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ORIGINAL ARTICLE

Haemodynamic changes during labour: continuous minimally invasive monitoring in 20 healthy parturients

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ABSTRACT
Background: There are few studies on maternal haemodynamic changes during labour. None have used continuous cardiac output monitoring during all labour stages. In this observational study, we monitored haemodynamic variables continuously during the entire course of labour in healthy parturients.
Methods: Continuous haemodynamic monitoring with the LiDCOplus technique was performed in 20 healthy parturients during spontaneous labour, vaginal delivery and for 15 minutes postpartum. Cardiac output, stroke volume, heart rate, systemic vascular resistance, and systolic arterial pressure were measured longitudinally at baseline (periods between/without contractions) and during contractions in early and late stage 1, stage 2, during delivery, and postpartum, and were analysed with marginal linear models.
Results: Twenty parturients were included. In early stage 1, baseline cardiac output was 6.3 L/min (95% CI 5.7 to 6.9). Baseline values were similar across both labour stages and postpartum for all haemodynamic variables. During stage 2 contractions, cardiac output decreased by 32%, stroke volume decreased by 44%, heart rate increased by 52%, systemic vascular resistance increased by 88%, and systolic arterial pressure increased by 36% compared to baseline. During stage 1 contractions, haemodynamic changes were less profound and less uniform than during stage 2.
Conclusion: Progression of labour had no major effect on haemodynamic baseline values. Haemodynamic stress during contractions was substantial in both labour stages, yet most pronounced during the second stage of labour. The absence of an increase in stroke volume and cardiac output postpartum questions the common belief in an immediate rise in cardiac output after delivery due to autotransfusion from the contracted uterus.

Keywords: Haemodynamic changes; Continuous monitoring; Cardiac output; Labour; Vaginal delivery

Introduction

Literature on haemodynamic changes during labour and vaginal delivery is sparse and inconsistent.[1,2] In particular, the haemodynamic impact of bearing down during stage 2 has been studied very little.[1,2] Haemodynamic monitoring during labour is challenging to perform, particularly in parturients with high levels of pain, stress and restlessness, and long duration of labour. Most studies were performed between the 1950s and 1970s.[3–9] Cardiac output (CO) measurement techniques used at that time suffered from disadvantages such as a high degree of invasiveness, a low number of measurements per time, limited reliability for the detection of rapid changes and high sensitivity to noise.[2] To our knowledge, none of the previous studies used continuous measurement during all stages of labour.

More recently, new technologies for CO measurement have become available. In this observational study, we used the LiDCO*plus* monitor (LiDCO Ltd., Cambridge, UK); currently this is the only minimally-invasive device that provides calibrated continuous data, and is valid in detecting rapid changes in haemodynamic trends.[10] The aim of this study was to examine haemodynamic changes during the entire course of active labour and vaginal delivery in healthy parturients.

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Methods

The Regional Committee for Medical and Health Research Ethics of Southern Norway (Oslo, Norway), and the Data Inspectorate at Oslo University Hospital approved the study protocol. This prospective study was conducted according to Good Clinical Trial Practice and the principles of the Declaration of Helsinki. The data are reported according to STROBE guidelines. All subjects gave written consent after oral and written information.

Healthy women at term with a singleton pregnancy, in active labour at Ullevål, Oslo University Hospital, Norway, between November 2011 and December 2012, were asked to participate. Inclusion criteria were spontaneous active labour with contractions and cervical dilatation >3 cm, age 18–40 years, height 155–180 cm, pre-pregnancy body mass index $\leq$35 kg/m^2. Exclusion criteria were pre-existing or gestational hypertension, preeclampsia, cardiovascular or cerebrovascular disease, psychiatric or somatic disease (other than well-treated mild asthma or thyroid hypofunction). Trial inclusion did not alter obstetric routines or therapeutic decisions. Heterogeneity with regards to mobility, oral intake, administration of intravenous fluids, pain, pain management with epidural analgesia, oxytocin augmentation of labour and method of delivery was accepted.

On arrival in hospital, each parturient was examined by a midwife with regards to stage of labour and cervical dilatation. After the parturient's admission to the labour ward and her consent to study participation, the investigating anaesthesiologist followed her during the entire course of labour and delivery. An 18-gauge intravenous cannula and a 20-gauge radial arterial line were inserted after topical local anaesthesia. The LiDCO*plus* monitor was connected for continuous haemodynamic monitoring.[11] This device is based on pulse power analysis, and employs two algorithms: a continuous arterial waveform analysis system (PulseCO) coupled to a single-point lithium indicator dilution calibration system (LiDCO). PulseCO continuously calculates beat-to-beat stroke volume (SV) by analysing the arterial blood pressure trace.[12] The algorithm used by the PulseCO system is based on the assumption that the net power change in a heartbeat is the balance between the input of a mass of blood (SV) and the loss of a mass of blood to the periphery during the beat. The employed autocorrelation is a time-based method, avoiding a frequency approach as used in Fourier analysis, hence limiting the effects of arterial damping. The LiDCO system measures absolute SV using a small single dose of lithium chloride, injected via a peripheral venous line. The resulting absolute SV value is used for calibration of the PulseCO. LiDCO*plus* has been described and used in several recent studies in obstetric patients,[10,13–18] and its accuracy has been shown to be comparable to pulmonary arterial catheter measurements in preeclamptic parturients.[19]

Calibration of CO monitoring was performed after the women had been lying supine with left lateral tilt for five minutes, and haemodynamic variables were identified as stable by the LiDCO*plus* device. Continuous haemodynamic measurements were performed during the entire course of labour and delivery, and only paused on parturient demand, for mobilisation or rest during the first stage of labour. Parturients were encouraged to keep their upper limb still and relaxed, also under bearing down effort during stage 2 and delivery, and postpartum. Cervical dilatation was re-examined regularly by the midwife, at intervals between once every 15 minutes and once per hour, depending on the parturient's clinical progress. Labour was divided into the following stages: early stage 1 (cervical dilatation 3–7 cm), late stage 1 (cervical dilatation 8–10 cm), stage 2 (cervical dilatation 10 cm and bearing down effort), delivery (last 3–10 bearing down efforts in stage 2 leading to parturition), and early postpartum stage (up to 15 minutes after delivery). Changes in stage of labour, cervical dilatation, posture, pain score between 0 and 10 on a numeric rating scale (NRS), administration and dose of intravenous fluids or medication, epidural analgesia, possible side effects of medication, and any other specific events during the course of delivery were continuously entered into the LiDCO*plus* and on a case report form at the time of their occurrence. Onset and duration of contractions, as continuously identified by electrical tocometry by a midwife, were recorded into the LiDCO*plus* by the investigating anaesthesiologist who was onsite during the entire course of labour.

An intravenous oxytocin infusion (0.1 I.U./mL) for augmentation of labour could be started and adjusted at any time during labour, depending on obstetric indications. Placement of an epidural catheter could be performed at any time during the first stage of labour, on parturient demand, or based on anticipation of a complicated delivery by the obstetrician. A 5 mL initial bolus of ropivacaine (1 mg/mL) with sufentanil (1 μg/mL) was given followed by infusion between 0–10 mL/h. No other pharmacological method of analgesia was used.

Immediately after delivery, the newborn was placed on the mother's chest. The midwife recorded Apgar scores at 1, 5, and 10 minutes. The parturient received an intramuscular bolus of 5 I.U. oxytocin between 5–10 min after delivery. The arterial line and epidural catheter were removed, and the study ended within half an hour after delivery, before transferring the parturient to the obstetric ward.

Statistical analysis

Outcome measures were CO, SV, heart rate (HR), systemic vascular resistance (SVR) and systolic arterial

pressure (SAP) at baseline (periods between/without contractions) and during contractions, in the first and second labour stages, during delivery, and postpartum. The study was performed in a convenience sample of 20 parturients.

Longitudinally measured haemodynamic data were stored in the LiDCO*plus* monitor and downloaded both as visual (.lvu) files and as comma-separated values (.csv) text files for each parturient. Construction of the final dataset was performed using Excel version 14.3.2 (Microsoft Corp, Redmond, WA, USA). Outliers and artefacts were excluded from the individual datasets by the following criteria: all data identified as corrupted records ('bad status') by the LiDCO*plus*, all CO values <2 or >20 L/min, all HR values >200 beats/min, all SVR values corresponding to systemic vascular resistance index (SVRI) values >6000 dyne.s/cm^5, and all SAP values <60 mmHg. In a second round of data cleansing, artefacts defined as single-point values with sharp deviation from the adjacent beat-to-beat values were removed. From these cleansed datasets, only measurements taken with the parturient in the supine position with left lateral tilt were included for haemodynamic analysis of stage 1; measurements performed in the dorsal lithotomy position were included for analysis of stage 2. For each haemodynamic variable, repeated individual baseline values from each stage, and repeated individual minima/maxima during contractions from each stage were considered. For the extraction of these individual values, the following definitions were made: each individual baseline value should represent the mean value from one 30-s period without contraction, maternal activities or medical procedures, and without obvious noise in haemodynamic traces; each individual minimum/maximum value should represent the absolute minimum/maximum during one contraction, in the absence of other activities, procedures, or noise. All individual baseline, minimum and maximum values meeting these definition criteria, were included in the final dataset. This final dataset was then entered into statistical software.

Each haemodynamic variable was analysed separately within each stratum (baseline, minimum, maximum). Changes across stages were assessed with marginal linear models that account for dependency between repeated measures within subjects.[20] The model assessed haemodynamic variables as dependent factors, and stage, parity, epidural analgesia, and oxytocin infusion as factors. Epidural and oxytocin infusion were considered as binary factors (yes/no), and could vary within parturients during the course of labour. No effect of multicollinearity was found between the included factors. Standard errors were estimated by the robust variance estimator to allow for within-subject correlation. Mean values of the outcomes by stage were adjusted for included factors, and presented with 95% confidence intervals (CI). Sensitivity analyses were performed by running the same marginal linear models without adjusting for any factors other than stage. Haemodynamic data were analysed using Stata (StataCorp. 2015. Stata Statistical Software: Release 14, College Station, TX, USA).

Descriptive statistics are provided as frequencies and proportions for categorical variables, and as mean and standard deviation (SD) or as median and range as appropriate for continuous variables. Determination of normality was based on visual assessment of normality plots (histogram and q-q) or, when in doubt, on the Kolmogorov–Smirnov test. Comparisons were performed using Pearson chi-squared test for proportions, and using independent samples t-test or the Kruskal–Wallis test for continuous data. Descriptive data were analysed using IBM SPSS Statistics for Windows, Version 22.0 (IBM Corp Armonk, NY, USA). A P value <0.05 was considered statistically significant.

Results

Twenty healthy parturients were included in the study (Table 1). Continuous haemodynamic measurements from the first stage of labour until the early postpartum period were obtained in 19 parturients. For one parturient who delivered by caesarean section, only stage 1 measurements are included. The final dataset for all 20 parturients consists of 311 130 haemodynamic beat-by-beat measurements in total, 62 226 measurements per haemodynamic variable. These single measurements were transformed into 1366 summary measures per haemodynamic variable, consisting of 356 baseline measures (mean 17.8 measures per parturient), 505 contraction minimum and 505 contraction maximum measures (mean 25.3 measures per parturient).

Baseline values were similar across early and late stage 1, stage 2, and postpartum, for all haemodynamic variables (Tables 2 and 3). During stage-1 contractions, all haemodynamic variables changed significantly compared with baseline values (representative traces Figs. 1–3). For CO, SV and SVR, the direction of change was different between parturients: CO increased in most parturients (representative trace Fig. 2), but decreased in some (representative trace Fig. 1). Stroke volume decreased and SVR increased in most parturients (representative trace Fig. 1). Heart rate and SAP increased in all parturients.

The primary change in haemodynamic variables was often followed by a short change in the opposite direction before values returned to baseline. Minimum and maximum values for each variable are shown in Table 2. In late stage 1, minimum SV was lower and maximum HR and CO were higher than in early stage 1 (Table 3). During the peak of stage-2 contractions, a decrease in CO and SV, and an increase in HR, SVR and SAP were

Table 1 Maternal and neonatal characteristics

	All (n=20)	Nulliparous (n=11)	Parous (n=9)
Age (years)	29 ± 5	28 ± 4	30 ± 6
Height (m)	1.67 ± 0.06	1.66 ± 0.07	1.69 ± 0.05
Weight (kg)	76 ± 8	74 ± 9	79 ± 7
Body mass index (kg/m^2)	27 ± 3	27 ± 3	28 ± 4
Time with continuous monitoring (h)	6.2 ± 4.1	8.3 ± 4.3	3.7 ± 1.9
Cervical dilatation at inclusion (cm)	5 ± 2	5 ± 3	5 ± 2
Oxytocin infusion	14 (70%)	8 (73%)	6 (67%)
Epidural analgesia	12 (60%)	9 (82%)	3 (33%)
Vacuum assisted delivery	4 (20%)	3 (27%)	1 (11%)
Caesarean section	1 (5%)	1 (9%)	0 (0%)
Gestational age (weeks)	40 ± 1	40 ± 1	40 ± 1
Birthweight (g)	3591 ± 463	3595 ± 544	3587 ± 374
Apgar score 1 min	9 [6–10]	9 [6–10]	10 [8–10]
Apgar score 5 min	10 [9–10]	10 [9–10]	10 [10]

Data are as mean ± SD, median [range], or number of patients (%). No values were missing. Group comparisons of nulliparous versus parous by Pearson chi-squared test (patient numbers), Kruskal–Wallis test (Apgar values) or independent samples t-test (all other variables) did not reveal any significant differences (significance level=0.05; no *P*-values displayed).

found in all parturients. Comparing mean minimum/maximum values with stage 2 baselines (Table 2), CO decreased by 32%, SV decreased by 44%, HR increased by 52%, SVR increased by 88% and SAP increased by 36%. In stage 2, minimum CO and SV were lower and maximum HR, SVR, and SAP were higher than in early and late stage 1 (Tables 2 and 3). During delivery, we found similar haemodynamic changes as during stage 2. Typical haemodynamic changes are illustrated by extracts from representative raw data traces (Figs. 1–4).

The effects of parity, epidural analgesia and oxytocin infusion, both on baseline values and on values during contractions, were minimal and non-significant for all haemodynamic variables. Sensitivity analyses showed similar haemodynamic values with slightly more pronounced mean minima/maxima (data not shown) compared with the adjusted analyses. In parturients who had epidural analgesia, successful placement of the catheter was confirmed by a reduction of pain score during contractions (mean decrease by 3.6 points on a numerical rating scale, $P < 0.001$).

Discussion

This observational study demonstrated profound haemodynamic changes during contractions in active labour and vaginal delivery in healthy parturients.

The influence of advancing labour on haemodynamic baseline values has been controversial. While some authors[2,3,6,7] observed a cumulative increase in resting CO up to 50% during labour stages 1 and 2, others reported no change.[4,5,8] These differences might be due partly to the use of non-continuous or unreliable monitoring techniques.[2] In our parturients, baseline values across stages were similar for all haemodynamic variables, suggesting that progression of labour has no major effect on haemodynamic baseline values.

Literature on haemodynamic changes during contractions in labour stage 1 is inconsistent. While HR[2,5,9] and SAP[5–8] increased in most previous studies, and in all of our parturients, there has been controversy with regards to SV and CO. Increases of 20–30% in CO[3,4,7,8] and increases in SV[2,6,8] have been reported by most previous authors, whereas both increases and decreases in CO and SV have been observed by others.[5] The reliability of CO results in the early studies has been considered limited, as measurements were performed intermittently, and with monitoring techniques that are not reliable to detect rapid haemodynamic changes.[2,5] However, continuous invasive measurements with the LiDCO*plus* technique in our study suggest that CO may both increase (Figs. 2 and 3) or decrease (Fig. 1) during stage-1 contractions, depending on the relative extent of changes in HR and SV. Decreases in SV might be caused by the concomitant increases in afterload in many of our parturients (Fig. 1). Because we found parallel decreases in SV and afterload in a few cases, we assume that decreased filling time due to tachycardia or decreased venous return due to caval compression might also play a role.[21] The range of haemodynamic changes during labour is caused by complex interactions of mechanical, neurophysiological, and endocrine factors,[5] and owing to the lack of scientific evidence, interpretation of the physiological mechanisms behind our observations is difficult. Larger studies with synchronised continuous haemodynamic and tocodynamic monitoring might improve understanding of haemodynamics during labour.

Our observations during stage 1 illustrate that contractions induce considerable haemodynamic changes in early active labour, and suggest that the extent of

Table 2 Haemodynamic variables at baseline and during contractions in different labour stages

	Early stage 1 (n_{bl}=83; n_c=125)		Late stage 1 (n_{bl}=90; n_c=144)		Stage 2 (n_{bl}=80; n_c=154)		Delivery (n_c=82)		Postpartum (n_{bl}=103)	
	Mean	95% CI	Mean	95% CI	Mean	95% CI	Mean	95% CI	Mean	95% CI
Cardiac output (L/min)										
Baseline	6.3	(5.7 to 6.9)	6.5	(6.1 to 6.9)	6.6	(6.1 to 7.1)			6.3	(5.6 to 7.0)
During contractions, Minimum	6.0	(5.3 to 6.7)	5.8	(5.4 to 6.2)	4.5	(4.0 to 5.1)	4.6	(4.1 to 5.1)		
During contractions, Maximum	9.2	(7.9 to 10.4)	9.9	(9.3 to 10.6)	10.6	(10.1 to 11.2)	11.1	(10.3 to 11.8)		
Stroke volume (mL)										
Baseline	77	(70 to 84)	79	(75 to 82)	77	(73 to 81)			75	(70 to 79)
During contractions, Minimum	72	(64 to 80)	65	(51 to 71)	43	(35 to 51)	39	(31 to 48)		
During contractions, Maximum	104	(93 to 116)	108	(103 to 113)	101	(96 to 106)	96	(89 to 102)		
Heart rate (beats/min)										
Baseline	82	(78 to 85)	83	(79 to 86)	86	(82 to 90)			86	(79 to 92)
During contractions, Minimum	67	(61 to 72)	67	(63 to 72)	70	(65 to 76)	79	(73 to 84)		
During contractions, Maximum	102	(95 to 110)	115	(104 to 126)	131	(124 to 137)	141	(134 to 149)		
Systemic vascular resistance (dyne.s/cm^5)										
Baseline	1139	(971 to 1307)	1139	(1075 to 1203)	1124	(1036 to 1212)			1105	(986 to 1224)
During contractions, Minimum	854	(708 to 1001)	759	(689 to 828)	736	(669 to 803)	680	(612 to 747)		
During contractions, Maximum	1265	(1043 to 1488)	1417	(1258 to 1575)	2110	(1906 to 2313)	2126	(1887 to 2364)		
Systolic arterial pressure (mmHg)										
Baseline	134	(126 to 141)	136	(132 to 141)	136	(130 to 142)			129	(122 to 136)
During contractions, Minimum	130	(122 to 137)	124	(118 to 130)	118	(114 to 122)	116	(112 to 121)		
During contractions, Maximum	164	(154 to 174)	171	(164 to 178)	185	(179 to 191)	179	(172 to 187)		

Mean values are adjusted for parity, epidural analgesia and oxytocin infusion, and are based on marginal linear models which allowed for dependency between repeated measures within parturients. n=number of observations per haemodynamic variable. For baselines (bl), each observation represents the mean value from one individual 30-second period between contractions (stage 1 and 2) or without contractions (postpartum). For contractions (c), each observation represents the minimum/maximum during one individual contraction.

Table 3 Changes in haemodynamic baselines, minima, and maxima across stages

	Baseline			During contractions					
				Minimum			Maximum		
	Mean	95% CI	*P* value	Mean	95% CI	*P* value	Mean	95% CI	*P* value
Cardiac output (L/min)									
Early stage 1	ref			ref			ref		
Late stage 1	0.2	(−0.2 to 0.6)	0.27	−0.2	(−0.6 to 0.2)	0.35	0.8	(0.1 to 1.4)	0.02
Stage 2	0.3	(−0.2 to 0.8)	0.19	−1.4	(−1.9 to −0.9)	<0.001	1.5	(0.9 to 2.0)	<0.001
Delivery				−1.4	(−1.9 to −0–9)	<0.001	1.9	(1.1 to 2.6)	<0.001
Postpartum	0.0	(−0.7 to 0.7)	0.98						
Stroke volume (mL)									
Early stage 1	ref			ref			ref		
Late stage 1	1	(−2 to 5)	0.46	−7	(−13 to −1)	0.03	4	(−1 to 8)	0.16
Stage 2	−1	(−4 to 3)	0.75	−29	(−37 to −21)	<0.001	−3	(−8 to 2)	0.27
Delivery				−33	(−41 to −24)	<0.001	−8	(−15 to −2)	0.01
Postpartum	−3	(−7 to 2)	0.22						
Heart rate (beats/min)									
Early stage 1	ref			ref			ref		
Late stage 1	1	(−2 to 4)	0.56	1	(−4 to 5)	0.76	13	(2 to 24)	0.02
Stage 2	4	(0 to 9)	0.05	4	(−2 to 9)	0.16	28	(22 to 35)	<0.001
Delivery				12	(6 to 18)	<0.001	39	(32 to 46)	<0.001
Postpartum	4	(−3 to 11)	0.25						
SVR (dyne.s/cm^5)									
Early stage 1	ref			ref			ref		
Late stage 1	0	(−64 to 64)	0.99	−95	(−165 to −26)	0.007	151	(−7 to 310)	0.06
Stage 2	−14	(−103 to 73)	0.74	−119	(−186 to −52)	0.001	844	(641 to 1048)	<0.001
Delivery				−175	(−242 to −107)	<0.001	860	(621 to 1099)	<0.001
Postpartum	−33	(−152 to 85)	0.58						
SAP (mmHg)									
Early stage 1	ref			ref			ref		
Late stage 1	3	(−2 to 7)	0.24	−6	(−12 to 0)	0.04	7	(0 to 14)	0.06
Stage 2	2	(−4 to 9)	0.42	−12	(−15 to −8)	<0.001	21	(15 to 27)	<0.001
Delivery				−13	(−18 to −9)	<0.001	16	(8 to 23)	<0.001
Postpartum	−5	(−12 to 2)	0.16						

Mean values are changes from the mean value in the reference stage. Each haemodynamic variable was analysed separately within each stratum (baseline, minimum, maximum), with marginal linear models that allowed for dependency between repeated measures within parturients. The model assessed the haemodynamic variables as dependent factors, and stage, parity, epidural analgesia, and oxytocin as independent factors. SVR: systemic vascular resistance; SAP: systolic arterial pressure. CI: confidence intervals. *P* values <0.05 were considered statistically significant.

these changes increases slightly between early and late stage 1.

There are few studies on the haemodynamic impact of bearing down during labour stage 2.[3,5,7] They observed larger increases in HR and SAP than during stage-1 contractions,[5,7] but none achieved reliable CO measurements during bearing down.[1,2,22] Continuous invasive monitoring in our study revealed profound decreases from baseline in CO and SV, and marked increases from baseline in HR, SVR, and SAP during stage-2 contractions and delivery (Figs. 1–4). Our findings correspond to observations in a recent case report that was based on the same monitoring method.[1] Prominent decreases in CO and SV are probably partly due to reduced venous return, caused by increased intrathoracic pressure during expulsive effort.[3] However, we also found decreases in CO and SV during stage-1 contractions in some cases, in the absence of bearing-down (Fig. 1).

Comparison of haemodynamic changes during contractions in stage 2 versus stage 1 demonstrates that haemodynamic stress is more substantial during stage 2.

Increasing HR, SVR, and SAP during contractions have mainly been attributed to anxiety, stress and pain.[23] In a previous study, similar norepinephrine levels were found in parturients with and without epidural analgesia.[24] In another study, synchronised monitoring of blood pressure and uterine activity revealed increases in SAP before parturients' awareness of their contractions.[5] In a recent case report, significant haemodynamic changes were observed in a calm parturient with epidural analgesia and little pain.[1] These findings suggest that maternal haemodynamic changes during contractions occur, in part, independently of pain or subjective stress.

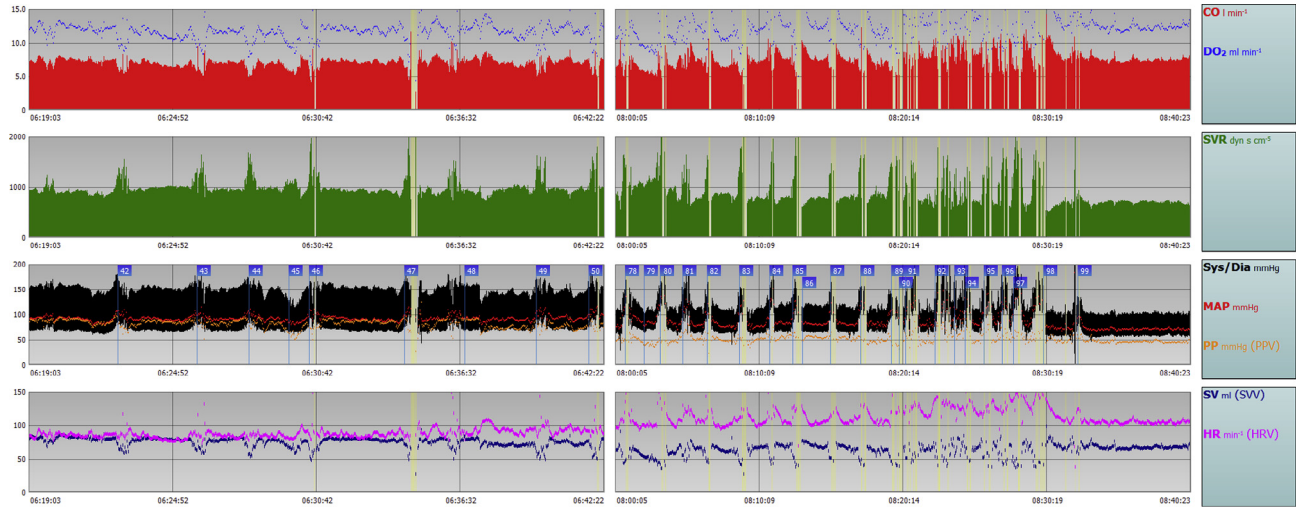

Fig. 1 Haemodynamic changes during the course of labour, representative trace 1: Extract from late stage 1 (flags 42–50), stage 2 (flags 78–87) with delivery (flags 88–99), and early postpartum stage in one of the parturients

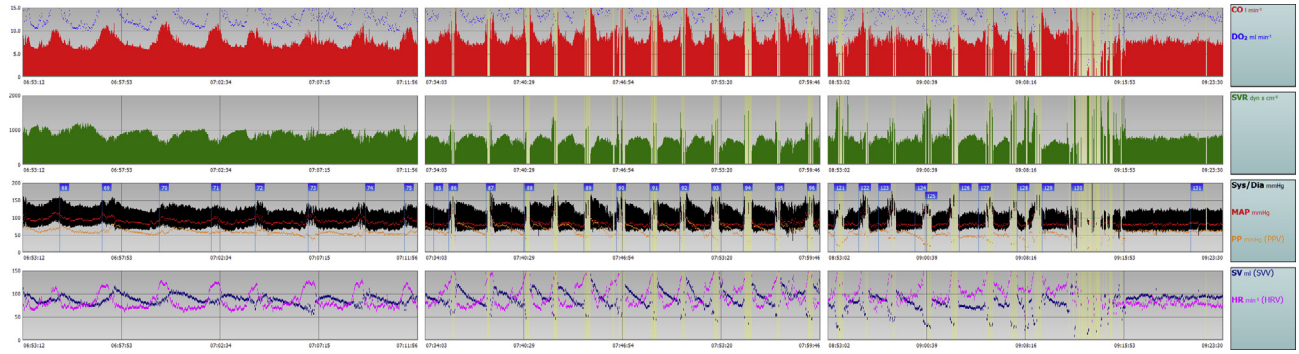

Fig. 2 Haemodynamic changes during the course of labour, representative trace 2: Extract from late stage 1 (flags 68–75), stage 2 (flags 85–96 and 121–124) with delivery (flags 125–130), and early postpartum stage in one of the parturients

In light of small effect estimates for epidural analgesia in marginal linear model analyses, and of pronounced haemodynamic changes during contractions in parturients with epidural analgesia in our study, we hypothesise that epidural analgesia does not eliminate the effect of contractions on maternal circulation. Our study was not designed for comparison of parturients with and without epidural analgesia, and further research on this topic is needed.

Immediately after delivery, all haemodynamic variables returned to values that were similar to baseline values in stage 1 and 2 (Figs. 1–4). We observed no significant increase in SV and CO directly postpartum, and therefore question the common belief[25–27] that autotransfusion due to uterine contraction after delivery results in a major immediate increase in venous return, SV and CO. This theory has originated from publications between the 1950s and 1970s that reported an important increase (of up to 80%) in resting CO during the first few minutes after delivery,[3,7,9,28] but has been doubted by other authors who found increased[8] or unchanged postpartum CO.[2,4,5] The theory of an immediate autotransfusion after delivery has also been challenged by a recent publication which compared haemodynamic changes among caesarean section patients receiving a postpartum bolus of either oxytocin, carbetocin or placebo. Stroke volume increased in the two former groups, but was stable in the placebo group.[29] Nonetheless, current literature relies on the concept that uterine autotransfusion, and even rapid mobilisation of extracellular fluid, create an important postpartum increase in venous return and SV.[30] Critics of the theory hypothesise that the increase in CO in respective older studies might be a side effect of an intravenously administered oxytocin bolus postpartum.[10] In our study, oxytocin was administered intramuscularly a few minutes after delivery, and was therefore unlikely to exert any haemodynamic effect within the monitoring period.

Ethical consent to this study was given on the premise that study inclusion would not alter general obstetric routines and decisions during the course of labour. As

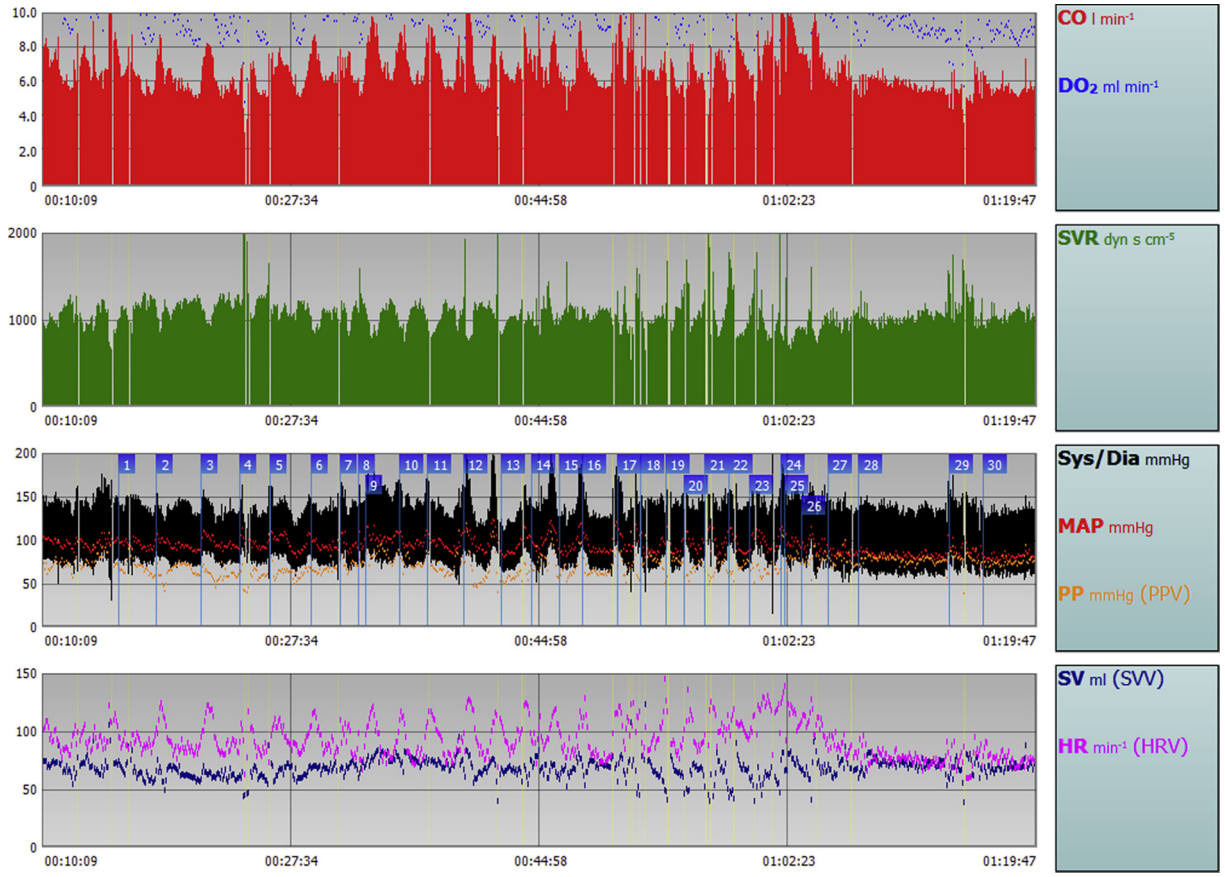

Fig. 3 Haemodynamic changes during the course of labour, representative trace 3: Extract from late stage 1 (flags 1–11), stage 2 (flags 12–21) with delivery (flags 22–26), and early postpartum stage in one of the parturients

the study was performed in parturients presenting for spontaneous delivery, heterogeneity with regards to cervical dilatation at the time of inclusion, duration of labour, mobilisation, fluid and food intake, eventual oxytocin infusion or epidural analgesia could not be eliminated.

Parturients were included after labour had started, and consequently there are no haemodynamic data before labour. The study size is limited with regards to the number of participants. However, both recruitment for, and successful completion of continuous invasive monitoring in parturients during the entire course of labour are challenging. Including 124 hours of continuously monitored haemodynamic data, this database is to our knowledge the largest such in healthy labouring parturients so far.

Our study was not designed to quantify the influence of epidural analgesia on haemodynamic changes. Valid detection of minor haemodynamic differences between parturients with and without epidural analgesia would require a large randomised controlled trial, but in addition to challenging practical aspects, this might be complicated from ethical and clinical perspectives, and lead to high dropout rates from protocol violations.

Our data do not include tocometry records, so haemodynamic measurements are not synchronised with the exact extent of uterine activity. Onset and duration of contractions were identified by electrical tocometry through a midwife, and recorded manually into the LiDCO*plus* by the investigating anaesthesiologist who was on-site during the entire course of labour. Because of possible minor drift in terms of accurate interpretation of absolute SV, recalibration of the LiDCO device every 8–12 h is recommended. In participants with a longer duration of labour, measured SV might thus differ slightly from the true absolute values. Since drift is unidirectional, this would not affect the extent of haemodynamic changes during contractions.

Because of parturient movement during painful labour, a considerable number of outliers and artefacts had to be removed. In some parturients, arterial curves and LiDCO traces were compromised at the time of peak effort during several stage 2 and delivery contractions, even though the arterial cannula was patent and the parturients' upper limb relaxed. As the same observation was made in a few individual contractions when no voluntary Valsalva effort was performed, we assume that disruptions were partly caused by intense vasocon-

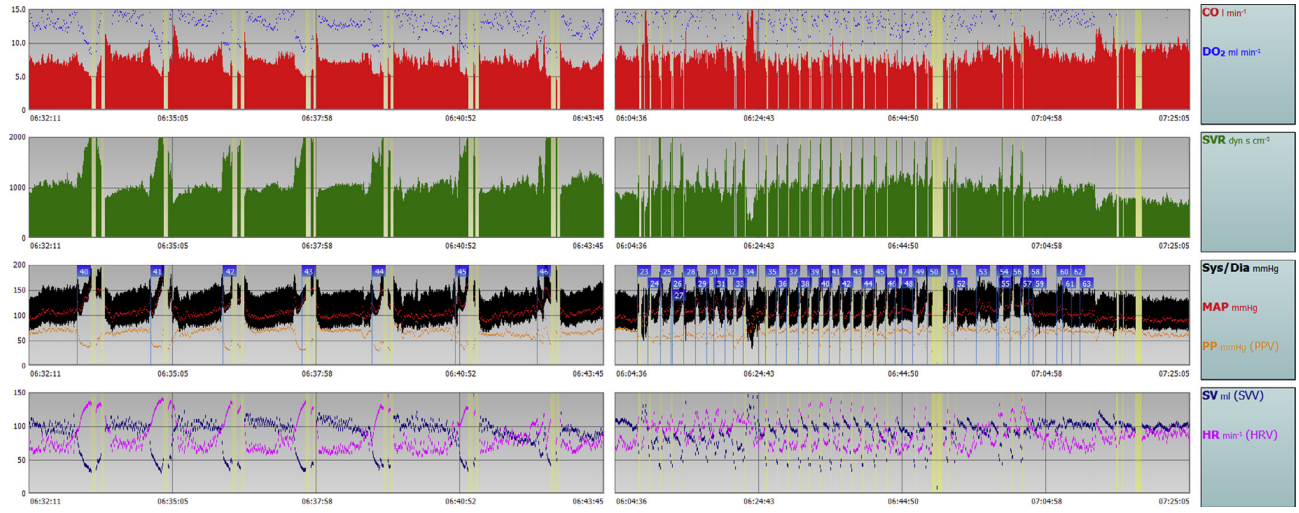

Fig. 4 Haemodynamic changes during the course of labour, representative trace 4: Extract from stage 2 (flags 23–47) with delivery (flags 48–59), and early postpartum stage in one of the parturients, in the right panel of the figure. In the left panel of the figure, flags 40–46 from the right panel are displayed in enlarged view

striction. Corrupted records were not included in the final dataset.

This study revealed profound haemodynamic changes during contractions both in early and late active labour. Maternal haemodynamic stress was most substantial when bearing down in stage 2, with pronounced decreases in SV and CO, and marked increases in HR, SVR and SAP. Baseline values were similar across labour stages in all haemodynamic variables. Absence of an increase in SV and CO during the first 15 minutes after delivery in our study questions the common belief[25–27,30] that delivery itself leads to a significant postpartum autotransfusion. Further research is required, to improve insight into maternal haemodynamics during vaginal delivery.

Disclosure

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