GA for CS, with no apparent adverse maternal or neonatal effects. These agents offer the advantage of more rapid approximation of alveolar to inspired tensions as the anaesthetic effect of the i.v. induction dose declines. Maternal recovery was not demonstrably faster with either of the newer agents (sevoflurane was compared with isoflurane, desflurane with enflurane).

Crawford argued that using halothane for induction risked a dangerous tissue accumulation of the drug, and the threat of inadequate uterine contraction. He pointed out that the uterine response to discontinuing halothane was dependent upon the duration of its administration.

Sevoflurane has a conspicuously low propensity for airway irritation and been use successfully for inhalational induction of anaesthesia for CS in the absence of vascular access and in status asthmaticus. The smooth, rapid onset and lack of coughing or vomiting suggest that the technique does not necessarily incur an increased risk of aspiration.

The following approach is suggested:

- 20-30° head-up tilt;
- breathing system primed with sevoflurane 8% in N$_2$O/O$_2$ (2:1);
- tidal respiration;
i.v. canulation after loss of consciousness;
neuromuscular blockade, tracheal intubation.

Non-depolarising neuromuscular block at induction
Awake fibre-optic intubation should be considered if the history or evaluation of the airway suggest that tracheal intubation might be difficult. The principal reason for the continuing use of succinylcholine, the rapid recovery of maternal neuromuscular function in the event of an airway crisis, was never originally considered potentially advantageous. It has been estimated that a serious reaction to succinylcholine might be expected once in every 4000 inductions. It has been shown to produce acceptable intubating conditions after thiopental 6 mg kg\(^{-1}\) within 90 s, without untoward neonatal effects. The onset of rocuronium 0.6 mg kg\(^{-1}\) is even faster than succinylcholine 1.5 mg kg\(^{-1}\). Doubling the intubating dose of rocuronium would, predictably, further shorten the onset time, but incur a duration of action excessive for CS. It might be expected that the mass of rocuronium transferred across the placenta would increase in proportion to the maternal dose, but it is uncertain whether the fraction of 1.2 mg kg\(^{-1}\) transferred in the event of protracted surgical delivery would be innocuous. In the event of failed tracheal intubation, it might be argued, controversially, that inserting a laryngeal mask airway (LMA) and ventilating the lungs with cricoid pressure applied is no less safe overall than using succinylcholine and attempting to proceed with inhalational anaesthesia by spontaneous respiration. Unlike the case with succinylcholine, optimal conditions will be sustained for airway management and maintenance of oxygenation, and vomiting cannot occur. Maintenance of a slight head-up tilt will allow gravity to reduce the risk of reflux of gastric contents. The evolving role of the LMA in obstetrics has been reviewed. The ProSeal™ LMA incorporates a second tube, which makes a sealed junction against the upper oesophageal sphincter, allowing continuity with the gastro-intestinal tract and isolation from the airway. Gastric insufflation during IPPV is minimised, and it is possible to pass a gastric tube. There are reports of use of the ProSeal™ LMA a rescue airway at CS. In the most unlikely event of airway obstruction at the glottis causing a ‘can’t intubate, can’t ventilate’ scenario, a better chance of successful cricothyrotomy will be afforded by profound neuromuscular block.

Sugammadex
The rocuronium antagonist sugammadex was launched in Europe in late 2008. In a non-obstetric population, recovery evoked by sugammadex 16 mg kg\(^{-1}\) (administered 3 min after rocuronium 1.2 mg kg\(^{-1}\)) was swifter than the same degree of spontaneous recovery from succinylcholine. Data on placental transfer of sugammadex are extremely sparse, and limited to animals: the manufacturer has indicated that <2-6% is transferred in rat and rabbit populations. There is no information about placental transfer of the sugammadex-rocuronium complex. However, adverse fetal effects of sugammadex-encapsulated rocuronium are unlikely.

Awareness and uterine relaxation
The tenet of Crawford's opposition to halothane was the feared effect on uterine tone. A dose-dependent reduction in uterine muscle contractility has been shown in vivo with sevoflurane and desflurane. This finding concords with earlier studies of the halothane, enflurane and isoflurane. However, myometrial responsiveness to Syntocinon at different end-tidal vapour tensions has never been studied in vivo. Experience with ex utero intrapartum treatment (EXIT) of babies with potential airway obstruction has provided evidence that the effect of deep inhalational anaesthesia with modern, insoluble agents is rapidly reversible. 2 MAC (end-tidal) vapour concentration (nevertheless requiring supplementation with glyceryl trinitrate) has been used to relax the uterus whilst allowing fetal oxygenation by continued uteroplacental perfusion. An OAA Controversies debate in 1996 explored whether awareness during GA for CS is negligent. If the uterus is failing to contract, haemorrhage is life-threatening and the volatile agent is discontinued, N\(_2\)O should nevertheless be continued, and anaesthesia maintained by opioids or benzodiazepines. Ketamine is perhaps the ideal agent in this situation. I suggest that in 2009 with inhalational agent monitoring now universally available, the risk of awareness in obstetric anaesthesia should have been consigned to history. A prospective study from Australia and New Zealand found that the awareness remains a significant but avoidable complication of GA for CS. To maintain bispectral index (BIS) values <60 (for ‘adequate’ depth of anaesthesia) at CS, end-tidal vapour concentration >0.75 MAC (+ 50% N\(_2\)O) has been recommended.

Pre-eclampsia
Uncorrected coagulopathy, or symptoms consistent with impending eclampsia (severe headache or visual disturbance) are potential maternal indications for GA. Potential benefit (smaller UA base deficit) has
been shown for compromised fetuses when mothers received GA as opposed to single-shot spinal anaesthesia.\textsuperscript{70} Prior communication with a paediatrician is essential in order that preparation can be made for antagonism of opioid or provision of ventilatory support for the neonate.

Laryngeal oedema is a real risk – beware the patient with recent voice change. Any dubious notion of light GA for the baby’s benefit should be overridden by efforts to protect the mother’s cerebral circulation.\textsuperscript{71} Although the onset and duration of succinylcholine are unaffected by therapeutic serum magnesium concentrations, the durations of action of all non-depolarising drugs are potentiated.

- Have a low threshold for direct arterial pressure monitoring;
- Attenuate the pressor response to intubation with alfentanil 10 µg kg\(^{-1}\) or remifentanil 2 µg kg\(^{-1}\) before rapid sequence induction with a generous dose of thiopental;
- Peripheral nerve stimulator essential to ascertain the degree of neuromuscular block;
- Before extubation, consider additional antihypertensive therapy (e.g. labetalol in 10-20 mg increments) to avert a dangerous pressor response.

Any patient whose larynx was noted to be swollen at laryngoscopy, or in whom intubation was traumatic is at particular risk of laryngeal oedema. Postoperative care must be undertaken in an ICU or high-dependency area with an anaesthetist immediately available. Midwives should be alerted to the ominous significance of stridor.

### Placenta praevia & accreta

UK\textsuperscript{72} and American\textsuperscript{73} retrospective studies and a small prospective randomised trial in Korea\textsuperscript{74,75} have compared regional and general anaesthesia for CS with placenta praevia. The commonly held obstetric view that placenta praevia necessarily dictates GA is not supported. Regional anaesthesia was associated with reduced estimated blood loss and transfusion requirements. However, risk factors for placenta accreta (anterior placenta praevia in women over 35 who have had previous CS\textsuperscript{s})\textsuperscript{76} flag up a particularly high risk of massive haemorrhage. GA with provision for postoperative ICU admission might be considered prudent.\textsuperscript{77}

### Conclusion

Although the CS rate has increased, use of GA has declined and trainee hours of work have been decreased. Most anaesthesia trainees have minimal experience of GA in obstetrics, and should take every opportunity to practise the technique. Increased presence of consultant anaesthetists on obstetric units should help optimize training opportunities. Training adjuncts, including medical simulation, may have a useful role.

GA for operative obstetrics, especially elective cases, is not inherently unsafe. If trainees are instilled with excessive anxiety about the potential hazards of GA, complications (especially failed intubation) will remain prevalent.\textsuperscript{77}

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Session 6: Caesarean Section
Managing the difficult airway in obstetrics

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Difficult and failed intubation in obstetrics
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Definitions
Difficult intubation – No consensus
• Grade 3 or 4 CCL
• More than one attempt

Incidence 1 in 30

Failed Intubation
Inability to intubate the trachea and subsequent abandonment as means of airway management

Incidence of Failed Intubation

<table>
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<th>Study</th>
<th>Location</th>
<th>Year</th>
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<tr>
<td>Lyons</td>
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<td>1985</td>
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<td>Hawthorne et al.</td>
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<td>1996</td>
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<td>Barneodo &amp; Jenkins</td>
<td>UK</td>
<td>2000</td>
<td>1 in 249</td>
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<td>Rahman &amp; Jenkins</td>
<td>UK</td>
<td>2005</td>
<td>1 in 238</td>
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<tr>
<td>McDonnell et al</td>
<td>AUSTR</td>
<td>2008</td>
<td>1 in 274</td>
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<tr>
<td>Rocke et al.</td>
<td>S Africa</td>
<td>1992</td>
<td>1 in 750</td>
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</table>

Failed intubation in obstetrics
Incidence higher than in general population
• Anatomical & physiological changes in pregnancy
• Failed intubation drill instituted more readily

Anatomical/physiological changes
• Increased TBW - swelling of pharynx and larynx
• Change in Mallampati score during pregnancy
• Laryngeal oedema – with/without pre-eclampsia
• Increased thoracic diameter & enlarged breasts
• Full dentition or awkward maxillary incisors

Failed intubation drill instituted more readily
• Emergency out of hours anaesthesia
• Trainee anaesthetists (less experience)
• Changing patient population

What are the consequences of failed intubation?
What are the consequences?

- Safe outcome if protocols followed
- Examples of protocol violation
  - Airway not pre assessed
  - Repeated muscle relaxants
  - Repeated intubation attempts
  - Failure to use appropriate equipment
  - Poor follow up

CEMD: 2000 – 2002 Anaesthesia Deaths
6 + (1) - All GA, all emergencies, all trainees

- Oesophageal intubation n = 3
  - SHO anaesthetists, equipment unavailable/not used, Auscultation OK
- Hypoventilation; n = 2
  - Trainees, isolated sites, help late, Obesity
  - Capnography not used in 1
  - Aspiration/Failed intubation; n = 1
  - Anaphylaxis; n = 1

How to reduce the incidence of failed intubation and its consequences?

**Individual Issues**
Pre operative assessment Planning and Procedures for
- Anticipated difficult airway
- Unanticipated difficult airway

**Organisation issues**

Pre operative Airway Assessment
How?
When and by whom?
- Duty Anaesthetist - ? Every woman/ anaesthetic intervention
- Midwives/obstetrician
- Antenatal referral (education)

How?
History
Notes
Bedside tests
- Eyeball test (obvious difficulty)
- Difficult laryngoscopy
- Difficult face mask
- Difficult LMA

Recognition of difficult laryngoscopy
- Mouth opening
- Mallampati class
- Jaw protrusion-
- Thyromental
- BMI - at booking
- Pathological airway - airway oedema due to PET

BMI
airway oedema

OAA: Three-day Course on Obstetric Anaesthesia and Analgesia, London, 9 – 11 November 2009

72
Pre operative Airway Assessment
Which tests?
When and by whom?
- Duty Anaesthetist - ?
- Every woman/anaesthetic intervention
- Midwives/obstetrician
- Antenatal referral (e.g. obese)

How to reduce the incidence of failed intubation and its consequences?

Pre operative assessment Planning and procedures for
- anticipated difficult airway
- unanticipated difficult airway

Anticipated difficult airway
- Antenatal team planning
e.g. help from colleague
Obstetric plan may need to be changed
- Regional anaesthesia is safe option

PLAN A - Regional Anaesthesia for C/S
Good reasons
- Anaesthetic expertise
- Patient preference
- Sedation uncommon
- Airway accessible
Problems
- May be contraindicated
- Inadequate block
- High/total spinal
PLAN B must always be worked out in advance

Anticipated difficult airway
PLAN B Options
- Awake intubation
- GA with supraglottic device +/- intubation
- Awake look and GA
- Local infiltration anaesthesia

Awake fibreoptic intubation skills in obstetric patients: a survey of anaesthetists in the Oxford region.
Choice of anaesthetic technique if regional anesthesia fails in a patient with a known difficult airway
Awake intubation 75/83(88%)
GA with LMA 5/83
GA with mask and airway 1/83
LA infiltration 1/83

Elective Caesarean section
Plan A Combined spinal-epidural
Plan B Awake Fibreoptic Intubation (AFI)

FAILED PLAN A
AFI following failed RA
for C- section in a parturient with Still’s disease
Popat M, Chipa J, Russell R
Eur J Anaesth 2000; 17: 211-14
**Flexible fibreoptic laryngoscope - advantages for awake intubation**

- Flexibility/continuous visualisation
- Ability to apply local anaesthetic
- Well tolerated by patients
- High success rate

**AFI in Obstetrics: Practical issues**

- Patient related
  - Regurgitation and aspiration
  - Nasal bleeding
  - Fetal/neonatal welfare
- Environment related
  - Equipment, expertise

**Practical Issues: Patient related**

1. Patient preparation
2. Position of patient
3. Monitoring & oxygenation
4. Sedation
5. Intubation route
6. LA of upper airway

**Confidence in performing AFI in OB patients**

| All anaesthetists | 30/68 (44%) |
| OB anaesthetists  | 12/31 (40%) |

Awake fibreoptic intubation skills in obstetric patients: a survey of anaesthetists in the Oxford region

**1. Patient Preparation**

- Psychological
- Pharmacological
  - Aspiration prophylaxis
  - Antisialogogue e.g. glyco 0.2mg iv

**2. Position of patient**

Advantages of sitting position
- Reduced risk of aspiration
- Venacaval occlusion prevented
- Topical anaesthesia better tolerated
- Airway obstruction minimal

**3. Monitoring and oxygenation**

**4. Sedation**

Benefits
Patient comfort, amnesia and anxiolysis
Risks
- Effect on laryngeal competence
- Airway obstruction
- Effect on baby
5. Oral Intubation route

<table>
<thead>
<tr>
<th>Author</th>
<th>Indication</th>
<th>Route guide</th>
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<tr>
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<td>Ankylosing spondylitis</td>
<td>Bermen</td>
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<td>D’Alessio 1995</td>
<td>Pre-eclampsia / HELLP</td>
<td>Williams</td>
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<td>Stills disease</td>
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<tr>
<td>Popat 2001</td>
<td>Previous failed intubation</td>
<td>Safe Bite</td>
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</table>

6. Local anaesthesia of upper airway

- Nerve blocks
- Nebulised LA
- Topical anaesthesia

My recipe

- 2% lignocaine gargle
- Spray as you go -
  4\% via epidural catheter
- Back up
- Cricothyroid puncture

1. Should all Consultant Obstetric Anaesthetists’ be confident in performing an AFI?
2. Can an AFI be performed rapidly for category 1 C/S

How to reduce the incidence of failed intubation and its consequences?

Pre operative assessment
Planning and procedures for
- anticipated difficult airway
- unanticipated difficult airway
Planning and procedures for Unanticipated difficult airway

• Delivering a ‘safe obstetric’ GA
• Following agreed ‘failed intubation drill’

Delivering a ‘safe obstetric’ GA

• Preparation
• Positioning the mother
• Pre oxygenation : at least 3 min
• Perfect technique

Preparation

At the start of every shift
• Airway equipment
• Drugs
• Help – Who? How?

Airway equipment Routine

• Laryngoscope - short handle & long blade
• ETT - 7.0 mm or smaller
• Gum elastic bougie
• Laryngeal mask airway
• Check cricothyroidotomy equipment

Delivering a ‘safe obstetric’ GA

• Preparation
• Positioning the mother
• Pre oxygenation : at least 3 min
• Perfect technique

Positioning the mother

• Head & neck
• Breasts
• Hands
• Thoracic tilt

Ramping for obese patients

Table tilt

Wedge
Delivering a ‘safe obstetric’ GA

- Preparation
- Positioning the mother
- Pre oxygenation: tight mask
- Perfect technique

Perfect technique

1st attempt = best attempt

Cricoid pressure (correct technique)
Induction agent (Give enough)
Suxamethonium (Give enough and wait to act)
Good laryngoscopy technique
Confirmation of intubation (visual, CO2, Auscultate)

Difficult Airway Society Guidelines

- Consensus
  (discussion & feedback)
- Evidence
- Experience


Routine induction
Rapid sequence induction

FAILED INTUBATION

- DECISION - tracheal tube cannot be passed and the lungs must be ventilated
Failed intubation
Ventilation successful

Decision to continue anaesthesia or awaken
Indications for continuing
Definite Maternal (haemorrhage, cardiac arrest)
Definite RA contraindicated e.g. coagulopathy & expertise not available for awake intubation
May be Sudden severe fetal distress (cord prolapse, abruption)

In all other cases, Awaken & perform surgery under RA

Some thoughts

- LMA before bag and mask
- If LMA is positioned well
  Should surgery always proceed
- If so
- Should LMA be used for intubation?

Rapid sequence induction

*Why not plan B?*
- Cricoid pressure and LM devices
- Short apnoea time

Failed Intubation
Failed Ventilation (face mask, LMA)
Increasing hypoxaemia

Maintenance of oxygenation

Use face mask, oxygenate and ventilate
- If spontaneous ventilation is not adequate
  Consider mechanical ventilation or intubation

Preoperative surgery and awake patient may proceed
- LMA™ or Proced LMA™ - if condition immediately escalating

What needle do you use?

- 13G Ravussin
- 6.0F Cook
- 14G Venflon

What do you connect the needle to?

- Manujet 3
Techniques for CICV

Surgical cricothyrotomy

American College of Surgeons. ATLS course manual, 6th Ed 1997

New(er) Toys

Intubation
Airtraq
GlideScope
Pentax Airway scope
Video laryngoscopes
(McGrath, Storz)
Optical stylets

Ventilation
Proseal
LMA supreme
I–gel
Ventilation & intubation
LMA C Trach®

Proseal LMA™

High seal pressure
Separates glottis & oesophagus
Prevent aspiration of regurgitated material
Oro-gastric tube
Better for IPPV

Learning curve

LMA Supreme
Single use
Shape = ILMA
Function = Proseal LMA

Intubating LMA

How to reduce the incidence of failed intubation and its consequences?

Individual Issues
Pre operative assessment
Planning and procedures for
• Anticipated difficult airway
• Unanticipated difficult airway

Organisation issues

OAA: Three-day Course on Obstetric Anaesthesia and Analgesia, London, 9 – 11 November 2009

Johnson et al. Anaesthesia 2000

GA for caesarean section

Johnson et al. Anaesthesia 2000
**Incidence of Failed Intubation**

<table>
<thead>
<tr>
<th>Name</th>
<th>Location</th>
<th>Year</th>
<th>Incidence</th>
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<tbody>
<tr>
<td>Lyons</td>
<td>UK</td>
<td>1985</td>
<td>1 in 300</td>
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<tr>
<td>Hawthorne et al.</td>
<td>UK</td>
<td>1996</td>
<td>1 in 250</td>
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<tr>
<td>Barnardo &amp; Jenkins</td>
<td>UK</td>
<td>2000</td>
<td>1 in 249</td>
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<tr>
<td>Samsoon &amp; Young</td>
<td>UK</td>
<td>1987</td>
<td>1 in 280</td>
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<tr>
<td>Rocke et al.</td>
<td>S Africa</td>
<td>1992</td>
<td>1 in 750</td>
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</table>

**Technical skills**

Can be taught on non obstetric patients

Non technical skills (attitudes/behaviour)

Need to be taught on delivery suite

e.g. Communication, decision making, situation awareness

How?

Manikins/Simulators

Role play

**Competency Based Training**

- knowledge
- skills
- attitude & behaviour

**Role of Consultant Anaesthetists**

**Service**

Antenatal planning

Back up for failed intubation

Skills – Awake intubation, Alternative techniques

**Training**

Good role models – safe GA/failed intubation drills

Educating others – obstetricians, midwives

**OAA: Three-day Course on Obstetric Anaesthesia and Analgesia, London, 9 – 11 November 2009**
We cannot eliminate general anaesthesia for obstetrics
We cannot eliminate failed intubation
Safe outcome is ensured by appropriate management of
Anticipated and unanticipated difficult airway
The physiological changes of pregnancy, including an initial gradual increase in cardiac output, followed by the development of increasing aortocaval compression in the third trimester, as well as co-morbidities such as preeclampsia, have generated considerable research into maternal haemodynamics. The use of the pulmonary artery catheter allowed a better understanding of the physiology of the healthy parturient and the haemodynamics of preeclampsia, including the effects of epidural analgesia in labour. Early use of dye dilution techniques gave clinicians insight into the haemodynamic changes during spinal anaesthesia for caesarean section. More recently, minimally invasive pulse wave form analysis has been used in the assessment of haemodynamic changes during this procedure.

Heart rate and blood pressure are appropriately used as surrogate markers of maternal cardiac output in all routine obstetric anaesthesia deliveries, and in most of the clinically valuable obstetric anaesthesia research to date. Intra-arterial monitoring provides a useful indicator of beat by beat changes in the unstable patient. During regional anaesthesia for caesarean delivery, maintenance of baseline maternal blood pressure, using phenylephrine, has been shown to produce the closest to zero umbilical arterial base deficit, the currently accepted short-term marker of neonatal wellbeing. This is despite the fact that phenylephrine reduces maternal cardiac output. The effectiveness of phenylephrine may be related to the limited susceptibility of the uterine artery to the vasoconstrictive effects of alpha agonists in advanced pregnancy. However, the maximum change in cardiac output has been shown to correlate better with uteroplacental blood flow than upper arm blood pressure. The maintenance of blood pressure and maternal cardiac output are therefore both important for maternal safety and comfort, and fetal wellbeing.

For clinical management and research purposes, there has been an increasing awareness of the potential complications of invasive monitoring. In addition the importance of the effects of fluid and vasopressor administration on flow rather than pressure, is now recognised in the non-obstetric population. In particular, central venous pressure and pulmonary wedge pressure are unlikely to predict the response to fluid administration, and pulse pressure variation and stroke volume variation may be better indicators of fluid resuscitation. These factors have led to a resurgence of interest in minimally invasive techniques of cardiac output monitoring.

Non-invasive methods of cardiac output measurement used in obstetric anaesthesia, have provided valuable information on maternal and fetal wellbeing and haemodynamics in the critical care setting and during regional anaesthesia for caesarean section. These techniques include transthoracic echocardiography, transoesophageal echocardiography, transoesophageal suprasternal aortic and uterine artery Doppler ultrasound techniques, and transthoracic and whole body electrical bioimpedance. All these methods have disadvantages, including expense, the requirement for user education, movement artefact, and in the case of bioimpedance techniques, the potential for inaccuracy in terms of absolute cardiac output values in advanced pregnancy and in the presence of increased lung water. None provide beat by beat data.

Arterial pulse wave form analysis methods are attractive to the obstetric anaesthetist, in that they provide beat by beat assessment of cardiac output, and could be used both in critical care monitoring (e.g. in complicated severe preeclampsia) and for research purposes (e.g. the effects of fluids, vasopressors and oxytocic drugs), in the labouring patient or during anaesthesia. Of crucial importance in the acceptance of these monitors are the precision and reliability of the employed algorithm in following changes in cardiac output (including in the setting of rapidly changing systemic vascular resistance), and the ability to predict ventricular preload response, through the derivation of fluid responsive parameters. When interpreting published data, usually involving Bland and Altman’s recommendation for the use of bias and precision statistics, the reader should bear in mind that in view of the +/- 10-20% accuracy of thermodilution, limits of agreement of up to +/- 30% between the new and the accepted technique are generally regarded as acceptable. Currently commercially available methods consist of calibrated devices (LiDCOplus [LiDCO, Cambridge, United Kingdom] and
PiCCOplus [Pulsion Medical Systems, Munich, Germany], and the uncalibrated Vigileo monitor (Edwards Lifesciences, Irvine, California).12

The PulseCO algorithm employed in the LiDCOplus monitor requires only a peripheral arterial and venous line and calculates stroke volume by analysis of the arterial blood pressure trace using a pulse power algorithm. This a three step process, involving initial transformation (compliance correction) of the arterial pressure into a volume-time wave form, followed by derivation of a nominal stroke volume and beat duration, employing autocorrelation, and finally calibration/scaling of the stroke volume with a lithium indicator dilution curve measurement of cardiac output, which is comparable to intrapulmonary thermodilution.12 It is important to note that the PulseCO pulse wave form algorithm described in 2001 used the first harmonic of the blood pressure wave form and related this mathematically to the cardiac output; this bears no relation to the commercially employed algorithm.13 Despite concerns regarding the ability of this method to respond to acute changes in SVR, it is noteworthy that this original algorithm was employed successfully to examine the detailed time based effects of a 5ug IV dose of epinephrine.14

Several publications have shown the ability of the subsequently commercially employed pulse power algorithm to trend changes in stroke volume accurately, avoiding the requirement for frequent recalibration.15,16 Three studies have demonstrated acceptable agreement11 between the LiDCOplus-derived cardiac output and Lithium dilution16,17, or thermodilution18 in the setting of systemic vascular resistance changes of up to 200%. Only one published investigation in obstetric anaesthesia has examined cardiac output trend changes during spinal anaesthesia for caesarean section in patients with severe preeclampsia, as well as acute responses to phenylephrine and oxytocin.19 This study elicited considerable debate,20 but current knowledge suggests that this monitor is valuable in the setting of acute changes in systemic vascular resistance. Recently this technology has been used to investigate the optimal combination of spinal bupivacaine and sufentanil, and phenylephrine infusion, in order to preserve optimal maternal haemodynamics.4 A small dose of bupivacaine and opioid, in combination with a low dose phenylephrine infusion, gave the best haemodynamic stability in the 4 combinations studied, with minimal maternal symptoms. Induction of spinal anaesthesia was associated with a decrease in blood pressure and, interestingly, an increase in cardiac output. In a recent randomised trial, cardiac output changes were estimated by transthoracic bioimpedance changes and the LiDCCOpus monitor in the same patient, in order to corroborate the data.5 Once again, spinal anaesthesia for elective caesarean section was associated with a reduction in systemic vascular resistance, and a partial compensatory increase in cardiac output. A bolus of 80µg phenylephrine administered in response to a 20% decrease in mean arterial pressure, reduced maternal cardiac output (but not below baseline values), and decreased cardiac output when compared with 10mg ephedrine. Cardiac output changes correlated with heart rate changes following vasopressor administration, emphasizing the importance of heart rate as a surrogate indicator of cardiac output. Co-administered phenylephrine obtunded hemodynamic responses to oxytocin. Using suprasternal Doppler flow measurements, a dose dependent reduction in cardiac output has been demonstrated, in parturients receiving an infusion of phenylephrine at 100µg per minute during SA for CS.21 These studies suggest that doses of phenylephrine which induce increases in blood pressure to above baseline values, and sinus bradycardia, result in depression of maternal cardiac output, and should be avoided.

The LiDCCOpus monitor has recently been shown to predict fluid responsiveness in mechanically ventilated patients by providing reliable measurements of pulse pressure variation and stroke volume variation.5 This attribute could be useful in the management of patients with complicated severe preeclampsia, in whom pulmonary artery catheterisation has a significant complication rate.2

The recently developed PiCCO system employs an algorithm which analyses the systolic component of the arterial wave form. The calibration method, transpulmonary thermodilution, requires cannulation of a proximal artery and a central vein, which is problematic for obstetric anaesthesia research, but may be feasible for critical care management. An acute rise in systemic vascular resistance induced by phenylephrine, has resulted in increased bias when compared with thermodilution in cardiac surgical patients.22

The Vigileo monitor uses a specialised arterial transducer which is connected to a monitor that samples the pressure recording at a frequency of 100Hz, and analyses wave form characteristics by a multivariate polynomial equation. Patient demographic characteristics are used to estimate inter-patient variability and thereby reduce bias. It does not require calibration, and any artery can be used. There is currently little published comparative data. Moderate accuracy has been shown in comparisons with thermodilution. However, the administration of
phenylephrine has been shown to increase the cardiac output measurement using the Vigileo monitor when transpulmonary thermodilution showed a decrease in output, in cardiac surgical patients. This would suggest that the monitor may not be suitable for the study of rapid haemodynamic changes associated with obstetric anaesthesia.

Pulmonary artery catheterisation has only a limited application in the management of complex cardiac defects or multiple organ failure in the parturient. Less invasive cardiac output monitors will have an important role to play, both in the haemodynamic management of parturients with co-morbidities, and in research into the haemodynamics of healthy and critically ill mothers. The prediction of fluid responsiveness using clinical assessment and pulse wave form monitors, may prove to be of greater value than the measurement of filling pressures.

Heart rate and noninvasive blood pressure measurement, as well as communication with the awake patient during regional anaesthesia, remain the most important monitors for the obstetric anaesthetist. It is however also important that the anaesthetist is constantly mindful of the effect of his/her interventions on both flow and pressure. In addition, research in the area of cardiac output contributes to a knowledge of the mechanism of spinal anaesthesia, and to the correct choice of vasoppressors when the patient is haemodynamically unstable.

References

20. Pauca AL: Pressure wave analysis is useful to understand the pathophysiology of preeclampsia but perhaps not the rapid changes during cesarean delivery. Anesthesiology 2008; 108: 773-4
Session 6: Caesarean Section

Spinal anaesthesia for caesarean section – dose, position & baricity

Marc Van de Velde, MD, PhD.
Professor of Anesthesiology, University Hospitals Gasthuisberg, Leuven, Belgium

Introduction
The spread of local anaesthetic solution within the spinal space can be modified by various factors or depends on various non-modifiable factors which need to be taken into consideration when designing an optimal neuraxial block strategy. The present manuscript reviews some of these factors in parturients undergoing a Caesarean delivery.

When local anaesthetic solutions are injected into the subarachnoid space the uptake of the anaesthetic determines which neuronal functions are affected, while the duration of the effect is determined by the elimination process of the local anaesthetic solution. The distribution of the anaesthetics within the spinal space determines the extent of altered neuronal function. To evaluate the extent of distribution ideally we should measure cerebrospinal fluid concentration of local anaesthetic. This is however in humans impractical and unethical to do. Therefore the distribution is measured by determining the spinal segmental level of anaesthesia.

To establish the spinal segmental level of anaesthesia various methods can be used: cold, pinprick or touch, but also heat and transcutaneous electrical nerve stimulation (1,2). Block to cold sensation extends usually higher then pinprick which extends usually higher then block to touch. Although the mean difference between touch and cold is 3 segments, while the mean difference between touch and pinprick is 2 segments (1). However large patient variability exists and many authors conclude that pinprick and cold are incapable to reliably detect a sufficient anaesthetic block (1,2,3).

Many factors may influence the distribution of local anaesthetic in the intrathecal space (table 1) (4). In the present manuscript, we will review some of these factors in relationship to spinal anaesthesia for Caesarean section.

Height and weight
Greene noted that common sense and clinical experience tell us that patient height and weight influence the distribution of local anaesthetic (4). However, the data supporting this is limited. McCulloch et al. reported in non-obstetric patients that increased weight and obesity increased block height (5). But in obstetric patients several investigations could not confirm these results.

Norris published two studies in which he administered 12 or 15 mg heavy bupivacaine intrathecally for Caesarean section and noted no relationship between block height and patient height, weight, vertebral column length and body mass index (BMI) (6,7). Ekelof more recently confirmed this using plain bupivacaine (8). However in an even more recent trial, Harten and co-workers compared a fixed dose of heavy bupivacaine with a weight and height adjusted dose (9). These authors however observed significant differences between the two groups, indicating that height and weight do play a role. With the adjusted dose onset time was longer, there was less hypotension and there were fewer patients having a segmental block higher than T1. In contrast to previous studies, they used considerably lower doses. Perhaps this can explain why height/height adjusted doses performed better then a fixed dose. More studies are needed to determine the need to adjust the dose to weight or height when low bupivacaine doses are administered.

It is well known that due to pregnancy the lordotic curvature of the spinal column is accentuated (4). This results in a reduced cephalad spread of the local anaesthetic solution due to pooling of the solution in the deepest part of the S-shaped curve.

The position in which the patient is during spinal puncture and the position during induction of the block also are factors that influence anaesthetic spread. Of course anaesthetic baricity influences the effect of positioning. Several studies have been performed in parturients scheduled for Caesarean section evaluating the effect of the sitting, lateral or Oxford positions. Other studies looked at the effect of left lateral versus right lateral positioning, while other studies evaluated whether a positional variation after the puncture but during establishment of the block played a role. It is difficult to draw firm conclusions since the literature is confusing and contradictory (10,11). However some suggestions can be made. Initiation of spinal anaesthesia in the left or right lateral position does not affect local anaesthetic spread (12,13). With hyperbaric solutions the sitting position has the following effects: the block extends less cephalad, produces less
<table>
<thead>
<tr>
<th>Table 1: factors influencing distribution of local anaesthetic solution in the cerebrospinal fluid (CSF).</th>
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<tbody>
<tr>
<td><strong>Patient characteristics</strong></td>
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<td><strong>Technique of injection</strong></td>
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<td><strong>Diffusion characteristics of CSF</strong></td>
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<td><strong>Characteristics of anaesthetic solution</strong></td>
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<tr>
<td><strong>Modulation of the epidural space</strong></td>
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</tbody>
</table>

Modified according to Greene. Anesth Analg 1985; 64, 715 – 730.
hypotension, better umbilical artery pH values, more epidural supplementation is required (when CSE with low spinal doses is performed), the block is technically easier to perform and onset time of anaesthesia is slightly prolonged (14,15). These effects are especially noted when lower spinal doses (< 9 mg) are used and become less clear or more variable when higher doses are used. Also experience of the anaesthetist with the position plays an important role in the eventual effects of the position.

Although Greene suggested that needle direction and spinal needle opening should influence the block height (4), no clinical studies in parturients could actually confirm these hypotheses. Massé et al. studied 40 parturients undergoing elective Caesarean section under spinal anaesthesia with a Whitacre spinal needle using hyperbaric bupivacaine (16). In 20 patients the orifice of the needle was directed cephalad, whilst in the other 20 patients the orifice pointed caudad. These investigators noted no difference between the groups.

Epidural volume extension (EVE) is the injection of an epidural solution (saline or local anaesthetic) into the epidural space with the intention to extend the block height of a spinally injected local anaesthetic solution (17). Most studies have demonstrated that, if EVE is performed within the first 5 – 10 minutes after the spinal injection, the upper dermatomal spread of the local anaesthetic can be increased by 1 to 5 dermatomes (17). The mechanisms involved to explain these effects remain theoretical and include a direct passage of epidural local anaesthetic through the dural hole, neutralisation of the negative epidural pressure due to the Tuohy needle inertion and increased epidural pressure due to the injection of epidural solution resulting in a reduced CSF volume.

Local anaesthetic baricity is the ratio of local anaesthetic density and cerebrospinal fluid density. CSF density decreases in pregnancy (18). Most “plain” local anaesthetics are isobaric or even slightly hyperbaric at room temperature, but quickly become hypobaric following intrathecal injection due to a decreasing baricity with increasing temperature (19,20). If one discusses the effect of baricity one cannot see this free from posture while injecting. Many studies are flawed because they only used one position and compared local anaesthetics of different baricity. Hallworth et al. however performed an excellent study randomising 150 patients undergoing CSE anaesthesia for Caesarean section to 6 study groups (21). Half of the patients underwent CSE anaesthesia whilst seated, 75 patients received a CSE whilst in the right lateral position. They concluded that in the lateral position baricity did not influence local anaesthetic spread, whilst in the sitting position the block spread higher with decreasing baricity. They and other authors also concluded that hypobaric solutions were much more unpredictable and resulted in more hypotension (21,22).

Dose is also an important determinant in the clinical effects of a spinal anaesthetic for C-section. Several authors have determined the full dose-response relationship of bupivacaine with opioids for spinal anaesthesia for Caesarean section. Significantly different results were obtained. Ginosar et al. and Carvalho et al. noted that the ED95 was > 11 mg, whilst Roofthooft et al. observed an ED95 of < 9 mg (23,24,25). These differences can be explained by differences in study population and study design, but emphasize that clinical practice and local experience is probably more important than an exact dose ! However, what is important to note is that with low dose spinal anaesthesia (< 9 mg or < 7.5 mg) bupivacaine, less hypotension occurs as was nicely summarized in a recent review (26). However, all authors agree that when low intrathecal doses are used, they should be administered as part of CSE technique.

Conclusions
Many factors influence the spread of the spinal block. Often the literature is confusing and contradictory. An important reason is that each and every factor is directly influenced by many of the other factors and by physician experience with a certain technique. Some conclusion can however be drawn: 1) Hyperbaric bupivacaine seems to give a more controllable and predictable block then is/hypobaric solutions; 2) The sitting position, especially with hyperbaric solutions, results in less hemodynamic perturbations; 3) Large variability exists in the literature concerning the ED95 dose of bupivacaine for C-section. Probably local practice and study design can account for these differences. However, low dose spinal anaesthesia has repeatedly been shown to provide effective anaesthesia conditions with limited duration. Therefore it is advised that when a low dose technique is used, a back-up epidural catheter would be placed; 4) Low dose spinal anaesthesia results in less hemodynamic side-effects.

References

1. Russell IF. A comparison of cold, pinprick and touch for assessing the level of spinal block at caesarean section. IJOA 2004; 13, 146 – 152.
2. Russell IF. At caesarean section under regional anaesthesia, it is essential to test
sensory block with light touch before allowing surgery to start. IJOA 2006; 15, 294 – 300.


20. Richardson MG, Wissler RN. Densities of dextrose-free intrathecal local anesthetics, opioids, and combinations measured at 37°C. Anaesth Analg 1997; 84, 95 – 99.


Session 6: Caesarean Section

The obese parturient

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Municipal Hospitals of Munich, Germany
Maternal Mortality During Pregnancy

Deaths per 100,000 Life Births: UK 2003-2005

<table>
<thead>
<tr>
<th></th>
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<tbody>
<tr>
<td>Early in pregnancy</td>
<td>14</td>
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<tr>
<td>Postpartum hemorrhage</td>
<td>14</td>
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<td>Amniotic fluid embolism</td>
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<tr>
<td>Thromboembolism</td>
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<tr>
<td>Cardiac causes</td>
<td></td>
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</table>


Obesity in Pregnancy and Use of Health Care Resources

<table>
<thead>
<tr>
<th>Variable</th>
<th>Normal BMI 20-29 (kg/m²)</th>
<th>Overweight BMI 30+ (kg/m²)</th>
<th>Class II obesity</th>
<th>Class III obesity</th>
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<tbody>
<tr>
<td>Gestational age</td>
<td>25-45</td>
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<td>20-29</td>
<td>30-40</td>
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<td>1.7%</td>
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<td>Sepsis</td>
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<td>Thromboembolism</td>
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<td>2.4%</td>
<td>2.7%</td>
<td>3.6%</td>
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<tr>
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<td>2.4%</td>
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<td>Hospital admission</td>
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Increased risks
- Limited cardiac and pulmonary reserve
- Sleep apnea
- Increased gastroesophageal reflux
- Preeclampsia
- Recurrent miscarriage
- Fetal macrosomia
- Caesarean delivery
- Postpartum hemorrhage
- Wound infections
- Thromboembolism
- Hypoxemia

High risk pregnancy

Obesity: maternal and fetal outcome

<table>
<thead>
<tr>
<th>BMI &gt; 30 kg/m²</th>
<th>BMI &gt; 40 kg/m²</th>
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<tbody>
<tr>
<td>2</td>
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</table>

Increased rate of congenital heart defects (atrial and ventricular septal defects)

- All defects
  - BMI > 29 kg/m² OR 1.18 (1.09 - 1.27)
  - BMI > 35 kg/m² OR 1.41 (1.22 - 1.64)
Severe Obesity and Neural Tube Defects

Rasmussen et al., Am J Obstet Gynecol 2008; 611-618

Maternal Gestational Diabetes and Childhood Obesity

Schäfer-Graf et al., Diabetes Care 2005; 28: 1745-1750

Maternal Weight and Failure of Epidurals

Retrospective analysis 1997-2005, 13299 epidurals

Dresner et al., BJOG 2006; 113: 1178-1181

Subcutaneous Catheter Movement During Labor with Tight Catheter Fixation

Carrie & Russel, Anaesthesia 2000; 55: 1231-1233

BMI and Depth of the Epidural Space

2009 parturients 1995 with normal Tuohy needle 14 with longer Tuohy needle (13 cm)

Clinkscales et al., Int J Obstet Anesth 2007; 16: 323-327

BMI and Local Anaesthetic Requirements

Panni et al., Br J Anaesth 2006; 96: 106-110

Spinal Anaesthesia and Maternal Vital Capacity

Von Ungern-Sternberg et al., Anaesthesia 2004; 59: 743-749

8 anaesthesia-related deaths (2.3% of all maternal deaths)

- 6 parturients with severe obesity
- 2 anaesthesia nurses (supervised by obstetrician)
- 6 anesthesia nurses (supervised by anesthesiologist, only in 2 cases present)
- 4 x cardiac arrest under spinal anaesthesia
- 5 x hypotension, airway obstruction after anaesthesia
- 2 x cardiomyopathy, myocardial infarction
- lack of standard monitoring postoperatively (pulsoximetry)
Laryngoscopy and Morbid Obesity: Sniffing versus Ramped Position
Collins et al., Obes Surg 2004; 14: 1171-1175

Comparison of Different Videolaryngoscopes in Morbid Obesity (BMI 43 kg/m²)
Maassen et al., Anesth Analg 2009; 109: 1560-1565

Gastric Emptying in Obese Parturients
Managing pain & distress during caesarean section under regional anaesthesia

Dr Geraldine O’Sullivan
Consultant Anaesthetist, Guy's & St Thomas' Hospitals, London

Pain. An unpleasant feeling caused by injury

Distress. Acute physical or mental suffering

Pain during caesarean section, which has not been appropriately managed, is one of the commonest reasons for complaints and litigation in obstetric anaesthesia.

From a woman’s perspective a caesarean section is never ‘sensation free’; it should however never be painful. Therefore at the pre-operative visit (or in the operating theatre in the event of an emergency caesarean section) the anaesthetists must carefully explain to the woman that her caesarean section will be ‘pain free’ but not ‘sensation free’. If these facts are not carefully explained the more anxious and nervous woman may believe that her anaesthesia has failed.

A complaint of pain (as opposed to discomfort) during caesarean section requires immediate action by the anaesthetist. If the pain is severe, this fact will probably be immediately apparent to all who are present in the operating theatre!! If the pain is less severe but is causing distress to the mother it can be helpful to ask her to rate her pain on a numerical scale, e.g. 0 - 10. In either event the anaesthetist must ask the surgeon to stop operating and offer the woman the option of continuing her surgery under general anaesthesia. If she accepts this option, general anaesthesia should be induced immediately using a rapid sequence induction. Surgery can recommence once the airway has been secured with a cuffed endotracheal tube. If the pain is severe it is unlikely that the woman will refuse the offer of a general anaesthetic. However in some situations, the mother may initially refuse a general anaesthetic. In this instance the anaesthetist must carefully record that general anaesthesia was offered and refused. Every effort must then be directed at relieving the pain. It will often be appropriate to make further offers of general anaesthesia to the woman. The times of these offers and the woman’s response must be recorded. This documentation may help deal with complaints and even legal action over the forthcoming months.

Why does regional anaesthesia fail during caesarean section?

1. Failure (or apparent failure) to check the level of anaesthesia.

   If an anaesthetist has not recorded the level of anaesthesia on the anaesthetic chart, any subsequent complaints about inadequate anaesthesia will be indefensible.

2. Inadequate block assessment

   The appropriate level of anaesthesia for a caesarean section and its method of assessment, has been one of the ongoing debates in obstetric anaesthesia. However, loss of cold sensation to T4 and touch to T5 usually implies an adequate level of anaesthesia. The lower level of anaesthesia should also be assessed, e.g. was the mother completely pain free when the urinary catheter was inserted? The level of motor block should also be assessed, if the women is fully mobile, the block is probably inadequate! Warm dry feet indicate that a sympathetic block has been achieved, and without which surgery should not be allowed to commence.

Should surgery ever commence before an adequate level of anaesthesia has been achieved?

Logic might suggest that the answer to this question would always be negative. However some bending of the rules can be permitted in the event of an emergency caesarean section in a woman who has had very effective epidural analgesia during labour. If such a woman requires an emergency section and the anaesthetist has satisfactorily demonstrated to the woman and him/herself that the level of anaesthesia is rising satisfactorily; then it might, in the event of severe fetal distress and after a thorough explanation to the woman, be permissible to make the skin incision before the block has reached T4 to cold.

Failure of Anaesthesia during caesarean section

Spinal vs Epidural anaesthesia

Local anaesthetic and opioid are required to produce adequate anaesthesia for both spinal and epidural anaesthesia for caesarean section. In the UK 0.5% heavy bupivacaine is the local anaesthetic of choice for spinal anaesthesia whilst fentanyl or preferably diamorphine are the most appropriate opioids.
Bupivacaine 0.5%, L-Bupivacaine 0.5% and Ropivacaine in combination with opioids are all effective agents for epidural analgesia. Subarachnoid anaesthesia produces a denser block than epidural anaesthesia and complaints of discomfort are therefore more commonly associated with epidural anaesthesia.

Approximately 66% of caesarean sections in the UK are performed as emergency procedures during labour. Many of these women will already have an epidural catheter in-situ for analgesia in labour. If the analgesia during labour has not been 100% satisfactory the epidural must not be topped-up for surgery. Inadequate analgesia can rarely be converted to satisfactory anaesthesia. In this situation the catheter must be removed and the surgery should be performed under spinal anaesthesia.

**Treatment of pain/distress during caesarean section**

1. Epidural top-up, if appropriate, e.g. 0.75% ropivacaine with either fentanyl 50-100.
2. IV fentanyl 50-100µg
3. 50:50 mixture oxygen and nitrous oxide
4. IV Ketamine 10-20mg
5. IV midazolam 1–1.5mg or Propofol 10-20mg. These drugs will not relieve pain, but ally anxiety.
6. General anaesthesia will be necessary if pain persists or if the woman requests to be put to sleep

**References**

Lucas DN. Extending low-dose epidural analgesia for emergency caesarean section – a comparison of three solutions. Anaesthesia 199; 54:1173
Session 7: The Baby 1
The decision to deliver - the obstetrician's viewpoint

Professor Andrew Shennan
Professor of Obstetrics, Guy's & St Thomas' Hospitals, London

When to delivery a baby is far from a precise science. Operative intervention rates, particularly Caesarean Section, have escalated in recent decades, with little evidence of improvements in perinatal outcomes. Caesarean Section rates have more then doubled since the 1980s. Even the recent trend to staff labour wards with senior obstetricians has little impact on intervention rates. Increasing trends in maternal choice for Caesarean Section cannot account for this increase. So why has the intervention rates increased?

The expectation of a normal baby is high in modern society, yet absolute risks of serious morbidity in childbirth remains relatively high compared to risks generally accepted in society: a 1:1000 risk of dying in labour is not accepted in other aspects of life (This is equivalent to x2 plans crashing each week leaving Heathrow). This is confounded by an inability to accurately judge fetal wellbeing. For all the apparent sophistication of fetal monitoring, the obstetrician is in fact waiting for gross and serious abnormality resulting in detectable biophysical changes (acidosis influences the autonomic system for example, causing abnormal fetal heart rate changes.)

The decision to deliver from the obstetricians viewpoint is therefore fraught with anxiety and uncertainty. Poor monitoring techniques result in frequent false positive decisions. The consequences of “getting it wrong” are substantial. There is only a small window of opportunity from acidosis resulting in detectable biophysical changes and neurological damage. This talk will outline the factors that inform the decision to deliver, including the uncertainty of current fetal monitoring.
Session 7: The Baby 1
Emergency caesarean section following the decision to deliver - the anaesthetist’s viewpoint

Dr Chris Elton
Consultant Anaesthetist, University Hospitals of Leicester

Emergency Caesarean Section - the anaesthetist's viewpoint

OAA Three-day Course in Obstetric Anaesthesia and Analgesia: 9 - 11 November 2009
10th November 2009
Dr Chris Elton
University Hospitals Of Leicester

Categories of Caesarean Section (US)

- Stat
  - Condition that is immediately life threatening for mother or fetus
  - Massive haemorrhage
  - Ruptured uterus
  - Cord prolapsed with fetal bradycardia
  - "Agonal" fetal distress
- Urgent
  - Maternal or fetal physiology is unstable but not immediately life threatening
  - Hypoxia
  - Failed trial of forces
  - Good prognosis without fetal distress
- Stable
  - Stable physiology
  - Chronic unexplained insufficiency
  - Abnormal fetal presentation with ruptured membranes

Grade of Caesarean Section (%)
Sentinel Audit 2001

Don’t Believe Everything You’re told!

- Sentinel (2001)
  - Presumed Fetal Compromise
  - CTG Normal (8%)
  - Abnormal 89% (but NOT severely abnormal)
  - FBS performed pH greater than 7.2 (19%)
  - Half the Grade 1 CS were reclassified (8% not 16%)
  - Emergency CS makes up about 2% of births

“Grade 1 CS” -Sentinel 2001

- All births 3 months England and Wales
- 152,139 births; 32,222 caesarean sections
- Median delivery time 27 minutes
- 25% delivered in 18 minutes
- 75% delivered in 45 minutes
- STANDARD NOT ACHIEVED (30 mins)
Don’t Believe Everything You’re told!

- Davies and Collis 2007
  - 20 patients had GA section for Grade 1 CS
  - Assessment by independent consultants
  - 4/20 actually needed GA
  - 10/20 could have had regional
  - 6/20 did not need a caesarean section

Decision to Delivery Timing

- 30 min rule was/is “pragmatic” standard
- International standard BUT 20 mins in Germany
- More likely to be met if patient in theatre within 10 minutes of decision
- Availability of 2nd theatre
- Shortening decision to delivery does not reduce admission to NICU or neonatal acidaemia

- Tuffnell et al BMJ 2001 1330
- MacKenzie et al BMJ 2001 1334
- Dunphy et al J Obstet Gynecol 1991 211

Decision to Delivery Timing

- Decision to Delivery Times less than 20 mins associated with INCREASED fetal compromise
  - (Hillemans et al 2005 Arch Gynecol Obstet 161-5)
  - Analysis of Sentinel Study patients showed NO difference provided DDI was less than 75 minutes
- (Thomas et al BMJ 2004 665)

“Special Cases”

- Placental Abruption with Fetal Bradycardia
  - 20 mins vs 30 mins (OR 0.44 0.22-0.86)
  - Kayani et al 2003 BJOG 679-83
- Bleeding vasa/placenta praevia
  - (Cord Prolapse with absent pulsation)


- 873 Intrapartum deaths in 1994-5 report
- 54 cases commented on anaesthesia (6.2%)
- 25 anaesthetic “delay” identified (2.9%)
- 19 GA, 3 Spinal, 3 Epidural

CESDI (2000)
No evidence in recent reports of “rushed” anaesthesia causing death

- SHO’s
- Equipment problems
- Women opted for GA (2000-2)
- Drug Problems
- Postopcare (2003-5)
- Are we getting the balance right?

Strategies for Improving Outcome

1. Proactive approach in labour
2. In utero resuscitation
3. Expedite arrival in theatre
4. Use appropriate anaesthetic technique

Proactive Anaesthetic Management

- Attend Ward Rounds to Identify those at risk of intervention
- “87% of 360 consecutive emergency CS can be anticipated”
  - (Morgan et al 1990 BJOG 420)
- Insert epidural- Regular follow up!
- Communicate (Distress, FBS, Progress)
- Coordinate

Proactive Anaesthetic Management - CEMACH 2007

- Identify those at risk of anaesthetic complications
  - Preconception
  - During Pregnancy
  - During labour (ward rounds)
- Particular Concerns
  - Obesity
  - Recent migrants
    - Language, Social Issues, Undiagnosed/Untreated Illness
  - Sepsis
  - Pre-eclampsia
  - Occult Haemorrhage

What has to be done between decision to deliver and delivery!-

Tuffnell BMJ 2001 1330

- Informed consent: Consent form signed
- Intravenous access
- Blood samples to be taken
- Blood forms to be filled in
- Bloods to laboratory
- Intravenous fluids running
- Premedication to be given from drug cupboard
- Premedication drawn up
- Premedication injected
- Anaesthetist informed
- Operating department assistant informed
- Consultant to be informed
- Anaesthetist to arrive
- Operating department assistant to arrive
- Theatre to be set:
  - Scrub nurse to scrub
  - Packs to be opened
  - Sutures to be opened
  - Monitoring to be discontinued
  - Intravenous lines to be secured
  - Fetal scalp clip to be removed
- Woman to be moved to theatre:
  - Anaesthetist to scrub
  - Spinal pack to be opened
  - Spinal drugs to be drawn up
  - Intravenous fluids running
  - Premedication to be given
  - Monitoring to be attached
- Woman to be moved on to theatre table
- Spinal anaesthesia
  - Wait for block to work
  - Patient to be present
  - Resuscitator to be checked
  - Catheter
  - Shave
  - Surgeons to scrub
  - Skin preparation
  - Skin incision
  - Sheath incision
  - Peritoneum opened
  - Bladder reflected
  - Uterine incision
  - Deliver baby
  - Syntocinon off
  - Position full left lateral (allows rapid spinal)
  - Oxygen
  - I.v. infusion of 1 litre crystalloid
  - Low blood pressure: I.v. vasopressor
  - Tocolysis: terbutaline 250 μg (s.c), glyceryl trinitrate 400 μg (metered aerosol doses)
  - Monitor Fetus It may get better!
  - Thurlow JA, Kinsella M UOA 2002 11 105-16

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98
What Anaesthetic Technique?

- GA associated with significantly shorter DDI
- GA associated with 16.7X (now 1.7) greater risk of maternal death
  - Hawkins et al 1997 Anesthesiology 273-6
- Overwhelming preference for RA for maternal safety-CEMACH
- Risk for fetus?

General Anaesthesia vs Spinal with Fetal Distress

Dyer et al Anesthesiology 2003 99 561-9
- Seventy patients randomised to GA or SAB
- “Non reassuring” fetal heart trace
- Neonatal indicators worse in SAB group

<table>
<thead>
<tr>
<th></th>
<th>GA</th>
<th>SAB</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neonatal umbilical BE</td>
<td>4.68 +/- 3.3</td>
<td>7.13 +/- 4.0</td>
</tr>
<tr>
<td>Umbilical pH</td>
<td>7.23</td>
<td>7.20</td>
</tr>
</tbody>
</table>

Topping up an epidural

- Is this epidural working?
- How Much? 20mls, Divided doses?
- What mixture?
  - Laevobupivacaine (10 mins)
  - Ropivacaine (10 mins)
  - Lignocaine/Adrenaline/Bicarbonate (7 mins)
- Fentanyl/Sufentanil only if not used in labour
  - Malhotra, Yentis Anaesthesia 2007 667
  - Tortosa et al BJA 2003 532


- Beware complacency with “less toxic agents”
- Aspirate
- Give intravascular test dose (1mg/kg lignocaine)
- Intralipid should be available
**“Single Shot Spinal”**

- 15-20 mins to surgical anaesthesia
- May be shorter with “rapid sequence spinal” (Scrutton, Kinsella)
  - Theatre in left lateral
  - IVI (obstetrician)
  - Pre O2
  - Prep, “No touch”
  - No diamorphine

*Scrutton, Kinsella IJOA 2003, 12, 144*

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**General Anaesthesia**

- Trained Anaesthetist/Skilled Anaesthetic Assistant
- Fully Equipped Theatre (Advanced airway aids)
- Antacids
- Pre O2 (Sat Up/30 degree head up)
- TILT
- RSI/Cricoid
- Thiopentone (sleep dose)/Suxamethonium/ETT
- MODIFY in Preeclampsia/Cardiac/Cerebrovascular Disease
  - Pressor response to intubation AND extubation
- Give an anaesthetic (ie 1.3 MAC!)
- Intubating Aids/Failed Intubation Drill

---

**Conclusion**

- High risk patients should be seen antenatally and in labour
- Emergency CS can often be anticipated
- Retrospective studies suggest that DDI in grade 1 CS should be less than 75 minutes but NOT less than 20 minutes except in extreme circumstances
- Anaesthetists should not feel bullied to achieve unsafe decision to delivery times
- Anaesthetists should work with not against obstetricians
Session 7: The Baby 1
Intrapartum fetal assessment and resuscitation – what the anaesthetist needs to know

Professor Steve Robson
Professor of Fetal Medicine, Royal Victoria Infirmary, Newcastle-upon-Tyne

Fetal Resuscitation in Labour
Aims

- Review assessment of fetal status in labour
- Review effects of maternal positioning, maternal oxygen and iv fluids
- Review use of tocolytics for nonreassuring FHR pattern
- Review use of amnioinfusion for nonreassuring FHR pattern
- Consider impact of mode of anaesthesia for emergency CS for potentially acidaemic fetus
- Develop guidance for fetal resuscitation in labour

Diagnosis of intrapartum ‘fetal distress’
- Baseline rate
- Baseline variability
- Decelerations
  - Relationship to contraction
  - Depth / recovery
- Meconium
- Response to stimulus
  - Vibroacoustic stimulation
  - Vaginal exam / PBS
- ECG
  - ST waveform analysis
- Acidaemia
  - pH < 7.20
  - Rate of decline
    - Metabolic / respiratory
- Lactate

Diagnosis of intrapartum ‘fetal distress’
Scalp pH vs. Lactate

- Focus on pH (acidaemia) – DD or lactate required to interpret
  - only metabolic acidaemia associated with neonatal mortality
- Conventional pH analysis requires 30-50 mL vs. 5 mL for lactate
- Sampling failure rates higher with pH (10-20%) vs. lactate (1.2%)
  (OR 15.1 [5.0-44.7])
- For pH decision-result interval >20 min in 35% cases (< 30 min in 9%)
  - Sampling and analysis quicker with lactate.
- No differences in metabolic acidaemia or short term neonatal outcomes
- Combined measurement pH & lactate no better at predicting outcome but increases number of abnormal results (and hence OOFD)

Diagnosis of intrapartum ‘fetal distress’
Scalp pH or lactate

<table>
<thead>
<tr>
<th>Lactate</th>
<th>pH</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.2%</td>
<td>10.4%</td>
</tr>
<tr>
<td>3.7%</td>
<td>3.6%</td>
</tr>
<tr>
<td>1.5%</td>
<td>1.6%</td>
</tr>
<tr>
<td>6.6%</td>
<td>9.5%</td>
</tr>
<tr>
<td>35.8%</td>
<td>38.2%</td>
</tr>
<tr>
<td>0%</td>
<td>0%</td>
</tr>
</tbody>
</table>

Diagnosis of intrapartum ‘fetal distress’
Decision-to-delivery interval

- DDI ‘standard’ of < 30 min widely adopted (e.g. RCOG, ACOG) and accepted as medical/legal benchmark
- DDI very poor correlation with outcome
- Some studies better cord arterial pH with longer DDI
  (Chapman et al. 1997, Mantero et al. 2002)

- Methods diagnosing ‘FD’ high F+ rate
- Ignore causes of ‘FD’ e.g. Inverse vs ‘potentially’ reversible
- Selection bias ( clinicians tend to deliver more severe cases quicker)
- Focus on DDI vs ‘Bradyarrhythmia-to-Delivery interval’
Intrapartum fetal distress

**Decision-to-delivery interval**

| Decision-to-delivery interval | 236 'crash' CS (5.7% at Em CS) - 90% under GA | Median (IQR) intervals - BDI 16 [14-16] min, DDI 11 [10-13] min | All delivered within 19 min of decision |

| Cord arterial pH | 500 mL !000 mL |

| Maternal oxygen therapy for nonreassuring FHR pattern |

- Umbilical & placental vessels vasoconstrict with hyperoxia
- Maternal hyperoxygenation immediately prior to CS does not affect umbilical cord pH
- Healthy may be immediately prior to CS does not affect umbilical cord pH

| Maternal position |

- Placing mother in left lateral or Sim’s position alleviates aortocaval compression
- Fetal oxygen saturation highest in left lateral position

| Supplementary oxygen for Emergency CS under regional anaesthesia |

- Oxygen via Venturi-type mask
- Rate of drop 0.013 / min

| Maternal oxygen therapy for nonreassuring FHR pattern |

- Umbilical & placental vessels vasoconstrict with hyperoxia
- Maternal hyperoxygenation immediately prior to CS does not affect umbilical cord pH

| Overstimulation with oxytocin |

- 'Aim for 3-4 contractions every 10 min'
- Cesu, RCOG 2001
Tocolysis for nonreassuring FHR pattern

- Terbutaline - 250 µg i.v. or sc
- Ritodrine – 1-2 mg i.v.
- Hexoprenaline – 5-10 µg i.v.
- Magnesium sulphate 4 g i.v.
- Atosiban – 6.75 mg i.v.

Terbutaline associated with reduced uterine activity in greater proportion of women than Mg (RR 0.07 (0.0-1.1))
Magann et al. 1993

Tocolysis for suspected intrapartum fetal distress

Terbutaline 250 mcg s/c

Overview of 3 RCT's

<table>
<thead>
<tr>
<th>Study</th>
<th>Control</th>
</tr>
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<tbody>
<tr>
<td>n=11</td>
<td>n=9</td>
</tr>
<tr>
<td>Fetal Capillary UA</td>
<td>P&lt;0.025</td>
</tr>
<tr>
<td>pH increase &gt; 0.05</td>
<td></td>
</tr>
<tr>
<td>pH decreased or 'unchanged'</td>
<td></td>
</tr>
</tbody>
</table>

Labour allowed to continue (unless other obstetric criteria present)
Delivery expedited

Patriarco et al. 1987

Tocolysis for fetal distress

Terbutaline

- 250 µg in 5 ml saline i.v. over 5 min.
- Rapid (1-2 min) prolonged (15-20 min) tocolysis
- Few maternal side effects [pH ↑ 20 bpm, 10% palpitations]
- Propanolol 1-2 mg after delivery baby
- 1-2 mg nebulized no effect

Kuller & Hofmeyer, CDSR 2009

Tocolysis for recurrent decelerations

Cabero et al. 1988

- 1126 Pathological FHR traces
- Late decelerations
- Variable decelerations
- Severely reduced BLV

Cabero et al. 2007

Pullen et al. 2007

Terbutaline vs nitroglycerin

Prolonged deceleration (> 2 min)
Severe variable decelerations
Tachysystole with reduced BLV

Patriarco et al. 1987

Kulier & Hofmeyer, CDSR 2009

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Amnioinfusion for recurrent decelerations
Operative delivery for fetal distress

- Recurrent decelerations (post FBS)
  - scalp pH ≥ 7.20 – review FHR after 15 min
  - scalp pH < 7.20 (prior to transfer to theatre – review FHR in theatre)

- Bradycardia
  - Prior to transfer to theatre – review FHR in theatre

Tocolysis for fetal distress

- Prolonged bradycardia
  - FHR < 100 bpm for ≥ 3 min
  - or < 80 bpm for ≥ 2 min
  - No change with oxygen, position change

- Abnormal FHR pattern
  - FHR not improved (3)
  - FHR improved (30)
  - FHR continuous later (6)

- Injection-recovery time
  - ≥ 2 min (23)
  - 4-6 min (47-9 min)
  - ≥ 9 min (1)

Intrapartum amnioinfusion

- Initial bolus (250-600 mL) or 10-20 mL/min
- Maintenance - 3 mL/min

- Outcomes not influenced by:
  - gravity or infusion pump
  - warmed (37°C) or room temperature

- Risks / complications
  - Maternal / neonatal infection – no evidence risk increased
  - AF embolism – 2 cases reported
  - Uterine tone – Mean increase 4 mm Hg
  - Isolated cases of hyperactivity

Technique of amnioinfusion

- Initial bolus (250-600 mL) or 10-20 mL/min
- Maintenance - 3 mL/min

- Outcomes not influenced by:
  - gravity or infusion pump
  - warmed (37°C) or room temperature

Amnioinfusion for potential or suspected umbilical cord compression in labour

Review 14 RCT’s transcervical amnioinfusion

- Persistent variable decelerations
  - CS for suspected fetal distress
  - Forceps/VE for suspected fetal distress
  - Apgar score <7 at 1 min
  - Apgar score <7 at 5 min
  - Cord artery pH <7.2
  - Admission to NICU
  - Neonatal hospital stay for > 3 days

Amnioinfusion for recurrent decelerations
Operative delivery for fetal distress

- Infusion associated with improvement in: UA pH (7.27 vs 7.23), UA pCO2 (63 vs 66) + 1 min Apgar scores: 8 vs 34%
- Reduced IP stay (2.5 vs 3.0 dy)

SA fetus
Growths restricted fetus
- abnormal UA Doppler
- Preeclampsia (abnormal uterine artery Doppler)

Abnormal intrapartum CTG

Is spinal anaesthesia safe?
Conclusions

- Lateral position, oxygen and fluid
- Oligohydramnios & recurrent decelerations
- Timescale / experience key

Resuscitation

- Tocolytic (all cases acidemia pre-CS)
- Amnioinfusion (oligohydramnios with borderline acidemia)
- Spinal anaesthesia reasonable
  - Phenylephrine to maintain BP ≥ 90% baseline
  - Timescale / experience key

Anaesthesia for CS in acute fetal compromise

- As fast as possible
  (< 15 minutes)
- Less than 30 minutes

Phenylephrine for hypotension
Maternal and fetal haemodynamics (Doppler)

Comparison of phenylephrine infusion regimes for maintaining BP during spinal anaesthesia for CS

PE - 100 µg/min for 2 min then 100 µg each min if SBP ≤ 7.1 (4.0) Max fall in CO (L/min) 0.91 (1.18) 0.98 (1.08) Max increase in UA PI 0.2 (0.4) 0.5 (1.0) No statistically significant differences between PE & SGA groups

Dyer et al. 2003

RT of epidural vs spinal anaesthesia for non-immediate CS in women with potential fetal compromise

<table>
<thead>
<tr>
<th>Spinal Group</th>
<th>Epidural Group</th>
<th>Mean difference (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.26 (0.11)</td>
<td>7.28 (0.07)</td>
</tr>
<tr>
<td>BE (mmol/L)</td>
<td>-3.85 (2.93)</td>
<td>-3.77 (2.60)</td>
</tr>
</tbody>
</table>

Spinal anaesthesia for Caesarean Section
Phenylephrine and rapid crystalloid cohydration

Kaplan-Meier plot showing proportion of patients remaining not hypotensive until uterine incision

<table>
<thead>
<tr>
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<th>BD (mEq/L)</th>
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</thead>
<tbody>
<tr>
<td>7.20</td>
<td>14.4 (17.2)</td>
</tr>
<tr>
<td>7.15</td>
<td>4.7 (14.3)</td>
</tr>
</tbody>
</table>

RT of epidural vs spinal anaesthesia for non-immediate CS in women with potential fetal compromise

Propective RT comparing general with spinal anaesthesia for CS in PE with a nonreassuring FHR pattern

Kaplan-Meier plot showing proportion of patients remaining not hypotensive until uterine incision

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</thead>
<tbody>
<tr>
<td>7.20</td>
<td>17.6 (29.5)</td>
</tr>
<tr>
<td>7.15</td>
<td>9.7 (18.1)</td>
</tr>
</tbody>
</table>

Spinal anaesthesia for Caesarean Section
General vs. regional anaesthesia
1 min Apgar scores ≤ 7 in 49% GA, 22% regional

Comparison of phenylephrine infusion regimes for maintaining BP during spinal anaesthesia for CS

Phenylephrine for hypotension
Maternal and fetal haemodynamics (Doppler)

Maternal choice

General
(n=71)
Spinal
(n=33)
Epidural
(n=22)

Marx et al. 1984

Spinal Group (n=27) Epidural Group (n=30) Umbilical artery pH 7.24 (0.11) 7.26 (0.07) NS Umbilical vein pH 7.28 (0.10) 7.31 (0.05) NS

Entry criteria

Mother No Mother No With SGA Yes With SGA No Randomised n = 60

Power

To detect difference in UA pH 0.05 in difference in BE 1.75 mmol/L need 25 in each group (p = 0.05, 0.5 or 0.9)

Decision - delivery interval

- As fast as possible
  (< 15 minutes)
- Less than 30 minutes

Maternal choice

12.5 mg hyperbaric bupivacaine 0.5% + 300 µg diamorphine

Kee et al. 2004

Kee et al. 2004

Spinal Group (n=27) Epidural Group (n=30) Umbilical artery pH 7.24 (0.11) 7.26 (0.07) NS Umbilical vein pH 7.28 (0.10) 7.31 (0.05) NS

Power

To detect difference in UA pH 0.05 in difference in BE 1.75 mmol/L need 25 in each group (p = 0.05, 0.5 or 0.9)

Decision - delivery interval

- As fast as possible
  (< 15 minutes)
- Less than 30 minutes
Session 8: Challenges for the Obstetric Anaesthetist

Major trauma in pregnancy

Dr Paul Howell
Consultant Anaesthetist, St Bartholomew's & Homerton Hospitals, London

Trauma in Pregnancy

Trauma is the leading cause of death in under 45 year olds (USA)
= Leading cause of non-obstetric death in pregnant women

Prevalence

- Around 8% pregnancies suffer some trauma
- Up to 20% require emergency surgery

Pregnant vs. NON-pregnant Women:

- More likely to be admitted with lesser injuries
- More likely to test positive for alcohol / drugs (USA)
- Higher rates of injury than non-pregnant women:
  - 2.6 x Assault
  - 1.9 x Motor Vehicle Accident
  - 1.5 x Firearm injury
  - 1.5 x Falls

Causes:

Blunt Trauma:
Commonest
70% = Motor Vehicle Accidents
12% = Domestic Violence
9% = Falls

Penetrating Injuries:
Uncommon
More serious injuries
More maternal deaths

Three Lessons:

- Maternal resuscitation is the best form of fetal resuscitation – carry on as usual!
- Remember uterine tilt
- Consider early emptying of the uterus to facilitate maternal / fetal resuscitation
# Trauma in Pregnancy

## Causes: Motor Vehicle Accidents
- Morbidity & Mortality for Mother & Baby when seat belts not used (properly)
- Fetal death $\uparrow \times 3$ if no belt
- NB: 1 in 3 Pregnant women in USA don’t use seat belt / properly

Airbags OK


## Causes: Domestic Violence
- Previously poorly recognised / reported
- Major cause of maternal mortality
- $\leq 20\%$ Pregnancies affected

Shadigan E. Obstet Gynecol Surv 2005; 60: 183-90

## Causes: Suicide
- Previously under-recognised / under-reported
- Major cause of maternal mortality
- Often atypical violent deaths


## Outcome: Maternal
- Most injuries are minor
- Mortality pregnant women > non-pregnant women
- Most deaths due to head injury + haemorrhage
- Obstetric Complications:
  - Abruption (20-50%)
  - Preterm labour (25%)
  - Uterine rupture (0.6% but ..)
  - Amniotic fluid embolism ??
  - Feto-maternal haemorrhage

Patteson SK. Am Surg 2007; 73: 824-7
Rothenberger D. J Trauma 1978; 18: 173-9

## Outcome: Fetal / Neonatal

### 1st Trimester:
- Pelvis + thick uterus protect fetus
- = miscarriage + isoimmunisation

### 2nd Trimester:
- Uterus emerges into abdomen
- Protected by amniotic fluid

### 3rd Trimester:
- Uterus thin-walled
- = risk of direct injury to fetus

## Resuscitation: New Concepts
- “Scoop & Run”
- Hypovolaemic resuscitation (fluid restriction)
- Permissive hypotension
- Relative anaemia (Hb 7-9 g.dL$^{-1}$)
- Damage Control Surgery
- Conservative management of solid viscera injury
- Hypertonic saline

No data for pregnant patients
**Resuscitation:**

**Multidisciplinary Care:**
- Obstetrician
- Emergency Physician
- Anaesthetist
- Midwife
- ± General / Orthopaedic Surgeon
- ± Internal Medicine
- ± Intensivist
- ± Social Worker
- ± Psychiatrist

**Trauma in Pregnancy**

**Resuscitation:**

**ATLS**

**Primary Survey**
- ABCDE (Airway, Breathing, Circulation, Disability, Exposure)

**Resuscitation**
- ATLS

**Definitive Care**
- Data / Information / Response to Therapy

**American College of Surgeons, 2004**

**Resuscitation:**

**ABCDE**

**Airway:**
- Clear obstruction / foreign body
- NB: Pulmonary aspiration risk
- Give oxygen
- Protect airway if necessary (ETT)
- Rapid sequence induction (usually)
- Remember C-spine …….

**Breathing:**
- Assess adequacy
  - ? Head injury / drugs / alcohol
  - ? C-spinal cord injury
  - ? Haemo/pneumo-thorax
  - ? Open / frail chest
- Intubation + IPPV if necessary
- Avoid hyper / hypo-ventilation
- Aim for $P_{aCO_2}$ ~ 25-30 mmHg
- Use minimum airway pressures
- Avoid PEEP if possible ± Chest drain (insert at higher level)
  - Spahn DR. Crit Care 2007; 11: R17

**Circulation:**
- Remember uterine tilt
- Late decompenation in hypovolaemia
- Poor FH may indicate hypovolaemia
- May lose 35% of circulating volume before showing classic signs of hypovolaemia
- Stop compressible haemorrhage
- $\geq 2 \times$ Large bore IV lines upper limbs
- ± CVP (beware in hypovolaemia)
- ± Arterial line
- Beware pelvic fractures:
  - → XS Blood loss + XS Mortality

**Circulation:**

**Crystalloids vs. Colloids**

SAFE Study

- Saline versus Albumin Fluid Evaluation Study:
  - 7000 Non-pregnant trauma patients in ICU
  - No difference in overall mortality
  - $\uparrow$ Mortality if brain injury + albumin

**Hypertonic Saline**

- → Controversial
  - No evidence of improved survival
  - Studies ongoing ……..
  - → No evidence for use in pregnancy

**OAA: Three-day Course on Obstetric Anaesthesia and Analgesia, London, 9 – 11 November 2009**
Resuscitation: ABCDE
Circulation: Blood Products
• NB: Physiological anaemia of pregnancy
• Access to Group O Rh-ve blood
• Aggressive Rx blood components in massive blood loss
  → ↓ Mortality (Military, non pregnant patients)
  → 1 unit FFP per unit blood
Borgman MA. J Trauma 2007; 63: 805-13

Resuscitation: ABCDE
Circulation: Blood Products
• NB: Physiological anaemia of pregnancy
• Access to Group O Rh-ve blood
• Aggressive Rx blood components
• Recombinant Factor VIIa (rFVIIa)
• Tranexamic Acid ?
• Cell Salvage

Resuscitation: ABCDE
Circulation:
• Hypothermia → XS Coagulopathy → poor outcome
  Warming is important!
Warming is important!

Resuscitation: ABCDE
Circulation: Cardiac Arrest
• Remember TILT!
  - Sandbag under hip
  - Cardiff Wedge
  - Kneeling rescuer
  - Manually pulling uterus across
Katz VL. Obstet Gynecol 1986; 68: 571-6

Resuscitation: ABCDE
Disability: Traumatic Brain Injury
• Most common severe injury in motor vehicle accidents
• Leading cause of maternal death (with haemorrhage)
  ↓ Level of Consciousness = Intracranial pathology
  Intoxication
  Metabolic # (e.g. Diabetes)
  Post-ictal state
  Hypovolaemia
  Low cardiac output
Reg ular assessment is vital

Resuscitation: ABCDE
Disability: Traumatic Brain Injury
• CT Scanning
• Avoid IV colloids (SAFE study)
• Raised intracranial pressure →
  Transient effect on ↓ ICP
  ↓ Utero-placental perfusion
  Ventilate to normal (for pregnancy) P\textsubscript{a}CO\textsubscript{2}

Resuscitation: ABCDE
Disability: Traumatic Brain Injury
• CT Scanning
• Avoid IV colloids (SAFE study)
• Raised intracranial pressure →
  Aggressive Management of hypotension & hypoxia is vital
  Raise head
  Avoid hypoventilation
  Avoid hyperventilation

  Mannitol / frusemide?
  Hypertonic saline-dextrose
  Corticosteroids?
Resuscitation: ABCDE

Exposure:
- Check for hidden injuries
- Convulsions or↓Level of Consciousness

- Eclampsia
- Epilepsy
- Intracranial pathology
- Metabolic (e.g. hypoglycaemia)
- Drugs (e.g. cocaine)

Fetus: Monitoring

Cardiotocography (CTG):
- Fetal Heart Rate + Variability

NB: Non-reassuring CTG may be 1st sign of hypovolaemia
NB:↓↓↓FHR Variability with opioid analgesia

Obstetrician looks for:
- Uterine activity
- Ruptured membranes
- Placental abruption

Fetal Heart ST Analysis (STAN)


Fetus: Isoimmunisation

Rhesus +ve fetus Rhesus –ve mother

Feto-maternal Haemorrhage

Kleihauer-Betke Test

Give Rh(D) Immunoglobulin to mother

Radiographic Imaging:

- Do what needs to be done – Mother comes first!!
- Risks of teratogenesis / malignancy / gene mutation = SMALL
- Maximum risk of teratogenesis in 1st Trimester
- Protect fetus where possible (lead shield over pelvis)

Multidetector CT Scan (MDCT) = Gold Standard
64 Slice MDCT → Whole body CT in ~ 30 secs

NB: Escort to CT Scan
Remember uterine tilt!

Ahvenjarvi L. Acta Radiol 2005; 46: 177-83

Opioids:
- OK = mainstay in trauma
- NB: flat CTG / neonatal depression

NSAIDs:
- Generally discouraged
- NB: Platelet / renal effects ?? Short term use OK

Paracetamol:
- Safe (IV Preparation available)

Tramadol:
- Embryotoxic in animal studies but ….?

Regional analgesia ± peripheral nerve blocks ± intercostal nerve blocks for rib fractures

Analgesia:

Ultrasonography:
e.g. FAST Scan (Focused Assessment with Sonography for Trauma)
- Use for ? intrabdominal haemorrhage (has replaced DPL)
- Useful for pericardial fluid / haemothorax
- Sensitivity similar in pregnant / non-pregnant
- May also be used to check fetal heart

Training:

Advanced Trauma Life Support (ATLS)
American College of Surgeons
www.facs.org/trauma/atls/index.html

Managing Obstetric Emergencies and Trauma (MOET)
Advanced Life Support Group (ALSG) / RCOG
Endorsed by CEMACH
www.alsg.org

Primary Trauma Course (PTC)
Developing world project
www.primarytraumacare.org

European Trauma Course (ETC)
European Resuscitation Council
New, simulator based course, in development
www.erc.edu

OAA: Three-day Course on Obstetric Anaesthesia and Analgesia, London, 9 – 11 November 2009
Surgery: Do what is necessary!

- Remember tilt – to minimise aortocaval compression
- Antacid prophylaxis
- Rapid Sequence Induction
- IPPV to normal (for pregnancy) $P_aCO_2$
- CTG - before / after surgery (+ during…..?)
- Maintain BP / cardiac output (+ uteroplacental perfusion)
- Consider transoesophageal doppler (TEE)
- Consider cell salvage if available
- If caesarean section – beware IV bolus of syntocinon
- Post-operative HDU / ITU care

Three Lessons:

- Maternal resuscitation is the best form of fetal resuscitation – carry on as usual!
- Remember uterine tilt
- Consider early emptying of the uterus to facilitate maternal / fetal resuscitation
Session 8: Challenges for the Obstetric Anaesthetist
Pulmonary oedema in the third trimester – a perspective from South Africa

Professor Rob Dyer
Professor & Second Chair, Department of Anaesthesia, University of Cape Town

Introduction
In the developing world, the diagnosis and management of the causes of pulmonary oedema in the third trimester are made difficult due to the high prevalence of co-morbidities such as preeclampsia, valvular heart disease and HIV/AIDS. This presentation describes several case presentations which highlight key diagnostic and management points in this challenging area of obstetric anaesthesia.

A brief summary of anaesthesia considerations relating to peripartum cardiomyopathy (PPCM) is presented as an introduction.

Clinical presentation and pathophysiology of PPCM
The diagnosis of PPCM depends upon the following criteria:
(1) The development of cardiac failure in the last month of pregnancy or within 5 months postpartum,
(2) The absence of an identifiable cause of cardiac failure,
(3) The absence of heart disease before the last month of pregnancy, and
(4) Echocardiographic evidence of left ventricular systolic dysfunction, including left ventricular ejection fraction < 45%, and/or fractional shortening < 30%, and/or left ventricular end diastolic dimension >2.7cm/m².

PPCM remains a diagnosis of exclusion; there are no specific criteria distinguishing PPCM and other forms of dilated cardiomyopathy. The incidence is about one case per 1000 live births in South Africa, and one in 2289 to one in 4000 live births in the USA. Although relatively rare, this condition is associated with mortality rates of 20-85%. The largest prospective case series reports have not confirmed previously suggested risk factors for PPCM, namely older age, multiparity, and long-term use of tocolytic agents. Preeclampsia has been thought to be a risk factor, although some authors state that the presence of hypertension or preeclampsia precludes the diagnosis of PPCM.

The pathogenesis remains poorly understood. Autoimmune and inflammatory mechanisms have been proposed and studied since the 1970’s. Myocarditis has been implicated, but no definite link established. Biopsies found an inflammatory component in only <10% of cases. A viral origin for the disease has also been postulated. Another theory is that male chromosomal DNA originating from fetal cells in the maternal circulation (microchimerism) may initiate an autoimmune myocarditis. Further evidence of immune activation is the finding of increased levels of tumour necrosis factor, C-reactive protein and a plasma marker of apoptosis, Fas/Apo-1. Prolactin cleavage is now thought to be implicated, influencing a pathway essential for the protection of the heart from postpartum stress. Yet another study has demonstrated that apoptosis of cardiomyocytes may be the crucial defect, in that inhibition of such apoptosis improves cardiac function in mice.

The clinical presentation in South Africa consists of a displaced hypodynamic apex in 72% of cases, and a gallop rhythm in 92%. Mitral regurgitation was found in 43%. ECG changes included voltage criteria for LVH in 66% and ST-T wave abnormalities in 96% of cases. Q waves, non-specific ST segment changes, atrial or ventricular arrhythmias, and left bundle branch block have all been reported. Other signs and symptoms are oedema, dyspnoea, orthopnoea, paroxysmal nocturnal dyspnoea, cough, abdominal discomfort, precordial pain and postural hypotension. As the heart dilates, thrombus formation, particularly in the right ventricle and left atrium, becomes an increasing risk. Peripheral embolisation can then involve lower extremities, the cerebral circulation, the splanchnic circulation, and the coronary or pulmonary arteries. The differential diagnosis includes myocardial infarction, sepsis, severe preeclampsia, amniotic fluid embolism and pulmonary embolism.

Pharmacological management
Treatment objectives are to reduce preload and afterload and increase contractility. Diuretics are safe and usually the first line therapy. Hydralazine is preferable to ACE inhibitors during pregnancy. Vasodilators in the form of nitrates and amlodipine may also have a place. The positive inotropic effects of dobutamine and even adrenaline may be necessary...
Digitalis is useful for contractility, rate control and employed. Inhibitor enoximone has also been successfully fetal effects 14. ACE inhibitors are avoided in pregnancy because of improve survival in dilated cardiomyopathy, but treatment after delivery. Heparinisation, particularly who dobutamine did not improve haemodynamics. The use of the β-blocker carvedilol may improve survival in dilated cardiomyopathy, but there is no data on its use in PPCM. Amiodarone and ACE inhibitors are avoided in pregnancy because of fetal effects, but ACE inhibitors are the mainstay of treatment after delivery. Heparinisation, particularly if EF is <35%, is often necessary, followed by warfarin in the postpartum period.5

Obstetric and anaesthesia management
Although this condition is uncommon, the anaesthetist should have a high index of suspicion of PPCM in any parturient presenting with symptoms attributable to cardiac failure in the last month of pregnancy. There are two well documented cases of undiagnosed PPCM who received conventional doses of thiopentone for induction of anaesthesia for CS and collapsed, requiring extensive cardiopulmonary resuscitation.15,16

Delivery of the fetus should reduce haemodynamic stress. A multidisciplinary team, including obstetrician, cardiologist and anaesthetist, should decide on the safest mode and timing of delivery in severe cases. It is important that the mother’s condition should be stabilised as best possible before delivery is attempted. Vaginal delivery is usually associated with minimal blood loss and less chance of postoperative infection and pulmonary complications. Effective analgesia is essential to minimise haemodynamic stress. Also, carefully titrated epidural analgesia can be used to carefully reduce afterload and preload, and decrease the fluctuations of cardiac output in labour. Caesarean delivery is reserved for fetal distress, failure to progress, or severe heart failure unresponsive to maximal medical therapy. For caesarean section, first and foremost the coagulation status determines whether regional anaesthesia can be used. Assuming no contraindication in this regard, the next consideration is whether the degree of severity of cardiac failure will permit a sensory block to the level of T4 (which implies sympathectomy to at least T2), as is required for CS, in the presence of some degree of aortocaval compression. Epidural or combined spinal-epidural are preferable to single shot spinal anaesthesia, allowing gradual titration of the block, while permitting judicious use of fluids and vasopressors. The authors of a series of 4 case reports gave cogent reasons for the use of carefully performed epidural anaesthesia for CS. However, recent case reports suggest that even very low doses of intrathecal local anaesthetic can cause major haemodynamic effects,1,19 and regional anaesthesia for CS in PPCM should probably be reserved for NYHA Class 1-2, and occasionally Class 3 patients in experienced hands. In patients with pulmonary oedema and hypoxaemia, or refractory heart failure, general anaesthesia is advisable,20 optimally with maintenance of low to normal heart rates and minimal fluctuation in blood pressure, and avoidance of negative inotropes. Remifentanil has been shown to be useful in this setting.21

Invasive monitoring (arterial and central venous lines), are advisable in NYHA Class 3 and 4 patients. In critically ill patients, pulmonary artery catheterisation may have benefits intraoperatively6 and in the postoperative management of pulmonary oedema and/or renal failure.10 In patients with very poor ventricular function, left ventricular assist devices have been used to aid recovery or as a bridge to cardiac transplantation.22, 23

The outcome of PPCM is better than that of other non-ischaemic cardiomyopathies. However, in one recent prospective South African study, 15/100 patients died, and only 23% had normal LV function after 6 months.24 Follow-up should consist of echocardiography, 6 monthly until either recovery or until function reaches a plateau. Medication, either ACE inhibitors or β-blockers, should continue for at least 1 year. Immunosuppressive or immunomodulatory therapy remain controversial. Symptoms of heart failure have been shown to occur in 21% of patients who enter a subsequent pregnancy with normal LV function, and in 44% in whom LV function is abnormal at the start of the subsequent pregnancy.

Summary
- Although rare, anaesthetists should have a high index of suspicion for PPCM in any patient presenting with symptoms compatible with heart failure in the last month of pregnancy.
- A combination of clinical judgement, exclusion of other causes, and investigations (ECG, chest radiograph and echocardiography where available), should point to the diagnosis.
- The cornerstones of stabilisation of the mother include diuretics, vasodilators (within the constraints of individual exclusions in pregnancy), positive inotropes and anticoagulants.
Timing and mode of delivery are a multi-disciplinary decision.

Careful epidural anaesthesia for labour has major benefits.

Anaesthesia for CS may be hazardous, and regional anaesthesia (epidural or CSE) in patients with NYHA class greater than 2 should only be undertaken by experienced practitioners.

In patients of NYHA Class 3 and 4 with PPCM, particularly if complicated by respiratory or renal failure or other co-morbidity, a balanced general anaesthesia technique which includes opioids such as remifentanil, with invasive monitoring and facilities for postoperative ventilation, has a significant role to play.

References


Session 8: Challenges for the Obstetric Anaesthetist
Prevention and treatment of postdural puncture headache

Professor Cynthia Wong
Associate Professor, Chief of Obstetrical Anesthesia,
Northwestern University Feinberg School of Medicine, USA

Prevention and Treatment of Postdural Puncture Headache

Cynthia A. Wong, MD
November 11, 2009

- Postdural puncture headache (PDPH)
  - Incidence
  - Prevention
  - Treatment

Incidence of PDPH after Unintentional Dural Puncture

Risk factors
- Young
- Female
- (Pregnancy)
- Non-obese body habitus

Incidence of PDPH after Unintentional Dural Puncture

Risk factors
- Incidence
  - Needle type
  - Bevel direction
  - LOR to saline vs. air

Incidence of PDPH: Needle Type

- Harrison [long]
- Tunsky [medium]
- Very short Crawford [tined]

Choi PT. Can J Anaesth 2003;50:466
Incidence of PDPH

Needle type | Fluid leak (mL/15 min)
--- | ---
17-g Hustead | 516 ± 319
17-g Tuohy | 405 ± 209
18-g Tuohy | 420 ± 191
20-g Tuohy | 100 ± 112*
18-g Special Sprotte | 360 ± 208
18-g Crawford | 356 ± 121

*P < 0.05 compared to all other needles

Incidence of PDPH: Needle Bevel Orientation

<table>
<thead>
<tr>
<th>Author</th>
<th>N</th>
<th>Lateral</th>
<th>Cephalad</th>
</tr>
</thead>
<tbody>
<tr>
<td>Norris, 1989</td>
<td>1558</td>
<td>1.4% (DP 41)</td>
<td>2.1% (BP 0.05%)</td>
</tr>
<tr>
<td>Richardson, 1999</td>
<td>534</td>
<td>0.4% (DP 15)</td>
<td>0.7% (BP 0%)</td>
</tr>
</tbody>
</table>

*P < 0.05 compared to cephalad

Incidence of PDPH after Unintentional Dural Puncture

- Risk factors
- Incidence:
  - Needle type
  - Bevel direction
  - LOR to saline vs. air

Prevention of PDPH

- Avoid dehydration
- Placement of an intrathecal catheter
Prevention of PDPH

- Avoid dehydration
- ?Placement of an epidural catheter
- ?Avoid 2nd stage pushing

Prevention of PDPH

- Prophylactic saline injection
  - Subarachnoid
  - Epidural

Prevention of PDPH

- Prophylactic blood patch

---

**Incidence of PDPH (BP) with an Intrathecal Catheter**

<table>
<thead>
<tr>
<th>Author</th>
<th>N</th>
<th>Intrathecal</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paech, 2001</td>
<td>100</td>
<td>87% (43%*)</td>
<td>86% (80%)</td>
</tr>
<tr>
<td>Norris, 1990</td>
<td>56</td>
<td>55% (11%)</td>
<td>52% (19%)</td>
</tr>
<tr>
<td>Rutter, 2001</td>
<td>72</td>
<td>71% (50%)</td>
<td>81% (73%)</td>
</tr>
<tr>
<td>Spiegel, 2001</td>
<td>153</td>
<td>70% (53%)</td>
<td>81% (62%)</td>
</tr>
</tbody>
</table>
| Ayad, 2003        | 115 | 30%* (18%*) | 91.9% (81.1%)

*P < 0.05 compared to control

---

**Avoid 2nd stage pushing**

<table>
<thead>
<tr>
<th>Author</th>
<th>N</th>
<th>No Push</th>
<th>Push</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stride, 1993</td>
<td>460</td>
<td>75%</td>
<td>86%</td>
</tr>
<tr>
<td>Angle, 1999</td>
<td>33</td>
<td>10%* (0%*)</td>
<td>74% (57%)</td>
</tr>
</tbody>
</table>

*P < 0.002

---

**Subarachnoid Saline Injection (10 mL)**

<table>
<thead>
<tr>
<th></th>
<th>N</th>
<th>Saline</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Charsley, 2001</td>
<td>54</td>
<td>25%*</td>
<td>62%*</td>
</tr>
</tbody>
</table>

(4%*) (38%*)

*P < 0.05 compared to control

---

**Prophylactic Blood Patch Study: Methods**

- Double-blinded (N = 64)
- Therapy recommendation by protocol
  - VRSP 1 – 3: conservative
  - VRSP 4-6, +ADL: conservative
  - VRSP 4-6, -ADL: therapeutic EBP
  - VRSP > 6: therapeutic EBP
- Followed for at least 5 days

Scavone BM. Anesthesiology 2004
Effect and Treatment of PDPH

Data presented as median (range) unless specified

<table>
<thead>
<tr>
<th></th>
<th>SHAM N = 18</th>
<th>EBP N = 18</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset of PDPH (d)</td>
<td>1.5 (1–6)</td>
<td>2.0 (1–4)</td>
<td>0.57</td>
</tr>
<tr>
<td>Maximum VRSP (0–10)</td>
<td>6 (2–10)</td>
<td>7 (1–9)</td>
<td>0.21</td>
</tr>
<tr>
<td>Inability to perform ADL (n)</td>
<td>13</td>
<td>10</td>
<td>0.69</td>
</tr>
<tr>
<td>Recommend therapeutic EBP (n)</td>
<td>15</td>
<td>11</td>
<td>0.14</td>
</tr>
<tr>
<td>Therapeutic EBP (n)</td>
<td>14</td>
<td>9</td>
<td>0.08</td>
</tr>
<tr>
<td>PDPH alleviated with therapeutic EBP (n)</td>
<td>9</td>
<td>3</td>
<td>0.15</td>
</tr>
<tr>
<td>2nd therapeutic EBP (n)</td>
<td>1</td>
<td>2</td>
<td>0.30</td>
</tr>
</tbody>
</table>

Prevention of PDPH

- Avoid dehydration
- Placement of an epidural catheter
- Avoid 2nd stage pushing
- Saline injection
- Prophylactic blood patch
- Epidural/intrathecal morphine

Prevention of PDPH: Epidural Morphine

- Placebo
- Caffeine 75 mg q6h
- Caffeine 125 mg q6h

Prevention of PDPH: Prophylactic Caffeine

- Placebo
- Caffeine 75 mg q6h
- Caffeine 125 mg q6h

Prevention of PDPH

- Avoid dehydration
- Placement of an epidural catheter
- Avoid 2nd stage pushing
- Saline injection
- Prophylactic blood patch
- Epidural/intrathecal morphine
- Prophylactic epidural Dextran 40 (Salvador, 1992)
- Prophylactic caffeine

Prevention of PDPH

- Postdural puncture headache (PDPH)
  - Incidence after inadvertent dural puncture
  - Prevention
  - Treatment
Differential Diagnosis of Postpartum Headache

- PDPH
- Migraine headache
- Tension headache
- Caffeine withdrawal
- Preeclampsia
- Pneumoencephalos
- Sinusitis

Differential Diagnosis of Postpartum Headache

- Meningitis
- Subarachnoid hemorrhage
- Intracranial hemorrhage
- Subdural hematoma
- Cortical vein thrombosis
- Posterior reversible encephalopathy syndrome (PRES)

Incidence of Postpartum Headache

<table>
<thead>
<tr>
<th>Headache Type</th>
<th>Number (% of postpartum headache)</th>
<th>Incidence of HA:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tension type</td>
<td>146 (28.3%)</td>
<td>39%</td>
</tr>
<tr>
<td>Migrainous</td>
<td>102 (20.9%)</td>
<td></td>
</tr>
<tr>
<td>Mucocutaneous</td>
<td>43 (11.3%)</td>
<td></td>
</tr>
<tr>
<td>Undetermined</td>
<td>31 (8.1%)</td>
<td></td>
</tr>
<tr>
<td>Migraine without aura</td>
<td>24 (6.3%)</td>
<td></td>
</tr>
<tr>
<td>Ovarian puncture</td>
<td>18 (4.7%)</td>
<td></td>
</tr>
<tr>
<td>Cervicogenic</td>
<td>13 (3.4%)</td>
<td></td>
</tr>
<tr>
<td>Migraine with aura</td>
<td>4 (1%)</td>
<td></td>
</tr>
<tr>
<td>Cluster, secondary</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>481 (100%)</td>
<td></td>
</tr>
</tbody>
</table>

Goldszmidt E. Can J Anaesth 2005 N = 985

Incidence of HA: 39%

Treatment of Postdural Puncture Headache

- Conservative
  - Prevent dehydration
  - Oral analgesics
  - Abdominal binder
  - Supine position

Treatment of Postdural Puncture Headache

- Conservative
- Caffeine: temporary

Treatment of Postdural Puncture Headache

- Conservative
- Caffeine
- Sumatriptan: ∅

ACTH for treatment of PDPH

<table>
<thead>
<tr>
<th>Author</th>
<th>Mode</th>
<th>N</th>
<th>ACTH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Foster, 1994</td>
<td>IV</td>
<td>20</td>
<td>70%</td>
</tr>
<tr>
<td>Kshatri, 1997</td>
<td>IV</td>
<td>2</td>
<td>2/2</td>
</tr>
<tr>
<td>Gupta, 1997</td>
<td>IM</td>
<td>48</td>
<td>83%</td>
</tr>
</tbody>
</table>
Treatment of Postdural Puncture Headache
- Conservative
- Caffeine
- Sumatriptan
- ACTH
- Epidural saline: ∅
- Epidural Dextran

Dextran 40 for treatment of PDPH

<table>
<thead>
<tr>
<th>Author</th>
<th>N</th>
<th>Success</th>
</tr>
</thead>
<tbody>
<tr>
<td>Barrios-Alarcon, 1989</td>
<td>56 (28 CS)</td>
<td>100% (within 24 h)</td>
</tr>
<tr>
<td>Aldrete, 1993</td>
<td>13</td>
<td>100% (within 24 h)</td>
</tr>
</tbody>
</table>

?? Neurotoxicity of Dextran??

Gelfoam and Fibrin Glue for Treatment of PDPH

<table>
<thead>
<tr>
<th>Author</th>
<th>Material</th>
<th>N</th>
<th>Success</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ambesh, 1991</td>
<td>Gelfoam 2 (18-g Tuohy)</td>
<td>2</td>
<td>100%</td>
</tr>
<tr>
<td>Gerritse, 1997</td>
<td>Fibrin glue 3 (catheter)</td>
<td>3</td>
<td>100%</td>
</tr>
<tr>
<td>Crul, 1999</td>
<td>Fibrin glue 1 (25-g PP)</td>
<td>1</td>
<td>100%</td>
</tr>
</tbody>
</table>

?? Neurotoxicity of Gelfoam and fibrin glue??

Epidural Blood Patch
- Gold standard
- Only one randomized, double-blinded study

Epidural Blood Patch for Treatment of PDPH

<table>
<thead>
<tr>
<th>Author</th>
<th>N</th>
<th>EBP</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Seebacher, 1989</td>
<td>12</td>
<td>83%*</td>
<td>0%</td>
</tr>
</tbody>
</table>

*P < 0.05 compared to control

Possible Mechanisms of Action of EBP
- Plug dural hole/prevent leak
- Initiate inflammatory reaction in dura
- Acute increase in CSF pressure
Mechanism of Action of EBP
- Plug dural hole/prevent leak
- Initiate inflammatory reaction in dura
- Adenosine receptor inactivation (Raskin, 1990):
  - Acute ↑CSF pressure ➔
  - inactivation of adenosine receptors ➔
  - vasoconstriction ➔
  - ↓headache
  (adenosine receptor activation → vasodilation)

EBP: Success Rate

<table>
<thead>
<tr>
<th>Author</th>
<th>N</th>
<th>1st EBP</th>
<th>2nd EBP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abouleish, 1975</td>
<td>118</td>
<td>105 (89%)</td>
<td>10 (98%)</td>
</tr>
</tbody>
</table>

Factors Associated with Success of Epidural Blood Patch
- Dural puncture needle size

Factors Associated with Success of Epidural Blood Patch
- DP needle size
- Interval from dural puncture to epidural blood patch

Dural Puncture to EBP Interval

Success Rate of Therapeutic Blood Patch
<table>
<thead>
<tr>
<th></th>
<th>N</th>
<th>&lt; 24 h</th>
<th>&gt; 24 h</th>
</tr>
</thead>
<tbody>
<tr>
<td>Loeser, 1978</td>
<td>8 E, 16 L</td>
<td>29%*</td>
<td>96%</td>
</tr>
</tbody>
</table>

Success = partial or complete relief of PDPH
*P < 0.05

Factors Associated with Success of Epidural Blood Patch

Safa-Tisseront V. Anesthesiology 2001

N = 504

OAA: Three-day Course on Obstetric Anaesthesia and Analgesia, London, 9 – 11 November 2009
Factors Associated with Success of Epidural Blood Patch

- DP needle size
- Interval from DP to EBP
- Volume of blood injected

Volume of Epidural Blood Patch

### Volume of Autologous Blood

<table>
<thead>
<tr>
<th></th>
<th>N</th>
<th>Low (10 mL)</th>
<th>High (10-15 mL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Taivainen, 1993</td>
<td>49</td>
<td>68%</td>
<td>66%</td>
</tr>
</tbody>
</table>

Randomized, unblinded

### Volume of Autologous Blood

<table>
<thead>
<tr>
<th></th>
<th>N</th>
<th>10 mL</th>
<th>15 mL</th>
<th>20 mL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wu, 1994</td>
<td>162</td>
<td>98%</td>
<td>98%</td>
<td>100%</td>
</tr>
</tbody>
</table>

Observational study

Factors Associated with Success of Epidural Blood Patch

- DP needle size
- Interval from DP to EBP
- Volume of blood injected
- Spinal level of EBP relative to DP
- Duration in lateral position after EBP
- Avoid Valsalva after EBP

Complications of Epidural Blood Patch

- Back pain
- Nerve root or radicular pain
- Transient neck pain/meningeal symptoms
- Lumbovertebral syndrome
- Impairment of subsequent epidural analgesia/anesthesia
Bibliography

4. Richardson MG, Wissler RN. The effects of needle bevel orientation during epidural catheter insertion in laboring parturients. Anesthesia & Analgesia 1999; 88: 352-6
22. Aldrete JA. Epidural dextran for PDPH. Regional Anesthesia 1993; 18: 325-6
37. Hebl JR, Horlocker TT, Chantigian RC, Schroeder DR. Epidural anesthesia and analgesia are not impaired after dural puncture with or without epidural blood patch. Anesthesia & Analgesia 1999; 89: 390-4
Resuscitation of the neonate has been bedevilled by well-intentioned practice with little evidence base. However, in the last 10 years there have been multicentre controlled trials which have begun to answer some of the urgent questions. In addition, The International Liaison Committee on Resuscitation (ILCOR) has published guidelines for Neonatal resuscitation (1, 2) and these are drawing on what little evidence is available to give advice to frontline clinicians.

It is rare for newborn infants to suffer a primary cardiac arrest. Ventilation alone is usually effective in resuscitating the neonate at any gestation. Self-inflating bags, flow-inflating (anaesthesia) bags, and T-piece devices all may be used to provide effective ventilation after birth. Although non-invasive techniques, such as “bag and mask”, are widely taught, they are not always carried out effectively. Intubation (perhaps after preliminary stabilisation) has the merit of delivering intratracheal surfactant to the extreme preterm infant. Whichever method is used, ventilation is likely to be delivered more consistently if a pressure-monitoring device is incorporated. The best indication of successful ventilation is a prompt increase in heart rate. The role of positive end-expiratory pressure during resuscitation requires further research, particularly in preterm infants, in whom it may protect against lung injury. Atelectotrauma can be damaging to the extreme preterm’s alveolar epithelium.

Should neonatal resuscitation be carried out with air or oxygen? There are very few studies in humans which attempt to answer this question. There is a case for initiating positive pressure ventilation using air, especially since primary apnoea is commoner than terminal apnoea. If the baby remains blue and bradycardic there is no evidence that increasing the inspired oxygen concentration is of benefit, but neither has it been shown to fail! It may be that cardiac compressions would be more effective at this stage. More studies are required to inform our decisions.

The management of meconium associated deliveries has become clearer in the last decade. Suction of vigorous infants at birth does not prevent meconium aspiration syndrome (3). Furthermore suction of meconium “on the perineum” does not improve outcome (4). It is still not known what role suctioning of thick meconium has in the resuscitation of the unresponsive or “depressed” infant, and perhaps we should continue to advocate this intervention.

Cold injury to preterm infants is independently associated with neonatal death. The use of clean polythene bags can largely eliminate evaporative heat losses from the infant’s skin. Consequently every baby should now arrive at the Neonatal Unit with a temperature above 36.5 deg C.

Drugs are rarely required in neonatal resuscitation. Adrenaline can be administered intravenously at a dose of 10 microgm/kg, increasing if necessary, but a dose of 30 microgm/kg should not be exceeded (5). Intratracheal adrenaline has not been shown to be effective but it is possible that this route might be beneficial if higher dosages are used (5).

References:
2) International Liaison Committee on Resuscitation 2005.
Drugs given to a mother during labour may affect the baby either after placental transfer (a direct effect) or via effects on maternal physiology and biochemistry (indirect effects). Clearly the former mechanism is potentially important for drugs that act systemically, while neuraxial analgesia does not depend for its effect on drug in maternal blood, so placental drug transfer is less important, while indirect effects may be more important.

It is widely assumed that any pharmacological analgesia must have adverse effects on the baby, and that unmodified labour is relatively harmless. In fact, it has long been known that painful labour produces adverse changes in maternal physiology and biochemistry (Fig 1) that are potentially harmful to the fetus, while effective analgesia may reverse these changes.1

### Effects of systemic analgesia

Opioids given to provide systemic analgesia in labour produce dose-dependent direct fetal and neonatal effects. Pethidine has been extensively studied (table 1). All neonatal effects are maximum if pethidine is given 4-5 h before delivery, but negligible if given only within 1 h. In much of the research involving neonatal outcome, systemic opioids have been compared with neuraxial analgesia, but a recent randomised comparison of i.v. pethidine with placebo showed that umbilical artery respiratory and metabolic acidosis were more severe with pethidine.2 As systemic opioid analgesia is less complete than neuraxial analgesia, opioids are less able to mitigate the adverse effects of labour pain.

Evidence that diamorphine or remifentanil offer any neonatal advantage over pethidine is conflicting. Risk of neonatal respiratory depression and the need for monitoring remain.3,4

### Table 1. Effects of pethidine on the baby

<table>
<thead>
<tr>
<th>Fetal effects</th>
<th>Neonatal effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>muscular activity ↓</td>
<td>Apgar scores ↓</td>
</tr>
<tr>
<td>aortic blood flow ↓</td>
<td>respiration ↓</td>
</tr>
<tr>
<td>short-term variability ↓</td>
<td>neurobehavioural score ↓</td>
</tr>
<tr>
<td>oxygen saturation ↓</td>
<td>muscle tone ↓</td>
</tr>
<tr>
<td>maternal saturation ↓</td>
<td>suckling behaviour ↓</td>
</tr>
</tbody>
</table>

---

**Fig. 1.** Effects of unmodified labour on maternal physiology and biochemistry.
**Direct effects of neuraxial analgesia**

Adverse direct fetal drug effects are likely only if maternal systemic effects of the drugs given neuraxially reach a detectable threshold. Thus when lidocaine was used to provide continuous epidural analgesia, drowsiness was observed in both mother and baby. Longer-acting local anaesthetics (bupivacaine, ropivacaine, levobupivacaine), correctly sited, are more slowly absorbed, and systemic effects are usually observed only after accidental i.v. administration. Although opioids are well recognised to produce direct fetal and neonatal depression and to impair breast-feeding when used systemically, their potential to do so when used for neuraxial analgesia is less, though still present with large doses (see below).

**Indirect effects of neuraxial analgesia**

Neuraxial analgesia may cause maternal changes that can affect the baby (Table 2). Focus on the potentially adverse effects may prompt the belief that neuraxial must be the worst type of analgesia for the baby, but this ignores its potentially favourable effects. Neonatal outcome depends on the balance between the two. No assumptions should be made as to overall neonatal effect without direct neonatal assessment.

<table>
<thead>
<tr>
<th>Potentially unfavourable effects</th>
<th>Potentially favourable effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal hypotension</td>
<td>Reduced maternal stress and hyperventilation</td>
</tr>
<tr>
<td>Maternal fever</td>
<td>(reversal of all factors in fig 1)</td>
</tr>
<tr>
<td>Increased need for oxytocin</td>
<td>Uterine vasodilatation (sympathetic blockade)</td>
</tr>
<tr>
<td>Prolonged second stage</td>
<td>Fewer episodes of haemoglobin desaturation</td>
</tr>
<tr>
<td>Increased need for instrumental delivery</td>
<td>Placental drug transfer is unimportant</td>
</tr>
</tbody>
</table>

**Assessment of the baby**

Even fetal effects are poor predictors of neonatal outcome.

**Fetal heart rate**

Despite its shortcomings, cardiotocography is often considered mandatory for mothers receiving neuraxial analgesia.

In consequence loss of short-term variability, decelerations and major bradycardia may be noted, although meta-analysis of controlled trials shows that fetal heart rate abnormalities are not increased by epidural compared with systemic opioid analgesia, or by epidural fentanyl, though they are increased by intrathecal opioids. Fetal bradycardia may be associated with the rapid onset of analgesia, and various mechanisms have been considered to explain this.

- Some bradycardias are associated with uterine hypertonus, which may result from a decline in circulating epinephrine concentration, itself a myometrial inhibitor.
- In theory, a reduction in epinephrine would allow unopposed norepinephrine to cause uterine vasoconstriction.
- Direct fetal effects from opioids (unlikely with small intrathecal doses).
- Maternal hypotension, reduced cardiac output, hypoventilation.
- Omission of local anaesthetic; sympathetic blockade causes uterine vasodilatation.

- Omission of fluid preload. An i.v. fluid load may inhibit uterine contractions and reduces the incidence of fetal heart decelerations.
- An external tocodynamometer belt is commonly removed before siting an epidural. Until the belt is replaced, once analgesia is achieved uterine contractions may be undetected.
- The obstetrician may be keen to continue the administration of oxytocin. This should be resisted during epidural insertion, to avoid undetected uterine hypertonus.

Provided the bradycardia is brief and does not reflect poor placental perfusion resulting from uterine hypertonus or vasoconstriction, maternal hypotension or aortocaval compression, its presence is claimed to be irrelevant to fetal outcome, though more attention has been paid to caesarean section rate than to actual neonatal welfare.

**Fetal blood flow**

Early Finnish studies using radio-Xenon suggested that, provided maternal aortocaval compression and hypotension were avoided, maternal placental flow rose following epidural local anaesthetic, particularly in the presence of preeclampsia. Since then, Doppler flow technology, a better predictor than cardiotocography of neonatal wellbeing, has spawned numerous studies, some again showing favourable changes with epidural analgesia in preeclampsia but none demonstrating any
correlation with neonatal outcome. One study purports to demonstrate increased flow resistance with epidural analgesia, but, as there were no controls, the findings could simply reflect changes associated with labour.

**Apgar score**

Apgar score reflects the intrauterine environment but applies only to the first few minutes of life, a stimulating time for the newborn, so it may fail to detect delayed neonatal depression. Meta-analysis of controlled trials showed that there were significantly fewer low Apgar scores at one and five minutes with epidural than with systemic opioid analgesia.

**Neurobehavioural assessments**

Over the years various neurobehavioural tests have therefore been developed to assess the newborn further into the post-natal period. The most used by anaesthetists, the neurological and adaptive capacity score (NACS), may not be very sensitive or reliable, although it tends to correlate with breast feeding (Table 3). Meta-analyses of randomised trials revealed no significant difference in NACS between epidural and systemic opioid analgesia, though higher doses of epidural fentanyl may be associated with lower scores.

**Acid-base balance**

Umbilical artery acid-base balance is a valid index of the recent fetal environment. Fetal pH is increased by maternal hyperventilation, so base excess is a better marker of fetal metabolic status in painful labour. In 1974, three studies found that, whereas normal labour was associated with a progressive deterioration in base excess in mothers and babies, epidural analgesia appeared to mitigate this adverse effect, particularly in long labour. Meta-analysis of controlled trials conducted since then, comparing umbilical acid-base values after epidural and systemic analgesia, showed that both UA pH and base excess are better following epidural than systemic analgesia. The same neonatal benefit is seen when comparing epidural with no analgesia. This beneficial effect applies to both old-fashioned epidurals with local anaesthetic alone, and modern low-dose combinations.

Neuraxial analgesia comes in many guises, that have only minor neonatal impact. One randomised trial showed that umbilical artery pH was significantly lower following single-shot spinal analgesia using a low-dose bupivacaine-opioid combination, than with paracervical bupivacaine. Combinations including clonidine or ephedrine also have significant adverse neonatal effects.

**Breast feeding**

The numerous unrandomised trials that have been published demonstrate variable effects of neuraxial analgesia on breast feeding (Table 3), but only those purporting to show adverse effects receive much publicity. The many confounding variables (intention to breast-feed, parity, age, local tradition, social class, education, delivery type and the amount of help and support offered) make results of unrandomised trials unreliable. One observational study and one randomised trial, however, have suggested (perhaps unsurprisingly) an adverse effect on breast feeding of large epidural doses of fentanyl. More evidence is needed. Meanwhile, when prolonged epidural analgesia for labour using a local anaesthetic-opioid combination is extended for emergency caesarean section, it is unwise for the baby’s sake to give further opioid either epidurally or systemically.

**Maternal anaesthesia**

While neuraxial analgesia in labour has a favourable neonatal effect (usually ignored), and spinal anaesthesia is preferred for the mother undergoing caesarean section for many reasons, it cannot be assumed to be better also for the baby. General anaesthesia sometimes depresses the Apgar score, but only at one minute. Traditional spinal anaesthesia, despite lack of maternal sedation, small dose requirement and careful attention to maternal haemodynamics, is associated with adverse fetal flow changes and more neonatal acidosis than either general or epidural anaesthesia, an effect only partly explained by the use of ephedrine with spinals. Using low-dose local anaesthetic-opioid combinations and phenylephrine as a vasopressor may improve neonatal outcome after spinal anaesthesia. There is no advantage to the baby in using a combination of ephedrine and phenylephrine, rather the reverse. The upshot is that it is better to use general than spinal anaesthesia, but rather that if, for any reason, a mother is compelled to receive general anaesthesia, she should be reassured that it is not more damaging to the baby than neuraxial anaesthesia.

**References**

3. Rawal N, Tomlinson AJ, Gibson GJ, Sheehan TM. Umbilical cord plasma concentrations of free morphine following single-dose diamorphine analgesia and their...


<table>
<thead>
<tr>
<th>Type of study</th>
<th>Epidural type (n)</th>
<th>Controls (n)</th>
<th>Outcome measures</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rajan 1994</td>
<td>Various</td>
<td>No epidural</td>
<td>Questionnaire at 6 weeks, breast, bottle or a mixture</td>
<td>Epidurals and Entonox no effect on breast feeding. Abnormal delivery, induction, short second stage, general anaesthesia and pethidine reduced it</td>
</tr>
<tr>
<td>Halpern 1999</td>
<td>CSE or epi bup+suf or fentanyl (113)</td>
<td>No epidural (74) (some had systemic opioids)</td>
<td>Telephone interview: breastfeeding success at 6 weeks</td>
<td>72% fully breast-feeding at 6-8 weeks. No factors relating to labour analgesia had any significant effect on feeding when leaving hospital or 6 weeks later</td>
</tr>
<tr>
<td>Albani 1999</td>
<td>n/k</td>
<td>No analgesia</td>
<td>Feeding at discharge</td>
<td>Vaginal: epidural 96.5% v. controls 97.8% (NS) caesarean section: regional 95% v. general anaesthesia 85% (P=0.002)</td>
</tr>
<tr>
<td>Riordan 2000</td>
<td>Various drugs and doses, usually bup+fent (27)</td>
<td>No mediation (37); i.v. (52); both (13)</td>
<td>IBFAT, LATCH assessment not properly blinded. Duration of breast-feeding at telephone at 6 weeks</td>
<td>LATCH score better with no medication. IBFAT score no med&gt;i.v.=epidural&gt;both. Low IBFAT = shorter feeding but unmedicated did not feed longer than the rest.</td>
</tr>
<tr>
<td>Ransjo-Arvidson 2001</td>
<td>Epidural bupivacaine or pethidine (?)systemic or combination (12)</td>
<td>No analgesia (10); mepivacaine pudendal block (6);</td>
<td>Video of infant behaviour immediately after delivery, assessed blindly. Age and duration of first suckling</td>
<td>Unmedicated infants more active</td>
</tr>
<tr>
<td>Radzyminski 2003</td>
<td>Ultra low dose bupivacaine + fentanyl (12)</td>
<td>No analgesia</td>
<td>Premature infant breast feeding behaviour scale at birth and at 24 h</td>
<td>No significant difference between groups in breast-feeding behaviour</td>
</tr>
<tr>
<td>Henderson 2003</td>
<td>PCEA bup+fent 5 µg/ml (690)</td>
<td>Support + N₂O + peth (302)</td>
<td>Timing + quality of 1st feed. Duration of feeding or still feeding at 6/12</td>
<td>Still feeding at 6/12: epidural 38%; controls 51%. Factors favouring longer breast feeding: tertiary education&gt;older mothers&gt;non-smoker &gt;no epidural</td>
</tr>
<tr>
<td>Baumgardner 2003</td>
<td>Not stated (115)</td>
<td>No epidural (116)</td>
<td>2 successful feeds in 24 h LATCH score</td>
<td>Success in 24 h: epidural 69.6%; controls 81%; OR 0.53 (NS). Odds of success increased with duration of epidural, to &gt;1!</td>
</tr>
<tr>
<td>Volmanen 2004</td>
<td>Bupivacaine (30) +occasional fentanyl (8)</td>
<td>No epidural (34)</td>
<td>Postal questionnaire Fully breast-feeding in 1st 12 weeks</td>
<td>Fully breast-feeding: epidural 33%; controls 71% (usual reason for failure: not enough milk) Factors favouring full breast feeding: younger mothers, no epidural, NOT rooming in, early skin contact, education.</td>
</tr>
<tr>
<td>Author</td>
<td>Year</td>
<td>Study Type</td>
<td>Interventions</td>
<td>Breast-feeding outcomes</td>
</tr>
<tr>
<td>-----------------</td>
<td>----------</td>
<td>----------------</td>
<td>-------------------------------------------------------------------------------</td>
<td>------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Chang 2005</td>
<td>prospective cohort; mixed parity</td>
<td>Not stated (52)</td>
<td>No analgesia 63</td>
<td>8-12 h initiation (LATCH) 4/52 continuation + NACS 8-12 h</td>
</tr>
<tr>
<td>Wang 2005</td>
<td>observational parity n/k</td>
<td>Not stated (96)</td>
<td>No epidural 74</td>
<td>Analgesia; mental state; time starting lactation; milk quantity; prolactin</td>
</tr>
<tr>
<td>Jordan 2005</td>
<td>retrospective cohort nullipara</td>
<td>Neuraxial (232) [containing opioid 158]</td>
<td>N₂O and/or i.m. pethidine (570)</td>
<td>Feeding on hospital discharge in discharge summary (exclusive or partial breast-feeding cf bottle-feeding)</td>
</tr>
<tr>
<td>Beilin 2005</td>
<td>randomised double-blind multiparae who had previously breast-fed</td>
<td>Infusion of bupivacaine with low-dose fentanyl (LF, n=59) or high-dose (HF, n=57)</td>
<td>Epidural infusion: bupivacaine alone (OF, n=60)</td>
<td>Day 1: maternal questionnaire, lactation consultant assessment, NACS. Telephone enquiry 6 weeks</td>
</tr>
<tr>
<td>Torvaldsen 2006</td>
<td>prospective cohort; singleton mixed parity. No selection of those intending to breast-feed</td>
<td>PCEA bup+ fent 3.3 µg/mL, ALL had i.m. pethidine (416)</td>
<td>Non-pharmacologic/ N₂O/pethidine (762)</td>
<td>Questionnaires on discharge and 8, 16 and 24 weeks: fully, partial or not breast-feeding. Hazard ratio of stopping feeding in 24 weeks</td>
</tr>
<tr>
<td>Willund 2009</td>
<td>retrospective all epidurals; controls matched for parity, age and gestation</td>
<td>Usually bup+sufentanil bolus or infusion ± paracervical and/or pudendal block (351)</td>
<td>Nothing or paracervical and/or pudendal block No opioid (351)</td>
<td>Breast feeding in 1st 4 h; artificial milk given; breast feeding at discharge</td>
</tr>
<tr>
<td>Wilson 2009</td>
<td>COMET plus no-epidural controls matched for parity, del type and ethnicity</td>
<td>CSE+low-dose bup+fent bolus (351) or LDI: low-dose bup+fent infusion (350) or high-dose bup bolus (353)</td>
<td>Pethidine (151) or no analgesia (200)</td>
<td>Interviewed 24-48 h ?initiated breast feeding. Postal questionnaire 12 months: duration of breast feeding</td>
</tr>
</tbody>
</table>

Breast-feeding effectiveness α NACS, no difference between groups. Still feeding 4/52: epidural 86%, controls 81% (NS)

Epidural better analgesia and mental state; quicker lactation; more milk; higher prolactin

Bottle-feeding on discharge: N₂O only: 32%; i.m. pethidine: 41%; neuraxial local anaesthetic alone: 44%; neuraxial with opioid 53% (NS). Final logistic regression model predictors of bottle-feeding: maternal age, feeding intention, caesarean section and fentanyl (dose-dependent ↑ bottle feeding)

Maternal assessment at 24 h correlated with NACS and subsequent performance, lactation consultant’s did neither. Not breast-feeding at 6 weeks: 0F: 2%; LF: 6%; HF: 19% (P=0.002)

Only less educated did not breast-feed at all. Predictors of partial breast-feeding 1st week: young mothers<less education<delivery type & analg. GA>epi>N₂O=none>pethidine! After adjusting for delivery type and parity, epidural effect NS. Breast-feeding at 24 weeks: no analg 72%; no epidural 64%; epidural: 52%. Predictors of stopping in 24 weeks: young<education<analg

Babies in the epidural group were less likely to breast feed in the first 4 h (OR 3.79), more likely to be given artificial supplement (OR 2.19) and less likely to be breast feeding at discharge (OR 1.79)

Number initiating breast feeding: all epidural groups and no-pethidine group NS; pethidine group lower initiation rates. Duration NS
**Session 10: Pre-eclampsia Revisited**

**Obstetric issues**

Dr Maggie Blott  
*Consultant Obstetrician, University College London Hospitals*

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**Pre-eclampsia revisited obstetric issues**

Maggie Blott  
*Consultant Obstetrician, University College Hospital London*

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**Areas to discuss**

- Aetiology & pathogenesis
- Prediction & prevention
- Antenatal management
- Timing of delivery

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**Pre-eclampsia – Global**

- >4 million women worldwide
- >100,000 cases of eclampsia
- 90% in developing countries
- 40,000 women die/year

**Pre-eclampsia**

- 1.8 stillbirths
- Fetal and maternal mortality highest
- Early onset disease
- Severe essential hypertension
- 12,000 pregnancies
- 20% special care cots
- 2% of all pregnancies
- 5-7% of primiparous

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**Pathogenesis**

- Two theories
  1. Two Stage Process
     a. Abnormal trophoblast invasion
     b. Poor placental perfusion
  - Release of circulating factors
  - Abnormal endothelial function
  - Clinical syndrome
  2. Altered immune response – ‘pro-inflammatory’

**Risk Factors for Developing Pre-eclampsia**

- **Risk Factors**
  - Maternal factors: multiparity, pre-existing hypertension, previous pre-eclampsia, severe preeclampsia in previous pregnancy, older age
  - Fetal factors: macrosomia, oligohydramnios, growth restriction
  - Placental factors: placentomegaly, abruption, insufficiency

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**Pathogenesis**

- Risk Factors
  - Fetal: oligohydramnios, growth restriction
  - Maternal: hypertension, proteinuria
  - Placental: insufficiency, infarction

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**Pathogenesis**

- Risk Factors
  - Fetal: oligohydramnios, growth restriction
  - Maternal: hypertension, proteinuria
  - Placental: insufficiency, infarction

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**Genetic factors**

- First-degree relative with pre-eclampsia
- Partner previously delivered premature baby

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**Personal medical history**

- Obesity
- Chronic renal disease
- Chronic hypertension
- Diabetes mellitus
- Thrombocytopenia
Trophoblast invasion

- CTBs invade and extend through the decidua and inner third of myometrium.
- Remodeling of the spiral arteries probably begins in the late first trimester and is completed by 18-20 weeks of gestation.

Soluble angiogenic factors – ‘sFlt-1’

- Produced from placenta – a receptor that binds:
  - PlGF (Placental Growth Factor)
  - VEGF (Vascular Endothelial Growth Factor)
- sFlt-1 ↑ in months 8-9 in normal pregnancy
- Significant ↑ in preeclampsia
- Changes are detectable 5 weeks before clinical disease

So what does sFlt-1 do?

1. When present in circulation, sFlt-1 BINDS to VEGF and PlGF
2. Prevents them from binding to cell surface receptors
3. Leads to a state of ENDOTHELIAL DYSFUNCTION

Prediction

UTERINE ARTERY DOPPLER VELOCIMETRY

- Bilateral UA ‘notching’
- Best PPV – 21% for proteinuric PIH
- RR 26 (11-64) for ‘severe PE & delivery <34 wks’
‘sFlt-1’ – A Predictive Role?

Stepan et al. NEJM 18 Nov 2004

Prior studies

Number of women with pre-eclampsia

Intention-to-treat

Completed study

Placebo

Vitamins C and E

Adjusted OR: 0.39 (0.17-0.90) 0.24 (0.08-0.70)

p=0.02 p=0.002

Aspirin in pre-eclampsia

• Meta analysis >32,000 women & babies

<table>
<thead>
<tr>
<th>Treatment</th>
<th>n</th>
<th>n/N</th>
<th>Number</th>
<th>Relative risk (95% CI)</th>
<th>Relative risk (95% CI)</th>
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<tbody>
<tr>
<td>Pre-eclampsia</td>
<td>103/15481</td>
<td>139/14934</td>
<td>24</td>
<td>0.90 (0.84-0.97)</td>
<td></td>
</tr>
<tr>
<td>Delivery ≥24th gestation</td>
<td>101/13767</td>
<td>111/1252</td>
<td>26</td>
<td>0.90 (0.84-0.97)</td>
<td></td>
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<tr>
<td>Total baby death before discharge</td>
<td>48/15181</td>
<td>53/15680</td>
<td>25</td>
<td>0.90 (0.84-0.97)</td>
<td></td>
</tr>
<tr>
<td>Neonatal death or hypotension</td>
<td>154/17872</td>
<td>164/18164</td>
<td>20</td>
<td>0.90 (0.80-1.00)</td>
<td></td>
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<tr>
<td>Preeclampsia or severe preeclampsia</td>
<td>85/9864</td>
<td>126/9868</td>
<td>12</td>
<td>0.90 (0.80-1.00)</td>
<td></td>
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</table>

Antenatal Management

1. Screen for women at high risk
2. Interventions
3. Medical management
4. Timing of delivery
5. Mode of delivery

NICE Guidelines – ‘Low risk’ Antenatal Care

OAA: Three-day Course on Obstetric Anaesthesia and Analgesia, London, 9 – 11 November 2009

135
Munjuluri BMJ 2005

Treatment for mild-moderate hypertension in late pregnancy (15 trials) Magee et al 1999

Eclampsia in the UK - 2005

M Knight (UKOSS) 2007

When to deliver the baby?

Hyaline membrane disease

NEC

Mode of Delivery

Caesarean section

Induction of labour

Conclusions I

- Beware of early onset pre-eclampsia
- Need to recognise and treat high SBP
- Early involvement of Consultant Obstetrician
- Watch for fluid overload
- Substandard care in hospital & community
- Institute PRECOG and NICE
- Beware of late-onset eclampsia

Munjuluri BMJ 2005

Consequences for women’s health

Threshold for vascular or metabolic disease

Complicated pregnancy e.g. pre-eclampsia, gestational diabetes

Satтар & Greer BMJ 2002

When to deliver the mother?

Renal failure

Placental abruption

Conclusions II

- Be aware of atypical/presentations
- Prediction is possible in various risk groups
- The future - consider the long-term health implications of pre-eclampsia
- The future - prevention with aspirin, calcium (but not anti-oxidant vitamins)
- Perhaps the complex mechanisms underlying preeclampsia are becoming clearer.....
Session 10: Pre-eclampsia Revisited
Medical management of pre-eclampsia

Dr Lucy Mackillop
Locum Consultant Obstetric Physician, John Radcliffe Hospital, Oxford

Medical Management of Pre-eclampsia

Lucy Mackillop
Locum Consultant Obstetric Physician
John Radcliffe Hospital
Oxford, UK

Leading causes of direct deaths
CEMAC, 2003-05

Pre-eclampsia – potential crises

- Cerebral haemorrhage (10/18)
- Eclampsia (6/18)
- DIC
- HELLP (5/18)
- Renal failure
- Hepatic failure / liver rupture
- Pulmonary oedema
- Cortical blindness
- Placental abruption
- IUD

Goals of Antenatal Management

Avoid maternal complications
- Control hypertension
- Early detection of complications
- Prevention of thromboembolism

Improve fetal survival & prognosis
- Prolong pregnancy
- Steroids for fetal lung maturity
- Regular Ultrasound to assess growth, arterial & venous dopplers and amniotic fluid index

Deliver if:

- Fetal syndrome
  - survival ex-utero > in-utero
- Complications with maternal disease
  - Fulminating pre-eclampsia
  - Placental abruption
  - Eclampsia
  - HELLP syndrome
  - Worsening renal function
  - Pulmonary oedema
  - Uncontrollable hypertension despite 3 agents

Maternal mortality from eclampsia and pre-eclampsia

Source: Confidential enquiries into maternal deaths, Figure 3.1, OAA Report 2009

Fulminating Pre-eclampsia

Visual disturbances
Headache
RUQ/epigastric pain

Pre-eclampsia protocol
Obstetric High Dependency Unit
Deliver baby

Pre-eclampsia Protocol

Blood Pressure Control
Fluid Management
Seizure Prophylaxis

Hydralazine
Fluid restrict to 85ml/h
MgSO4 loading & maintenance

Complications in PET

Stroke
Eclampsia
Pulmonary Oedema
Renal Failure
HELLP Syndrome

Yorkshire Critical Care Group

• n = 210,631; 16 units; 1999 - 2003.
• 1087 severe pre-eclampsia or eclampsia (5.2/1000)
• 151 serious complications
  – 82 (39/10,000) having eclamptic seizures and
  – 49 (23/10,000) requiring ICU admission.
• 82 eclampsia
  – 45 occurred antenatally (55%)
  – 18 before admission to the maternity unit
  – 11 in labour (13%)
  – 26 following delivery (32%).
• 25 pulmonary oedema (2.3% of cases)
• 6 renal dialysis (0.55% of cases).

Stroke in Pre-eclampsia

n = 18 deaths due to pre-eclampsia / eclampsia
– 10/18 due to ICH, 2/10 due to cerebral infarction

Mean rise in BP baseline
pre stroke

Mean rise in BP baseline
pre stroke

Systolic 64.4 mmHg
Diastolic 30.6 mmHg

Seizure prophylaxis to reduce the risk of eclampsia

Hypertension in Pre-eclampsia

• Pressor affects of intubation
• Active management of 3rd stage – avoid ergometrine

Blood Pressure control to reduce the risk of stroke

Mean rise in BP baseline
pre stroke

Systolic 64.4 mmHg
Diastolic 30.6 mmHg
Eclampsia

- Affects 1 in 2000 deliveries/1-2% of women with pre-eclampsia
- ~30% precedes hypertension/proteinuria
- up to 50% postnatal
- 2.2% mortality

MAGPIE

MgSO₄ for prevention of eclampsia
- 175 centres, 33 countries, 10,141 women

<table>
<thead>
<tr>
<th>MgSO₄</th>
<th>placebo</th>
<th>RR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eclampsia</td>
<td>0.8%</td>
<td>1.9%</td>
</tr>
<tr>
<td>Death</td>
<td>0.2%</td>
<td>0.4%</td>
</tr>
</tbody>
</table>

Halves rate of eclampsia

MAGPIE

No difference in rate of:
- baby death / SCBU
- CS (ie. not a tocolytic)
- Severe morbidity
- Antihypertensive therapy
24% of MgSO₄ side effects eg. flushing / N+V

Need to treat 1000 women to prevent 11 cases
NNT = 70 for severe pre-eclampsia, 90-100 for less severe pre-eclampsia

Renal failure in pre-eclampsia

- 1.1%
- Cohort study 1995-1998 Groote Schuur, Cape Town 28000 del/yr → 588 admitted to obstetric HDU
- 89 severe PET + ARF (obtained 72 case notes)
  - Cr>100+ oliguria <100ml/4hrs
  - Median max creatinine = 341 μmol/l

Renal failure in PET

- 9 yrs, n = 37 (78% black)
- Incidence: 2.9% (older, multips)
- Antenatal 30%
- Postnatal 70%
- Maternal Mortality 11%
- Fetal Mortality 53%

Dialysis: 1:13500

Short term <2wk: 10% (3/4 HELLP)
Long term dialysis 0%
Renal Transplant 0%

Predictor: creatinine doubles in 1st 1-2d post admission

Pulmonary oedema in PET

- 9 yrs, n = 37 (78% black)
- Antenatal 30%
- Postnatal 70%
- Maternal Mortality 11%
- Fetal Mortality 53%

Drakeley et al. AJOG 2002; 186: 253
**HELLP - Diagnosis**

- Low grade haemolysis
- Raised LDH > 600 U/L
- Raised unconjugated bilirubin
- Abnormal LFTs
- Raised transaminases AST ≥ 70 U/L
- Low / falling platelets (< 100,000)
- DIC (20%)

**Radiology**

- Hepatic infarcts
- Subcapsular haematoma

**Liver Biopsy**

- Necrosis
- Haemorrhage

---

**Maternal morbidity in HELLP**

- n = 170
  - ARF 13.5%
  - Abruption 6.6%
  - Pneumonia 3%
  - Liver haematoma 2.3%
  - Pulmonary oedema 2.3%
  - DIC 1.7%
  - Cerebral haemorrhage 1.2%

  Morbidity increased with lower platelet counts

  *Romero Arauz et al. Ginecol Obstet Mex 2001; 69: 189*

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**Corticosteroids in HELLP**

- 5 studies (n=170) – 3 antepartum, 4 vs placebo
  - Dexamethasone (12 mg iv 12 h) superior to betamethasone
  - No difference in maternal mortality/morbidity
  - (abruption, pulmonary oedema, liver haematoma/rupture)
  - Reduced hospital stay (-4.5 [-7.1,-1.9] day)
  - Longer interval to delivery (41 vs 15 h p=0.007)
  - Tendency to greater platelet count increase over 48h (40.6 [-26.2, 107.3])
  - Greater mean birthweight (247 [65,428] g)

*O’Brien JM et al. Am JOG 2002; 186:475-9*

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**RCT Steroids for HELLP syndrome**

- N = 132, dexamethasone 10 mg 12hrly + 3 doses postpartum
- Non significant shortening of hospitalization 6.5 vs 8.2 days
- No difference in
  - time to recovery of platelet counts
  - lactate dehydrogenase
  - aspartate aminotransferase
  - development of complications
- Results were found in both pregnant and puerperal women.

*Fonseca JE et al. Am JOG 2005; 193: 1587*

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**Effect of corticosteroids on regional anaesthesia rates**

69 patients with HELLP

<table>
<thead>
<tr>
<th>Mean fall plts (ad to del)</th>
<th>4 x 10⁹</th>
<th>15 x 10⁹</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. with ↑ plts</td>
<td>15</td>
<td>1</td>
</tr>
<tr>
<td>Mean plts at deliv</td>
<td>88 x 10⁹</td>
<td>72 x 10⁹</td>
</tr>
<tr>
<td>Regional anaesthesia (%)</td>
<td>43%</td>
<td>35%</td>
</tr>
</tbody>
</table>

*O’Brien JM et al. Am JOG 2002; 186:475-9*

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**Effect of corticosteroids on regional anaesthesia rates**

37/69 patients with HELLP and plts < 90 x 10⁹

<table>
<thead>
<tr>
<th>Regional anaesthesia</th>
<th>Steroid (42%)</th>
<th>no steroid (0%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>GA for CS</td>
<td>46%</td>
<td>100%</td>
</tr>
<tr>
<td>24 hr latency</td>
<td>54%</td>
<td></td>
</tr>
<tr>
<td>RA if 24 hr latency</td>
<td>57%</td>
<td>0%</td>
</tr>
<tr>
<td>GA if 24 hr latency</td>
<td>22%</td>
<td>100%</td>
</tr>
</tbody>
</table>

*O’Brien JM et al. Am JOG 2002; 186:475-9*
Blood Pressure Control

**Chronic**
- Methyl dopa
- Nifedipine, hydralazine
- Labetalol, alpha blocker

**Acute**
- IV labetalol
- IV hydralazine
- Oral nifedipine (not with MgSO₄)

**Drugs not used**
- ACEI
- ARB
- Diuretics

Keep BP < 160/100

Fluid Management

- Post delivery oliguria is common
  - ADH↑/Angiotensin II↑/ANP↑↓
  - Oxytocin
  - Vasodilators
  - Low albumin
  - ‘Third space’ losses
  - Non-steroidal anti inflammatory drugs

- Anuria is a blocked catheter or obstructed ureters until proved otherwise
- Measure Mg levels if on MgSO₄

Fluid Management

- **Assess patient first**
  - Clinical – Thirst, JVP, Hand veins, Lung bases
  - Non-invasive monitoring - Pulse oximeter, chest x-ray
  - Invasive monitoring - CVP (2-6)
  - Urine vol <0.5ml/kg/hr over 4 consecutive hours (80 mls/4hr)
  - Check biochemistry, exclude DIC, blood loss

- **Reassess patient**
  - Hartmann’s @ 1ml/kg/hr if not taking oral fluids
  - Do not use dextrose unless diabetic
  - No advantage of colloid, albumin and blood unless abnormal losses

- **Repeat assessment at least every 4 hours**

Seizure Control & Prevention

**Acute Management of seizure**
- Lorazepam 2mg IV
- MgSO₄ Infusion

**Prevention**
- MgSO₄
- IV 4g loading dose (over 5 minutes)
- IV infusion 1g/hr
- 2-4g for recurrent seizures
- Monitor level if:
  - Oliguria
  - Renal impairment
  - Signs of toxicity
  - Low BMI

**HELLP - Management**

- **Control BP**
- **Fluid restrict**
- **Correct coagulopathy (FFP ± Platelets)**
- **Deliver**

- Steroids of no benefit
**Additional management**

- Thromboprophylaxis
- No NSAIDs

**Postpartum Hypertension**

- BP rises after normal delivery, reaching a peak at 3-4 days
- Methyldopa should be stopped / avoided
- Atenolol / Calcium antagonist / ACEI

**Summary**

**Pre-eclampsia**

- is common and dangerous
- always surprises
- Endothelial damage leads to a multi-system disorder of protean manifestations
- requires clear and agreed management plans
- should be managed on the labour ward
Session 10: Pre-Eclampsia Revisited
Anaesthetic Issues

Dr Robin Russell
Consultant Anaesthetist, John Radcliffe Hospital, Oxford

Hypertensive disease in pregnancy remains one of the leading causes of maternal mortality in the UK. In the 2003-05 CEMACH report there were 18 deaths directly attributed to eclampsia or pre-eclampsia, a small increase from the previous report. Although the number of deaths has fallen in recent enquiries, nearly half of the reported cases showed features of substandard care.

**Antenatal Assessment**
The 2000-02 CEMACH report highlighted the need for early diagnosis and treatment of hypertension. There was a call for clear written protocols and input from senior obstetricians, anaesthetists and intensive care specialists. Poor communication and teamwork, and a lack of appreciation of the severity of illness, with failure to involve senior staff, have been recurring themes in both CEMACH and CEMD reports. For effective multi-disciplinary care, women with significant co-existing disease should be referred for anaesthetic assessment as early as possible during pregnancy. Those with hypertensive disease are more likely to require the services of an anaesthetist during labour and delivery and early consultation allows time to assess the severity of illness and current management following which options for analgesia and anaesthesia can be discussed. A recent claims that antepartum continuous infusion of epidural ropivacaine reduces uterine artery vascular resistance warrants further investigation.

The 2003-05 report emphasised the need to control blood pressure adequately before inducing general anaesthesia for caesarean section (see below) especially when there was a desire to delivery the baby urgently.

**Labour Analgesia**
Regional analgesia is recommended for women with hypertensive disease. It provides superior pain relief when compared to systemic or inhalational analgesia, and prevents further surges in blood pressure associated with autonomic activity. Epidural analgesia promotes uteroplacental blood flow and results in improved umbilical blood gases. Furthermore, it can be readily extended to provide regional anaesthesia for instrumental delivery or caesarean section.

Regional blocks are contraindicated in the presence of abnormal coagulation as vascular trauma with either needle or catheter has the potential to cause neurological damage, although in the obstetric population this is extremely rare. As thrombocytopenia is a recognized feature of hypertensive disease a recent platelet count should be performed to assess suitability of regional analgesia. Ideally this should be taken on admission to the labour ward and, for those with severe disease, repeated every six hours. Both the absolute number and rate of decline guide the anaesthetist to the risk of spinal haematoma. If the total platelet count is below 75 x 10^9/L or the number has dropped by more than 50 x 10^9/L in the previous 12 hours, many anaesthetists would avoid performing a regional block. Faced with such results the risks and benefits should be assessed on an individual basis by senior clinicians. Despite the publication of a number of case reports in which neuraxial blocks have been placed without complication in thrombocytopenic patients the risk of spinal haematoma must not be overlooked.

Risk assessment should include history, examination, and evaluation of blood tests. Clotting studies are often performed when the platelet count falls below 100 x 10^9/L but are frequently normal. Bleeding times are not considered to be of benefit. Thromboelastography (TEG) measures whole blood coagulation although does not take into account the initial reaction between platelets and vessel wall. Changes in the maximum amplitude on the TEG are seen in pre-eclamptic women with platelet counts below 100 x 10^9/L. However, a normal TEG cannot predict the safety of regional anaesthesia in the presence of pre-eclampsia. Platelet function analyzers have so far failed to gain widespread popularity in clinical practice, although in severe pre-eclampsia they have been shown to detect impaired haemostatic function. Further assessment of coagulation is also necessary before removing an epidural catheter as there is again a risk of vascular damage and haematoma.

**Anaesthesia for Delivery**
Women with pre-eclampsia are more likely to require caesarean section. The decision whether to use regional or general anaesthesia is based both on the severity of pre-eclampsia and the urgency of delivery. Regional anaesthesia is usually preferred as it is associated with reduced maternal mortality and
morbidity. General anaesthesia is reserved for cases where regional block is contraindicated (see below), has failed or there is inadequate time to perform or extend a block.

**General Anaesthesia:** In the presence of coagulopathy, pulmonary oedema or impaired consciousness, general anaesthesia is usually preferred. Pre-operative assessment should be as thorough as time permits. Difficult intubation should be anticipated. Upper airway narrowing is more common in pre-eclamptic women, and laryngeal oedema has been reported. At induction an exaggerated hypertensive response to laryngoscopy is to be expected; this increases the risk of precipitating pulmonary oedema or intracerebral bleeding. A number of drugs have been used to obtund this reflex, including lidocaine, short-acting opioids, magnesium and labetolol. Of these alfentanil is most widely used in the UK, possibly reflecting increased familiarity with this drug. A combination of alfentanil and magnesium has been shown to be more effective than either used alone. More recent interest has focused on remifentanil which blunts the hypertensive response to laryngoscopy in both non-hypertensive women and those with pre-eclampsia, although transient neonatal respiratory depression may result. The hypertensive response to extubation should not be overlooked. Potentiation of non-depolarising muscle relaxants by magnesium is not usually problematic, although monitoring neuromuscular function is necessary.

**Regional Anaesthesia:** Despite concerns over hypotension, the effects of vasoconstrictors and excessive intravenous fluid administration, it is now widely accepted that, in the hands of an experienced anaesthetist, a regional technique is preferred for caesarean section in the pre-eclamptic patient. It was originally believed that because of reduced maternal plasma volume and the need to avoid large intravenous fluid boluses in pre-eclampsia, the rapid onset of autonomic block with spinal anaesthesia was best avoided. Fears that spinal, compared to epidural, anaesthesia might provoke excessive hypotension have been dispelled by a number of studies. Furthermore it has been demonstrated that compared to healthy women, those with pre-eclampsia experience less hypotension and require smaller doses of vasoconstrictors with spinal anaesthesia. The effect of spinal anaesthesia on cardiac output has also been assessed. Using lithium dilution cardiac output (LiDCO) measurement in 15 women with severe pre-eclampsia, it has been shown that cardiac output remains stable during caesarean section under spinal anaesthesia, rising only after administration of an oxytocin bolus. However accompanying editorials questioned whether cardiac output was the most appropriate cardiovascular parameter to study and if LiDCO was the best method of assessment.

Two theories as to why hypotension should be seen less frequently in pre-eclampsia have been suggested. Firstly it may partly result from decreased aortocaval compression due to the relatively smaller weight of the baby, although this has been discredited. More likely is a humoral mechanism. Endothelial vasoconstrictor systems are altered in pre-eclampsia decreasing the relaxation of small resistance vessels. Also increased local production of factors with a potent pressor effect and increased sensitivity to pressor agents produce widespread vasoconstriction which is not altered by the autonomic block associated with spinal anaesthesia. The preferred vasoconstrictor in pre-eclampsia has yet to be determined. In healthy pregnancy there is now good evidence that phentolamine has advantages over ephedrine. However, given the increased sensitivity to alpha agonists in pre-eclampsia, caution is still required until more evidence to support their advantage is forthcoming.

**High Dependency Care**

Maternal plasma volume expansion seen in healthy pregnancy is usually much reduced in pre-eclampsia. Blood pressure and systemic vascular resistance are increased with low or normal central venous pressure. Correlation between right and left sided pressures are poor. Consequently, over reliance on CVP values can lead to over transfusion and it is recognised that inappropriate fluid management in the peripartum period can lead to adverse outcome, especially if pulmonary oedema is precipitated. Indeed many women with severe pre-eclampsia require high dependency or intensive care and early involvement of intensive care specialists has been recommended. Fluid management in pre-eclamptic patients requires a careful balance between restriction with possible exacerbation of end-organ hypoperfusion and renal dysfunction and volume overload with pulmonary oedema. The latter is more likely in pre-eclampsia because of increased vascular permeability from endothelial damage, decreased plasma oncotic pressure and in some women, left ventricular dysfunction. The situation is more hazardous when renal dysfunction is present. Oliguria is common after delivery, more so if oxytocic drugs have been used, and does not necessarily imply volume depletion. Acute renal failure is fortunately extremely uncommon. Although a number of protocols have been suggested, there is still no consensus on the optimal type or volume of fluid that should be given. Diuretics should be used with
extreme caution and only after discussion with senior staff.

Fluid preloading before regional analgesia in labour is usually unnecessary especially when low-dose local anaesthetic mixtures are used. Aggressive preloading before regional anaesthesia for caesarean section should be avoided as it is largely ineffective and increases the risk of pulmonary oedema. For particularly severe cases invasive monitoring may be useful. Opinions differ as to precise indications but it is useful when significant fluid resuscitation is required, in severe refractory hypertension, pulmonary oedema and oliguria unresponsive to fluid therapy.34 Central venous pressure monitoring may pick up hypovolaemia but used in isolation is an unreliable guide to fluid management. Pulmonary artery catheters allow monitoring of pulmonary artery wedge pressure and cardiac output and have been advocated in cases where assessment of left ventricular pre-load is important.35 However, recent evidence has questioned their effectiveness.36 Less invasive haemodynamic monitoring, such as whole body impedance cardiography, oesophageal Doppler and LiDCO are likely to become increasing popular.30,37

References


31. Langesaeter E. Is it more informative to focus on cardiac output than blood pressure during spinal anesthesia for caesarean delivery in women with severe preeclampsia? Anesthesiology 2008; 108: 771-2.

32. Pauca AL. Pressure wave analysis is useful to understand the pathophysiology of preeclampsia but perhaps not the rapid changes during caesarean delivery. Anesthesiology 2008; 108: 773-4.


Notes
FUTURE MEETINGS & COURSES

2010

3 March 2010  Cases and Controversies in Obstetric Anaesthesia, London
19 - 21 May 2010  Obstetric Anaesthesia 2010 (Reception 19 May, Annual Meeting 20 & 21 May), The Sage, NewcastleGateshead
13 October 2010  Refresher Day on Obstetric Anaesthesia and Analgesia, London
8 - 10 November 2010  Three-day Course on Obstetric Anaesthesia and Analgesia, London
10 December 2010  Joint meeting with Royal Society of Medicine (Section of Anaesthesia and Section of Obstetrics and Gynaecology), London

2011

25 - 27 May 2011  Obstetric Anaesthesia 2011 (Reception 25 May, Annual Meeting 26 & 27 May), Edinburgh

2012

23 - 25 May 2012  Obstetric Anaesthesia 2012 (Reception 23 May, Annual Meeting 24 & 25 May), Liverpool

2013

22 - 24 May 2013  Obstetric Anaesthesia 2013 (Reception 22 May, Annual Meeting 23 & 24 May), Bournemouth

Obstetric Anaesthetists’ Association
Promoting the highest standards of anaesthetic practice in the care of mother and baby

Obstetric Anaesthesia 2010
20 - 21 May 2010  (evening reception 19 May) The Sage, NewcastleGateshead

Enquiries: OAA Secretariat T: +44 (0) 20 8741 1311  F: +44 (0) 20 8741 0611  www.oaa-anaes.ac.uk

Obstetric Anaesthetists' Association
Three-day Course on Obstetric Anaesthesia and Analgesia
Organised by Dr Roshan Fernando, University College London Hospitals and Dr Chris Elton, University Hospitals of Leicester

9 – 11 November 2009
Church House Conference Centre, Westminster, SW1P 3NZ
LONDON

LECTURE ABSTRACTS