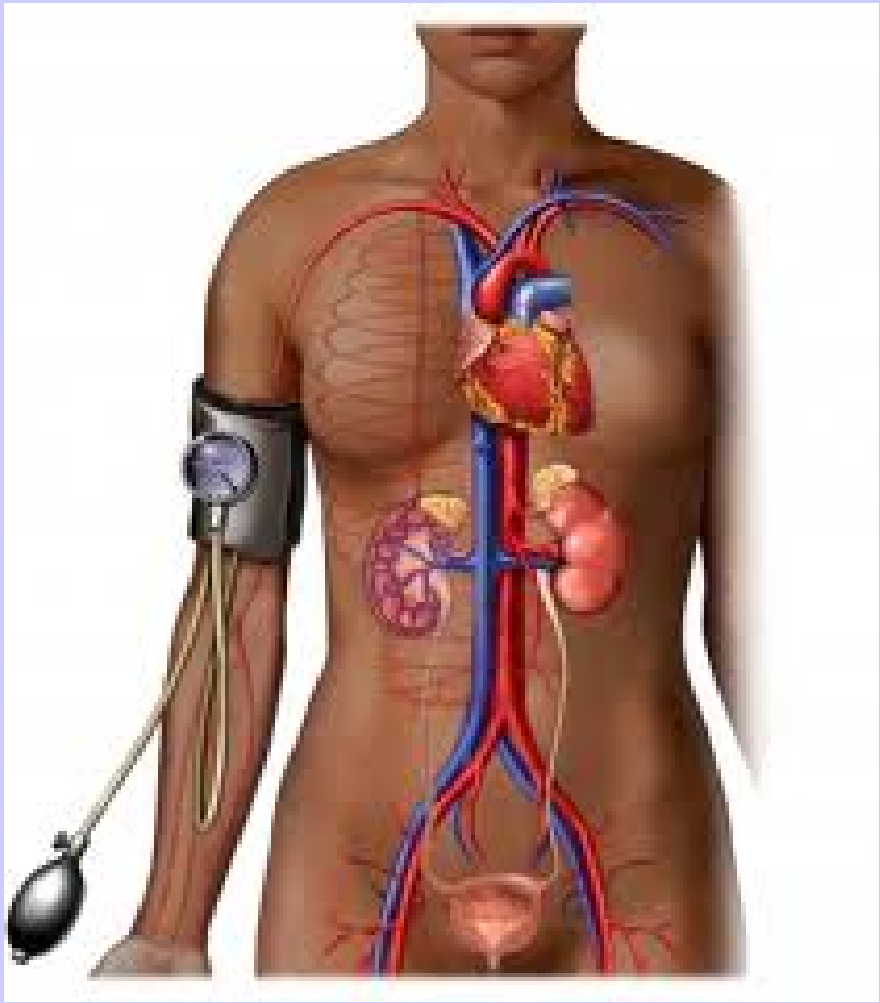
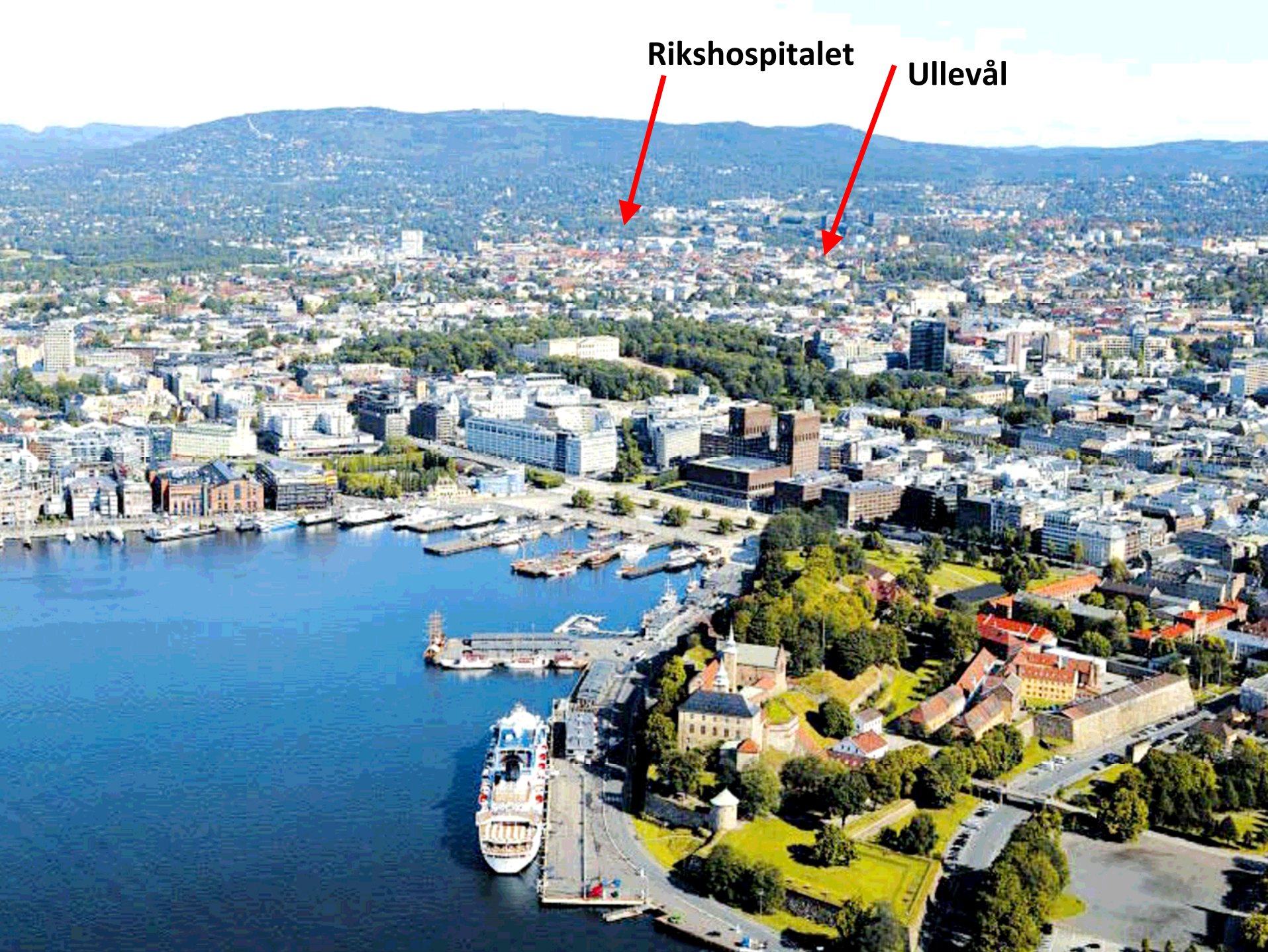
HAEMODYNAMIC MONITORING in SEVERE PREECLAMPSIA



Eldrid Langesæter MD PhD
Consultant Anaesthesiologist
Oslo University Hospital
Rikshospitalet


Rikshospitalet
Ullevål

CASE 1

- Anxiety, otherwise healthy pregnant woman
- Remifentanil during labour
  - 1 p.m: Spontanous vaginal delivery
- 2 p.m: GA for retained placenta
  - “hysterical”, HR 150, SBP 180 mmHg
  - 1000 ml bleeding

CASE 1

- 3 p.m: Postoperative
  - Circulatory stable
  - Tachycardia > 120
  - Considered anxious

- 8.30 p.m
  - Hb 7
  - Platelets 77
  - Difficulties inserting an arterial line
    - SAP 220 mmHg
  - Volume
  - MgSO4

CASE 1

- 9.30 p.m
  - Circulatory collaps, intubated without any medication
  - Hb 4

- HELLP with liver capsule rupture and DIC

AGENDA

- CMACE
- Guidelines
- Haemodynamic monitoring
- Anaesthetic implications
- Future research

Confidential Enquiry into Maternal and Child Health

Improving care for mothers, babies and children

Why Mothers Die 2000–2002

Table 1.16 Numbers and percentage of *Direct* deaths assessed as having substandard care; United Kingdom 2000–02

Cause of death and chapter number	Major substandard care (*n*)	Minor substandard care (*n*)	Total cases in chapter (*n*)	Overall substandard care	
				(*n*)	(%)
2. Thrombosis	12	5	30	17	*57*
3. Hypertension	6	1	14	7	*50*
4. Haemorrhage	10	2	17	12	*71*
5. Amniotic fluid embolism	0	3	5	3	*60*
6. Early pregnancy	10	0	15	10	*67*
7. Sepsis	2	8	13	10	*77*
8. Other *Direct*	4	2	8	6	*75*
9. Anaesthetic	6	0	6	6	*100*
Total	50	21	106	71	*67*

Confidential Enquiry into Maternal and Child Health

CEMACH

Saving Mothers' Lives:

Reviewing maternal deaths to make motherhood safer - 2003-2005

December 2007

The Seventh Report of the Confidential Enquiries into Maternal Deaths in the United Kingdom

Table 1.11
Numbers and percentage of *Direct* and *Indirect* deaths assessed as having substandard care by cause of death; United Kingdom: 2003-05.

Cause of death	Numbers with substandard care			Total number of cases	*Percentage of all cases with substandard care*
	Major	Minor	Major and Minor		
Direct					
2. Thromboembolism	19	4	23	41	*56*
Pulmonary embolism	18	4	22	33	*67*
Cerebral thrombosis	1	0	1	8	*13*
3. Pre-eclampsia/eclampsia*	9	4	13	18	*72*
4. Haemorrhage**	10	0	10	17	*59*
5. Amniotic fluid embolism	6	1	7	17	*41*
6. Early pregnancy	10	1	11	14	*79*
7. Sepsis	12	2	14	18	*78*
8. Anaesthetic	6	0	6	6	*100*
All *Direct*	**72**	**12**	**84**	**132**	***64***
Late *Direct*	**6**	**0**	**6**	**11**	***55***
Indirect					
9. Cardiac	15	7	22	48	*46*
10. Other *Indirect*	25	5	30	86	*35*
11. Psychiatric***	3	5	8	19	*42*
12. Cancer****	2	3	5	10	*50*
All *Indirect*	**45**	**20**	**65**	**163**	***40***


Volume 118, Supplement 1, March 2011
BJOG
An International Journal of Obstetrics and Gynaecology
Saving Mothers' Lives
Reviewing maternal deaths to make motherhood safer: 2006–2008
March 2011

Table 1.12. Numbers and percentages of cases of *Direct* and *Indirect* deaths by cause and degree of substandard care (SSC); UK: 2006–08

Cause	Numbers of cases			Percentages of cases			Percentage of cases with no SSC	Total number of cases
	Major	Minor	Total	Major	Minor	Total		
Direct								
Thrombosis and thromboembolism	6	4	10	33	22	56	44	18
Pre-eclampsia, eclampsia and acute fatty liver of pregnancy	14	6	20	64	27	91	9	22
Haemorrhage	4	2	6	44	22	67	33	9
Amniotic fluid embolism	2	6	8	15	46	62	38	13
Early pregnancy deaths	6	–	6	55	–	55	45	11
Sepsis	12	6	18	46	23	69	31	26
Anaesthesia	3	3	6	43	43	86	14	7
Total *Direct*	47	28*	75*	44	26	70	30	107*
Indirect								
Cardiac disease	13	14	27	25	26	51	49	53
Other *Indirect* causes	17	11	28	33	21	54	46	52
Indirect neurological causes	11	12	23	31	33	64	36	36
Psychiatric causes	6	1	7	46	8	54	46	13
Total *Indirect*	47	38	85	31	25	55	45	154
Total *Direct* and *Indirect*	94	66	160	36	25	61	39	261

Rise in maternal mortality in the Netherlands

JM Schutte,[a] EAP Steegers,[b] NWE Schuitemaker,[c] JG Santema,[d]* K de Boer,[e] M Pel,[f] G Vermeulen,[g] W Visser,[b] J van Roosmalen,[h,i] the Netherlands Maternal Mortality Committee

BJOG 2009

Table 7. Modes of death in 80 cases of maternal mortality caused by pre-eclampsia, the Netherlands, 1993–2005

Mode of death	*n* (%)
Cerebral complication	49 (61)
Cerebral haemorrhage	36 (45)
Cerebral oedema	8 (10)
Encephalopathy	5 (6)
Adult respiratory distress syndrome	8 (10)
Disseminated intravascular coagulation	6 (8)
Multiple organ failure	3 (4)
Liver rupture	7 (9)
Miscellaneous	7 (9)

Substandard care 96%

GUIDELINES AND RECOMMENDATIONS

- Evidence-based mangement for preeclampsia
  Dadelszen et al. Frontiers in Bioscience **2007**;12:2876-2889
- Diagnosis,Evaluation, and Management of the Hypertensive Disorders of Pregnancy
  J Obst Gyn Can **2008** (3):S1-S48
- Guidelines for the management of hypertensive disorders of pregnancy
  Aust NZJ Obst Gyn **2009**;49:242-6

GUIDELINES AND RECOMMENDATIONS

1. **Treat SAP $\geqq$ 160 mmHg**
2. MgSO4
   - Prevent Eclampsia
   - Treat Eclampsia
3. Fluid restriction

Stroke and Severe Preeclampsia and Eclampsia: A Paradigm Shift Focusing on Systolic Blood Pressure

Martin et al. Obstet Gynecol **2005**:1052-54

Few years ago, obstetricians focused on diastolic blood pressure only

Is systolic blood pressure of 160 mmHg an adequate target?

Non-invasive devices for BP-monitoring may underestimate BP in preeclamptic patients

Confidential Enquiry into Maternal and Child Health

Improving care for mothers, babies and children

Why Mothers Die 2000–2002

the pressure during systole which causes intracerebral haemorrhage. Recognition of this concept should be incorporated into clinical guidelines to try to ensure effective reduction of systolic pressure. **It is, therefore, recommended that clinical protocols identify a systolic blood pressure above which urgent and effective antihypertensive treatment is required**. Some would recommend 160 mmHg as a useful guide to treatment.

CMACE 2003-2005

Box 3.1
Learning points: pre-eclampsia and eclampsia

Fulminating pre-eclampsia occurs at term and post term, as well as pre-term.

Systolic blood pressures over 160 mm/Hg must be treated.

Syntometrine should not be given for the active management of the third stage if the mother is hypertensive, or her blood pressure has not been checked.

The anaesthestist should be given as much time as possible to try to prevent the pressor effects of intubation in the pre-eclamptic woman, even when there are pressing fetal reasons for urgent caesarean section under general anaesthesia.

Systolic hypertension

The single major failing in clinical care in pre-eclampsia in the current triennium was inadequate treatment of systolic hypertension. The sequel, intracranial haemorrhage, occurred in several cases. The following

CMACE 2006-2008

- **SAP ≥ 150 mmHg** require effective antihypertensive treatment.

Treatment of systolic hypertension

The single major failing in clinical care in the current triennium was, again, inadequate treatment of hypertension, with subsequent intracranial haemorrhage. For example:

NON-INVASIVE BLOOD PRESSURE

Pre-eclampsia/eclampsia: learning points

Eclampsia is important because it is a marker for severe disease, but seizures also carry additional intrinsic risks. Some automated blood pressure monitoring systems systematically *underestimate* systolic pressure in pre-eclampsia.

GUIDELINES AND RECOMMENDATIONS

1. Treat SAP ≥ 160 mmHg
2. **MgSO4**
   - **Prevent Eclampsia**
   - **Treat Eclampsia**
3. Fluid restriction

Do women with pre-eclampsia, and their babies, benefit from magnesium sulphate? The Magpie Trial: a randomised placebo-controlled trial

Lancet 2002; **359:** 1877–90

*The Magpie Trial Collaborative Group**

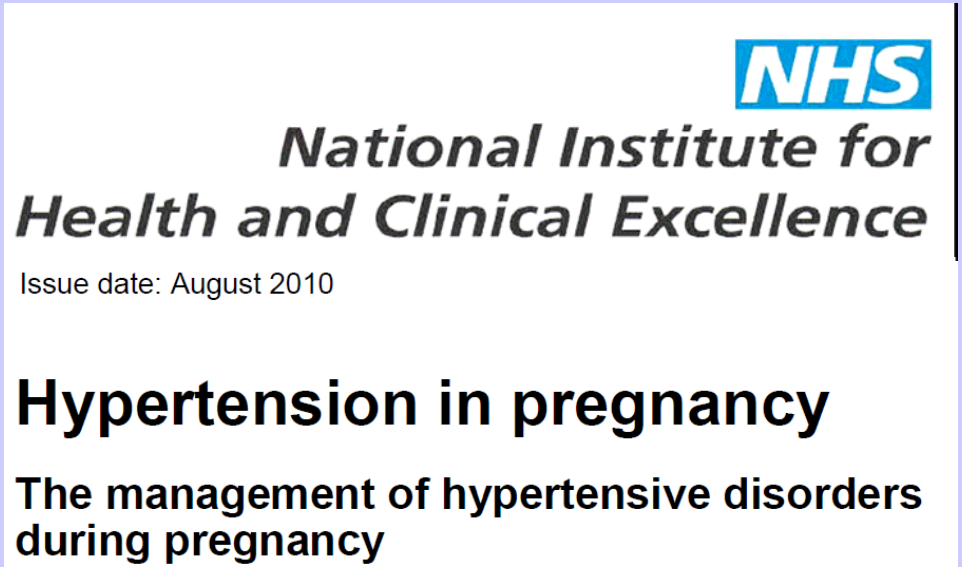
Included 10 000 patients

NNT 69

Interpretation Magnesium sulphate halves the risk of eclampsia, and probably reduces the risk of maternal death. There do not appear to be substantive harmful effects to mother or baby in the short term.

MgSO4

- Severe hypertension and proteinuria **or**
- Mild/moderate hypertension and proteinuria and (1)
  - Severe headache
  - Vision disturbance (blurring, flashing)
  - Severe epigastrial pain or vomiting
  - Papilloedema
  - Signs of clonus
  - Liver tenderness
  - HELLP
  - Platelets < 100 000
  - ASAT/ALAT > 70



Acta Obstetricia et Gynecologica. 2006; 85: 929–936

Taylor & Francis
Taylor & Francis Group

ORIGINAL ARTICLE

Eclampsia in Scandinavia: incidence, substandard care, and potentially preventable cases

ALICE BEATHE ANDERSGAARD[1], ANDREAS HERBST[2], MARIANNE JOHANSEN[3], ANNA IVARSSON[2], INGEMAR INGEMARSSON[2], JENS LANGHOFF-ROOS[3], TORE HENRIKSEN[4], BJØRN STRAUME[5] & PÅL ØIAN[6]

From the [1]Department of Obstetrics and Gynaecology, Sykehuset Innlandet Health Authority, Norway, [2]Department of Obstetrics and Gynaecology, Lund University Hospital, Lund, Sweden, [3]Department of Obstetrics and Gynaecology, Rigshospitalet University Hospital, Copenhagen, Denmark, [4]Department of Obstetrics and Gynaecology, Rikshospitalet University Hospital, Oslo, [5]Institute of Community Medicine, University of North Norway, Tromsø, and [6]Department of Obstetrics and Gynaecology, University Hospital of North Norway, Tromsø, Norway

2 years (1998-2000)

2/210 had prophylactic MgSO4

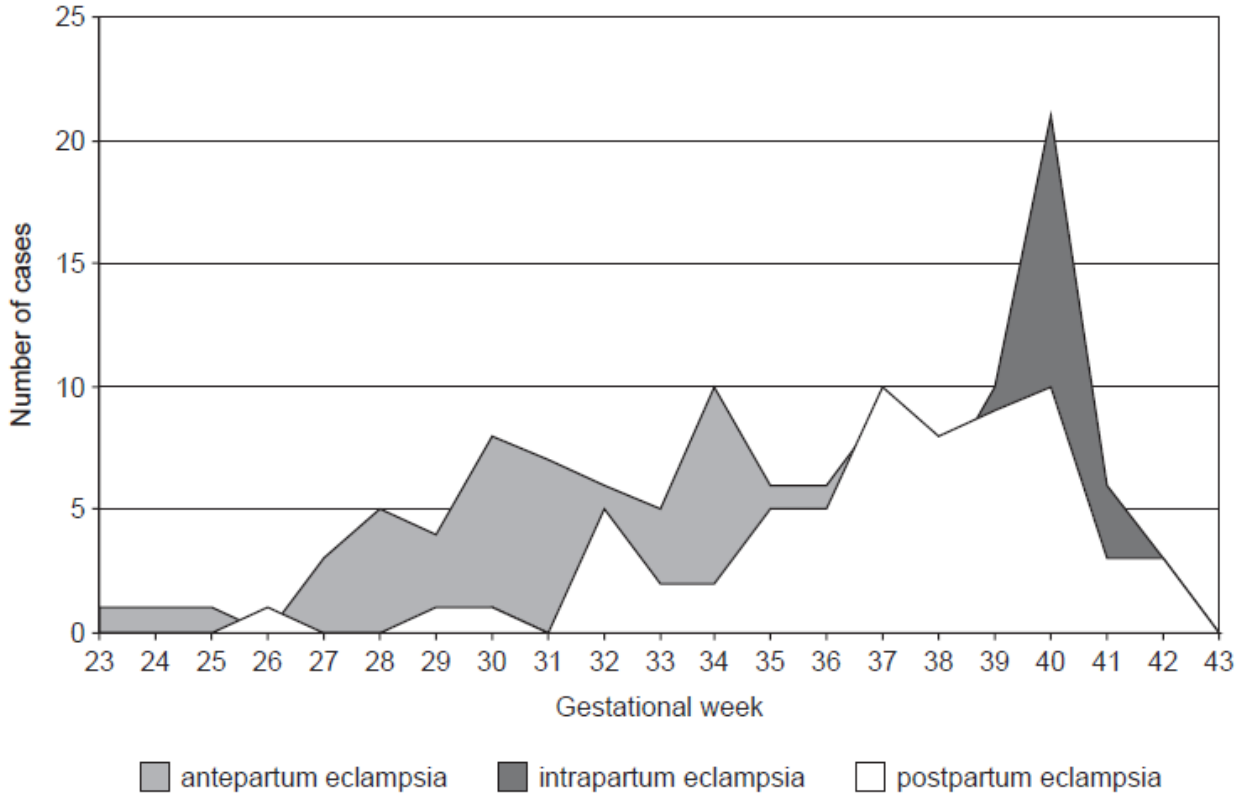
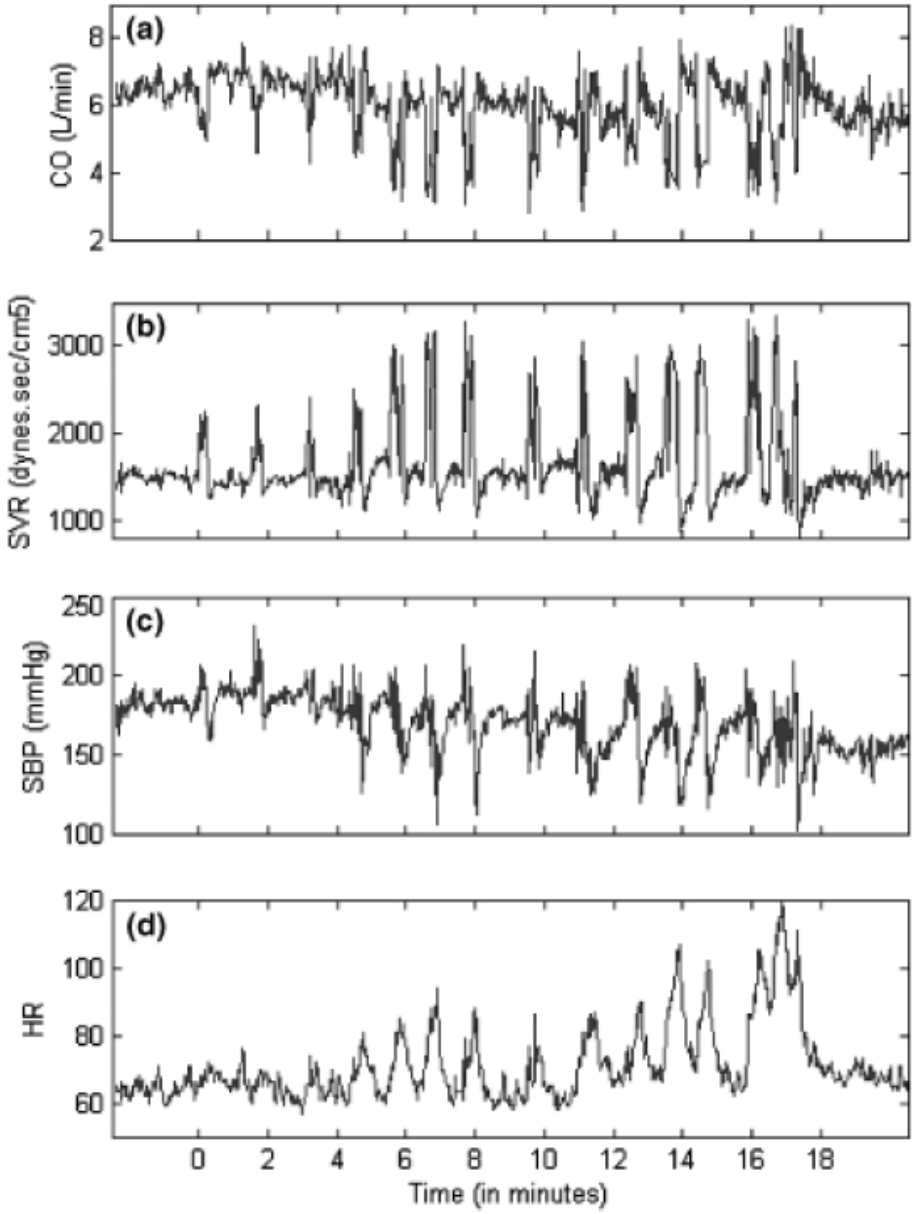


Figure 1. Number of cases with eclampsia by gestational age at first seizure.



Langesæter ACTA Anaesth Scand 2009

GUIDELINES AND RECOMMENDATIONS

1. Treat SAP $\geq$ 160 mmHg
2. MgSO4
   - Prevent Eclampsia
   - Treat Eclampsia
3. **Fluid restriction**

FLUID RESTRICTION

- Recommendations based on one study (Evidence level 1)

A randomised controlled trial comparing two temporising management strategies, one with and one without plasma volume expansion, for severe and early onset pre-eclampsia

Ganzevoort el al. BJOG **2005**;112:1358-68

- 200 patients
- Randomisation based on diastolic BP
- Treatment group: Plasma volume expansion and antihypertensive treatment
- Control group: Antihypertensive treatment

Conclusion: No group differences in fetal and maternal outcome

Preeclampsia and anaesthesia

Wiebke Gogarten

Current Opinion in Anaesthesiology 2009, 22:347–351

”..evidence currently does not support plasma volume expansion as it may promote pulmonary oedema due to capillary leakage with no consistent effect on outcome.”

(ref. CEMACH)

Table 3.2
Numbers of deaths from pre-eclampsia or eclampsia; United Kingdom 2003-05.

Triennium	Cerebral					Pulmonary				Hepatic				Total
	Intracranial haemorrhage	Subarachnoid	Infarct	Oedema	All	ARDS	Oedema	Other	All	Rupture	Failure/ necrosis	Other	All	
1985-87	11	0	0	0	**11**	9	1	2	**12**	0	1	3	**4**	**27**
1988-90	10	2	2	0	**14**	9	1	0	**10**	0	1	2	**3**	**27**
1991-93	5	0	0	0	**5**	8	3	0	**11**	0	0	4	**4**	**20**
1994-96	3	1	0	3	**7**	6	2	0	**8**	2	1	2	**5**	**20**
1997-99	7	0	0	0	**7**	2	0	0	**2**	2	0	5	**7**	**16**
2000-02	9	0	0	0	**9**	1	0	0	**1**	0	0	4	**4**	**14**
2003-05	10	0	2	0	**12**	0	0	0	**0**	0	2	4	**6**	**18**

Evidence for recommendation against plasma volume expansion?

Table 3.1. Numbers and underlying cause of death due to eclampsia and pre-eclampsia, UK: 1991–2008

Cause of death	1997–99			2000–02			2003–05			2006–08		
	n	Rate	95% CI	*n*	Rate	95% CI	*n*	Rate	95% CI	*n*	Rate	95% CI
Cerebral												
Intracranial haemorrhage	7	0.33	0.16–0.69	9	0.45	0.23–0.87	9	0.43	0.22–0.82	9	0.39	0.20–0.75
Subarachnoid	0	0.00		0	0.00		0	0.00		0	0.00	
Infarct	0	0.00		0	0.00		1	0.05	0.01–0.34	0	0.00	
Oedema	0	0.00		0	0.00		0	0.00		0	0.00	
Eclampsia	0	0.00		0	0.00		0	0.00		5	0.22	0.09–0.52
Subtotal	7	0.33	0.16–0.69	9	0.45	0.23–0.87	10	0.47	0.25–0.88	14	0.61	0.36–1.03
Pulmonary												
Adult respiratory distress syndrome	2	0.09	0.02–0.38	1	0.05	0.01–0.36	0	0.00		0	0.00	
Oedema	0	0.00		0	0.00		0	0.00		0	0.00	
Subtotal	2	0.09	0.02–0.38	1	0.05	0.01–0.36	0	0.00		0	0.00	
Hepatic												
Rupture	2	0.09	0.02–0.38	0	0.00		0	0.00		1	0.04	0.01–0.31
Failure/necrosis	0	0.00		0	0.00		1	0.05	0.01–0.34	2	0.09	0.02–0.35
Other	5	0.24	0.10–0.57	4	0.20	0.08–0.53	3	0.14	0.05–0.44	2	0.09	0.02–0.35
Subtotal	7	0.33	0.16–0.69	4	0.20	0.08–0.53	4	0.19	0.07–0.50	5	0.22	0.09–0.52
Overall total	16	0.75	0.46–1.23	14	0.70	0.42–1.18	14	0.66	0.39–1.12	19	0.83	0.53–1.30
Acute fatty liver of pregnancy	4	0.19	0.07–0.50	3	0.15	0.05–0.47	1*	0.05	0.01–0.34	3*	0.13	0.04–0.41

*As a result of the very small numbers these cases are counted in this Chapter 3. Before 2003 they were counted in the now nonexistent chapter 'Deaths from other *Direct* causes.'

PLASMA VOLUME EXPANSION

- Old studies using PA-cather
  - Belfort et al. BJOG **1989**;96:634-41
  - Visser et al. Eur J Obst Gyn Repr Bio **1995**;63:147-54
- Recent studies using eccocardiography
  - Valensise et al. Ultrasound Obst Gyn **2008**;31:55-64

  – Plasma volume expansion and vasodilatation improved maternal and fetal circulation

  – Blood pressure alone is not enough to guide treatment

Hypertensive Disorders and Severe Obstetric Morbidity in the United States

Kuklina et al. Obst Gyn **2009**;113:1299-306

Table 3. Estimated Odds Ratios of Severe Obstetric Complications for Delivery Hospitalizations With Hypertensive Disorders Compared With Delivery Hospitalizations Without Hypertensive Disorders: The 1998–2006 Nationwide Inpatient Sample (N=36,537,061)

	Estimated Odds Ratio (95% Confidence Interval)*				
	Eclampsia/Preeclampsia		Hypertension		
	Severe	Mild	Chronic	Gestational	Any†
Acute renal failure	34.8 (30.4–39.9)	5.8 (4.8–6.9)	9.9 (8.4–11.6)	2.2 (1.7–2.9)	10.7 (9.6–11.8)
Pulmonary edema	9.4 (7.9–11.3)	3.7 (3.0–4.5)	3.7 (2.9–4.7)	1.5 (1.1–2.1)	4.7 (4.1–5.3)
ARDS	11.9 (10.7–13.2)	2.9 (2.5–3.4)	3.7 (3.2–4.3)	1.2 (0.9–1.4)	4.1 (3.7–4.4)
PCD	16.6 (14.2–19.5)	2.5 (1.9–3.1)	4.2 (3.3–5.2)	1.4 (1.1–1.9)	5.1 (4.5–5.7)
DICS	14.8 (13.6–16.2)	3.3 (3.0–3.7)	2.3 (2.0–2.6)	1.7 (1.5–1.9)	4.5 (4.2–4.7)
Ventilation	11.0 (9.9–12.1)	2.3 (2.0–2.6)	3.4 (3.0–3.9)	1.1 (0.9–1.3)	4.0 (3.7–4.3)
Mortality	6.9 (5.2–9.2)	1.3 (0.8–2.0)	2.9 (2.0–4.0)	0.9 (0.5–1.5)	2.7 (2.3–3.3)

ARDS, adult respiratory distress syndrome; PCD, puerperal cerebrovascular disorder; DICS, disseminated intravascular coagulation syndrome.

IMPORTANT QUESTIONS

- Who are at risk for complications?
- Can recommendations be generalized in this patient group?
- Is monitoring of blood pressure enough in the high risk patients?

- Can long time morbidity and mortality be reduced?
  - Bellamy et al. BMJ **2007**; 335(7627)
  - McDonald et al. Am Heart J **2008**; 156 (5):918-30

PREDICTORS FOR INCREASED RISK

- **Early onset preeclampsia** (< 34 weeks)
  - Valensise et al. Hypertension **2008**;52:873-80
- **Increased systemic vascular resistance**
  - Valensise et al. BJOG **2006**. 1045-52
  - Mei et al. J Obst Gyn Res **2008**. 34(2):179-88

Early and Late Preeclampsia

Two Different Maternal Hemodynamic States in the Latent Phase of the Disease

Herbert Valensise, Barbara Vasapollo, Giulia Gagliardi, Gian Paolo Novelli

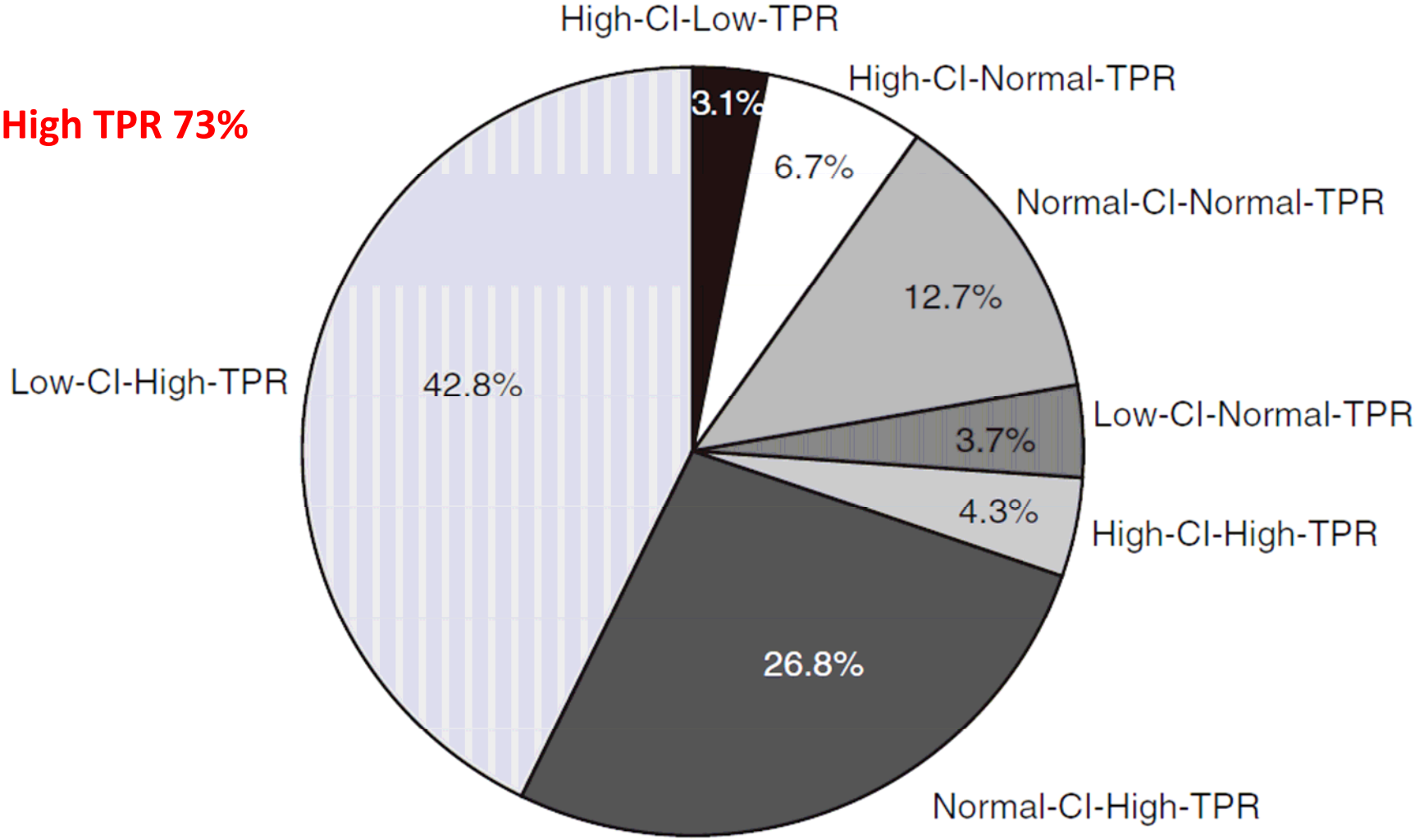
Hypertension **2008**;52:873-80

- **EARLY (n=75)**
  - **TPR 1605**
  - **CO 4.5**
- **LATE (n=35)**
  - **TPR 739**
  - **CO 9**

Pre-eclampsia outcomes in different hemodynamic models

Mei et al. J Obst Gyn Res **2008**.34(2):179-88

High TPR 73%



Assessment of left ventricular structure and function in preeclampsia by echocardiography and cardiovascular biomarkers

Rangeen Rafik Hamad[a], Anders Larsson[b], John Pernow[c], Katarina Bremme[a] and Maria J. Eriksson[d]

Journal of Hypertension 2009, 27:2257–2264

Table 4 The level of biomarkers in the study groups at inclusion and follow-up

	Preeclampsia group		Normal pregnancy group		*P*	
	Pregnancy	Follow-up	Pregnancy	Follow-up	Groups	Time
NT-pro-BNP (ng/l)	477 (152)	76 (16)	46 (6)	61 (6)	<0.0001	0.004
Cystatin C (mg/l)	1.29 (0.03)	0.82 (0.04)	0.95 (0.03)	0.78 (0.04)	<0.0001	<0.0001
Cystatin GFR (ml/min per 1.73 m^2)	58 (4)	112 (5)	95 (4)	117 (5)	<0.0001	<0.0001
CRP (mg/l)	7.93 (3.19)	4.60 (1.56)	6.17 (0.81)	2.87 (0.56)	0.58	<0.0001
Troponin I (ug/l)	<0.0022	<0.0022	<0.0022	<0.0022	NA	NA

The values are in mean (SE). CRP, C-reactive protein; NA, not applicable.

Early PE 1243 vs Late PE 583

European Journal of Obstetrics & Gynecology and Reproductive Biology 153 (2010) 12–15

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journal homepage: www.elsevier.com/locate/ejogrb

Amino-terminal pro-brain natriuretic peptide (NT-proBNP) is a biomarker of cardiac filling pressures in pre-eclampsia

Leonie Speksnijder [a], Joost H.W. Rutten [b], Anton H. van den Meiracker [b], René J.A. de Bruin [b], Jan Lindemans [c], Wim C.J. Hop [d], Willy Visser [a,*]

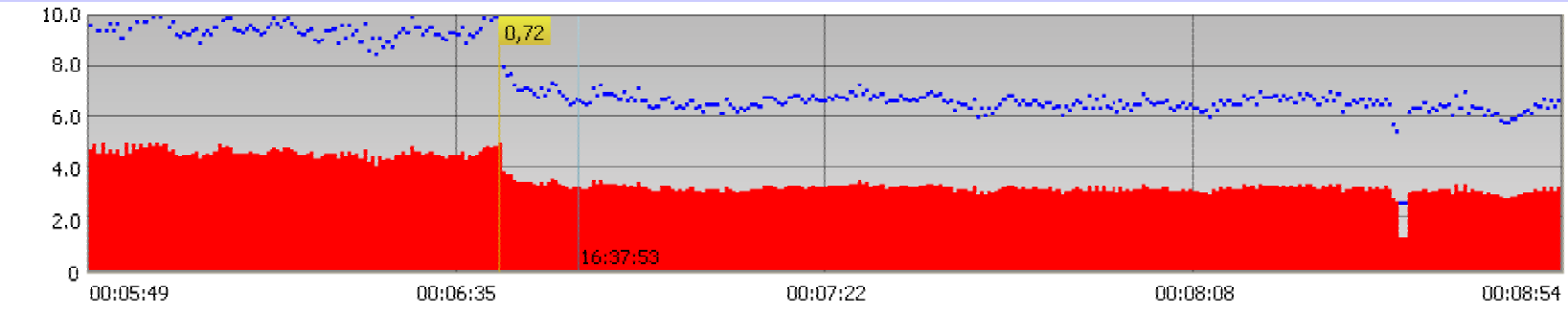
[a] *Department of Obstetrics and Gynecology, Division of Obstetrics and Prenatal Medicine, Erasmus Medical Center, Rotterdam, The Netherlands*
[b] *Department of Internal Medicine, Erasmus Medical Center, Rotterdam, The Netherlands*
[c] *Department of Clinical Chemistry, Erasmus Medical Center, Rotterdam, The Netherlands*
[d] *Department of Biostatistics, Erasmus Medical Center, Rotterdam, The Netherlands*

CASE 2

- 32 years, Nullipara, GA 36.5, BMI 36
  - Hypertension 14 days
  - Dyspnoe 4 days
- Ecco cor
  - hypokinesi in septum and apex, left ventricle not dilatated
  - EF (ejection fraction) 27%
  - NYHA IV
- Hb 14.4, platelets 241 000, ASAT 81, ALAT 74, haptoglobin < 0.2, U-totalprotein/kreatinin-ratio 548.
- **SVRI > 5500** dynes.sec.cm-5
- **Pro-BNP > 4000**

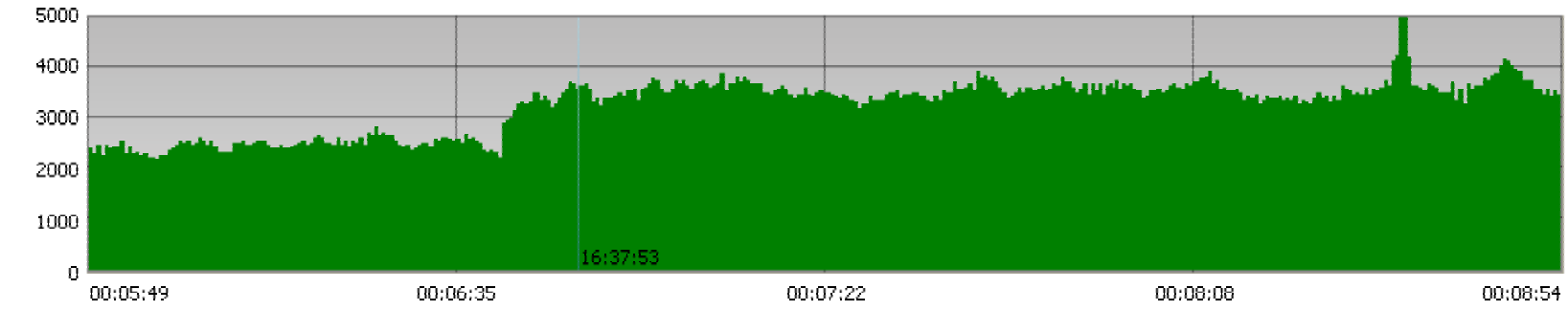
CASE 2

- IABP (intraaortic balloon pump)
- MgSO4
- Caesarean section
  - continuous spinal anaesthesia
- Bleeding 1600 ml, B-lynch suture
- After delivery: no dyspnoe lying flat
- Ecco cor 6 weeks postpartum: EF 45%, NYHA I-II

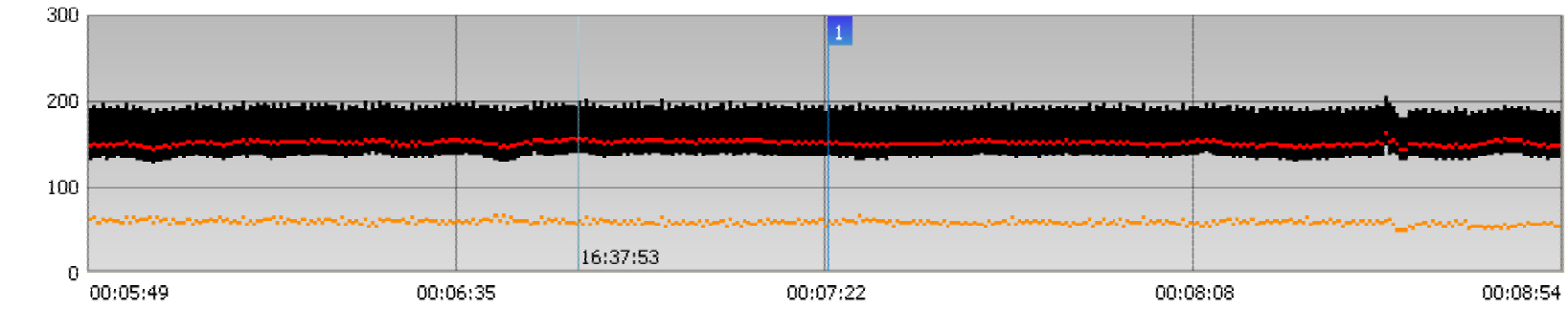


CO l min^{-1}
3,3

DO_2 ml min^{-1}
673



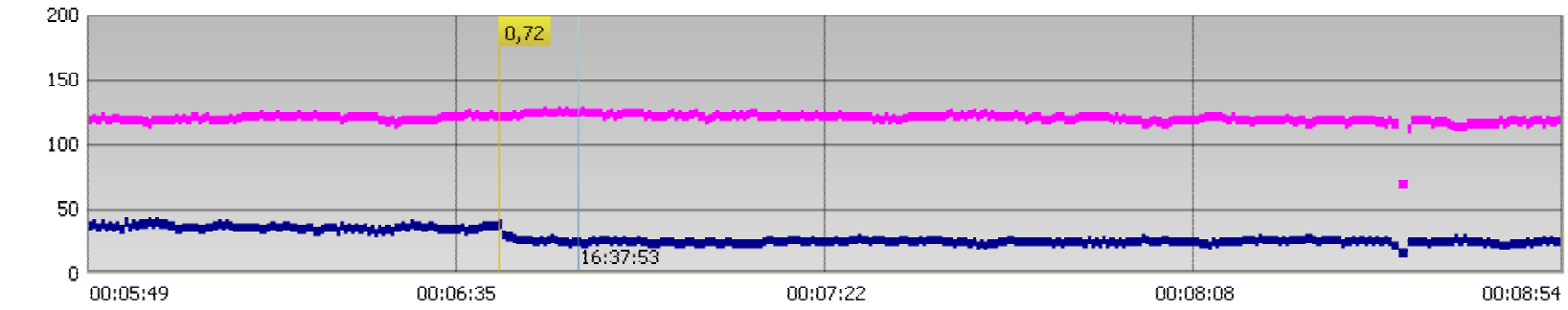
SVR dyn s cm^{-5}
3674



Sys/Dia mmHg
198/140

MAP mmHg
157

PP mmHg (PPV)
58 (20%)



SV ml (SVV)
25 (20%)

HR min^{-1} (HRV)
129 (1%)

Invited critical review

New markers in preeclampsia

Alexandre Hertig [a,b,c,*], Philippe Liere [d]

[a] Inserm U702, Hôpital Tenon, Paris, France
[b] APHP, Hopital Tenon, Urgences Néphrologiques et Transplantation Rénale, Paris, France
[c] UPMC, Université Paris 6, France
[d] UMR788 Inserm-University Paris Sud XI, France

Clinica Chimica Acta **2010** Nov 11;411(21-22):1591-5

↑ **sFlts** – the soluble form of the type-1 receptor of the vascular endothelial growth factor (VEGF)

Endoglin (sEng) – an endothelial receptor for transforming growth factor beta

↓ PIGF – placental growth factor

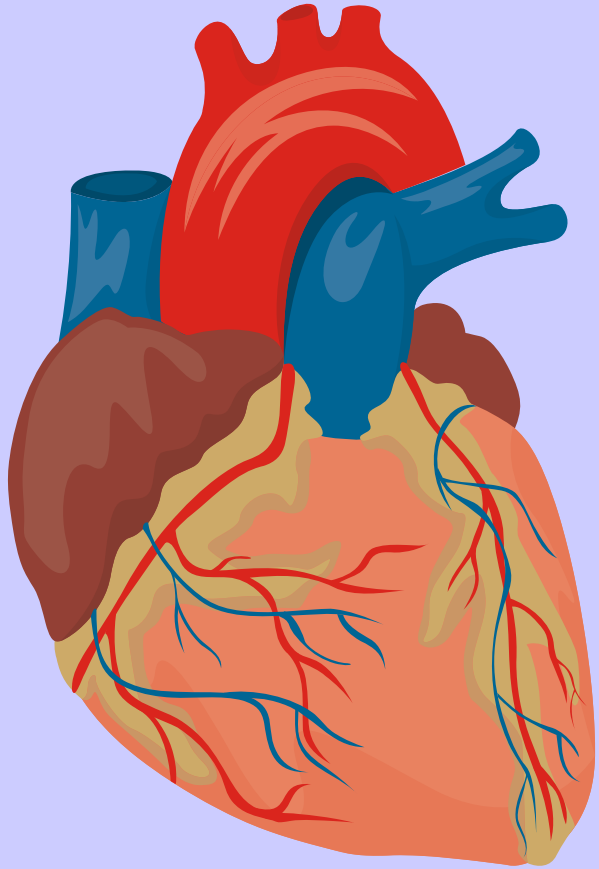
VEGF – vascular endothelial growth factor

WHY MONITORING CARDIAC OUTPUT?

- No studies verify a better outcome using CO-monitoring in the ICU

- ”..we are left with the physiological rationale as primary defense for monitoring…”
  Pinsky M. Curr Opin Crit Care **2003**

- **CO** = HR x SV
- MAP = **CO** x SVR

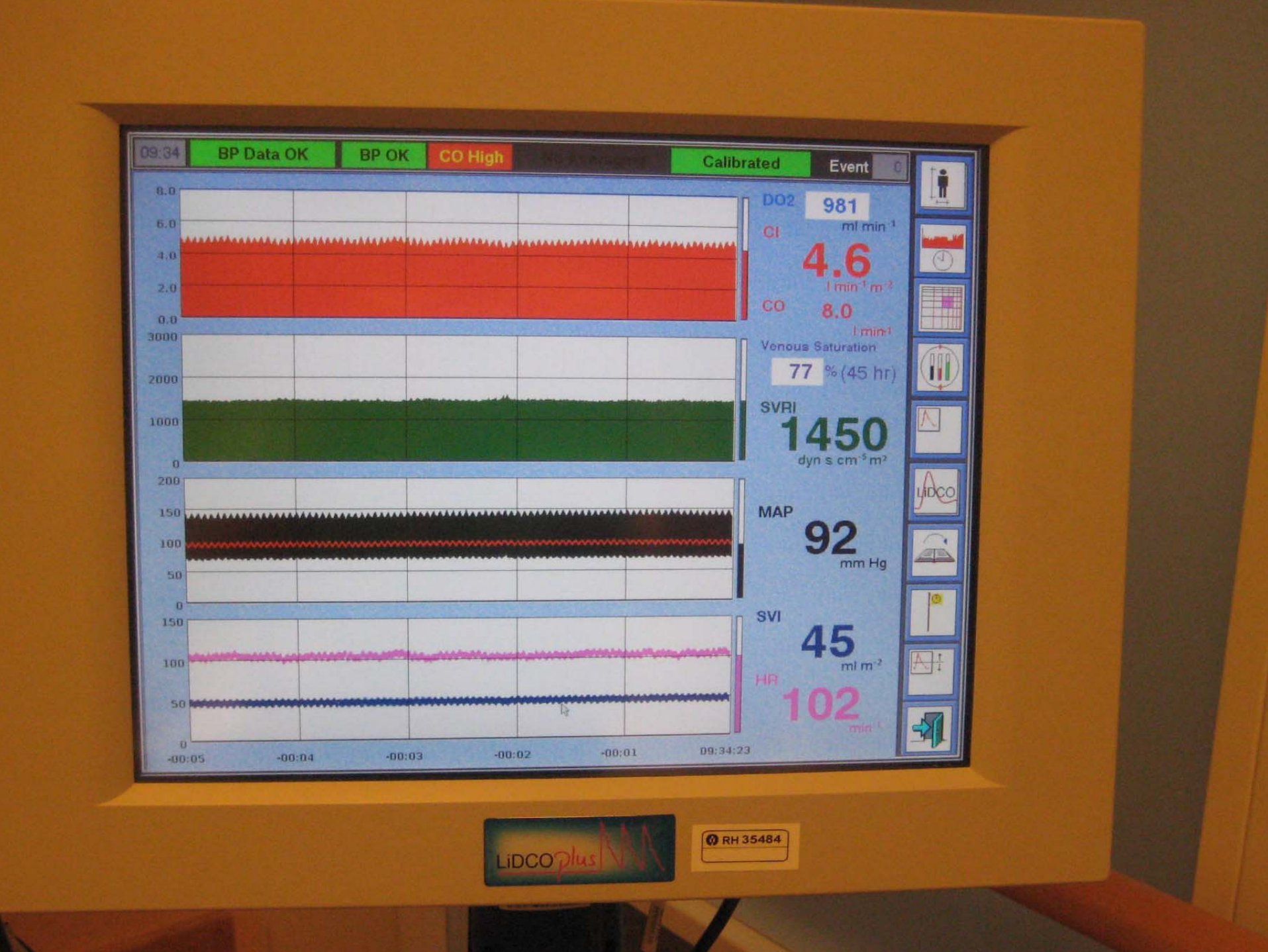


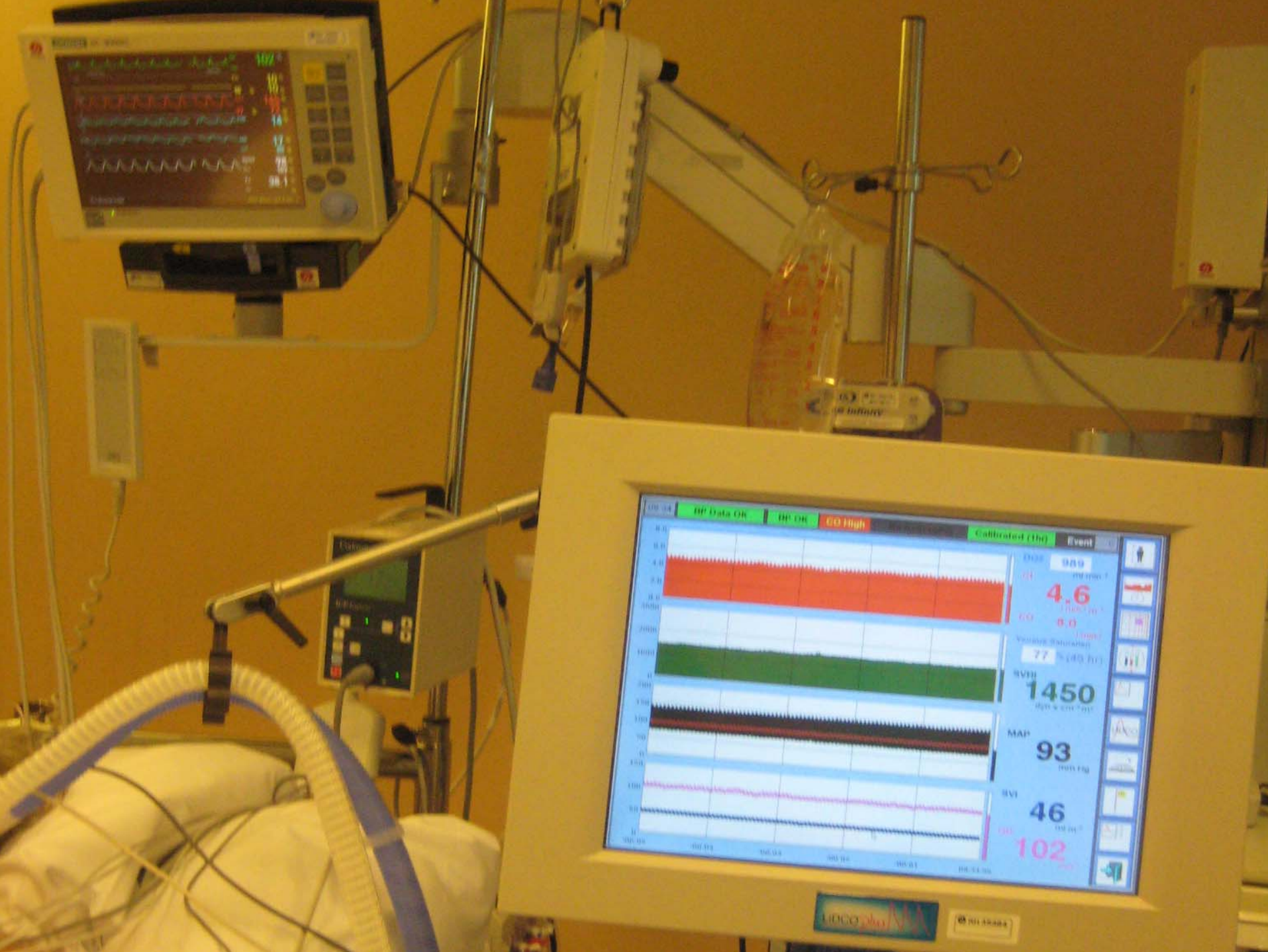
The LiDCOplus monitor – 2 algorithms

- **PulseCO** – continuous pulse power analysis
- **LiDCO**
  - Measuring SV with lithium dilution technique
  - Calibrated into the PulseCO

- A peripheral venous and arterial line

- Recalibration after delivery (?)


09:34
BP Data OK
BP OK
CO High
Calibrated
Event
8.0
6.0
4.0
2.0
0.0
3000
2000
1000
0
200
150
100
50
0
150
100
50
0
-00:05
-00:04
-00:03
-00:02
-00:01
09:34:23
DO2
981
ml min-1
CI
4.6
l min-1 m-2
CO
8.0
l min-1
Venous Saturation
77
% (45 hr)
SVRI
1450
dyn s cm-5 m2
MAP
92
mm Hg
SVI
45
ml m-2
HR
102
min-1
LiDCO
LiDCOplus
RH 35484


BP Data OK
BP OK
CO High
Calibrated (1hr)
Event
989
4.6
77
SVRI
1450
MAP
93
SVI
46
102

THE LITHIUM CHLORIDE INDICATOR DILUTION METHOD OF MEASURING CARDIAC OUTPUT

Fig 1

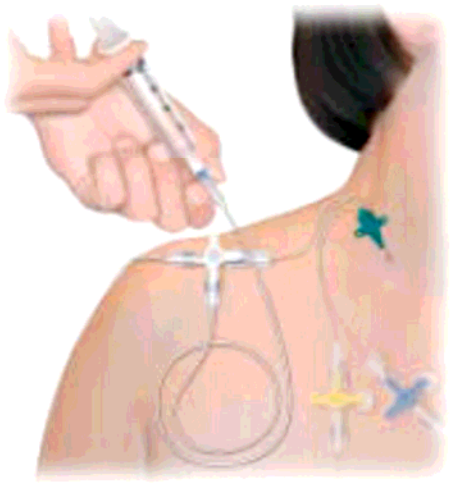


Fig 2

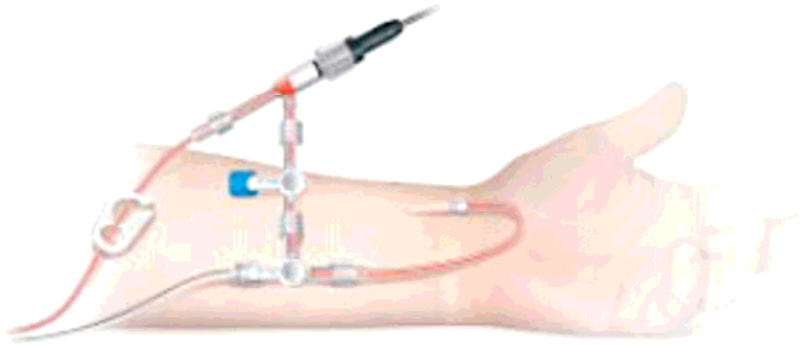
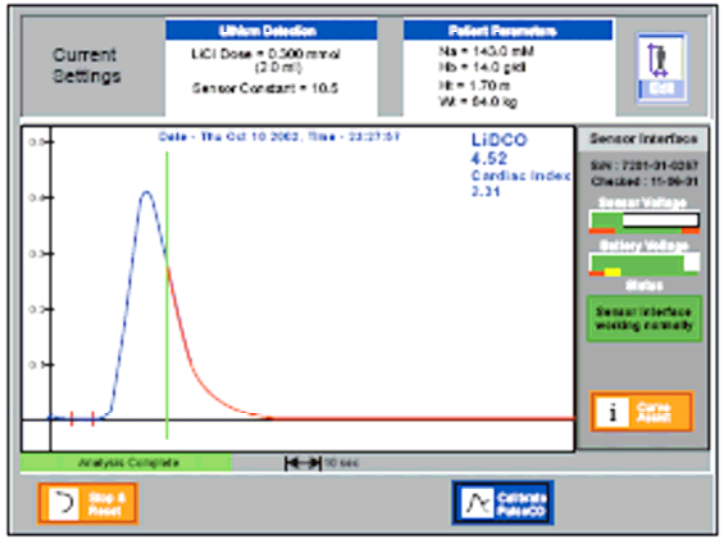


Fig 3



CLINICAL INVESTIGATIONS

Anesthesiology 2008; 108:802–11

Hemodynamic Changes Associated with Spinal Anesthesia for Cesarean Delivery in Severe Preeclampsia

Robert A. Dyer, F.C.A. (S.A.), Jenna L. Piercy, F.C.A. (S.A.),† Anthony R. Reed, F.R.C.A.,† Carl J. Lombard, Ph.D.,‡ Leann K. Schoeman, F.C.O.G. (S.A.),§ Michael F. James, Ph.D.‖*

EDITORIAL VIEWS

Anesthesiology 2008; 108:771–2

Is It More Informative to Focus on Cardiac Output than Blood Pressure during Spinal Anesthesia for Cesarean Delivery in Women with Severe Preeclampsia?

E.Langesæter

Anesthesiology 2008; 108:773–4

Pressure Wave Analysis Is Useful to Understand the Pathophysiology of Preeclampsia, but Perhaps Not the Rapid Changes during Cesarean Delivery

A. Pauca

Comparison between pulse waveform analysis and thermodilution cardiac output determination in patients with severe pre-eclampsia

R. A. Dyer[1*], J. L. Piercy[1,2], A. R. Reed[1], G. W. Strathie[2], C. J. Lombard[4], J. A. Anthony[3] and M. F. James[1]

Key points

- Cardiac output (CO) monitoring may be of value in patients with complicated severe pre-eclampsia.
- In view of potential risks of pulmonary artery catheter (PAC) insertion, thermodilution CO measurements were compared with values obtained from the minimally invasive LiDCOplus monitor.
- The comparison showed a statistically but not clinically significant bias after central venous calibration with lithium, and no significant bias after peripheral venous calibration.
- These findings support the use of LiDCOplus for haemodynamic monitoring in patients with complicated severe pre-eclampsia.

Dyer et al. BJA 2010

2 cases with severe PE – the same blood pressure

- **CASE A**

- **BP 193/90 (135)**
- HR 66

- CO 5.3 l/min
- SVR 1900

- **CASE B**

- **BP 189/96 (130)**
- HR 103

- CO 9.3 l/min
- SVR 1100

POSTPARTUM

- **CASE A**
- BP 145/86 (110)
- HR 71

- **SV 49**
- **CO 2.9 l/min**
- **SVR 2800**

- Vasodilatation
- Plasma volume-expansion
  - 10 litre crystalloids (24 hours)

- **CASE B**
- 147/79 (105)
- HR 64

- **SV 141**
- **CO 9.0 l/min**
- **SVR 900**

- Diuretics

The role of the anaesthetist in the management of the pre-eclamptic patient

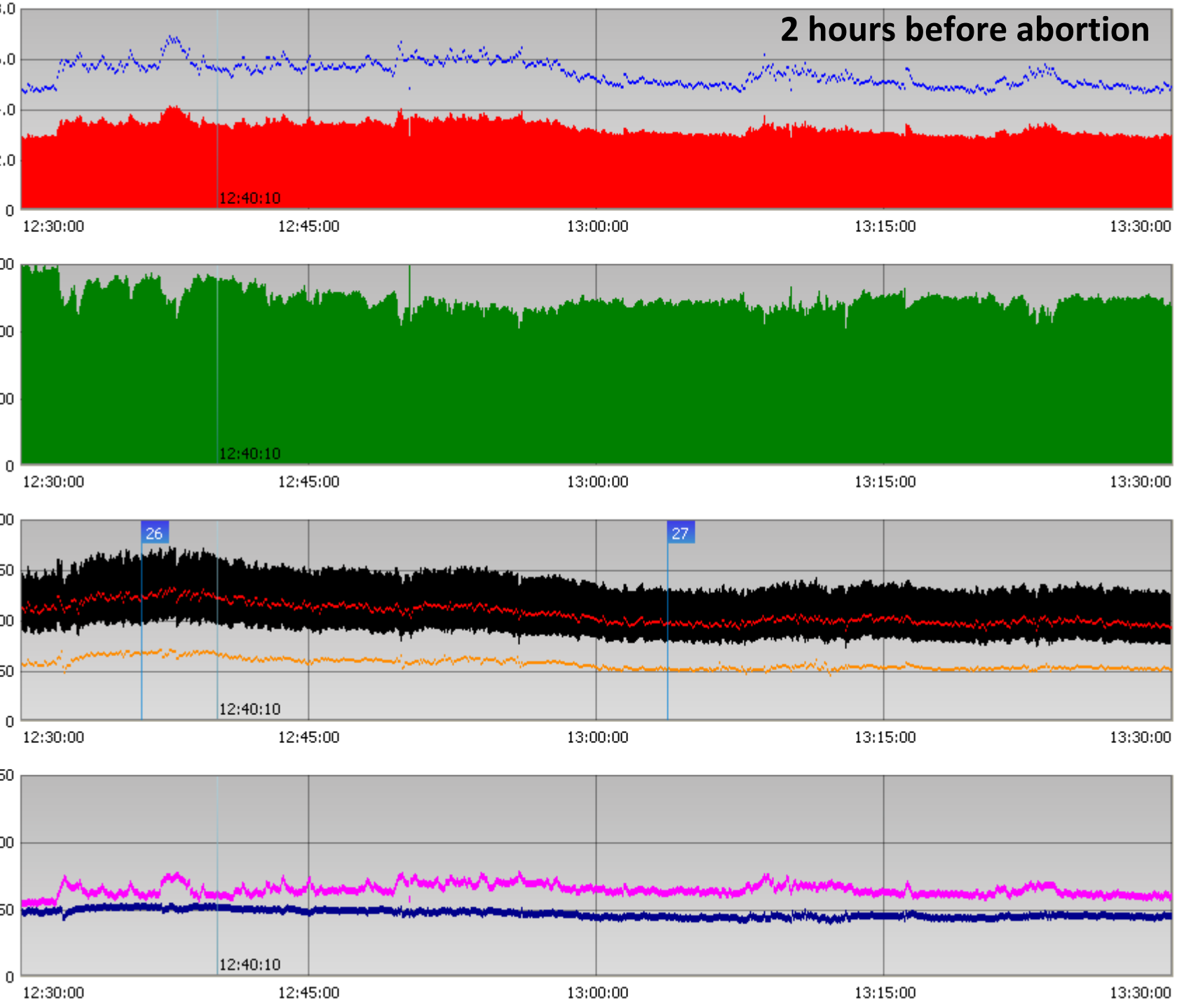
Robert A. Dyer, Jenna L. Piercy and Anthony R. Reed

Current Opinion in Anaesthesiology 2007, 20:168–174

”.., calibrated pulse contour or pulse power algorithms **may have a valuable future role,** because only an arterial and peripheral line are required. Simply measuring cardiac output and blood pressure allows calculation of the systemic vascular resistance, which **guides the appropriate use of fluids, diuretics, vasodilators** and, occasionally, inotropes.”

ABORTION GA 21.6

- **Renal causes or preeclampsia?**



CO l min⁻¹
2,9

DO_2 ml min⁻¹
491

SVR dyn s cm⁻⁵
2884

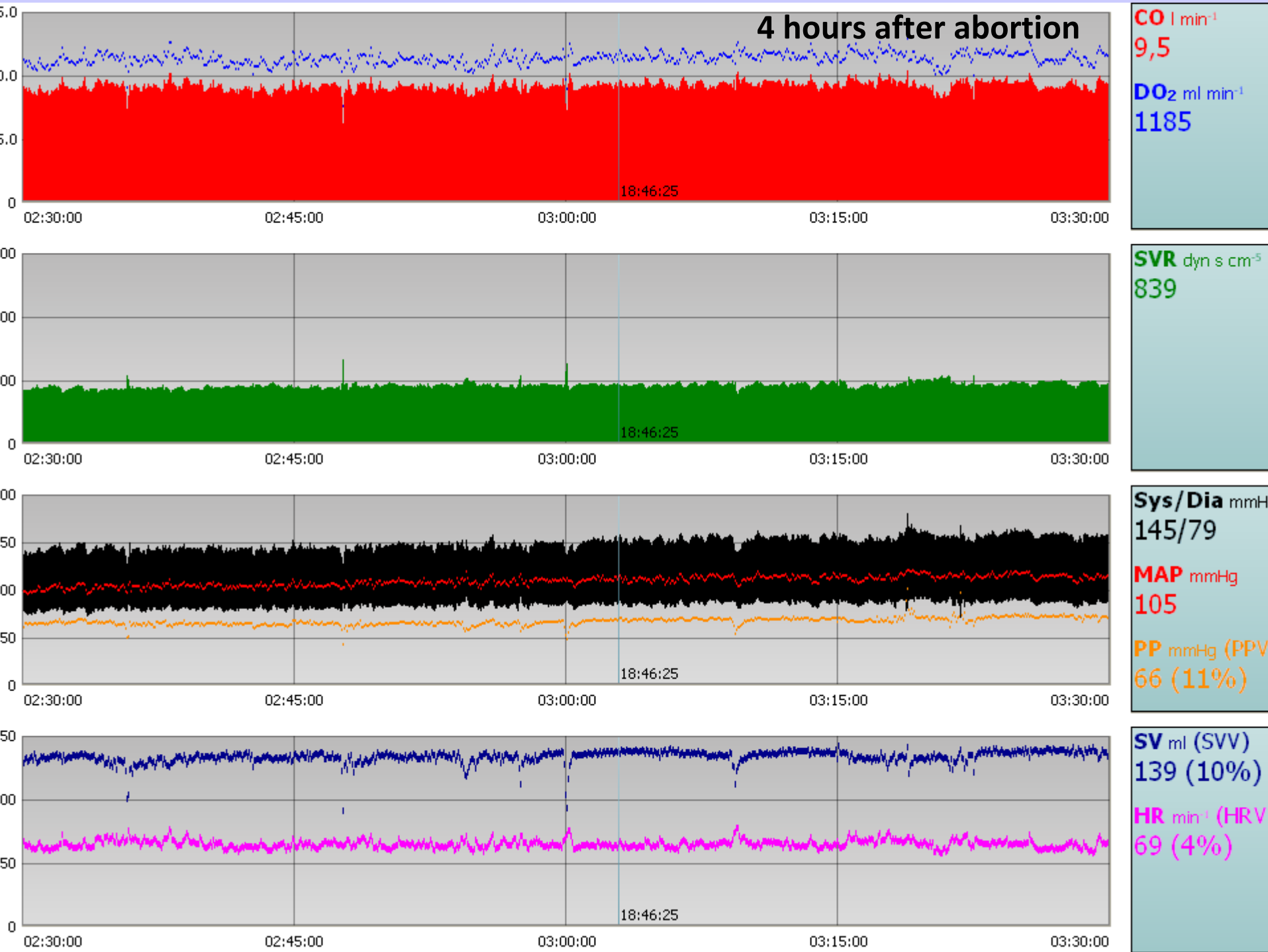
Sys/Dia mmHg
144/88

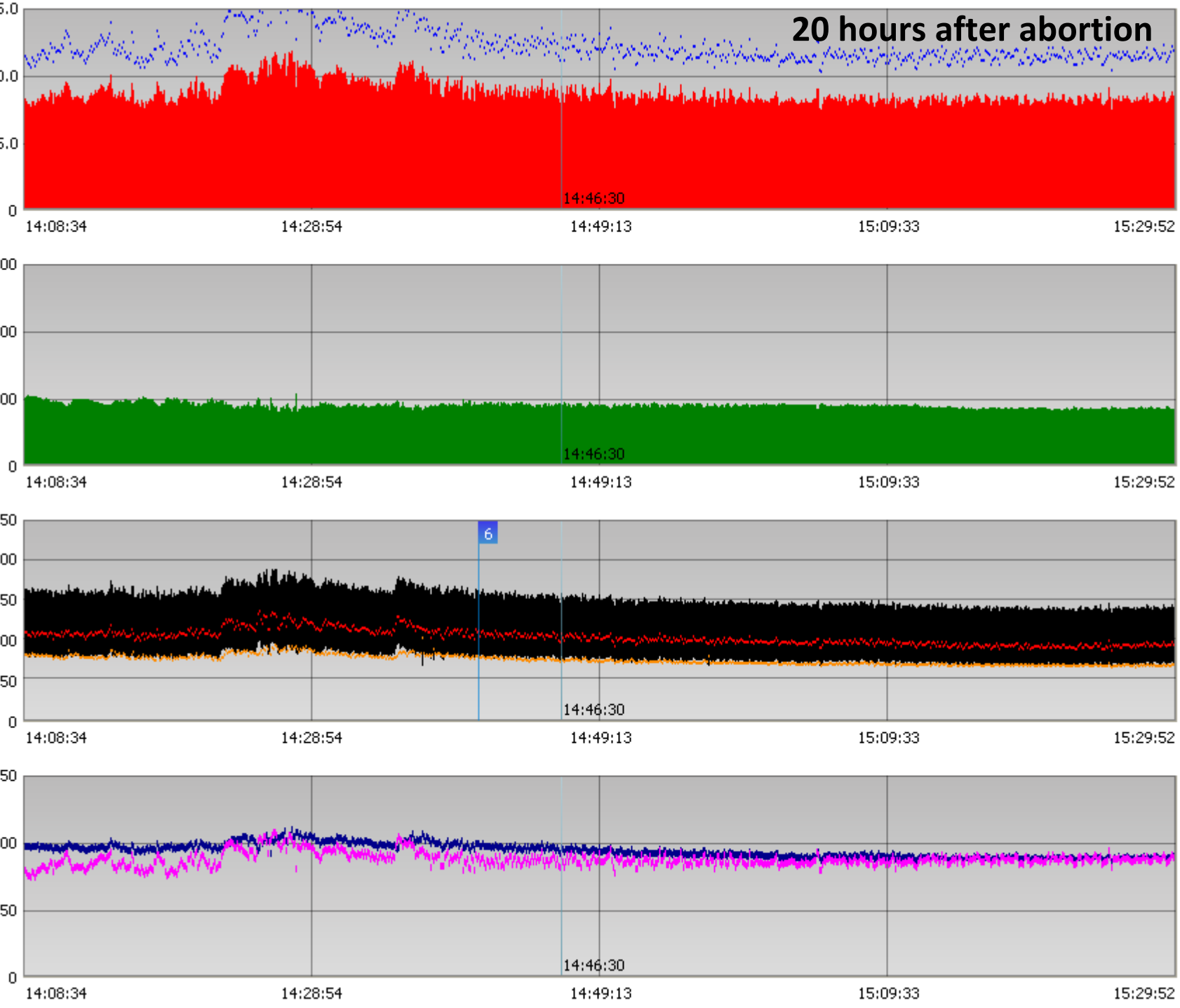
MAP mmHg
111

PP mmHg (PPV
56 (7%)

SV ml (SVV)
50 (6%)

HR min⁻¹ (HRV)
59 (1%)


4 hours after abortion
CO l min-1
9,5
DO2 ml min-1
1185
SVR dyn s cm-5
839
Sys/Dia mmH
145/79
MAP mmHg
105
PP mmHg (PPV
66 (11%)
SV ml (SVV)
139 (10%)
HR min-1 (HRV
69 (4%)
18:46:25
02:30:00
02:45:00
03:00:00
03:15:00
03:30:00



CO l min^{-1}
8,7

DO_2 ml min^{-1}
1217

SVR dyn s cm^{-5}
883

Sys/Dia mmH
149/76

MAP mmHg
103

PP mmHg (PPV
73 (5%)

SV ml (SVV)
94 (5%)

HR min^{-1} (HRV
92 (2%)

ANAESTHESIA FOR CAESAREAN SECTION

- Spinal anaesthesia
- Oxytocin

BJA

"I would have everie man write what he knowes and no more."–Montaigne

BRITISH JOURNAL OF ANAESTHESIA

Volume 102, Number 3, March 2009

British Journal of Anaesthesia **102** (3): 291–4 (2009)
doi:10.1093/bja/aep003

Editorial I

Hypotension in obstetric spinal anaesthesia: a lesson from pre-eclampsia

G. Sharwood-Smith & G.B. Drummond

STUDIES ON AUTONOMIC BLOCKADE. I. COMPARISON BETWEEN THE EFFECTS OF TETRAETHYLAMMONIUM CHLORIDE (TEAC) AND HIGH SELECTIVE SPINAL ANESTHESIA ON BLOOD PRESSURE OF NORMAL AND TOXEMIC PREGNANCY [1]

By N. S. ASSALI AND HARRY PRYSTOWSKY [2]

(From the Department of Obstetrics, University of Cincinnati College of Medicine, and the Cincinnati General Hospital, Cincinnati)

(Submitted for publication March 23, 1950; accepted, July 18, 1950)

12 Healthy – all hypotensive after SA

15 PE – 4 hypotensive after SA (2 superimposed PE)

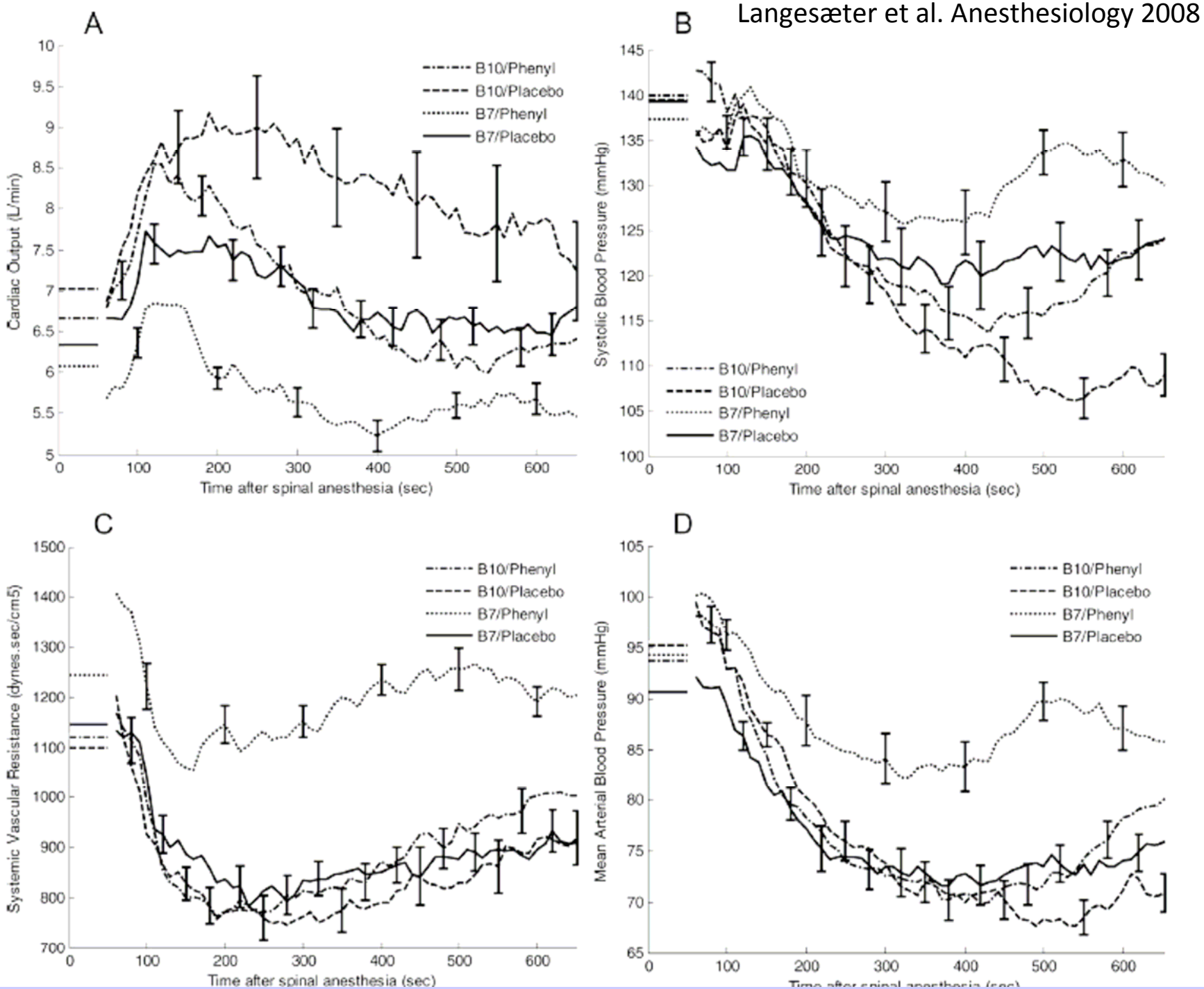
Spinal catheter L3-4, 16 G

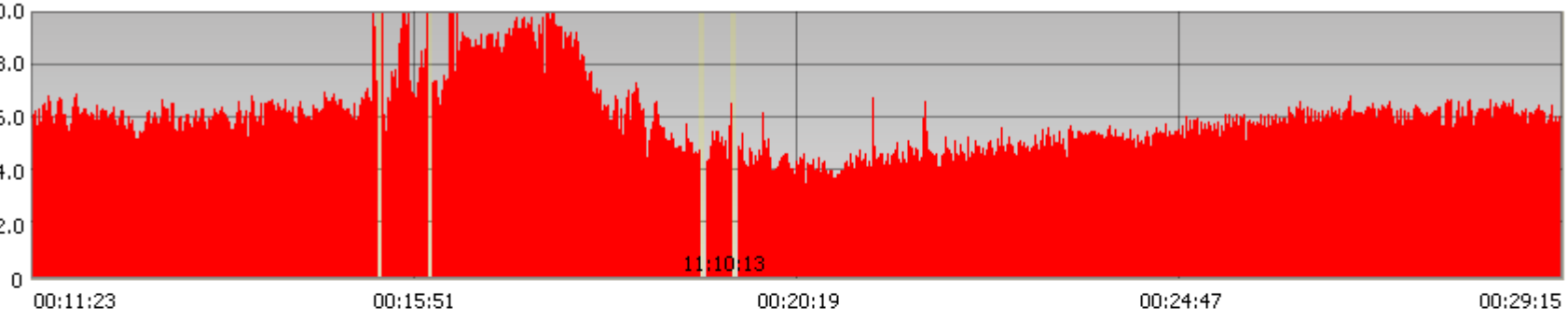
- Procaine 0.2%, 5-8 ml (C8-T2)

In cases where the blood pressure fell to dangerous levels with spinal anesthesia and the patient's condition seemed seriously affected, raising the legs 90 degrees was the only measure used to improve the patient's condition.

(2) *Side effects:* At the height of the depressor response with the prepartum spinal test, nausea, vomiting, dizziness, weakness, fatigue, yawning, generalized numbness, perspiration of the forehead, cyanosis, hyperpnea, and other signs of imminent collapse occurred in all the patients.

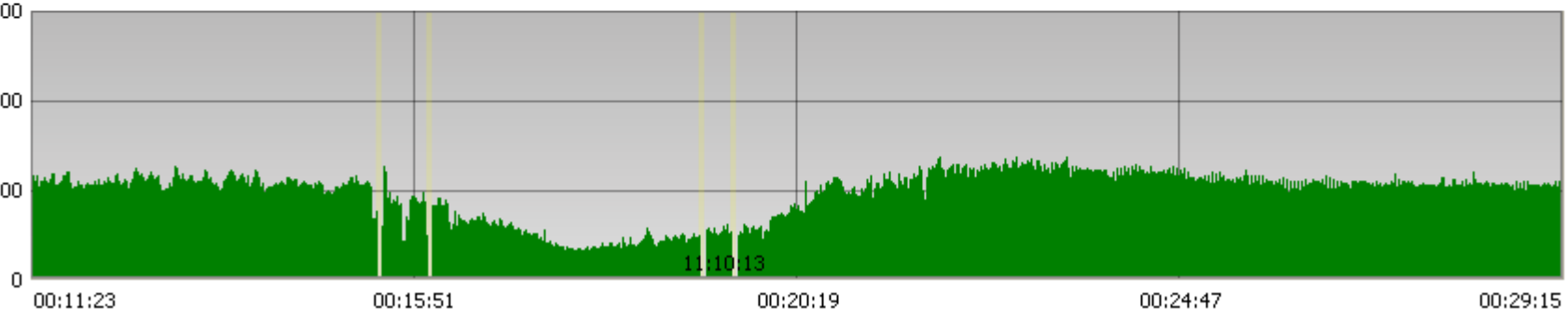
Langesæter et al. Anesthesiology 2008



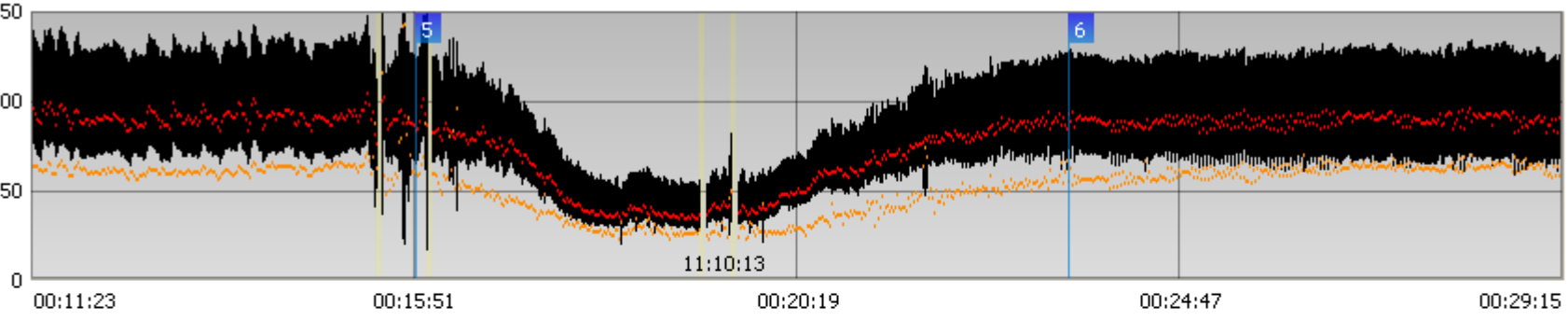


CO l min^{-1}
6,2

DO$_2$ ml min^{-1}
0



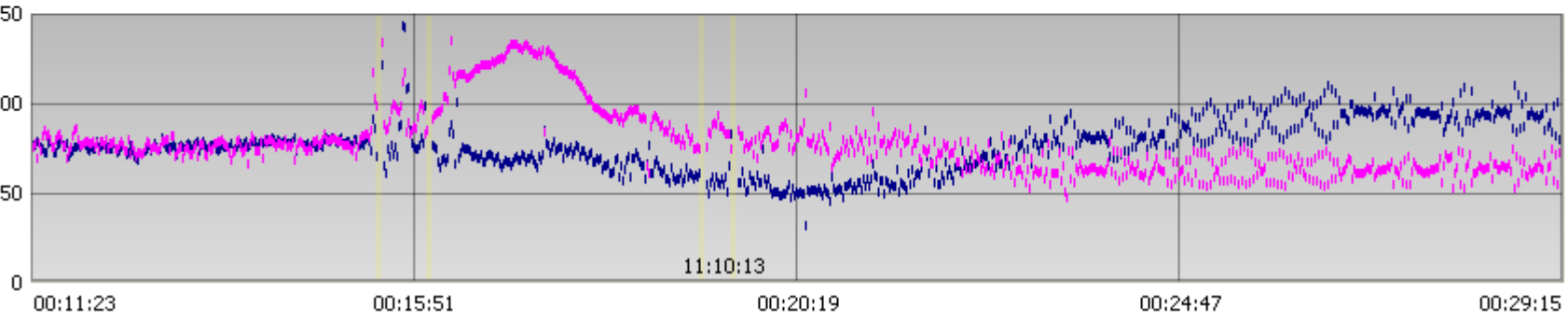
SVR dyn s cm^{-5}
368



Sys/Dia mmH
53/21

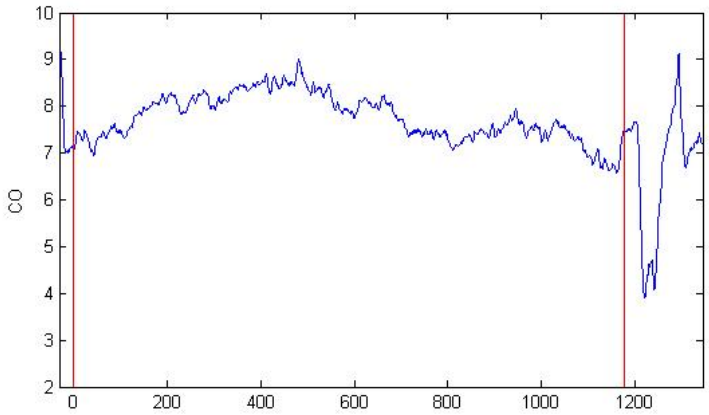
MAP mmHg
35

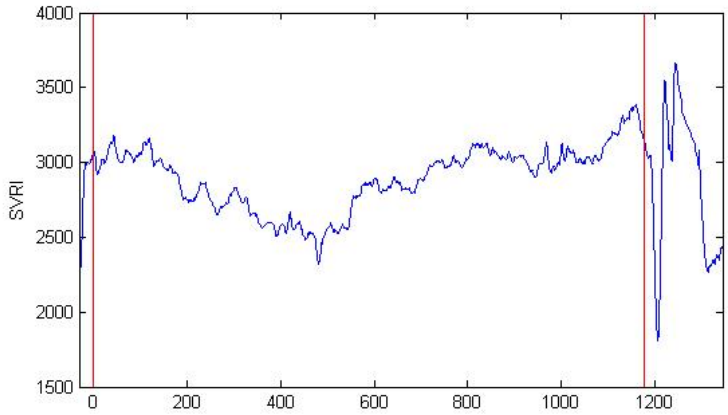
PP mmHg (PPV
32 (56%)

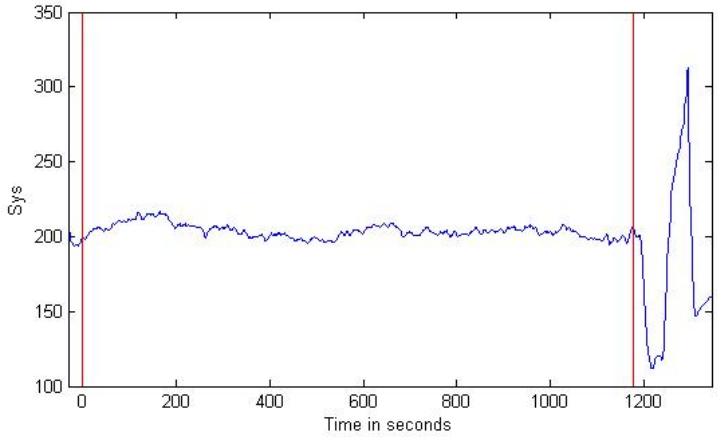


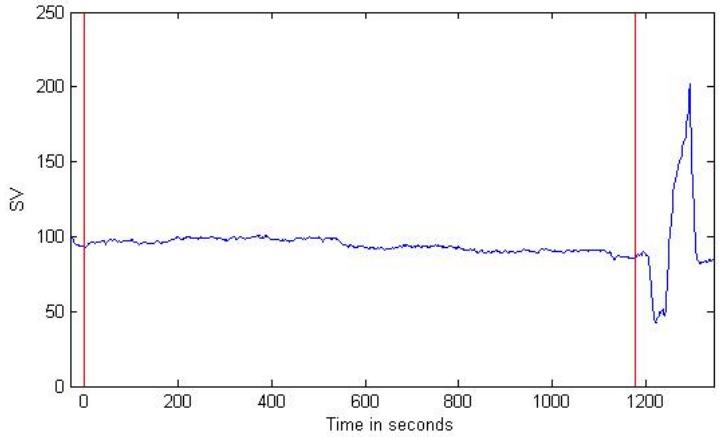
SV ml (SVV)
67 (38%)

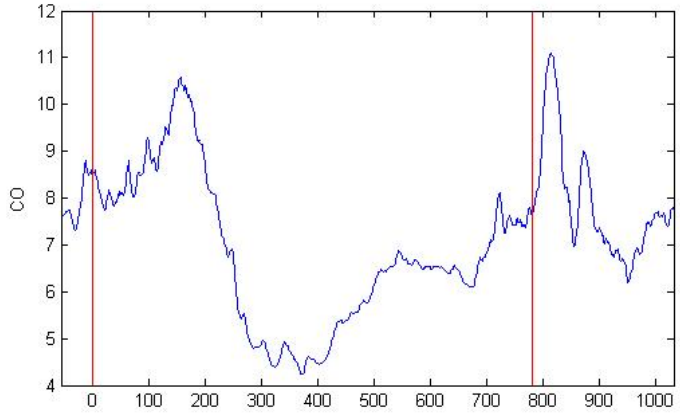
HR min^{-1} (HRV
94 (3%)

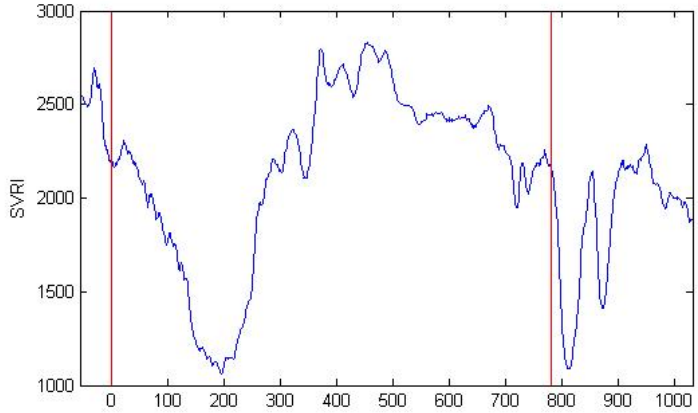

CO
10
9
8
7
6
5
4
3
2
0
200
400
600
800
1000
1200

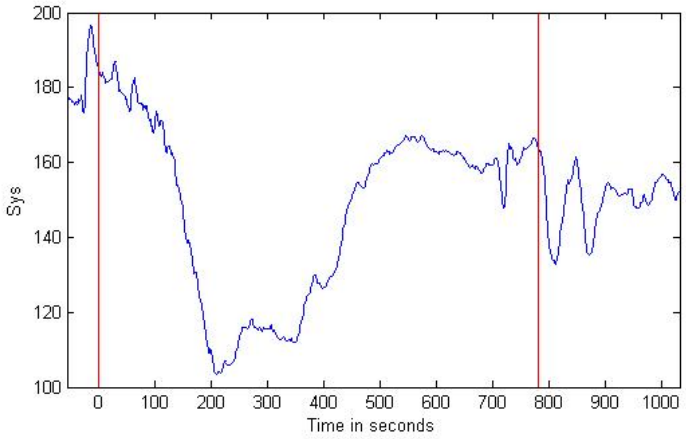

SVRI
4000
3500
3000
2500
2000
1500
0
200
400
600
800
1000
1200

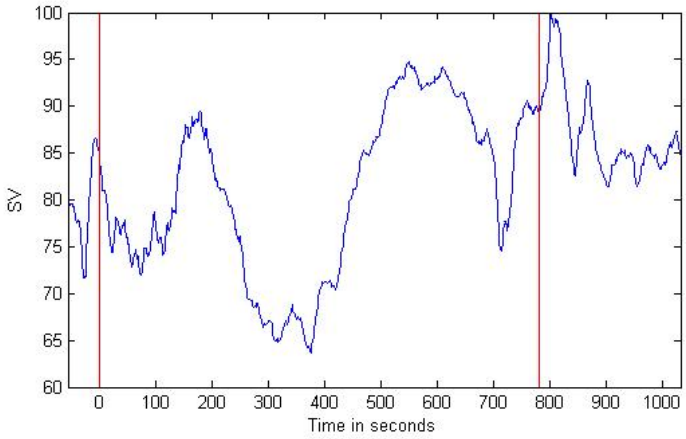

Sys
350
300
250
200
150
100
0
200
400
600
800
1000
1200
Time in seconds

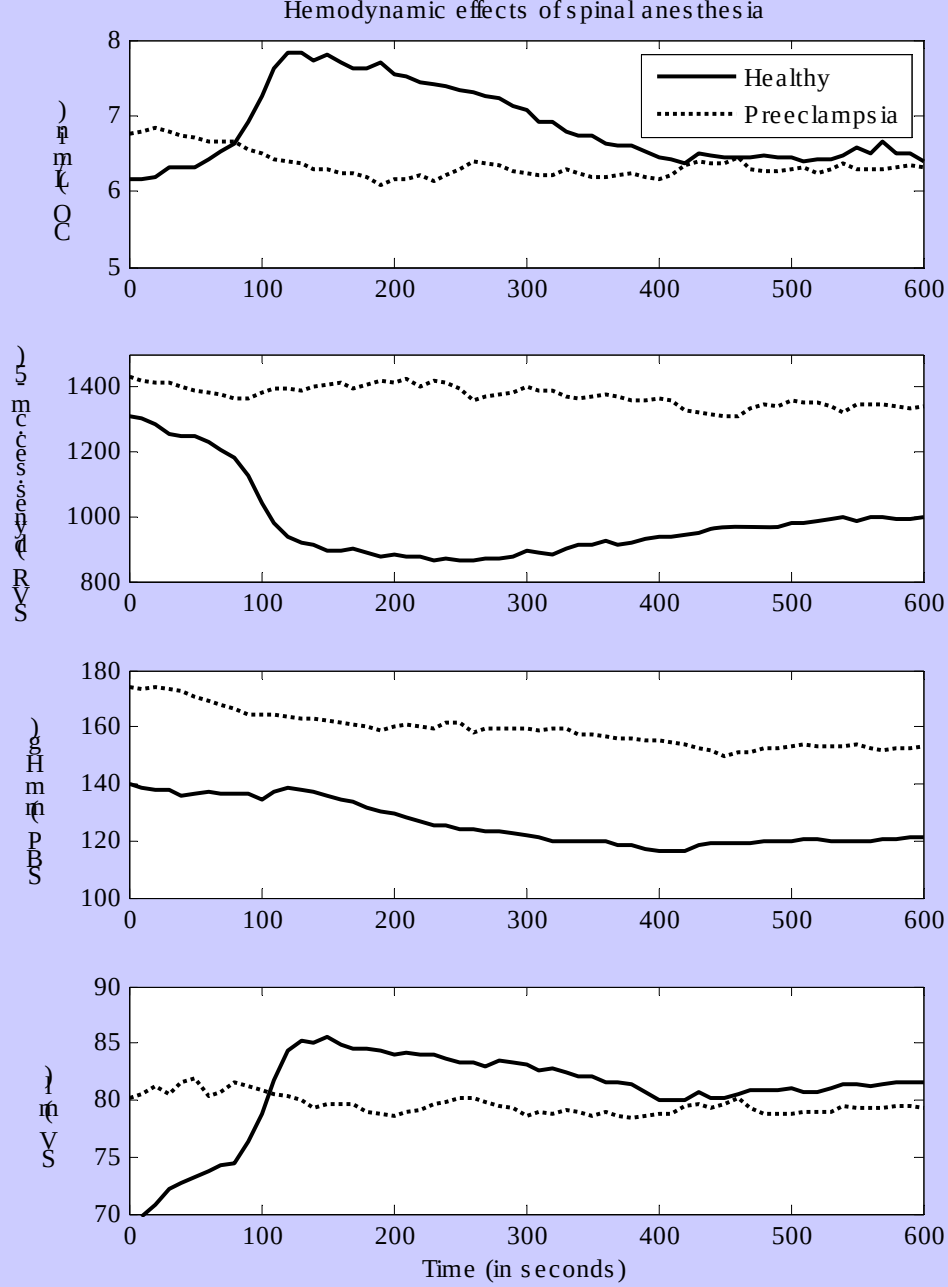

SV
250
200
150
100
50
0
0
200
400
600
800
1000
1200
Time in seconds


CO
12
11
10
9
8
7
6
5
4
0
100
200
300
400
500
600
700
800
900
1000


SVRI
3000
2500
2000
1500
1000
0
100
200
300
400
500
600
700
800
900
1000


Sys
200
180
160
140
120
100
0
100
200
300
400
500
600
700
800
900
1000
Time in seconds


SV
100
95
90
85
80
75
70
65
60
0
100
200
300
400
500
600
700
800
900
1000
Time in seconds



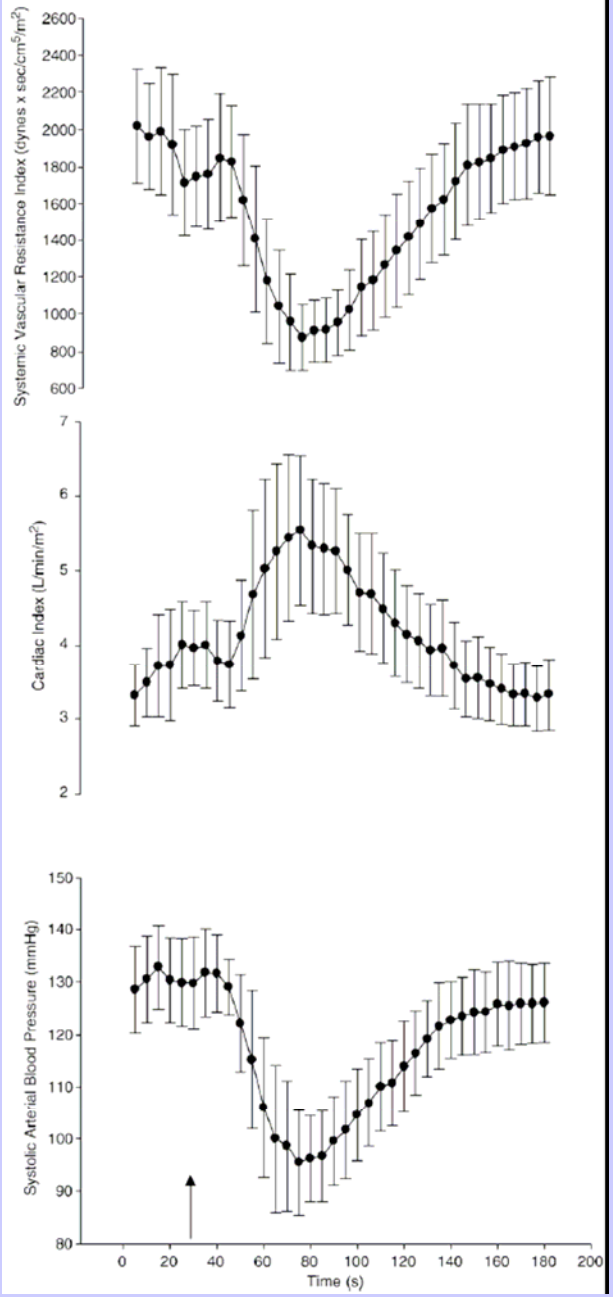
SPINAL ANAESTHESIA

31 women with severe PE

80 healthy pregnant women

(unpublished data)

OXYTOCIN



OXYTOCIN 5 IE i.v

Langesæter et al. Int J Gyn Obst **2006**;95:46-7

ED95% 0.35 units (not in labour)

ED95% 3 units (in labour)

Carvalho et al. Obst Gyn **2004**;104;1005-10

Balki et al. Obst Gyn **2006**;107:45-50

Signs of myocardial ischaemia after injection of oxytocin: a randomized double-blind comparison of oxytocin and methylergometrine during Caesarean section

M. C. Svanström[1], B. Biber[3], M. Hanes[3], G. Johansson[3], U. Näslund[2] and E. M. Bålfors[4]*

BJA 2008

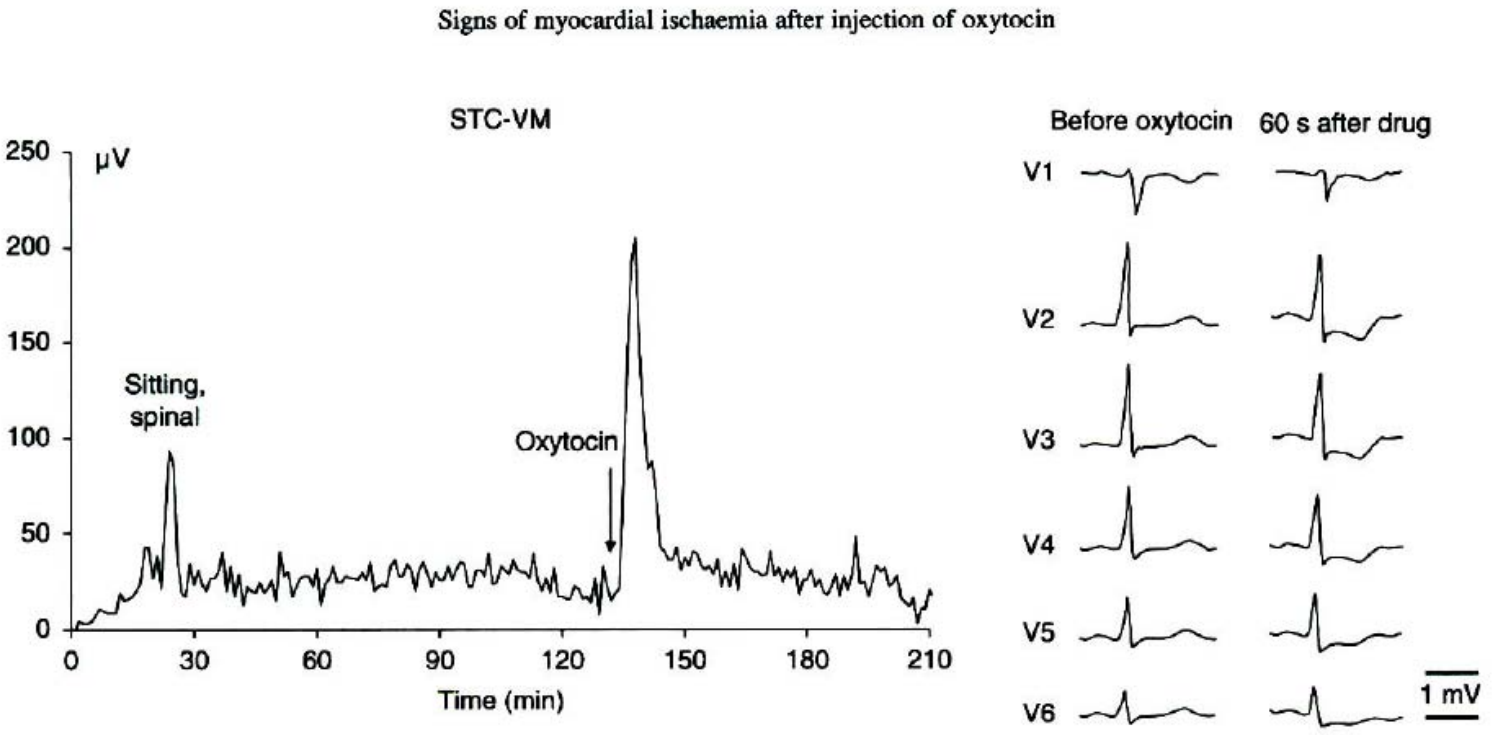
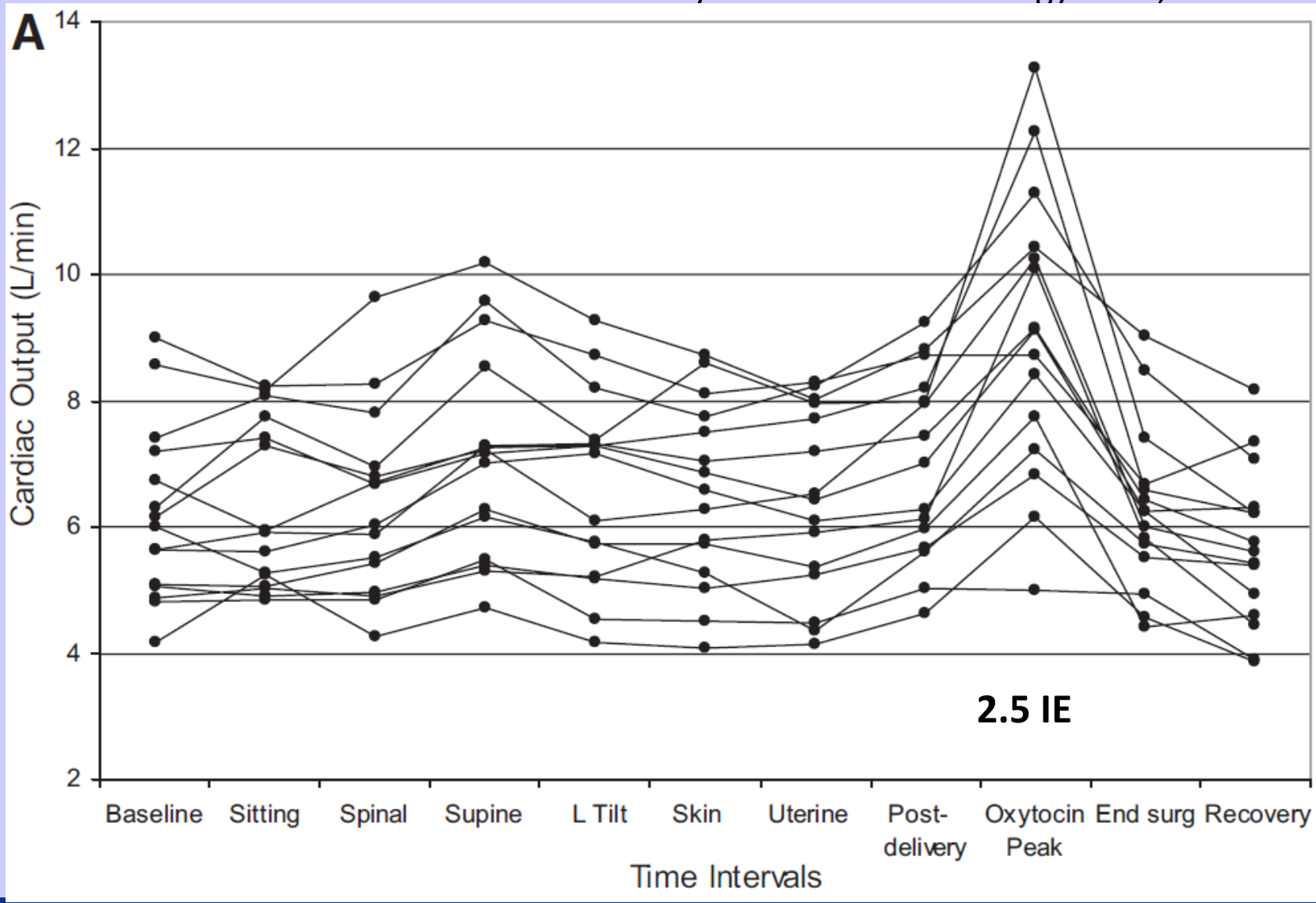


Fig 3 Recordings of scalar ECG and VCG in one OXY-CS woman after 10 IU of oxytocin i.v. Note the pronounced peak in STC-VM in the VCG trend curve and the ST-segment depression in the scalar ECG.

Hemodynamic Changes Associated with Spinal Anesthesia for Cesarean Delivery in Severe Preeclampsia

Dyer et al. Anesthesiology **2008**;108:802-11



International Journal of Obstetric Anesthesia (2011) **20**, 26–29
0959-289X/$ - see front matter
doi:10.1016/j.ijoa.2010.10.004

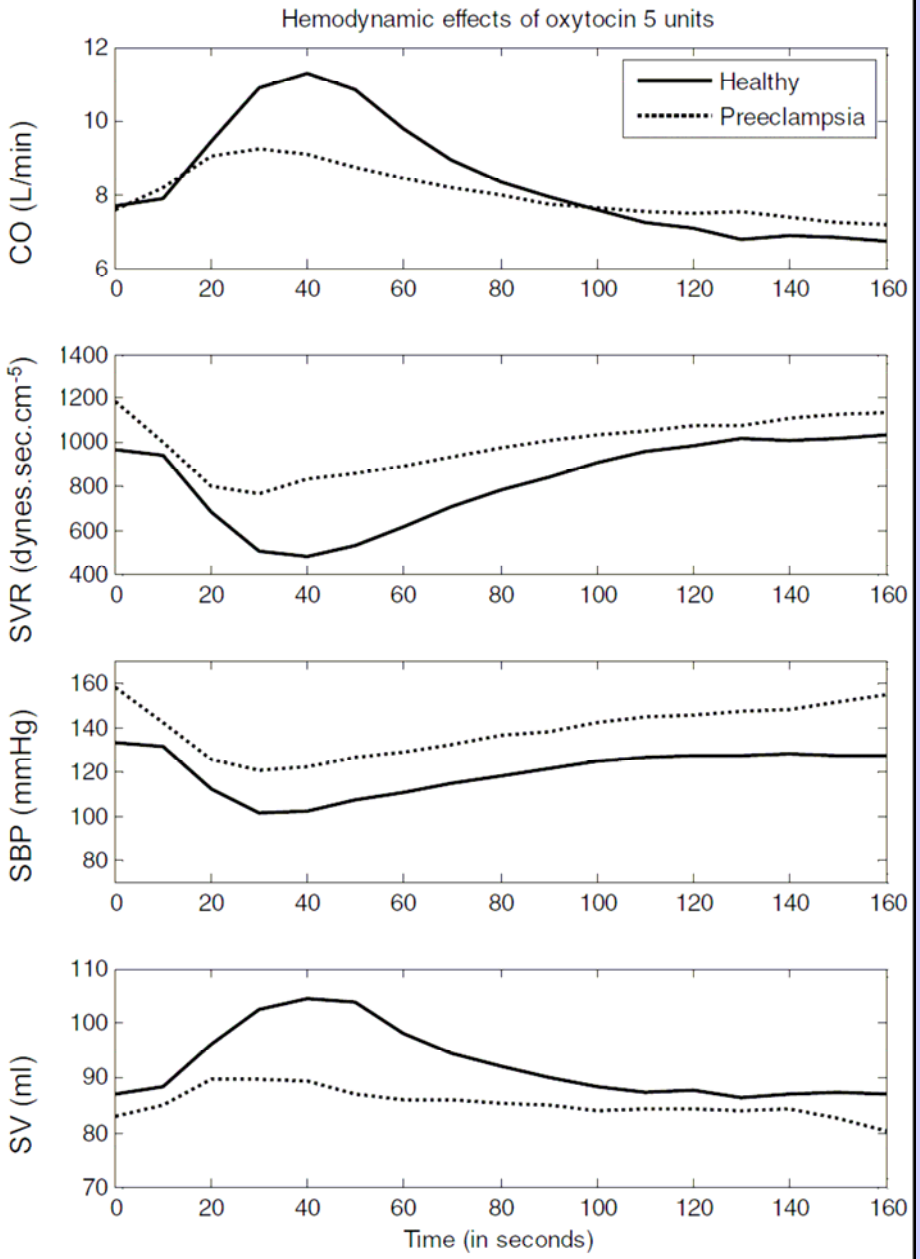
www.obstetanesthesia.com

ORIGINAL ARTICLE

Haemodynamic effects of oxytocin in women with severe preeclampsia

E. Langesæter, L.A. Rosseland, A. Stubhaug
Division of Anaesthesia and Intensive Care Medicine, Oslo University Hospital – Rikshospitalet, Oslo, Norway

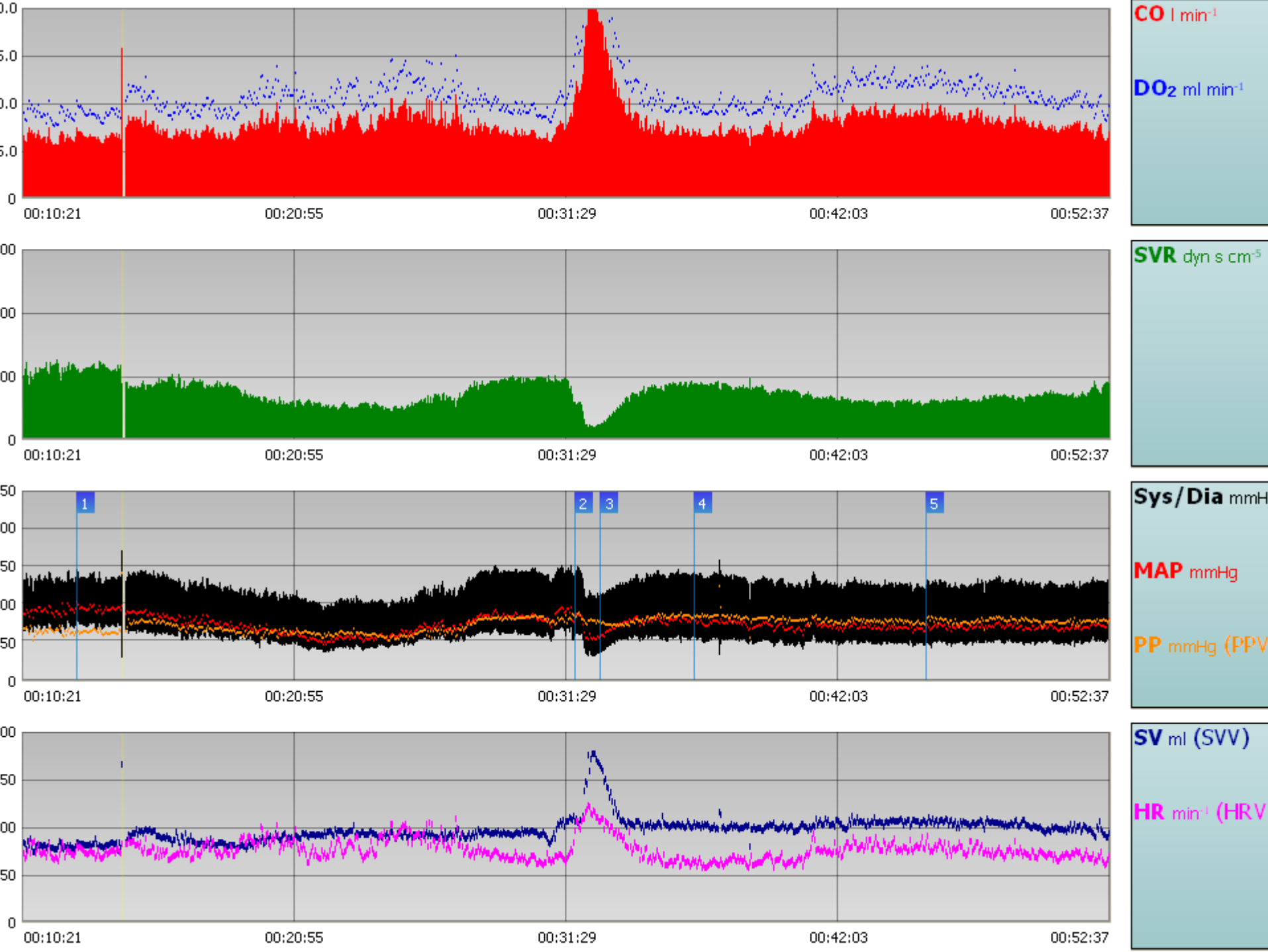
5/18 women with severe preeclampsia had a decrease in cardiac output after 5 units oxytocin i.v

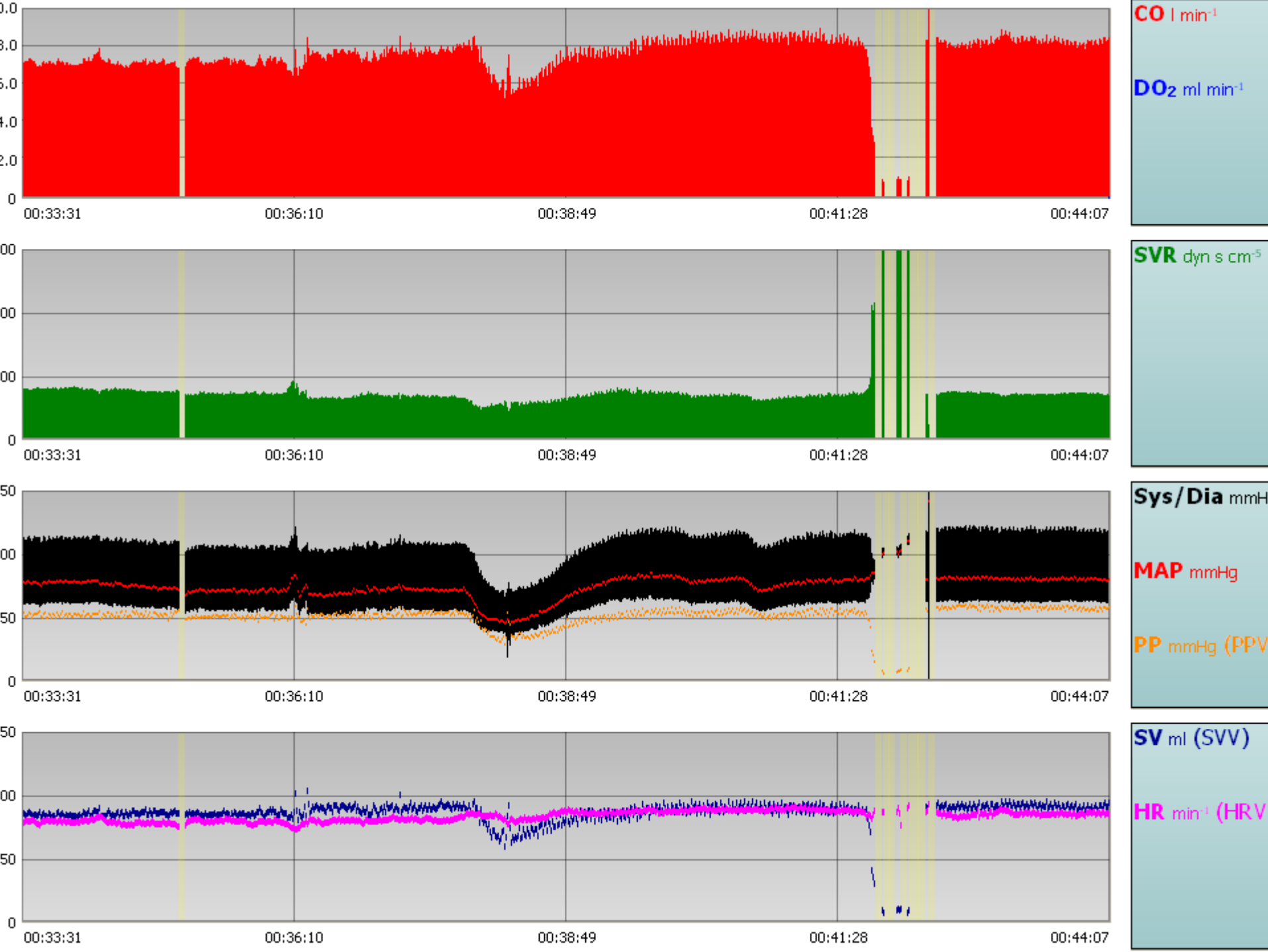


5 units oxytocin i.v

18 women with severe preeclampsia

80 healthy pregnant women


CO l min-1
DO2 ml min-1
SVR dyn s cm-5
Sys/Dia mmH
MAP mmHg
PP mmHg (PPV
SV ml (SVV)
HR min-1 (HRV
00:10:21
00:20:55
00:31:29
00:42:03
00:52:37
1
2
3
4
5


CO l min-1
DO2 ml min-1
SVR dyn s cm-5
Sys/Dia mmH
MAP mmHg
PP mmHg (PPV
SV ml (SVV)
HR min-1 (HRV
0
2.0
4.0
6.0
8.0
00:33:31
00:36:10
00:38:49
00:41:28
00:44:07

Maternal haemodynamic changes during spinal anaesthesia for caesarean section

Eldrid Langesæter[a] and Robert A. Dyer[b]

[a]Division of Critical Care, Department of Anaesthesiology, Oslo University Hospital, Oslo, Norway and [b]Department of Anaesthesia, University of Cape Town, Cape Town, South Africa

Correspondence to Eldrid Langesæter, MD, PhD, Consultant Anaesthesiologist, Division of Critical Care, Department of Anaesthesiology, Oslo University Hospital, Oslo, Norway
Tel: +47 230700 00;
e-mail: eldrid.langesaeter@oslo-universitetssykehus.no

Current Opinion in Anaesthesiology 2011, 24:000–000

Purpose of review

Maternal haemodynamic changes during spinal anaesthesia for caesarean section have traditionally been evaluated by noninvasive blood pressure and heart rate. Recent publications have addressed the importance of cardiac output measurement in the assessment of the maternal circulation. In this review, a physiological approach is suggested for the prevention and treatment of haemodynamic instability during caesarean section in healthy women and in those with preeclampsia or cardiac disease.

Recent findings

A better understanding of the maternal haemodynamic effects of spinal anaesthesia and the effects of vasopressors has emerged from the monitoring of cardiac output during caesarean section in healthy women and in those with severe preeclampsia or cardiac disease. Based on maternal physiological arguments, phenylephrine is the vasopressor of choice in healthy pregnant women. New work demonstrating cardiac dysfunction in some women with severe preeclampsia has implications for risk assessment and anaesthesia. Recent publications suggest that combined spinal-epidural and continuous spinal anaesthesia is well tolerated in pregnant women with cardiac disease.

Summary

The most frequent response to spinal anaesthesia for elective caesarean section is a marked decrease in systemic vascular resistance and partial compensation from increased stroke volume and heart rate. Early administration of phenylephrine by bolus or continuous infusion is indicated in most cases. Recent work has expanded our knowledge of the therapeutic range of phenylephrine and indicates that the heart rate response to vasopressors is a good surrogate marker for cardiac output. Further research should examine haemodynamic changes during spinal anaesthesia in high-risk pregnant women with early onset preeclampsia or cardiac disease.

Oxytocin for labour and caesarean delivery: implications for the anaesthesiologist

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Purpose of review

The implications of the obstetric use of oxytocin for obstetric anaesthesia practice are summarised. The review focuses on recent research on the uterotonic effects of oxytocin for prophylaxis and management of uterine atony during caesarean delivery.

Recent findings

Oxytocin remains the first-line agent in the prevention and management of uterine atony. In-vitro and in-vivo studies show that prior exposure to oxytocin induces uterine muscle oxytocin receptor desensitization. This may influence oxytocin dosing for adequate uterine tone following delivery. Oxytocin has important cardiovascular side-effects (hypotension, tachycardia and myocardial ischaemia). Recent studies suggest that the effective dose of oxytocin for prophylaxis against uterine atony during caesarean delivery is significantly lower than the 5–10 IU historically used by anaesthesiologists. Slow administration of small bolus doses of oxytocin minimises maternal haemodynamic disturbance. Continuous oxytocin infusions are recommended for maintaining uterine tone after bolus administration, although ideal infusion rates are still to be established. The efficacy of the long-acting oxytocin analogue carbetocin requires further investigation. Recommendations are presented for oxytocin dosing during caesarean delivery.

Summary

Oxytocin remains the first-line uterotonic after vaginal and caesarean delivery. Recent research elucidates the therapeutic range of oxytocin during caesarean delivery, as well as receptor desensitization. Evidenced-based protocols for the prevention and treatment of uterine atony during caesarean delivery are recommended.

- Anaesthetist involved early
- Screening with ecco cor (pro-BNP)
- Focus on fluid balance (diuresis)
- Magnesium Sulphate
- Antihypertensive treatment postpartum
- Long-time follow up

- Impact on outcome? Reduced morbidity?

- **Antihypertensive treatment**, SAP 150 mmHg
- **MgSO4** and volume
- Continuous invasive blood pressure monitoring
- Titrated doses of oxytocin
- **Continue antihypertensive therapy postpartum**
  - SAP < 140 mmHg
    - Labetolol, Adalat Oros 30 mg
- Diuresis
  - Volume
  - Diuretics if dyspnoe
  - Low treshold for ecco cor

FUTURE RESEARCH

- **Predicting risk** in women with preeclampsia
  - Use of non-invasive techniques (echocardiography)
- **How to randomise** in this heterogenous patient group?
  - Plasma volume expansion?
- PPCM and PE

Imitators of Severe Pre-eclampsia

Baha M. Sibai, MD

Semin.Perinatol. **2009**, **33:**196-205.

There are many obstetric, medial, and surgical disorders that share many of the clinical and laboratory findings of patients with severe pre-eclampsia–eclampsia. Imitators of severe pre-eclampsia–eclampsia are life-threatening emergencies that can develop during pregnancy or in the postpartum period. These conditions are associated with high maternal and perinatal mortalities and morbidities, and survivors may face long-term sequelae. The pathophysiologic abnormalities in many of these disorders include vasospasm, platelet activation or destruction, microvascular thrombosis, endothelial cell dysfunction, and reduced tissue perfusion. Some of these disorders include acute fatty liver of pregnancy, thrombotic thrombocytopenic purpura, hemolytic uremic syndrome, acute exacerbation of systemic lupus erythematosus, and disseminated herpes simplex and sepsis syndromes. Differential diagnosis may be difficult due to the overlap of several clinical and laboratory findings of these syndrome. It is important that the clinician make the accurate diagnosis when possible because the management and complications from these syndromes may be different. Because of the rarity of these conditions during pregnancy and postpartum, the available literature includes only case reports and case series describing these syndromes. This review focuses on diagnosis, management, and counseling of women who develop these syndromes based on results of recent studies and my own clinical experience.

KEYWORDS severe pre-eclampsia, acute fatty liver, TTP, HUS

ACUTE FATTY LIVER OF PREGNANCY

Systemic Arterial Vasodilation, Vasopressin, and Vasopressinase in Pregnancy

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J Am Soc Nephrology 2010

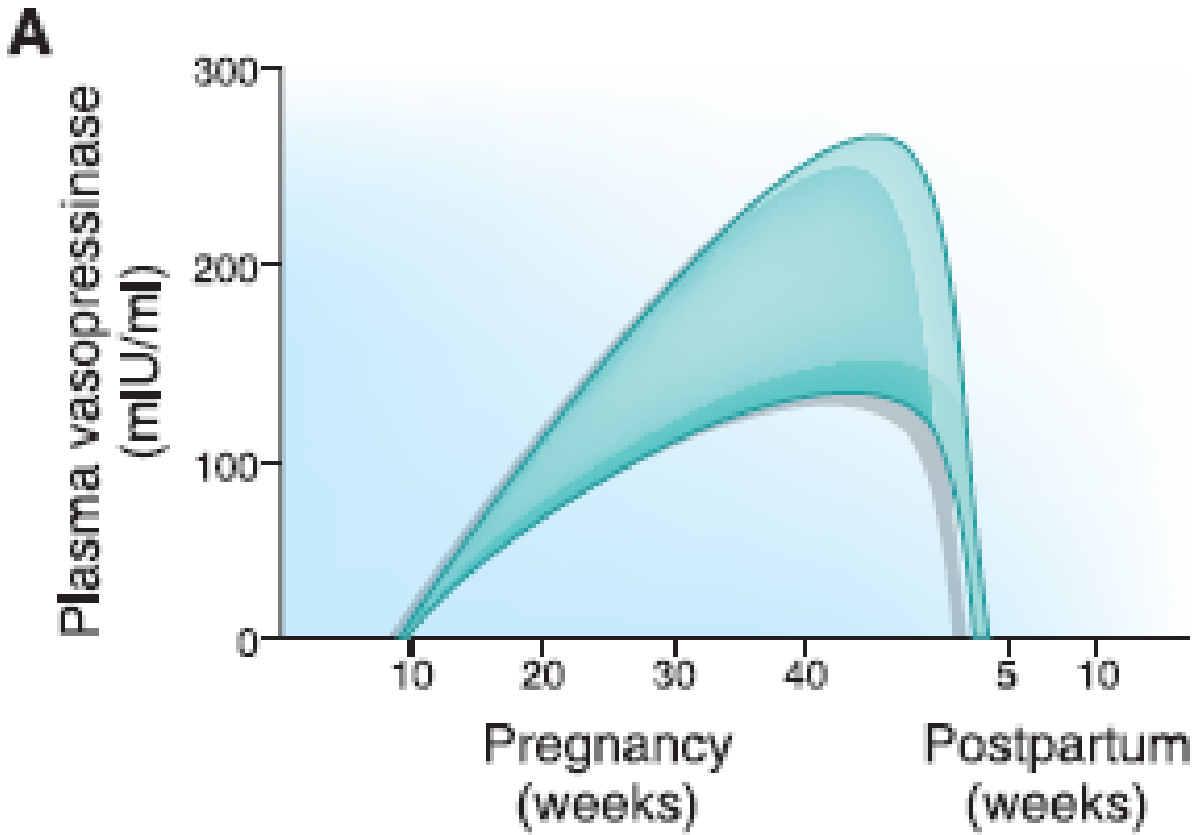


TABLE 2. Hemodynamic Profile in Untreated and Treated Patients With Severe Preeclampsia

	Untreated			Treated		
	Nulliparous ($n=74$)	p	Parous ($n=13$)	Nulliparous ($n=32$)	p	Parous ($n=15$)
Systemic circulation						
Heart rate (beats·min^{-1})	74 (57–110)	NS	78 (51–95)	84 (64–131)*	NS	87 (62–135)
Mean intra-arterial pressure (mm Hg)	125 (92–156)	NS	123 (96–143)	120 (80–138)*	NS	120 (105–154)
Cardiac index (l·min^{-1}·m^{-2})	3.4 (2.1–5.3)	NS	3.1 (2.0–4.2)	4.3 (3.0–7.6)*	NS	3.8 (2.4–7.0)†
Stroke volume index (ml·beat^{-1}·m^{-2})	48 (25–75)	<0.05	35 (29–56)	52 (32–82)*	NS	52 (33–65)†
Systemic vascular resistance index (dyne·sec·cm^{-5}·m^2)	2,951 (1,771–5,225)	NS	3,331 (1,827–4,753)	2,162 (1,057–3,325)*	NS	2,581 (1,177–3,688)†

Visser, Hypertension **1991**

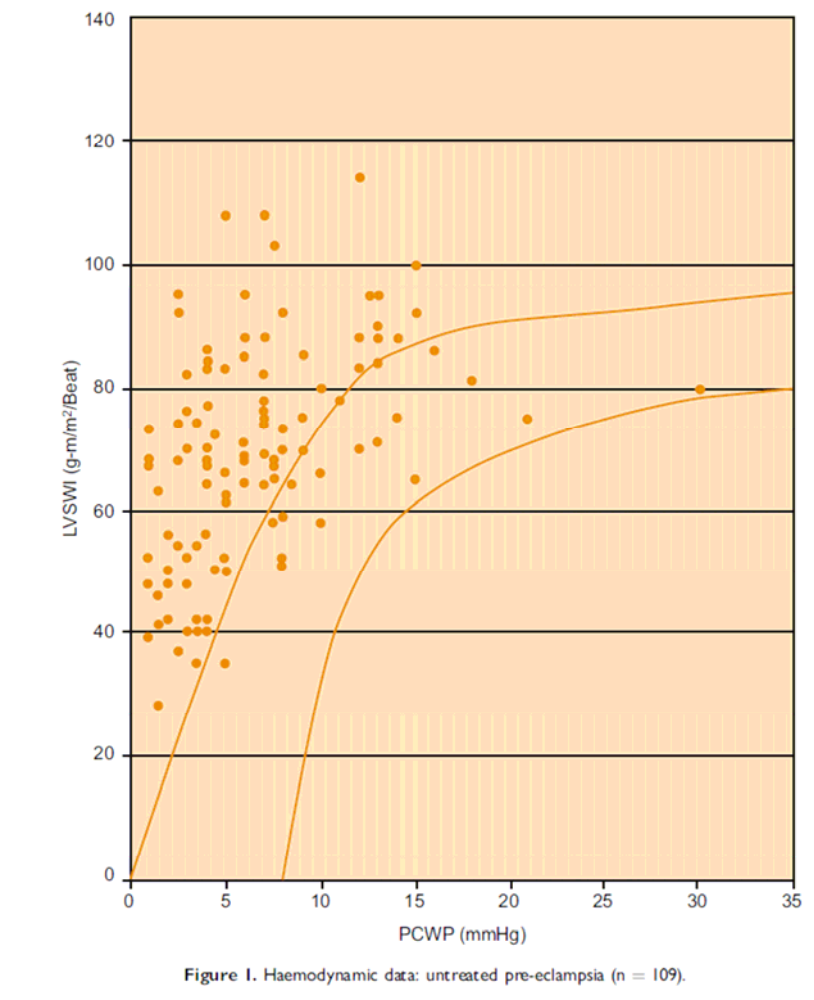


Figure 1. Haemodynamic data: untreated pre-eclampsia (n = 109).

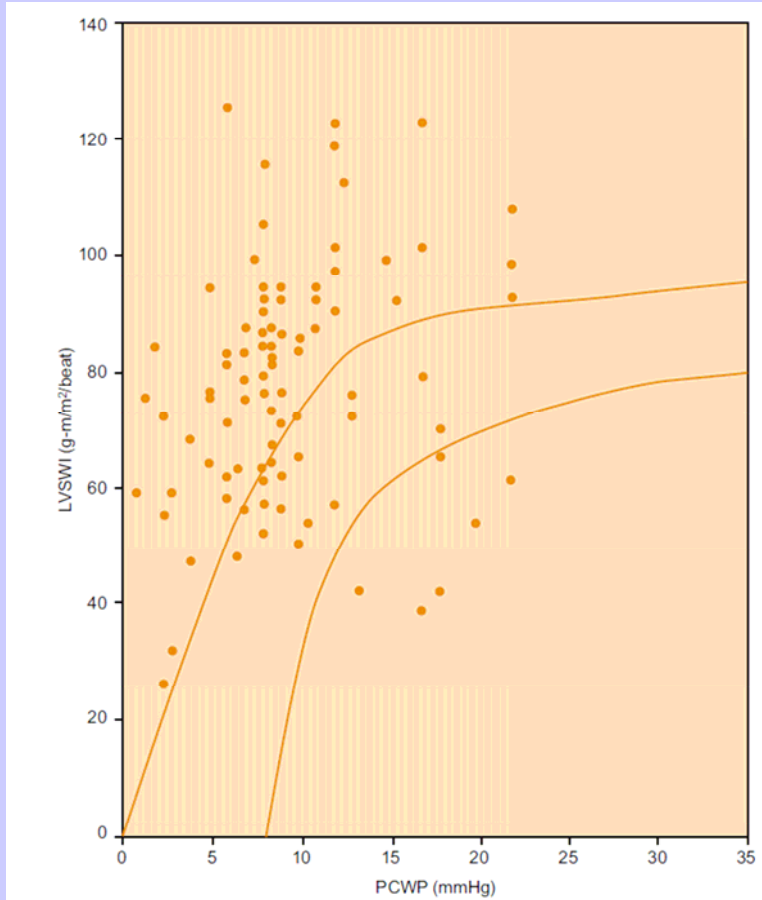


Figure 2. Treated pre-eclampsia.

Table 4. Haemodynamic data in treated pre-eclampsia.

Trial	CVP (mmHg)	PCWP (mmHg)	SVR (dyne.cm.sec^{-5})	PVR (dyne.cm.sec^{-5})	CO (l.min^{-1})
Untreated					
Visser and Wallenburg[56] n = 87[a]	2	7	3003	131	(CI = 3.3 l.min^{-1}.m^{-2})
Cotton et al[12] n = 45	4	10	1496	70	7.5
Wallenburg[22] n = 44[a]	1	4	2970	82	(CI = 3.1 l.min^{-1}.m^{-2})
Treated					
Mabie et al[25] n = 41	4.8	8.3	1226	65	8.4
Visser and Wallenburg[56] n = 47[a]	1	7	2212	101	(CI = 4.3 l.min^{-1}.m^{-2})
Wallenburg[22] n = 22[a]	2	8	2475	128	(CI = 3.8 l.min^{-1}.m^{-2})

Results expressed as mean.
[a] Results expressed as median.

Can umbilical pH be used as end point?

- Umbilical pH
  - Can not be used for evaluation of “the best anaesthesia”
- Apgar
- Not sensitive enough…

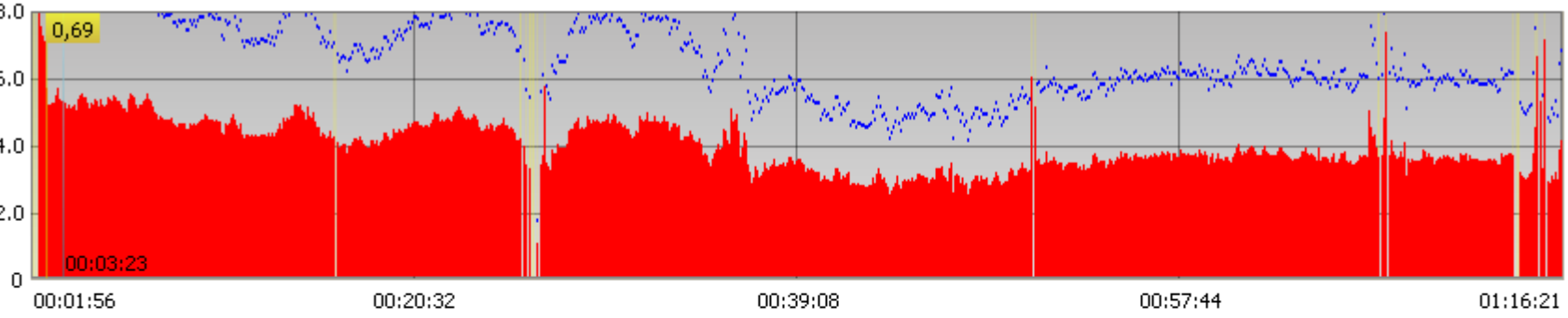
Clark 1989

Table 2. Central hemodynamic changes in normal pregnancy

Measurement	Nonpregnant	Pregnant	Change From Nonpregnant
Cardiac output, L/min	4.3 ± 0.9	6.2 ± 10	43%
Heart rate, beats/min	71 ± 10	83 ± 10	17%
Systemic vascular resistance, dyne · cm · sec^{-5}	1530 ± 520	1210 ± 266	−21%
Mean arterial pressure, mm Hg	86.4 ± 7.5	90.3 ± 5.8	NS
Pulmonary artery occlusion pressure, mm Hg	6.3 ± 2.1	7.5 ± 1.8	NS
Central venous pressure, mm Hg	3.7 ± 2.6	3.6 ± 2.5	NS
Colloid oncotic pressure, mm Hg	20.8 ± 1.0	18.0 ± 1.5	−14%

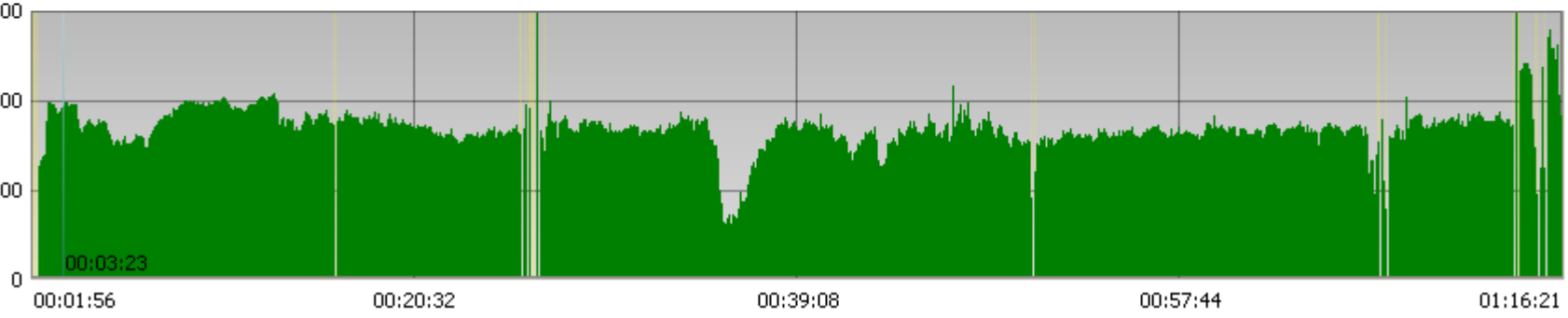
NS, not significant. Reproduced from Ref. 6; Copyright © 1989, with permission from Elsevier.

Cardiac output 6.5
SVR 1129
MAP 93.5
HR 93

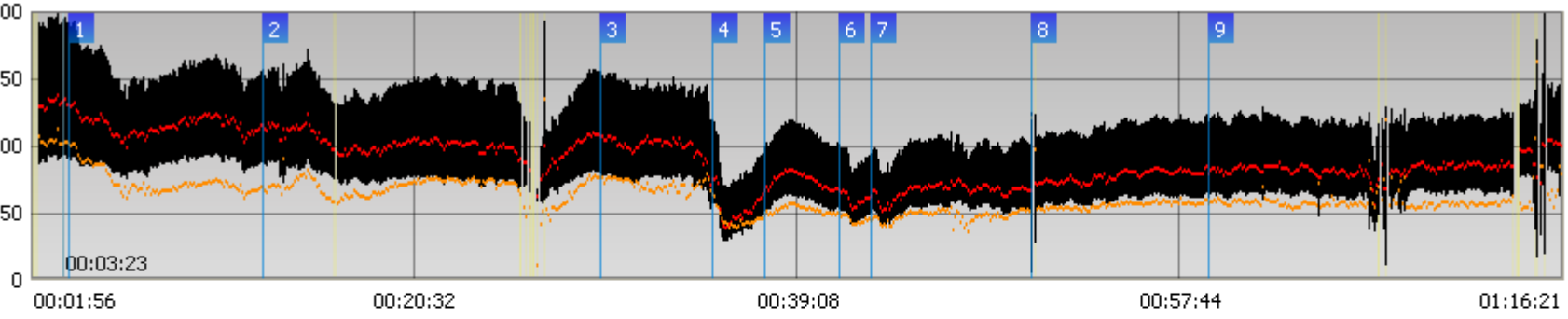


CO l min^{-1}
5,4

DO_2 ml min^{-1}
897



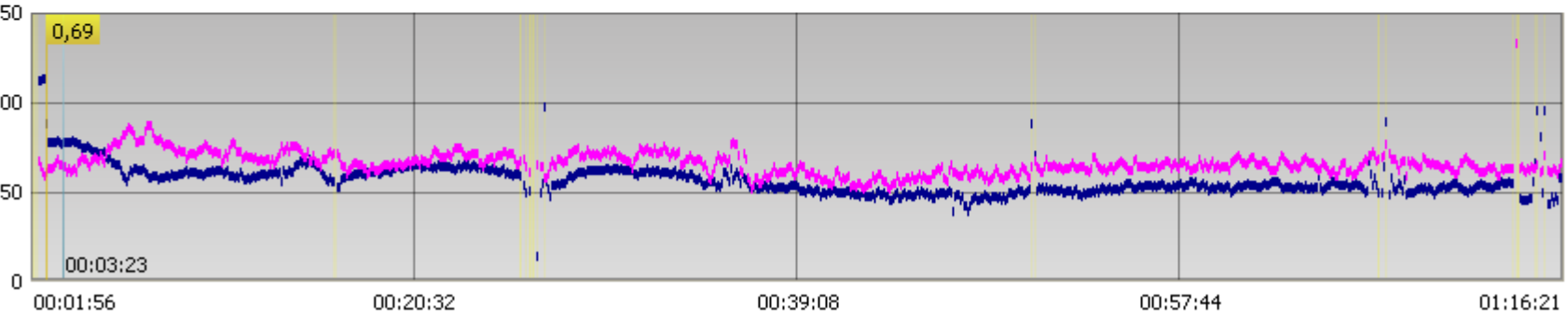
SVR dyn s cm^{-5}
1933



Sys/Dia mmH
195/92

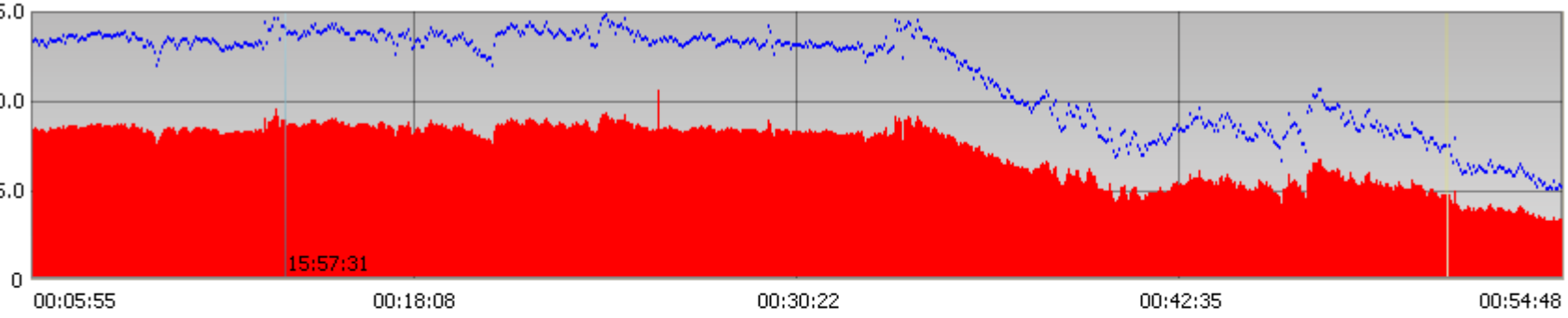
MAP mmHg
135

PP mmHg (PPV
103 (5%)



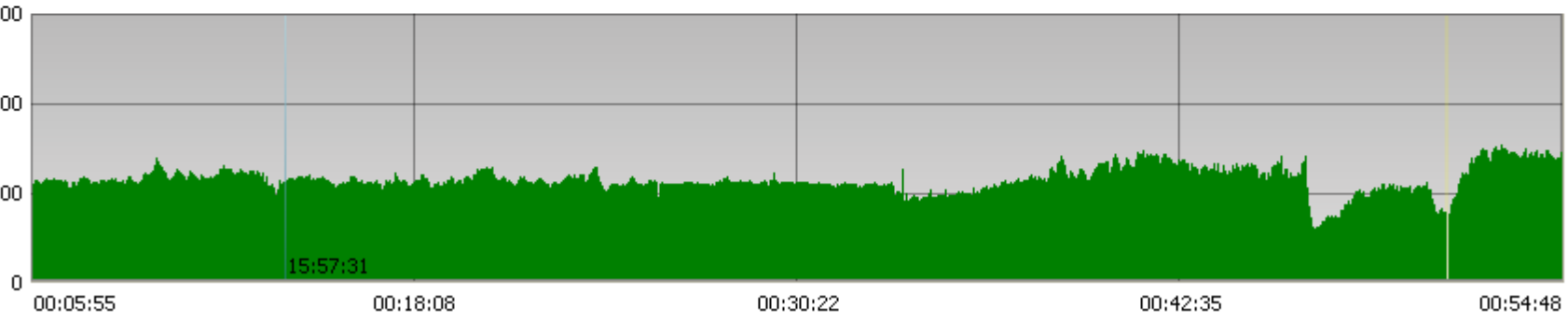
SV ml (SVV)
80 (3%)

HR min^{-1} (HRV
67 (2%)

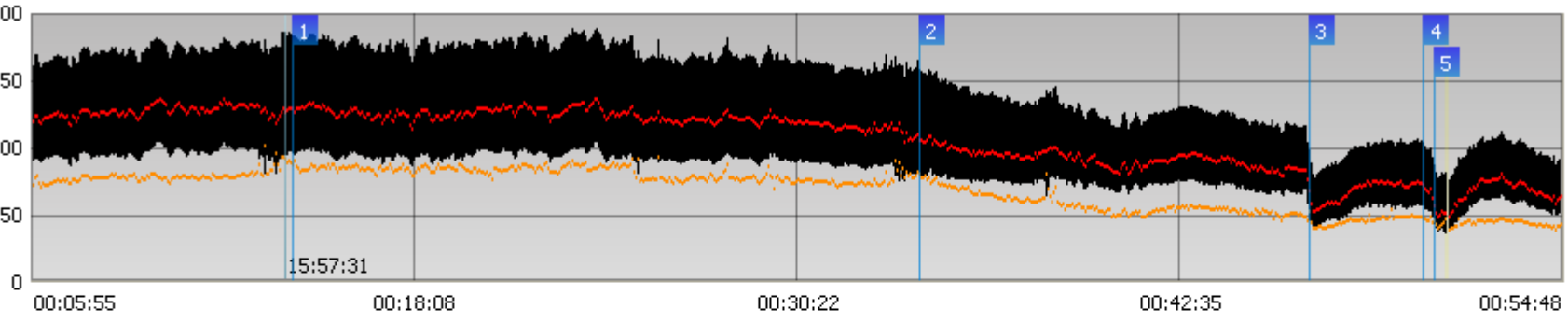


CO l min^{-1}
8,9

DO$_2$ ml min^{-1}
1421



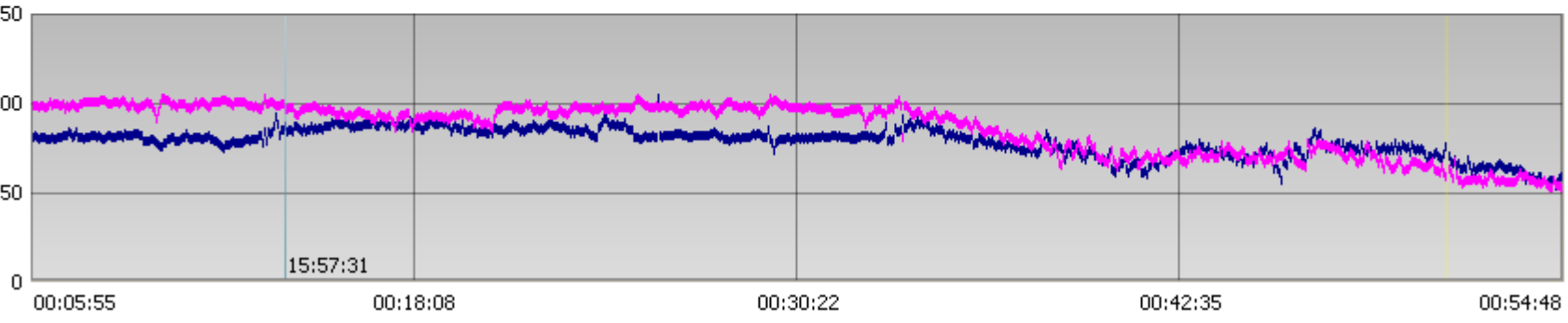
SVR dyn s cm^{-5}
1131



Sys/Dia mmH
189/96

MAP mmHg
130

PP mmHg (PPV
93 (30%)



SV ml (SVV)
88 (24%)

HR min^{-1} (HRV
101 (1%)