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REGIONÁLNÍ ANESTEZIE
PŘI ZÁVAŽNÝCH ONEMOCNĚNÍCH

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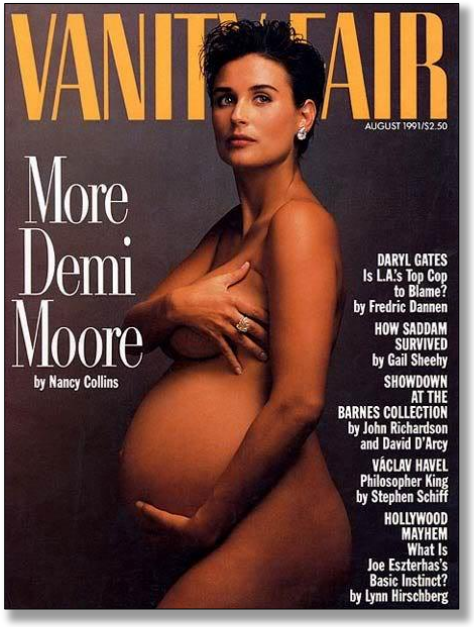
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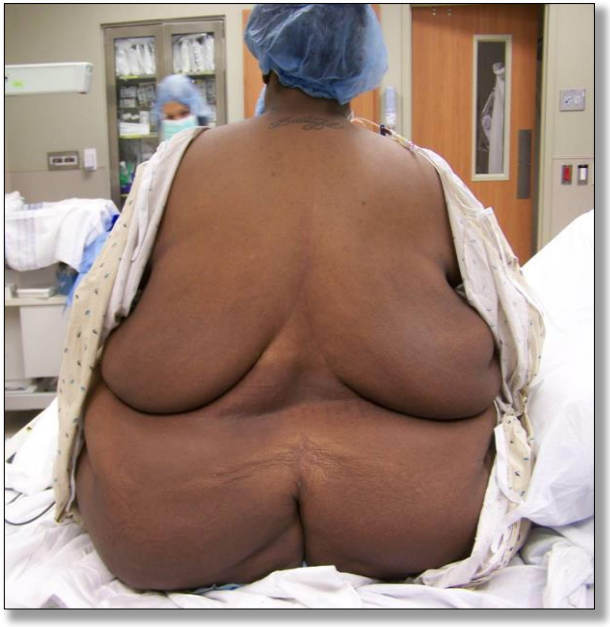


REGIONÁLNÍ ANESTEZIE
PŘI ZÁVAŽNÝCH ONEMOCNĚNÍCH

- Koagulační porucha
- Hemodynamická nestabilita

NE

- Neurologické onemocnění
- Abnormální placentace
- Infekce

?

ABNORMÁLNÍ PLACENTACE

Caesarean section for placenta praevia

Table 2 The grade of the most senior anaesthetist present and type of anaesthesia used for emergency or elective Caesarean section for placental praevia. GA= general anaesthesia. †Includes one failed spinal anaesthesia converted to GA before delivery. ‡Includes two spinal anaesthesias for Caesarean section with GA for hysterectomy, one failed epidural converted to spinal anaesthesia preoperatively and two spinal anaesthesias for Caesarean section and subsequent hysterectomy. ‡‡Includes one Caesarean section proceeding to hysterectomy. ††Includes one combined spinal–epidural anaesthetic. **Includes one combined spinal–epidural anaesthetic where the spinal component failed to spread and the epidural was topped up preoperatively

	Emergency Caesarean section			Elective Caesarean section			Total
	GA	Spinal	Epidural	GA	Spinal	Epidural	
Consultant	12	20	6	13‡‡	53‡	13**	117
Associate specialist	4†	2	0	2	10		18
Senior registrar	4	1		2	7		14
Staff anaesthetist	3	5	1	0	2	0	11
Registrar	55	19††	5	10	20	9	118
SHO/clinical assistant	35†	11††	2**	5	11	8	72
Total	113	58	14	32	103	30	350

Parekh N et al. Br J Anaesth. 2000 Jun;84(6):725-30

ABNORMÁLNÍ PLACENTACE

However, this retrospective analysis of anaesthesia for placenta praevia finds no evidence to support the much quoted opinion that GA is mandatory in such cases. Indeed, to the contrary, this study adds to the growing literature[11 12 22 23] suggesting that, when RA is used, not only is there no reduction in maternal safety in relation to the management of blood loss but there may be a reduced blood loss and reduced need for postoperative blood transfusion.

Arcario T et al. Anesthesiology 1988; 69 (Suppl 3A):A659
McShane PM et al. Obstet Gynecol 1985; 65: 176–82
Chestnut DH et al. Anesthesiology 1989; 70: 607–10

Parekh N et al. Br J Anaesth. 2000 Jun;84(6):725-30

ABNORMÁLNÍ PLACENTACE

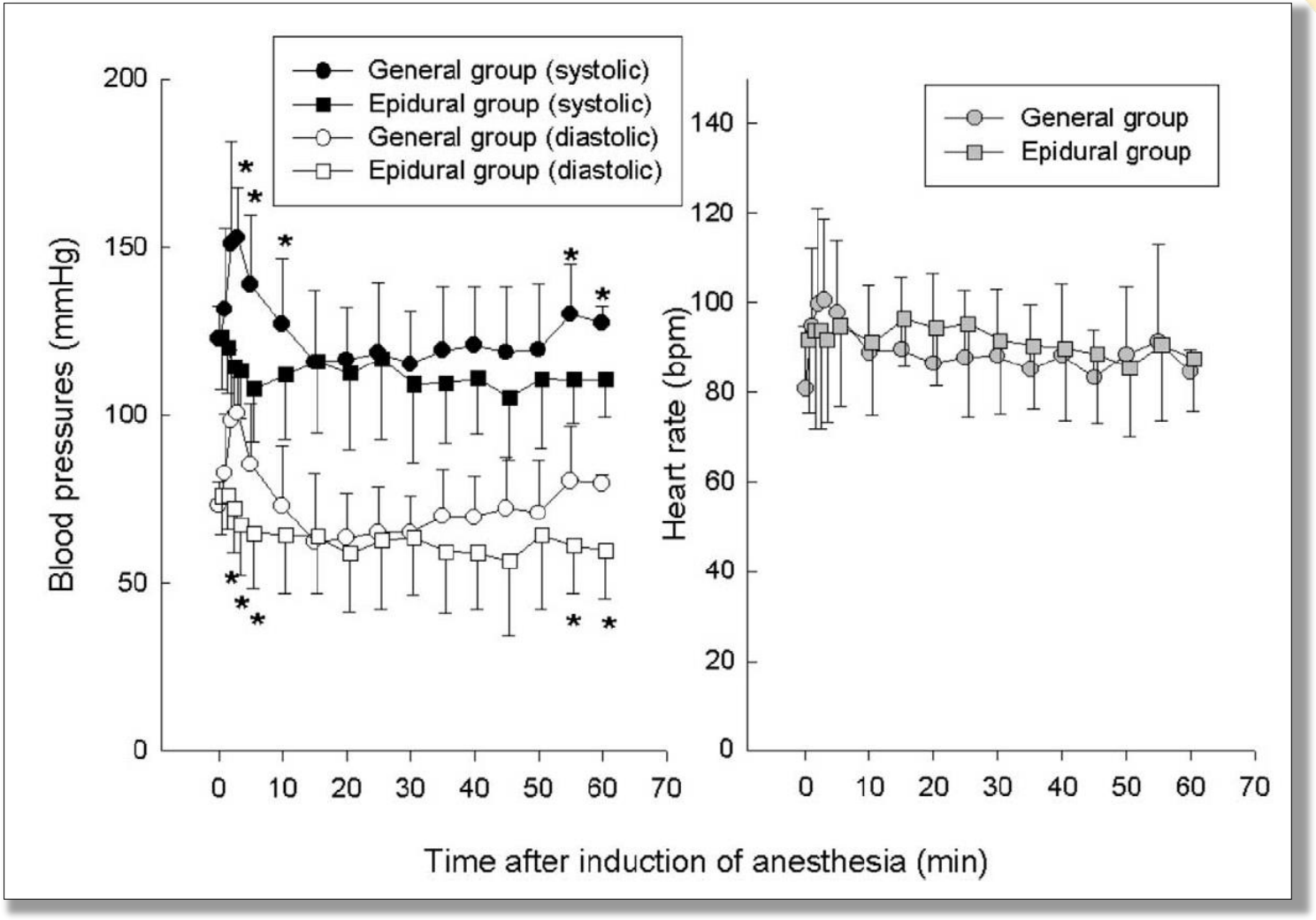
Table 2. Anesthetic and operative management

	General group ($n = 12$)	Epidural group ($n = 13$)
Operating time (min)	60.4 ± 10.4	70.2 ± 15.2
Incision-delivery time (min)	5.8 ± 1.5	6.0 ± 1.7
Estimated blood loss (mL)		
All patients	1623 ± 775	1418 ± 996
Posterior placenta	1459 ± 672	1269 ± 760
Anterior placenta	2094 ± 872	1000
Lateral placenta	880	2375 ± 2114
Collected amniotic fluid (mL)	329 ± 108	292 ± 103
Administered fluid (mL)	2042 ± 485	2218 ± 787
Postoperative transfusion (unit)	1.08 ± 1.6	0.38 ± 0.9*
Urine output (mL)	118 ± 73.6	153 ± 127
Ephedrine (mg)	0	5.4 ± 8.8*
Apgar score (1 min)	8 (4–9)	8 (7–9)
Apgar score (5 min)	10 (6–10)	9 (9–10)

Values are mean ± SD or median (range).
* $P < 0.05$ compared to the general group.

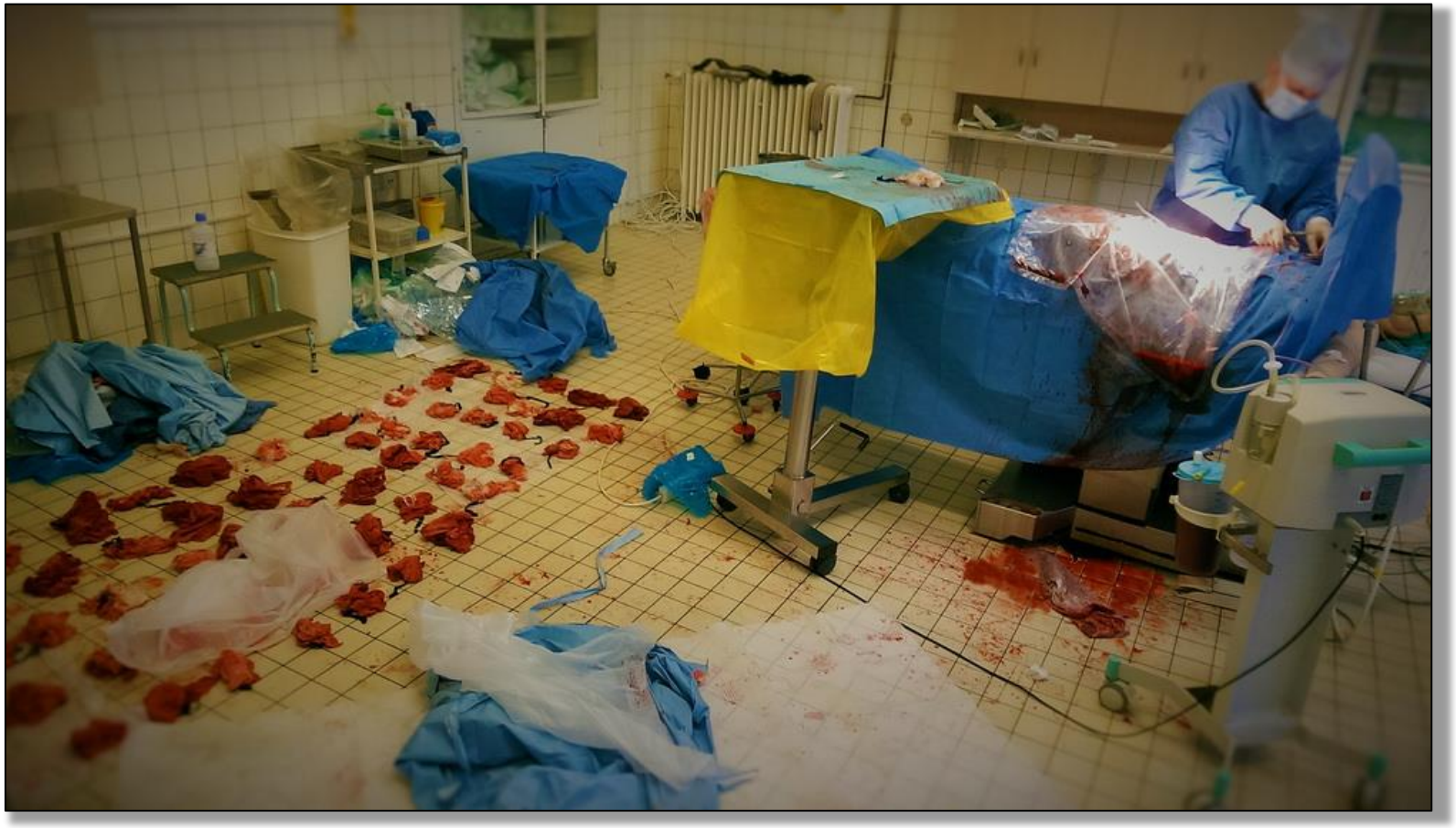
Hong JY et al. Int J Obstet Anesth. 2003 Jan;12(1):12-6

ABNORMÁLNÍ PLACENTACE



Hong JY et al. Int J Obstet Anesth. 2003 Jan;12(1):12-6

ABNORMÁLNÍ PLACENTACE



POROD & HOREČKA

Probably the best-supported and most plausible theory is based on the systemic effect of thermogenic inflammatory mediators, such as interleukin-6, released from the placental-myometrial interface into the maternal circulation.

Dashe JS et al. Obstet Gynecol 1999; 93:341– 4
Goetzl L et al. Am J Obstet Gynecol 2002;187:834 – 8
Riley LE et al. Obstet Gynecol 2011; 117:588 –95
Goetzl Let al. Am J Obstet Gynecol 2006; 195:1031–7

Frolich et al. Anesthesiology 2012; 117:302– 8

NEUROAXIÁLNÍ BLOKÁDA & HOREČKA

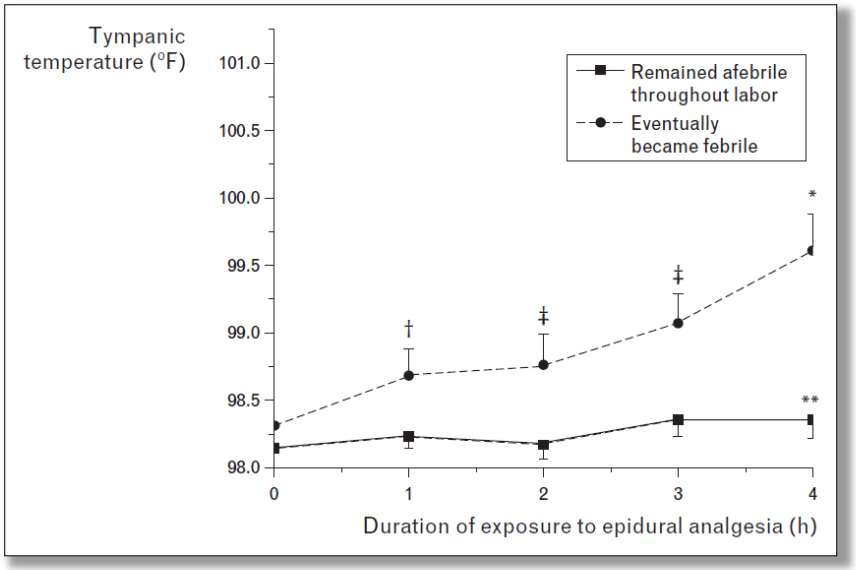


FIGURE 1. Maternal tympanic temperature in the 4 h immediately following initiation of epidural analgesia, stratified by ultimate intrapartum fever status. Temperature points that are significantly different between the two curves are marked (repeated measures analysis †, $P<0.05$; ‡, $P\leq 0.01$; *, $P<0.0001$.). Repeated measures analysis was used to evaluate changes in temperature over time in the afebrile group. No significant increase was observed (**, $P=0.26$). Adapted with permission from [16].

Goetzl L. Curr Opin Anesthesiol 2012, 25:292–299

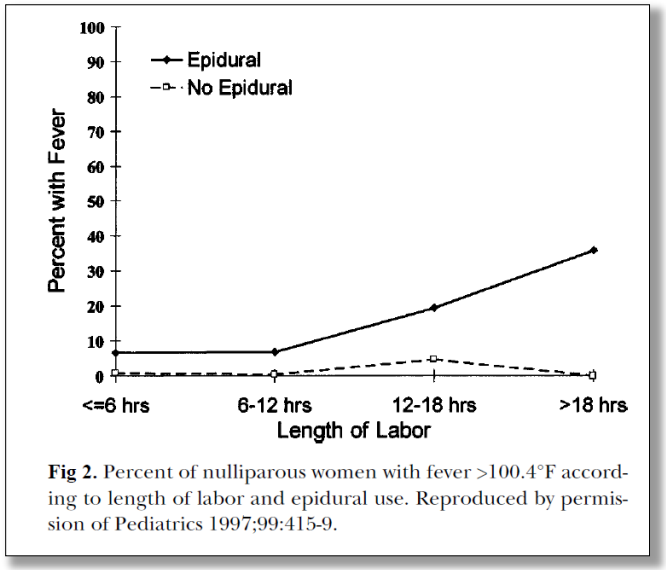
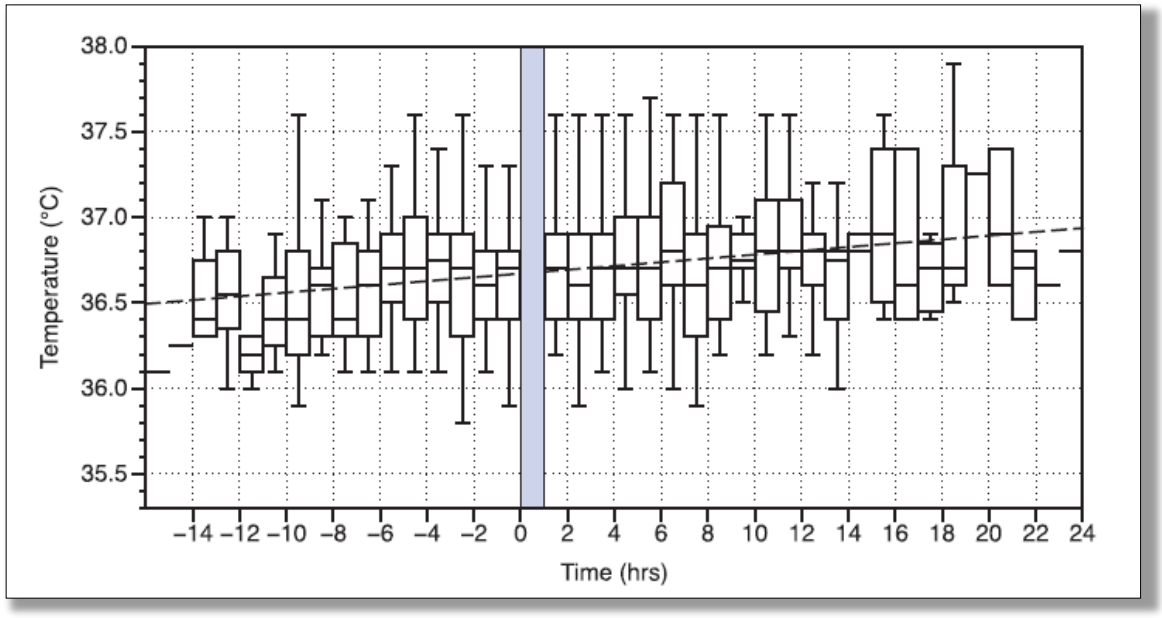


Fig 2. Percent of nulliparous women with fever >100.4°F according to length of labor and epidural use. Reproduced by permission of Pediatrics 1997;99:415-9.

Lieberman E. Am J Obstet Gynecol 2002;186:S31-68

NEUROAXIÁLNÍ BLOKÁDA & HOREČKA



Frolich et al. Anesthesiology 2012; 117:302– 8

Summarizing the key findings of our study, we conclude that induced labor is associated with a small temperature increase. We found an overall significant linear trend of temperature over time after correcting for heterogeneity among patients. Patients with higher BMI and longer duration from ROM to delivery are more likely to increase their body temperature during labor. In our study of women scheduled for induction of labor, epidural analgesia had no effect on maternal temperature.

NEUROAXIÁLNÍ BLOKÁDA & HOREČKA

[83] The mechanism of maternal hyperthermia following induction of epidural analgesia remains unclear. Possible explanations include cessation of hyperventilation that follows pain relief, increased incidence of shivering and decreased sweating. [88, 89]

Kuczkowski. Southern African Journal of Anaesthesia & Analgesia - November 2002

NEUROAXIÁLNÍ BLOKÁDA & HOREČKA

FEVER: THE SAFETY OF REGIONAL ANESTHESIA

It is a common concern among anesthesiologists that administration of regional anesthesia to a febrile parturient may spread the infectious agent to the central nervous system and lead to neurological sequelae. However, to date no epidemiologic study has documented a causal relationship between dural puncture in the presence of bacteremia and the subsequent development of complications such as meningitis and epidural abscess. [65]

Kuczkowski. Southern African Journal of Anaesthesia & Analgesia - November 2002

NEUROAXIÁLNÍ BLOKÁDA & HOREČKA

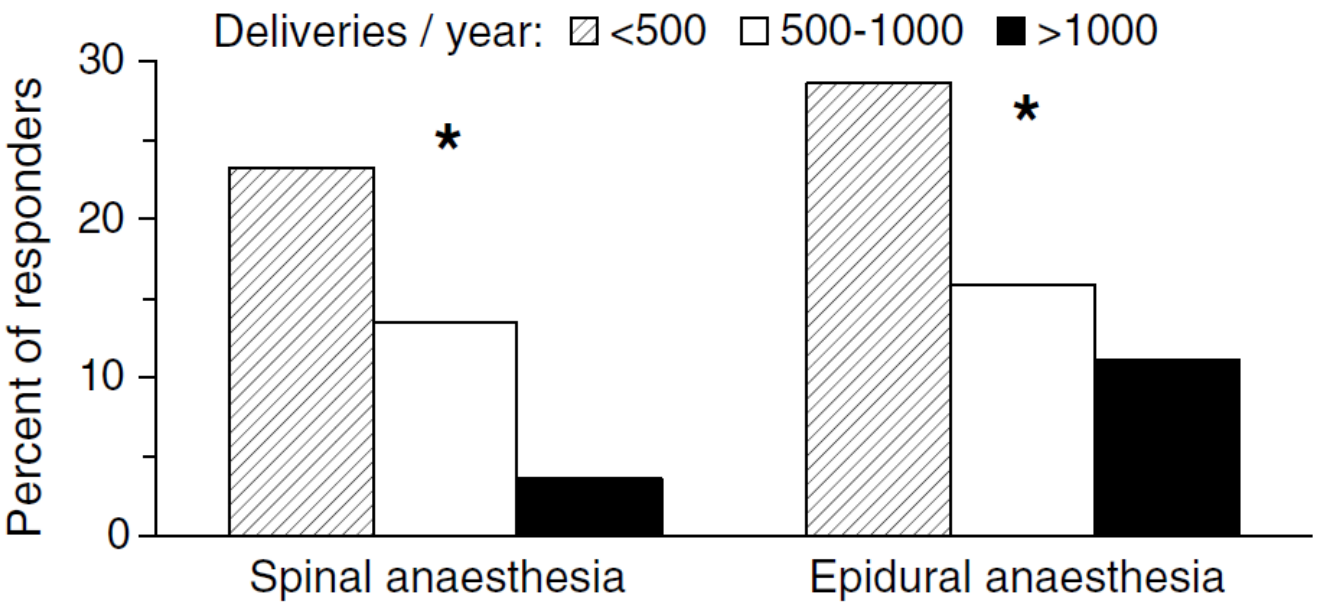


Fig. 2 Percent of responders who considered chorioamnionitis an absolute contraindication to spinal or epidural block. $^{*}P < 0.05$.

Stamer et al. International Journal of Obstetric Anesthesia (2007) 16, 328–335

NEUROAXIÁLNÍ BLOKÁDA & HOREČKA

Reg Anesth. 1996 Sep-Oct;21(5):436-41.

Safety of spinal and epidural anesthesia in parturients with chorioamnionitis.

Goodman EJ, DeHorta E, Taguiam JM.

Department of Anesthesiology, Case Western Reserve University, School of Medicine, Cleveland, Ohio, USA.

Abstract

BACKGROUND AND OBJECTIVES: The safety of spinal and epidural anesthesia in patients with chorioamnionitis was explored.

METHODS: A retrospective study was made of the charts of 517 parturients who had received epidural anesthesia and 14 who had received spinal anesthesia before delivery and whose placentas had subsequently been found to be positive for chorioamnionitis.

RESULTS: Of the 146 blood culture results that were reported, 13 were positive. Of these 13 blood cultures, 5 had been drawn within 6 hours after placement of the epidural block, and four of the five bacteremic patients did not receive antibiotics until after the regional anesthetic was administered. One quarter (11/45) of the patients who were febrile and three quarters (174/229) of those who had leukocytosis before their block received no antibiotics before the block was placed. After the epidural block was performed, the catheter was left in place for over 24 hours in 18% (46/260) of the women who spiked a fever and in 14% (18/130) of those who exhibited leukocytosis during that period. There was no report of an epidural or spinal abscess or of meningitis in any of the women in the study.

CONCLUSION: Conduction anesthesia may be safe in parturients with chorioamnionitis without prior antibiotic therapy.

PMID: 8896004 [PubMed - indexed for MEDLINE]

NEUROAXIÁLNÍ BLOKÁDA & HOREČKA

A meta-analysis of 267 articles conducted by Ruppen et al[1] concluded that there was no evidence that regional anesthesia should be contraindicated in parturients with chorioamnionitis. There were no studies that could delineate a causal relationship between regional anesthesia and complications such as meningitis and epidural hematoma formation in bacteremic patients.[1]

Osborne et al. AANA J. 2008 Jun;76(3):221-6

NEUROAXIÁLNÍ BLOKÁDA & HOREČKA

Study	No.	Study Design	Findings	Comments/Conclusions
Bader et al[4] (1992)	10,047 cases	Retrospective review	319 patients with chorioamnionitis; no complications associated with anesthesia	Risks of neuraxial anesthesia unsubstantiated, especially considering risk of general anesthesia
Carp and Bailey[12] (1992)	40 rats	Animal study	Meningitis resulted after cisternal puncture in rats intentionally infected with *Escherichia coli*.	Gentamicin-treated group had no cases of meningitis; cisternal puncture much more invasive than epidural administration in humans
Beilin et al[10] (1996)	177	Survey	Practice patterns of US anesthesiologists	Majority (74%-89%) would place CLE if temperature <101°F and antibiotic therapy initiated
Goodman et al[9] (1996)	517 epidural and 14 spinal anesthetics	Retrospective review	No reports of anesthetic infection, epidural abscess, or meningitis	Lumbar puncture in the face of infection does not seem to increase risk of meningitis.
Wang et al[11] (1999)	17,372	Survey	9 spinal epidural abscesses, none in obstetric cases; overall incidence of complications: 1:1,930 in SAB; 1:5,661 in CLE	Spinal epidural abscess associated with long duration of anesthesia, immunosuppression, and use of low-molecular-weight heparin
Bajwa et al[13] (2002)	1 case	Case report	Diskitis and osteomyelitis after cesarean delivery with spinal anesthesia	No cases of infectious diskitis associated with pregnancy and spinal anesthesia are reported.
Phillips et al[6] (2002)	5,000 in 5 y	Retrospective review	No case of epidural abscess	Low incidence of epidural abscess makes it difficult to show positive effect of prophylactic antibiotics.
Kuczkowski and Reisner[7] (2003)	Review	Review article	Chorioamnionitis incidence, 0.5%-10%	The presence of infection and fever in labor does not always contraindicate the administration of regional anesthesia.
Wlody[2] (2003)	Review	Review article of complications of regional obstetric anesthesia	Anesthetic mortality 17 times greater in cesarean deliveries performed under general anesthesia compared with regional anesthesia	Stresses the importance of risk-benefit analysis and use of sterile technique
Moen et al[3] (2004)	1990-1999: 1,260,000 SAB cases; 450,000 CLE cases	Retrospective review	Complications of obstetric CLE, 1:20,000-30,000	Greater complication rate of CLE compared with SAB; obstetric patients had a lower risk
Schroeder et al[5] (2004)	1 case	Case report	Spinal epidural abscess in postpartum patient after epidural anesthesia	Surgical decompression resulted in no neurological deficits.
Baer[8] (2006)	Review	Review of postdural puncture meningitis	Postdural puncture meningitis most often results from exogenous inoculation, not hematological spread.	Antibiotic prophylaxis is advised and postoperative follow-up is necessary; sterile precautions emphasized.
Ruppen et al[1] (2006)	27 studies	Meta-analysis of epidural complications	Hematoma formation, 1:168,000; deep epidural infection, 1:145,000; persistent neurological injury, 1: 240,000; transient neurological injury, 1:6,700	Provides some best-estimate information for complications; risk-benefit analysis should guide decision to proceed with regional anesthesia

Table. Literature of Chorioamnionitis and Neuraxial Anesthesia

SAB indicates subarachnoid block; CLE, continuous lumbar epidural.

NEUROAXIÁLNÍ BLOKÁDA & HOREČKA

TABLE II Maternal outcome, oxytocin augmentation, and maternal fever during labour

	Group I (amnionitis, no LEA)	*Group II (amnionitis, + LEA)*	*Group III (no amnionitis, + LEA)*	*P*
Delivery mode (%)				
Cesarean section	14.5	16.0	16.4	0.938
Vaginal delivery	85.5	84.0	83.6	0.938
Vacuum/forceps	8.1	14.0	15.9	0.298
Oxytocin (%)	9.7	24.0	39.3*	0.000
Fever during labour (%)	100.0	100.0	1.0 *#	0.000

*P <0.05 compared to Group I; #P <0.05 compared to Group II, fever during labour=fever plus one or more criteria used for the diagnosis of amnionitis (see text). For other abbreviations see Table I.

TABLE III Neonatal outcome

	Group I (amnionitis, no LEA)	*Group II (amnionitis, + LEA)*	*Group III (no amnionitis, + LEA)*	*P*
One-minute Apgar <7 (%)	35.5	20.0	17.4*	0.010
Five-minute Apgar <9 (%)	33.9	30.0	27.4	0.607
Birth weight (kg)	2.57 ± 1.19	2.46 ± 0.70	2.24 ± 0.51* #	0.003
LBW (<2.5 kg) (%)	32.3	52.0	72.6*#	0.000
Overall NSER (%)	32.3	52.0	55.0*	0.007
LBW neonates with NSER (%)	100.0	100.0	76.0*#	0.001

LBW=low birth weight; NSER=neonatal sepsis evaluation rate; *P <0.05 compared to Group I; #P <0.05 compared to Group II; for other abbreviations see Table I.

Vallejo et al. CAN J ANESTH 2001 / 48: 11 / pp 1122-1126

NEUROAXIÁLNÍ BLOKÁDA & HOREČKA

Table 1. Inhibitory Properties of Local Anesthetics for Various Bacteria

	C. albicans	*E. coli*	*E. faecalis*	*H. influenzae*	MRSA	*P. aeruginosa*	*S. aureus*	*S. epidermidis*	*S. pneumoniae*	Other pathogens	Reference
Bupivacaine	+	+	+		+	+	+	+	+	*Bacillus* spp., *B. cereus*, *Candida* spp., *Corynebacterium* spp., MSSA, *Micrococcus* spp., *E. faecalis*, *S. pyogenes*	8–18, 20, 30
Lidocaine	+	+	+	+	+	+	+	+	+	*Bacillus* spp., *B. subtilis*, *B. catarrhalis*, *B. cepacia*, *Candida* spp., *Corynebacterium* spp., *Enterobacter* spp., *K. pneumoniae*, *Micrococcus* spp., *M. osloensis*, *S. marcescens*	11, 13, 16, 22–26, 28, 34, 37, 38
Lignocaine		+		+		+	+	+	+	*A. niger*, *B. subtilis*, *M. catarrhalis*	21, 27, 3
Ropivacaine	+	+			+	+	+	+		*K. pneumoniae*	17–19, 29,
Cocaine				+		+	+		+	*B. catarrhalis*, *Enterobacter* spp., *K. pneumoniae*	34, 35
Tetracaine						+	+				35, 36
Amethocaine										*A. niger*, *B. subtilis*	21
Amylocaine										*A. niger*, *B. subtilis*	21
Benzydamine	+									*Candida* spp.	20
Cincochaine										*A. niger*, *B. subtilis*	21
Levobupivacaine			+				+	+			10
Mepivacaine					+						13
Oxybuprocaine		+		+		+			+		31
Prilocaine		+				+	+				16
Procaine										*A. niger*, *B. subtilis*	21
Proparacaine						+	+				35

MRSA = methicillin resistant *Staphylococcus aureus*; MSSA = methicillin susceptible *S. aureus*.

Johnson et al. Surg Infect 2008; 9(2):

NEUROAXIÁLNÍ BLOKÁDA & HOREČKA

Infectious complications after a neuraxial anesthetic are more closely related to failure of pure aseptic technique rather than a hematological spread of an infective agent.[2,4] Infections occur due to contaminated solutions and contaminated catheters.[6] In addition, skin colonization of bacteria and respiratory droplets from the anesthesia provider are other sources of potential infection.[6]

Osborne et al. AANA J. 2008 Jun;76(3):221-6

NEUROAXIÁLNÍ BLOKÁDA & HOREČKA

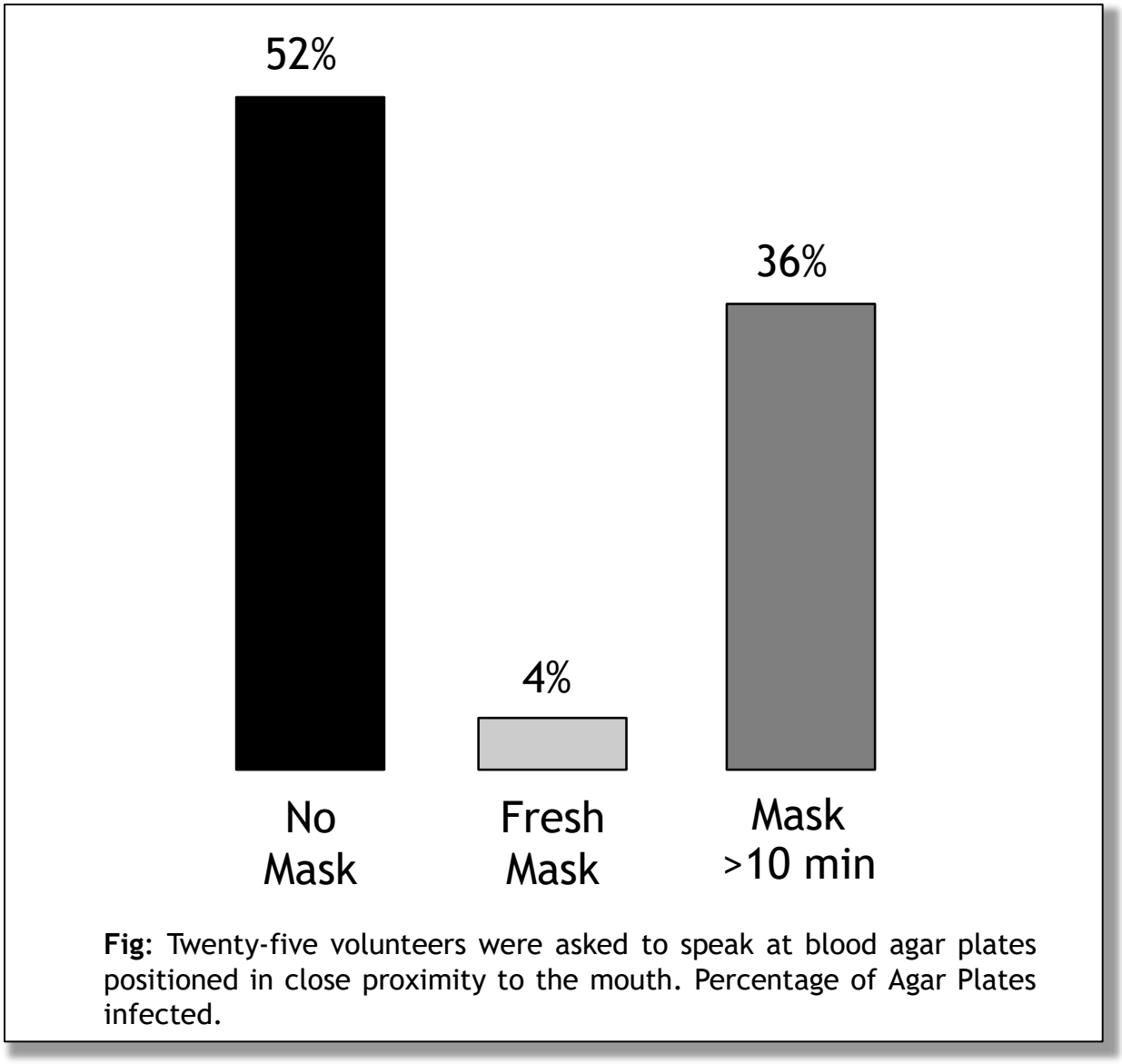


Fig: Twenty-five volunteers were asked to speak at blood agar plates positioned in close proximity to the mouth. Percentage of Agar Plates infected.

Philips BJ et al. Br J Anaesth 1992 Oct;69(4):407-8

NEUROAXIÁLNÍ BLOKÁDA & PREEKLAMPSIE

Table 1
Numbers and rates per 100,000 maternities of maternal deaths reported to the Enquiry by cause; United Kingdom: 1985-2005.

Cause of death	1985-87	1988-90	1991-93	1994-96	1997-99	2000-02	2003-05	1985-87	1988-90	1991-93	1994-96	1997-99	2000-02	2003-05
	Numbers							*Rates per 100,000 maternities*						
***Direct* deaths**														
Thrombosis and thromboembolism	32	33	35	48	35	30	**41**	*1.41*	*1.40*	*1.51*	*2.18*	*1.65*	*1.50*	*1.94*
Pre-eclampsia and eclampsia*	27	27	20	20	16	14	**18**	*1.19*	*1.14*	*0.86*	*0.91*	*0.75*	*0.70*	*0.85*
Haemorrhage*	10	22	15	12	7	17	**14**	*0.44*	*0.93*	*0.65*	*0.55*	*0.33*	*0.85*	*0.66*
Amniotic fluid embolism	9	11	10	17	8	5	**17**	*0.40*	*0.47*	*0.43*	*0.77*	*0.38*	*0.25*	*0.80*
Early pregnancy deaths	16	24	17	15	17	15	**14**	*0.71*	*1.02*	*0.73*	*0.68*	*0.80*	*0.75*	*0.66*
Ectopic	11	15	9	12	13	11	**10**	*0.48*	*0.64*	*0.39*	*0.55*	*0.61*	*0.55*	*0.47*
Spontaneous miscarriage	4	6	3	2	2	1	**1**	*0.18*	*0.25*	*0.13*	*0.09*	*0.09*	*0.05*	*0.05*
Legal termination	1	3	5	1	2	3	**2**	*0.04*	*0.13*	*0.22*	*0.05*	*0.09*	*0.15*	*0.09*
Anaesthetic	6	4	8	1	3	6	**6**	*0.26*	*0.17*	*0.35*	*0.05*	*0.14*	*0.30*	*0.28*
Cardiac	23	18	37	39	35	44	**48**	*1.01*	*0.76*	*1.60*	*1.77*	*1.65*	*2.20*	*2.27*

Saving Mothers' Lives. Confidential Enquiry into Maternal and Child Health, UK. 2003-2005

NEUROAXIÁLNÍ BLOKÁDA & PREEKLAMPSIE

PROČ **NE**:

- preeklampsie sama je komplikací, proč přidávat další rizika spojená s RA.
- pokles TK bývá náhlejší a vzhledem k předchozí hypertenzi i významnější než u „zdravé" rodičky.
- výrazný pokles TK může vést k významné uteroplacentární insuficienci, navíc již u kompromitovaného těhotenství = RA není vhodná k tlumení porodních bolestí.
- zavedení RA je navíc často komplikováno trombocytopenií (a koagulopatií)

Lindheimer MD, Katz AI. Hypertension pregnancy. N Engl J Med 1985; 313:675-680
Andrew M et al. Pre-eclampsia and eclampsia: Anesthetic management. Annu Refresher Course Lectures 1991; 421:12-16

NEUROAXIÁLNÍ BLOKÁDA & PREEKLAMPSIE

PROČ **ANO**:

- pokles tlaku není nižší než u „zdravých rodiček".
- není rozdíl v počtu nepostupujících porodů, císařských řezů či distresu plodu proti skupině bez RA
- naopak pro event. císařský řez je k dispozici již zavedený epidurální katetr
- u hypertenzních rodiček RA blok sympatiku lépe stabilizuje výkyvy TK a tím působí proti komplikacím způsobených hypertenzí (vzestup ICP s CMP, arytmie, rozvoj plicního edému, pokles uteroplacentárního průtoku …).

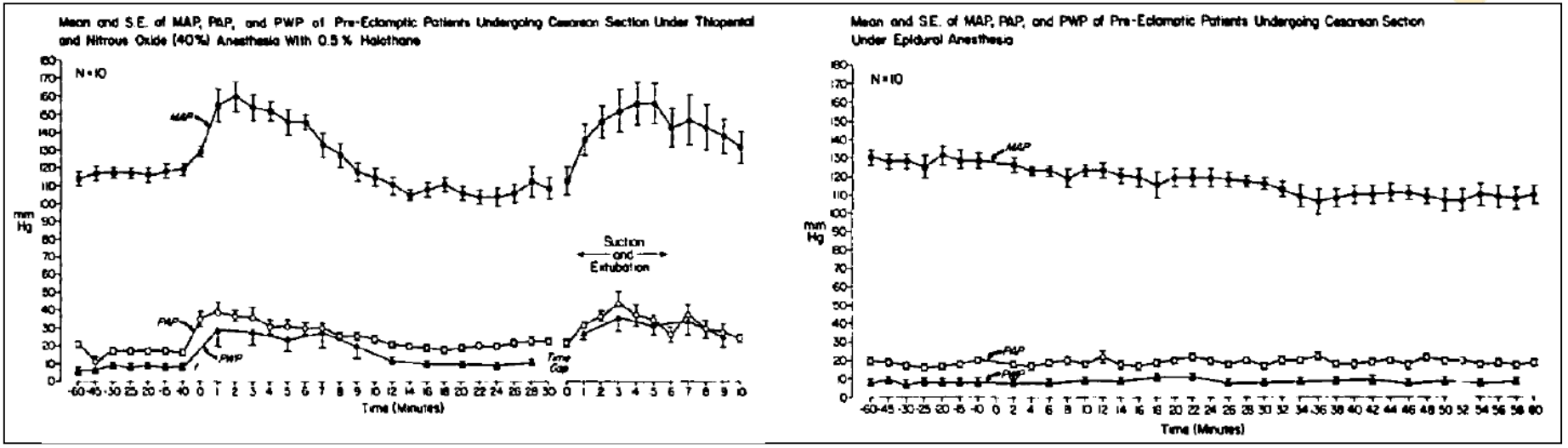
Hogg et al. Am J Obstet Gynecol 1999;181:1096-1101
Lucas et al. Am J Obstet Gynecol 2001;185: 970-5
Patel et al. J Obstet Gynaecol Res 2005 Aug; 31, 4:291-5

NEUROAXIÁLNÍ BLOKÁDA & PREEKLAMPSIE

Table 3.1 Number of deaths by cause due to eclampsia and pre-eclampsia; United Kingdom 1988–2002

	Triennium				
Cause of death	**1988–90**	**1991–93**	**1994–96**	**1997–99**	**2000–02**
Cerebral:					
Intracranial haemorrhage	10	5	3	7	9
Subarachnoid	2	0	1	0	0
Infarct	2	0	0	0	0
Oedema	0	0	3	0	0
Subtotal	14	5	7	7	9
Pulmonary:					
ARDS	9	8	6	2	1
Oedema	1	3	2	0	0
Subtotal	10	11	8	2	1
Hepatic:					
Rupture	0	0	2	2	0
Failure/necrosis	1	0	1	0	0
Other	2	4	2	5	4
Subtotal	3	4	5	7	4
Overall total	27	20	20	16	14

NEUROAXIÁLNÍ BLOKÁDA & PREEKLAMPSIE



Hodgkinson et al. Can J Anesth 1980 27: 389-394.

Table 3.1 Number of deaths by cause due to eclampsia and pre-eclampsia; United Kingdom 1988–2002

	Triennium				
Cause of death	**1988–90**	**1991–93**	**1994–96**	**1997–99**	**2000–02**
Cerebral:					
Intracranial haemorrhage	10	5	3	7	9
Subarachnoid	2	0	1	0	0
Infarct	2	0	0	0	0
Oedema	0	0	3	0	0
Subtotal	14	5	7	7	9

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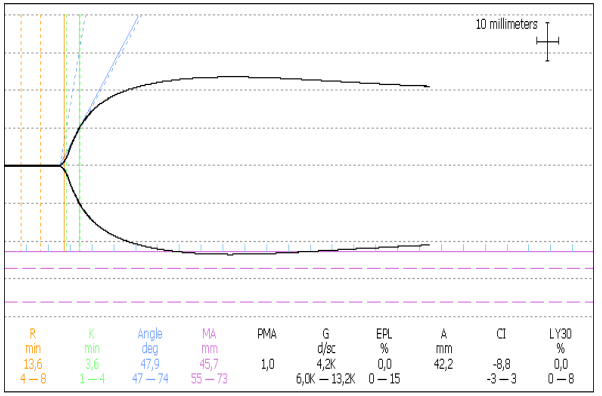
	Normal Pregnant (n = 52)	Mild Preeclampsia (n = 140)	Severe Preeclampsia (n = 114)
Platelet count			
≥150,000/mm^3	47 (90)	126 (90)	64 (56)
100,000/mm^3–149,000/mm^3	4 (8)	10 (7)	16 (14)
<100,000/mm^3	1 (2)	4 (3)	34 (30)
MA < 54 mm	0	0	10 (9)
Abnormal coagulation profile*	NA	0/4	5/34 (12)

Values are n (%).

MA = maximum amplitude, mm (clot strength); NA = not available.

* Abnormal coagulation profile = at least one abnormal value in the coagu-



	Normal Pregnancy (n = 52)	Mild Preeclampsia (n = 140)	Severe Preeclampsia with PC ≥ 100,000/mm^3 (n = 80)	Severe Preeclampsia with PC < 100,000/mm^3 (n = 34)
r (mm)	26.5 ± 7.5 (13–42)	29.5 ± 7.2 (13–42)	29.6 ± 7.6 (15–42)	37.4 ± 9.9 (19–63)*
K (mm)	10.2 ± 3.5 (5–20)	10.8 ± 3.7 (5–25)	11.6 ± 4.2 (6–22)	20.9 ± 7.4 (7–37)*
MA (mm)	66.5 ± 7.1 (54–80)	69.3 ± 7.3 (54–86)†	66.1 ± 6.9 (54–80)	52.1 ± 10.6 (34–78)*
α angle (°)	43.1 ± 9.1 (27–64)	41.6 ± 9.8 (26–72)	39.9 ± 9.4 (26–64)	23.6 ± 9.1 (13–47)*

Values are mean ± SD (range).

PC = platelet count; r = reaction time; K = clot formation time; ∝ angle = clot formation rate (°); MA = maximum amplitude (clot strength) (mm).

* $P < 0.001$ *versus* normal pregnant, mild preeclamptic and severe preeclamptic women with PCs ≥ 100,000/mm^3.

† $P < 0.05$ *versus* normal pregnant and all severe preeclamptic women.

Sharma et al. Anesthesiology 1999;90:385-90

NEUROAXIÁLNÍ BLOKÁDA & PREEKLAMPSIE

Table 3. Hematological and Clotting Variables

	Controls ($n = 93$)	Mild PET ($n = 23$)	Severe PET ($n = 27$)
Hct (%)	35 (4)	34 (4)	33 (5)
Platelet count ($\times 1000/mm^3$)	257 (89)	230 (83)	177 (81)*
PT (s)	13.2 (0.9)	13.2 (0.9)	13.3 (1.2)
APTT (s)	28.7 (3.0)	30.2 (4.3)	31.3 (3.4)†
Fibrinogen (g/L)	4.4 (1.0)	4.1 (1.0)	4.0 (0.9)
VWF activity (IU/L)	312 (97)	335 (119)	360 (93)
VWF antigen (IU/L)	349 (105)	371 (122)	409 (110)‡

Hct = hematocrit; PT = prothrombin time; APTT = activated partial thromboplastin time; VWF = von Willebrand Factor; PET= preeclamptic.

Data are mean (±SD).

* $P < 0.001$ (control versus severe PET).

† $P < 0.01$ (control versus severe PET).

‡ $P < 0.05$ (control versus severe PET).

Davies et al. Anesth Analg 2007 Feb;104(2):416-20

NEUROAXIÁLNÍ BLOKÁDA & PREEKLAMPSIE

Gogarten W. Preeclampsia and anaesthesia. Curr Opin Anaesthesiol. 2009 Jun;22(3):347-51.

Preeklampsie je spojena s vasokonstrikcí, snížením CO, a snížením plnících tlaků. Neuroaxiální anestezie proto pomáhá hemodynamiku stabilizovat.

Ve srovnání se zdravými rodičkami je výskyt hypotenze a potřeba vasopresorů nižší, CO je beze změny.

V přítomnosti trombocytopénie je vzhledem k riziku spinálního hematomu výhodnější užití SAB.

SUMMARY:
Intrakraniální krvácení je hlavní příčinou mortality u žen s preeklampsií. Každý vzestup TK >160 mmHg při úvodu do celkové anestezie by měl léčen. Tradičnímu bleskovému úvodu je proto vhodné se vyhnout – neuroaxiální technika je tak metodou volby.

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ORIGINAL ARTICLE

Haemodynamic effects of oxytocin in women with severe preeclampsia

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Conclusions: The haemodynamic effects of oxytocin in women with severe preeclampsia may be less predictable compared to findings in healthy pregnant women, suggesting that oxytocin should be given with caution in women with severe preeclampsia.

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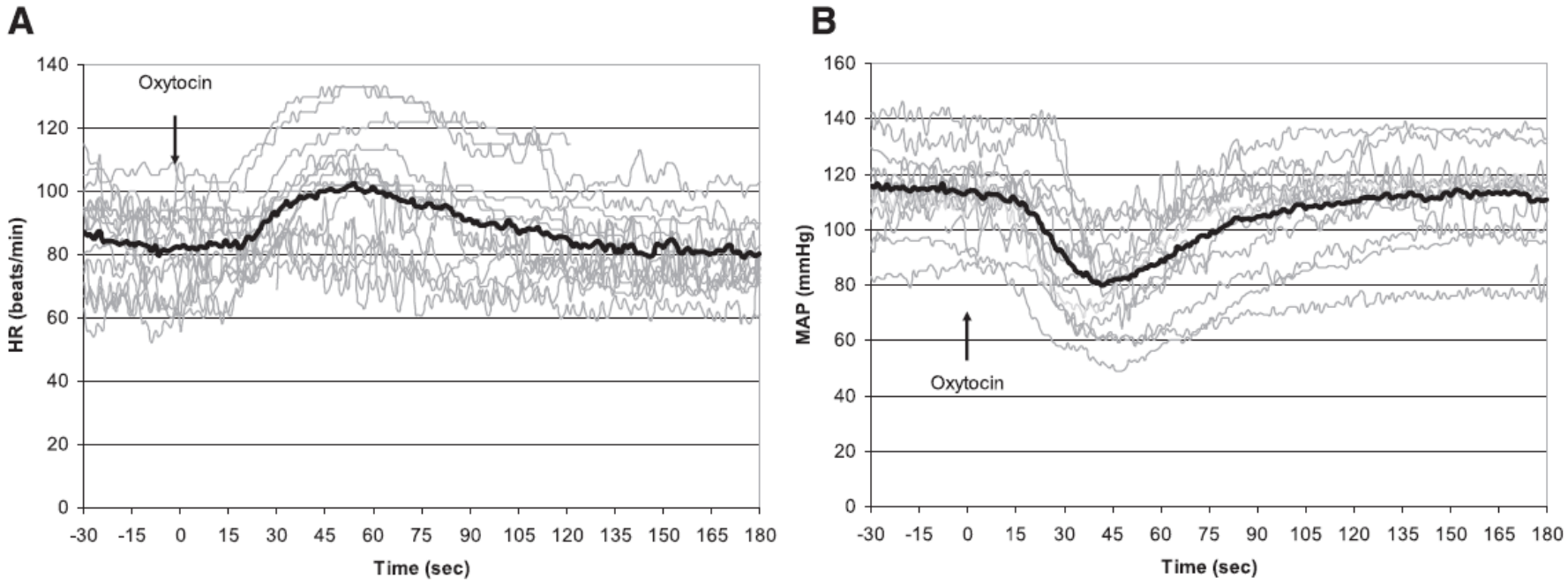
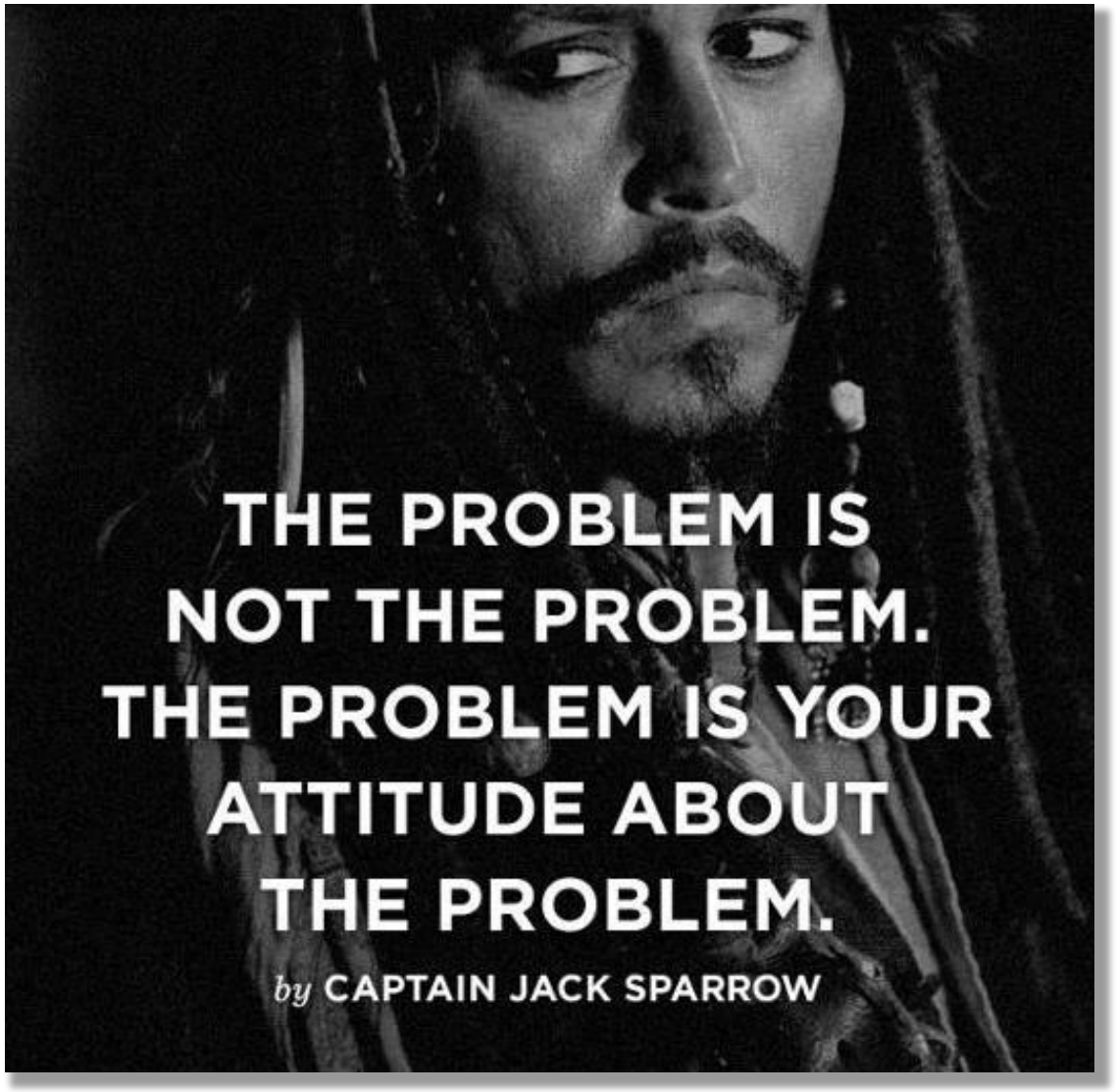


Fig. 3. Ensemble of hemodynamic changes after the administration of oxytocin. (*A*) Heart rate (HR). At peak effect, HR increased from 84.7 (12.6) to 101.5 (15.9)* beats/min. (*B*) Mean arterial pressure (MAP). At peak effect, MAP decreased from 114.3 (15.0) to 80.6 (15.3)* mmHg. (*C*) Stroke volume (SV). At peak effect, SV increased from 82.9 (15.9) to 89.1 (17.3) (not significant). (*D*) Cardiac output (CO). At peak effect, CO increased from 7.0 (1.5) to 9.1 (2.3)* l/min. (*E*) Systemic vascular resistance (SVR). At peak effect, SVR decreased from 1,295 (252) to 718 (282)* dyn · s · cm^{-5}. * Value significantly different from baseline and postdelivery; $P < 0.01$.

Dyer RA et al. Anesthesiology 2008; 108:802–11



DĚKUJI ZA POZORNOST