


RESEARCH ARTICLE

Intrapartum Care

Impact of obesity on uterine contractile activity during labour: A blinded analysis of a randomised controlled trial cohort

Tuija Hautakangas^{1,2}  | Jukka Uotila³ | Joel Kontiainen² | Heini Huhtala⁴ |
Outi Palomäki³

¹Department of Obstetrics and Gynaecology, Central Finland Health Care District, Tampere, Finland

²Faculty of Medicine and Health Technology, Tampere University, Tampere, Finland

³Department of Obstetrics and Gynaecology, Tampere University Hospital and Faculty of Medicine and Health Technology, Tampere University, Tampere, Finland

⁴Faculty of Social Sciences, Tampere University, Tampere, Finland

Correspondence

Tuija Hautakangas, Department of Obstetrics and Gynaecology, Central Finland Health Care District, Hoitajantie 3, 40620 Jyväskylä, Finland.
Email: tuija.hautakangas@ksshp.fi

Funding information

Financial support was received from Special Grant funding provided by the Government of Finland and the Finnish Medical Foundation. Funding sources have not been involved in the collection, analysis or interpretation of data or writing of the manuscript.

Abstract

Objective: To investigate the impact of severe obesity (body mass index [BMI] ≥ 35 kg/m²) on uterine contractile activity. The hypothesis was that obese parturients might have weaker uterine activity and need more oxytocin than leaner parturients.

Design: Exploratory, blinded analysis of a randomised controlled trial cohort.

Setting: Two labour wards, one in a university tertiary hospital and one in a central hospital.

Population: In all, 686 parturients with singleton pregnancies, gestational age ≥ 37 weeks, fetus in cephalic presentation, and intrauterine tocodynamometry during labour. [Correction added on 6 June 2022, after first online publication: the number of parturients has been corrected to 686.]

Methods: Uterine contractile activity was assessed as intrauterine pressure, frequency of contractions and basal tone of uterine muscle. The use of oxytocin and cervical dilatation were recorded simultaneously.

Main outcome measures: Primary outcome: uterine contractile activity. Secondary outcomes: use of oxytocin, labour outcomes.

Results: Obese parturients reached intrauterine pressure ≥ 200 Montevideo units (MVU) during the first stage of labour more often than leaner parturients; 62% vs 49%; odds ratio [OR] 1.67 (95% CI 1.05–2.67) and had higher basal tone of uterine muscle. However, obese parturients without previous vaginal delivery were not able to reach the active stage of labour as often as leaner ones, and their vaginal delivery success rate was lower. If a parturient had had previous vaginal delivery, obesity did not influence uterine activity, nor was there a risk of caesarean section. Doses and total consumption of oxytocin did not differ between BMI groups.

Conclusions: Obese nulliparas have stronger uterine contractile activity than leaner ones, but they more often fail to reach the active phase of labour and their vaginal delivery success rate is lower.

KEYWORDS

body mass index, caesarean section, contraction, dystocia induction of labour, intrauterine pressure, intrauterine pressure catheter, mode of delivery, Montevideo unit, obesity, oxytocin

Linked article: This article is commented on by Mikko Tarvonen, pp. 1798 in this issue. To view this minicommentary visit <https://doi.org/10.1111/1471-0528.17153>.

This is an open access article under the terms of the [Creative Commons Attribution-NonCommercial](https://creativecommons.org/licenses/by-nc/4.0/) License, which permits use, distribution and reproduction in any medium, provided the original work is properly cited and is not used for commercial purposes.

© 2022 The Authors. BJOG: An International Journal of Obstetrics and Gynaecology published by John Wiley & Sons Ltd.

Tweetable abstract: Despite higher strength of uterine contractions among obese parturients, they reach the active stage of labour less often than leaner ones.

1 | INTRODUCTION

Obesity is associated with slow progression of labour and an increased risk of unplanned caesarean section (CS).^{1–5} High maternal body mass is associated with admission to hospital at an earlier stage of cervical dilatation, prolonged duration of labour and risk of arrested labour.^{1,6–12} Some studies have shown more need for oxytocin during labour among obese parturients,^{11,13} but there are also results showing no difference.^{4,14} The reasons for failure in labour progress among obese parturients are unclear; mechanical occlusion by excess fat in the maternal pelvis, a macrosomic fetus and inefficient uterine contractions have been suggested to be some explanations.^{12,13} As poor or uncoordinated contractions have been considered as plausible causes of dystocia, contractions of obese parturients have been studied in *in vitro* experiments^{15–17} and also in clinical studies.^{4,13,18} Hormonal and metabolic imbalances of obese women—such as hyperlipidaemia and high levels of leptin—and their effects on Ca²⁺ signalling in the myometrium have been found to decrease contractility of uterine muscle in *in vitro* studies^{15–17} and have been suggested as explanations for dystocia.^{19–22} The effect of obesity on hormonal balance and onset of labour and contractility activity^{19–22} is shown in a simplified form in Figure S1.

Uterine contractile activity can be assessed as intrauterine pressure (IUP), reflecting the power of contractions during labour, the frequency of contractions and basal tonus of the uterus. Guidelines on use of an IUP catheter (IUPC) during augmented labours²³ were drawn up on the basis of the results of fairly small study,²⁴ and according to those results, IUP should exceed 200 Montevideo units (MVU) during labour to achieve vaginal delivery (VD). There are only a few studies on uterine contractile activity during labour of obese parturients and the results are contradictory. In cohort and registry studies, the progress of labour and uterine contractility of obese parturients has been found to be ineffective, but these conclusions were not based on measurements of contractions.^{2,17} However, Chin et al. and Nuthalapaty et al. have shown that obese parturients are as able to reach IUP levels of ≥ 200 MVU as lean parturients, but despite that they have slower progress of labour and an increased risk of unplanned CS.^{4,13} In a study by Buhimschi et al.,¹⁸ the duration and contractile power of the second stage of labour did not differ between obese and leaner parturients.

The aim of this study was to discover whether the contractions of obese parturients are less powerful and whether they need more oxytocin than leaner parturients to achieve VD. We carried out analysis for 4 h before parturition to see how contractility changes towards the end of labour.

2 | METHODS

We carried out an exploratory analysis of a prospective cohort from our randomised controlled trial (RCT) in which we compared intrauterine and external tocodynamometries with respect to labour outcomes. The entire RCT study protocol and methods have been published.²⁵ The RCT was conducted in 2012–2017, and we recruited a total of 1504 parturients—originally 736 in intrauterine and 768 in external groups. To be able to study the power of contractions properly, only women with intrauterine tocograms were included into the present study. Of the former RCT study material, 686 intrauterine tocograms of sufficient duration (≥ 1 hour before birth) and interpretable quality were selected for analysis. Two investigators interpreted the tocograms retrospectively without knowing the basic characteristics of the parturient or neonate or the outcome of labour.

Basic data on parturients and newborns were collected from medical records and material from the original RCT.²⁵ The study parturients were either nulliparas, parous women with oxytocin augmentation in the first stage of labour or parturients who underwent trial of labour after CS. Parturients with singleton pregnancies, gestational age ≥ 37 weeks with the fetus in cephalic position and cervical dilatation of ≤ 7 cm were recruited. Parturients with an estimated fetal birthweight of >4500 g, signs of intrauterine infection or fetal distress at recruitment, or positive results in serological tests for human immunodeficiency virus or hepatitis B or C virus, were not eligible.

As our goal was to analyse uterine contractile activity among parturients with clinically significant obesity, we first carried out analysis of basic labour outcomes and delivery modes within WHO BMI classes, presented in Table S1. A BMI of ≥ 35 kg/m², indicating severe obesity (WHO classes II and III), was found to be a critical cut-off point, beyond which the proportion of cases of unplanned CS rose significantly. That cut-off point was chosen for more detailed analyses—and parturients with a BMI of ≥ 35 kg/m² will be called obese in this publication.

In the RCT protocol,²⁵ oxytocin was administered by midwives in increasing doses, i.e. addition of 2.0–2.5 IU/min every 20–30 minutes until 150–300 MVU had been reached or the progression of labour was adequate. The basal tonus of uterine muscle had to be under 20 mmHg and relaxation of the uterus had to be seen between contractions. A sensor-tipped catheter was used (Koala, Clinical Innovations) and it was inserted by a doctor during the first vaginal examination after randomisation. Monitoring of contractions was carried out by using Philips Avalon FM30 or FM50 Gemini equipment (Koninklijke Philips N.V., Amsterdam, the Netherlands).

We analysed uterine contractile activity as IUP (MVU), the frequency of contractions/10 min and basal tonus of the uterine muscle (mmHg) in 30-min periods, i.e. 15 min before and after exact times, 4, 3, 2 and 1 h before birth or a decision to carry out CS. We chose this method of 30-minute periods to minimise the fluctuations due to contractions falling out of the measurement period. The doses of oxytocin (IU/min) and dilatation of the cervix (cm) were recorded similarly. The definition of an MVU is the sum of contraction amplitudes as mmHg above basal uterine tonus for a 10-min period. For the analyses, the parturients were divided into subclasses according to WHO BMI classification.²⁶

2.1 | Outcome measures

The primary outcomes were uterine contractile activity measured as IUP, frequency of contractions and basal tonus of the uterine muscle, and the impact of BMI on these variables. Secondary outcomes were cervical dilatation and oxytocin administration, the rates of different delivery modes and composite adverse neonatal outcome defined as umbilical artery pH < 7.05, Apgar scores < 7 after 5 min or admission to a Neonatal Intensive-Care Unit.

Core outcome sets (COSs) did not exist at the time of designing this study, and a relevant COS still does not exist, or is in development at the time of submission.

2.2 | Statistical analysis

Data were analysed in BMI subgroups and according to the presence of previous VD. All statistical analyses were performed using SPSS for Windows 26 software (IBM SPSS Statistics for Windows, Version 26.0; IBM Corp.). Continuous variables were expressed as means with standard deviations, or medians with quartiles, depending on the skewness of distribution, which was investigated by using the Shapiro–Wilk test. Differences between groups were tested by using appropriate tests, i.e. the Mann–Whitney U-test, Fisher's exact test, and the chi-square test. Repeated measures analysis of variance (ANOVA) was used to compare differences between BMI groups (Figure 1). A *p*-value of < 0.05 was considered statistically significant. All *P*-values were two-sided.

Ethics approval for the study was given by the Ethics Committee of Pirkanmaa Hospital District (R12229) in October 2012.

2.3 | Patient and public involvement

The staff of the labour and prenatal wards were informed of the RCT before beginning the study. The staff then had a chance to give advice and hints regarding the study and the data-collection form. Parturients were not involved in planning the study, and there was no questionnaire for them. The results of the study will be presented at annual meetings of

obstetricians, midwives and neonatologists, but not to study participants personally.

3 | RESULTS

Table 1 includes the characteristics of the study population divided using the BMI limit of 35 kg/m². Obese parturients more often had diabetes (OR 5.62, 95% CI 3.48–9.09) and more often needed induction of labour (OR 4.85, 95% CI 2.58–9.12), with prostaglandins as an induction method, compared with leaner parturients (OR 2.90, 95% CI 1.77–4.75). The median BMI among leaner parturients was 24.3 kg/m² (min–max 16.6–34.9) compared with 39.1 kg/m² (min–max 35.1–57.3) in the obese group.

Uterine contractile activity of all obese parturients, measured as basal tonus of the uterine muscle, was higher than among leaner parturients. Obese parturients were able to achieve IUPs of ≥ 200 MVU more often than leaner ones (OR 1.67, 95% CI 1.05–2.67) (Table 2). Among parturients achieving IUPs of ≥ 200 MVU, the CS rate among obese women without a previous VD was 37.3% and among leaner women without a previous VD 16.5% (OR 3.01, 95% CI 1.49–6.07; *p* = 0.002); if there had been a previous VD, obesity did not affect the delivery modes when using this IUP limit (*p* = 0.797). Among obese parturients without a previous VD, IUP was higher but there were no differences in the frequency of contractions, in cervical dilatation or doses of oxytocin. If VD was achieved, the progression of cervical dilatation, use of oxytocin, and IUP levels were almost identical in the two groups (Figure 1). If the labour ended in CS, obese parturients without a previous VD tended to have higher IUPs and they received more oxytocin.

We also compared uterine contractility according to vaginal parity. Uterine contractile activity was significantly higher among parturients without previous VD (IUP 200 MVU [median; IQR 157–25], basal tone 20 [IQR 20–30], frequency 5 [IQR 4–5]) than in parturients with previous VD (IUP 170 MVU [IQR 120–210], basal tone 20 [IQR 15–20], frequency 4 [IQR 4–5]) regardless of BMI (all *p*-values < 0.001), but the use of oxytocin (maximum dose 12 [range 8–18] versus 10 [range 8–15] mIU/min; *p* = 0.128, total consumption 3.0 [range 1.5–5.0] versus 3.0 [range 2.0–5.0] IU; *p* = 0.149) did not differ in relation to vaginal parity.

Associations between BMI and labour outcomes are presented in Tables 3, S1 and S2. The need of vacuum extraction because of dystocia or fetal distress did not differ in cases above and below the chosen BMI limit. Obese parturients did not achieve the active stage of labour, i.e. ≥ 6 cm dilatation of the cervix, as often as leaner parturients did (OR 2.36, 95% CI 2.39–4.37; *P* = 0.005). However, if they reached the active stage, their labour proceeded as fast as among the leaner parturients. The subgroup of obese parturients who had had a previous VD reached full cervical dilatation as often as leaner parturients did (92.4% versus 90.0%; OR 0.74, 95% CI 0.15–3.64; *P* = 0.660). Induction of labour did not influence IUP when compared

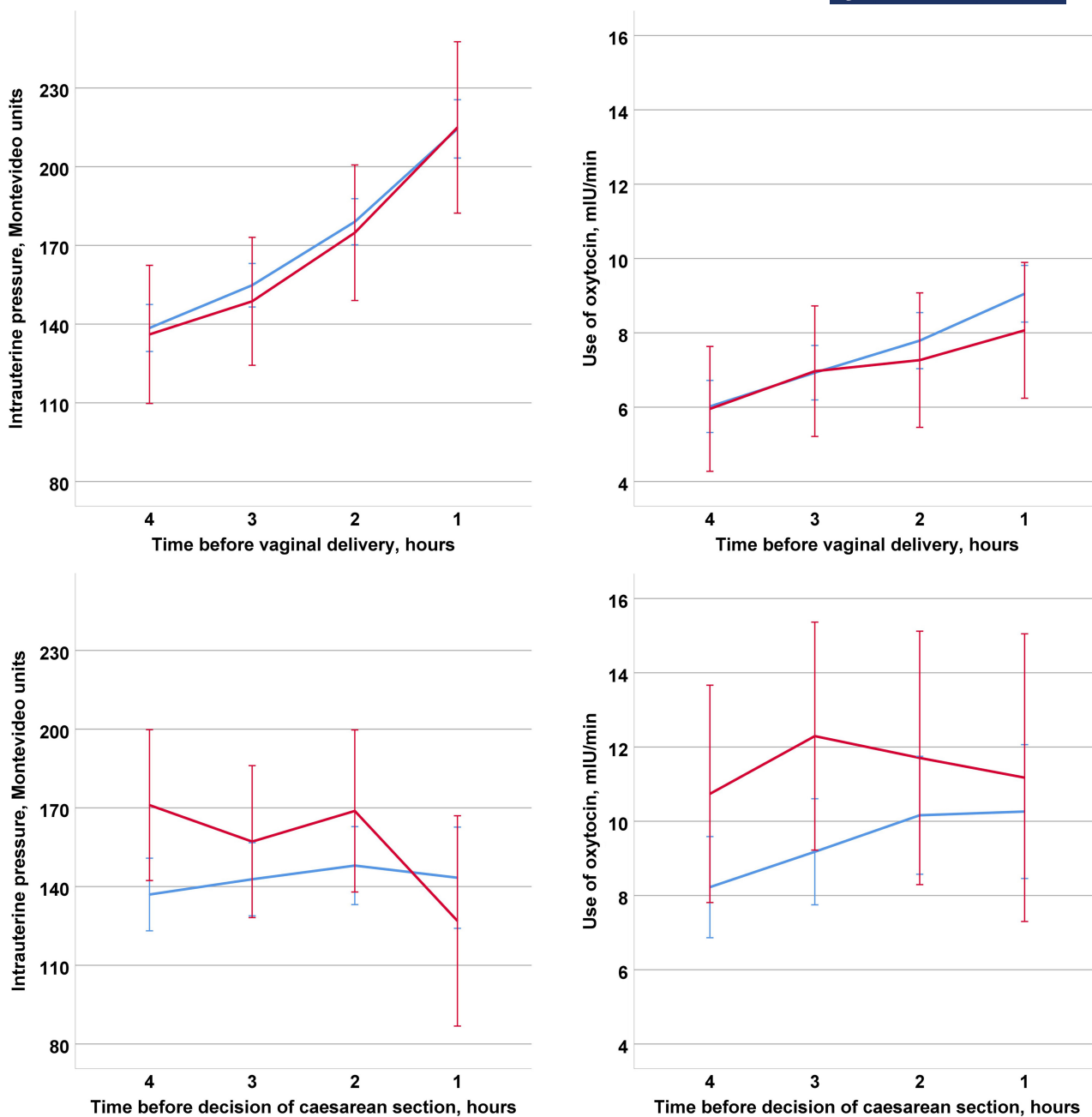


FIGURE 1 Intrauterine pressure and use of oxytocin at different time points during the last 4 hours before vaginal birth or before a decision on caesarean section, with a BMI limit of 35 kg/m²; blue line <35 kg/m², red line ≥35 kg/m²

with spontaneous onset of labour (Table 2; BMI <35 kg/m², $P = 0.390$, BMI ≥35 kg/m², $P = 0.711$).

Chorioamnionitis was more common among obese parturients (Table 3). Chorioamnionitis did not substantially impair contractile activity otherwise, but maximum frequency among parturients without previous VD was lower during infection (median 4.3 [IQR 4.0–5.0] vs. 5.0 [4.0–5.0], $p = 0.029$).

4 | DISCUSSION

Contrary to our hypothesis, obese parturients had even stronger contractions than leaner parturients, and a higher

proportion of them achieved IUPs of ≥200 MVU in our study. However, although obese parturients achieved IUPs of ≥200 MVU, the risk of unplanned CS was higher than among leaner parturients.

Only a few investigators have sought an association between uterine activity and obesity. Although our method of uterine contractility analysis in relation to BMI differs from those in past studies,^{4,13,18} because we recorded values in a particular time frame, not only the highest IUP reached during labour, the results of those earlier studies^{4,13,18} are mainly in line with our findings, showing that uterine contractile activity of obese parturients is not worse than that among leaner parturients.

TABLE 1 Characteristics of the study population, with BMI limit of 35 kg/m²

	BMI <35 kg/m ² n = 602		BMI ≥35 kg/m ² n = 84		P-value
	n/median	%/Q ₁ –Q ₃	n/median	%/Q ₁ –Q ₃	
At least one previous vaginal delivery	132	22	20	24	0.697
Maternal age (years)	30.0	26–33	29.0	24–33	0.135
Maternal height (metres)	1.65	1.62–1.70	1.65	0.06/1.60–1.70	0.810
Gestational age (days)	282	275–288	280	273–288	0.144
Pregestational or gestational diabetes	135	22.4	52	61.9	<0.001
Induction of labour ^a	333	55	72	86	<0.001
Prostaglandin E2 and/or Foley catheter	146	24	38	45	<0.001
Prostaglandin E2	101	17	31	37	<0.001
Foley catheter	75	12	15	18	0.170
Amniotomy and/or oxytocin	186	31	34	40	0.078
Cervical dilatation at recruitment	3.5	3.0–4.0	3.0	3.0–4.0	0.231

BMI, body mass index, kg/m²; SD, standard deviation.

^aMore than one method per patient was used when needed.

Despite obese parturients having as strong or even stronger contractions than non-obese parturients, they more often fail to achieve VD or reach the active stage of labour. This is probably attributable to the properties of cervical tissue and its ability to dilate in response to uterine activity, with possible explanations including elevated levels of leptin, disturbing the degradation of cervical collagen, and apoptosis^{19–22} (Figure S1). In addition, cervical tissue is connected to uterine muscle fibres and cervical status has an influence on tension of the uterine wall. With non-compliant and firm cervical tissue—which may be the case among obese nulliparas—tension of the uterine wall is high during contraction, resulting in higher IUP.²⁷ However, our results support the findings from some previous studies showing that if obese nulliparas can achieve the second stage of labour, the risk of second-stage CS is the same as in normal-weight nulliparas.^{1,28,29} However, there are also differing results.¹⁷ Altogether, contractility is only one factor in the complex system of parturition.

Obese parturients without a previous VD need more powerful contractions to reach the active stage of labour than leaner parturients do, but among women with previous VD this phenomenon does not exist. The force needed for cervical dilatation of obese nulliparas seems to be greater, but full dilatation in a previous delivery might influence cervical tissue, making it more elastic without a need for as powerful contractile activity as among nulliparas. One of the main findings of the present study concerning everyday work in labour wards was that if an obese parturient had had a previous VD, obesity did not influence uterine contractile activity or the risk of CS. Another clinically important finding was that when obese parturients reach the active stage of labour, labours can be as fluent as those of leaner parturients.

Our results on labour outcomes are affected by the fact that our cohort was selected. The most straightforward labours were not included in the study (exclusion criteria >7 cm dilatation at recruitment; parous women without

need of oxytocin augmentation during the first stage), and this might also explain our finding of no difference in CS rate with a dystocia indication in relation to BMI. Another explanation could be that a diagnosis of dystocia was not made until 6 cm dilatation was reached, so the obese parturients' difficulties to reach the active stage of labour were not included in these numbers; they were mainly in the 'failed induction of labour' category. Not only is reaching the active stage of labour more difficult among the obese, failed induction of labour is also common.

Our result indicating that induction of labour fails more often among obese parturients is supported by the results of a recent meta-analysis and systematic review.³⁰ Obese parturients often need induction of labour for several reasons such as pre-eclampsia, gestational diabetes or a macrosomic fetus. In our study cohort, the incidence of diabetes was high, especially among obese women, and so was the need for induction. The rate of inductions in our study cohort is overrepresented as a result of the inclusion criteria in the original RCT.²⁵

The obesity of a parturient may bias decision-making of physicians when considering CS for dystocia—the physician knows the risks of CS for obese parturients and therefore a decision on CS is not made lightly. On the other hand, CS of obese parturients may be carried out early with the intention to anticipate and avoid emergency and technical problems in anaesthesia and surgery. However, in our study, cervical dilatation at the time of deciding on CS (dystocia) was greater among obese parturients (6.3 vs. 5.5 cm, Table S2, NS).

As shown in previous studies^{5,31} as well as the present one, chorioamnionitis is more common among obese parturients. Chorioamnionitis did not impair contractility other than having an effect on the frequency of contractions. However, it may be impossible to know whether the infection is slowing down labour or is associated with prolonged labour.

TABLE 2 Uterine contractile activity and the use of oxytocin during the last 4 hours of labour, with BMI limit of 35 kg/m²

	BMI <35 kg/m ² n = 602		BMI ≥35 kg/m ² n = 84		P-value
	n/median	%/Q ₁ -Q ₃	n/median	%/ Q ₁ -Q ₃	
Intrauterine pressure, MVU					
≥200, n					
All parturients	297	49	52	61	0.031
No previous VD	249	53	43	67	0.032
Previous VD	48	36	9	45	0.457
Highest					
All parturients	190	144-240	222	155-263	0.120
Spontaneous onset of labour	200	150-250	194	125-278	0.997
Induction of labour	190	147-240	220	152-263	0.071
No previous VD	200	150-250	226	170-278	0.039
Previous VD	170	120-210	165	103-228	0.812
Spontaneous vaginal delivery	190	140-240	205	135-260	0.597
Vacuum extraction	225	170-288	230	218-343	0.226
Caesarean section	177	130-220	214	133-260	0.075
Frequency/10 min, maximum					
All parturients	4.7	4.0-5.0	5.0	4.0-5.0	0.432
Basal tonus of uterine muscle, mmHg					
Maximum					
All parturients	20	20-25	25	20-30	<0.001
No previous VD	20	20-30	25	20-30	0.001
Previous VD	20	15-20	20	20-25	0.033
Use of oxytocin					
Use during stage I, n	569	95	83	99	0.108
Dose >15 mIU/min during stage I, n	145	24	24	29	0.371
Total consumption, IU, all parturients	3.0	1.5-5.0	3.2	2.0-5.5	0.296
No previous VD	3.0	1.5-5.0	4.0	2.0-6.0	0.111
Maximum dose, mIU/min, stage I					
All parturients	10.0	5.0-15.0	10.0	5.0-15.0	0.098
No previous VD	10.0	5.0-10.0	11.3	5.0-17.5	0.051
Present delivery VD	7.5	4.0-12.5	7.5	5.0-12.5	0.549
Present delivery CS	12.5	5.0-17.5	12.5	9.5-20.4	0.126
Mean dose, mIU/min, stage I, all parturients	6.0	2.5-10.8	6.9	3.1-12.5	0.158
No previous VD	5.9	2.3-11.3	8.8	3.1-14.4	0.078
Present delivery VD	5.0	1.9-9.4	5.3	2.3-10.8	0.528
Present delivery CS	8.1	3.1-14.1	9.6	5.7-14.4	0.293

BMI, body mass index; CS, caesarean section; Q, quartile; MVU, Montevideo unit; VD, vaginal delivery.

4.1 | Strengths and limitations

The strength of our study is that interpretation of the tocodynamometry data was made by people, not a computer, so this reflects the real situation in most labour wards and makes interpretation realistic. Analyses were made blinded, i.e. the investigators did not know the basic characteristics of the parturients or neonates or the outcome of labour. To our

knowledge, this is only the second study, and the largest in cohort size (besides the study by Nuthalapaty et al.¹³) in which IUP has been assessed in connection with maternal weight and vaginal parity. As highlighted in a review concerning obesity and induction of labour,³⁰ the presence of previous VD has not been taken into account in many studies and, on the basis of our results, it would seem to be a very important factor as regards the success of trial of labour and induction of labour.

TABLE 3 BMI limit of 35 kg/m² and its association with labour outcomes

	BMI <35 kg/m ² n = 602		BMI ≥35 kg/m ² n = 84		OR	95% CI	P-value
	n/mean/ median	%/SD/Q ₁ -Q ₃	n/mean/ median	%/SD/Q ₁ -Q ₃			
Cervical dilatation 10 cm not reached	108	18	24	29	1.81	1.09–3.03	0.021
Active stage of labour not reached, i.e. cervical dilatation ≥6 cm ^a	55	10	16	20	2.36	1.28–4.37	0.005
Caesarean section	119	20	26	31	1.82	1.10–3.01	0.019
No previous VD	108	23	23	36	1.88	1.08–3.27	0.024
Previous VD	11	8	2	10	1.22	0.25–5.97	0.804
Indication of CS							
Dystocia, failed IOL excluded	66	11	10	12	1.10	0.54–2.23	0.797
Failed IOL	18	3	10	12	4.38	1.95–9.86	0.001
Fetal distress	38	6	7	8	1.35	0.58–3.13	0.483
Vacuum extraction	92	15	10	12	0.75	0.37–1.50	0.415
Chorioamnionitis ^b	68	11	18	21	2.14	1.20–3.82	0.009
Birthweight >4 kg	151	25	23	27	1.13	0.67–1.88	0.650
Composite adverse neonatal outcome	77	13	6	7	0.52	0.22–1.25	0.137

BMI, body mass index; CS, caesarean section; IOL, induction of labour; VD, vaginal delivery.

^a32 missing values, 28 (4.7%) in BMI group <35 kg/m² and 4 (4.8%) in BMI group ≥35 kg/m².

^bIntrapartum temperature more than 38.0°C or a combination of fetal tachycardia and maternal C-reactive protein more than 20 g/L.

There are a few limitations of our study. The population involved was selected. As the limit for recruitment was ≤7 cm dilatation of the cervix, and parous women without the need of oxytocin augmentation were not included, the most rapidly progressed labours were not recruited. These parturients are often lean and young, which may have biased our results concerning the duration of labour, CS rates and indications for operative deliveries. For the same reason, the incidence of obese women was higher (12.2%) than in our study hospitals generally (6.4%). However, the selection of our cohort did not influence the main outcome we focused on, i.e. contractility analysis.

4.2 | Interpretation

Uterine activity of obese parturients is not weaker than among leaner parturients. In particular, among parturients without previous VD, obesity worsens the success of induction of labour with an unfavourable cervix. We speculate that the response of uterine and pelvic tissues of obese nulliparas to powerful contractions is different from that of leaner parturients—and IUP is higher in the presence of firm cervical tissue of obese parturients without previous VD. When obese parturients achieve the active stage of labour, it is as fluent as leaner parturients' labours. The future challenge is to find an effective and safe method for induction of labour of obese nulliparas. However, in the complex system of parturition, contractility is only one factor.

5 | CONCLUSIONS

If there has been a previous VD, obesity does not influence uterine contractile activity or the risk of CS. The uterine contractile activity of obese parturients is equal to or even greater than that of leaner parturients, but obese parturients less often achieve vaginal delivery. However, if the active stage of labour is reached, obese parturients have labours that are as straightforward as those among leaner parturients.

ACKNOWLEDGEMENTS

We thank midwife and illustrator Kaisa-Maija Antila for her drawing and work with Figure S1. We thank all the women who participated in this trial, and both participating institutions and their staff for their contribution to this study, particularly midwives and doctors for their excellent work.

CONFLICT OF INTERESTS

All authors have completed the ICMJE uniform disclosure form at www.icmje.org/coi_disclosure.pdf. The authors report no conflicts of interest. There are no relationships or activities that could appear to have influenced the work.

AUTHOR CONTRIBUTION

TH contributed to the whole process: providing the initial idea for the study structure, planning the study and the material for it, recruitment of parturients of the original RCT, collection of data, analysis of data and reporting the work and results. JK contributed to the planning the study, collection of data, analysis of data and reporting phases of the study. HH contributed

to analysis of the data and reporting the work and results. JU contributed to the planning, conduction and reporting phases of the study. OP contributed to the planning, conduction and reporting phases of the study. The corresponding author attests that all listed authors meet authorship criteria and that no others meeting the criteria have been omitted.

ETHICAL APPROVAL

Ethics approval for the study was given by the ethics Committee of Pirkanmaa Hospital District (R12229), in October 2012.

DATA AVAILABILITY STATEMENT

The full dataset is available from the corresponding author on reasonable request.

ORCID

Tuija Hautakangas  <https://orcid.org/0000-0002-5124-6735>

REFERENCES

1. Carlhäll S, Källén K, Blomberg M. Maternal body mass index and duration of labor. *Eur J Obstet Gynecol Reprod Biol.* 2013;171:49–53.
2. Cedergren MI. Non-elective caesarean delivery due to ineffective uterine contractility or due to obstructed labour in relation to maternal body mass index. *Eur J Obstet Gynecol Reprod Biol.* 2009;145:163–6.
3. Knight M, Kurinczuk JJ, Spark P, Brocklehurst P. Extreme obesity in pregnancy in the United Kingdom. *Obstet Gynecol.* 2010;115:989–97.
4. Chin JR, Henry E, Holmgren CM, Varner MW, Branch DW. Maternal obesity and contraction strength in the first stage of labor. *Am J Obstet Gynecol.* 2012;207:129e1–6.
5. Hautakangas T, Palomäki O, Eidstø K, Huhtala H, Uotila J. Impact of obesity and other risk factors on labor dystocia in term primiparous women: a case control study. *BMC Pregnancy Childbirth.* 2018;18:304.
6. Kominiarek MA, Vanveldhuisen P, Hibbard J, Landy H, Haberman S, Learman L, et al. The maternal body mass index: a strong association with delivery route. *Am J Obstet Gynecol.* 2010;203:264.e1–7.
7. Zhang J, Troendle JF. Maternal prepregnancy overweight and obesity and the pattern of labor progression in term nulliparous women. *Obstet Gynecol.* 2004;104:943–51.
8. Denison FC, Price J, Graham C, Wild S, Liston WA. Maternal obesity, length of gestation, risk of postdates pregnancy and spontaneous onset of labour at term. *BJOG.* 2008;115:720–5.
9. Arrowsmith S, Wray S, Quenby S. Maternal obesity and labour complications following induction of labour in prolonged pregnancy. *BJOG.* 2011;118:578–88.
10. Ehrental DB, Jiang X, Strobino DM. Labor induction and the risk of a cesarean delivery among nulliparous women at term. *Obstet Gynecol.* 2010;116:35–42.
11. Soni S, Chivan N, Cohen WR. Effect of maternal body mass index on oxytocin treatment for arrest of dilatation. *J Perinat Med.* 2013;41:517–21.
12. Kominiarek MA, Zhang J, Vanveldhuisen P, Troendle J, Beaver J, Hibbard JU. Contemporary labor patterns: the impact of maternal body mass index. *Am J Obstet Gynecol.* 2011;205:244.e1–8.
13. Nuthalapaty FS, Rouse D, Owen J. The association of maternal weight with cesarean risk, labor duration, and cervical dilation rate during labor induction. *Obstet Gynecol.* 2004;103:452–6.
14. Ramö Isgren A, Kjölhede P, Carlhäll S, Blomberg M. Maternal body mass index and oxytocin in augmentation of labour in nulliparous women: a prospective observational study. *BMJ Open.* 2021;11:e044754.
15. Higgins CA, Martin W, Anderson L, Blanks AM, Norman JE, McConnachie A, et al. Maternal obesity and its relationship with spontaneous and oxytocin-induced contractility of human myometrium in vitro. *Reprod Sci.* 2010;17:177–85.
16. Zhang J, Kendrick A, Quenby S, Wray S. Contractility and calcium signaling of human myometrium are profoundly affected by cholesterol manipulation: implications for labor? *Reprod Sci.* 2007;14:456–66.
17. Zhang J, Bricker L, Wray S, Quenby S. Poor uterine contractility in obese women. *BJOG.* 2007;114:343–8.
18. Buhimschi CS, Buhimschi IA, Malinow AM, Weiner CP. Intrauterine pressure during the second stage of labor in obese women. *Obstet Gynecol.* 2004;103:225–30.
19. Carlson NS, Hernandez TL, Hurt KJ. Parturition dysfunction in obesity: time to target the pathobiology. *Reprod Biol Endocrinol.* 2015;13:135–49.
20. Lowe NK, Corwin EJ. Proposed biological linkages between obesity, stress, and inefficient uterine contractility during labor in humans. *Med Hypotheses.* 2011;76:755–60.
21. Arrowsmith S, Kendrick A, Hanley J, Noble K, Wray S. Myometrial physiology - time to translate? *Exp Physiol.* 2014;99:495–502.
22. Prendergast C. Maternal phenotype: how do age, obesity and diabetes affect myometrial function? *Curr Opin Physiol.* 2020;13:108–16.
23. ACOG. Dystocia and augmentation of labor. *ACOG practice bulletin no. 49.* *Obstet Gynecol.* 2003;102:1445–54.
24. Hauth JC, Hankins GDV, Gilstrap LC, Strickland DM, Vance P. Uterine contraction pressures with oxytocin induction/augmentation. *Obstet Gynecol.* 1986;68:305–9.
25. Hautakangas T, Uotila J, Huhtala H, Palomäki O. Intrauterine versus external tocodynamometry in monitoring labour: a randomised controlled clinical trial. *BJOG.* 2020;127:1677–86.
26. WHO. World Health Organization BMI classification [Internet] 2016. <http://www.euro.who.int/en/health-topics/disease-prevention/nutrition/a-healthy-lifestyle/body-mass-indexbmi>
27. Oláh KS, Steer P. The use and abuse of oxytocin. *Obstet Gynaecol.* 2015;17:265–71.
28. Fyfe E, Anderson NH, North RA, Chan EHY, Taylor RS, Dekker GA, et al. Risk of first-stage and second-stage cesarean delivery by maternal body mass index among nulliparous women in labor at term. *Obstet Gynecol.* 2011;117:1315–22.
29. Robinson BK, Mapp DC, Bloom SL, Rouse DJ, Spong CY, Varner MW, et al. Increasing maternal body mass index and characteristics of the second stage of labor. *Obs Gynecol.* 2011;118:1309–13.
30. Ellis JA, Brown CM, Barger B, Carlson NS. Influence of maternal obesity on labor induction: a systematic review and meta-analysis. *J Midwifery Women's Health.* 2019;64:55–67.
31. Hadley EE, Discacciati A, Costantine MM, Munn MB, Pacheco LD, Saade GR, et al. Maternal obesity is associated with chorioamnionitis and earlier indicated preterm delivery among expectantly managed women with preterm premature rupture of membranes. *J Matern Neonatal Med.* 2019;32:271–8.

SUPPORTING INFORMATION

Additional supporting information may be found in the online version of the article at the publisher's website.

How to cite this article: Hautakangas T, Uotila J, Kontiainen J, Huhtala H, Palomäki O. Impact of obesity on uterine contractile activity during labour: A blinded analysis of a randomised controlled trial cohort. *BJOG.* 2022;129:1790–1797. <https://doi.org/10.1111/1471-0528.17128>